Clinical Avian Medicine

VOLUME I

GREG J. HARRISON, DVM
Diplomate Emeritus American Board of Veterinary Practitioners (Avian)
Diplomate European College of Avian Medicine and Surgery (n.p.)
Palm Beach, Florida

TERESA L. LIGHTFOOT, BS, DVM
Diplomate American Board of Veterinary Practitioners (Avian)
Florida Veterinary Specialists
Tampa, Florida

Spix Publishing, Inc.,
Palm Beach, Florida

Library of Congress Control Number: 2005922214

Clinical Avian Medicine (2-volume set) ISBN: 00-9754994-0-8

Production: Zoological Education Network

Cover Design: Jean Coffinberry

Cover photos: Mimi Walling, We Shoot Birds (cockatiel, black-capped lory, double yellow-headed Amazon, African grey parrots, Quaker parrot, sun conure, budgerigar); Loro Parque, Tenerife, Spain (blue and gold macaws)

Proofreading: K. Stormy Hudelson, DVM, Dipl ABVP-Avian, Paul Hudelson

Indexing: Ann Truesdale, DVM

Administrative Assistants: Lesley Flahie, Patty Wolf

Technical Assistance: Chris Lane

Printing and Binding: Four Colour Imports

Advertising and Sales: HBD Intl Inc. www.harrisonsbirdfoods.com

©2006 Spix Publishing, Inc., Palm Beach, Florida
All rights reserved. No part of this book may be reproduced or used in any form or by any means, electronic or mechanical, including photocopying, or by any information storage or retrieval system, without permission in writing from the publisher. Address any inquiries to Spix Publishing Inc, 3610 South Ocean Blvd #601, Palm Beach, FL 33480-5877.

Last digit is the print number: 9 8 7 6 5 4 3 2 1

CONTRIBUTORS

HEATHER L. BOWLES, DVM, Diplomate American Board of Veterinary Practitioners (Avian), Certified in Veterinary Acupuncture (Chi Institute)
Hunt Valley Animal Hospital, Hunt Valley, Maryland
Evaluating and Treating the Reproductive System, Surgical Resolution of Soft Tissue Disorders

MARCELLUS BÜRKLE, DVM
Tierärztliche Praxis, Karlsruhe, Germany
Management of Zoo and Park Birds

GARY D. BUTCHER, BS, MS, DVM, PhD, Diplomate American College of Poultry Veterinarians
Poultry Medicine and Management, College of Veterinary Medicine,
University of Florida, Gainesville, Florida
Management of Galliformes

CARL D. CHENEY, PhD
Department of Psychology, Utah State University, Logan, Utah
Concepts in Behavior, Section I: The Natural Science of Behavior

PETER COUTTEEL, DVM
Dierenartsenpraktijk Trigenio, Nijlen, Belgium
Management of Canaries, Finches and Mynahs

LORENZO CROSTA, DVM
Avian, Exotic and Zoo Animal Consulting, Como, Italy
Management of Zoo and Park Birds

ROBERT D. DAHLHAUSEN, DVM, MS
Veterinary Molecular Diagnostics, Inc., Milford, Ohio
Implications of Mycoses in Clinical Disorders

BOB DONELEY, BVSc, FACVSc (Avian Health)
West Toowoomba Veterinary Surgery, Toowoomba, Queensland, Australia
Maximizing Information from the Physical Examination, Management of Captive Ratites

M. SCOTT ECHOLS, DVM, Diplomate American Board of Veterinary Practitioners (Avian)
Adjunct Professor, Texas A&M University College of Veterinary Medicine,
Westgate Pet and Bird Hospital, Austin, Texas
Evaluating and Treating the Kidneys

THOMAS M. EDLING, DVM, MSpVM
Director, Veterinary Medicine, Petco Animal Supplies, Inc., San Diego, California
Concepts in Behavior, Section I: The Natural Science of Behavior, Updates in Anesthesia and Monitoring

GWEN B. FLINCHUM, BS, MS, DVM
All Bird Clinic of Palm Beach County, Lake Worth, Florida
Clinical Practice, Emergency and Critical Care, Management of Waterfowl

SUSAN G. FRIEDMAN, PhD
Department of Psychology, Utah State University, Logan, Utah
Concepts in Behavior, Section I: The Natural Science of Behavior

MICHAEL M. GARNER, DVM, Diplomate American College of Veterinary Pathologists
Adjunct Associate Professor, Washington State University, Northwest ZooPath, Monroe, Washington
Overview of Tumors Section II: A Retrospective Study of Case
Submissions to a Specialty Diagnostic Service

STACEY GELIS, BSc, BVSc (Hons), MACVSc (Avian Health)
Kingston Veterinary Clinic, Mentone, Victoria, Australia
Evaluating and Treating the Gastrointestinal System

PHOEBE GREENE LINDEN, BA, MA
Santa Barbara Bird Farm, Santa Barbara, California
Concepts in Behavior, Section II: Early Psittacine Behavior and Development

KENDAL E. HARR, DVM, MS, Diplomate American College of Veterinary Pathologists
Associate Director Aquatic Animal Health, Clinical Assistant Professor,
College of Veterinary Medicine, University of Florida, Gainesville, Florida
Diagnostic Value of Biochemistry

LORI DANIELLE HARRISON, DVM
Avian and Exotics at Central Animal Hospital, Eden, North Carolina
Evaluating and Treating the Liver

GREG J. HARRISON, DVM, Diplomate Emeritus American Board of Veterinary Practitioners (Avian),
Diplomate European College of Avian Medicine and Surgery (n.p.),
Avian Consultant, Palm Beach, Florida
Clinical Practice, The Companion Bird, Nutritional Considerations Section II: Nutritional Disorders, Emergency and Critical Care, Maximizing Information from the Physical Examination, Surgical Resolution of Soft Tissue Disorders

PETER HELMER, DVM, Diplomate American Board of Veterinary Practitioners (Avian),
Chief of Staff, Avian and Animal Hospital of Bardmoor, Largo, Florida
Advances in Diagnostic Imaging, Surgical Resolution of Orthopedic Disorders

CLAUDIA HOCHLEITHNER, Dr med vet
Private practitioner, Vienna, Austria
Evaluating and Treating the Liver

MANFRED HOCHLEITHNER, Dr med vet, Diplomate European
College of Avian Medicine and Surgery
Private practitioner, Vienna, Austria
Evaluating and Treating the Liver

JAN HOOIMEIJER, DVM
Clinic for Birds, Meppel, The Netherlands
Management of Racing Pigeons

K. STORMY HUDELSON, BS, DVM, Diplomate American Board of Veterinary Practitioners (Avian)
Private practitioner, Tucson, Arizona
Endocrine Considerations

PAUL M. HUDELSON, BA, MA
Tucson, Arizona
Endocrine Considerations

JACK J. KOTTWITZ, DVM
Underwood Animal Hospital, Heath, Ohio
Surgical Resolution of Soft Tissue Disorders

MARIA-ELISABETH KRAUTWALD-JUNGHANNS, Prof Dr med vet, Dr habil,
Diplomate European College of Avian Medicine and Surgery, German Veterinary Specialist for Birds/Reptiles
University Professor, Scientific Assistant, Clinic for Birds and Reptiles, University Leipzig, Leipzig, Germany
Evaluating and Treating the Cardiovascular System

ANGELA M. LENNOX, DVM, Diplomate American Board of Veterinary Practitioners (Avian)
Avian and Exotic Animal Clinic, Indianapolis, Indiana
The Companion Bird

TERESA L. LIGHTFOOT, BS, DVM, Diplomate American Board of Veterinary Practitioners (Avian)
Florida Veterinary Specialists, Tampa, Florida
Concepts in Behavior Section II: Early Psittacine Behavior and Development, Concepts in Behavior Section III: Pubescent and Adult Psittacine Behavior, Maximizing Information from the Physical Examination, Emergency and Critical Care, Integument, Overview of Tumors Section I: Clinical Avian Neoplasia and Oncology

MICHAEL LIERZ, Dr med vet, MRCVS, Certified Specialist Poultry (Avian Medicine), Certified Species Conservation
Institute for Poultry Diseases, University of Berlin, Berlin, Germany
Diagnostic Value of Endoscopy and Biopsy

KEATH L. MARX, DVM
Seven Seas Veterinary Services, Blacksburg, Virginia
Therapeutic Agents

DEBRA McDONALD, PhD, BSc (HONS I)
Zoo Nutrition Advisory Group (NAG), Healesville, Victoria, Australia
Nutritional Considerations, Section I: Nutrition and Dietary Supplementation
Nutritional Considerations, Section II: Nutritional Disorders

ROBERT D. NESS, DVM, Certified in Chiropractic (American Veterinary Chiropractic Association), Certified in Veterinary Acupuncture (Chi Institute)
Ness Exotic Wellness Center, Lisle, Illinois
Integrative Therapies

ESPEN ODBERG, DVM
Private practitioner, AviVet, Oslo, Norway
Surgical Resolution of Soft Tissue Disorders

JOANNE PAUL-MURPHY, DVM, Diplomate American College of Zoo Medicine
Associate Professor, School of Veterinary Medicine, University of Wisconsin, Madison, Wisconsin
Pain Management

MICHAEL PEES, Dr med vet, German Veterinary Specialist for Birds/Reptiles
Scientific Assistant, Clinic for Birds and Reptiles, University of Leipzig, Leipzig, Germany
Evaluating and Treating the Cardiovascular System

DAVID N. PHALEN, DVM, PhD, Diplomate American Board of Veterinary Practitioners (Avian)
Department Large Animal Medicine and Surgery, Texas A&M University College of Veterinary Medicine, College Station, Texas
Preventive Medicine and Screening, Implications of Macrorhabdus in Clinical Disorders, Implications of Viruses in Clinical Disorders

SIMON R. PLATT, BVM&S, MRCVS, Diplomate American College of Internal Medicine (Neurology), Diplomate European College of Veterinary Neurology, RCVS Specialist in Veterinary Neurology
The Animal Health Trust, Centre for Small Animal Studies, Kentford, Newmarket, Suffolk, United Kingdom
Evaluating and Treating the Nervous System

CHRISTAL G. POLLOCK, DVM, Diplomate American Board of Veterinary Practitioners (Avian)
Department of Clinical Sciences, College of Veterinary Medicine, Kansas State University, Manhattan, Kansas
Implications of Mycobacteria in Clinical Disorders

DIANA POST, VMD
Executive Director, Rachel Carson Council, Inc., Silver Spring, Maryland
Low-risk Pest Management

MADELINE A. RAE, BS, DVM, MS, Diplomate American Board of Veterinary Practitioners (Avian)
Amazon Veterinary Diagnostics, Salem, Oregon
Diagnostic Value of Necropsy

PATRICK T. REDIG, DVM, PhD
Professor and Director, The Raptor Center, College of Veterinary Medicine, University of Minnesota, Saint Paul, Minnesota
Surgical Resolution of Orthopedic Disorders

JILL A. RICHARDSON, DVM
Associate Director, Consumer Relations and Technical Services, The Hartz Mountain Corporation, Secaucus, New Jersey
Implications of Toxic Substances in Clinical Disorders

PETER SANDMEIER, Dr med vet, Diplomate European College of Avian Medicine and Surgery
Private practitioner, Baden-Dättwil, Switzerland
Management of Canaries, Finches and Mynahs

JAIME SAMOUR, MVZ, PhD, Diplomate European College of Avian Medicine and Surgery
Fahad bin Sultan Falcon Center, Riyadh, Kingdom of Saudi Arabia
Diagnostic Value of Hematology, Management of Raptors

ROBERT E. SCHMIDT, DVM, PhD, Diplomate American College of Veterinary Pathologists
Zoo/Exotic Pathology Service, Greenview, California
Integument

MICHAEL STANFORD, BVSc, MRCVS
Birch Heath Veterinary Clinic, Tarporley, Cheshire, United Kingdom
Calcium Metabolism

JENS STRAUB, Dr med vet, German Veterinary Specialist for Birds/Reptiles
Scientific Assistant, Clinic for Birds and Reptiles, University of Leipzig, Leipzig, Germany
Evaluating and Treating the Cardiovascular System

LINDA TIMOSSI, DVM
Avian, Exotic and Zoo Animal Consulting, Como, Italy
Management of Zoo and Park Birds

THOMAS N. TULLY, Jr., MS, DVM, Diplomate American Board of Veterinary Practitioners (Avian), Diplomate European College of Avian Medicine and Surgery
Louisiana State University School of Veterinary Medicine, Baton Rouge, Louisiana
Update on Chlamydophila psittaci: A Short Comment

LIZ WILSON, CVT
Levittown, Pennsylvania
Concepts in Behavior Section II: Early Psittacine Behavior and Development, Concepts in Behavior Section III: Pubescent and Adult Psittacine Behavior

---

Note to the Reader

The National Research Council (NRC) has not established standards for nutritional products fed to birds; therefore, claims of nutritional completeness can not legally or ethically be made. Human medical textbooks and those of domestic animals have established normal laboratory values, recommended treatment regimes and nutritional disease states — all based on individuals meeting minimum standards of nutrition. These are not available in avian medicine.

Disclaimer

The publisher, editors, authors, reviewers and distributors involved assume no legal responsibility for use and make no claims or warranties regarding results that may be obtained from information in this text, nor necessarily endorse the procedures, medications, medication dosages or their uses as offered in this work. The above parties shall not be liable to any party for any damages, nor considered negligent for any misstatement or error obtained in this text.

Foreword

If you do something long enough, you will eventually be able to step back and observe the real progress that has been made in your field of endeavor. After almost thirty years of avian practice I am amazed and gratified how far we have come. When I started practicing in 1976, in a given year, it was an easy task to read every thing published about clinical avian medicine. That has obviously and dramatically changed. The formation of the Association of Avian Veterinarians increased the generation of published materials considerably. Over the years, as each major text neared the publication date, an excitement was generated. Greg Harrison, with the assistance of co-editors Linda Harrison and Branson Ritchie with previous texts, and Teresa Lightfoot with Clinical Avian Medicine, has learned to distill the massive amount of material into a manageably useful and cohesive text.

Clinical Avian Medicine is predominantly written by clinicians for clinicians with a wonderful degree of clinical relevance. The number of high quality color photos inserted into the body of the text makes it easy to follow the ideas being presented. Thank you to doctors Harrison and Lightfoot and the fifty international authors for their hard work and perseverance in generating this most current and comprehensive avian medical reference text.

Donald W. Zantop, DVM, Diplomate American Board of Veterinary Practitioners (Avian)
Fallston Veterinary Clinic, Fallston, Maryland

Preface

The future of avian medicine and stewardship is increasingly bright. Importation of wild-caught birds has been largely curtailed, and the vestiges of unethical (yet legal) loopholes as well as illegal venues for smuggling are being actively and more effectively opposed.

With the cessation of mass importation, along with vastly improved nutritional provisions, increased diagnostic and therapeutic techniques and a better understanding of the emotional needs of our pet birds, we are privileged to observe the first generations of captive-born psittacines that may survive to attain their optimal quality and length of life.

Clinical Avian Medicine offers knowledge gleaned from years of clinical experience, combined with cutting edge research. New methods of prevention, diagnosis and treatment of various diseases are included in the areas of virology, neoplasia, dermatology, neurology, necropsy techniques, hepatic disease, pain management, behavior, soft tissue and orthopedic surgery, anesthesia, endoscopy, reproductive disorders, nephrology, hematology, biochemistry, endocrinology, gastroenterology, and therapeutics. Novel topics such as low-risk (i.e., environmentally safe) pest control and integrative (alternative) avian medicine are provided. The approaches and coverage of all these topics by contributing authors are both enlightening and readily applicable to clinical practice.

Anecdotal, yet often invaluable, clinical information regarding avian disease syndromes, treatment, husbandry and nutrition are included in Clinical Avian Medicine. The exclusion of these essentials would be an impediment to the practitioner searching for information to provide treatment for his or her avian patient. Anecdotal reports can create concern with efficacy and safety if not appropriately identified. Therefore, throughout this text, we have attempted to indicate the source of the stated information, allowing the individual veterinarian to determine its applicability. Unarguably, strict scientific methods are needed to document the validity of diagnostic and treatment protocols. Yet we must not squelch the introduction of innovative and potentially life-saving techniques in clinical practice due to delays in the completion of scientific validation. Our duty as authors and editors is to present the facts as they currently stand. We believe this availability of information has the potential to enhance the quality of avian medicine, surgery and husbandry when placed in the discriminating hands of veterinary practitioners.

Greg J. Harrison, DVM, Diplomate Emeritus American
Board of Veterinary Practitioners (Avian),
Diplomate European College of Avian Medicine and Surgery (n.p.)

Teresa L. Lightfoot, DVM, Diplomate American Board of
Veterinary Practitioners (Avian)

Vision

The vision of clinical avian practice as stated in the Preface mirrors the history and achievements of Dr. Greg Harrison. This book is first, last and foremost, the result of his lifetime dedication to avian medicine and stewardship.

When Dr. Harrison began the practice of avian medicine in the 1970's, he was one of a very few individuals in this field. Nearly all of the accepted "facts" in avian medicine were extrapolated (correctly or incorrectly) from companion animal medicine or poultry science. Dr. Harrison's early work precipitated the subsequent exponential increase in avian medical and surgical innovations throughout the following three decades. The discoveries and improvements in avian endoscopic techniques, microsurgery, radiosurgical modifications and applications, and a multitude of other current standard avian therapies are the direct result of his early work in these fields.

The publication in 1986 of the textbook, Clinical Avian Medicine and Surgery (Harrison and Harrison), provided practitioners with a much needed avian veterinary reference text. Throughout the development of that book, Dr. Harrison enlisted colleagues within the USA and abroad, involving some of the foremost authorities in this burgeoning field. The contributions of these authors, combined with Dr. Harrison's vast experience (and due in no small part to Linda Harrison's unsurpassed organizational and editorial skills), created a text with a depth of knowledge and information invaluable and previously unavailable to veterinarians in avian practice.

This has been Dr. Harrison's modus operandi throughout his career: to put forth ideas, solicit input and encourage innovation in others, and to listen open-mindedly to alternative theories. Dr. Harrison has consistently walked the difficult line between improving the application of current techniques, while seeking alternative treatments and more fundamental data.

In recent years, Dr. Harrison has dedicated his time and energies to preventive avian medicine, specifically in the area of nutrition. The outcome of this commitment has improved the health, quality and length of life of untold numbers of beloved pet birds.

As is common in innovative persons of all fields, Dr. Harrison's intense focus and drive have created ardent followers and admirers, as well as skeptics. It is again to his credit that both reactions are welcome. Advancements in avian medicine will be accelerated by those whose research validates Dr. Harrison's hypotheses, and by those whose work may contradict various theories. Within Clinical Avian Medicine, Dr. Harrison's third major avian text, the reader will encounter this broad perspective in the presentation of contrasting ideas presented by various authors.

Greg Harrison has been my role model, teacher, mentor and friend. I have been and will remain awed by him. The world of avian veterinary medicine and the world in general is a better place thanks to his contributions and his presence among us. This publication is a reflection of his life's commitment.

Teresa L. Lightfoot, DVM, Diplomate American Board of Veterinary Practitioners (Avian)

June 2005

Acknowledgements

Any undertaking that involves 50 authors is touched by many others, all of whom, unfortunately, may not be mentioned. So for all those who contributed in any way, I thank you. I offer deep appreciation to all the authors for their patience and tenacity in repeatedly revisiting and reworking material to create this exceptional text.

Stormy and Paul Hudelson were very supportive, reviewing all and revising much of the text. Patty Wolf was there for every chapter over and over. Vanessa Rolfe for doing a final read through. Heather Bowles, Lori Harrison, Jack Kottwitz, Michael Lierz, Michael Pees and Michael Stanford stepped up to work on subjects that offered challenges late in the process and came through in rapid fashion.

We welcome the breakthrough contributions in this text of Debra McDonald, Robert Ness, Diana Post, Susan Friedman, Liz Wilson, Phoebe Greene Linden, Heather Bowles, Peter Sandmeier and Peter Coutteel.

To our contributors whose first language is not English, including Espen Odberg, Michael Lierz, Michael Pees, Peter Sandmeier, Manfred Hochleithner, Claudia Hochleithner, Jan Hooimeijer, Marcellus Bürkle, Peter Coutteel, Maria-Elisabeth Krautwald-Junghanns, Jens Straub, Lorenzo Crosta, and Linda Timossi: the extra skill and effort required to write in a non-native language is appreciated and admired.

Teresa Lightfoot made the whole process possible. Her talent, brilliance, humor and tenacity were my saving graces. I love her beyond words. Teresa enrolled the follow reviewers: Keeley McNeal, DVM; Lisa Teske, DVM; Krysta Deitz, DVM; Benjamin Welbourne, DVM; Hugues Lacoste, DVM; Raimon Duran-Struuck, DVM; Lisa Dixon, DVM; Valerie Sadler, DVM, Diplomate ACVR; Neil Shaw, DVM, Dipl ACVIM; John Meeks, DVM, Dipl ACVIM (Neurology); Tracy LaDue, DVM, Dipl ACVIM (Oncology), Dipl ACVR (Radiation Oncology); Mark Levy, DVM, Dipl ACVS; Eric Mears, DVM, Dipl ACVIM; Amy Benedict, CVT; Brenda Fulcher, CVT; Lee Burstiner, DVM, Dipl ABVP. Teresa also wishes to thank Florida Veterinary Specialists for the latitude and attitude to tap the knowledge and experience of the above individuals, and most of all, never-ending thanks to her husband, Chris Lane — for everything.

Nigel Harcourt-Brown is acknowledged for his art work and review of portions of the text. Additional reviewers to whom we are grateful are: David Rupiper, DVM; Jürgen P. Schumacher, Dr med vet, Dipl ACZM; and Rebecca E. Gompf, DVM, MS, Dipl ACVIM (Cardiology). Thanks to Dr. Carolyn Cray for contributions of the University of Miami to hematology and chemistry reference ranges. To others who reviewed material without acknowledgement, I offer my thanks.

Thanks to Mimi Walling for her beautiful bird photos, to Scott Echols for the use of his surgery photos and to the following for their photographic support: Auckland Zoo, E. Alberti, Lucy Bartlett, Peter Helmer, Chris Griffin, Nancy Boedeker, Carol Canny, Friedrich Janeczek, Judy St. Ledger, Eduardo Nycander, Drury Reavill, Nico Schoemaker, Michael Walsh and Don Zantop. Special thanks to Jean Coffinberry for promotional and cover concepts and to Ray Kersey, Chris Lane and Friedrich Janeczek for cover and presentation ideas.

Thank you to Tanya Harrison-Coffinberry for assistance with the organizational and financial management, production, legal issues and sale of this work. I appreciate the clients of The Bird Hospital and Harrison's Bird Foods who helped create a need for this information in the veterinary field.

My love and gratitude to Linda Harrison for encouraging me to spread my own wings with this book. There is no greater communicator of practical veterinary information than you, Linda.

Greg J. Harrison, DVM, Diplomate Emeritus American
Board of Veterinary Practitioners (Avian),
Diplomate European College of Avian Medicine and Surgery (n.p.)

Clinical Avian Medicine

CONTENTS VOLUME I

CHAPTER
1

Clinical Practice

GREG J. HARRISON, DVM, Dipl ABVP-Avian, Dipl ECAMS;
GWEN B. FLINCHUM, BS, MS, DVM

Avian medicine has seen dramatic growth and development over the past several years. Veterinarians who wish to incorporate avian medicine into their practices now have numerous resources available to help them get started. The Association of Avian Veterinarians (AAV) is currently the most recognized avian veterinary professional organization in the United States, Europe and Australia. AAV national conferences provide veterinarians opportunities to share information and advice, along with programs that reveal the latest innovations in avian medicine. Other organizations in the USA include the American Federation of Aviculture, Mid-Atlantic States Association of Avian Veterinarians and statewide professional associations. The European Association of Avian Veterinarians meets every other year on the odd years. The Australian Committee of the AAV (AAVAC) meets yearly (see Table 1.1).

Education

There are numerous journals and educational materials that provide information about avian medicine. The AAV's Journal of Avian Medicine and Surgery is an international journal that provides information on the medicine and surgery of captive and wild birds and publishes scientific articles, book reviews and information regarding upcoming AAV meetings in its accompanying Newsletter. There are other resources listed in Tables 1.2 and 1.3.

Information sharing with veterinary colleagues is also available on the Internet. Forums such as Veterinary Information Network, Avian Medicine On-Line and Exotic DVM Readers' Forum (see Table 1.2) allow veterinarians from all over the world to communicate directly concerning their cases and experiences.

Fig 1.1a | A veterinary clinic's heart is its staff, who ideally are devoted, dependable, friendly, compatible and committed to patient care, service and hospital quality from floors and plants to attire. From record keeping to honesty and controlling costs, a veterinarian's dream can come true if everyone works at these goals and the work is made enjoyable.

Fig 1.1c | Educating the public is the goal of a good staff. Brochures[1], newsletters from the clinic or product distributors, web sites, clinic-sponsored classes and guest lectures all contribute to client education.

Fig 1.1b | Educating one's self and staff is much easier with access to current textbooks, references, newsletters, brochures and CDs[1].

For those veterinarians who are interested in specializing in avian medicine, The American Board of Veterinary Practitioners (ABVP) has approved residency programs leading to board certification. Alternately, experienced practitioners may apply for ABVP-Avian certification without completing a residency. In either case, the current procedure for becoming a Diplomate of ABVP-Avian Practice includes writing case studies, passing rigorous written and practical examinations, as well as satisfying other required criteria. A similar but more academically oriented credentialing system has been established in Europe and is called the European College of Avian Medicine and Surgery (ECAMS).

KNOWLEDGE AND EXPERIENCE

The first challenge is to acquire the knowledge and experience necessary to practice avian medicine in a manner on par with other branches of veterinary medicine. One needs to study the available reference materials, including the journals and proceedings from the professional organizations listed in Table 1.1. Attending meetings of these organizations will provide opportunity for hands-on experience via wet lab attendance to improve one's practical skill, and direct interaction with others in avian veterinary medicine. Ideally, a novice avian practitioner would work directly with an experienced avian veterinarian. When this is not possible, a referral/phone consultation relationship with an experienced avian veterinarian will allow one to comfortably refer complicated cases, and to properly handle those cases that are within one's current skill level, while one's diagnostic and therapeutic abilities increase. Wild bird rehabilitation organizations are often in need of veterinary services. In exchange for these services, practitioners will gain valuable experience in handling and will build confidence in their surgical abilities without fearing the loss of a beloved pet bird. Ownership of birds of various species will make the practitioner comfortable with behavioral differences among species.

The goal of this text is to assist the veterinarian in becoming a competent avian practitioner. Knowledge of one's limitations regarding both equipment and experience is part of this qualification.

The American Animal Hospital Association (AAHA) 2003 study on standards and patient care states that failure to educate clients is the leading reason pets do not receive optimal care (this study is available on the AAHA web site, see Table 1.1)[1] A knowledgeable staff and appropriate educational support material is the underpinning of client compliance (Figs 1.1a-c).

MARKETING SERVICES

Once the decision has been made to incorporate avian medicine into the practice, attracting prospective clients is a priority. This can be accomplished through advertisements in various modalities, including the telephone directory, various Internet group listings, newspapers,

pet stores and in the "veterinary services" section of bird magazines. A guide to help locate additional advertising venues is available.[4]

The following questions can be used to define specific emphases within avian medicine. Some items may present areas for future expansion of your knowledge and the scope of your practice's client services.

What makes your practice distinctive?
Does your practice possess specialized equipment and procedures (eg, endoscopy, laser unit, microsurgery)?
Does your veterinary or technical staff have specific training in the areas of avian medicine, surgery, nursing or behavior?
Are classes or consultations offered to clients for behavior disorders?
Does your practice carry recommended diets, bird-safe and appropriate toys, perches or caging?
Are house calls offered?
Is emergency service for avian patients provided?

Colleagues who do not treat birds can be a good source of referrals. Pet store owners and bird breeders may refer their customers if a good relationship is developed with your practice.

Bird marts, bird shows and bird fairs are very popular in the USA. Educating the public of the inherent dangers of these bird fairs should be a goal of avian veterinarians, responsible bird breeders and bird clubs. Currently, such events involve bird traders, neophyte hobbyists (the latter group often having the best of intentions) and a number of uneducated individuals, who often purchase birds on impulse. Birds at these fairs sometimes are poorly bred, incorrectly fed and/or improperly raised. Husbandry practices prior to the fair are unknown, and the co-mingling of species from various breeders that occurs at the fairs can be a major source of disease exposure and transmission. If a bird is purchased from a bird show or bird fair, it should be quarantined from other birds in its new household, although this seldom is accomplished. Sadly, the volume-oriented, discount-minded pet store often is a similar source of potentially diseased and compromised birds. Fortunately, awareness of these problems is increasing in both the USA and the EU. In the interim, encouraging clients to at least consider the methods of breeding, raising, feeding, testing and hygiene that are practiced by the seller prior to purchase will decrease the risk of acquiring a sick or debilitated bird. See Chapter 3, Concepts in Behavior for comments on proper husbandry and raising techniques. For guidelines on helping owners to choose a pet bird, see Chapter 2, The Companion Bird.

Despite all these problems, avian and exotic animal expositions are an acknowledged source of potential clients.

Jan Hooimeijer

Fig 1.2 | Bird expositions are a common source of sick birds in the pet industry.

Renting a booth and showcasing your clinic will allow you to meet hundreds of bird enthusiasts. Although this is a high-visibility opportunity that may bring new clients, there are disadvantages. The most obvious problem is that the very presence of an avian veterinarian at these shows implicitly condones their existence. (Fig 1.2). Therefore, conflicts of interest may arise between sellers, buyers and veterinarians at such an event.

Dr. Jan Hooimeijer of The Netherlands has developed a unique offering for bird owners who desire to share their passion for their birds. He implements a strict client education program and regimented standards that must be met by the clients. These include recommendation of an organic formulated diet, disease testing and behavior classes. Log on to www.harrisonsbirdfoods.com for further information. Dr. Hooimeijer's clients are then invited to attend a "Bird Walk," a yearly celebration held in a local park, and the public and media are invited to attend. Some 400 clients and their family members attended in 2002, and the event was covered by national television (Figs 1.3a,b).

Another modality for attracting clients is to offer free educational classes after hours in the clinic, a local pet store, local university or other continuing education facility. (Fig 1.4) The classes can cover topics such as choosing a bird, bird behavior, nutrition and first aid. Flyers for the classes can be placed in pet stores, university bulletin boards, supermart bulletin boards, and advertisements can be placed in local newspapers. The classes can be also be promoted with flyers in the hospital, on the clinic's web site and in the clinic newsletter. This will attract current clients as well as prospective ones who are interested in becoming more knowledgeable about bird care.

Client referral is an excellent method to increase clientele, reflecting the satisfaction of an existing client and generating positive advance impressions in a new client. The extent of this "word of mouth" client referral will be

Jan Hooimeijer

Jan Hooimeijer

Fig 1.3a,b | Dr. Jan Hooimeijer has an annual "Bird Walk" to proudly show off his clients and their family members. More than mere pets, Jan molds his patients into a cohesive unit. Bicycle rides and family outings like this strengthen the clinic-client bond. Clinic support of bird clubs or bird-oriented groups concerned with parrot welfare provides opportunities for the clinic to impact the bird world in a very important way.

based to a great extent on client satisfaction. Client satisfaction starts with a clean, comfortable, bird safe, well-planned clinic with a friendly, knowledgeable staff and minimal waiting time. When technicians and doctors have ample time to spend discussing educational material and answering questions, this reflects concern and genuine interest in the patient, which pleases clients and impresses upon them that the staff and doctors are competent. Clients also are impressed with professional achievement awards, certifications achieved by the hospital (eg, AAHA) or staff with advanced training. They appreciate knowing about areas of special interest and the number of years that the staff and doctors have been involved in bird treatment.

Let clients know about your continuing education efforts, any papers you publish or news items written about your clinic. These items can be put on web sites, in newsletters or in waiting room binders for the client to see.

Fig 1.4 | Classes at public meeting places and sanctioned by clubs, schools and other public groups serve as the bedrock of educated clients. This gathering at Pine Jog Environmental Center in 1970 led to a university course on aviculture at Florida Atlantic University. From this small group came leaders of aviculture, founding secretary of Avian and Caged Bird Society of Ft. Lauderdale, FL (Ellen Tannerhill); a president of the American Federation of Aviculture (Tom Ireland); head curator of birds at Sea World, Orlando (Sherry Branch); Harvard Botanical collection director (George Staples); organizational president, AAV and HBD (Greg Harrison); president of ZEN, editor, Exotic DVM (Linda Harrison); owner, Palm Beach County, FL, oldest bird pet shop, Fins, Furs and Feathers (Charlie Holland); four other pet shop owners and a dozen avid aviculturists. One lady later became the wife of the head of veterinary services at Busch Gardens, Tampa, FL — she and her husband still breed birds some 20 years later.

Mimi Walling/We Shoot Birds

Fig 1.5 | A client waiting room with a pleasant presentation of safe toys, educational materials and veterinarian-only dispensed bird foods makes a strong statement about the value of these items. Selling only items endorsed by the staff reduces confusion and improves client compliance.

Having photos of recommended pet birds at various locations throughout the clinic lets clients know of your special interests. A photo album containing photos either solicited from your clients, or taken at your hospital of the clients and their birds, is a very popular item in the waiting room.

FOOD, TOYS AND OTHER BIRD PRODUCT SALES

Clinic service and income can both be increased through the sales of recommended bird food, toys, books, perches, carriers and various other products. Toy selection should be based on bird-safe toys (Figs 1.5, 1.6a,b)

Fig 1.6a | Unsafe toys include clips like the one caught on this African grey's beak. For more on safe toys, see Chapter 6, Maximizing Information from the Physical Examination.

Fig 1.6b | Unsafe toys can trap a bird by the foot or leg. With its owners in the next room, this Amazon was totally silent as it chewed its leg free from being caught in a toy's rings.

Fig 1.7 | Just because a toy is made for a bird does not ensure it is safe. The Amazon in fig 1.6b mangled its foot with this set of rings (top). Similar metal circles are used on this key ring toy (bottom), which could easily trap a toe or foot.

Fig 1.8 | Staff at The Bird Hospital removes unsafe metal (galvanized clanging metal bells and clips) and replaces them with stainless steel chain and chain "quick" links. Avoid man-made fibers like nylon in toys, and instead use natural cotton, leather, sisal or hemp.

Fig 1.9 | This boarding facility offers an observation window from the reception area. Plants, up-to-date periodicals, and a clean, fresh presentation attract clients.

such as woven palm leaves, leather, wood and unbreakable plastic. Since many chains, mirrors and toy clips (Fig 1.7) are made of unsafe metals, clips should be switched to stainless steel C-clips (Fig 1.8). These can be bulk ordered and the cost can be incorporated into the price of the toy.

AFTER-HOURS EMERGENCY SERVICES

Service availability is another factor in client satisfaction. Weekend and late-night office hours are a convenience for clients who work during weekdays. An answering service allows clients to talk to a real person after hours when needed. Clinic brochures can be offered to give information on office hours, special services and directions to the clinic.

Offering a 24-hour emergency service is an advantage that clients value and appreciate. One author (GJH) employs a reasonable policy that requires clients to have physical examinations performed on their birds at least once yearly to be eligible for after-hours emergency services. The receptionist informs callers, shoppers, food and toy buyers that a physical exam and current testing are required for eligibility for emergency service and boarding services (Fig 1.9). This maintains client commitment to routine care for their birds and reassurance that they have access to emergency services when needed.

Qualified veterinarians may give special training in avian emergency medicine to local emergency clinic personnel. Such training should emphasize stabilization of sick birds and include basic nursing care such as the administration of injections, selection of medications frequently administered in avian emergencies, gavage-feeding techniques and general supportive care. Avian veterinarians in several major cities provide training programs including procedure manuals for emergency clinicians in their area. This often allows the avian veterinarian to consult on emergencies via telephone, decreasing the demands of providing 24-hour emergency care.

Fig 1.10a | House call kit.

Fig 1.10b | Needles, blood collection tubes, styptic and materials for record keeping.

Fig 1.10c | Suture, simple surgical instruments, heparin, slides for smears and injectable antibiotics.

Fig 1.10d | Rotary drill, stethoscope, culturettes, syringes and medications round out the kit. Fresh, clean towels and species-specific equipment like nets are included as called for by the house call.

COLLEAGUE RELATIONSHIPS

Developing and maintaining a positive professional relationship with colleagues benefits everyone. Local veterinarians can be excellent sources of referral cases. Lectures given at local veterinary associations will increase referrals from these colleagues.

HOUSE CALLS

House calls are a convenience that clients appreciate. This is especially important for clients with numerous birds or birds that are too large (swans, geese) to transport. A house call kit (Figs 1.10a-d) can be easily made using a tool kit from a hardware store. Significant data regarding husbandry and potential disease can be obtained with a visit to the home or aviary. (Figs 1.11a-e) A video of the facility can be a useful alternative if a house call is not possible. See Chapter 6, Maximizing Information from the Physical Examination for an ideal psittacine aviculture setup. For an example of a mobile practice see Figs 1.12a-g. Be certain to check with your state regulatory authority regarding any additional requirements or permits that may be necessary when house calls are added to your current practice.

Practice Equipment

The operating microscope is one of the most useful tools in avian practice (Fig 1.13). A variety of types are available, and some have automated pedal switches that allow hands-free adjustment of focus, magnification power and zoom-in view. Some operating microscopes have photographic capabilities so that pictures can be taken of the image seen through the microscope. The operating microscope greatly enhances visualization during surgery. It is useful for physical examinations of smaller birds such as canaries and finches.

Head loupes (Fig 1.14) are helpful for enhancing visualization, and are less expensive and more portable than operating microscopes. A magnifying loupe allowing a minimum of 4 to 8x magnification is needed for many avian surgeries. Various of types are available, with and

Fig 1.11a | House calls reveal a world one would not suspect unless seen: several tens of thousands of dollars of champion-bred show canaries packed into 70 cages, stacked in one large room of a physician's home. An outbreak of polyomavirus allowed a rare visit into this private aviculture world. Infectious disease will rapidly spread in this type of inadequate housing.

Fig 1.11b | The husbandry of an aviculture facility is best observed firsthand. Note the hardware cloth door. This type of wire is zinc coated and potentially toxic. The feeding of seeds and allowing debris to accumulate are not ideal practices.

Fig 1.11c | Turtle eggs incubated in the same room with parrots. Salmonella and other problems are invited by such measures.

Fig 1.11d | Housing mixed ages and species together can potentiate disease outbreaks that a house call can identify and prevent.

Fig 1.11e | Rodents leave a black streak from body oil on the wall over this perch in the cockatoo facility of a prominent US zoo. The heavy stain indicated a lack of effective pest control measures.

without light sources. Head loupes make the field of view appear closer, rather than actually magnifying what is seen. Endoscopy equipment is another essential in avian practice (Figs 1.15a-h). Currently (and since the inception of avian endoscopy) the most commonly used endoscope is a rigid 2.7-mm with operating sheath (see Chapter 24, Diagnostic Value of Endoscopy and Biopsy). Several types of biopsy forceps are available to use with the operating sheath. Most endoscopes can be equipped with cameras and video monitors, and photographs can be taken from the image seen through the endoscope. These images can be recorded to a disc or printed as a hard copy, which allows documentation of the endoscopic procedure. There also is a 1.2-mm semi-flexible endoscope[a] that is especially useful for endoscopy of the trachea of small birds such as cockatiels and canaries. A flexible endoscope[b] is available and comes equipped with grasping forceps useful for gastrointestinal endoscopy and foreign-body removal.

Microsurgical equipment also should be a part of avian practice. This is discussed in Chapter 35, Surgical Resolution of Soft Tissue Disorders.

AVIAN ANESTHESIA EQUIPMENT

In avian medicine a semi-open, non-rebreathing system is recommended. Supplemental heat capability is critical for success in extended avian surgeries. For further information, see Chapter 33, Updates in Anesthesia and Monitoring. A mobile field units for endoscopy (Figs 1.16a-f) and anesthesia (Figs 1.16g) can be made.

ULTRASOUND

Ultrasound can be a useful diagnostic modality, although its use in birds is limited by patient size, conformation and by the presence of air sacs.[5] Ultrasound is particularly useful in differentiating abdominal swellings.[7] Further information on avian ultrasonography can be found in Chapter 25, Advances in Diagnostic.

Thomas M. Edling

Fig 1.12a | A mobile house call practice allows the veterinarian an opportunity to bring state-of-the-art technology to various clinics, malls, zoos, aviculturists and private owners.

Thomas M. Edling

Fig 1.12b | Miniature laptop computers and compact equipment powered by generators, batteries or plugging into an outside source allow all laboratory procedures to be performed.

Thomas M. Edling

Fig 1.12c | Intensive care cages with heat, humidity, oxygen and nebulizer capabilities in the mobile clinic.

Thomas M. Edling

Fig 1.12d | Treatment area in the mobile clinic.

Thomas M. Edling

Fig 1.12e | Mobile clinic surgery.

Thomas M. Edling

Fig 1.12f | Storz endoscopy video monitoring equipment.

Thomas M. Edling

Fig 1.12g | Isoflurane, sevoflurane, oxygen, monitors, scavengers and surgical support equipment in the mobile clinic.

Fig 1.13 | A complete surgical suite in The Bird Hospital, allowing video from rigid or flexible endoscopes and operating microscopes.

Espen Odberg

Fig 1.14 | The General Scientific Corporation's° ergonomically designed optical magnification system allows comfortable operating distance, lighting and magnification for microsurgery and delicate examinations.

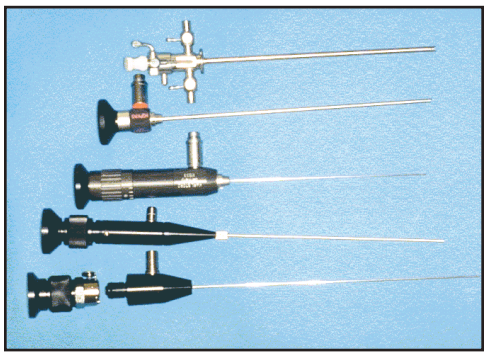


Fig 1.15a | A set of four rigid endoscopes in sizes to accommodate procedures on various size birds and a multiport sheath (top).

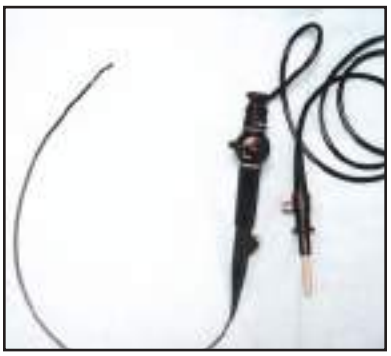


Fig 1.15b | Flexible endoscope, allowing flushing, suction and biopsy of sites difficult to reach with a rigid scope.

Fig 1.16a | Steps to custom power an endoscope for portability. The embedded magnification focus lens is removed from over the light of a Surgitel magnification scope.

Fig 1.16b | Close-up of removed lens (left) and the custom-made adapter (right) to allow this unit to power an endoscope.

Fig 1.16c | The modified light is ready to be attached to the endoscope.

Fig 1.16d | Light source snapped onto the endoscope.

Fig 1.16e | The final scope with battery pack for full mobile endoscopy. The author (GJH) uses it for tracheoscopy without anesthesia on canaries. This is a portable, lightweight field unit and it also produces more than enough light.

Fig 1.16f | Alternatively, an electrical plug can run the unit instead of battery power. The total unit price is several thousand dollars less than a traditional light source and flexible fiberoptic cable.

Fig 1.16g | A field anesthetic setup. A plastic drink bottle is filled with cotton wool and two straws or tubes are glued in place, one in the opening of the bottle neck and the other in a hole made near the neck. The neck tube is attached to a second bottle fashioned as a facemask induction chamber. The bird is placed in the induction mask and a towel seals around the neck. The anesthetist puffs (blows) on the second tube in the bottle with the cotton that has had 10 cc of isoflurane, halothane or ether added to the cotton. The anesthetic gas enters the induction mask. No more "blowing" is done until the level of anesthesia is so light that more gas is needed. **Human exposure to volatile anesthetic gases must be considered when using an open system such as this without suction or exhaust**. This semi-closed system avoids the waste of a mask with cotton and anesthesia in the mask itself. In a plain mask system it is difficult to control the anesthetic depth. With the anesthetic generator chamber one has more control, which in this case required covering the chamber with a stocking cap to keep the chamber temperature lower, as the sun produced heat that was causing too much anesthetic in the air-gas mixture, and birds were going down too fast and too deep.

Fig 1.17a | Metal toxicosis is seen commonly in avian practice. A "bag rad" technique is used as a preliminary scout film to make scout films fast, affordable and safe.

Fig 1.17b | The bird is placed in a bag sealed with tape and the bag is placed on the cassette; the exposure is made.

Fig 1.17c | Bird in bag on the cassette being exposed.

Fig 1.18a | Heart-liver-coelomic area view of a cockatiel, with no metal densities.

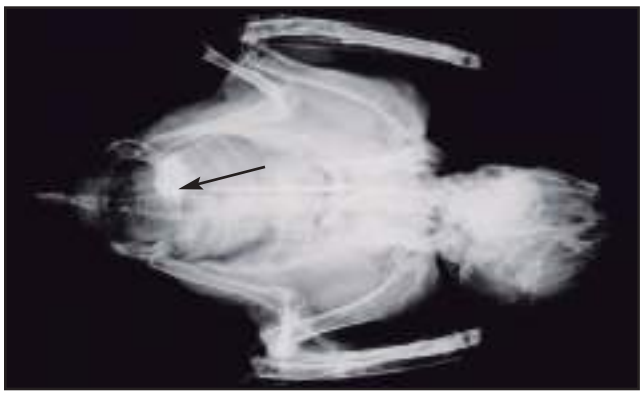


Fig 1.18b | "Metal" density (arrow) in ventriculus area in left caudal lateral aspect, liver swollen, increased shadowing of lungs and heart area.

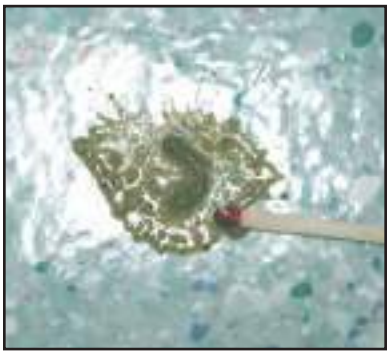
RADIOLOGY

Radiology in avian medicine is discussed in Chapter 25, Advances in Diagnostic Imaging. In cases where radiology is used to identify metal-dense particles, anesthesia is not needed, since proper positioning is not required. Most birds can be safely placed in paper bags (Figs 1.17a-c) and placed directly on the cassette for radiography. This allows a scout film to be safely made and metal densities to be visualized (Figs 1.18a,b).

GRAM'S STAINING

Laboratory sampling is discussed elsewhere; however, Gram's staining will be mentioned here and in Chapter 4, Nutritional Considerations. In past decades, Gram's stains of feces and choanal swabs were used as a major method of testing newly imported birds. The paucity of knowledge at that time regarding the methods of safe venipuncture and subsequent interpretation of results made Gram's stains and cultures the only available diag-

Fig 1.19 | A simple Gram's staining rack that confines the stains to the sink area.



Figs 1.20a,b | Placing a wooden-stemmed match head into a bird's cloaca stimulates the production of a fresh sample. Feces are usually produced moments after placing the match in the cloaca.

Fig 1.21a | Small birds can be restrained as shown to make jugular venipuncture easier. It stretches the neck out in an ideal fashion for small to medium-sized birds. One pulls the right wing caudally to be held with the leg on the same side as the wing. At the same time the head is held. A second person draws the blood sample.

Fig 1.21b | Lovebird being restrained using clamps to hold a towel around the neck like a whiplash collar, immobilizing the bird.

Fig 1.21c | Pelican bill restraint.

Fig 1.21d | A restraint device for positioning a bird for radiography or surgery.

nostic tools. In retrospect, this use of Gram's staining was less than optimal, and the results of the Gram's stain often were misinterpreted or over-interpreted.

Currently, in these authors' practices, Gram's staining of fecal material is done routinely and prophylactically. These authors have found that a bird's intestinal bacterial balance is an excellent reflection of its general nutritional state and thus more of a determinate of wellness than of illness.

The procedure can be very messy, and various racks (Fig 1.19) make this more manageable. Gram's stain solutions can be purchased in large-quantity containers, making the cost per test minimal. Ideally, the client is instructed to bring fresh, voided feces as part of the patient evaluation (see Chapter 4, Nutritional Considerations). If the client lives a long distance from

Fig 1.22 | **a.** Single-handed restraint to start a wing clip **b-d.** The foot and wing are restrained simultaneously. Wing trim in a light-bodied bird.

Fig 1.23a | Capture of an Amazon in a towel.

Fig 1.23b | Positioning the wing for trimming in a heavy-bodied bird.

Fig 1.23c | In an Amazon trim, secondaries only (distal to elbow) are clipped on one wing.

the clinic, there is a recommended method for getting a fresh sample (Figs 1.20a,b).

RESTRAINT

The restraint of birds such as raptors and other birds of prey is discussed in the special species chapters. The towel restraint is discussed in Chapter 6, Maximizing Information from the Physical Examination. For grooming, the towel makes single-person restraint and procedural accomplishment possible. Old towels can be purchased inexpensively at thrift or secondhand stores. Washcloths work well for smaller birds such as budgerigars or parrotlets, hand towels are ideal for cockatiels and lovebirds, and larger birds such as Amazons and macaws need to be wrapped in full-sized bath towels.

Small birds can be held for jugular bleeding as shown (Fig 1.21a). Towel clamps can be used to secure the towel once the bird is wrapped (Fig 1.21b). Waterfowl, wading birds and water birds in general need the beak controlled (Fig 1.21c) while raptors need foot restraint that will prevent taloning or "footing" of either the handler or of the bird itself. A plastic restraining board[c] (Fig 1.21d) is ideal for restraining birds for radiography and certain other procedures under anesthesia.

AVIAN GROOMING

Grooming birds consists of wing trimming, nail cutting, beak trimming and bathing. Wing trimming for large birds involves taking off a variable number of the primaries (see Chapter 6, Maximizing Information from the

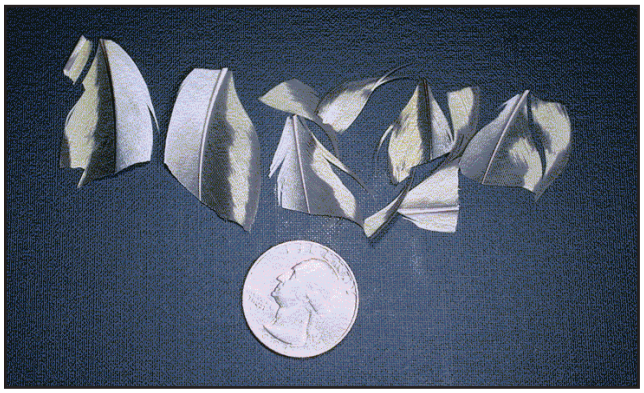


Fig 1.24 | Number of feathers removed from a cockatiel to stop flight. The bird was flying after an incorrect wing trim.

Fig 1.25a | Sternal ulcer from falling due to over-clipped wings.

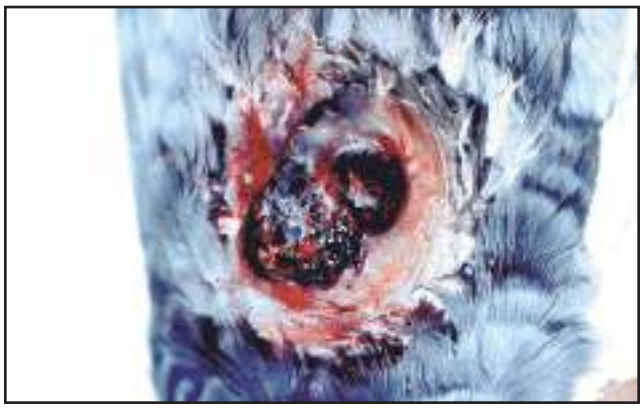


Fig 1.25b | After removing scab.

Fig 1.25c | An African grey, a heavy-bodied bird, that was falling and creating a sternal ulcer as seen in Fig 1.25a. The bird is having trimmed feathers pulled under anesthesia to speed feather regrowth. Use a firm grip with a needle holder, pull gently and steadily to avoid ripping the follicle and surrounding membranes.

Fig 1.25d | This photo shows the number of feathers pulled from the bird in Fig 1.25c. This is a form of forced (non-drug) molt. New feathers will grow in over the following few weeks. The bird must be kept on the floor or in a perch-less cage during regrowth to avoid falling.

Fig 1.25e | Several weeks after the wing feathers have regrown, the bird regained stability and then flight. At this time, the wing should be properly trimmed. If additional sternal trauma can be avoided for several weeks, the ulcer will heal by second intention. Proper diet speeds up feather regrowth and healing.

Physical Examination and Figs 1.22a-d and 1.23a-c). The primaries are cut as closely to their insertion as possible. Currently, the most popular method of wing trimming is to remove a variable (4-7) number of distal primary feathers from both wings. In the authors' practice, large-bodied birds have primary feathers cut from their right wing only, from the elbow to the tip of the wing. Small birds can usually be groomed by clipping both the primaries and some of the secondaries. Since there are many modifications to wing trims, it is NOT safe for birds to be carried free outdoors after ANY wing-trimming procedure. Fig 1.24 shows the limited mass of feathers needed to achieve flight in a cockatiel that was inadequately trimmed.

Fig 1.26a | Beak and nail trimming. The stone on this electric hand-held rotary tool® has been "loaded" with plastic or paint by grinding such a surface in such a manner as to "load" the stone, creating a smooth surface. This smooth surface generates heat for cautery if a beak or nail starts to bleed during trimming.

Fig 1.26b | Positioning the upper beak (maxilla) inside the lower beak (mandible) to accomplish a controlled trim and avoid tongue damage. Note the bracing of fingers one against another to allow fine controlled motions.

Fig 1.27 | Nail trim in a small bird, single-person hold. Brace fingers against one another for maximum control.

Fig 1.28a | Grinding a baby macaw's nails and accustoming it to the rotary tool. Older birds unaccustomed to this procedure should always be restrained to avoid biting or grabbing the drill or causing injury to the bird.

Fig 1.28b | Using the towel restraint (see Fig 1.23), the foot and toes can be controlled. Note the leveraging of the fingers one against another to provide accurate control.

Fig 1.28c | Changing angles and finger positioning to allow complete honing of each nail.

Conversely, excessive feather removal in heavy-bodied birds can result in sternal ulcers from falling onto hard surfaces (Fig 1.25a-e). African greys (*Psittacus erithacus*) and some macaws, cockatoos and Amazons develop necrotic ulcers of the skin over the distal wing that heal when the cut feather stubs are pulled under anesthesia. Personality changes from the sudden instability created by a wing trim can occur, especially in sensitive species such as African greys. It takes weeks for new feathers to emerge. In the authors' experiences, sternal ulcers usually heal successfully by avoiding further trauma (keeping the bird from falling repeatedly) and allowing feather regrowth. No medical or surgical (sternal keel reduction) treatment is usually needed if recurrence of the trauma can be prevented.

Beak trimming is not necessary in birds unless the beak is overgrown due to underlying health problems or malocclusion. Some clients will request the beak be dulled to mitigate mutilation or the pain to themselves or others from being bitten.

Cement (concrete) perches are useful for providing a rough surface on which the bird can clean its beak. Even birds that will not stand on a cement perch will often utilize one for cleaning food debris and excess keratin off the beak. These perches can be hung vertically in the cage to provide the bird with better beak access. Some of the available commercial cement perches are too rough and can cause plantar surface irritation, especially if the cement perch is located where the bird elects to perch for prolonged periods of time. Other brands are too smooth and are therefore not effective in providing an abrasive surface for the nails or beak (T. Lightfoot, personal communication, 2003). A hand-held rotary

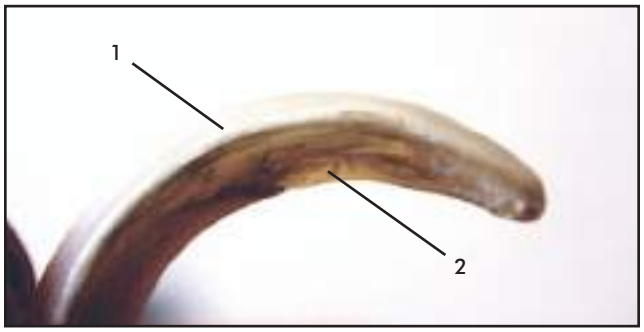


Fig 1.29 | Close-up view of a nail. Simply cutting the parrot's nail tip does not address the two sharp edges of the nail that are normally present (1,2). Controlled honing with a grinding or filing device leaves a smooth-surfaced nail and provides weeks, longer satisfaction for the client. Explaining this to the client and keeping towels and rotary tools sterile justifies the additional charges needed for a veterinary facility to provide grooming.

Fig 1.30a | Bathing of birds is a part of grooming. Thorough rinsing, towel drying and, in some birds, blow-drying may be used.

Fig 1.30b | Some "advisors" suggest blow-drying can be a cause of dry skin and that birds do not like blow-dryers. "Papagei," a pampered Moluccan cockatoo, thoroughly enjoys his bimonthly bath and blow-dry. He moves on his own to get each spot dry. He does not have a dry skin problem.

Fig 1.30c| The use of a feather cleansing solution provides a good vehicle for topical medications (ie, heparin, F10).

tool[e] is ideal for trimming beaks and nails because it files the tissue back in a smooth, controlled manner. Furthermore, the heat generated by friction from the filing tip facilitates cauterization if bleeding occurs. A conical-shaped rasping tip (Fig 1.26a) works best for avian grooming. Caution must be used when honing the sides of the rhinotheca, as the final protective/germinal layer is very thin and can easily be ground through or burned, creating a long-standing deficit. To facilitate shortening of mandibular rhamphothecal length, the maxillary rhamphotheca can be inserted inside the mandibular rhamphotheca (Fig 1.26b). This positioning not only makes the mandibular rhamphotheca more accessible, but also helps prevent the patient's tongue from making contact with the rotary bit.

Restraint for nail trimming is demonstrated (Figs 1.27, 1.28a-c). As the rotary tool is held, fingers should be positioned to allow maximum dexterity. Birds have sharp ridges on the sides of their nails, and this is why they remain sharp when only the tip of the nail is removed (Fig 1.29). The rotary tool is ideal for smoothing ridges so nails do not feel sharp. Again, as with wing trims, clients should be forewarned that the bird will be less able to maintain its balance after a nail trim and will be more likely to fall from its cage or the owner's shoulder when the nails are dull.

Giving birds a bath with soap and water is not a routine part of the grooming process as with cats and dogs. However, some birds really enjoy the process, as do the owners. The type of soap that is used for fine washable garments has been noted as safe and effective for bird-baths for the past three decades. It is recommended that one drop of soap per 250 cc water be used for routine bathing. Up to 3 cc soap in 250 cc water may be used to clean more heavily soiled feathers. The bird should be subsequently rinsed in warm water, towel dried, then blow-dried with an electric dryer (assuming the bird accepts this procedure) (Figs 1.30a-c), taking care not to burn the skin.

Fig 1.31a | An aluminum leg band.

Fig 1.31b | If the band is too large it can be easily slipped up over the ankle and removed intact.

Fig 1.31c | Bring digit 1 of the banded leg up along the metatarsus and slip the band down over that digit.

Fig 1.31d | Continue to slip the band off.

Fig 1.31e | The band is off. Reversing the process allows the band to be put back on for travel needs.

Fig 1.32a | Metal cutting pliers can cut only aluminum bands. They will only slightly dent the stainless steel band shown here.

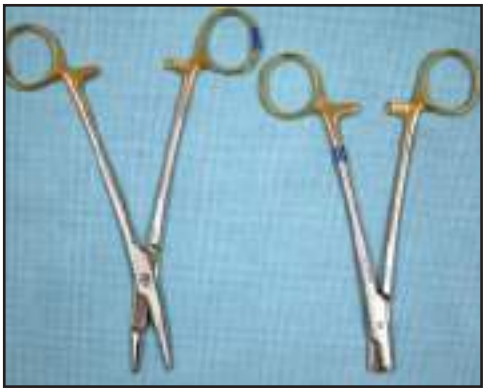


Fig 1.32b | To custom-make a very small bird band remover an Olsen-Hager needle holder on the left can have the needle holder plates ground off as shown in Fig 1.32c.

Fig 1.32c | The flat grind wheel on a hand-held rotary tool[e] can be used to remove the needle holder's tips and make the cuts shown in Fig 1.32d. This makes an excellent band remover for the fine plastic bands and some aluminum bands used for birds like canaries.

Fig 1.32d | Close-up of the cuts made to create a band remover from a needle holder.

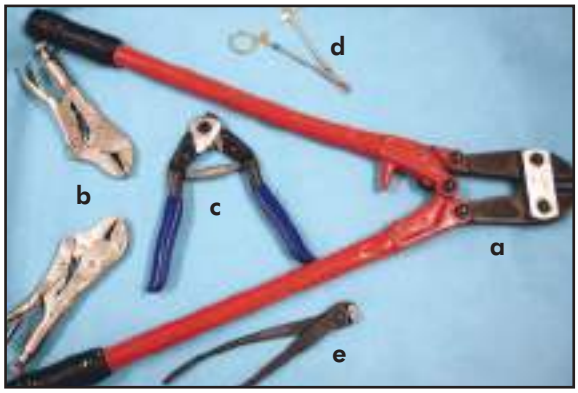


Fig 1.32e | A collection of tools used to remove various leg bands: Bolt cutter (a), vise or locking pliers (b), metal snips (c), modified needle holder (d), Kras cutter[f] (e).

Fig 1.32f | Side cutters and needle-nose pliers are examples of tools used to cut bands and hold metal for bending or cutting.

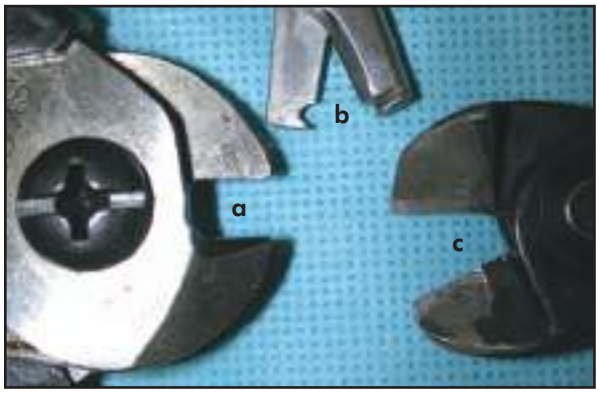


Fig 1.32g | Close-up of band-cutter heads. Metal snip (a), modified needle holder (b), Kras cutter[f] (c).

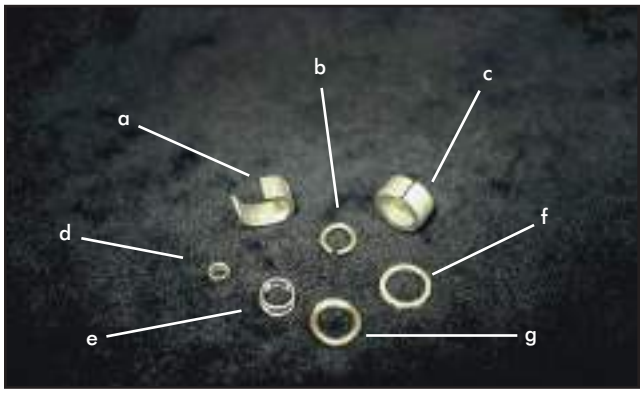
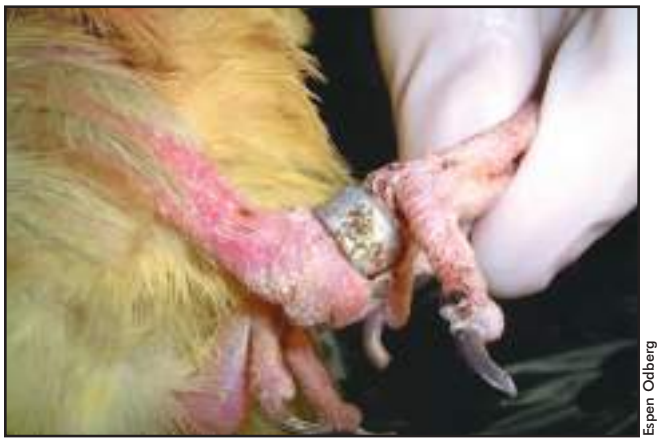
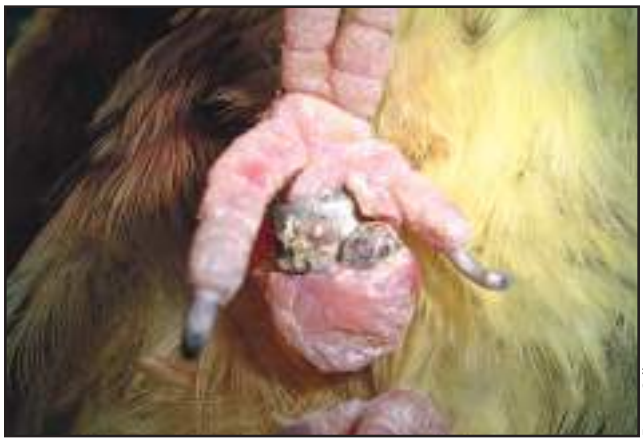


Fig 1.33 | Three split bands (a,b,c): (b) A stainless steel band that requires a bolt cutter or a two-vise-grip twist to be removed. (d,e,f) Aluminum bands: (d) and (e) can best be cut with modified needle holders; (f) requires one of the two tools shown in Fig 1.32e. (g) A solid stainless steel ring that requires bolt cutters once or twice and then vise grips.



Espen Odberg

Fig 1.34a | A leg constricted by a band due to accumulation of exfoliated skin under the band.



Espen Odberg

Fig 1.34b | Malnutrition creates a proliferation of the scales that can accumulate under a band. Over time, a depression in the leg's structure is formed by the proliferating skin mounding between the band and the leg. Pressure from this accumulation under the band causes constriction of the leg vessels. The foot can be lost due to necrosis. The custom-made needle holder band remover usually works best on this problem if the band is aluminum.

Espen Odberg

Fig 1.34c | A metal snip also can be used to cut aluminum bands constricting the leg.

Espen Odberg

Fig 1.34d | Pressure necrosis caused by tissue proliferation makes a deep groove that severely compromises circulation.

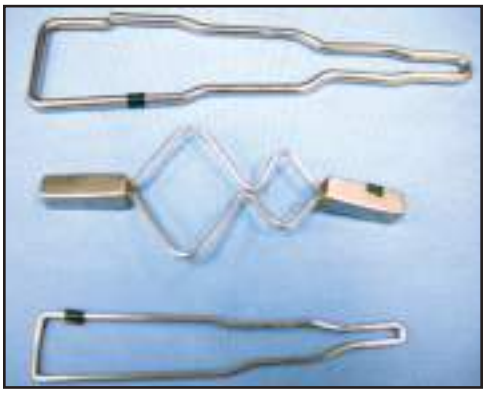


Fig 1.35 | A variety of mouth specula.

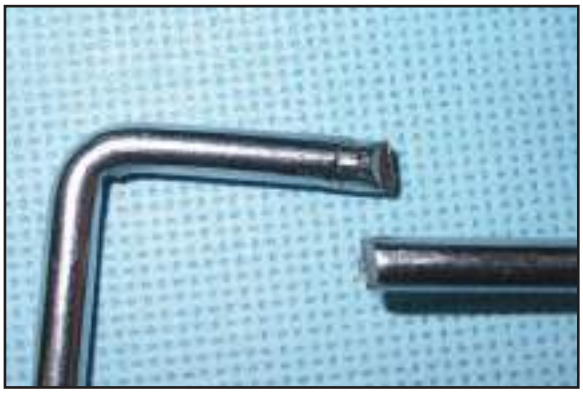


Fig 1.36 | A mouth speculum cut to reduce beak cracking.

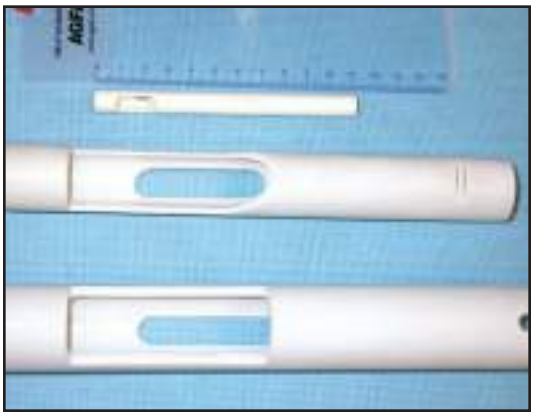


Fig 1.37 | Sections of plastic pipe used to make a speculum with a hole through which a gavage device can be passed.

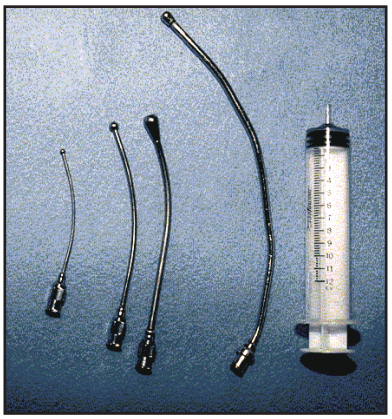


Fig 1.38 | Various metal gavage tubes.

Fig 1.39 | Silicone feeding tubes[h].

BAND-REMOVAL INSTRUMENTS

Oversized bands can be slipped off the foot. The band is slipped proximally over the tibiotarsal-tarsometatarsal joint (hock) as high as possible (Fig 1.31a-e). Digit 1 is retracted along the joint as shown and the nail is placed under the band. If needed, a lubricant can be applied. With light pressure the band slips off. It can be replaced later if need be. Band cutters[f] work well for removing most small, closed bands. (Figs 1.32a-g) Bands that are too small (Fig 1.33) or build up layers of keratin under them from nutritional disorders can cause constriction (Figs 1.34a-d). The larger steel bands are too strong to be cut with most band cutters and must be twisted off using vise grips or split with heavy metal (bolt) cutters (large red-handled device in Fig 1.32e).

OTHER EQUIPMENT

Specula

Stainless steel specula have the advantages of being indestructible and easy to sterilize (Fig 1.35). However, if the bird bites down aggressively on the speculum, damage to the mandibular beak can occur. Cutting one side of the speculum so that it is slightly moveable can minimize this potential problem (Fig 1.36). Specula are also made from tubular plastic material (Fig 1.37). Plastic specula are commercially available and are found useful by some practitioners to obtain pharyngeal swabs for PCR tests or culture. Two pieces of gauze also can be used to hold the beak open. For most procedures, a speculum is not necessary.

Feeding Tubes

Because they are indestructible and easily disinfected, stainless steel feeding tubes[g] (Fig 1.38) work best for most pet birds. Disposable silicone tubes[h] (Fig 1.39) work well for tube feeding neonates, waterfowl or other birds that cannot bite the tube. Red rubber catheters[g] are ideal for long-necked birds such as seabirds. See Chapter 14, Evaluating and Treating the Gastrointestinal System for photos of damage avoidance and proper tube placement.

Microchipping Equipment

Microchips are small electronic devices that can be injected into the body. The left pectoral muscle is the accepted placement of microchips in psittacines. These devices contain a code that, when scanned with a reader, will provide identification of the bird. One disadvantage of

Fig 1.40 | A Moluccan cockatoo on the perch on a scale.

Fig 1.41 | Disposable paper bags make easy restraint devices to weigh small birds and require no cleaning.

Fig 1.42 | A plastic tube perch in a cage.

microchips is that there is no industry standard, and one model of microchip reader cannot identify all microchips.[6] The USA's FDA and USDA have approved a combination AVID code and Fecava code 125-kHz chip reader.[i,i1] In Canada, Western Europe and Australia, the chips operate at 134.2 kHz. This ISO chip can be read with a global scanner, but not by the USA 125-kHz scanner, which only reads the corresponding chip.

Gram Scale

Pet birds need to be weighed in grams. Modern digital scales allow placing the bird on a perch (Figs 1.40). The tare function on these scales will allow automatic deduction of the weight of a box, bag or towel from the digital readout. These digital scales can be fitted with an AC adapter to avoid constant battery replacement. Most pet birds will sit on a perch to be weighed. For those that do not, various bags (Fig 1.41) work and they are replaceable without cleaning.

Examination and Clinic Cage Perches

Acrylic perches[j] (Fig 1.40) are ideal clinical perches for the exam room because they are relatively inexpensive and can be easily disinfected. Plastic tube perches are inexpensive, easy to clean and the material with which to construct these can be purchased at any local hardware store in multiple diameters. This plastic piping can be cut to any length to be custom fit to cages (Fig 1.42). Wrapping these plastic (PVC) perches in removable self-adhering bandage material (ie, VetWrap) offers the bird better traction while perching, and hygiene can be maintained by changing the wrap between patients.

PATIENT PRESENTATION

Birds should be presented to the clinic in travel cages (Figs 1.43, 1.44; see Fig 1.51b), not on the owner's shoulders (Fig 1.45). This is to prevent injury to bird or owner. Restraint for assisting doctors, grooming or administration of treatments is most effectively achieved as previously discussed using a towel.

Avian Practice Staff Members

All clinic staff should sign clinic confidentiality agreements; professional staff should include this agreement as part of their employment contract. Contracts should have buy-outs and exit strategies well outlined and should be renewed to keep them effective. Without these procedures chaos is invited.

The delineation of duties within the course of an avian appointment will vary between practices. The relative experience of staff, technicians and doctors, as well as the ratio of these and the current level of activity, makes cross training and flexibility advantageous, as in any veterinary practice. In order to give concrete examples of how the various steps involved in a patient visit can proceed, the authors have assigned specific responsibilities to each staff member in the following examples.

RECEPTIONIST

The demands of avian medicine are different from the demands of small animal practice; therefore, staff members should be appropriately trained. The receptionist is the client's first contact with your hospital and a major representative of the clinic. The receptionist should make clients feel welcome and comfortable and should be educated enough to answer questions concerning general

Fig 1.43 | Bird in a carrier made of hardware cloth, which is a potential source of zinc or lead. Lead will will cause a wet test strip [i1] to turn red.

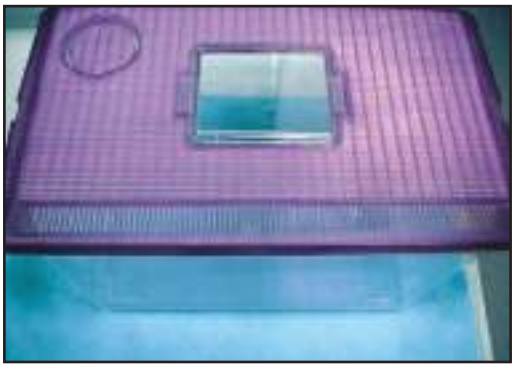


Fig 1.44 | A plastic storage box used to transport birds and other small pets.

Fig 1.45 | A hyacinth macaw on its owner's shoulders. An improper presentation mode for a clinical visit.

bird care. A competent receptionist should be able to successfully complete paperwork and expedite clients as they move in and out of the waiting room. Other responsibilities for the receptionist may include debt collection, retail ordering of food and toys, organizing reminder cards, initiating client callbacks and other communication to be sent out to clients. A skilled receptionist can turn the telephone shopper into an appointment.

VETERINARY ASSISTANTS

Finding certified technicians with avian experience often is very difficult. Therefore, one is often forced to train assistants to support the veterinarian in certain duties. While seldom competent in all areas taught to veterinary technicians, enthusiastic assistants can provide a great deal of support. Assistants often can be taught many of the technical duties: client questioning, performing fecal Gram's stains, administering drugs, and assisting in radiology and surgery. Assistants can perform procedures but seldom understand all the ramifications or anatomical, physiological, pathological and pharmacological reasons for their actions. Depending on the size of the hospital and the presence and extent of avian boarding, the veterinary assistant and kennel assistant may have overlapping duties (see the following section on Kennel Assistants).

Technicians, on the other hand, know why they are doing what they are doing. They often can support the veterinarian by offering suggestions based on practical understanding, demonstrating the huge difference between assistants and technicians.

VETERINARY TECHNICIANS

Veterinary technicians are certified as to their special training. They often need further experience and guidance to adapt to birds. Training opportunities are available at special seminars or conferences, such as the Association of Avian Veterinarians' annual conference and the International Conference on Exotics (ICE). There also are programs available through technical colleges throughout the USA. A complete list of veterinary technology programs in the USA is available in the American Veterinary Medical Association Directory and Resource Manual (Table 1.1). Although there currently are no technical programs that specialize in avian medicine, most programs do cover exotic animal care. Following are descriptions of responsibilities that technicians can be trained to perform.

Client Communication

There is a recognized lack of accurate information available to pet bird owners. This can be overcome by training technicians to give short talks during appointments on dietary requirements, husbandry and home care. See Chapter 6, Maximizing Information from the Physical Examination for a form that covers these topics. Technicians also can become involved with writing and distributing educational handouts and presenting classes.

Initial Examination

After reception, the technicians or assistants project the clinic's second image to the client. They take the bird into the exam room, review the case history and records, and verify that the paperwork is correct including name, phone number, bird's name, age, sex, species and color. They should present the clinic philosophy of wellness and education, offer take-home educational material and start the medical record. (See Chapter 6, Maximizing Information from the Physical Examination for a form that can be completed by the support staff.) Procedures are more readily understood and accepted by the client if visual educational material is presented. Diet, preventive practices, and the physical exam are discussed and performed. An information video on the value of proper

Fig 1.46 | A steam cleaner[k] makes dried matter easier to remove.

diet can be shown. A sample of fresh feces is Gram's stained in the laboratory. The veterinarian can then come in to evaluate the records, complete the physical examination, record the clinical signs, and form and record a list of differential diagnoses. These are then discussed and the diagnostic tests necessary are explained to the owner. The technician records these and prints out an estimate, including the diagnostic and treatment protocols. This is explained to the client by the technician, then approved by the client. The owner can then sign treatment permission and price quotation forms.

Under veterinary supervision, technicians draw blood, perform laboratory tests and collect, prepare and properly submit laboratory samples. Technicians also review cases daily with the veterinarian.

Technicians should monitor boarding and hospitalized birds on a daily basis noting and recording appetite, fecal production, weight and behavior. If any abnormalities are noted, these should be discussed with the doctor. In addition, technicians should become competent at performing routine treatments, preparing medications to be dispensed, administering injections, gavage-feeding, administrating oral and topical medications, and performing radiography, cytology and blood collection. Technicians are usually familiar with medical terminology so they can take notes for doctors during exams, surgery and necropsies. Taking radiographs, ordering medical and surgical supplies, contacting owners for follow-ups on laboratory reports and preparing instructions for patients being released are tasks a technician often masters. A competent technical staff with such skills is irreplaceable. Technicians also assist in surgery, including preparing the operating room for surgery, monitoring anesthesia and patient recovery, and cleaning and sterilizing instruments after surgery. A new small steam generator[k] is available for equipment cleaning and is efficient, effective and very affordable. The steam sterilizes as it loosens debris and reduces the need for chemicals (Fig 1.46).

Hospital inventory

Technicians should develop a rapport with several medical supply company representatives, increasing the chances of finding the best prices on drugs and hospital supplies. In addition, a pharmacy inventory should be kept so that stock can be kept up to date and reordered when needed. Shelves should be checked on a regular basis to remove expired food and drugs.

KENNEL OR HOSPITAL ASSISTANTS

Kennel or hospital assistants are responsible for weighing birds daily, and for clinic sanitation including cleaning cages and rooms. Assistants should be comfortable around birds because cleaning often necessitates handling birds without getting bitten, injuring the bird or permitting the birds to escape. Assistants should be astute enough to observe any abnormalities in feces, appetite or weight changes, so that technicians and doctors can be notified. These employees must be able to recognize proper placement of perches, food and water bowls within the cage to make certain the bird is accessing these. For example, a nervous bird may sit on a high perch in an unfamiliar animal hospital environment and not venture down to the level where the food and water are placed. Assistants also may be responsible for making sure that food and toy supplies are stocked on shelves and ensuring that any such items offered to hospital and boarding birds are safe and appropriate. Individual items such as cups, toys and carriers should be marked with the owner's name so that they are returned when the patient leaves the hospital.

AVIAN BEHAVIORIST

Biting, screaming and feather picking are frequently encountered problems with pet birds. Owners often do not know how to react correctly without reinforcing the problem. It is ideal to have an in-house bird behaviorist to work with clients to overcome these types of problems. Behaviorists also can train birds (and their owners) to overcome such bad habits as refusal to perch and insisting on staying on the owner's shoulders. Training for behavioral work can be obtained by attending conferences and seminars and by consulting with behavioral experts. For further information see Chapter 3, Concepts in Behavior.

Fig 1.47 | Padlock cage.

Fig 1.48 | White butcher paper.

Fig 1.49 | Central vacuum.

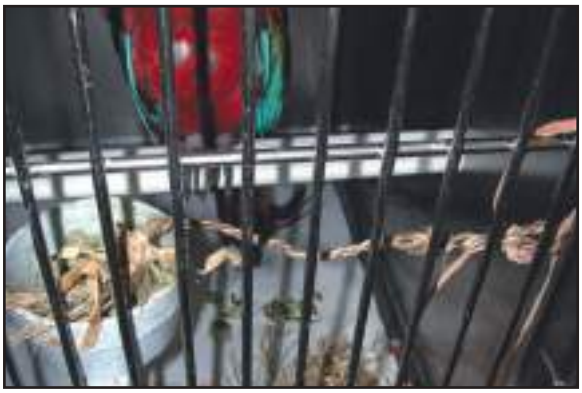


Fig 1.50 | Shredder toys.

Hospital Facilities

BOARDING

Fig 1.9 shows boarding birds that can be seen from the reception area. The presence of a "clinic bird" in a mixed small animal practice often serves to announce the practice's interest in birds.

Boarding birds should be housed in an area located away from noise and excitement. This is easier to do in an exclusively avian practice versus a small animal practice, where birds must have a special area away from dogs, cats or other exotic animals. Stainless steel or fiberglass cages, such as those used for dogs and cats, work well for housing birds because they are indestructible and easy to clean. Some stainless steel cages can be padlocked (Fig 1.47), which is useful to safeguard "escape artists" and help ensure the security of very expensive birds. Plastic pipe for perches can be cut to any size to fit in cages. White butcher paper (Fig 1.48) is non-staining and can be used for the cage lining. The white paper allows quick visualization of fecal and urate color and consistency, and this helps monitor the bird's health. For smaller birds, cages can have the bottoms removed and be placed directly on the butcher paper, thus facilitating cleanup.

Central vacuum systems (Fig 1.49) make cleaning easier. Central vacuum access in the surgical preparation area is also an advantage, allowing feathers to be immediately vacuumed as they are plucked.

Preliminary Testing for Boarding Birds

The policies that are implemented for testing prior to boarding should be tailored to the individual bird and its particular risk factors. For example, juvenile birds, birds that are exposed to other birds at bird shows and those that accompany their owners to pet stores, should be tested more frequently. It is good policy to require every boarding bird to have a physical examination and a minimum laboratory base-line, including a Gram's stain, within 6 months of boarding. It may be prudent to require a psittacosis PCR test at the same interval. This not only reduces the potential that sick birds will be housed with healthy birds, but also provides client loyalty for the clinic. Birds that have not been tested or have test results pending must stay in an isolation room until the test results have been received. An extra daily fee is justified for housing in isolation.

TOYS FOR BIRDS

Many clients will purchase toys for their boarding and recovering birds. This is good for the bird and also generates income for the clinic. Alternatively, the clinic can provide inexpensive toys such as native nontoxic woods or palm leaf pieces (Fig 1.50).

Fig 1.51a | A cockatiel in plexiglas container.

Fig 1.51b | Acrylics are lighter and more scratch resistant than plexiglas.

Fig 1.51c | Hospital intensive care unit[p] with toys.

Fig 1.52 | A plastic tub, its top replaced with wire and with a heating pad underneath, makes a practical homecare unit.

Fig 1.53 | A shower stall is made into a cage by placing a flexible panel used to block doors for toddlers. This makes a practical, easily cleaned unit for water birds that need frequent cleaning with large volumes of water.

HOSPITALIZATION

The hospitalization room should be a quiet environment, separated from the boarding and isolation areas. Birds requiring supplemental heat should be housed in incubator-type enclosures. Many of these are equipped with a heat and humidity source. Most sick birds should be kept at a temperature of 26.7 to 29.4° C (80-85° F) and humidity of 70% (Fig 1.51a-c). A plastic container can be used as a brooder by cutting a section out of the top and replacing it with wire (Fig 1.52). It is convenient to have hospital supplies, such as frequently used medications, syringes, needles and electrolyte solutions, located in the hospitalization room. Supplemental oxygen should be located in the hospitalization room for use with patients requiring oxygen therapy. Commercial brooder-incubators[p] are available that allow control of humidity and temperature. The authors have found it easier to keep sick and recovering birds warm if air conditioning vents are not installed in hospital rooms; this also reduces air from these rooms circulating into well-bird areas. A simple electric space heater can be used to increase the temperature of bird rooms needing warmer temperatures. Heating pads under cages for heat and other forms of dry heat can cause dehydration. Humidity should be added as needed.

ISOLATION

An isolation area is necessary for housing untested birds or birds with unknown illnesses away from other birds in the clinic. Ideally, the isolation room should have a ventilation system that is separate from the main clinic ventilation system. Staff should be instructed to strictly adhere to precautionary measures associated with isolated birds. These include treating isolated birds last and having scrubs, gowns, gloves, shoe coverings and cold sterilization dishes that are to be used only for isolated birds. Staff also should be instructed on proper room disinfection procedures.

The authors' clinic uses a chemical autoclave that employs a form of alcohol rather than water for vapor. This machine is kept in the isolation room and birds are moved to a bathroom when autoclaving; thus, the fumes help keep this room sterile. Other than the bathrooms, this is the only room with an exhaust fan in the clinic. The clinic's isolation area contains a shower to house large waterfowl such as pelicans and swans (Fig 1.53). This area can be hosed down as needed to help contain odor and mess.

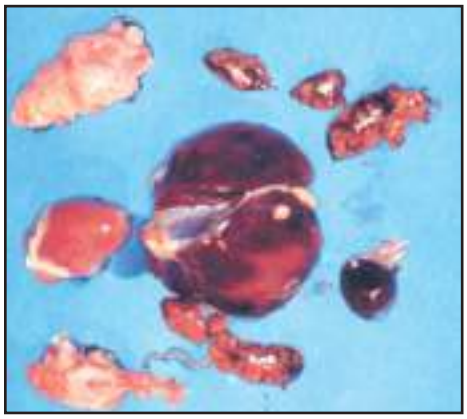


Fig 1.54 | Necropsies should be performed as often as possible in avian medicine to add to both the individual veterinarian's and the avian veterinary community's collective knowledge. A wide selection of tissues must be collected for histopathologic confirmation.

Fig 1.55 | A locked safe is used to store controlled drug and may be bolted to the floor.

In larger hospitals and those that also offer hospitalization and/or boarding for dog and cat patients, physical distance can serve as additional separation between multiple sick birds that need to be isolated. Fortunately, few contagious diseases of birds are commonly contagious to pet dogs and cats. Attention must then be paid to appropriate temperature, humidity, and lack of stressful stimuli when traditional companion pets are present with clinically ill avian patients.

CLIENT RELATIONS

Once a clientele has been established in the practice, it is important to make sure they return on a regular basis for continued care of their birds. Appointment reminders for semi-annual exams and laboratory testing, including Gram's stains and other recommended tests, should be sent out every 6 months. Client information folders[l] (see Fig 1.1b) can be given to clients so they have a record of body weights, health history and identifying characteristics of their bird. These are updated at each visit. Designing a client library that includes an avian veterinary medical text is also a good idea (see Fig 1.1b), although avian textbooks contain medical terminology that clients might have difficulty understanding.

Education and communication are important aspects of maintaining good client relations. Handouts with current information on pet bird health and disease are useful in clarifying information that was covered during the office visit. Topics that might be addressed include metal toxicosis in birds, importance of nutrition, safe diet conversion and zoonotic diseases of concern. Behavioral issues become more prevalent with increased length of ownership and are becoming more pervasive in our current pet bird population. Based on the bird's species, age, temperament and relationship with its owners, each visit may be accompanied by the discussion of a specific behavioral concern. In addition, there are educational newsletters[m] available for client distribution. Development of a clinic newsletter can be a positive addition to client education, retention and recruitment. This can cover new innovations in bird health as well as inform clients about new developments within the practice. Newsletters can be placed on the clinic web site. Educational brochures can be ordered[m] to place in the reception area. These contain information regarding various pet bird species, first aid, signs of illness, specific diseases, nutrition and hand-feeding[n]. Bird species identification books tend to be popular with clients. Having both normal and abnormal feathers available will demonstrate to clients the difference (see Figs 6.57k,l) between healthy and unhealthy conditions of their bird's feathering.

Educational material is available through companies that manufacture pet bird products such as food, cages and toys. Furthermore, most of the companies have web sites, and clients can communicate directly with company representatives via e-mail. With the advent of computer technology, web sites have become powerful tools for providing information to people all over the world. Setting up a clinic web site enables thousands of people to see pictures of clinic facilities and staff and to communicate questions directly via e-mail. A hospital web site with recommended links can also direct clients and potential clients to reliable sources of information on the Internet, providing a counterpoint to misleading and inaccurate information that can be encountered elsewhere on the web. As a convenience, appointments can be made via e-mail as well. E-mails can be sent to clients for follow-up office visits, and appointment reminders

also can be sent via e-mail. A clinic web site can provide an opportunity for clients to download the clinic newsletter, calendar of events or other educational information about pet bird health.

Another aspect of client satisfaction is conveying the message that the staff and veterinarians take a personal interest in the client. This can be accomplished by sending thank you notes for referrals and to thank new clients for their business. Clients also are very appreciative of expressions of sympathy such as cards, flowers or donations in their bird's honor when they have lost a beloved pet.

Record Keeping

COMPUTER SOFTWARE

Increased computer technology has brought many advances to veterinary medicine. One of the most important has been the increased efficiency and accuracy of record keeping through computer software developed for veterinary practices. Although there is no computer program currently available specifically for avian practice, it is possible to adapt small animal programs for use in avian practice. Estimates for future hospital services can be written and stored in the computer program. When an estimate is needed, the information can be easily accessed and modified, and presented quickly and accurately. Photographs and radiographs can be loaded into the patient's record. This is an advantage when records must be transferred or a patient is being referred, because the information can be electronically transmitted to another clinic, saving time and eliminating lost records. Computer programs are able to keep track of each client's visit and generate timely reminder cards. Computer programs store billing history and highlight bills that are overdue, making billing more consistent. Computer software also prints and records drug labels, which precludes mistakes due to illegible handwriting on labels, and allows directions for previous therapeutics prescribed to be retrieved. In addition, software allows auditing, individual access and monitoring for security measures.

DIGITAL CAMERAS

Another advance in record keeping is the development of digital cameras. Digital images allow information to be shared quickly among veterinarians via the Internet. With digital cameras, it is possible to capture images of the patient and any related conditions or procedures. Radiographs can be digitally recorded and the file sent to another practitioner without concern for the physical possession of the radiographs and associated legal implications.

Additionally, the use of digital cameras for documenting necropsy findings can be invaluable. The complete necropsy shown in Fig 1.54 involves opening the skull and demonstrates the value of the camera while illustrating a critical portion of the necropsy that is often omitted by many practitioners.

For the highest quality pictures, digital cameras should have a built-in macro capability (to enable good close-ups) and should be capable of image densities of two megapixels or greater.[3]

There are special adapters that allow cameras to be mounted on microscopes to capture microscopic images, while the addition of macro lenses and built-in macro functions of some digital cameras will create exceptional images when placed directly onto one of the eyepieces of a microscope.

PAPERWORK

Although increasing numbers of veterinary hospitals have become virtually paperless, there are still several situations that are best addressed by signature authority. Authorization forms document the client's understanding of and consent to having a particular service or services performed and the terms of that service. Authorization forms are commonly used in avian practice, as are forms that limit liability.

SECURE ITEMS

Keeping cash, controlled drugs and vital records in a fireproof safe is important for security. Fireproof containers have a lining that generates moisture upon heating, making combustion less likely (Fig 1.55). These containers are guaranteed for only 5 years, as this function deteriorates over time.

CONTROLLED DRUGS

Controlled drugs, radiology monitoring, medical waste and OSHA training are areas covered by federal and state laws in the USA. Such subjects need to be reviewed with your national, state and local authorities.

General Information Avian Practitioners Should Have Available

TRAVEL FORMS

On many occasions, people wish to take their pet birds

with them while traveling. In some situations, it is necessary to have travel forms that permit the bird to accompany the owner.

Travel by car within the USA requires Interstate Health Certificates.

When traveling by airplane within the USA: Form SA-B (Official Certificate of Veterinary Inspection for Interstate Movement of Dogs, Cats, and Other Non-livestock Species) must be filled out by an accredited veterinarian. APHIS form 7001 (US Interstate and International Certificate of Health Examination for Small Animals) also may be used for this purpose.

Travel by airplane outside the United States: An accredited veterinarian must fill out APHIS form 7001. The bird owner must then mail or hand-carry this completed form to the designated USDA-APHIS office, along with a processing fee. The form will then be approved by a USDA veterinarian and sent back to the owner to accompany the bird during travel. Because of the time needed to process this form, owners should be advised that planning ahead is imperative in order to receive the completed paperwork in time for travel. Additionally, the country to which the bird is being exported may have its own forms that must be completed prior to exportation, and may have differing requirements regarding testing, vaccination and identification. The owner should check with that country's embassy in the USA prior to making travel plans.

Travel with poultry or hatching eggs for export: There is a special form for poultry, VS form 17-6. Unless the birds are hatching eggs and newly hatched poultry, the veterinarian must go to the premise to inspect the birds in order to validate the travel form.

Contacts for Further Information

Division of Animal Husbandry (850-488-8280)

The United States Department of Agriculture (305-526-2926)

www.Aphis.USDA.gov/travel/pets.html

www.Aphis.USDA.gov/us/sregs - US State and Territory Animal Import Regulations

PSITTACOSIS INFORMATION

The Committee of the National Association of State Public Health Veterinarians has compiled a compendium of psittacosis control. The compendium discusses psittacosis infection among humans and birds, transmission, clinical signs and symptoms, case definitions, diagnosis, treatment, and recommendations and requirements. Copies of the compendium can be accessed at the Center for Disease Control web site (www.cdc.gov/ncidod) and at the web site for the American Veterinary Medical Association (www.avma.org).

IMPORT-EXPORT OF PET BIRDS

Most parrots are classified as endangered species, which require a special Conference on Endangered Species (CITES) permit to be imported or exported to or from the USA. See Table 1.1 and the discussion on Regulations in Chapter 2, The Companion Bird.

Products Mentioned in the Text

a. Karl Storz Veterinary Endoscopy-America, Inc, Goleta, CA, US, 805-968-7776, www.karlstorz.com

b. Endoscopy Support Services, Brewster, NY, US, 800-fix-endo, www.endoscopy.com

c. Miami Vise, Veterinary Specialty Products, 800-362-8138, vetspecpro@aol.com, www.vet-products.com

d. Poly Perches, 888-765-5971, www.pollyspetproducts.com

e. Dremel, Racine, WI, US, 800-437-3635, www.dremel.com

f. Kras Avian Leg Band Cutter, Veterinary Specialty Products, P.O. Box 812005, Boca Raton, FL, US, 33481, 800-362-8138, vetspecpro@aol.com, www.vet-products.com

g. Feeding Tube and Urethral Catheter, Sovereign, Sherwood Medical, St. Louis, MO, US

h. Silicone feeding tubes or human female catheters, Veterinary Specialty Products, 800-362-8138, vetspecpro@aol.com, www.vet-products.com. Also catheters and tubes A/S, DK-3540 Lynge Denmark or AUV Veterinary Surgeons Cooperative, The Netherlands (31)4855-3355-55

i. AVID, 3179 Hamner Ave, Norco, CA, US 92860, 800-336-2843, www.avidid.com

i1. Home Again. Schering Animal Health, www.homeagainid.com/vets/index.cfm

j. Acrylic perches. Lyon Electric Company, Chula Vista, CA, US, 619-216-3400

j1. Leadcheck Kit/Hybrivet Systems, Inc., Farmingham, MA

k. Steamfast - a small steam generator available from appliance stores

l. Zoological Education Network, PO Box 541749, Lake Worth, FL, US, 33454-1749, 800-946-4782, www.exoticdvm.com, info@exoticdvm.com

m. Avian Examiner, Harrison's Bird Foods, HBD International, Inc. Brentwood, TN, USA, 615-221-9919, www.harrisonsbirdfoods.com

n. Juvenile Hand Feeding Formula, Harrison's Bird Foods, HBD International, Inc. Brentwood, TN, USA, 615-221-9919, www.harrisonsbirdfoods.com

o. General Scientific Corp., 800-959-0153, www.surgitel.com

p. Lyon Electric Co. Inc., 1690 Brandywine Ave., Chula Vista, CA 91911-6021 US, www.lyonelectric.com

Table 1.1 | Avian Veterinary Medical Organizations and Web Sites

Organization	Website
American Animal Hospital Association (AAHA)	www.aahanet.org
American Board of Veterinary Practitioners (ABVP)	www.abvp.com
American Veterinary Medical Association (AVMA)	www.avma.org
Association of Avian Veterinarians (AAV)	www.aav.org
Association of Avian Veterinarians Australian Committee (AAVAC)	www.vet.murdoch.edu.au/birds/aav/join/htm
European College of Avian Medicine and Surgery (ECAMS)	www.ECAMS-online.org
Mid-Atlantic States Association of Avian Veterinarians	www.masaav.org/contact.htm
USDA-APHIS US State and Territory Animal Import Regulations	www.aphis.usda.gov/vs/sreqs/
APHIS - US Fish and Wildlife Service	www.permits.fws.gov (click on Import/Export)

Table 1.2 | Journals and Sources of Information on Avian Medicine

Information	Contact
American Journal of Veterinary Research	www.AVMA.org
Avian Diseases - Journal of the American Association of Avian Pathologists	www.aaap.info/audis
Avian Examiner	www.avianmedicine.net
Avian Pathology (UK)	www.tandf.co.uk/journals/tf/03079457.html
Veterinary Record - In Practice	www.vetrecord.co.uk
Compendium of Small Animal Practice	www.VetLearn.com
Exotic DVM Magazine and Exotic DVM Readers' Forum	www.exoticdvm.com
Journal of Avian Medicine and Surgery	www.aav.org
Proceedings American Association of Zoo Veterinarians and Journal of Zoo and Wildlife Medicine	www.aazv.org/aazv_001.htm
Proceedings Association of Avian Veterinarians (AAV)	www.aav.org
Proceedings Association of Avian Veterinarians Australian Committee	www.vet.murdoch.edu.au/birds/aav
Proceedings European College of Avian Medicine and Surgery	www.ecams-online.org
Proceedings of the European Committee of the Association of Avian Veterinarians	www.eaav.org
Seminars in Avian and Exotic Pet Medicine	www.us.elsevierhealth.com/product.jsp?isbn+1055937X

Table 1.3 | Avian and Veterinary-oriented Web Sites

Subject	Comments	Contact
Avian Medicine On-line	Password needed to enter	www.avianmedicine.net
Birdmed Discussion List	Password needed to enter For veterinarians, students, technicians, free archives	www.vet.murdoch.edu.au /birds/birdmed.htm
Exotic DVM	For veterinarians and veterinary students only	www.exoticdvm.com
Veterinary Information Network	Password needed to enter By subscription, access to archives	www.vin.com
Exotic Animal Network	Free	www.exoticanimal.net/
Avian and avian medicine-related links	Part of Birdmed – see above	www.vet.murdoch.edu.au /birds/aav/avi-links.htm

References and Suggested Reading

1. Cantarzare TE: Compliance begins and ends with the veterinary team. DVM Best Practices Supplement of DVM Magazine, July 2003, pp 4-7.
2. Johnson-Delaney CA (ed): Avian Emergency Care: A Manual for Emergency Clinics. Seattle, Assoc of NW Avian Vet, 1991.
3. Kramer M: Practical digital imaging for the exotic animal practitioner. Exotic DVM 4(4):33-35, 2002.
4. Herschell GL: Big Profits from Small Budget Advertising. Publishing information about this book may be found at www.amazon.com.
5. McMillan MC: Imaging techniques. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1994, p 260.
6. Perry RA: The avian patient. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1994, pp 42-43.
7. Zantop DW: Differentiating abdominal swelling in birds with ultrasonography. Exotic DVM 2(3):11-12, 2000.

CHAPTER
2

The Companion Bird

ANGELA M. LENNOX, DVM, Dipl ABVP-Avian and
GREG J. HARRISON, DVM, Dipl ABVP-Avian, Dipl ECAMS

Jan Hooimeijer

More veterinarians than ever are willing to treat companion birds. The quality of avian medicine has greatly improved over the years as evidenced by the increase in numbers of professional publications, scientists pursuing companion bird research and continuing education opportunities. In the United States, the Association of Avian Veterinarians (AAV) began in 1980 as a group of 175 veterinarians. Today, membership tops 3300 veterinarians from 43 countries.[5] The explosion of new information, treatment and surgical protocols provides opportunities to practice avian medicine at very high levels. This also represents a double-edged sword, as those wishing to provide veterinary care for companion birds must be willing to practice at this advanced level and stay abreast of the current standard of care. It is no longer acceptable for veterinary professionals to proclaim the pet to be "just a parakeet" and inform the owner there is not much that can be done.

In 1993, the American Board of Veterinary Practitioners established a board specialty for avian practice, with rigid requirements for certification. In 2002, there were 92 board-certified avian specialists in 22 states, and in Canada and the Netherlands.[1] Outside the USA, the European College of Avian Medicine and Surgery (ECAMS), established in 1993, and Australia's avian veterinary specialist program provide similar advanced degrees.[8] Certified avian specialists should be viewed as a valuable resource for those desiring second opinions and referrals on difficult cases. As in any medical referral situation, human or veterinary, referring veterinarians should provide complete medical records including radiographs and laboratory results to the referral veterinarian and expect a timely response, written report and complete follow-up recommendations.

Pet Bird Ownership in the United States

The popularity of pet birds, especially psittacines, remains strong.[2] However, an American Veterinary Medical Association (AVMA) survey covering 1991 to 2001 showed that the percentage of households owning pet birds and the actual numbers of birds kept as pets had decreased. In that time period, numbers of birds owned as pets decreased from 11 to 10.1 million.[4] According to most recent statistics, more than half of USA households own a companion animal, and of these households, approximately 4.6% own pet birds. Yet another survey covering 2001 to 2002 by the American Pet Products Manufacturing Association (APPMA) showed 6.9 million homes owned a bird. This survey conflicts with data generated by AVMA and actually reports the number of households owning birds as slightly increasing.

Birds remain the most popular specialty or exotic pet, second only to fish. As a comparison, less than 2% of households owned pet rabbits. All other exotic pets surveyed, including ferrets and reptiles, were well below one half of 1%. Practitioners seeing even modest numbers of pet birds will affirm APPMA statistics proclaiming the cockatiel as currently the most popular pet bird species in the USA (Table 2.1).

Bird ownership was strongest in the Pacific and Mountain regions of the USA in 2001, while the lowest percentage of bird owners lived in the West, North and Central regions.

Table 2.1 | Numbers and Types of Avian Patients Seen in 1 Year at a Busy Avian Practice[16]

Patient	#	Patient	#	Patient	#
Blue-fronted	44	Nanday	9	Hybrid	14
Double yellow-headed	32	Mitred	6	Bronze-winged	3
		Sun	22	Maximillian	7
Lilac-crowned	15	Red-sided	11	White-capped	9
Orange-winged	8	Solomon	20	Budgerigar	99
Red-lored	11	Grand	2	Cockatiel	240
Spectacled	10	Vosmaeri	9	Lovebirds	50
Yellow-naped	40	Blue and gold	90	Senegal	33
Goffin's	38	Blue-throated	2	Indian ring-necked	10
Lesser sulfur-crested	10	Green-winged	19		
		Hahn's	11	Canary	20
Moluccan	46	Hyacinth	12	Congo A. Grey	122
Umbrella	59	Illiger's	1	Timneh A. Grey	16
Galah	15	Military	7	Chicken	18
Blue-crowned	20	Scarlet	13	Duck	16
Green-cheeked	20	Severe	14	Dove	5
Jenday	7	Yellow-collared	8	Flock consult	39

Typical pet bird owners do not fit a general profile, although a few statistical generalizations can be made. More owners are couples rather than single, and the majority have at least two children. Personal income does not seem to influence the likelihood of bird ownership, but level of education apparently does. Persons with advanced college degrees are much *less* likely to own pet birds. Bird owners are slightly more likely to live in urban rather than rural areas. Therefore, the "typical" bird owner in the USA may be a young couple with undergraduate college degrees, with two children, living in a large metropolitan area with a single pet bird.

Companion bird ownership appears to be popular outside the USA as well. In Australia, pet bird ownership apparently is even more popular as approximately 17% of households own a bird, with an average of 8.7 birds per household (R. Doneley, personal communication).

Frequency of Veterinary Care

The AVMA survey indicated both good and bad news for avian practitioners. On the negative side, pet bird owners overall are not likely to seek veterinary care. In 2001, only 11.7% of bird owners in the USA reported at least one veterinary visit. In comparison, 83.6% of dog owners and 65.3% of cat owners reported at least one veterinary visit in 2001. On the positive side, however, a 6-year survey indicated the average number of veterinary visits for pet birds actually increased. An estimated 2 million avian veterinary visits occurred in 2001, compared to 1.6 million in 1996. This represents a solid increase in demand for the services of avian veterinarians. More evidence for this conclusion can be seen in the fact that veterinary expenditures for bird owners increased dramatically from 37 million dollars in 1991 to 135 million dollars in 2001.

It is interesting to note those veterinary services most commonly purchased for pet birds. Examinations are purchased most frequently, followed by laboratory tests, then emergency care. In comparison, emergency care is not even listed in the top five services most commonly purchased by dog and cat owners. This does not suggest that emergency care for dogs and cats is uncommon. However, it does support what many avian practitioners already suspect. While many bird-owning clients appreciate the value of preventive medicine, far too many others consult the avian veterinarian only in time of medical crisis.

Slightly more than half of surveyed clients selected their regular dog and cat veterinarian to provide care for their

Greg J. Harrison

Fig 2.1 | While undoubtedly popular, birds like canaries and finches are not presented as frequently for veterinary care as are birds more likely to bond with their owners.

avian pets. Encouragingly enough, 24.2% made their selection based on the fact that the veterinarian was a bird specialist. (Note that this survey does not distinguish between veterinarians who are board-certified specialists and those claiming a "special interest" in avian medicine). Discouragingly, just as many clients chose a veterinarian based simply on location.

It is obvious avian practitioners have a great deal of work to do to catch up to our fellow dog and cat practitioners. While bird owners who do seek regular veterinary care are generally seeking a higher quality of care and more frequent visits for their pets, it is obvious the great majority of bird owners either are unaware such services are available or not convinced of their value.[4]

The Human-Bird Bond

There is no doubt that many owners develop a deep attachment to their birds, due in part to their relative longevity. A recent survey of bird-owning clients of a busy avian practice revealed that most owners consider their pets equal in importance to family members.[11] This must be contrasted, however, to the growing problem of unwanted birds, to which organizers of parrot rescue facilities can readily attest. Human-bird interaction studies indicate that birds play many of the same roles for people as do dogs and cats. Some significant differences between human-bird and human-dog/cat interactions exists. More effort is required by the bird owner to elicit a positive response from their pet. Birds require more time to train than dogs and cats and lose pet quality faster when there is no regular interaction. It has been theorized that birds may be a more consistent stimulus for calming interaction than other pets, as owners must approach birds in a quiet, non-threatening manner to maintain a satisfying relationship.[6] Birds that require less interaction and typically do not bond to owners, like finches and canaries, are less often seen by veterinarians (Fig 2.1).[16]

Pet Loss, Grieving and Euthanasia

Most owners bonded to their pets go through a grieving process of variable intensity in the face of loss of their pet. Many choose to be present with their bird during euthanasia. This necessitates that the avian veterinarian be competent and comfortable with an anxiety and pain-free euthanasia process. In 2000, AVMA published a guide to humane euthanasia techniques for many pet species. Included in the list of acceptable techniques for birds was thoracic compression.[15] The AAV responded with an editorial requesting this technique be stricken from the list. In most situations, euthanasia can be best accomplished by first inducing general inhalant anesthesia. Euthanasia solution can subsequently be administered by intravenous injection. This technique, performed in a quiet, private area with veterinary personnel relating to the patient in a gentle, compassionate manner, is usually gratefully accepted by grieving owners.

The same survey of pet bird owners mentioned above indicated that the majority of owners would, in the event of the death of their pet bird, choose private burial on their own property. A surprising number, however, stated they would select individual cremation with return of ashes.[11]

Very few owners indicated they had provided for their pet in a formal or legal will in the event of their own death. The great majority of owners, however, said they had already discussed the possibility and made informal arrangements for continued care of their pet.[11]

History of Pet Bird Ownership

The literature is full of tantalizing, although not completely documentable, references to pet parrots in history. The earliest reference may be Ctesia's *Indica*, which contains a reference to a bird resembling a plum-headed parakeet (*Psittacula cyanocephala*). Aristotle gave the name Psittace to a similar bird he described. Frederick II (1194-1250) was said to enjoy the company of an umbrella cockatoo given to him by the Sultan of Babylon. In 1492, Columbus brought back a pair of Cuban Amazons to Queen Isabella of Spain. The first sighting of an Australian bird by a European was said to

be on August 22, 1699, when William Dampier spotted a flock of little corellas off the northwestern coast of Australia.[12] A reference on Aztec burial customs reports the burial of a prince together with a macaw. It is fascinating to consider the improbable relationship of the stereotypical pirate and his pet parrot.

Regulations Concerning Pet Birds

While regulations exist in the USA prohibiting the ownership of some native wild birds, there are few restrictions concerning pet bird ownership. Some communities prohibit the keeping of birds that are considered to be farm animals, such as geese and chickens. Some apartment dwellings and condominiums include birds in pet ownership restrictions. International trade in birds for the pet trade, however, is regulated by the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES). CITES is an international agreement between governments to ensure that trade does not threaten the survival of wild animals and plants. CITES entered into force in 1975 and includes over 150 participating governments protecting approximately 5000 species of animals and 25,000 species of plants. Since the adoption of CITES, not a single protected species has become extinct as a result of international trade. Protected species are classified into three Appendices, listed I, II and III. Appendix I species are threatened with extinction, and trade is prohibited with exceptions made for specific circumstances, such as scientific research. Appendix II species are not threatened but may become so if trade is not restricted. Trade in these species must be approved and an export permit granted. Appendix III species may be legally traded, but are listed in order to solicit cooperation of other countries to ensure trade is not unsustainable. Specific permits also are required.

Under the classification Psittaciformes, 44 species are listed under CITES Appendix I, including 13 Amazon and 6 macaw species. Within the USA, CITES is enforced by United States Fish and Wildlife Service division of Management Authority.[7] Legal importation of CITES I-, II- and III-listed species to the USA officially ended in 1993 with passage of the Wild Bird Conservation Act. Birds legally imported to the USA prior to this act still may bear an import band placed at the time of entry into a USA quarantine station. Quarantine bands are easily recognized and are imprinted with three letters and three numbers. Once birds leave quarantine, there is no legal requirement to retain the band, and most have since been removed. Domestically bred birds are commonly

Greg J. Harrison

Fig. 2.2 | The solid aluminum breeder band is often inscribed with letters (breeder and state) and numbers (year of birth and ID number) The "TX" means this bird was bred in Texas.

close-banded within weeks of birth. (Fig 2.2) Closed bands are difficult to remove. They have no legal meaning other than identification. Using one of these bands to trace the origin of a bird is nearly impossible.

Although illegal importation exists, the proliferation of large corporate and small "backyard" parrot breeders supplies the pet trade with an ample number of birds at reasonable prices. Many breeders place a band on their birds when they are very young. Breeders may inscribe bands with any combination of symbols. A typical breeder band may contain a set of letters identifying the breeder, a two-letter combination indicating state of hatch, and two numbers signifying year of hatch. Interstate movement of any bird, including pet birds vacationing with owners, requires a state-issued health certificate completed by a licensed and accredited veterinarian indicating the bird is free of signs of illness. In addition, the destination state may require additional testing before the bird can cross state lines. Requirements are obtained by phoning the destination state's Board of Animal Health, or looking up requirements on each state's individual web site. That being said, many owners are oblivious to these regulations and do not request health certificates when they travel.

The destination country similarly determines requirements for entry into foreign countries. Most countries require the bird to be identified with a leg band or microchip. Requirements can be obtained by phoning the consulate office of the destination country. In the USA, international requirements can be obtained by calling the local US Department of Agriculture-Animal and Plant Health Inspection Service office (USDA-APHIS). Alternatively, requirements are posted on the USDA-APHIS web site, which also contains information for traveling with birds into the USA from foreign countries.[17]


Greg J. Harrison

Fig 2.3 | The Endangered Species Act forbids the keeping of Queen of Bavaria (or golden) conure (*Aratinga guarouba*) for commercial purposes. Feather picking is common in this species.


Chris Migliore

Fig 2.4 | Moluccan cockatoos (*Cacatua moluccensis*) appreciate human attention, even if it means wearing a costume.

The movement of CITES-protected species is not regulated within the USA; however, international travel with these species requires a special permit. Information on travel from the USA with CITES-protected species may be obtained from the United States Fish and Wildlife Service (USFWS).[9]

The Endangered Species Act was passed in 1973. This act is enforced by USFWS, which regulates commerce concerning endangered species. Any bird listed as threatened or endangered may not be traded in interstate commerce. The Queen of Bavaria or golden conure (*Aratinga guarouba*) is listed as endangered as well as several other species occasionally seen in pet practice (Fig 2.3). Under the provisions of the Endangered Species Act, these species may not be sold in interstate commerce or kept for commercial purposes.[9] Government regulations concerning companion birds are further discussed in Chapter 1, Clinical Practice.

Birds in Schools and Care Facilities

Birds are gaining in popularity in nursing homes and other care facilities. Birds most commonly seen in care facilities are canaries, budgerigars, cockatiels and lovebirds (Fig 2.4).[13] Many studies suggest the benefits of birds and other animals in care facilities. One experimental study documented better attendance and less hostility in group therapy meetings of psychiatric patients in rooms containing finches.[6] Local health departments may require veterinary examinations and periodic testing for animals in contact with residents. Veterinarians performing these exams must be aware they cannot certify that any bird is completely free of potentially zoonotic diseases, in particular, chlamydiosis (see Chapter 27, Update on *Chlamydophila psittaci*: A Short Comment). Facility and health department personnel, along with the veterinarian, must determine a reasonable amount and frequency of testing to minimize health risk due to the presence of pet birds, especially in the presence of persons with diseases compromising the immune system. New additions should be quarantined away from existing birds and residents for at least 45 days.[9] Attention must be given to proper cage construction, dietary requirements and cage additions, such as perches and toys. Birds in care facilities occasionally are subjected to multiple caregivers, or are in the care of persons with no training or interest in their well-being. Caretakers must receive adequate training in all aspects of care of their charges and be familiar with common signs of illness.

Birds and Animal Welfare

Many well-known animal welfare groups have taken stands for and against pet bird ownership. In 2002, the American Society for the Prevention of Cruelty to Animals (ASPCA) proclaimed January as "Adopt a Rescued Bird Month." The ASPCA web site contains links to a directory of birds available for adoption and worthy information on the pros and cons of pet bird ownership.[3] People for the Ethical Treatment of Animals (PETA), an organization known for aggressive animal rights positions, encourages formulated diets and regular veterinary care for pet birds. The same organization, however, advises against any captive breeding of parrots and encourages owners to allow pet birds to select a companion bird of its own species and to free fly.[14] The Humane Society of the USA, the largest animal welfare

Table 2.2 | General Guidelines for Recommended Pet Birds Based on General Pet Quality

Common name and/or representative species	General traits	Potential concerns	
African Grey - Congo (*Psittacus erithacus*)	The most demonstrably intelligent psittacine sp. Greatest potential for range of vocalizations and increasing vocabulary throughout their lives. Research has documented cognitive association between learned words and both actions and objects.	Intelligent and emotionally sensitive; i.e., prone to remember negative experiences and make associations with people and objects that may develop into phobias/neuroses. Feather destructive behavior is a very common condition in captive African greys.	OW
African Grey - Timneh (*Psittacus e. timneh*) (Darker grey and smaller than the nominate Congo species)	Similar in appearance and characteristics to the nominate species noted above.	Both positive traits (learning ability) and negative traits (neurosis, obsessive behavior) are usually somewhat reduced in this subspecies compared to the nominate species.	OW
Amazons (*Amazona* spp.) • Yellow-naped (*A.ochrocephala auropalliata*) • Blue-fronted (*A.aestiva*) • White-fronted (*A.albifrons*) • Orange-winged (*A. amazonica*) • Mealy (*A.farinosa*) • Festive (*A.festiva*)	Active, fairly hardy. Tend to become bonded to certain individuals and aggressive toward others. Some are excellent talkers (eg, yellow-nape, double yellow-head, blue-fronts).	Screaming, territoriality, and aggression are common. Learn quickly to use lunging or biting to relay their negative opinions. Quieter species include: spectacled (white-front) orange-winged, mealy, festive	NW
Budgerigars (*Melopsittacus undulatus*)	Can be interactive, enjoyable pets.	Genetic predisposition to many diseases and neoplastic conditions.	OW
Caiques (*Pionites* sp.) • Black-capped (*P. melanocephala*) • White-bellied (*P. lucogaster*)	Beautiful, small parrots, playful personalities	Although difficult to locate, this genus is a favorite recommended species. Not known for their talking ability.	NW
Cockatiels (*Nymphicus hollandicus*)	Intelligent, popular pets. Can become very attached to the owner or to conspecifics.	Chronic egg-laying in some females. Aggression may develop in males as they mature (especially toward children). Color mutations may be more prone to illnesses.	OW
Cockatoos general (*Cacatua* sp.)	Enjoy physical contact. Vocabulary is limited but intelligible and often endearing.	Screaming, mate aggression (conspecific or surrogate) may be severe. Occasional unpredictable severe biting episodes, even with humans to which they are bonded. (Note: Powderdown production is pronounced, and is not only a cleaning concern, but can cause allergic reactions in some people and in some macaws)	OW
Smaller *Cacatua* sp. • Goffin's (*C. goffini*) • Red-vented (*C. haematuropygia*) • Bare-eyed (*C. sanguinea sanguinea*)	More active than other, larger cockatoos.	Can be hyperactive. Not as predictably accepting of cuddling and petting.	OW
Larger *Cacatua* sp. • Umbrella or white cockatoo (*C. alba*)	Enjoy cuddling, petting, and prolonged physical contact.	Can develop behavioral and medical problems, (screaming, feather destructive behavior, self-mutilation, vent prolapse) related to their demand for physical stroking and/or other psychological captive abnormalities.	OW
Moluccan or salmon-crested cockatoo (*C. moluccensis*)	As with umbrellas, but can be less predictable and even aggressive.	Often escape artists. Behavior problems, as with umbrella cockatoos above, can occur. May be very destructive with their beaks.	OW
Conure (*Aratinga* sp.) • Sun (*A. solstitialis*) • Jenday (*A. jandaya*) • Gold-capped (*A. auricapilla*)	Beautiful, intelligent birds.	Loud, high resonance screams. Can become territorial. Not known for their talking ability.	NW
Conure • Nanday (*Nandayus nenday*)	Historically, were common imports and relatively inexpensive. Captive-raised individuals can make excellent pets.	Established feral colonies of nanday conures exist in parts of south Florida. May develop loud and persistent screaming behavior.	NW
Conure • Patagonian (*Cyanoliseus patagonus*)	Beautiful, larger conure. Relatively quiet.	Historical documentation as carriers of Pacheco's disease virus has made owners wary of introduction into their collection. This may still be a valid concern in multiple-bird households.	NW
Conure (*Pyrrhura* sp.) • Green-cheeked (*P. molinae*) • Black-headed (*P. rupicola*) • Maroon-bellied (*P. frontalis*)	Smaller and generally quieter than *Aratinga* sp. conures.		NW
Doves, Pigeons (Columbiformes)	Gentle, excellent pets. Although the degree of interaction (vocal, body posturing) is limited, there is little or no danger of injury to humans from bites.	If raised by humans may have no fear or defense against dogs or cats	OW NW
Eclectus sp. (ten subspecies) • Red-sided (*E. roratus*) • Vos (*E. vosmaeri*) • Solomon Island (*E. solomonensis*)	Most pronounced sexual dimorphism of any psittacine. "Pensive" when considering novel items or situations in a secure environment, leading to the misconception that eclectus are dull-witted. Moderately good talkers. Males tend to be more docile than females.	Unless socialized early, may become alarmed by new situations or locations. Feather destructive behavior common. In breeding situations, females will often traumatize males.	OW
Finches, Canaries (Passerines)	Easy to care for, quiet, pleasant vocalizations. Limited ability to interact with their owners as compared to psittacines.	Inbreeding has created genetic predispositions to multiple disease syndromes in some lines.	OW

Table 2.2 | General Guidelines for Recommended Pet Birds Based on General Pet Quality

Common name and/or representative species	General traits	Potential concerns	
Hyacinth macaw (*Anodorhynchus hyacinthinus*)	Largest psittacine. Beautiful bird. Temperament can be calmer than other macaws. The attending veterinarian needs to be aware of specific nutritional needs and pharmacologic sensitivities.	Possibly due to genetics or captive rearing limitations, this species can become neurotic/phobic. Research into the parents' temperament is recommended. Expensive.	NW
Lories and Lorikeets • Rainbow (*Trichoglossus haematodus*) • Red (*Eos bornea*) • Dusky (*Pseudeos fuscata*)	Ring-necks and lories were previously considered aviary birds, but can be quite tame when captive raised. Beautiful colors and brilliant sheen to feathers.	Fruit and nectar diet makes droppings messy. As with *Aratinga* sp, their beak sharpness and their speed make bites, if they occur, painful.	OW
Lovebirds (*Agapornis* sp.) • Fischer's (*A. fischeri*) • Peach-faced (*A. roseicollis*)	Can be very tame and bonded to people or other birds.	Can be very aggressive during breeding season.	OW
Macaws (*Ara* sp.) • Blue and gold (*A. ararauna*) • Green-winged (*A. chloropterus*) • Scarlet (*A. macao*)	Large, physically active, vocal birds. Intelligent, highly interactive and energetic. Require frequent training and structured play to focus their energies.	Need physical outlets for their abundant energy. Loud; screaming can become a problem. Generally develop a limited vocabulary. Learn tricks readily. Require a knowledgeable owner.	NW
Mini-macaws • Yellow-collared (*Ara auricollis*) • Noble (*A. n. cumanensis*) • Severe or chestnut-fronted (*Ara severa*)	Can be excellent, affectionate and intelligent pets.	Common as imports in previous decades. Few were bred in captivity following cessation of importation. Therefore the current availability is low and the genetic pool is limited for many species.	NW
Mynahs • Indian hill mynah (*Acridotheres tristis*)	Excellent mimics. Have the same interactive limitations as the small passerines.	Stools are projectile and messy. Prone to iron storage disease.	OW
Grass parakeets (*Neophema* sp.) • Bourke's (*N. bourkii*) • Turquosines (*N. pulchella*)	Quiet, easily maintained birds, often kept in aviaries.	Not as readily bonded to people as many other parrots.	OW
***Pionus* sp. Parrots** • White-headed (*P. seniloides*) • Bronze-winged (*P. chalcopterus*) • Dusky (*P. fuscus*)	Usually gentle, smaller and quieter than the related Amazons.	Generally, limited ability to mimic speech compared to Amazons. Produce a rapid "sniffing" sound when frightened that is often mistaken for respiratory disease.	NW
***Poicephalus* sp. Parrots** • Senegal (*P. senegalus*) • Myers (*P. meyeri*)	Playful, active, usually gentle, fairly hardy.	Can become territorial with sexual maturity.	OW
Quaker parakeet (Monk) (*Myiopsitta monachus*)	Intelligent, feisty birds, with moderate talking ability. Hardy, including tolerance of colder environments. Colony breeders.	Can become aggressive. Tendency to become obese and a relatively high incidence of pancreatic problems. Illegal in some US states due to their propensity for establishing feral populations, even in temperate climates.	NW
Ring-necked Parrots (*Psittacula* sp.) • Mustached (*P. alexandri*) • Derbian (*P. derbiana*)	Generally quiet, can be tame and personable. Were previously thought to be "aviary birds" until captive breeding produced tame, human-oriented individuals.	Few, except Old World species disease susceptibility. Some new color mutations may be genetically predisposed to problems.	OW
Toucans (*Ramphastos* sp.) • Keel-billed (*R. sulfuratus*) • Toco (*R. toco*) • Channel-billed (*R. vitellinus*)	Beautiful, fascinating birds. Recognize owners, but limited interaction (may "clack," but do not mimic speech or posture as do psittacines).	Dietary requirements can be difficult to fulfill, including low iron and some live prey. Prone to iron storage disease. Voluminous, messy stool.	NW
Waterfowl • Geese (*Anser* sp.) (*Branta* sp) (*Nettapus* sp.) • Ducks, mallard (*Anas platyrhynchos*) • Muscovy (*Cairina moschata*)	Usually gentle, may be aggressive during breeding. Outdoor environment highly recommended.	Require water for swimming/bathing/drinking. Voluminous stools	NW OW
Waterfowl Swans (*Cygnus* sp.)	Beautiful, but often aggressive.	Not usually tame as adults	

Note: Since disease susceptibility (eg, circovirus and sarcocystosis), nutritional needs and/or dietary sensitives may be dependent upon the area of origin, Old World (OW) vs. New World (NW) is noted in the final species column.

organization in America, considers only canaries, finches, budgerigars, lovebirds and cockatiels suitable as pets. Larger birds are not recommended, and reasons stated against ownership include longevity, specialized needs and demands for care.[10]

The Ideal Pet Bird

While opinions vary on what constitutes the ideal pet bird, Table 2.2 lists commonly kept birds and some of their characteristics relating to pet qualities (Figs 2.5-2.45).

Selective Breeding, Color Mutations and the Future of Companion Birds

Selective breeding has produced a variety of desirable physical and behavioral traits in many species of companion animals. Along with these desirable traits, however, come some that are less desirable or even detrimental to the health of the animal. So-called "puppy mills" in the USA in the 1960-1970s produced large numbers of dogs for the pet market without regard to the quality of animals produced. Practitioners are beginning

to recognize this phenomenon in pet birds, particularly cockatiels (Figs 2.36, 2.37), lovebirds (Fig 2.42) and budgerigars (Fig 2.45). Anecdotal reports indicate these mass-produced birds have an increased incidence of disease, unthriftiness and shorter life spans. In some cases, mass-produced birds are given prophylactic antibiotic and antifungal medications without a medical diagnosis. Pathogen resistance is a clear risk with this practice. Many bird breeders are producing new and novel color mutations of common species. Unusual varieties of cockatiels, budgerigars and lovebirds have been available for many years. Avian practitioners now are seeing unusually colored Quaker parrots and conures as well. Whether or not these mutations are less healthy than their normal counterparts remains to be determined.

An unusual color variation has been seen in African grey parrots. These birds fledge with or later develop pink or red contour feathers over various portions of the body (Fig 2.46). This coloration has been linked to circovirus or a dietary imbalance (see Chapter 4, Nutritional Considerations, Section II and Chapter 13, Integument). Many of these birds, however, do not appear to develop other clinical evidence of illness.

Selecting Healthy Pet Birds

The average pet owner has at least a few choices with regard to selection of a pet bird. The ideal source is a breeder with limited numbers of hand-reared offspring of just a few species. For purposes of disease control, the ideal breeder does not raise larger psittacines in the same premises as smaller birds such as cockatiels, lovebirds and budgerigars. The ideal breeder selects for characteristics that maximize pet quality, such as calmness and docility and spends significant amounts of time raising and socializing young birds and feeds a formulated diet (Fig 2.47). The ideal breeder consults with an avian veterinarian and may offer birds that have been examined or even screened for underlying disease conditions. Table 2.3 summarizes the ideal characteristics of breeding facilities that produce parrots for the pet trade. In many cases, the only source of birds available locally may be those found in pet stores or bird fairs. Buyers must be aware of the potential for disease when unweaned young birds from varying sources are mixed together. Buyers should question bird vendors carefully and obtain a health guarantee. Not all health guarantees are alike and should be examined carefully. Some guarantees offer to pay veterinary bills if a health problem is discovered within a certain time period. Some merely offer to replace the ill bird with another from the same source, which often is unsatisfying to purchasers who may quickly bond to their new pet.

Table 2.3 | Summary of Characteristics of Breeders of Parrots for the Pet Trade

Ideal	Not Ideal
Raises small numbers of birds	Raises many birds
Specializes in a few species	Many species intermixed at same facility
Does not mix larger parrots with small species, such as cockatiels, lovebirds and budgerigars	Larger hand-fed parrots mixed with smaller species
Selects breeders to maximize ideal pet characteristics	Breeders selected for reasons other than to maximize ideal pet characteristics: only birds available, "bargain birds," unwanted pets with problems such as phobias and feather plucking
Sells only weaned, hand-fed parrots	Sells unweaned young birds
Sells birds directly to clients, and not through pet stores or bird fairs	Sells birds through venues where young birds are co-mingled: pet stores and bird fairs
Raises birds on pellets	Raises birds on seeds

Increased computer access has allowed people to search for and purchase parrots over the Internet. While purchasing birds in this manner has many advantages, disadvantages include potentially shipping young birds long distances and in some cases, the inability to fully scrutinize the source.

Many commercial hatcheries produce healthy ducks, chickens, geese and other exotic fowl that can be purchased in small numbers for the pet trade. These facilities tend to follow strict disease prevention protocols, and often are much safer sources than backyard breeders or animal auctions (see Chapter 21, Preventive Medicine and Screening).

Comments on Life with Birds

Sharing life with pet birds is not for everyone. Experienced bird owners understand that birds can produce a great deal of dust, dander and mess, require constant handling to remain tame and in many cases are long-lived. Many birds naturally tend to dunk food into water bowls and shred toys into tiny bits. Some owners can be frustrated by the demands of pet birds and endless cleaning routines. Overall, birds require more intensive training to remain social than do most dogs and cats. A well-cared-for parrot may live for many decades.

All parrots make noise, and while this fact doesn't seem to bother parrot lovers, it can bother many neighbors. It's important to find out in advance if the noise is likely to cause problems. Sometimes the quiet but constant beeping of a cockatiel may be more offensive than the


Jan Hooimeijer

Fig 2.5 | The human/avian bond can occur with common birds, such as pigeons (*Columba livia*).


Greg J. Harrison

Fig 2.6 | Doves, such as this pied ringneck dove (*Streptopelia risoria*), are gentle and quiet.


Angela Lennox

Fig 2.7 | Often overlooked as pets, some chicken breeds may be good pets for children.


Greg J. Harrison

Fig 2.8 | The rose-breasted cockatoo or galah (*Eolophus roseicapillus*) is considered a pest in its native Australia, where free-ranging birds are captured for the pet trade.


Greg J. Harrison

Fig 2.9 | The black palm cockatoo (*Proboscigar aterrimus*) is a rare, expensive and endangered species that is uncommon in captivity and seldom seen in clinical practice.


Greg J. Harrison

Fig 2.10 | These pied Bengalese (or society) finches (*Lonchura domestica*), while commonly kept as pets, are also used as foster parents to chicks of more exotic finch species.

Greg J. Harrison

Fig 2.11 | The appealing cordon bleu finch (*Uraeginthus* spp.) has become expensive due to bans on wild-caught birds and aviculture challenges.

Greg J. Harrison

Fig 2.12 | The yellow-collared macaw (*Ara auricollis*) is one of the so-called "mini" macaws that exhibits characteristics similar to larger macaws but in moderation.

Mimi Walling/We Shoot Birds

Fig 2.13 | Like most mini macaws, this severe (or chestnut-fronted) macaw (*Ara severa*) can be hard to find, because breeders are often few in numbers.

Mimi Walling/We Shoot Birds

Fig 2.14 | Although this white phase scarlet macaw (*Ara macao*) is rare and valuable, mutations like this are often less resistant to disease.

Greg J. Harrison

Fig 2.15 | Green-winged macaws (*Ara chloroptera*) are beautiful and gregarious, but they need special homes because of their size, noise level, destructive habits and demand for attention.

Loro Parque

Fig 2.16 | The blue and gold macaw (*Ara ararauna*) is the most common macaw species kept as a pet in the United States.


Mimi Walling/We Shoot Birds

Fig 2.17 | The caninde (or blue-throated) macaw (*Ara glaucogularis*) is smaller than the blue and gold macaw and is rarely seen in captivity.


Mimi Walling/We Shoot Birds

Fig 2.18 | The spix macaw (*Cyanopsitta spixii*) is the rarest macaw and likely no longer exists in the wild.


Mimi Walling/We Shoot Birds

Fig 2.19 | Despite the initial investment to purchase, it is not uncommon for the hyacinth macaw (*Anodorhynchus hyacinthinus*) to be moved from home to home due to the great demands of upkeep.


Mimi Walling/We Shoot Birds

Fig 2.20 | The military macaw (*Ara militaris*) is often confused with the Buffon's macaw, but is slightly smaller and equally rare in captivity.


Greg J. Harrison

Fig 2.21 | The white-fronted Amazon (*Amazona albifrons*) is one of the few sexually dimorphic parrots; red feathers are found on the wings of the mature male and not on the female.


Friedrich Janecheck

Fig 2.22 | The blue-fronted Amazon (*Amazona aestiva*) is probably the most popular Amazon parrot because of its gregarious nature and ability to mimic, but like the larger Amazons, is frequently abandoned to a rescue facility.

Mimi Walling/We Shoot Birds

Fig 2.23 | The double yellow-headed Amazon (*Amazona ochrocephala oratrix*) is one of the least commonly seen Amazon parrots due to depletion in the wild and their aggressive personalities in captivity.

Mimi Walling/We Shoot Birds

Fig 2.24 | Yellow-naped Amazons (*Amazona auropalliata*) are successfully bred in captivity and are popular pets because they are entertaining talkers, singers and clowns.

Mimi Walling/We Shoot Birds

Fig 2.25 | Some communities have banned the Quaker (or monk) parakeet (*Myiopsitta monachus*) because escapees have established free-ranging breeding colonies even in temperate climates.

Mimi Walling/We Shoot Birds

Fig 2.26 | While the black-headed caique (*Pionites melanocephala*) is recommended as a pet, it is relatively rare and hard to find.

Mimi Walling/We Shoot Birds

Fig 2.27 | The white-bellied caique (*Pionites leucogaster*) may be threatened with extinction in the wild and should not be kept as a single pet.

Greg J. Harrison

Fig 2.28 | Small Australian parrots, including the superb parrot (or barraband parakeet) (*Polytelis swainsonii*), are usually viewed as aviary birds. However, if an individual is hand-raised in a family environment, it can be a good pet.

Greg J. Harrison

Fig 2.29 | A dusky-headed conure (*Aratinga weddellii*) is considered an ideal parrot because of its size, temperament, hardiness, lack of mutations and potential for human bonding.

Mimi Walling/We Shoot Birds

Fig 2.30 | Maroon-bellied conures (*Pyrrhura frontalis*) are smaller and generally more acceptable as a pet than the slightly larger *Aratinga* species.

Greg J. Harrison

Fig 2.31 | The sun conure (*Aratinga solstitialis*) is one of several conure species that are commonly bred in captivity, but even hand-raised individuals are loud and somewhat aggressive.

Greg J. Harrison

Fig 2.32 | Blue-headed pionus parrots (*Pionus menstruus*) are noted for their calm behavior and quiet nature, but are subject to stress-related disorders.

Greg J. Harrison

Fig 2.33 | Although infrequently seen in practice, pionus parrots (*Pionus* spp.) represent ideal pet characteristics: predictability, reserved nature, quiet, tidy, gentle and tolerant.

Mimi Walling/We Shoot Birds

Fig 2.34 | In the USA, African grey parrots (*Psittacus erithacus*) are being domestically bred and managed to eliminate negative characteristics that are still prevalent in imported greys in Europe: feather-picking, screaming and respiratory infections.

Loro Parque

Fig 2.35 | Eclectus parrots (*Eclectus roratus*) are the most pronounced example of sexual dimorphism: the female is red and the male is green. Eclectus appear to have some unique disorders that are not yet fully understood.

Mimi Walling/We Shoot Birds

Fig 2.36 | The cockatiel (*Nymphicus hollandicus*) is the most common patient seen by avian veterinarians. A male is shown. For best results a cockatiel should be purchased from a reputable breeder with less emphasis on developing color mutations.

Greg J. Harrison

Fig 2.37 | The popularity of cockatiel color mutations brings an increase in disease and unthriftiness. Lutinos (with reddish eyes) seem to have immune deficiencies and short lives, whereas pieds (such as this pied white-faced cockatiel with dark eyes) have fewer health problems.

Fig 2.38 | The meyer's parrot, a member of the *Poicephalus* genus, can be an enjoyable pet.

Mimi Walling/We Shoot Birds

Fig 2.39 | Because of their bright colors and clown-like antics, lories (*Loricus* and other species) in general are appealing, but their traditional nectar diets result in loose, messy droppings. This particular species, the black-capped lory, is rare and thus should not be kept as a pet.

Friedrich Janeczek

Fig 2.40 | The Bourke's parrot (*Neophema bourkii*) has all the same characteristics as other Australian small parakeets and is becoming more popular as an aviculture bird because of mutations, such as this rosy Bourke.

Fig 2.42 | Lovebirds (*Agapornis* spp.) are best obtained from a reputable breeder who has not concentrated on developing mutations and has paid more attention to their long-term health. Many breeding birds have endemic circovirus.

Mimi Walling/We Shoot Birds

Fig 2.44 | Creating color mutations, such as this lutino ring-necked parakeet, result in weaker birds with more health problems.

Greg J. Harrison

Fig 2.41 | Toucans can be entertaining clowns but are not generally recommended as pets because of their special dietary and large housing requirements.

Greg J. Harrison

Fig 2.43 | The hardy Indian ring-necked parakeet (*Psittacula krameri*) is a common pest bird in its native India. It is dimorphic: the male has a distinct ring around the neck, whereas the female's ring is not a full collar.

Mimi Walling/We Shoot Birds

Fig 2.45 | The budgerigar (*Melopsittacus undulatus*) is the most popular pet parrot in the world. Budgerigars bred for show are often grossly overweight and have reduced life spans. The wild-type green color reflects sexual dimorphism: the cere is blue in males and brown in females.

Nico J. Schoemaker

Fig 2.46 | Unusual red-colored feathers in African grey parrots may be linked to a dietary deficiency or circovirus infection. In many cases, however, these birds remain apparently healthy.

Greg J. Harrison

Fig 2.47 | Weaning a budgerigar to a formulated diet often is easier using a mirror. Seed diets are the major cause of illness in pet birds.

occasional yell from Amazons. Canaries, pigeons, doves, finches, and even female ducks and chickens have been found in homes where neighbors never suspected they lived. In many cases, as far as neighbors are concerned, the best bird is a quiet bird.

Appropriately sized cages can take up considerable space, especially for larger birds. Large cages, play gyms and toys can be prohibitively expensive. Some owners seek to cut costs by buying used cages, which may not be safe if the previous inhabitant died of a communicable illness. Wooden perches and porous items cannot be properly disinfected and should not be reused.

Pets often are restricted for owners living in apartments or condominiums. However, one ingenious owner rescued a boisterous Moluccan cockatoo, took it to the swimming pool and put it on the fence, declaring the bird to be the condo mascot. While the condo had a no-pet rule, regulations apparently did not cover mascots.

Medical care for birds tends to be less expensive than that for other domestic species. An example was heard on National Public Radio program update on veterinary costs for pets entitled, "How Much is that Doggie in the Window?" The woman interviewed had just spent over $20,000 to treat her cat for cancer. Although many avian vets have never come close to that sum with a sick bird, the interviewee's expenditure may have been due to her inability to let go of her pet and not reflective of an expensive but successful treatment protocol.

With the above in mind, many things must be considered before acquiring a pet bird. The biggest birds do not automatically make the best companions. Most of the birds that these authors generally recommend are medium to small birds, which are easier to manage, house, feed and train than are large psittacines. While beauty is in the eye of the beholder, the finches and small parrots often are the most ornate. If song is most inspiring, only one bird has held the title of "Elvis of Birds" for so long: the humble canary. In many cases, for beauty, size and song, one has to look no further than to the tiniest birds to fulfill many desires.

So why does one choose to cohabit with a bird? It generally comes down to what seizes a person's heart. For some people, birds fill the void in a way no other pet can.

Conclusion

Avian companions clearly occupy more than just a niche in their caregivers' homes and lives. The importance and expertise of avian medical practice must continue to expand to meet the demands of this multi-species discipline. Bird ownership increasingly embraces large and small companions, where value often is not related to the cost of the bird. The proliferation of birds in other non-home settings, debates regarding animal rights and the wide variety of opinions generated by these issues will continue to occupy avian medical practitioners and caregivers alike.

References and Suggested Reading

1. American Board of Veterinary Practitioners web site: www.abvp.com.
2. American Pet Products Manufacturer's Assoc. 2001-2002. National Pet Owner Survey. APPMA web site: www.appma.org. Jan 2003.
3. American Society for the Prevention of Cruelty to Animals web site: www.aspca.org.
4. American Veterinary Medical Association: US Pet Ownership and Demographics Sourcebook. 2002.
5. Association of Avian Veterinarians web site: www.aav.org.
6. Beck A: Bird-human interaction. J Am Vet Med Assoc 3:152-153, 1980.
7. Convention on International Trade in Endangered Species web site: www.cites.org.
8. European College of Avian Medicine and Surgery web site: ecams.online.org/public/introduction/asp.
9. Fish and Wildlife Division of Management Authority web site: www.fws.gov.
10. The Humane Society of the United States, Armstrong M, personal communication, web site: www.hsus.org.
11. Independent survey, Avian and Exotic Animal Clinic of Indianapolis, 2002.
12. McMillan RJ: The Parrot Society UK web site: www.theparrotsocietyuk.org.
13. Orosz S: Veterinary management of birds in care facilities. Proc Assoc Avian Vet, 2002, pp 369-374.
14. People for the Ethical Treatment of Animals web site: www.peta-online.org.
15. Report of the AVMA Panel on Euthanasia. J Am Vet Med Assoc, 218:669-696, 2000
16. Speer B: Bird Numbers, e-mail from AVNVET 12/21/02, 1:45:48 PM EST.
17. United States Department of Agriculture Animal and Plant Health Inspection Service web site: www.aphis.usda.gov.

CHAPTER

3

Concepts in Behavior

Section I: The Natural Science of Behavior

Section II: Early Psittacine Behavior and Development

Section III: Pubescent and Adult Psittacine Behavior

Edwardo Nycander

Concepts in Behavior: Section I

The Natural Science of Behavior

SUSAN G. FRIEDMAN, PhD; THOMAS M. EDLING, DVM, MSpVM; CARL D. CHENEY, PhD

Of all the many facets of parrots' total wellness supported by veterinarians, perhaps the most challenging of all is behavior. Having adapted over eons for survival in the free-range environment, many parrot behaviors run counter to those necessary for success in our homes. This challenge is intensified by parrots' extraordinary ability to learn maladaptive behaviors from their often-unwitting caretakers. Veterinarians also face educational challenges as their pursuit of a comprehensive and cohesive knowledge of behavior often is made difficult by the fractured development of the science itself — the natural science of behavior historically crosses two disciplines, zoology and psychology, each with its own purpose and methods. Finally, among professionals and laypersons alike, there is a general lack of awareness that a science of learning and behavior exists within the field of psychology. A sound understanding of this science, known as behavior analysis, is critical to successfully keeping parrots as companions. These challenges contribute to the current state of affairs in which too many pet parrots unnecessarily fail to thrive due to behavior problems.

In this chapter, we provide the foundation for a comprehensive and cohesive understanding of behavior as it relates to facilitating the lives of companion parrots. To meet this goal, the following topics are discussed: free-range behaviors as a basis for predicting and interpreting the behavior of parrots in captivity, a simplified model for systematically analyzing the functional relationships between behavior and environmental stimuli, and the teaching technology based on the fundamental principles of learning and behavior. With this information, veterinarians will be able to better guide their clients to proactively teach their parrots successful companion behaviors and effectively analyze and resolve behavior problems that inevitably arise.

What is Behavior?

Fundamental to all science is the task of explaining phenomena by identifying observable, physical events that produce them. This is true with behavioral science as well, where the goal is to explain behavioral phenomena. In this scientific context then, behavior is anything an animal does that can be observed and measured. This includes overt behaviors that can be directly observed by others (such as preening and eating) as well as covert behaviors, which can only be directly observed by the individual so behaving (such as thinking and feeling). As a result, covert behaviors are of limited use in our work with parrots due to their inaccessibility. And, considering the difficulty most of us have guessing what members of our own species are thinking in the absence of direct measures, accurate interpretation of parrots' covert behaviors is all the more remote.

Similarly, the practice of describing what an animal *is* rather than what it *does* is an obstacle to understanding and changing behavior. Labels, such as "*is* territorial," "*is* dominant," and "*is* spoiled," do not describe behaviors, they describe ideas. These ideas, called hypothetical psychological constructs, are largely untestable theories about mental processes believed to explain behavior. Focusing on constructs often gets in the way of identifying straightforward behavior solutions. To change behavior, clients must work with behavior directly, and they should be encouraged to move past inferences of covert behaviors and construct labels to observe and describe what their birds actually do. For example, the frequently used label "is territorial" often describes a bird that bites; "is dominant" often describes a bird that does not step up; and "is spoiled" often describes a bird that

screams for intolerable durations. Territoriality, dominance and the degree to which the bird is spoiled can't be changed directly because they have no tangible form; however, biting, stepping up and screaming are all behaviors birds *do*, which we can do something about.

Behavior is the result of the indivisible blend of heredity and learning. These two processes work toward the same end, ie, coping with environmental change through adaptation. Adaptation through heredity, phylogenetic adaptation, occurs slowly over generations at the species level. Through the process of evolution by natural selection, phylogenetic adaptation equips each species for common lifestyles in their natural habitat. Alternatively, adaptation through learning is an individual process that occurs within the short span of a lifetime. As defined by Chance, learning is a change in behavior due to experience.[8] Learning is the astonishing mechanism that equips each individual within a species to meet life's ever-changing circumstances with rapid modifiability.

Observations from the Field

Parrots are most brilliantly adapted for the free-range environment. For example, the physiology of wings, beaks and vocal structures prepares them well for the natural behaviors of flight, nest carving and long-distance contact calls. Clearly these and many other behaviors are supported by parrots' genes and are part of their natural history. From an evolutionary perspective, the genes that enable these behaviors likely serve survival functions related to food gathering, courtship and mating and protection from predators. It is worth noting, though, that the evolutionary origins of many behaviors often are easier to hypothesize than to prove.

Ethology, a discipline within zoology, is the field of behavior science most concerned with the study of behavior patterns characteristic of different animal species as they occur in their free-range environments. More complex than reflexes, ethologists call these species-specific behavior chains "fixed action patterns." Fixed action patterns are displayed by nearly all members of a species under similar environmental conditions, with very little variability in the way in which they are performed across individuals or instances. According to Gray, these behavior patterns are fixed in the sense that the "controlling mechanisms are 'fixed' in the animal's nervous system by heredity and are relatively unmodifiable."[12] In this sense, we call them innate behaviors.

There is some debate about how unmodifiable fixed action patterns actually are, as few, if any, behaviors can be said to be immutable or impervious to experience. Some researchers reason that "flexible action patterns" is a more accurate description of species-specific behavior chains.[21] For example, fledglings' flight skills certainly improve with practice, as does perching and climbing. Even simple reflexes can be modified through habituation[26] (eg, cats[28]) and through sensitization (eg, blowflies[9]). These studies add to the evidence that heredity and learning are inextricably entwined. Nonetheless, knowledge of the behavior patterns of free-range parrots, as well as the environmental conditions that elicit and shape them, greatly increases our ability to predict, interpret and manage many parrot behaviors in captivity. For these reasons, knowledge of the free-range behavior of parrots is important to improving the care of captive birds.

SOCIAL SIGNALS

Among the many things we can learn from the behaviors of free-range birds, perhaps the most important are those that serve a communication function among parrots. This is a language very unfamiliar to many caretakers, to the detriment of their birds and themselves. In an interesting study on cross-species communication, it was found that dog pups only a few weeks old were more skillful at reading human social cues (such as pointing, looking and touching) to locate hidden food than were chimpanzees and wolf pups.[13] The researchers theorize that dogs uniquely possess this skill due to the process of domestication in which communication skills with humans were selected.

Unfortunately, our parrots' current lack of domestication leaves them unprepared to innately interpret human signals. This puts the onus on us to accurately interpret their communications at the same time they are learning to interpret our signals. Observations from the field confirm that parrots have a rich and subtle communication system that involves nearly every feather on their bodies. Head, eye and neck movements, body posture, wings and tail and leg and foot gestures are all used as signals to communicate desires, intentions and general comfort or discomfort with current events and conditions.

Caretakers often misunderstand the behaviors used to communicate the boundaries of personal space, especially those that function to back intruders away. Most species of parrots use threatening stances rather than outright aggression to drive off perceived intruders in the wild, and many of these behaviors are seen in captivity as well. These behavior patterns are made up of various vocalizations and both overt and subtle movements and postures. Depending on the species, such warnings include raised nape feathers with wings slightly lifted, a raised foot held open at chest level, directed hacking motions with an open beak, and growling.[18] By not

heeding these warnings, caretakers push parrots to escalate their message to serious biting. As a result, stress is unnecessarily increased and trust is decreased for both birds and humans. Learning to perceive, interpret and respond to these signals is essential for building relationships with captive parrots. Veterinarians can help caretakers become more astute observers of their parrots' "messages" by discussing social signals with them.

ACCOMMODATING INNATE BEHAVIORS

Other innate behavior patterns common to free-range parrots, such as loud contact calls, wood chewing, food flinging and territorial biting, can be challenging to deal with in the captive setting. Changing the environment to accommodate them to the greatest extent possible, rather than attempting to change the bird, often best manages these behaviors. For example, simply answering a bird's calls, even from another room, often deters parrots from screaming. Arranging challenging body and brain activities provides alternatives to chewing household woodwork. Offering smaller, more frequent food servings in cages fitted with aprons reduces the mess and waste of food flinging. Allowing birds to climb out of their cages when the door is opened, rather than insisting they step onto intruding hands, reduces the opportunity for biting. By keeping natural behavior repertoires in mind and arranging the environment to manage them, caretakers can focus on engaging appropriate behavior rather than disengaging problem behavior.

THE LIMITS OF LEARNING

Another important reason for clients to understand parrots' free-range behaviors is to guide the general limits of what our parrots can reasonably be expected to learn. A classic article on behavior, lightheartedly entitled "The Misbehavior of Organisms," reported the breakdown of novel trained behaviors in favor of fixed action patterns, even though food reinforcement was contingent solely on the performance of the trained responses.[3] The authors called this phenomenon "instinctive drift," as they observed that raccoons tended to rub coins with their paws in a characteristic washing motion rather than deposit them into a bank; pigs tended to toss coins with their snouts in a characteristic rooting motion rather than carry them in their mouths; and chickens tended to scratch the floor with their feet in a characteristic wiping motion rather than stand still.

Instinctive drift is consistent with Seligman's continuum of preparedness, described by Chance[6]: "An organism comes to a learning situation genetically prepared to learn (in which case learning proceeds quickly), unprepared (in which case learning proceeds steadily but more slowly), or contraprepared (in which case the course of learning is slow and irregular)." Too often, unknowing caretakers simply expect too many behaviors for which parrots are contraprepared. This occurs when, for example, caretakers insist that parrots be petted by strangers (or for some birds, petted at all), or when birds are left in cages for interminably long durations with nothing to do (from the birds' perspective). Of course, the particular limits of parrots' behavioral preparedness to learn vary greatly across species and between individuals within species; still, knowledge of species-typical behaviors observed in the free-range environment is an excellent starting point for predicting and interpreting the behavior of different species of captive parrots. It also is essential to helping clients set reasonable expectations for parrot behavior in their homes.

Applied Behavior Analysis

Ethology informs us about the behavioral norms of different parrot species in the free-range environment. While this information is important to successful companion parrot care, it is not sufficient to meet the challenges of living with captive parrots. It also is essential to have expertise in applying the fundamental principals of learning and behavior applicable to all species of animals. This is true for several reasons. First is the extent to which individuals of the same species are known to vary from one another and from expected behavioral norms: Any particular African grey (*Psitticus erithacus erithacus*) may exhibit the cuddly behaviors of the average umbrella cockatoo (*Cacatua alba*); and, any particular sun conure (*Aratinga solstitialis*) may be as quiet as the average dusky Pionus (*Pionus fuscus*). Second is the wide variability across captive environments in which companion parrots are challenged to live: Ranging from quiet, routine lives with a single caretaker to noisy, unpredictable lives full of kids and other pets, no two home environments are alike. Third, parrots' extraordinary longevity means most birds will be confronted with decades of changing circumstances for which they need to be extremely flexible learners.

When we change our focus from the species level to the individual level and from innate responses to learned responses, the natural science of behavior is (much like veterinary practice itself) a "study of one." The field of behavior science that most explicitly concentrates on the learned behavior of individuals is applied behavior analysis; it primarily is the applied science of teaching and learning, which is why it is so very relevant to companion parrots and their caretakers.

ACCOUNTING FOR BEHAVIOR

For lack of knowledge about the fundamental principles of learning and behavior, many people are utterly baffled by the things their parrots do. Caretakers often describe their birds as inscrutable creatures that behave in completely unfamiliar and unpredictable ways. People don't realize that many of their birds' behaviors are the direct result of the environments they provide and the pattern of interactions they have with their birds. A different problem is a general resistance to the idea of training animals. To some people, training carries the connotation of forcing an animal to succumb to the will of their human captors. They believe parrots should be taught as little as possible so they remain "natural." On the contrary, parrots' tendency to learn is as natural as their tendency to eat and sleep. Learning enables parrots to adapt to life in captivity and in the wild. It also is the mechanism through which we can provide enrichment activities to our birds to improve what might otherwise be a stultifying life in captivity. Concerns about force are immediately dispelled when people learn the teaching technology of applied behavior analysis, which facilitates positive-first learning solutions.

The focus of applied behavior analysis is on the environmental elements that account for behavior. By changing what we do and the environments we provide, we can facilitate behaviors more suited to life in captivity and reduce problem behaviors. This is the way to protect captive parrots from lives locked in cages, multiple homes and eventual homelessness. To make the most of every teaching/learning opportunity, clients need to know how behaviors are learned, how to functionally analyze behavior, how to teach new behaviors, and how to reduce problem behaviors with effective, non-forceful behavior intervention plans. As veterinarians often are the first and only professionals parrot caretakers turn to for help, this information is critical to providing the gold standard of veterinary care and support to companion parrots.

THE ABCs OF BEHAVIOR

Behavior doesn't randomly spurt out of a behaving organism from some internal fount, nor is it performed in a vacuum or broadcast into a void. On the contrary, behavior has function. The function of any particular behavior is related to the environmental stimuli that precede and follow it, called antecedents and consequences. Antecedents are those events or conditions that immediately precede a behavior, which set the occasion for the behavior to occur. Not all preceding events or conditions are functionally related antecedents, just those specifically related to the ensuing behavior. For example consider three common parrot behaviors - screaming, stepping up and biting. Below are examples of antecedents that may be functionally related to these behaviors in many situations:

When I leave the bird room, then the bird screams.
When I offer an open hand, then the bird steps up.
When I pet the bird, then the bird bites.

In these examples, leaving the bird room, offering an open hand and petting are all antecedents that are functionally related to the specific behaviors that immediately follow them (eg, screaming, stepping up, biting). Antecedents signal to each individual which behavior to exhibit in any given circumstance. Without the relationship between antecedents and behavior, humans would indeed behave willy-nilly, tossing out behaviors without rhyme or reason; or we may just sit there doing nothing at all.

In day-to-day conversation, the word "consequence" often is used to mean something punitive, as in, "Suffer the consequences!" In behavior analysis, consequences are those events or conditions that affect the future rate of the behaviors they immediately follow. Consequences are outcomes produced by an individual's behavior and provide environmental feedback about whether the behavior just performed should be repeated or modified in the future, when similar circumstances (antecedents) next arise. Of course, consequences don't always come from people. For example, when a new fledgling bumps against a branch when it first takes flight, it will quickly adjust the angle of its wings. No behavior emitted goes without some consequence in return, and all learners actively sift through the feedback to discover how to make behavior "work." We can add the following consequences to our examples of bird behavior from the previous section:

When I leave the bird's room, if the bird screams, then I return.
When I offer an open hand, if the bird steps up, then I praise it.
When I pet the bird, if the bird bites me, then I remove my hand.

Behaviors that produce valued consequences (such as our return to the bird room, praise and the removal of an unwanted hand) tend to be repeated or increased. Behaviors that result in consequences of no value or negative value tend to be modified, decreased or abandoned. In this way, individuals learn to operate on their environment to produce certain outcomes. Skinner called this process "operant" conditioning to emphasize the learning process in which the learner is an *active participant*.[27] (This is in contrast to respondent or Pavlovian conditioning in which the animal is a passive participant, responding reflexively to eliciting stimuli.)

As can be seen in the examples above, consequences also strengthen the antecedent-behavior relationship.

For example, if stepping up consistently produces something of value to your bird, offering your hand will become a strong antecedent for stepping up, as it signals the availability of a valued consequence.

These three terms, *antecedent, behavior* and *consequence*, comprise the ABCs of behavior. Skinner called this three-term contingency the smallest meaningful unit of analysis. In other words, no behavior can be understood in isolation of its related antecedents and consequences. Focusing on our birds' behavior alone has no meaning because their behaviors are not performed in the absence of antecedents and consequences.

FUNCTIONAL ASSESSMENT/ANALYSIS

The process of hypothesizing the functionally related antecedent, behavior and consequence is called functional assessment. It is an important tool for understanding problem behaviors and for devising specific plans to teach new behaviors. With functional analysis, caretakers can determine exactly what leads to and maintains specific parrot behaviors by systematically making changes and evaluating the effect on behavior. Finally, caretakers can design new antecedents and/or consequences to facilitate successful behaviors — their own and their birds'. When caretakers consider behavior in light of this behavior-analytic approach, the causes of problem behaviors and workable solutions often become very clear. Functional assessment and analysis reduce the likelihood that caretakers will resort to unverifiable, hypothetical constructs to explain their parrots' behavior, which may lead them further astray from practical solutions.

There are six basic steps to conducting a functional assessment/analysis:

Step 1: Operationally define the target behavior. A target behavior is the response you want to maintain, increase or decrease. To operationally define the target behavior, describe it in clear, observable terms. Ask: What does the bird actually *do*?

Step 2: Identify the antecedents that set the occasion for the target behavior. Ask: What event or condition immediately precedes or "leads" the bird to exhibit this behavior?

Step 3: Identify the consequence that immediately follows the target behavior. Ask: What happens immediately after the behavior is exhibited? What do *you* do or how does the environment respond?

Step 4: Predict the probable future behavior that most likely will occur as a result of the current consequence. Ask: Will the behavior likely be repeated, increased or decreased?

Step 5: Devise and implement a new antecedent and/or consequence to facilitate a different behavior. Ask: What can we do instead?

Step 6: Evaluate the outcome, reanalyze and adjust the teaching program as needed. Ask: Was the desired outcome achieved?

Below are three examples of functional assessments for one very common problem, a bird that refuses to step up from the top of his cage:

Functional Assessment #1: Parrot Refuses to Step Up from Top of Cage

Antecedent: Caretaker says, "Up!" and offers hand to bird on top of cage.

Behavior: Bird performs evasive maneuvers running around the cage top.

Consequence: Caretaker gives up chasing bird and walks away.

Prediction: Bird will continue to run away from his caretaker's hand in the future to avoid being removed from cage top.

Many people ascribe to hypothetical constructs to explain such "misbehavior." One pervasive theory repeated in many popular parrot magazines is that birds are asserting dominance over their caretakers by refusing to step up from the tops of their cages and are vying for control of the human-parrot flock. Caretakers are told that to solve this problem, they need to increase their rank in the eyes of their birds and disallow them from making any important decisions about what they do and when, and never allow their birds higher than the caretaker's heart level. Alternatively, a functional assessment, which adheres to describing the observable relationships between antecedents, behaviors and consequences, suggests a more plausible hypothesis, as described below:

Functional Assessment #2: Bird Willingly Steps Up When Requested

Antecedent: Caretaker says, "Up!" and offers hand to bird on top of cage.

Behavior: Bird steps up.

Consequence: Bird is returned to cage.

Prediction: Bird will step up less in the future to avoid being returned to the cage.

This functional assessment suggests that this bird has learned to run away from the offered hand simply to avoid being locked in its cage. It seems an intelligent choice from the bird's point of view, given the consequences of complying with the request. Unlike the construct explanation, this behavior-analytic explanation meets the scientific criterion of a good hypothesis:

1. We can test it by changing the consequence and see if the behavior changes;
2. it is as simple as possible, but no simpler;

3. it allows us to predict future events; and,
4. it is useful, as it implies workable, positive alternatives. For example, most parrots would be very responsive to stepping up from their cage tops if they valued the consequence for doing so. A few moments of attention before being returned to the cage and a treasured food treat after entering the cage are usually all it takes.

Of course, human behavior also is a function of its consequences. Below is a functional assessment of the caretaker's behavior whose bird refuses to step up:

Functional Assessment #3: Caretaker Leaves Bird in Cage

Antecedent: Bird is playing on cage top.

Behavior: Caretaker says, "Up!" and offers hand to bird on top of cage.

Consequence: Bird runs away.

Prediction: Caretaker asks bird to step up less often to avoid refusal.

Chances are, in the long run, this caretaker either will leave his bird in its cage more and/or become more forceful when retrieving the bird. As a result, many birds escalate their initial refusal to biting. All this caging, force and refusal are unnecessary when a simple positive strategy like offering a food treat or a few minutes of uninterrupted attention before being returned to the cage can solve the problem of birds refusing to step up from their cage tops.

Before considering how to change a behavior, caretakers should conduct a functional assessment to determine the function the behavior likely serves for the parrot. The question is not, "Why is the bird behaving this way?" but rather, "What valued consequences result from performing the behavior for this particular bird in this situation?" By changing antecedents and consequences, we change target behaviors. As antecedents and consequences most often are stimuli or conditions we control, changing our birds' behavior always is the direct result of first changing our own behavior.

INCREASING AND MAINTAINING BEHAVIOR

When you think about it, consequences influence behavior in one of two basic ways: Consequences function to maintain/increase the frequency of a behavior or they function to eliminate/decrease the frequency of a behavior. In this section, we are concerned with consequences that function to increase behavior, called reinforcers, and with the process of delivering reinforcers, called reinforcement.

The relationship between behavior and reinforcers is clear, as we see the effect of this principle all around us. When we fasten our seat belts and the buzzer stops, we learn to fasten our seat belts more often to stop the buzzer; when the cat sits in front of the door and we let it out, the cat learns to sit at the door more often to be let out; when the parrot steps up and we take it out of its cage, the parrot learns to step up more often to be removed from its cage.

Characteristics of Effective Reinforcement

Less well considered are the characteristics of effective reinforcement, the most important of which are clear contingency, close contiguity and attention to individual differences. Contingency refers to establishing the dependency between a behavior and its reinforcing consequence. Some people refer to it as "Grandma's Law," which states, "*If* this is your behavior, *then* this is your consequence." Thus, reinforcement is the process of delivering a reinforcer *contingent* upon the performance of a particular behavior. Consistency is important to establishing clear contingency between a behavior and a reinforcer.

Contingency also is clearer when reinforcers are delivered with close contiguity, the second characteristic of effective reinforcement. Contiguity refers to immediacy; that is, the shorter the interval of time between the behavior and the reinforcer, the more effective it will be in increasing the future rate of that behavior. Lattal demonstrated the importance of contiguity in an interesting study with pigeons.[19] In an effort to teach a pigeon to peck a disk, Lattal arranged to deliver a food pellet each time the pigeon moved toward the disk. However, he purposely delayed the delivery of the pellet for just 10 seconds after the target behavior was exhibited. After 40 days of 1-hour training sessions, the pigeon never learned to peck the disk. Subsequently, when the delay between the behavior and the reinforcer was reduced to 1 second, the bird learned to peck the disk in less than 20 minutes.

Reinforcers also are highly individual. Some people are not reinforced by the cessation of the car buzzer and so do not increase the behavior of buckling their seat belt; some cats are not reinforced by going outside, thus, they do not sit by the door; and some parrots are not reinforced by coming out of their cages, preferring instead to drive away the caretaker with a serious bite. Reinforcers are not what we think "should" increase the frequency of a particular behavior; rather, reinforcers are those consequences that actually do increase the frequency of a particular behavior they contingently follow. The only way to know for sure which consequences will be reinforcing for any particular bird is to try them and then observe the future frequency of the behavior.

Developing New Reinforcers

Some consequences such as food, water and warmth are inherently reinforcing to all animals from the moment they are born. These consequences are called unconditional reinforcers (also called unconditioned or primary reinforcers); they are unconditional in the sense that they are not dependent on prior experience (learning), but they do require certain conditions or "establishing operations" to function as reinforcers, eg, hunger, thirst and cold. Surely these unconditional reinforcers are part of nature's clever plan to kick-start behavior at birth for survival.

As soon as an animal starts to interact with its environment, learning begins, and many different consequences become reinforcing by being paired with existing reinforcers. These learned reinforcers are called conditional reinforcers (also called conditioned or secondary reinforcers); they are conditional in the sense that their reinforcing properties are acquired and maintained by being paired with existing reinforcers. Praise, petting and toys are examples of conditional reinforcers for many companion parrots and have become reinforcing though association with food or other valued stimuli.

The more reinforcers an individual parrot has, the more tools we have to influence its behavior, as novelty and variety are essential to effective reinforcement.[30] New reinforcers can be conditioned throughout the lives of all animals, and caretakers can make use of this process by pairing existing reinforcers with new stimuli to build a rich pool of reinforcers with which to teach and enrich their parrots' lives. Providing a constant supply of new treats, toys and activities allows our birds to sample new stimuli that may prove to be reinforcing.

Caretakers often complain that they have no way to teach their bird desirable behaviors because the bird has no reinforcers. Of course if that were the case, their bird would have no behavior. It sometimes takes sharp powers of observation to notice what reinforces a particular bird's behavior. Subtle outcomes like being set down or returned to the cage, or a caretaker's retreat, are often conditional reinforcers for poorly socialized birds. We can use even these reinforcers to increase their adaptive behavior, and condition more positive ones by association. For example, to teach a fearful bird to remain calm in our presence, we might start by withdrawing ourselves from its cage for a few seconds contingent on quiet, still behavior. If our removal functions as a reinforcer, we will see calm behavior increase over several repetitions. Again, if our removal functions as a reinforcer, saying "Good!" at the same moment we retreat will result in the word "good" acquiring reinforcing properties for this bird. Eventually, we can advance one small step at a time, reinforcing calm behavior with the word "good."

Positive and Negative Reinforcement

Admittedly, distinguishing two types of reinforcement with the terms "positive" and "negative" is at best esoteric and at worst utterly confusing. It is tempting just to avert the discussion, define reinforcement precisely and leave it at that. The distinction is pursued here because these terms are so commonly misunderstood and misused, and because positive reinforcement is the preferred strategy for changing behavior, as explained below.

Foremost, reinforcement is reinforcement. That is, regardless of type, positive or negative, reinforcement results in an overall increase in the behavior it follows when next the occasion (antecedent) is set for the behavior to be performed. A positive reinforcer is something that an individual behaves in a particular way to produce (+, add to its environment). It is gaining the reinforcer that functions to increase the behavior with positive reinforcement. Alternatively, a negative reinforcer is something that an individual behaves in a particular way to remove (-, subtract from its environment). It is the removal or escape from the reinforcer that functions to increase behavior with negative reinforcement. The example of increasing a bird's calm behavior contingent upon the caretaker's withdrawal is an example of negative reinforcement, functionally analyzed below:

Antecedent: Caretaker approaches cage.
Behavior: Bird flails.
Consequence: Caretaker remains near cage.
Antecedent: Caretaker remains near cage.
Behavior: Bird stops flailing for an instant.
Consequence: Caretaker steps back 5 paces from cage.
Prediction: Perching calmly will increase to remove caretaker from cage.

Below are additional examples of positive and negative reinforcement to make this distinction clear. Notice two things:

1. In all cases, the target behavior is increased or maintained as these examples all describe reinforcement;
2. With negative reinforcement, an aversive stimulus has to be present in the environment in the first place in order to increase behavior by its removal.

Examples of Positive and Negative Reinforcement #1:

Background: Beaker is a parrot that lunges at Grace's hand every time she puts her hand in or near Beaker's cage. Grace has decided to teach (increase) Beaker's behavior of perching on the branch farthest from the food cups so she can replenish them without Beaker's lunging.

Positive reinforcement solution:
Antecedent: Grace says, "Perch!"
Behavior: Beaker perches.
Consequence: Grace puts food and the food bowl in cage.
Prediction: Beaker will go to the perch more often to add (+) the food to the environment.

Negative reinforcement solution:
Antecedent: Grace herds Beaker to a particular perch in his cage with a stick.
Behavior: Beaker perches.
Consequence: Grace puts down stick.
Prediction: Beaker will go to the perch more to remove (-) the stick from the environment.

Examples of Positive and Negative Reinforcement #2:

Background: Of course, Grace also has a problem getting Beaker to step up from inside the cage without lunging.

Positive reinforcement solution:
Antecedent: Grace offers her hand.
Behavior: Beaker steps up.
Consequence: Grace praises Beaker enthusiastically and sets Beaker on top of the cage.
Prediction: Beaker will step up more to result in Grace's attention and cage-top location.

Negative reinforcement solution:
Antecedent: Grace holds a towel in one hand while offering her free hand.
Behavior: Beaker steps up on free hand.
Consequence: Grace sets down towel.
Prediction: Beaker will step up more to result in the removal of the towel.

As can be seen with these examples, a condition of negative reinforcement is the presence of an aversive stimulus in order for the animal to have something to work to escape. Indeed, another name for negative reinforcement is escape/avoidance learning. Research over decades with many different species of animals has shown that procedures that rely on aversive stimuli, such as negative reinforcement and punishment, tend to be associated with negative behavioral side effects. As you read the common types of side effects, consider how well they describe the behavior of many unfortunate parrots in captivity:
1. escape/avoidance behavior,
2. aggressive behavior,
3. response suppression, and,
4. fear of people or things in the environment in which the aversive stimuli are presented.[2]

The fact that these four general side effects are common descriptions of captive parrots suggests that many birds experience their environments as negatively reinforcing or outright punishing. Caretakers are encouraged to be analytical about the approaches they employ when interacting with their birds, so that they can deliberately decrease their use of aversive procedures. Positive reinforcement occasions none of this "aversive fallout," clearly making it the preferred behavior change strategy.[30]

SHAPING NEW BEHAVIOR

So far, we have discussed using positive reinforcement for maintaining or increasing the frequency of behaviors that a bird already performs. Shaping, also called Differential Reinforcement of Successive Approximations, is a procedure to teach new behaviors. To shape a new target behavior, start by contingently reinforcing the response already exhibited by the bird that most closely resembles (approximates) the target behavior. Once mastered (ie, performed without hesitation), reinforcement then is withheld for that behavior. Withholding reinforcement for a previously reinforced behavior is called extinction. Extinction results in an initial increase in responding and effort, which offers natural variability in the way the behavior is offered. Careful observation of this variability allows us to "catch" the next closer approximation with reinforcement. This process of ignoring one behavior (the mastered approximation) and subsequently reinforcing another behavior (the next closer approximation) is called differential reinforcement of successive approximations. Differential reinforcement of successive approximations is continued until the final target behavior is displayed and reinforced.

Many new behaviors required of successful companion parrots can be simply shaped and different dimensions of existing behaviors can be shaped, too. For example, proximity to a feared person or object can be increased; duration staying on a play gym or under a shower can be increased; and latency in responding to the requests "step up" or "off there" can be reduced. With shaping, an endless number and variety of adaptive behaviors can be taught and problem behaviors solved, all with positive reinforcement, thus avoiding the negative side effects that occasion more forceful or coercive methods.

Here's an example of the approximations that can be differentially reinforced to teach a parrot to play with foot toys:
1. Look at toy;
2. move toward toy;
3. touch toy with beak;
4. pick up toy with beak;
5. hold toy with foot;
6. hold toy with foot and manipulate with beak;
7. hold toy with foot and manipulate with beak for longer durations;

Table 3.1.1 | Intermittent Schedules of Reinforcement

	Fixed (set)	Variable (on average)
Ratio (number)	FR - reinforcement occurs after every "nth" response. FR 3 means that every third response will be reinforced.	VR - the number or responses required before reinforcement varies unpredictably around some average. VR 3 means the number or responses required will average around 3 but will vary.
Interval (time)	FI - reinforcement occurs after a fixed period of time elapses. FI 6" reinforcement will occur after 6 seconds elapse.	VI - the period of time that must elapse before a response is reinforced varies unpredictably around some average. In a VI 10" schedule, the average period required before the next response is reinforced is 10".

8. repeat with other toys until the behavior is generalized to all toys.

Unfortunately, negative behaviors can unwittingly be shaped as well. We inadvertently teach our birds to bite harder, scream louder and chase faster through the subtle mechanism of shaping. For better or worse, shaping is endlessly applicable to teaching our birds, limited only by our imagination and our commitment to practicing its use.

SCHEDULES OF REINFORCEMENT

Schedules of reinforcement are the rules we follow to determine when a particular instance of the target response will be reinforced out of the many responses that occur. Several so-called simple schedules are relevant here, as research demonstrates that different ratios of "behavior-to-reinforcement" result in remarkably different, but extremely predictable, patterns of behavior.

A continuous reinforcement schedule (CRF) is one in which each and every occurrence of the target behavior is reinforced. With CRF, the ratio of "behavior-to-reinforcement" is 1:1. Generally speaking, continuous reinforcement is the best reinforcement schedule to use with our birds, especially when the goal is to teach a new behavior or increase the rate of an existing behavior.[30] CRF is the clearest way of communicating exactly what behavior we want to see again. Research also has demonstrated that individuals behave in proportion to the reinforcement available for a given response.[15] There is little doubt that the more you positively reinforce your bird's desirable behavior, the more frequently your bird will exhibit desirable behavior. We get what we reinforce.

On the other end of the spectrum is a schedule called extinction (EXT), discussed previously as it applies to shaping. With an extinction schedule, no instances of the behavior are reinforced, ie, the ratio of behavior-to-reinforcement is 1:0. As the name suggests, when the particular reinforcer that maintains a behavior is withheld, the rate of that behavior will predictably decrease to prereinforcement levels. When human attention is the reinforcer maintaining a particular behavior, extinction is synonymous with ignoring, ie, we withdraw attention. Using extinction for the purpose of decreasing an unwanted behavior is not a simple procedure to properly implement. There is much to learn about the correct use of ignoring, which is briefly discussed in a subsequent section.

Somewhere between continuous reinforcement (1:1) and extinction (1:0) is another category of simple schedules of reinforcement known as intermittent reinforcement schedules. With intermittent schedules, only some (as opposed to all or none) of the target behaviors are reinforced. There are two basic dimensions along which intermittent schedules can be arranged: The first dimension regards what is being counted, either frequency of responses (called ratio schedules) or time elapsed (called interval schedules). The second dimension along which intermittent schedules can be arranged regards the predictability of reinforcement, either fixed or variable. With fixed schedules, the ratio (frequency of responses) or interval (length of time) that must occur for reinforcement to be delivered is predetermined and unchanging, ie, it remains the same throughout the program. With variable intermittent schedules, reinforcement fluctuates around a preset average and the learner never knows how many responses, or how long they must wait, for each reinforcer.

Crossing the two dimensions of intermittent reinforcement schedules results in four basic types of intermittent schedules of reinforcement: Fixed ratio (FR), variable ratio (VR), fixed interval (FI) and variable interval (VI). Numbers follow these acronyms to indicate the exact value of the unit of measure (Table 3.1.1). For example, FR 3 means every third response will be reinforced; VR 3 means the number of responses required for reinforcement will vary unpredictably around an average of every third response. An FI 6" means 6 seconds must elapse between the first reinforced response and the next. In a VI 10" schedule, the average period required before the next response is reinforced is 10 seconds.

Intermittent schedules of any kind are known to cause more persistent behavior than continuous schedules under conditions of extinction or very lean reinforcement. For example, many birds try to clamber out of their cages when the door is opened. Every once in a while they make it to the top of the cage. This intermittent reinforcement maintains their persistent effort to "escape" every time the door is opened.

The now classic analogy of the different rates of putting coins in machines observed with a coke machine vs. slot machines is a sound demonstration of the effects of dif-

ferent schedules of reinforcement: With the continuous reinforcement provided by the typical coke machine, most of us do not keep putting money in the slot if nothing comes out. Yet, many people continue to drop coins into slot machines with a very lean schedule of reinforcement. All things considered, our birds benefit most from our ability to "catch them being good" at as high a rate as possible and reinforcing them for it. One important benefit of this approach is that people who deliver dense schedules of reinforcement are more likely to become valued reinforcers themselves.

OBSERVATIONAL LEARNING

Observational learning describes the process of learning by observing the experience of another individual. As described in Chance,[7] it was not until the 1960s that research on observational learning really took off after initial results with monkeys were reported.[32] Since that time, research has demonstrated observational learning takes place with many different species including cats,[14] octopi,[10] bats,[11] children and adults.[16,17]

Irene Pepperberg's work with Alex, the African grey parrot, suggests the effectiveness of observational learning.[24] Her work also confirms that observational learning has enormous relevance to increasing adaptive behaviors with parrots that display limited companion repertoires or seriously maladaptive behaviors.

BEHAVIORAL MOMENTUM

Nevin hypothesized that the physics principle of momentum is a good metaphor for behavior.[22] He asserts that compliance to demanding or undesirable tasks can be increased by first requesting a series of easy or high-probability behaviors. He calls this procedure behavioral momentum. Behavioral momentum appears to be an effective positive strategy for increasing parrots' compliance to requests they initially balk at doing. For example, one author observed master trainer Phung Luu using this approach with a kea (*Kea nestor*) learning the husbandry behavior of entering a crate. Having a known negative history with crates (learned during the initial transport to the zoo), the kea ignored the cue to crate several times. Rather than forcing the bird into the crate or accepting that it wouldn't enter the crate, the trainer cued bird to several different perches in rapid succession, something the kea did without hesitation. Once the kea built up behavioral momentum by complying with the easy cues, the trainer asked it to crate at which point the bird actually leaped into the crate where a jackpot of food reinforcers was delivered. Caretakers can use the same procedure to build behavioral momentum with fun, easy behaviors before asking their birds to do something they are less than willing to do. Behavioral momentum is a positive and effective solution to overcoming behavioral resistance, much preferred over force.

Decreasing Behaviors

Scientifically speaking, punishment is the process by which a consequence decreases the behavior it follows and the consequence itself is called a punisher. As you can see, this simple, functional definition is quite different from common use, which often has more to do with venting anger than actual behavior change. Just like reinforcement, the effect of punishment depends on contingency and contiguity between the behavior and the consequence, as well as the schedule with which the punisher is delivered. Also, just like reinforcement, punishment is a very individual matter. A consequence that is punishing to one bird may not be punishing to the next bird. As always, the function of a consequence can be demonstrated only by observing the future rate of the behavior. If the behavior doesn't decrease over time, the procedure is not punishment.

There also is a distinction between positive (+) and negative (-) punishment. Positive punishment is the process of adding an aversive stimulus to the environment to decrease behavior; negative punishment is the process of removing something of value (ie, a reinforcer) from the environment to decrease behavior. Negative punishment includes relatively mild behavior-decreasing techniques such as extinction and time out from positive reinforcement, both of which are further discussed below.

Unfortunately, positive punishment is all too commonly applied to birds. To reduce unwanted behaviors, people rely on what they know, their "cultural knowledge," which is learned over a lifetime of personal experience with punishment. For lack of alternative information and skills, people often force their birds out of cages in towels, squirt them with water to move them off unapproved perches, and cover their cages to stop them from screaming. They are unaware or skeptical that positive reinforcement solutions are readily available to influence these behaviors.

NEGATIVE SIDE EFFECTS OF PUNISHMENT

As with negative reinforcement, people must be made aware of the predictable side effects occasioned by punishment. These devastating side effects are most likely to result from positive punishment procedures in environments with little opportunity for positive reinforcement. The negative fallout of all aversive strategies is important enough to repeat here:

1. escape/avoidance behavior,
2. aggressive behavior,

3. response suppression, and,
4. fear of people or things in the environment in which the aversive stimuli are presented.

Notice that one of the problems with punishment is *not* that it doesn't work. Punishment works to decrease behavior when executed correctly. This fact results in perhaps the most detrimental side effect of punishment — whenever punishment works to decrease an unwanted behavior, the person delivering the punishment is reinforced for using it. Therefore, s/he is more likely to use punishment in the future. This is not only disconcerting, it explains at least one reason punishment is so pervasive in our society, punishment often is reinforcing to the punisher.

DIFFERENTIAL REINFORCEMENT OF INCOMPATIBLE/ALTERNATIVE BEHAVIORS

Fortunately, there are effective alternatives to punishment for decreasing unwanted behaviors, which make use of differential reinforcement. Differential reinforcement first was introduced in the section on shaping, where continuous reinforcement was combined with extinction to advance from one approximation to the next closer approximation of the target behavior. In this section, two differential reinforcement strategies to decrease an unwanted behavior in favor of a desirable alternative are discussed.

With differential reinforcement of an incompatible behavior (DRI), we reinforce a behavior that is incompatible or mutually exclusive with the unwanted behavior, which we ignore. For example, if continuous screaming is targeted for reduction, we can reinforce talking because the two behaviors cannot occur at the same time. If biting people is targeted for reduction, we can reinforce chewing a foot toy because chewing a toy and biting a person are incompatible. DRI allows us to decrease the frequency of the undesirable behavior by increasing the frequency of an incompatible behavior with positive reinforcement. In this way, we take a positive reinforcement approach to decreasing undesirable bird behaviors.

Differential reinforcement of alternative behavior (DRA) is another way to indirectly decrease an unwanted behavior using positive reinforcement. With DRA, the behavior that is reinforced is not necessarily incompatible with the unwanted behavior, but is a more acceptable alternative. For example, a bird that bites to get you to remove your hand instead can be reinforced for a vocalization to make its protests known. Differential reinforcement is a highly effective approach to decreasing unwanted behavior without negative side effects and with all the benefits that positive reinforcement affords.

FUNCTIONAL MISBEHAVIOR

The example of a bird biting its caretaker's hand to result in the caretaker removing her hand from the cage brings up an interesting point: Problem behavior is often a misguided attempt by our birds to communicate a need and/or to get desired reinforcers such as our attention. For example, birds sometime display more raucous vocalizations and increased nippiness communicating that they are tired and ready for sleep. If we teach our birds more acceptable ways to communicate with us, we can decrease their undesirable behavior. This strategy has been validated in several studies with children who were self-injurious, aggressive to others and otherwise disruptive.[5] The problem behaviors the children exhibited served a valid communication function as evidenced by the significant decrease in the problem behaviors after the children learned more acceptable alternatives to gain objects, activities and attention.

With this hypothesis in mind, Alberto and Troutman[1] developed three criteria for selecting incompatible and alternative behaviors for DRI and DRA strategies that can be applied to solving behavior problems with our birds:

1. Always first analyze the inappropriate behavior to determine if it serves an important function for the bird. If it does, then a replacement behavior should be found that serves that function, but in a more appropriate way.
2. The alternative behavior should give the bird the same amount or more reinforcement than the unwanted behavior or it will just revert back to the inappropriate behavior in the long run.
3. DRI and DRA strategies work best if the incompatible or alternative behavior already is something the bird knows how to do. In this way, the effort the bird expends can be on replacing an unwanted behavior with a desirable behavior, rather than learning something new.

EXTINCTION

Extinction as it relates to shaping and differential reinforcement of alternative behavior already has been discussed, but it also can be used as a procedure to decrease an unwanted behavior by *permanently* withholding the reinforcement that has maintained it in the past. When human attention is the reinforcer maintaining a behavior, extinction is in effect when the behavior is ignored. Ignoring an unwanted behavior sounds easy enough, however, it actually is one of the most difficult techniques to use effectively.

First, many problem behaviors just cannot be ignored, such as extreme biting, screaming or chewing on woodwork. Second, extinction initially produces a reliable but

temporary increase in both frequency and intensity of the unwanted behavior during the beginning stages of the procedure, called an extinction burst. Extinction bursts give new meaning to the phrase, "It's going to get a lot worse before it gets any better." Therefore, when considering using extinction, the critical issue is not whether you can ignore current levels of the behavior, but whether you can ignore significantly escalated levels of the behavior until it finally begins to decrease. Extinction is a relatively slow process and people often inadvertently reinforce unwanted behaviors at these escalated intensities, resulting in worse problems than before they began extinction.

Another challenge using extinction is that we are not always in control of the source of reinforcement that maintains unwanted behaviors. Parrots can derive reinforcement from the feeling they get when they bite our skin and from the reaction of other birds, pets or children in the environment; even an echo in a particular room can reinforce screaming. In these cases, where "bootleg" reinforcement is available to the bird, our efforts to pay no attention to the behavior will have no effect.

Finally, even after a behavior is successfully extinguished, we can count on its sudden reappearance over time. If we prepare caretakers for this "spontaneous recovery," they will more likely reinstitute extinction immediately rather than conclude the initial procedure failed. The good news is that with each reapplication of extinction the behavior is less likely to reappear in the future. Nonetheless, for these reasons, our best strategy for reducing unwanted behavior is differential reinforcement, ie, the combination of extinction of the unwanted behavior and reinforcement of a more adaptive behavior alternative. A sound axiom to guide caretakers in their choice of managing difficult behavior is, "Replace rather than eliminate." By following this rule, we teach the bird what to do instead of solely what not to do, we maintain a higher level of reinforcement and we preserve the function for the bird that was served by the original unwanted behavior.

TIME OUT FROM POSITIVE REINFORCEMENT

Time out from positive reinforcement (TO) is another negative punishment procedure used to decrease unwanted behavior. With TO, behavior is decreased by *temporarily* removing access to desired reinforcers. For example, birds can be taught to leave shirt buttons alone by setting the bird down for a few seconds contingent on the bird moving toward or touching a button. If being with the caretaker is reinforcing, removal from the caretaker will decrease the biting behavior given good delivery of the consequence (ie, contingency and contiguity).

A functional analysis of this program might look like this:
Antecedent: Caretaker is holding bird.
Behavior: Bird puts beak on button.
Consequence: Caretaker removes bird to the nearby counter for several seconds.
Prediction: Bird will bite button less to stay with caretaker.

The most common way people fall short with this strategy is by not really removing access to reinforcement at all.

For example, consider the following analysis:
Antecedent: Caretaker is busy preparing dinner.
Behavior: Bird flies to newly reupholstered couch.
Consequence: Caretaker gets bird and walks down the hall, up the stairs, steps over the sleeping dog, passes the ringing phone, passes through the door of the bird room and returns bird to its cage.
Prediction: Bird will fly to newly reupholstered couch to get more time with the caretaker on the way to a distant cage.

At that point, the bird hardly could be aware of the contingency between the misbehavior and the consequence meant to reduce it.

Three additional ways TO is commonly used ineffectively is when:
1. birds are removed from reinforcing activities for too long,
2. birds are not given another chance to behave appropriately soon after the "infraction," and,
3. the caretaker adds reinforcing emotional reactions including brusque movements, strained voices and angry faces.

The effectiveness of TO is greatly increased by following these suggestions:
1. Ensure clear contingency and contiguity by selecting a nearby TO location.
2. Keep TO short, no more than a few minutes or the bird likely will forget the connection between his behavior and the consequence.
3. After a short TO, bring the bird right back to the "scene of the crime" to earn reinforcement for doing it right.
4. Let TO do all the work for you. There is no need for other consequences or histrionics, which likely will reinforce the unwanted behavior.

Although TO is a punishment procedure, there is some evidence with children that suggests it can be used without producing the negative side effects of positive punishment.[25] In this sense, well-executed TO is a relatively

mild strategy for reducing negative behavior. Even so, antecedent arrangements and positive reinforcement strategies should always be tried first before using any other strategy. If strategies such as extinction or TO are used, special attention should be paid to arranging and reinforcing positive behaviors at a high rate to maintain a positive total environment.

Conclusion

Were it not for parrots' extraordinary ability to adapt on an individual level, one might conclude that at the species level they are genetically ill equipped for the captive environment. Indeed, this may well prove to be the case for some species of parrots. Their high-decibel shrieking, ratchet beaking, food flinging, exclusive bonding, wood remodeling and long-distance flying ways make them demanding animals to care for in our homes. Ensuring parrots' success as companions will require an increased awareness of their species tendencies to set the behavioral context, and a sound working knowledge of how animals learn in order to teach them behaviors well adapted to our homes.

For years, the pervasive approach with companion parrots has been little more than a reflection of cultural beliefs about behavior. The application of scientific information has been scarce. Based on these beliefs, many people assume that behavior is caused by invisible forces originating inside the bird rather than the perpetual interaction between the individual and the environment. For example, one commonly advanced theory is that parrots are driven by a desire for dominance. This is not a benign theory, as it predisposes people to interpret behavior as a struggle for position in some supposed hierarchy and, therefore, to advocate management practices designed for caretakers to win the struggle. Such practices often are forceful and coercive, relying heavily on negative reinforcement and positive punishment, both of which are defined in part by the presence of aversive stimuli.

As a result of this dominance-drive theory, caretakers have been endlessly instructed how to take charge of their birds' behavior, issue commands and establish their superior rank. They've been encouraged to establish control by prying their bird's toes off perches, threatening their birds with towels and ignoring their bird's bites of protest. One of the most disturbing aspects of this dogma is the repeated use of an analogy to sound parenting practice so described: "You wouldn't allow a small child to decide whether or not to take a bath, now would you?" No, we would not; however, the method of choice to facilitate children's bathing would not be to pry, threaten or ignore cries of protest to get them into the tub. The first step in solving behavior problems is to identify the stimuli in the environment that set the occasion for and reinforces resistance to a reasonable request. The next step is to create an environment that sets the occasion for and reinforces adaptive, cooperative behaviors.

A common criticism voiced by advocates of negative reinforcement and punishment is that positive reinforcement results in increased permissiveness. On the contrary, the skills we want our captive parrots to exhibit do not have to change with this urgent call to change the strategies we use to teach them. For example, with positive reinforcement, parrots can quickly and easily be taught to step up from *all* perching areas; with differential reinforcement of an alternative behavior, parrots can be taught to voice their displeasure rather than bite; and with shaping, parrots can be taught to play independently for a reasonable duration rather than scream incessantly for attention.

Over the course of decades researching and teaching about positive reinforcement, we have heard many unfounded trepidations. Countless times caretakers have asked if teaching with positive reinforcement solutions diminishes intrinsic motivation, results in reward addictions, suppresses the root causes of behavior while addressing mere symptoms, exchanges one symptom for another, promotes bribery, works only with intelligent learners, works only with simple behaviors, requires massive amounts of treats and takes too much work. We are confident to report that given the extensive experimental research base, combined with decades of successful application in schools, zoos and other settings, it is clear that positive reinforcement increases our teaching efficacy in myriad ways and that these concerns are unfounded. And, we are heartened to observe among the parrot-owning public that more and more people are questioning the drawbacks and limitations of using punishment.

Foremost among the many benefits of positive-first teaching is that parrots are taught what *to do* rather than *not do*, and they are empowered to operate on their environment in ways that result in competence and self-reliance. These benefits are especially important in light of the extensive research on learned helplessness, a class of behaviors that results from having little effect on one's own outcomes when repeatedly exposed to aversive events.[20] Not only does learned helplessness result in a loss of motivation to improve one's condition when improvement is possible, it is also associated with deficits in learning, performance, and emotional problems. As this research has been replicated with cockroaches,[4] dogs, cats, monkeys, children and adults,[20,23] we

have every reason to believe that these effects also are common among parrots.

Finally, applied behavior analysis not only empowers parrots but caretakers as well. Caretakers learn that behavior is functionally related to environmental antecedents and consequences, not some immutable force within. They know where to look to affect behavior directly with positive-first solutions, one behavior at a time, and they understand that to change their birds' behavior, they must change what they do. With a beginning knowledge of the principles of learning and behavior, caretakers also are better able to make reasoned, informed decisions about alternative, less positive approaches, as needed.

Veterinarians often are in the position of being the first and most credible authority parrot owners turn to for guidance on the behavior of their birds. We could do no better than to turn to the dual sciences of ethology and applied behavior analysis to lead us into a new era of understanding and skill with behavior. In this way, realistic expectations for companion parrots will emerge, as will the commitment to apply scientifically validated, positive-first behavior management strategies. Veterinarians who are knowledgeable about species-level behavior and individual learning will dramatically change the future of companion parrots and their caretakers.

Web Sites Recommended by the Author

1. www.avi-train.com
2. www.naturalencounters.com
3. www.thegabrielfoundation.org
4. www.groups.yahoo.com/group/Bird-Click
5. www.parrottalk.com

Books Recommended by the Author

1. Animal Training: Successful Animal Management through Positive Reinforcement, by Ken Ramirez (1999).
2. Clicking with Birds: A Beginners Guide to Clicker Training Your Companion Parrot by Linda Morrow (available at www.avi-train.com/manual.html).
3. Clicker Training with Birds, by Melinda Johnson.
4. Don't Shoot the Dog: The New Art of Teaching and Training (revised edition), by Karen Pryor.
5. Good Bird! A Guide to Solving Behavioral Problems in Companion Parrots! by Barbara Heidenreich. 2004, Avian Publications, Minneapolis, MN, www.avianpublications.com.
6. The Power of Positive Parenting A Positive Way to Raise Children, by Glen Latham.

References and Suggested Reading

1. Alberto PA, Troutman AC: Applied Behavior Analysis for Teachers 5th ed. Merrill-Prentice Hall, 1999, pp 287-288.
2. Azrin NH, Holz WC: Punishment. *In* Honeg WK (ed): Operant Behavior: Areas of Research and Application. New York, Appleton-Century-Crofts, 1966.
3. Breland K, Breland M: The misbehavior of organisms. Am Psychol 16:681-684, 1961.
4. Brown GE, Hughs GD, Jones AA: Effects of shock controllability on subsequent aggressive and defensive behaviors in the cockroach (*Periplaneta americana*). Psychol Reports 63:563-569, 1988.
5. Carr E, Durand VM: Reducing behavior problems through functional communication training. J Applied Behav Anal 18:111-126, 1985.
6. Chance P: Learning and Behavior 5th ed. California, Thomson Wadsworth, 2003, 439.
7. Chance P: Learning and Behavior 5th ed. California, Thomson Wadsworth, 2003, 266.
8. Chance P: Learning and Behavior 5th ed. California, Thomson Wadsworth, 2003, 24.
9. Dethier VG, Solomon RL, Turner LH: Sensory input and central excitation and inhibition in the blowfly. J Comp Physiol Psychol 60:303-313, 1965.
10. Fiorito G, Scotto P: Observational learning in *Octopus vulgaris*. Science 256:545-547, 1992.
11. Gaudet CL, Fenton MB: Observational learning in three species of insectivorous bats (Chiroptera). Anim Behav 32:385-388, 1984.
12. Gray P: Psychology 3rd ed. New York, Worth, 1999, p 73.
13. Hare B, et al: The domestication of social cognition in dogs. Science 298:1634-1636, 2002.
14. Herbert M J, Harsh CM: Observational learning by cats. J Comp Psychol 37:81-95, 1944.
15. Herrnstein RH: Relative and absolute strength of response as a function of frequency of reinforcement. J Exper Anal Behav 4:267-273, 1961.
16. Higgins ST, Morris EK, Johnson LM: Social transmission of superstitious behavior in preschool children. Psychol Rec 39:307-323, 1989.
17. Kanfer FH, Marston AR: Human reinforcement: Vicarious and direct. J Exper Psychol 65:292-296, 1963.
18. Lantermann W: The New Parrot Handbook. New York, Barron's, 1986, pp 91-94.
19. Lattal KA: Contingency and behavior analysis. Behav Analyst 24:147-161, 1995.
20. Maier SF, Seligman MEP: Learned helplessness: Theory and evidence. J Exper Psychol: General 105:3-46, 1976.
21. Manning A, Stamp Dawkins M: An Introduction to Animal Behavior. Australia, Cambridge U Pr, 1992, pp 77.
22. Nevin JA: The momentum of compliance. J Applied Behav Anal 29:535-547, 1996.
23. Overmier JB, Seligman MEP: Effects of inescapable shock upon subsequent escape and avoidance responding. J Comp Physiol Psychol 63:28-33, 1967.
24. Pepperberg IM: The Alex Studies: Cognitive and Communicative Abilities of Grey Parrots. Massachusetts, Harvard U Pr, 2000.
25. Rortvedt AK, Miltenberger RG: Analysis of a high probability instructional sequence and time-out in the treatment of child noncompliance. J Applied Behav Anal 27:327-330, 1994.
26. Sharpless SK, Jasper HH: Habituation of the arousal reaction. Brain, 79:655-680, 1956.
27. Skinner BF: The Behavior of Organisms. BF Skinner Foundation, 1938.
28. Skinner BF: Science and Human Behavior. New York, Macmillan, 1953.
29. Skinner BF: Selection by consequences. Science 213:501-504, 1981.
30. Sulzer-Azaroff B, Mayer GR: Behavior Analysis for Lasting Change. Florida, Harcourt Brace Jovanovich, 1991, p 180.
31. Thorndike EL: Animal Intelligence: Experimental Studies. New York, Hafner, 1911.
32. Warden CJ, Jackson TA: Imitative behavior in the rhesus monkey. J Genetic Psychol 46:103-123, 1935.

Concepts in Behavior: Section II

Early Psittacine Behavior and Development

LIZ WILSON, CVT, PHOEBE GREENE LINDEN, BA, MA;
TERESA L. LIGHTFOOT, BS, DVM, Dipl ABVP-Avian

Ethologists have yet to map out the stages of development for psittacine birds. As a consequence, the information about companion psittacine behavior is predominantly anecdotal and experiential. The wide variation in maturation rates between species as well as between individuals within species, as exemplified in Table 3.2.1, further complicates this issue. As a general rule, the smaller the species, the faster an individual of that species will mature. For example, most cockatiels (*Nymphicus hollandicus*) are sexually mature by 6 months of age, whereas the average 6-month-old hyacinth macaw (*Anodorhynchus hyacinthinus*) has not yet developed the physical coordination to consistently walk without stumbling.

Generally speaking, small species like budgerigars (*Melopsittacus undulatus*) and cockatiels fledge around 3 to 4 weeks, wean around 6 to 11 weeks and enter puberty at 4 to 6 months. Medium-sized birds (*Psittacus erithacus* and *Amazona* sp.) fledge at 10 to 12 weeks, wean around 12 to 16 weeks and enter puberty at 3 to 4 years of age. Larger psittacines such as *Ara* spp. fledge at about 12 to 15 weeks, wean around 16 to 20 weeks and enter puberty at about 4 to 5 years. Table 3.2.1 presents more detailed information regarding representative species.

Table 3.2.1 | Stages of Development

Species	Fledge (weeks)	Wean (weeks)	Puberty Onset	Sexual Maturity (years)	Geriatric** (years)	Life Span[47] (years)
Budgerigar (*Melopsitticus undulatus*)	3-4	6-7	4-6 months	1	6-12	18
Cockatiels (*Nymphicus hollandicus*)	3-6	7-11	4-7 months	1	12-18+	32
Sun conure (*Aratinga solstitialis*)	6-7	8-9	9-18 months	2	18-25	25
Green-cheeked conure (*Pyrrhura molinae molinae*)	4-6	6-12	9-18 months	2	12-15	25
Peach-faced lovebird (*Agapornis roseicollis*)	3-6	7-11	7-8 months	1	10-15	12
Yellow-naped Amazon (*Amazona ochrocephala auropalliata*)	11-13	15-18	4-6 years	7	35-45	
Blue-fronted Amazon (*Amazona aestiva*)	10-12	12-16	3-5 years	6	25-35	80
Congo grey (*Psittacus erithacus*)	10-12	12-16	3-5 years	6	20-25	50
Eclectus parrot (*Eclectus roratus*)	10-11	14-16	3-5 years	6	15-20	20
Galah (rose-breasted cockatoo) (*Eolophus roseicapillus*)	8-10	11-18	1-2 years	4	18-20	20
Umbrella cockatoo (*Cacatua alba*)	10-12	12-18	3-4 years	8	20?	
Moluccan cockatoo (*Cacatua moluccensis*)	12-15	16-25	3-5 years	10	25?	
Yellow-collared macaw (*Ara auricollis*)	9-10	10-12	1-2 years	4-5	22-27	
Blue and gold macaw (*Ara ararauna*)	10-12	14-22	4-6 years	8	30-40	50
Green-winged macaw (*Ara chloroptera*)	12-15	16-35	5-7 years	10-11	35-45	

**True onset of "geriatric" in psittacines is subjective and requires several generations of captive-bred individuals of each species to determine.

Behavior Development and Changes

The larger parrots are generally sexually mature by 3 to 5 years of age. This slow rate of maturation often confuses those who assume that the behaviors displayed in this prolonged babyhood will be permanent rather than transient. For instance, a 2-year-old scarlet macaw (*Ara macao*) is not yet an adult and remnants of youthful behaviors exist. This scarlet macaw can be expected to undergo significant behavioral changes in the future.

As a consequence, some owners are startled and upset at the behaviors displayed as their parrots mature. Those caregivers who are unprepared for avian adulthood routinely complain to veterinarians and behavior consultants that they "want their sweet baby back." The concept of neoteny is powerfully appealing to humans.[5] However, the reality is that parrots continue to grow, mature and change. Psittacine behavior is readily influenced by positive interactions, so caregivers need not lament passing babyhood, but instead embrace life-long learning.

THE NEONATE

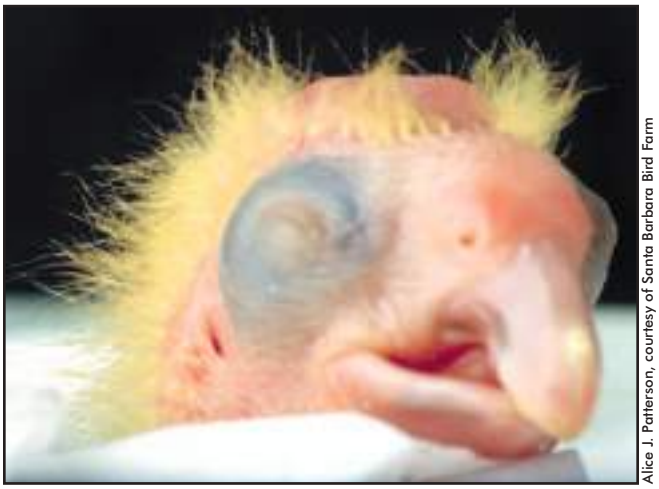
Parrots are altricial and are unable to thermoregulate, even in those species that are born with down feathers. Baby parrots are considered neonates from hatching until their eyes open, and during that time their physical needs are simple but absolutely critical: a warm environment and warm food (Fig 3.2.1).[15]

Feeding

Many breeders feed their babies on a rigid schedule of every 1 to 4 hours, depending on their ages.[24] Waking baby birds to feed them or forcing hungry babies to wait for food until the next scheduled feeding is needlessly stressful for young birds. The optimal feeding schedule is sensitive to the bird's needs, including their rapid increases in weight and the concurrent increase in the volume of food required at each feeding.

Young budgerigars and cockatiels have been documented to solicit feeding with typical baby cries only upon the entrance into the nest box of a parent bird. Occasionally a nestling will cry to solicit feeding from a sibling. The only other vocalizations made are the hissing sound (made by cockatiels even at a young age) at the presence of an intruder in the nest box. Extrapolating this to captive bred nestling parrots would indicate that healthy young psittacine hatchlings should not be prone to indiscriminate or long-lasting crying binges.

Concern with the prevention of sour-crop has promoted the axiom that the crop should be completely empty



Alice J. Patterson, courtesy of Santa Barbara Bird Farm

Fig 3.2.1 | Despite its blindness, this Moluccan cockatoo hatchling is still exquisitely sensitive to its environment. At this age, temperature is everything, both in environment and food.

prior to the next feeding. Several pitfalls are inherent if this guideline is followed. Liquid food that is fed tends to stretch the crop, and a small pendulous area of crop may retain liquid food for a prolonged period. Waiting for this material to empty produces a hungry baby and creates both emotional and physical stunting. *(Ed. Note: Retention of a desiccated portion of the hand-feeding formula may indicate a poorly formulated product, improper temperature of the formula when fed, or prolonged crop-emptying related to illness.)* Observation of parent-fed babies demonstrates the normal state of marked crop distention.

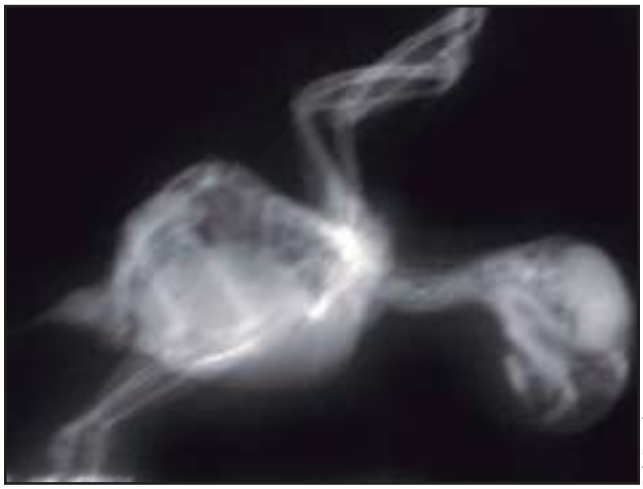
Chronically whining baby parrots (especially within the cockatoo family) may be related to prolonged hunger. Breeders who no longer adhere to the rigid scheduled feeding style report neonates that sleep soundly for consecutive hours and awaken eager to eat. Rather than forcing babies to adhere to schedules tailored to human convenience, feeding babies on demand is best for proper physical and emotional growth *(Ed. Note: Recent research on African Greys* (Psittacus erithacus) *and* Pionus *spp., showed that many babies from parents fed a seed-based diet had some degree of osteomalacia radiographically. Figs 3.2.2a-b demonstrate an advanced case of bony deformation in a ring-necked parakeet. In addition to physical malformations in severe cases of osteomalacia, chronic pain may be present in subclinical cases. Chronic pain at a young age may contribute to excessive crying and potentially to future behavioral problems. See Chapter 5, Calcium Metabolism for further discussion and references.)*

Touch

Psittacines wild-caught as adults and not socialized to humans may be adverse to touch. However, naturally

Bob Doneley

Fig 3.2.2a | An extreme example of osteomalacia from a malnourished diet.



Bob Doneley

Fig 3.2.2b | The radiograph of the bird in Fig 3.2.2a. Such birds are in pain based on similar problems in species that show more of a pain response, such as monkeys.

occurring parental touch has been reported in many psittacine species. Aviculturist Katy McElroy collects video nest box documentation of Moluccan cockatoos as they lay, incubate, hatch and nurture a baby to fledging. She and subsequent observers are impressed by the "lavish attention" the parents paid to their baby, continuously preening and touching their offspring all over its body between feedings. In one film, McElroy's parent birds are seen with their single baby; all three birds are asleep in the nest box with the baby tucked under its father's wing, its head laid across his back. Like most animals, parrots are most likely adverse to rough handling, but readily accept appropriate touching, especially when raised to do so from an early age.[20]

There is an obvious positive reaction to touch, with neonates responding to soft stroking and wing tip massage by pushing their heads into the human hand. Prior to the opening of their eyes, they also will respond to the sound of a familiar step and voice, popping their heads up on wobbly necks. One author (PL) has noted the importance of duplicating the weight of the parent bird's wing when raising babies in incubators, especially when a single chick is housed alone. The baby or babies should be placed into a secure container and covered with towels that rest lightly on the neonates. Soft weight on the back quiets a baby after feeding.

McElroy reports that her video has documented the extreme sensitivity of baby cockatoos to parental and sibling touch. "They sit upright on their bottoms a few hours after hatching and use their feet propped out like stabilizers to keep from tipping over. The slightest touch will cause them to spin around in that direction, using one foot as a pivot as they search for food or a warm body. You rarely see a baby that isn't snuggled up against a sibling or parent. If two blind neonates get accidentally kicked apart when the parents leave the box, they will both stretch their necks out and lurch around in circles until making contact" (K. McElroy, personal communications/e-mail, 2002). The importance of physical contact for neonates should not be ignored. The keeping of multiple chicks together increases normal physical stimulation. Incubator-hatched birds should have touch massage incorporated into their daily care.

Light

Prior to their eyes opening, neonatal parrots are responsive to light. Biologically designed to begin life in the darkness of a tree cavity, baby parrots react adversely to strong light by flinching, hiding or trembling. As a consequence, the popular practice of keeping babies in glass aquariums under fluorescent lights not only is potentially detrimental to the developing eyes of neonates, but also might cause psychological distress.[15] Because their eyes need time to develop slowly in a darkened cavity, many aviculturists supply neonates with a darkened container in which to grow and develop. Hand-raised psittacine babies actually gain weight faster when kept in the dark.[25]

THE NESTLING

After the neonatal psittacine's eyes open, the baby is categorized as a nestling. Psittacine birds with recently opened eyes seem to be myopic, which is consistent with most newborn animals. Certainly babies who are confined in a closed container have no need to see across vast distances. If given the opportunity to do so too early in the development process, nestlings will blink, recoil and seek a dark corner. They do, however, move toward and touch objects in close proximity, so boxes can be enriched at this stage to encourage visual development.

Fig 3.2.3a | This African grey baby was raised in a plastic incubator with its physiologic needs met. However, the excessive exposure to light, lack of parental or sibling weight, heat and support, are potentially psychologically devastating. Here the baby is hiding after being frightened by a ringing phone.

Layne Dicker, courtesy of Santa Barbara Bird Farm

Fig 3.2.3b | Baby blue and gold macaws benefit from an enriched cardboard box environment with a covered corner for hiding when needed.

As feathers develop and open, the need for supplemental heat decreases, and young psittacines can be moved out of brooders into unheated containers that allow more movement but still resemble a nest. The limited space has been demonstrated by Nigel Harcourt-Brown to be a critical factor in the prevention of valgus deformities of the legs in neonatal birds.[15a] When the babies are fully feathered, they can be housed at room temperature, (72-78° F, 22-26° C), which should be carefully monitored. Various containment systems employ the judicious use of towels to provide darkness, privacy, traction and hiding places that appear to be critical to a stress-free environment. For further comfort and physical and psychological safety, food sources must continue in a dependable manner and the environment must remain secure.

Visual Stimulation

Appropriately stimulating environments are vital to mental development; the use of bright colors and accessible, touchable toys are enrichments that are simple to incorporate into young psittacine environments. Designs on nursery walls and colorful mobiles are examples of visual enrichment for birds at the peri-fledgling stage when they are perching intermittently on the edge of the nest box.

As the baby bird develops increased visual ability and physical mobility, increased opportunities for learning must be provided. Fearless curiosity is characteristic of young animals,[5] and this characteristic is best utilized in teaching the young bird to competently deal with the world. In addition to the previously noted necessities of warmth, food and security, the neonates' environment needs increasing stimulation in terms of vision, touch, sound and interaction.

Borrowing again from early development of more extensively studied species, it is fair to assume the existence of a window of opportunity for the development of visual recognition, learning and acceptance in psittacines. Therefore, to make the view more interesting, one can hang bright posters and add plants (either real or artificial) to the nursery. People can wear bright colors when working with babies. Colorful towels that cover the containers are simple enrichments. By rotating the towels every couple of days, caretakers can ensure the babies become accustomed to different patterns and colors. In this manner, an early foundation is laid that encourages the birds to be receptive to change.

Baby parrots raised in opaque plastic tubs receive no visual stimulation. Cutting a notch in the side of the tub (melting the edges so they are not sharp) so the babies can see out can counteract this lack of visual stimulation. Organic containers are more natural and stimulating: simple cardboard boxes (which can easily be cut to provide a view and disposed of when soiled) or inexpensive natural-colored baskets. Whatever the environment, caretakers should cover most of the container with towels throughout the day, and cover it completely at night. This provides privacy as well as darkness, should a baby become overstimulated (Fig 3.2.3a). The towel coverings also influence thermoregulation and should be adjusted as needed (Fig 3.2.3b). Both the need to withdraw from stimulation and the need for warmth decrease as the birds continue to develop.[27]

Most veterinarians use towels to restrain birds. Acclimating a bird to being restrained in a towel will reduce the stress of veterinary visits and aid in grooming at home. Initially, cover the baby with the towel and let


Bob Doneley

Fig 3.2.4a | Picking a safe color towel and teaching a bird restraint will make veterinary exams and training easier as a bird matures. Here a bird is introduced to a towel.


Bob Doneley

Fig 3.2.4b | Slowly covering the bird with the towel in a reassuring way.


Bob Doneley

Fig 3.2.4c | Adding pressure and restraint slowly over time allows grooming and examination or restraint whenever needed in a safe unfrightening way.

it sleep. As the bird nears fledging, it can be carried around in the towel. It can then be introduced to different areas of the house, people, objects, and other pets, using withdrawal into the towel for security. It is likely, as is documented in dogs and cats, that if a bird is not introduced to certain animal species by a certain age, it will have difficulty accepting the presence of this species in the future without fear. The positive and negative results of creating this fearless state need to be considered (See Socialization and Co-parenting Section to follow [Figs 3.2.4a-c]).

Parrots spend large amounts of time with their faces close to their babies. As the babies develop, visual contact—face-to-face and eye-to-eye—soon expands into vocal interactions post feeding, as the babies respond to gentle murmuring of the hand-feeder. Introduction to adult foods during the neonatal period will increase acceptance and prevent the development of food rigidities in the future (Figs 3.2.5a-b).

Tactile Stimulation

Periodically stroking baby birds with warm hands simulates parental attention. One author (PGL) has observed the following regarding toenail sensitivity in baby birds:

"Touch the toenail clipper to the nail while holding the baby securely. Some birds do not flinch at all, while others react with varying degrees of withdrawal. Interestingly, this relative sensitivity is consistent into fledging."

Subsequent training exercises are used to desensitize the tender-footed neonate at an early age, while continuing to increase the amount of reinforcement the less reactive baby receives.

Aural Stimulation

Vocal communication between parents and chicks begins early. Hand-feeders are encouraged to talk to the babies in their care, accustoming them to human voices and language. Varieties of other types of sounds also are healthy and useful. The positive aspects of music have been proven repeatedly with animals as well as people.

Reactive Attachment Disorder

Reactive attachment disorder involves children 5 years old or younger. This condition was known in previous centuries as orphanage baby syndrome and is better known in the current lay literature as failure to thrive. Defined as "a disturbance of social interaction and relatedness", this condition is associated with "grossly pathological care, with persistent disregard for a child's basic emotional needs for comfort, stimulation and affection, as well as repeated changes of the primary caregiver that prevent the formation of appropriate bonds."[2] This severe absence

Greg J. Harrison

Fig 3.2.5a | Shown are examples of the most valuable types of moist foods to be offered to baby birds to teach variety. Starting at mid left going clockwise: organic acorn squash, lettuce, beets and beet tops, broccoli, carrots, yams (sweet potatoes) and butternut squash.

Greg J. Harrison

Fig 3.2.5b | An example of the proper kinds, amounts and types of organic dry foods to teach a baby bird to accept, for macaw-sized birds on the left and Amazon-sized birds on the right. These bowls show the amount of food offered to a pair of the respective birds mentioned. The limiting of seeds and nuts is vital; these are offered only to breeding birds until the babies are weaned. Then the nuts and seeds should be stopped altogether for the babies and suspended until the next breeding period for the parents.

of care can result in serious psychological and *physical* problems in children, such as stunted growth, the inability to socialize appropriately and increased potential for self-destructive behaviors later in life.[13]

Currently, many captive-bred, hand-fed parrots do not know how to play, accept appropriate touching, interact or even how to eat a variety of foods. Perhaps as a result of improper or incomplete early development, increasing numbers of parrots engage in feather destruction and even self-mutilation as adults. Increasing numbers of young domestically bred and hand-fed parrots seem unable to form a healthy relationship with humans. The authors wonder if these increasing problems are related to psittacines being raised in an assembly-line fashion in a cold, clinical nursery. If this is the case, the production-raising of psittacine birds is not the best technique for producing an emotionally and physically stable companion animal. Happily, the industry seems to be turning away from production techniques, as evidenced by the important work being done in large facilities such as the University of California-Davis and Texas A&M.[26]

The dangers of creating "failure to thrive" are lessened to an extent by raising neonates together rather than in individual enclosures. When aviculturists have substantiated the health of their babies (see Chapter 21, Preventive Medicine and Screening), the young of certain species may be housed together; in these mixed-species settings animation and interaction increase. Early work on raising psittacines in mixed-species groups yielded such good results —youngsters that seek touching, sleep readily, play with and seem curious about others —that the practice is widely accepted by many breeders today.

Socialization and Co-parenting

Socialization is a process by which an individual forms an attachment to other species.[3] Time frames for socialization in birds are not established as they have been for dogs and cats. The hand-feeding of psittacine chicks certainly provides exposure to humans. Conversely, provision of all the natural elements outlined in this chapter—feeding, warmth and tactile, visual and vocal stimulation—can be difficult for the human caregiver to provide. Ongoing studies at the University of California-Davis with co-parenting have shown great promise. Pairs of orange-winged Amazons (*Amazona amazonica*) were raised by their parents through fledging, with university students interacting with the young in the nest box for brief but regular periods of time. The study is ongoing, but preliminary results show that limited handling by humans for short periods, several times a week, may produce offspring that are socialized to humans, but benefit from all the inherent advantages of parent-raised birds. Extreme caution should be exercised in the selection of parents and young for this protocol to ensure that parental infanticide, abandonment or abuse does not occur.

THE FLEDGLING

Prior to fledging, babies show increasing interest in the world outside their enclosure. A partial covering of towels will enable the babies to see out of their container or to retreat and hide. As they get braver, they will spend more time looking out of the container and less time in concealment. There is often a great deal of wing flapping that happens inside the nest as babies start building up their pectoral muscles.

As their foot and leg strength develops with exercise, low perches should be added to the inside of the container. In this way, babies can perch when they wish and also stand flat-footed when they prefer.

As their distance vision improves, objects that stimulate vision can be added to the nursery walls. When possible, a view of the outdoors should be provided. Visual acuity continues to increases as the bird spends more time observing objects and movement outside of the nest box. For instance, immediately after opening their eyes, Eclectus nestlings will track food as it comes toward their eyes, but remain largely unresponsive to noiseless activities farther away. As the weeks progress, these birds track movements at greater distances. Just prior to fledging, birds can be seen scanning the horizon, (e.g., tracking an airplane's progress in the distant sky), and then quickly adapting their vision to objects presented at close range. Play behavior increases with developing visual acuity. McElroy describes a 6-week-old Moluccan chick that plays with a parent's molted feather in the nest box, flapping her wings wildly and rolling around as if she had another bird as a playmate.

A fledgling is a young bird that is learning to fly. In nature, fledging happens prior to weaning, which is logical when one realizes that a parrot baby must develop the physical strength and dexterity to learn controlled flight before it follows its parents to various and distant food sources. Only then is it sufficiently developed to achieve the complex manual dexterity necessary to eat on its own. Flight competence therefore precedes weaning in psittacine birds.

In the past, aviculturists automatically clipped the wings of baby parrots at their first attempt at flight. The popular belief was that the psittacines would not miss flight if never allowed to fly. However, in the last few years it has been recognized that fledging can make a marked difference in a bird's physical and emotional development, even when the wings are later clipped.[17] Fledging is a normal part of psittacine development and allowing this stage to progress naturally makes the weaning process much easier.[16] An excellent example of this phenomenon is the African grey parrot. African grey babies formerly were perceived as being awkward and prone to falling. Actually, this species is amazingly adept at flight and readily learns to maneuver in mid-air if given adequate opportunity. After all, wild babies that fall frequently surely could not survive. The African grey's reputation for clumsiness has more to do with early wing clipping than with any inherent lack of coordination.

The fledgling psittacine is a creature obsessed. Though still food-dependent, the fledgling often loses interest in eating. Flight becomes all-consuming and weight loss

Lori Reilly Walton, courtesy of Santa Barbara Bird Farm

Fig 3.2.6 | A blue and gold fledgling displays a partial wing clip that slows flight but does not curtail it. Graduated clipping done over a period of several days is recommended over a severe clip that ends flight abruptly.

is normal at this stage, as the youngsters lose baby fat and slim down to a more streamlined, aerodynamic figure. Inexperienced hand-feeders often are confused by this phase if they assume that the lack of interest in food indicates that fledglings are starting to wean. This is not the case. The process of weaning doesn't begin for another week or more after fledging, so caretakers should continue to hand-feed appropriate foods at every possible opportunity. Although requiring a dedicated area to avoid serious damage to the bird or one's environment, fledging confers significant developmental advantages. The young psittacine's coordination, muscular structure and social skills increase as the fledgling learns to interact with a wider variety of flock members once flight is achieved.

Should a new owner wish trimmed wings on his young parrot, a gradual wing clip is preferable to the abrupt curtailment of flight. Instead of a drastic clipping of flight feathers, graduated clips, several days apart, should be performed, gradually limiting flight (Fig 3.2.6).

THE WEANLING

"To wean" is defined as "to accustom to take food other than by nursing" and second as "to detach from a source of dependence."[22] A weaned parrot is capable of survival with little or no guidance in procuring adequate nutrition. Weaned wild birds, therefore, find and supply themselves with a variety of nutritious foods in sufficient quantity.[14] The best weaning process for psittacine companions allows eating skills to develop gradually over a period of several weeks. Unfortunately, many pet stores and breeders consider a baby parrot "weaned" as soon as it shows interest in eating on its own; which is a regrettable and potentially fatal misconception. The weaning process is a gradual process wherein a baby parrot learns where, what and how to eat. There are

Laura Price, courtesy of Santa Barbara Bird Farm

Fig 3.2.7 | A young female eclectus enjoys a meal of squash and the novelty of new foods. Just as in human children, messy eating and experimentation are part of the process of learning to accept and enjoy foods of various textures, shapes and tastes.

numerous motor skills necessary to accomplish this, and these take time to develop.

Psittacine parents assist in the weaning process by holding food in their beak and feet for their babies. Human caretakers can assist parrot weanlings by finger-feeding warm, wet food. Ideal foods for this technique include chunks of cooked squash, carrots or yams and mango. Weaning pellets, or the type of formulated diet that is to be fed when the bird is weaned, can be soaked and offered in this same manner (Fig 3.2.7). All foods are warmed and moistened in hot water or fruit juice. The temperature range is critical for maximum palatability and digestion. Foods should be warmed to 104 - 105° F (38-38.5° C). Candy thermometers work well for this and are available in any kitchen supply store.

Using a camera in the nest box of a pair of wild-caught Moluccan cockatoos (*Cacatua moluccensis*), the process of raising a youngster was videotaped for 11 months. Although the baby was noted to be typically noisy while being fed, it was never noted to cry for food or to get the parent's attention (K. McElroy, personal communications, 2002).

Conversely, Harrison's observation of a trio of black cockatoos in Australia depicted the parents and their young flying from tree to tree. While the parents located and ingested food, they ignored the crying of their accompanying youngster for 30 minutes. The youngster finally chose and ingested food on its own. This was obviously the final stage of weaning, as the baby was not easily differentiated from the adults.[10]

A possible explanation for variations of parental participation in feeding young after fledging may be reflected by the environmental conditions of the species. Dissimilar environments might produce substantial disparities in the urgency of achieving food-independence. Development is faster in parrots from dry regions than in those from rain forests. This is because the period in which food is most abundant is shorter in an arid environment like the Australian hinterland than on an Indonesian island where there is considerably more tree cover.[25] Differences in parental post-fledging feeding have been noted among cockatiels, lovebirds, budgerigars and *Eclectus* sp. (Kavanau 1987).

Reasons for these differences are still speculative. Therefore, we must astutely analyze the circumstances associated with crying in the baby bird in order to respond appropriately.

VOCALIZATION

During a specific developmental period, young psittacine birds develop a loud, repetitive, plaintive call, which would, in the wild, signal parent birds to locate those fledglings. Key developmental events collide at this point: young fledglings learn to fly, navigate, land, come and follow and practice independent eating skills. Therefore, conscientious caregivers are challenged to carefully observe crying fledglings to determine what the cries signal. Does this young psittacine need comfort, exercise or food?

If comfort is needed, the bird should be held only until it settles down. If the bird needs exercise, playtime can be initiated wherein the fledgling is encouraged to try new physical skills such as flapping or climbing. If the bird is hungry, it should be fed a moderate amount of food and then shown how to forage to find accessible foods.

While some parent birds might actually let their babies cry in order to have them practice making a verifiable retrieval signal, this cry has a function separate from the cry of a hungry youngster. In no case should young fledglings be ignored or allowed to go hungry. Teaching a bird to whisper and to hum can be immediately rewarding and contribute to acceptable vocalization for years to come.

Selling Unweaned Parrots and Force-weaning

Avian veterinarians see a multitude of potentially serious medical problems when unweaned parrots are sold to inexperienced caregivers. Some cases of aspiration pneumonia and crop burns are treatable but some are fatal, and most are preventable through ethical breeding and sales practices. Many feel strongly that the practice of selling unweaned birds should be made illegal, as it is with puppies and kittens. However, should that happen, the fear is that baby parrots will be "force-weaned" in an effort to expedite the weaning process.

Force-weaning entails withdrawing hand-feeding when parrots are still begging for it, based on the belief that hunger will *force* the babies to start eating on their own. A multitude of behavioral problems may be associated with forced or stressful weaning.[16]

Baby parrots that are force-weaned often later become high strung, hyper-responsive to stimuli, prone to stress and rigid in their eating habits. When eating skills are not firmly in place and readily practiced, underlying undesirable behaviors manifest. Force-weaned African greys, for instance, seem much more prone to developing phobic behaviors later in life than do abundantly weaned African greys.[28] Cockatoos that are force-weaned often become chronic whiners, which may contribute to cockatoo prolapse syndrome. The large macaws, probably not truly "food-independent" in the wild until they are at least 6 to 9 months old, often are the victims of force-weaning. When macaws are force-weaned, they generally get into patterns of obsessive food begging, often with repetitive wing flicking and a typical macaw begging sound well into adulthood.[4] Birds that are weaned prematurely will exhibit chronic begging behaviors. It is common for them to flap one wing and bob their heads for food while crouched down.[1] In contrast, properly weaned birds will run to a food bowl, investigate its contents, and select a morsel and consume it, albeit wastefully. The aberrant behaviors of force-weaned birds are assumed to result from deprivation during a critical period of their development.

THE JUVENILE OR PREADOLESCENT

The hallmark of this period of psittacine development is often the bird's refusal to cooperate. Increased independence and increased athleticism both necessitate training and learning that is critical to the future development of the parrot and cohabitation with humans. Most of the medium and large species of parrots listed in the "Pets for Sale" section in local newspapers will be between 8 and 24 months of age (Fig 3.2.8). When properly reinforced for desirable behaviors, this need not be an overly stressful time for bird or owner (see Chapter 3, Behavior, Section I, The Natural Science of Behavior).

Juvenile Molt

Juvenile parrots often experience a heavy and uncomfortable molt that may make them irritable when touched. Molting parrots can be testy at any age, so one must assume that they are uncomfortable. Owners should be cautious when petting their birds, as it is easy for human fingers to accidentally inflict pain. Gentle stroking with a feather or toothbrush is suggested at these times, as this may decrease the bird's discomfort.[6] Frequent bathing also can help during this period; either self-bathing,

Courtesy of Santa Barbara Bird Farm

Fig 3.2.8 | A juvenile Moluccan cockatoo in full display. Juveniles can be a challenge, but less so if clear, consistent controls and limits are established for their behavior.

enclosure in a bathroom with the shower running to create high humidity, or exposure to actual, warm rainfall. Increased humidity will soften the keratin sheaths of pinfeathers and enable them to open more easily.

Bathing Skills

Bathing skills are critical for good feather and skin health, and a thoroughly soaked parrot is inspired to healthy feather grooming. Many parrots are rain forest species that evolved in environments where annual rainfall is measured in feet, not inches. Even those from arid regions often are found within flying distance of water pools or rain-drenched microhabitats where bathing opportunities abound. With artificial heat and air conditioning, human environments are seriously dry, so periodic soakings are needed to counteract these conditions. Bathing skills are important for young parrots to learn, and caretakers must be patient and creative in discovering an individual bird's preferred bathing technique. Some parrots prefer pool bathing in a shallow dish, and some leaf bathe in wet kale, romaine and chard. Others prefer rain bathing via a hand-held sprayer, the human's shower stall, a garden hose or natural rain on warm summer days. It is important for caretakers not to frighten a young parrot with the introduction to bathing. This may create fears that can be difficult to overcome.[11,12] Additionally, parrots who bathe or shower with vigor also are adept at exercise because the two vital functions reinforce each other. *(Ed. Note: Some believe that blow-drying should be vehemently discouraged, as this can negate the positive effects of bathing by drying out feathers and skin. Others disagree, believing that blow-drying is acceptable if it is tolerated by the bird and carefully regulated to prevent burns or overheating.)*

Exercise

Exercise is important to parrots of all ages, and it is

Courtesy of Santa Barbara Bird Farm

Fig 3.2.9 | Under close supervision, wing-trimmed fledgling macaws play in juniper bush while showering. Exuberant exercise is critical to physical and psychological health for psittacines as well as functionally decreasing problems in the captive environment. Tired parrots tend to be quiet parrots.

Layne Dicker, courtesy of Santa Barbara Bird Farm

Fig 3.2.10 | Prey species like psittacines need the choice to be visible or not, so providing hiding places can greatly decrease stress for any age of psittacine. With his softened face feathers, this fledgling yellow-naped Amazon displays the comfort level allowed by providing such choices.

especially important for juveniles. Parrots evolved to fly many miles each day in the wild, and this inherent need for exercise is critical for success in the captive environment. Flying and/or flapping exercises are a daily necessity, and caretakers should encourage these activities in their parrots. Exercise also is enhanced by the use of movable perches and the provisions of branches, rather than the thick, stationary perches (Fig 3.2.9).

Juveniles should be encouraged to chew, shred and otherwise pulverize a variety of destructible toys. Natural branches from non-toxic, non-sprayed trees, complete with bark and leaves, are ideal for parrots, and caretakers should be encouraged to find a constant source of such things as bamboo and willow for their birds.

The Human Environment and the Companion Parrot

Cage Placement

The ideal area in which to place a bird's cage is dependent on the personality of the specific bird. Most parrots enjoy being in the center of human activity, but care should be taken to allow for the instinctive insecurities of a prey animal. Placing a cage against a solid wall provides security, but many parrots enjoy a window view. Cages may be placed partially against a window and partially against a solid wall to provide the advantages of both security and stimulation.

Extroverted parrots that are caged away from human activities often scream excessively. Anxious, skittish parrots may start showing feather-destructive behaviors if caged in the middle of a high-traffic area, especially if the bird is startled by people appearing without warning.

The height of the cage also is important. Many parrots appear comfortable when allowed to perch at human chest or shoulder level. If caged too low, an insecure parrot can become seriously frightened. If caged too high, headstrong individuals may be more difficult to handle. Hiding places also are important, so parrots are allowed the choice of whether or not to be visible (Fig 3.2.10). Hiding places can include branches wired to the outside of the cage to produce a "thicket-like barrier" (M.S. Athan, personal communications, 2000), a fabric cover over one corner or wooden boxes attached to the side of the cage.

Environmental Enrichment

Because of the psittacine's need to forage and shred, suitable objects are necessary components of the enriched environment. Destructible objects, such as safe branches with leaves and bark intact, paper cups, tongue depressors and cotton-tipped applicators, also can keep parrots quietly and inexpensively absorbed.[7] One author's own blue and gold macaw hen (*Ara ararauna*) methodically works its way through an old phonebook once or twice a year, spending several weeks of intensive work to render the entire publication into thumbnail-sized pieces. This activity appears to diffuse aggression.[23]

Four categories of parrot toys have been described: chew toys, climbing toys, foot toys and puzzle toys.[9] A small number of stimulating toys, rotated on a weekly basis, seems to hold a parrot's interest. One toy from each category might satisfy most parrots' need to play, investigate and destroy, and also leave sufficient room

for the bird to move around its cage. In a very large cage or aviary, toys might be placed randomly about in order to encourage the parrots to use their entire territory.

Foraging

A recent study demonstrates the value of environmental enrichment in psittacine birds.[21] The use of foods and toys for foraging activities and environmental enrichment is a long-standing tradition in many companion and avicultural situations. Foraging is encouraged when foods are offered in new and challenging ways, such as stuffing an empty tissue box with greens or hiding a food within view but not within reach, eg, inside a puzzle toy. Parrot owners must devise methods to keep their birds occupied, especially during the long hours spent alone.

Toys also are useful as deflectors of aggressive energy, especially with species like Amazons. These birds may interact roughly with their toys, dissipating potentially aggressive energy.

Adequate Sleep

Sleep is another important consideration, especially with a young parrot. The actual sleep requirements and the presence of active (REM) vs. slow-wave sleep have not been determined in various psittacine species. In dogs the "active sleep-quiet sleep," or slow-wave (REM) sleep cycle, is only 20 minutes as compared to 90 minutes in humans. In the absence of controlled data on normal sleep rhythms, extrapolation and observation must be used to tentatively determine a pet bird's sleep requirements. As tropical and neotropical species, most companion parrot species evolved in an environment that provided 12 hours of darkness and daylight, year-round.

As previously mentioned, due to their social nature, parrots often are caged in high-traffic areas. This places them in locations with extended hours of noise and artificial light. When questioned about sleep, owners generally believe that covering the bird initiates sleep. More accurate information would be derived from asking what time the noise ceases and the lighting is extinguished in the evening.[29]

Rather than declare major rooms off limits past a certain hour to give a parrot more sleep, veterinary ethologist Andrew Leuscher originated the concept of the "sleep cage." A sleep cage is a small, sparsely equipped cage that is kept in a room that is unoccupied by humans at night, and it allows parrots to be put to bed at a reasonable hour. This allows them to get the hours of dark and uninterrupted sleep that they appear to need. Behavioral manifestations of sleep deprivation in parrots include hyperactivity, aggression, excessive screaming (especially after sunset) and feather-destructive behaviors such as plucking.

TRAINING THE YOUNG PSITTACINE

Young parrots need reinforcement for appropriate behavior. Parrots, like other animals, will perform best for positive reinforcement and will soon discard behaviors for which they receive no reinforcement. Without training, parrots do not understand how to be good companions and people do not understand how to be good caregivers.

Activities appropriate for the young bird to learn include physical lessons such as swinging, flapping and climbing. With these athletic adventures, young birds learn to burn their calories in appropriate ways and don't have massive amounts of energy left over at the end of each day for screaming, pacing or hyperactivity. In addition to physical activities, young birds should be encouraged to develop social skills that allow them to take food from human hands, to play with toys with various people, and to step up on either an offered hand or a hand-held perch. Vocal skills also benefit young psittacines who are encouraged to modulate their contact calls with more pleasing and less repetitive, less plaintive vocalizations, such as soft chortling and whistles.

The owner should train the young parrot to accept handling that facilitates life as a successful companion, such as entering and exiting the cage and stepping up and down upon request. Ideally, parrots should be trained to tolerate procedures such as grooming; this can greatly minimize stress but is difficult for most people to accomplish with their pet bird.

Parrots that do not receive rudimentary training are apt to lose their homes for two reasons: (1) caregivers tend to lose interest in "unmanageable" birds; (2) untrained parrots shape their own behaviors into less compatible actions such as screaming and biting.

Parrots should not be making decisions, such as whether or not they wish to go back in their cages or whether or not they wish to get off of the owner's shoulders. Parrots that learn to respond to reasonable requests are those that consistently benefit most from positive reinforcement, and caregivers should be aided in finding positive ways to teach their birds.

It is also important to understand that parrots are independent creatures. While parrots should step on the human hand on command when they exit their cages, the act of compliance with this command should be made a positive experience. For instance, rather than wait until the last possible second to return birds to their cages when owners are stressed and pre-occupied by being late for work, they will have greater success if they choose to re-cage their parrots earlier under more relaxed circumstances. For example, to remove a parrot

from its cage, the owner may approach the cage and ask the bird if it wants to come out. Observation of body language readily answers this question. If the answer is affirmative, the parrot generally moves forward, and/or picks up a foot. If so the owner opens the cage door and uses the "Up" command to which the bird has been trained. A negative response is equally obvious—the bird moves away and/or turns its back. If the response is negative, the interaction is ended. No command has been given, so no control has been lost.[8]

Birds vary in their reaction to food as a motivator for behavior. Most owners cannot and do not wish to withhold food from their birds in order to stimulate food-motivated behavior. However, some who manipulate delivery of a favorite food report surprisingly good results. Therefore, rewards should be selected for their efficacy in eliciting and reinforcing desired behaviors.[21]

Juvenile Behavior Problems

For detailed analysis of the various problem behaviors seen in companion parrots, refer to Section III of this chapter. Many of the problems seen in older birds have their foundation in mishandling of the youngster.

Inappropriate behaviors become problems when they are inadvertently reinforced, such as the baby that lunges at a stranger, only to be hugged and soothed (and therefore rewarded for aggression) by the owner. Some birds will hold onto their owner, or their cage door, as the owner attempts to return them to their cage. This should be recognized as early defiant behavior and addressed.

SUMMARY

Aviculture undoubtedly will continue to raise psittacine birds destined to become human companions as long as humans demand them. Accordingly, the need continues for examination of psittacine development. Appropriate diets, stimulation, security, regular and consistent sleep, appropriate lighting and sufficient exercise are important for the development of young parrots. Fledging should be part of the optimal psittacine development. Training techniques that enhance success in the human environment include basic handling skills (such as cage entrance and exit competence). When we properly educate ourselves, we can raise young psittacines that have an excellent basis for success in their captive environment.

References

1. Abramson J: The Large Macaws. Abramson, Speer and Thomsen, Raintree Publications, Fort Bragg, CA, 1995:205.
2. American Psychiatric Association: The Diagnostic and Statistical Manual of Mental Disorders Washington, DC, APA, 1994:116-118.
3. Beaver B: Feline Behavior: A Guide for Veterinarians. Philadelphia, PA, WB Saunders, 1992.
4. Blanchard S: Handfeeding trauma in young macaws. Pet Bird Report #17 9/94:46-47.
5. Budiansky, S: The Covenant of the Wild: Why Animals Chose Domestication, Yale University Press, New Haven, CT, 1992:77-78.
6. Cravens EB: The essence of touch. Parrots Magazine No 33 April/May 200:22-24.
7. D'Arezzo C, Shannon-Nunn L: Parrot Toys and Play Areas. Crow Fire Pub, Springfield, VA, 2000.
8. Davis C: Spring is in the air. Bird Talk 19(4)2001:80-83.
9. Foushee D: Play therapy. The Pet Bird Report 5-3(23)1995:30-32.
10. Harrison G: Perspective on parrot behavior. *In* Avian Medicine: Principles and Application. Ritchie B, Harrison G, Harrison L (eds). HBD Int'l Inc, Brentwood, TN, 1999:97.
11. Hefton D: Showering your parrot. Pet Bird Report 25:14-17, 1996:14-17.
12. Hefton D. The fine art of bathing the psittacine. Pet Bird Report 31:18-23, 1997.
13. Kaplan H, Sadock B, Grebb J: Synopsis of Psychiatry ed 7. Williams & Wilkins, Philadelphia, 1994, chapter 45.3:1109.
14. Linden, PG: Abundance weaning. Pet Bird Report 13(3) 5: 18-22, 1993.
15. Linden PG: Teaching Psittacine Birds to Learn. Sem Avian Exotic Pet Med: Behavior. Alan Fudge (ed); Christine Davis (guest ed), Oct 1999 8(4):154-164.

15a. Harcourt-Brown NH: Development of the skeleton and feathers of dughy parrots (*Pionus fusous*) in relation to their behavior. Vet. Rec 154, 42-48. 2004.

16. Linden PG: The developmental impact of weaning, The Pet Bird Report, Vol 4 #6, Issue 20, 1995:4-11.
17. Linden P: The lasting behavioral effects of fledging. The Pet Bird Report #29, 9/96:40-45.
18. Martin S: Lecture: Things That Make You Go 'Hmm!' Annu Conf Int Aviculturists Soc, Orlando, FL, March 1998.
19. May D: Greys of the Congo basin. Bird Talk Magazine, January 2002 20(1):37-47.
20. McElroy K: Parent-rearing Moluccan cockatoos and breeding cockatoos outdoors in a northern climate. Proc 9th Ann Conf Midwest Avian Research Expo, 1998:51-56.
21. Meehan C, Millan JR, Mench J: Foraging opportunity and increased physical complexity both prevent and reduce psychogenic feather picking by young Amazon parrots. Applied Anim Behav Sci, 80(1):71-85, 2003.
22. Merriam-Webster's Collegiate Dictionary 10th Ed, 2000,1334.
23. Murphy J: Shadow boxing or play fighting in Amazon parrots. Amazona Quarterly (2):14-16, 1997.
24. Schubot R, Clubb K, Clubb S: Psittacine Aviculture. Avicultural Breeding and Research Center, Loxahatchee, FL, 1992.
25. Silva T: Psittaculture: The Breeding, Rearing and Management of Parrots. Ontario, Canada, Silvio Mattacchione & Co, 1991:63.
26. Styles D: The Importance of socialization and flocking practices in shaping psittacine behavior in the home and aviary. The Gabriel Foundation, Parrots in the New Millennium 2002, February 8, 2002, San Diego, CA. Verbal presentation.
27. Wilson L: The NEW and IMPROVED Baby Parrot. The Watchbird, J Amer Fed Aviculture, Vol XXVI No 2, Mar/April 1999:47-49.
28. Wilson L: Phobic Parrots: An Increasing Phenomena? Proc Assoc Avian Vet 1998:125-131.
29. Wilson L: Sleep: How Much Is Enough For A Parrot? The Pet Bird Report, April 1999 43:60-63.

Recommended Reading

1. Blanchard S (ed): The Companion Parrot Quarterly (formerly The Pet Bird Report). www.companionparrot.com, Alameda, CA
2. Brown SD, Dooling RJ: Perception of conspecific faces by budgerigars (*Melopsitticus undulatus*): II. Synthetic models. Comp Psychol 1993 Mar 107(1):48-60.
3. D'Arezzo C, Shannon-Nunn L: Parrot Toys and Play Areas. Crow Fire Pub, Springfield, VA, 2000.
4. Davis C: Basic considerations for avian behavior modification. Sem Avian Exotic Pet Med (8)4:183-195, 1999.
5. Davis CS: Parrot psychology and behavior problems. Vet Clin No Amer Small Anim Pract (6):1281-8, 1991.
6. Diamond J, Bond AB: Kea, Bird of Paradox. The Evolution and Behavior of a New Zealand Parrot Univ of CA Press, USA, 1999.
7. Foster S, Hallander J: Cockatoos and African greys: Phobic behavior. The Pet Bird Report 8(4)42 :18-23.
8. Harris LJ: Footedness in parrots: Three centuries of research, theory, and mere surmise. Can J Psychol 1989 Sept 43(3):369-96.
9. Kavanau JL: Behavior and evolution: Lovebirds, cockatiels and budgerigars. Science Software Systems, Inc, California, USA, 1987.
10. Lightfoot T: Avian Behavior. Proc Assoc Avian Vet, 2002.
11. Linden PG: Abundantly Avian, The Compiled Works of Phoebe Linden Green. Pet Bird Report, Santa Barbara, CA, 1999, www.santabarbarabirdfarm.com.
12. Linden PG: Behavioral Development of the Companion Psittacine Bird. Proc Annu Conf AAV, 1998:139-143.
13. Linden PG: Teaching psittacine birds to learn. Sem Avian Exotic Pet Med (8)4:154-164, 1999.
14. Pearn SM, Bennett AT, Cuthill IC: Ultraviolet Vision, Fluorescence and Mate Choice in a Parrot, the Budgerigar *Melopsitticus undulates*, Proc R Soc Lond B Biol Sci 2001 Nov 7;268(1482):2273-2279.
15. Pepperberg IM: The Alex Studies: Cognitive and Communicative Abilities of Grey Parrots, Harvard Univ Press, Massachusetts, USA, 2000.
16. Pepperberg IM, Wilcox SE: Evidence for a Form of Mutual Exclusivity During Label Acquisition by Grey Parrots (*Psitticus erithacus*). J Comp Psychol, 2000 Sept 114(3):219-31.
17. Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1994.
18. Shields KM, Yamamoto JT, Millam JR: Reproductive Behavior and LH Levels of Cockatiels (*Nymphicus hollandicus*), Associated with Photostimulation, Nest-Box Presentation, and Degree of Mate Access, Hormones and Behavior 23:68-82, 1989.
19. Sibley DA: The Sibley Guide to Bird Life and Behavior, Chantilleer Press, NY, 2001.
20. Styles, D: Captive Psittacine Behavioral Reproductive Husbandry and Management: Socialization, Aggression Control, and Pairing Techniques. Proc AAV, Selected Topics in Non-Infectious Disease (Specialty Advanced Program), 2001:3-19.
21. Wanker R et al: Discrimination of Different Social Companions in Spectacled Parrotlets (*Forpus conspicillatus*): Evidence for Individual Voice Recognition. Behav Ecol Sociobiol 1998 43(3):197-202.
22. Welle KR: Avian Obedience Training. Proc Annu Conf AAV 1997:297-303.
23. Welle KR: Psittacine Behavior. Proc Annu Conf AAV 1998:371-377.
24. Wilson L: Handbook of Avian Articles. www.upatsix.com/liz.
25. Wilson L: Phobic Psittacines Birds-An Increasing Phenomenon? Proc Annu Conf AAV 1998:125-131.
26. Wilson L: Restraint in the Animal Hospital. Vet Clin NA-Exotic Sept 2001, 4(3):633-640.
27. Wilson L: Screaming and Biting in the Psittacine Pet Bird. Vet Clin NA-Exotic Sept 2001, 4(3):641-650.
28. Wright TF, Wilkinson GS: Population Genetic Structure and Vocal Dialects in an Amazon Parrot. Proc R Soc Lond B Biol Sci 2001 Mar 22, 268(1467):609-616.

Concepts in Behavior: Section III

Pubescent and Adult Psittacine Behavior

LIZ WILSON, CVT; TERESA L. LIGHTFOOT, BS, DVM, Dipl ABVP-Avian

The prevalence of captive-raised psittacines as pets and the ensuing problem behaviors have become critical issues for veterinarians as well as aviculturists and parrot owners. Ethological considerations should occupy a large area of future study, concern and advancement.

The terms adolescent and puberty in this chapter are used to identify the period of development during which hormonal changes play an active role in both the initiation of reproductive capacity and associated variations in temperament. Recognition of the role of sexual hormones in psittacine behavior is critical to the interpretation and modification of behavior.

Puberty

As with other psittacine developmental stages, the onset of puberty varies with the species and the individual (see Table 3.2.1 in Section II of this chapter). If erratic behavior is inadvertently rewarded it may continue after the sexual hormonal fluctuations of puberty have subsided.[9]

Owners are often unprepared for the prolonged developmental changes that their pet psittacine will undergo. Clearly established behavioral boundaries will decrease but not eliminate hormone-induced behavior. To aid in establishing rules and guidelines, the types of behavior for which the animal is reinforced must be examined. Pubescent parrots can learn to behave in ways that mitigate obnoxious behaviors, hormonal or not, and should be appropriately reinforced for non-reproductive behaviors if they are meant to be non-reproductive companions.

Increasing sexual hormone levels may lead to the first instances of territoriality and perceived aggression (Fig 3.3.1).[1] In the adolescent parrot, fluctuating hormones add to the inherently high energy level to produce a bird that needs directed, acceptable methods for dissipating this energy. Daily flapping exercise, play with toys and showers consume natural energy and provide learning opportunities with inherent reinforcement. Once through puberty, the adult psittacine may display a more predictable pattern of behavior (Fig 3.3.2).

The Adult Psittacine Companion

In the wild, constant challenges test the parrot's athleticism, ability to avoid predation and to find food. To succeed in such a demanding environment, parrots must be flexible, adaptable and continue to learn. It has been estimated that the average parrot's day is divided thusly: 50% of waking daylight hours spent locating and consuming food; 25% spent interacting with one's mate and/or other flock members; and 25% spent in preening (J. Harris, personal communication, 1996). In contrast, there are few challenging activities in the human habitat. Alone in a cage all day, the monotony is pronounced. Enrichments, especially ones that encourage foraging behavior, will provide appropriate learning and athletic experiences for captive psittacines.

Management issues can significantly contribute to problem behaviors. Inadequate diet or sleep and boredom can all be problematic. Cage size, location and height are also important. Refer to Chapter 2, The Companion Bird, for discussions of these subjects.

TRAINING

Psittacines should be trained to step on and off both the hand and a hand-held perch and to enter and exit their cages on command. They also should be trained to step from one hand to the other, an exercise called "laddering." When training a parrot, it is preferable to work out

Greg J. Harrison

Fig 3.3.1 | Sexually hormonal female yellow-naped Amazon parrot that is agitated. Note the erect neck feathers and forward-leaning stance as she defends her position on a cage door.

Greg J. Harrison

Fig 3.3.2 | Umbrella cockatoo exhibiting "alert" display. Species and individual variation dictate how a parrot in this "state" should be approached.

of sight of a parrot's perceived territory. Lessons should be short and upbeat, with rewards that are meaningful to the bird. Positive interactions and praise work well for parrots that already like their trainers. Correct responses should be instantly rewarded. The reward will vary with the personality of the individual parrot. It may be praise, desired physical contact such as head scratching, or a food treat (see Section I of this Chapter).

The species most prone to feather destruction and self-mutilation in domestic life often inhabit large flocks in the wild, and these same animals may therefore have evolved extensive interactions within a social order. When social order is ambiguous or absent in the human habitat, high levels of stress may result.[27]

Color Preferences and Applications

Many birds have preferred colors and other colors that are associated with fear or avoidance. To evaluate an individual bird's color preferences, place the parrot in a confined area with six or seven identical children's colored wooden or plastic blocks or balls. Note the colors the parrot avoids and those with which it plays. Remove the objects in the colors it ignores or avoids. Repeat this test several times to verify the preferences.

The positive colors can be supplied in the form of other items in the parrot's environment. During training, utilize these favorite colors in the selection of clothing, perches and food cups (Fig 3.3.3). This color selection can aid in the acclimation and acceptance of toweling in parrots (see Chapter 6, Maximizing Information from the Physical Examination). Teaching of toweling to young parrots can include playing "hide and seek" with towels of preferred colors, while gradually increasing the parrot's acceptance of restraint.

EXERCISE AND PLAY

The importance of exercise and play increases with the adolescent parrot and continues to be an important skill throughout psittacine life. Behaviorists have equated the amount of play in which the young engage with a species' general adaptability. Studies of keas (*Nestor notabilis*) are particularly compelling in this regard.[23] Play behavior is consistently demonstrated in properly raised captive psittacines. According to one biologist, "The question of 'play' in animals is an intriguing one. We use the word to connote activity that has no obvious use in daily life — almost a frivolous pastime. In fact, play has an important function for the animals that engage in it, humans included, and particularly for their young. It is a way of developing dexterity and motor coordination, and of discerning the boundaries of social behavior, skills that are critical to success as an adult. Play, as we know from watching our children, is a form of learning."[15]

Problem Behaviors in Captivity

An understanding of normal psittacine behavior is necessary when attempting to address problem behaviors in captivity (Fig 3.3.4). For example, it is perfectly normal for a parrot to use its beak when climbing, even when climbing onto a human hand. If that hand jerks in

Fig 3.3.3 | This lovebird is well-adapted to toweling by its owner and prefers the color blue.

Greg J. Harrison

Fig 3.3.4 | This is a common species threat posture displayed by a nervous green-winged macaw willing to defend against entry into its cage.

anticipation of a "bite," and the parrot doesn't reach its destination, the next time it might try a different (harder, perhaps) grip before it steps. It has been observed that inexpert handling by humans fuels fear in young parrots. Further, psittacines are normally loud, destructive and messy animals. While these behaviors may distress the people who live with them, they are not aberrant.

When one examines a "problem" behavior, the first question to ask is; "What function does the behavior serve?" Behavior has a function, which means that every learning-based animal performs certain behaviors based on what the animal gets as a result of that behavior (see Section I of this chapter).

INAPPROPRIATE BONDS

Some birds are reinforced when they show aggression toward less-favored people. Their actions may be accompanied by screaming, laughing or crying, culminating in a rescue by the favored person. All this vocalization and activity is positive reinforcement.

Owners can work with their parrots to prevent over-bonding to one person. A useful technique is Blanchard's handling exercise called the warm potato game.[14] A neutral room is ideal for this purpose. The parrot is passed from person to person with each member interacting positively (ie, praise, petting, treats) with the bird. This handling exercise should be continued for the duration of the parrot's life. To avoid alarming the timid parrot, the socialization process can begin by stepping onto the shoe of a less-favored person (sitting with one leg crossed), then be stepped back to the familiar hand and rewarded.[30]

People may inadvertently allow their parrots to form a mate bond with them (Fig 3.3.5). Owners often stroke their companion parrot's back and tail.[18] Breeding

Greg J. Harrison

Fig 3.3.5 | This yellow-naped Amazon has been sexually regurgitating its food to the owner, and it has some of this food on its beak.

parrots perform these behaviors during courtship and we therefore assume that companion parrots interpret this type of petting as sexual. Panting and masturbatory behavior often follow such intimate touching.[6] Serious aggression may follow, when the bird attempts to drive all creatures except the perceived mate from its territory. When unable to reach the targets of its attack, this bird may displace its aggression, often in the form of a severe bite, to the perceived mate or even to themselves.

COCKATOO VENT PROLAPSE

This syndrome is extremely common in adult umbrella (*Cacatua alba*) and Moluccan (*Cacatua moluccensis*) cockatoos. Generally, these parrots are strongly bonded to a human. Several veterinarians[38] (Van Sant, personal communication, 2002) have made the observation that

Fig 3.3.6 | Prolapsed cloaca on an umbrella cockatoo.

Fig 3.3.7 | Closer look at the prolapsed cloaca in Fig 3.3.6.

Fig 3.3.8 | After prolaspe is reduced, loss of elasticity and dilation of the cloacal lips are evident.

delayed weaning appears related to this phenomenon (see below). Although the etiology is still speculative, several characteristics have been noted in most of these cases. They:

- Are hand-raised individuals of the noted cockatoo species.
- Experienced delayed weaning and/or continued begging for food.
- Are psychologically attached to at least one person.
- Have a tendency to hold stool in their vent for prolonged periods (ie, overnight) rather than defecating in their cage. This may be exaggerated by potty training these parrots.

Prolonged begging for food causes straining and dilation of the vent. Misplaced sexual attraction to their "human" mate also will cause vent straining and movement. Retention of stool in the vent for prolonged periods stretches and dilates the cloaca. The vent lips in these birds are often distended and flaccid (Figs 3.3.6-3.3.8).

Behavioral modification is difficult for owners to accomplish, since it involves altering the tight bond that they have with their parrot. Behaviors that increase this inappropriate bonding in affected cockatoos include stroking the parrot, especially on the back (ie, petting), feeding the parrot warm foods by hand or mouth, and cuddling the parrot close to the body.

If the parrot perceives the owner as either its parent or its mate, it will continue to strain and the prolapse will likely recur despite surgical correction (see Cloacopexy in Chapter 35, Surgical Resolution of Soft Tissue Disorders). Some veterinarians have found that a total change of environment and human companionship (ie, finding the parrot a new home, either temporarily or permanently) is necessary to correct this problem.

PROLONGED HORMONAL STIMULATION

Captive-bred parrots display sexual behaviors at earlier ages than their tree-ranging counterparts. Eclectus hens (*Eclectus roratus*), for example, may lay eggs in captivity at 3 years of age. In the wild, eclectus hens generally don't begin to lay until about 6 years of age.[18] Sexually mature companion psittacines are also developing what some avian veterinarians call "hormone toxicity." Instead of going into nesting behavior once a year, circumstances in the human habitat can cause parrots to remain hormonally stimulated indefinitely. In the wild, seasonal triggers, like changes in photoperiod, suitable nesting sites and ample food, initiate nesting behavior. Similar triggers initiate nesting behavior in captivity (Table 3.3.1), but the artificial environment provides not only safety from predation but also confusing signals. Artificial light mimics the long days of summer, and seasonal variations are lacking. The same is true of temperature, as indoor parrots are protected from cyclic change. Food is abundant and some parrots are fed daily rations that provide many times their caloric needs.[18]

In parrots that become aggressive when in breeding mode, pupillary constriction, filoerection of the nape or

Table 3.3.1 | Foods that Encourage Breeding or Foods to Avoid to Reduce Breeding Behavior

Item	Description
High fat items	• Seeds such as a sunflower, safflower, hemp, niger, thistle, spray millet • Nuts • Meats • Oils
Sweet items	• Immature items such as corn, beans and peas • Apples • Grapes • Citrus • Bananas
Refined carbohydrates	• Pasta • Breads and other baked goods

crest feathers, or flexion of the joints of the wing will convey a warning of the parrot's agitation and potential reaction to further approach (Figs 3.3.1, 3.3.2). If these warning signs are not recognized, both physical and emotional damage may occur.[7] Some companion psittacine species become reproductively active once a year and during the remaining months are not so frenetic.[22]

In recent years, sexual hormone manipulation has been attempted with compounds such as chorionic gonadotropin or a GnRH agonist.[b] These medications may temporarily ameliorate the problem behaviors of hormonal aggression, as well as physical problems like chronic egg laying (see Chapter 19, Endocrine Considerations and Chapter 9, Therapeutic Agents). However, if the parrot's environment contains triggers for nesting behavior, sexual hormone levels may remain high despite the administration of these medications. Environmental triggers must be eliminated before lasting relief can be found.[28]

One aviculturist cites the following example of "energy management." He suggests redirecting psittacine energy into survival behaviors instead of reproductive behaviors. In the case of one excessively hormonal female umbrella cockatoo, he randomly modified the heat and light in the parrot room, the amounts and types of food offered, and the bird's perception of safety (by not covering the cage every night). Paper was provided during the day to shred. Within a week, the hen had returned to normal behavior. (G. Wallan, personal communication, 2002).

CHEWING

Chewing is not something that parrots "grow out of," as do puppies. Owners who complain that their psittacine is chewing the wall behind the cage should move the cage, supervise their parrots when out of their cages, and teach their parrots to chew on well-designed toys or other appropriate items. Dried pine cones, natural fresh branches from unsprayed trees such as apple, citrus, melaleuca, Australian pine, ash, beech, and aspen are also ideal. Indeed, bark chewing and eating behaviors have been observed in African greys in Africa.[31]

EXCESSIVE VOCALIZATION

People frequently ask how to "teach their parrots how to be quiet." Parrots are loud animals and they cannot be taught to be otherwise. When lay bird magazines tout a particular species as being "quiet," this does not mean that parrots of that species make no noise; it means they make less noise than the species that are known to be very loud.

Normal noise levels vary from species to species, but generally speaking, parrots tend to vocalize loudly several times a day for 5 to 15 minutes. The large macaws, Amazons and cockatoos normally will produce 15- to 20-minute bursts of screams several times per day, especially morning and evening.

Case Study: Excessive Screaming

A 9-year-old male umbrella cockatoo (*Cacatua alba*) was screaming for hours on end and neighbors were beginning to complain.

Through the process of taking a detailed history the following information was discovered. The bird's cage backed up to a half-wall that allowed visibility from all directions and the bird had no hiding place in its cage. The cockatoo was getting only 6 hours of dark, uninterrupted sleep and was eating a high-sugar, low-nutrition diet. The owners also were unknowingly allowing sexual behaviors such as masturbation and had little control over the cockatoo.

The approach to this problem was multifaceted. The owners purchased a sleep cage and began putting the bird to bed at a much earlier hour, enabling 10 hours of sleep. A large towel was positioned over one end of the parrot's cage, allowing the bird to withdraw from sight whenever he wished, and the owners discovered that the bird chose to spend a great deal of time behind the barrier. The diet was adjusted and the owners began having daily lessons to establish better control. Sexual behaviors were curtailed with no punishment or drama. Within a couple of weeks, the screaming had abated tremendously, and 2 years later, the owners (and their neighbors) continued to be very pleased with the decrease in the bird's vocalizations to expected levels.

Incessant screaming is not normal behavior. Once again, the first step to changing a behavior is to analyze its function: what does the parrot achieve by screaming? Does the screaming parrot yell to get the owner's attention, to obtain food or to alleviate boredom? Many parrots that scream excessively do so to liven up their monotonous existence.[43] See Section I of this Chapter for more information on inadvertent reinforcement of excessive screaming.

When taking a behavioral history, ask the owners *what they do* when their bird screams. They may let the parrot out of its cage, pick the parrot up, give it a treat to quiet it or yell at the bird. Unfortunately, all these responses are rewards and reinforce the behavior.

Modifying Screaming Behavior

There are inherent problems with most cases of excessive screaming. First is duration, because the more time

parrots have been reinforced for screaming, the more ingrained is the behavior. It is also essential that all people involved understand that changing screaming behavior requires participation by all the humans in the area. If one person in the environment continues to reward the screaming, the behavior will not change.

The resolution of any behavior problem requires a step-by-step approach (see Section I of this chapter for more information). Circumstances that precede the behavior should be documented. These may include:

- Time of day
- Day of the week
- Activity/mood/noise level in the household
- The parrot's body language
- Relation to feeding time

Reactions that accompany or follow the screaming should be recorded (as these may be the inadvertent reinforcers of the behavior).

Owners are instructed to collect but not interpret data for 10 to 14 days. If a pattern is detected, steps can be taken to change what leads up to the behavior and the response to the behavior, therefore changing the behavior itself[43] (see Section I of this chapter for an in-depth discussion of this behavioral model).

To successfully decrease excessive screaming, owners must be consistent and patient. If a parrot screams while caretakers are in the room, they can turn their backs and momentarily withdraw their attention. This maneuver is classified as a "time out." The second the racket quiets, they can turn back to the parrot and gently, so as not to reignite a screaming fit, reward with smiles, praise or acceptable food treats. If the screaming begins again, caretakers should turn their backs and leave the room, leaving the parrot alone. They should not return until the parrot has temporarily quieted.

If they are not in the same room when a screaming episode begins, they are to do nothing until the parrot quiets briefly and preferably makes some acceptable type of noise. Then they can reenter the room and reward the parrot for either silence or acceptable vocalizations. Extinguishing excessive screaming will not produce a completely quiet parrot.[25]

Parrots often scream excessively when company arrives. Owners can take the following preemptive steps: (1) Do not feed the parrot for 4 to 6 hours prior to company arriving; (2) Encourage the parrot to engage in flapping session, followed by a drenching shower. Once accomplished, move them to a sleep cage in a separate room (for more information on sleep cages, see Chapter 2, The Companion Bird); and (3) Give the bird a meal and some special treats hidden in a difficult-to-get-at toy.

There are exceptions to the rule of ignoring excessive screaming. When the human "flock" reunites, parrots tend to celebrate this occasion with raucous noise. Owners should not ignore the bird in this situation. Instead, they should greet their parrots and spend a couple of minutes interacting with them. Owners should then ignore any noise that happens after a parrot is suitably greeted. A second exception has been termed the "contact call."[24] In the wild, a parrot's flock represents the safety and protection of numbers. When other flock members are not visible, for example, when a flock is feeding in the heavy foliage of the rain forest canopy, they may use the contact call. Its function is to make certain they have not become separated from the flock. Companion parrots also do this, and they are simply making certain they are not alone. When contact calls are not answered, they often escalate to a scream. If a scream is required to receive a response, this inadvertently reinforces screaming.

African greys (*Psittacus erithacus erithacus*) have been described as learning human contact calls, such as the ringing of the phone and the beep of a microwave. It is postulated that they mimic these sounds when they are seeking contact with the members of their human flock.[26] Cage location also can influence levels of psittacine vocalization.

Without an area in which to hide, vigilance behavior may be displayed as excessive vocalization.

BITING

Unlike excessive noise, biting may not be grounded in instinct. According to observations made of parrots in the wild, the beak is used for eating, preening and social interaction, not as a weapon against other flock members. Wild parrots use complicated body language, feather position and voice to express themselves in situations of conflict with others in their flock. When a confrontation is not quickly resolved, they simply fly away rather than engage in actual combat.[36,37,39]

Most captive parrots are caged and have clipped wings, so instinctive responses such as flight are not an option. Hence, biting becomes a common behavior in captivity.

The Functions of Biting

As was previously mentioned, it is critically important to analyze the function of a behavior before appropriate recommendations can be made. A detailed history must be obtained. There are wide varieties of stimuli that motivate a parrot to bite, and these need to be identified and analyzed.

Fear-based Biting

The issue of fear is a critical consideration with a parrot. Psittacines tend to be frightened of novel objects or situations, as their survival in the wild would be enhanced by approaching new things with extreme caution. Owners may not realize this and become impatient with their parrot, increasing rather than dissipating its fear. One needs to look for techniques to gradually desensitize the parrot to fear stimuli.

Biting and Territoriality

Parrots, like most animals, have territorial impulses.[3] If a parrot starts to lunge when around any perimeter, it is labeled as "territorial." Caregivers can alter the configuration of that perimeter, preferably with enrichment, decreasing the territorial response. Preferred behaviors should simultaneously be reinforced. Commonly identified perimeters frequently include nest boxes, feeding stations, play gyms, dark places, cage interiors and cage doorways. The territory outside of these locations can be made more enriching than the protected areas. For example, the parrot that balks at leaving its cage can be shown, before the cage exit is requested, that the reward it will get contingent upon removal from the cage is greater than that of solitude.

As exemplified by the frequent springtime attacks of small songbirds on substantially larger predators, like raptors, dogs and people, parrots take their territoriality quite seriously. Owners who allow their parrots total freedom in the home unwittingly exacerbate this problem, as the parrots may then defend wider areas from non-favored people.[21] In extreme cases, parrots assume the entire house as their territory, attacking anyone who enters.

By training parrots to enter and exit the cage on command, caretakers maintain control over the cage space, preventing territorial aggression. This is easily accomplished by teaching parrots that they must step onto the offered hand or hand-held perch if they wish to exit the cage.[13] If parrots do wish to come out of the cage, owners should not just open the cage door and walk away, allowing the parrots to exit whenever they choose. Instead, parrots should be taught to politely step onto a perch or the hand prior to exiting the cage. Feeding such a parrot away from the cage makes the effort of getting the parrot out of its cage easier.

Parrots that have a very small living area (cage) may have increased territorial instincts. Setting up a number of other play areas can decrease territorial instinct back to a more normal level.

Case Study: Fear-Based Biting:

Normally sweet and unaggressive, Lily, a 9-month-old African grey (*Psittacus erithacus erithacus*) hen abruptly started biting when her owner's friends tried to handle her. She became especially aggressive with her person's new boyfriend, striking quickly and biting hard.

Lily was biting from fear-based aggression. Frightened by non-flock members, she needed to be better socialized. If not identified and handled properly, a shy parrot like Lily can blossom into a determined fear biter. Lily's person needed to reassure her that she was safe when interacting with others.

Introducing her to other people in neutral territory with patience and sensitivity, the owner taught Lily that new people are fun and interesting. Initially, she expected Lily only to step onto the outsider's hand politely, and then step right back onto her trusted caretaker's hand. Lily's good manners were then lavishly rewarded with smiles and praise. Each time Lily did this successfully, she discovered that positive things happened when she was compliant with new people. As a result, Lily learned to enjoy interactions with non-flock members.

Case Study: Biting as a Learned Defense

An extremely polite and unflappable 3-year-old male African grey had always been very sweet and easy to handle, so this author (LW) was quite startled when the owner recently complained that the grey had been biting her.

When questioned regarding the circumstances, the owner revealed that the bird was biting only when she was trying to pet him. Upon further questioning, the owner admitted that on each occasion, the bird had pushed her hand away several times before it would bite. This was a clear example of biting as a learned defense. The bird obviously had tried to communicate to the owner that it did not wish to be petted, but the owner did not understand. When the frustrated bird finally resorted to biting, the woman ceased her attempts to fondle it.

In this manner, the owner was very clearly sending the message that nothing short of violence would get her to stop forcing herself on the bird. It was explained to the owner that the parrot had politely refused her attentions and that request should be honored. The domesticated dog is the only animal that is always in the mood to be petted, and a parrot is not, after all, a dog with feathers.

Case Study: Territorial Aggression

Clients complained that their 14-year-old male yellow-naped Amazon (*Amazona ochrocephala auropalliata*) was lunging and biting at family members as they walked by his cage.

The location of cages and playgyms can strongly influence parrot behaviors. In this situation, questioning revealed that the Amazon's cage was in the middle of a high-traffic intersection, on the same wall with doors. The consultant explained that while these social creatures like being in the middle of the action, they can get overstimulated by constant traffic flowing by their cages, as well as being startled repeatedly by the abrupt appearance of humans. Owners need to remember that parrots are prey animals that often react aggressively when frightened. This aggressive behavior was eliminated when the owners relocated the cage out of the middle of the traffic flow over to a far wall where the Amazon could see people coming.

Parrots are often not comfortable with strangers invading their cage space. If interaction with a stranger is to be attempted, the parrot should be removed from the cage first and then introduced to the stranger. When the protected territory is a person to which the bird is strongly bonded, owners must be vigilant to prevent injury to other persons, the bird or themselves (see Mate-bonding).

HEIGHT

No true dominance has been documented in birds as relates to their relative perching height. Ornithologist Jim Murphy commented, "Height does have its advantages, and behaviors can and do change as a result of it. Height alone confers a temporary advantage if there is the determination on the part of the involved individual parrot to exercise that advantage."[34] Parrots that become incorrigible when above eye level are already out of control, and this becomes more obvious with the increased altitude.[46]

In some situations, a temporary easement of problems can result simply from raising the people. For example, a footstool placed next to a high play gym can enable shorter people to more easily reach and therefore have better control over a parrot.[20] Whether being at an increased height changes a parrot's perception of its status or decreases its perceived vulnerability is still under debate. Regardless of the psychology behind it, most parrot species that are incompletely trained are more compliant on the floor than when perched on top of a tall cage.

SHOULDERING

A potentially dangerous but extremely popular practice is allowing parrots onto people's shoulders. A popular tradition over centuries of parrot ownership, this practice probably did not become especially dangerous until the advent of captive-bred parrots.

It has long been understood by raptor specialists that there is a substantial difference between raptors that have been captured and tamed and those that are domestically raised and socialized to humans. Birds of prey that have been socialized to humans have no fundamental fear of people and they can become extremely dangerous when in nesting season, aggressively attacking people who encroach on their territory (M.J. Stretch, personal communications, 2000). Imprinted hawks may need to be tethered during nesting season to prevent them from attacking people. Due to their potential aggression toward humans, these raptors cannot be released into the wild. The similar reaction seems to occur in captive-bred parrots. Allowing parrots on the shoulder can be particularly hazardous.

Additionally, a psittacine on a human's shoulder easily can reach vulnerable parts of the owner's anatomy (eyes, ears, noses, lips). Facial injuries can be severe and also permanently damage the parrot-human bond. Parrots should not be allowed on a human's shoulder. This is one of the few issues on which all experienced persons involved in parrot behavior agree.[4,10,19,20,40]

BEHAVIORAL MODIFICATION FOR BITING

Undesired behaviors often can be avoided by interpretation of psittacine body language. Careful observation is the first step in this process. Once experienced in reading body language, people will find that it is easier to anticipate and avoid a bite.

A parrot most often resorts to biting when other potentials for communication have been exhausted. It then continues to bite because it has been reinforced for doing so. For example, a parrot often will respond to unwanted human attention by raising its foot to fend off the hand, or by pushing the hand away with their beak. Humans will get bitten if they ignore these warnings.

Owners reaching for a parrot while they are on the phone may trigger a biting reaction. Psittacines often behave as if they dislike telephones. From the bird's perspective, the ringing telephone elicits an immediate response. The owner holds the phone to their ear and interacts with it verbally. It is not surprising that parrots act so negatively to this apparatus.

An owner can often prevent a bite by making direct eye contact and putting a hand or finger up as a counterpoint of attention, while quietly but firmly saying something like, "Be good" or "Settle down." Seldom will a parrot break eye contact in order to bite. Other methods of prevention include giving the parrot something on which to place its beak while stepping it onto the hand, or approaching from behind instead of offering the hand from the front.

When a parrot attempts to bite while sitting on a hand or arm, there are two techniques that can be effective. Athan's "Wobble Correction" entails a tiny rotation of the arm or hand.[5] Davis' "Little Earthquake" involves performing a slight drop of the hand or arm.[10] If used consistently, parrots soon make the connection between biting and losing their balance.

Laddering can be can effective deterrent to biting. This is the exercise described previously in "Training" in this section. The technique is the same, but the handler's facial expression and tone of voice is quiet and firm and the handler is frowning. When the parrot follows the command, the owners tone turns positive, and the bird is then placed on a perch. Laddering works well with many cases of aggression when firmly implemented.

Some owners are not aware that parrots normally use their beaks as hands rather than weapons. Afraid of being bitten, these people often pull away when a bird reaches for them with their beaks. Repetition of this exchange will teach young parrots that if they wish to climb onto a hand they must grab it quickly. At some point the bird may grab the human hand with enough force to cause pain. If this is rewarded with vocalization by the person (such as "OW, BAD BIRD, NO BITE!"), the bird has received positive reinforcement. Instead of verbal reinforcement, a stern look and a quiet, firm, "No" *without* withdrawing the hand is most effective. Once reprimanded, the parrot can be given a toy with which to play, offering it a healthy outlet for exploration with its beak.

Neurotic Fears or Phobias

According to human psychology, a phobia is defined as "any unfounded or unreasonable dread or fear."[2] It is not unfounded or unreasonable for a prey animal such as a parrot to be afraid of a predator such as a human, and this reaction would be expected in wild psittacines. However, most pet psittacines are acclimated to the human captive environment. When there is no precipitating incident and the parrot is suddenly terrified of people, noises, shadows, or comparable non-threatening items, the definition of phobic would seem to apply.

There are few situations as frustrating as dealing with the phobic or neurotic parrot. The classic signalment is a high-strung young parrot that suddenly begins reacting to humans as if they are deadly predators. This is especially upsetting when the formerly beloved owner is an object of their terror. The parrot may flail around its cage, screaming and trying to escape when the owner approaches. A "phobic" parrot is not simply afraid of new toys or new people, it also overreacts to noise, movement, and even direct eye-contact from humans. These parrots often break multiple blood feathers, and cause extensive soft tissue damage to their keel and wing tips.[12]

Ordinarily, aggressive parrots are not phobic (J. Doss, personal communications, 1997-1998). There has been discussion concerning whether these are two different responses to the same stimulus. If so, insecure parrots that perceive themselves as threatened can become either phobic or aggressive, depending on individual personality type (P. Linden, L. Dicker, personal communications, 1997). Some species are particularly prone to phobic behaviors, including small cockatoos like the rose-breasted (*Eolophus roseicapillus*), citron-crested (*Cacatua sulphurea citrinocristata*) and triton (*C. s. triton*); small *Poicephalus* (Meyers [*P. meyeri*] and Senegal parrots [*P. senegalus*]); African greys (especially the Congo [*Psittacus erithacus erithacus*]); and eclectus parrots (*Eclectus roratus*). These same species also are predisposed to feather-destructive behaviors.

The etiologies of phobic behavior are unknown. A particular incident may precipitate phobic behavior, but the actual underlying causes may be species-specific tendencies and developmental problems (see Relative

Case Studies: The Non-Phobic Phobic

Care must be taken to accurately diagnose phobics, since they are handled so differently from the more common problem behaviors seen in companion parrots. One author (LW) worked with a "phobic" yellow-naped Amazon (*Amazona ochrocephala auropalliata*) that turned out to have an idiopathic medical problem that predisposed the parrot to falling from the hand because it could not grip properly with its feet. Multiple falls taught the parrot a direct correlation between handling and pain. The result was a dramatic fear response when people approached. Interestingly enough, the Amazon's screaming and flailing was eliminated by the use of the dopamine antagonist, haloperidol[c] (D. Kupersmith, personal communications, 1997-1998).

Table 3.3.2 | Sodium Contents in Foods

Food Serving	Sodium (mg)	Food Serving	Sodium (mg)
Fruits/Vegetables		**Peanuts**	
Peppers (sweet) (1 cup)	3	Dry-roasted (unsalted)	0
Banana (one)	1	Oil-roasted (unsalted)	0
Apples (one)	0	Roasted-in-shell (unsalted)	0
Pineapples (fresh) (1 cup)	2	**Sunflower seeds**	
Raisins (1 cup)	17	Unsalted	0
Pumpkin (1 cup)	2	**Popcorn** (movie theater style)	
Oranges (one)	0	Light (1 cup)	97
Cantaloupe (1/8)	6	**Crackers**	
Strawberry (1 cup)	2	Crackers w/cheese	101
Bananas (one)	1	Whole wheat	105
Papaya (1 cup)	4	Saltines® (4)	156
Grapes (European) (10)	3	Saltines® low salt (4)	75
Watermelon (1 cup)	3	**Chips - Bread**	
Green beans (1 cup)	4	Chips (1 oz)	216
Romaine (1 cup)	4	Whole grain bread (1 slice)	127
Olives (5)	192	**Butter - Cheese**	
Yams (fresh no skin) (1 cup)	2	Butter (1 Tbsp)	117
Yams (canned heavy syrup) (1 cup)	76	Cheddar (1 oz)	176
Brussel sprouts (frozen) (1 cup)	36	American (processed) (1 oz)	405
Cabbage (1 cup)	13	Mozzarella (whole milk) (1 oz)	150
Beet greens (1 cup)	347	**Meat**	
Corn (cob) (1 cup)	8	Beef fresh (3 oz)	56
Corn (canned) (1 cup)	571	Beef (dried) (1 oz)	984
Celery (1 cup)	104	Turkey (breast white) (3 oz)	54
Carrots (1 cup)	39	Chicken (breast white) (1/2)	64
Broccoli (fresh) (1 cup)	24	Bacon (3 slices)	303
Spinach (1 cup)	24		
Coconuts (1 cup)	24		
Potatoes (french fries) (10)	15		
Mixed vegetables (frozen)	64		
Mixed vegetables (canned)	243		
Beans (canned)	700-1000		

* Products with 3 mg of sodium or more per serving should be avoided in feather picking birds. Restrict foods to recommended yellow fruits and vegetables (G.J. Harrison, personal communication, 2003).

Case Study: Feather Destruction

A 3-year-old male African grey (*Psittacus erithacus erithacus*) was boarded with this author (LW), and on presentation the bird had removed all its contour feathers on its chest, leaving only down.

The impetus for this behavior became obvious when the bird yanked out feathers whenever its wishes were frustrated. When the grey apparently realized that no one would acquiesce to its desires, the motivation for the plucking was removed and the behavior ceased. The bird went home 3 weeks later with a dramatic increase in contour feathers on its chest, and the owners were delighted. However, they renewed their behavior of complying with the bird's wishes whenever it touched a feather, so the bird promptly reduced its feather coat to only down again.

The owners opted to do a consult at this point, and learned how to avoid reinforcing the bird and the behavior ceased once again. There were periodic episodes of plucking over the next few years (such as when the owners purchased a Great Dane puppy), but when the owners did not reward the feather destruction with attention, it ceased after a short period. At this time, the bird is fully feathered.

It is felt that success in a feather picker is that it does not escalate, may cease, or does not mutilate. It has been noted in many cases that during times of stress, which varies from case to case (a storm, a new pet, new people, a new home or changes in the home) the bird will revert at least temporarily to picking (G.J. Harrison, personal communication, 2003). Guaranteeing a cure is seldom fulfilled.

Attachment Disorder in Section II of this chapter). Potential exacerbating events may include physical or psychological trauma such as aggressive capture and restraint techniques.[42] Ethologists agree that aggressive handling or "punishment" is not the only reason that parrots become phobic (A. Luescher, J. Oliva-Purdy, L. Seibert, personal communications, 2003). Often there is no discernible history of abuse.

Rehabilitation of a phobic parrot can be a painfully slow and difficult process. Misinterpretation and mishandling of phobic behavior may increase the severity of the problem.[44] Various psychotropic drugs may be used in conjunction with behavioral modification to aid in the treatment of phobic parrots. Many of these drugs are also used for parrots with feather destructive behavior. Species specificities seem to exist but much of this information is still anecdotal. One author (TLL) has had relatively good results in with several African greys using a combination of diazepam and fluoxetine, or diazepam and amitriptyline. Conversely, haloperidol has worked in a number of *Cacatua* species, but the same dose caused excessive agitation in a male black-headed caique (*Pionites melanocephalus*), an eclectus (*Eclectus roratus*) female and one eleanora cockatoo (*Cacatua eleanori*) (see Chapter 9, Therapeutic Agents).

Feather Destruction and Self-mutilation

Feather destructive behavior is a condition seen only in, and therefore caused in some manner by, captivity.[17] Species predilections exist, with African greys and cockatoos being over represented. Psychological as well as

physical etiologies contribute to feather-destructive behavior.[27]

Feather-destructive behavior is a clinical sign, not a disease entity. Nutritional causes, including malnutrition and food, pesticide, dye or preservative sensitivities, and excess salt or sodium may be causative (Table 3.3.2). Environmental factors including low humidity, inappropriate light cycles, and noxious aerosols, are all possible potentiating factors. Primary dermatologic disease as well as metabolic disease and parasitism may be causative. The reader is referred to the appropriate chapters in this and other references for more information.

The observation that "the best kept parrots often develop feather-destructive behavior" is applicable, and frustrated owners find comfort in this adage. With all their food, attention and security needs met, some parrots will redirect their energies. Normal preening then becomes prolonged and exaggerated and may lead to feather mutilation. Psychotropic medications seldom are a complete answer but may be used as an aid during environmental and behavioral modification.

Additionally, owners often unconsciously reward parrots for feather destruction. Psittacines may recognize the potential for feather picking or destruction to obtain internal stimulation or external reinforcement. The first step in behavioral modification is to ignore this behavior.[41]

The Geriatric Parrot

Despite the USA having imported hundreds of thousands of parrots in the 1970s and 1980s, there is little information to be found on geriatric parrots. Additionally, the physical and emotional health of these older import parrots may not equate to that of captive-bred psittacines that will be reaching geriatric status in the coming decades.

A study of the large macaws at Parrot Jungle (Miami, FL) found their "functional breeding life-span" to be about 30 years, and their survival life-span was about 45 years. Physical signs in older macaws included increased scaliness of feet, thickening of the skin of the feet, thinning of the facial skin, appearance of warts, cataracts and other ocular color changes. It was noted the yellow iris thinned so the dark retina could show through, producing a dark ring within the iris. Macaws over the age of 35 showed postural changes due to arthritis and degenerative neurological disease. Causes of death in these older birds included tumors, renal failure and other degenerative diseases.[16]

Fig 3.3.9 | Cataract in an older yellow-naped Amazon. Cataracts may cause behavioral changes related to decreased vision.

Gradual development of cataracts allows compensation by the bird as long as its environment remains static. Sudden onset of cataracts may cause behavioral abnormalities that mimic aggression, lethargy, or even seizures (Fig 3.3.9). Thousands of imported parrots that should be growing old in captivity have died of malnutrition and husbandry related disease. Documentation of the physical and behavioral changes of our current psittacine pets as they age will improve our understanding of their medical and emotional needs. Hopefully, we will have an increasing captive geriatric parrot population from which to learn.

Conclusion

Psittacine birds have been sharing the human habitat for hundreds of years. However, these are still not domestic creatures. It will take additional research and application of existing knowledge to integrate parrots into the human environment. A growing number of owners, particularly of cockatoos, are "donating" these parrots to shelters and rescue organizations due to excessive screaming, plucking or biting behavior.[33] Other birds are condemned to life in a cage due to the owner's fear or inability to handle them. Early education of owners may prevent incompatible behaviors that lead to abandonment or neglect.

Products Mentioned in the Text

a. Chorionic gonadotropin, hCG, American Pharmaceuticals Inc., Los Angeles, CA, USA
b. GnRH agonist Depo-Lupron (Lupron Depot, leuprolide acetate), TAP Pharmaceuticals, Inc., Lake Forest, IL, USA
c. Haldol, Henry Schein, Melville, NY, USA
d. Prozac, Eli Lilly, Indianapolis, IN

References and Suggested Reading

1. Abramson J: Captive breeding. *In* Abramson J, Speer BL, Thomsen JB: The Large Macaws. Fort Bragg, CA, Raintree Publications, 1995, p 150.
2. American Psychiatric Association: The Diagnostic and Statistical Manual of Mental Disorders 4th ed. Washington, DC, APA, 1994, p 443.
3. Ardrey R: The Territorial Imperative. New York, Kodansha America Inc, 1966.
4. Athan MS: The importance of being "tall." Guide To A Well-Behaved Parrot. Hauppauge, NY, Barrons, 1993, pp 64-66.
5. Athan MS: Beyond biting. *In* Guide To Companion Parrot Behavior. New York, Barrons, 1999, p 157.
6. Athan MS: Guide to Companion Parrot Behavior. Hauppauge, NY, Barrons Pub, 1999, p 6.
7. Bauck L: Growing pains. Bird Times Magazine 8(1):10-11, 2000.
8. Bebe A: Seminar on dog and cat behavior, Harcum College, Bryn Mawr, PA, 1997.
9. Blanchard S: Games parrots play. Bird Talk Magazine 9:48-54, 1991.
10. Blanchard S: Problems with parrots on shoulders. Pet Bird Report 25(5):5, 1995.
11. Blanchard S: Sexual behavior in companion parrots: Myths and realities. Pet Bird Report 32:50-54, 1997.
12. Blanchard S. Phobic behavior in companion parrots. Proc Ann Conf Int Avicult Soc, Orlando, FL, March 1998.
13. Blanchard S: Cage etiquette. Companion Parrot Handbook. Oakland, CA, Abbey Press, 1999, p 69.
14. Blanchard S: Maintaining flock acceptance. Companion Parrot Handbook. Oakland, CA, Abbey Press, 1999, p 177.
15. Burger J: The Parrot Who Owns Me. New York, Random House Inc, 2001, pp 134-135.
16. Clubb S: Geriatrics in macaws. Ann Conf Amer Fed Avicult, Baltimore, MD, 1998.
17. Cooper JE, Harrison GJ: Dermatology. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles And Application. Lake Worth, FL, Wingers Publishing Inc, 1994, pp 607-633.
18. Cravens EB: Early sexuality in captive psittacines. Birdkeeping Naturally. The Perch: Newsletter of the Peninsula Caged Bird Society, Yorktown, VA, 12/2002.
19. Davis C: New techniques in pet avian behavior modification. Proc 1989, pp 183-189.
20. Davis C: Caged bird behavior, taming, and psychology with common behavioral problems and solutions. *In* Rosskopf W, Woerpel R (eds): Diseases of Cage and Aviary Birds 3rd ed. Philadelphia, Williams and Wilkins, 1996, pp 19-29.
21. Davis C: Today the sofa, tomorrow the world! Bird Talk Magazine 14(6):44-47, 1996.
22. Davis C: Spring is in the air. Bird Talk Magazine 19(4):80-83, 2001.
23. Diamond J, Bond A: Kea: Bird of Paradox. Los Angeles, University of California Press, 1999.
24. Forshaw J: Parrots of the World. Melbourne, Australia, Landsdowne Press, 1993, pp 423.
25. Friedman, S: Lecture 1, Introduction to behavior. Online course: Living and Learning with Parrots. www.ParrotTalk.com, Jan-Feb 2003.
26. Hallander J: Contact calls: The communication link with our parrots. Pet Bird Report 42:62-65, 1999.
27. Jenkins J: Feather picking and self-mutilation in psittacine birds. Vet. Clini No Am: Exotic Ani Practi 4(3):656, 2001.
28. Jenkins T: Lecture: Pet Bird Information Council Conf, Minneapolis, MN, May 2002.
29. Lightfoot T: Captive-raised psittacine birds and species differences in the animal hospital environment, Proc Assoc Avian Vet, 2002. pp 25-31.
30. Linden P: Bird Behavior Q&A. Original Flying Machine 2:18-20, 2000.
31. May D: Lecture: Where the wild greys are. Camelot, Atlanta, 2001.
32. Meehan C, Millan JR, Mench J: Foraging opportunity and increased physical complexity both prevent and reduce psychogenic feather picking by young amazon parrots. Applied Anim Behav Sci 80(1):71-85, 2003.
33. Murad JW: The sad reality: The need for avian humane facilities. Proc Assoc Avian Vet, 2001, pp 429-436.
34. Murphy J: The above eye-height position. Amazona Quarterly 13(1):9, 1997.
35. Parrot Education and Adoption Center "Ask The Experts" roundtable discussion. San Diego CA, November, 2002.
36. Shade R: Bronze-winged pionus research project. Original Flying Machine 2(42):42-45, 2000.
37. Shindlinger M: Lecture: Tracking the wild Amazons. D Grindol's Pet Bird Seminar, San Jose, CA, March 1999.
38. Speer B: The clinical consequences of routine grooming procedures. Proc Assoc Avian Vet, 2001, pp 109-115.
39. Williams J: Steve Martin, trainer. Bird Talk 3(25):25-28, 1985.
40. Wilson L: Behavior problems in adolescent parrots. Proc Assoc Avian Vet, Conf AAV, 1995, pp 415-418.
41. Wilson L: Feather destruction: A non-medical approach to the behavioral feather plucker. Proc Assoc Avian Vet, 1996, pp 3-9.
42. Wilson L: Phobic psittacines: An increasing phenomenon? Proc Assoc Avian Vet, 1998, pp 125-131.
43. Wilson L: What to do about biting and screaming. Proc Assoc Avian Vet, 1999, pp 71-76.
44. Wilson L: Behavior problems in pet parrots. *In* Olsen G, Orosz S (eds): Manual of Avian Medicine. Philadelphia, Mosby, 2000, pp 124-147.
45. Wilson L: The one-person bird: Prevention and rehabilitation. Proc Assoc Avian Vet, 2000, pp 69-73.
46. Wilson L: Height dominance revisited. Original Flying Machine 5:11, 2001.
47. Zantop, D: Anafranil for prolapsing cloacas. Exotic DVM 5(5):2003.

CHAPTER
4

Nutritional Considerations

Section I: Nutrition and Dietary Supplementation

Section II: Nutritional Disorders

Nutritional Considerations Section I

Nutrition and Dietary Supplementation

DEBRA McDONALD, PhD, BSc (HONS I)

Selection or formulation of appropriate diets for companion and aviary birds is based on wild feeding ecology, digestive anatomy and physiology, and nutritional requirements of related species. Research indicates that requirements of some key nutrients for psittacines vary from those of poultry. Apart from vitamin E, there is no evidence to suggest that vitamin and trace mineral requirements for psittacines are greater than those recommended for poultry.[54] While there are substantial differences between production species and companion bird species, dietary requirements of poultry remain the standard for estimating the needs of companion birds. Individual nutrient classes will be discussed with particular focus on recent research into the nutritional requirements of companion birds. See Nutritional Disorders, Section II of this chapter for aspects of malnutrition and nutritional diseases commonly diagnosed in companion birds.

Water

Calculated water intake of adult Australian parrots does not correlate with observed water intake (Table 4.1.1).[37] Desert-adapted birds require less water intake than tropical birds. Changes in diet or environmental temperatures can alter water intake.

Table 4.1.1 | Water Intake Per Day of Various Birds

Species	Body Weight (g)	Calculated Water Intake (ml)	Actual Water Intake (ml)
Budgerigar[86]	30-35	0.7-0.8	4
Canary[85]	18-24	0.4-0.6	4
Lovebird[86]	55	1.3	10
Cockatiel[86]	100	2.4	13.6
Cockatoo[86]	300-900	7-22	15
Amazon/Grey[86]	350-600	8-14	17-35

WATER REQUIREMENTS OF PSITTACINE NEONATES

The ratio of feed to water to maximize survivability in the growing chick is dependent on age; insufficient water within the first few days after hatch leads to high mortality, and insufficient solids result in slow growth rates.[31] Studies of cockatiels indicate changes in the proportions of solids to water from 7:93 for the first 4 days after hatch, increasing to 30:70 thereafter.[65]

NUTRITIONAL SUPPLEMENTATION OF DRINKING WATER

Supplementation of vitamins and minerals via the drinking water is not recommended. Water intake can vary inter- and intraspecifically and is influenced by environmental temperatures and diet. The high redox potentials of minerals, such as zinc, iron and copper, can destroy vitamins, and some vitamins are light-sensitive. It is impossible to standardize intake of vitamins via drinking water. Vitamin A and D toxicoses have been reported in macaws and conures being supplemented with liquid vitamins.[7,67,78] Dehydration may result if the additives decrease water intake due to unpleasant taste or unfamiliar coloration.

Energy

When calculating energetic requirements of birds, the following equations are used:

Passerine BMR
kcal/day = $114.8 \times kg^{0.726}$
kJ/day = $480 \times kg^{0.73}$

Non-passerine BMR
kcal/day = $73.5 \times kg^{0.734}$
kJ/day = $kg^{0.73}$

Basal metabolic rate (BMR) is calculated from energy expended when a bird is sleeping. Perching can increase energy expenditure in budgies 2-fold; preening, eating and shuffling locomotion cause 2.3-fold increase and flight increases as much as 11 to 20 times over BMR.[10] Energy requirements of free-living birds are greater than those of their captive counterparts due to the increased energy required for thermoregulation, food procurement and territorial defense. However, the daily needs for amino acids, minerals and vitamins are relatively constant regardless of energy expenditure.

Climate can influence BMR. Psittacines from temperate climates have BMRs approximately 20% higher than those of tropical species, with seasonal changes in thermoregulation varying from 3.07 times BMR in winter (5.9° C) to only 2.77 times BMR in summer (20.7° C).[10]

ENERGY REQUIREMENTS

Daily consumption of calories must exceed daily energy expenditure for a sustained period in order for overweight or obese body conditions to develop. A diet for weight loss should be replete in all nutrients so that protein, essential fatty acids, vitamins and minerals are present in amounts sufficient to support normal physiological processes and to retain lean body tissue. Reducing fat content of the diet too quickly or too far has led to obsessive eating behaviors in obese double yellow-headed Amazons.[22] Formulated calorie reduction diets generally contain lower levels of fat with simultaneous increase in indigestible fiber, air or moisture. Increased levels of dietary fiber slow gastric emptying. Insoluble fibers have a greater effect on slowing gastrointestinal tract (GIT) transit time than soluble fibers. Fiber also increases excretion of bile acids and fat.

ENERGETIC COSTS OF MOLT

Feather replacement requires energy and specific nutrients, as well as metabolic and physiological adaptations.[24] Energy costs of the molt include the caloric content of the new feathers and feather sheath, the energy required in their synthesis, and the energy required to produce and maintain feather pulps. Approximately 3 to 10% of total body mass (20 to 30% total lean body mass) of passerines is replaced during a complete molt.[24] Daily energy expenditure of passerines undergoing a rapid complete molt can increase from 3% (early and late in the molt) to 20% (at peak molt),[52] with BMR doubling in some passerines at peak molt.[34] Increases in energy expenditure due to rapid molt can be partly offset by reductions in other activities such as locomotion or singing.[28,34]

Feathers grow throughout the day and night at similar rates,[50] but the feather material deposited at night when most birds are fasting is of a slightly different quality. Feather synthesis at night requires complex and costly modifications to the metabolism of amino acids, compared to daytime synthesis, so the overall costs of molt may be lower in areas with relatively longer day length.[51]

Carbohydrates

Carbohydrates (Fig 4.1.1) are used to produce energy in the form of adenosine triphosphate (ATP) from glycolysis

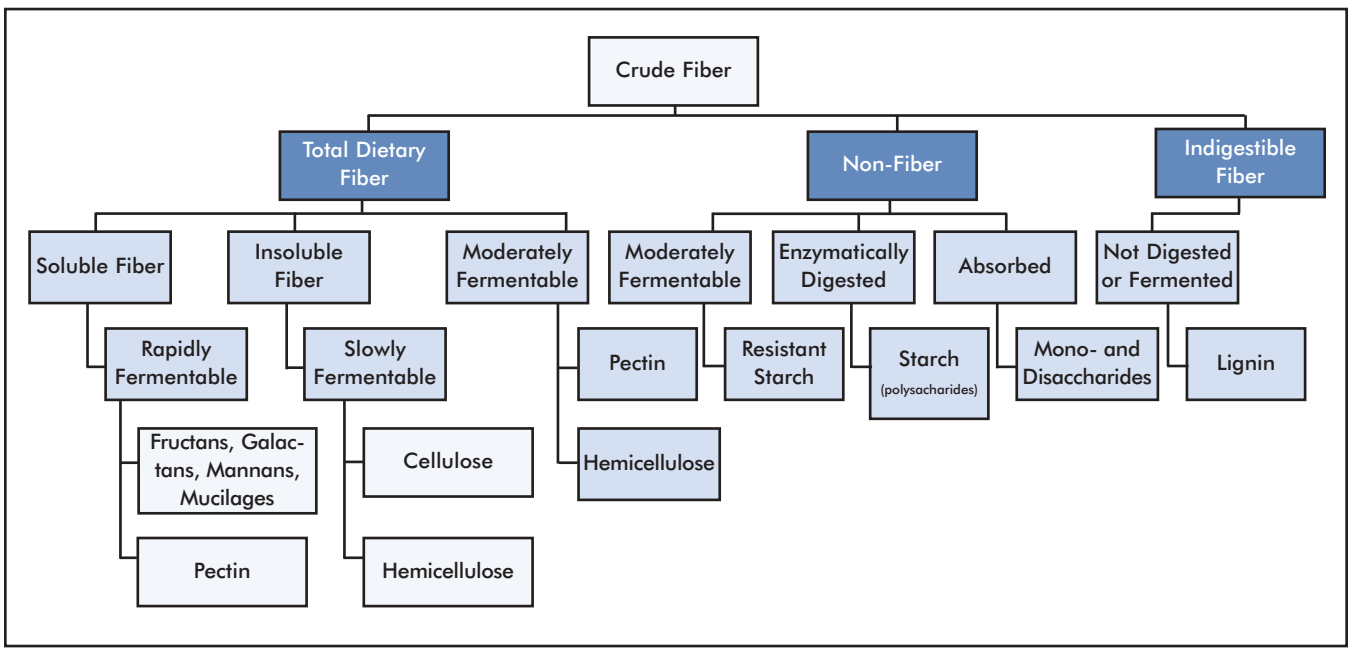


Fig 4.1.1 | Classification of carbohydrates.

Table 4.1.2 | In the Absence of Adequate Dietary Carbohydrate, Glucogenic Amino Acids are used to Manufacture Carbohydrates

- Alanine
- Arginine
- Asparagine
- Aspartic Acid
- Cysteine
- Glutamic Acid
- Glutamine
- Glycine
- Histidine
- Methionine
- Phenylalanine
- Proline
- Serine
- Threonine
- Tryptophan
- Tyrosine
- Valine

and the TCA (tricarboxylic acid) cycle, and produce heat from the oxidation of glucose to CO_2 and H_2O. They are also used to produce precursors of other nutrients, synthesize glycogen or fat from glucose, decrease luminal pH through production of short-chain fatty acids, and increase the population of anaerobic flora. The antibacterial properties of short-chain fatty acids may decrease pathogenic intestinal bacteria and may be important in prevention of, and recovery from, intestinal disorders.

The central nervous system and erythrocytes require glucose for energy, in contrast to muscles that can utilize substrates such as fatty acids. In the absence of adequate dietary carbohydrates, amino acids (glucogenic amino acids via the gluconeogenic pathway) are shunted away from growth and production to be used for glucose synthesis (Table 4.1.2).

SUCRASE AND FRUIT SUGARS

Sucrose, one of the predominant disaccharides of fruit sugars (Figs 4.1.2, 4.1.3), is easily digestible. However, some insectivorous passerines, such as thrushes that feed on diets high in protein/fat and low in carbohydrate, lack the sucrase enzyme necessary for the digestion of these simple sugars. The differences in proportions of fruit mono- and disaccharides are important for species that lack the sucrase enzyme. Avoid feeding these birds fruits high in disaccharides such as mango, apricot, nectarine and peach.

POLYSACCHARIDES

While simple sugars such as the monosaccharides and disaccharides are readily absorbed, the α-bonds of starches require further digestion. Heat from extrusion processes gelatinizes the starch molecule, increasing its digestibility. The ß-bonds of some of the complex carbohydrates, such as those comprising the fibrous fraction of foods, require microbial degradation that may not be sufficient in psittacines and passerines. Cellulose is composed of ß-bonds and is generally unavailable as a source of energy, while hemicellulose consists of varying proportions of α- and ß-bonds, depending on the source. Hemicellulose is therefore partially digestible without microbial breakdown, but its close association with the polyphenolic lignin often makes it indigestible. The carbohydrate content of psyllium fiber consists predominantly of hemicelluloses, so is partially digestible.

Fermentation of Polysaccharides

Postgastric microbial fermentation of polysaccharides occurs in nectarivorous, frugivorous and florivorous species. The process of enzymatic digestion taking place prior to fermentation benefits species that feed on easily digested foods such as nectar and fruit, as fermentation of nutrient-rich foods is not energetically efficient. In contrast, pregastric fermentation occurs in the hoatzin, which feeds on mature leaves, stems and branches.[18] Such bulk foods are generally incompatible with flight.

Chitin

Chitin is a naturally occurring polysaccharide similar in structure to cellulose with an additional amino group. It is the principal polysaccharide of cell walls of fungi and

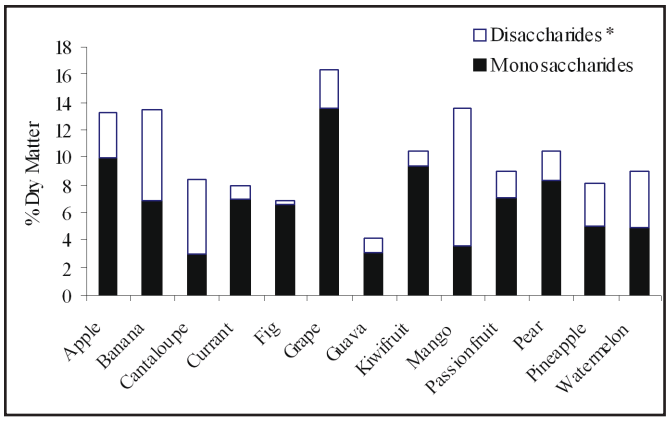


Fig 4.1.2 | Sugar content of fruits commonly fed to birds.
*Require sucrase for digestion

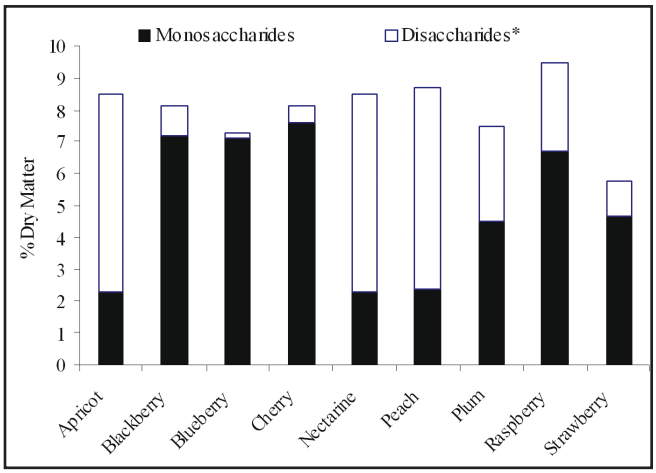


Fig 4.1.3 | Sugar content of stone fruits and berries.
*Require sucrase for digestion

Table 4.1.3 | Chitin Content of Various Invertebrates and Fungi

Food Item	Chitin Content
Fungi	5-20%
Worms	20-38%
Squid	3-20%
Scorpion	30%
Spider	38%
Cockroach	35%
Crab	70%

the primary constituent of the exoskeletons of crustaceans and invertebrates (Table 4.1.3). The digestion of chitin is considered low, but it still presents a useful energy source for some species. While chitinase activity has been identified in starlings, raptors and a variety of seabirds, it is low in chickens and absent in African grey parrots and pigeons. Measurements of crude protein may overestimate protein availability for birds that lack chitinase enzymes.

Protein

Proteins are composed of the nitrogen-containing molecules, amino acids. They can be manufactured from dietary precursors (non-essential) or are required as dietary constituents (essential). Amino acids that are deemed to be essential for birds include arginine, isoleucine, leucine, lysine, methionine, phenylalanine, valine, tryptophan and threonine. In addition, glycine is known to be essential for budgerigars (*Melopsittacus undulatus*),[79] and histidine and proline are essential for chickens. It is presumed that the digestion of proteins by psittacines reflects that of poultry. Lorikeets digest only 13.3% of complete protein (egg white).[16] Digestion of proteins is more efficient in nestlings than in adult birds.

MEASUREMENT OF PROTEIN CONTENT OF FOODS

Protein values of food (crude protein) are measured using the Kjeldahl technique with the assumption that all nitrogen in the food sample is present as protein and that all proteins contain 16% nitrogen. Crude protein is calculated as follows:

$$CP\ (\%) = g\ N/kg \times 6.25$$

While this value is commonly used for converting nitrogen values to protein, it is not applicable to all foods, especially some seeds.

DIETARY REQUIREMENTS FOR PROTEIN AND AMINO ACIDS

The dietary requirement for protein varies with age and physiological state, being highest in hatchlings and females laying large clutches, and lowest in adults at maintenance. In granivorous birds protein requirement is correlated with body size,[29] with larger species such as macaws possibly requiring more protein than smaller birds. Preliminary studies with African grey parrots (*Psittacus erithacus erithacus*) indicate a protein requirement of 10 to 15% (dry matter).[27] Protein requirement for the adult budgerigar is 6.8% (balanced protein diet).[14] Sunflower seeds, safflower seeds and peanuts are deficient in the amino acids required by poultry.[54]

High fiber diets can increase fecal nitrogen content due to bacterial digestion, which can confound the results of a protein digestion study.[63] Nectarivorous and frugivorous species have lower obligatory protein losses. Rainbow lorikeets (*Trichoglossus haematodus*) may require as little as 2.9% protein when fed high-quality, readily digested protein.[16]

While high dietary protein has been implicated in renal dysfunction and gout in psittacines, studies of male cockatiels (*Nymphicus hollandicus*) indicate that protein levels of 20, 30 or even 70% do not result in renal insufficiency.[32] However, 70% dietary protein is not recommended, as excessively high protein is strongly correlated with a significant increase in liver lesions.[32] Excess protein has been associated with overgrowth of beaks and nails.[35] Introduction of birds to new diets with varying levels of protein should be undertaken gradually, as sudden changes in dietary protein levels may result in nephritis and gout.[32] Since birds are uricotelic, an overload of the excretory ability of the kidneys, caused by excessive intake of proteins or nucleic acids, may lead to hyperuricemia. Dehydration exacerbates this problem and may produce visceral and articular gout even without excessive dietary protein.[35]

PROTEIN REQUIREMENTS FOR REPRODUCTION

Protein requirements during egg production are influenced by clutch size/frequency and the protein composition of eggs. There is little taxonomic variation in amino acid composition of avian eggs.[39,49] Protein requirements for species that lay only single eggs are similar to maintenance requirements, but that may increase if essential amino acids are lacking. Birds laying eggs require dietary amino acids for maintenance, growth of the oviduct and accretion of egg proteins, at least a week prior to the first oviposition.[29,49] Budgerigars can maintain breeding performance on 13.2% protein. However a diet of white millet, canary seed and hulled oats (13.4% protein) containing only half the necessary lysine, methionine and cysteine is not sufficient to support reproduction.[1]

PROTEIN REQUIREMENTS FOR CHICKS

Protein requirements for growth are highest at hatch and decrease over time as growth rate slows. Altricial chicks have a higher fractional growth rate and may have higher total amino acid requirements. Protein requirement for growth of cockatiels has been estimated at 20%. In addition, the protein must include 0.8 to 1.5% lysine.[64,65] Many birds in the wild supplement their diets with insects, which will often provide additional protein; however, some wild birds just increase their food intake. While there is generally sufficient crude protein, lysine and arginine in commercial hand-rearing mixes for psittacines, most lack sufficient quantities of the sulphur amino acids, methionine and cysteine, which leads to stunted feather growth.[85]

PROTEIN REQUIREMENTS FOR FEATHERS

Feathers comprise a large percentage of total body protein (22% lovebirds; 28% budgies) with approximately 15% by mass contained in the sheath.[50] However, the amino acid composition of whole plumage differs from that of the sheath and calamus.

Dietary deficiencies in some of the sulphur amino acids cause a pronounced curvature and periodic restriction of the rachis, abnormal persistence of the basal sheath, and misshapen vanes.[50] Feather strength is correlated with adequate dietary lysine (0.203%).[80] Cysteine is abundant in the epidermal structure and feather barbs.[51] While cysteine is frequently cited as a potentially limiting nutrient in the growth of plumage, cysteine reserves may be sufficient for keratin synthesis during overnight periods of fasting and short-term sulphur amino acid deficiencies. Dietary deficiencies of methionine result in dark, horizontal "stress lines" on feathers,[38] while threefold excesses are correlated with soft, weak feathers.[80] Tyrosine is an important factor in melanogenesis, and a deficiency in phenylalanine also impairs melanogenesis.[83]

Production of sheaths during molt can increase protein requirements 4 to 8% per day above maintenance requirements.[51] The additional energy required for thermoregulation may increase food intake to provide sufficient protein for feather growth without increasing the proportion of protein in the diet. A number of products on the market promoted as supplements during periods of molt are deficient in the required amino acids.[87]

Lipids

Lipids supply energy, essential fatty acids and facilitate the absorption of fat-soluble vitamins. In addition, they are precursors of many hormones and eicosanoids.

CLASSIFICATION OF FATS

Fats are described by the length of the carbon chain, the number of double bonds and the exact position of the double bonds. Short-chain fats contain 2 to 4 carbons, medium-chain fats 6 to 10 and long-chain fats 12 to 24 carbons. Fatty acids with 4 to 12 carbons are found in milks; those with 10 to 12 carbons are found in certain seed oils. Longer chain fatty acids are common in plants of marine origin.

ESSENTIAL FATTY ACIDS

Saturated fatty acids (SFA) are those where all carbons of the fat are satisfied with a single bond to another element. If one double bond is introduced, they are monounsaturated fatty acids (MFA). Those with two or more double bonds are polyunsaturated fatty acids (PUFA). The preferred nomenclature for fatty acids is based on the position of the double bond from the methyl end (Fig 4.1.4). For example, linoleic acid 18:2 (*n*-6) contains 18 carbons with two double bonds, placed six carbons from the last double bond with reference to the terminal methyl end. Higher evolved animals are unable to manufacture fatty acids of the *n*-3 or *n*-6 families and must obtain these from dietary sources. Fatty acid composition of seeds and nuts that are available for companion birds may not resemble that of wild diets (see Fig 4.1.6).

PUFA profiles are not amenable to dietary manipulation. Grains and seeds are generally rich in linoleic acid (*n*-6), grasses and leaves in α-linolenic acid 18:3 (*n*-3) and docosahexaenoic acid (*n*-3) (DHA). Fish and other aquatic insects are rich in 20:5 (*n*-3) or 22:6 (*n*-3).

FATTY ACIDS AND EICOSANOIDS

Certain membrane fatty acids have specific roles in the regulation of cell functions. Arachidonic acid (AA), γ-linolenic acid and eicosapentaenoic acid (EPA) act as precursors for the synthesis of eicosanoids, an important group of immunoregulatory molecules that function as local hormones and mediators of inflammation. Changes in characteristics of fatty acids available to cells modify the fatty acid composition of the membrane phospholipids of those cells and may influence inflammatory processes (Fig 4.1.5).

Cis and *Trans* Fatty Acids

While most natural fatty acids occur in the *cis* form (carbons on same side), processing such as extensive heat or partial hydrogenation can convert fats to the *trans* form

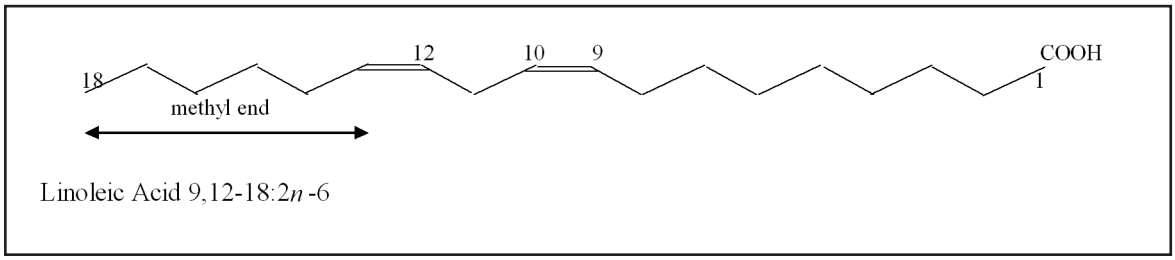


Fig 4.1.4 | Structure of *n*-6 fatty acid.

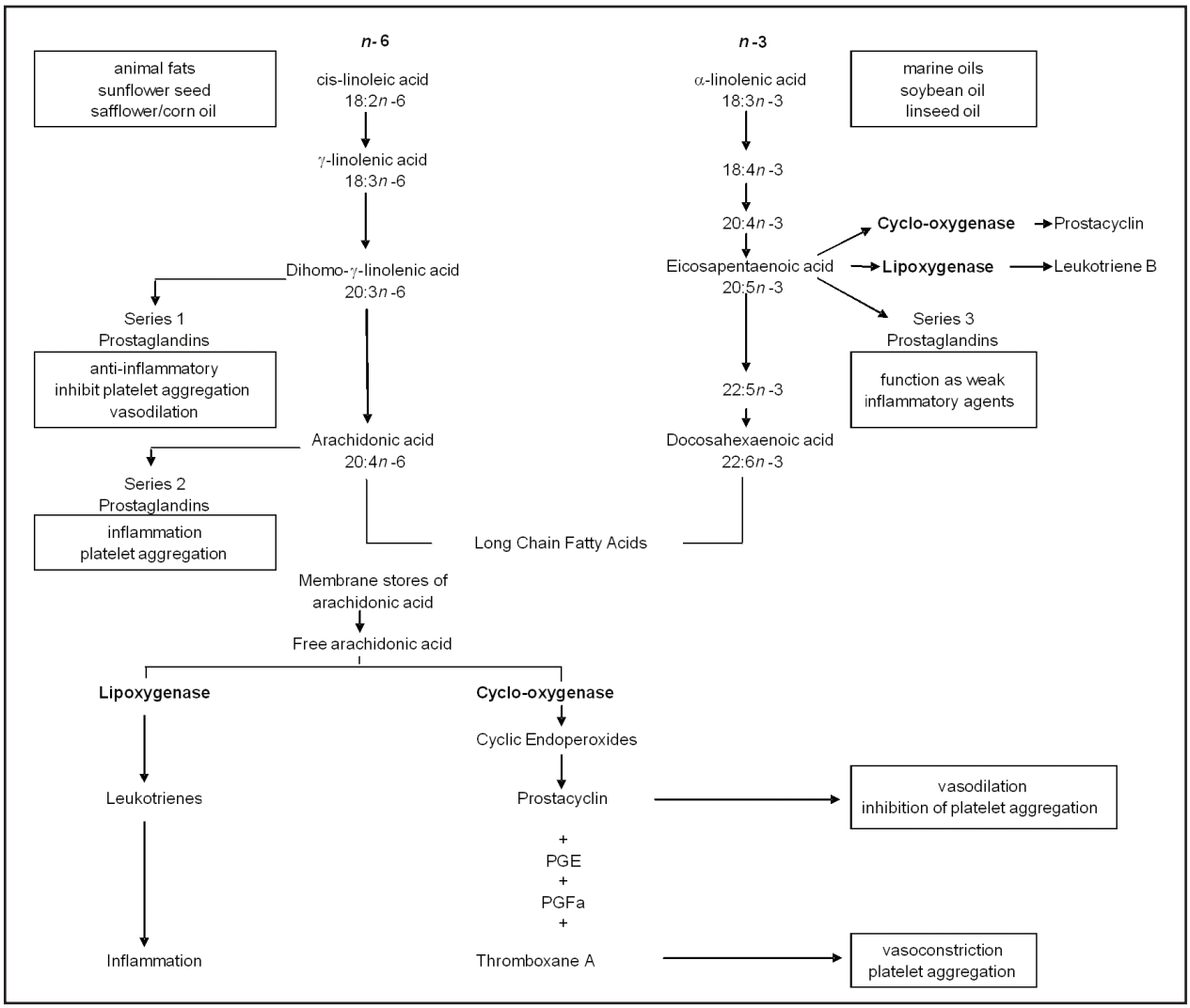


Fig 4.1.5 | Production of eicosanoids from essential fatty acids.

(carbons on opposite sides), which affects their biological activity. Although both *cis* and *trans* forms are metabolized for energy, *trans* isomers cannot function as essential fatty acids.

Antioxidants and Lipid Peroxidation

Insufficient antioxidants such as vitamin E in the feed also may enhance lipid peroxidation during storage.[48] Diets high in PUFA require additional antioxidant protection to prevent rancidity. There are a number of naturally occurring substances in food that have antioxidant properties including vitamins A, C, E, and yellow-colored carotenoids such as ß-carotene.

Antioxidants help to counter the detrimental effects of oxygen-free radicals. Oxygen-free radicals have been implicated in the development of cancer, inflammatory conditions and heart disease. A deficiency of antioxidants may promote peroxidation of membrane phospholipids.

OBESITY

Obesity can lead to congestive heart failure or hepatic lipidosis and may predispose a bird to diabetes mellitus or exacerbate this illness. Body weight relative to a bird's

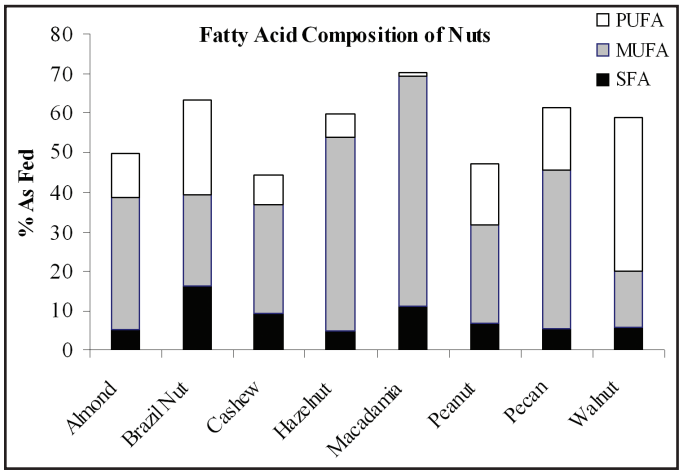


Fig 4.1.6 | Fat composition of nuts commonly fed to birds.

Table 4.1.4 | Lipid Content of Egg Yolk of Various Birds

Species	Chicken	Gull	Pigeon
Mode	Precocial	Semi-precocial	Altricial
Triacylglycerol	71.4%	35.1%	58%
Phospholipid	20.7%	24.6%	30.7%
Cholesterol Ester	0.8%	1.1%	5%
Free Cholesterol	5.6%	5.9%	4.7%

Table 4.1.5 | Fatty Acid Composition of Seeds of Wild Food Plants of the Orange-bellied Parrot (*Neophema chrysogaster*)[44]

	Linoleic Acid C18:2n-6	α-linolenic Acid C18:3n-3	AA C20: 4n-6	EPA C20: 5n-3	DHA C22: 6n-3
Introduced Mainland					
Atriplex prostrata	0.04	0.13	0.27	0	0.30
Cakile maritima	0.06	23.8	0	0	1.23
Chenopodium glaucum	0.06	5.28	0.14	0.02	0.06
Indigenous Mainland					
Halosarcia pergranulata	0	1.6	0	0	0
Samolus repens	0	1.53	0	0	0
Sarcocornia quinqueflora	0	2.0	0	0	0
Suaeda australis	0.23	2.39	0	0	0.04
Indigenous Tasmania					
Baumea tetragona	0	4.48	0	0	0
Gahnia grandis	0	0.37	0	0	0
Restio complanatus	0	3.55	0	0	0
Commercial	0	1.42	0	0	0.08

optimal weight has been used as a defining criterion for obesity because body weight is easier to measure than body fat. Body weight in excess of optimal body weight of 1 to 9% is acceptable, 10 to 19% is considered overweight and greater than 20% is defined as obese.

DEPOSITION OF FATS IN EGG YOLK

The yolks of precocial and altricial birds vary in lipid composition (Table 4.1.4). However, determination of specific fatty acid dietary requirements has been undertaken only on granivorus species. In contrast to fatty acid profiles of commercial grains, fatty acid composition of wild seeds on which the orange-bellied parrot (*Neophema chrysogaster*) feeds is characterized by a distinct lack of *n*-6 fatty acids (Table 4.1.5).[44]

BRAIN LIPIDS

There is a surge of brain growth in the second half of the embryonic/early neonatal stage, with specific uptake of docosahexaenoic acid (DHA) by embryonic brain tissue. The selective depletion of yolk phospholipid DHA results in a range of cognitive, behavioral and visual impairments. The high proportions of amino acids in brain tissues imply a requirement for adequate levels of both *n*-3 and *n*-6 fatty acids in yolk lipids. While the avian embryo may be able to synthesize DHA from α-linolenic acid, this ability may be species-specific. Yolk lipids of the domestic chicken maintained on formulated diets are almost devoid of α-linolenic acid, while those of the goose, pheasant and ostrich on similar diets have high levels of this fatty acid, providing sufficient precursors for conversion to DHA.

SPERMATOZOA LIPIDS

The high PUFA content of avian semen predisposes them to lipid peroxidation. Susceptibility is increased in membranes high in DHA, which is present in duck spermatozoa and can be exacerbated by low vitamin E. However, similarities in lipid peroxidation of chicken and ducks suggest the presence of high levels of antioxidant enzymes. Age-related decreases in sperm output are reduced with supplementation of 200 mg/kg vitamin E. Supplementation with longer chain essential fatty acids is also beneficial.

Vitamins and Supplementation Requirement

WATER-SOLUBLE VITAMINS

Water-soluble vitamins include the B complex and vitamin C. As dietary requirements for B vitamins have not been evaluated for companion birds, further discussion will be confined to disease entities that specifically impli-

cate imbalances in the B vitamins (see Section II Nutritional Disorders).

Vitamin C

Vitamin C (ascorbic acid) is involved in the syntheses of collagen, carnitine and catecholamine; in tyrosine, histamine, steroid, fatty acid and drug metabolism; and in the prevention of peroxidation. Birds under stress, including the stresses associated with high temperatures, growth and reproduction may have increased requirements for vitamin C.

Sources of Vitamin C

Most birds synthesize vitamin C in the kidney, liver or both. Evolutionarily, enzymatic activity occurs in the kidneys of birds in older orders and becomes localized in the liver of more advanced Passeriformes. Some passerines such as the vented bulbul (*Pycnotus* sp.) are unable to synthesize vitamin C. Vitamin C is concentrated in fresh fruits, green leafy vegetables and animal organs, with only small amounts in skeletal muscle.

Vitamin C Deficiency

Reproduction and growth increase the demand for collagen. Supplementation of 100-200 mg/kg vitamin C improves growth, egg production and eggshell strength of young chicks and hens exposed to high environmental temperatures.[68] Dietary requirements also may vary with age, as willow ptarmigan (*Lagopus lagopus*) adults are able to synthesize sufficient vitamin C, whereas chicks require supplementation with 750 mg/kg.[21] Berries that form part of the diet of the willow ptarmigan during the breeding season can contain up to 5000 mg/kg vitamin C.[21]

Vitamin C is susceptible to destruction with handling and processing. While it is stable when exposed to boiling water for short periods, a greater proportion is destroyed when heated at low temperatures for long periods. Any form of processing that ruptures tissue (such as freezing and thawing) exposes vitamin C to air losses due to oxidation, but vitamin C is generally stable during normal pelleting processes.

Vitamin C Toxicity

Metabolites of L-ascorbic acid such as oxalic acid can bind calcium. Excesses of vitamin C also can bind copper, resulting in growth deficiencies and increases in the incidence of aortic rupture and decreases in elastin content of the aorta if diets are also deficient in copper. It is important to minimize dietary vitamin C content for species that are susceptible to iron storage disease, as vitamin C improves the absorption of iron by facilitating the reduction of the ferric form to the more absorbable ferrous state.

FAT-SOLUBLE VITAMINS

It is important to maintain an appropriate balance with the fat-soluble vitamins as they all compete for sites of uptake. Dietary excess of one vitamin can diminish uptake and availability of another, despite adequate dietary intake.

Vitamin A

Vitamin A is involved in vision, reproduction, immunity, membrane integrity, growth, embryogenesis and the maintenance of epithelial cells. Vitamin A is of animal origin and does not occur in plant tissues. Some carotenoids can be converted to vitamin A in the intestinal wall via a specific enzyme.

Forms of Vitamin A

The most active form (retinol) is susceptible to moisture, heat and light. The retinal aldehydes are incorporated in rhodopsin and influence dim light vision. Retinoic acid supports growth and tissue differentiation but not vision. Although retinoic acid appears to play a role in testosterone synthesis, it does not support the production of sperm.

Dietary Requirement

Vitamin A requirements vary among production species (Table 4.1.6). Dietary requirements of cockatiels (*Nymphicus hollandicus*) at maintenance are 2000-4,000 IU/kg.[30] Levels below 10,000 IU/ kg do not significantly influence plasma levels in cockatiels.[30] Dietary deficiencies of vitamin A may not impact immunocompetence for up to eighteen months in birds previously maintained on sufficient dietary Vitamin A. Dietary vitamin A can be adequately provided from 2.4 mg/kg ß-carotene in cockatiels. Recessive white canaries (*Serinus canaria*) are unable to convert ß-carotene to vitamin A and require three times as much vitamin A as colored canaries.[88]

Sources of Vitamin A

The vitamin A content of animals varies. Vitamin A levels in invertebrates are extremely low (Table 4.1.7).[4] Fish store large amounts of vitamin A in the liver and fatty tissue. Supplementation with cod liver oil is not recommended. Seeds and nuts are generally low in carotenoids (Table 4.1.8), while some fruits can provide large quantities (Table 4.1.9).

The vitamin A content of many formulated products, most parrot foods and nectar-replacement products (Fig 4.1.7, Table 4.1.10) exceed dietary recommendations for poultry[54] and cockatiels.[30] While the vitamin A content of some products is higher than data reported by manufacturers, poor packaging may result in breakdown of the vitamin.[41]

Table 4.1.6 | Vitamin A Requirements of Birds, Expressed as IU/kg of Feed Dry Matter

	Growing	Laying
Chicken[54]	1,670	4,440
Turkey[54]	5,550	5,550
Quail, Coturnix[54]	1,830	3,670
Duck, Pekin[54]	2,780	4,440
Goose[54]	1,670	4,440
Cockatiel (Maintenance)[32]	2,000	

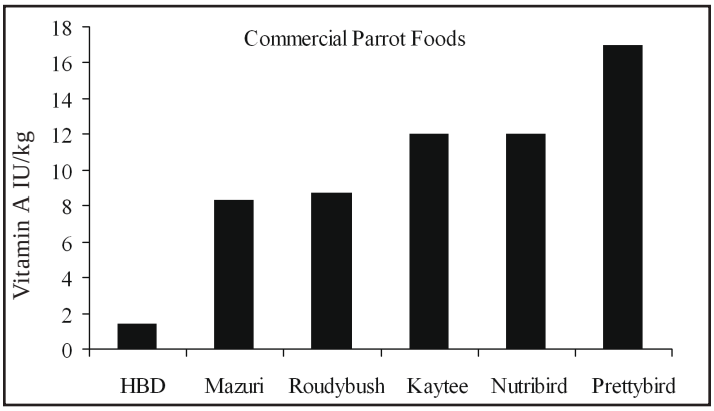


Fig 4.1.7 | Vitamin A content of various commercial parrot foods.

Table 4.1.7 | Fat-soluble Vitamin Content of Various Invertebrates Commonly Fed to Birds

Invertebrate	Vitamin A (IU/kg)	Vitamin E (mg/kg)
Mealworm, larvae	811	30
Cricket, adult[3]	811	80
Cricket, juvenile[3]	471	70
Earthworm, wild-caught[4]	2400	70
Earthworm, commercial[4]	328	230
Fruit Fly[4]	0	23
Waxworm[4]	150	500

Table 4.1.8 | Carotenoid Content of Nuts/Seeds

Nut/Seed	Carotenoid (RE/g)
Flax	0
Safflower	0.53
Sesame	0.09
Sunflower	0.53
Almond	0
Brazil	0
Hazel	0.71
Macadamia	0
Peanut	0
Walnut	1.29

Table 4.1.9 | Carotenoid Content of Fruits[41]

Nut/Seed	Carotenoid (RE/g)
Apple	3.3
Banana	3.15
Cantaloupe	315.0
Grape	3.76
Honeydew	3.87
Kiwi Fruit	10.32
Mango	212.9
Raspberry	9.68
Strawberry	3.2
Watermelon	43.11

Data reprinted with permission from Elsevier Science[41]

Table 4.1.10 | Vitamin A Content of Various Commercial Nectar Products[41]

Product name	Vitamin A (IU/kg)	Vitamin E (mg/kg)
Aristopet*	5,994	6
Aves Nectar†	24,150	22
Avione*	4,296	20
Elliots Dry*	666	2.8
Elliots Wild nectar*	666	1.3
HBD Adult Lifetime Fine (Maintenance)‡	1,400	215
HBD High Potency Fine (Breeding)‡	1,500	240
Lory Life Nectar§	52,900	54
Lory LifePowder§	10,130	11
Marion Lory¶	8,500	250
Nekta Plus§	12,470	12
Nekton Lori†	60,550	34
Nekton Lori and Gelb†	244,820	136
Noah's Kingdom§	330	25
Passwells*	9,990	29
Quicko Nectar§	400	5
Rainbow Landing Lorikeet Nectar§	22,467	45
Roudybush 15% Protein§	19,500	33
Roudybush 9% Protein§	18,860	81
Sheps Lori dry*	167	1.7
Sheps Wet*‖	12,500 (333)	25 (1.8)
Wombaroo Nectar†¶	26,640 (28,740)	89 (27)

Data reprinted with permission from Elsevier Science[41]

*Independent laboratory analyses, Australia 2002
†Author's laboratory analyses, 1999
‡Independent laboratory analyses, USA 2002
§Laboratory analyses (Graffam, 1999)
¶Manufacturer's data, 1999
‖Manufacturer's data, 2002

Measuring Vitamin A

Approximately 90% of total-body vitamin A is contained in the liver.[46] Storage levels of 2-5 IU vitamin A/g liver are deemed to be adequate.[46] Plasma retinol does not change dramatically with marginal vitamin A status, and reduced levels are generally not detected until animals reach a severe deficiency. It is not adequate to evaluate vitamin A status from blood samples. Liver biopsies are recommended.

Interpreting Laboratory Data

Units are reported in *international units* (IU) for vitamin A or *retinol equivalents* (RE) for provitamin A.

The equivalencies are as follows:

0.6 μg ß-carotene is equivalent to 1 IU provitamin A activity

1 RE = 1 μg retinol = 6 μg ß-carotene or 12 μg of other provitamin A carotenoids

1 RE = 3.33 IU retinol = 1.818 IU vit. A palmitate = 10 IU ß-carotene

Data that is reported as μg/dl retinol can be converted to SI units (μmol/L) as follows:

μg/dl x 0.0349 = μmol/L

Approximately 10% of the more than 500 carotenoids have provitamin A activity.

Signs of Vitamin A Deficiency

Clinical signs of vitamin A deficiency often resemble those of toxicity, and distinguishing between the two requires careful evaluation of dietary intake and other influencing factors. While turkey chicks exhibit signs of deficiency after only five weeks[2], cockatiels can be maintained on a diet devoid of vitamin A for up to two years before demonstrating clinical signs.[30] Signs of vitamin A deficiency can generally be classified into five categories:

Vision

Retinol is utilized as the prosthetic group in iodopsin (cones). Vitamin A deficiency results in a loss of opsin, the protein that converts vitamin A to rhodopsin, from the outer segments of the rods, leading to their eventual degeneration. Even in the late stages of vitamin A deficiency, it is possible to regenerate rods, but cones eventually disintegrate and result in blindness. Vitamin A deficiency also results in decreased secretions of tear glands.[40]

Bone

A deficiency results in reduced activity of osteoclasts, leading to excessive deposition of periosteal bone by the unchecked function of osteoblasts.

Maintenance of Epithelial Tissue

Dietary deficiencies of vitamin A can alter the permeability of lipoprotein membranes of cells and intracellular particles. Low levels of liver vitamin A are correlated with symptoms of focal metaplasia of the excretory duct as well as the glandular epithelium of salivary glands. Loss of membrane integrity also interferes with water retention, and renal uric acid deposits can result. Coccidiosis can lead to the destruction of vitamin A in the gut and injure microvilli of the intestinal wall, decreasing the absorption of vitamin A.[69] Vitamin A deficiency in chicks is characterized by poor feathering on the head, neck and breast regions, as well as facial dermatitis.

Reproduction

Defects in reproduction, including increased time between clutches, reduced hatchability, increased embryonic mortality, decreased survival time of progeny, decreased testes size, failure of spermatogenesis and a decline in sexual activity in males, are correlated with deficiencies of vitamin A. These may be associated with failure to maintain healthy epithelium.[17]

Immune Function

Both deficiency and excess of dietary vitamin A suppress immune function. Vitamin A deficiency in chicks leads to a rapid loss of lymphocytes. Diarrhea, pneumonia and blunted immune response are characteristic of vitamin A deficiency in cockatiels.[33] Deficiencies lead to phagocytic activity in macrophages and neutrophils, and impair intestinal IgA response.[33]

Vitamin A Toxicity

In the wild, noncarnivorous birds are rarely exposed to dietary excess of vitamin A. These birds probably depend on the conversion of carotenoids to biologically active vitamin A. Toxicities are avoided because the efficiency of conversion of vitamin A from ß-carotene decreases with higher levels of intake. Conversion efficiency in the chicken drops from a ratio of 2:1 to 5:1.[46] Studies of Japanese quail also indicate a saturation of the retinol-transporting system, as birds supplemented with ß-carotene do not develop increased levels of retinyl palmitate.[60] Cockatiels at maintenance are more susceptible to vitamin A toxicity than deficiency.[30] Perhaps ß-carotene would be a superior source of vitamin A in some psittacine diets; however, ß-carotene may not be appropriate for hand-rearing mixes because chicks may not efficiently convert ß-carotene to vitamin A. Toxicities have been reported in cockatiels maintained at 10,000 IU/kg of vitamin A. Many commercial diets exceed this level.[30] Some hand-rearing diets have levels in excess of 47,000 IU/kg.[85] In lorikeets, commercial diets high in vitamin A and deficient in vitamin E are correlated with high rates of infertility, decreased hatching and survivability of chicks.[42] The author hypothesizes similar levels may contribute to the increased incidence of iron storage disease in these birds. Vitamin A toxicity causes epithelial damage and keratinization of squamous cells.[33] Epithelial damage results from penetration of retinol into the lipid portion of the membrane, causing it to expand. Weakening of the membrane results from the inelastic protein portion of the membrane resisting expansion and increases access to pathogens and infection. Clinical signs of Vitamin A toxicity include:

Vocalization Patterns

Cockatiels maintained on excess dietary vitamin A exhibit frequent stress calls of greater intensity and duration.[30] Vitamin A toxicities may contribute to behavioral problems in companion birds. Vocalization changes were observed in psittacines maintained on diets that contained recommended levels of vitamin A.[47,75]

Iron Storage Disease

See Section II Nutritional Disorders for the potential contribution of excess vitamin A to iron storage disease. Splenic hemosiderosis has been correlated with excess vitamin A in cockatiels.[30]

Pancreatitis

Pancreatitis was diagnosed in cockatiels fed excessively high levels of vitamin A.[33] Hypervitaminosis A increases the activity of sucrase and eliminates the duodenum's ability to regulate this enzyme in the small intestine[71] which may lead to diabetes and digestive difficulties.

Table 4.1.11 | Ultraviolet Light

Type of light	Distance (ft)	UVA (microwatts/cm²)	UVB* (microwatts/cm²)
Reptisun 5.0	1	23	10
Vitalite	1	6	1.6
Blacklight	1	153	2.6
Active UV Heat			
100 watt flood	1	400	50
100 watt flood	2	110	12.5
160 watt flood	1	640	85
160 watt flood	1.5	480	55
160 watt flood	2	320	25
160 watt flood	3	142	11
275 watt flood	2	720	66
275 watt flood	2.5	520	48
275 watt flood	3	320	30
275 watt flood	4	180	20
100 watt spot	2.5	1130	70
100 watt spot	3	562	40
100 watt spot	4	400	30
160 watt spot	3	1500	137
160 watt spot	4	1200	100
Location	**Time**	**Direct/ Shade**	**UVB (microwatts/cm²)**
Natural Sunlight			
Equator	noon	direct sunlight	265
Germany	noon	direct sunlight	175
Yucatan	noon	direct sunlight	250
Illinois	noon	direct sunlight	260
Illinois	7:00 am	direct sunlight	12
Illinois	7:00 pm	direct sunlight	17
Illinois	1:00 pm	shade	54
Illinois	5:00 pm	shade	22

*UVB is the biologically active ultraviolet light

Reproduction

Excesses of vitamin A may interfere with uptake of vitamin E, compromising fertility, hatchability and survivability of chicks.

Antioxidant Status

Vitamin A supplementation of laying hens increases liver, egg yolk and embryonic liver concentrations at the expense of vitamin E, compromising the antioxidant status of progeny. High levels of vitamin A reduce uptake of astaxanthin, a powerful carotenoid antioxidant that protects mitochondria from damage by Fe^{+2} catalysed lipid peroxidation.

Vitamin D

Vitamin D is a group of closely related compounds that possess antirachitic activity. They are obtained directly from the diet or from irradiation of the body. The two major natural sources (provitamins) are cholecalciferol (D_3 in animals) and ergocalciferol (D_2, predominantly in plants). Both D_2 and D_3 forms also can be ingested and further metabolized to 25-hydroxyvitamin D_3 through hydroxylation first by the liver, and then again to 1,25-dihydroxyvitamin D_3 in the kidneys. Cholecalciferol can be produced in the skin of most mammals from provitamin 7-dehydrocholesterol via activation with ultraviolet light in as little as 11-15 minutes daily. Vitamin D enhances intestinal absorption and mobilization of calcium and phosphorus through the hormone 1,25-dihydroxyvitamin D_3. Circulating levels of 25-hydroxyvitamin D_3 are indicative of vitamin D status. One IU of vitamin D activity is equivalent to the activity of 0.025 μg vitamin D_3. As vitamin D_2 has only 1/10 the activity of vitamin D_3 in chicks the International Chick Unit (ICU) is used with reference to vitamin D_3 in poultry. Plasma half-lives vary with the form of the vitamin ranging from 5 to 7 days (vitamin D) to 20 to 30 days (25-(OH)D_3) (see Chapter 5, Calcium Metabolism). In mammals and reptiles, activation depends on UVB radiation (290-320 nm) (Table 4.1.11).

Table 4.1.12 | Vitamin D and Calcium Content of Various Commercial Foods*

Product	Manufacturer	Vitamin D_3 (IU/kg)	Calcium (%)
Avipels	Blue Seal	4170	1.11
Bird of Paradise	Zeigler	3970	1.34
Bird of Prey (frozen)	Animal Spectrum	3470	1.05
Chick Starter	Blue Seal	4000	1.19
Crane	Mazuri	10830	2.62
Exotic Game Bird	Mazuri	2500	0.89
Flamingo	Mazuri	6670	1.72
HPC	HBD International	150	0.69
Nutribird Parrot	Nutribird	1200	0.9
Palm Cockatoo	SSP	1900	1.1
Psittacine Breeder	Roudybush	1560	1.0
Psittacine Handfeeder	Roudybush	1560	1.0
Psittacine Maintenance	Roudybush	890	0.44
Scenic Bird	Marion	1600	1.2
Poultry	NRC	200	0.99
Turkey	NRC	900	0.5

*From manufacturer's published data 1999-2002.

Dietary Requirement

The dietary requirement for vitamin D in poultry is 200 IU/kg. While higher dietary requirements are evident for the turkey (900 IU/kg) and Japanese quail (1200 IU/kg), optimum levels for companion birds have yet to be established. It has been suggested that dietary levels for poultry are adequate for breeding African grey parrots[76] (Table 4.1.12), but many formulated foods exceed these levels.

Vitamin D Deficiency

Vitamin D synthesis can be affected by liver malfunction; intestinal disorders can reduce absorption of the vitamin and kidney failure can prevent synthesis of 1,25-$(OH)_2D_3$. Inadequate exposure to UVB radiation prevents production of vitamin D in the skin. Glass windows block the penetration of UVB rays. The first signs of vitamin D deficiency include decreased egg production, thinning or

absence of eggshells, and an increased incidence of embryonic death. Inadequate maternal transfer of vitamin D_3 and 25-(OH)D_3 results in the failure of development of the upper mandible and failure to pip.

Vitamin D Toxicity

Vitamin D toxicity may arise from an excess of dietary vitamin D or a deficiency in the other fat-soluble vitamins. Toxicity leads to widespread calcification of soft tissue. Toxic levels can be transferred maternally to the embryo, leading to abnormalities in chick development. Safe upper limits in chicks less than 60 days old are 40,000 IU/kg and 2800 IU/kg in birds older than 60 days.[69]

Vitamin E

Vitamin E consists of tocopherols and tocotrienols in four isomeric forms, α, ß, δ, and γ. α-tocopherol has the highest vitamin E activity followed by ß, δ and γ, respectively. Dietary requirements of vitamin E are dynamic, with increased requirements with diets high in PUFA, oxidizing agents, vitamin A, carotenoids, trace minerals and decreased requirements in diets high in other fat-soluble antioxidants, sulphur-containing amino acids and selenium. Vitamin E is one of the least toxic vitamins, however, high doses decrease absorption of vitamins A, D and K, resulting in reduced hepatic and egg yolk storage of vitamin A,[77] impaired bone mineralization[20] and coagulopathies.[53] Studies of pelicans indicate that 500-10,500 IU vitamin E/kg result in decreased growth and coagulopathy.[53] Japanese quail under heat stress (34° C) require 250 mg/kg vitamin E and 0.2 mg/kg Se.[66] Various researchers have recommended up to 60 mg α-tocopherol per gram of PUFA. Many formulated foods have less than 200 mg/kg of vitamin E. Vitamin E status can be evaluated from single blood samples, but the magnitude of body stores may not be reflected in α-tocopherol concentrations.[45,81] High dietary concentrations of vitamin E elevate vitamin E levels in the blood. Deficiencies affect the neuromuscular, vascular and reproductive systems.

Vitamin K

Vitamin K plays a major role in blood clotting factors and is involved in the synthesis of osteocalcin, with deficiencies resulting in increased bleeding times and toxicities resulting in kidney tubule degeneration. Vitamin K is available as phylloquinone (K_1) from plants, menaquinone (K_2) from bacteria and menadione (K_3) which is synthetic. Vitamin K_1 is present as the fat-soluble portion of plant chlorophyll (Table 4.1.13). An energy-dependent process absorbs vitamin K_1 from the intestine, whereas vitamins K_2 and K_3 are passively absorbed. Estrogens stimulate the absorption of vitamin K_1. Vitamin K_3 has twice the potency of natural vitamin K_1 on a weight-to-weight basis. Vitamin K_3 is the most common form in commercial bird foods. Choline can impact the activity of water-soluble K_3, destroying up to 80% within 3 months. γ-irradiation of foods to increase storage life also inactivates vitamin K, while heat treatment can increase its bioavailability (Table 4.1.14).

Table 4.1.13 | Vitamin K Content of Fruit on a Dry Matter Basis

Fruit	Vitamin K (mg/100 g)
Apple peel, green	60.0
Apple peel, red	20.0
Apples, no skin	0.4
Avocado, raw	40.0
Banana, raw	0.5
Grapefruit, raw	0.02
Grapes, raw	3.0
Kiwifruit, raw	25.0
Melon, raw	1.0
Orange, raw	0.1
Peach, raw	3.0
Pineapple, raw	0.1
Plum	12.0

Table 4.1.14 | Influence of Heat Treatment on Vitamin K Content on a Dry Matter Basis

Vegetables	Vitamin K (mg/100 g) raw	Vitamin K (mg/100 g) cooked
Broccoli	205	270
Carrot	5	18
Cauliflower	5	10
Coriander leaf	310	1510
Mint leaf	230	860
Parsley	540	900

Table 4.1.15 | Conversion of Carotenoids to Vitamin A Relative Rat-biopotency[46]

Carotenoid	Biopotency
α-carotene	25
β-carotene	100
γ-carotene	14
Cryptoxanthine	29

It has been suggested that mortality from cerebral hemorrhage in some species of fig parrots (*Opopsitta* spp.) is the result of dietary deficiency of vitamin K.[11] If fig parrots have developed a dependency on vitamin K_2 produced by the gut microbes of termites, they may be unable to process sufficient vitamin K_1 from plant sources. Daily supplementation of 300 μg vitamin K_1 per fig parrot appears to alleviate clinical signs.[11]

Nutritional Influence on Feather Pigmentation

The dietary pigments utilized by passerines for their coloration are referred to as carotenoids. Psittacines do not use carotenoids for feather pigmentation. Each carotenoid produces a specific color. Carotenoids are subdivided into carotenes and xanthophylls (Table 4.1.15). Carotenoids may be used directly in the plumage or modified to other forms prior to incorporation into the feathers or skin (Fig 4.1.8, Tables 4.1.16-4.1.18). Each carotenoid appears to have its own individual pattern of absorption, plasma transport and metabolism. There are considerable species differences in the types of carotenoids that are preferentially absorbed and metabolized. Many carotenoids act as potent antioxidants and stimulate the immune system.

The yellow pigmentation of the helmeted honeyeater

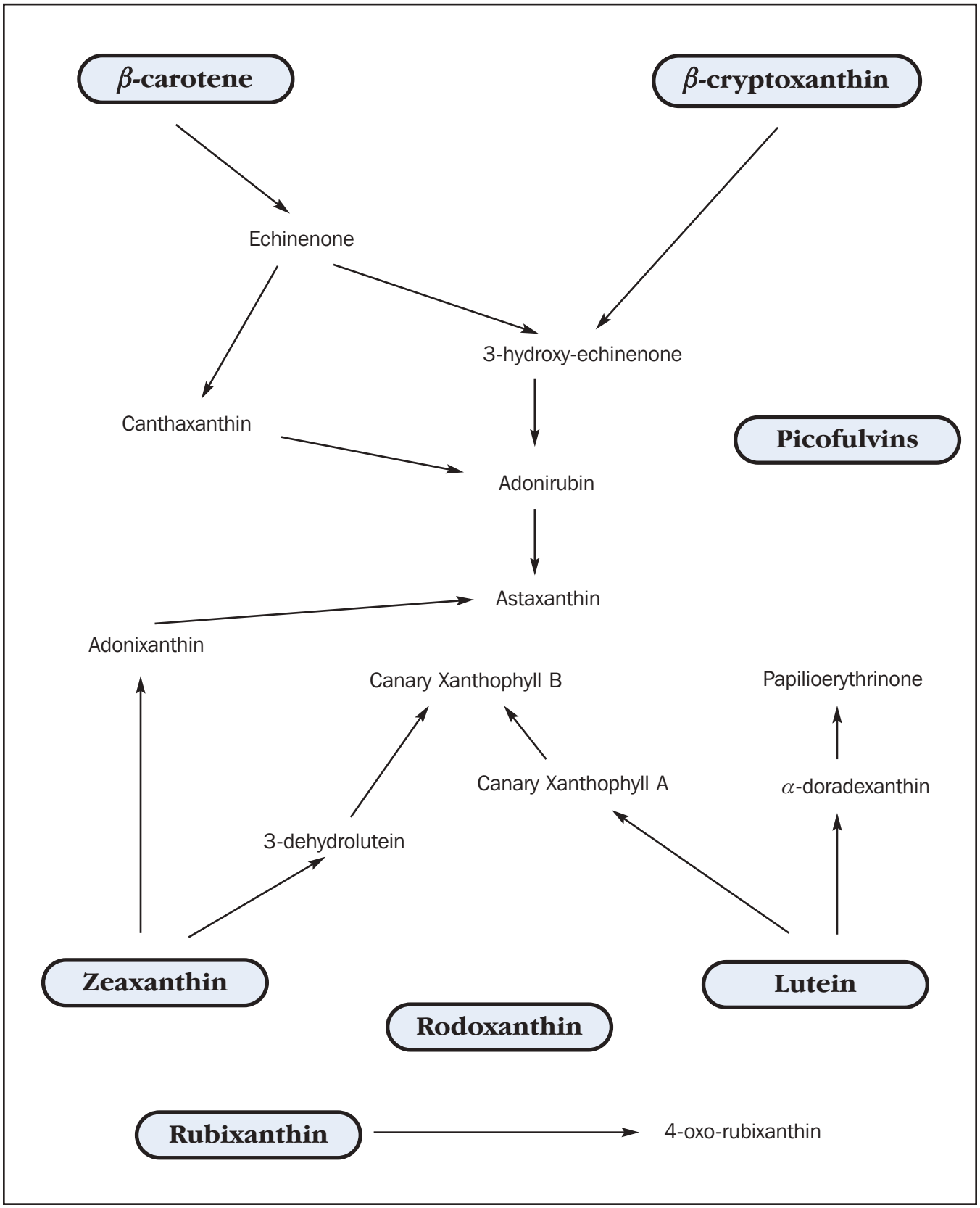


Fig 4.1.8 | Metabolic pathways for various dietary carotenoids. Rodoxanthin and picofulvins are deposited directly from the food into the feathers and are not metabolically transformed.

Table 4.1.16 | Metabolic Pathways of Various Carotenoids Responsible for Pink-colored Feathers of Passerines

Species Name	Common Name	Feather Pigment	Metabolism	Original Pigment	Feather Pigment
Aegithalos caudatus	Long-tailed tit	3-hydroxy-echinenone	oxid	ß-crypto/ß-carot	pink
Carduelis cannabina	Common redpoll	3-hydroxy-echinenone	oxid/hydrox	ß-crypto/ß-carot	carmine red
		4-oxo-rubixanthin	oxid	rubix	
		4-oxo-gazaniaxanthin	oxid	rubix	
Carduelis flammea	Linnet	3-hydroxy-echinenone	oxid/hydrox	ß-crypto/ß-carot	carmine red
		4-oxo-rubixanthin	oxid	rubix	
		4-oxo-gazaniaxanthin	oxid	rubix	
Carpodacus roseus	Pallas's rosefinch	3-hydroxy-echinenone	oxid/hydrox	ß-crypto/ß-carot	bright pink
		4-oxo-rubixanthin	oxid	rubix	
		4-oxo-gazaniaxanthin	oxid	rubix	
Fringilla coelebs	Chaffinches	3-hydroxy-echinenone	oxid	carots	copper-pink
		4-oxo-rubixanthin			
		dehydrolutein			
		astaxanthin			
Pyrrhula pyrrhula	European bullfinch	α-doradexanthin	oxid	ß-carot	pinkish-red
		astaxanthin			
		adonirubin			
Rhodospiza obsoleta	Desert finch	canthaxanthin	oxid	ß-carot	pink

Oxid=oxidation, hydrox=hydroxylation, carots=carotenoids, ß-crypto=ß-cryptoxanthine, ß-carot=ß-carotene, rubix=rubixanthin

Table 4.1.17 | Metabolic Pathways of Various Carotenoids Responsible for Red-colored Feathers of Passerines

Species Name	Common Name	Feather Pigment	Metabolism	Original Pigment	Feather Pigment
Bombycilla cedrorum	American waxwing	rodoxanthin	direct	rodox	red
Carduelis carduelis	Gold finch (face mask)	canary xanthophylls B/C/D	dehydrog	lut/keratin	red
Carduelis cucullata	Red siskin	α-doradexanthin	oxid	lut	red
		canthaxanthin			red
Colaptes auratus	Northern flicker	astaxanthin	oxid	carots	red
		lutein/zeaxanthin	direct	lut/zea	yellow
		ß-cryptoxanthin	direct	ß-crypto	yellow
Dendrocopos major	Great spotted woodpecker	astaxanthin	oxid	lut/zea/carots	red
		α-doradexanthin			
		adonirubin			
Hirundo rustica	Swallow	lutein	direct	lut	red
		zeaxanthin	direct	zea	
		3-hydroxy-echinenone	oxid	ß-crypto/ß-carot	
Loxia curvisrostra	Common crossbill (m)	3-hydroxy-echinenone	oxid	ß-crypto	red
Loxia leucoptera	White-winged crossbill	4-oxo-rubixanthin	oxid	rubix	red
		4-oxo-gazaniaxanthin		gazan	
Luscinia calliope	Siberian rubythroat	astaxanthin	oxid	carots	ruby red
		α-doradexanthin			
		adonirubin			
Pericrocotus flammeus	Scarlet minivet (m)	astaxanthin	oxid	carots	red
		α-doradexanthin			
		adonirubin			
		canthaxanthin			
Picus viridis	Green woodpecker	α-doradexanthin	oxid	lut	red
		picofulvins		lut/zea/ß-crypt	green/yellow
Pinicola enucleator	Pine grosbeaks (m)	3-hydroxy-echinenone	oxid	ß-crypt	red
		4-oxo-rubixanthin		rubix	
Pyrrhula erythaca	Grey-headed bullfinch	canary xanthophylls A/B	dehydrog	lut	orange-red
Serinus pusillus	Red-fronted serin	canthaxanthin	oxid	ß-carot	red
Trichodroma muraria	Wallcreeper	astaxanthin	oxid	zea	red
Uragus sibiricus	Long-tailed rosefinch	3-hydroxy-echinenone	oxid	ß-crypt/ß-carot	red

Rodox=rodoxanthin, lut=lutein, zea=zeaxanthin, gazan=gazaniaxanthin

Table 4.1.18 | Metabolic Pathways of Various Carotenoids Responsible for Yellow-colored Feathers of Passerines

Species Name	Common Name	Feather Pigment	Metabolism	Original Pigment	Feather Pigment
Bombycilla garrulus	Bohemian waxwing	canary xanthophylls (tail)	dehydrog	lut/zea	yellow
		astaxanthin (wing patches)		ß-caro	
C. sinica/C. spinoides	Asiatic finches	canary xanthophylls	direct	lut	yellow
Carduelis atrata	Black siskin	canary xanthophylls A/B	dehydrog	lut	yellow
Carduelis carduelis	Gold finch (wing bars)	canary xanthophylls B/C/D	dehydrog	lut	yellow
Carduelis chloris	Greenfinch	canary xanthophylls	dehydrog	lut	yellow
Carduelis citrinella	Citril finch	canary xanthophylls A/B	dehydrog	lut	yellow
Carduelis spinus	Siskin finch	canary xanthophylls A/B	dehydrog	lut	yellow
Emberiza citrinella	Yellowhammer	lutein/zeaxanthin	direct	lut/zea	yellow
E. melanocephala	Black-headed bunting	lutein/zeaxanthin	direct	lut/zea	yellow
Fringilla coelebs	Chaffinches (secondaries)	lutein	direct	lut	yellow tinge
Fringilla coelebs	Chaffinches (rump)	lutein	direct	lut	
Leiothrix lutea	Peking robin	dehydrolutein	dehydrog	lut/zea	yellow
		α-doradexanthin	oxid	carots	
		astaxanthin	oxid	carots	
Loxia curvisrostra	Common crossbill (f)	canary xanthophylls A/B	dehydrog	lut	yellow
Motacilla flava	Yellow wagtail	lutein	direct	lut	yellow
		zeaxanthin		zea	
Oriolus oriolus	Golden oriole	lutein	direct	lut	yellow
		zeaxanthin	direct	zea	yellow
		oxo-carotenoids	oxid	lut/zea	bright yellow
Parus ater	Coal tit	lutein	direct	lut	yellow
		zeaxanthin		zea	
Parus ceruleus	Blue tit	lutein	direct	lut	yellow
		zeaxanthin		zea	
Parus major	Great tit	lutein	direct	lut	yellow
		zeaxanthin		zea	
Pericrocotus flammeus	Scarlet minivet (f)	lutein/zeaxanthin	direct	lut/zea	yellow
Pinicola enucleator	Pine grosbeaks (f)	lutein	direct	lut	yellow
		dehydrolutein	dehydrog	zea	
Regulus regulus	Goldcrest	lutein	direct	lut	yellow/green
		zeaxanthin	direct	zea	orange
Serinus mozambicus	Yellow-fronted canary	canary xanthophylls A/B	dehydrog	lut	yellow
Serinus serinus	Serin	canary xanthophylls	dehydrog	lut	yellow

(*Lichenostomus melanops cassidix*) (Fig 4.1.9) is replaced with near white pigmentation (Fig 4.1.10) when maintained on commercial nectar mixes high in vitamin A. Feather structure and light refraction can influence feather color. Carotenoids also can act synergistically with melanin pigments. Dark colors (black, brown, gray and related tints) produced by melanin and porphyrin pigments complexed with trace minerals are influenced by amino acid nutrition. Stress can influence feather quality and color.

Minerals

CALCIUM AND PHOSPHORUS

Adequate dietary calcium can be negated by high phosphorus content. The dietary ratio of Ca:P should range from 1:1 to 2:1.[61] Calcium availability can be influenced by solubility and particle size. Foods high in oxalic acid form insoluble calcium oxalates, while phytates bind phosphorus and decrease its availability. Additional oxalic acid can be produced from excesses of vitamin C. Fats can form insoluble calcium soaps.

The calcium content of nuts and seeds is variable (Fig 4.1.11). Some green leafy vegetables and tubers that are high in calcium also are high in oxalic acid (Figs 4.1.12, 4.1.13), which decreases the calcium availability.

Invertebrates generally have poor Ca:P ratios (Table 4.1.19). Crickets should be maintained on 80% poultry mash and 20% calcium carbonate. Water should be provided *ad lib* from produce (such as a slice of apple) or free water. If die-off of crickets occurs from constipation, restrict gut loading to 48 hours prior to feeding. When

Fig 4.1.9 | Helemeted honeyeater, full pigmentation (in-house nectar mix).

Fig 4.1.10 | Helemeted honeyeater, light pigmentation (commercial nectar mixes).

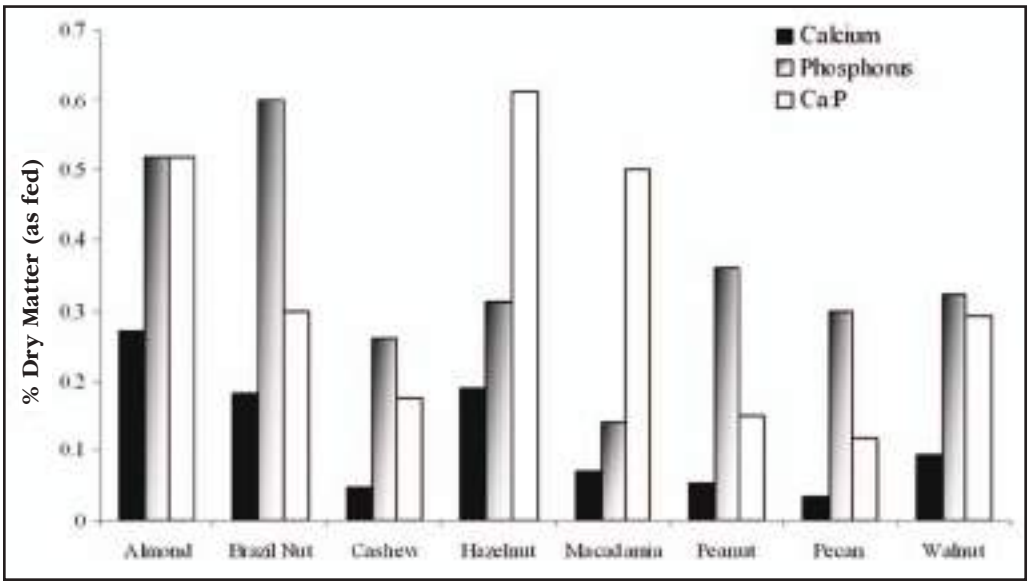


Fig 4.1.11 | Calcium and phosphorus content of nuts and seeds.

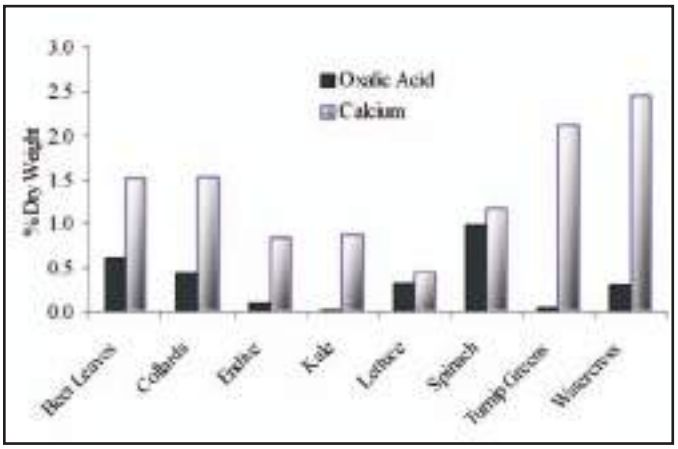


Fig 4.1.12 | Oxalic acid and calcium content of greens.

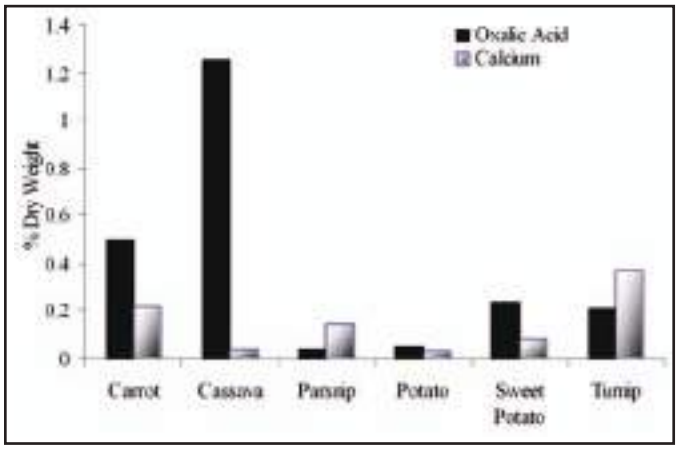


Fig 4.1.13 | Oxalic acid and calcium content of tubers.

Table 4.1.19 | Calcium and Phosphorus Content of Invertebrates

Invertebrate	Part	Calcium (% DM)	Phosphorus (% DM)	Ca:P
Bogong Moth (*Agrotis infusa*)	Abdomen	0.64		
	Wings	0.17		
	Whole	0.25		
Cricket (*Acheta domesticus*)	Adult	0.21	0.78	0.27
	Pinhead	1.29	0.79	1.63
Fruit fly (*Drosophila melanogaster*)	Pupae	0.77	2.73	0.28
	Larvae	0.59	2.3	0.26
	Adult	0.1	1.05	0.10
Mealworm (*Tenebrio molitor*)	Larvae	0.11	0.77	0.14
	Beetle	0.07	0.78	0.09
	Pupae	0.08	0.83	0.10

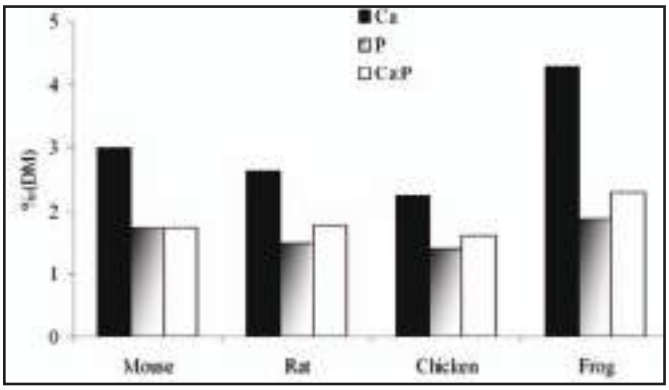


Fig 4.1.14 | Calcium and phophorus content of whole vertebrate prey.

feeding whole vertebrate prey, it is not necessary to supplement with calcium (Fig 4.1.14).

It has been suggested that optimum levels of calcium in feed are between 0.3 to 0.7%.[58] Many wild seeds provide 0.1 to 0.3% calcium (Table 4.1.20). Companies continue to provide formulated feeds in excess of 0.7%. It is possible that the high calcium content of formulated foods contributes to the development of renal disease that is observed in color mutation cockatiels.

IRON

Body iron is either hemal or non-hemal (Table 4.1.21). Hemal iron forms part of the porphyrin group and comprises 70 to 75% of total iron. Iron from animal sources is generally more available than that from plant sources. Iron in soy isolates may be unavailable. Pectin, vitamins A and C, and amino acids such as cysteine, histidine and lysine enhance iron uptake. High levels of calcium and/or phosphate decrease iron absorption in chicks. Cellulose and oxalate, as well as the heat and pressure of food processing, increase the bioavailability of iron. (Tables 4.1.22-4.1.25).

SELENIUM

Selenium (Se) and vitamin E function synergistically as

Table 4.1.20 | Calcium Content of Wild Food Resources of the Orange-bellied Parrot[44]

Species	Common Name	Calcium (%)	Seed Weight (mg)
Mainland Indigenous			
Halosarcia pergranulata	Black-seed glasswort	0.1	0.3
Samolus repens	Creeping brookweed	0.68	0.017
Sarcocornia quinqueflora	Beaded glasswort	0.28	0.3
Suaeda australis	Austral seablite	0.08	0.4
Introduced Species			
Atriplex prostrata	Hastate orache	0.08	1-3
Cakile maritima	Beach rocket	0.16	7.4-9.6
Chenopodium glaucum	Goosefoot	0.07	0.4
Tasmanian Species			
Baumea tetragona	Square-twig rush	0.04	0.3
Gahnia grandis	Brickmaker's sedge	0.3	7.7
Restio complanatus	Flat cord rush	0.2	0.5

antioxidants; the actions of vitamin E cannot be replaced with selenium. Selenium toxicity decreases hatchability, growth and reproductive success and results in deformed embryos, diminished immune function, abnormal feather loss, emaciation and liver lesions.[23,74] Dietary selenium affects whole blood levels.[56] Mallards fed more than 10 mg/kg Se developed bilaterally symmetrical alopecia of the scalp and dorsal cervical midline, broken nails and necrosis of the tip of the beak.[56] Chicks raised on diets depleted of both vitamin E and Se show signs of exudative diathesis on the superficial pectoral muscles. Deficiencies of selenium are characterized by increases in heterophils and decreases in lymphocytes, basophils and hemoglobin. Selenium deficiency depresses plasma T_3 concentrations.[25] Dietary selenium up to 0.4 mg/kg appears to be adequate for large psittacines maintained on extruded diets with 200 mg/kg vitamin E.[43]

ZINC

Zinc is involved in cell replication and in the development of cartilage and bone. Normal serum zinc concentrations reported for parrots range between 0.5 and 5.8 ppm (7.65-84 μmol/L).[82,45] However, plasma and serum concentrations above 2 ppm (30 μmol/L) have been considered diagnostic for zinc toxicity in most species.[5,15,55,59,72,82] A lack of correlation between hepatic zinc levels and clinical diagnosis of zinc toxicity also has been reported.[12]

Zinc toxicosis usually arises from ingestion of zinc-coated aviary wire or metallic foreign bodies.[62] Clinical signs of zinc intoxication include anorexia, acute gastroenteritis, ataxia, lethargy, yellow-colored feces, vomiting, extreme loss of plumage and hepatomegaly.[72,85] It can cause pancreatic cell necrosis.[72] Excess dietary zinc negatively impacts tissue concentrations of α-tocopherol.[36] Zinc is more toxic in iron-deficient chicks than in properly iron-supplemented birds.[6,55] Liver biopsy is

Table 4.1.21 | Forms of Organic Iron

Hemal Iron	Non-hemal iron
Hemaglobin	Transferrin
Myoglobin	Ferritin
Cytochromes	Hemosiderin
Cytochome oxidase	Iron Proteinates
Catalase	—
Peroxidase	—

Table 4.1.22 | Iron and Vitamin C Content of Native Australian Fruits

Fruits	Iron mg/kg	Vitamin C mg/kg
Lillypilly, *Acmena smithii*	15.15	303
Wild ginger, *Alpinia caerulea*	5.7	199
Davidson plum, *Davidsonia pruriens*	127.66	BDL
Quandong, *Santalum acuminatum*	108.61	BDL
Wild fig, *Ficus platypoda*	80.12	59.35
Native gooseberry, *Physalis minima*	265.82	63.29

Data reprinted with permission from Elsevier Science[41]

Table 4.1.23 | Iron and Vitamin C Content of wild Australian Insects (expressed as dry matter)

Insect	Iron mg/kg	Vitamin C mg/kg
Honeypot ant, *Melophorus spp*	35	15
Lerp scale, *Psylla eucalypti*	78	100
Witchety grub, *Cossidae spp*	102	127
Bogong moth, *Argrotis infusa*	159	20
Green tree ant, *Oecophylla smaragdina*	400	58

Data reprinted with permission from Elsevier Science[41]

Table 4.1.24 | Iron Content of Invertebrates

Invertebrate	Diet	Iron mg/kg
Mealworm	wheat, grain, carrots	40
Mighty Mealy	wheat, brain, supplements	26
Super Mealworm	wheat, grain, carrots	50
Cricket, adult	cornmeal, wheat, soybean hulls, meat meal molasses, fish meal	110
Cricket, juvenile	shipped with raw potato	200
Wax Worm	none	80
Fruit Fly	commercial feed	450
Earth Worm, wild		11,100
Earth Worm, commercial	peat humus soil	5,800

Table 4.1.25 | Iron and Vitamin C Content of Fruits Commonly Fed to Birds

Fruit	Iron mg/kg	Vitamin C mg/kg
Apple, apricot, banana, fig, grape, raisin	—	Low <1,000
Watermelon	20	1130
Canteloupe	21	4130
Orange	9	4344
Papaya	9	5530
Strawberry	45	6725

Data reprinted with permission from Elsevier Science[41]

Table 4.1.26 | Concentrations of Hepatic Zinc in Birds

Species	Zn (mg/kg) Physiological Status	Zn (mg/kg) Intoxication	Reference
Budgerigar (*Melopsittacus undulatus*)	50.5 ± 12.7 (37.6-70.5) n=10	153 250	7
Budgerigar (aviary bred) (*Melopsittacus undulatus*)	64.7 ± 37 (29-126) n=8	No clinical signs of toxicity	12
Monk Parakeet (*Myiopsitta monachus*)	57.9 ± 34.5 (28.1-156) n=14	179 ± 73.7 (n=7)	13
Lovebird (*Agapornis roseicolli*)	42.5 ± 8.9 (37.5-50.2) n=5	75 156	62
Macaws (*Ara chloroptera, A. macao*)	38.9 ± 22 (12.0-115) n=77	150 ± 37.0 (n=3)	13
Rosellas and Lorikeets	74 ± 63 (27-166) n=4	Wild-caught	12
Galah (*Eolophus roseicapilla*)	31.6 ± 5.4 (24-45) n=16	Wild-caught	45
Sulphur-crested Cockatoo (*Cacatua galerita*)	37.5 ± 7.8 (25-59) n=21	Wild-caught	45
Long-billed Corella (*Cacatua tenuirostris*)	37.3 ± 9.8 (29-64) n=13	Wild-caught	45

not definitive as a diagnostic tool (Table 4.1.26). Signs of zinc deficiency include reduction in immune response, alterations to cell division, early embryonic death, fetal abnormalities, weak chicks at hatching, retarded growth, alopecia, dermatitis, delayed sexual development, abnormal skeletal formation and feathering.

Specific Diets

FRUIT AND POLLEN

Nectarivorous birds feed on a variety of pollens, plants, insects and their exudates.[9,19,57,89] Pollen is high in protein (Table 4.1.27), while its digestibility is relatively low in hummingbirds and lorikeets (4.5-6.6%).[8] As the sugar content of fruits increases, the volumetric intake and passage rate decrease.[84] The protein content of fig species is variable (4-25%).[26]

WHOLE PREY

Whole prey fed to birds in captivity can differ from that available in the wild (Tables 4.1.28, 4.1.29; Figs 4.1.15, 4.1.16). and may require supplementation. Feeding individual pieces of prey or eviscerated meat can contribute to nutrient imbalances. Feeding high proportions of liver can result in hypervitaminosis A. Supplement meat-based diets with $CaCO_3$, which has

Table 4.1.27 | Protein and Amino Acid Content of Pollen Sources in Australia[73]

Pollen source	Poultry require-ments	Eucalypts	Banksias	She-oak	Hakea	Wattles	Almond	Black /Spear thistle	Lavender	Onion weed	Saffron thistle
Amino acids (% protein)											
Threonine	3.77	3.66 (3.38-4.11)	4.06 (3.81-4.3)	3.67	4.26	3.97 (3.01-4.63)	4.58 (4.47-4.7)	3.25 (1.7-3.6)	4.17	3.27	4.05
Valine	3.47	4.94 (4.38-5.83)	4.92 (4.7-5.4)	4.07	4.78	4.70 (3.95-5.49)	5.21 (4.83-5.4)	4.33 (3.4-5.1	4.54	11.08	5.69
Methionine	1.68	2.14 (1.0-2.69)	2.24 (2-2.273)	2.44	2.04	2.58 (2.21-2.84)	1.77 (0.7-2.57)	1.78 (1.2-2.1)	2.21	1.30	2.55
Leucine	5.51	6.60 (5.97-7.63)	6.49 (5.6-7.6)	6.03	6.59	6.54 (5.35-7.28)	6.81 (6.41-7.4)	5.98 (4.6-6.4)	6.04	6.91	6.94
Isoleucine	3.35	3.97 (3.36-5.47)	3.89 (3.5-4.5)	3.34	3.93	3.89 (2.94-4.64)	4.3 (4-4.7)	3.98 (3.2-4.5)	3.59	4.56	5.03
Phenylalanine	2.99	3.94 (3.48-5.37	4.43 (3.71-5.4)	3.29	3.81	3.76 (3.21-4.24)	3.57 (2.3-4.9)	3.55 (2.6-4.1)	4.11	3.38	4.18
Lysine	4.01	5.65 (5.17-6.34)	5.74 (5.1-6.5)	4.37	4.66	5.3 (4.66-6.19)	4.97 (3.1-6.48)	3.93 (1-6.8)	6.38	3.77	6.77
Histidine	1.44	2.31 (1.8-3.84)	2.58 (2.37-2.98)	1.73	2.4	2.05 (1.73-2.36)	1.95 (1.82-2.1)	2.7 (1.4-3.1)	3.67	1.64	4.43
Arginine	5.51	6.2 (4.13-7.18)	7.36 (6.7-8.6)	6.44	6.41	5.92 (4.66-7.2)	5.05 (4.6-5.48)	4.5 (3.7-6.5)	4.31	7.40	4.48
Crude protein (%)	16.7	24.87 (20.5-29.4)	33.06 (31.2-36.9)	12.50	18.4	23.75 (21.7-24.9)	25.94 (23.3-30.7)	20.94 (16.1-31.8)	19.4	18.25	18.1
Fat (%)		2.01 (0.48-3.9)	2.18 (1.9-2.45)	1.93	2.82	1.52 (0.9-2.52)	2.32 (1.89-2.74)	2.42 (2.25-2.59)	2.9	4.50	3.86

Note: Data in parentheses indicate ranges.

Table 4.1.28 | Mineral Content of Whole Vertebrate Prey

	Ca	P	Ca:P	Mg
Mouse	3.0	1.7	1.7	0.16
Rat	2.6	1.5	1.8	0.08
Chicken	2.2	1.4	1.6	0.5
Frog	4.3	1.9	2.3	2.47

Table 4.1.29 | Fat-soluble Vitamin Content of Whole Prey

	Vitamin A (IU/g)	Vitamin E (mg/kg)
Mouse (12weeks)	657	74
Rat (adult)	335.3	152
Chicken (6 weeks)	35.59	61
Frog, green	25.11	82.2

Table 4.1.30 | Calcium Content of Various Supplements

Supplemental source	Ca (%)	P (%)
Calcium borogluconate	8.32	0
Calcium carbonate (ground limestone, oyster shell, cuttlebone)	40.04	0
Calcium gluconate	9.31	0
Calcium glucobionate (4.6% Ca)	23mg/ml	0
Calcium lactate	18.31	0
Calcium phosphate (monobasic)	17.12	24.47
Calcium phosphate (dibasic)	29.46	22.77
Calcium phosphate (tribasic)	38.76	19.97
Bone meal, steamed	31.74	15

Table 4.1.31 | Iron and Vitamin A Content of Invertebrates

Invertebrate	Diet	Iron (mg/kg)	Vitamin A (IU/kg)
Mealworm	Wheat, grain, carrots	40	810
Mighty mealy	Wheat, grain, supplements	26	160
Super mealworm	Wheat, grain, carrots	50	970
Cricket, adult	Cornmeal, wheat midds, soybean hulls, meat meal, molasses, fish meal	110	810
Cricket, juvenile	Shipped with raw potato	200	470
Waxworm	None	80	150
Fruit fly	Commercial feed	450	not detected
Earthworm, wild		11,100	2400
Earthworm, commercial	Peat humus soil	5800	330

Table 4.1.32 | Calcium Content of Insects

	Supplement	Ca:P
Mealworm	nil	1:9
Cricket	nil	1:16
Cricket	Gut loaded	1:5
Cricket	Gut loaded/dusted	1:3

the highest calcium content (Table 4.1.30).

INSECTS

There are limited varieties of invertebrates for captive birds, with mealworms, earthworms and crickets forming the bulk of the available diet. Hard-bodied insects that contain up to 50% of their body weight as chitin may be important sources of dietary fiber, as chitin is chemically similar to cellulose. Chitinase activity has been identified in starlings, raptors and a variety of seabirds. Vitamin E content of many insects is adequate, but vitamin A content is relatively low or undetectable (Table 4.1.31).[4] Insects (especially from colder climates) contain high levels of polyunsaturated fatty acids. Insects generally concentrate a number of carotenoids that may be important for pigmentation or antioxidant activity. Insects generally have poor Ca:P ratios (Table

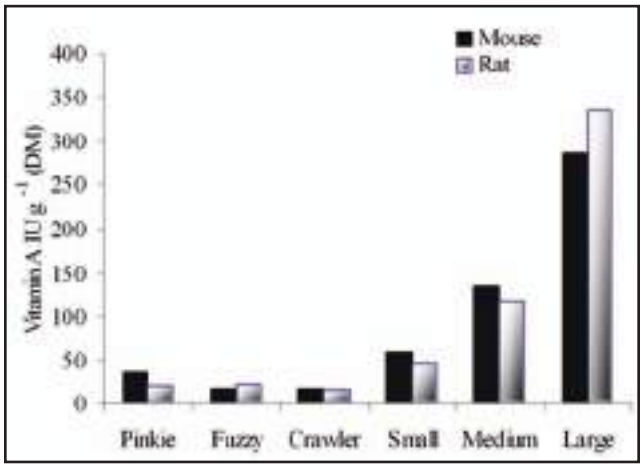


Fig 4.1.15 | Vitamin A content of rodents at various stages of development.

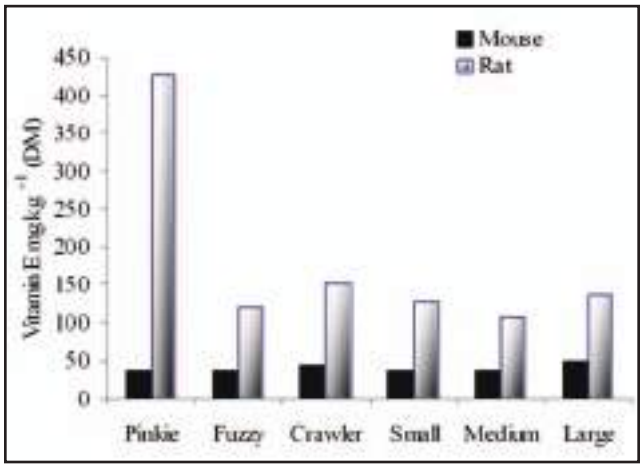


Fig 4.1.16 | Vitamin E content of rodents at various stages of development.

4.1.32). Calcium content of termites is low, but the content of their nest material (1.7%) provides a valuable source of calcium to birds, such as fig parrots, that nest in termitaria.[70]

FISH

Fish must be stored below -18° C to maintain nutritive value. Fish should be dry-thawed at <4° C up to 48 hours before use, and emergency thaws should be undertaken in plastic bags under cold running water to prevent the loss of nutrients. Supplementation of piscivorous diets is required for some key nutrients, depending on the species of fish fed and the method of preparation. Feeding whole fish is imperative to maintain proper Ca:P ratio. Iodine content of marine fish is considered adequate (0.9 mg/kg), while that of freshwater fish may be as low as 0.03 mg/kg. Sodium levels of marine fish are adequate if fish are not thawed in fresh water. Heat stress may increase a bird's sodium requirement. Vitamin A content of fish commonly fed to birds is adequate (Fig 4.1.17). Supplementation may be required if eviscerated fish are fed. Vitamin E levels of frozen fish are generally inadequate (Fig 4.1.18). Thawing of fish in water will deplete water-soluble vitamins. However, there is no data to support the supplementation of fish with water-soluble vitamins other than thiamine (B_1) if they are dry-thawed. Many fish contain thiaminase; these require 25 to 30 mg of thiamine per kg of fish fed.

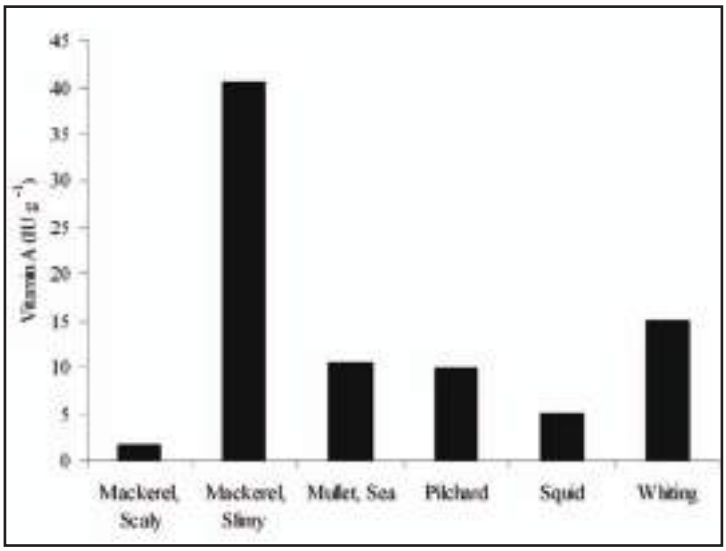


Fig 4.1.17 | Vitamin A content of fish stored in a frozen state (-10°C).

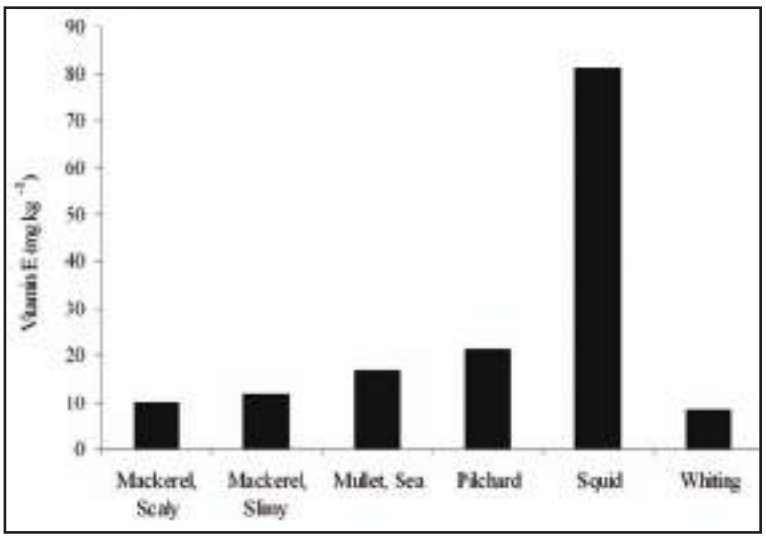


Fig 4.1.18 | Vitamin E content of fish stored in a frozen state (-10°C).

Labeling

Most products will display information regarding only a "guaranteed analysis." This provides an indication of maximum or minimum levels of crude protein, fat and fiber. However, there is no legal requirement for every batch to be chemically evaluated, rather these values often are derived from calculated values and thus may not be accurate. Furthermore, a value for "crude protein" provides no information about the digestibility of the protein or the proportions of various essential amino acids. A crude protein value for products containing invertebrates does not account for the proportion of nitrogen bound up in chitin. A crude fat content indicates neither whether the fats are saturated or unsaturated, nor the proportions of essential fatty acids. A crude fiber value does not delineate the proportion of soluble or digestible fiber, and provides no information about the lignin content.

SUMMARY

Nutrition is the single most important aspect of bird husbandry. Nutrition impacts the health, longevity, appearance and behavior of birds in captivity. The complex biochemistry and interactions between levels of nutrients coupled with the paucity of research in companion birds make choosing an appropriate diet very difficult.

Species differences between various psittacines makes dietary recommendations even more complex. Table 4.1.33 offers current estimated nutritional requirements for psittacines. A summation of critical factors in the selection of a diet should include:

1. Consideration of any species — specific dietary requirements or sensitivities.
2. Avoidance of excessive amounts of nutrients, as well as assuring adequate mineral levels.
3. Analysis of the components and availability of the listed manufacturers, "crude" protein, fat, calcium, etc., and knowledge of what method is used to measure these levels.
4. Awareness, as in human, dog and cat nutrition of the potential dangers inherent in preservatives and additives.
5. Recognition of the discrepancy between wild natural food sources and substitutions made by many commercial manufacturers.

Table 4.1.33 | Estimated Nutritional Requirements for Psittacines

Nutrient	Unit	Maintenance	Breeder
Protein			
Crude protein	%	10-15	15-22
Lysine	%	0.8-1.5	
Lipid			
Crude fat	%	5	10-15
Macrominerals			
Calcium	%	0.3-0.7[a]	0.7-1.2
Magnesium	%	0.15	
Phosphorus	%	0.3-0.7	0.5-0.8
Potassium	%	0.7	
Sodium	%	0.2	
Microminerals			
Copper	mg/kg	4-12	
Iron	mg/kg	100[b]	100
Manganese	mg/kg	65	
Selenium	mg/kg	0.30	0.4-0.5
Zinc	mg/kg	40-50	50-80
Vitamins			
Vit A	IU/kg	4000[c]	6000
Vit D_3	IU/kg	200-1200	2000
Vit E	mg/kg	200-250	250-350
Vit K_1	mg/kg	0.5[d]	0.5

[a]Calcium requirements established for budgerigars, some species may have higher requirements.
[b]Species susceptible to ISD may require less than 80 mg/kg.
[c]Beta carotene 22.4 mg/kg.
[d]Supplementation of 300 μg daily for fig parrots susceptible to vitamin K deficiency. Expressed on a dry matter basis. These values are estimates only and may not apply to all species.

References and Suggested Reading

1. Angel R, Ballam G: Dietary protein effect on parakeet reproduction, growth and plasma uric acid. Proc First Annu Conf Nutrition Advisory Group, Toronto, p 91, 1995.
2. Aye PP, et al: Induction of vitamin A deficiency in turkeys. Avian Dis 44(4):809-817, 2000.
3. Barker D: Preliminary observations on nutrient composition differences between adult and pinhead crickets, *Acheta domestica*. Bull Assoc Reptile Amphib 7(1):10-13, 1997.
4. Barker D, Fitzpatrick MP, Dierenfeld ES: Nutrient composition of selected whole invertebrates. Zoo Biol 17:123-134, 1998.
5. Bauck L, LaBonde J: Toxic diseases. *In* Altman RB, et al: Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 604-316.
6. Blalock TL, Hill CH: Studies on the role of iron in zinc toxicity in chicks. Biol Trace Element Res 17:17-29, 1988.
7. Bourke AM: Vitamin A toxicity in conures. AAV Newsletter 3-5, 1996.
8. Brice AT: Protein nutrition of captive hummingbirds. Proc 9th Dr Scholl Conf Nutr of Captive Wild Animals, 1991, pp 10-24.
9. Brice AT: The essentiality of arthropods in the diet of hummingbirds. Comp Biochem Physiol 101A(1):151-155, 1992.
10. Buttemer WA, et al: Time-budget estimates of avian energy expenditure: physiological and meteorological considerations. Physiol Zool 59:131-149, 1986.
11. Crosta, personal communication.
12. Doneley R: Zinc toxicity in caged and aviary birds - "new wire disease." Aust Vet Pract 22:6-11, 1992.
13. Dorrestein GM, et al: Zinc intoxication in two parrot flocks. Proc Deutsche Veterinärmedizinsche Gesellschaft e.V.125-133;2002
14. Drepper K, et al: Unteruschunen zum protein- aund energiebedarf adulter welesittche (*Melopsittacus undulatus*). Kleintierpraxis. 33, 1988.
15. Dumonceaux G, Harrison GJ: Toxins. *In* Ritchie BW, Harrison G J, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 1034-1052.
16. Frankel TL, Avram DS: Protein requirements of rainbow lorikeets, *Trichoglossus haematodus*. Aust J Zool 49:435-443, 2001.
17. Fu Z, et al: Retinoic acid accelerates the development of reproductive organs and egg production in Japanese quail (*Coturnix coturnix japonica*). Biol Reprod 63:1795-1800, 2000.
18. Grajal A: The nutritional ecology and digestive physiology of the hoatzin, *Opisthocomus hoazin*, a folivorous bird with foregut fermentation. PhD, Uni of Florida, 1991.
19. Grant B: Pollen digestion by Darwin's finches and its importance for early breeding. Ecology 77:489-499, 1996.
20. Gross KL, et al: Nutrients. *In* Hand MS, et al: Small Animal Clinical Nutrition. Kansas, Mark Morris Institute, 2000, pp 21-110.
21. Hanssen I, et al: Vitamin C deficiency in growing willow ptarmigan. J Nutr 109:2260-2278, 1979.
22. Harrison G, personal communication.

23. Hoffman DJ: Role of selenium toxicity and oxidative stress in aquatic birds. Aquat Toxicol 57:11-26, 2002.
24. Jenni L, Winkler R: Moult and Ageing of European Passerines. London, Academic Press, 1994.
25. Jianhua H, Ohtsuka A, Hayashi K: Selenium influences growth via thyroid status in broiler chickens. Br J Nutr 84:727-732, 2000.
26. Kalina J: Ecology and behavior of the black-and-white-casqued hornbill (*Bycanistes subcylkindricus subquadratus*) in Kibale Forest, Uganda. PhD. Michigan State Uni, 1988.
27. Kamphues J, Otte W, Wolf P: Effects of increasing protein intake on various parameters of nitrogen metabolism in grey parrots (*Psittacus erithacus erithacus*). *In* Kamphues J, Wolf P, Rabehl N: First Int Symp of Pet Bird Nutrition, 1997, pp 118.
28. King JR: Energetics of avian moult. Acta 17th Congr Int Orn Berlin, 1980, Vol. 1978, pp 312-317.
29. Klasing KC: Comparative Avian Nutrition. UK, CAB International, 1998.
30. Koutsos EA, Klasing KC: Vitamin A nutrition of cockatiels. Proc Joint Nutr Symp, Antwerp, Belgium, 2002, p 141.
31. Koutsos EA, Matson KD, Klasing KC: Nutrition of birds in the order Psittaciformes: A review. J Avian Med Surg 15:257-275, 2001.
32. Koutsos EA, et al: Adult cockatiels (*Nymphicus hollandicus*) metabolically adapt to high protein diets. J Nutr 131:2014-20, 2001.
33. Koutsos EA, et al: Adult cockatiels (*Nymphicus hollandicus*) at maintenance are more sensitive to diets containing excess vitamin A than to vitamin A deficient diets. J Nutr 2003 Jun; 133(6): 1898-1992.
34. Lindström Å, Visser GH, Daan S: The energetic cost of feather synthesis is proportional to basal metabolic rate. Physiol Zool 66:490-510, 1993.
35. Lowenstine LS: Nutritional disorders of birds. *In* Fowler MF (ed): Zoo Animal Medicine, Philadelphia, WB Saunders Co, 1986, pp 202-212.
36. Lü J, Combs GFJ: Excess dietary zinc decreases tissue α-tocopherol in chicks. J Nutrition 118:1349-1359, 1988.
37. MacMillen RE, Baudinette RV: Water economy of granivorous birds: Australian parrots. Funct Ecol 7:704-712, 1993.
38. MacWhirter P: Malnutrition. *In* Ritchie BW, Harrison GJ and Harrison LR (eds): Avian Medicine: Principles and Application, Lake Worth, Florida, Wingers Publishing, 1994, pp 842-861.
39. Massey DM, Selwood EHB: The amino-acid composition of budgerigar diet, tissues and carcass. Vet Rec 72:283-287, 1960.
40. Maynard LA, et al: Animal Nutrition 7th ed. New York, McGraw-Hill, 1979.
41. McDonald DL: Feeding Ecology and Nutrition of Australia Lorikeets. *Seminars in Avion and Exotic Pet Medicine; 12(4): 195-204; 2003*.
42. McDonald DL: Dietary considerations for iron storage disease in birds and implications for high vitamin A contents of formulated bird foods. Proc Joint Nutr Symp, Antwerp, Belgium, 2002, p 162.
43. McDonald DL: Evaluation of the use of organic formulated bird foods for large psittacines. Proc Joint Nutr Symp, Antwerp, Belgium, 2002, p 110.
44. McDonald DL: Failure of the wild population of the endangered Orange-bellied Parrot (*Neophema chrysogaster*) and implications for nutritional differences in indigenous and introduced food resources. Proc Aust Assoc Avian Vet, 2004.
45. McDonald DL, Nutritional status of the Australian long-billed corella (*Cacatua tenuirostris*) and sulphur-crested cockatoo (*Cacatua galerita*): an evaluation of hepatic and serum vitamin and mineral concentrations. Proc Aust Assoc Avian Vet, 2004.
46. McDowell LR: Vitamins in Animal and Human Nutrition. Ames, Iowa State University Press, 2000.
47. Meade J: Amazons. Proc Smokey Mountain Cage Bird Society Meeting, 1998.
48. Mezes M, et al: Nutritional metabolic diseases of poultry and disorders of the biological antioxidant defense system. Acta Vet Hung 45:349-360, 1997.
49. Murphy ME: Amino acid composition of avian eggs and tissues: Nutritional implications. J Avian Biol 25:27-38, 1994.
50. Murphy ME, King JR: Diurnal constancy of feather growth rates in white-crowned sparrows exposed to various photoperiods and feeding schedules during the postnuptial molt. Can J Zool 66:1403-1413, 1986.
51. Murphy ME, King JR: Nutritional aspects of avian molt. Acta 20th Congr Int Orn, Christchurch, pp 2186-2193, 1991.
52. Murphy ME, King JR: Energy and nutrient use during moult by white-crowned sparrows, *Zonotrichia leucophrys gambelii*. Ornis Scand 23:304-313, 1992.
53. Nichols DK, et al: Coagulopathy in pink-backed pelicans (*Pelecanus rufescens*) associated with hypervitaminosis E. J Zoo Wildl Med 20(1)57-61,1989.
54. NRC: Nutrient Requirements of Poultry. Washington, National Academy Press, 1994.
55. Osofsky A, et al: Determination of normal blood concentrations of lead, zinc, copper and iron in Hispaniolan Amazon parrots (*Amazona ventralis*). Avian Med Surg 15:31-36, 2001.
56. O'Toole D, Raisbeck MF: Experimentally induced selenosis of adult mallard ducks: Clinical signs, lesions, and toxicology. Vet Pathol 34(4):330-340, 1997.
57. Paton DC: The diet of the New Holland honeyeater, *Phylidonyris novaehollandiae*. Aust J Ecol 7:279-298, 1982.
58. Phalen D: Study determines safe diets for companion birds. Animal News Vol III, 2001.
59. Puschner B, St Leger J, Galey FD: Normal and toxic zinc concentrations in serum/plasma and liver of psittacines with respect to genus differences. J Vet Diagn Investig 11:522-527, 1999.
60. Pusztai A, Agota G, Bardos L: Effect of large dietary doses of beta-carotene on plasma retinoid and beta-carotene levels and on progesterone production in the granulosa cells of Japanese quail. Acta Vet Hung 48: 81-7, 2000.
61. Rabehl N: Untersuchungen zur Körperzusammensetzung und deren Entwicklung bei vershiedenen Ziervogelarten (Kanarien, Wellensittiche, Agaporniden, Nymphensittiche, Amazonen und Graupapagein). Hannover, Institut für Tierernährung der Tierärzlichen Hochschule, 1995.
62. Reece RL, Dickson DB, Burrowes PJ: Zinc toxicity (new wire disease) in aviary birds. Aust Vet J 63:199, 1986
63. Remington TE: Why do grouse have ceca? A test of the fiber digestion theory. J Exp Zool Suppl 3:87-94, 1989.
64. Roudybush TE, Grau CR: Lysine requirements of cockatiel chicks. Proc 34th West Poult Dis Conf, 1985, vol. 34, pp 113-115.
65. Roudybush TE, Grau CR: Food and water interrelations and the protein requirement for growth of an altricial bird, the cockatiel (*Nymphicus hollandicus*). J Nutr 116:552-559, 1986.
66. Sahin K, et al: Protective role of supplemental vitamin E and selenium on lipid peroxidation, vitamin E, vitamin A, and some mineral concentrations of Japanese quails reared under heat stress. Bio Trace Elem Res 85:59-70, 2002.
67. Schoemaker NJ, Lumeij JT, Beynen AC: Polyuria and polydipsia due to vitamin and mineral oversupplementation of the diet of a salmon crested cockatoo (*Cacatua moluccensis*) and a blue and gold macaw (*Ara ararauna*). Avian Pathol 26:201-209, 1997.
68. Scott ML: Environmental influences on ascorbic acid requirements in animals. Annals NY Acad Sci 258:151-155, 1975.
69. Scott ML, Nesheim MC, Young RJ: Nutrition of the Chicken. Ithaca, NY, 1982.
70. Sergio E, et al: Nutrition of the tamandua: I. Nutrient composition of termites (*Nasutitermes* spp.) and stomach contents from wild tamanduas (*Tamandua tetradactyla*). Proc 1st Ann Conf NAG, Toronto, 1995, pp 170-171.
71. Sheshukova T, et al: The adaptation of the digestive system to carbohydrates in experimental A hypervitaminosis. Fiziol Zh SSSR Im I M Sechenova 77:70-76, 1991.
72. Smith A: Toxicosis in a flock of Hispaniolan Amazons. Proc Conf Assoc Avian Vet, 1995, pp 447-453.
73. Somerville DC: Nutritional Value of Bee Collected Pollens. RIRDC, Victoria, 2001.
74. Spallholz JE, Hoffman DJ: Selenium toxicity: Cause and effects in aquatic birds. Aquat Toxicol 57:27-37, 2002.
75. Stobart, personal communication.
76. Stanford M: Calcium metabolism in grey parrots: The effects of husbandry. Proc Assoc Avian Vet, 2002.
77. Sunder A, Flachowsky G: Influence of high vitamin E dosages on retinol and carotenoid concentration in body tissues and eggs of laying hens. Arch Tierernahr 55:43-52, 2001.
78. Takeshita K, Graham DL, Silverman S: Hypervitaminosis D in baby macaws. Proc Assoc Avian Vet: 1986, pp 341-345.
79. Taylor EJ, Nott HMR, Earle KE: Dietary glycine: Its importance in growth and development of the budgerigar (*Melopsittacus undulatus*). J Nutr 124:2555S-2558S, 1994.
80. Tsiage VK, et al: Identification of volatile sulfur derivatives released from feathers of chicks fed diets with various levels of sulfur-containing amino acids. J Nutr 117:1859-1865, 1987.
81. Ullrey DE, et al: Limitations of plasma or serum analysis in assessing vitamin E status of domestic and wild animals. First Annu Conf Nutrition Advisory Group (NAG) of the Amer Zoo and Aquarium Assoc (AZA), 1995, pp 195-202.
82. Van Sant F: Zinc and parrots: More than you ever wanted to know. Proc Assoc Avian Vet, 1998, p 305.
83. Voitkevich AA: The Feathers and Plumage of Birds. London, Sidgwick and Jackson, 1966.
84. Witmer MC: Ecological and evolutionary implications of energy and protein requirements of avian frugivores eating sugary diets. Physiol Zool 71:599-610, 1998.
85. Wolf P: Handfeeding of young parrots. Techniques, diets and recommendations. Proc Joint Nutrition Symposium. Antwerp, Belgium, 2002, pp 113-136.
86. Wolf P, Kamphues J: Die futter- und wasseraufnahme bei kanarien - Einflußfaktoren und abhängigkeiten-. Kleintierpraxis 37:545-552, 1992.
87. Wolf P, Wendler C, Kamphues J: Untersuchungen zur Zusammensetzung handelsüblicher Mauserpräparate für Ziervögel. Kleintierpraxis 41:669-670, 1996.
88. Wolf P, et al: Vitamin A metabolism in recessive white canaries. Animal Welfare 9(2):153-165, 2000.
89. Wooller R, Richardson K, Pagendham C: The digestion of pollen by some Australian birds. Aust J Zool 36:357-362, 1988.

Nutritional Considerations Section II

Nutritional Disorders

GREG J. HARRISON, DVM, Dipl ABVP-Avian, Dipl ECAMS
DEBRA McDONALD, PhD, BSc (HONS I)

Nutritional disorders can result from malabsorption, a deficient diet, over-supplementation and/or overeating. Deficiencies and excesses of nutrients can both be harmful to birds.

Companion birds have been maintained for decades on diets that, while nutritionally inadequate, support limited breeding in a few species. While there are numerous publications regarding nutritional requirements of agricultural species, captive passerine energetics and feeding ecology, there are few controlled scientific studies on aviary and companion birds or their wild conspecifics. Variations in lifestyle and breeding ecology result in differing nutritional requirements. Clinically, many health problems are correlated with nutritional disorders. This chapter will provide an overview of these conditions observed in companion birds, with reference to anecdotal observations in a clinical context and summaries of nutrient implications that have been predominantly studied in agricultural species. Specific studies of companion and wild birds will be discussed. Parallels may exist between the following description of the improper diet cascade and the metabolic syndrome of humans and rats.[90b]

The Improper Diet Cascade (IDC)

The 'improper diet cascade' (IDC) (Table 4.2.1) has been postulated by the author (GJH) from decades of clinical experience, reports from pathologists and nutritionists, as well as consultations with companies that produce commercially formulated diets. The IDC expresses itself in a highly individualistic fashion. The most common thread is the history of a basic seed and table food diet. Generally, at presentation of a "sick" bird, the IDC patient exhibits pansystemic clinical signs that often include various behavioral problems. Typically though, the earliest clinical signs are reflected in the integument, followed closely by the digestive system. Often birds are not presented for evaluation until the reproductive or respiratory system is affected. Behavioral problems can be the proximal cause of veterinary presentation when other clinical signs have been missed or ignored.

The IDC can be initiated from a nutrient imbalanced diet as well as from influences, such as improper husbandry, diet handling and storage or over-supplementation of nutrients in formulated diets. Therefore, when evaluating nutritional disorders, consider the composition of the diet eaten, as well as the stability or availability of nutrients in that diet. Pathological influences such as parasite infestation, metal toxicoses, malabsorption syndromes, pancreatitis and gastroenteritis produce clinical signs similar to those seen in IDC, and therefore need to be ruled out (Table 4.2.2a).

The IDC is the result of improper nutrient utilization, usually from malnutrition that weakens the body immunologically and structurally. This can allow invasion of low level pathogens or commensals of viral, bacterial, or fungal origin.

Recent research by Dr. M. Beck, University of North Carolina[15], showed that when the host is affected by a nutritional deficiency, the invading pathogen is affected as well. By sequencing the viral isolates recovered from selenium-deficient mice, she demonstrated mutations in the viral genome associated with increased pathogenesis of the virus affected by nutrient deficiency. Bhaskaram

Table 4.2.1 | Improper Diet Cascade (IDC)

Nutritional Imbalance

⇩

MULTISYSTEMIC ABNORMALITIES

Cellular	Structural	Functional	Immunologic
Impaired metabolism	Metaplasia of columnar epithelium	Goblet cells mucin production impaired	Commensal organisms normally bound to mucus are not excreted
Altered cell wall permeability	Increased mucous viscosity	Loss of cleansing ability of mucous	Relationship with commensal organisms disrupted
Cellular autointoxication Change in GI pH (less acidic)	Loss of normal collagen elasticity	Normal glandular production of various systems suppressed	Bone marrow suppression Decreased IgA, decreased lymphocytes
Chronic: eg, • Hepatic lipidosis, fibrosis, cirrhosis • Iron storage disease • Irreversible degradation of retinal cones leading to blindness	Chronic: eg, • Abnormal cilia • Renal tubular nephrosis • Follicular atresia • Cataract formation • Bone/muscle abnormalities	Chronic: eg, • Diabetes mellitus • Deposits of high density lipids in vasculature • Exocrine pancreatic insufficiency • Infertility, decreased hatchability of chicks • Secondary hyperparathyroidism	Chronic: eg, • Secondary microbial infections • Increased susceptibility to neoplasia

⇩

ABNORMALITIES OF SPECIFIC SYSTEMS

Integument	Gastrointestinal	Respiratory	Renal	Endocrine	Reproductive	Cardiovascular
• Skin • Feathers • Beak • Nails • Fat deposits	• Oropharyngeal • Pancreatic • Hepatic • Intestinal	• Nares • Infraorbital sinus • Syrinx • Air sacs	• Glomeruli • Renal tubules • Ureters • Urodeum	• Pancreatic • Thyroid • Parathyroids • Intestinal • Gonadal	• Ovarian • Uterovaginal • Testicular • Cloacal • Egg abnormalities	• Vasculature • Myocardium • Air capillaries • Pericardium

Biochemical	Hematological	Behavioral
• AST, ALT • Bile acid • Glucose • HDL, LDL, Triglycerides • Cytokines	• Increased WBC • Altered total WBC	(see subsequent section)

Table 4.2.2a | Commonly Encountered Etiologies of Improper Nutrient Intake or Utilization

Congenital Developmental	Individual	Complicating Factors	Rule outs that impair digestion and/or absorption
Improper parental diet	Provision of improper diet	Little or no sunlight	Pancreatitis or organ failure
Improper handfeeding diet	Consumption of improper diet	Lack of bathing	Malabsorption syndromes
Weaned to improper diet	Improper diet supplementation	Lack of exercise	Viral, bacterial, fungal, or parasitic gastroenteritis
Diet constituents interfere with nutrient utilization	Improper food packaging/handling or storage		Metal toxicosis

expanded this theory by showing that several micronutrients such as vitamin A, ß-carotene, folic acid, vitamin B_{12}, vitamin C, riboflavin, iron and selenium could be involved in such a scenario in humans.[17] These micronutrient-compromised viruses can lead to the emergence of new infections.[17] This hypothesis was further advanced by Lavender[61], who showed that, at least for RNA viruses, host nutrient deficiencies and excesses can influence the genetic make-up of the pathogen. The majority of viruses are RNA viruses.[61]

The importation of wild caught psittacines has traditionally involved weeks to months of stress including severe nutrient imbalance. Such birds imported into the USA in the 1970s and 1980s were a part of a pandemic of new viral diseases. Psittacine beak and feather disease, proventricular dilatation disease and papillomatosis are three that still plague us. The research community has not adequately addressed the role of malnutrition in viral pathogenesis. It is interesting to ponder this hypothesis in light of the new expressions of these same viruses occurring in the European Union countries that still import wild-caught birds.

IMPROPER DIET FORMULATION

There is a general perception that 'fresh' is best. However, presenting a bird with an array of fresh produce, seeds and nuts does not necessarily provide a nutritionally balanced diet. Commonly fed seeds are deficient in a number of nutrients (Table 4.2.2b). Much of the produce is sold in its immature state of growth, and even when mature, it does not have the equivalent nutrient profiles of wild food items. Thus such produce is unable to improve the nutrient profile of the diet.

It is imperative that bird owners be informed of the nutritional inadequacies of such diets. In the wild, psittacines usually balance their diets by feeding on a variety of seeds and other plant parts. Primary issues of concern with captive diets are vitamin levels (vitamins A, D, E, and K and the water-soluble vitamins—biotin and B_{12}) and minerals. Seeds do not contain vitamin A and are generally low in the vitamin A precursor ß-carotene. Hypovitaminosis A is particularly prevalent in birds on all-seed diets. Mineral levels of seeds can vary among plant species as well as geographically, depending on the composition of the parent soil. Calcium is deficient in most seeds and, while adequate phosphorous may appear to be present, up to 70% may exist in phytate form that is generally indigestible. Fatty acid composition will also vary among seed species and an imbalance can be an important cause of a number of health issues. Many seeds provide adequate total protein but do not contain the complete set of essential amino acids. A diet of predominantly millet seed will result in a lysine deficiency not seen on other seed-based diets. The composition of commercially raised seeds differs dramatically from wild seeds (see Section I of this Chapter).

Table 4.2.2b | Nutrient Deficiencies of Seeds
The seeds most commonly fed birds, such as oats, corn, sunflower, safflower and millet, are generally missing 32 ingredients (from eight groups) needed to keep birds healthy. These include:

- **Vitamins** - choline, niacin, pantothenic acid, riboflavin (B_2), cyanocobalamine (B_{12}), biotin (H), D_3, E, K, and folic acid (M)
- **Minerals** - calcium, phosphorous (70% tied up as non-digestible phytates in plant products, such as grains), sodium
- **Trace minerals** - selenium, iron, copper, zinc, manganese, iodine, chromium, vanadium, bismuth, tin, boron
- **Pigments** - chlorophyll, canthaxanthin
- **Protein** - (amino acids) lysine, methionine
- **Fiber** - (mucopolysaccharide) both soluble and insoluble
- **Vitamin precursors** - ß-carotene, converted to vitamin A in liver
- **Omega 3 Fatty Acids**

Birds do not exhibit nutritional wisdom when selecting dietary ingredients; they show a preference for high-energy, lipid-rich seeds, high carbohydrate seeds and fruits. The advent of formulated foods has diminished the incidence of nutritional disorders in the author's (GJH) practice. Yet not all formulated diets are created equal (Tables 4.2.2c-e). For example, products that offer the opportunity for selecting favored food items are poorly formulated and can be just as imbalanced as a seed-based diet in the end.

The Association of Avian Veterinarians (AAV) formed a committee of nutrition experts who developed a list of recommendations to assist veterinarians and owners in feeding pet birds (Table 4.2.2f).

While some essential nutrients are higher in organically certified plant products, a diet composed solely of organic seeds will present as many nutritional problems as a diet solely composed of non-organic seeds.

There are also the issues of diminished availability of some nutrients by interference from other nutrients and potential breakdown of key nutrients.

OVER-SUPPLEMENTATION

Vitamin toxicity is an aspect of dietary management that is frequently overlooked, but can be responsible for a number of clinical signs of a disease. Many commercially formulated products contain excessive levels of the fat-soluble vitamins A and D. The addition of vitamin supplements with high concentrations of these two vitamins compounds that excess. The generally low levels of

Table 4.2.2c | Provision of Improper Diet - Common Presentations

Excessive quantity of seeds or nuts provided (minimal vitamin A precursors, lysine deficient, decreased vitamin E absorption, inverted Ca:P ratio, excessive calories)	Excessive percentage of fruits and vegetables (deficient in essential amino acids and essential fatty acids, contain excessive sucrose) *Nutritional deficiencies vary widely between fruits and vegetables - see Figs 4.1.2, 4.1.3 and Tables 4.1.8, 4.1.9 in section 1)	Excessive quantity of "table foods" such as the carbohydrate rich pastas and breads (in addition to the aforementioned deficiencies, these provide a medium for yeast overgrowth in susceptible individuals)	Improper/excessive vitamin-mineral supplementation	
			Potential toxicities eg, vitamin A,D_3, iron, selenium	Competitive nutrient absorption, eg, excessive fatty acids, phytates, and fat soluble vitamins

Table 4.2.2d | Consumption of Improper Diet - Common Presentations

Formulated diet over-supplemented with vitamins (vitamin A) or minerals (iron). Deficiencies: lysine, L-carnitine	Diet provided requires bird to consume all components to achieve balance	Supplements needed to balance diet are provided as a coating on food that is not entirely consumed

Table 4.2.2e | Preparation, Packaging and Storage Problems of Formulated Diets

Problems in Preparation	Packaging Concerns	Improper Storage
Inclusion of raw soybeans, oats or brown rice. Cooking soybeans improves the availability of methionine & cystine[14b] & destroys trypsin inhibitors. Oats & brown rice are high in lipase [break down fats to free fatty acids & lipoxygenase (oxidizes fatty acids to hydroperoxides)][43b]	Use of oxygen-permeable packaging Oxidation → Rancidity	Continued mycotoxin production
Inclusion of mycotoxin producing agents	Exposure to light	
Poor quality control	Insect contamination	
Over cooking → degradation of nutrients and conversion of *cis* to *trans* fatty acids	Pesticide contamination	Insect infestation (eg, transmission of Sarcocystosis)
Addition of artificial coloring/dyes long term effects unknown	Soft plastics may act as phytoestrogens	Degradation of nutrients
Preservatives (such as ethoxyquin) may be toxic or teratogenic. However, in the absence of preservatives, proper packaging and storage are imperative to maintain quality and prevent rancidity.		

vitamin E in both commercial diets and vitamin supplements may exacerbate toxicity. Dietary supplementation should be undertaken only if there is an extensive knowledge of the nutrient composition of both the diet and the supplement. The common clinical practice of injecting vitamins into sick birds may not be defensible, especially if the bird has been on a formulated and/or supplemented diet. See Section 1, Nutrition and Dietary Supplementation for a more in-depth discussion.

RANCIDITY

Altering tissue structure mechanically (hulling, grinding, and crushing in the case of vegetable matter or maceration in the case of animal tissue) releases lipases.

Grains damaged at harvest also allow this lipase release to occur. Similarly, micro-organisms (fungal contaminants) contain lipases that cause hydrolysis of fats.[43b] So quality control of source products is essential. The exposure to oxygen, moisture and heat act with the catalysts naturally present in grains (iron, copper) to accelerate the deterioration process at all stages of grain handling and product manufacturing.

These lipolytic enzymes act on lipids to release free fatty acids and triglycerides. In the presence of oxygen, heat and moisture, these fatty acids and triglycerides are autooxidized or acted upon by enzymes (primarily stored in the germ) called lipoxygenases. Polyunsaturated fatty acids (oleic, linoleic, and linolenic) are the most likely to be oxidized, and they are usually the most abundant fatty acids in nuts and seeds.[43b] This oxidation process produces free radicals in a dark environment. A similar but slightly different reaction occurs when exposed to light. Both reactions end with the production of lipid hydroperoxides which further break down, causing rancidity. This process is often self perpetuating, starting

Table 4.2.2f | AAV Feeding Brochure

Association of Avian Veterinarians
Feeding Recommendations

FEEDING COMPANION BIRDS
Feeding of companion birds has been one of the most challenging aspects of their care, primarily because of limited nutritional research on all species. However, based on studies of poultry and other animals, generalizations can be made on adequate feeding practices for companion birds.

FORMULATED DIETS
Formulated bird food products are available from the pet food industry as a convenience to the owner and to ensure a more nutritionally balanced diet than that offered by seeds alone. The current trend is toward specific formulations addressing age, activity, therapeutic, and stress-related needs of the bird. For example, birds have special nutritional needs during molting, egg laying, or raising young. However, improving a diet in the short term in anticipation of these life stages is not effective; the feeding practices must be optimal year round.
Commercial bird food products may be purchased as pellets, nuggets, crumbles, or hand feeding premixes. Converting a seed-eating bird to a formulated diet must be done with care because new items in the cage may not be immediately recognized as food. Your veterinarian can recommend a commercial formulated bird diet and help you with the conversion process.

ALTERNATIVE HOMEMADE DIETS
Where commercial diets are not available, attempts are made to produce a homemade diet. While not ideal for pet birds, these usually offer an improvement over an exclusive seed diet. Overall, however, homemade diets are often lacking in calcium, iodine, selenium, protein, fatty acid balance, fiber, pigments, and vitamins A, B complex, E, and D_3 while providing an excess of carbohydrates, and phosphorus. Additionally, homemade diets with moist ingredients tend to spoil easily and lose nutrients if not stored properly or if made too far in advance of feeding. The time and effort involved in preparing foods and the difficulty in balancing the nutrients make homemade diets impractical for the pet bird owner. Owners choosing a fresh food plan tend to offer too much variety and quantity of food each day, permitting birds to pick out what they like. Birds will not choose a balanced diet if given free choice. Consult your avian veterinarian for specific recommendations on items and quantities to feed.

FRESH WATER
Fresh water must be provided at all times. Some aviculturists and companion bird owners have had success using pet water bottles for birds, thereby limiting soiling of water.

FEEDING TIPS
- Carefully monitor TOTAL food consumption during any diet change.
- Introduce small amounts of a new food at a time.
- Gradually reduce the total volume of seeds as you increase the volume of more nutritional foods.
- Clean all food and water cups and remove old food from the cage daily.
- Do not provide supplemental vitamins unless recommended by your avian veterinarian.

BEHAVIORAL ENRICHMENT
A consistent daily feeding program contributes to physical and mental health as much as a varied diet. The availability of natural items such as branches, empty nutshells, leather pieces and coconut shells create a stimulating environment.

GRIT
Grit is small non-dissolvable rock. The necessity of grit in the diet is debatable. Some birds, such as pigeons, fowl, canaries and finches, appear to need the availability of grit. In psittacine species, an occasional grit particle is harmless but it is not necessary for healthy maintenance of pet parrots, macaws, parakeets and similar species.

SALT
Salt licks are not necessary for birds.

DEPRAVED EATING HABITS
Birds that routinely eat inappropriate materials (eg, feces, enclosure substrate) should be examined by a veterinarian. This behavior may be associated with disease or nutritionally deficient diets and is often prevented by the feeding of a more balanced formulated food product.

SPECIAL REQUIREMENTS
Lories and lorikeets require specialized diets in captivity. These nectar diets attract insects and result in liquid and messy feces. Your avian veterinarian can recommend a diet for these species. Soft-billed birds, waterfowl, backyard poultry and gamebirds Commercial foods are available for these birds. Some toucans and mynahs may have a special dietary requirement for a low-iron formula. Consult your avian veterinarian for recommendations.

© Association of Avian Veterinarians 2002
Used with permission

slowly and increasing rapidly as reaction chemicals become available.

Expressing the oil from seeds increases the surface area being exposed to oxygen, which can increase the possibility of rancidity occurring.

The production of lipid hydroperoxides does not appear to alter flavor. Lipid hydroperoxides deteriorate to aldehydes in the presence of oxygen.[43b] These do alter flavor and finally palatability. Alcohols and hydrocarbons are also produced. These latter products have been reported to be mutagenic.[16] Rancid fats can lead to selenium and vitamin E deficiencies implicated in encephalomalacia, pancreatitis, myocardial necrosis, hepatic necrosis and general myopathy. Biochemical analysis of affected birds' blood may show anemia, elevated lactate dehydrogenase (LDH), aspartate aminotransferase (AST), creatinine phosphate (CK) and phosphorous levels. Many of these clinical conditions are not reversible.[98]

Chickens fed diets with increased rancidity parameters (peroxide and aldehyde concentrations) experienced increased mortality from fatty liver syndrome (FLS). Total blood proteins of affected chickens were elevated, as were lipoproteins and total lipids.[28]

HANDLING AND STORAGE

Wild birds naturally feed on an array of fresh foods while their captive counterparts are provided with foods that have been stored for extended periods. Nutritionally imbalanced food supplies are not uncommon in wild situations. Agriculture produces seeds and nuts only at the end of the growing season, usually in the fall. Storage increases the potential for nutrient degradation. Nitrogen flushing and storage under refrigeration are steps that discourage oxidation.

COLD DARK STORAGE HELPS PREVENT RANCIDITY

Storing walnuts in the light at 21° C resulted in profound oxidative changes.[51] However, walnuts stored in the dark at 5° C for 25 weeks, even in 50% oxygen, were without a trace of rancid taste.[51] However, it should be remembered that rancidity, as determined by chemical analysis, precedes taste detection.

Storing corn oil at room temperature for 48 months resulted in rancid oil, whereas storage in the refrigerator did not.[16] A specific strain of mice fed the rancid corn oil showed significantly increased expression of oncogenes in all major organs. The results demonstrated that rancid oils, rich in *n*-6 polyunsaturated fatty acids, could initiate tumors and promote tumor growth.[86]

COOKING

In the preparation of a formulated diet, cooking (roasting, pelletizing or extrusion) is designed to stabilize oils. However, depending on the condition of the products being mixed, some combinations may cause flash rancidity (D. Jones, personal communication 2000). This is due to the presence of enzymes in items like grains and peanuts that cause natural fermentation when exposed to warm moist air. The lipase concentration in some grains is very high. Oats and brown rice are examples.[43b] Dehulling and milling these products causes rapid deterioration (rancidity) unless they are heat stabilized prior to storage or further processing. When these raw products are combined under the heat of processing, this flash rancidity can occur. For this reason, these ingredients need to be roasted or other wise partially cooked separately, then mixed with the other ingredients prior to final processing.

Raw soybeans contain trypsin inhibitors and can therefore be difficult to digest. This enzyme is a critical part of digestion in monogastric animals. Trypsin inhibitors are inactivated by heat.[14b,70b] Cooking also improves the availability of methionine and cysteine.[14b] Overcooking destroys or makes unavailable certain amino acids (lysine) and greatly reduces natural vitamin precursors such as tocopherols and carotenoids.

MOISTURE CONTENT

Lowering the moisture content of a product also acts as a stabilizer. Moisture plays a vital chemical role in most oxidation processes.[43b] Levels below 5% are often required to deter degradation. The author (GIH) has shown that these low moisture levels cause minor proventricular irritation evidenced by excessive regurgitation and minor weight loss in some pet umbrella cockatoos. Even at these low moisture levels, over time non-free 'water' is all lipases need to act. Non-free water cannot be removed by drying.[43b]

PACKAGING

Many bird foods are packaged in plastic, cellophane, coated paper or cardboard boxes. The latter two prevent exposure to light. Airtight containers (plastic, cellophane) prevent moisture from evaporating, but many do not stop oxygen from crossing into the food. The oxygen then breaks down essential nutrients or changes their biological activity. An advertised vitamin A content of 12,500 IU/kg may be reduced to as few as 1,500 IU/kg by inadequate packaging, with further deterioration once the package is opened. Even if one starts with a nutritionally sound, preservative-free formulated diet, the lack of proper packaging and resulting rancidity cancel its effectiveness.

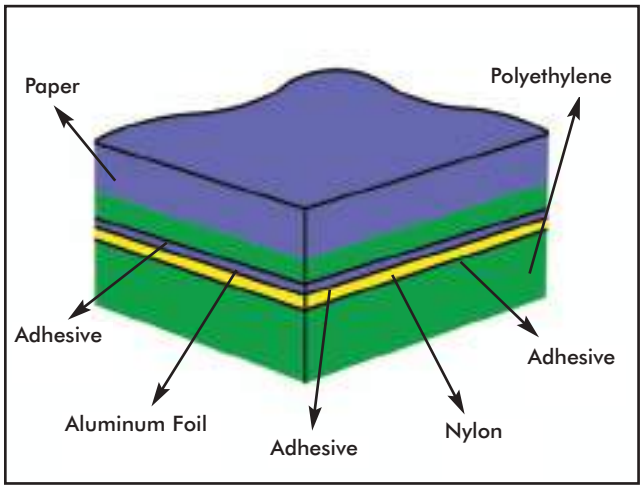


Fig 4.2.1 | Quadruple laminate packaging helps preserve the freshness of fomulate diets and prevents rancidity.

Fig 4.2.2 | A red-lored Amazon fed a seed and table food diet has an overgrown maxillary rhamphotheca that has been recently honed down to a more normal shape.

Fig 4.2.3a | A green-winged macaw fed a seed and table food diet. The red feathers are almost pink. Black pigment is co-mingled with green. The beak is hyperkeratotic. The breast and wing feathers are tattered and picked.

Fig 4.2.3b | A yellow-naped Amazon fed a seed and table food diet. The bird is obese, the maxillary rhamphotheca is overgrown and the feathers are abnormally pale green. The rectrices are tattered. Structural abnormalities make the coverts of the wing and body contour feathers lack the homogeneous interlocking appearance of a normal bird.

To avoid oxygen deterioration, chemical preservatives like ethoxyquin (originally used to soften rubber, later as a herbicide) and propylene glycol have been used for decades in dry animal foods. They have not been deemed safe for human foods. Recent public demand for more natural pet foods has led to a variety of newer techniques to avoid rancidity.

Lipid peroxidation can particularly affect products composed of organic ingredients that lack synthetic preservatives but is no less an issue for any products that are inadequately packaged.

For these reasons, all foods need to be smelled when first opened. If they smell like old frying grease or linseed oil they are rancid. A taste test should be observed when first offering a new bag of food to the bird. If the bird acts hungry but rejects the food it might be rancid. Rancid foods should not be fed. Following the manufacture's directions for handling the food and shelf life will usually prevent rancidity problems.

Table 4.2.3 | IDC and the Integument

Nutritional Imbalance

⇩

CLINICAL SIGNS

Feathers	Skin	Beak and Nails
• Frequent or incomplete molts • Retained feather sheaths • Abnormal coloration • Irritability due to feather discomfort • Deformities: - Cysts - Loss of elasticity - Barbules not interlocking • Chronic/Severe - Feather destructive behavior - Self-mutilation	• Flaky • Dry • Pruritic • Hyperkeratotic • Loss of elasticity (tears readily) • Plantar surface - loss of pattern • Chronic/Severe - Pododermatitis	• Excessive length • Exaggerated curvature • Friable texture • Splitting • Bruise readily • Chronic/Severe - Marked deformity - Secondary infections

INITIAL TREATMENT

Husbandry	Medical	Procedural	Diet Conversion
• Increase UVB • Increase outdoor exposure: - Humidity - Ventilation - Sunlight - Psychological stimulation • Improve available perches, increased variety of sizes and textures • Increase exercise, both physical and mental • Verify or improve hygiene	• Antipruritics: - Systemic - Topical • Treat secondary infection if present and significant • Psychotropic medications if self-mutilating • Parenteral vitamin supplementation for severe deficiencies • Blood work and other diagnostics if indicated	• Trim overgrown beak • Trim overgrown nails • Remove damaged feathers • Trim rachis of feathers if irritating bird • Mechanical barrier to self-mutilation if needed • Medicated padding and or bandaging for pododermatitis when present	• Evaluate for the following: - Essential amino acids - Balanced fat and CHO - Vitamins at physiologic levels (not excessive or deficient) - Free of dyes and preservatives - Need for supplemental essential fatty acids • Formulated diet is often most convenient and effective • Monitor weight during conversion

EVALUATION OF THERAPY

- Dietary conversion is necessary for long-term treatment and control.
- Anticipate exacerbation of clinical signs for 3-9 months (pruritus, flaking of skin and molting) with integumentary regeneration.
- If clinical signs worsen, perform diagnostic work-up for systemic sequelae to IDC.
- At 9-12 months, marked improvement should be noted in initial clinical signs.

There are few natural oxidative inhibitors. Tocopherols (vitamin E) and rosemary leaves have been tried. In the author's experience, preliminary studies of products containing rosemary had less than ideal acceptance, and the test subjects' had lower than desired body weights.

The natural antioxidants found in whole cereal grains have not been fully exploited.

The development of quadruple laminate bags (Fig 4.2.1), consisting of a layer of poly-coated extruded paper (blocking light), a layer of nylon for puncture resistance, a metal alloy as a barrier to oxygen and a polyethylene layer to resist changes in moisture and retain oils, have increased shell life of non-synthetically preserved products by up to 14 months. However, once the seal is broken and exposure to oxygen and moisture increases, these products are only viable for up to six weeks before clinical signs produced in birds resemble those of birds maintained on diets depicted in Figs 4.2.2-4.2.3a,b. It is important that clients adhere to the manufacturers'

Fig 4.2.4 | A blue and gold macaw that was fed a diet of pasta, crackers, cookies, pellets and vegetables. The feathers are tattered and lack symmetry. The blue feathers contain a black pigment. Under the contour body feathers, the bird had an excessive number of pinfeathers.

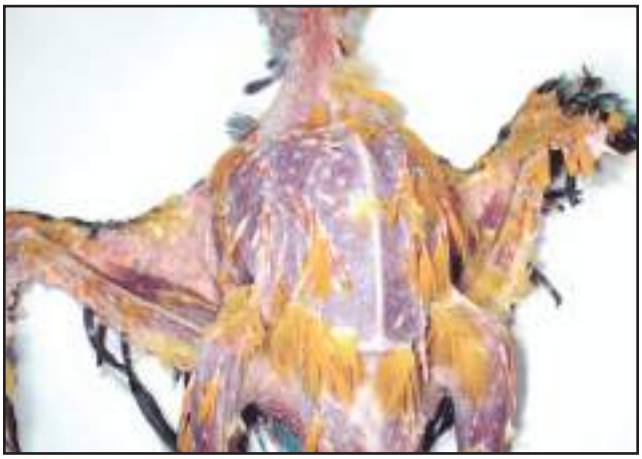


Fig 4.2.5 | This blue and gold macaw hen died after laying a clutch of 5 infertile eggs. Note the pinfeathers after all the body and extremity feathers were removed. Also note the black pigment in the normally blue feathers. The bird had been fed a seed and table food diet.

Table 4.2.4 | Using a Formulated Diet

Shelf Life	Use within 4-6 weeks of opening*
Storage	Store in manufacturer's packaging only if adequate**
	Express air to minimize oxidation
Feeding Frequency	Offer fresh food 2-3 times daily dependent on species
Feeding Amount	Ensure bird eats all food offered, including crumbs. Amount fed should maintain normal body weight.
Selective Feeding	Don't allow bird to favor individual particles
Supplementation	Follow manufacturer's instructions as to types and amounts of supplementary foods
Water	Don't allow birds to dunk food in water as this degrades vitamins and pollutes water leading to bacterial and fungal overgrowth

*Diets composed of nonorganic ingredients may have a longer shelf life due to synthetic preservatives.
**Many products are packaged in inferior packaging resulting in breakdown of key nutrients before packaging is even opened. Quadruple laminate packaging preserves nutrients for extended periods of time.

storage directions, as even nutritionally adequate diets have a limited shelf life once opened (see Table 4.2.4).

The IDC from a Systemic Point of View

Although birds seldom present with only one system affected by improper diet cascade, diagnosis, treatment and prevention are best discussed by looking at a single system at a time.

Early recognition by the clinician of the effects of IDC on various systems allows diagnosis and implementation of dietary therapy. This is a key element in avian preventive health care.

INTEGUMENTARY SYSTEM

The integument is the site where clinical signs of dietary inadequacy often appear to be noticed first, but these early stages are so commonly encountered that they may not be perceived as abnormal (Table 4.2.3). The stratified squamous epithelial (SSE) cells characteristic of skin are involved in the production of integumentary components such as the nails, beak, feathers, and feather follicles. In addition to the integument, SSE cells are found in the rhinal cavity, mouth, salivary duct junctions, tear ducts, ear canal, syrinx, air sac junctions to the lungs, bile duct, pancreatic duct, cloaca, renal tubules and vagina. Nutritional imbalance can influence the structure and function of any of these sites. While nutritional inadequacies are most often manifested in the integument, the clinical presentation can be complicated by more serious underlying illnesses. The development of nutritionally balanced formulated diets has dramatically reduced the incidence of dermal disorders, but such diets are far from successful in totally eliminating these problems once they have developed.

The Physical Exam Form outlined in Chapter 6, Maximizing Information from the Physical Examination, is a useful tool for identifying signs and common clinical presentations listed in Table 4.2.3. Minor integumentary signs are often overlooked by the bird care industry. It is important to establish a program of wellness with regular checkups, especially for new birds, to identify problems with nutritional inadequacies at an early stage.

KERATINIZATION

Hyperkeratosis is characterized by failure of the new cells to differentiate beyond the squamous stage.

Table 4.2.5 | IDC and the Digestive System

Nutritional Imbalance→ Pathology of gastrointestinal organs, liver, pancreas			
⇩			
CLINICAL SIGNS			
Body Condition	**Behavior**	**Flora**	**Digestive**
• Obesity • Loss of muscle mass • Bleeding • Chronic - Emaciation - Fatty liver - Cirrhosis - Hemochromatosis	• Regurgitation • Vomiting • Loss of appetite • Listlessness • Aggression	• Total # gram-positives decrease • Lower % gram-positive rods • Gram-negative bacteria increase • Yeast not budding • Chronic - Gram-negatives predominate - Budding yeast - Enterotoxemia (gram-positives explode) - Clostridial overgrowth	• Bilestained urine, urates and stool • Occult blood in stool • Undigested food and fiber in stool = pancreatic failure • Liver shadow increases or decreases • Chronic - Ileus - Diarrhea
TREATMENT			
Diagnostics for Secondary Infections	**Medical Treatment**	**Environmental Concerns**	**Dietary Conversion**
• Endoscopy and organ biopsy • Culture and sensitivity • Radiology • Hematology • Biochemistry • Ultrasound	• Fluids • Systemic treatment of secondary infections • GI stimulants • Bacteria, enzyme replacement • Lactulose • Milk thistle • SAMe • Apple cider vinegar • Chronic - Ultra clear®f - Hepasan®e	• Heat • Proper humidity	• Same as Table 4.2.3 • Formulated diet is often most convenient and effective

Dysfunctional, excessively keratinized cells replace normal cells. This can result in epithelial lesions and an increased susceptibility to infection. If the imbalance is severe and prolonged, columnar epithelium undergoes metaplasia to SSE. Keratinization can result in a loss of function of the tissues involved, including those of the alimentary, reproductive, respiratory and urinary tracts.

Clinical signs of hyperkeratosis involving the integumentary system can manifest as overgrowth of the beak and nails, which retain their outer covering due to a proliferation of basal cells. The keratinized outer coatings of pinfeathers are thicker, less flexible and retained much longer than normal. Retained coatings prevent pinfeathers from opening and such feathers appear to be painful to the birds if the unopened feathers are manipulated. Clients commonly report that birds with chronically retained pin feathers are irritable and vocalize as if in pain during preening (Figs 4.2.4 and 4.2.5).

While hyperkeratosis is generally associated with dietary deficiencies of vitamin A, excesses of vitamin A are also correlated with hyperkeratosis. The percent of squamous cells present in nasal flushes has been used as an indicator of vitamin A toxicosis.[58] It is important to obtain a full dietary history before prescribing vitamin A supplementation to treat hyperkeratosis. In rodents, oral supplementation with vitamin A failed to raise serum vitamin A levels in the absence of adequate vitamin E.[6] Therefore a mixture of both vitamin E and vitamin A may be required to treat hyperkeratosis due to a vitamin A deficiency. Deficiencies of zinc and biotin have been associated with hyperkeratosis. Biotin deficiencies, which can result from excess of salt, are correlated with hyperkeratosis on the footpad and the plantar surfaces of the toes.[7] Thus the caveat to not treat all hyperkeratosis with vitamin A injections is valid.

GASTROINTESTINAL SYSTEM

Secondary to the dermal system (and some behavioral traits), the avian clinician is likely to observe gastrointestinal tract (GIT) dysfunction next in the unfolding of the IDC (Table 4.2.5). Vitamin A deficiency may interfere with normal growth, rate by influencing functionality of the small intestine by altering the proliferation and maturation of cells of the intestinal mucosa.[104] Hyperproliferation of enterocytes, decreased number of goblet cells, decreased alkaline phosphatase activity, and decreased expression of brush-border enzymes are all correlated with vitamin A deficiencies.[104]

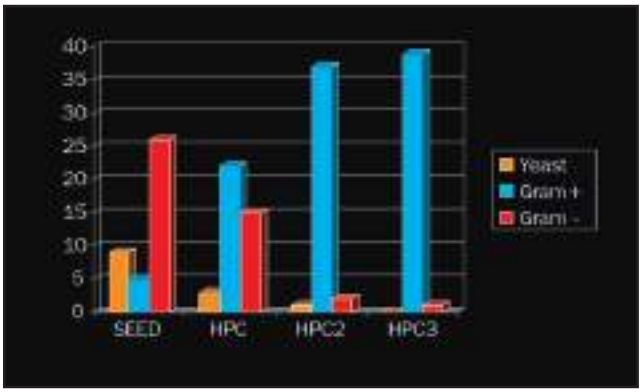


Fig 4.2.6 | Improvement of fecal Gram's stains over time with only the use of an organic formulated diet.[102]
Key:
Seed = Initial assessment of 100 birds after 12 months of eating a seed-based diet
HPC = Samples collected 4 weeks after changing 80 birds to Harrison's High Potency Coarse (HPC) pelleted formulation
HPC2 = Samples collected from same birds after 8 weeks on HPC
HPC3 = Samples collected from same birds after 12 weeks on HPC

The Fecal Gram's Stain in Psittacines

While early studies of captive birds indicate that they commonly have low levels of gram-negative bacteria in cultures of feces,[5,9,18,21,54,99] other researchers maintain that autochthonous flora in healthy parrots are not gram-negative.[37,38,45,46,47,102] Normal fecal flora of psittacines is comprised of 100% gram-positive, non-spore forming rods and cocci.[37,38,45,46,47,102] One study of wild yellow-naped Amazon chicks showed 60% of cloacal cultures had Enterobacteriacea.[99] The author (GJH) has hypothesized that this group of nesting birds were under undue stress from poachers, the presence of humans guarding the nests and a declining natural environment. For 20 years, this author (GJH) has used fecal Gram's stains to evaluate the normal flora of pet birds.[46] Studies of wild psittacines in a recent trial confirm the absence of gram-negative bacteria.[45,47] Gram-negative bacteria were reduced to almost zero after conversion of African grey parrots from a typical seed-based diet to a nutritionally balanced one.[102] (Fig 4.2.6) Glunder found (*E. coli* or *Klebsiella* spp.) it nearly impossible to colonize in the intestine of budgerigars on nutritionally balanced diets.[38] Joyner[54] whose MPVM thesis study looked at breeding pairs of aviary budgerigars on a seed-based diet, reported a 60% cloacal presence of gram-negative rods, and reported this as normal. This may be considered normal for a seed-eating budgerigar in this study, but it should not be considered normal for a healthy bird on a balanced diet.

Normal resident microflora maintains an acidic environment that inhibits the proliferation of gram-negative rods and yeast. An imbalance in the intestinal homeostasis results in alterations to the normal populations of microflora, and thus the distribution of bacteria in the

Table 4.2.6 | Techniques for Performing a Fecal Gram's stain

1. Fecal samples should ideally be collected at home and refrigerated until evaluated to prevent the proliferation of saprophytic gram-negative bacteria that may be interpreted as pathogenic.
2. A small amount of feces should be applied to a pre-cleaned glass slide using the wooden end of a cotton-tipped applicator. The sample should be spread into a uniform, thin, even film, using a single swath.
3. Heat fix
4. Place slide on staining tray:
   - Apply 3 drops of gentian violet to the sample and allow to stand for 30 seconds (stains all bacteria blue).
   - Rinse with water and drain excess water.
   - Apply 3 drops of Gram's iodine and allow to stand for 30 seconds (closes pores on gram-positive bacteria).
   - Apply 5 drops 95% ethyl alcohol to decolorize blue stain from gram-negative bacteria.
   - Rinse immediately with water and then add 5 drops saffron to stain gram-negative bacteria red.
   - Rinse immediately with water and blot dry with lens paper or tissue.
5. Scan slide under low microscope power for suitable evaluation site.
   - Using the oil immersion lens, scan several fields for a further idea of uniformity.
   - Choose a uniform field and begin to estimate the total number of bacteria, i.e., count 10 bacteria, assess the proportion of entire field occupied by those 10 bacteria and then estimate the total bacterial population per 1000x field.
6. Record results (see Table 4.2.7)
7. The presence of gram-negative cocci indicates improper staining technique.

With experience, the entire process should take less than 2-3 minutes to perform.

Table 4.2.7 | Recording Results of the Fecal Gram's Stain

Date _____
Species _____
Case ID _____
Results:
_____ total bacteria/1000x oil immersion field
_____ % G+ rods/field
_____ % G+ cocci/field
_____ % G- rods/field
_____ number yeast/field
_____ % budding yeast
_____ high fiber in feces
_____ undigested food
_____ parasites
_____ clostridial organisms
_____ hyperkeratotic cells
_____ normal intestinal cells
_____ RBC's
_____ WBC's

GIT. Normal intestinal flora of parrots, seen as gram-positive (blue) bacteria on a fecal Gram's stain, represent both aerobic and anaerobic bacteria such as *Bacillus*, *Corynebacterium*, *Streptomyces*, *Lactobacillus*, *Streptococcus* and *Enterococcus* spp.,[37] some of which are not able to be cultured using standard techniques.

Enterobacteriaceae are gram-negative (red) bacteria that include pathogens (eg, *Salmonella* spp., *E. coli*, *Acinetobacter* spp.) and non-pathogenic species. Enterobacteriaceae are not normal components of unstressed parrots' microflora[37] and are not detected in preliminary studies of wild parrots.[45,47] However, normal flora bacteria can become secondary pathogens depending on the functional state of the host defense system.[37] Systemic disease, including septicemia and death, can occur when bacteria leave the mucosal surface and penetrate the intestinal wall, a situation that can be precipitated by an imbalanced diet influencing the integrity of mucosal surfaces. Parrot-specific *Lactobacillus*[g] (currently only available in Europe) has been used successfully to treat chronic coliform infections, eliminating the incidence of *E. coli* on culture.[37] Techniques for performing and recording a fecal Gram's stain are outlined in Tables 4.2.6 and 4.2.7.

The fecal Gram's stain is an important component of complete patient evaluation of psittacines. Although not definitive in making a diagnosis, it provides a visual screen of the proportions of bacteria present in the GIT at the time of sampling. When interpreted in conjunction with a complete physical exam and diet history, it can determine the next diagnostic step: whether to proceed to a culture and aggressive therapy or to treat conservatively with husbandry changes.

Interpreting a Fecal Gram's Stain

Ideally, one should use a fecal Gram's stain in conjunction with culture and antibiotic sensitivity testing and only then, antibiotic therapy. Figs 4.2.7-4.2.22 represent a range of fecal Gram's stains commonly seen in clinical practice (1000x oil immersion field) from psittacines maintained predominantly on seed-based diets. Figs 4.2.23-4.2.27 are representative of wild Australian psittacines taken from birds in the December breeding season when diets include a number of wild blossoms (D. Brennan, personal communication). A healthy psittacine should have a predominance of gram-positive rods and cocci, with an absence of gram-negative rods.

Malnutrition and liver disease are characterized by changes in the number and distribution of bacteria on the fecal Gram's stain. In the early stages, the change is reflected by:

- Decrease in total bacteria
- Decrease in percentage of gram-positive cocci
- Increase in % gram-positive rods

In the later stages of malnutrition and liver disease, the Gram's stain generally shows:

- Increase in presence of gram-negative rods (generally speaking, the more gram-negative rods, the more pathologic the situation)
- Presence of yeast which are judged as to their clinical significance by the number of budding yeast per field. The greater the percentage of budding yeast found, the more likely that the immune system is compromised.

The fecal Gram's stain of the stool from pet passerines should be free of bacteria, yeast and *Macrorhabdus* sp. organisms.

Clostridia (gram-positive, anaerobic organisms), are commonly associated with fetid stools in both cockatoos with cloacal prolapse and macaws with cloacal papillomas. Both the clostridial organism and the underlying cause require treatment[106] (Figs 4.2.16 and 4.2.18).

Hepatobiliary System

Fatty Liver Syndrome

The following discussion is offered because the author (GJH) believes fatty liver hemorrhagic syndrome (FLHS) of poultry is similar to a common clinical disease in psittacines, which is primarily a result of malnutrition.[71] FLS, generally a consequence of an imbalance in energy metabolism, is associated with an accumulation of excessive abdominal and hepatic fat. Lipid infiltration weakens the hepatic cellular structure and results in hepatomegaly. Lipid deposits are also found in some skeletal muscles, alimentary tract, autonomic ganglia, CNS, pineal gland, kidney, heart and occasionally, small amounts are seen in the corneas, exocrine pancreas, adrenal medulla and epithelium of the thyroid follicles. Endogenous hypercholesterolemia and cessation of egg production are characteristic signs of a similar disorder in poultry, fatty liver hemorrhagic syndrome (FLHS).[100]

The numerous blood vessels of an enlarged friable liver are easily ruptured during egg laying. The rupture of large blood vessels can result in death. This disease (FLHS) is most often seen in apparently healthy poultry in a high state of egg production. It also affects young birds, especially chicks from young parents, with 50% higher mortality in females than males. Fatty liver syndrome is especially evident in older, overweight pet birds that are fed a diet of seeds or nuts, but can be seen in handfed chicks as well. Trauma associated with adult birds falling from a perch or being held for a routine clinical examination has caused hepatic rupture in FLS

Figs 4.2.7-4.2.12 | Fecal Gram's Stains (FGS) Commonly Observed in Psittacines in Clinical Practice (oil immersion 1000x).

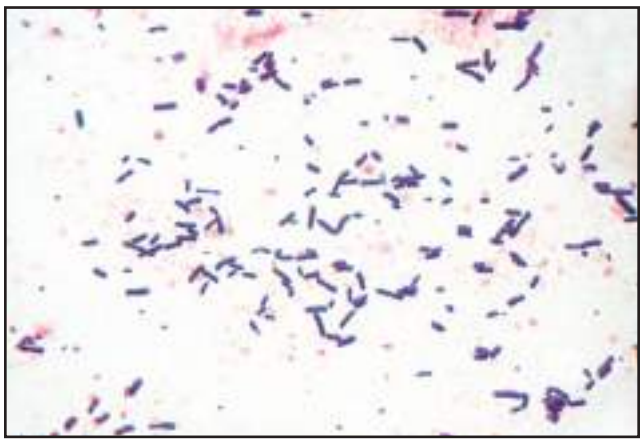


Fig 4.2.7 | Budgerigar, 4-year-old male: Hx = Apparently healthy bird, fed organic formulated diet.[b] Normal flora. Clinical Signs (CS) = none. FGS = Normal distribution of organisms: 157 total bacteria per field, 70% gram-positive rods, 30% gram-positive cocci, 0 gram-negative bacteria, 0 yeast. Digestion of food is complete.

Fig 4.2.8 | Cockatiel, 14-year-old male: Hx = Bird presented for boarding, seed diet. CS = Dull feather color, retained pin feathers. FGS = 55 bacteria per field; 90% gram-positive rods, 10% gram-positive cocci. Hyperkeratotic cell with characteristic straight sides suggests intestinal microflora imbalance, probably due to malnutrition, early liver disease. Rx = Conservative, diet change.

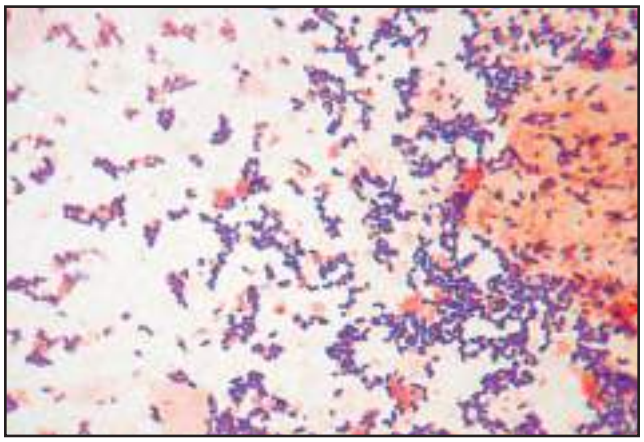


Fig 4.2.9 | African grey parrot, 4 years old, sex unknown: Hx = Intermittent vomiting or loose stool, not as playful. FGS = 400 bacteria per oil field, 95% gram-positive short rods, 5% gram-positive rods, 0 yeast. Overgrowth of intestinal bacteria, enterotoxemia, malnutrition. Rx = Aggressive, dietary change, antibiotics and supportive care.

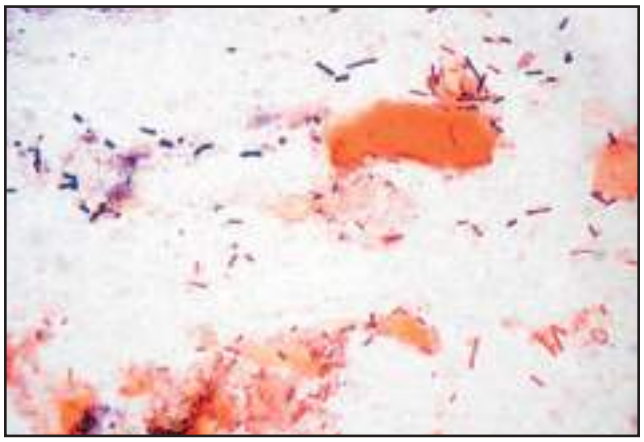


Fig 4.2.10 | Psittacine: Iatrogenic gram-negative rods due to staining error. An error is suspected when the demarcation of gram-positive and -negative is streaked and the groups are similar in shape and size, differing only in color. Note the presence of a normal intestinal epithelial cell, which is rounded and takes on a blue color. Compare this to the straight, pointed edges of the hyperkeratotic cell in Fig 4.2.8. Rx = None.

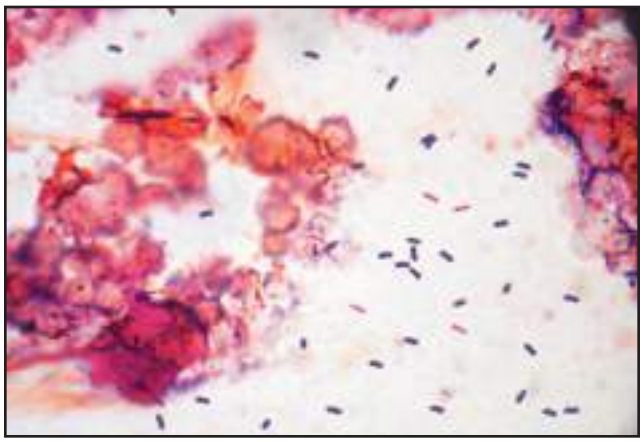


Fig 4.2.11 | Amazon parrot, 8-year-old, female: Hx = Finicky eater, occasionally grumpy. CS = Failure to molt correctly, balding of feet, obvious layering of beak, overgrowth of nails, minor feather-picking. FGS = 40 bacteria per field, 90% gram-positive rods, 0% gram-positive cocci, 10% gram-negative rods. (The normal binding of urates by protein is occasionally seen in fecal gram's stains). Rx = Conservative diet change.

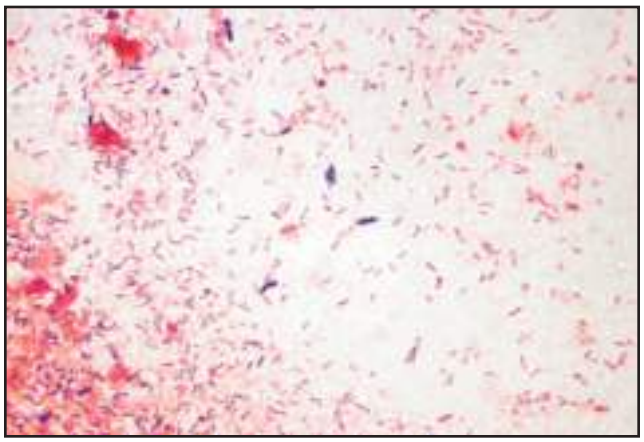


Fig 4.2.12 | Severe macaw, 7 years old, sex unknown: Hx = Depressed, not eating, weak. CS = Underweight, scant feces, dark yellow urine and urates, malcolored feathers. FGS = 200 bacteria per field, 1% gram-positive rods, 0% gram-positive cocci, 98% gram-negative rods. Rx = Aggressive for enteritis and septicemia.

Figs 4.2.13-4.2.18 | Fecal Gram's Stains Commonly Observed in Psittacines in Clinical Practice (oil immersion 1000x).

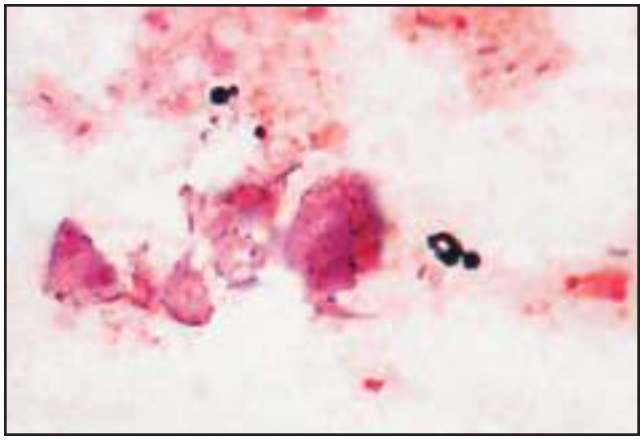


Fig 4.2.13 | Meyer's parrot, 6 years old, sex unknown: Hx = Diet of seeds and supplements, treated previously for "bacteria." CS = Depressed, fluffed, poor appetite. FGS = Scant bacteria, two budding yeast organisms, suggesting early malnutrition. Rx = Aggressive, antimicrobials, dietary change and supportive care.

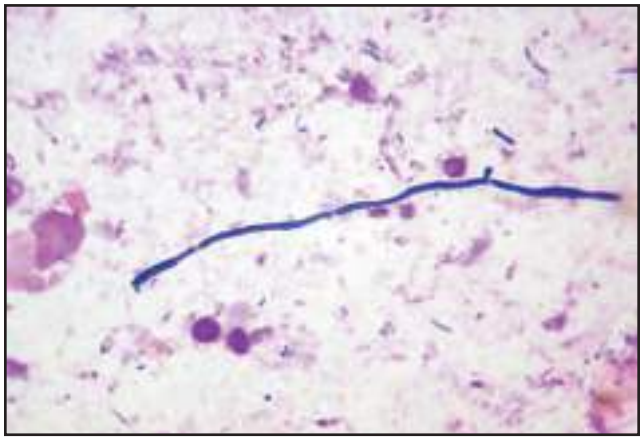


Fig 4.2.14 | Ring-necked parakeet, 9-year-old male: FGS = Scant gram-positive bacteria, occasional gram-negative, many apparent bacterial forms and colors; invasive filament of yeast budding bi-directionally. Rx = Aggressive, antimicrobials, supportive care, dietary correction.

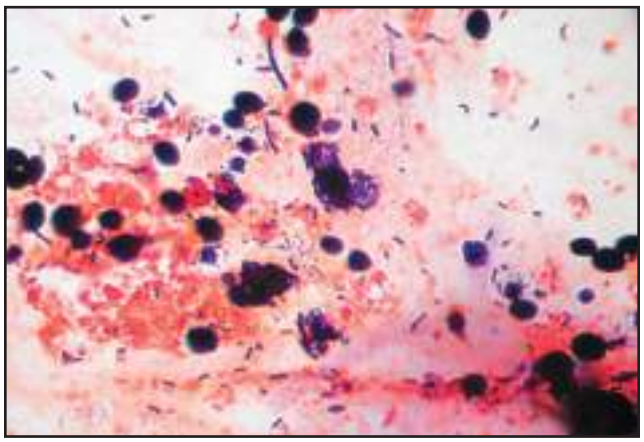


Fig 4.2.15 | Cockatiel, 8-year-old female: FGS = 80 bacteria per field, 80% gram-positive rods, 20% gram-positive cocci; 20 non-budding, yeast-like structures (possibly from bakery products in diet, not clinically significant). Rx = None.

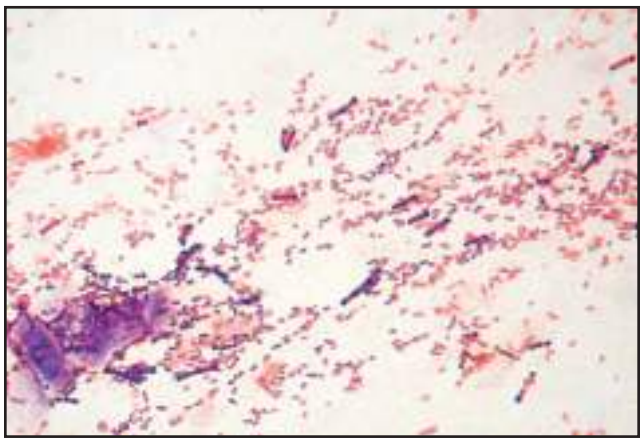


Fig 4.2.16 | Umbrella cockatoo, 6-year-old female: Hx = Exposure to carnivorous pets, seed only diet. CS = Fetid stool, weight loss, passing undigested food. FGS = 200 bacteria per field, 10% gram-positive rods of which 45% are *Clostridium* spp., 45% gram-negative rods. Rx = Aggressive antimicrobials, supportive care, dietary correction.

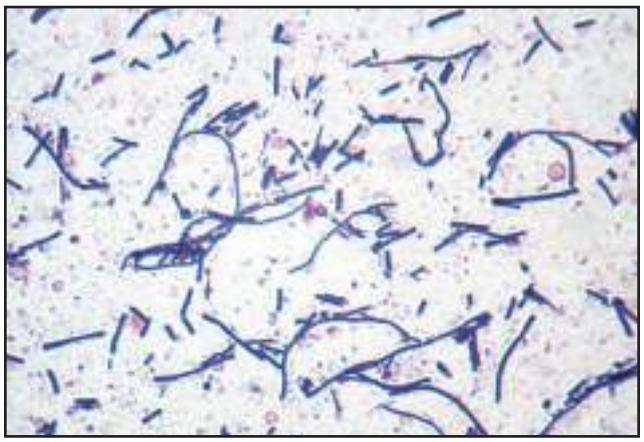


Fig 4.2.17 | Budgerigar, 4-year-old male: CS = Digestive upset. FGS = 200 bacteria per field, 5% gram-positive cocci, 95% gram-positive rods, of which half are large filamentous rods. Rx = Aggressive. See Chapter 30, Implications of *Macrorhabdus* in Clinical Disorders.

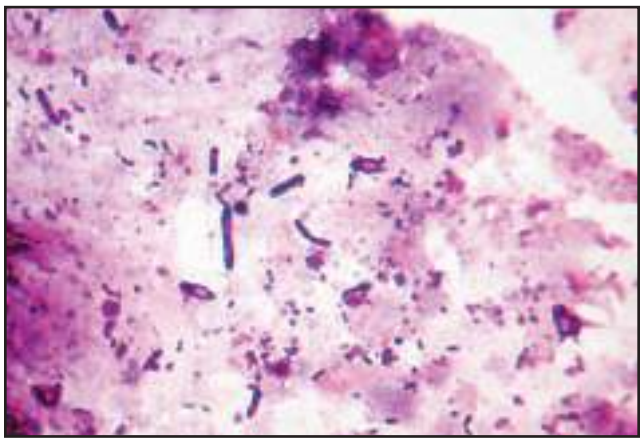


Fig 4.2.18 | Moluccan cockatoo, 7-year-old male: CS = smelly stool. FGS = 50 bacteria per field, 90% gram-positive rods, 10% gram-positive cocci, 30 *Clostridium* spp. organisms. Rx = Aggressive (see Fig 4.2.16).

Figs 4.2.19-4.2.22 | Fecal Gram's Stains Commonly Observed in Psittacines in Clinical Practice (oil immersion 1000X).

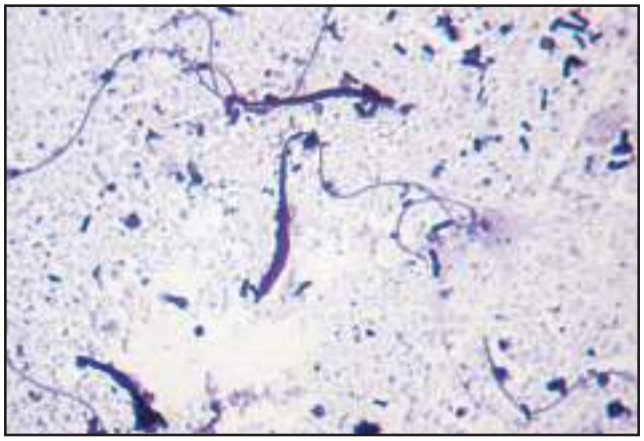


Fig 4.2.19 | Budgerigar, 3-year-old male: Hx = Frequent masturbation. FGS = Presence of sperm. Rx =None.

Fig 4.2.20 | Psittacine: Various forms of gastrointestinal diseases can be suspected if digestion of fiber or dietary ingredients is improper. Top slide = Normal fiber content of feces. Bottom slide = Undigested fiber.

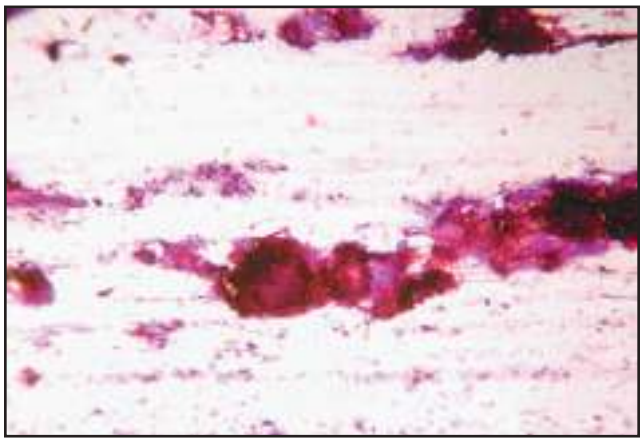


Fig 4.2.21 | Psittacine: FGS = Large amounts undigested fiber (low microscopic power).

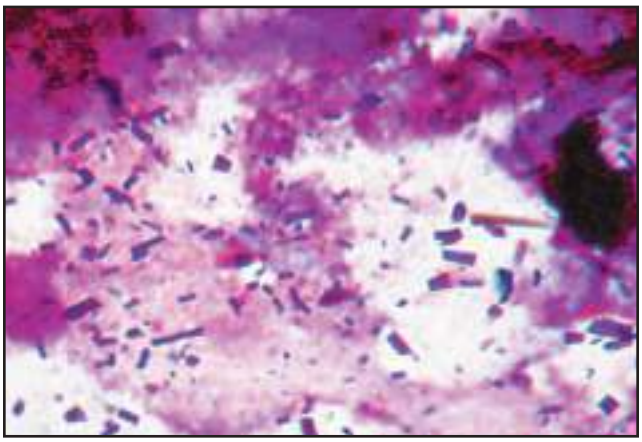


Fig 4.2.22 | Psittacine: FGS = 20 bacteria per field, 100% gram-positive rods, lots of undigested food particles cluttering field, suggesting some form of gastrointestinal disturbance.

Figs 4.2.23-4.2.27 | Fecal Samples from Free-ranging Australian Psittacines (oil immersion 1000x).

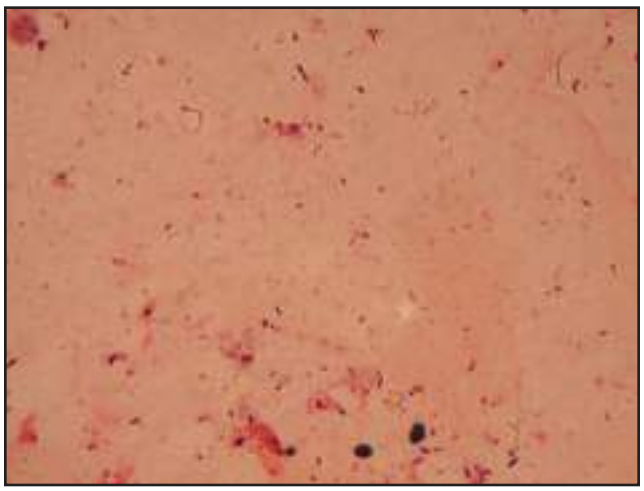


Fig 4.2.23 | *Eolophus roseicapillus*: 30 bacteria/field, 50% small to medium gram-positive rods, 50% gram-positive cocci, no gram-negative rods, no yeast, slight debris, digested particles, two yeast-like forms.

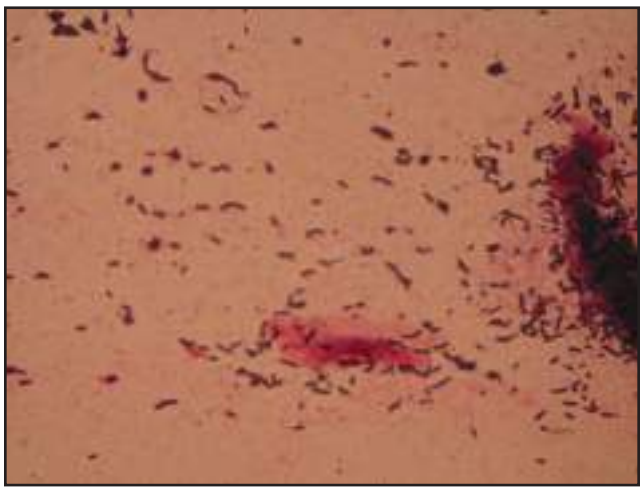


Fig 4.2.24 | *Eolophus roseicapillus*: 170 bacteria/field, 90% large gram-positive rods, 10% gram-positive cocci, no gram-negative rods, no yeast, moderate debris, digested particles.

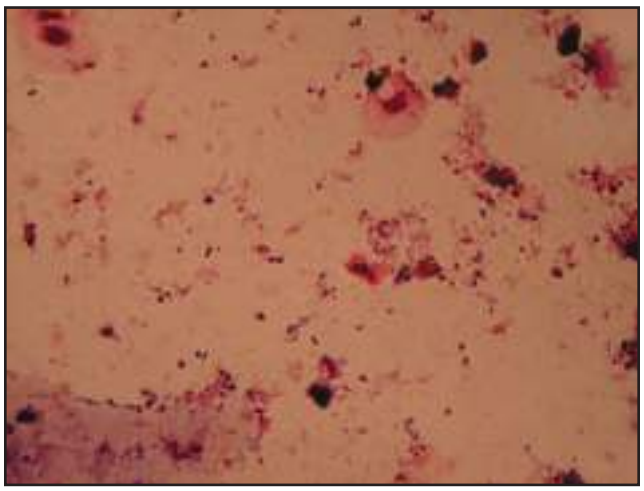


Fig 4.2.25 | *Cacatua tenuirostris*: 60 bacteria/field, 60% small to medium gram-positive rods, 40% gram-positive cocci, no gram-negative rods, no yeast, moderate amount debris, digested particles, two circular non-cornified cells with nucleus, one pollen-like form.

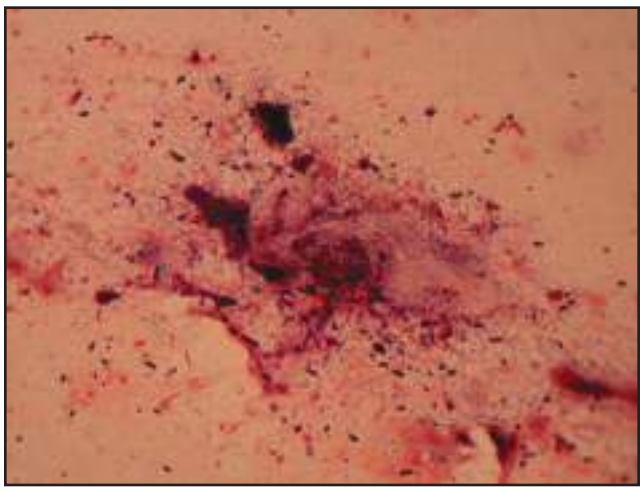


Fig 4.2.26 | *Cacatua tenuirostris*: 90 bacteria/field, 70% small to medium gram-positive rods, 30% gram-positive cocci, no gram-negative rods, no yeast, abundant debris (some not digested).

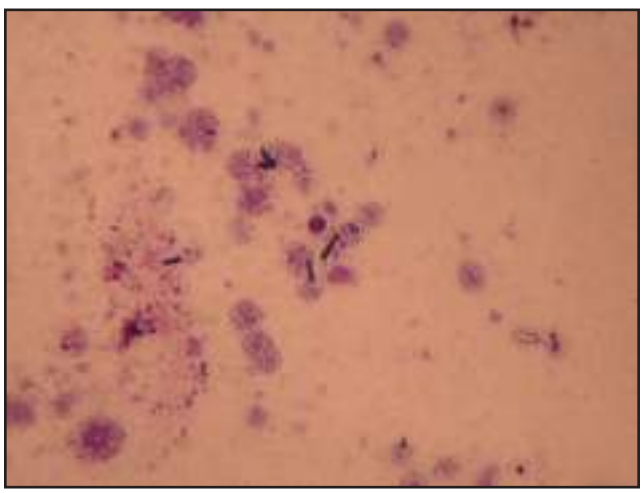


Fig 4.2.27 | *Cacatua tenuirostris*: 15 bacteria/field, 50% large gram-positive rods, 50% gram-positive cocci, no gram-negative rods, no yeast, moderate amount debris (cellular and digested particles).

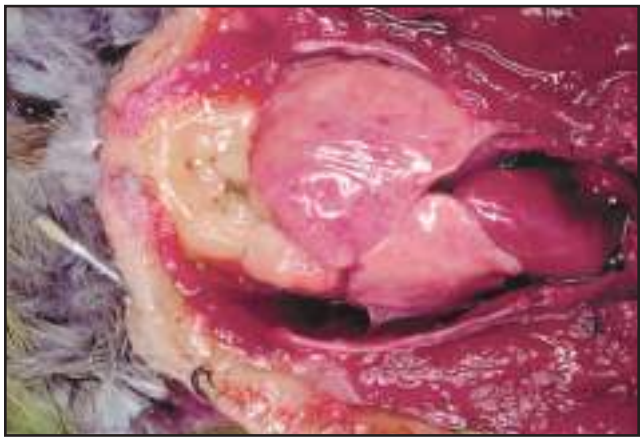


Fig 4.2.28a | Fatty liver in a Mexican red-headed Amazon. The bird died while being restrained for grooming. The serum was lipemic. The veterinarian never realized long nails and beak were a sign of a problem.

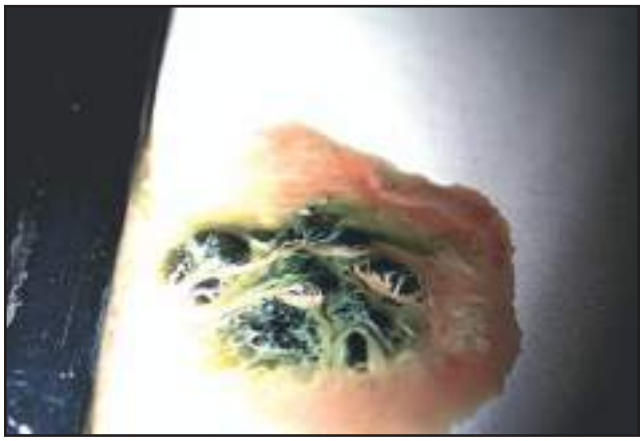


Fig 4.2.28b | The feces from an obese bird will on occasion have a red cream-colored urate after restraint. The renal vessel fragility is a strong prognostic indicator of fatty liver disease.

Fig 4.2.29 | Fatty liver in a baby cockatiel fed a commercial handrearing formula containing excessive fat.

Table 4.2.8 | Enzymatic Changes in Fatty Liver Affected Birds.[10,11,105]

Enzyme	FLHS-affected birds
Acetylcholinesterase	↑ activity
Aspartate aminotransferase	↑ activity
Aspartate transaminase	↑ activity
Glucokinase	↓ activity
Gluconeogenesis	↓ activity
Lactate dehydrogenase	↑ activity
Phosphofructokinase	↓ activity
Sorbitol dehydrogenase	↓ activity

birds. A fatty, swollen liver that compromises the abdominal and caudal thoracic air sacs can result in death from hypoxia (Figs 4.2.28a,b). Although there are some hereditary tendencies towards the disease, nutrition plays a major role in its development. There is little data on FLS in caged birds and a plethora of references on FLHS (Fig 4.2.29). Because FLHS is on the decline in poultry as a result of such data, we offer the following discussion for consideration.

Enzymatic Function and FLHS

A number of plasma enzymes increase with FLHS, such as AST, LDH and glutamate dehydrogenase (GDH). These can be used as indices of the syndrome in laying hens[27] (Table 4.2.8).

Signs and Symptoms

An overweight bird with a marked accumulation of fat is a likely candidate for hepatic lipidosis. Typically these birds may be considered behaviorally normal. Early signs include bile pigments in the urine, changes in the fecal Gram's stain and abnormal feather coloring. See Chapter 15, Evaluating and Treating the Liver for a further discussion.

Nutritional Implications for Development of FLHS

Dietary Fat

Liver fat and excess body weight (associated mainly with an accumulation of abdominal fat) are believed to be two predisposing factors contributing to the onset of FLHS in poultry.[42] Unnecessarily force-feeding birds can increase liver fat and plasma estradiol, producing FLHS.[11,42] A similar condition has also been observed in cockatoos and cockatiels fed improperly formulated diets (Fig 4.2.29). However, high liver lipid content alone may not be sufficient to cause FLS, as adequate dietary levels of lipid trigger a feed-back mechanism, enhanced by dietary starches, to prevent hepatic lipid accumulation.[43a] Long chain fatty acids, especially those of the *n*-3 family are beneficial in the diet as a preventative measure.[44] Ground flaxseed (100 g/kg), flaxseed oil (40 g/kg) significantly decrease hepatic fat.[95] Safflower phospholipids decrease liver triglycerides (hepatic triglycerides increase with liver hemorrhage score[84]), serum cholesterol and body weight.[3] Palm kernel oil at 2% of dry matter weight of diet decreases FLS.[76] Palm oil is rich in vitamin E and carotenoids. The vitamin E fraction (400 mg/kg) is in an approximate ratio of 30:70 tocopherols:tocotrienols.

Tocotrienols have an unsaturated side chain rather than the saturated chain of the more common tocopherols. Tocotrienols more effectively lower cholesterol and show stronger antioxidant activity than tocopherols. See earlier discussion under Rancidity for oxidation's possible role in FLHS in poultry.

L-cysteine

Deficiencies in essential amino acids can increase mortality from FLHS and may be prevented with supplementation of L-cysteine at 6 g/kg of feed.[26] N-acetyl-L-cysteine (NAC) is the pre-crystallized form of the simple amino acid cysteine. It is a powerful antioxidant and immune support substance that neutralizes the free radicals produced by normal metabolic activity. While both cysteine and methionine are precursors of glutathione, NAC is more effective. During digestion, approximately 85% of the sulfur groups of L-cysteine are lost (these contribute to the active portions of glutathione), while only 15% are lost from NAC, resulting in up to six times more sulfur groups after digestion (for detoxification). NAC is also a better source of glutathione than supplementation with glutathione itself, because less than half the supplemental glutathione leaves the digestive system for other organs. This greater efficiency is important since cellular glutathione levels tend to drop 30 to 35% with age.

S-adenosylmethionine (SAMe)

S-adenosylmethionine (SAMe), a natural metabolite of the amino acid methionine, was discovered as a pharmaceutical in Italy in the 1970s and has been available in Europe for over 20 years. It is the most active of all methyl donors arising from the amino acid methionine. While healthy livers synthesize sufficient methionine, liver disease can impair SAMe syntheses. On a cellular level SAMe maintains mitochondrial function, prevents DNA mutations and, restores cellular membrane fluidity so that cell receptors become better able to bind hormones and other factors.

SAMe's methyl groups make possible the production of the "fat burner" carnitine; the neuro nutrient acetyl L-carnitine; the primary ATP energy reservoir, creatine phosphate; the stress hormone and neurotransmitter, adrenaline; the neuro nutrient and chief membrane phospholipid, phosphatidyl choline; and the DNA bases methyl adenine and methylcytosine.

In addition to transmethylation, SAMe is involved in transsulfuration, which begins with the by-products of the transmethylation of S-adenosylhomocysteine (SAH). SAH yields homocysteine, which can be converted to cysteine and then to a family of key sulphur biochemicals: glutathione, glutathione peroxidase, glutathione-S-transferase and taurine. As much as 80% of dietary cysteine, low in many foods, can lose its bioactive sulfhydryl groups passing through the stomach. The glutathione compounds and taurine play important roles in liver detoxification.

SAMe production decreases with age. Dietary supplementation may be required for older birds prone to fatty liver disease. Without SAMe, the liver protective glutathione cannot be synthesized. While increasing glutathione levels through supplementation is desirable, glutathione alone is not a substitute for the combined actions of SAMe and glutathione.

Betaine and SAMe

Anhydrous betaine (trimethyl glycine, not to be confused with the digestive aid betaine hydrochloride), is a substance made from beet sugar that increases SAMe levels. Impairment of SAMe synthetase may result in SAMe being manufactured through the betaine pathway, an alternative to the SAMe synthetase-dependent methionine-plus-ATP route. Increasing levels of betaine reduce fatty infiltration and provide the precursors for the free radical scavenger glutathione.

It is recommended that SAMe supplementation in humans for a diet comprising 16% protein range between 100 to 500 mg, with higher requirements in females. Mild stomach irritation may result if not using a product with an enteric coating. There have been no clinical trials on the effectiveness of SAMe for birds with liver disease. Early empirical data is encouraging.

Vitamins and SAMe

Once a SAMe molecule loses its methyl group it breaks down to form homocysteine. On its own homocysteine can be extremely toxic, but the presence of vitamins B_6, B_{12} and folic acid convert homocysteine into glutathione or re-methylate it into methionine. Deficiencies of any of the active coenzyme forms of vitamins B_2, B_6, B_{12} or folic acid will disrupt SAMe production. Reciprocally, diminished SAMe production will impair conversion of folic acid and B_{12} to their coenzyme forms.

In order to maximize the effectiveness of the interlocking SAMe pathways, the addition of the water-soluble vitamins B_2 (20 to 200 mg), B_6 (40 to 400 mg), B_{12} (0.5 to 5.0 mg) and folic acid (0.8 to 2.0 mg) is required. Vitamins B_6, B_{12} and folic acid also convert the toxic homocysteine to glutathione or re-methylate it into methionine.

Biotin

While low dietary protein predisposes chicks to develop FLHS, high dietary protein can cause classical signs of biotin deficiency.[11] Biotin is an essential coenzyme involved in the conversion of protein to carbohydrate and the conversion of protein and carbohydrate to fat.

Biotin enzymes are important in protein synthesis, amino acid deamination, purine synthesis, and nucleic acid metabolism. Biotin itself is required for trans-carboxylation in the degradation of various amino acids; it also plays an important role in maintaining normal blood glucose levels when dietary intake of carbohydrate is low. Biotin deficiency is most severe in young chicks of heavier strain and greater rate of weight gain;[8] promoting higher growth rates in psittacines may predispose birds to FLS.

Many of the problems associated with biotin deficiencies and FLHS result from biotin's role as a cofactor for many enzymes. These include: a decreased rate of lipogenesis; depressed gluconeogenesis from lactate and glycerol; an increase in the activities of fatty acid synthase (FAS), citrate cleavage enzyme (CCE) and phosphokinase[11]; abnormal fatty acid composition of infiltrated lipid, with an increased proportion of monounsaturated fatty acids; severe hypoglycemia; and depleted hepatic glycogen.[105] Low fat or protein levels that increase the metabolic rate of biotin-dependent enzymes (pyruvate, acetyl CoA carboxylase) aggravate the condition. Biotin also serves as part of the prosthetic group, a transient carrier of CO_2, and is required for normal long-chain unsaturated fatty acid synthesis and is important for essential fatty acid metabolism.[59] FLHS is generally worsened by a high proportion of long chain saturated fatty acids.[48]

Biotin deficiencies can result from a dietary deficiency of biotin or other factors that impact on the stability of biotin. The richest sources of biotin include: royal jelly, liver, kidney, yeast, blackstrap molasses, peanuts and eggs, while poor sources include: corn, wheat, other cereals, meat and fish. However, the chemical form of biotin (bound or unbound) as well as its overall content in feed is important, as less than one-half of the biotin in various feeds is biologically available. Starvation of birds can lower liver biotin levels, leading to an increase in liver weight and lipid content.[10] The addition of raw egg white also decreases biotin availability as the proteinaceous avidin binds very tightly to biotin. While low mortality is seen in broilers fed freeze-dried egg white at 11.8 g/kg, mortality is high if dietary concentrations exceed 17.7 g/kg.

Not all forms of biotin are equivalent in their action; biotin contains three asymmetric carbonations, with eight different isomers. Only the isomer d-*biotin* contains vitamin activity; the stereoisomer l-*biotin* is inactive.[59] Biotin is inactivated by rancid fats and choline [97] and gradually destroyed by ultraviolet radiation. Structurally related analogues of biotin can vary in activity from anti-biotin activity, to no activity, to partial replacement.[7] Oxybiotin has 1/3 biotin activity for chicks whereas desthiobiotin and biotin sulfate are inhibitory to bacteria. Biotinidase, present in pancreatic juice and intestinal mucosa, releases biotin from biocytin during the luminal phase of proteolysis. Physiological concentrations of biotin are absorbed from the intestinal tract by a sodium-dependent active transport process, which is inhibited by desthiobiotin and biocytin.[92]

Cecal microorganisms do not supply chickens with significant amounts of biotin; they compete with the host animal for dietary biotin, thereby increasing the requirement. In poultry, polyunsaturated fatty acids, ascorbic acid, and B vitamins may influence the demand for biotin. Biotin is rapidly destroyed as feeds become rancid, with 96% inactivation occurring in as little as 12 hours if linoleic acid of a high peroxide number is added to the diet. Supplementary choline in biotin-deficient diets decreases biotin status in chicks and increases mortality from FLHS.[105] The use of sulfa drugs can also induce a deficiency. Conversely, α-tocopherol decreases inactivation of biotin.

Minimum biotin requirements have been established for a number of commercial species, with higher requirements for turkeys compared to chickens (NRC, 1994). The minimum dietary requirements of 120 μg/kg dietary dry matter determined for poultry increases to 160 μg/kg in order to prevent fatty liver development and as high as 240 μg/kg when sunflower seed meal is a dietary component.[75] Incorporation of 2% palm kernel oil can reduce the prophylactic dietary biotin requirements down to 120 μg/kg.[76]

Choline

Choline plays an essential role in fat metabolism in the liver. Choline prevents abnormal accumulation of fat by promoting fat's transport as lecithin or by increasing the utilization of fatty acids in the liver itself. While conversion to betaine is required before choline can be a methyl donor, betaine itself fails to prevent FLHS. The addition of choline can decrease the amount of fat in the liver. Diets high in fat exacerbate choline deficiencies, thus increasing the dietary requirement. This is particularly important for chicks, as they are unable to synthesize choline until approximately 13 weeks of age.[68] However, mortality increases in chickens that are supplemented with B vitamins (other than biotin), with higher mortality if choline is also supplemented.[105] Only 57% of biotin in multivitamin premixes[68] is retained if the supplement contains choline.

Normal dietary choline requirements for poultry range from 800 to 2,000 mg/kg. Choline is largely absent in fruit and vegetables and is low in corn. Wheat, barley and oats have higher levels of choline. Peanuts are a good source of choline as are cereal germs, legumes and

Table 4.2.9 | IDC and Iron Storage Disease

Nutritional Imbalance→ High iron content diet, presence of vitamin C and excess vitamin A	
⇩	
CLINICAL SIGNS	
Insectivore/Frugivores	**Parrots**
Early • Listless • Respiratory wheeze or click • Decreased exercise tolerance • Sudden death **Chronic** • Swollen coelomic cavity • Dyspnea with forward leaning posture	**Early** • None - most common • Found on necropsy • Intestinal cellblock is saturated **Chronic** • Sudden death
Diagnostics • Radiology - Pericardial effusion - Hepatomegaly • Ultrasound • Endoscopy and biopsy (caution due to potential coagulation disorders)	**Diagnostics** • Liver biopsy
Treatment • Low iron diet <80 mg/kg • Avoid vitamin C and excess vitamin A • Iron chelator • Black tea • Phlebotomy	**Treatment** • Low iron diet <80 mg/kg • Avoid vitamin C and excess vitamin A

Table 4.2.10 | Avian Families Diagnosed with ISD[20,25,35,36,57,70a,91,107*]

Order	Family	Common Name
Passeriformes	Sturnidae Paradisaeidae	Mynahs/starlings, tanagers Birds of Paradise
Galliformes	Cracidae	Guans, currasows
Ciconiformes	Ardeidae	Bitterns
Trogoniformes	Trogonidae	Quetzals
Piciformes	Rhamphastidae	Toucans, toucanettes, aracari
Ciconiformes	Phoenicopteridae	Roseate flamingos
Psittaciformes	Loridae Psittacidae	Lories Lorikeets Parrot/cockatoos

*See Chapter 4, Nutritional Considerations: Section I, Nutrition and Dietary Supplementation for further discussion

oilseed meals. Choline needs of poultry fed wheat-based diets are much lower than those fed on other grains. Wheat and sugar beets are high in betaine, which can spare choline for some reactions.

Vitamin E

Vitamin E powerfully combats the peroxidation of polyunsaturated fatty acids in the liver. Daily supplementation of 100 to 400 IU Vitamin E in conjunction with low dietary vitamin A is recommended for birds suffering from FLHS. See earlier discussion of rancidity for possible role of fat-soluble vitamin destruction and FLHS.

Silymarin (Milk Thistle)

Silymarin is a collective group of polyphenolic flavanolignans extracted from the seeds of the milk thistle *(Silybum marianum)*. The flavanoids are powerful antioxidants that increase levels of glutathione and protect the liver from oxidative damage. They may promote growth of new, healthy liver cells.[32,85] While clinical trials in birds have not been undertaken to evaluate the effectiveness of silymarin, it has proven effective empirically when administered twice daily to birds with liver disorders. See Chapter 10, Integrative Therapies, Chapter 9, Therapeutic Agents and Chapter 15, Evaluating and Treating the Liver for further information.

Environmental Influences

While nutritional imbalances are the main factors contributing to both FLS and FLHS, stress alone can initiate FLHS. When birds are subjected to mild stress and/or short-term fasting, liver glycogen reserves become rapidly depleted and a progressive hypoglycemia develops that can prove fatal. Stress-associated lipogenesis increases cholesterol synthesis and converts excess glucose to fatty acids, which are stored as triglycerides.

Pesticides

While pesticide levels of individual ingredients may be deemed safe, a combination of a variety of pesticides or an accumulation of pesticides in tissues can result in pesticide toxicity. Polychlorinated biphenyls (PCBs) increase liver and body weights of birds associated with FLHS and can increase total cholesterol[103]. Many pesticides have estrogenic actions; high estrogen levels are associated with FLHS.[42] These estrogenic pesticides mimic the action of normal endogenous hormones and influence ovarian function. A combination of estrogen

and excess dietary energy create sufficient fat deposition in the liver for FLHS to occur.[89]

Summary of Fatty Liver Disease

A variety of nutritional factors are implicated in the development of FLS and FLHS in chickens, but many are avoidable by providing a nutritionally balanced diet. Some factors are exacerbated by other dietary ingredients, environmental stimuli or infectious diseases.[19] While the name of the disease, "fatty liver (hemorrhagic) syndrome," implicates dietary fat levels as causative factors, there are many other dietary components that are important and warrant further consideration. We feel this model may serve the captive parrot industry well, as empirically the same corrections seem to apply.

IRON STORAGE DISEASE

Iron storage disease (ISD) is prevalent in many frugivorous and insectivorous birds maintained on commercially formulated foods (Table 4.2.9). Iron storage disease differs from one of its precursors[53], hemosiderosis, which is defined as the excessive accumulation of iron (hemosiderin) in hepatocytes or in free circulation in the blood, without alteration of normal tissue morphology or damage to any of the major organs. Various factors have been implicated in the development of this disease, including genetic predisposition, immunological stress, nutritional inadequacy and viruses; dietary iron content has been the main focus of nutritional investigations. The causative factors of ISD have been addressed by several authors.[29,36,70a] Further studies on the interactions of dietary sugars, copper and iron metabolism have been proposed.[87] The discussion here will be confined to nutritional implications in the development of ISD. There is a high correlation with commercially formulated foods and ISD. Table 4.2.10 highlights a number of avian families in which the disease has been reported.

While iron is essential for fundamental cell functions, it is also a catalyst for chemical reactions involving free radical formation that can lead to oxidative stress and cell damage. Uptake of iron from the diet is regulated in the intestine, so acute intoxication is not observed under natural conditions. Cellular iron levels are generally regulated to maintain adequate substrate levels while minimizing the pool of potentially toxic 'free iron.' The main control of body iron homeostasis is in the duodenum where dietary iron is absorbed, but no controlled means of eliminating unwanted iron has evolved in animals. Consequently, chronic ingestion of large amounts of absorbable iron can lead to the storage of iron in the liver in many species.

Iron storage disease results from the accumulation of iron in various tissues, with the liver most frequently involved. In severe cases, iron pigment is found in the liver, spleen, gut wall, kidney and heart; this leads to subsequent development of ascites, heart failure and multisystem pathology.[25] Iron may be found within the Kupffer cells in the liver[36] and the macrophage cells of the spleen, especially where concurrent diseases, such as hemolytic anemia, septicemia, neoplasia or starvation, are present.[25]

The syndrome of excessive iron overload in mynahs shares most of the important histopathologic characteristics with idiopathic hemochromatosis in human beings. Iron storage disease has been correlated with immunological stress,[25,36] as well as crowded conditions.[53] Reduced peristalsis or neuropathic gastric dilatation may increase iron absorption.[36] Stress increases lipid peroxidation and diminishes vitamin E levels, resulting in a lower level of antioxidant activity. Iron and vitamin E are involved in electron transfer in reduction/oxidation cycles; a dietary surplus of either iron or vitamin A decreases the α-tocopherol concentration. Therefore, any impact on vitamin E levels may reduce the protection of biological membranes against oxidation. In addition, diets high in saturated fats increase iron absorption.[79]

Nutritional Implications for ISD

Highly frugivorous or highly insectivorous birds have adapted to foods low in iron (fruits and insects). The high vitamin C content of many fruits enhances iron uptake from iron deficient diets. Consequently, high dietary iron has been implicated in the development of the disease and it is generally recommended that iron content of commercial diets be maintained below 100 mg/kg[53,] and in mynahs 19 to 25 mg/kg.[29,94] However, birds have been maintained on commercial foods that reflect the high values of iron in some dietary components of wild toucans[78] (150 mg/kg) with no evidence of iron storage disease. Diet is not implicated in the development of ISD in the Rothschild mynah.

Vitamin A and Iron Uptake

Some commercially formulated products have high vitamin A content (see Chapter 4, Nutritional Considerations, Section I Nutrition and Dietary Supplementation). These high vitamin A levels are in contrast to the low vitamin A content of fruits and insects. Vitamin A from plants arises from conversion of carotenoids, a regulated process that avoids potential vitamin A toxicity. Productivity of psittacines increases when birds are transferred to formulated diets low in vitamin A but high in carotenoids.[67] Additional vitamin A is either supplied from plant based carotenoids in the diet or else it may

Table 4.2.11 | IDC and the Respiratory System

Nutritional Imbalance → Squamous Metaplasia of Respiratory Epithelium			
CLINICAL SIGNS			
Oral/Rhinal	**Sinus**	**Trachea/Lungs**	**Air Sacs**
• Blunting or loss of choanal papillae • Increased mucus viscosity • Serous rhinal discharge • Sneezing, scratching nares • Chronic - Rhinolith formation: Can be sterile, bacterial, or fungal. Dark discharge = hemoccult+	• Infraorbital swelling • Respiratory wheeze or click • Lacrimal duct infection or impaction • Chronic - Secondary sinusitis: May be bacterial or fungal	• Voice change or loss • Decreased exercise tolerance • Dyspnea with forward leaning posture • Chronic - Tracheal obstruction: Often *Aspergillus* granuloma	• Increased panting • Decreased exercise tolerance • Mild, intermittent tail bob • Chronic - Secondary air sacculitis: *Aspergillus* is commonly isolated
TREATMENT			
Diagnostics	**Medical Treatment**	**Environmental Concerns**	
• Endoscopy (tracheal, coelomic for air sacs) • Cytology of respiratory exudate • Culture and sensitivity of exudate • Radiology • Hematology • Biochemistry • Serology (aspergillosis)	• Debride rhinoliths • Nasal/sinus flushes as needed • Infraorbital sinus drainage as needed • Nebulization, this can be utilized for humidity, mucolytic agents, or antimicrobial therapy delivery • Systemic treatment of secondary infections - *Aspergillus* • Diet correction - (see Fig 4.2.7: see diet conversion challenges at end of chapter)	• Proper humidity • Adequate ventilation of enclosure • Filtration of ambient air (medical grade filters) • Avoidance of exacerbating aerosols: - cigarette smoke - perfumes - cockatoo dust - pollens	

Recurrent or chronic respiratory infections are common with chronic IDC and marked changes in the respiratory epithelium.

be inferred that birds have an overall low requirement for vitamin A. The incidence of ISD is negligible in these birds.

Low serum retinol is associated with mild anemia in adult humans.[30] Retinol also plays a role in increasing levels of hemoglobin in children, especially those on iron supplementation.[2,63] Dietary iron also influences conversion of ß-carotene to retinol by enhancing ß-carotene 15,15'-dioxygenase activity in the small intestinal mucosa of rats.[31] High levels of dietary vitamin A may negatively influence availability of other fat-soluble vitamins such as vitamin E.

In contrast to vitamin A, the presence of carotenoids in microsomal membranes partially inhibits the loss of α-tocopherol, especially during the late phase of oxidative stress when ß-carotene decreases phospholipid hydroperoxide production.[73] However, despite its beneficial antioxidant activity, ß-carotene, like vitamin A, can increase the absorption of iron by preventing the inhibitory effect of phytate and tannins on iron absorption by forming complexes with iron that maintain solubility in the intestinal lumen.[62]

The antioxidant activity of the different carotenoids is variable. The inhibitory effect of ß-carotene on the production of lipid peroxidases is less than that of the extremely potent antioxidant astaxanthin from marine micro algae. Astaxanthin protects the mitochondria from damage by Fe^{++}-catalyzed lipid peroxidation during vitamin E deficiencies.[62] It is two-fold more effective than ß-carotene in inhibiting production of lipid peroxidases.[39] Peridinin, another carotenoid of marine micro algae, limits oxidative damage on iron-liposomes, possibly by decreasing membrane permeability to initiators.[12] While the direct benefits of the blue-green algae *Spirulina platensis* have not been evaluated in birds, the phycobilins (phycocyanins and allophycocyanin) of *S. platensis* act as potent free-radical scavengers (hydroxyl and peroxyl) and inhibit microsomal lipid peroxidation in humans.[88] Diets[b], low in vitamin A and containing micro algae, have demonstrated improvements in health and productivity of large psittacines.[67]

Canthaxanthin is a carotenoid compound that is supplemented to promote feather pigmentation in flamingos and scarlet ibis. A recent study suggests that canthaxanthin can substantially alter the antioxidant status of murine liver tissue in vivo. In mice, canthaxanthin reduces both cellular content of lipophilic antioxidants and the activity of enzymatic antioxidants, as well as increasing iron concentrations in the liver by up to 27%. ISD has been diagnosed in flamingos.[20] The addition of canthaxanthin to the diets of these birds may alter the protective ability of tissues against oxidative stress in vivo and increase iron storage in the liver. The carotenoid canthaxanthin is associated with eye and liver damage in humans.

Fig 4.2.30 | An old female budgerigar with IDC, chronic rhinorrhea and secondary rhinal infection with yeast and/or bacteria.

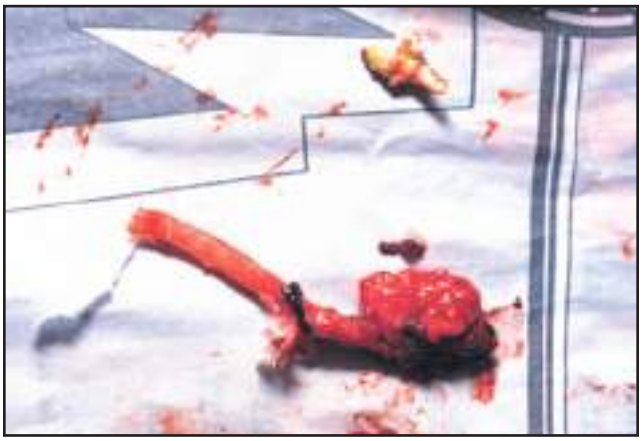


Fig 4.2.31 | Liths in the bronchial syrinx area or the air ostium to the lungs often become secondarily infected with yeast or fungal spores. This lesion was taken from a free-ranging Major Mitchell's cockatoo.

Table 4.2.12 | IDC in Geriatric Budgeriagar with Goiter

History	Clinical Signs	Treatment
• Diet Seed based	• Regurgitation • Crop mucus accumulation • Dyspnea • Swollen thyroid	• Dexamethasone (if severe) • Iodine • Diet change

Summary of ISD

While diets low in iron are advocated for birds susceptible to ISD, the high vitamin A content of commercially formulated foods may also be implicated in the development of the disease. It is recommended that diets low in iron, vitamin A and saturated fats be presented to birds susceptible to this disease in addition to supplementation with vitamin E. Furthermore, high levels of dietary ß-carotene may be detrimental. Vitamin A activity should be provided from other carotenoid sources, especially those present in blue-green algae, such as *Spirulina platensis*. Frugivorous species should be provided with fruits low in vitamin C to minimize uptake of iron from commercial diets. Recent recommendations on the use of tannins from tea to tie up iron have shown promise. It is evident that further research on this topic is required.

RESPIRATORY SYSTEM

Loss of function of epithelial tissue is a problem in the respiratory system because loss of cilia and mucus production decreases cleaning capacity (Table 4.2.11). In chronic cases, serum oozes from endothelial ulcerations and provides a culture media for bacteria, yeast or other fungi (Figs 4.2.30-4.2.31). The nares can reflect the internal condition.

Liths that roll up to form "balls" can develop from desquamated epithelial cells when proper cleaning function has not been maintained. These balls may randomly accumulate in such places as the connection between the cul-de-sacs of the infraorbital sinus or the layering of the rhinal cavity under the operculum. In the case of the sinus, they may act as one-way valves causing the cervicocephalic air sac to hyperinflate. In the rhinal cavity, the accumulation leads to rhinitis or rhinorrhea (often containing blood). The liths in the bronchial syrinx area or the air sac junction to the lungs can become secondarily infected with yeast or other fungal spores (Fig 4.2.31). Recent reports from Europe indicate this is a common respiratory sign seen in African greys with circovirus.

Liths are not confined to the respiratory tract. They can accumulate in the bile duct. When flecks of liths partially obstruct the duct in cockatiels, the duct balloons to resemble a gall bladder. Liths in the kidney are often associated with gout. In the uterus, they can form egg-like structures of various sizes and shapes.

BUDGERIGAR GOITER

It has been common knowledge since the 1950s, that budgerigars *(Melopsittacus undulatus)* develop goiter on a seed diet low in iodine. Table 4.2.12 shows the typical scenario. The bird may only show obesity. Most common clinical presentations are "vomiting" (regurgitation) of a thick liquid (mucus) that accumulates on the feathers of the head. Respiratory sounds (squeaking) from impingement on the syrinx by the swollen thyroid are also a frequent complaint. While diagnostic tests are possible, they are seldom used due to their expense and the ease of diagnosis using response to treatment. Dexamethasone may speed the response to injectable

Table 4.2.13 | Influence of Prostaglandins on Renal Function

	PGE_2	Prostacyclin	$TBXA_2$
Vasodilation	↑	↑	
Vasoconstriction			↓
Renal blood flow	↑	↑	↓
GFR	↑	↑	↓
Platelet function		↑	↓

iodine by decreasing thyroid swelling and may be life saving in severely dyspneic birds. Complete recovery is insured by adding iodine to the water or seeds. Since other deficiencies will soon manifest on such a diet, only geriatric birds and birds that refuse to convert to formulated diets are treated by iodine supplementation alone.

INGLUVITIS

Baby psittacines from parents on seed-based diets commonly are plagued by ingluvitis. Seed-based malnutrition is the primary cause and can be prevented with formulated diets. Secondary invaders (yeast and Enterobacteriaceae) may require specific antimicrobials and resemble liver and gastrointestinal disorders caused by the same agents. In these latter conditions, crop atony followed by ileus is not unusual, especially in cockatiels. These birds are very difficult to cure and frequently suffer a prolonged wasting type malaise. See Chapter 7, Emergency and Critical Care and Chapter 14, Evaluating and Treating the Gastrointestinal System, for further discussion.

RENAL DISEASE

Excesses of dietary protein and the accumulation of waste products derived from the catabolism of protein result in uric acidemia and to a lesser degree, uremia, in birds. Excessive dietary protein is catabolized to uric acid and other nitrogenous compounds normally excreted by the kidneys. Decreased renal function leads to accumulation of these compounds. One goal of nutritional therapy is to achieve nitrogen balance by proportionally decreasing protein intake as renal function declines, except in cases of protein losing nephropathy.

Gout is associated with the deposition of a white chalky substance (urate) on body organs (visceral), in joints (articular) or in ureters (renal). Urates are the end products of protein metabolism in birds. Their accumulation impairs the function of key organs and can eventually be fatal. While excess dietary protein has been implicated in the increase of serum uric acid, birds are usually able to excrete excesses.[58] However, fasting, dehydration or a diet deficient in lysine may increase serum uric acid. Alterations to normal elimination of uric acid, which can cause extensive renal damage, have also been associated with vitamin A deficiency leading to gout.[58]

Dietary lipids have been implicated in the progression of chronic renal disease, especially in relation to a change in the balance of renal prostaglandins. A diet rich in arachidonic acid leads to a predominance of PGE_2, prostacyclin and thromboxane A_2 ($TBXA_2$), resulting in a shift toward greater vasodilatation and less platelet aggregation (Table 4.2.13). For further discussion on this topic, see Chapter 16, Evaluating and Treating the Kidneys. The effects of excess vitamin A and/or D are discussed in Section I of this chapter.

CARDIOVASCULAR DISEASE

Atherosclerosis

Atherosclerosis is problematic for psittacines in captivity[52] that are provided with high-fat diets and little exercise compared to their free-ranging counterparts. Atherosclerosis involves the deposition of cholesterol within the innermost lining of the arteries and subsequent inflammatory response and fibrosis. While cholesterol-lowering agents and dietary changes are treatment mainstays in humans, the development of the disease may be prevented in birds with nutritionally balanced diets containing antioxidants.

Damage to the endothelial lining of the internal surfaces of the heart increases permeability to lipoproteins and macrophages. Increased endothelial permeability leads to the accumulation of lipoproteins within the subendothelial space, which initiates the formation of atherosclerotic plaques. The oxidation of lipoproteins retained within the subendothelial space is responsible for the inflammatory response seen in atherosclerosis. Lipoprotein oxidation is a necessary step in the development of atherosclerotic plaques; the oxidation of low-density lipoprotein (LDL) is implicated in lesion formation in the aorta. Vitamin A can attenuate the oxidation of LDL and consequently minimize or even reverse aortic plaque development and endothelial dysfunction.

Oxidants are produced by the normal metabolic functions of the endothelium, macrophages, and smooth muscle of the arterial wall. Lipoproteins that accumulate within the subendothelial space are exposed to these cellular oxidants. Although a small degree of lipoprotein oxidation may occur during circulation, most lipoprotein oxidation occurs within the arterial wall. It is therefore noteworthy that lipoproteins are themselves enriched with antioxidants such as vitamin E, coenzyme Q_{10} (ubiquinone) and carotenoids. Water-soluble antioxidants found in the plasma, such as vitamin C, may also be important in preventing oxidation of lipoproteins in the circulation.

In a recent abbreviated study, two high fat and two low fat diets were fed for periods of 24, 28 and 32 days. Results showed that palm kernel oil (derived from the seed) produced significantly higher levels of plasma cholesterol and phospholipid concentrations than did sunflower oil.[13] No conclusions on the effect on atherosclerosis could be drawn. The authors had previously shown 84% sudanophilic staining levels in aortas of parrots presented for necropsy.[14a] The authors recommended diets of up to 10% fat on a dry matter basis.

Nutritional Supplementation for the Treatment of Atherosclerosis

In studies on humans, palm oil (derived from the fruity coating outside the seed), which is distinct from palm kernel oil, has proven beneficial in the treatment of atherogenesis. Palm oil is very rich in carotenes, vitamin E and coenzyme Q_{10}. The cholesterol lowering action of palm oil is attributed to its high vitamin E content, especially the tocotrienol fraction. A typical palm oil vitamin E concentrate contains up to 30% α-tocopherol and 70% mixture of different tocotrienol isomers (20% α-tocotrienol, 20% γ-tocotrienol, 40% α-tocotrienol, 10% δ-tocotrienol and 10% others).[49]

Cardiomyopathy

Cardiomyopathy has been associated with a number of disease entities including malnutrition, as the pathogenesis in birds is similar to mammals, it is possible that similar nutritional inadequacies are implicated. While supplementation levels have not been established for birds, studies of mammals indicate that supplementation with taurine, vitamin B_6, coenzyme Q_{10} and carnitine are helpful.

Taurine

Taurine is an essential amino acid in cats as they have a limited ability to synthesize taurine from cysteine and methionine. Because there are many physiological similarities between carnivorous birds and felines, it is possible that taurine deficiency is implicated in the development of cardiomyopathy in carnivorous birds, especially those provided with commercial dog foods that are typically low in taurine. Plasma taurine concentrations can be influenced by food intake and food deprivation. Whole blood concentration is a more reliable index of taurine status, as this only declines after prolonged periods of depletion. While taurine levels for cats (plasma <20 to 30 nmol/ml; whole blood <150 nmol/ml) are indicative of dietary deficiencies, similar data is not available for carnivorous birds.

Vitamin B_6

Mild deficiencies in vitamin B_6 can interfere with taurine conversion. The heat treatment associated with the production of commercial pet foods degrades B_6.

Coenzyme Q_{10} (CoQ_{10})

Coenzyme Q_{10} is an antioxidant that plays a role in mitochondrial function. A deficiency of CoQ_{10} is correlated with deterioration in heart function. CoQ_{10} is similar in structure to vitamin K and can interfere with the blood-clotting mechanism. Studies with humans indicate that supplementation at 2 mg/kg body weight reduces symptoms associated with cardiomyopathy and heart failure. ß-blockers and cholesterol-lowering drugs from the statin family can interfere with the body's production of CoQ_{10}.

Carnitine

Carnitine is a small, water-soluble, vitamin-like quaternary amine found in high concentrations in mammalian heart and skeletal myocytes. L-carnitine is synthesized primarily in the liver from the amino acids lysine and methionine. Long-chain fatty acids are important for maintaining a constant energy supply to the heart. Carnitine is a critical component of the mitochondrial membrane enzymes that transport activated fatty acids. In addition to its role in fatty acid transport, free carnitine serves as a mitochondrial-detoxifying agent. Because of the high-energy requirements of the heart muscle, it is particularly vulnerable to carnitine deficiencies. A recent report shows the potential for L-carnitine to reduce the percent body weight and lipoma size in budgerigars.[14c]

OPHTHALMIC DISORDERS

Cataracts

While causative factors have not been identified clearly, cataracts have been described in aging macaws.[24] Nuclear cataracts associated with aging occur in the center of the lens. The nucleus of the lens is particularly sensitive to nutrient deficiencies. Nuclear cataracts are associated with deficiencies in the fat-soluble vitamins A and α-tocopherol and the water-soluble vitamins B_2 (riboflavin) and B_3 (niacin). Carotenoids have potent antioxidant activity with marginal inverse associations between the carotenoids lutein and cryptoxanthine and the development of nuclear cataracts. Riboflavin is important in the production of glutathione peroxidase; deficiencies in glutathione peroxidase have been correlated with cataracts. Selenium and vitamins C and E are also helpful in preserving glutathione levels. However, supplementation with selenium is not recommended as cataracts have been correlated with both deficiencies and excesses of this trace mineral. Taurine deficiency (particularly in animals fed heat-processed diets) has also been correlated with cataracts.

Table 4.2.14 | The IDC and the Reproductive System

History	Signalment	Diagnosis	Treatment
Diet • Seeds, nuts, produce, supplemental vitamins, table foods	**Eggs** • No eggs, small clutches, soft/rough-shelled eggs, infertile eggs **Hatchlings** • Failure to hatch, (need assistance), early neonatal death, bent legs/beaks, crop/digestive disorders, require hand raising (from day 1) **Adults** • Egg binding, masturbation, regurgitation, nest building, mate mutilation	**Blood** • Increased cholesterol, hyperlipidemia **Diet Change** • Response takes up to a year **Reproductive Tissue** • Culture uterus, biopsy uterus/testis, endoscopy (cystic ovaries) **Eggs** • Necropsy, culture	• Treat specific disorder **Diet** • Nutritionally balanced formulated organic foods, limit supplements: low sugar produce - Breeding Birds: 5-10% high fat items (sunflowers seeds, nuts) sugary fruits & vegetable (necessary for breeding stimulation) **Genetics Cockatiels** • Choose birds that are not chronic egg layers - Deplete body stores of nutrients • Hormone therapy **Salpingohysterectomy**

The cortical cataract occurs in the cortex of the lens and a subcapsular cataract starts as opacity under the capsule, usually at the back of the lens. The prevalence of cortical cataracts is reduced in the presence of polyunsaturated fatty acids. Insufficient *n*-3 fatty acids or excess saturated or '*trans*' (hydrogenated) fats may impact on the progression of eye disease. While high levels of α-tocopherol reduce the risk of nuclear opacity, medium levels are associated with a reduced risk of cortical opacities.

Macular Degeneration

While macular degeneration has not been reported in birds, there are different kinds of macular problems. In other animals the most common is age-related macular degeneration. Zinc deficiencies can exist in older birds from poor absorption from food. Zinc is highly concentrated in the eye, particularly in the retina and tissues surrounding the macula. Zinc is necessary for the action of over 100 enzymes, including chemical reactions in the retina. While zinc supplementation may be beneficial if there is a dietary deficiency or malabsorption problem, excess zinc may also interfere with other trace minerals such as copper. The xanthophylls lutein and zeaxanthin selectively accumulate in the macula, providing what is known as the macular pigment. Singlet oxygen production in the eye can be increased by UV light exposure, but this is yet to be established in birds. It is assumed this increased oxidative damage is due to increased free radicals in the retina. Lutein and zeaxanthin are scavengers of these free radicals. Anti-oxidants (vitamin A, C and E) may also help slow down macular degeneration and other aging factors associated with activated oxygen from exposure to light, but this has yet to be established.

REPRODUCTIVE SYSTEM

Various reproductive problems can be caused by a nutritionally imbalanced diet (Table 4.2.14). A high fat and sugar based diet is strongly suspected to be involved in the overly stimulated breeding bird. See Chapter 3, Concepts in Behavior, Section III Pubescent and Adult Psittacine Behavior for a table of foods hypothesized to be stimulatory.

Protein and Amino Acids

Amino acid requirements increase at least one week prior to the first oviposition for growth of the oviduct and accretion of egg proteins. While overall protein requirements for birds laying small clutches may be little more than maintenance requirements, a deficiency in essential amino acids may increase overall protein requirements. Budgerigars maintained on seed-based diets that provided only half the lysine, methionine and cysteine required, produced fewer hatchlings, fledglings, fertile eggs, and total eggs.[4]

Vitamins

Fat-soluble vitamins can influence breeding potential; a reduction in vitamin A and E reduces antioxidant function and increases exposure of lipid-rich tissues to peroxidation. Both deficiencies and excesses of Vitamin A influence epithelial integrity. The resulting hyperkeratosis can influence mucin production as well as proper tone and elasticity of reproductive tissue. Vitamin A deficiencies can result in failure of spermatogenesis, decreased size of testes and decreased sexual activity in males.

Dietary excesses of vitamin A may negatively influence reproductive output of birds. Improved productivity has been recorded in a variety of larger psittacines maintained on diets low in vitamin A and high in vitamin E.[67] A study of blue and gold macaws *(Ara ararauna)* highlights the importance of maintaining breeding birds year round on nutritionally balanced diets. Productivity significantly decreased when birds were transferred to seed-based diets during the nonbreeding season.[67] Maternal diets can influence nutrient transfer to embryos, as well as the antioxidant status of the developing embryo and hatchlings.

Table 4.2.15| Action of Pesticides at a Cellular Level

Action	Result	Pesticide
Altered membrane integrity	Interferes with fluid and electrolyte movement	DDT Pyrethrins
Altered cell volume regulation	Alters energy metabolism, reducing energy availability to drive active transport systems, synthesis of macromolecules and maintenance of osmotic balance	Dinitrophenol Chlorophenol fungicides Arsenates Tin fungicides
Results from metabolic defects	Abnormal accumulation of lipids and pigments	industrial estrogenic wastes
Alteration of protein synthesis	Denaturation or inactivation of enzymes	Oxalic acid Fluoroacetate Organophosphates Carbamates
Disturbance of growth regulation	DNA damage that is not properly repaired or exceeds homeostatic control	Damage documented but not the cause

Lipids

While obesity can be detrimental to breeding birds, an imbalance of essential fatty acids is also problematic. Linoleic acid (*n*-6) typically reduces liver fat and improves egg production.[42,43a] It is preferentially retained by laying hens. Conversely, high levels of linolenic acid reduce egg production in laying hens.[1]

Typical fatty acid profiles of chicken spermatozoa display considerable resistance to manipulation by dietary means.[56] Dietary supplementation of docosahexaenoic acid (DHA) may inhibit synthesis of *n*-6 fatty acids in the testes[23] resulting in an accumulation of DHA, a concurrent decrease in vitamin E concentrations, and increased susceptibility to lipid peroxidation. While supplementation with *n*-3 fatty acids may be beneficial for the treatment of inflammatory diseases (see Chapter 16, Evaluating and Treating the Kidney), it may influence fertility by impacting the integrity of the component fatty acids in the spermatozoa's phospholipid membranes.

Sperm output generally decreases with age, but this decrease can be prevented by supplementing diets with oils rich in either arachidonic acid or DHA, in conjunction with vitamin E (200 mg/kg). Testes mass can also be increased up to 1.5 times in aging birds when supplemented with essential fatty acids. However, supplementation with linoleic acid in the absence of vitamin E can result in 50% reduction in spermatozoa per ejaculate in aging birds.

The cloaca can transmit microbes retrograde into the reproductive tract. The hypothesis that there is a passage of beneficial microbes at the time of copulation is intriguing. It has been hypothesized that females copulate with multiple males to gain microfloral advantage for the offspring from flora transmitted at copulation.[64] The female is thought to benefit by protection from future as well as present infections that become overwhelmed by the inoculum.[64] This is another area that, if proven, shows the value of the microflora to the body.

ADVERSE FOOD REACTIONS

Food Allergies

The bird should undergo a complete physical examination including appropriate diagnostics to look for any underlying pathology, which may be exacerbated by or in addition to the suspected food allergy. Not all dermal problems are related to nutritionally imbalanced diets as some birds, like cats and dogs, have allergic reactions to certain dietary ingredients. Advanced stages often result in feather picking and self-mutilation. While early signs of these food allergies are yet to be described, failure of nutritional therapy may warrant skin allergy testing and/or a simplified organic diet[a] where ingredients such as corn and sunflower seeds are eliminated. While these ingredients have been incriminated in the development of dermal disorders[65], they are regularly included in both formulated and homemade diets as primary ingredients with no reports of digestive disorders. Organic formulated diets are free from pesticide residues and preservatives that could be potentially allergenic. Common allergens for mammals (wheat, gluten, egg and dairy products) should also be eliminated from sensitive birds, although no proof exists they are a problem. A commercial organic mash diet[a] is composed of the following ingredients: buckwheat, hulled gray millet, hulled white (proso) millet, spirulina, chia, alfalfa, clay, sea kelp, anise, natural sources of vitamins, minerals and trace minerals. (Can be wrapped in thin slices of banana for feeding). This mash has been clinically correlated with the abatement of pruritis in several birds suspected to be suffering from food allergies. Once the mash has been accepted, the proportion of banana can be gradually decreased until eliminated. However, a bird could potentially be allergic to any dietary ingredient.

DIETARY ANTIFEEDANTS AND XENOBIOTICS

As the practice of organic farming diminishes the detrimental impact of pesticides on wildlife and their habitats, the consumption of organic products minimizes the need for animals in captivity to modify and detoxify antifeedants in their diet. Evaluating the potential burden that residual pesticides in non-organic ingredients place on a bird requires understanding the biochemical complexities of detoxification mechanisms that enable animals to process these potentially harmful chemicals. This can apply equally to foodstuffs with high concentrations of antifeedants, such as alkaloids, cardiac glycosides and phenolic compounds, that require detoxification. However, pesticide tolerance has not been evaluated in

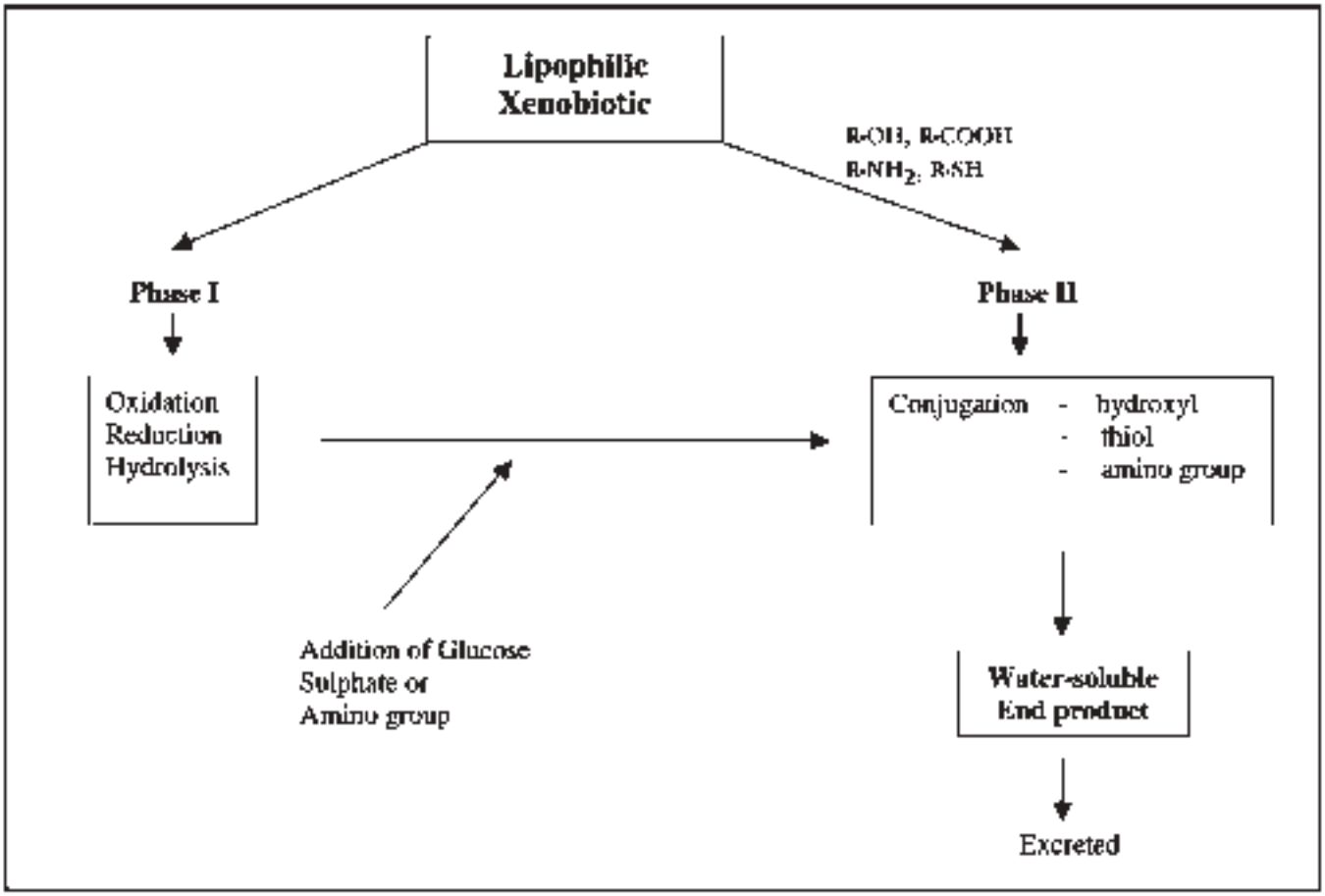


Fig 4.2.32 | Phase I and II detoxification of xenobiotics.

companion birds. Clinically significant improvements in health and productivity have been reported for birds maintained on organic diets.[67]

Actions of Chemical Contaminants

Most toxicological injury results in cellular damage. The toxic response to any specific chemical is the result of dysfunction of relatively few basic biologic processes. Normal processes can be suppressed or stopped completely, or they can be enhanced beyond normal physiologic limits and, in turn, affect other systems dependent on their controlled functions. Cellular responses to chemical toxins occur through both structural and metabolic mechanisms in the cell. A single response or a number of actions can be elicited from an individual pesticide (Table 4.2.15).

Detoxification of Xenobiotics

Xenobiotics are pharmacologically, endocrinologically, or toxicologically active substances not endogenously produced and therefore foreign to an organism. Pesticides acting as estrogens (xenoestrogens) are a common subject of discussion at wild bird disease seminars[33,34,40,41,103] (Fig 4.2.32).

The detoxification of xenobiotics is a biphasic process carried out by a suite of non-specific microsomal enzymes referred to as mixed function oxidases (MFOs) that act primarily in the endoplasmic reticulum. The detoxification process converts a lipophilic compound into a highly water-soluble product that is suitable for excretion in urine or feces. The two phases of detoxification place different demands on nutrient stores in the body. Therefore, the ability to digest and process foreign chemicals may vary from one individual to another, depending on species, feeding ecology, gender and developmental stage. If liver function is diminished from any other disease process, detoxification capability may also be affected.

Nutritional Requirements for Detoxification

Dietary nutritional deficiencies, such as minerals (calcium, copper, iron, magnesium, zinc), vitamins (E, C and the B complex) and proteins, can limit the chemicals necessary for the synthesis of enzymes or conjugating agents. Energy deficits induced by fasting decrease the activity of xenobiotic metabolizing enzymes. Protein malnutrition may also impair enzyme synthesis, MFO activity and hepatic glutathione concentration.

Detoxification may also have an impact on ascorbic acid (vitamin C) levels, as glucuronate is a precursor in the

Table 4.2.16 | Behavioral Traits Attributed to Nutritional Inadequacy

History	Signalment Individualistic	Treatment
Overly permissive owners **Behavior** •Craves sweets/fats **Diet** • Poorly balanced 30-60% warm table foods and numerous treats • Seed based **Husbandry** • Poor	**Distrustful** **Obsessive/Compulsive** **Depressed, Separation Anxiety** **Reproductive** • Masturbation, hiding, nest building, egg laying **Feather Picking, Mutilation** **Biting, Screaming** **Sweet/Lovable Clinging** **Aggression**	**Formulated Diet** • Nutritionally balanced **Environment** • Adjust, move/redecorate cage **Medication** • Human chorionic gonadotropin • Lupron, dexamethasone **Behavior** • Modification, tolerance training, exercise

biosynthesis of ascorbate in most animals. Deficiencies of ascorbate reduce the ability to detoxify foreign chemicals. Stress may also increase the rate of ascorbate metabolism. Any changes in environmental attributes (structural or nutritional) may impact on levels of ascorbate and thus a species' ability to detoxify antifeedants in its diet.

By consuming several different plant foods, and so using various rate-limiting detoxification pathways, an animal can ingest larger amounts of food per unit time enabling them to obtain more energy and nutrients. While varying the diet may be beneficial to some animals, there is the risk of exposure to a greater variety of biologically active compounds. If more than one xenobiotic is absorbed there can be an additive, synergistic or antagonistic interaction between them. Therefore, the ability to utilize a single food substrate or to incorporate various plant species into the diet will be based on the individual.

Implications for Pesticide Residues in Avian Diets

Birds maintained in captivity are confronted with a different set of stresses than their wild counterparts. In addition to adjusting to the stress of being maintained in a confined environment, they are generally supplied with foods that have been domesticated for the human palate or formulated foods composed of foreign ingredients. There are various natural antifeedants in foods that the avian system must detoxify, and the addition of residual pesticides in non-organic foods adds to this burden. Tannins differ in their actions. Condensed tannins have the potential to bind to proteins, rendering them insoluble and unavailable for assimilation; hydrolysable tannins do not bind to proteins, but require detoxification, placing a drain on nutrients associated with the detoxification system. A nutritionally balanced diet is still not optimal if levels of feed contaminants require extensive detoxification.

The toxicity of certain pesticides may be underestimated, as cumulative effects or delayed toxicity can occur long after exposure. This confuses the interpretation of signs credited to pesticide exposure. Synergistic effects may also be observed, if one chemical affects the solubility, binding, metabolism or excretion of another. Therefore, while studies on the effects of individual pesticides may not indicate any detrimental actions, a combination of two or more pesticides may enhance toxic actions. Potentiation occurs when one chemical enhances the toxicity of another, even though the toxicity of the potentiator is minor or nonexistent. See Chapter 11, Low-risk Pest Management for actions one might consider in avoiding pesticides.

Pesticides and Behavioral Abnormalities

Levels of pesticide contamination in bird foods composed of non-organic ingredients have yet to be evaluated. Behavioral abnormalities associated with pesticide exposure include: reduction in courtship behavior[33,34,74], reluctance of females to take food from males, changes of activity patterns in males[40], reduced levels of nest defense[33,34,41], alterations of incubation behavior, decreased parental attentiveness resulting in increased embryonic mortality[33,41,82], decreased time feeding young, fewer sorties to feed young and increased time away from nests.[33,34,82]

Eggshell thinning induced by dichlorodiphenyldichloroethylene (DDE) is species specific. Organochlorines reduce levels of androgens in males and estrogen and progesterone in females. Levels of thyroxine in both sexes decrease in a dose-related fashion. There are also links between hyperthyroidism, PCB's and dichlorodiphenyltrichloroethane (DDT).[41] Chlorinated hydrocarbons induce changes in the metabolism of steroid hormones by mixed function oxidases (MFO).[82]

BIRD BEHAVIOR

A range of behavioral traits can be attributed to nutritionally imbalanced diets, inappropriate dietary ingredients, and dietary exposure to pesticides (Table 4.2.16). These include changes to vocalization patterns and breeding behavior. Behavioral traits of birds also need to be evaluated when converting birds to formulated diets (see Chapter 3, Concepts in Behavior, Section III Pubescent and Adult Psittacine Behavior). The subjects of high-fat and high-sugar diets along with elevated sodium are presented.

Dietary Influences on Vocalization Patterns

Changes in vocalization patterns have been reported in cockatiels maintained on diets with deficient or excessive levels of vitamin A.[58] Excessively high levels of vitamin A (100,000 IU/kg) increase the number of vocalizations and reduce the peak frequency of vocalizations, while moderately excessive levels of vitamin A (10,000 IU/kg) result in a reduction in peak amplitude and total power. These changes may influence breeding behavior, social interactions and responses of adults to begging behavior of chicks.

Diet and Behavioral Changes

Reproductive behavior, normally regulated by photoperiod, can be influenced by dietary components. Additional seeds stimulate breeding behavior in African grey parrots.[69] Endocrine malfunction has been implicated in anecdotal studies of male budgerigars on all-seed diets that display continuous feeding behavior towards a mirror. Reducing the synthetic vitamin A content of food fed to lorikeets reduced defecation in nest boxes (Unpublished data, D. McDonald). See Chapter 3, Concepts in Behavior, Section III Pubescent and Adult Psittacine Behavior.

DIET CONVERSION CHALLENGES

Modifying a bird's diet is one of the biggest behavioral challenges. Most issues can be overcome with patience and perseverance. Educating the owner about the benefits of a formulated diet versus a seed-based diet is the first challenge. Feeding the new dietary items early in the morning when the birds are most hungry is beneficial, but dietary changes should be undertaken gradually. If birds continue to have problems with acceptance of new dietary items, placement near birds that are already feeding on similar foods is beneficial as birds usually mimic feeding habits of other birds. Mixing the new diet with a favorite fruit can be helpful; mushy fruit works best as it sticks to the formulated food. Remove the fruit after 4 to 6 hours to avoid consumption of spoiled food.

An important concern is the bird's refusal to eat or significant weight loss. Although weighing the bird in grams on a daily basis is the best method of monitoring adequate food consumption, monitoring droppings can also indicate if the bird is eating enough. Prior to the diet change, note the number and character of the droppings (color, amount, liquid, form, shape, lack of odor, staining). See examples in Chapter 6, Maximizing Information from the Physical Examination. Any change in volume and number of droppings, usually a dramatic decrease in amount, indicates insufficient consumption. Character of droppings will change as the bird consumes more formulated diet. Weight fluctuations greater than 10% are considered problematic. Even if provided with surplus food, birds can starve to death if they don't consume the food offered. Therefore, it is imperative to immediately return a bird to its original diet if it refuses to eat the new diet. The following guidelines should be given to owners when a diet conversion is being initiated.

Tips for Converting Birds to a Formulated Diet

- *Visit the avian veterinarian for a general health exam to decide if the bird is healthy enough to undergo a diet change at this time.*
- *Discuss which formulated product is best for the bird.*
- *Determine the goal body weight that is appropriate for the bird.*
- *Purchase a gram scale and learn how to use it correctly.*
- *Weigh the bird at the same time every morning for a week to establish normal fluctuations in weight. Report any serious fluctuations (10% or more) to the avian veterinarian.*
- *Mix half formulated diet and half the old diet. Expect the bird to exhibit negative behavior by throwing the pellets/nuggets at owner, screaming, yelling, and having tantrums. Talking to the bird may soothe it. As the bird starts to eat the formulated diet, gradually reduce the amount of the old diet and increase the proportion of the new diet.*
- *Remove all perches from cage so the bird is forced to sit on the food dish or put the bird in a plastic or glass box (aquarium) and sprinkle food on the floor. Provide a small water container—no toys.*
- *Place formulated food on a mirror located on the floor* (Figs 4.2.33a,b).
- *Place the bird in a cage with another bird that is already consuming a formulated diet. Do not provide any seed and separate birds at night. If the bird has not consumed any of the formulated food by evening, provide it with seed.*
- *Once it starts to eat, place a bowl of formulated food near the highest perch.*
- *Feed the old diet for 30 minutes in the morning, remove and replace with formulated food for remainder of the day. Feed the old diet for 30 minutes at night if formulated diet is not eaten.*
- *Grind formulated food in a blender (or purchase a mash product) and mix crushed millet in with the mash.*
- *Using a less palatable seed (hulled white millet), break the seeds up in a blender with the pellets. After a few days use less ground millet.*
- *Thoroughly mix bird's favorite fruit into formulated diet so bird gets a mouthful of new diet with fruit.*
- *Under the guidance of an avian veterinarian, try returning to hand feeding a juvenile formula with a*

Fig 4.2.33a | Budgerigar that has become infatuated with its image in a mirror.

Fig 4.2.33b | Cockatiel in a container with only a mirror. Food is placed on the mirror. Return the bird to its own cage at night for regular diet and water. Replace in mirror box next day after having at least 30 minutes to eat regular diet and drink. Diminish the time with seeds daily until weaned.

syringe and then wean to formulated food.

- *Very small particles of formulated foods seem more acceptable to small birds*

If patience and persistence doesn't pay off, it is best to board the bird under clinical supervision. The veterinarian can convert the bird to the new diet while carefully monitoring weight and health of the bird. Most birds switch diets very quickly when removed from the "comfort" of the home environment. Avian veterinarians generally have more experience with dietary changes. Birds should be left for a sufficient period of time to ensure conversion is complete ie, held for 2 to 10 days. Wait until the bird maintains body weight for at least two days after normal diet has been totally removed. It is best that the bird is not returned to the same routine at home (ie, move cage, redecorate cage, don't place in kitchen at mealtime).

Some of the IDC clinical signs are idiosyncratic in nature. A 20-year old cockatiel maintained solely on sunflower seeds or a 50-year-old Amazon provided with chicken bones, seeds, nuts and table foods may never present with the advanced signs of a clinical nutritional disorder. However, this should be seen as the exception and not the rule. Additionally, individual birds may develop clinical signs of the IDC at different rates or to varying degrees.

While problems associated with the integument can precede breakdown of other systems, they are seldom recognized in the early stages of the IDC. Therefore, digestive upsets associated with gastroenteritis and ileus are more likely to instigate an investigation into yeast or coliform bacterial infection rather than dietary history. A proliferation of meetings, publications, investigative consultancies, analytical laboratories and therapies aimed at the presenting problems over the past 30 years have all contributed to a better understanding of illnesses in pet birds. While secondary problems associated with any one of dozens of common, yet serious, secondary pathogens still warrant attention, preventive medicine remains the most effective therapy.

Products in Text

a. Adult Lifetime Mash. HBD International, Inc., Brentwood, TN, 1-800-346-026
b. Harrison's Bird Foods. HBD International, Inc., Brentwood, TN, 1-800-346-0269
c. Avian Enzyme- HBD International, Inc., Brentwood, TN, 1-800-346-0269.
d. Prozyme- www.prozymeproducts.com
e. Hepasan- www.vetpharm.unizhoch/tpp/oooo
f. Ultraclear. Metagenics, 1152 Ensell Rd. Lake Zurich, IL. www.metagenics.com
g. Parrot Specific Lactobacillus (Munich)- www.janeczek.de
h. Bird Builder, AVIx - www.exoticdvm.com

References

1. Adams RL Pratt DE, Lin JH, Stadelman WJ: Introduction of omega-3 polyunsaturated fatty acids to modulate arachidonic acid metabolism. Poultry Science 68S1:166A, 1989.
2. Ahmed F, Khan MR, et al: Concomitant supplemental vitamin A enhances the response to weekly supplemental iron and folic acid in anemic teenagers in urban Bangladesh. Am J Clin Nutr 74(1):108-115, 2001.
3. An B K, Nishiyama H, et al: Dietary safflower phospholipid reduces liver lipids in laying hens. Poultry Science 76(5):689-695, 1997.
4. Angel R, Ballam G: Dietary protein effect on parakeet reproduction, growth, and plasma uric acid. 1st An Conf NA Group, Toronto, 1995.
5. Avery P: Preliminary bacteriology in Psittaciformes birds with some procedures in the identifiction of the bacteria present. Proc Assoc Avian Vet 1982 58-90.
6. Ayres S J, Mihan R, et al: Synergism of vitamins A and E with dermatologic applications. Cutis 23(5):689-690, 1979.
7. Bain SD, Newbrey JW, et al: Biotin deficiency may alter tibiotarsal bone growth and modeling in broiler chicks. Poultry Science 67(4):590-595, 1988.
8. Balnave D, Pearce J: Lactate administration and fatty liver and kidney syndrome development in biotin-deficient chicks. British Poultry Science 20(1):109-116, 1979.
9. Bangert RL, Cho BR, Widders PR, et al: A survey of aerobic bacteria and fungi in the feces of healthy psittacine birds. Avian Dis 29:951-962, 1985.
10. Bannister DW: The biochemistry of fatty liver and kidney syndrome. Biotin-mediated restoration of hepatic gluconeogenesis in vitro and its relationship to pyruvate carboxylase activity. Biochemistry Journal 156(1):167-173, 1976.
11. Bannister DW, O'Neill IE, et al: The effect of biotin deficiency and dietary protein content on lipogenesis, gluconeogenesis and related enzyme activities in chick livers. British J Nut 50(2):291-302, 1983.
12. Barros MP, Pinto E, et al: Astaxanthin and peridinin inhibit oxidative damage in Fe(2+) - loaded liposomes: scavenging oxyradicals or changing membrane permeability? Biochem Biophys Res Commun 288(1):225-232, 2001.
13. Bavelaar FJ, Beynen AC: Influence of amount and type of dietary fat on plasma cholesterol concentrations in African grey parrots. J Applied Res Vet Med 1:1-8, 2003.

14a. Bavelaar FJ, Beynen AC: Severity of atherosclerosis in parrots in relation to fatty and composition of breast muscle on adipose tissue - biomarkers of fatty acid intake. Avian Dis 47(3):566-577, 2003.

14b Cowell CS: Making commercial pet foods. *In* Hand et al (eds) Small Animal Clinical Nutrition. Mark Morris Institute. Topeka, Kansas 2000 p130.

14c. Devo RS, Trogdon M, Flammer K: Preliminary Assessment of the Effect of Diet and L-carnitine supplementation on Lipoma Size and Bodyweight in Budgerigars (*Metopsittacus undulatus*). J Avain Med Surg. 2004; 18(1):12-18.

15. Beck, MA. Antioxidants and viral infections: Host Immune response and viral pathogenicity. JAM Coll. Nutr Oct; 20 (5suppl):384s-388s; discussion 396s-397s, 2001.
16. Bezard J, Blond JP, et al: The metabolism and availability of essential fatty acids in animal and human tissues. Reprod Nutr D 34(6):539-568, 1994.
17. Bhaskaram P: Micronutrient malnutrition, infection, and immunity: An overview. Nutr Rev May; 60(5 Pt 2):S40-45, 2002.
18. Bowman TA, Jacobson ER: Cloacal flora of clinically normal captive psittacine birds. J Zoo An Med 11:81-85, 1980.
19. Branton SL, Lott BD, et al: Fatty liver-hemorrhagic syndrome observed in commercial layers fed diets containing chelated minerals. Avian Dis 39(3):631-635, 1995.
20. Brayton C: Amyloidosis, hemochromatosis, and atherosclerosis in a roseate flamingo (*Phoenicopterus ruber*). An N Y Acad Sci 653:184-90, 1992.
21. Brown PA: Pros and cons of fecal Gram's stain. Proc Assoc Avian Vet 1996, p 331.
22. Campbell T: The Gram's stain in avian practive. J of Avian Med and Surgery 14 (3):194-198, 2000.
23. Cerolini S, Surai P, et al: Lipid composition of semen in different fowl breeders. Poult Avian Biol Revs 8(3/4):141-148, 1997.
24. Clubb SJ, KarpinskiL: Aging in macaws Proc Assoc Avian Vet 1992:83-86.
25. Cork SC: Iron storage diseases in birds. Avian Pathol 29:7-12, 2000.
26. Diaz GJ, Squires EJ, et al: Effect of selected dietary antioxidants on fatty liver-hemorrhagic syndrome in laying hens. Br Poult Sci 35(4):621-629, 1994.
27. Diaz GJ, Squires EJ, et al: The use of selected plasma enzyme activities for the diagnosis of fatty liver-hemorrhagic syndrome in laying hens. Avian Dis 43(4):768-773, 1999.
28. Dimitrov A, Antonov S, Stoianov P, Petrova L, Aleksandrova E: (Fatty liver syndrome in laying hens) Vet Med Nauki; 17(1):81-89, 1980.
29. Dorrestein GM, Mete A, et al: Hemochromatosis/iron storage: New developments. Proc Assoc Avian Vet, 2000, pp233-238.
30. Dreyfuss ML, Stoltzfus RJ, et al: Hookworms, malaria and vitamin A deficiency contribute to anemia and iron deficiency among pregnant women in the plains of Nepal. J Nutr 130(10):2527-2536, 2000.
31. During A, Fields M, et al: Intestinal beta-carotene 15,15'-dioxygenase activity is markedly enhanced in copper-deficient rats fed on high-iron diets and fructose. Br J Nutr 84(1):117-124, 2000.
32. Flora K, Hahn M, Rosen H, Benner K: Milk thistle (*Silybum marianum*) for therapy of liver disease. Am J Gastroenterol 93(2), 139-143, 1998.
33. Fox GA, Gilman AP, Peakall DB, et al: Behavioral abnormalities of nesting Lake Ontario herring gulls. J Wildl Manage 42:477-483, 1978.
34. Fyfe RW, Risebrough RW, Walker W: Pollutant effects on the reproduction of the prairie falcons and merlins of the Canadian prairies. Can Field Nat 90:346-355, 1976.
35. Garner M, West G, et al: Five lorikeets with hemochromatosis associated with high dietary concentrations of dietary iron. Proc Assoc Avian Vet, 2000 pp 215-216.
36. Gerlach H, Enders F, et al: Discussion of the increased iron content in the liver of some bird species (particularly parrots). M.A.R.E., Toledo, Ohio, 1998.
37. Gerlach H: Bacteria. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994.
38. Glunder G: Influence of diet on the occurrence of some bacteria in the intestinal flora of birds. Dtsch Tierarztl Wochenshr,109 (6):266-270, 2002.
39. Goto S, Kogure D, et al: Efficient radical trapping at the surface and inside the phospholipid membrane is responsible for highly potent antiperoxidative activity of the carotenoid astaxanthin. Biochim Biophys Acta 1512(2):251-258, 2001.
40. Grue CE, Shipley BK: Interpreting population estimates of birds following pesticide applications: Behavior of male starlings exposed to an organophosphate pesticide. Studies Avian Biology 6:292-296, 1981.
41. Grue CE, Powell GVN, McChesney MJ: Care of nestlings by wild female starlings exposed to an organophosphate pesticide. J Appl Ecol 19(2):327-335, 1982.
42. Haghighi-Rad F, Polin D: The relationship of plasma estradiol and progesterone levels to the fatty liver hemorrhagic syndrome in laying hens. Poult Sci 60(10):2278-2283, 1981.

43a. Haghighi-Rad F, Polin D: Lipid: the unidentified factor for alleviating fatty liver syndrome. Poult Sci 61(10):2075-2082, 1982.

43b. Hamilton RJ: The chemistry of rancidity in foods. *In* Allen JC and Hamilton RJ (eds): Rancidity in Foods. 3rd ed Aspen Publishers, Inc. Gaithersburg, Maryland, 1999, p 7, 146, 148,154.

44. Hargis P S, Van Elswyk ME, et al: Dietary modification of yolk lipid with menhaden oil. Poult Sci 70(4):874-883, 1991.
45. Harrison GJ: Preliminary field study of fecal Gram's stain results in two free-ranging Australian parrot species. Exotic DVM, 4:10-11, 2003.
46. Harrison GJ: Pet bird practice. Proc VMA Ann Conf, Corpus Christi, TX 1983.
47. Harrison G J, McDonald DL: A pilot field study evaluating the fecal Gram's stain technique on free-ranging cockatoos. ECAMS, Tenerife, 2003.
48. Hartroft WS: Proc Am Soc Exp Biol 14: 655, 1955.
49. Hussein ZF, Sundram K., Nor, R. Md., Pathmanathan. R, Wong KT Effect of dietary palm oil and its minor components on dimethylhydrazine (DMH) induced colon cancer in rats. Carotino Nutritional Research (no date).
50. Jefferies DJ: Induction of apparent hyperthyroidism in birds fed DDT. Nature 222(193):578-579, 1969.
51. JensenPN, Sorensen G, Engelsen SB, Bertelsen G: Evaluation of quality changes in walnut kernals (Juglans Regia L.) by VIS/NIR spectroscopy. J Agric Food Chem 49 (12):5790-5796, 2001.
52. Johnson JA, Phalen DN, et al: Atherosclerosis in psittacine birds. Proc Assoc Avian Vet, 1992 p 87-93.
53. Johnston GB: Iron storage disease (hemochromatosis) in softbilled birds. AFA Watchbird. 5, 1999.
54. Joyner KL: The Use of Gram's stain results in avian medicine. Proc Assoc Avian Vet, 1991, 78-97.
55. Joyner KL: Interpretation of yeast on a Gram's stain. J Assoc Avian Vet 3(2):69, 1989.
56. Kelso KA, Cerolini S, et al: The effects of dietary supplementation with docosahexaenoic acid on the phospholipid fatty acid composition of avian spermatozoa. Comp Biochem Physiol 118B(1):65-69, 1997.
57. Kincaid AL, Stoskopf MK: Passerine dietary iron overload syndrome. Zoo Biol 6:79-88, 1987.
58. Koutsos EA, Klasing KC: Vitamin A nutrition of cockatiels. Joint Nutrition Symposium, Antwerp, Belgium, 2002.
59. Kramer TR, Briske-Anderson M, et al: Effects of biotin deficiency on polyunsaturated fatty acid metabolism in rats. J Nutr 114(11):2047-2052, 1984.
60. Lane R: Use of Gram's stain for bacterial screening. J Assoc Avian Vet 4(4):214-217, 1990.
61. Lavender OA: Nutrition and newly emerging viral diseases: An overview. J Nutr May, 127(5suppl):948S-950S, 1997.
62. Layrisse M, Garcia-Casal MN, et al: New property of vitamin A and beta-carotene on human iron absorption: effect on phytate and polyphenols as inhibitors of iron absorption. Arch Latinoam Nutr 50(3):243-248, 2000.
63. Lin X, Tang Y, et al: Effects of vitamin A and iron supplementation on the improvement of iron status and immunological function in preschool children. Zhonghua Yu Fang Xue Za Zhi 35(6):374-377, 2001.
64. Lombardo MP, Thorpe PA, et al: The beneficial sexually transmitted microbe hypothesis of avian copulation. Behavioral Ecology 10(3):333-337, 1999.
65. Macwhirter P, Mueller R, Gill J,

Ongoing research report: Allergen testing as a part of diagnostic protocol in self-mutilating Psittaciformes, Proc Assoc Avian Vet 1999, pp 125-129.
66. March BE, MacMillan C: Linoleic acid as a mediator of egg size. Poultry Science 69(4):634-639, 1990.
67. McDonald DL: Improvements in health and productivity of large psittacines fed organic formulated diets low in vitamin A. Zoo Nutr News (No 3), 2003.
68. McDowell LR: Vitamins in Animal and Human Nutrition. Ames, Iowa State University Press, 2000.
69. Meade J: Amazons. Smokey Mountain Cage Bird Society Meeting, 1998.
70a. Morris PJ, Avgeris SE, et al: Hemochromatosis in a greater Indian hill mynah (*Gracula religiosa*): Case report and review of the literature. J Assoc Avian Vet 3(2):87-92, 1989.
70b. Morrison FB: Feeds and Feeding 22nd ed The Morrison Pub Co. Clinton IA, 1959.
71. Murphy J: Psittacine fatty liver syndrome. Proc Assoc Avian Vet, 1992 pp 78-82.
72. Mwanri L, Worsley A, et al: Supplemental vitamin A improves anemia and growth in anemic school children in Tanzania. J Nutr 130(11):2691-2696, 2000.
73. Nakagawa K, Kang SD, et al: Inhibition of beta-carotene and astaxanthin of NADPH-dependent microsomal phospholipid peroxidation. J Nutr Sci Vitaminol (Tokyo) 43(3):345-355, 1997.
74. Newton I, Meek ER, Little B: Breeding ecology of the merlin in Northumberland. Br Birds 71:378-398, 1978.
75. Oloyo R A: Studies on the biotin requirement of broilers fed sunflower seed meal based diets. Arch Tierernahr 45(4):345-353, 1994.
76. Oloyo RA, Ogunmodede BK: Effect of dietary palm kernel oil and biotin on the fatty liver and kidney syndrome in broiler chicken. Beitr Trop Landwirtsch Veterinarmed 27(3):347-355, 1989.
77. Oloyo RA, Ogunmodede BK: The biotin requirement of broilers feed maize-palm kernel meal based ration. Beitr Trop Landwirtsch Veterinarmed 29(2):223-233, 1991.
78. Otten BA, Orosz SE, et al: Mineral content of food items commonly ingested by keel billed toucans (*Ramphastos sulfuratus*). J Avian Med Surg 15(3):194-196, 2001.
79. Pabon ML, Lonnerdal B: Effects of type of fat in the diet on iron bioavailability assessed in suckling and weanling rats. J Trace Elem Med Biol 15(1):18-23, 2001.
80. Peakall DB: Pesticide-induced enzyme breakdown of steroids in birds. Nature 216(114):505-506, 1967.
81. Peakall DB: p.p'-DDT: Effect on calcium metabolism and concentration of estradiol in the blood. Science 168:592-594, 1970.
82. Peakall DB, Peakall ML: Effect of polychlorinated biphenyl on the reproduction of artificially and naturally incubated dove eggs. J Appl Ecol. 10:863-868, 1973.
83. Pearce J, Balnave D: Biotin deficiency and liver metabolism in relation to fatty liver and kidney syndrome. Br Poult Sci 19(4):431-439, 1978.
84. Pearson AW, Butler EJ: Environmental temperature as a factor in the aetiology of fatty liver-haemorrhagic syndrome in the fowl. Res Vet Sci 25(2):133-138, 1978.
85. Pepping J. Milk thistle: *Silybum mariana*. An J Health Syst Pharm 56(12):1195-1197, 1999.
86. Perjesi P, Pinter Z, Gyongyi Z, Ember I: Effect of rancid corn oil on some onco/suppressor gene expressions in vivo: A short-term study. Anticancer Res Jan-Feb;22(1A):225-230, 2002.
87. Pietrangelo A: Physiology of iron transport and the hemochromatosis gene. Am J Physiol Gastrointest Liver Physiol 282(3):G403-414, 2002.
88. Pinero Estrada JE, Bermejo Bescos P, et al: Antioxidant activity of different fractions of *Spirulina platensis* protean extract. Farmaco 56(5-7):497-500, 2001.
89. Polin D, Wolford JH: Role of estrogen as a cause of fatty liver hemorrhagic syndrome. J Nutr 107(5):873-886, 1977.
90a. Roland DA, Farmer M, et al: Calcium and its relationship to excess feed consumption, body weight, egg size, fat deposition, shell quality, and fatty liver hemorrhagic syndrome. Poult Sci 64(12):2341-2350, 1985.
90b. Reaven P Metabolic syndrome. J Insur Med 2004; 36(2):132-42 Review.
91. Rosskopf WJ, Woerpel RW, et al: Iron storage disease (hemochromatosis) in a citron-crested cockatoo and other psittacine species. Proc Assoc Avian Vet, 1992 pp 98-107.
92. Said HM, Derweesh I: Carrier-mediated mechanism for biotin transport in rabbit intestine: studies with brush-border membrane vesicles. Am J Physiol 261:R94-R97, 1991.
93. Sayle R: Evaluation of droppings. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery, Philadelphia, PA, WB Saunders Co, 1986 pp153-156.
94. Schmidt MK, Muslimatun S, et al: Vitamin A and iron supplementation of Indonesian pregnant women benefits vitamin A status of their infants. Br J Nutr 86(5):607-615, 2001.
95. Schuman BE, Squires EJ, et al: Effect of dietary flaxseed, fax oil and n-3 fatty acid supplement on hepatic and plasma characteristics relevant to fatty liver haemorrhagic syndrome in laying hens. Br Poult Sci 41(4):465-472, 2000.
96. Schoemaker NJ, Beynen AC: Composition of commercial feeds for mynah birds with particular attention to the iron content. Tijdschr Diergeneeskd 126(19):620-623, 2001.
97. Scott ML Nesheim, MC et al: Nutrition of the Chicken. Ithaca, New York, 1982.
98. Shivaprasad HL, Crespo R, Duscher B, Lyna S, Wright L: Myopathy in brown pelicans (*Pelicanus occidentalis)* Associated with rancid food. Vet Rec; 150(10):307-311, 2002.
99. Sigurdson SC, Joyner KL: The microflora of wild yellow-naped Amazon (*Amazona auropalliata*) chicks in Guatemala. Proc Assoc Avian Vet, 1995: pp123-124.
100. Simpson CF, Harms RH: Aortic atherosclerosis in nonlaying hens with fatty liver syndrome. Avian Dis 27(3):652-659, 1983.
101. Slifka KA: Visceral gout in birds and reptiles. 9th Dr Scholl Conf Nutr Captive Wild Animals, Chicago, 1991.
102. Stanford M: Effects of dietary change on fecal Gram's stains in the African grey parrot. Exotic DVM 4(6):12-13, 2003.
103. Summer CL, Giesy JP, et al: Effects induced by feeding organochlorine-contaminated carp from Saginaw Bay, Lake Huron, to laying White Leghorn hens: I. Effects on health of adult hens, egg production, and fertility. J Toxicol Environ Health 49(4):389-407, 1996.
104. Uni Z, Zaiger G, et al: Vitamin A deficiency interferes with proliferation and maturation of cells in the chicken small intestine. Br Poult Sci 41(4):410-415, 2000.
105. Whitehead CC, Bannister DW, et al: Metabolic changes associated with the occurrence of fatty liver and kidney syndrome in chicks. Br J Nutr 40(2):221-234, 1978.
106. Wilson GH, Fontenot DK, Greenacre, CB, et al: Enteric clostridial colonization in psittacine birds. Compendium 24(7):550-554, 2002.
107. Worell AB: Further investigations in rhamphastids concerning hemochromatosis. Proc Assoc Avian Vet, 1993, pp 98-107.

CHAPTER

5

Calcium Metabolism

MICHAEL STANFORD, BVSc, MRCVS

Calcium plays two important physiological roles in the avian subject. First, it provides the structural strength of the avian skeleton by the formation of calcium salts. Second, it plays vital roles in many of the biochemical reactions within the body via its concentration in the extracellular fluid.[3] The control of calcium metabolism in birds has developed into a highly efficient homeostatic system, able to quickly respond to increased demands for calcium required for both their ability to produce megalecithal eggs and their rapid growth rate when young.[2,3] Parathyroid hormone (PTH), metabolites of vitamin D_3 and calcitonin, regulate calcium as in mammals, acting on the main target organs of liver, kidney, gastrointestinal tract and bone.[2,3,11,24] Estrogen and prostaglandins also appear to have an important role in calcium regulation in the bird.[2,3]

There are distinct differences between the mammalian and avian systemic regulations of calcium. The most dramatic difference between the two phyletic groups is in the rate of skeletal metabolism at times of demand. The domestic chicken will respond to hypocalcemic challenges within minutes compared with response to similar challenges in mammals that can take over 24 hours.[11] This is best demonstrated by an egg-laying bird where 10% of the total body calcium reserves can be required for egg production in a 24-hour period.[10] This calcium required for eggshell production is mainly obtained from increased intestinal absorption and a highly labile reservoir found in the medullary bone, normally visible radiographically in female birds. The homeostatic control of the medullary bone involves estrogen activity.[2] Due to the rapid metabolic responses in the avian skeleton, it has become a common model for skeletal studies concerning the regulation of calcium.

Abnormalities of calcium metabolism are common in the poultry industry, leading to poor production and growth defects, in particular, tibial dyschondroplasia in broiler chickens kept indoors. Due to the economic status of the poultry industry, calcium metabolism has been well researched in production birds, including the assessment of the importance of dietary calcium, vitamin D_3 and their interaction with ultraviolet light, especially in the UV-B spectrum (315-280 nm).[1,4,14,24]

In captive pet birds, disorders of calcium metabolism also are common, ranging from osteodystrophy in young birds (due in part to the greater calcium requirement in young growing birds) to hypocalcemic seizures and egg binding in adults.[5,6,7,16] Although African grey parrots (*Psittacus erithacus*) are considered to be especially susceptible to disorders of calcium metabolism, problems have been reported in a variety of captive species. The husbandry requirements with respect to calcium have been poorly researched in pet birds in the past, and much of our present knowledge is extrapolated from work with poultry.

Calcium Regulation in Birds

CALCIUM

Calcium exists as three fractions in the avian serum: 1) the ionized salt, 2) calcium bound to proteins, and 3) as complexed calcium bound to a variety of anions (citrate, bicarbonate and phosphate). Ionized calcium is the physiologically active fraction of serum calcium, with a role to play in bone homeostasis, muscle and nerve conduction, blood coagulation and control of hormone secretions such as vitamin D_3 and parathyroid hormone. The majority of the protein-bound calcium is bound to albumin and considered to be physiologically inactive. Any change in serum albumin levels will directly affect the total calcium level. The binding reaction is strongly pH-dependent, so an increase or decrease in pH will respectively increase or decrease the protein-bound calcium fraction. The significance of the small, complexed calcium fraction is unknown at the present time, but any disease state that affects the levels of the binding anions would be expected to significantly alter calcium levels.[8,21,22]

The ionized calcium level appears to be maintained within a tight range in the normal individual compared with the total calcium level, which fluctuates with serum protein concentrations, and any major change in the serum ionized calcium level is likely to be of pathologic significance. Ionized calcium concentrations are regulated by the interaction of PTH, vitamin D_3 metabolites and calcitonin in response to changing demands.

VITAMIN D_3

The vitamin D_3 metabolism of birds has been extensively reviewed.[1,3,4,24,25] Following the identification of vitamin D_3 metabolic pathways, vitamin D_3 now is considered to be a steroid hormone exhibiting feedback relationships in response to calcium requirements.[8] The main role of vitamin D_3 is in the control of bone metabolism by strictly regulating mineral absorption, but more recently it has been found to have profound effects on the immune system, skin and cancer cells.[1] Due to the importance of the vitamin's role in bone development and the requirement for UV light in the metabolic conversion of provitamin D_3 to vitamin D_3, the commercial availability of dietary vitamin D_3 has been essential to allow the indoor production of poultry.[4] Vitamin D occurs naturally in plants as ergocalciferol (vitamin D_2) and this will function in mammals as well as vitamin D_3, but birds do not respond well to dietary vitamin D_2. This is due to increased renal clearance of vitamin D_2 rather than lack of intestinal absorption.[10]

It has been established that the domestic chicken secretes 7-dehydrocholesterol (provitamin D_3) onto the featherless skin. It has recently been shown that there are ten times more provitamin D_3 on the featherless leg skin than on the back, indicating the importance of this area for vitamin D_3 metabolism.[25] Conversion of the provitamin D to cholecalciferol (vitamin D_3) occurs by a UV-B light-dependent isomerization reaction. Cholecalciferol is a sterol prohormone that is subsequently activated by a two-stage hydroxylation process.

Cholecalciferol is initially metabolized to 25-hydroxycholecalciferol in the liver; 25-hydroxycholecalciferol is transported to the kidney via carrier proteins and converted to either 1,25-dihydroxycholecalciferol or 24,25-dihydroxycholecalciferol, the active metabolites of cholecalciferol in the domestic fowl. The most significant active metabolite of vitamin D_3 in domestic chickens is 1,25-dihydroxycholecalciferol, which displays a hypercalcemic action.[9,21] While not significant in mammals, 24,25-dihydroxycholecalciferol is thought to have a special active role in the laying hen.[2] The synthesis of 1,25- dihydroxycholecalciferol is tightly regulated by PTH, depending on the calcium status of the bird. The metabolite regulates calcium absorption across the intestinal wall by inducing the formation of the carrier protein calbindin-D28k. The presence of this protein reflects the ability of the intestine to absorb calcium. Calbindin-D28k also is found in the wall of the oviduct, and levels rise during egg laying, although this process is not directly related to the actions of 1,25-dihydroxycholecalciferol. Bone formation is stimulated by 1,25- dihydroxycholecalciferol, which induces osteoclastin production from osteoblasts. In egg-laying birds, 30 to 40% of the calcium required

for eggshell formation is acquired from medullary bone. The control of this highly labile pool of calcium involves both 1,25- dihydroxycholecalciferol and estrogen activity. The function of vitamin D_3 is reliant on the presence of normal vitamin D_3 receptors. The receptors have been found in bone, skin, skeletal muscle, gonads, pancreas, thymus, lymphocytes and pituitary gland.[2,3,4,8,11,14,24,25]

If vitamin D_3 is supplied in the diet, it can be absorbed with 60 to 70% efficiency. In most situations, there is a compromise between the amount of dietary vitamin D_3 administered (on the basis of economy and toxicity) and UV light provided for natural vitamin D_3 formation.[10]

PARATHYROID HORMONE (PTH)

The parathyroid glands produce PTH from the chief cells in response to a low ionized calcium level.[8,10] As in mammals, PTH has an essentially hypercalcemic action in birds, and if a parathyroidectomy is performed in quail, the birds respond with severe hypocalcemia unless calcium is given in the diet.[3] Birds appear more sensitive to PTH than mammals, reacting to intravenous injections of the hormone within minutes with a rise in blood calcium levels.[2,3,11] The blood ionized calcium level feeds back on the chief cells in the parathyroid gland to tightly control secretion of the hormone. The hormone uses the kidney and the bone as its main target organs in birds.[3] In the bone, the hormone has rapid effects measurable within 8 minutes of administration compared with hours in mammals, so PTH is probably at least partially responsible for the speed of calcium metabolism in the bird.[2]

PTH directly stimulates osteoclasts to resorb bone. The hormone also will actively stimulate osteoblast activity, and it is thought that PTH-stimulated osteoblasts regulate osteoclast activity, providing the precise control system necessary in avian skeletal turnover.[2,3]

PTH binds to osteoclasts, increasing bone resorption by stimulating their metabolic activity and division. PTH also has direct influence on both calcium and phosphorus excretion in the bird. Calcium excretion is increased and phosphorus decreased following parathyroidectomy. These changes can be reversed by injections of PTH.

PTH has a different structure in birds than in mammals. It consists of an 88-amino acid chain, compared with 84 in mammals, and there is a lack of homology between the two molecules. The greatest similarity is found in the 1-34 segment of the chain, which also is responsible for much of the activity of the hormone.[3,8] Unfortunately, this segment has a short half-life, and mammalian assays tend to assess the middle and carboxyl-terminal regions of the amino acid chain where homology with avian PTH is poor.[8] This may explain why PTH has appeared difficult to measure in the avian subject and is poorly researched at the present time, although circulating levels are believed to be low compared with mammals. An inverse relationship has been found between PTH concentrations and ionized calcium levels in the laying chicken, indicating that PTH has at least an important role in calcium regulation during this period. This response is very efficient during egg production, with PTH levels increasing due to the greater demand for calcium. A recent study in African grey parrots investigating hypocalcemia has used a mammalian 1-34 PTH enzyme-linked immunosorbent assay with consistent results.[18]

PARATHYROID HORMONE-RELATED PEPTIDE (PTHRP)

PTHrP is a second member of the PTH family, originally discovered as a cause of hypercalcemia in malignancy in man and now the subject of much clinical research. PTHrP has three isoforms of 139, 141 and 173 amino acids, all with identical sequences through to amino acid 139. The hormone has distinct structural homology with PTH, suggesting a common ancestral gene. There is distinct homology between the structure of mammalian and avian PTHrP in the 1-34 segment, as has been found with PTH. PTH and PTHrP share a common receptor. Many tissues in the chicken embryo contain levels of PTHrP. In chickens as in man, PTHrP is thought to play many regulatory and developmental roles in a variety of tissues. Concentrations of PTHrP have been shown to rise in the shell gland of the chicken during the calcification cycle, affecting smooth muscle activity in the gland. Levels of PTHrP return to normal once the egg has been laid. PTHrP has been shown to have effects on bone resorption in the chicken embryo.[3]

ULTIMOBRANCHIAL GLANDS

The ultimobranchial gland in birds produces calcitonin. It is a 32-amino acid hormone that exerts an essentially hypocalcemic effect in response to rising serum ionized calcium levels.[3] The levels of circulating calcitonin in birds, amphibians and possibly sub-mammals are high and readily detectable compared with PTH. The bioactivity of calcitonin also varies among phyletic groups, with fish calcitonin exhibiting the most potent effect. In the bird, calcitonin levels increase following injections of calcium. There is a direct correlation between calcitonin levels and dietary calcium concentrations (and, hence, serum calcium levels). Although calcitonin has been shown in man to exert its hypocalcemic effects mainly by inhibiting osteoclastic bone resorption, its biological action in the bird remains surprisingly unclear despite the high circulating levels of the hormone in this group.

PROSTAGLANDIN

Prostaglandins are powerful facilators of bone resorption, with hypercalcemic effects similar to PTH and vitamin D_3 metabolites. The osteoclast appears to be the site of action for prostaglandin. Injections of prostaglandin into chickens will produce hypercalcemia, and the use of prostaglandin antagonists will produce hypocalcemia.[3]

ESTROGEN

Estrogens promote the formation of the vitellogenins from the liver. These are lipoproteins that are incorporated into the egg yolk. They bind calcium, and their production is followed by a rise in serum calcium levels. This estrogen-controlled hypercalcemic effect is not seen in mammals and is thought to be due to the need to produce large calcified eggs, requiring a rapidly mobilized source of calcium. Estrogens also influence the mobilization of medullary bone during the egg-laying cycle (and also during the nocturnal fast). The effect of estrogen on avian medullary bone is a large research area due to the importance of estrogen in maintaining bone mass in postmenopausal women.[2,3]

Investigating Abnormalities of Calcium Metabolism

CALCIUM

The measurement of serum ionized calcium provides a more precise estimate of an individual's calcium status than does total serum calcium, especially in the diseased patient.[8,26] Unfortunately, the majority of veterinary pathology laboratories at the present time report only a total calcium value measured by spectrophotometer, reflecting the total combined levels of ionized calcium, protein-bound calcium and complexed calcium. This can lead to misinterpretation of calcium results in birds, as any change in protein-bound calcium levels is not thought to have any pathophysiological significance.[22,26] Measurement of total calcium in an avian patient with abnormal protein levels or acid-base abnormalities would not truly reflect the calcium status of the animal. Any state affecting serum albumin values in the blood will affect the total calcium concentration, leading to an imprecise result. In laying female birds, the serum albumin levels may rise by up to 100%. This would produce an inflated total calcium concentration due to an increased protein-bound calcium fraction, while not affecting the ionized calcium level. The binding reaction between the calcium ion and albumin is strongly pH-dependent, so acid-base imbalances will affect ionized calcium levels. Therefore, a patient with metabolic acidosis would be expected to show an ionized hypercalcemia level due to decreased protein binding. With an alkalotic patient, an ionized hypocalcemia would occur as the protein-binding reaction increases. Ionized calcium levels are therefore considered a far more accurate reflection of the patient's calcium status, especially in a diseased animal. In mammals, positive correlations have been found between albumin and total calcium levels.[8,26] This enables formulae to be developed that "correct" total calcium levels for fluctuations in albumin levels. Recent research has suggested these corrected estimates of free calcium are inaccurate in 20 to 30% of cases in mammals.[8] In mammals, the relationship between total calcium and albumin diminishes with the severity of the disease, and the correction formulae are now thought to be less useful. Previous research in psittacine birds found a positive correlation between albumin and total protein in African grey parrots, but not Amazons (*Amazona* spp.);[12,13] more recent research in healthy African grey parrots has not indicated a significant relationship.[21,22] In the majority of cases, the measurement of ionized calcium is preferred in both mammals and birds.

Ionized calcium levels are measured by ion-specific electrodes. Analyzers using ion-selective electrodes are increasingly available for use in veterinary clinics. The methodology employed by the analyzers is based on the ion-selective electrode (ISE) measurement principle to precisely determine the ion values. The analyzers are normally fitted with ISE electrodes to assay the electrolytes sodium, potassium and ionized calcium. Each electrode has an ion-selective membrane that undergoes a specific reaction with the corresponding ions contained within a particular sample. The membrane is an ion exchanger reacting to the electrical charge of the ion, causing change in the membrane potential or measuring voltage, which is built up in the film between the sample and the membrane. A galvanic measuring chain within the electrode determines the difference between the two potential values on either side of the membrane of the active electrode. The potential is conducted to an amplifier by a highly conductive inner electrode. The ion concentration in the sample is then determined by using a calibration curve produced by measuring the potentials of standard solutions with a precisely known ion concentration. Blood samples for ionized calcium assays should be drawn into heparin and analyzed as soon as possible after venipuncture, as changes in the blood pH of the sample will affect the accuracy of the ionized calcium levels (ie, contact with carbon dioxide in the air will reduce the pH of the sample). Despite this, a study in dogs suggests that samples will not be adversely affected if not assayed for up to 72 hours, so it is possible to use external laboratories.[17] This has been the

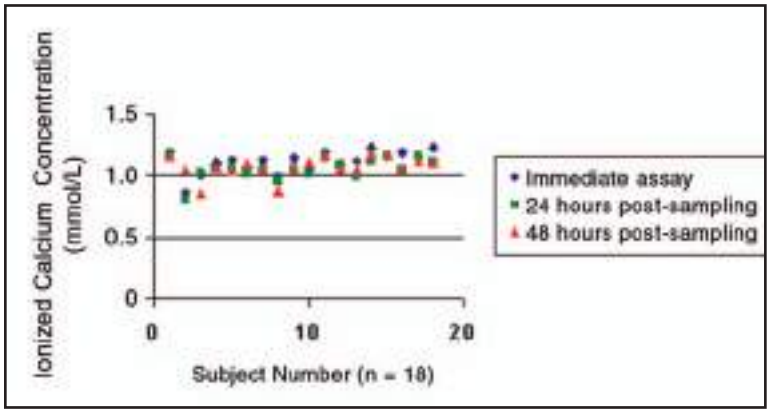


Fig 5.1 | Effect of sample handling delay on ionized calcium concentrations. There was no significant difference between ionized calcium concentrations in the same sample despite delays of up to 48 hours. The samples were stored at 3-5° C prior to analysis.[22]

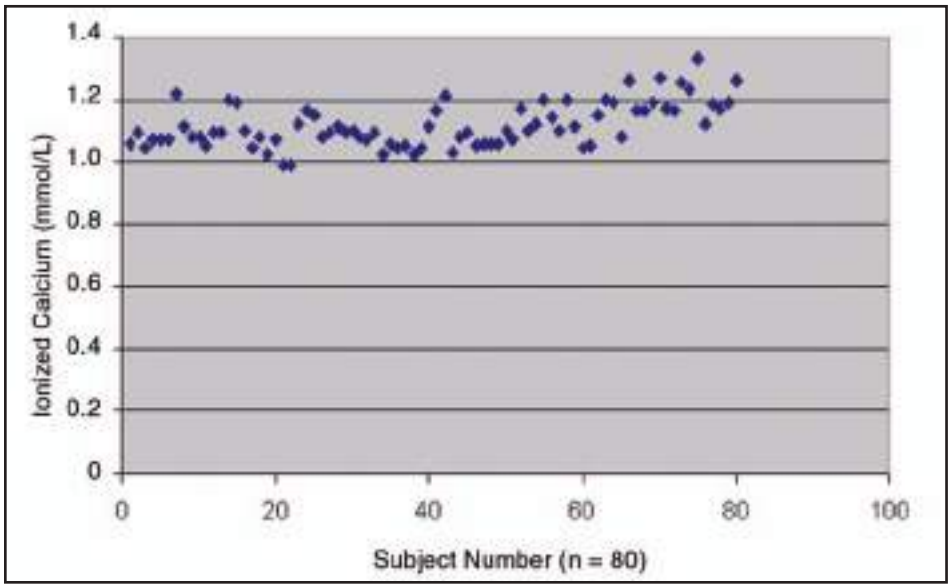


Fig 5.2 | Serum ionized calcium concentrations in 80 healthy captive African grey parrots. The ionized calcium concentrations are all within a tight range. The normal range was found to be 0.96-1.22 mmol/L.

author's experience with avian samples, but it is advisable to fill the sample tubes to minimize contact with the air[22] (Fig 5.1). It also is important to chill the samples immediately to reduce glycolysis by the red blood cells, which continue to produce lactic acid as a byproduct, reducing the pH of the sample.

A recent study in healthy African grey parrots indicated a narrow normal range for ionized calcium levels, but a wide variation in total calcium levels due to protein fluctuated among birds[21,22] (Figs 5.2, 5.3). The study did not reveal any significant correlation between albumin levels and total calcium levels in healthy birds. It was shown that using total calcium values in isolation would lead to misdiagnosis of disorders of calcium metabolism in this species. Of 394 samples submitted from African grey parrots into the practice laboratory in 2001, 54 samples (13.17%) had a low ionized calcium level despite having normal total calcium concentrations, suggesting that hypocalcemia is potentially under-diagnosed in this species.[22] In mature female birds, hypercalcemia might be over-diagnosed due to increased protein-bound calcium producing a high total calcium level, but ionized levels would not be affected. Due to the potentially large fluctuations in albumin levels in adult birds compared with mammals, it is considered essential wherever possible to measure ionized calcium levels.

VITAMIN D_3

The measurement of 25-hydroxycholecalciferol is considered the best assessment of vitamin D_3 status in the avian subject due to a longer half-life than other vitamin D_3 metabolites.[8] There are assays available for the other metabolites of vitamin D_3 and it is important to ensure that any assay has been optimized for the metabolite of interest. Until recently, radioimmunoassays were the

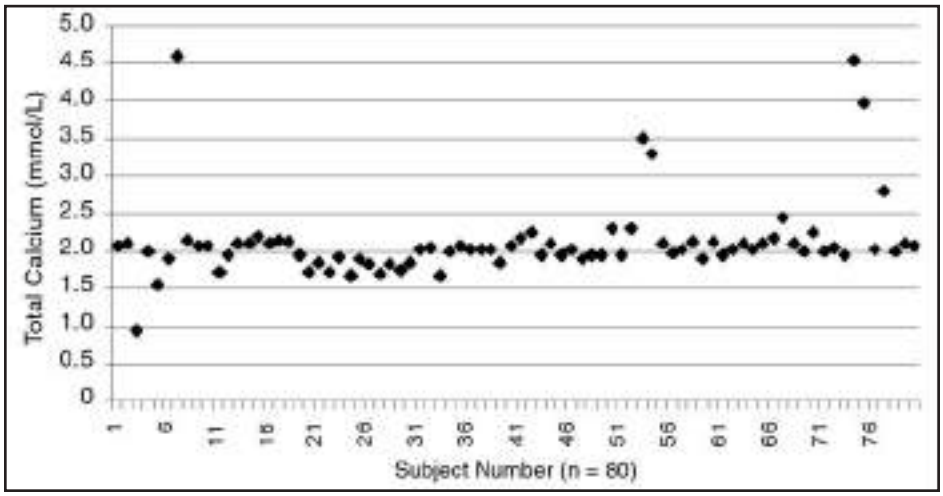


Fig 5.3 | Serum total calcium levels in the same 80 healthy captive African grey parrots as Fig 5.2. The total calcium concentrations were outside the normal range (2.0-3.0 mmol/L) in several birds despite normal ionized calcium levels. This fluctuation in the total calcium concentration was due to protein variations in individual birds and has no pathological significance. This demonstrates the importance of measuring ionized calcium wherever feasible rather than total calcium.

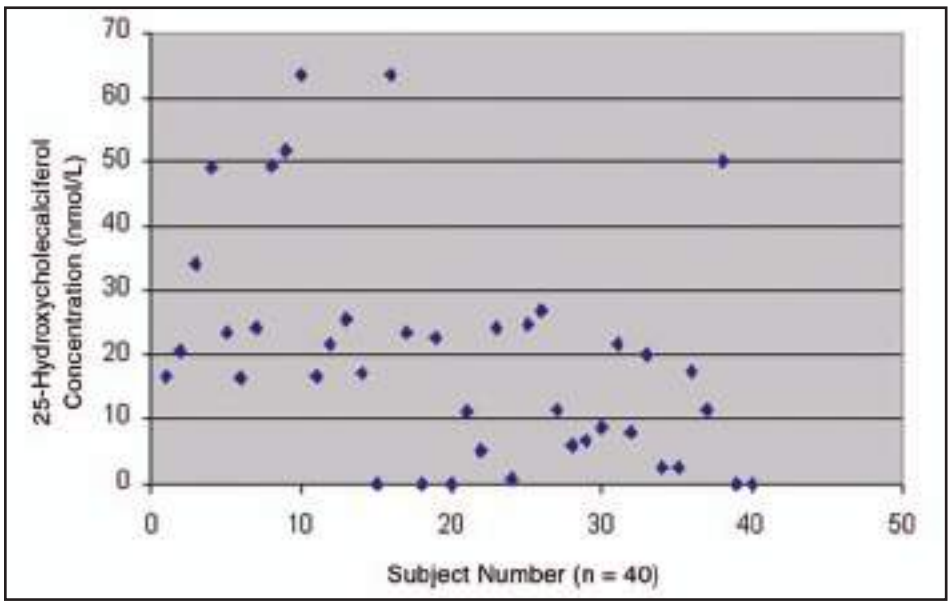


Fig 5.4 | 25-hydroxycholecalciferol concentrations in seed-fed African grey parrots. There was a wide variation in vitamin D levels. It is postulated that this is due to both fluctuations in UV-B levels received by individual birds and low dietary vitamin D.

only commercial assays available for both 25-hydroxycholecalciferol and 1,25-hydroxycholecalciferol. An ELISA test for 25-hydroxycholecalciferol has recently been developed with the advantages of both convenience and economy. This assay has been shown to correlate well with the radioimmunoassays.[8] This has allowed research to be carried out in the *Psittacus* species. A recent study in African grey parrots (a genus known to suffer from hypocalcemia) indicated a wide variation in 25-hydroxy-cholecalciferol levels in seed-fed birds[19,20] (Fig 5.4). Any 25-hydroxycholecalciferol results should be interpreted within the context of the diet and the levels of UV light received by the bird.[20] Recent research in psittacine birds has suggested that serum vitamin D_3 levels can be affected by both varying levels of dietary vitamin D_3 and UV-B light exposure.[23] Blood should be drawn into heparin and preferably frozen immediately after venipuncture until the assay can be performed. The use of vitamin D_3 assays will have an increasing use in avian medicine due to the common presentation of calcium disorders, both in terms of clinical disease and poor reproductive results. Vitamin D_3 assays could possibly be useful for reptiles — another group that suffers problems with calcium metabolism, in many cases due to poor husbandry.[15]

Fig 5.5 | A 5-week-old African grey parrot with severe nutritional osteodystrophy. There was radiographic evidence of pathological fractures in both tibiotarsi and humeri with severe spinal malformation. The bird was euthanized. Histopathology of the parathyroid glands confirmed vacuolated hypertrophic chief cells suggestive of nutritional secondary hyperparathyroidism. The bird had been parent-reared by adults fed an unsupplemented seed mix.

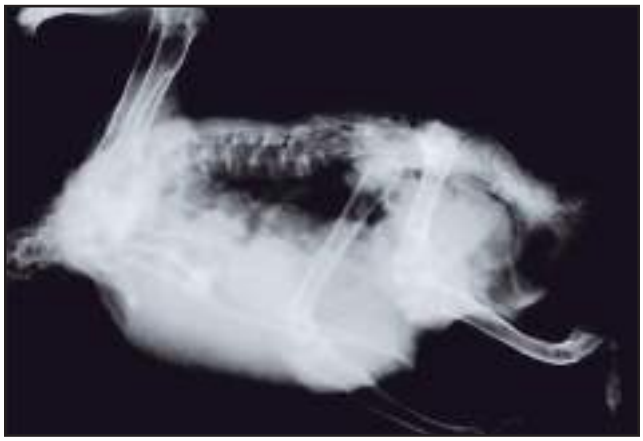


Fig 5.6 | Lateral radiograph of a 12-week-old African grey parrot with severe osteodystrophy. There is severe deviation of both tibiotarsi with marked deviation of the spine. The chick had been parent-reared by adults fed an unsupplemented seed-based diet. The bird was humanely euthanized.

ASSAY FOR PTH

PTH is very labile and any assay requires exacting sample handling to produce good results. Blood should be drawn into EDTA and either assayed immediately or frozen to -70° C within 1 hour of venipuncture. The majority of human measurements involve a two-site radioimmunoassay in an attempt to reduce the interfering effect of the many cleavage products of PTH that have long half-lives. Most human assays concentrate on the middle and terminal segments of the PTH molecule due to the very short half-life of the biologically active 1-34 section. A human research kit for 1-34N section PTH has been used to assay PTH in African grey parrots with consistent results. This study indicated that as ionized calcium levels rose in a group of 40 African grey parrots, PTH levels fell. Unfortunately, the assay for PTH 1-34N is too intricate for routine use at the present time and, in the author's opinion, intact 1-84 PTH assays are not useful in psittacine birds.[8,13,18]

Effects of Husbandry on Calcium Metabolism in Psittacine Birds

The effects of altering dietary vitamin D_3, calcium or exposure to different levels of UV light have been well researched in poultry.[3,4,10,14,24] Deficiencies in any of these parameters will lead to poor production and skeletal disorders such as tibial dyschondroplasia.[3] Extensive research has produced published results, enabling the poultry industry to select varying levels of dietary vitamin D_3 and calcium in accordance with the amounts of UV supplied. If domestic poultry are receiving no UV light, then all the cholecalciferol must come from the diet.[10] The domestic fowl does not have a dietary requirement for vitamin D_3 if it receives adequate UV-B radiation.[10]

Unfortunately, caged birds have not been extensively researched, but most would be expected to receive a poor diet with inadequate UV light. Seed-based mixes traditionally used for feeding captive parrots have low levels of both vitamin D_3 and calcium.[5,16,20,21] In addition, they can contain high levels of phosphorus that can bind the calcium in phytate complexes.[10] Chronic deficiency of vitamin D would be expected to lead to hypocalcemia and secondary hyperparathyroidism. Vitamin D is obtained by birds directly from the diet and by the action of UV-B on vitamin D precursors in the cutaneous tissues. Either a deficiency in dietary vitamin D or lack of available UV-B light would be expected to produce a vitamin D-deficient bird, potentially leading to subsequent problems with calcium metabolism.[3,10] Hypocalcemia is a common syndrome in avian subjects, with African grey parrots (*Psittacus erithacus*) in captivity appearing particularly susceptible, although the etiology is still unconfirmed.[5,6,7,16] Affected birds present clinically with a variety of neurological signs, ranging from slight ataxia to seizures, which respond to calcium therapy.[21,22] In adult females, egg binding or poor reproductive performance is a common presentation. In young African grey parrots, osteodystrophy also is a common presenting clinical sign[5] (Figs 5.5-5.8). The growing birds can be affected by bony deformities that range from subtle radiographic changes to obvious gross deformities in both the long bones and spinal column. Radiography has been routinely used to demonstrate juvenile osteodystrophy in many other species of birds. In a recent study,

Fig 5.7 | An adult African grey with osteodystrophy. The bird has characteristic bending in the tibiotarsi. The condition was successfully corrected by osteotomy and fixation of both legs.

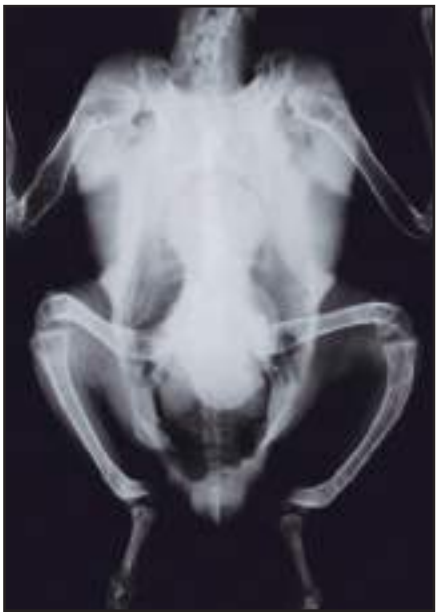


Fig 5.8 | Ventral dorsal radiograph of the bird in Fig 5.7. There is severe deviation in both tibiotarsi with folding pathological fractures. There also is evidence of abnormal skeletal development in both wings.

16 of 34 feather-plucking birds examined radiographically as part of a normal work-up revealed evidence of osteodystrophy.[5] The condition will be permanent and deteriorates as the birds increase in weight, which puts increased pressure on the deformed bones and frequently requires corrective surgery.

A recent study in African grey parrots has shown that it is possible to produce changes in calcium parameters by varying husbandry conditions as in poultry.[21,23,25] The study has demonstrated the effects of feeding a formulated nugget diet[a] with increased levels of vitamin D_3 and calcium compared with a seed-fed control group. The nugget diet produced a statistically significant increase in both the ionized calcium levels and 25-hydroxycholecalciferol levels over the seed-fed group[21,25] (Figs 5.9, 5.10). This was reflected in improved breeding performance in the nugget-fed group. These young birds developed without radiographic evidence of osteodystrophy.

In the same study, both the seed- and nugget-fed groups revealed a significant increase in 25-hydroxycholecalciferol and ionized calcium concentrations when they were exposed to artificial UV-B radiation[23] (Figs 5.11, 5.12). The nugget-fed group under the same UV levels revealed significantly higher concentrations of vitamin D_3 and ionized calcium over the seed control group.[23] The initial observations from this study suggest that UV light levels also are important factors in the vitamin D metabolism of captive birds in addition to diet. In the future, it will be possible to supply diets with varying amounts of vitamin D depending on the ambient supply of UV-B light available to captive birds. Initial work by the author with South American species has shown that they do not appear to be as dependent as African grey parrots on UV-B light for maintaining adequate vitamin D levels (M. Stanford, unpublished data). This might explain the prevalence of disorders of calcium metabolism in African grey parrots compared with other psittacine birds. South American rain forest has a dense canopy compared with African forests, reducing the UV-B light levels reaching the birds, so their vitamin D metabolism would be expected to be different from those of African birds.

These studies show that it is important that breeders feed their birds a diet that contains adequate calcium and vitamin D. It also is important to supply adequate UV-B radiation to captive birds kept indoors. This would be expected to help prevent the many expressions of hypocalcemia seen specifically in African grey parrots and other susceptible species. This includes the parent birds, because the female birds lay down medullary bone starting about 6 weeks prior to laying eggs. The nutrition of female birds appears to affect the early development of the chicks. In a recent study in African grey parrots, parent-reared juveniles showed radiographic evidence of osteodystrophy at 8 weeks if the parents were fed seed-based diets. The young from nugget-fed parents had no radiographic signs of osteodystrophy at 12 weeks [21,25] (Figs 5.13, 5.14). See Chapter 4, Nutritional Considerations, Section I Nutrition and Dietary Supplementation.

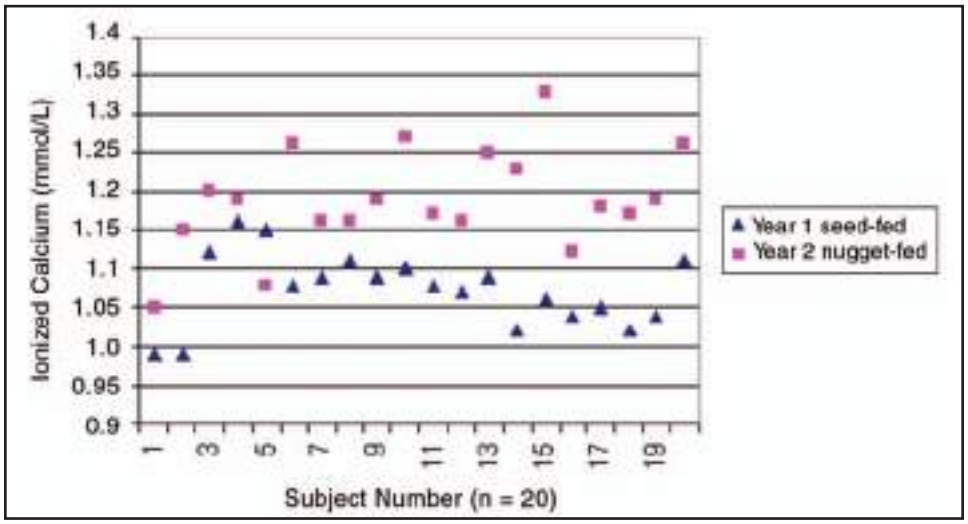


Fig 5.9 | Effect of diet on ionized calcium concentrations in African grey parrots. There was a significant increase in the serum ionized calcium concentrations in birds fed a formulated nugget diet[a] compared with the same birds fed a seed-based diet.

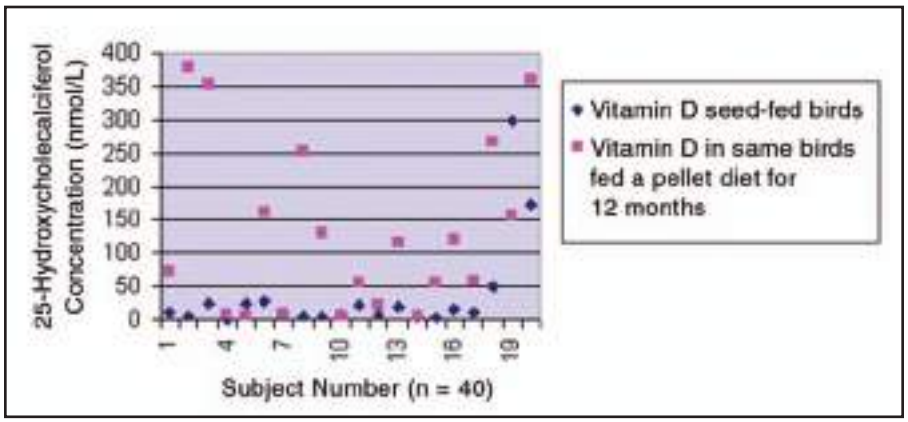


Fig 5.10 | Effect of diet on vitamin D concentrations in African grey parrots. There was a significant increase in the 25-hydroxycholecalciferol concentrations in birds fed a nugget diet[a] compared with the same birds fed a seed-based diet.

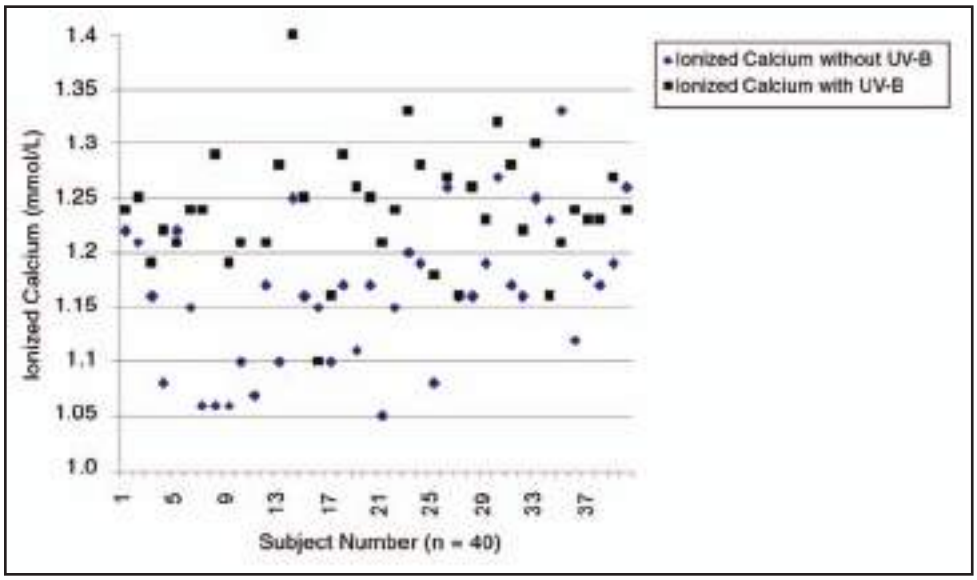


Fig 5.11 | Effect of UV-B supplementation on ionized calcium concentrations independent of diet fed. There was a significant increase in ionized calcium concentrations in both the seed- and nugget-fed[a] groups exposed to 12 hours of UV-B light in every 24 hours.

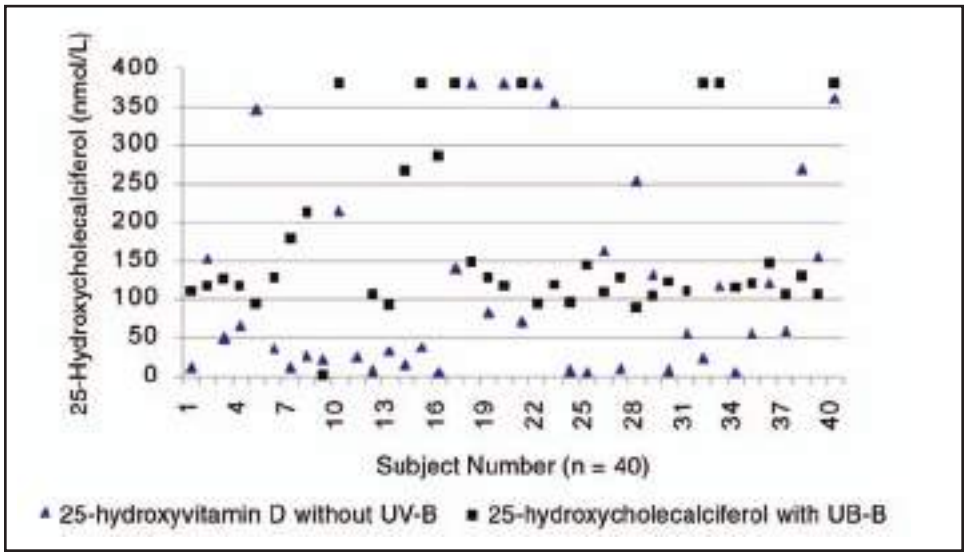


Fig 5.12 | Effect of UV-B supplementation on 25-hydroxycholecalciferol concentrations in African grey parrots independent of diet fed. There was a significant increase in 25-hydroxycholecalciferol concentrations in both seed- and nugget-fed[a] groups exposed to 12 hours of UV-B light in every 24 hours.

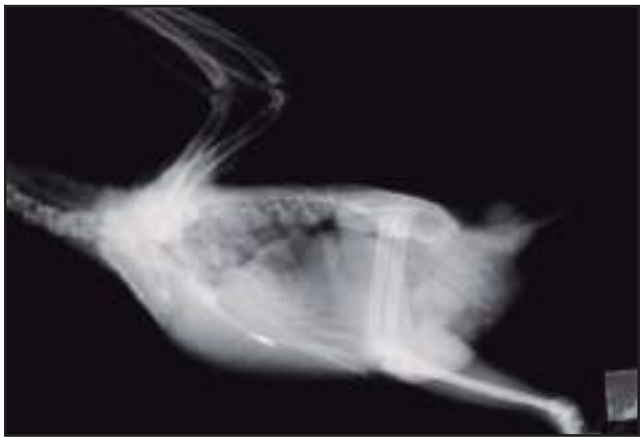


Fig 5.13 | Lateral radiograph of a 12-week-old African grey parrot with normal skeletal development. The bird was parent-reared until weaning by adults fed a formulated parrot food[a] until weaning.

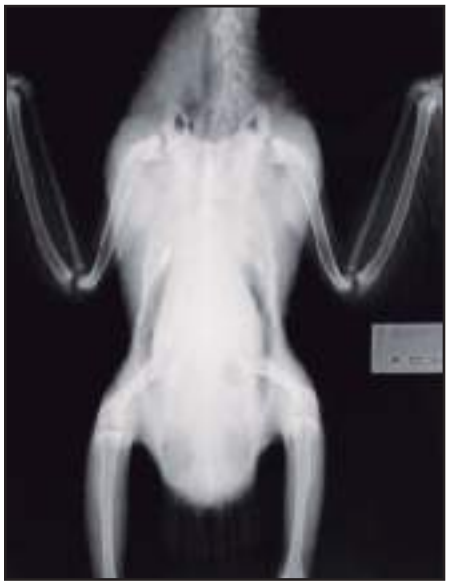


Fig 5.14 | Ventral dorsal radiograph of a 12-week-old African grey parrot with normal skeletal development. The bird was parent reared until weaning by adults fed a formulated parrot food[a] .

Products Mentioned in the Text

a. Harrison's High Potency Coarse, HBD International, 7108 Crossroads Blvd, Suite 325, Brentwood, TN 37027 USA, 800-346-0269 customerservice@harrisonsbirdfoods.com, www.harrisonsbirdfoods.com

References and Suggested Reading

1. Aslam SM, Garlich JD, Qureshi MA: Vitamin D deficiency alters the immune responses of broiler chicks. Poult Sci 77(6):842-849, 1998.
2. Bentley PJ: Comparative Vertebrate Endocrinology. Cambridge, UK, Cambridge Univ Press, 1998, pp 269-301.
3. Dacke GC: *In* GC Whittow (ed): Sturkie's Avian Physiology 5th ed. London, UK, Academic Press, 2000, pp 472-485.
4. Edwards JR, et al: Quantitative requirement for cholecalciferol in the absence of ultraviolet light. Poult Sci 73: 288-294, 1994.
5. Harcourt-Brown NH: Incidence of juvenile osteodystrophy in hand-reared grey parrots (*Psittacus eritbacus*). Vet Rec 152, 438-439, 2003.
6. Hochleithner M: Convulsions in African grey parrots in connection with hypocalcemia: Five selected cases. Proc 2nd Euro Symp Avian Med Surg, 1989, pp 44-52.
7. Hochleithner M, Hochleithner C, Harrison GJ: Evidence of hypoparathyroidism in hypocalcemic African grey parrots. The Avian Examiner Special Suppl. HBD International Ltd, Spring, 1997.
8. Hollis BW, Clemens TL, Adams JS: *In* Favus MJ (ed): Primer on the Metabolic Bone Disease and Disorders of Bone Metabolism. Philadelphia, Lippincott, Williams & Wilkins, 1997.
9. Hurwitz S: Calcium homeostasis in birds. Vitam Horm 45:173-221, 1989.
10. Klasing KC: Comparative Avian Nutrition. New York, CAB Intl, 1998, pp 290-295.
11. Koch J, et al: Blood ionic calcium response to hypocalcemia in the chicken induced by ethylene glycol-bis-(B-aminoethylether)-N,N'-tetra-acetic acid: Role of the parathyroids. Poult Sci 63:167-171, 1984.
12. Lumeij JT: Relationship of plasma calcium to total protein and albumin in African grey (*Psittacus erithacus*) and Amazon (*Amazona* spp.) parrots. Avian Pathol 19:661-667, 1990.
13. Lumeij JT, Remple JD, Riddle KE: Relationship of plasma total protein and albumin to total calcium in peregrine falcons (*Falco peregrinus*). Avian Pathol 22:183-188, 1993.
14. Mahout A, Elaroussi, et al: Calcium homeostasis in the laying hen. Age and dietary calcium effects. Poult Sci 73:1581-1589, 1994.
15. Mitchell M: Determination of plasma biochemistries, ionised calcium, vitamin D_3 and haematocrit values in captive green iguanas (*Iguana iguana*). Proc Assoc Reptile Amphib Vet, 2002, pp 87-95.
16. Rosskopf WJ, Woerpel RW, Lane RA: The hypocalcemic syndrome in African greys: An updated clinical viewpoint. Proc Assoc Avian Vet, 1985, pp 129-132.
17. Schenck PA, Chew DJ, Brooks CL: Effects of storage on serum ionised calcium and pH values in clinically normal dogs. Am J Vet Res 56:304-307, 1995.
18. Stanford MD: Development of a parathyroid hormone assay in grey parrots. Proc Brit Vet Zool Soc, 2002.
19. Stanford MD: Measurement of 25 hydroxycholecalciferol in grey parrots (*Psittacus e. erithacus*). Vet Rec 153:58-61, 2003.
20. Stanford MD: Measurement of 25 hydroxycholecalciferol in grey parrots: The effect of diet. Proc Euro Assoc Avian Vet, 2003.
21. Stanford MD: Measurement of ionised calcium in grey parrots: The effect of diet. Proc Euro Assoc Avian Vet, 2003.
22. Stanford MD: Measurement of ionised calcium in grey parrots (*Psittacus e. erithacus*). Vet Rec 2004. In Press.
23. Stanford MD: The Effect of UVb supplementation on calcium metabolism in psittacine birds. Vet Rec 2004. In Press.
24. Taylor TG, Dacke CG: Calcium metabolism and its regulation. *In* Freeman BM (ed): Physiology and Biochemistry of the Domestic Fowl Vol 5. London, UK, Academic Press 1984, pp #125-170.
25. Tian XQ, Chen, et al: Characterisation of the translocation process of vitamin D_3 from the skin into the circulation. Endocrinology 135(2):655-6121, 1994.
26. Torrance AG: Disorders of calcium metabolism. *In* Torrance AG, Mooney CT (eds): BSAVA Manual of Small Animal Endocrinology. Cheltenham, UK, BSAVA Publications, 1995, pp 129-140.

CHAPTER

6

Maximizing Information from the Physical Examination

BOB DONELEY, BVSc, FACVSc (Avian Health);
GREG J. HARRISON, DVM, Dipl ABVP-Avian, Dipl ECAMS;
TERESA L. LIGHTFOOT, DVM, Dipl ABVP-Avian

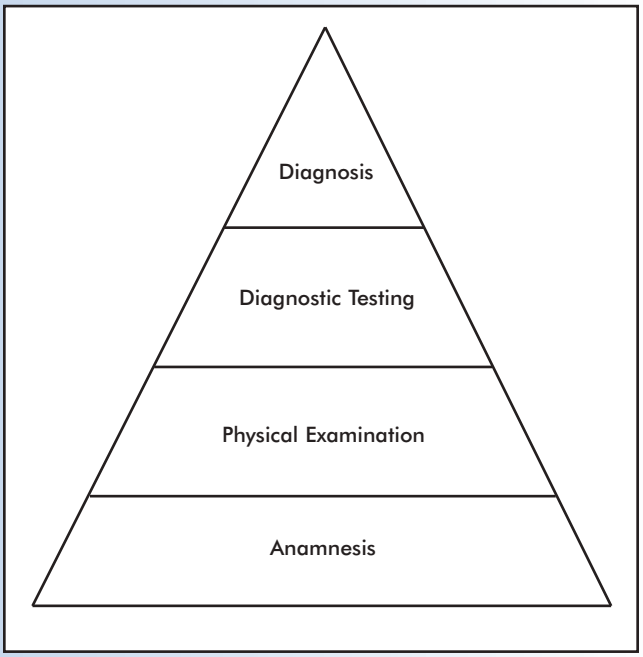


Fig 6.1 | The Diagnostic Pyramid

The last ten years have seen amazing advances in diagnostic testing capabilities in avian medicine. Tests that were not even considered a decade or two ago are now commonplace. However, the cornerstone of good avian medicine is still the careful evaluation of the patient. Diagnostic tests are an important part of that evaluation, but they are not the whole evaluation. Their use is only a part of a continuum that starts with a careful anamnesis and concludes with a diagnosis. This can be illustrated by the diagnostic pyramid shown (Fig 6.1).

Astute clinicians will move carefully through these levels, refining their differential list through careful history taking, physical examination and selection of diagnostic testing until a final diagnosis (or at least a much shortened list of differentials) is reached.

Understanding the Masking Phenomenon

A common misconception held by many bird owners and veterinarians is that birds are not very resistant to illness. To the novice it often appears that birds show signs of illness one day, are at the bottom of their cage the next, and dead the day after. This misconception has stemmed from two sources.

First, many of the birds seen in practice are only a few generations descended from wild birds. As such, they retain many of the protective instincts inherited from their forebears. Many avian species kept as companions are relatively low on the food chain. These protective instincts have been developed to avoid drawing the

Greg J. Harrison

Fig 6.2a | The bird's natural masking phenomenon misleads owners and many veterinarians. They think the bird is fine, until the bird decompensates and shows overt signs of illness. This blue budgerigar is showing the signs of the latter stages of illness and is "sleeping" and "fluffed up." In the same environment, the yellow bird is keenly alert.

Greg J. Harrison

Fig 6.2b | Observing a caged bird is an art well worth perfecting. Outwardly, this dead canary appeared fine to the owner who brought the bird to the veterinarian. The bird died moments after examination.

Greg J. Harrison

Fig 6.2c | This canary's illness has wasted away all the pectoral muscle. One must observe the bird in a quiet situation — waiting a few minutes for the masking behavioral defense to wane and the fluffing to recur. A history of a small bird that has not eaten in a day and has scant feces denotes a very sick bird. See discussion under Fig. 6.42a-d prior to handling such a sick bird.

attention of predators. One such instinct is often known as the "masking phenomenon." Predators are naturally drawn to prey that look or behave differently from others. Unusual coloring, weakness or lameness can single out a bird and make it attractive to a predator. A natural instinct is therefore to avoid appearing "different." A sick bird will make a determined effort to look healthy, even in the absence of predators. The classical "sick bird look" we usually associate with illness — fluffed feathers, closed eyes, lethargy — only develops when the bird is incapable of masking these signs (Fig 6.2a). Therefore, many of the patients presented to veterinarians are past the initial stages of their illness and are now decompensating rapidly.

Secondly, due to lack of experience, most owners and many veterinarians may miss subtle changes in a bird's behavior or appearance that are indications of a health problem. Overlooking these early signs, combined with the bird's efforts to mask obvious clinical signs, invariably leads to the delayed detection of illness and the presentation of the bird in extremis (Figs 6.2b,c). It is important that veterinarians learn to recognize early signs of illness, and educate their clients, so that illnesses can be detected before becoming too advanced. A physical examination form, used routinely by veterinarians and support staff, allows thorough and methodical documentation of the essential parameters of the physical examination (Fig 6.2c). The "masking phenomenon" and the ease with which early clinical signs can be overlooked highlight the importance of regular health examinations for the companion bird. Long-standing conditions such as malnutrition can be detected and corrected before the bird begins to decompensate and shows overt signs of illness.

History Taking

The collection of an accurate and thorough history of a patient is as crucial to making a diagnosis as is the physical examination and comprehensive diagnostic testing. A good history can alert the clinician to likely problems and allow him/her to refine the diagnostic approach.

SIGNALMENT

The first step in obtaining a history is to gain as much information about the bird itself as possible. A good receptionist or technician can often obtain such information. The clinician should be familiar with avian taxonomy, sexual dimorphism, and species-specific behaviors. Clients are often unaware of some basic facts about their bird (such as species, sex and age). Veterinarians intending to see avian patients regularly need to acquire a working knowledge of commonly kept bird species and their physical characteristics. Such knowledge, while available in some publications, is generally acquired

through experience and through being proactive and inquisitive. This may require visiting other veterinarians, aviaries, avian pet stores and zoologic collections and will take some time to acquire.

The species of the bird is the first key piece of information. Many clients are unaware of the correct identification of their bird, or may refer to it by a local name unfamiliar to others. The cockatiel (*Nymphicus hollandicus*) is known all over the world as the cockatiel or 'tiel, except in Australia (the bird's native land), where it is more commonly known as the Quarrion or, in Western Australia, as the Weero (Figs 6.3a-f). The clinician needs to be able to recognize common species and have access to illustrated literature that will enable the clinician to confirm the scientific name and to identify unfamiliar species (see Chapter 2, The Companion Bird for selected photos). Knowledge of the species can offer the clinician vital clues to likely differential diagnoses for the sick bird and appropriate preventive medicine and husbandry during wellness examinations. For example, black cockatoos (*Calyptorhynchus* spp.) and some macaws require more fat in their diet than many other psittacines. The same diet fed to galahs (*Eolophus roseicapillus*) and budgerigars (*Melopsittacus undulatus*) can result in obesity, atherosclerosis or hepatic lipidosis. Species-specific behaviors, such as the alarm "snuffing" typical of *Pionus* spp., is often mistaken by new owners and veterinarians unfamiliar with this behavior as a pathologic respiratory condition.

The age of the bird, while usually difficult to determine once adult plumage has been attained, is another important part of the anamnesis. Juveniles are more susceptible to conditions such as rickets, polyomavirus, circovirus and bacterial infections, while adult birds are more likely to suffer from neoplasia, chronic malnutrition and degenerative conditions. While owners may not be sure of the exact age of their pet, they usually know how long they have owned it, and that may be sufficient for establishing an age-specific list of differential diagnoses.

Many psittacines are sexually monomorphic—or at least appear that way to their owners. Knowing the sex of a bird can be vital. For example, abdominal hernias are almost non-existent in male birds, while being relatively common in females; yolk-related peritonitis and cystic ovarian disease are obviously seen only in hens; cocks do not become egg bound. Do not accept the owner's assertion of their bird's sex unless they have proof of sex identification (ie, a history of egg laying or a certificate of sex identification that can be correlated with this bird). If such proof is not available, or the sex cannot be positively diagnosed by physical characteristics, surgical or DNA sexing may be required (Figs 6.4a,b).

PATIENT HISTORY

How long have the clients owned this bird? (Fig 6.5)

Birds that have been in the owner's possession for many years, with no recent exposure to other birds, are less likely to have infectious diseases (Fig 6.6) and are more likely to be suffering from underlying chronic conditions such as malnutrition or inadequate exercise. Recently obtained birds are more likely to have been in close contact with other birds and, as such, have possibly been exposed to infectious diseases (Figs 6.7-6.9).

Where did the owner obtain the bird?

With experience, the clinician will be able to identify "problem sources" of birds in the local area (ie, a certain breeder with a history of circovirus, bird fairs or a pet shop renowned for chlamydial problems). Developing a working knowledge of the quality of the sources of pet birds in your area can be a key element of patient evaluation in your practice (Fig 6.10) (see Chapter 2, The Companion Bird).

Is the bird aviary bred or wild-caught? (Fig. 6.11)

Wild-caught psittacines, although less common in recent times, are still coming into the pet market in certain parts of the world, such as in Europe. In countries such as Australia, Africa and South America, it is reasonably easy to obtain a bird from the wild. The challenge of taming a wild-caught bird can be daunting. Paradoxically, wild-caught (and therefore parent-raised) parrots that survive the rigors of capture and have adapted to interaction with humans, are much less likely to demonstrate the common behavioral problems of our domestically raised parrots. Feather plucking, vent prolapse, inappropriate screaming, and learned biting are all more prevalent in captive-raised African greys, *Cacatua* spp., and macaws (*Ara* spp.) Fortunately, improved techniques for raising these birds are being implemented, and both veterinary and client awareness of the need for continued attention to the emotional well-being of our parrots is increasing. As veterinarians, our responsibility is to inform the owners of the need for attention to and modulation of behaviors in their newly-acquired psittacine pet prior to the onset of problems. Suggesting and providing a contact for a knowledgeable parrot behavioral consultant can make the difference between a decades-long rewarding human-pet bond and years of frustrated and dissatisfied co-existence (see Chapter 2, The Companion Bird and Chapter 3, Concepts in Behavior).

Parasitic infections (both gastrointestinal and hematogenous parasites) are more common in wild-caught birds. However, areas with outdoor aviaries in the USA often have significant and occasionally fatal super infections from various nematode species.

Mimi Walling/We Shoot Birds

Fig 6.3a | Knowing the species, the sex and the name of the color mutation, such as in this cockatiel, makes an owner more comfortable with the avian veterinarian's knowledge. While most immature cockatiels resemble each other, mature cockatiels are usually sexually dimorphic. A mature gray male is shown here.

Mimi Walling/We Shoot Birds

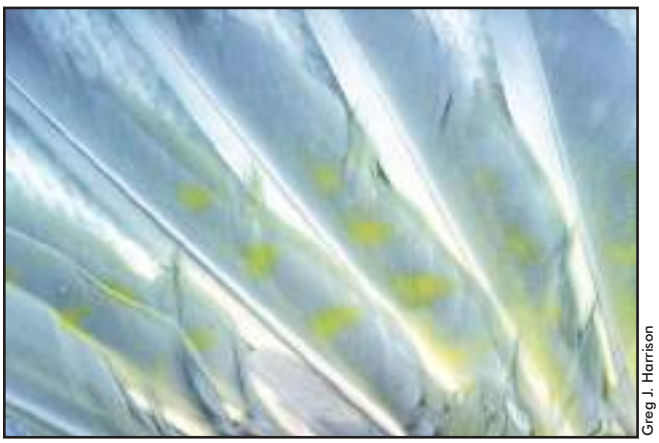
Fig 6.3b | These birds, if mature (over 9-12 months of age) represent the typical coloration of females of this species and color type. Females are lacking the bright yellow head in the normal gray colored cockatiel. Today's birds are seldom pure, but rather hybrid mutations. Coloration is not as dependable a factor in determination of gender in these mutation cockatiels. The bird on the left has some yellow on the head and would be said to be a hybrid — demonstrating normal grey female markings like the bird on the right but with the additional yellow of a pied. Females of the normal color have horizontal striping on the tail. When compared to the male in Fig 6.3a the body color is not the usual gray. This is a sign of a split. The coloration illustrated is likely to be "split" to cinnamon as well as lutino in order to demonstrate these colors.

Greg J. Harrison

Fig 6.3c | This yellow-headed cockatiel was thought to be a male until "he" laid an egg.

Greg J. Harrison

Fig 6.3d | Young pearl-colored cockatiels (pearlies) have this color pattern. The males revert to being very similar to Fig 6.3a, (normal grey), while the females retain the pearl pattern at maturity.



Greg J. Harrison

Fig 6.3e | Yellow (in this case) or white wing bars are present on the ventral surface in both cockatiel sexes until maturity. In immature females, these bars are present from the axillary region to the distal wing; in immature males they are present from the elbow joint to the distal wing. The bars are lost in mature males during their first adult molt.

Mimi Walling/We Shoot Birds

Fig 6.3f | Pied-colored cockatiels are not as distinctly sexually dimorphic as normal grey cockatiels. Behavior may aid in gender determination in these mutations. Male cockatiels carry a melody while females usually produce only one or two notes.


Greg J. Harrison

Fig 6.4a | Mature budgerigars are sexually dimorphic in the nominate green birds and most color mutations. The male shown here has the characteristic male blue cere.


Greg J. Harrison

Fig 6.4b | A mature female budgerigar with normal brown hypertrophy of the cere. Note the lack of hyperkeratosis, despite her being an egg producing hen for several years.


Jan Hooimeijer

Fig 6.5 | Some bird species, when properly cared for by the avian veterinarian and the owner, can be safely co-mingled, especially in spacious outdoor housing.


Mimi Walling/We Shoot Birds

Fig 6.6 | Boarding facility at an avian veterinary hospital. Proper testing prior to boarding is critical. Testing for infectious diseases helps prevent disease outbreaks. A complete history and physical examination at the time of admission allows underlying disease, such as chronic malnutrition, to be detected, brought to the owner's attention and addressed. This protocol minimizes the chance that a bird will expire during boarding, and the subsequent assumption by the owner that the boarding was causative. Trained staff and attention to proper husbandry and monitoring of the birds are essential.


Greg J. Harrison

Fig 6.7 | A free-flying Amazon in an English aviculture facility is exposed to untested budgerigars and cockatiels. In this environment, this Amazon may serve as a carrier, intermediate host or mechanical vector between the untested budgerigars and cockatiels and the balance of the aviary. As such, it poses as much or more danger to other aviary birds as exposure to wild birds or infestation with rats or roaches.


Greg J. Harrison

Fig 6.8 | Wild birds at feeders outside bird facilities can be a source of infection. Wild lories at Currumbin Bird Park in Australia (see Chapter 21, Preventive Medicine and Screening).


USDA

Fig 6.9 | A cap, mask, coveralls or gown, boots, disposable containers and air filtration with HEPA-filters are used at a USDA pet bird quarantine facility. This demonstrates measures that must be taken to impose a true quarantine and limit disease transmission.

Friedrich Janeczek

Fig 6.10 | These pet store birds have been properly tested and are kept housed as a single species. Individuals for display, sale and breeding are maintained in the store. When a collection is kept closed and is disease-free for several years, the incidence of disease will be much lower than in birds from pet stores with mixed, untested, open collections.

Greg J. Harrison

Fig 6.11 | Dr. Jan Hooimeijer examines a confiscated Jamaican parrot that was headed for the black market. Such birds are a constant source of diseases in the pet bird market and of diseases potentially devastating to the poultry industry.

Friedrich Janeczek

Fig 6.12 | Perches over a fish pond and rock substrate over drains facilitate cleaning. The natural perches, artificial plants, and reed walls are difficult to sterilize. However, in a properly managed, disease-free collection, disinfection is not required.

Old World species of psittacines are also particularly susceptible to infection and death from sarcocystosis.

In Australia, the probability of a wild-caught cockatoo being affected with psittacine beak and feather disease (PBFD) is much higher than that of an aviary-bred bird. The same is true of African greys imported into the European Union (EU) from the wild. In the USA and the EU, captive African greys, eclectus, lories, sun conures and assorted other species can be infected with proventricular dilatation disease (PDD) (see Chapter 32, Implications of Viruses in Clinical Disorders). Macrorhabdosis is common in captive-raised budgerigars and lovebirds (see Chapter 30, Implications of *Macrorhabdus* in Clinical Disorders).

Is the bird hand-reared or parent-reared? If hand-reared, at what age was it pulled from the nest, or was the egg incubator hatched?

The health of juvenile birds is dependent largely on the health and nutrition of the parents (Fig 6.12). Hand-reared birds are dependent upon the quality of the hand-rearing formula being fed and the skill of the person performing the hand-feeding. Parent-reared birds tend to be more difficult to tame, unless they are handled on a regular basis before fledging. Hand-reared birds on the other hand, while usually more closely bonded to people, often have behavioral disorders associated with poor socialization, especially if reared in isolation. If a large number of chicks from different sources are reared in one facility, there is a higher probability of the spread of diseases such as polyomavirus and chlamydiasis. All of these factors need to be assessed during the history collection, especially when examining juvenile birds. Birds sold prior to weaning, often to inexperienced owners, are predisposed to develop aspiration pneumonia. Behavioral problems are also common in babies hand-fed with the traditional methods (see Chapter 3, Concepts in Behavior).

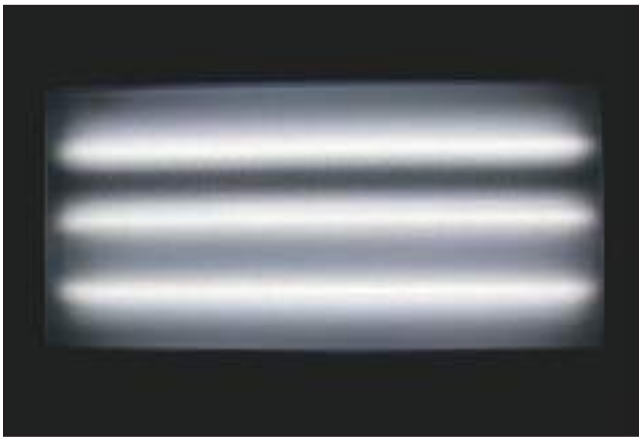
Although incubator hatched eggs, when harvested by knowledgeable aviculturists, have a far less chance of accidental breakage or parental destruction, several negative consequences arise. Inappropriate incubator temperature or humidity can cause congenital deformities. The lack of antibodies from initial crop feedings by the parents may create an increased susceptibility to many diseases, some of which can prove fatal. Additionally, the consequence of deprivation from parental contact during the first days and weeks of life is not documented in birds, but extrapolation from other species would make it likely that a serious negative impact may arise from this isolation.

Does the client have other birds? Are they in contact with each other? Are any of these birds affected with similar clinical signs?

Other birds in the household may have many effects on the environment of the presenting patient. These could be a source of infection or susceptible to infection from a bird presenting to the practitioner with a contagious disease. It would be significant if a blue and gold macaw (*Ara ararauna*) presenting for dyspnea was being kept in the same room with a powderdown-producing species, such as an umbrella cockatoo (*Cacatua alba*). With feather destructive or self-mutilation behaviors, the presence or absence of other birds may affect (positively

Greg J. Harrison

Fig 6.13a | Birds are exposed to natural sunlight yet protected from over-exposure in this aviculturist's facility.



Greg J. Harrison

Fig 6.13b | Fluorescent lights may have cyclic flickering that is potentially harmful, both physiologically and psychologically.

or negatively) the emotional state of the affected bird.

Previous Medical History

The avian patient's previous medical history should be determined to the extent possible.

- Has the bird been ill before?
- If so, what were the clinical signs, tests performed, results of those tests, diagnosis and therapy administered?
- What was the bird's response to this therapy?
- Has the bird recently been treated with remedies purchased at a pet shop or supplied by a breeder? If so, what medication and for what period of time was or is it being administered?
- If the bird is presented for a second opinion or as a referral, may we request copies of the previous medical record?

HUSBANDRY

Is the patient an aviary bird, a companion bird, or a zoological specimen?

The husbandry of aviary birds and zoological specimens are discussed elsewhere in this book, and the reader is referred to the appropriate chapters. Assessment of a companion bird's husbandry must include the cage, the environment around the cage, and the bird's interaction with its environment.

Is the cage in which the bird was transported the bird's permanent cage?

If it is not, ask the owner to describe the permanent cage, using the guidelines contained in the following paragraphs.

Where is the cage located in the house?

In the kitchen, living room, individual's bedroom, or screened porch?

Is the bird able to remove itself from view?

Is it supplied with a hide box or comparable visual screen? Is it kept isolated, away from family activities, or in the middle of constant activity?

Is the bird permitted to get adequate sleep at night?

A common owner misconception is that covering a bird's cage, while that cage is located in a room with the lights and television on, is providing the bird with an environment conducive to sleep. Whether covered or not, the number of hours that a bird is supplied with sufficient darkness and quiet is critical to health — both emotional and physical.

Does the bird have access to direct, unfiltered sunlight on a regular basis? (Figs 6.13a,b)

Increased calcium absorption and metabolism from ultraviolet (UV) light exposure is beneficial to birds as it is for many other species. Recent research shows that some species may have an absolute requirement for UV light (see Chapter 5, Calcium Metabolism). In the appropriate climates or seasons, regular outdoor exposure to sunlight can have many additional benefits, both physical and psychological. Increased humidity, the sounds and sights of nature, breezes and changes in barometric pressure all promote emotional well-being in birds when these are provided with adequate thought for the bird's safety and comfort. This leads to the next point:

If the bird is kept outside, is it safe from predators and diseases?

Are there potential disease vectors such as mosquitoes, roaches or rats that can access the bird's cage? If the bird is brought into the house at night, is the food and water removed and replaced to prevent contamination of the cage contents overnight and subsequent exposure to diseases such as sarcocystosis? (Note: Additional husbandry issues are covered in Figs 6.14-6.17a-c.)
Predators such as raccoons are notorious for reaching

into cages and severing legs and wings from parrots.

Does the bird come out of its cage? Is it supervised when out of the cage?
Remember that "supervision" is subjective. Many birds with heavy metal toxicity ingest the inciting material while being supervised. Owners are often adamant prior to the radiographic diagnosis that their bird has no access to metal.

Answers to questions concerning time out of the cage will also help establish the degree of interaction between the bird, its environment and its owners.

Are the bird's wings clipped or is it free-flighted? Is the bird exposed to potentially dangerous situations?
Sinks full of water, ceiling fans, open toilet bowls and sliding glass doors are among the potentially injurious, if not fatal, environmental hazards to which a bird may be exposed.

Different injuries and exposures occur in flighted and non-flighted birds. For example, heavy-bodied birds with severe wing clips are prone to beak and keel trauma. While keel and beak tip trauma may be obvious due to the presence of blood, the dislocation or straining of the quadrate bone and associated muscles may present only as depression and lack of appetite.

Conversely, concussive force to the head is most common in fully flighted birds that fly into glass doors or mirrors, as are ceiling fan injuries, burns from stovetops and drowning from accessible water sources.

Does the bird interact with other animals or birds?
Dogs and many other pets may carry clostridial bacteria normally, while pet birds often develop digestive disorders from exposure to such bacteria (see Chapter 4, Nutritional Considerations, Section II Nutritional Disorders). Also, cat claws and bites may cause penetrating wounds that do not leave obvious external marks but often cause internal damage and/or infection.

Is the bird subject to potential exposure to toxins?
Burning plastic, non-stick cooking pans, cigarette smoke, aerosol sprays or household plants are some of the common substances causing toxicity in pet birds. Additionally, the metal wicks of candles may contain lead. Smoke (including cigarette, incense, candles and barbeque grills) and other strong smells are potentially hazardous to pet birds, and exposure to these should be avoided (see Chapter 31, Implications of Toxic Substances in Clinical Disorders [Figs 6.18-6.24]).

DIET

Underlying many health problems in pet birds is the common thread of malnutrition. For many generations, bird owners have accepted as fact that seed is a complete diet for birds (Fig 6.25-6.41). This is reinforced by advertising from many producers and vendors of bird seeds. Historically, there has been a lack of readily available information on the deleterious effect of all-seed diets. Birds, as many other animals, often prefer high fat diets. Given a choice between seed, vegetables, formulated diets and fruits, nearly all birds will consume the seed first. Given this preference, it is not surprising to hear many bird owners state, "All he will eat is the seed, so that is all that I give him." Chapter 4, Nutritional Considerations, gives more details on nutrition and nutritional disorders. The reader is urged to study this carefully.

How much food is being consumed?

An important part of history taking is to ascertain not what the owner offers the bird, but more importantly, what the bird actually consumes. The clinician needs to be aware that there are major species differences in dietary requirements. For example, some species may have a higher requirement for fat than others. There is no single diet that is appropriate for all avian species any more than there is one diet suitable for all mammalian species.

In addition to the type of food offered and consumed, feeding practices need to be reviewed. Some birds, even when provided with an excellent diet, will consume an excessive quantity and become obese.

Is the food prepared fresh daily? Are dishes cleaned each day? Does the bird dunk food into its water dish?
This last practice of dunking food creates a nutrient-rich broth ideal for bacterial contamination. Sterilization is not necessary in the healthy home environment, but reasonable cleanliness should be employed.

Does the bird get any treats?

Treats can supply sufficient calories to pet birds that any attempt at weight control is sabotaged. Also, certain foods, such as guacamole dip, are not always perceived by the owner as potentially toxic. Other foods, such as snacks containing excessive sodium, may cause or exacerbate feather-destructive or mutilation behaviors (see Chapter 2, Concepts in Behavior Section III p82).

Are vitamin and mineral supplements being administered?

If so, what are the contents of the supplement and in what form is it provided (ie, in the water or on the food)? Vitamins administered in the water may decrease a bird's water consumption, discolor the urates or feces, and either be ineffective due to dilution and lability, or cause hypervitaminosis if administered in conjunction with vitamin-enriched seeds and/or formulated diets.

Greg J. Harrison

Fig 6.14 | Cage with natural perches. This bird's cage is too small to allow for flight. Note that two sources of calcium (mineral blocks) are provided in addition to a formulated diet. This may lead to nutritional imbalance due to excess calcium (see Chapter 4, Nutritional Considerations).

Fig 6.15 | Here sand is used as a cage substrate. Note the accumulation of feces and food indicating poor husbandry and potential microorganism overgrowth. Sand can be used but needs to be raked clean often and replaced when dirty.

Greg J. Harrison

Fig 6.16 | Newspaper is a good substrate if changed daily. The number and character of droppings can be determined readily, yielding information reflecting the state of the bird's health. These "popcorn" droppings are not normal.

Greg J. Harrison

Fig 6.17a | Cement and sandpaper perches. Sandpaper perches are generally ineffectual and may cause or add to plantar surface abrasion. Overgrown nails are a sign of metabolic disease and require addressing the underlying problem. Sharp nail points are normal and necessary in the wild. Owners often request trimming of these tips to prevent discomfort to their skin. Some blunting may be necessary to prevent trauma to the owner, but the bird will be less stable when perching and the owners must be forewarned of this. In some cases cement perches can work to keep the tips blunted when the proper type, size, texture and placement in the cage is accomplished.

Greg J. Harrison

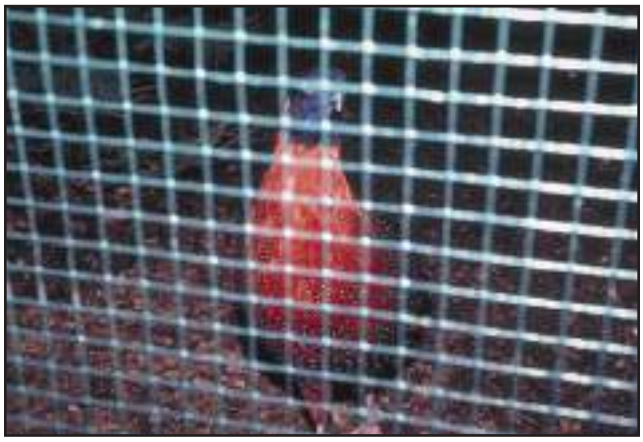
Fig 6.17b | Plastic perches with ridges to allow a better grip can be wedged into stainless steel cages. They are easy to clean and sterilize.

Greg J. Harrison

Fig 6.17c | Natural hard woods like manzanita make long lasting perches. However, these do not offer a rough surface on which the birds can clean their beaks, nor is such a hard slick surface the ideal perch.

Greg J. Harrison

Fig 6.18 | Some dog and cat flea products can be a danger to birds.



Greg J. Harrison

Fig 6.19a | "Hardware cloth" is galvanized iron. The galvanized coating is usually predominately zinc and may also contain lead. While usually safe for non-chewing birds like this *Tragopan* sp., it is not safe for members of the psittacine family.

Greg J. Harrison

Fig 6.19b | Toys are frequently made of polished galvanized metal. Here a magnet is used as a diagnostic tool — if it attaches, the underlying metal is iron. This is a common metal that is galvanized to prevent rust. Galvanization is primarily zinc. The circular shaped magnet has attached to a chain and anchor screw, indicating galvanization is likely and these objects should be removed.

Greg J. Harrison

Fig 6.19c | Toys can have the galvanized hook replaced with brass, the chain replaced with stainless steel, leather or natural fibers (hemp, sisal or cotton), and the link replaced by one of stainless steel.

Greg J. Harrison

Fig 6.20 | A decorative palm tree is a safe house plant but items in the soil such as fertilizer beads and other slow release items can be toxic.

Greg J. Harrison

Fig 6.21a | Lead weights used to balance car tires and sink fish bait. These are extremely toxic, being rapidly dissolved in the ventriculus and absorbed into the blood stream.

Greg J. Harrison

Fig 6.21b | Curtain weight made of lead.

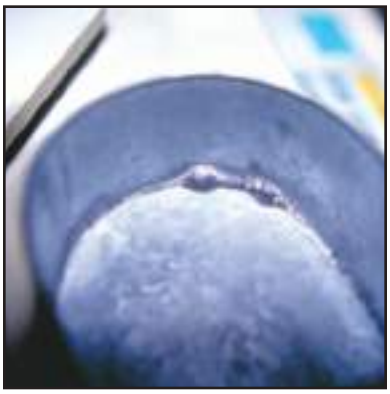


Fig 6.21c | Lead soldered galvanized water dish.


Greg J. Harrison

Fig 6.22 | A dangerous toy's clip has gotten stuck on this African grey's beak.


Greg J. Harrison

Fig 6.23a | While tobacco can be toxic if ingested, smoke from cigarettes is the product's greatest danger to birds. Contact with nicotine has been considered as a cause of dermatitis.


Greg J. Harrison

Fig 6.23b | Shelled nuts often get rancid unless refrigerated or stored in glass or foil bags.


Greg J. Harrison

Fig 6.23c | Seeds infested by insect larvae that hatch often become "webby" due to a fungus (usually *Aspergillus*) and can produce mycotoxins. Do not freeze webby seed and then feed it. The larvae from a grain moth has penetrated this sunflower seed.

Greg J. Harrison

Fig 6.23d | Chocolate contains dangerous levels of theobromine and caffeine. Chocolate containing less sugar generally contains a higher level of toxic substances.


Greg J. Harrison

Fig 6.23e | Although pothos is considered poisonous in small animals, it has never been a proven cause of systemic toxicity in healthy pet birds.


Greg J. Harrison

Fig 6.24 | Mineral blocks, especially when provided to birds on formulated diets, could add toxic levels of calcium (see Chapter 4, Nutritional Considerations).

Greg J. Harrison

Fig 6.25 | Palm nuts are eaten by wild hyacinth macaws. Cattle consume and digest the sticky, fleshy outer coating. The seed on the left has been passed out in feces; birds prefer to eat the nut in this form.

Greg J. Harrison

Fig 6.26 | Animal protein (meat, egg, cheese) has been removed from pet bird diets for 15 years with excellent results. Whether adding scientifically formulated amounts of animal protein to the diet of breeding birds is warranted is still unknown. Vegetable protein diets have been empirically proven to be sound.

Greg J. Harrison

Fig 6.27 | Offering excessive amounts of unbalanced foods allows the bird to choose its diet and nutritional disorders result. The amount of food shown was offered twice a day. The immature corn (sweet corn), baby beans, zucchini, and squash are of little nutritional value. The broccoli, kale and carrots are difficult to digest. While no sunflower seeds are offered, safflower is just as imbalanced, being even higher in fat than sunflower seeds. Peanuts are also high in fat, and when fed without the shell, often become rancid. Peanuts are a common source of mycotoxins. If they are fed at all, a human grade of peanuts certified free of mycotoxins should be used.

Greg J. Harrison

Fig 6.28 | An aviculture diet used commonly in the 1980's. Birds on sunflower seeds, apples, oranges, grapes, pound cake and bread rapidly developed nutritional disorders, especially the breeding females. Nutritionally deprived parent birds were unable to raise their young. Incubation of the eggs and hand-feeding from hatching had to be employed. The associated developmental problems in the young disappeared when a formulated diet was instituted.

Greg J. Harrison

Fig 6.29a | Sunflower seed, millet and canary seed are the historic staples of the bird food industry. Only one current manufacturer of formulated diets uses these century-old ingredients and improves their nutritional balance with appropriate additives. This has improved acceptance and avoided potential problems that one may get when attempting to incorporate novel ingredients. New, untested ingredients can create unforeseen problems that may take decades to discover.

Greg J. Harrison

Fig 6.29b | Since no standards have been officially declared, diets such as the one pictured above are incorrectly marketed as "complete diets" in many pet stores. Colored seed is the largest part of the pet bird food market in the USA. Newer versions of these colored seed diets have added shaped and colored pellets but empirically are not nutritionally superior to plain seeds.

Fig 6.29c | A scarlet macaw taken from a USA pet store magazine ad for a product that claims to have been developed by their researchers "to make a new all natural... treat (sic) premier supplement for all caged birds — and better than all other brands." The abnormal color and physical characteristics of the bird's feathers would indicate the company, their researchers and/or advertisement personnel are uninformed as to the desired physical attributes of a healthy bird.

Edwardo Nycander

Fig 6.29d | Free-ranging scarlet macaws hand-raised on an organic formula in Tambopata, Peru. Notice the tight feather structure and brilliant colors compared to Fig 6.29c.

Greg J. Harrison

Fig 6.30 | An aviculturist's food bowls for macaws and large cockatoos on the left and Amazons and African greys on the right. The organic formulated nuggets are a high potency formula. During the non-breeding season the seeds and nuts are stopped and for some species the nuggets are changed to a maintenance product. After ten years of this diet, the common avicultural problems of infertility, incubation, raising, hand-feeding from day one, congenital and developmental conditions and chronic "infections" are no longer encountered. The parents incubate and raise the babies until a week or so prior to weaning, when hand feeding is initiated to assure tameness. With production increased and problems decreased, profits at this aviary have soared.

Greg J. Harrison

Fig 6.31 | Twenty-five grams of whole grain (dense) organic nuggets (on the right) compared to an equal weight of traditional extruded brand made from refined flours that expand more readily. The fiber and other ingredients in whole grain flours lack the refined carbohydrates necessary to get the extrusion-induced expansion (fluff) attained with extrusion of these less-nutritious refined flours. These less expensive refined flours are by-products of the oil industry. The end product produces larger, lighter bags of food at a reduced cost. Such products require more additives than whole grains to establish balanced nutrition.

Greg J. Harrison

Fig 6.32 | Quantity of pellets consumed in a day by a cockatiel for $.01 (USA). Many owners waste food as they are not properly educated on proper food volumes.

Greg J. Harrison

Fig 6.33 | While many theme parks' cage labels state that the birds eat a formulated diet, the bowls are often full of a seed mix with colored pellets. Nutritional disorders are commonly observed in such a facility. Incentives such as free foods, cash donations and revenue sharing may be given to the facility for reciprocal endorsements that have little to do with the actual food fed or its nutritional value.

Greg J. Harrison

Fig 6.34 | Seeds top-dressed with a powdered mineral/vitamin supplement show the powder on the bottom of the food cup, which is subsequently disposed of with the seed hulls.

Greg J. Harrison

Fig 6.35 | Veterinarians in the USA often recommend a pressed seed cake. The waste in this bird's cage shows that most has been discarded except for the oat hearts. The food coating (egg, minerals, and vitamins) was discarded untouched. This bird had liver, respiratory and orthopedic problems that improved when switched to an organic formulated nugget that could not be selectively consumed.

Greg J. Harrison

Fig 6.36 | Organic formulated nuggets are low in synthetic vitamins and have natural sources of nutrients (alfalfa and spirulina for carotinoids, kelp for iodine and trace minerals, sea salt, natural clay with naturally chelated minerals, high soluble fiber from digestible hulls and psyllium). These nuggets are free of pesticides, food coloring, by-products and preservatives. The absence of preservatives requires cool, dry, dark storage in airtight containers to prevent rancidity or loss of nutrient value (see Chapter 4, Nutritional Considerations, Section II Nutritional Disorders).

Greg J. Harrison

Fig 6.37 | Vitamins in this bird's water color it yellow. Many birds will not drink freely from water with this color and taste, and dehydration can result. Bacterial and fungal growth is also encouraged in this medium. Finally, the dilution of the vitamins and exposure to water, air, heat and light degrade many of the labile vitamins.

Greg J. Harrison

Fig 6.38 | Water is often contaminated by improperly designed purifiers. Many allow bacterial proliferation or fail to remove pollutants. In the USA, reverse osmosis (blue and white canisters), bottle and tap water are commonly provided. Only water labeled "USDA drinking water" is regulated and must meet government standards.

Greg J. Harrison

Fig 6.39a | Plastic pipe and water hoses have been incriminated in chronic *Pseudomonous* spp. infections.

Greg J. Harrison

Fig 6.39b | Inadequately cleaned plastic bowls are potential sources of bacterial infections. Crock and stainless steel are less porous and thus less likely to be a problem. Water bottles are best constructed of non-porous glass.

Greg J. Harrison

Fig 6.40 | Salt/mineral spool, mineral block, powdered calcium, grit and oyster shell are not necessary for birds fed formulated diets, and can be harmful (see Chapter 4, Nutritional Considerations).

Mimi Walling/We Shoot Birds

Fig 6.41 | Male cockatiel on a seed and table food diet. Note the bent tail, ruffled feathers, and excessive pin feathers over the shoulder and crown. The same bird is shown in Fig 6.3a 6 months after conversion to an organic formulated diet.

Pediatric Patients

Concerns with babies still being hand-fed add additional elements to the anamnesis.

What hand feeding formula is being used? Is the formula being prepared according to the manufacturer's recommendations? Is anything extra being added to an already balanced diet?
It is common to see a hand-feeding formula designed for macaws used on a baby *Cacatua* spp., with resultant hepatic lipidosis. Likewise, additives such as peanut butter, macadamia oil and sunflower seeds can add additional fat and detract from other necessary nutrients.

Conversely, many prepared formulas for hand-feeding have insufficient calories per unit volume for some species of psittacines, notably macaws.

What is the temperature of the food when fed? What are the quantity, frequency and method (syringe, tube, spoon, cup, weaning pellets) of feeding?
Using a microwave to heat the formula can lead to crop burns. What is not commonly understood is the following: food heated in a microwave oven can have hot spots due to uneven heating. When water is heated in a microwave oven and then poured into another container to be mixed with the formula, the temperature of the resulting formula will be more uniform. The temperature of the formula should still be accurately assessed with a thermometer.

However, if the same container in which the water is heated is used to receive the powered formula, and multiple syringes are extracted over several minutes from this container, disaster often occurs. Many containers hold heat from the microwave, and gradually transfer this heat into the formula, causing the subsequent syringes that are delivered to be much hotter than the initial temperature reading indicated. Severe crops burns can result from this practice.

Conversely, baby birds may refuse formula that is not warm, and cold formula can delay crop emptying.

Various methods of administering the formula are used. Most common still is the use of a syringe, which allows an accurate determination of the quantity of formula being consumed. Spoon and cup feeding are also used successfully by some. Soft plastic or rubber tubing can be used, and this method does decrease the mess produced by bobbing, but carries the inherent risk of accidental ingestion of the tube if it becomes detached from the syringe. It also may not be as psychologically satisfying as having food that can be tasted.

The use of soft, warm, solid foods for feeding and weaning is advocated to more closely approximate the natural feeding patterns of these birds. This technique has been extensively used by Phoebe Linden, of Santa Barbara Bird Farms, with promising results.

Inexperienced hand feeders often cause aspiration or inadvertent starvation of birds while hand feeding. Generally, a baby bird can accept roughly 10% of its body weight per feeding. The frequency of the feedings and the quantity of each feeding will vary with the age, species and individual.

Common problems to look for in the history and physical examination of hand-feeding baby birds include:

1. Insufficient calories being given. Often a new bird owner will adhere to guidelines that state a bird should have a reduced number of feedings per day at a given age. The owner may fail to understand that this is based on the supposition that the bird is starting to eat on its own at this age. Some birds have not yet even been offered food, but the frequency of feedings has been severely reduced, leading to weight loss and debilitation.
2. Over-distention of the crop. This can have many causes, but over-distention may be obvious on physical examination and may be associated with a history of feeding an excessive volume at a given feeding.

BEHAVIOR

A behavioral history is becoming increasingly important as pet birds move out of their cages and into their owners' lives. Just as countless dogs and cats are euthanized every year because of behavioral problems, many birds suffer a similar fate or are transferred from household to household.

As psittacine behavior is determined largely by the interaction between the bird, its owners and its environment, questioning must focus on these areas. Who is the primary caretaker? Whom does the bird seem to prefer? Does the bird dislike anyone? How many hours per day does the bird spend alone? What does the bird see and hear when it is alone? Does the bird spend time with other birds or other pets?

Many factors that may influence pet psittacine behavior are yet to be determined. For example, recent work has indicated that fluorescent lights are perceived as a constant flickering by the eyes of birds, and both physiologic and behavioral problems may arise from previously unrecognized sources such as these. Also, there is increasing concern that our traditional methods of hand-feeding may by laying the groundwork for the development of behavioral problems later in life (see Chapter 3, Concepts in Behavior).

It is important to clarify the bird's interaction with its

owners (human flock). How tame is the bird? Does it readily step up onto a proffered hand? Does it always try to move up onto a person's shoulder? Is this allowed or encouraged? Does the bird talk? Does it like to be petted? Where? How does it react to different family members? How does it react to strangers? For the potential significance of the answers to these questions, see Chapter 3, Concepts in Behavior.

The Presenting Complaint

Once the bird's background has been established, it is time to assess the reason(s) the bird has been presented. Ask the client to describe the problem. Do not interrupt the client other than to seek clarification of details. You may need to repeat back to the client what they have said to ensure that a mutual understanding and clarification of the owner's concerns are reached. For example, many birds presented by their owners for "diarrhea" are actually polyuric. An explanation of the difference between diarrhea and polyuria, and a determination of which is actually present, should be made prior to the physical examination and diagnostic testing.

Once the practitioner has identified the problem(s), appropriate questions must be asked of the client to determine duration, severity, progression, previous diagnostics if known, previous therapeutics, and response to prior treatments. When did it start? Is this the first time it has happened? Have other treatments been tried? Who prescribed these treatments? Did they work? Is this condition static, progressive, or resolving? Are other birds affected?

The history taking as described above is not comprehensive. As one gains information, areas of concern will become apparent, and additional questions may be needed for clarification. The clinician must not dominate the conversation — rather, he or she should ask short questions and listen carefully to the client's reply. However, the clinician must be prepared to guide the discussion, to ensure that the maximum amount of useful information is obtained.

The Distant Examination

During the history taking, the clinician should be observing the bird and noting its behavior prior to restraint.

Most birds, no matter how ill, will make an effort to mask their clinical signs when first brought into the examination room. The clinically ill bird will be unable to maintain this pose for any length of time. Avoid disturbing the bird until it has settled, otherwise valuable clinical signs may be overlooked. The practitioner should be seated in order to remove any predatory threat to the bird. This will expedite its relaxation and therefore accelerate the demonstration of clinical signs.

Observe the bird's respiration (Fig 6.42a). Once the bird has settled in its cage in the examination room, there should be no open-mouthed breathing, marked tail bobbing, increased respiratory effort or audible respiratory noise. The presence of these signs should alert the clinician to potential respiratory compromise. Respiratory compromise may be due to true respiratory disease, cardiac disease, space-occupying mass or fluid within the coelom, anemia or severe debilitation. Care must obviously be taken with handling patients that are demonstrating respiratory distress.

The bird's posture should be observed (Figs 6.42b-d). Sick birds that are hypothermic will fluff their feathers in an attempt to conserve body heat. They sit still to conserve energy and, as they weaken, they sleep more (Fig 6.42e). Such signs are the classic "sick bird look," but not all sick birds will display these signs.

Evidence of a wing droop, lameness or reluctance to bear weight on one leg may indicate a musculoskeletal problem or a central or peripheral neurologic affectation. Spinal deformities can often be detected by an abnormal positioning of the tail. An upright position with a wide-legged stance may indicate egg binding or a similar space-occupying mass in the coelom or cloaca. Birds that hold both wings away from their body and pant are usually heat stressed or severely oxygen deprived.

The bird's plumage can be cursorily examined prior to restraint. Normally the plumage should be sleek, well groomed and clean. Untidy or dirty plumage may indicate that the bird is not grooming itself, or that there is some type of feather abnormality. Discolored feathers can reflect a variety of problems, including PBFD, chronic liver disease, excessive handling with oily hands or malnutrition. Closer examination of the feathers is warranted when the bird is removed from its cage.

Note the condition of the beak and toenails. Overgrown or flaky beaks or overgrown and twisted nails may be associated with PBFD, poor husbandry, chronic liver disease or malnutrition.

Watch as the bird defecates, looking for signs of straining or discomfort and listening for any accompanying flatulence or vocalization. Birds do not generally pass any gas and should be able to defecate effortlessly.

Observe the bird's behavior, assessing how tame the bird


Greg J. Harrison

Fig 6.42a | Prior to handling this dyspneic female cockatiel oxygen is warranted (see Chapter 7, Emergency and Critical Care).


Greg J. Harrison

Fig 6.42b | The owner noted that this pearl female cockatiel was fluffed and not eating its seed diet.


Greg J. Harrison

Fig 6.42c | The same bird as in Fig 6.42b is shown preening. Physical exam demonstrated an egg in the reproductive tract. This exemplifies the ability of a bird to mask symptoms.


Greg J. Harrison

Fig 6.42d | A 20-year-old lutino cockatiel is shown after having the jugular vein wet with alcohol for venipuncture. At this age, geriatric considerations are pertinent. Even this minor restraint has caused the bird to appear listless and sleepy. Geriatric (or otherwise stressed) birds may require more gentle handling and for shorter periods of time. Geriatric birds may benefit from the addition of fatty acids and herbs to the diet (see Chapter 10, Integrative Therapies). Geriatric pet birds are becoming more commonly seen in practice.


Greg J. Harrison

Fig 6.42e | This finch (or any bird that is motionless and on the bottom of the cage) is a high risk patient. The clients need to be informed in advance of the danger of handling such a sick bird. Heat and oral electrolytes, glucose and caffeine are often the best first steps. The prognosis for such birds is guarded, and a simple breast muscle evaluation will determine if emaciation is present. This commonly encountered finding confirms the chronicity and severity of the bird's condition (see Fig 6.2c).

is and whether it is showing any overt sexual display towards the owner, objects in its cage or other people present in the room. Note the owner's interaction with the bird, as this may reveal valuable clues to relationships at home.

THE CAGE

The cage must be of sufficient size to allow the bird to extend and flap its wings and to turn around without damaging feathers. The bird should ideally be able to express its normal behaviors within its cage, unless the cage is used only as a roosting space while the bird has access to a larger environment. The cage should be constructed of materials that are safe and appropriate for the size and power of the bird's beak. Small gauge wire, while suitable for smaller psittacines, is readily chewed and eaten by larger birds, often leading to heavy metal toxicity. Similarly, poorly galvanized cheap wire often has tags of zinc on it that are easily picked off and swallowed by psittacines of all sizes. Unsealed wooden cages are inappropriate, not only because many birds will chew the wood, but also because wood is impossible to disinfect, making it difficult to maintain adequate hygiene.

The floor of the cage should not be covered with grit, sand or wood shavings. The substrate of a cage is rarely changed with sufficient frequency, and ingestion of substrate material can lead to blockage of the gastrointestinal tract. Newspaper is a non-toxic and readily available substrate. It is also inexpensive, which encourages frequent changing.

Many cages are sold with plastic or wooden dowel perches. These are rarely suitable, since the smooth, unchanging surface and diameter offer little exercise for the feet and toes, and the symmetry can create constant pressure on selected areas of the feet, leading to pododermatitis. The uninformed owner may be reluctant to discard these perches, assuming that since they were supplied with the bird cage that they are appropriate. Just as variation in diameters and surfaces of perches are important for the individual bird, birds of various sizes and species require different ranges of perch diameters.

The positioning of the perches within the cage is also important. Birds tend to sit on the higher perches, so the diameter and texture of these perches may need to be alternated. Perches should not be placed so that a bird will be defecting into its food or water dish. A bird may be encouraged to sit on a concrete perch if a food dish or treat cup is placed so that access to this cup is achieved by perching on the desired surface.

Dishes should be constructed of a material appropriate to the species using them, and free of contaminants such as lead, which has been used as a solder to repair cheap galvanized dishes. Galvanized dishes should not be used, as the zinc in the galvanized coating may leech into the food or water. Dishes should be cleaned daily and positioned where they are unlikely to be soiled. Food and water dishes should not be placed alongside each other, as many birds will drop their food into the water, producing a broth within a few hours. Birds that tend to dunk their pellets into their water may need to have the water and food dishes placed on opposite sides of the cage.

Toys should be appropriate for the bird, and should not be so numerous as to restrict the bird's movement within the cage. Cheap toys, especially bells, are a common source of lead or zinc. Metal items that can be attached to a magnet are iron based. Shiny silver-appearing metals are often galvanized and polished and are a potential source of zinc toxicity (see Figs 6.19a-c). Toys should be made of natural materials, such as rope and wood, and should be replaced as soon as they become frayed. Bathing or misting should be available on a regular basis (see Chapter 3, Concepts in Behavior).

ODORS

Birds that are exposed to significant cigarette smoke will absorb the odor of smoke onto their feathers. Problems ranging from respiratory disease to feather destructive behavior have been linked to excessive exposure to smoke.

The feces of birds with enteritis, especially due to clostridial species overgrowth, have a distinctive, fetid odor. This seems to be most prevalent in cockatoos with fecal retention and cloacal prolapse and in birds with extensive and restrictive cloacal papillomatosis, although it may occur in any bird. The detection of this odor in a bird's stool should be pursued diagnostically, usually by first performing a Gram's stain on the feces.

Owners may present their bird, commonly an Amazon, for "bad breath." This is usually the natural smell of these species, and not related to disease.

Examination Room Equipment

Appropriate equipment for use in the examination room includes:

- A supply of freshly laundered towels of different sizes (or paper towels) for restraining birds.
- Scales capable of weighing in grams, preferably with a detachable T-perch (to allow birds to perch on while being weighed), and a container in which to weigh smaller, fully flighted birds.
- A training perch for the bird to perch on while being examined.
- Clinical equipment, such as stethoscope, a focal light source, magnifying loupes, needles and syringes, blood collection tubes and culture swabs.

The use of heavy gloves to catch and restrain birds should be discouraged. With these gloves on, the clinician cannot be sensitive to small movements of the bird, and can easily hurt or even kill the patient. Additionally, these gloves cannot be cleaned or sterilized.

Ensure that the room is escape proof, and that clinic staff will not enter the room unexpectedly. Avoid stressful sights and sounds such as dogs, cats and other potential predators.

Handling and Restraint

Once a thorough history has been obtained and the bird observed in its cage, the next step is to examine the patient more closely. In order to do so, the bird will

need to be handled and restrained. In the case of aviary birds, this should be done with a view of minimizing stress to the bird, while at the same time, avoiding injury to the handler. Many companion birds, on the other hand, have learned to trust humans and regard them affectionately. Destroying this trust through aggressive catching and handling techniques can adversely affect the bond between owner and bird. This relationship must be preserved, and handling techniques for closely bonded birds should emphasize minimal stress and fear (see Chapter 3, Concepts in Behavior).

As the oils on human skin can be detrimental to the feathers of many species, a light dusting of unscented talcum powder on the clinician's hands is appropriate before beginning an examination.

Be aware that the scrubs or other clothing worn by the technicians and practitioner will be exposed to powder down and fecal material during handling and restraint. The potential for disease transmission to subsequent patients should be considered and clothing changed when appropriate.

By the time the clinician is ready to examine the bird, the general temperament of that bird should have been established. If the cage is the bird's home, its territorial instincts may drive the bird to protect its cage from the intrusion of strangers. In many cases, therefore, it may be appropriate to ask the owner to remove the bird from the cage. If the owner is unwilling (or unable) to do so, the clinician should study the cage to determine the best means of removing the bird. Tame birds may simply step through an open door. At other times, the cage may need to be dismantled rather than trying to catch and remove a bird through a small door. If the bird is friendly, the clinician should gently introduce a hand into the cage, with the back of the hand to the bird. If there is no aggression, the forefinger is extended and placed under the bird's chest. A tame bird will usually step onto the finger. Restrain the foot or a toe by gently pressing on it with a thumb against the finger, keeping the bird steady, and gently bring it out of the cage. During this procedure, the clinician should be talking to the bird, praising it, and maintaining eye contact. Once the bird has been removed from the cage, continue to praise it and, depending on the species and individual, scratch its head, its axilla, or simply continue talking to it while raising it to a height at which it appears comfortable. If the bird has to be physically caught, it is usually best to use a small hand (or paper) towel to gently envelop and then restrain the bird. Show the bird the towel and let it become accustomed to its appearance. If possible, the clinician should envelop the bird in the towel from below — an approach from above is potentially a very intimidating experience for a pet bird. Keep talking in a friendly voice, and maintain eye contact (see Chapter 3, Concepts in Behavior).

Birds that are not tame can be caught using a towel as described above. These birds will rarely stay still during capture, so a quick capture is the best approach. The most dangerous part of the bird's body should be immobilized first (ie, psittacines = the head and beak; raptors = the feet). Once the dangerous areas are immobilized, the bird is wrapped in the towel and removed from the cage.

When the bird has been removed from the cage, the next step will be determined by the bird's tolerance of handling. Very tame birds can be placed on scales to be weighed, while less tame birds may need to be examined first while still restrained and weighed just before being returned to their cage.

At all times the clinician must be aware of the bird and how it is handling the stress of restraint and examination. Many birds are presented for evaluation of an illness, having been ill for a period prior to the owner's recognition of disease, and the stress of restraint can exceed their ability to compensate. Collapse and death are, unfortunately, not uncommon with critically ill birds. If there is any doubt as to the bird's ability to cope with the stress, it should be immediately returned to a perch or the cage and allowed to regain its composure before proceeding. Critically ill birds should not be handled initially (see Chapter 7, Emergency and Critical Care).

THE "PUT IT DOWN" LIST

Panting and increased respiratory rate while being examined warrant attention. It may be that these are normal compensation techniques for a stressed or obese bird, but during restraint, it is difficult to determine the extent of the stress without reducing the effectiveness of the restraint.

1. If the bird is panting or breathing rapidly, first alter the grip on the head so the head is free to move. The bird should immediately begin to turn its head in search of something to bite. If it doesn't, PUT IT DOWN.
2. A paper towel or a corner of the towel being used to restrain the bird can be placed into its mouth. It should immediately begin to bite at this, demonstrating that it has sufficient oxygen reserves to do so. If the bird lets the material lay limply in its mouth, PUT IT DOWN.
3. Have the bird grasp your hand or finger with both of its feet. (This should be part of the physical exam anyway, to determine symmetry and strength of grip). If the bird's grip is weak or non-existent, PUT IT DOWN.
4. If the bird's eyes close during the physical exam, PUT


Greg J. Harrison

Fig 6.43 | Disposable paper bags provide a dark area for restraint for all but the largest of birds. This can be used to obtain a body weight on untamed birds.


Greg J. Harrison

Fig 6.44 | For birds that will perch a digital scale with an easily disinfected perch stand is ideal. In this case, the wooden perch is coated with an epoxy sealer.

IT DOWN. Conversely, do not be reassured if the bird has its eyes open — many birds have held their eyes open as they drew their last breaths.

5. If in doubt, PUT IT DOWN. Return the bird to the location (cage, owner) where it is most comfortable, and observe it while talking to the owner.

The Physical Examination

"You will miss more by not looking, then you will ever miss by not knowing."

The old veterinary adage expressed above is as true for avian medicine as it is for any other species. A thorough, systematic physical evaluation of the patient is essential to obtaining information regarding the bird's problem and diagnosis. Clinicians should develop a thorough examination protocol with which they are comfortable, and use it for every (stable) patient, regardless of the reason for presentation. A physical examination form may be useful in ensuring that nothing is overlooked.

BODY CONDITION

All birds should be weighed during each visit to the veterinarian, and at the same time each day while hospitalized. Monitoring an individual bird's weight will often detect potential disease prior to the demonstration of clinical signs. The veterinarian will also develop an appreciation for the normal body weight ranges of various species. The weight should be recorded in grams, as this allows accurate monitoring (Figs 6.43, 6.44).

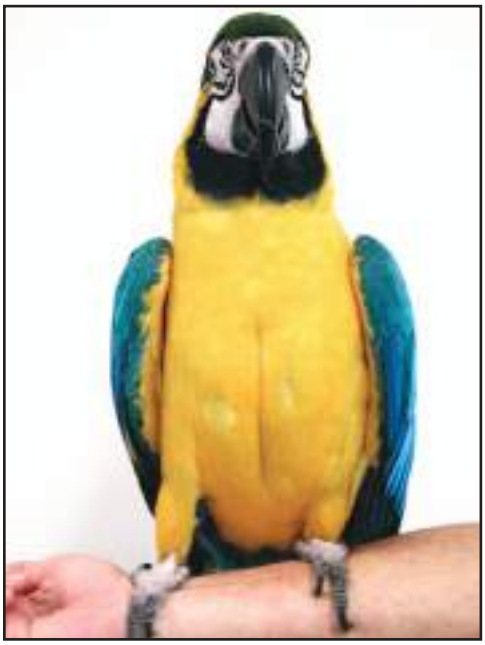
Traditionally a bird's body condition was determined by palpation of the pectoral muscles and allocating a body score based on the muscle and fat coverage of the sternum. Although useful as a cursory determination of emaciation, this technique fails to take into account that most birds do not store fat in their pectoral region and can be carrying significant fat deposits while still having an apparently normal body score. Wetting the feathers over the abdomen, flanks, thighs and neck with alcohol allows visualization of subcutaneous fat deposits, seen as yellow fat under the skin rather than pinkish-red muscle (Figs 6.45a,b-6.47a,b).

The combination of body-weight recording, pectoral muscle palpation and examination of subcutaneous fat allows an accurate assessment of body condition.

BLEEDING

Bleeding or bruising may be encountered during or produced by the physical examination. Excessive, prolonged or abnormal bleeding or bruising in birds is often related to one or more manifestations of malnutrition. The following is a brief list of the most common presentations and associated etiologies:

- Conjunctival hemorrhage or "red tears" are commonly seen in African greys and Quaker parakeets (see Fig 6.47a2 for information on potential etiologies).
- Denatured blood in the nasal debris of psittacines. This seems particularly prevalent in mutation cockatiels (see Fig 6.47b2). Malnutrition causing squamous metaplasia and secondary bacterial and fungal infections is a likely cause in many birds. In these cockatiels, however, there may also be a decrease in



Greg J. Harrison

Fig 6.45a | Body condition scores on simple pectoral profiling are not accurate. This blue and gold macaw is 1300 g and has cleavage in the area of the keel's carina. When the bird's feathers are wetted down with alcohol, no fat in the sub-cutaneous tissues is evident. However, on the commonly proposed body score technique, this bird would be called obese because the breast muscle exceeds the keel's carina in depth. Obese birds are considered high risk birds. This is a large blue and gold, but it is not obese.

Greg J. Harrison

Fig 6.45b | Same bird as in Fig 6.45a at a distance. Note the appearance of the feathers. They appear as a unit, not a collection of individual feathers. The colors are clear and crisp. The feather margins are smooth and sharp. The feathers are strong and straight. The skin is reptilian and boldly patterned. The nares, facial skin, eyes and nails are all exemplary. Max has been on a high fat organic formulated nugget for 10 years with limited fruits and vegetables. Natural sunlight and showers are frequently provided. Note the lack of flaking or layering of the beak. No flaking is present on the facial skin and no debris has accumulated in the nares.

intrinsic clotting factors. Verification and etiology of this coagulopathy have not been determined, but malnutrition and hepatopathy, as well as genetic predisposition, should be considered.

- Facial skin bruising is often noted in macaws and African grey parrots (see Fig 6.47c2). This can be the result of restraint that is too aggressive or an inherent bleeding dyscrasia. The same condition likely occurs in other species, but the presence of feathers in the periorbital area prevents observation of the bruising. Malnutrition is likely to be the major underlying cause of overly fragile dermal tissue and decreased coagulation factor production.
- Beak injuries (Fig 6.47d2).
- Broken blood feathers (Fig 6.47e2).
- Blood from the cloaca.
- Blood in the urine.
- Bite wounds (Fig 6.47f2).

PLUMAGE

Normal feather development in a baby cockatoo is shown in Figs 6.48a-g. Normal adult feathers are seen in Figs 6.49-6.56a-e and Chapter 2, The Companion Bird.

Attention should be paid to the following areas (Figs 6.57a-z-6.59c):

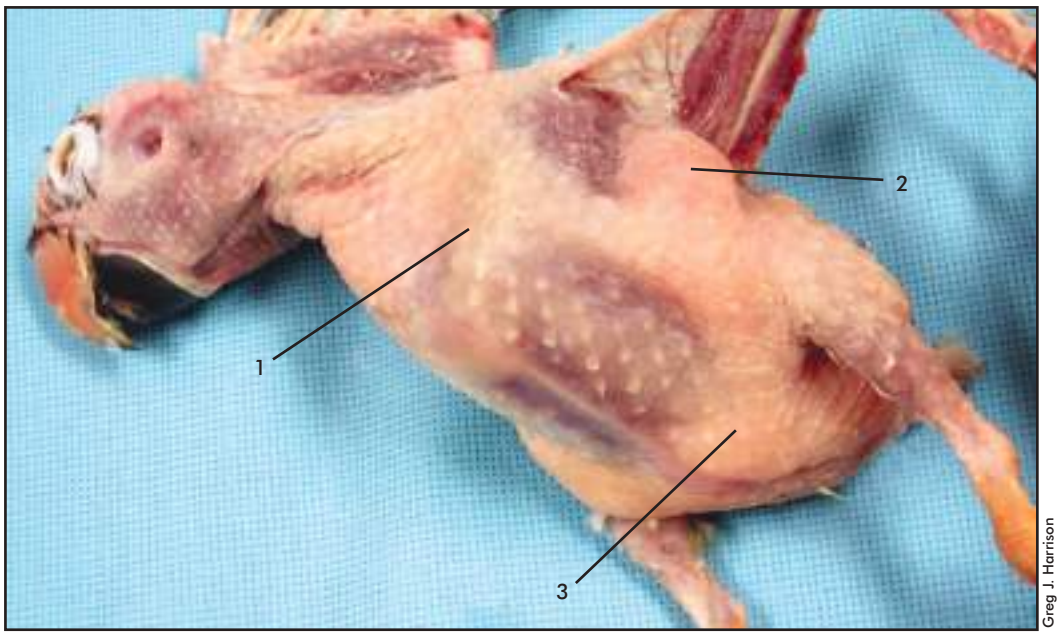
- **Color of the Feathers.** Abnormal coloration of feathers can be due to a multitude of causes. PBFD can cause green feathers to turn yellow and blue feathers to turn white. It will also lead to a generalized dirtiness of the feathers, especially in cockatoos. Chronic liver disease and/or malnutrition can cause darkening of feathers and a decrease of powderdown production in applicable species. Frequent handling of birds by the owner can leave a deposit of oil on the feathers, which then encourages fungal overgrowth. This causes a black discoloration on these feathers. This is not seen in birds with powderdown, presumably because the powder keeps the feathers clean.
- **Tidiness of the Plumage.** Birds generally keep their plumage well groomed and tidy. If the plumage is untidy, with no immediately obvious cause (eg, recent handling), the clinician should suspect that either the bird is unable to groom itself properly, or a generalized feather dystrophy (eg, PBFD) is present.
- **Evidence of Feather Damage.** Chewed and/or broken

Greg J. Harrison

Fig 6.46a | Alcohol can be used to part the feathers to ascertain the absence or presence of body fat. This bird has an accumulation in area three (the abdomen, just anterior to the vent). Other areas should be observed.

Greg J. Harrison

Fig 6.46b | This young budgerigar has a bulging fat mass at the furculum (area one).



Greg J. Harrison

Fig 6.46c | This euthanized blue-crowned conure has had its feathers removed to show the three fat areas coalescing. 1 is area one. 2 is area two. 3 is area three. Area two, the axilla, is still discrete. Note the fat is deposited subcutaneously to the feather tracts.

Greg J. Harrison

Fig 6.46d | Dorsal view of bird in 6.46c. Fat depositions continue in feather tracks over the wing, scapula and pelvis.

Greg J. Harrison

Fig 6.47a | A very ill conure is barely able to keep its eyes open and maintain its balance. Despite the presence of strangers it remains fluffed during clinical presentation. This implies a grave prognosis.

Greg J. Harrison

Fig 6.47b | Same conure as in Fig 6.47a. When the feathers are wet with alcohol, there is obviously little remaining breast musculature. This severe emaciation carries a grave prognosis. Handling such a bird without ascertaining its tolerance for restraint via proper distance observation will often precipitate a crisis. If the bird dies, the crisis will be with the owner. The owner will assume the bird died due to inappropriate veterinary care. Proper evaluation can avoid such a crisis. Most birds in this condition will not survive, but a few, when gradually and cautiously approached with treatment and diagnostics, will respond. In either case, the owner must be informed in advance of the severity of their bird's condition.

Greg J. Harrison

Fig 6.47a2 | Quaker (monk) parakeet with a drop of conjuctival blood post-examination. This phenomena is commonly observed in African greys when restrained. A nutritional disorder is usually involved. Subclinical rhinitis and sinusitis have been incriminated in this production of bloody tears from conjunctival hemorrhage, Squamous metaplasia of the respiratory system is likely underlying the respiratory disease. The degree of blood pressure elevation that restraint creates may also be a factor in more sensitive species (see Chapter 4, Nutritional Considerations).

Peter Coutteel

Fig 6.47b2 | Cockatiel with black rhinal discharge that is hemoccult positive. Secondary invaders are common and often require therapy. This therapy often includes nutritional correction.

Greg J. Harrison

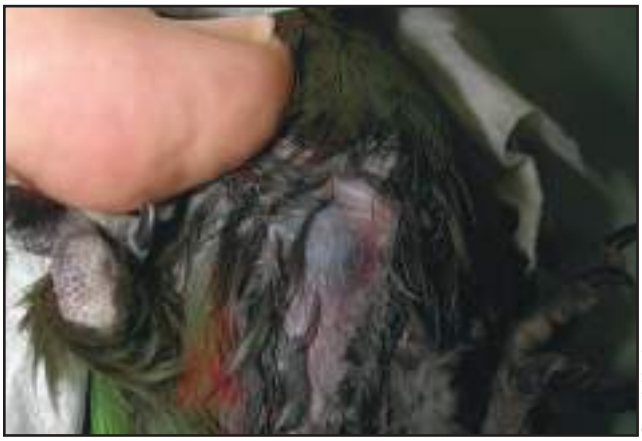
Fig 6.47c2 | Facial erythema and subsequent scabs like those shown in this African grey are often seen in macaws following restraint. While the immediate cause is the handling, the underlying skin fragility usually responds to nutritional correction.

Greg J. Harrison

Fig 6.47d2 | Budgerigar with beak bleeding after trimming. A nutritional disorder is likely. Budgerigars and other parrots do not normally require beak trimming. The vessels in a nutritionally imbalanced bird's beak grow closer to the tip and bleed more profusely. Styptics will usually control the bleeding.

Greg J. Harrison

Fig 6.47e2 | Mealy Amazon with a fractured primary remige. Pulling the feather is no longer recommended. Damage to the delicate structure of the feather follicle (see Fig 6.56c,e) may occur. Clip off the distal portion and apply baking flour to the stub until bleeding stops. Birds that bleed excessively may respond to injections of vitamin K. Nutritional disorders need to be addressed. In a well bird the feathers are less likely to fracture and if they do, the subsequent bleeding is generally self-limiting.



Greg J. Harrison

Fig 6.47f2 | According to the owner, this lovebird was mouthed but not bitten when "picked" up by their dog. Wetting feathers to examine for hematomas associated with tooth penetrations is an easy technique. The owner's assumptions were proven wrong in this case and antibiotics are indicated.

feathers should lead the clinician to suspect overgrooming, self-mutilation, cage mate trauma or malnutrition. Saw-toothed edges can indicate a failure to molt normally; hence, old, worn feathers are being retained. It should be noted in cases of feather destructive behavior, whether the feathers have been bitten off level with the skin, plucked out, or if the shaft is being chewed.

- **Evidence of Feather Dystrophies.** Retained feather sheaths, retained pulp, hemorrhage in the shaft of feathers, strictures of the calamus and twisted feathers are indicative of feather dystrophies, often of nutritional, genetic, traumatic or viral origin (ie, polyomavirus, circovirus).
- **Wing Clipping (if present).** The wings should be examined to determine if the bird's wings have been clipped and, if so, if that clip is appropriate to the species and temperament of the bird. The degree of lift that the bird is achieving with the current clip should be determined by asking the owner and by a "test flight" in a safe area, if needed. The owner's satisfaction and the effectiveness of the last clip should be determined. The clip should be examined to determine if the cut ends of feathers could be bothering the bird.
- **Absence or Presence of Powder Down.** Powderdown is produced by the powderdown feathers on the thighs of many species of birds, particularly cockatoos and African grey parrots. It is easily recognized by the presence of a fine white powder on the clinician's hands and clothing after handling the bird. A lack of powderdown leads to staining of the feathers and a shiny appearance to the beak and feet. The most common causes of loss of this powderdown include: malnutrition, hepatic disease, genetic mutations (notably in cockatiels) and circovirus in *Cacatua* spp.
- **Molting Patterns.** Most birds will molt heavily twice yearly, in spring and autumn — the so-called "pre-nuptial" and "post-nuptial" molts. Outside of these annual molts, there is a steady and progressive turnover of old feathers. The end result in psittacines is that each feather is normally replaced once a year. Continual heavy molts or the sudden loss of many feathers is abnormal, as is the failure to molt (seen as the retention of worn and broken feathers).
- **The Presence of Stress Lines or Stress Bars.** Stress or disease at the time a feather is growing will lead to a transverse "break" in the vane of the feather. The presence of many feathers with such stress lines is indicative of a problem in the bird's recent past.
- **The Condition of the Skin.** The presence of erythema, excessive scale or areas of skin trauma should be noted. This can be done by parting the feathers with a cotton-tipped applicator, or gently blowing on the feathers.
- **Areas of Trauma.** The skin should be thoroughly examined for areas of trauma, especially on the wing tips, sternum, cere, ventral pygostyle and axillae.
- **Flexibility of the Feather** (Figs 6.58a-c). The shaft of the feathers of a healthy bird on a good diet should flex rather than break when the tip is drawn down towards the base; the feather should spring back to a normal position when released.
- **Parasites.** The presence of parasites on the feathers should also be noted; microscopic examination may be necessary for detection (Figs 6.59a-c).


Greg J. Harrison

Fig 6.48a | Baby Umbrella cockatoo at day 2.


Greg J. Harrison

Fig 6.48b | At 2 weeks of age this bird shows a minor prognathism developing and crooked toes. This was likely a result of the seed-based diet fed to the parents.


Greg J. Harrison

Fig 6.48c | At 4 weeks of age. The organic hand-feeding formula has overcome the beak and toe problems.


Greg J. Harrison

Fig 6.48d | At 6 weeks of age the slight weakness of the abductor muscles in previous figures has been corrected and the baby is standing.


Greg J. Harrison

Fig 6.48e | Eight weeks of age.


Greg J. Harrison

Fig 6.48f | Ten weeks of age.


Greg J. Harrison

Fig 6.48g | Twelve weeks of age.


Mimi Walling/We Shoot Birds

Fig 6.49 | A perfectly feathered goffin cockatoo in a defense (attack) posture stimulated by the toy owl.

Mimi Walling/We Shoot Birds

Fig 6.50 | A Moluccan (salmon-crested) cockatoo displays a greeting feather erection and reaches out with the foot to be picked up. The bird has been fed an organic formulated diet since hatching, 8 years ago. A healthy bird can normally separate its feathers in this manner. Observe the beak, skin around the eye and the feet. Note the strength and vibrance of the feathers. The bird's attitude is upbeat and active. These are some determinations one needs to master in avian medicine. This requires either seeing normal specimens, such as this bird, or transferring in one's mind's eye the characteristics seen in most wild birds. If only seed-eating pet birds are seen in practice, normal is not appreciated.

Mimi Walling/We Shoot Birds

Fig 6.51 | Two grey-eyed (immature) African grey parrots. Color tones and feather scalloping on these birds are ideal.

Mimi Walling/We Shoot Birds

Fig 6.52 | This citron-crested cockatoo is normal except for a mild unzipping of the crest feathers.

Jan Hooimeijer

Fig 6.53a | This severe macaw is showing a defensive posture. The feathers are of poor color and structure. Such feathers often dramatically respond to diet change.

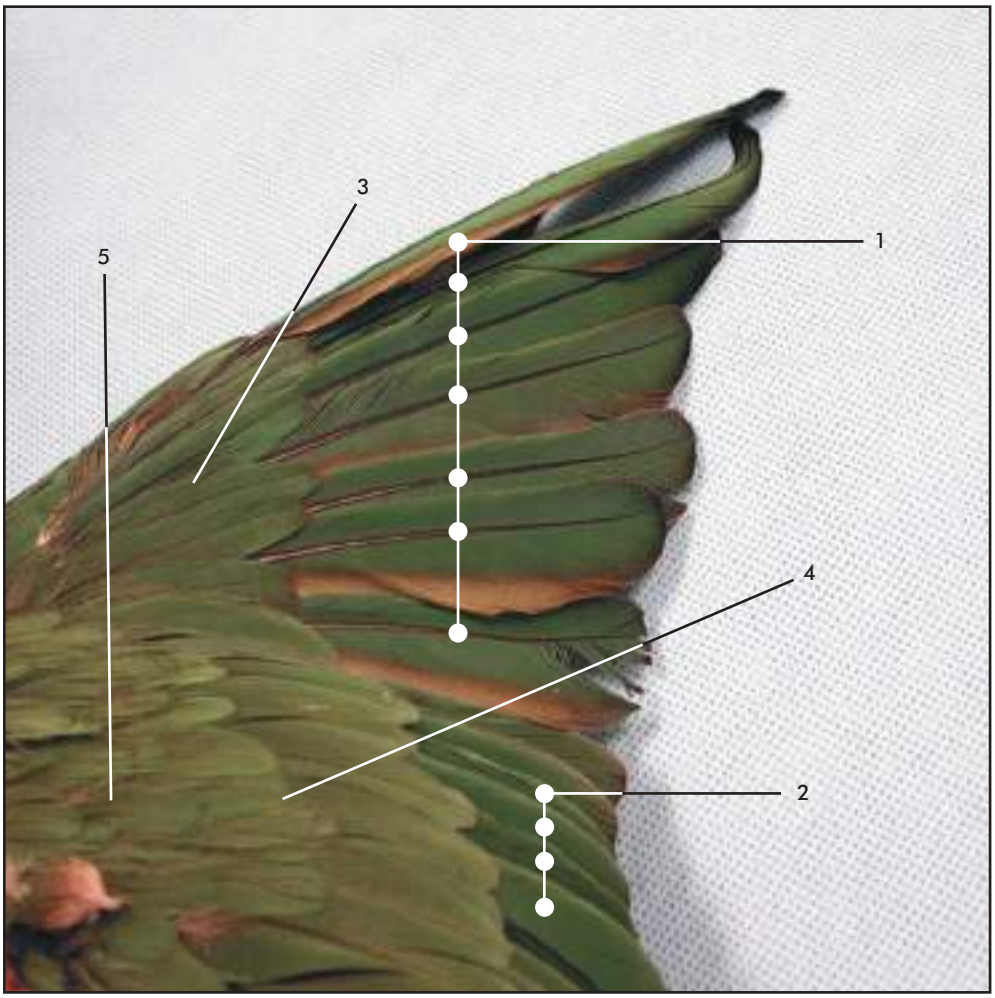
Jan Hooimeijer

Fig 6.53b | Approximately 1 year later, the bird in Fig 6.53a has been guided by Jan Hooimeijer into the specimen seen here. Owners were instructed in an hour long office consultation on nutrition, husbandry and behavior. The diet was changed to an organic nugget. Periodic evaluations were scheduled to assure secondary problems were not becoming clinically significant. When an avian practitioner has repeatedly seen improvement in these cases with only dietary correction, recommendations for diagnostics in future cases are often altered. A CBC may be warranted to determine if concurrent infection is present. Serum chemistries with bile acids may be performed. However, abnormalities in hepatic enzyme levels, calcium levels and other parameters are often a reflection of the effects of chronic malnutrition. In the absence of clinical disease, the practitioner may elect to institute a wellness program, with the primary emphasis on dietary correction. In this case an organic formulated diet, lactulose and milk thistle were administered. This is a particularly judicious approach if the bird is stable but likely suffering from malnutrition-induced decreased hepatic function. These birds are not ideal candidates for venipuncture due to potential clotting deficiencies. Even more significant, hepatic biopsy, although it may be diagnostic, can be a fatal procedure in the presence of hepatic insufficiency (see Chapter 4, Nutritional Considerations).

Greg J. Harrison

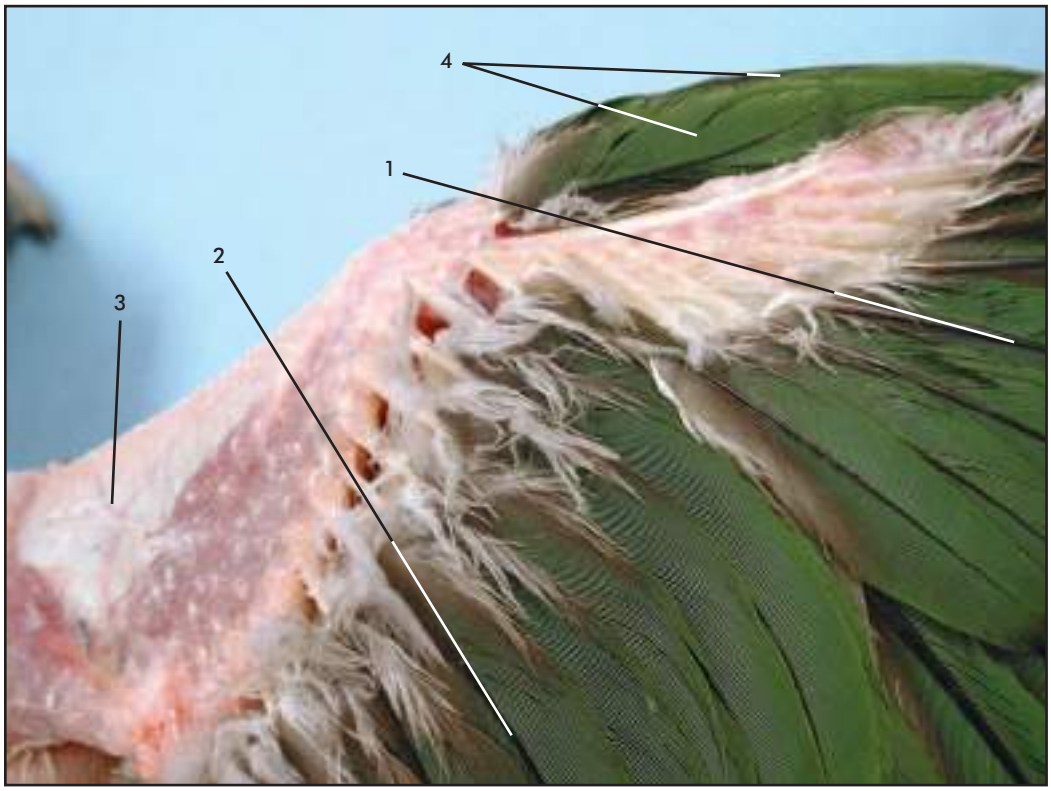
Fig 6.54 | A 17-year-old seed-eating cockatiel shows hyperkeratotic follicles, generalized weakness, worn feathers and retained pin feathers.

Fig 6.55 | A wild-caught sulfur-crested cockatoo (*Cacatua galerita galerita*) which is fed a seed diet, displays poor feathering. Note the fluffed appearance, unzipped crest feathers and its position on the bottom of the cage. The physical examination revealed a retained egg. Malnutrition was addressed over the next few visits.



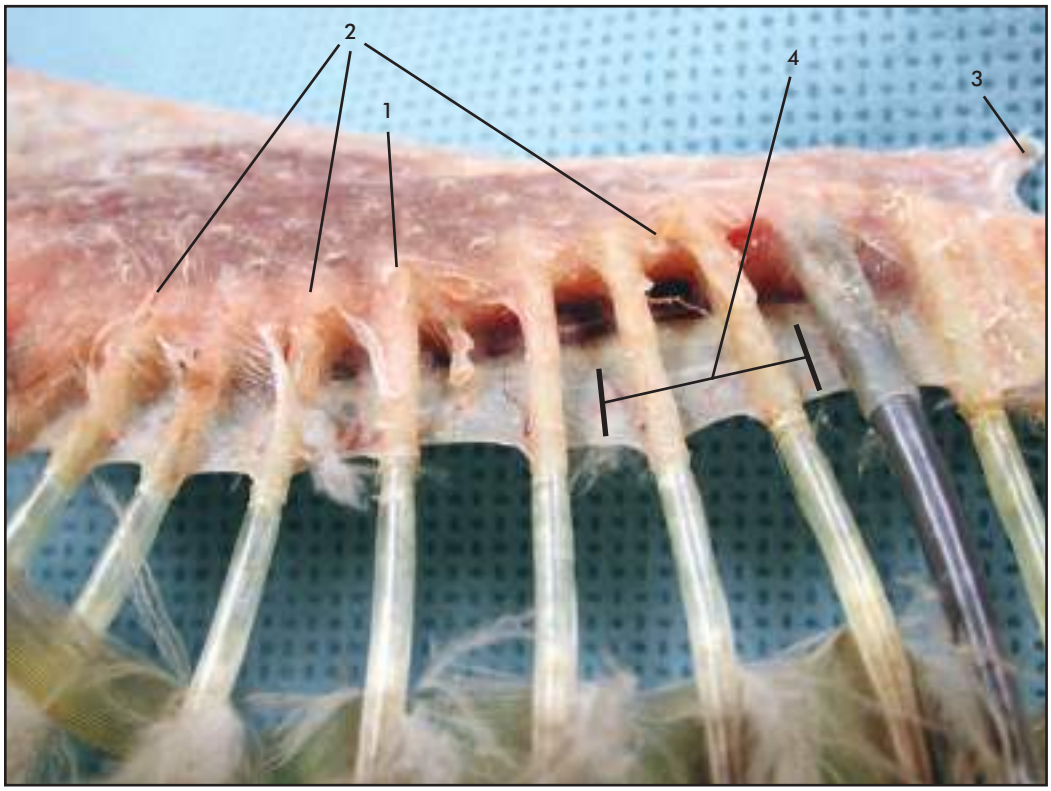
Greg J. Harrison

Fig 6.56a | Plumage of the extended right wing. Dorsal view. 1. Primary remiges 2. Secondary remiges 3. Primary coverts 4. Secondary coverts 5. Upper marginal coverts of the propatagium and manus.



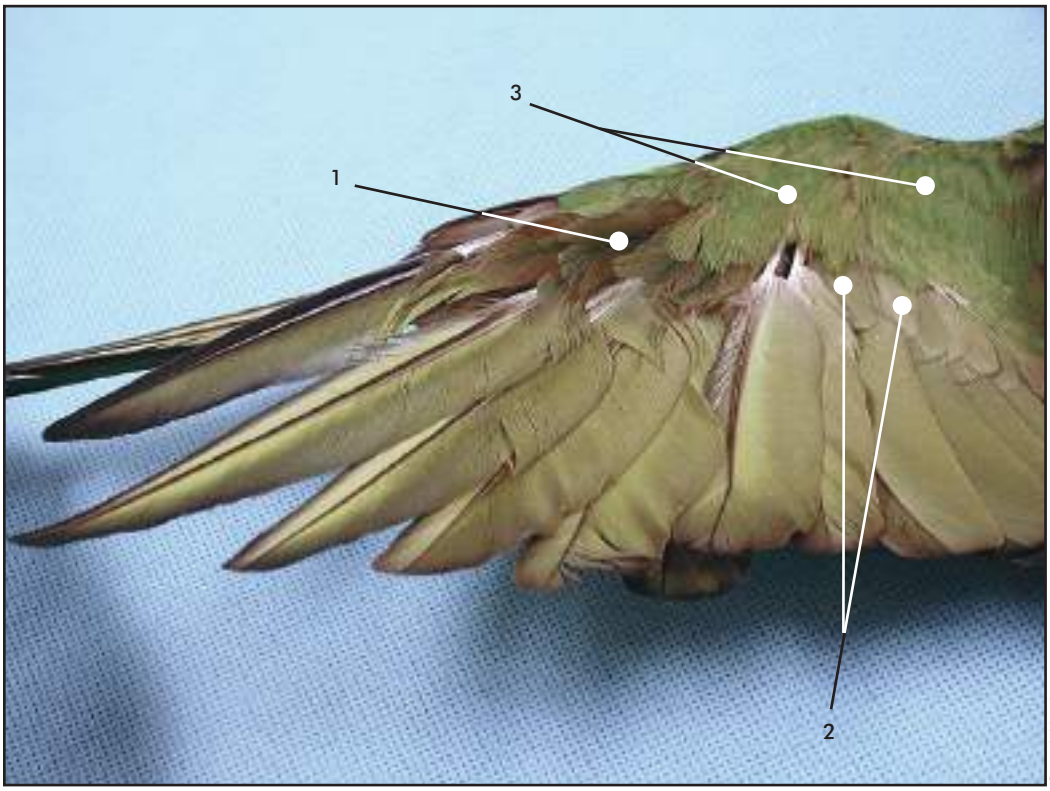
Greg J. Harrison

Fig 6.56b | Plumage of the extended right wing. Dorsal view. Upper marginal coverts of the propatagium and the manus removed. 1. Major primary coverts 2. Major secondary coverts 3. Propatagium 4. Alular remiges.



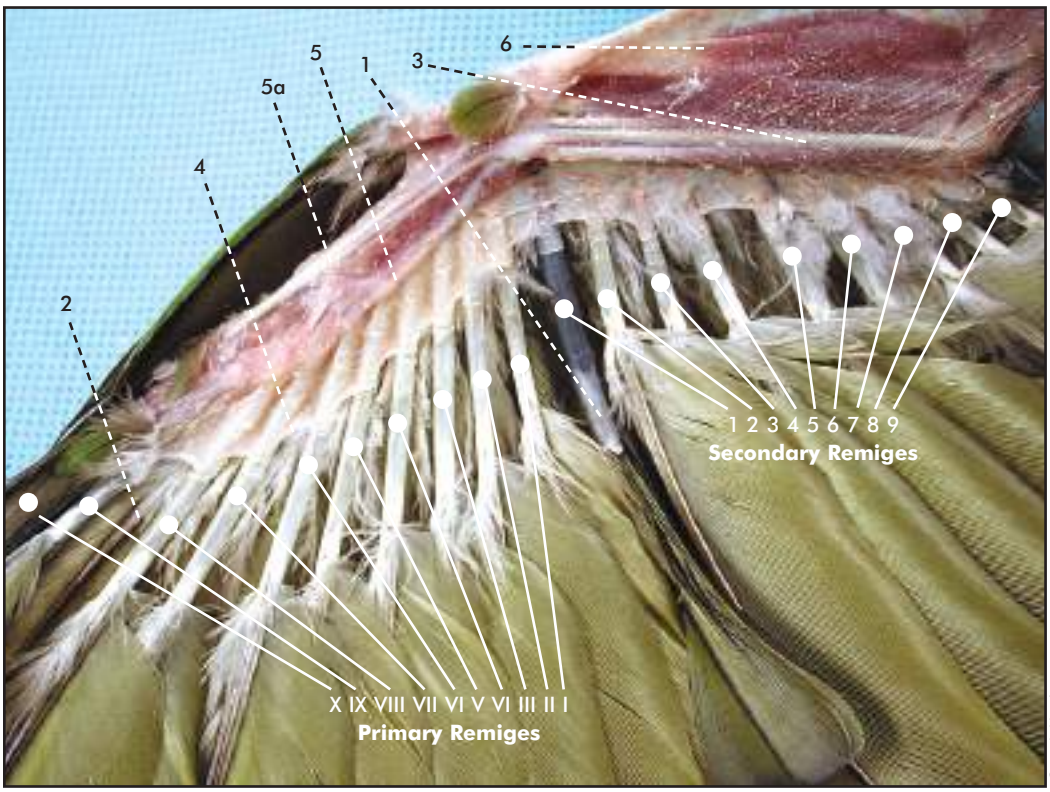
Greg J. Harrison

Fig 6.56c | Plumage of the right wing. Dorsal view. Major primary and secondary coverts removed. 1. Secondary remex inserting on dorsal surface of ulna 2. Follicles of insertion for covert feather 3. Alular digit 4. Postpatagium.



Greg J. Harrison

Fig 6.56d | Plumage of right wing. Ventral view. 1. Under wing primary coverts 2. Under wing secondary coverts 3. Under wing marginal coverts of propatagium.

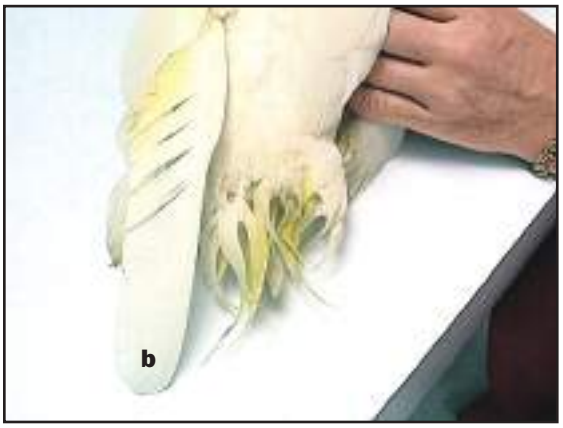


Greg J. Harrison

Fig 6.56e | Plumage of right wing. Ventral view. Under wing primary and secondary coverts and the marginal coverts of the propatagium have been removed. 1. Axial secondary pin feather inserting on the dorsal ulna. 2. Upper primary covert 3. Ulna 4. Postpatagium 5. Minor metacarpal III 5a. Major metacarpal II 6. Propatagium. I-X Primary remiges. (I - VI insert on metacarpals; VII the minor digit; VIII-X the major digit) 1-9. Secondary remiges (several additional ones were pulled).

Greg J. Harrison

Fig 6.57a | Four-year-old female umbrella cockatoo fed a seed and table food diet. The crest feathers are failing to shed the keratin surrounding the underlying pin feathers. The bald crown area is normal.



Greg J. Harrison

Fig 6.57b | Same bird as in Fig 6.57a demonstrating tattered and broken tail feathers compared to a normal umbrella's wing feather (b).

Greg J. Harrison

Fig 6.57c | This close-up demonstrates the difference between stained feathers developed by a bird fed a seed and table food diet compared to a perfect feather being held for comparison.

Greg J. Harrison

Fig 6.57d | These wing feathers show a lack of opacity and have a soiled appearance when compared to a perfect feather.

Greg J. Harrison

Fig 6.57e | Poor quality rump and tail feathers in a blue and gold macaw fed only seeds and table foods. The feathers lack a sharp vane margin (unzipped appearance). The feather color lacks uniformity and many transverse (stress) lines are present.

Greg J. Harrison

Fig 6.57f | Budgerigar on a seed diet with tail feathers unzipped. The lateral rectricies are a dull off-brownish-white compared to those they cover, which are pure normal white but still unzipped.


Greg J. Harrison

Fig 6.57g | The parents of this palm cockatoo were fed seeds, vegetables and rancid pine nuts soaked in chlorine bleach to remove molds. This baby was raised on a high protein commercial hand rearing product making up 70% of the diet with the other 30% consisting of 20 g of sunflower seed kernels, 40 g apple and 40 g broccoli. In addition to abnormal plumage there was a deficiency of normal flora in the fecal Gram's stain.


Greg J. Harrison

Fig 6.57h | Same bird as in Fig 6.57g 2 months after being removed from described diet. The bird was placed on an organic high fat diet. The head, neck and some wing coverts have molted and regrown in normal texture and color. The bird's timid and nervous attitude was replaced with a jolly playful one. Fecal bacteria were returning to normal.


Greg J. Harrison

Fig 6.57i | Same bird as in Fig 6.57g 6 months post-diet change. The color is uniformly normal. The beak has shed much of its retained keratin. The normal red cheek patch took three more months to appear.

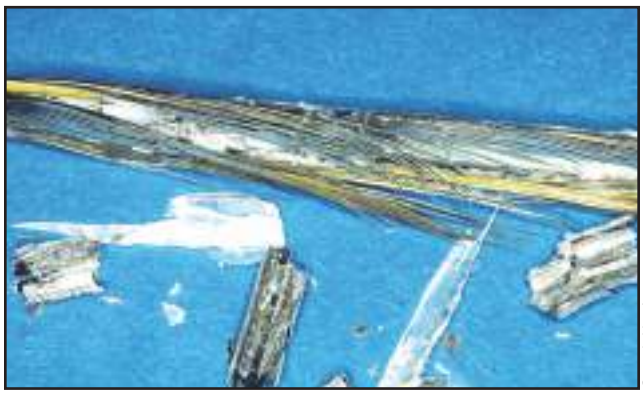

Greg J. Harrison

Fig 6.57j | A rectrice with retained sheath and pulp material that is normally shed. This is due to improper nutrient availability to the feathers and a resultant hyperkeratosis. Seed and table food based diets are the major cause (see Chapter 4, Nutritional Considerations).


Greg J. Harrison

Fig 6.57k | The two lateral feathers are from a bird with a nutritional disorder. The bird was fed a seed and table food diet. Compare these to the central feather of a bird with normal development on a proper diet. Color, texture, strength, and structure (width of vein) are compared on the feathers' ventral view.


Greg J. Harrison

Fig 6.57l | Dorsal views of three normal and three abnormal feathers from the same dietary situation(s) as described in Fig 6.57k.


Greg J. Harrison

6.57m | A sun conure and a gold-capped conure fed the same formulated diet. The sun conure developed yellow primary remiges. The addition of red palm oil, high in carotinoids and vitamin E, allowed the development of new blue feathers. The addition of wheat germ for vitamin B did not produce this coloration, nor did other omega 3-6 oils, including fish, flax, borage, evening primrose, corn and sunflower.


Greg J. Harrison

Fig 6.57n | Normal lilac-crowned Amazon fed an organic formulated diet.


Greg J. Harrison

6.57o | Lilac-crowned Amazon fed a seed and table food diet. The overgrown beak and black pigmentation of the feathers has empirically been associated with advancing liver disorders (see Chapter 4, Nutritional Considerations and Chapter 15, Evaluating and Treating the Liver).


Greg J. Harrison

Fig 6.57p | Sulfur-crested cockatoo picks at its neck feathers and is able to pull its crest feathers with feet to chew them. Diet, behavior and integrative therapies (Chapter 10, Integrative Therapies) are often of benefit. A total cure is rare in such feather disorders unless caught at an early stage.


Greg J. Harrison

Fig 6.57q | This yellow-naped Amazon with a history of an all seed, nut, table food diet has keratin accumulating on the feet and beak. While fungus (usually *Aspergillus*) can be cultured from the black feathers, correcting the diet has treated hundreds of birds under the author's (GJH) care with no specific therapy instituted for the fungus. Supportive care is often used, such as milk thistle and lactulose, for regeneration of the liver.

Greg J. Harrison

Fig 6.57r | Budgerigar fed a vitamin-enriched seed only diet. While liver disorders in budgerigars are common, this blue budgerigar has black in the blue rump feathers, which is rare. Most budgerigars with liver disorders do not show any indications in their feather color.

Greg J. Harrison

Fig 6.57t | A lutino cockatiel with staining of the tail from being dipped in vitamin water, oiled seeds top dressed with vitamins and feces, as the perch was too low. A fungus is growing in the black feather coating but the problem cleared when the sources of contamination of the tail feathers were removed.

Greg J. Harrison

Fig 6.57v | A lutino cockatiel four months after therapy for liver disease showing a return to white feathers.

Greg J. Harrison

Fig 6.57s | A pied/lutino cockatiel in the later stages of disease. The dark yellow color is associated with a suspected hereditary liver disease. While too late for this bird, a formulated organic diet and liver support in the form of lactulose and milk thistle may be curative if diagnosed early.

Greg J. Harrison

Fig 6.57u| An 8-year-old lutino pied with advanced gold coloring of the feathers. This bird was fed a seed diet.

Greg J. Harrison

Fig 6.57w | Budgerigar with oiled ventral body feathers. Such birds become hypothermic and suffer digestive disorders from preening the oil. Bathing, drying and placing in an incubator at 86° F is minimum therapy.

Greg J. Harrison

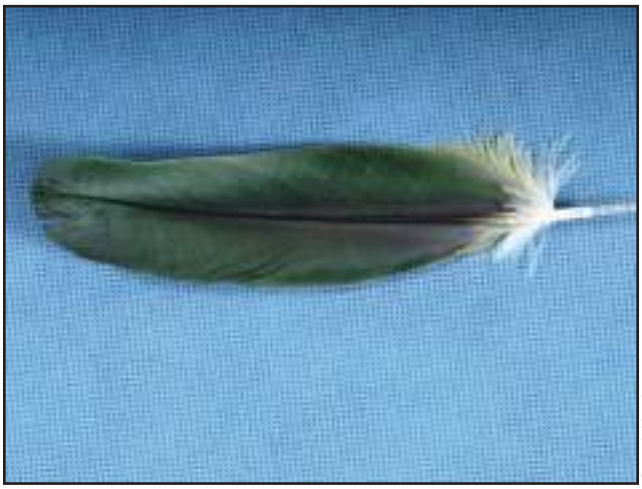
Fig 6.57x | An 27-year-old yellow-naped Amazon with abnormal yellow coverts developed while being fed a seed, table food and nut diet.

Greg J. Harrison

Fig 6.57y | Same bird as in Fig 6.57x with malcolored yellow primary remiges.

Phoebe Linden

Fig 6.57z | Same bird as in Fig 6.57x 6 months after correcting the diet. The abnormal yellow areas are gone.



Greg J. Harrison

Fig 6.58a | Healthy feathers are flexible with uniform color and structure.

Greg J. Harrison

Fig 6.58b | Holding the feather by the tip, the feather is slowly flexed tip to shaft. It should rebound to a normal position as in (a).

Greg J. Harrison

Fig 6.58c | A narrow veined feather that broke (fractured) at the mid-shaft on the flex test. Such easily damaged feathers indicate a nutritional disorder.

Peter Coutteel

Fig 6.59a | Feather lice eggs ("nits") in a canary's tail feathers.

Greg J. Harrison

Fig 6.59c | Louse under magnification (100x).

Greg J. Harrison

Fig 6.59b | A single louse on a finger. A few drops of alcohol are safe on a healthy bird to obtain lice to show the owners. Debilitated birds should not have alcohol used on them. (20x)

Table 6.1 | Uropygial Gland by Author Observations (GJH)

Uropygial Gland Absent	Uropygial Gland Present
• Argus pheasant	• African grey
• Bustards	• Blue and gold macaw
• Cassowary	• Budgerigar
• Cormorant	• Cockatiel
• Citron cockatoo	• Eclectus
• Double-headed Amazon	• Goffin cockatoo
• Yellow-fronted Amazon	• Gold-capped conure
• Green-winged macaw	• Indian ringneck
• Grey-cheeked parakeet	• Moluccan cockatoo
• Hyacinth macaw	• Most Psittacines
• Red-cheeked conure	• Red-masked conure
• Emu	• Rose-breasted cockatoo
• Ostrich	• Severe macaw
• Tailless domestic fowl	• Sun conure
• White carnean and rumples pigeon	• Umbrella cockatoo
• Woodpeckers	• Rock dove (wild pigeon)

Abnormalities of the feathers and skin should be recorded in a detailed manner. Veterinarians should familiarize themselves with the descriptive terminology used for the external anatomy of a bird. Such precise terminology is essential for later comparisons of progression or resolution of lesions and for describing a case to another veterinarian (see the physical exam form at end of chapter).

The uropygial (or preen) gland is located on the dorsal base of the tail. It is bilobed and is not present in all species (it is absent in many Columbiformes and psittacines, notably *Amazona* spp. and hyacinth macaws but prominent in budgerigars, cockatoos and waterfowl) (Table 6.1).

The uropygial gland should be assessed for evidence of enlargement or inflammation. Impaction, abscessation and neoplasia, all of which may be followed by self-trauma, are potential causes of uropygial gland abnormalities.

THE HEAD

The head should be first visualized in profile from a number of different angles, looking for asymmetry. Such asymmetry may arise from exophthalmus, enophthalmos, sinus swelling or depression of the skin over the sinuses. Pupillary size, iris color, lens clarity, feathers surrounding the external acoustic meatus (ear) and relative size of the ears, asymmetry of the cere, size of the nares, appearance of the nasal opercula, rhinothecal or gnathothecal deviations or overgrowth all need to be noted (Figs 6.60a,b) (see physical examination form). Loss of feathers on the head can be due to a variety of conditions. Some cockatiel mutations, especially lutinos, have a bald spot behind the crest. Feather loss in other species can be associated with fungal or bacterial dermatitis, infestation with ectoparasites, allergic dermatitis,

PBFD or excessive grooming by a cage mate. Feather loss around the eyes can indicate facial rubbing associated with conjunctivitis or sinusitis. Matting of the feathers over the crown and nape may indicate the bird has been regurgitating or vomiting.

The conformation of the beak should be assessed (Figs 6.60c-6.61e) for the presence of congenital or acquired abnormalities such as scissor (wry) beak, prognathism and bragnathism. Trauma to the beak or localized sinus infections can result in anatomical abnormalities (eg, longitudinal grooves in the keratin). Excessive keratin flaking of the beak can reflect poor nutrition or simply a lack of opportunity to rub the beak on a suitably abrasive surface (ie, a cement perch). Overgrowth of the beak can occur with PBFD, *Knemidocoptes* spp., congenital or acquired malalignment of the upper and lower beaks, chronic liver disease or malnutrition. It is rarely the result of a lack of objects to chew on. It is important to note that some species, such as the long-billed corella, *Cacatua tenuirostris*, naturally have elongated beaks. This should not be mistaken for an overgrown beak.

The cere (Figs 6.62a-c), the fleshy skin at the top of the beak, is not present in all species. In the normal green budgerigar (*Melopsittacus undulatus*) cere color can be used to sex the bird, with cocks having a blue cere and hens a brown cere. However, this will vary with the age of the bird, the color mutation, and the degree of health. Cere hypertrophy — a thickening of the brown cere in the budgerigar hen — may reflect a normal or pathologic hyperestrogenic state.

ORAL EXAM (Figs 6.63a-f)

Examination of the oropharynx can be accomplished by using roll gauze, plastic or metal speculums to open the mouth. In many birds equipment is not needed, as the approach of a light source toward the oral cavity will produce a wide open-mouth reaction and allow visualization. The choana (the slit in the roof of the oropharynx) should be free of excessive mucus or discharge and fringed with well-defined papillae. There should be no abscesses or diphtheritic membranes present. In larger birds, the infundibular cleft can be visualized in the hard palate of the choana. In some cases of severe sinusitis or otitis media, the infundibular cleft will be dilated and contain purulent debris (see Chapter 26, Diagnostic Value of Necropsy).

THE CROP

The crop can be palpated in most birds at the base of the neck, just cranial to the thoracic inlet. It should be carefully and gently palpated to assess if:

- Food is present (ie, Is the bird eating?)
- It feels doughy or fluid-filled, indicating that crop stasis may be present
- Ingluvioliths or other foreign objects are present
- The crop mucosa is thickened
- There is excessive water present
- The crop is overly distended

Care must be taken, especially in debilitated birds, that fluid or ingesta is not propelled retrograde from the crop into the oropharynx and aspirated by the bird.

THE BODY

Palpation of the skin over the trunk occasionally reveals the crackling or air-filled distention caused by subcutaneous emphysema. While this is normal in species such as pelicans, in most species it is the result of trauma or infection in the air sacs that allow the escape of air under the skin.

The abdomen in the normal bird is concave between the end of the sternum and the pubic bones. If this area is convex, then distention is present. The clinician needs to distinguish between internal and external distension of the abdomen. Internal distension of the abdomen can be due to fat, organ enlargement, ascites or the presence of an egg. External distension can be due to subcutaneous fat, neoplasia (especially lipomas), xanthomas or hernias. Radiology may be required to distinguish between internal and external abdominal distension and between different etiologies of both. The use of GI contrast material (barium) may help determine whether herniation is present and what structures may be incorporated into the hernia (Figs 6.64a,b). Abdominal pain or discomfort can occasionally be elicited by careful palpation. In passerines and juvenile psittacines, wetting the ventral abdomen with alcohol may allow visualization of internal organs. The liver should not extend pass the caudal border of the sternum in adult birds. If it does, liver disease should be suspected (eg, atoxoplasmosis in canaries).

If ascites is suspected, careful abdominocentesis may be indicated. After disinfecting the skin over the abdomen, a 23-27 g needle is gently introduced along the midline. If the needle is inserted lateral to midline, ascitic fluid may then communicate with the abdominal air sacs. In larger psittacines, a suitably sized intravenous catheter can be used. Negative pressure with a syringe is applied, and the fluid obtained is processed for cytology, culture and protein analysis. Care must be taken when abdominocentesis is performed that the loss of protein and/or the sudden change in abdominal pressure do not cause serious or fatal results. See Table 6.2 for a cursory list of fluid characteristics and causes. A more complete discussion is available in other texts.

Table 6.2 | Abdominocentesis Abbreviated Results

Nature of Fluid	Diagnostic Possibilities
Yellow-pink, turbid fluid. Cytology shows fat droplets, proteinaceous material, meso-epithelial cells, macrophages, occasional heterophils.	Yolk-related peritonitis
Light colored, clear fluid. Cytology shows few cells of any description.	Ovarian cyst Ascites Various neoplasias
Dark brown fluid. Cytology shows meso-epithelial cells, occasional erythrocytes, heterophils and macrophages.	Renal or hepatic cyst Degenerating ovarian follicles
Thick, gelatinous fluid.	Salpingitis
Fluid of variable color and consistency. Cytology shows macrophages, erythrocytes and heterophils, possibly bacteria.	Intestinal perforation Serositis

The back should be carefully palpated for evidence of scoliosis, lordosis or kyphosis. As the thoracic and lumbar vertebrae are predominantly fused, flexibility of the spine cannot be assessed as it is in dogs and cats.

The carina of the sternum should be palpated for evidence of distortion, trauma or congenital defects such as splitting. Distortion of the carina, often indicating a history of rickets or other metabolic bone disease, should lead the clinician to recommend radiographic evaluation of the rest of the patient's skeletal system.

The ventral area between the cloaca and the tail should be assessed for splitting of the skin (avulsed pygostyle). This condition may be mistaken for a cloacal prolapse on initial examination, until it is noted that the vent is present cranial to the red, protruding tissue that is actually muscle. This condition is commonly seen in pet psittacines and is associated with a poor diet, obesity and/or excessively clipped wings. Obesity and an excessively severe wing clip can cause the bird to land awkwardly, avulsing the tail from the pygostyle. Malnutrition causes the skin to lose its elasticity. The result is that the skin and underlying muscle in this area split. The initial injury may not be noticed by the owner, but the subsequent bleeding and picking at the affected tissue usually alert the owner to a problem.

THE WINGS (Figs 6.65a-e)

Each wing should be carefully extended and flexed to assess mobility and should be compared to the contralateral wing. The bones and joints should be palpated for swelling or crepitus. Recent trauma may be evident as greenish discoloration of the soft tissue. This is bruising and should not be mistaken for infection or tissue death. If the cause of a wing droop is not detectable after careful palpation, radiology is required to assess the pectoral girdle. The bones of this girdle are covered by strong muscles, and fractures are often not detectable by palpation alone. The patagium should be evaluated for loss of elasticity, trauma or scarring and for the presence of a tattoo (indicating the bird has been surgically sexed).

THE LEGS AND FEET

Each leg should be carefully palpated to detect abnormalities, such as fractures, healed bony calluses, or angular deformities of the long bones. Soft tissue swelling may be palpable or be suspected by the bird's reaction to palpation. Suspicious areas should be examined for bruising. Each joint should be extended and flexed to assess mobility and range of motion. Joints should also be examined for swelling or the presence of subcutaneous and intra-articular deposition of chalky white uric acid crystals (ie, articular gout). This condition is extremely painful and the bird will often be lame and react violently to digital pressure applied to the affected areas. All aspects of the legs should be compared with the contralateral side for symmetry, length, strength of grip and degree of muscling (Figs 6.66a-d).

The toes should be examined for abnormalities including:

- Missing digits or nails
- Annular constrictions
- Swelling of interphalangeal joints, occasionally with the deposition of uric acid crystals
- Avascular necrosis
- Excessive thinness, especially in neonates
- Abnormal position and conformation of the toes
- Excessively long or twisted nails

The skin of the foot is an ideal reflection of the rest of the dermis (Figs 6.67a-k). The plantar surface of each foot should be examined and the condition of the metatarsal pads and digital pads noted (Figs 6.68a-g). Abnormalities seen here include: loss of definition of the epidermis (seen as a shiny, reddened surface), swelling, erosions, ulcers and scabs. Pododermatitis (bumblefoot) is common in captive raptors (bumblefoot is never seen in wild, even one-legged birds - S. Hudelson, personal communication, 2004), but can be seen in any bird. A unilateral lameness causes increased weight-bearing on the unaffected leg. This in turn can lead to pressure necrosis, infection and subsequent pododermatitis. Consequently, in cases of a unilateral lameness, the opposite leg should always be closely examined. Bilateral pododermatitis is frequently encountered in older, obese psittacines with a history of poor diet, inadequate exercise and/or unsuitable perches. Note the discussion under Figs 6.67f-i for evaluating nails.

Occasionally, due to the nature of the injury or the disposition of the bird, a full examination may require general anesthesia. If this is the case, radiographs can be taken at the same time, minimizing handling of the conscious (and therefore stressed) patient.

Respiratory/Cardiovascular

AUSCULTATION

When to auscult is the prerogative of the clinician but may be better performed before the bird has been extensively restrained. The heart rate is usually rapid, although that of some pet birds can be surprisingly slow compared to wilder birds. Murmurs, arrhythmias, muffled heart sounds from pericardial effusion, severe tachycardia and bradycardia are occasionally detectable.

Lung and air sac noises can be auscultated, and occasionally friction rubs associated with air sacculitis can be detected. Since the avian lung is basically motionless, the typical mammalian auscultation parameters do not extrapolate well.

CLINICAL PRESENTATIONS

Open-mouthed breathing, abdominal movements and tail bobbing are due to respiratory distress but may also be due to the presence of space-occupying coelemic masses, ascites, anemia, cardiovascular disease, polycythemia, obesity, egg binding and other non-respiratory conditions.

Thyroid disorders in budgerigars can resemble either respiratory or crop/gastric disorders. Dyspnea is not uncommon.

The avian respiratory system is commonly affected by subclinical chronic disease. This is usually due to complications from stress disorders. Malnutrition is the most common cause (see Chapter 4, Nutritional Considerations, Section II Nutritional Disorders). A common presentation is mild nasal discharge that stains the feathers over the nares (Figs 6.69a,b). The discharge usually starts out serous in nature and may progress to mucoid in nature. This tendency seems to be species related. For example, budgerigars tend to be more serous, Amazons are more mucoid. African grey parrots and lovebirds seldom have nasal discharge but build up debris and form rhinoliths (Fig 6.69c). This can lead to atrophic rhinitis.

If the nasal condition is not treated, the lower respiratory tract may be affected (see Fig 6.69d). Sinusitis with resulting ocular discharge may occur (Figs 6.69e,f). If it is confined to sinuses of the head a sinus flush is beneficial for diagnosis. Sinus infections may present with swelling over the infraorbital sinus. There is no simple method to evaluate this diverse air sac system.

Generally, tracheal obstruction can be differentiated from lower or generalized respiratory disease by the sound of the respiration (tracheal noise) and the forward-leaning, neck-extended posture that is assumed by these birds. In cockatiels, tracheal obstruction may be due to seed or seed hull aspiration, especially when the illness is truly acute in nature (as judged by the presentation of a well-fleshed bird). In other cases, cockatiels may demonstrate a loss of voice and/or a squeaky sound produced for several days to weeks prior to the onset of more pronounced dyspnea. These cases are more likely to be due to a granuloma (eg, *Aspergillus* spp.) located at the syrinx, but endoscopy may be necessary to differentiate between these etiologies.

Older and larger birds (African greys, macaws and cockatoos) often suffer from chronic malnutrition with accompanying vitamin A deficiency. This leads to squamous metaplasia and a respiratory environment conducive to *Aspergillus* spp. propagation and granulomas of the trachea and/or syrinx. The eventual prognosis for these birds is guarded at best due to the long standing pathology and the potential for systemic disease.

Upper respiratory disorders tend to show inspiratory dyspnea. Lower respiratory tract disorders tend to show expiratory dyspnea that rarely have audible sound.

The nares, which are normally covered by feathers in many species, are the openings into the rhinal cavity located at the top of the beak. The nares should be symmetrical, open and dry. The normal opercula should not be mistaken for rhinoliths. Blockage of the nares or an area of communicating cul de sacs of the infraorbital sinus may result in a subtle inflation and deflation of the infraorbital sinus. This causes the up and down motion of the skin over the infraorbital sinuses with respiration (see Chapter 4, Nutritional Considerations, Section II Nutritional Disorders).

Neurologic Sensory Assessment

A cursory evaluation of the nervous system should be part of the physical examination. Birds presented for neurologic problems require a thorough neurologic assessment (see Chapter 17, Evaluating and Treating the Nervous System):

- Abnormal conformation or posture
- Paresis or paralysis of any or all limbs
- Fractures of limb bones
- Weakness or inability to grip with one or both feet
- Head tilt, opisthotonos, torticollis
- Altered mentation
- Decreased visual acuity


Greg J. Harrison

Fig 6.60a | Normal beak in a blue and gold macaw.


Greg J. Harrison

Fig 6.60b | Gray-cheeked conure with amputated maxilla. This will not likely regrow due to the proximal location of the amputation. The excessive pin feathers indicate that a diet or nutrient assimilation evaluation is needed.


Greg J. Harrison

Fig 6.60c | Close-up view of beak flaking. It may take a year after diet correction for this flaking to abate. This bird is deceased and is demonstrating post-mortem prognathism, a common occurrence, which has no clinical significance after death.


Greg J. Harrison

Fig 6.60d | Amazon four weeks post-diet change from seeds and table food to a formulated diet. New yellow head and green neck feathers and the flaking beak are positive signs that the bird is responding to the nutritional therapy. Sneezing and itching often accompany this period. A higher fat and protein diet speeds this recovery.

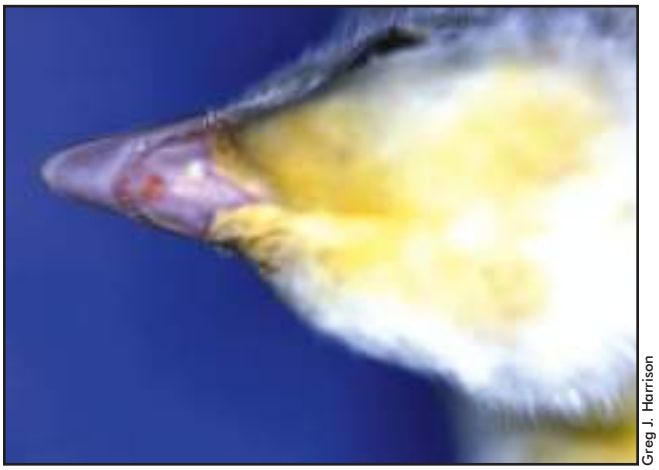

Greg J. Harrison

Fig 6.60e | Cockatiel with overgrown maxilla. Such gross overgrowths may be the result of symphyseal fractures of the mandible and the lack of normal wearing of the maxilla. Nutritional deficiencies and trauma may also result in severe maxillary beak overgrowth.


Greg J. Harrison

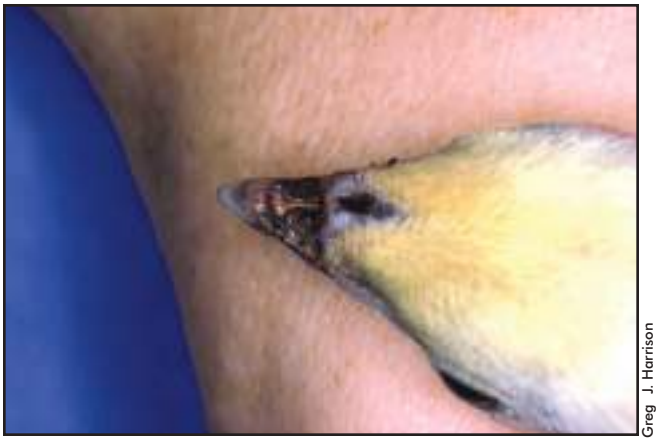
Fig 6.60f | Overgrowth of the rhamphotheca and interlaminal hemorrhage are common in budgerigars fed seed diets with liver disorders. Grinding or trimming is only a temporary solution. Diet change and liver therapy are needed.

Fig 6.60g | Cockatoo with PBFD, necrosis of the maxilla and exposure of the periosteum under the rhamphotheca. Euthanasia is strongly recommended for this presentation of circovirus.



Greg J. Harrison

Fig 6.60h | Peafowl chick traumatized by clutch mates.



Greg J. Harrison

Fig 6.60i | If not detected early, this traumatized bird will likely be further attacked and killed by its clutch mates.

Fig 6.60j | Scissor beak in a stunted Alexandrine parakeet. Harrison feels this is a form of rickets and myositis from an unhealthy oral epithelium and facial muscle infections, often emanating from nutritional disorders.

Greg J. Harrison

Fig 6.61a | A wild rehabilitating starling shows ideal head structures. This bird demonstrates a glass smooth rhamphotheca, impeccable nares and pristine facial plumage.

Greg J. Harrison

Fig 6.61b | Another passerine (warbler) shows the model beak and nares, but at this magnification demonstrates retained pin feathers over the crown. This presentation is common in wild birds with toxic substance-related debilitation.


Friedrich Janeczek

Fig 6.61c | Long beaks are normal for a species like the little (slender-billed) corrella.


Greg J. Harrison

Fig 6.61e | Delaminating beak in this Toco toucan is a reflection of a metabolic disorder as it is in other species.


Greg J. Harrison

Fig 6.62b | Cere of a yellow-fronted Amazon. These swellings in the cere tissue resemble sebaceous cysts of mammals. They may decrease in size with dietary improvement and weight loss if the bird is obese. They can be expressed but they tend to refill, or they can be surgically removed. Often no treatment is necessary since progression of the cysts is usually halted after a diet correction.


Greg J. Harrison

Fig 6.61d | This 20-year-old toucan was fed a toucan pellet for a decade and then an organic low iron nugget for the second half of his life. He had free flight and ate 1/4 of a raw organic papaya daily. Two years after this he died of a pancreatic carcinoma.

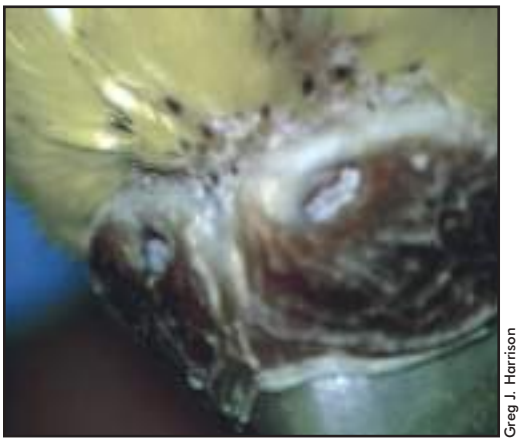
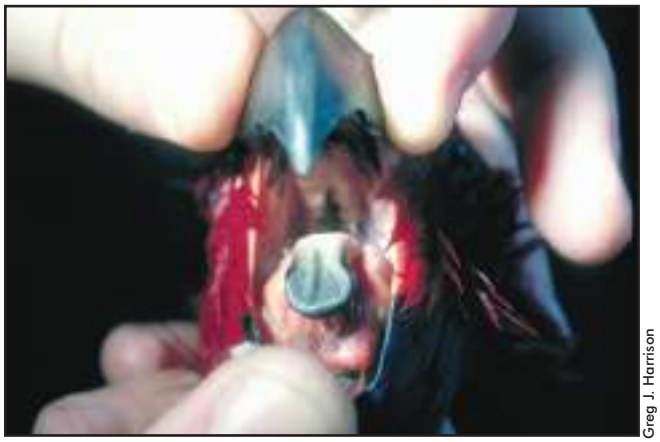

Greg J. Harrison

Fig 6.62a | The cere of a mature egg-laying budgerigar fed a seed diet. Compare this to the normal cere in Fig 6.4b. The cere is dry, flaky and lacks turgidity. The forehead feathers are predominately immature and have retained sheaths. Malnutrition and other systemic disorders should be on the differential diagnostic list (see Chapter 4, Nutritional Considerations, Section II Nutritional Disorders for the Improper Diet Cascade).

Fig 6.62c | Scaly face (*Knemidocoptes* spp.) mites cause a powdered look to the beak and a raised honeycomb mass, either on the cere, eyelids, beak, feet or other body locations. Magnification shows pinpoint tunnels in these powdery masses. This is where the mites live. The tunnels are pathogomonic and help differentiate this from other causes of similar lesions. Scrapings may be negative even when mites are present.

Fig 6.63a | The "gape" in a nighthawk. Note the lack of a choanal slit. There are leukoid plaques in the oropharnyx. Candidiasis or trichomoniasis is suspected.



Greg J. Harrison

Fig 6.63b | Female eclectus with palatine and sublingual hyperkeratotic salivary "abscess." While usually sterile, the condition is reported to be due to hypovitaminosis A. Therapy with vitamin A and dietary correction containing sufficient vitamin A precursors is appropriate treatment. (Some cases of "foot stomping" in eclectus parrots eating spirulina in the diet improve on dilution of the diet, while others find a correlation with PDD. If not PDD then the "stomping" may decrease over time with no therapy.)

Greg J. Harrison

Fig 6.63c | The intermandibular space found in all parrots.

Greg J. Harrison

Fig 6.63d | A swelling of the sublingual salivary gland on the left side of the intermandibular space. This lesion is usually a collection of amorphous cellular debris. Surgical resection may be necessary if these sterile abscesses interfere with tongue movement.

Susan Kelleher

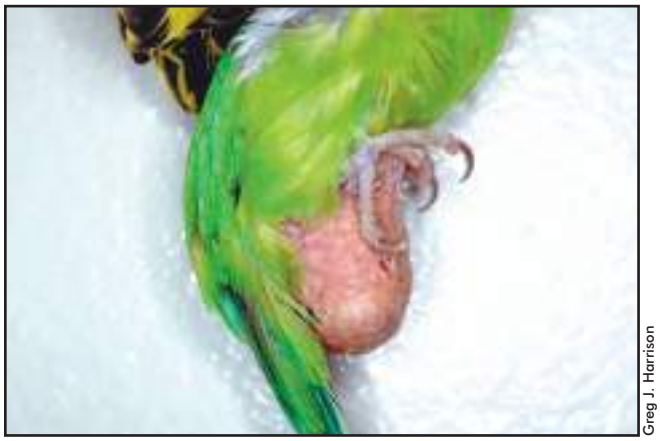
Fig 6.63e | An oral speculum in an Amazon with a fungal infection.

Susan Kelleher

Fig 6.63f | The same Amazon as in Fig 6.63e after therapy and dietary correction. While the choanal papillae are reduced in stature and depigmented, they have regrown their pointed characteristic tips.

Greg J. Harrison

Fig 6.64a | Anesthetized female budgerigar with an early abdominal hernia. Hernias are correlated with fat accumulating in the hernia area, xanthoma of the hernia sac's skin, ovarian cystic accumulation of estrogen laden fluids and nutritional disorders. Nutritional and hormone therapy should be instituted prior to considering surgery (see Chapter 18, Evaluating and Treating the Reproductive System).



Greg J. Harrison

Fig 6.64b | Budgerigar hernia — see Fig 6.64a. A simple skin removal several weeks post-diet change aids in correction. However, if the tissue is xanthomatous, it will not hold sutures well.

Peter Coutteel

Fig 6.65a | Dermatophytosis in a finch. Skin and feather fungal infections that cause lesions are very rare in birds. Most finches are fed a primarily seed diet. Combine that with crowding, poor air quality, lack of sun and inability to bathe, and the bird's defenses may succumb. Topical and systemic medications are needed, and the primary husbandry issues need to be addressed.

Greg J. Harrison

Fig 6.65b | Patagial dermatitis is not uncommon in birds. In birds with nutritional disorders, the skin (and all tissues) lose elasticity. As a result small tears can occur. While viruses have been suspected in lovebirds with similar lesions, none have been reported in most other parrots. Treating for fungal and bacterial infections, topical dressing and splinting to stop motion while changing the diet and husbandry may be curative.

Greg J. Harrison

Fig 6.65c | Sternal ulcers are seen in large bodied birds that have had their wing(s) clipped too short and are housed over hard surfaces. The resultant falls cause pressure necrosis (see Chapter 13, Integument and Figs 1.25a-e).


Greg J. Harrison

Fig 6.65d | Xanthomas may be seen in birds with nutritional disorders. If treated early, they may respond to dietary correction. Hormonal therapy may be of value in reproductively active hens with xanthoma. In advanced cases, surgical resection is often required.

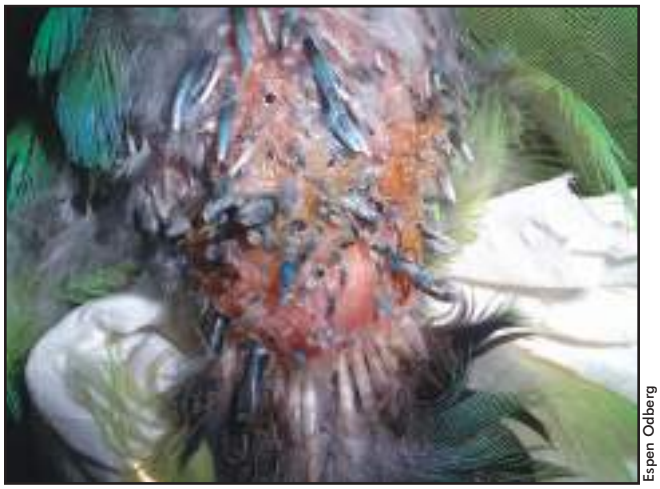

Espen Odberg

Fig 6.65e | Lovebird with polyfolliculitis. Multiple feathers form in a single follicle. A necrotizing dermatitis is also present (see Chapter 13, Integument).


Greg J. Harrison

Fig 6.66a | Bruising from vascular injury associated with a tibial fracture can be accessed if the feathers are wet or removed.


Greg J. Harrison

Fig 6.66b | Peafowl chick hock shows bruising from post-traumatic repair. Subcutaneous blood in birds is often green (biliverdin).


Greg J. Harrison

Fig 6.66c | Peafowl chick with a dislocated Achilles tendon. Providing an improved diet, surgical repair and providing the enclosure with a substrate that is not slick may be curative. However, the prognosis is progressively guarded as the species size increases.


Greg J. Harrison

Fig 6.66d | A wild passerine in rehabilitation shows the normal nail length, with the nail tapered and needle sharp. This bird has a toxic neuropathy.


Greg J. Harrison

Fig 6.67a | Normal Amazon dorsal foot skin patterns.


Greg J. Harrison

Fig 6.67b | Dorsal surface of the foot of a free-ranging slender-billed corrella. A severe drought led to water holes drying up and very low natural food sources. The feet are dry but the patterns remain bold. This is believed to be a temporary phenomenon as a result of environmental stress.


Greg J. Harrison

Fig 6.67c | Acute phase of Amazon foot necrosis can occur in a matter of minutes. The condition usually affects just Amazons (see Chapter 13, Integument).


Greg J. Harrison

Fig 6.67d | Peracute phase of Amazon foot necrosis. The skin will slough over several weeks. No therapy is effective at this stage, but prevention of self trauma and secondary infection may be necessary (see Chapter 13, Integument).


Greg J. Harrison

Fig 6.67e | Permanent pigmental scars 3 months after the initial lesions of Amazon foot necrosis.


Greg J. Harrison

Fig 6.67f | A free-ranging slender-billed corrella shows the natural nail length and tapering needle points of the nail. Note that while the foot has dry skin, the plantar patterns are still bold. One would expect the dry skin to be replaced in a few weeks if the drought is broken.


Greg J. Harrison

Fig 6.67g | This Amazon's nails are thickened and overgrown (see Chapter 4, Nutritional Considerations, Section II Nutritional Disorders).


Greg J. Harrison

Fig 6.67h | Nails of a captive canary on a seed-based diet. In addition to being long and twisted with no tapering, or sharp point, the feet scales are hypertrophic and dry. This could indicate metabolic, parasitic or bacterial disease and/or malnutrition (eg, *Knemidocoptes* spp., staphylococcal or fungal infection).


Greg J. Harrison

Fig 6.67i | Finch supplied with synthetic nest material (nylon) has a strangulated digit that needs to be amputated. Natural fibers or nest pads avoid this unnecessary calamity.


Peter Coutteel

Fig 6.67j | Papillomatosis of both feet of a finch.

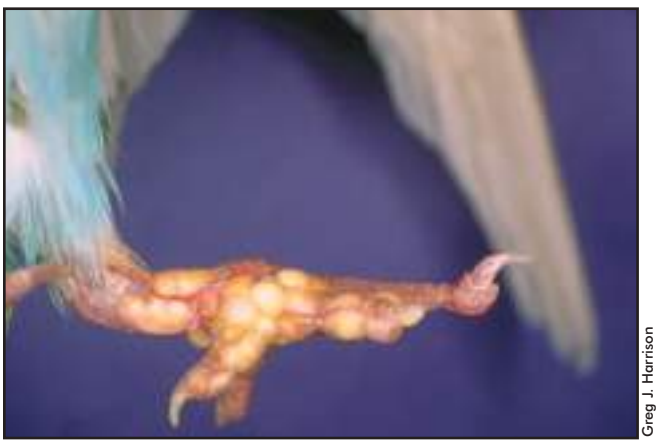

Greg J. Harrison

Fig 6.67k | Gout tophi is a painful collection of uric acid crystals in the joints and subcutaneous areas of the feet.


Greg J. Harrison

Fig 6.68a | Normal Amazon plantar foot skin patterns.


Greg J. Harrison

Fig 6.68b | This warbler shows the normal bold but delicate pattern of a small passerine's plantar foot surface.


Greg J. Harrison

Fig 6.68c | Plantar surface of a slender billed corrella. As previously mentioned, the skin is dry but the bold pattern is undisturbed.


Greg J. Harrison

Fig 6.68d | Tarsal pad loss of epithelial pattern is common in parrots with nutritional disorders. As they become physically inactive, their tendons seem to weaken and they perch on their hocks. Dietary correction and supportive care, correction of husbandry issues, and alteration of perches may improve this condition.


Greg J. Harrison

Fig 6.68e | The degree of development of bald patterns on the plantar surface can be an indication of nutritional disorders. Abrasive perches can exacerbate the problem, but these surfaces do not generally cause plantar excoriation, ulceration or pododermatitis in healthy birds. Large bodied birds may be an exception.


Greg J. Harrison

Fig 6.68f | Advanced bumblefoot in a female lutino cockatiel. Anecdotally females develop bumblefoot more often than males in several species (cockatiels, swans, flamingoes and chickens). Nutritional and hormonal disorders need to be addressed.

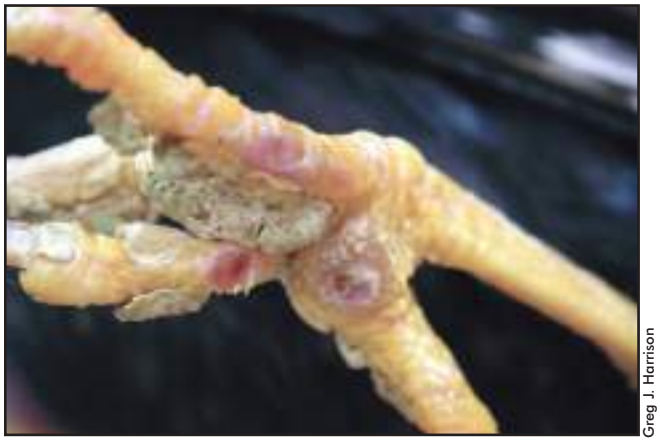

Greg J. Harrison

Fig 6.68g | Mynah bird with bumblefoot and massive tarsal scale proliferation indicative of a metabolic disorder. Nutritional and husbandry issues must be addressed and any secondary infection treated.


Greg J. Harrison

Fig 6.69a | A 20-year-old male cockatiel fed a seed based diet shows accumulation of discharge over the left naris and around the eyes, which is typical of sinusitis.


Greg J. Harrison

Fig 6.69b | Budgerigar female with discharge from nares accumulated in frontal feathers. The cere is dry and appears to have fungal growth around the right naris. The bird was old and had obstructive bowel problems that lead to its demise. It was presented for panting.


Greg J. Harrison

Fig 6.69c | Lovebird rhinolith. Nutritional correction and nasal flushes, systemic antibiotics and antifungals are often required. The addition of hyaluronidase to the flush can expedite the breakdown of caseated debris (see Chapter 9, Therapeutic Agents).

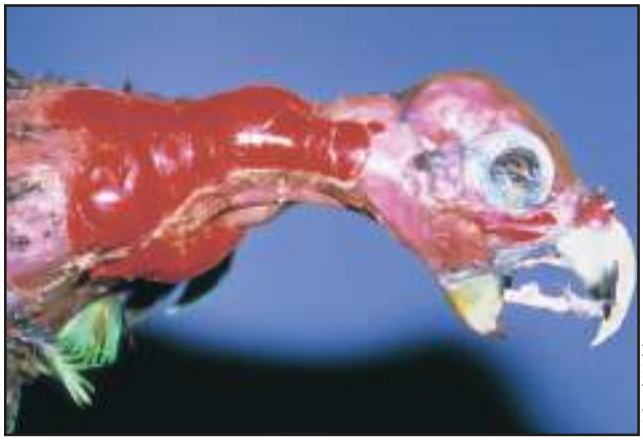

Michael Walsh

Fig 6.69d | Latex injection mold of the cervicocephalic air sac and the infraorbital sinus (red latex under eye, around naris and the cranial aspect of the beak). A section of a wooden applicator is being used to prop open the oral cavity. (Some latex has run down the rhamphotheca from the nares and more has run into the oral cavity at the commissure of the mouth and on the leading edge of the beak).


Gwen Flinchum

Fig 6.69e | Budgerigar with an extensive accumulation of infraorbital sinus serous fluid. This is a very difficult problem to correct. Mycoplasma has been incriminated in some cases.


Peter Coutteel

Fig 6.69f | Canary with infraorbital sinusitis. Mycoplasma was isolated.


Greg J. Harrison

Fig 6.70a | The iris of an immature blue and gold macaw.


Greg J. Harrison

Fig 6.70b | The iris of a mature blue and gold macaw.


Greg J. Harrison

Fig 6.70c | The iris of an immature African grey.


Greg J. Harrison

Fig 6.70d | The iris of a mature African grey.

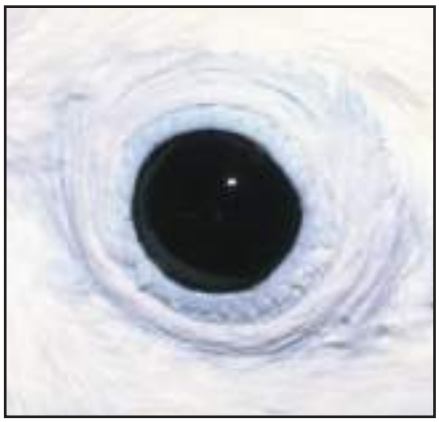

Greg J. Harrison

Fig 6.70e | The iris of an immature umbrella cockatoo. This appears the same as one would see in a mature male umbrella cockatoo.

EYES

The eyes should be bright and clear. Ocular discharge and loss or matting of the feathers around the eye indicates either conjunctivitis or sinusitis. Conjunctival hypertrophy is common in chronic conjunctivitis, especially in cockatiels. Mycoplasma has been implicated in this syndrome in cockatiels. Focal light, magnification and fluorescein stain is needed for a detailed ocular examination. Severe or non-responsive ocular disease should be referred to an ophthalmologist familiar with the avian eye when possible.

Determination of the integrity of the globe and neurologic pathways necessary for vision can be difficult. Consultation with or referral to an ophthalmologist familiar with the avian eye may be necessary.

Iris color can indicate sex and/or age. Young birds tend to have a dark iris that lightens as the bird matures. This is true for blue and gold macaws and African grey parrots among others. In many white cockatoos the dark iris lightens to brownish red or even reddish orange in the mature female, while in the male it remains dark. This is not as prevalent in the umbrella and Moluccan cockatoos as it tends to be in the yellow or citron crested members of this genus. Additionally, it is not a guarantee of gender, and a DNA determination of sex should be made if the gender is questionable (Figs 6.70a-e).

Both nuclear sclerosis and true cataracts occur in birds. In larger psittacines, cataract removal can be accomplished by selected veterinary ophthalmologists. As in dogs and cats, the degree to which the bird is affected by the decreased or lost vision will often dictate whether attempting surgery for cataract removal is warranted.

Various congenital and acquired diseases of the eyelids, cornea, iris, and fundus exist in birds as in other species. Again, there are increasing numbers of veterinary ophthalmologists who are knowledgeable regarding avian ocular anatomy and disease, and a referral may be indicated.

EARS

The ears can be examined by parting the ear coverts with the wooden end of a cotton-tipped applicator or similar

Greg J. Harrison

Fig 6.71a | Cloaca everted in a green-wing macaw in order to check for papillomatosis. The small raised area at 9:00 needs to be tested with acetic acid and/or biopsy. Blanching, in response to the application of vinegar, is reportedly diagnostic.

Greg J. Harrison

Fig 6.71b | Speculum in the cloaca of an anesthetized umbrella cockatoo. The hypertrophied opening of the ductus deferens onto a prominent papilla is indicative of a male.

appliance. The ears should be open and free of discharge or erythema. Visualization of the tympanic membrane is difficult in most species without the use of an endoscope (see Chapter 24, Diagnostic Value of Endoscopy and Biopsy). Note that the tympanum of birds is normally convex, as opposed to the normal concavity that is found in mammals.

DIGESTIVE AND URINARY SYSTEM

The cloaca can be assessed externally for enlargement and dilation (often indicative of reproductive behavior in a hen), prolapse, ulceration or inflammation around the mucocutaneous junction, and the presence or loss of sphincter tone. Moistened cotton-tipped applicators can be introduced into the cloaca and used to isolate and evert the cloacal mucosa. The mucosa is normally thin, pink and smooth (Figs 6.71a,b). Gently everting the cloaca allows a cursory examination of the mucosa, possibly revealing papillomas in susceptible species. These may be obvious pedunculated protrusions or more subtle thickenings with a cobblestone appearance to the tissues. Suspicious areas can be painted with dilute acetic acid; blanching indicates the presence of a papilloma. A more thorough evaluation of the cloaca requires endoscopy.

REPRODUCTIVE SYSTEM

Refer to the physical examination form, Chapter 4, Nutritional Considerations, Section II Nutritional Disorders and Chapter 18, Evaluating and Treating the Reproductive System for in-depth discussions of reproductive anatomy, physiology and disease (Figs 6.72a,b,g).

Fecal Examination

Birds' droppings are made up of three components: feces, urates and urine (Figs 6.72a2,b2). In a healthy bird, the fecal portion should be formed and homogenous, with little odor (except for poultry, waterfowl and carnivorous birds). The color should be various shades of brown when the bird is fed a pelleted diet. Seed diets will cause the stool to be a more greenish color. Various fruits, especially those with strong pigments such as cranberries and blueberries, and artificially colored foods including colored pellets, may affect the stool color (Fig 6.72c).

The urates should be a crisp white and slightly moist. If the bile pigments are not adequately resorbed from the GI tract and reused by the liver, the excess of bile pigments, mainly biliverdin, will leach out of the feces and into the urates, causing the urates to develop a greenish tinge. Diet, species, and state of excitation may alter the ratio of urine to feces present in a dropping. Do not confuse true polyuria with "excitement polyuria," the excess urine produced by an excited or nervous bird. See Chapter 16, Evaluating and Treating the Kidneys, for information on polyuria and further diagnostics. Lorikeets, due to their liquid diet, will normally produce large amounts of urine. A close examination of the droppings is a valuable and non-stressful starting point for the clinical examination. Examination of the droppings in the bird's cage that have been collected from the past 24 hours will yield more information than limiting the examination to stress droppings produced enroute to or in the hospital.

Some abnormalities commonly encountered in the avian dropping include:

- Diarrhea — unformed fecal portion (Figs 6.72c,f2)
- Undigested food in feces (Fig 6.72f)
- Very bulky droppings — maldigestion; malabsorption; reproductively active hens; abdominal growth; pelleted diet (Figs 6.72a2,d,e)
- Melena

Fig 6.72a | Prolapsed uterus in a finch.

Greg J. Harrison

Fig 6.72a2 | Normal Amazon dropping seen when the bird consumes an organic formulated diet.

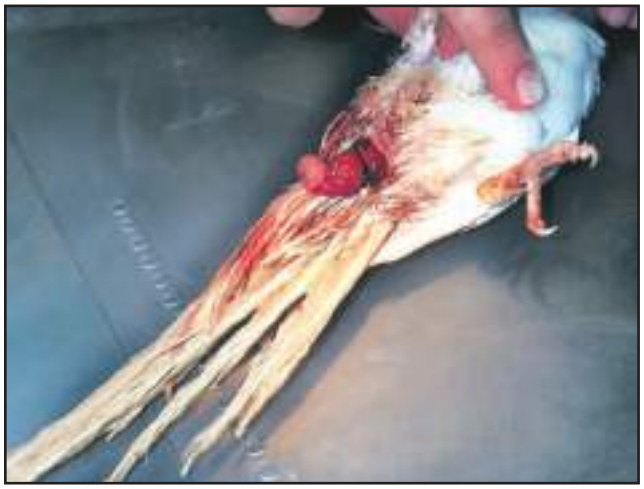


Fig 6.72b | Prolapsed uterus in a budgerigar.

Greg J. Harrison

Fig 6.72b2 | Normal passerine dropping.

Greg J. Harrison

Fig 6.72c | Passerine droppings with artificially colored food items and mild enteritis.

Greg J. Harrison

Fig 6.72d| An elderly malnourished female budgie with an obstructive cloacal condition from a uterine tumor. When the obstruction was manipulated, 8 cc of feces were expressed. The owner elected euthanasia.

Greg J. Harrison

Fig 6.72e | The normal stress changes observed in the droppings of a parrot that traveled a long distance prior to examination. Droppings at home (bottom); droppings at the clinic (top). The dark color at home (a sign of melena) is not normal.

Greg J. Harrison

Fig 6.72f | Passing whole seeds in feces. Various causes of proventriculitis, ventriculitis, and pancreatitis must be considered.

Greg J. Harrison

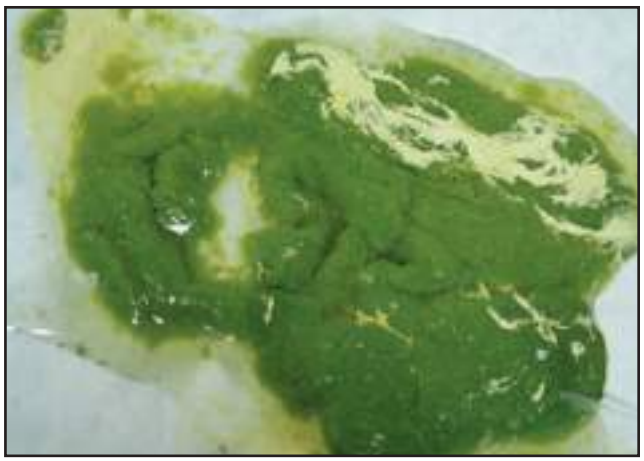
Fig 6.72f2 | A dropping from a parrot with diarrhea is a rare observation.

Greg J. Harrison

Fig 6.72g | Egg-bound pearly cockatiel passes a loose liquid dropping after having the egg removed. Biliverdinuria reflects the liver stress of the situation.

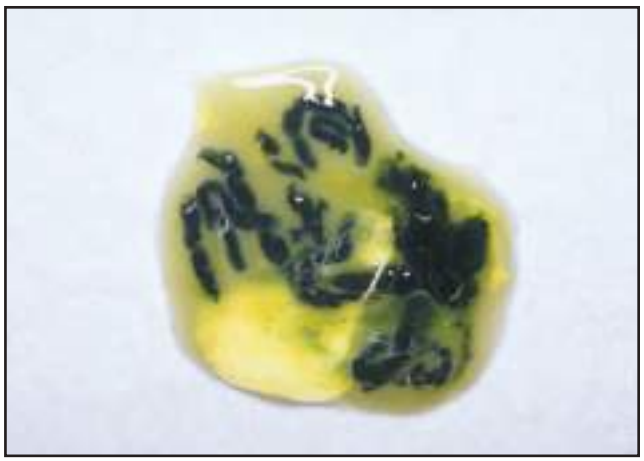
Greg J. Harrison

Fig 6.72h | A parrot's droppings show excess urates with the beginning of biliverdinuria.



Greg J. Harrison

Fig 6.72i | Loose feces from polydipsia in a large parrot.



Greg J. Harrison

Fig 6.72j | Polyurates with biliverdinuria and feces typical of enteritis.


Greg J. Harrison

Fig 6.72k | Parrot dropping with PU/PD and biliverdinuria.


Greg J. Harrison

Fig 6.72l | The droppings from a cockatiel with PU/PD. Hyperglycemia is a common cause of PU/PD, especially in obese adult cockatiels on poor diets.


Greg J. Harrison

Fig 6.72m | A car in Florida (USA), parked under a tree where roosting passerine birds have been eating a local tree's seasonal fruit, demonstrates that they pass large amounts of urates on such a diet.


Greg J. Harrison

Fig 6.72n | Polyurates in a bird recently switched to an organic high fat and high protein diet. The pansystemic repair occurring requires massive tissue replacement leading to an increased nitrogen load.

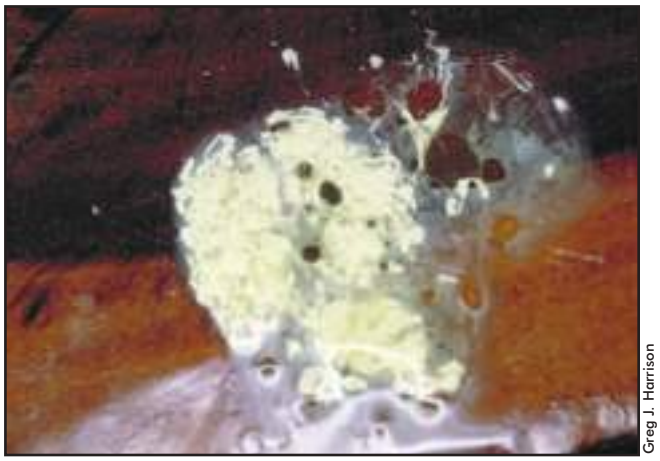

Greg J. Harrison

Fig 6.72o | Polyurates with fat. Fat in the urine is rare. Severe kidney damage has occurred, and this bird did not survive.

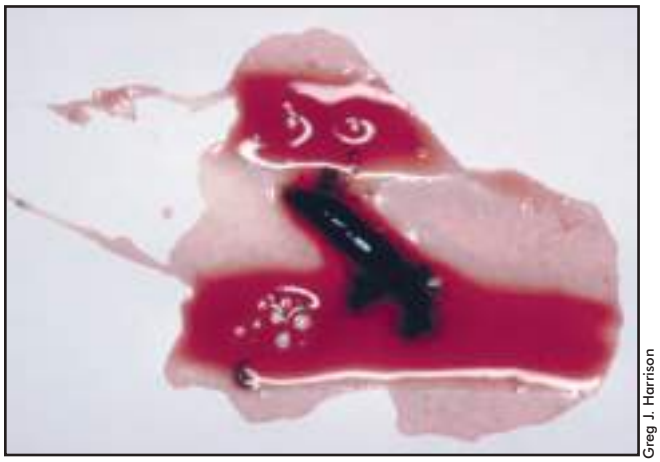

Greg J. Harrison

Fig 6.72p | Passing whole blood in the urine after the bird ate the back of an old mirror (mercury). Similar presentations have been seen in lead toxicosis.

- Malodorous droppings — bacterial (clostridial or other) or fungal overgrowth
- Aerated droppings — also called "popcorn stool" seen most commonly in cockatiels with giardiasis (see Fig 6.16)
- Green urates — often indicative of liver disease and biliverdinuria (Figs 6.72e,f,j,k)
- Yellow urates — associated with anorexia and liver bilirubin excess (Fig 6.72h)
- Pink/red urates — blood, hemoglobin or denatured hemoglobin that may be associated with renal disease. Some species, such as Amazons, eclectus and galahs will demonstrate pink to brown urates with lead poisoning
- Orange urates — may be due to vitamin B injection in the last few hours or artificial colors in the diet
- Thick, pasty urates — dehydration
- Polyuria — multiple etiologies, including: heavy metal toxicity, renal disease, sarcocystosis, diabetes mellitus, and pituitary adenoma (Figs 6.72i-l)
- Polyurates (Figs 6.72m-o)
- Anuria — etiologies include:
  Obstruction: Fecoliths or uroliths, egg-binding, cloacal prolapse, papillomatosis
  Functional: Renal disease, severe dehydration
- Fresh blood — cloacal pathology (Fig 6.72p)

A fresh fecal sample should be collected for a fecal Gram's stain (see Chapter 4, Nutritional Considerations: Section II, Nutritional Disorders), flotation and wet smear evaluation. If there is polyuria, a urine sample can be collected for urinalysis.

Urine evaluation and the urinary system in general is covered in depth in Chapter 16, Evaluating and Treating the Kidneys.

Diagnostic Testing

Veterinarians treating birds (and other exotic species) are faced with challenges often not encountered by their colleagues treating dogs and cats. Many birds are presented to veterinarians only when near-terminally ill, and a rapid tentative diagnosis is often the difference between life and death. Birds are often limited in their range of expression of clinical signs and many clinicians, through no fault of their own, lack the experience to conduct a thorough physical examination. The combination of these factors has led to an increasing tendency in avian medicine to conduct exhaustive diagnostic tests on patients, often with scant attention paid to a complete history and a careful physical examination and with little attempt to refine or focus the diagnostic efforts. The selection of diagnostic tests should be based on a solid understanding of the species in question and the results of a thorough history taking and physical examination that enable the practitioner to develop an abbreviated list of differential diagnoses.

Before proceeding with diagnostic tests, the clinician should first ask:

- Is the patient sufficiently stable to undergo diagnostic testing, or does it require supportive care prior to sampling?
- Are the physical risks to the patient justified by the likely clinical value of the results?
- Are the test(s) appropriate to the patient (ie, species, age, sex) and its clinical signs?
- Has the test been validated to ensure that the result obtained is likely to be both accurate and meaningful?

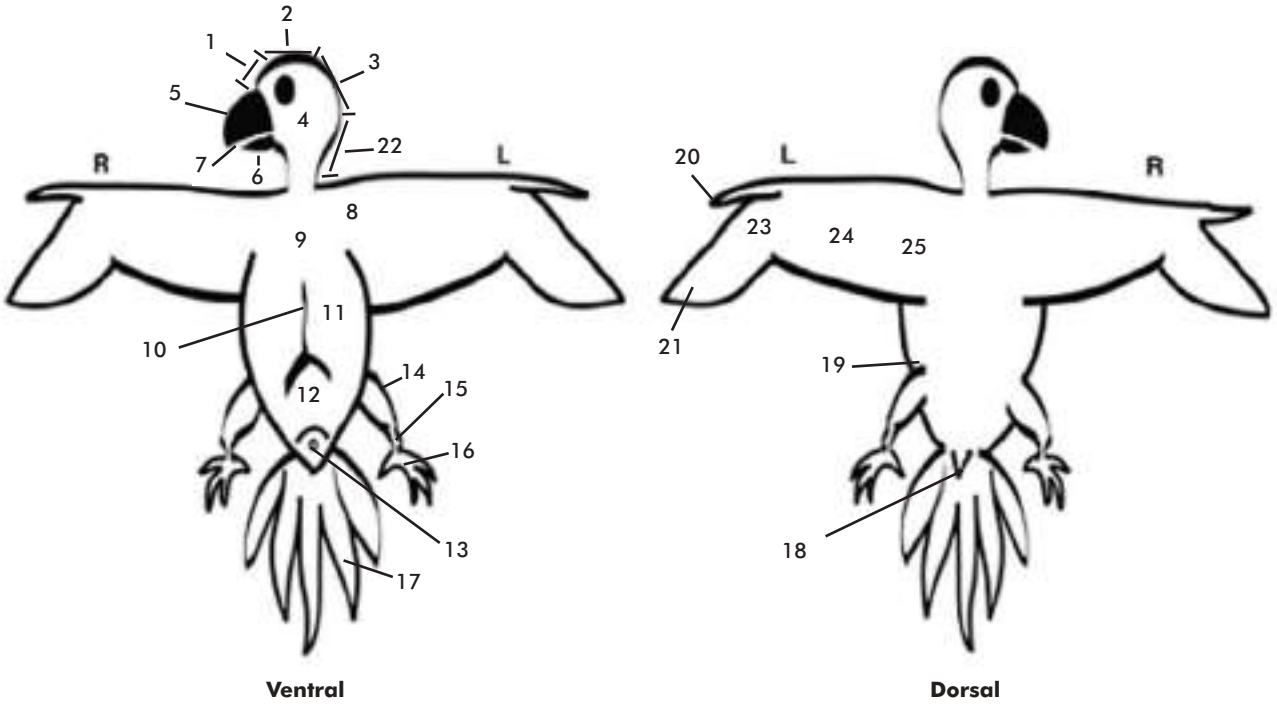
If the answer to these questions is 'yes,' then diagnostic testing should proceed.

Diagnostic testing should be done in steps, with the results of each test allowing interpretation and reevaluation of the subsequent diagnostic procedure. Where appropriate, the clinician should endeavor to start with minimally invasive tests (ie, fecal wet smears, fecal flotations and Gram's staining) before moving on to more invasive tests. As each tier is passed, the information gained should allow the clinician to narrow the differentials and perform tests leading to a definitive diagnosis.

The practitioner should be aware that the "normal" values provided by laboratories for serum chemistries, hematology and other parameters are generalizations and are not species-specific. Some of these values are inaccurate and are extrapolated from canine or feline values. The practitioner should be familiar with normals for the species in question, or have a reputable reference text available (see also Appendix).

Clinicians also need to be familiar with the advantages, disadvantages and accuracy of the diagnostic test(s) they employ. There is controversy and healthy debate within avian medicine concerning many of the tests that are currently in use. Examples include:

- Are fecal Gram stains an appropriate diagnostic test to use on healthy patients (see Chapter 4, Nutritional Considerations, Section II Nutritional Disorders)?
- What is the significance of yeast in a Gram's stain?
- How should one interpret cloacal and choanal cultures? Should these cultures be routinely performed?
- Should zinc levels be assessed in patients not showing clinical signs consistent with zinc toxicosis?
- Can a diagnosis of zinc toxicosis be made from a single blood level evaluation?
- Can aspergillosis be diagnosed or ruled out in a patient based solely on serologic tests or culture?



1. Front	6. Mandible	11. Breast (pectoral)	16. Claw (foot)	21. Hand
2. Crown	7. Gnathothea	12 Abdomen	17. Tail	22. Neck
3. Nape	8. Axilla	13. Vent	18. Uropygial gland	23. Wrist
4. Ear	9. Area of crop	14. Thigh (shank)	19. Flank	24. Forearm
5. Rhamphothea	10. Sternal carina (keel)	15. Shin (metatarus)	20. Alular digit	25. Upper arm

Harcourt-Brown

Fig 6.73 | Physical Exam

- How accurate is the differentiation between species of *Mycobacterium* via serology? What is the true zoonotic potential of *Mycobacterium avium*? Should birds diagnosed with this disease be treated?
- Can disease be diagnosed from a positive DNA or PCR test on an asymptomatic patient?
- What is the appropriate interpretation of plasma electrophoresis?
- In what cases are biopsies (eg, renal, hepatic, and pancreatic) warranted? What is the risk/benefit ratio?
- Should crop biopsies be obtained for potential diagnosis of PDD?
- Is skin testing for birds sufficiently advanced to yield clinically useful information?

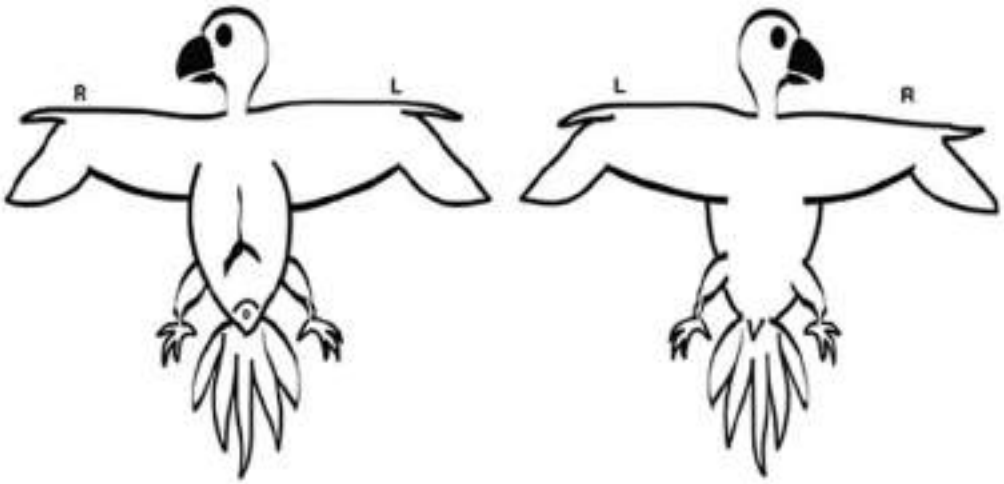
As we move into the 21st century, it may no longer be sufficient to just have a textbook as a reference on these issues and others. Access to discussion lists on the Internet is an invaluable tool for keeping abreast of current issues. Topics are often discussed on the Internet several years before they appear in even the most recent textbook. As with all non-peer reviewed information, accuracy may be in question. It is important to trace information to the original source to adequately evaluate the material. Journals, educational CDs, wet labs, annual conferences and regular discussions with colleagues also contribute to the maintenance of current knowledge. Conversely, a solid knowledge of the basics of avian anatomy, physiology and disease generally requires more in-depth study than can be obtained through the above sources. A combination of core textbook study, experience, and Internet and journal-based current information will yield the optimal breadth and scope of knowledge necessary for avian practice.

Once again, "you will miss more by not looking than you ever will by not knowing." It is only by a careful evaluation of the patient's history, a thorough physical examination, and the judicious use of appropriate diagnostic tests that the clinician can arrive at the correct diagnosis and implement successful treatment.

Physical Examination Form

©Spix Publishing Inc, Adapted from Harrison GJ, Lightfoot TL: Clinical Avian Medicine, 2005.

MAP OF FINDINGS



Harcourt-Brown

BODY CONDITION

Body weight _____ g

Hydration: ☐ Normal
Dehydration: ☐ <5% ☐ >5-10% ☐ >10%

Emaciation . . . . . ☐ yes ☐ no

Underweight. . . . . ☐ yes ☐ no
(percent or by how many grams? _____% _____ g

Amount of body fat: ☐ None ☐ Trace ☐ Light ☐ Obese

Lipoma(s). . . . . ☐ yes ☐ no
Where located? _____ (see diagram)

BLEEDING

IF BLEEDING IS OR HAS BEEN PRESENT

Bleeding/bruising of

Sternum. . . . . ☐ yes ☐ no

Distal wing . . . . . ☐ yes ☐ no
(note: bleeding from wing tips may be from skin tears, bruising or damaged blood feathers and these must be differentiated)

Skin . . . . . ☐ yes ☐ no
Location: _____

Beak . . . . . ☐ yes ☐ no
If yes from beak tip, trauma? . . . . . ☐ yes ☐ no
Bite wound . . . . . ☐ yes ☐ no
Skin at commissure . . . . . ☐ yes ☐ no

Blood feathers . . . . . ☐ yes ☐ no

Cloacal blood

Frank red blood in feces . . . . . ☐ yes ☐ no

Occult blood in feces. . . . . ☐ yes ☐ no

Black feces . . . . . ☐ yes ☐ no

Frank blood from cloaca independent of droppings . . . . . ☐ yes ☐ no

Hemolyzed blood in urine . . . . . ☐ yes ☐ no

Occult blood in urine . . . . . ☐ yes ☐ no

"Chocolate milk" methemoglobin in urine . . . . . ☐ yes ☐ no

FEATHERS

Clipping of Wings

IS BIRD CURRENTLY FULL-FLIGHTED? . . . . . ☐ yes ☐ no

Owner declines clipping . . . . . ☐ yes ☐ no

Wing clipped: Now. . . . . ☐ yes ☐ no
Previously . . . . . ☐ yes ☐ no

Wing clipped: Right . . . . . ☐ yes ☐ no
Left . . . . . ☐ yes ☐ no
Both. . . . . ☐ yes ☐ no

Feather Structure/Color

Abnormal molt . . . . . ☐ yes ☐ no
Describe _____

Chronic pinfeathers that fail to open . . . . . ☐ yes ☐ no

Retained keratin in the feathers of head . . . . . ☐ yes ☐ no
Feathers of body . . . . . ☐ yes ☐ no

Saw-toothed edges to feathers (failure to zip) . . . . . ☐ yes ☐ no

Broken, malformed or bent feathers . . . . . ☐ yes ☐ no

Lack of powder down when applicable . . . . . ☐ yes ☐ no

Dull appearance to feathers . . . . . ☐ yes ☐ no

Stained or dirty . . . . . ☐ yes ☐ no
☐ Generalized ☐ Localized

Stress lines/bars . . . . . ☐ yes ☐ no

Flexibility of feather at 180° tip to base: (test of feather integrity)
- Breaks when bent . . . . . ☐ yes ☐ no
- Bends and remains bent . . . . . ☐ yes ☐ no
- Indents when flexed. . . . . ☐ yes ☐ no
- Straightens back to normal when released . . . . . ☐ yes ☐ no

Are there malcolored feathers (abnormal for species, i.e., black on normally green or blue feathers, pink or red feathers; yellow coloration to normally blue, green or white feathers; white discoloration of hyacinth feathers; red pigment in grey feathers) ☐ yes ☐ no
If yes, describe (color, location, onset): _____

Over-preening, picking, or other feather destructive behavior ☐ yes ☐ no

Feather dystrophy ☐ yes ☐ no

Multiple feathers in follicles ☐ yes ☐ no

BEAK

Is beak symmetrical ☐ yes ☐ no

If no, describe abnormality (scissors beak, prognathism, beak trauma, groove in beak from naris, previous rhinitis, other) _____

_____

Overgrown ☐ yes ☐ no

Friable ☐ yes ☐ no

Hyperkeratinization ☐ yes ☐ no

Small scratch abrasions from concrete perch evident on beak ☐ yes ☐ no

NAILS

Missing nails ☐ yes ☐ no
List: _____

Abnormally curled ☐ yes ☐ no

Otherwise deformed ☐ yes ☐ no
If so, describe: _____

SKIN

Flaking ☐ yes ☐ no

Pruritic ☐ yes ☐ no

Other lesions (erythema, excoriations, scabs, lacerations, necrotic areas)
List and see diagram: _____

_____

Cutaneous or subcutaneous masses ☐ yes ☐ no
Describe: _____

Loss of normal foot patterns (thin shiny skin) ☐ yes ☐ no
Where located: _____

Pododermatitis ☐ yes ☐ no
Where located and degree _____

Self-cannibalized (mutilation) ☐ yes ☐ no
Where located: _____

Burn ☐ yes ☐ no
Where located: _____

Bite wounds ☐ yes ☐ no
Where located: _____
(Note: with a history of an encounter with a dog or cat, one should assume that a bite wound has occurred whether or not a wound is detected)

UROPYGIAL GLAND

Is a uropygial gland normally present or absent in this species? ☐ yes ☐ no
If present, is the uropygial gland normal in size and symmetry ☐ yes ☐ no

Able to express small amount of sebum from papilla ☐ yes ☐ no

AXIAL SKELETON

Is the spine completely immobile ☐ yes ☐ no

If mobile, identify areas of bruising: _____

_____

_____

ABAXIAL SKELETON

Wings

Symmetrical at rest (i.e., no wing droop) ☐ yes ☐ no

Bilaterally symmetrical on extension ☐ yes ☐ no

Symmetrical range of motion ☐ yes ☐ no

Pain on palpation, extension or flexion ☐ yes ☐ no

Swelling or thickening of any joints ☐ yes ☐ no

Skin of patagium healthy and elastic ☐ yes ☐ no

Legs

Tibiotarsal length _____

Symmetry of legs when extended ☐ yes ☐ no

Range of motion of leg joints - bilaterally symmetrical ☐ yes ☐ no

Pain on extension or flexion ☐ yes ☐ no

Weakness of grip when perched ☐ yes ☐ no

Symmetrical grip strength ☐ yes ☐ no

Favoring one leg when perched or ambulating ☐ yes ☐ no

Feet abnormally warm ☐ yes ☐ no

Posture (erect, drooped, unstable) ☐ yes ☐ no
If yes, describe: _____

Toes

Toes missing ☐ yes ☐ no
Which one(s): _____

Toes deformed/luxated ☐ yes ☐ no
Which one(s): _____

Sternum

Carina of keel - smooth, straight ☐ yes ☐ no

Breast muscle bilaterally symmetrical ☐ yes ☐ no

ABDOMINAL PALPATION

Normal or increased sterno-pubic distance ☐ yes ☐ no

Palpable fluid in sterno-pubic area ☐ yes ☐ no
Severity/extent of fluid? _____

Masses palpable in sterno-pubic area ☐ yes ☐ no

RESPIRATORY/CARDIOVASCULAR

Nares

Dirty feathers over nares ☐ yes ☐ no

Nasal discharge ☐ yes ☐ no
Character: _____

Nares asymmetrical ☐ yes ☐ no
Describe: _____

Dry (lith), hard mass in nares ☐ yes ☐ no

Infraorbital sinus swollen ☐ yes ☐ no
Describe: _____

Excessive sneezing ☐ yes ☐ no

Dyspnea ☐ yes ☐ no
If yes, characterize the dyspnea: _____

_____

Is neck extended and does the bird vocalize with inspiratory dyspnea ☐ yes ☐ no

Is there increased abdominal movement ☐ yes ☐ no

Open mouth breathing ☐ yes ☐ no

Tail-bobbing ☐ yes ☐ no

Panting with exercise ☐ yes ☐ no

Cessation of panting within 2-3 minutes ☐ yes ☐ no

Auscultation

Respiratory Rate _____ Heart Rate _____

Cardiac murmur . . . . . ☐ yes ☐ no

Arrhythmia . . . . . ☐ yes ☐ no
Describe: _____

Air sacs audible . . . . . ☐ yes ☐ no
Describe: _____

Lung sounds audible . . . . . ☐ yes ☐ no
Describe: _____

Nasal or tracheal noise/fluid/wheeze . . . . . ☐ yes ☐ no
Describe: _____

NEUROLOGIC - SENSORY

Ears

Presence of symmetrical openings . . . . . ☐ yes ☐ no

Discharge or matting of feathers . . . . . ☐ yes ☐ no

Pruritus, excessive scratching at ears . . . . . ☐ yes ☐ no

Fluid or material visible beneath tympanic membrane . . . . . ☐ yes ☐ no

Head tilt . . . . . ☐ yes ☐ no

Eyes

Symmetrical size when viewed head-on . . . . . ☐ yes ☐ no
(If not, R/O glaucoma, exophthalmos, sinusitis, microphthalmia, retrobulbar mass)

Redness or hyperplasia of conjunctiva . . . . . ☐ yes ☐ no

Blepharospasm . . . . . ☐ yes ☐ no

Corneal opacity . . . . . ☐ yes ☐ no

Clarity of lens . . . . . ☐ yes ☐ no

Iris color consistent with age, species and sex . . . . . ☐ yes ☐ no

Pupillary light response . . . . . ☐ yes ☐ no
(Note: consensual response is not present in birds, and voluntary constriction can occur, so interpret carefully.)

Eyelid margins normal . . . . . ☐ yes ☐ no

Does the bird appear visual . . . . . ☐ yes ☐ no

Egg-yolk stroke . . . . . ☐ yes ☐ no

Neurologic exam - use special form not included

REPRODUCTIVE SYSTEM

Female

Abdominal palpation suggestive of egg retention . . . . . ☐ yes ☐ no

Evidence of cystic ovarian disease . . . . . ☐ yes ☐ no

Egg-yolk peritonitis . . . . . ☐ yes ☐ no

Male

Is the vent irritated . . . . . ☐ yes ☐ no

Change in cere color (budgerigars) . . . . . ☐ yes ☐ no

DIGESTIVE SYSTEM

ORAL EXAMINATION

Choana

Choanal papilla normal . . . . . ☐ yes ☐ no

Papillomas in oral cavity . . . . . ☐ yes ☐ no

Presence of plaques . . . . . ☐ yes ☐ no

Abscesses near glottis at base of tongue . . . . . ☐ yes ☐ no

Infundibular cleft visible . . . . . ☐ yes ☐ no

Infundibular cleft swollen or discharge present . . . . . ☐ yes ☐ no

Mucous membrane color appropriate for species . . . . . ☐ yes ☐ no

Sublingual area abscess/masses . . . . . ☐ yes ☐ no

Tongue symmetrical and mobile . . . . . ☐ yes ☐ no

Submandibular space abscess . . . . . ☐ yes ☐ no

Wounds . . . . . ☐ yes ☐ no
Describe: _____

Regurgitation

Passive or active regurgitation noted . . . . . ☐ yes ☐ no

Passively regurgitates water when handled . . . . . ☐ yes ☐ no

Delayed crop emptying . . . . . ☐ yes ☐ no

Food retained in crop/crop distention . . . . . ☐ yes ☐ no

Odor to crop contents . . . . . ☐ yes ☐ no

Lesions/burns/fistulas on crop skin . . . . . ☐ yes ☐ no

Droppings

Odor to feces . . . . . ☐ yes ☐ no

Decreased/increased amount . . . . . ☐ yes ☐ no

Yellow or green in urine . . . . . ☐ yes ☐ no

Yellow or green in urates . . . . . ☐ yes ☐ no

Change in feces color . . . . . ☐ yes ☐ no

Increased liquid in urine . . . . . ☐ yes ☐ no

Increased powdered urates . . . . . ☐ yes ☐ no

White, fluffy droppings . . . . . ☐ yes ☐ no

Undigested food in feces . . . . . ☐ yes ☐ no

Dark brown, black tarry or coffee ground-colored feces . . . . . ☐ yes ☐ no

Parasites (correlate with laboratory fecal exam for eggs) . . . . . ☐ yes ☐ no

Bubbly, gaseous droppings . . . . . ☐ yes ☐ no

Scant feces . . . . . ☐ yes ☐ no

Diarrhea . . . . . ☐ yes ☐ no

Pasting of vent . . . . . ☐ yes ☐ no

Gram's Stain of Droppings

Normal numbers of digestive bacteria (100-150/high power field) . . . . . ☐ yes ☐ no

Decreased number of bacteria (_____/field) . . . . . ☐ yes ☐ no

High % of gram-positive rods (>90%) . . . . . ☐ yes ☐ no

Low % of gram-positive cocci (<10%) . . . . . ☐ yes ☐ no

Gram-negative rods . . . . . ☐ yes ☐ no
☐ >1% ☐ >10% ☐ >30% ☐ >90%

More than 5-10 yeast per field . . . . . ☐ yes ☐ no

More than 10% budding yeast . . . . . ☐ yes ☐ no

Clostridium spp. present . . . . . ☐ yes ☐ no

Undigested fiber . . . . . ☐ yes ☐ no

RBCs in Gram's stain . . . . . ☐ yes ☐ no

WBCs in Gram's stain . . . . . ☐ yes ☐ no

Megabacteria (macrorhabdosis) in Gram's stain . . . . . ☐ yes ☐ no

Fungal or yeast hyphae in Gram's stain . . . . . ☐ yes ☐ no

Cloaca

Vent lips normal . . . . . ☐ yes ☐ no

Diameter of vent and tone normal . . . . . ☐ yes ☐ no

Mucosa of cloaca normal thin, clear tissue . . . . . ☐ yes ☐ no

Irritation, ulceration, cobblestone appearance or papillomas noted . . . . . ☐ yes ☐ no

Acknowledgements

Spix Publishing Inc acknowledges the following veterinarians for their input into the physical examination form: Drs. Greg J. Harrison, Teresa Lightfoot, Bob Doneley, Nigel Harcourt-Brown, Jan Hooimeijer and Thomas Tully.

CHAPTER
7

Emergency and Critical Care

GREG J. HARRISON, DVM, Dipl ABVP-Avian, Dipl ECAMS;
TERESA L. LIGHTFOOT, DVM, Dipl ABVP-Avian;
GWEN B. FLINCHUM, BS, MS, DVM

Fig 7.1 | This dyspneic female cockatiel was unresponsive to stimuli and had flexed claws. This bird died shortly after presentation.

Emergency Stabilization

Avian emergencies are typically more challenging than dog and cat emergencies.[10] This is because birds tend to hide illness such that by the time they are brought in to be examined they are in an advanced state of debilitation. At this point, handling or other stress may be fatal. Some birds will have such grossly severe clinical signs that handling for examination is contraindicated (see the "Put It Down" List in Chapter 6, Maximizing Information From The Physical Examination). Such clinical signs include pronounced dyspnea (Fig 7.1), prolonged panting or gasping for air, inability to grasp with feet, weakness, inability to bite, closing the eyes during the examination (listlessness), lack of normal response to stimuli and incoordination (see Fig 7.3), marked abdominal swelling, frank blood in feces (Fig 7.2) and fluffed appearance. A lack of fecal material in the droppings (Fig 7.4) and anorexia, especially in smaller birds, can foreshadow impending death, which may be hastened by handling. For this reason, these clinical signs have been referred to as a "put it down" list.[21] If such signs are noted during the examination, the bird should be released immediately back into its cage. Very critical patients, especially those that are dyspneic, often will benefit from oxygenation prior to handling. This is especially important with inhalation toxicosis or in cases of tracheal obstruction. The least stressful method of oxygen administration is to place the patient in a chamber connected to an oxygen source to create an environment of 40 to 50% oxygen concentration.[31] When possible, the bird should remain in the carrier in which it is presented, and the carrier with the bird inside is placed into an oxygen chamber (Fig 7.5). The bird should remain in the chamber until it is stabilized. Oxygen toxicity from prolonged exposure has been recorded and should be avoided.

Fig 7.2 | Frank blood in the urine portion of the droppings is frequently an indication of renal disease. Mercury and lead toxicoses have been reported etiologies for hematuria. Blood from the cloacal mucosa may be confused with blood in the urine.

Fig 7.3 | In an emergency situation with a dyspneic bird, minimal handling is required to avoid further stress. Placement of the bird into a container and then placing that container into a plastic bag will allow for direct delivery of oxygen.

Fig 7.4 | Psittacine and passerine birds seldom pass pure urates and urine coated by mucous. Excessive fruit consumption, increased water consumption due to heat, or stress may produce such a dropping in an otherwise healthy bird. Passing such multiple droppings often indicates prolonged anorexia. Also note the yellow-colored bile pigment stain in the urates, indicating hepatic disease. All these indicate a grave prognosis.

Fig 7.5 | Covering the cage of a dyspneic bird with a plastic bag and flushing with oxygen can be life saving.

In these critical cases it is best not to handle the patient immediately. Explain to the client possible differentials, prognosis and a plan of action. During this conversation, the client will have time to comprehend the severity of the bird's condition. Also, the owner may add information to the history that will alter the list of differential diagnoses, preliminary treatment or the prognosis. It is essential that the owner have a clear understanding of the prognosis and cost estimation before treatment is begun.

Once the owner has agreed to hospitalize the bird, a plan of action should be formulated. Severely ill birds may die from handling, and one must proceed in a stepwise fashion. One treatment or procedure is performed, and the bird is then placed back in its enclosure and allowed to recover prior to attempting the next diagnostic or therapeutic step.

AIR SAC TUBE PLACEMENT

In some cases, emergency placement of an air sac tube (Fig 7.6a-c) in the caudal thoracic or abdominal air sac is necessary. This should be done in cases of tracheal or syringeal obstruction. Clinical signs of upper respiratory obstruction include gasping for air, often with the neck extended (see Fig 7.1), and/or making "squeaking" sounds with each breath. Air sac tubes are not indicated for respiratory disease below the syrinx or for non-respiratory origins of dyspnea (ascites or organomegaly). For example, primary lung disorders, such as polytetrafluoroethylene (PTFE) toxicity, will not be improved by the placement of an air sac tube, since the air capillaries will still not be able to absorb and exchange oxygen. Abdominal air sac tube placement is contraindicated in lower respiratory conditions such as air sacculitis. These birds may present with whole-body movement during respiration and/or crackling sounds on lung auscultation.

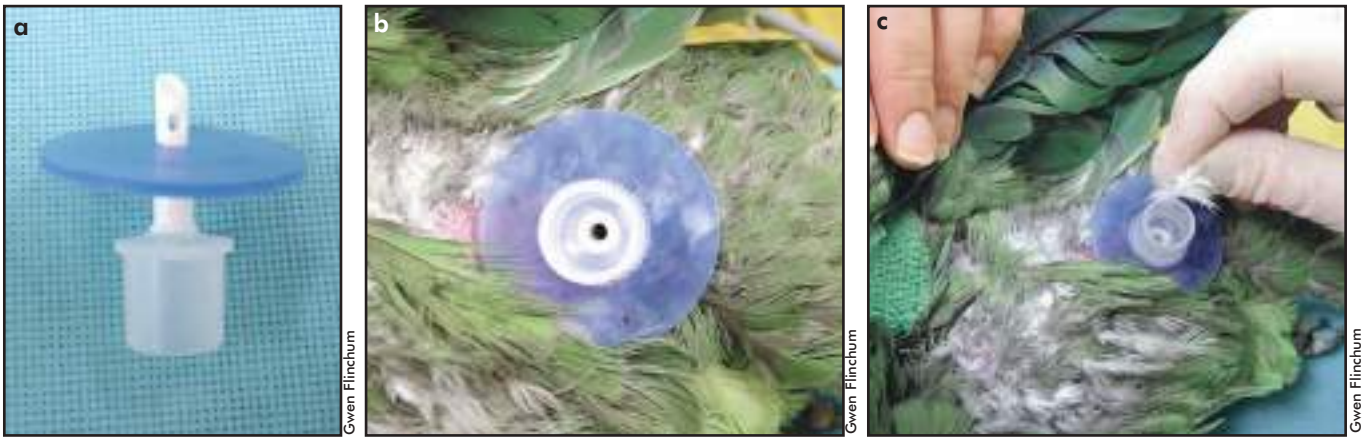


Fig 7.6 | Air sac tube. **a)** Non-cuffed tube[a] and retention disk that is sutured to the skin. **b)** The tube inserted anterior to the leg (or in the left paralumbar fossa). **c)** A down feather plucked and used to detect airflow through the tube.

The approach to lacement of an air sac tube is similar to preparation for lateral laparoscopy (see Chapter 24, Diagnostic Value of Endoscopy and Biopsy). The patient is placed in right lateral recumbency with the dorsal leg pulled caudally. The caudal edge of the eighth (last) rib is palpated and the skin over this site is surgically prepped. A small skin incision is made behind the eighth rib. Using mosquito forceps, the muscle wall is bluntly dissected and the body wall is penetrated. A small tube is placed into the hole (Fig 7.6b). To check if the tube is properly seated, place a down feather over the tube opening. The feather should move with each breath (Fig 7.6c). Keep a tight hold on the feather so it is not sucked into the air sac. Care must be taken to avoid iatrogenic organ damage caused by pushing the tube in too far. The diameter of the tube should be the approximate diameter of the patient's trachea. Modified red rubber catheters work well for air sac tubes, as do endotracheal tubes for medium to larger birds. Avian air sac surgical catheters[a] are ideal because there is a retention disc attached to the tube that makes suturing the tube to the skin easy and secure (see Tracheal Obstruction later in this chapter).

Supportive Care

SICK-BIRD ENCLOSURES

Sick birds are often hypothermic and should be placed in heated (brooder-type) enclosures[b] (Fig 7.7) in a quiet environment (see Chapter 1, Clinical Practice). A temperature of 85° F (29° C) with 70% humidity is desirable for most sick birds. If brooders are not equipped with a humidity source, placing a small dish of water in the enclosure will often supply adequate humidity. A moist towel that is heated and placed on the bottom of a cage or incubator rapidly humidifies the environment, as indicated by the fogging of the acrylic cage front.

Fig 7.7 | A commercial incubator[b] that allows regulation of temperature and humidity. Oxygen can be attached if needed.

FLUID THERAPY

Oral Administration

Oral administration is the ideal method of giving fluids. This method is more commonly used in mildly dehydrated birds or in conjunction with subcutaneous (SC) or intravenous (IV) therapy. Oral rehydration (30 ml/kg PO q 6-8 h) also may be used in larger birds (eg, waterfowl) that are difficult to restrain for parenteral fluid therapy.

Subcutaneous Administration

Subcutaneous fluid therapy is probably the most common method of administration, although administration in very critical patients must be done judiciously. With experience, warm fluids can be given over the dorsum in very depressed birds without restraint or altering of the bird's position within its incubator. Studies have shown that adding hyaluronidase[e] to fluids (150 IU/L fluids) greatly facilitates the absorption of these fluids.[17] Subcutaneous fluids are most commonly given in the intrascapular area, the flank, and the area over the pectoral muscles

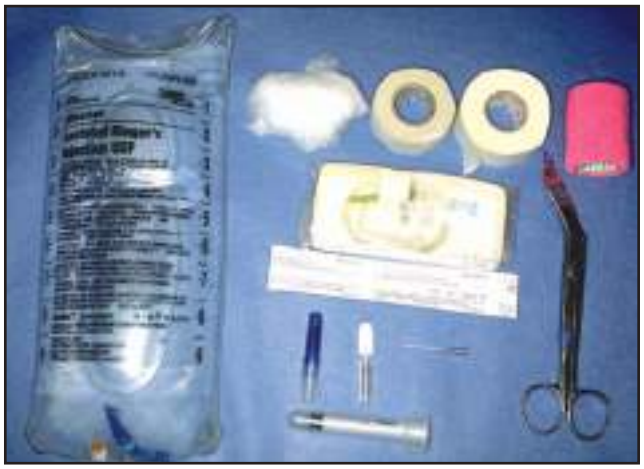


Fig 7.8a | Materials needed for placement of an intravenous catheter in a bird: isotonic warm fluids, syringe and needle for administering flush, heparin flush (not pictured), plastic-coated intravenous catheter[ff] (24-gauge pictured here), a catheter adapter set (extension), bandage scissors, cotton wool, porous tape, stretch fabric tape[z], flexible bandage material[aa] (Note: needle and catheter sizes are based on medium to large psittacine patients).

Fig 7.8b | A butterfly catheter (24-gauge) and a much larger 22-gauge, three-quarter-inch catheter for larger birds are possible alternative indwelling catheters for birds.

or the axilla. Maintaining fluids on a heating pad or in an incubator, so they are available at the correct temperature for emergencies, is important. Warm fluids are both an adjuvant treatment for hypothermia and less painful on administration. However, as in mammals, a severely debilitated or dehydrated bird will not absorb SC fluids.

Intravenous Administration

Intravenous administration of fluids is necessary in cases of severe debilitation or severe hypovolemia. However, when dealing with critical cases in avian medicine, difficult decisions must often be made. For example, some patients may die from the stress of being restrained for injection or catheter placement. On the other hand, IV therapy may be imperative in saving a bird's life. Careful consideration must be given to the bird's history and physical condition. Intravenous hetastarch (10-15 ml/kg q 8 h for 1 to 4 treatments) is indicated for hypoproteinemic patients (total solids <2.0 g/dl).

Intravenous Catheter Placement

Intravenous catheterization facilitates fluid administration; however, catheterization can be challenging due to the small size of bird veins. Avian veins also are more subject to rapid hematoma formation than are mammalian veins. This is especially true with the basilic (wing) vein. Catheter placement may be more easily accomplished with the bird anesthetized, although the risk of anesthesia must be considered. Furthermore, catheter maintenance may be difficult, especially if the bird is prone to chew at the site. Elizabethan collars can be placed to prevent this, but often result in further stress to the patient.

Intravenous plastic-coated catheters (IVC) (Figs 7.8a,b) and the related plastic tubing and various rubber adapters — all derived from human pediatric medicine — have made catheter placement in small birds possible (Figs 7.9a-g). Anesthesia may be required for catheter placement and proper securing of the catheter and line. Once placed, the security of the adhesive materials incorporated into these devices, combined with the severe debilitation of the patients in which these catheters are utilized, makes additional restraint or mechanical barriers usually unnecessary.

With the advent of the use of hyaluronidase in subcutaneous fluids the IVC is seldom required. The multidose IV fluid technique also works on most cases in which subcutaneous fluids with hyaluronidase are inadequate. The disadvantage to bolus IV fluids is that they cause hypervolemia with subsequent polyuria; therefore, less fluid is retained than with a constant rate infusion IV drip. This drawback is minimized by the use of a syringe pump that will deliver as little as 1 cc of fluids over periods of up to 1 hour.

Other Fluid Therapy Methods

An alternative to IV catheterization is intraosseous (IO) catheterization of the distal ulna or proximal tibia. This method is very useful during the first 24 to 48 hours of initial hydration and shock therapy. It also is used to maintain hydration and IV access during prolonged procedures, such as complicated orthopedic repairs. In the latter case, the IO catheter can be placed after the patient is anesthetized, avoiding the pain and stress related to its placement. Insertion of IO catheters tends to be painful and often necessitates anesthesia for placement.

Fig 7.9a | Placing an indwelling catheter in the medial metatarsal vein on a lovebird's leg. The vein is occluded at the proximal tibial-tarsus.

Fig 7.9b | A 24-gauge catheter needle has entered the metatarsal vein. Note the entry site is just the distance of the catheter hub length proximal to the hock. This creates maximum catheter stability when the taping snugs the hub into the depression proximal to the tibioltarsus' distal condyles.

Fig 7.9c | The catheter is advanced to the hub. The hub is taped with a 3- to 4-mm-wide and a 4- to 5-cm-long piece of waterproof adhesive[bb] tape in the fashion shown.

Fig 7.9d | A small tuft of cotton wool is wrapped around the leg.

Fig 7.9e | An extension with an injection port has been attached to the catheter and it is folded along the skin and covered with cotton wool.

Fig 7.9f | A layer of cohesive flexible bandage[aa] is wrapped over the catheter, extension and injection port.

Fig 7.9g | The site is wrapped with stretch fabric tape. Fluids can now be administered by bolus as often as desired, or an IV pump can be attached and a continuous drip administered.

Fig 7.10 | An intraosseous needle and the insertion handle.

This may be too stressful for critically ill birds. An IO needle with a handle[a1] (Fig 7.10) is available and makes catheter placement easier and more precise.

Rectal fluids also may be effective. Posturetal urine can be modified in the rectum after retrograde movement from the coprodeum and subsequently be reabsorbed. In an experimental study, four out of six pigeons were rectally infused with a hypotonic solution, which successfully maintained hydration.[23]

PROTECTIVE DEVICES

Various traumas, postsurgical patients and cases of self-mutilation may require the placement of a mechanical barrier to prevent self-trauma. Some birds require little physical distraction (Fig 7.11a). Historically, circles of exposed radiographic film were designed to encircle the neck, forming a type of Elizabethan (E.) collar. Various types of padding used in conjunction with these collars creates a safe and effective barrier to self-mutilation.

Such devices have several drawbacks, however: they are time-consuming to custom make; one must ensure that the collar itself does not abrade or otherwise injure the bird; if the padding is not properly applied, the film's sharp edge may cause abrasions or lacerations circumscribing the cervical area; additionally, the collar must be designed such that the bird does not destroy it. Radiographic film is not very durable and is challenging to apply (Fig 7.11b).

Plastic disks are commercially available, but many are heavy and cumbersome (Fig 7.11c); birds may stumble and fall, have difficulty perching and/or accessing food and water (Fig 7.11d). These collars may become entrapped in the cage or on cage items. Almost all birds will go through a period of agitation in response to application of these "collars." Flapping in a circular, spastic manner for a prolonged period is not uncommon. Additional padding can be applied to avoid damage to the propatagium during these violent episodes; however, this again adds additional weight to the collars and makes them more cumbersome.

Regardless of the material used, E. collars can be applied facing one of two directions. Applying them with the wide portion forward (cranial) allows for better balance and does not interfere with wing movement; however, it is more likely to impede eating and drinking, and the visual presence of the collar surrounding the bird's head often frightens the bird. If the collar is applied facing backward (with the wide end caudal), the bird can more readily access food and water and can see its surroundings without visual impairment; however, with the collar in this position, the wings cannot be extended for balance. In some cases, the collar must be left long enough to prevent the bird's access to its legs and feet. In these cases, the length of the collar is often significant and the bird will tend to trip on it as it attempts to ambulate.

Plastic disks that provide a neck ring of harder plastic or metal are available. These neck rings may break and some of these devices have a screw-type clamshell neck portion that is very difficult to apply without anesthesia.

Various custom-made, padded cervical collars have been utilized (Fig 7.11e). These are measured from the thoracic inlet — with the neck fully extended —to the mandible. A compressible material consisting of a rubber or plastic foam, such as a swimming pool float or the insulation used to encase refrigeration piping and covered with an elastic tape, has worked well in this author's (GJH) experience. Disposable baby diapers or similar human pads used to absorb urine and feces also can be shaped into lightweight, flexible neck collars (Figs 7.11f,g).

An additional device is a plastic "lego"-like, locking, ballooned out collar[f] (Fig 7.11h). Care must be taken to avoid entrapping the bird's feathers in the device, preventing it from locking. If the bird falls or hits this device it may snap open and fall off.

Regardless of the protective device selected, a primary concern is that the application of the collar does not cause injury to the patient. Various combinations or devices and alternating applications may be necessary.

Removing the collar for a period of time each day so the bird may preen will decrease the stress to some birds during prolonged use of a collar.

The use of collars for feather-destructive behavior is seldom warranted, although their use in cases of self-mutilation can be life saving. This is where the clinical distinction between feather destruction and self-mutilation becomes critical.

Fig 7.11a | This cockatiel needed only the minor distraction of the decorative portion of a child's sock to discourage picking.

Fig 7.11b | A severe case of Amazon foot necrosis that occurred prior to the advent of newer treatment techniques. Such cases faced months of collaring. This collar was reinforced with a heavy-duty fabric tape used in the construction industry. Note that the bird managed to pull in the disk's edge and remove part of the tape. This collar also produced a weight burden for the bird.

Fig 7.11c | Plastic circular disks use a plastic clamshell shape to envelop the neck. The sleeve comes in two parts and is joined by metal screws. Variations of this collar have a padded, rubber-like cervical edge and metal snaps.

Fig 7.11d | An Amazon is collared to prevent feather-destructive behavior. The bird's nutritional disorders, hormonal imbalances and behavior problems were not being addressed. The use of a collar without addressing the underlying problem is inhumane.

Fig 7.11e | A cervical brace-type protection device made from a padded material. While still requiring a period of adjustment, this variation seems superior to the extensive Elizabethan collar for comfort and rapid return to normal function.

Fig 7.11f | A section of black refrigeration pipe insulation that can be cut into various lengths to fit the cervical area and prevent mutilation. Elastic fiber tape makes a durable coating.

Fig 7.11g | Human diaper pads can be used as padding material that is taped to offer durability and attached for a custom-made cervical brace protective device.

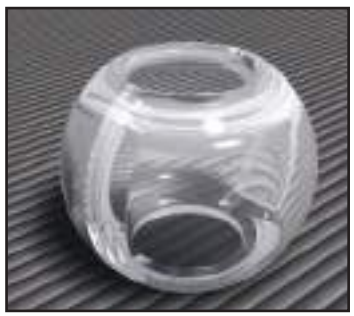


Fig 7.11h | A plastic clamshell protective device[f] with a soft bubble section shaped to prevent the bird from reaching its body. The manufacturer reports faster adaption and a higher rate of comfort.

Birds that have E. collars applied should be kept in the hospital until they have acclimated to their presence. Some birds have an initial reaction of constant flipping and flailing. These birds should be placed in a padded but otherwise empty incubator and observed. The use of a medication such as medazolam prior to the application of an E. collar may aid in its acceptance. Acclimation here is defined as being able to move around a normal cage and access food and water. The collar may require modification or reinforcement, either to facilitate ambulation or to provide a more extensive barrier to self-mutilation. Chewing at the collar may persist to some degree throughout the period it is worn.

Applying a protective barrier to a bird without consideration for and treatment of the underlying etiology is both poor medicine and potentially cruel. If a less cumbersome device can be used, it should be applied.

ANTIBIOTIC THERAPY

The unnecessary use of antibiotics in veterinary medicine is a current concern. When practical, diagnostic testing should be done prior to the initiation of therapy. Minimally, confirmation of the need for antimicrobial therapy should be determined by response to treatment. Complete blood counts should be performed if collecting blood will not endanger the patient. Culture and sensitivity tests can identify infective organisms and the appropriate medication choices; however, it may take several days to get culture results, which is unacceptable for emergency therapy. Fecal, throat or crop Gram's stains can be done quickly, are relatively non-invasive and can be useful in making therapy decisions. Although indiscriminate use of antibiotics is discouraged, prophylactic use may be indicated on the basis of clinical signs and history for birds that cannot undergo further testing.

Antibiotics are commonly given intramuscularly (IM). Although this route is quick and effective, recently there has been concern about muscle necrosis and pain associated with IM administration. It has been suggested that medications be given SC, especially in smaller birds with less muscle mass. Controlled studies are needed to see if absorption from this location will provide adequate blood levels of antimicrobials.

Many critically ill or septicemic birds require IV injections. This requires repeated venipunctures or catheter placement. Disadvantages of IV injections include the possibility of extravasation, hematoma formation and stress to the patient.

ANTIFUNGAL THERAPY

Ideally, antifungal therapy is based on culture and sensitivity testing. This is almost never done in practice due to the extended period of time involved, the lack of laboratory support for such studies and the lack of a wide-range of therapeutic choices. Gram's stain results, such as budding yeast or fungal hyphae, or confirmation of aspergillosis or other fungal infection through cytology or histopathology will help confirm a diagnosis. Antifungal medications also may be indicated during antibiotic therapy to decrease the risk of secondary yeast infections, especially in immunosuppressed birds. Birds with a history of malnutrition and chronic respiratory disease may benefit from prophylactic use of antifungal medications until a diagnosis can be made. Aspergillosis secondary to chronic vitamin A deficiency and subsequent squamous metaplasia may be an underlying problem in these birds. Among pet birds, Amazons (*Amazona* spp.),[1,14] African grey parrots (*Psittacus erithacus*)[1,25] and macaws (*Ara* spp.)[6] appear to be especially susceptible to aspergillosis. Antifungal medications exhibit diversity in their mechanisms of action, toxicities and cost. An understanding of these properties will enable intelligent decisions as to which drug is appropriate for a particular situation.

MISCELLANEOUS MEDICATIONS

Corticosteroids

Corticosteroids have been widely debated due to the potential complications arising from their use.[26] These side effects include immunosuppression, adrenal suppression, delayed wound healing and gastrointestinal ulceration. Most of these side effects are seen following a prolonged course of administration of corticosteroids, although reports of potential glucocorticoid-induced adrenal suppression after topical use have been cited.

However, single doses of corticosteroids have been reported to improve the prognoses in cases of shock, trauma and toxicity[31] without the occurrence of clinically observable adverse side effects. The use of corticosteroids is not recommended in birds that have had a history of immunosuppression or fungal disease. Dexamethasone sodium phosphate (2 mg/kg IM or IV once) is the preferred steroidal anti-inflammatory for birds.[4] Prednisolone sodium succinate (0.5-1.0 mg/kg IM or IV once) has been reported to be most effective in cases of neurologic emergencies (see Stress in Chapter 19, Endocrine Considerations).

Glucose Therapy

Hypoglycemia may be present in cases of malnutrition or starvation, sepsis or hepatic dysfunction. This is seen most often in passerines, but seldom in psittacines and occasionally in raptors. Blood glucose levels can be determined on a laboratory panel, and levels will fall to below half of the normal for the species in hypoglycemic patients.[5] Severe cases must be treated immediately, as

symptoms may include seizures, weakness and depression. Treatment includes IV administration of 25% dextrose (1-2 ml/kg slowly to effect), although dextrose alone should not be used in dehydrated patients.[5] Oral solutions such as honey or Karo® syrup also can be used in birds that are not prone to aspiration. The underlying cause, if hypoglycemia, must be subsequently determined and corrected.

o,p'-DDD

Mitotane, or o,p'-DDD, is an adrenal-blocking drug that has been shown to inhibit stress-induced hypersecretions of corticosteroid, thus bolstering immunocompetency during stressful situations.[13] Adrenal blockers also have been shown to arrest tumor growth and inhibit stress-induced tumor metastasis.[11] o,p'-DDD is used in cases of suspected immunosuppression and for neoplastic conditions. Its use should be avoided in cases of suspected pesticide toxicity.

ORAL NUTRITIONAL SUPPLEMENTS

Below are listed some of the oral nutritional supplements that can be gavage-fed to debilitated birds. Various hand-feeding formulas are on the market and, as a whole, are far superior to the homemade formulas used decades ago that contained monkey biscuits, peanut butter and ground seeds. Commercially available hand-feeding formulas for baby birds are often utilized in the treatment of sick and debilitated adult birds. The quantity that can be fed at one time to a sick bird is greatly reduced from that of baby birds. On the average, a baby parrot can accommodate 10% of its body weight per feeding due to the elasticity of the crop and its rapid emptying. Adult birds have a greatly decreased crop capacity, averaging 3% of their body weight. Additionally, sick birds are less tolerant of food in the crop and care must be taken to avoid regurgitation and/or aspiration.

A sick or debilitated bird should always have its hydration corrected prior to attempting to initiate oral gavage-feeding.

Some formulas that are used and the indications for these are summarized below. None of these formulas is indicated in the presence of ileus. Many ill birds are captive-raised and were hand-fed, and these patients may respond to hand-feeding techniques (Fig 7.12). This facilitates both feeding and medication administration.

Carbohydrate Supplement

A simple carbohydrate powder[i] (fructose and malt dextran) can be mixed with water, Tyrode's solution[j] or Normosol-R to make a non-viscous tube-feeding solution. This is useful for birds to resolve crop stasis because the thin consistency of the carbohydrate supplement mixture is more digestible than regular hand-feeding formulas. It also is useful for hypoglycemic patients or those with primary liver disease. Another similar product[k] is recommended for patients with primary hypoglycemia or for birds needing additional calories provided as highly digestible carbohydrates.

Fig 7.12 | Hand-feeding a sick bird using a warmed hand-feeding product may be attempted prior to tube-feeding. Hand-feeding will be more time-consuming but less stressful for the patient if accepted.

Following rehydration in sick birds, the liver needs adequate carbohydrates to carry out its functions, so carbohydrates are next in line as nutriceuticals for ill birds. Carbohydrates are generally followed by a more complex oral nutritional formula within 24 to 48 hours.

Carbohydrate Metabolic Detoxifier[h]

Based on a rice protein concentrate, this product works as a metabolic detoxifier for the liver while also providing calories. This is useful in cases of chronic liver disease and when severe biliverdinuria is present. It is used for several days following the initial use of the carbohydrate-only formula. This is usually followed by the use of the hand-feeding formula or a sick-bird support formula.

Sick-bird Support Formulas

Medical and surgical patients are often undergoing pansystemic debilitation. A product[l] with simple proteins (isolated soy protein) and simple familiar fats (hi-oleic sunflower oil) is added to the carbohydrate supplementation listed above to further meet the bird's nutritional requirements. See the discussion on carbohydrates in Chapter 4, Nutritional Considerations for the raffinose values of soy.

Many surgical and medical patients have been fed a seed diet and need the additional nutritional support gained from the administration of a balanced formula.

The method of administration of the supplemental formula will depend on both the patient's condition and the experience of the person administering the food.

1. Hand-feeding can be an ideal way to supplement birds that revert to baby begging and feeding behavior when ill. However, the administration of a sufficient quantity of supplement at each feeding requires that the patient be strong enough to demonstrate an adequate feeding response, and that the person feeding be experienced enough to avoid causing aspiration of the material.
2. Gavage-feeding also requires experience. When gavage-feeding is performed, the experienced person will be capable of avoiding the following inherent dangers:
   a) Mechanical damage to the oropharynx with the metal crop feeder
   b) Damage to the beak with inappropriate use of oral speculums
   c) Accidental tracheal gavage
   d) Reflux of the formula from the crop into the oral cavity

(See Chapter 1, Clinical Practice).

Precautions: Feeding sick birds should not be attempted by inexperienced persons. Do not attempt to hand-feed birds that are not aggressively feeding. Do not use formulas that are too cold or too liquid. Feedings must be accomplished promptly to avoid appetite loss. Failure to heed these cautions could result in aspiration and death.

Hand-feeding Formula[d] (Fig 7.13)

Although originally formulated for hand-feeding juvenile birds, these formulas have been ideal for nutritional support of malnourished birds or as a tube-feeding formula for sick birds. The average dose for psittacines is 30-60 ml/kg per day, divided and dosed q 6-8 h for an adult bird, in addition to parenteral fluid administration, assuming normal gastrointestinal motility.

TOTAL PARENTERAL NUTRITION (TPN)

Total parenteral alimentation is the IV administration of all essential nutrients. Indications for use include gastrointestinal stasis, severe head trauma that precludes oral alimentation, and malabsorption or maldigestion.[31] Although parenteral alimentation has been described,[9,30] it is not routinely utilized in avian medicine. There can be difficulties in placing and maintaining a catheter, and sepsis and bacteremia can occur if the catheter becomes contaminated. The constant monitoring of the electrolyte and acid/base blood values for adjustments to this formula is blood volume-prohibitive in pet birds.

Total parenteral nutrition given intraosseously (TPN/IO) was reported in 1995 and was tried experimentally on pigeons. Hyperglycemia and glucosuria were encountered and resulted in the death of one bird. Another bird had complications at the site of administration. No further reports are currently available. Financial constraints may be an additional reason that parenteral nutrition is not being administered to avian patients. A recent e-mail (TPN January 9, 2003 xoticvet@shaw.ca on exoticdvm@yahoogroups.com) stated that the cost of having a pharmacist compound a TPN formula was $100 Canadian for 300 ml, and the shelf life was very short (several days).

Fig 7.13 | A formulated diet designed for hand-reared psittacines. The psyllium in this product[d] cleans the digestive system, improves hydration and helps restore electrolytes while reducing toxins and cholesterol. This product delivers 1.5 Kcal/ml when reconstituted.

FREQUENTLY SEEN EMERGENCIES

Liver Failure/Malnutrition

Malnutrition is a major cause of liver disease in birds eating seed-only diets. This is due to the high fat and low nutrient content of seeds. As with many diseases in birds, no clinical signs may be apparent until the patient is in an advanced stage of decompensation. Clinical signs may include lethargy, inappetence, diarrhea with loose green feces and green or gold urates (Fig 7.14) (see Chapter 4, Nutritional Considerations, Section II). There may be no abnormalities on the serum chemistries of these patients, although elevations in either the hepatic enzymes or bile acids may be present.[16] Hepatic disease with malnutrition as the underlying cause may present commonly as hepatic lipidosis, fibrosis and cirrhosis.

Routine dorsoventral and lateral radiographs can be helpful in identifying hepatomegaly or decreased liver size. Bag radiographs (see Figs 1.17a,b,c, 1.18a,b) are non-invasive and can be done by placing the bird in a paper bag for radiography. This technique is useful for

Fig 7.14 | Bilirubin in the urates, an indication of hepatopathy.

Fig 7.15 | Milk thistle[m] with a vegetable glycerin base is used as an oral supplement to aid hepatic regeneration.

screening for the presence of heavy metal densities, but not reliable for determination of hepatic size (see Chapter 1, Clinical Practice for information on bag radiographs).

Response to treatment for malnutrition-induced hepatic disease depends on the duration of the disease and the extent of organ dysfunction. Treatment includes parenteral fluid administration, treatment of any concurrent infection and gavage-feeding with an appropriate formula. In one author's (GJH) practice, this is more specifically defined as the administration of subcutaneous fluids, and tube-feeding with sick-bird support formulas[d,1]. If recovery is possible, a response to treatment should occur within 24 to 48 hours. In the presence of a leucocytosis, with or without hepatic enzyme or bile acid elevations, antimicrobials are routinely used by one author (TLL). These are empirically based on the CBC differential and fecal Gram's stain and may often include both antibiotic and antifungal agents.

Milk thistle (silymarin[m,12,34,37]) has been shown to improve liver function and enhance regeneration of damaged liver cells (Fig 7.15).

Various other medications may be added to aid in hepatic function (see Chapter 15, Evaluating and Treating the Liver).

A diet change from seeds to a formulated diet is imperative in long-term successful treatment.

Egg Binding

Egg binding is common in lovebirds, budgerigars, cockatiels, eclectus parrots and older Amazon parrots (10 years or older) that are usually fed seed-based diets. Clinical signs include swollen abdomen, fluffed appearance and lethargy. Often, birds will act as if they are trying to defecate but cannot; therefore, the client concern may be "constipation." A retained egg may be palpated in the abdomen, although care should be taken not to confuse a caudally displaced ventriculus for an egg during palpation of the sterno-pubic area. Radiographs are recommended to help identify soft-shelled eggs or double eggs, as well as secondary problems such as an enlarged liver shadow or coelomic fluid accumulation.

Treatment includes parenteral calcium supplementation (if the shell is soft), SC fluids and supplemental heat (see Chapter 5, Calcium Metabolism). Oxytocin (5 IU/kg IV or SC once) has been recommended to help in egg expulsion. It has been reported that oxytocin can have adverse side effects and should be used only if the uterovaginal sphincter is well dilated and the uterus is free of adhesions. However, since oxytocin is not the primary hormone of uterine contraction in birds, it is only partially effective, and the effect of exogenous administration is diminished. Both prostaglandin F-2 alpha and prostaglandin E are reported to be effective in cases of egg binding. Prostaglandin F-2 alpha is more readily available, although it also carries the concern of oviduct rupture if the utero-vaginal sphincter is not dilated. Prostaglandin E is used in human obstetrics. Egg extraction via the cloaca is the least invasive method and can be utilized in most cases (Fig 7.16). When dealing with soft-shelled eggs, eggs adhering to the oviduct or uterus, or cranially located eggshell fragments, laparotomy may be necessary. Collapsing the egg with a syringe and needle (implosion) may facilitate removal. When the egg cannot be visualized via the cloaca, this may be due to complications such as an impacted uterus or mummified egg. During egg extraction all shell material must be removed. If left in the uterus, shell fragments tend to migrate proximally, making retrieval very difficult. The use of delivery forceps, feeding needle, syringe, and cotton-tipped swab facilitates egg or egg fragment removal (Figs 7.17a-c). The collapsed egg can occasionally be

Fig 7.16 | An egg-bound bird with the eggshell adhered to the uterus. This is typical in birds with nutritional disorders.

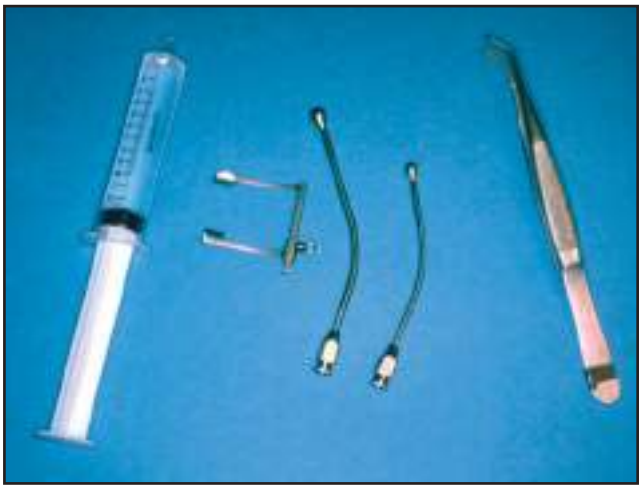


Fig 7.17a | Equipment pictured here may be useful in treating egg-bound birds; a syringe for flushing the uterus with normal saline, an ophthalmic speculum, metal feeding needles for flushing, and a curved-tip forceps for dilating the uterus and grasping the shell of a collapsed egg.

Fig 7.17b | Anesthetized female positioned over a towel to aid in proper cloacal inspection. The tail is perpendicular to the spine.

Fig 7.17c | A lateral view of the forceps dilating the cloaca and the feeding needle in place to puncture the egg, aspirate the contents or flush out shell fragments.

Fig 7.18 | Expressing a collapsed egg after it has been aspirated transabdominally.

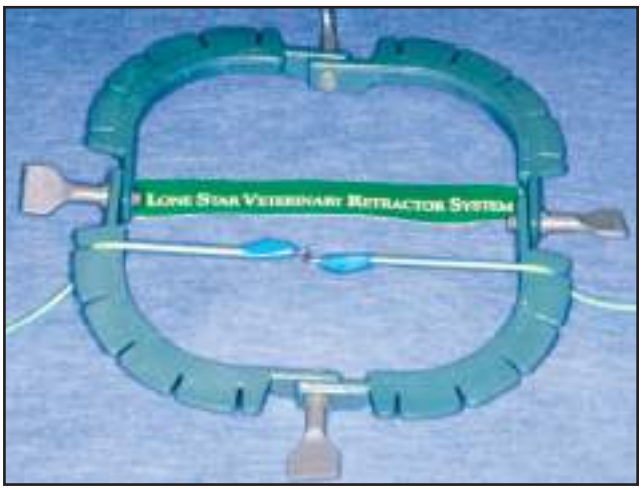


Fig 7.19 | A retractor[n] makes cloacal examination much easier.

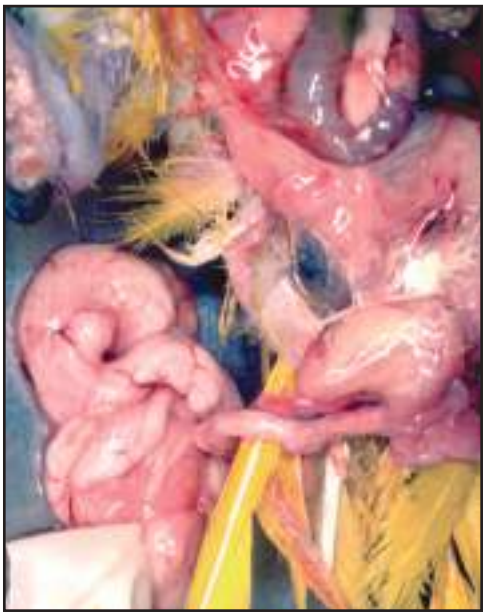


Fig 7.20 | A ruptured uterus allowed the egg to become retro-coelomic, causing death in this cockatoo.

removed intact (Fig 7.18). Use of a retractor[n] (Fig 7.19) makes visualization much easier. (See Chapter 35, Surgical Resolution of Soft Tissue Disorders for more on retractor use. See Chapter 18, Evaluating and Treating the Reproductive System for forceps and procedures). Occasionally the uterus will rupture and the egg will be free within the coelomic cavity (Fig 7.20).

The rigid, semi-flexible[o] (Fig 7.21) or flexible endoscope for egg delivery is useful. Warm saline/air flushing and insufflation with the endoscope can make visualization and subsequent egg particle removal much less stressful than blind forceps delivery. This is especially true for eggs that have migrated proximally and for retrieval of eggshell fragments. Once the egg has been removed and the bird is stabilized, a series of leuprolide acetate (500-1000 μg/kg IM q 2 weeks, 3 times)[3,38] or chorionic gonadotropin (HCG) (500 IU/kg IM on days 1, 3, 7, 14 and 21);[22] injections have been recommended. One author (GJH) has found diet change from seeds to a formulated diet mandatory for full recovery and prevention of recurrence. Dexamethasone has empirically improved the response to HCG injections (see Chapter 18, Evaluating and Treating the Reproductive System for an excellent review).

Tracheal Obstruction

Tracheal obstruction is frequently seen in cockatiels subsequent to seed inhalation. In larger species of psittacines, tracheal obstruction is often due to the presence of tracheal granulomas or aspergillomas that impinge on the tracheal lumen. The history of these birds often includes primarily a seed diet, with subsequent vitamin A deficiency. Clinical signs include open-mouthed breathing with a high-pitched squeaking noise emitted each time the bird breathes. The bird will often have a forward-leaning posture as it attempts to increase air intake (see Fig 7.1). Flexible and rigid endoscopes can be used for oral approaches to the larynx and trachea. A 1.2 mm x 20 cm semi-flexible endoscope[o] (see Fig 7.21) is a valuable tool for visualizing and identifying the tracheal obstructions in birds under 100 g. Nebulization can be used to soften obstructive material. A needle can be placed through the trachea to prevent distal migration of the material. The bird is then held upside down and suction is applied until the obstruction is resolved. With cockatiels, a tom cat catheter can be utilized to provide a tube of appropriate diameter for aspiration of the material. A surgical approach to the thoracic inlet has been described and may be used to remove foreign material, but the surgical risk is significant.[2] A final option is to push the foreign body into one main-stem bronchus and into the lung, and follow up with aggressive antibiotic and antifungal therapy. Air sac tube placement (discussed earlier in this chapter) will likely be necessary for any attempted treatment. Without an air sac tube, the bird's respiration may be severely compromised by the presence of equipment in the trachea (see Chapter 24, Diagnostic Value of Endoscopy and Biopsy and the previous section of this chapter regarding air sac tube placement).

Gwen Flinchum

Fig 7.21 | A semi-flexible endoscope for examining the salpingo-uterine area in an egg-bound bird.

Vomiting and Regurgitation

Differentiation must be made between behavioral and pathologic regurgitation. Behavioral regurgitation is usually done in a controlled fashion and directed at an object in the cage or the owner. Pathologic regurgitation is not controlled and the patient will often sling its head as it regurgitates. Causes for regurgitation include toxicity, especially heavy metal, foreign body ingestion, bacterial, fungal or protozoal gastrointestinal infection, ingestion of oral irritants (ie, biting into a common houseplant of the *Pothos* sp.), goiter in budgerigars, rancid food ingestion, gastrointestinal obstruction, pancreatitis, renal

failure, hepatopathy, septicemia, dehydration, polydipsia, motion sickness and proventricular dilatation disease. Diagnostic tools include radiography (plain film and barium series), blood metal level tests, Gram's stains of fecal, crop or regurgitated material, and culture and sensitivity. Determining and correcting the underlying cause is necessary for long-term treatment. Initial therapy may include parenteral fluid administration, metoclopramide parenterally, crop flushing, frequent gavage-feeding of small amounts of a non-viscous formula.

Gastrointestinal Stasis

The most important aspect of resolving gastrointestinal stasis is to identify the underlying cause. Differential diagnoses include proventricular dilatation disease, bacterial or fungal crop infection, foreign body ingestion, septicemia, gastrointestinal obstruction, toxicity or other underlying disease. The first step in treatment of gastrointestinal stasis is to remove excessive material from the crop if present. A Gram's stain or culture and sensitivity of the flush contents will help yield a diagnosis if primary crop infection is present. Rehydration must be accomplished via parenteral fluids prior to initiating gavage feeding. Once this is accomplished and the crop is empty, the bird should be tube-fed small amounts of a dilute solution such as non-lactated multielectrolyte solution and 5% dextrose or carbohydrate substitute.[j,k] If obstruction is not suspected, gastrointestinal stimulants such as cisapride[p] (0.5-1.5 mg/kg PO q 8 h) or metoclopramide (0.5 mg/kg IM q 8-12 h as needed) may improve motility. Although cisapride has been taken off the market in the United States, it is still available through compounding pharmacies. It also is important to note that metoclopramide has been shown to cause seizures in macaws at 0.5 mg/kg; however, when given at 0.1 mg/kg both PO and IM, no hyperexcitability or seizures have been noted by one author (TLL). As gastrointestinal motility improves, tube-feedings can be made more concentrated until normal consistency is tolerated (see Oral Nutritional Supplements in this chapter). Identification and treatment for the underlying cause of gastrointestinal stasis must be accomplished (see Chapter 14, Evaluating and Treating the Gastrointestinal System).

Blood in Droppings

It is important to note whether blood in the droppings originates from the urine, feces, reproductive tract or cloaca. Blood in the urine (see Fig 7.2) is a sign of kidney disease and is commonly caused by toxicity or tumors. Some birds will pass blood in the urine transiently after restraint. This vessel's fragility is not normal and, like occult blood in the feces, may be a sign of liver or gastrointestinal disease. A fecal test[q] will identify blood versus artifact.*

*(*Ed. Note: The authors have performed tests on 15 clinically normal boarding birds: no blood was grossly or microscopically noted in the stools and all were negative on the occult blood test. It has been stated that all birds' stools test positive for occult blood, however, we did not find this to be true. Positive blood on a fecal in a normal bird may be found when the diet includes meat products.)*

Melena is the passage of black, tarry feces containing blood that has been acted upon by the digestive system. This is commonly seen in advanced liver disease, gastrointestinal ulceration and starvation. It also may be seen with gastric and hepatic adenocarcinoma and pancreatitis. Treatment should include treatment for the underlying cause, vitamin K_1 injections (0.2-2.5 mg/kg IM as needed) and parenteral fluid administration. Tube-feeding with barium sulfate suspension concentration (0.05 ml/g body weight PO as needed) provides a protective and anti-inflammatory coating to the gastrointestinal tract, assuming no perforation exists.

Hematochezia is the passage of frank blood in the feces. Causes include foreign body ingestion, reproductive disease, neoplasia, papillomas, cloacitis and gastroenteritis. The appearance of frank blood that is separate from the fecal portion and the urine portion when multiple droppings are examined is generally either reproductive or cloacal in origin. In South American psittacines, the presence of cloacal papillomatosis should be suspected and a cloacal examination done to rule out this disorder. Frank blood or hemoglobinuria may be seen in the urine in some cases of lead and other heavy metal toxicosis.

Cloacal Prolapse

Cloacal prolapse has been associated with behavior problems (see Chapter 3, Concepts in Behavior, Section III, Pubescent and Adult Psittacine Behavior), poor nutrition, neoplasia, papillomatosis, infection and reproductive complications. Prolapsed tissue should be inspected for necrosis or tears. Viable tissue can be gently replaced using a gloved finger or blunt swab lubricated with activated yeast gel[r]. In this author's (GJH) experience, when there is severe tissue inflammation, the topical application of a dexamethasone-dimethyl sulfoxide (DMSO) solution (4 mg:10 ml) will help reduce swelling with no apparent clinical adverse effects; however, inflamed tissue in addition to DMSO may allow systemic uptake of these drugs causing potential adrenal suppression. Hyaluronidase also can be utilized to reduce tissue swelling. Its effect is assumed to last for several days, as has been documented in people (TLL).

Suture placement to reduce the cloacal opening has been described to maintain reduction of a prolapse; however,

if the underlying cause is not corrected, this will be only a temporary solution. In recalcitrant cases (ie, sexual behavior/masturbation), 2-0 nylon may be necessary, as smaller sutures will not hold. Care must be taken to avoid damage to the innervation of the vent. Do not place sutures directly within the vent lips. Do not inhibit defecation by having excessive restriction of cloacal outflow. Cloacal atony associated with these conditions has been reported to respond to cisapride.[p,39]

Many birds with cloacal prolapse may present for foul-smelling diarrhea. A Gram's stain will often demonstrate overgrowth of clostridial organisms when this fetid diarrhea is present in association with cloacal prolapse and straining. Although the clostridial overgrowth is likely secondary to irritated cloacal mucosa and fecal retention, it still requires treatment when encountered. Gram-negative overgrowth also can occur, and a culture and sensitivity of stool would then be indicated.

More persistent or recurrent prolapses may require a cloacopexy. When the cloacal margins have been excessively stretched, surgical reduction may be necessary to reduce the diameter of the opening (see Chapter 35, Surgical Resolution of Soft Tissue Disorders).

The owner should be forewarned that there is danger of stricture and/or impediment to defecation or urination when a cloacopexy is performed, and unless underlying causes are addressed and corrected, the prolapse is likely to recur.

Uterine Prolapse

Uterine prolapse may occur as a sequel to egg binding or in conjunction with cloacal prolapse. Frequently, an egg will be visualized within the prolapsed tissue. Prolapsed tissue must be carefully flushed with sterile saline and examined for tears and necrotic areas. Treatment includes supplemental heat, warm SC fluid administration, parenteral calcium and broad-spectrum antibiotics. Secondary yeast infections are commonly seen in these cases, so antifungal medication also may be indicated. Once tissue has been debrided and inspected, it can be gently replaced using a gloved finger or sterile swab. However, the suspensory ligament has usually been damaged by the prolapse, and suturing the cloaca to hold the uterus as it involutes or performing a cloacal hysterectomy may be the best approach. Severely necrotic uterine tissue may require resection. Subsequent ovulation would, therefore, potentially allow intracoelomic follicle deposition and egg-yolk peritonitis in some species and individuals. Birds with uterine prolapses frequently have poor prognoses, since they are often malnourished and may have advanced liver disease or other underlying health problems.

Metal Toxicities

Toxicities are frequently seen in birds, especially in those that are allowed to roam freely in the house. Metal toxicity is common, with zinc toxicosis seen most often in the USA. Clinical signs may include general malaise, polyuria and diarrhea, and polydipsia with subsequent passive regurgitation of water. Neurologic signs are more common with lead toxicosis than with zinc toxicity. Radiographs can be helpful in identifying metal particles, however, metallic foreign bodies will not be seen in some zinc-toxic birds.[36] Conversely, not all ingested metal is toxic. Placing a bird in a paper bag for radiography facilitates screening for metal densities with minimal stress. Blood metal concentration tests[s] are dependable for the diagnosis of metal toxicosis;[35] however, significant blood elevations of metal may not always occur in clinically ill birds.[15] Furthermore, there is disagreement as to what constitutes accurate reference levels for metal toxicities.[28] Cases are best diagnosed and treated after careful consideration of history, clinical signs and test results.

Treatment for metal toxicity involves removal of the metal pieces from the gastrointestinal tract and/or chelation of metal ions in the bloodstream. Cathartics, such as lactulose or 1% psyllium in a hand-feeding formula,[d] speed elimination of smaller metal pieces. Oils such as peanut butter are much less effective. Magnets are used in larger birds for removal of sizable iron pieces that have been galvanized with lead or zinc. For chelation therapy, the following drugs are commonly used:

1. Injectable edetate calcium disodium injection, USP[t], (CaEDTA) (35 mg/kg IM or IV q 12 h for 5 days, then repeat as needed) is an effective chelator. Its disadvantages include expense, pain at the injection site and potential nephrotoxicity (although this has not been documented in birds to date). The advantage of CaEDTA is that its parenteral formulation allows administration even in birds that are regurgitating.
2. Succimer (30 mg/kg PO q 12 h for 14 days) is an oral chelating compound that is safe and effective, especially for lead. It is usually made by a compounding pharmacy into a 25-mg/ml suspension.[36]
3. D-penicillamine[u] is the author's (GJH) drug of choice (55 mg/kg PO q 12 h for 1 to 2 weeks, off 1 week, then repeat as needed). It is an oral chelating compound that is safe and effective. However, it is available only as an oral medication, and therefore may not be used for the initial therapy if the patient is regurgitating. Also, it has been reported to cause regurgitation in some birds. Prolonged use may create a copper deficiency.
4. DMSA and dimercaprol.

Airborne Toxicities

Superheated, Non-stick Plastic Fume Toxicity

Superheated, non-stick plastic fume toxicity, or polytetrafluoroethylene (PTFE) gas poisoning, can be rapidly fatal. Clinical signs include severe dyspnea, weakness and coma. Treatment should include supplemental oxygen, SC fluids, aminophylline (10 mg/kg IV q 3 h, after initial dose, other doses can be given PO), dexamethasone sodium phosphate (0.8 mg/kg IV once), heparin (40-50 IU/kg IV once) and nebulization with heparin, lactated Ringer's solution and dexamethasone. The prognosis for severely affected birds is extremely poor (see Chapter 31, Implication of Toxic Substances in Clinical Disorders).

Air Fresheners

Clinical symptoms similar to PTFE toxicity have been observed in smaller birds (budgerigars, canaries) after exposure to plug-in or wick air fresheners. Treatment is the same as for PTFE toxicity.

Scented Candles

Sneezing and nasal discharge may occur after exposure to lighted, scented candles. Some scented candles contain wicks made with lead. When these are lit, the resulting odor can be poisonous to birds as well as humans.

Candy Cooking Flavoring

Flavorings such as peppermint and spearmint used in candy cooking while birds were present in the kitchen caused death in two budgies that were located 10 feet from the stove in an open cage (K. Marx, personal communication, 2003).

It is likely that other aerosolized agents may be toxic to birds. Limiting the exposure of psittacines to any aerosolized substance is a reasonable precaution.

General Recommendations for Toxicity

When toxicity is suspected, a complete anamnesis should be collected to determine the substance that is most likely responsible. If the bird is not regurgitating, activated charcoal solution[v] can be given (10-20 ml/kg PO) to prevent or reduce toxin absorption in the gastrointestinal tract. Subcutaneous or IV fluid administration encourages the elimination of toxins through urination. If cholinergic toxicity is suspected, atropine (0.1-0.2 mg/kg IM or SQ q 4 h PRN) is indicated.

Poisoning by plants is infrequently seen; other references including human and veterinary poison control centers are recommended if such a case is encountered.

BEAK, NAIL AND FEATHER TRAUMA

The tip of the upper beak may be cracked without any blood or obvious displacement of the rostral fragment. The bird may present for clinical signs such as decreased appetite, hesitancy to climb or failure to preen and history of a fall or other trauma. When digital pressure is applied to the rostral portion of beak, the distal damaged fragment may shift.

One method of correction is to use an electric grinding tool and hone off the fractured tip. If bleeding occurs, the practitioner may use the grinding stone's heat, or other cautery method, to cauterize the tip of the newly exposed beak. Soft foods may be used for a short period to encourage the bird to eat. It may be best to perform this procedure under a light plane of inhalation anesthesia. Damage to the beak tip is painful, and the anesthesia will prevent elevation of the bird's blood pressure and associated increase in bleeding.

Broken nails are a concern if they bleed excessively. Cutting off any remaining distal portion and cauterizing as discussed above is usually corrective. Similarly, applying flour as a first aid measure may be palliative until attended by a veterinarian. Telephone advice for this condition includes suggesting that the owner place flour in the bottom of a shoebox or other container and place the bird inside. This way, the bird's nail will come in contact with the flour without the increased blood pressure (and subsequent increased bleeding) that can occur with restraint.

Broken and bleeding feathers are handled similarly to the beak and nails. Distal fragments can be removed, and if still actively bleeding, place flour directly on and in the follicle. Ligating the proximal portion of the feather may be necessary. Caution must be used in advising clients to attempt chemical cautery at home. Inadequate restraint and insufficient application of hemostatic agents combined with the increased blood pressure generated by the restrained bird often result in prolonged and repeated bleeding episodes.

Cautery or pulling the broken feather is best avoided if possible. Pulling the feather may result in damage to the feather follicle's generative tissue, resulting in a crooked or cystic feather.

Excessive bleeding from any source should be investigated. Hepatic disease is frequently an underlying cause of coagulopathy.

More severe beak injuries are discussed in Chapter 14, Evaluating and Treating the Gastrointestinal System.

TRAUMA

Head Trauma

This is most often seen in free-flying birds encountering a ceiling fan or sliding glass doors. Cranial trauma may

Fig 7.22 | Micro-encapsulated ammonia solution[w] for postsurgical or traumatic pain, burns and swelling. It is the author's (GJH) observation that the solution also aids in preventing bacterial overgrowth.

cause neurologic clinical signs. Prednisolone sodium succinate is a rapid-acting steroid that helps decrease central nervous system swelling. Dexamethasone sodium phosphate also can be given (2 mg/kg IM once). Head trauma cases should be placed in quiet, dark places with no heat. Minor cases usually recover within 24 to 48 hours.

Limb or Body Trauma, Bites, Lacerations

Non-penetrating traumas can be greatly aided by the use of a topical ammonia solution[w] (Fig 7.22). This product reduces pain, swelling and the incidence of infection. (GJH)

In the case of fractures and deep penetrations, stabilization of the patient is the first priority. If there is no active or severe bleeding, warm, quiet and oxygenation may be the logically provided emergency care. While the bird is stabilizing in the warm, humidified oxygen chamber, the practitioner can be collecting information from the client that will aid in subsequent determination of the location and extent of the injuries, and in their treatment.

If significant active bleeding is present, this must be controlled. The application of direct pressure to the wound for several minutes may accomplish this. Remember that a bird's blood pressure can undergo a threefold increase in response to painful stimuli. Therefore, if the bleeding has stopped, it may be wise initially to refrain from exploring the injured area until the patient is stable (see earlier portion of this chapter). Repeated manipulation and restraint may cause the resumption of bleeding.

If bleeding persists, however, one or more of the following steps should be taken:

1. Application of a pressure wrap.
2. Radiosurgical cautery.
3. Application of ferric subsulfate.
4. The use of Yunnan Piao[x], a Chinese herb that also is an excellent hemostatic agent.[24] It can be used as a topical agent or mixed with a non-lactated multielectrolyte solution and gavage-fed prior to surgery. This decreases blood loss in surgeries where excessive bleeding may occur (GBF).
5. Vitamin K_1 injection and evaluation for hepatopathy.

Once bleeding is controlled and the patient is stable, wounds should be debrided and cleaned. Any feathers that are sticking in the wound should be cut or removed. Wounds can be flushed with 50% dextrose for its bacteriocidal properties.[19] Sterile saline also can be used. If there is dirt and dried blood caked in the wound, gentle cleaning with gauze and 0.5 to 1.0% povidone-iodine is helpful. In birds, many wounds will heal very well by second intention without sutures. Patients that pick at their wounds may require a collar.

BANDAGING

Modern gel-type dressings (Figs 7.23a-d) have made bandaging of bird injuries much easier and more successful. When combined with the improved properties of adhesive tapes (see Figs 7.9f,g), bandage removal has been made difficult to impossible for the bird and relatively easy for the veterinarian.

If needed, protective neck devices or collars can augment the bandage for additional safety. The bandages can usually be left on for 2 days. The hydroscopic or gel dressing can be reapplied at the time of bandage change.

Wounds that are deep, extensive or obviously contaminated require parenteral antibiotic administration as well as topical debridement. Puncture wounds, often from dog or cat bites, may appear minor but have a great potential for infection and septicemia.

Split Vent or Ruptured Pygostyle

This condition is commonly seen in malnourished cockatiels and other psittacines, and results from trauma to the region caudal to the vent during a fall (Fig 7.24). When the bird's tail feathers strike the floor, the skin and connective tissue surrounding the vent is stretched to the point of tearing. The skin in malnourished birds is not flexible and it tears easily. These wounds often have significant exposure of the subcutaneous tissue and muscle. Treatment includes debridement and flushing of the wound. Transecting the tail feathers so that they do not touch the floor as readily helps minimize repeated stretching trauma. The bird must be housed so that repeated trauma does not occur. If the wings were severely clipped, they should be allowed to grow out to allow some glide and decrease the severity of impact. Amazingly, these wounds may heal very well without sutures by second intention. In selected patients, suturing may be indicated. Critical to the long-term prevention of recurrence of this condition is changing the

Fig 7.23a | An example of a gel dressing with a biosynthetic absorbent coating[cc] that helps to maintain wound hydration. This type of dressing does not adhere to skin and is ideal for wounds without appreciable necrotic debris.

Fig 7.23b | A hydroactive dressing[dd] is soft and spreads out as it absorbs serum from a wound and molds to the area. This "gel" is an excellent debriding agent and also forms a matrix in which cells can grow. If there is copious serum production, the bandage should be changed daily. To apply, the protective backing of the dressing is removed and the yellow-brown gel side is applied to the wound. A flexible bandage[aa] is then applied over the dressing.

Fig 7.23c | A section of the gel dressingd[dd] has been cut into the desired shape and the protective backing is removed. The red necrotizing tissue of Amazon foot necrosis is readied for the bandage with the bird under anesthesia.

Fig 7.23d | The H-shaped dressing is placed on the plantar surface of the claw. The four legs of the H fold around the metatarsus and the third digit, covering all surfaces of the wound. A coating of a cohesive, flexible bandage[aa] and an over-covering of elastic-cloth tape[z] is applied as shown in Figs 7.9 f,g.

bird's diet to correct the malnutrition and obesity that were contributing factors.

ACUTE BLOOD LOSS AND ANEMIA

Acute blood loss is more rapidly life threatening in malnourished birds because they frequently have decreased liver function with associated coagulopathies. Severely anemic birds will appear pale on the skin covering the nares and feet, and have pale mucous membranes of the cloaca and oral cavity. Tachypnea and tachycardia also may be present.

As with other species, the administration of oxygen will increase the efficiency of the remaining red blood cells.

Fig 7.24| A cockatiel with a split in the tissue just caudal to the cloaca's yellow feathers. This is a sign of a nutritional disorder that has diminished the skin's elasticity. Improvement of the bird's diet, preventing the bird from falling on hard surfaces and treatment as an open wound are indicated.

Transfusions are indicated when the packed cell volume (PCV) is below 20%. Homologous transfusions, from the same genus and species, yield the best result.[7,8] Transfusion volume should be 10 to 20% of the patient's calculated blood volume.

Studies showed that using washed RBCs (no plasma) in conures the same genus as a donor was as efficacious as homologous transfusions (white-eyed conure to sun conure, sun conure to sun conure) for a single transfusion.[8] In another study, however, cockatiels received Amazon blood (a heterologous transfusion), and these washed and labeled RBCs were short-lived.[7]

Other alternatives for blood transfusions have recently become available. Hemoglobin glutamer-200 (bovine) (Oxyglobin[y]) (30 ml/kg IV once) has been shown to be effective in the resolution of clinical signs in dogs, cats, sheep, rats, horses and a number of exotic animals.[27] Its use in avian medicine has been investigated,[20] although it is not labeled for use in birds. Hemoglobin glutamer-200 is expensive due to the lack of availability of small packaging, but it is potentially useful in treating avian anemia.

Hetastarch

Hetastarch (10-15 ml/kg IV q 8 h up to 4 treatments)[4,20] is used primarily for intravascular volume restoration; however, it can be used in anemic patients when blood products are unavailable.[29]

Erythropoietin[g]

Erythropoietin[g] (100 IU/kg SC 3 times per week until desired PCV is attained) has been reported to improve success in treating chronic anemia by stimulating erythrocyte production in some mammals.[29]

Erythropoietin[g] is a hormone that stimulates erythropoiesis in severely anemic patients. Avian erythropoietin, which is stimulated and released following hypoxia and suppressed by induced polycythemia, is structurally different than the mammalian type. Whether or not mammalian erythropoietin acts to stimulate RBC production in birds is not documented.

Interferon and F10

In the United Kingdom, preliminary results of clinical trials on circovirus-positive African greys with precipitous decreases in WBC and RBC counts show promise using interferon, F10[ee], and Oxyglobin[y] injections (M. Stanford, personal communication, 2003).

As with all critical conditions in birds, the stress of treatment must be considered prior to initiating intensive therapy. Birds with severe anemia have little oxygen reserve, and treatment that causes stress with increased oxygen demand may prove fatal.

FRACTURES

In cases of fractured limbs, it is a priority to stabilize the patient before attempting fracture repair. Birds will die from stress, anesthesia, debilitation or septicemia rather than from the fractured limb. Topical analgesic[w] (Fig 7.23) helps reduce soft tissue swelling and pain. Analgesics such as butorphanol are indicated for systemic pain relief in fracture patients (see Chapter 8, Pain Management).

For emergency care of fractures, bandaging, and fracture repair see Chapter 34, Surgical Resolution of Orthopedic Disorders.

Products Mentioned in the Text

a. 20 Fr Non-cuffed Tube with Retention Disk, Cook Veterinary Products, Bloomington, IN, USA, 1-800-777-222, custserv@cookaust.com.au

a1. Intraosseous Needle and Handle, Cook Veterinary Products, Bloomington, IN, USA, 1-800-777-222, custserv@cookaust.com.au

b. Animal Intensive Care Unit, Lyon Electric Company, Chula Vista, CA, USA, 619-585-9900, lyonelect@aol.com

c. Tyrode's solution, Zoological Education Network, www.exoticdvm.com

d. Juvenile Formula, Harrison's Bird Foods, Brentwood, TN, USA, 615-221-9919, www.harrisonsbirdfoods.com

e. Wydase, Wyeth Laboratories Inc, Philadelphia, PA, USA, 1-800-934-5556, www.wyeth.com

f. Spherical cervical collar, Gary Nelson, DVM, DRGHN@aol.com

g. Epogen (epoetin alfa), Jansen C.lag. Agmen-Roche

h. UltraClear Plus, UltraBalance Medical Foods, Gig Harbor, WA, USA, 1-800-843-9660, www.ultrabalance.com

i. Ultrafuel, Twin Laboratories, Ronkonkoma, NY, USA

j. Tyrode's powder to make solution, Zoological Education Network, 800-946-4782, www.exoticdvm.com

k. Critical Care; Nutri-Support; Carbo-Boost, Lafeber Co, Cornell, IL, USA, 1-800-842-6445, www.Lafeber.com

l. Recovery Formula, HBD International, 7108 Crossroads Blvd, Suite 325, Brentwood, TN 37027, customerservice@harrisonsbirdfoods.com, www.harrisonsbirdfoods.com

m. Milk thistle, Zoological Education Network, www.exoticdvm.com

n. Lone Star Medical Products, Inc, 713-796-0505, www.LSMP.com

o. Karl Storz, 1-800-955-7832, www.karlstorzvet.com

p. Propulsid, Janssen-Cilag, www.janssen-cilag.com. Must be obtained in the US from a compounding pharmacy

q. Hemoccult Fecal Test, SmithKline Diagnostics, San Jose, CA, USA

r. Preparation H Gel, Whitehall-Robins Healthcare, Madison, NJ, USA

s. Louisiana Veterinary Medical Diagnostic Laboratory, Baton Rouge, LA, USA, 1-504-346-3193

t. Calcium versenate, 3M Pharm, Northridge, CA, USA 91324

u. Cuprimine, Merck, www.Merck.com

v. Toxiban, Vet-A-Mix, Shenandoah, IA, USA

w. Penetran, Zoological Education Network, www.exoticdvm.com

x. Mayway Corp, Oakland, CA, USA, 1-800-2-Mayway, www.mayway.com

y. Oxyglobin, Biopure Corp, Cambridge, MA, USA

z. Elastikon, Johnson & Johnson Medical, www.jnj.com/home.htm

aa. Equi-flex, Burns Vet Supply Inc, Rockville Centre, NY 11590

bb. Nex-care Waterproof adhesive tape, 3M Healthcare, St. Paul, MN, 55144-1000

cc. BioDres, DVM Pharmaceuticals, Miami, FL, USA www.dvmpharmaceuticals.com/about_dvm.html

dd. DuoDERM, ConvaTec, Bristol-Myers Squib, PO Box 4000, Princeton, NJ 08543-4000, USA

ee. F10, Health and Hygiene (Pty), Ltd, Sunninghill, South Africa, www.healthandhygiene.co.za/products-select.asp

References and Suggested Reading

1. Bauck L: Mycoses. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l Inc, 1999, p 1001.
2. Bennett A: Approach to the thoracic cavity of birds. Exotic DVM 1(3):71, 1999.
3. Bowles H: Update of management of avian reproductive disease with leuprolide acetate. Proc Assoc Avian Vet, 2001, pp 7-10.
4. Carpenter JW, Mashima TY, Rupiper DJ: Exotic Animal Formulary 2nd ed. Philadelphia, WB Saunders Co, 2001, pp 161, 221.
5. Clippinger TL, Platt SR: Seizures. *In* Olsen GH, Orosz SE (eds): Manual of Avian Medicine, St. Louis, Mosby, 2000, p 173.
6. Clubb SL: Psittacine pediatric husbandry and medicine. *In* Altman RB, Clubb SL, Dorrestein GM, Quesenberry KE, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, p 94.
7. Degernes LA, et al: Autologous, homologus homologous and heterolgus heterologous red blood cell transfusions in cockatiels (*Nymphilus hollandicus*) J Avian Med Surg 13(1):2-9, 1999.
8. Degernes LA, et al: Autologous, homologus homologous and heterologous heterologous red blood cell transfusions in conures of the genus *Aratinga*. J Avian Med Surg 12(1):10-14, 1999.
9. Degernes LA: A preliminary report on IV TPN in birds. Proc Assoc Avian Vet, 1995, p 25.
10. Degernes LA: Trauma medicine. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l Inc, 1999, p 42, Fig 7.
11. Deguchi M, et al: Usefulness of metyrapone treatment to suppress cancer metastasis facilitated by surgical stress. Surgery 125(1):120, 1995.
12. Duke JA, duCellier J, Beckstrom-Sternberg S: Western herbal medicine: Traditional materia medica. *In* Schoen AM, Wynn SG (eds): Complementary and Alternative Veterinary Medicine, St. Louis, Mosby, 1998, pp 323,351.12.
13. Flinchum GB, Harrison GJ: Observations on the effect of adrenal-blocking drugs in terminally ill pet birds. Exotic DVM 2(3):71-75, 2000.
14. Forbes NA: Respiratory problems. *In* Beynon PH, Forbes NA, Lawton MPC (eds): Manual of Psittacine Birds. British Small Anim Vet Assoc, 1996, p 152.
15. Fudge AM, Speer B: Selected controversial topics in avian diagnostic testing. Sem Avian Exotic Pet Med 10(2):97-98, 2001.
16. Fudge AM: Avian clinical pathology: Hematology and chemistry. *In* Altman RB, Clubb SL, Dorrestein GM, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, p 149.
17. Griffin C, Snelling LR: Use of hyaluronidase in avian subcutaneous fluids. Proc Assoc Avian Vet, 1998, pp 239-240.
18. Harrison GJ, Ritchie BW: Making distinctions in the physical examination. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l Inc, 1999, p 151.
19. Kelleher S: Wound and abscess management in rabbits. Exotic DVM 2(3):49-51, 2000.
20. Lichtenberger M, et al: Administration of Oxyglobin and 6% hetastarch after acute blood loss in psittacine birds. Proc Assoc Avian Vet, 2001, p 15.
21. Lightfoot TL: Avoiding disaster in the critical patient. Proc Assoc Avian Vet, 1998, pp 265-271.
22. Lightfoot TL: Clinical use and preliminary data of chorionic gonadotropin administration in psittacines. Proc Assoc Avian Vet, 1996, pp 303-306.
23. Lumeij JT, Ephocrti C: Theory and practice of rectal fluid therapy in birds. Proc Europ Com Assoc Avian Vet, London, 1997, p 101.
24. McCluggage D: Holistic medicine in exotic species practice. *In* Schoen AM, Wynn SG (eds): Complementary and Alternative Veterinary Medicine, St. Louis, Mosby, 1998, pp 655,658.
25. McMillan MC, Petrak ML: Aspergillosis in pet birds: A review of 45 cases. Proc Assoc Avian Vet, 1988, p 35.
26. Orcutt C, Flinchum G: Contrasting views on corticosteroid use in birds. Exotic DVM 3(5):43, 2001.
27. Orcutt C: Oxyglobin administration for the treatment of anemia in ferrets. Exotic DVM 2(3): 44-46, 2000.
28. Osofsky A, et al: Determination of normal blood concentrations of lead, zinc, copper and iron in Hispaniolan Amazon parrots. J Avian Med Surg 15(1):31-36, 2001.
29. Plumb DC: Veterinary Drug Handbook 3rd ed. Ames, Iowa State Univ Press, 1999, pp 284,376.
30. Pollock C: Practical total parental nutrition. Proc Assoc Avian Vet, 1997, pp 263-276.
31. Quesenberry KE, Hillyer EV: Supportive care and emergency therapy. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l Inc, 1999, pp 393,396.
32. Romagnano A, et al: Treatment of a hyacinth macaw with zinc toxicity. J Avian Med Surg 9(3):185-189, 1995.
33. Romagnano A: Avian obstetrics. Sem Avian Exotic Pet Med 5:180-188, 1996.
34. Saller R, Meier R, Brignoli R: The use of silymarin in the treatment of liver disease. Drugs 61(14):2035-2063, 2001.
35. Tully T, Hoefer H, VanSant F: Heavy metal toxicosis. J Avian Med Surg 11(2):115-118, 1997.
36. VanSant F: Zinc and clinical disease in parrots. Proc Assoc Avian Vet, 1997, pp 387-391.
37. Wellington K, Jarvis B: Silymarin: A review of its clinical properties in the management of hepatic disorders. Biodrugs 17(7):465-489, 2001.
38. Zantop D: Using leuprolide acetate to manage common avian reproductive problems. Exotic DVM 2(3):70, 2000.
39. Zantop D: Cisapride Use in birds. HBD's Avian Examiner - The 4-Year collection, 1998, p 35. Available online at avianmedicine.net.

CHAPTER

8

Pain Management

JOANNE PAUL-MURPHY, DVM, Dipl ACZM

Greg J. Harrison

Fig 8.1 | With such a devastating lesion, pain would seem obvious, but the canary did not show evidence of pain until this band was removed.

What is Pain?

The International Association for the Study of Pain (IASP) defines pain as an unpleasant sensory and emotional experience associated with actual or potential tissue damage.[2] The IASP also includes under this definition an important note: *The inability to communicate in no way negates the possibility that an individual is experiencing pain and is in need of appropriate pain-relieving treatment* (Fig 8.1). This is a very important statement that gives credibility to pain experienced by non-verbal populations of species, including humans as well as all animal species.

All animals have neuroanatomical and neuropharmacological components required for nociception: transmission, perception and response to noxious stimuli. But when does nociception become pain? This is not easily determined, but for the purposes of this chapter it will be accepted that not only do birds perceive and respond to noxious stimuli, but also that birds feel pain. Pain is always subjective and the emotional component is difficult for us to translate because birds do not share a verbal language with humans. In humans, we accept that pain is what the patient says it is, but in birds as well as other animal species, it is what people think it is.

RECOGNIZING PAIN

The challenge is to recognize avian behaviors that occur with painful stimuli, both acute and chronic. Perhaps birds have evolved behaviors to minimize painful displays as a way of minimizing the attention of predators. We are not yet fully capable of recognizing when a bird is affected by pain; therefore, it may be best to err on the side of over-estimation and assume that conditions that would be painful to humans also are painful to

birds. Nonetheless, having an identified behavior or set of behaviors that correlate with pain provides a means to monitor response to pain treatment. It is difficult to say we have effectively treated something if we cannot measure it before and after therapy. But there is no reliable measurement or gold standard for the assessment of pain in animals, including birds, either for research subjects or clinical patients. Assessment of pain must give consideration to species, gender, age, strain, environment and concurrent disease. Significance must be given to the type of pain, such as acute, chronic, somatic, visceral, clinical or neuropathic.

Behaviors that occur with acute noxious stimuli are the easiest to identify. There is an immediate cause-effect relationship between activation of nociceptors (a receptor preferentially sensitive to a noxious stimulus), withdrawal reflexes and behaviors that correspond to conscious perception of the stimulus.[12] Noxious stimuli that have been used to study avian pain are similar to those used in mammalian studies, such as electrical stimulation, thermal stimulation and pressure, but other studies have employed noxious stimuli unique to birds, such as feather removal, beak amputation and comb pinching, as well as research models for chronic pain using intra-articular injection of sodium urate.[12,18] Physiological measurements are not always consistent or specific to painful stimuli and are not useful for assessment. Respiratory rate, heart rate and blood pressure will increase during painful stimuli but also can be affected by light, sound, temperature, restraint and other external stimuli. Avian withdrawal behaviors to acute painful stimuli include escape reactions (foot lifting, wing flapping), vocalizations, decreased head movements or extreme movements (jumping). When parrots were given a mild electrical stimulus to the foot using a specially designed test perch, the expected response was foot withdrawal with a reluctance to place the foot back on the perch; but there also were several individual parrots that did not lift the foot, but began flapping their wings when a stimulus was delivered.[31] Chickens can display a distinctive behavior termed crouching immobility, which occurs experimentally when they are exposed to repeated noxious stimulation.[12,14,37]

How do we know when a bird is experiencing prolonged pain or chronic pain? It is generally accepted that when a bird is in pain there is a change in or absence of one or more normal behaviors. To accurately assess pain, the observer must be familiar with normal and pain-associated behavior of a given species of bird as well as that of the individual bird. Social species of birds will decrease their social interactions. This may be as subtle as a reduction in social grooming behaviors or as obvious as perching away from the flock. For social species of birds housed as single pets, there may be a reduction in interactions with the owner. This is often manifested by a decrease in vocalizations or chattering that birds often do with the owner or reluctance to seek stroking or petting from the owner. Guarding behavior to protect a painful body part is seen in birds and can subtly manifest in similar antisocial behaviors. Aggression has been linked in individual birds to painful conditions because often the aggressive behaviors dissipate once the clinical problem is resolved. A behavioral change in feather grooming is common to both solitary and social birds. The change can be either an increase or decrease in grooming. Decreased self-grooming is a withdrawal behavior that occurs when a bird's focus is on pain and not daily activities. Increased grooming behavior to themselves or other birds in their environment can be an intentional distraction. Studies using chickens with induced sodium urate arthritis demonstrate that shifting attention can reduce the severe pain and potentially reduce peripheral inflammation.[13] Alternatively, grooming and feather picking may increase over a specific area, often directly related to the region of discomfort or painful stimulus.

Decrease in appetite and weight loss often accompany painful conditions. In a study of rats observed postoperatively, dehydration and weight loss due to decreased food and water consumption was significantly lower in rats given adequate perioperative analgesia.[9] Unfortunately, dehydration and weight loss can occur under numerous stressful conditions, from something as simple as changing the perches in a bird's cage to a variety of known diseases. Weight loss is not unique to pain, but because it can be quantified it is very valuable to record a bird's daily weight. If weight gain or stabilization occurs following administration of analgesia, then it was a useful gauge.

Tonic immobility is an innate fear response characterized by a profound and reversible state of motor inhibition. In chickens, crouching immobility has been associated with prolonged pain, stress and fear responses. Studies using chickens demonstrated that repeated feather plucking caused an initial agitated response, which progressed to sustained crouching immobility of the chicken.[12,14] A later study using chickens with ulcerated oral lesions placed an irritating substance on the lesions, which caused several birds to go into a crouch-like posture, with the head held close to the body and a significant reduction in head movements.[11] The immobility reaction is prolonged when fear is present and reducing the fear component decreases the time of immobility. Companion birds may display similar tonic immobility postures postoperatively or under chronically painful conditions. It has been suggested that tonic immobility evolved as a response of a prey species to reduce potential damage produced brought on by struggling. In

guinea pigs, it has been demonstrated that tonic immobility induces an endogenous analgesia involving opioid synapses, which increases the animals' threshold to noxious stimuli.[21]

The correlation of elevated corticosterone has been studied in its relationship to pain. It is a hormonal indicator known to become elevated with ACTH (adrenocorticotropic hormone) release and stress in birds and other animals.[24] Pain can induce both acute and chronic stress. Fecal corticosterone levels may offer a measurable response that can be collected without interfering with the bird's normal activities.[22] Studies are currently in progress to determine if measurable amounts of corticoids in avian feces correlate to painful stressors.

PAIN ASSESSMENT SCALES

Pain assessment scales are used in human medicine for verbal as well as non-verbal segments of the population. Pain scales can be sensitive and repeatable, and a similar process could be used to develop pain scales for birds with the understanding that each would have to be species-specific. As an example, a recent study with pigeons noted that body trembling consistently occurred for several hours after recovery from general anesthesia, orthopedic surgery and opioid analgesia, but it did not occur with controls given the same period of general anesthesia and opioid, but *without* orthopedic surgery. Additionally, all pigeons received the opioid after surgery, but the group of pigeons that also received opioids prior to surgery stopped trembling earlier in the recovery period than the other surgical group (J. Paul-Murphy, unpublished data). Body trembling may be specific to pigeons or it may be specific to orthopedic surgery, or it may be specific to some other variable not measured in this early attempt to develop a pain scale for pigeons. There are currently more than 26 published pain scales for children, each trying to manage variables that may confound pain assessment.[23] A pain scale can be a very useful assessment tool under consistent conditions, and it may be necessary to develop numerous species-specific scales until a more accurate method is found to assess pain.

PHYSIOLOGY OF PAIN

The physiology of pain in all animals involves the peripheral process of detecting a noxious stimulus (mechanical, thermal or chemical) and transmission of the impulses to the spinal cord. Here they are modulated and projected to the brain for central processing of the information, which determines the perception of the noxious stimulus. Nociceptive pain involves the activation of the pain receptors, is usually localized and transient, and usually activates a withdrawal response, both reflexive and conscious. All other types of pain are considered clinical or pathological, often involving tissue damage with inflammation or nerve damage. Pathological pain can originate from a gentle tactile stimulus, causing an exaggerated or prolonged pain response, or can persist in the absence of noxious stimuli.

Peripheral sensitization occurs when inflammation at the site of injury creates an increased response to a normally painful stimulus. Cell damage and leakage leads to a series of responses resulting in increased sensitivity of the peripheral receptors and may even activate "silent" nociceptors, which magnify the pain response. In addition to sensitization at the peripheral tissue and pain receptor environment, the central nervous system can be sensitized as well. Central sensitization is an increase in the excitability of spinal cord neurons and a recruitment of neurons not involved in pain perception under normal circumstances. When stimulation from the peripheral nociceptors to the spinal cord continues for an extended time period, a wide range of spinal neurons become sensitized and hyper-responsive. The response to additional noxious stimuli becomes heightened and prolonged. Stimulation from the periphery that was previously non-noxious, such as a tactile stimulus, becomes painful.

Understanding the mechanism of peripheral and central sensitization helps to explain why prevention of sensitization is critical and provides multiple places in the process that could be altered by different classes of analgesic therapy. When sufficient analgesia is provided at an early stage of tissue trauma, such as with surgery, it can greatly reduce the degree of postoperative discomfort.[33] Although this has not been experimentally demonstrated in birds, animal and human studies have demonstrated that when analgesics are given prior to a painful event rather than after the start of the stimulation, the spinal excitability can be dampened.[35,36] The earlier pain is treated, the less total drug will be required to maintain analgesia both during and after surgery. It has been shown in mammalian species that a shorter period of analgesia is needed in the postoperative period when analgesics are given in the preoperative period.[33] A study of pigeons undergoing orthopedic surgery showed that the pigeons receiving butorphanol before and after orthopedic surgery returned to normal behaviors sooner than those pigeons receiving butorphanol only in the postoperative period (J. Paul-Murphy, unpublished data).

RELIEF OF PAIN

Pain is a combination of both peripheral inputs and neurophysiological processes within the central nervous system. Diagnosing the cause of pain can be difficult at times, but identifying the disease process or site of tissue damage will influence the choice of analgesic drugs

and supportive care. Therapy should be directed at resolving the disease process or injury as well as reducing pain signals coming from the periphery and its effects on the central neural processing of the pain. For example, immobilizing a bird's fracture maximizes bone healing, but it also reduces micromovement irritation and inflammation, thus reducing noxious stimuli projected from the site of trauma. Analgesia therapy may include an opioid plus an NSAID, but the immobilization itself greatly reduces the pain. Conversely, we may recognize the signs of pain in a patient before we actually diagnose the cause, and in these cases, treatment of the pain becomes part of the symptomatic approach to therapy. Selection of analgesic drugs should be done conservatively in situations where the cause of pain is still under investigation.

Supportive care is very important to the management of pain. This includes keeping the avian patient warm, dry and clean. Environmental stressors should be kept to a minimum by providing a quiet, soothing hospital environment away from barking dogs or strong smells of "predators," such as cats and ferrets. If the bird is a pet, provide human contact and speak in a soothing voice.

Pharmacological Approach to Pain Control

LOCAL ANESTHETICS

Local anesthetics are used to produce regional anesthesia and analgesia by blocking the transmission of noxious impulses. Local anesthetics such as lidocaine and bupivacaine block sodium channels in the nerve axon, which interferes with the conduction of action potentials (pain impulses) along the nerve. Reducing the number of pain impulses reduces the nociceptor sensitization, which has the beneficial effect of minimizing central sensitization. Local anesthetics do not need to be distributed systemically for the analgesic effect, but will slowly be absorbed by the vasculature in the region being blocked.

Regional infiltration or a local line or splash block are among the most common methods used. A 25-gauge needle is used to make several subcutaneous (SQ) injections of small volumes of dilute solution into the operative area. A line block follows the course of the intended incision by injecting a modicum of dilute anesthetic SQ, then withdrawing the needle and reinserting it at the edge of the raised area. Another modicum is delivered under the skin and the process is repeated until the length of the incision is blocked.

Lidocaine has a commercial preparation of 2% (20 mg/ml), and the formulation without epinephrine is recommended. The commercial preparation should be diluted 1:10 with sterile water or diluted further to the volume required for the block. The total dosage should not exceed 2 to 3 mg/kg. For example, a 500-g cockatoo can receive 1 mg lidocaine; 0.05 ml is diluted to 1 ml and this solution is used to block a 2-cm surgical skin incision. The commercial preparation of bupivacaine is prepared as 0.25% (2.5 mg/ml), 0.5% (5 mg/ml) or 0.75% (7.5 mg/ml) solution. No bupivacaine dosage has been established for birds, but 1 mg/kg total dose has safely been administered to large birds.

Local anesthetic dosage recommendations for birds are lower than for mammals because birds may be more sensitive to the effects of the drug. Systemic uptake of the drug may be rapid in birds, increasing the potential for onset of systemic reactions. There can be acute toxic effects if these drugs are accidentally injected intravenously. Toxic side effects can include fine tremors, ataxia, recumbency, seizures, stupor, cardiovascular effects and death. Chickens were injected with high doses of bupivacaine (2.7-3.3 mg/kg) and showed immediate signs of toxicity such as drowsiness and recumbency.[18]

The duration of action of local anesthetics depends on the molecular properties of each drug, especially the lipid solubility of the drug. Neither the time to effect nor duration of action has been determined for these drugs in birds. In mammals, bupivacaine lasts much longer (4-10 hours) than lidocaine (1-3 hours).

Intra-articular administration of bupivacaine (3 mg in 0.3 ml saline) was studied for its analgesic effects in chickens with experimentally produced acute arthritis. Chickens given bupivacaine were able to feed, peck and stand on the affected limb similar to birds without arthritis.[18]

Topical benzocaine has been used for minor wound repair in small birds.[3] Topical bupivacaine has been studied when applied to the amputation site in beak-trimmed chickens and provided 4 hours of analgesia.[16] In mammals, local anesthetics also are used in the form of transdermal patches and transdermal creams, but the use of these patches and creams has not been studied with birds. Local anesthetics also are used in mammalian medicine for epidural infusions, spinal blocks, intravenous blocks and peripheral nerve blocks and as an antiarrhythmic agent, but none of these applications has been reported for use in birds.

OPIOIDS

Opioids reversibly bind to specific receptors in the central and peripheral nervous systems. There are three major classes of opioid receptors associated with analge-

sia: mu, delta and kappa. Chickens have similar proportions of each receptor type as do humans.[7] The distribution, number and type of opioid receptors are conserved across species in the brainstem and spinal cord but vary substantially in the forebrain.[27] In mammals, mu and kappa receptors primarily are associated with pain relief, with mu receptors being widely distributed in the forebrain, midbrain and hindbrain, whereas, delta and kappa receptors are numerous in the spinal and supraspinal regions of the central nervous system (CNS). Autoradiography was used to identify mu, kappa and delta opioid receptors in the forebrain of rats, mice and humans, and kappa receptors represented 9, 13 and 37% of the total opioid receptor population, respectively. In contrast, the forebrain of pigeons has a relatively high proportion (76%) of kappa receptors.[27]

All drugs in the opioid classification have morphine-like effects, which may be mediated primarily by an increase in serotonin synthesis. Given at appropriate dosages, opioids do not cause a loss of consciousness but can cause sedation and respiratory depression. Opioids vary in their receptor specificity and efficacy at different receptors, which results in a wide variety of clinical effects in different mammalian species, and is influenced by the commercial preparation of opioid, the dose and the species receiving the drug. It stands to reason that the opioid and the dose also will have a wide range of clinical effects in different avian species.

Physiological and analgesic effects of opioids have been studied in parrots using the isoflurane-sparing technique. This method anesthetizes healthy birds with isoflurane and determines the minimum anesthetic concentration (MAC) by using a noxious stimulus (toe pinch) and observing a withdrawal response with a cognitive body movement. Each bird then is injected with an analgesic and the MAC is redetermined. If the concentration of isoflurane can be lowered, then this "sparing effect" is considered due to the analgesic effects of the drug being tested. Butorphanol (1 mg/kg) was administered to three species of parrots, and the isoflurane MAC could be significantly lowered in cockatoos and African grey parrots but not Amazon parrots.[5,6] The addition of butorphanol also caused lowering of the heart rate, tidal volume, and inspiratory and expiratory times. A similar type of study compared mu and kappa opioids in chickens, and both drugs had isoflurane-sparing effects.[4]

The effect of different opioids on conscious parrots was evaluated by studying the change in withdrawal threshold from noxious electrical and thermal stimuli before and after receiving an opioid. Butorphanol, 1 and 2 mg/kg IM, had an analgesic effect on African grey parrots, and 1 and 3 mg/kg had an analgesic effect on Hispaniolan Amazon parrots.[30,31] Plasma concentration of 2 mg/kg butorphanol in African grey parrots has a mean residence time of less than 2 hours (J. Paul-Murphy, unpublished data). Buprenorphine at 0.1 mg/kg IM in African grey parrots did not show an analgesic effect when tested by analgesimetry, but recent pharmacokinetic analysis suggests that this dose may not achieve effective plasma levels, and the mean plasma residence time is only 1 hour (J. Paul-Murphy, unpublished data). Higher doses of buprenorphine using different avian species may help to clarify if buprenorphine is an effective analgesic for birds. Clinical use of buprenorphine suggests it has an analgesic effect.

Fentanyl was evaluated using cockatoos and, although 0.02 mg/kg provided plasma levels similar to the concentration found to be analgesic in cats, it did not affect the withdrawal response of the cockatoos.[30] A ten-fold increase in the dosage of fentanyl (0.2 mg/kg SQ) did produce an analgesic response, but many birds were hyperactive for the first 15 to 30 minutes after receiving the high dose.[30] The duration of analgesic effect from fentanyl has not been established, but 0.02 mg/kg given IM to cockatoos maintained a plasma concentration considered analgesic in people for 2 hours.

Butorphanol (1-3 mg/kg IM) is the current recommendation for opioid analgesia in parrots. Butorphanol is a mixed agonist-antagonist with primarily kappa agonist action. This supports the one study finding of a high percentage of kappa receptors in the forebrain of pigeons. The dosage of butorphanol for effective analgesia needs to be balanced with sedation and respiratory depression, which may vary with other avian species and need further evaluation.

NSAIDs

Non-steroidal anti-inflammatory drugs (NSAIDs) are among the most commonly used classes of drugs in small animal medicine. NSAIDs interfere with eicosanoid synthesis by the inhibition of cyclooxygenase (COX) enzymes. COX enzymes initiate a cascade of reactions that results in polyunsaturated acids being converted to eicosanoids such as prostaglandins and thromboxane.[34] These are released at sites of tissue injury and cause inflammation and sensitization of nerve endings.

Reduction of prostaglandins and thromboxanes decreases inflammation at the site of injury and also has a modulating effect within the CNS. The physiological mechanisms involving prostaglandins, their role in inflammation and their ability to sensitize sensory neurons to physical or chemical stimuli in birds are similar to those in mammals.[29] The COX-1 enzyme is part of normal cell composition in numerous tissue types and the

concentrations are fairly stable, keeping prostaglandin levels at homeostatic levels. In mammals, COX-2 concentrations increase when stimulated at sites of inflammation, but both COX-1 and COX-2 are expressed in the CNS and participate in spinal pain transmission. The relative expression of COX-1 and COX-2 enzymes varies among species, and both enzymes are important in avian pain at the site of peripheral inflammation as well as in spinal pain transmission. However, more information is needed to differentiate their physiological effects in avian species.

NSAIDs can be used to relieve musculoskeletal and visceral pain, acute pain (trauma or surgical) and chronic pain such as osteoarthritis. The most common NSAIDs used in avian medicine are piroxicam, carprofen, ketoprofen and meloxicam. These compounds vary in their structure, but all inhibit COX enzymes. Generally, the current NSAIDs are safe drugs but should be monitored and evaluated in each individual patient. Increased monitoring may be indicated with high-risk patients: establishing a base-line set of hepatic enzymes and uric acid prior to NSAID administration and reevaluating these parameters at fixed intervals (see Chapter 16, Evaluating and Treating the Kidneys). NSAIDs should not be used if there is any indication of renal impairment, hepatic dysfunction or severe hypovolemia or if gastric ulceration is present. Only one NSAID should be used at a time, but in cases of chronic pain, frequently review the response to therapy and change to a different formulation NSAID if response is poor or diminishing. For treatment of mild to moderate chronic pain, NSAIDs can be given on an as-needed basis. The dose and frequency of administration has not been determined for any clinical NSAID use in birds. Ketoprofen (5 mg/kg IM) administered to ducks anesthetized with isoflurane had an analgesic effect 30 to 70 minutes after administration.[25] Carprofen given to rapidly growing chickens with chronic lameness improved their ability to walk and navigate a maze.[28] Carprofen (1 mg/kg SQ) given to chickens raised their threshold to pressure pain for at least 90 minutes.[8] Peak plasma levels occurred at 1 to 2 hours after SQ administration of carprofen to chickens.[28] Plasma levels do not provide sufficient information to indicate physiological activity of the NSAID because NSAIDs tend to accumulate in areas of inflammation. In mallard ducks, 5 mg/kg flunixin and 5 mg/kg ketoprofen suppressed thromboxane B_2 levels for 12 hours, indicating a prolonged physiological effect.[26]

MULTIMODAL THERAPY

Information specific to birds is minimal, and many current therapies for birds are extrapolated from species in a different class of vertebrates. Some drugs are handled similarly across species lines and other drugs may have very different mechanisms of action; they may be effective in one species and range from ineffective to toxic in another species.

Combining analgesics that act by different mechanisms can maximize the analgesic effect. Administration of two or more analgesics frequently produces a synergistic effect, such as a local anesthetic at the surgical incision or site of recent trauma, a NSAID plus an opioid. This combination of drugs usually allows the dosage of each drug to be reduced, thereby reducing the side effects. Additionally, opioids such as butorphanol will reduce the concentration of inhalation anesthetic needed for a surgical plane. Adjunctive drugs, such as tranquilizers used in conjunction with analgesic drugs, can potentiate the analgesic effects by reducing anxiety. The most common sedatives used in avian medicine are the benzodiazepines, which calm the bird and may reduce anxiety.

CHRONIC PAIN

Treatment of chronic pain in birds opens more questions than there are answers. How to treat chronic pain in a non-verbal patient such as the bird is the ultimate challenge. It is difficult to evaluate response to treatment when the condition itself may be progressive, such as chronic degenerative joint disease or neoplasia. Response to analgesic therapy is based on evaluation of a set of behaviors particular for each individual bird.

NSAIDs are the first course of therapy for chronic disorders. Carprofen is the current drug of choice because of its widespread use and low incidence of reported toxicities. Carprofen is an orally administered drug and can be initiated at the low-end dosage and monitored for response to treatment. As pain gradually increases over time, the dosage of carprofen can be increased, and monitoring the renal and hepatic serum values every 2 to 4 months is recommended, especially in an older bird. If pain recurs over several months of treatment, the next set of options includes changing to another NSAID, such as meloxicam or piroxicam. There is a risk when switching NSAIDs that the side effects can be potentiated, so a no-drug period of 7 to 10 days is recommended. Piroxicam may have synergistic action with anticancer drugs and also is an effective NSAID for degenerative joint disease in birds. Piroxicam is noted for renal toxicity and gastric ulceration in mammals, but its long-term use (months of treatment) in cranes with chronic degenerative joint disease has not caused clinical problems. If pain persists or increases, especially with neoplasia, opioid therapy may be indicated. Unfortunately, most parenteral forms of opioids reach "effective" plasma levels rapidly, but these levels are maintained for only a few hours. No information is available regarding oral opioids in birds, but in mammals much higher dosages are required for oral administration to reach effective plasma levels.

References and Suggested Reading

1. Beynon PH, Forbes NA, Harcourt-Brown NH: BSAVA Manual of Raptors, Pigeons and Waterfowl. Ames, Iowa State Univ Press, 1996.
2. Merskey H, Bogduk N (eds): Classification of Chronic Pain, 2nd ed. IASP Task Force on Taxonomy. Seattle, IASP Press, 1994, pp 209-214.
3. Clubb SL: Round table discussion: Pain management in clinical practice. J Avian Med Surg 12(4):276-278, 1998.
4. Concannon KT, Dodam JR, Hellyer PW: Influence of a mu and kappa opioid agonist on isoflurane minimal anesthetic concentration in chickens. Am J Vet Res 56:806-812, 1996.
5. Curro TG: Evaluation of the isoflurane-sparing effects of butorphanol and flunixin in Psittaciformes. Proc Assoc Avian Vet, 1993, pp 17-19.
6. Curro TG, Brunson D, Paul-Murphy J: Determination of the ED_{50} of isoflurane and evaluation of the analgesic properties of butorphanol in cockatoos (*Cacatua* spp.). Vet Surg 23:429-433, 1994.
7. Danbury TC, Hudson AL, Waterman-Pearson AE: Saturation binding of υ, γ, and κ opioid ligands in chicken brains. Arch Pharmacol 358(Suppl 35):105, 1998.
8. Danbury TC, et al: Self-selection of the analgesic drug carprofen by lame broiler chickens. Vet Rec 146(11):307-311, 2000.
9. Flecknell PA, Roughan JV, Stewart R: Use of oral buprenorphine (buprenorphine jello) for postoperative analgesia in rats: A clinical trial. Lab Anim 33(2):169-74, 1999.
10. Gentle MJ: Pain in birds. Animal Welfare 1:235-247, 1992.
11. Gentle MJ, Hill FL: Oral lesions in the chicken: Behavioral responses following nociceptive stimulation. Physiol Behav 40(6):781-7833. 1987.
12. Gentle MJ, Hunter LN: Physiological and behavioural responses associated with feather removal in *Gallus gallus* var. *domesticus*. Res Vet Sci 50(1):95-101, 1991.
13. Gentle MJ, Tilston VL: Reduction in peripheral inflammation by changes in attention. Physiol Behav 66(2):289-292, 1999.
14. Gentle MJ, Jones RB, Woolley SC: Physiological changes during tonic immobility in *Gallus gallus* var. *domesticus*. Physiol Behav 46(5):843-847, 1989.
15. Gentle MJ, Hunter LN, Waddington D: The onset of pain-related behaviors following partial beak amputation in the chicken. Neurosci Lett 128:113-116, 1991.
16. Glatz PC, et al: Analgesia therapy of beak-trimmed chickens. Aust Vet J 69(1):18, 1992.
17. Graham J, et al: Pharmacokinetics of ketoprofen in adult Japanese quail (*Coturnix japonica*). Proc Assoc Avian Vet, 2001, pp 19-21.
18. Hocking PM, et al: Evaluation of a protocol for determining the effectiveness of pretreatment with local analgesics for reducing experimentally induced articular pain in the domestic fowl. Res Vet Sci 63(3):263-267, 1997.
19. Jenkins JR: Postoperative care of the avian patient. Sem Avian Exot Pet Med 2:97-102, 1993.
20. Johnson-Delany CA: Exotic Companion Medicine Handbook for Veterinarians. Lake Worth, FL, Zoological Education Network, 2000.
21. Leite-Panissi CR, et al: Endogenous opiate analgesia induced by tonic immobility in guinea pigs. Braz J Med Biol Res 34(2):245-250, 2001.
22. Ludders JW, et al: Fecal corticosterone reflects serum corticosterone in Florida sandhill cranes (*Grus canadensis pratensis*). J Wildl Dis 37(3):646-652, 2001.
23. Paul-Murphy J, et al. The need for a cross-species approach to the study of pain in animals. Mar 1;224(5):692-697, 2004.
24. Lumeij JT, et al: Action of ACTH upon plasma corticosterone concentrations in racing pigeons. Avian Pathol 1:199-204, 1987.
25. Machin KL, Livingston A: Assessment of the analgesic effects of ketoprofen in ducks anesthetized with isoflurane. Am J Vet Res 63(6):821-826, 2002.
26. Machin KL, et al: Pharmacodynamics of flunixin and ketoprofen in mallard ducks. J Zoo Wildl Med 32:22-29, 2001.
27. Mansour A, et al: Anatomy of CNS opioid receptors. TINS 11(7):308-314, 1988.
28. McGeown D, et al: Effect of carprofen on lameness in broiler chickens. Vet Rec 144:668-671, 1999.
29. Nicol GD, Klingberg DK, Vasko MR: Prostaglandin E2 increases calcium conductance and stimulates release of substance P in avian sensory neurons. J Neurosci 12:1917-1927, 1992.
30. Paul-Murphy J, Ludders J: Avian analgesia. Exot Anim Prac 4(1): 35-45, 2001.
31. Paul-Murphy JR, Brunson DB, Miletic V: A technique for evaluating analgesia in conscious perching birds. Am J Vet Res 60(10):1213-1217, 1999.
32. Paul-Murphy JR, Brunson DB, Miletic V: Analgesic effects of butorphanol and buprenorphine in conscious African grey parrots (*Psittacus erithacus erithacus* and *Psittacus erithacus timneh*). Am J Vet Res 60(10):1218-1221, 1999.
33. Roughan JV, Flecknell PA: Effects of surgery and analgesic administration on spontaneous behavior in singly housed rats. Res Vet Sci 69:283, 2000.
34. Vane JR, Bakhle YS, Blotting RM: Cyclooxygenases 1 and 2. Annu Rev Pharmacol Toxicol 38:97-120, 1998.
35. Woolf CJ: A new strategy for the treatment of inflammatory pain: Prevention or elimination of central sensitization. Drugs 47(Suppl 5):1-9, Discussion:46-47, 1994.
36. Woolf CJ, Chong MS: Preemptive analgesia: Treating postoperative pain by preventing the establishment of central sensitization. Anesth Analg 77(2):362-379, 1993.
37. Woolley SC, Gentle MJ: Physiological and behavioral responses in the hen (*Gallus domesticus*) to nociceptive stimulation. Comp Biochem Physiol 88A(1):27-31, 1987.

CHAPTER
9

Therapeutic Agents

KEATH L. MARX, DVM

Editors' Disclaimer:

Every attempt has been made to ensure accurate citation of dosages and references. However, the reader must assume ultimate responsibility for the use of any medication. Indications and usage listed are as per the reference(s) cited, and may not bear pharmacodynamic data verifying efficacy or safety.

Dosages that vary significantly (by a factor of 50x or greater) from other empirical or pharmacokinetic studies may be omitted from the table, but may be found in the references.

No claims to efficacy or safety are intended or implied.

See abbreviations key at the end.

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Acepromazine Maleate	Avian	0.5-1	IM	QD	E	53, 704	Add ketamine
Acepromazine Maleate	Cassowary, Double-wattled	0.63	IM	PRN	G	447	Add etorphine
Acepromazine Maleate	Hawk	2.0	NL	PRN	D	1401	Add ketamine
Acepromazine Maleate	Ostrich	0.16-0.19	IM	PRN	B	447	Add etorphine
Acepromazine Maleate	Ostrich	25 MG TD	NL	PRN	D	1401	Add etorphine + xylazine
Acepromazine Maleate	Owl	2.0	NL	PRN	D	1401	Add ketamine
Acepromazine Maleate	Raptor	2.2	IM	PRN	E	1386	Add ketamine
Acepromazine Maleate	Ratite	0.25-0.5	IM	PRN	G	418	
Acepromazine Maleate	Ratite	0.1-0.2	IV	PRN	G	418	
Acetylcysteine	Amazon, Red Lored	22 mg/ml	Nebulize	QD	G	700	10% concentration
Activated Charcoal	Avian	52	PO	Once	E	1526	Give after first dose of fluids for oiled birds
Activated Charcoal	Avian	2-8 g/kg	PO	NL	E	1554, 1745	
Activated Charcoal	Psittacine	2-8 g/kg	PO	PRN	E	1240	Absorb ingested toxins
Activated Charcoal	Raptor	2-10 g/kg	PO	PRN	E	1400	Antidiarrheal, absorbs toxins
Acyclovir	Avian	1 g/L water	Drink	NL	A	435	
Acyclovir	Avian	400 mg/kg food	Feed	Once	A	435	
Acyclovir	Avian	80	PO	TID	E	1470, 1473	For herpesvirus, (also ref # 1560, 1650)
Acyclovir	Avian	25	IM	NL	G	435	
Acyclovir	Avian	40	IM	TID	G	1306	For Pacheco's disease
Acyclovir	Avian	80	PO	TID	G	1306	For Pacheco's disease
Acyclovir	Avian	330	Gavage	BID	G	1352	
Acyclovir	Avian	0.4 g/L	Drink	QD	G	1352	
Acyclovir	Chicken	10	IM	QD	B	435	
Acyclovir	Macaw	80	PO	TID	A	435	
Acyclovir	Parakeet, Quaker	40	IM	TID	A	1060	
Acyclovir	Parakeet, Quaker	1 g/L feed	Feed	QD	A	1060	Change feed BID
Acyclovir	Psittacine	0.4 g/L water	Drink	NL	E	763	
Acyclovir	Psittacine	80	PO	TID	E	1365	For Pacheco's virus
Acyclovir	Raptor (Peregrine falcon)	80	PO	TID	G		
Acyclovir	Raptor	333	PO	BID	G	94	Juvenile dosage
Albendazole	Avian	100	PO	Once	E	1526	For nematodes and Capillaria
Albendazole	Avian	50	PO	QD	E	1526	For nematodes and Capillaria
Albendazole	Bird, Aquatic	100	PO	Once	E	1503	For nematodes and Capillaria
Albendazole	Bird, Aquatic	50	PO	QD	E	1503	For nematodes and Capillaria

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Albendazole	Crane	20	PO	QW	E	1361	For trematodes
Albendazole	Emu	10	NL	NL	G	1471	For fascioliasis
Albendazole	Penguin	50	PO	QD	E	1478	For nematodes and flukes
Albendazole	Penguin	100	PO	Once	E	1478	For nematodes and flukes
Albendazole	Ratite	0.264	PO	BID	E	1240	Repeat in 2 weeks, antiprotozoal
Alfaxalone + Alfadolone	Avian	10-36	IM-IV-SC	PRN	E	704	
Alfaxalone + Alfadolone	Avian	5-10	IV	PRN	E	1181	Short duration, may be apnea
Alfaxalone + Alfadolone	Avian	36	IM-IP	PRN	E	1181	Short duration
Alfaxalone + Alfadolone	Buzzard (Augur, Lizzard)	10	IV	PRN	B	1610	
Alfaxalone + Alfadolone	Chicken	10	IV	PRN	B	1610	
Alfaxalone + Alfadolone	Crane	4-8	IV	PRN	E	1240	May cause transient apnea, surgical anesthesia for 8 to 10 min
Alfaxalone + Alfadolone	Eagle (African Fish, Hawk, Tawny) and Falcons	10	IV	PRN	B	1610	
Alfaxalone + Alfadolone	Flamingo	4-8	IV	PRN	E	1240	
Alfaxalone + Alfadolone	Goshawk, African	10	IV	PRN	B	1610	
Alfaxalone + Alfadolone	Owl, Great-horned	6-12	IV	PRN	B	1174	
Alfaxalone + Alfadolone	Pelican	5.4	IV	PRN	G	595	
Alfaxalone + Alfadolone	Pigeon	5-7	IM-IV	PRN	G	232	
Alfaxalone + Alfadolone	Psittacine	5-10	IM-IV	PRN	E	1240	
Alfaxalone + Alfadolone	Raptor	10	IV	PRN	B	1568	For wild injured raptors anesthesia induction
Allopurinol	Avian	10-15	IM, PO	QD	G	58, 704	
Allopurinol	Budgerigar	0.33 g/L	Drink	NL	G	111	
Allopurinol	Budgerigar	0.67 g/L	Drink	NL	E	763	For gout
Allopurinol	Psittacine	10	PO	QD	B	1035	For hyperuricemia
Allopurinol	Psittacine	0.33 g/L	Drink	NL	E	1240	To treat gout
Allopurinol	Psittacine	10-30	PO	BID	E	1756	
Aloe Vera	Avian	16 ml/L	Drink	QD	G	1682	
Aloe Vera	Avian	30 ml/L	Drink	QD	G	111	
Amantadine HCl	Avian	0.001 g/L	Drink	NL	E	1650	For orthomyxoviruses
Amantadine HCl	Avian	25	PO	NL	E	1650	For orthomyxoviruses
Amantadine HCl	Poultry	2	PO	NL	G	435	
Amantadine HCl	Turkey	10	PO	QD	G	435	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Amikacin Sulfate	Amazon, Blue-fronted	10-15	IM	BID-TID	A	697	
Amikacin Sulfate	Amazon, Orange-winged	13-20	IM/IV	BID	A	736	
Amikacin Sulfate	Avian	10-15	IM-IV-SC	BID-TID	A	1473, 111	
Amikacin Sulfate	Bird, Aquatic	18-20	IM	BID	E	1478, 1559	
Amikacin Sulfate	Chicken	20	IM	TID	A	1058	For hens
Amikacin Sulfate	Cockatiel	15-20	IM	BID-TID	A	32, 697, 879	
Amikacin Sulfate	Cockatoo, Goffin	15-20	IM	BID-TID	A	697	
Amikacin Sulfate	Crane	10	IM-SC	BID	E	629	
Amikacin Sulfate	Fowl, Domestic	10-20	IM	QD-TID	A	1631	For salmonellosis
Amikacin Sulfate	Gull	15	IM	BID	E	1357	
Amikacin Sulfate	Ostrich	7.6	IM	TID	A	747	
Amikacin Sulfate	Parrot, Grey	10-20	IM	BID-TID	A	112, 692	
Amikacin Sulfate	Penguin, African	15	IM	BID	G	1353	Monitor hydration
Amikacin Sulfate	Pigeon	15-20	IM-IV	BID	G	590	
Amikacin Sulfate	Psittacine	15-40	IM	QD-BID	E	565	
Amikacin Sulfate	Raptor	15-20	IM	QD	G	1314	
Amikacin Sulfate	Ratite	7.6-11	IM	BID	G	1308	
Amikacin Sulfate	Stork, Saddle-billed	10	IM	BID	G	1645	
Aminoloid	Raptor	0.25-0.75	IM	q2w	E	111	Induce molting
Aminonitrothiazole	Raptor	20-40	PO	NL	E	1463	For trichomoniasis
Aminopentamide Sulfate	Avian	0.05	IM-SC	BID	E	111, 1240. 1473	Control vomition
Aminophylline	Avian	0.5	Nebulize	BID-TID	G	1199	Add acetylcysteine, antibiotic, 5 ml NaCl
Aminophylline	Avian	10	IV-PO	q3h	E	1151	
Aminopromazine	Avian	6.75	IM	NL	F	1209	
Aminothiazole	Pigeon	5 ml/L	Drink	QD	E	1240	
Amitriptyline HCl	Psittacine	1-5	PO	B-QID	D	1446, 111, 1240	For behavioral feather picking
Ammonia Solution	Avian	10 ml/L	Topical	PRN	G	111	Add aloe vera + 4 drops sulfonate detergent, control skin pruritis
Amobarbital Sodium	Anseriformes	2	PO	PRN	G	4	Soak food in solution
Amobarbital Sodium	Duck, Mallard	3.6 g/L feed	Feed	Once	G	1398	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Amoxicillin	Avian	150-175	PO	QD-BID	E	111	
Amoxicillin	Avian	250	IM	QD	E	704	Long-lasting preparation
Amoxicillin	Avian	0.2-0.4 g/L	Drink	QD	E	704	Soft feed
Amoxicillin	Avian	167	PO	QD-BID	E	741	
Amoxicillin	Avian	150	IM-PO	NL	E	924	Broad spectrum
Amoxicillin	Avian	100	PO	BID	E	1431	
Amoxicillin	Avian	150-175	PO	QD-BID	E	1473	
Amoxicillin	Avian	100-150	SC	QD	E	1526	Broad spectrum
Amoxicillin	Avian	100-150	IM	NL	G	1618	For open fractures
Amoxicillin	Bustard	100	IM-SC	BID	E	1240	
Amoxicillin	Canary	0.3 g/L	Drink	NL	A	1139	
Amoxicillin	Canary	0.5 g/kg feed	Feed	Once	A	1139	
Amoxicillin	Chicken	16	PO	QD	B	1006	
Amoxicillin	Currawong, Pied	25-50	PO	BID	G	1609	Post-surgical
Amoxicillin	Duck	20	Drink	QD	C	705	
Amoxicillin	Falcon	100	Parenteral	q2d	G	1128	
Amoxicillin	Galliformes	0.17 g/L	Drink	QD	E	704	
Amoxicillin	Goshawk	10	PO	BID	G	127	Precede with ticarcillin
Amoxicillin	Gull	100	PO	QD-BID	E	1357	
Amoxicillin	Penguin, African	200	PO	TID	G	1353	
Amoxicillin	Pigeon	20-90	PO	QD-BID	A	493	Use capsules
Amoxicillin	Pigeon	100	PO	QD-BID	A	565	Gram negative bacteria
Amoxicillin	Pigeon	50	IM	QD-BID	A	565	Gram positive bacteria
Amoxicillin	Pigeon	18-91	PO	QD-BID	A	733	Use high dosage for Enterobacteriaceae
Amoxicillin	Pigeon	1.5 g/L	Drink	NL	A	800	For *Streptococcus bovi* s
Amoxicillin	Pigeon	150	IV-PO	NL	A	800	For *Streptococcus bovis*
Amoxicillin	Pigeon	0.2 g/L	Drink	NL	A	993	
Amoxicillin	Pigeon	75-100	PO	BID	A	1066	
Amoxicillin	Pigeon	20	Drink	QD	C	704	
Amoxicillin	Pigeon	25-50	PO	QD	G	47	
Amoxicillin	Poultry	15-20	Drink	QD	C	705	
Amoxicillin	Poultry	55-110	PO	BID-TID	G	585	
Amoxicillin	Psittacine	150-175	PO	QD-BID	E	565	
Amoxicillin	Psittacine	250	IM	QD	E	1140	
Amoxicillin	Psittacine	100	SC	NL	G	1613	
Amoxicillin	Raptor	150	IM-PO	BID	E	1400	Bacterial infections
Amoxicillin	Raptor	50	IM	BID	E	1612	
Amoxicillin	Ratite	15-22	PO	BID	G	1308	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Amoxicillin + Tylosin	Avian	10 g/L	Drink	QD	E	1492	For flock treatment of bacterial and mycoplasmal infections
Amoxicillin + Tylosin	Avian	3000	PO	QD	E	1492	
Amoxicillin + Tylosin	Avian	10 g/L	Drink	QD	E	1650	
Amoxicillin + Tylosin	Avian	3000	PO	QD	E	1650	
Amoxicillin Depot	Bustard	100-250	IM-SC	q3-7d	A	1127, 1240	
Amoxicillin Depot	Pigeon	150	IM	q2d	A	493	Long-lasting formulation
Amoxicillin Depot	Pigeon	150-200	SC	QD	A	991	Long-lasting oil based susp.
Amoxicillin Sodium + Clavulanate Potassium	Amazon Parrot	125	NL	TID	A	1491	
Amoxicillin Sodium + Clavulanate Potassium	Avian	125	PO	QID	E	704	Dosage based on amoxicillin
Amoxicillin Sodium + Clavulanate Potassium	Avian	100	PO, IM	BID	E	1431	Useful in young birds
Amoxicillin Sodium + Clavulanate Potassium	Avian	50	IM	BID-QID	E	1470	For bacterial infection with liver disease
Amoxicillin Sodium + Clavulanate Potassium	Avian	150-175	IM-PO	BID	E	1474	For neonatal septicemia
Amoxicillin Sodium + Clavulanate Potassium	Bird, Aquatic	125-150	IM, PO	BID	E	1478	Pre- and post-operative
Amoxicillin Sodium + Clavulanate Potassium	Canary	125	Gavage	BID	G	1139	For GI infection
Amoxicillin Sodium + Clavulanate Potassium	Chicken	0.5 g/L	Drink	NL	A	801	
Amoxicillin Sodium + Clavulanate Potassium	Hawk, Red-tailed	50	PO	BID	G	1626	Post-operative
Amoxicillin Sodium + Clavulanate Potassium	Owl, Great-horned	125	PO	BID	G	173	
Amoxicillin Sodium + Clavulanate Potassium	Passerine	200	NL	TID	G	1489	For frostbite associated skin infection
Amoxicillin Sodium + Clavulanate Potassium	Pigeon	125	Gavage	BID	B	751	For localized gut infection
Amoxicillin Sodium + Clavulanate Potassium	Psittacine	100	PO	BID	D	1446	For staphylococcal dermatitis
Amoxicillin Sodium + Clavulanate Potassium	Raptor	150	PO, IM	BID	E	1400, 1433	Can be nephrotoxic
Amoxicillin Sodium + Clavulanate Potassium	Raptor	125	PO	BID	G	234	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Amoxicillin Sodium + Clavulanate Potassium	Ratite	11-15	IV-PO	TID	G	1308	
Amphotericin B	Amazon, Yellow-naped	50-75 mg TD	IT	QD	G	740	
Amphotericin B	Avian	1	IT	TID	E	625	Add flucytosine + IV amphotericin B, aspergillosis therapy
Amphotericin B	Avian	1.5	IV	TID	E	625, 674	See above
Amphotericin B	Avian	100	PO	BID	E	1492	For macrorhabdosis (formerly megabacteria or avian gastric yeast)
Amphotericin B	Avian	0.83g/ L	Nebulize	BID	E	1526	For aspergillosis therapy. Must use sterile water - NaCl inactivates Amphotericin
Amphotericin B	Avian	1	IP	QD	G	861	
Amphotericin B	Bird, Aquatic	100-200	PO	TID-QID	E	1503	For GI candidiasis
Amphotericin B	Bird, Aquatic	1.5	IV-SC	QD	E	1503	For aspergillosis therapy
Amphotericin B	Budgerigar	109	PO	BID	B	589	For macrorhabdosis (formerly megabacteria or avian gastric yeast)
Amphotericin B	Crane	0.33-0.67 g/L	Nebulize	TID	G	1361	
Amphotericin B	Gull	1	IT	BID	E	1357	
Amphotericin B	Gull	1.5	IV	TID	E	1357	
Amphotericin B	Psittacine	1.5	IV	BID-TID	E	111, 745	
Amphotericin B	Psittacine	1	IT	BID-TID	E	111, 741, 1309	
Amphotericin B	Raptor	1.5	IV	TID	B	1178	Add IT amphotericin B + flucytosine + rifampin for aspergillosis
Ampicillin	Amazon Parrot	150-200	PO	BID-TID	A	567	May give combination of both oral and parenteral
Ampicillin	Anseriformes	100	IM	q4h	E	1318, 1358	
Ampicillin	Avian	0.25g/kg	Feed	QD	E	741	
Ampicillin	Avian	1.041 g/L	Drink	QD	E	741	
Ampicillin	Canary	1-2 g/L	Drink	QD	G	49	
Ampicillin	Canary	2-3 g/kg food	Feed	QD	G	49	In soft food
Ampicillin	Crane	15-20	IM-SC	BID	A	596	
Ampicillin	Crane, Greater Sand Hill	20	IM	BID	A	847	
Ampicillin	Emu	20	IM	BID	A	847	
Ampicillin	Falcon	100	IM-PO	BID	E	1027	
Ampicillin	Parrot, Blue-naped	150-200	PO	BID-TID	A	567	May give combination of both oral and parenteral
Ampicillin	Pigeon	25-120	PO	QD-BID	A	493	Use capsules
Ampicillin	Pigeon	100	IM	q2d	A	493	Long-lasting formulation

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Ampicillin	Poultry	55-110	Parenteral	BID-TID	G	585	
Ampicillin	Psittacine	100	IM	q4h	E	111	
Ampicillin	Psittacine	100-200	PO	QID	E	1240	Poorly absorbed
Ampicillin	Raptor	100-250	IM	NL	E	1463	
Ampicillin Sodium	Amazon Parrot	50-100	IM	q4-8h	A	567	May give combination of both oral and parenteral
Ampicillin Sodium	Pigeon	150	IM	QD-BID	A	493	
Ampicillin Sodium	Pigeon	773	IM	QD-BID	A	733	For *Enterobacteriaceae*
Ampicillin Sodium	Pigeon	155	IM	QD-BID	A	733	
Ampicillin Sodium	Ratite	0.1 - 0.2 g/L	Drink	NL	G	418	Clostridial treatment
Amprolium	Avian	0.048-0.096g/L	Drink	QD	E	111	
Amprolium	Avian	0.08 g/L	Drink	QD	E	1492	Coccidiostatic, overdose reversed by administration of vitamin B complex
Amprolium	Chicken	40-250 mg/kg food	Feed	QD	E	564	No slaughter withdrawal
Amprolium	Crane	0.06 g/L	Drink	QD	E	1361	For coccidiosis
Amprolium	Pigeon	0.2 g/L	Drink	QD	G	232	
Amprolium	Poultry	1.25 cc/L	Drink	QD	G	585	Use 20% powder formulation
Amprolium	Psittacine	0.25 g/L	Drink	QD	E	1365	For sarcocystosis
Apramycin	Chicken	0.5 g/L	Drink	QD	B	881	For colibacillosis
Arecoline Hydrobromide	Pelican, Brown	1-1.6	PO	QH	B	1089	Give thiabendazole previous day
Arsanilic Acid	Poultry	0.1 g/kg food	Feed	QD	E	564	Nutritional use, 5-day slaughter withdrawal
Ascorbic Acid	Avian	20-40	IM	QD-QW	E	1473	
Ascorbic Acid	Avian	20-50	IM-PO	NL	E	924	For viral infection, liver disease, stress
Ascorbic Acid	Avian	20-40	IM	QD-QW	E	1473	
Ascorbic Acid	Ostrich	20-50 mg TD	IM	q2d	G	401	For chicks
Ascorbic Acid	Raptor	250	PO	QD	E	1359	Antioxidant therapy, administer during lead chelation therapy
Asparaginase	Avian	0.4 KIU/kg	IM	QW	E	1470	
Asparaginase	Owl, Great-horned	1.7 KIU/kg	SC	Once	F	125	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Aspirin	Amazon, Red Lored	0.5-1	PO	QD-BID	G	1756	Add fatty acid omega 6:omega 3 < 6:1 ratio, use until renal histology normalizes for renal disease
Aspirin	Amazon, Yellow-naped	0.5-1	PO	BID	G	1033	
Aspirin	Avian	5	PO	TID-QID	D	1533	
Aspirin	Avian	1.2 g/L	Drink	QD	E	1650	
Aspirin	Avian	5	PO	TID	G	1341, 1671	
Aspirin	Psittacine	0.5-1	PO	QD-BID	E	1756	
Aspirin	Stork, Saddle-billed	1	PO	QD	G	1645	
Astragalus	Avian	10 drops/kg	PO	TID	E	1205	Chronic GI problems and infections
Astragalus	Avian	33-66 drops/kg	PO	QD	E	1435	Immune stimulant
Atipamezole							
Atipamezole	Anseriformes	0.25	IV	PRN	D	1503	Add flumazenil to reverse ketamine + medetomidine + midazolam
Atipamezole	Avian	0.25-0.38	IM	PRN	E	1181	Reverse ketamine + xylazine or ketamine + medetomidine
Atipamezole	Duck, Mallard	0.25 mg TD	IV	PRN	B	764	Add flumazenil
Atipamezole	Pigeon	0.5	IM	PRN	B	1588	Reverse medetomidine
Atipamezole	Psittacine	0.25-0.38	IM	PRN	E	1240	Reverse xylazine or medetomidine
Atropine	Anseriformes	0.1	IM-IV	q3-4h	D	1150	For anticholinesterase poisoning
Atropine	Avian	0.04-0.1	IM-SC	Once	G	1309	Preanesthetic
Atropine	Avian	0.1-0.2	IM-SC	q4h	E	1151	For cholinergic toxin
Atropine	Avian	0.2-0.5	IV	q3-4h	E	1554	For anticholinesterase toxicity
Atropine	Avian	0.01-0.02	Parenteral	q3-4h	E	1650	For organophosphate toxicity
Atropine	Avian	0.1-0.2	IM-SC	PRN	G	1309	For organophosphate poisoning
Atropine	Canary	0.2	Parenteral	q4h	G	1714	For convulsions possibly caused by organophosphate toxicity
Atropine	Pigeon	0.27	IM-IV	q4-8h	G	590	
Atropine	Psittacine	0.05	IM-SC	QH	E	1240	For acetylcholinesterase poisoning
Atropine	Psittacine	0.5	IM-SC	NL	E	1688	
Atropine	Raptor	0.5	IM-IV	PRN	G	61	[May thicken secretions and increase risk of airway obstruction - KLM]
Atropine	Raptor	0.1	IM-IV	q3-4h	E	1240, 1400	For acetylcholinesterase poisoning
Atropine	Raptor	0.02-0.05	IM-IV	PRN	E	1359	For organophosphate toxicity
Azamethiphos	Poultry	5-20 g/L	Topical	NL	E	1479	Apply to animal facilities, insect spray

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Azaperone	Ostrich	4-6	IM	PRN	B	522	Add metomidate, chick dosage
Azaperone	Ostrich	3.3-6.6	IM	PRN	G	283	Add metomidate
Azaperone	Ratite	0.5-2	IM	Once	G	1226	Add ketamine + diazepam after 10 to 15 minutes, sedation
Azithromycin	Avian	50-80	PO	QD	G	702	For chlamydophilosis
Azithromycin	Avian	43	PO	QD	G	1154	Add ethambutol + rifabutin for mycobacteriosis
Azithromycin	Falcon	40	PO	QW	F	1157	For chlamydophilosis
Bach flower	Avian	1 drop	Drink	QID	G	912	For calming effect, also apply topically as spray
Bach flower	Avian	45-75 drops/L	PO	QID	G	912	See above
Bacitracin	Poultry	110-220 mg/kg feed	Feed	QD	E	564	Nutritional use, no slaughter withdrawal
Bacitracin	Ratite	0.4 g/L	Drink	NL	G	418	Clostridial therapy
Bambermycin	Chicken	3.3 mg/kg feed	Feed	Once	B	400	
Bambermycin	Ostrich	0.07 mg/kg feed	Feed	Once	C	283	Growth promotant
Barium Sulfate	Avian	50 ml/kg	PO	NL	E	1151	G.I. protectant and antiinflammatory
Barium Sulfate	Avian	10-20 ml/kg	PO	NL	E	1482	Mix 50:50 with hand-feeding formula for GI contrast rads
Benzyl Benzoate	Pigeon	25% solution	Topical	PRN	E	704	Apply to lesion
Benzyl Benzoate	Raptor	10% solution	Topical	QD	E	1612	For *Knemidocoptes*
Betamethasone	Avian	0.1	IM	NL	D, E, G	201, 1533, 1573	
Betamethasone	Bird, Aquatic	0.1	IM	NL	E	1503	
Betamethasone	Pigeon	1	IM	Once	E	704	Anaphylaxis
Biotin	Raptor	0.025	PO	QD	E	1068	During regrowth of claw
Biotin	Raptor	0.05	PO	QD	E	1240	Aid in beak and claw growth
Bismuth Subsalicylate	Avian	35	PO	BID	E	1240	May help remove ingested toxins
Bismuth Subsalicylate	Avian	35-88	PO	Once	E	1526	Enteric coating for oiled birds

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Brewers yeast	Anseriformes	75 g/kg feed	Feed	QD	E	1358	For niacin deficiency with leg deformities, weakness, poor growth
Bromhexine HCl	Avian	3-6	IM	NL	E	704, 1434	
Bromhexine HCl	Avian	1.5	IM	QD-BID	E	1526, 1617	Liquefy respiratory mucus
Bromhexine HCl	Raptor	1.5	IM	QD	E	1612	Mucolytic
Bunamidine HCl	Raptor	25	PO	NL	E	1463	Anthelmintic
Bupivacaine HCl	Parrot, Grey	2	IM	PRN	A	1339	Maintain plasma concentration for less than 2 hours
Buprenorphine HCl	Avian	0.01-0.05	IM-IV-SC	BID	E	1167	
Buprenorphine HCl	Pigeon	0.5	IM	q5h	B	1656	
Butorphanol Tartrate	Amazon, Hispaniolan	1-3	IM	NL	A	1339	
Butorphanol Tartrate	Cockatoo	1	NL	PRN	B	1678	Isoflurane sparing
Butorphanol Tartrate	Parrot, Grey	1-2	IM	NL	A	1339	Plasma levels maintained less than 2 hours at 2 mg/kg
Butorphanol Tartrate	Parrot, Grey	1	IM	NL	B	586, 1339	
Butorphanol Tartrate	Pigeon	4	IM	q3h	B	1656	
Butorphanol Tartrate	Psittacine	3-4	IV-PO	NL	E	111, 1249	
Butorphanol Tartrate	Psittacine	3-4	IV-PO	TID	E	1240	Also analgesic
Butorphanol Tartrate	Raptor	2-4	IM-SC	QD	E	1359	
Butorphanol Tartrate	Rhea	0.7	IM	PRN	G	1628	Add ketamine + medetomidine
Butorphanol Tartrate	Toucan, Toco	2	IM	Once	G	1379	Post-surgical pain
Calcitriol	Avian	0.000025 (0.025ug/kg)	Gavage	QD	G	1183	Add calcium source to gavage for hypocalcaemia
Calcium Borogluconate	Avian	200	IM-IV	QD	E	1431	Used during egg laying problems, often with oxytocin
Calcium Borogluconate	Avian	100-500	IM-SC	NL	E	1434	For egg binding and hypocalcemic tetany
Calcium Borogluconate	Goshawk	300	IV	NL	G	234	
Calcium Borogluconate	Pigeon	100-500	IM-SC	NL	E	1432	For egg binding and hypocalcemic tetany
Calcium Borogluconate	Psittacine	100-200	IM-IV	NL	E	1240	
Calcium Borogluconate	Raptor	100-500	IV-SC	Once	E	1240	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Calcium Borogluconate	Raptor	10-50	IV-SC	Once	E	1400	For egg binding or hypocalcaemia, give IV slowly
Calcium Carbonate	Raptor	10 g/kg food	Feed	Once	D	1612	For osteodystrophy
Calcium Glubionate	Avian	0.44 g/L	Drink	NL	E	1492	For egg binding and calcium deficiencies
Calcium Glubionate	Avian	150	PO	BID	G	54	
Calcium Glubionate + Calcium Lactobionate	Avian	363	PO	QD	E	1617	
Calcium Gluconate	Avian	100-300	IM-SC	NL	E	1480	Add phytonadione for bleeding syndrome
Calcium Gluconate	Avian	5-10	IM-SC	BID	E	1473	
Calcium Gluconate	Avian	50-100	IV	NL	E	1473	Slowly to effect
Calcium Gluconate	Avian	5-10	IM	NL	E	1474	
Calcium Gluconate	Avian	50-100	IV	NL	E	1474	Give slowly, follow with oxytocin or dinoprostone for egg binding, may add vitamin E + selenium
Calcium Gluconate	Avian	100-300	SC	NL	E	1481	
Calcium Glycerophosphate + Calcium Lactate	Avian	5-10	IM-SC	NL	E	1570	For egg binding
Calcium Glycerophosphate + Calcium Lactate	Avian	0.5-1	IM	QW	G	54	
Calcium Glycerophosphate + Calcium Lactate	Lory, Red	7	IM	NL	G	1621	Add oxytocin for egg binding
Calcium Glycerophosphate + Calcium Lactate	Parrot, Grey	5-10	IM-SC	BID	G	88	Acute therapy
Cambendazole	Avian	60-100	PO	QD	G	57	Avoid during breeding season
Cambendazole	Pigeon	75	PO	QD	E	1051	For ascarids and capillaria
Caprylic Acid	Avian	270	PO	NL	E	111	Adjunct to aspergillosis therapy
Carbaryl	Avian	N/A	Topical	NL	E	111	5% mixture, powder formulation

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Carbaryl	Avian	N/A	Topical	NL	E	1240	5% mixture, lightly dust feathers for ectoparasites
Carbenicillin Disodium	Avian	150	IM-PO	NL	E	924	Gram negative, *Pseudomonas*
Carbenicillin Disodium	Avian	100	IM	TID	G	55	
Carbenicillin Disodium	Avian	100-200	IM	BID	G	861	Freeze small portions indefinitely
Carbenicillin Disodium	Crane	100	IM-IV	BID-TID	E	629, 1361	
Carbenicillin Disodium	Owl, Great-horned	150	PO	BID	G	173	
Carbenicillin Disodium	Pigeon	100	IM	BID-TID	G	260	
Carbenicillin Disodium	Psittacine	100-200	IM	TID-QID	E	565	
Carbenicillin Disodium	Psittacine	100	IM	BID	E	763	
Carbenicillin Disodium	Raptor	100-200	IM	TID	E	1240	
Carbenicillin Disodium	Raptor	100	IT	QD	E	1240	For Gram negative bacteria, synergistic w/aminoglycosides
Carbenicillin Disodium	Raptor	100-200	IM	TID	E	1400	
Carbon Dioxide Gas	Avian	N/A	IH	NL	E	1564	Euthanasia
Carboplatin	Amazon, Yellow-naped	125 mg/m^3	IV	q2-3w	G	1033	Dilute with 5% dextrose
Carboplatin	Budgerigar	5	IV	QM	G	1259	Dilute 1:10 with sterile water
Carboxymethylcellulose Sodium	Avian	0.1% solution	Drink	QD	G	1457	Add amphotericin B, mix fresh daily, allow to stand overnight, dissolve drug in the AM
Carfentanil Citrate	Ostrich	3 mg TD	IM	PRN	B	521	Add xylazine
Carfentanil Citrate	Ostrich	0.024	NL	PRN	G	465	
Carnidazole	Avian	33	PO	Once	E	1526	For trichomoniasis, no food/water night before treatment plus no water 2 to 3 hours after treatment
Carnidazole	Avian	20-30	PO	Once	E	1554	For trichomoniasis and giardiasis
Carnidazole	Bird, Aquatic	33	PO	Once	E	1503	
Carnidazole	Bustard	20-25	PO	Once	E	1240	
Carnidazole	Dove	30	PO	NL	G	61	
Carnidazole	Falcon	25	PO	Once	B	1420	For Trichomoniasis, less effective than multiday dimetridazole
Carnidazole	Finch	20-30	PO	Once	E	1572	
Carnidazole	Pigeon	20-30	PO	Once	D	1221	For trichomoniasis
Carnidazole	Pigeon	200	PO	Once	E	111, 1473	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Carnidazole	Pigeon	12.5-25	PO	Once	E	1240	Use lower dosage for juveniles
Carnidazole	Pigeon	20-30	PO	QW-q2w	E	1364	
Carnidazole	Psittacine	20-30	PO	Once	E	1240	For trichomoniasis, hexamitiasis, histomoniasis, use lower dosage for juveniles
Carnidazole	Raptor	30	PO	QD	G	61	
Carprofen	Amazon, Orange-winged	4	IM	BID	G	1377	Post-surgical
Carprofen	Anseriformes	4	SC	QD	E	1190	
Carprofen	Anseriformes	5-10	IM	NL	G	1345	
Carprofen	Avian	1-2	IM-PO	BID	D	1533	
Carprofen	Avian	4	IM-IV-PO	NL	E	1167	
Carprofen	Avian	5-10	IM	QD	E	1431	Pre-operative painkiller
Carprofen	Bird, Aquatic	1-2	IM-PO	BID	E	1503	
Carprofen	Pigeon	5-10	IM	NL	G	1345	
Carprofen	Psittacine	2-10	IM-IV-SC	QD	E	1240	Post-surgical pain
Carprofen	Raptor	1-2	IM-IV-PO	BID	D	1400	
Carprofen	Raptor	2-10	IM-IV-SC	QD	E	1240	Post-surgical pain
Cefadroxil	Pigeon	100	PO	BID	G	260	
Cefazolin Sodium	Crane	25-30	IM-IV	TID	E	629	
Cefazolin Sodium	Poultry	11-55	NL	BID-TID	G	585	
Cefazolin Sodium	Raptor	100	IM	Once	E	1359	Perioperative, use amoxicillin/clavulanate post-operative
Cefotaxime Sodium	Amazon Parrot	75-100	IM	q4-8h	A	697	
Cefotaxime Sodium	Amazon, Blue-fronted	100	IM	TID	A	748	
Cefotaxime Sodium	Avian	75-100	IM-IV	TID-QID	A	1473	
Cefotaxime Sodium	Bird, Aquatic	100	IM	TID-QID	E	1478	For hock joint pressure sore infections
Cefotaxime Sodium	Crane	50-100	IM-SC	TID	E	629	
Cefotaxime Sodium	Hawk, Red-tailed	100	IM	NL	G	1626	Intra-operative
Cefotaxime Sodium	Pigeon	100	IM	BID-TID	G	260	
Cefotaxime Sodium	Psittacine	50-100	IM	TID	E	565	
Cefotaxime Sodium	Psittacine	50-100	SC	TID	E	632	Neonate dosage
Cefotaxime Sodium	Psittacine	100	IM	BID	E	763	Frozen reconstituted drugs lasts 12 weeks in freezer, 10 days in refrigerator
Cefotaxime Sodium	Raptor	100	IM-IV	Once	E	1359	Perioperative, use amoxicillin/clavulanate post-operative
Cefotaxime Sodium	Ratite	25	IM	TID	G	1308	For young birds

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Ceftazidime	Psittacine	75-200	IM-IV	BID-QID	E	1756	For bacterial nephritis
Ceftiofur Sodium	Amazon, Orange-winged	10	IM	BID-TID	A	788	
Ceftiofur Sodium	Avian	50-100	IM	QID	E	1240	
Ceftiofur Sodium	Chicken	0.16 mg TD	IM	QD	A	788	
Ceftiofur Sodium	Cockatiel	10	IM	q4h	A	788	
Ceftiofur Sodium	Psittacine	100	IM	TID	E	1756	For bacterial nephritis
Ceftiofur Sodium	Ratite	10-20	IM	BID	G	1308	
Ceftriaxone Sodium	Amazon Parrot	75-100	IM	q4-8h	A	697	
Ceftriaxone Sodium	Avian	75-100	IM-IV	TID-QID	E	111	
Celecoxib	Avian (Macaw)	10	PO	QD	G	1750	For proventricular dilatation disease symptoms
Cephalexin	Avian	35-50	PO	q4-6h	E	111	
Cephalexin	Avian	40-100	IM-PO	TID-QID	E	1240	For *E. coli* and *Proteus*
Cephalexin	Avian	35-50	PO	q4-6h	E	1473	
Cephalexin	Avian	35-50	PO	QID	E	1492	
Cephalexin	Bird, Aquatic	35-50	PO	QID	E	1503	
Cephalexin	Bustard	40-100	IM-PO	TID-QID	E	1240	For *E. coli* and *Proteus*
Cephalexin	Crane	35-50	PO	QID	A	596	
Cephalexin	Crane	100	IM	QID	A	697	
Cephalexin	Crane	100	PO	q4-6h	E	111	
Cephalexin	Crane	35-50	PO	QID	A	457, 847	
Cephalexin	Duck	35-50	PO	q2-3h	A	457	
Cephalexin	Duck	100	IM	BID-TID	A	697	
Cephalexin	Duck	35-50	PO	QID	A	847	
Cephalexin	Emu	35-50	PO	QID	A	418, 457, 847	
Cephalexin	Emu	100	IM	QID	A	697	
Cephalexin	Pigeon	35-50	PO	QID	A	847	
Cephalexin	Pigeon	100	PO	BID-TID	G	260	
Cephalexin	Poultry	55-110	NL	BID	G	585	
Cephalexin	Psittacine	35-50	PO	QID	E	763	
Cephalexin	Psittacine	100	PO	TID	G	697	
Cephalexin	Quail	100	IM	BID-TID	A	697	
Cephalexin	Quail, Bobwhite	35-50	PO	q2-3h	A	457	
Cephalexin	Quail, Japanese	35-50	PO	QID	A	847	
Cephalexin	Raptor	40-100	IM-PO	TID-QID	E	1240	
Cephalexin	Raptor	50-100	IM-PO	TID	E	1400	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Cephalexin	Raptor	50	PO	QID	G	234	
Cephalexin	Ratite	15-22	PO	TID	G	1308	
Cephalothin Sodium	Avian	100	IM-IV	QID	A	1473	
Cephalothin Sodium	Avian	100	IM-IV	QID	E	111	
Cephalothin Sodium	Avian	100	IM	q2-6h	E	565	
Cephalothin Sodium	Avian	100	IM	QID	E	741	No gastrointestinal absorption
Cephalothin Sodium	Ratite	30-40	IM-IV	QID	G	1308	
Cephradine	Avian	35-50	PO	q4-6h	E	111, 1473	
Cephradine	Crane	100	PO	q4-6h	E	111	
Cephradine	Emu, pigeon	100	PO	q4-6h	E	1473	
Chitosan	Avian	< 50	Topical	q3d-q2w	B	1633	Rinse and apply sparingly to wound surface or pack suppurating wound, promote wound healing
Chloral Hydrate	Avian (budgerigar, canary, chicken, crow, crane)	106.5	IM	PRN	B	1084	Add magnesium sulfate + pentobarbital sodium, reduce dose 15 to 20% in debilitated birds
Chloral Hydrate	Eagle, Golden	80.9	IM	PRN	B	1086	
Chloral Hydrate	Falcon, Prairie	64	IM	PRN	B	1086	
Chloral Hydrate	Gull (California, Laughing, Herring)	106.5	IM	PRN	B	1084	
Chloral Hydrate	Hawk (Marsh, Red-tailed, Swainson's)	64-68	IM	PRN	B	1086	
Chloral Hydrate	Owl, Saw-whet	106.5	IM	PRN	B	1084	
Chloral Hydrate	Pea Fowl, pheasant	106.5	IM	PRN	B	1084	
Chloral Hydrate	Pigeon, sparrow, toucanet	106.5	IM	PRN	B	1084	
Chloral Hydrate	Rail, Wood	106.5	IM	PRN	B	1084	
Chloralose	Anseriformes	0.4-0.48g/L bait	Feed	Once	G	1230	Add diazepam for anesthesia
Chloralose	Bird, Seed-eater	2 g/kg grain	Feed	Once	E	4	
Chloralose	Blackbird, Red-winged	0.02-0.025 mg TD	Feed	Once	G	1230	Add secobarbital
Chloralose	Crane	1.54-1.79 g/L corn	Feed	Once	G	1361	
Chloralose	Duck	0.4-0.5 g/L bait	Feed	Once	G	1386	Add diazepam
Chloralose	Duck, Mallard	15	Feed	Once	B	1095	
Chloralose	Duck, Mallard	40	PO	PRN	D	1401	
Chloralose	Turkey	8 g/L corn	Feed	Once	E	1386	Sedation in 40-60 min, 9% mortality

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Chloramphenicol	Avian	50-100	PO	QD	E	704	
Chloramphenicol	Avian	200 mg	Nebulize	TD	E	704	Dilute in 15 ml 0.9% saline solution
Chloramphenicol	Avian	50-100	Parenteral	QD	E	704	
Chloramphenicol	Budgerigar	50	IM	BID	A	394	
Chloramphenicol	Canary	0.1-0.15 g/L	Feed	QD	E	695	In soft food
Chloramphenicol	Chicken	50	IM	BID	A	394	
Chloramphenicol	Conure (Nanday, Sun)	50	IM	QID	A	394	
Chloramphenicol	Crane	100	SC	TID	A	596	
Chloramphenicol	Duck, Chinese	100	IM-IV	QID	A	508	
Chloramphenicol	Duck, Chinese	25	IM-IV	q3h	A	508	
Chloramphenicol	Duck, Chinese	240	IM-IV	TID	A	508	
Chloramphenicol	Duck, Muscovy	50	IM	BID	A	394	
Chloramphenicol	Eagle	50	IM	QD	A	394	
Chloramphenicol	Goose, Egyptian	50	IM	BID	A	394	
Chloramphenicol	Hawk	50	IM	BID	A	394	
Chloramphenicol	Macaw	50	IM	QID	A	394	
Chloramphenicol	Ostrich	10	IM	BID	G	401	Double dosage for birds below 5 kg
Chloramphenicol	Owl, Barred	50	IM	BID	A	394	
Chloramphenicol	Pea Fowl	50	IM	QD	A	394	
Chloramphenicol	Pigeon	95	PO	QID	E	111	With grit in diet
Chloramphenicol	Pigeon	50	PO	TID	E	704	
Chloramphenicol	Pigeon	30	PO	QID	E	1473	Without grit in diet
Chloramphenicol	Pigeon	80	IM	BID	E	1650	For salmonellosis
Chloramphenicol	Psittacine	50	IM-IV	TID-QID	E	111	
Chloramphenicol	Raptor	50	IM	TID	E	1240	
Chloramphenicol	Ratite	35-50	IM-IV-PO-SC	TID	G	1308	Not for food animals
Chloramphenicol Palmitate	Avian	80-100	Gavage	BID	E	741	
Chloramphenicol Palmitate	Avian	50	PO	TID	G	55	
Chloramphenicol Palmitate	Avian	150-200	PO	TID-QID	G	739	For large birds
Chloramphenicol Palmitate	Psittacine	100	PO	BID-QID	E	741	
Chloramphenicol Palmitate	Turkey	50	PO	QID	E	741	
Chlordiazepoxide HCl	Cowbird, quail	150 g/kg food	Feed	Once	B	1094	Anesthesia

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Chlorhexidine Gluconate	Avian	2.5 ml/L of 2% solution/L	Drink	NL	E	741	For candidiasis or antimicrobial prophylaxis
Chlorhexidine Gluconate	Avian	2.5-7.5 ml of 2% solution/L	Drink	NL	E	1240	Prevent or treat mild intestinal candidiasis
Chlorhexidine Gluconate	Avian	0.5% solution	Flush	NL	E	1240	Wound flush
Chlorhexidine Gluconate	Avian	0.005 g/L	Drink	QD	E	1492	For candidiasis
Chloroquine	Avian	0.4 g/L	Drink	QD	E	1180	For blood parasites
Chloroquine	Avian	10	PO	TID	G	57	Usually in combination with primaquine
Chloroquine	Avian	10	PO	QD	G	61	Add primaquine phosphate
Chloroquine	Falcon	10	PO	Once	B	1177	Add primaquine, follow 1st dose with 5 mg/kg at hours 6,18 and 24
Chloroquine	Passerine, Small	2.1 g/L	Drink	NL	E	1187	Therapeutic but not curative for *Plasmodium*
Chloroquine	Penguin	5	PO	QID	E	111, 1240	Loading dosage 10 mg/kg
Chloroquine	Penguin, African	5	PO	QD	B	262	
Chloroquine	Raptor	5	NL	NL	D	1612	
Chloroquine	Raptor	25	IM	QD	E	1400	*Plasmodium* and *Leucocytozoon*
Chloroquine	Raptor	10	PO	QD	G	61, 94	Add primaquine phosphate
Chlorpromazine	Avian	0.0125 g/L	Drink	NL	E	1492, 1650	For feather picking and anxiety
Chlortetracycline HCl	Amazon, Blue-fronted	2.5 g/kg corn	Feed	QD	A	230	For therapy less than 2 weeks duration
Chlortetracycline HCl	Amazon, Green-cheeked	2.5 - 5.0 g/kg food	Feed	Once	B	1062	For *Chlamydophila*
Chlortetracycline HCl	Amazons	5 g/kg food	Feed	QD	B	1079	For *Chlamydophila*
Chlortetracycline HCl	Avian	1 g/L	Drink	QD	E	704	Prophylactic dosage
Chlortetracycline HCl	Avian	5 g/L	Drink	QD	E	704	Therapeutic dosage
Chlortetracycline HCl	Avian	100	PO	QID	E	704	
Chlortetracycline HCl	Avian	0.5 g/L	Drink	QD	E	741	For chlamydophilosis initial therapy
Chlortetracycline HCl	Avian	100	IM-PO	NL	E	924	
Chlortetracycline HCl	Avian	34	IV	NL	F	1209	
Chlortetracycline HCl	Budgerigar	5 g/kg seed	Feed	QD	A	731	Hulled medicated millet
Chlortetracycline HCl	Budgerigar	500 mg/kg food	Feed	QD	E	565	Antichlamydophilial
Chlortetracycline HCl	Canary	1.5 g/kg food	Feed	QD	E	695	
Chlortetracycline HCl	Chicken	2.5 g/L	Drink	NL	A	802	Add chlortetracycline feed also
Chlortetracycline HCl	Chicken	2.5 g/kg food	Feed	Once	A	802	Add chlortetracycline drinking water also
Chlortetracycline HCl	Chicken	20-60	PO	NL	C	705	
Chlortetracycline HCl	Chicken	220 mg/kg food	Feed	QD	E	564	No slaughter withdrawal

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Chlortetracycline HCl	Cockatiel	2.4 g/kg food	Feed	QD	A	738	Hulled millet + oats for chlamydophilosis
Chlortetracycline HCl	Cockatoo, G Sulfur-crested	4.4 g/kg food	Feed	QD	B	1079	For chlamydophilosis
Chlortetracycline HCl	Conure (Maroon-bellied, Nanday)	2.4 g/kg food	Feed	QD	A	738	Hulled millet + oats for chlamydophilosis
Chlortetracycline HCl	Finch	1 - 1.5 g/L	Drink	QD	E	1572	
Chlortetracycline HCl	Finch	1.5 g/kg food	Feed	QD	E	1572	
Chlortetracycline HCl	Galliformes	30	Drink	QD	E	704	
Chlortetracycline HCl	Lorikeet	0.5 g/kg fruit	Feed	QD	A	1077	For chlamydophilosis
Chlortetracycline HCl	Lorikeet	0.5 g/L liquid food	Feed	Once	A	1077	For chlamydophilosis
Chlortetracycline HCl	Lory (Ornate, purple-naped)	0.5g/kg liquid food	Feed	Once	A	1077	For chlamydophilosis
Chlortetracycline HCl	Lovebird, Abyssinian	2.4 g/kg food	Feed	QD	A	738	
Chlortetracycline HCl	Macaw, Buffon's	10-15 g/kg	Feed	Once	A	781	
Chlortetracycline HCl	Parakeet (Bourke's, red-front, ring-necked)	2.4 g/kg food	Feed	QD	A	738	Hulled millet + oats for chlamydophilosis
Chlortetracycline HCl	Passerine	1.5 g/kg soft food	Feed	QD	E	1437	Add drug to drink at the same time, use for 30 days for chlamydophilosis
Chlortetracycline HCl	Passerine	1.0 - 1.5 g/L	Drink	QD	E	1437	Add drug to food at the same time, use for 30 days for chlamydophilosis
Chlortetracycline HCl	Pigeon	40-50	PO	B-TID	A	565	Without grit in diet
Chlortetracycline HCl	Pigeon	100	IM	QD	G	232	
Chlortetracycline HCl	Pigeon	50	PO	TID-QID	G	260	With or without tylosin
Chlortetracycline HCl	Pigeon	0.5 g/L	Drink	QD	G	590	Change daily
Chlortetracycline HCl	Poultry	0.106-0.264 g/L	Feed	QD	C	564	Prophylactic
Chlortetracycline HCl	Poultry	110-550 mg/kg feed	Feed	QD	C	564	
Chlortetracycline HCl	Psittacine	2.5-10 g/kg food	Feed	QD	A	693	Dosage based on weight of food
Chlortetracycline HCl	Raptor	33	PO	TID	E	1612	For chlamydophilosis
Chlortetracycline HCl	Ratite	15-20	PO	TID	G	1308	
Chlortetracycline HCl	Turkey	2.5 g/L	Drink	NL	A	802	Add chlortetracycline feed also
Chlortetracycline HCl	Turkey	10-30	PO	NL	C	705	
Choline	Avian	500-1300	Feed	QD	E	1470	For hepatic lipidosis
Chromium Picolinate	Avian	10 drops/kg	PO	QD	E	1435	Stock solution 1 pill per 30 ml lactulose for diabetes
Chromium Picolinate	Avian	0.1	PO	BID	E	1205	
Cimetidine	Penguin, African	35	PO	QD	G	128	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Ciprofloxacin HCl	Avian	80	PO	QD	D	1470, 1571	Add ethambutol + rifampin for liver mycobacteriosis
Ciprofloxacin HCl	Avian	20	PO	BID	D	1571	Add clofazimine + cycloserine + ethambutol for avian mycobacteriosis
Ciprofloxacin HCl	Avian	10-15	PO	BID	G	1319	
Ciprofloxacin HCl	Canary	20	PO	BID	G	48	
Ciprofloxacin HCl	Chicken	5	PO	NL	A	797	
Ciprofloxacin HCl	Cockatiel	30	PO	BID	G	44	
Ciprofloxacin HCl	Gull	20	PO	QD	E	1357	
Ciprofloxacin HCl	Hawk, Red-tailed	50	PO	BID	A	168	
Ciprofloxacin HCl	Ostrich	20	NL	BID	E	628	For oviduct infection
Ciprofloxacin HCl	Pigeon	0.25 g/L	Drink	QD	G	590	Change daily
Ciprofloxacin HCl	Pigeon	5-20	PO	BID	G	590	
Ciprofloxacin HCl	Psittacine	20-40	PO	BID	E	111	
Ciprofloxacin HCl	Toucan, Toco	10	PO	BID	G	1379	
Cisapride	Avian	0.5-1	PO	TID	E	1151	To improve GI motility
Cisapride	Psittacine	1	NL	BID	G	1263	For ileus
Cisapride	Raptor	0.25	PO	TID	E	1400	Increase gut motility
Cisplatin	Macaw, Blue and Gold	0.3 mg/cm^3	Parenteral	QW	G	1369	Intralesional-fibrosarcoma
Citric Acid	Avian	0.5 g/L	Drink	QD	E	704	Add oxytetracycline
Citric Acid	Passerine, Small	1.0 g/L	Drink	NL	E	1187	
Clanobutin	Psittacine	0.2	PO	QD	G	1722	Liver support
Clarithromycin	Avian	85	PO	QD	G	1154	Add ethambutol + rifabutin, dose allometrically for mycobacteriosis
Clazuril	Anseriformes	5-10	PO	q3d	D	1150	For coccidiosis
Clazuril	Pigeon	5-10	PO	QD	G	57	Repeat after 2 days
Clazuril	Pigeon	6.5	PO	Once	G	109	
Clazuril	Pigeon	5	PO	NL	G	109	
Clazuril	Poultry	5-10	PO	QD	G	57	Repeat after 2 days
Clazuril	Psittacine	7	PO	QD	E	1240	Wait 2 days then repeat for coccidiosis
Clazuril	Raptor	5-10	PO	q3d	E	1240	For coccidiosis
Clindamycin HCl	Avian	100	PO	QD	D	1221	For clostridiosis
Clindamycin HCl	Avian	150	PO	QD	E	1431	For osteomyelitis

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Clindamycin HCl	Eagle, Little	40	PO	BID	G	1568	For post-operative orthopedic surgery
Clindamycin HCl	Goshawk, Brown	40	PO	BID	G	1568	
Clindamycin HCl	Gull	100-150	PO	QD	E	1357	
Clindamycin HCl	Owl, Barn	40	PO	BID	G	1568	
Clindamycin HCl	Owl, Great-horned	12.5	PO	BID	G	173	Add enrofloxacin
Clindamycin HCl	Pigeon	100	PO	QD	A	1473	
Clindamycin HCl	Pigeon	100	PO	QD	E	111	
Clindamycin HCl	Psittacine	100	PO	QD	E	1240	For osteomyelitis and tendon sheath infections
Clindamycin HCl	Psittacine	50	NL	BID	F	1170	Used to treat anaerobic osteomyelitis
Clindamycin HCl	Psittacine	25	NL	TID	F	1170	
Clindamycin HCl	Raptor	30-40	PO	BID	B	1568	
Clofazimine	Avian	6	PO	QD	D	1470	Add ethambutol + rifampin for liver mycobacteriosis
Clofazimine	Avian	1.5	PO	QD	D	1571	
Clofazimine	Psittacine	1.5	PO	QD	E	1240	For mycobacteriosis
Clofazimine	Raptor	1-5	PO	QD	E	1240, 1154	
Clomipramine HCl	Avian	0.5-1	NL	QD-BID	D	1475	Control of self-mutilation, mixed results
Clomipramine HCl	Cockatoo	3	PO	BID	G	1278	Cloacal prolapse
Clomipramine HCl	Psittacine	0.5-1	PO	BID	B	43	
Clomipramine HCl	Psittacine	0.5-1	PO	QD-BID	E	111	Gradually increase dose over 4 to 5 days
Clopidol	Chicken	125-250 mg/kg food	Feed	QD	E	564	5-day slaughter withdrawal, prophylactic
Clopidol	Poultry	0.25 g/kg food	Feed	QD	C	564	Prophylactic
Clorsulon	Anseriformes	20	PO	q2w	D	1150	For cestodes and nematodes
Clorsulon	Avian	20	PO	q2w	G	818	For tapeworms
Clorsulon	Psittacine	20	PO	q2w	E	111	
Clorsulon	Raptor	20	PO	q2w	E	1240	For trematodes and cestodes
Clotrimazole	Crane	10 g/L	Nebulize	TID	E	1361	Antifungal
Clotrimazole	Raptor	70-100 g/L	Nebulize	BID	E	1359	
Cloxacillin Sodium	Avian	100-200	IM	QD	E	565	
Cloxacillin Sodium	Avian	250	PO	BID	E	1234	For bumblefoot
Cloxacillin Sodium	Avian	100	PO	QD	E	1470	
Cloxacillin Sodium	Raptor	250	PO	BID	E	1240	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Cloxacillin Sodium	Raptor	100-250	IM	NL	E	1463	For bacterial infections
Coenzyme Q10	Avian	1	PO	QD-BID	E	1205, 1435	For heart, geriatric, diabetic, immune deficient birds
Colchicine	Avian	0.04	PO	QD	D	1470	For hepatic fibrosis
Colchicine	Macaw	0.01	PO	BID	G	45	
Colchicine	Psittacine	0.2	PO	BID	E	111, 1473	
Colchicine	Psittacine	0.04	PO	QD-BID	E	1756	
Corticotropin	Avian	16 IU TD	IM	Once	B	88, 532	
Crotamiton	Psittacine	10%	Topical	NL	E	1240	For knemidocoptic mites
Cupric Sulfate	Avian	51% powder	Topical	PRN	E	1240	For ulcerative dermatitis
Cupric Sulfate	Ostrich	0.5 g/L	Drink	QD	G	1254, 283	For candidiasis, use acidified copper sulfate
Cupric Sulfate	Psittacine	1:2000 dilution	Topical	q2w	D	1446	For dermatomycosis
Cyanocobalamin	Avian	0.25-0.5	IM	QW	E	111	
Cyclophosphamide							**Check current literature prior to use
Cyclophosphamide	Owl, Great-horned	25	PO	Once	F	125	Maximum dosage listed
Cyclophosphamide	Psittacine	200 mg/m^2	IO	QW	E	1470	Lymphosarcoma
Cycloserine	Avian	5	PO	BID	D	1571	Add ciprofloxacin or enrofloxacin + clofazimine + ethambutol for avian mycobacteriosis
Cycloserine	Raptor	5	PO	BID	E	1240, 1154	
Cyclosporine	Duck, Pekin	60	IV	QD	A	835	
Cyclosporine	Duck, Pekin	10	IV	TID-QID	A	835	
Cypermethrin	Pigeon	0.05% solution	Topical	QD	E	704	Apply as spray
Cypermethrin	Psittacine	2.0% solution	NL	NL	E	1240	Spray premises for *Dermanyssus*, avoid contact with skin
Cypermethrin	Raptor	2% solution	NL	NL	E	1240	
Danofloxacin Mesylate	Chicken	0.05 g/L	Drink	NL	A	837	For *Mycoplasma gallisepticum*
Danofloxacin Mesylate	Chicken	5	PO	QD	B	893, 990	For colisepticemia

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Decoquinate	Chicken	30 mg/kg food	Feed	QD	E	564	No slaughter withdrawal, prophylactic
Decoquinate	Pheasant, Ring-necked	0.03 g/kg food	Feed	QD	B	895	For coccidiosis
Deferiprone	Toucan	50	PO	BID	G	1280	For iron storage disease, zinc supplementation may be required
Deferoxamine Mesylate	Avian	100	SC	QD	D	1470	To reduce liver iron levels
Deferoxamine Mesylate	Avian	20	PO	Once	G	87	Continue IM q4h as needed
Deferoxamine Mesylate	Mynah	40	IM	QD	G	340	Repeat qow for series of 10 treatments
Deferoxamine Mesylate	Toucan	100	SC	QD	G	1280	For iron storage disease
Deferoxamine Mesylate	Toucan	1.28 mg/kcal	IM	QD	E	1180	Iron chelation
Delmadinone Acetate	Avian	1	IM	NL	E	1434	
Delmadinone Acetate	Pigeon	6.67	IM	NL	E	1432	
Detomidine HCl	Avian	0.3	IM	NL	G	1320	
Detomidine HCl	Chicken	0.3	IM	PRN	E	1573	Sedation
Detomidine HCl	Ostrich	0.3	IM	PRN	E	1573	Sedation
Dexamethasone	Avian	1-2	IM	NL	D	1533	
Dexamethasone	Avian	2-4	IM	QD	E	1431	Use sparingly
Dexamethasone	Avian	N/A	Topical	NL	E	1151	Add dimethyl sulfoxide for cloacal or uterine prolapse
Dexamethasone	Crane, Sarus	0.5	IV	Once	G	1087	
Dexamethasone	Falcon	2	IM-IV	Once	E	1027	For shock, trauma, toxic conditions
Dexamethasone	Heron, Great Blue	0.5	IV	Once	G	1087	
Dexamethasone	Pigeon	0.15-1.5	IM-IV	BID	E	1432	
Dexamethasone	Pigeon	0.3-3	IM-IV	BID	E	1432	For shock
Dexamethasone	Psittacine	2-4	IM	QD-BID	E	1240	
Dexamethasone	Psittacine	4	IM-SC	NL	G	632	Neonate dosage, shock or sepsis
Dexamethasone	Raptor	2-4	IM	QD	E	1433	Use with great care
Dexamethasone	Raptor	0.3-3	IM	q2d	E	1240	Long-lasting formulation, reduce inflammatory response and shock
Dexamethasone Sodium Phosphate	Amazon, Yellow-naped	1	IM	q3-7d	G	1033	
Dexamethasone Sodium Phosphate	Anseriformes	2	IM	QD	E	1240	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Dexamethasone Sodium Phosphate	Avian	2	IM-IV	Once	E	1151	
Dexamethasone Sodium Phosphate	Bustard	2	IM	QD	E	1240	
Dexamethasone Sodium Phosphate	Psittacine	2-4	IM-IV	NL	E	1688	
Dexamethasone Sodium Phosphate	Raptor	2	IM	QD	E	1240	
Dextrose	Avian	N/A	Topical	PRN	E	1151	For flushing wounds
Dextrose	Avian	50	IV-PO-SC	NL	E	1240	Isotonic for hypoglycemia and dehydration, IV slowly
Dextrose	Avian	50-100	IV	NL	E	111	Give slowly
Dextrose	Avian	50	IV-PO-SC	NL	E	1240	
Dextrose	Avian	50-100	IV	NL	E	1473	Give slowly
Dextrose	Avian	1	IV	NL	G	1311	For hypoglycemia, 50% solution, may be deleted
Dextrose	Psittacine	2 ml/kg	IV	NL	G	632	Neonate dosage, use 50% solution, slow bolus
Dextrose	Raptor	500-1000	IV	Once	E	1400	Give slowly for hypoglycemia
Diatrizoate Meglumine	Budgerigar	0.2 ml TD	IP	NL	G	95	Goiter therapy
Diatrizoate Meglumine + Diatrizoate Sodium	Amazon, Double Yellow-head	400	IV	Once	D	1756	For IV excretory urography
Diazepam	Anseriformes	1	NL	PRN	D	1403	Add ketamine
Diazepam	Anseriformes	1.2-1.6 g/L bait	Feed	Once	G	1230	Add chloralose
Diazepam	Anseriformes	0.5-1	IM-IV	BID-TID	E	1240	Control seizures
Diazepam	Avian	0.5-1	IM	NL	E	1120, 1492	For seizures related to lead toxicoses
Diazepam	Bird, Aquatic	0.6	IM-IV	PRN	E	1503	
Diazepam	Bird, Aquatic	0.5-1	IM-IV	PRN	E	1559	For lead poisoning to control convulsions
Diazepam	Crane	0.5-1	NL	PRN	E	1189	For tranquilization 4 to 6 hours
Diazepam	Crane	0.2-0.5	NL	PRN	E	1189	Add ketamine for anesthesia
Diazepam	Duck	1.3-1.7 g/kg bait	Feed	Once	G	1386	Add chloralose
Diazepam	Emu	0.6	IV	PRN	G	283	Add pentobarbital after 1 hour
Diazepam	Emu	5	IV	NL	E	4	Sedation
Diazepam	Falcon	1.2	IV	PRN	D	1401	Add ketamine
Diazepam	Hawk, Rough-legged	1-1.5	IV	PRN	G	1092	Combine with ketamine

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Diclofenac Sodium	Pigeon	12.5 mg/TD	PO	NL	E	1240	For arthritis
Diethylcarbamazine Citrate	Raptor	50	PO	NL	E	1463	Anthelmintic
Diethylstilbestrol	Avian	0.025-0.075	IM	NL	E	111	
Diethylstilbestrol	Avian	0.1-0.3	IM	NL	E	1473	
Digoxin	Amazon, Yellow-naped	0.02	PO	BID	G	1033	
Digoxin	Avian	0.02	NL	NL	D	1470	
Digoxin	Avian	0.01	PO	QD-BID	E	1152	
Digoxin	Avian	0.02-0.05	PO	QD-BID	E	1152	
Digoxin	Avian	0.0033 g/L	Drink	QD	G	1323	Change daily
Digoxin	Budgerigar	0.02	PO	QD	A	882	
Digoxin	Sparrow	0.02	PO	QD	A	882	
Dimercaprol	Avian	2.5	IM	q4h	E	1120, 1236	Continue BID for 10 days (or longer if needed) for lead toxicoses
Dimercaprol	Avian	25-35	PO	QD-q2d	E	1240, 1473	Give 5 days per week for lead toxicosis
Dimethyl Sulfoxide	Avian						
Dimethyl Sulfoxide	Avian	5 ml	Nebulize	NL	E	704	Add tylosin + 10 ml 0.9% saline solution
Dimethyl Sulfoxide	Avian	10 ml/L	Nebulize	TID-QID	E	1650	Add appropriate antimicrobial, adjunct to parenteral therapy for air sacculitis
Dimethyl Sulfoxide	Avian	Ointment	Topical	NL	E	1650	For prolapsed phallus
Dimethyl Sulfoxide	Avian	1 ml/kg 50 % solution	PO	NL	G	1717	Also apply topically
Dimethyl Sulfoxide	Avian	20	IM	BID-TID	G	861	For heavy metal poisoning
Dimethyl Sulfoxide	Chicken	Mix 50:50	Topical	NL	B	1342	Add bupivacaine, prepare 50:50 solution
Dimethyl Sulfoxide	Raptor	N/A	Topical	NL	E	1240	Reduce swelling, vehicle to carry drugs through skin particularly on legs
Dimethylglycine	Avian	5	PO	TID	E	1205	Brain disorders
Dimethylglycine	Avian	2-4	PO	QD-BID	E	1435	Immune and energy booster
Dimetridazole	Avian	50	PO	QD	E	704	
Dimetridazole	Avian	1.25 ml powder/L	Drink	NL	E	741	For trichomoniasis, giardiasis and histomoniasis

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Dimetridazole	Avian	50	PO	NL	E	924	For protozoa
Dimetridazole	Avian	30	Gavage	Once	E	1650	For trichomoniasis, giardiasis and histomoniasis
Dimetridazole	Finch	0.1 g/L	Drink	QD	G	1334	For cochlosomiasis
Dimetridazole	Lorikeet	0.1 g/L	Drink	QD	E	1479	
Dimetridazole	Pigeon	0.4 g/L	Drink	NL	A	791	
Dimetridazole	Pigeon	50	PO	QW	E	1432	
Dimetridazole	Pigeon	20	PO	QD	G	232	
Dimetridazole	Poultry	500 mg/kg food	Feed	QD	C	564	Prophylactic
Dimetridazole	Poultry	0.5-1.0 g/L	Drink	QD	G	585	
Dimetridazole	Raptor	125	PO	NL	D	1612	For trichomoniasis
Dimetridazole	Raptor	100	PO	NL	E	1463	For trichomoniasis
Dinitolmide	Chicken	40-187 mg/kg food	Feed	QD	E	564	No slaughter withdrawal, prophylactic
Dinoprost Tromethamine (Prostaglandin F-2a)	Anseriformes	0.02-0.1	IM-Topical	Once	D	1150	Apply to cloacal mucosa topically, egg binding
Dinoprost Tromethamine	Anseriformes	0.02-0.1	IM	Once	E	1240	For egg binding
Dinoprost Tromethamine	Avian	0.02-0.1	IM	Once	E	1473	Also intracloacal
Dinoprost Tromethamine	Ostrich	5 mg TD	Parenteral	Once	G	1224	For caseous salpingitis, add appropriate antimicrobials for 1 week
Dinoprost Tromethamine	Psittacine	0.02-0.1	Vent	Once	E	1240	Apply to cloacal mucosa for egg binding
Dinoprost Tromethamine	Psittacine	0.02-0.1	IM	Once	E	1240	For egg binding
Dinoprost Tromethamine	Raptor	0.02-0.1	Vent	Once	E	1240	
Dinoprostone (Prostaglandin E)	Avian	0.2	Vent	NL	E	1474	Precede with calcium gluconate for egg binding, apply into cloaca, may add vitamin E + selenium
Dinoprostone	Cockatiel	0.02	Vent	Once	G	1699	For egg binding
Diphenhydramine HCl	Avian	2	IO	NL	E	1470	Give before asparaginase and doxorubicin to minimize anaphylaxis
Diphenhydramine HCl	Avian	4	IM	TID	E	1554	For anticholinesterase toxicity
Diphenhydramine HCl	Avian	2-4	PO	BID	D	1475	Control of self-mutilation
Diphenhydramine HCl	Avian	2-4	NL	BID	E	1477	For feather, suture and bandage picking
Diphenhydramine HCl	Psittacine	2-4	PO	BID	E	1473	
Diphenhydramine HCl	Raptor	4	IM	TID	G	50	
Diprenorphine	Cassowary, Double-wattled	2 mg/mg etorphine	NL	PRN	D	1401	Etorphine antagonist

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Diazepam	Kestrel	2.2	IV	PRN	D	1401	Add ketamine
Diazepam	Ostrich	1	IV	PRN	D	1401	Smooth anesthesia recovery
Diazepam	Ostrich	5	PO	PRN	E	4	Standing sedation
Diazepam	Ostrich	1-2	IV	PRN	G	481	Give just prior to recovery from tiletamine/zolazepam
Diazepam	Owl	1	IV	PRN	D	1401	Add ketamine
Diazepam	Passerine	0.5	PO	NL	E	1439	Anxiolytic and hyperphagic for wild fractious species
Diazepam	Pigeon	2.5	IM	PRN	E	1432	Add ketamine, 20 to 30 minutes deep sedation
Diazepam	Pigeon	0.5-1	IM-IV	PRN	G	590	Add ketamine
Diazepam	Psittacine	2.5-4	PO	PRN	E	1473	
Diazepam	Psittacine	0.5-1	IM-IO-IV	PRN	E	1688	
Diazepam	Psittacine	1-1.5	IM-IV	PRN	G	824	
Diazepam	Raptor	1	IM	PRN	D	1401	Add ketamine
Diazepam	Raptor	1-1.5	IV	PRN	D	1533	Add ketamine
Diazepam	Ratite	1	NL	PRN	D	1403	Add ketamine
Diazepam	Ratite	0.1	IM	PRN	E	4	Add ketamine
Diazepam	Ratite	0.2-0.3	IV	PRN	E	243	Add ketamine
Diazepam	Ratite	0.3	IV	PRN	G	418	Tranquilization particularly during anesthesia recovery
Diazepam	Rhea	5	IV	NL	E	4	Sedation
Diazepam	Swan	1.3-1.7 g/kg bait	Feed	Once	G	1386	Add chloralose
Dibutyltin Dilaurate	Chicken	200 mg/kg food	Feed	QD	E	564	10 day slaughter withdrawal
Dibutyltin Dilaurate	Poultry	374 mg/kg food	Feed	QD	C	564	
Dichlorophen	Pigeon	100 mg/TD	PO	q10d	G	232	
Dichlorvos	Avian	N/A	IH	QD	E	704	Impregnated strip, 30 m^3 minimum
Dichlorvos Strip	Kakariki	N/A	IH	QD	G	1609	For *Knemidocoptes*
Diclazuril	Chicken	0.5-1.0 mg/kg food	Feed	QD	G	564	
Diclazuril	Crow	10	PO	QD	D	1438	Dose on days 0, 1, 2, 4, 6, 8 and 10 for toxoplasmosis
Diclazuril	Partridge, European grey	0.366	Feed	QD	B	891	For *Eimeria*
Diclazuril	Passerine	10	PO	QD	G	1334	Give on days 0, 1, 2, 4, 6, 8 and 10
Diclazuril	Pheasant, Ring-necked	0.369	Feed	QD	B	891	For *Eimeria*
Diclazuril	Quail, Japanese	0.29-0.56	Feed	QD	B	891	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Diprenorphine	Ostrich	0.05-0.06	IV	PRN	B	447	Opioid antagonist
Diprenorphine	Ostrich	4.5-24 mg TD	IM	PRN	G	481	Reverse etorphine
Docusate Sodium	Psittacine	0.3 g/L	Drink	NL	E	763	To aid in expelling lead or prevent constipation following cloacal surgery
Doxapram HCl	Anseriformes	10	IV	Once	D	1150	Stimulant
Doxapram HCl	Avian	5-10	IM-IV	Once	E	111, 1473	
Doxapram HCl	Avian	5	IM-IO-IV	NL	E	243	
Doxapram HCl	Avian	6	IV	NL	G	496	
Doxapram HCl	Avian	20	IM-IO-IV	NL	E	1183	Also drop onto tongue for cardiac arrest
Doxapram HCl	Falcon	10	IM-IV	Once	E	1027	
Doxapram HCl	Ostrich	0.5-1.5	IV	PRN	G	522	
Doxapram HCl	Psittacine	20	IM-IO-IV	NL	E	1688	
Doxapram HCl	Raptor	10	IM-IV	Once	E	1240	
Doxapram HCl	Raptor	5-20	IT-IV	Once	E	1359	For anesthetic emergencies
Doxepin HCl	Avian	0.5-1	PO	BID	E	111	Control feather picking
Doxepin HCl	Avian	0.5	PO	BID	E	704	Or add to drinking water
Doxorubicin							**Check current literature prior to use
Doxorubicin	Avian	30 mg/m^2	IO	q3w	E	1470	Give 30 mg/m² of body surface for lymphosarcoma
Doxycycline	Amazon Parrot	1 g/kg food	Feed	QD	A	693	
Doxycycline References followed by ** are based on the long acting formula)	Avian	100	IM	QW	A	55	European formulation. For chlamydophilosis, may be spaced 5 to 7 days for first 4 weeks
Doxycycline	Amazon Parrot	40-50	PO	QD-BID	E	111	
Doxycycline**	Amazon Parrot	100	IM	q5d-QW	A	230	For chlamydophilosis
Doxycycline**	Amazon	50	IM	q4-5d	B	1062	
Doxycycline	Anseriformes	0.24 g/kg food	Feed	QD	D	1150	
Doxycycline	Anseriformes	50	PO	BID	D	1150	For chlamydophilosis
Doxycycline**	Avian	100	IM	q10d	B	583	
Doxycycline	Avian	15	Drink	QD	C	705	
Doxycycline**	Avian	75-100	IM-SC	q5d-QW	E	565	Antichlamydophilial
Doxycycline**	Avian	75-100	IM	q5d	E	703	For chlamydophilosis, may be spaced 5 to 7 days for first 4 weeks

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Doxycycline	Bird, Aquatic	25-50	PO	QD	E	1503	Broad spectrum, food for respiratory infections, treat for 45 days for chlamydophila
Doxycycline	Bird, Aquatic	10 g/L	Drink	QD	E	1503	See above
Doxycycline**	Bird, Aquatic	75-100	IM	QW	E	1503	See above
Doxycycline**	Bustard, Houbara	100	IM	QW	A	1240	See above
Doxycycline	Bustard, Houbara	22-44	IV	Once	A	1240	Continue therapy with IM or PO dosage for severe chlamydophilosis
Doxycycline	Chicken	20	PO	QD	A	762	
Doxycycline	Chicken	0.1 g/L	Drink	NL	A	803	
Doxycycline	Chicken	10	Drink	QD	B	1009	For fowl cholera
Doxycycline	Cockatiel	0.83 g/L	Drink	QD	A	1638	For chlamydophilosis
Doxycycline	Cockatiel	0.5 g/kg seed	Feed	QD	A	1638	For chlamydophilosis, may cause toxicosis
Doxycycline	Cockatiel	1 g/kg mash	Feed	QD	A	1638	For chlamydophilosis
Doxycycline**	Cockatiel	100	IM	q10d	A	1638	For chlamydophilosis, may not maintain adequate drug plasma levels
Doxycycline	Cockatiel	40-50	PO	QD	E	111, 703	For chlamydophilosis
Doxycycline	Cockatoo	1 g/kg food	Feed	QD	A	693	
Doxycycline	Cockatoo, Goffin	25	PO	BID	A	697	
Doxycycline	Cockatoo, Goffin	0.4 g/L	Drink	NL	A	749	
Doxycycline	Cockatoo, Goffin	1 g/kg mash	Feed	QD	A	1639	
Doxycycline	Macaw	25	PO	BID	A	697	
Doxycycline	Macaw, Blue and Gold	1 g/kg corn mash	Feed	QD	A	730	Each bird fed 100 g mash
Doxycycline	Ostrich	10	NL	BID	E	628	Oviductal infection therapy
Doxycycline	Parrot, Grey	25	PO	BID	A	697	
Doxycycline	Parrot, Grey	0.4 g/L	Drink	NL	A	749	
Doxycycline	Parrot, Grey	1 g/kg food	Feed	QD	A	1639	
Doxycycline	Passerine	0.25 g/L	Drink	QD	E	1437	Add drug to food at the same time, use for 30 days for chlamydophilosis
Doxycycline	Passerine	1 g/kg food	Feed	QD	E	1437	Add drug to drink at the same time, use for 30 days for chlamydophilosis
Doxycycline	Penguin	10	NL	QD	G	1353	For babesiosis
Doxycycline	Penguin, African	20	PO	BID	G	1353	
Doxycycline **	Pigeon	75	IM	Once	A	379	
Doxycycline**	Pigeon	75-100	IM-SC	q5d-QW	A	565	
Doxycycline	Pigeon	25	PO	BID	A	565	With grit in diet
Doxycycline	Pigeon	15	Drink	QD	C	704	
Doxycycline	Poultry	0.05 g/L	Drink	QD	C	564	
Doxycycline	Poultry	0.25-0.5 g/L	Drink	QD	G	585	Add tylosin, antimycoplasmal

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Doxycycline	Psittacine	25-50	IV	NL	E	111	
Doxycycline**	Psittacine	100	IM	QW	E	632	Neonate dosage
Doxycycline	Psittacine	50	PO	QD	E	632	Neonate dosage
Doxycycline **	Psittacine	20	IV	Once	E	632	Neonate dosage
Doxycycline	Raptor	50	IM	NL	B	1568	
Doxycycline	Raptor	25	PO	BID	G	94	Juvenile dosage
Doxycycline	Ratite	2-3.5	PO	BID	G	1308	For chlamydophilosis
Doxycycline Hyclate	Avian	0.4 g/L	Drink	QD	A	703	For chlamydophilosis
Doxycycline Hyclate	Avian	200-400 mg/kg food	Feed	QD	E	565	Antichlamydophilial
Doxycycline Hyclate	Cockatiel	0.2-0.4 g/L	Drink	QD	A	703	For chlamydophilosis
Doxycycline Hyclate	Cockatoo, Goffin	0.8 g/L	Drink	QD	A	703	
Doxycycline Hyclate	Parrot, Grey	0.8 g/L	Drink	QD	A	703	
Doxycycline Hyclate	Pigeon	55	PO	BID	G	47	
Edetate Calcium Disodium	Anseriformes	10-40	IM-IV	BID	D	1150	For lead poisoning
Edetate Calcium Disodium	Avian	30-50	IM-IV	QID	D	1221	For lead or zinc toxicosis, may use in conjunction with penicillamine
Edetate Calcium Disodium	Avian	20-40	IM-IV	BID	D	1470	To reduce liver lead and zinc
Edetate Calcium Disodium	Avian	20-40	IM	BID-TID	E	111	
Edetate Calcium Disodium	Bird, Aquatic	35	IM	BID	D	1478	Give 3 to 4 days per week for lead poisoning
Edetate Calcium Disodium	Crane, Sand Hill	35	IM	BID	B	1088	Repeat 4 days later for lead poisoning
Edetate Calcium Disodium	Lory, Chattering	30	SC	BID	G	42	
Edetate Calcium Disodium	Macaw	35	IM	BID	G	40	
Edetate Calcium Disodium	Pigeon	30	IM	BID	G	590	
Edetate Calcium Disodium	Psittacine	10-40	IV	BID	E	1240	Lead and heavy metal poisoning
Edetate Calcium Disodium	Raptor	35-50	NL	BID	E	1188	For lead toxicity
Edetate Calcium Disodium	Raptor	10-40	IM-IV	BID	E	1240	Lead and heavy metal poisoning

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Enilconazole	Avian	2 g/L NaCl	Topical	NL	E	924	For skin mycosis
Enilconazole	Psittacine	2 g/L NaCl	Nebulize	BID	G	1034	Dilute 100 mg/ml 1:50 in normal saline
Enilconazole	Psittacine	0.1-1% solution	IT	NL	E	1240	
Enilconazole	Raptor	5	IT	QD	E	1240	Dilute 1:10
Enilconazole	Raptor	0.2% solution	Topical	BID	E	1240	
Enilconazole	Ratite	10% solution	Flush	NL	G	1224	For mycotic dermatosis
Enrofloxacin	Amazon Parrot	7.5-15	IM-PO	QD-BID	A	1473	
Enrofloxacin	Amazon	15	IM	QD	A	749	
Enrofloxacin	Amazon, Blue-fronted	0.5 mg/kg food	Feed	Once	A	858	For chlamydophila
Enrofloxacin	Amazon, Red Lored	10	IM	BID	G	700	2.5% concentration
Enrofloxacin	Anseriformes	10-15	IM-PO	BID	D	1150, 1358	
Enrofloxacin	Aracari, Black-necked	10	PO	BID	G	1367	Pre-surgical
Enrofloxacin	Avian	5-15	IM-PO-SC	QD-BID	A	916	
Enrofloxacin	Avian	30	PO	NL	E	1492	For *Mycoplasma, Chlamydophila*
Enrofloxacin	Avian	10 g/L	Nebulize	TID-QID	E	1650	Add dimethyl sulfoxide, adjunct to parenteral therapy for air sacculitis
Enrofloxacin	Avian	0.25-1 g/kg food	Feed	QD	F	916	
Enrofloxacin	Avian	0.05-0.5 g/L	Drink	NL	F	916	
Enrofloxacin	Avian	15	PO	QD	G	580	Add ethambutol + rifampin, antimycobacterial
Enrofloxacin	Bird, Aquatic	30	IM	BID	E	1478	Post-trauma infections
Enrofloxacin	Budgerigar	30	Feed	Once	A	920	For chlamydophilosis
Enrofloxacin	Bustard, Houbara	15-30	IM-IV-PO	BID	A	790	
Enrofloxacin	Chicken	10	Drink	NL	A	811	
Enrofloxacin	Duck, Muscovy	15	Drink	QD	B	842	Pulse dose for 4 hours each day
Enrofloxacin	Duck, Pekin	10	Drink	QD	A	854	For *Pasteurella* and coliforms
Enrofloxacin	Gull	10	IM	BID	E	1357	
Enrofloxacin	Hawk, Red-tailed	5	IV	QD	A	978	
Enrofloxacin	Hornbill, Great	10-20	PO	BID	G	1587	
Enrofloxacin	Lory, Red	15	PO	BID	G	1621	
Enrofloxacin	Ostrich		PO	TID	G	418	
Enrofloxacin	Owl, Great-horned	5	IM-PO	BID	G	173	Add clindamycin or cephalothin
Enrofloxacin	Parrot, Grey	0.5 g/L	Drink	QD	A	921	For salmonellosis
Enrofloxacin	Parrot, Grey	0.5 g/kg food	Feed	Once	A	921	Higher blood levels in neonates
Enrofloxacin	Penguin, Rockhopper	9	PO	QD	G	1646	
Enrofloxacin	Pheasant	20	IM-PO	NL	G	678	
Enrofloxacin	Pheasant, Chinese Ringneck	7.5	IM	QD	G	1296	Continue therapy orally
Enrofloxacin	Pigeon	15	IM-SC	QD	A	179	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Enrofloxacin	Pigeon	10	PO	QD	A	179	
Enrofloxacin	Pigeon	5-10	IM-IV-PO-SC	QD-BID	A	866	
Enrofloxacin	Pigeon	10-20	IM-PO-SC	QD-BID	A	919	
Enrofloxacin	Pigeon	5	Drink	QD	A	919	
Enrofloxacin	Pigeon	5	PO	BID	A	1473	
Enrofloxacin	Poultry	15	NL	BID	G	585	
Enrofloxacin	Psittacine	2.5-20	PO	QD	A	179	
Enrofloxacin	Psittacine	11-17.5	Drink	NL	B	750	
Enrofloxacin	Psittacine	10-20	IM	NL	E	632	Neonate dosage, may cause joint defects
Enrofloxacin	Raptor	15	IM-PO	BID	E	1240	Broad spectrum including *Pseudomonas, Klebsiella, Mycoplasma*
Enrofloxacin	Ratite	1.5-2.5	PO-SC	BID	G	1308	For Gram negative infections
Epinephrine	Avian	0.1	IO-IP-IT-IV	NL	E	1473	
Epoetin Alfa	Avian	0.1	SC	q2-3d	E	1151	For anemia
Ergonovine Maleate	Avian	0.06	IM	Once	E	111, 1473, 1650	With or without calcium gluconate for egg binding
Erythromycin	Avian	0.125 g/L	Drink	NL	E	111	
Erythromycin	Avian	25	IM	QD	E	565	
Erythromycin	Avian	100	PO	NL	E	704	
Erythromycin	Avian	200 mg/kg	Feed	QD	E	704	Soft food
Erythromycin	Avian	10-20 g/L	Nebulize	TD	E	704	Diluted in 15 ml 0.9% saline solution
Erythromycin	Chicken	0.102 g/L	Drink	QD	A	544	Chick dosage
Erythromycin	Finch	0.2 g/kg food	Feed	QD	E	1572	
Erythromycin	Finch	0.125 g/L	Drink	QD	E	1572	
Erythromycin	Pigeon	100	PO	NL	A	1055	
Erythromycin	Pigeon	2.2 g/L	Drink	QD	E	704	Erythrocin Soluble®, 5 g per teaspoon
Erythromycin	Poultry	0.25 g/L	Feed	QD	C	564	
Erythromycin	Poultry	10-20	Drink	QD	C	705	
Erythromycin	Poultry	50-100	NL	BID	G	585	
Erythromycin	Psittacine	44-88	PO	BID	E	741	For sinusitis
Erythromycin	Psittacine	10-20	PO	BID	E	1240	For mycoplasmal sinusitis and air sacculitis
Erythromycin	Psittacine	10-20	Parenteral-PO	BID	E	1473	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Erythromycin	Ratite	5-10	PO	TID	G	1308	
Estradiol	Avian	10-15	IM	QW	B	677	Induce molting X 4 weeks
Ethambutol HCl	Amazon, Yellow-cheeked	30	NL	QD	G	713	Add rifampin + isoniazid
Ethambutol HCl	Avian	30	PO	QD	D	1470	
Ethambutol HCl	Avian	15	PO	BID	E	1473	
Ethambutol HCl	Avian	30	PO	QD	E	1650	Add enrofloxacin + rifabutin for mycobacteriosis
Ethambutol HCl	Avian	10	PO	BID	G	55	Add rifampin + streptomycin for mycobacteriosis
Ethambutol HCl	Crane, Whooping	30	PO	QD	G	207	Add rifampin
Ethambutol HCl	Psittacine	15-20	PO	BID	E	1240	For mycobacteriosis
Ethambutol HCl	Raptor	20	PO	BID	E	1240	
Etorphine	Cassowary, Double-wattled	0.15	IM	PRN	G	447	Add acepromazine
Etorphine	Ostrich	0.04-0.07	IM	PRN	B	447	Add acepromazine
Etorphine	Ostrich	0.04	IM	PRN	B	447	Add acepromazine + xylazine
F10	Avian	1:250 dilution	Nebulize	QD-BID	G	1197	Nebulize as antifungal, antichlamydophilial and antibacterial
F10	Avian	1:200 dilution	Topical	QD	G	1302	Daily spray onto feathers for yeast and supplement to feather picking therapy
F10	Avian	1:125 dilution	Nebulize	TID	B	1465	
F10	Avian	1:125 Dilution	Nebulize	BID	G	1268	Add omega interferon, prevents secondary infection
F10	Raptor	1:250 Dilution	Nebulize	BID-TID	G	742	Add itraconazole, 15 to 30 minutes per day for mild aspergillosis
Fatty Acids, Omega	Psittacine	0.22 ml/kg	PO	NL	E	1756	Add aspirin, use < 6:1 ratio omega 6:omega 3, use until renal histology normalizes for renal disease
Fatty Acids, Omega	Psittacine	0.11 ml/kg	PO	QD	G	1263	Mix 5:1 N-6:N-3 omega fatty acids for glomerulonephritis or acute pancreatitis
Febantel	Ostrich	20	NL	NL	C	283	
Febantel	Ostrich	5	PO	NL	G	481	
Febantel	Pigeon	30	PO	q3w	A	888	For ascarids and capillaria
Fenbendazole	Anseriformes	20	PO	Once	D	1150	Control nematodes

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Fenbendazole	Anseriformes	5-10	PO	QD	E	111, 1473	
Fenbendazole	Avian	25	NL	QD	D	1221	Repeat in 2 weeks for capillariasis
Fenbendazole	Avian	100	NL	q2w	D	1221	For capillariasis
Fenbendazole	Avian	2	PO	QD	D	1470	For liver parasitic disease
Fenbendazole	Avian	20-50	PO	q10d	E	111	For ascarids
Fenbendazole	Avian	20-50	PO	QD	E	111	For flukes and capillaria
Fenbendazole	Avian	50	PO	QD	E	1617	For ascarids, may need 5-day treatment for capillaria, repeat in 2 weeks,
Fenbendazole	Avian	20-100	PO	Once	G	57	Avoid during breeding season and molting
Fenbendazole	Bird, Aquatic	22	NL	NL	E	1478	
Fenbendazole	Bird, Aquatic	50	PO	QD	E	1503	Repeat in 2 weeks for ascarids
Fenbendazole	Bird, Aquatic	100	PO	q2w	E	1503	
Fenbendazole	Bustard	30	PO	Once	E	1240	
Fenbendazole	Bustard, Houbara	25	PO	Once	G	932	Prophylaxis for round worms
Fendendazole	Cockatiels	N/A			G		May have lethal reaction
Fenbendazole	Crane	100	PO	QD	E	629	Repeat q2w as needed
Fenbendazole	Crane	50-100	PO	QD	E	1361	Repeat in 2 weeks for gapeworms and capillarids
Fenbendazole	Falcon	20	PO	QD	E	1240	For *Serratospiculum*
Fenbendazole	Finch	10-25	PO	QD	E	1572	
Fenbendazole	Grouse	0.5-0.7	Feed	QD	C	704	For *Trichostrongylus*
Fenbendazole	Gull	50	PO	QD	E	1357	For capillariasis
Fenbendazole	Gull	25	PO	q2w	E	1357	For ascaridiasis
Fenbendazole	Ostrich	15	PO	NL	B	524	With or without resorantel
Fenbendazole	Ostrich	30	PO	NL	B	526	Add resorantel
Fenbendazole	Ostrich	15	PO	NL	G	481	
Fenbendazole	Parakeet, Australian	50	Gavage	Once	E	1186	For roundworms
Fenbendazole	Partridge	8	Feed	QD	C	704	For *Capillaria*
Fenbendazole	Partridge	12	Feed	Once	C	704	For *Syngamus, Heterakis*, ascarids
Fenbendazole	Penguin	50	PO	NL	E	1478	For nematodes and flukes
Fenbendazole	Pheasant	12	PO	Once	C	706	
Fenbendazole	Pheasant	8	PO	QD	C	706	
Fenbendazole	Pigeon	16	PO	Once	C	704	
Fenbendazole	Pigeon	20	PO	QD	C	706	For gastrointestinal roundworms
Fenbendazole	Pigeon	20-50	PO	QD	E	1240	For capillariasis
Fenbendazole	Pigeon	8	PO	Once	E	1240	At 8 weeks of age
Fenbendazole	Pigeon	7.5	PO	Once	E	1432	
Fenbendazole	Pigeon	7.5-20	PO	Once	G	232	
Fenbendazole	Pigeon	10-12	PO	QD	G	260	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Fenbendazole	Pigeon	50	PO	QD	G	590	
Fenbendazole	Pigeon	15	PO	q10d	G	1208	For ascarids or *Capillaria,* not for use during molting
Fenbendazole	Psittacine	20-50	PO	r2w	E	763	
Fenbendazole	Psittacine	15	PO	QD	E	1240	May medicate feed for 7 days
Fenbendazole	Psittacine	100	PO	q2w	E	1613	For ascaridiasis
Fenbendazole	Psittacine	50	PO	QD	E	1613	For capillariasis
Fenbendazole	Raptor	10-50	NL	q10d	E	1132	For ascaridiasis
Fenbendazole	Raptor	30-50	NL	Once	E	1132	For flukes
Fenbendazole	Raptor	25	PO	QD	E	1159	For nematodes
Fenbendazole	Ratite	15	PO	NL	G	418	With or without resorantel
Fentanyl Citrate	Avian	0.2	SC	NL	B	1341	Some hyperactivity for first 15 to 30 minutes
Ferric Subsulfate	Avian	N/A	Topical	PRN	E	1473	For hemorrhage
Fipronil	Avian	7.5-15	NL	Once	G	1729	For larger birds
Fipronil	Canary	N/A	Topical	Once	G	1729	Spray on gloved finger and rub under wings and on dorsal and ventral surfaces of body for red mites
Fipronil	Dove, Duck	7.5-15	NL	Once	G	1729	
Fipronil	Raptor	Light Spray	Topical	Once	B	1568	For wild injured raptors with ectoparasites
Fipronil	Raptor	N/A	Topical	Once	E	1359	For ectoparasites
Flax Seed Oil	Amazon, Yellow-naped	0.022 ml/kg body wt.	Feed	QD	G	1033	Dilute 1:4 with corn oil
Flax Seed Oil	Avian	0.1-0.2 ml/kg	PO	NL	G	1682	Mix 1:4 with corn oil prior to dosing for renal disease
Floxacillin	Hawk	125	PO	BID	G	1110	
Floxacillin	Raptor	250	PO	NL	E	1463	For bacterial infections
Flubendazole	Anseriformes	0.24 mg/kg feed	Feed	QD	D	1150	Control nematodes
Flubendazole	Galliformes	30-60 g/ton feed	Feed	QD	C	704, 706	For gastrointestinal roundworms, gapeworms and tapeworms

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Fluconazole	Amazon Parrot	20	IV-PO	q2d	D	1330	For ketoconazole-resistant *Candida*, tissue-based yeasts, eye, cerebral spinal, long-term *Aspergillus*
Fluconazole	Avian	8	NL	QD	D	1221	For cryptococcosis
Fluconazole	Avian	20	PO	q2d	D	1221	For candidiasis
Fluconazole	Avian	5	IV-PO	QD	D	1330	
Fluconazole	Avian	20	IV-PO	q2d	D	1330	Adult doase
Fluconazole	Avian	4-6	IV-PO	BID	D	1330	Juvenile dose
Fluconazole	Bird, Aquatic	15	PO	QD	E	1478	For prevention of aspergillosis, may also nebulize
Fluconazole	Bird, Aquatic	8	PO	BID	E	1503	
Fluconazole	Bird, Aquatic	15	PO	BID	E	1559	
Fluconazole	Chicken	100	Gavage	BID	G	1348	For macrorhabdosis (formerly megabacteria or avian gastric yeast), toxic to budgerigars
Fluconazole	Penguin, African	30	PO	BID	G	128	
Fluconazole	Pigeon	5-10	PO	BID	G	56	Prophylaxis
Fluconazole	Psittacine	5-15	PO	BID	D	1446	
Fluconazole	Psittacine	2-5	PO	QD	G	697	
Fluconazole	Raptor	2-5	PO	QD	E	1240	For generalized aspergillosis
Fluconazole	Raptor	5	PO	QD	E	1359	For candidiasis
Fluconazole	Raptor	2-5	PO	QD	E	1400	Aspergillosis
Flucytosine	Avian	250	PO	BID	D	1221	For candidiasis
Flucytosine	Avian	50-100	PO	BID	D	674, 1330	For *Aspergillus* or *Candida* prophylaxis, add amphotericin B or azoles for therapy
Flucytosine	Avian	40-50	PO	TID	E	626	Add IV and IT amphotericin B for aspergillosis therapy
Flucytosine	Avian	50-60	PO	BID	E	626	Aspergillus prophylaxis
Flucytosine	Avian	20-50	PO	BID	E	1492	
Flucytosine	Bird, Aquatic	50-60	NL	BID	E	1503	Aspergillosis prophylaxis
Flucytosine	Bird, Aquatic	100	PO	TID	E	1503	For aspergillosis adjunct therapy
Flucytosine	Hornbill, Great	250	PO	BID	G	1587	
Flucytosine	Penguin, African	250	PO	BID	G	1353	May give long-term
Flucytosine	Psittacine	75-120	PO	BID	D	1330	For *Aspergillus* or *Candida* prophylaxis, add amphotericin B or azoles for therapy
Flucytosine	Psittacine	100-250	PO	BID	D	1330	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Flucytosine	Raptor	120	PO	BID	B	1178	Add IT and IV amphotericin B + rifampin for aspergillosis
Flucytosine	Raptor	60	PO	BID	D	1330	For aspergillus or *Candida* prophylaxis birds > 500 g, add H1176amphotericin B or azoles for therapy
Flucytosine	Ratite	80-100	PO	BID	G	1308	
Flucytosine	Swan	50-75	PO	BID	G	86, 697	
Flumazenil	Anseriformes	0.05	IV	PRN	D	1503, 1533	Add atipamezole to reverse ketamine + medetomidine + midazolam
Flumazenil	Avian	0.1	NL	PRN	E	1481	Reverse benzodiazepines
Flumazenil	Avian	0.05	IV	PRN	E	1533	
Flumazenil	Pigeon	0.04	IM	PRN	G	1644	Reverse midazolam
Flunixin Meglumine	Anseriformes	1	SC	QD	E	1190	
Flunixin Meglumine	Anseriformes	1-10	IM	NL	E	1150	For joint lameness
Flunixin Meglumine	Avian	1-10	IM	QD	D	1533	May cause renal ischemia at higher dosages
Flunixin Meglumine	Bird, Aquatic	1-10	IM	QD	E	1503	May cause renal ischemia at higher dosages
Flunixin Meglumine	Bustard, Houbara	2	NL	NL	G	1594	Swelling in foot deformity of neonate
Flunixin Meglumine	Duck, Mallard	5	NL	NL	A	1339	Residual physiological effect 12 hours
Flunixin Meglumine	Duck, Mallard	5	IM	BID	A	1490	For frostbite
Flunixin Meglumine	Pigeon	1-10	IM-IV	QD	E	1432	
Flunixin Meglumine	Psittacine	1-10	IM	NL	E	1240	
Flunixin Meglumine	Raptor	2-10	IM	QD	D	1400	
Fluoxetine HCl	Avian	1	NL	NL	E	1186	For behavioral problems such as feather picking
Fluoxetine HCl	Avian	1	NL	QD	E	1477	For feather picking
Fluoxetine HCl	Psittacine	0.4	PO	QD	D	1446	For behavioral feather picking
Furaltadone	Avian	0.47 mg./kcal	PO	QD	E	1180	
Furaltadone	Pigeon	15-20	PO	QD	E	565	
Furaltadone	Pigeon	0.125 g/L	Drink	QD	E	1492, 1650	For salmonellosis
Furaltadone	Pigeon	0.4 g/L	Drink	QD	E	1240	Add tetracycline for trichomoniasis and hexamitiasis, not for adults feeding young < 10 days old
Furazolidone + Furaltadone	Pigeon	15-20	PO	QD	F	1061	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Furosemide	Amazon, Yellow-naped	0.15	PO	BID	G	1033	
Furosemide	Avian	0.165	IM	BID	G	1309	Diuretic, lories sensitive
Furosemide	Avian	0.1-0.2	IM-PO	QD-BID	E	1152	For diuresis, risk of toxicosis especially in small birds
Furosemide	Avian	0.15-2	IM	QD-BID	E	1650	For ascites and edema, not for lorikeets
Furosemide	Avian	0.15-2	IM-SC	QD-BID	E	111, 1470	
Furosemide	Avian	0.1-0.2	IM	BID	E	1431	
Furosemide	Avian	1-2	NL	QD-BID	E	1470	For congestive heart failure with liver disease
Furosemide	Pigeon	0.5-3	PO	BID-TID	G	590	
Furosemide	Pigeon	2.2	IM-IV-PO	BID	G	590	
Furosemide	Psittacine	0.15-2	IM-SC	QD-BID	E	1240	
Furosemide	Raptor	1.5	IM	QID	E	1240	
Furosemide	Raptor	1	IM-IV	PRN	E	1359	
Furosemide	Raptor	1.5	IM	TID-QID	E	1400	
Furosemide	Raptor	1.5	IM	PRN	G	234	
Furosemide	Secretary Bird	2.2	IM	BID	G	41	
Fusidate Sodium	Avian	2% ointment	Topical	BID	E	1240	For mild/early bumblefoot and other skin lesions, may penetrate intact skin, effective against *S. aureus*
Gadopentetate Dimeglumine	Avian	0.25 mmol/kg	IV	Once	G	1754	For head magnetic resonance imaging studies
Gentamicin Sulfate	Avian	10	IT	QD	E	704	
Gentamicin Sulfate	Avian	5-10	IM	BID-TID	E	704	
Gentamicin Sulfate	Avian	5 g/L	Nebulize	TID	E	741, 924	For sinusitis or airsacculitis
Gentamicin Sulfate	Avian	4	IM	NL	E	924	For *Pseudomonas*, nephrotoxic
Gentamicin Sulfate	Avian	5-10	NL	QD	E	1170	Relatively renal toxic particularly if dehydrated
Gentamicin Sulfate	Avian	5 g/L	Flush	BID	E	1183	Nasal or sinus
Gentamicin Sulfate	Avian	2.5	IM	BID	E	1434	
Gentamicin Sulfate	Avian	3-5	IM	QD-BID	E	1492	
Gentamicin Sulfate	Avian	3-5	IM	QD-BID	E	1650	For resistant organisms
Gentamicin Sulfate	Bustard	5	IM	QD-BID	E	1240	For first 3 days of life to prevent yolk sac infections after assisted hatch
Gentamicin Sulfate	Chicken	1.5	IM	TID	A	715	
Gentamicin Sulfate	Chicken	2	IM	BID	A	1056	For roosters

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Gentamicin Sulfate	Chicken	10	IM	BID-QID	A	1143	
Gentamicin Sulfate	Crane	5	IM	TID	A	458	
Gentamicin Sulfate	Duck	5	IM	QID	A	847	
Gentamicin Sulfate	Duck, Eagle, Macaw, Owl	10	IM	BID-QID	A	1143	
Gentamicin Sulfate	Emu	5	IM	TID	A	418	
Gentamicin Sulfate	Emu	2.5	IM	BID	A	847	
Gentamicin Sulfate	Hawk, Red-tailed	2.5	IM	TID	A	714	
Gentamicin Sulfate	Owl, Great-horned	2.5	IM	TID	A	714	
Gentamicin Sulfate	Pheasant	5	IM	TID	A	458	
Gentamicin Sulfate	Pigeon	10	IM	QID	A	847	
Gentamicin Sulfate	Pigeon	20	IM	BID-QID	A	1143	
Gentamicin Sulfate	Psittacine	10	IM	BID-TID	E	565	
Gentamicin Sulfate	Psittacine	5-10	IT	QD	E	741	Add carbenicillin for pneumonia
Gentamicin Sulfate	Quail	10	IM	QID	A	458	
Gentamicin Sulfate	Raptor	2.5	IM	TID	A	565, 714	
Gentamicin Sulfate	Ratite	5	IM	QD	G	1308	May cause visceral gout
Gentamicin Sulfate	Turkey	3	IM	BID	A	880	
Gentian Violet	Macaw, Hyacinth	N/A	Topical	NL	E	1240	Excellent for crop or skin fold candidiasis in chicks
Glipizide	Cockatoo, Rose-breasted	0.05 g/L	Drink	QD	G	1726	Crush tablet into water for diabetes mellitus
Glucosamine Sulfate	Avian	10 drops/kg	PO	TID	E	1205	For joint disease
Glyburide	Cockatiel	0.00125-0.0025 g/L	Drink	QD	G	1720	For polyuria/polydipsia
Glycerin	Raptor	< 5 ml/kg	PO	NL	E	1463	For impaction, may give cloacally
Glycopyrrolate	Pigeon	0.01	IM	PRN	G	1644	Add midazolam for anesthesia premed
Gonadotropin, Chorionic	Avian	500-1000 ug/kg	IM	QD	E	1474	Treat chronic egg laying, inject day one, on day 3 if egg, on day 7 if second egg, use up to 3-6 w
Gonadotropin, Chorionic	Goose	0.5 mg TD	Parenteral	q3-4d	B	894	Increase spermiation and early puberty in ganders
Griseofulvin	Ostrich	30-50	Drink	QD	G	531, 1308	
Griseofulvin	Pigeon	10	PO	QD	E	1240	For dermatophytosis

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Halofuginon HBr	Poultry		Feed	QD	C	705	For coccidial prophylaxis
Halofuginon HBr	Chicken	3 g/ton food	Feed	QD	E	564	Seven day slaughter withdrawal
Haloperidol	Avian	0.1	PO	BID	D	1475	
Haloperidol	Avian	0.17	Drink	QD	E	704	Body weight less than 1 kg, gradually increase dosage to 0.9 mg/kg
Haloperidol	Avian	0.2	Drink	QD	E	704	
Haloperidol	Avian	0.4	Drink	QD	E	704	Maintenance dose
Haloperidol	Avian	0.4	NL	NL	E	1186	For behavioral problems such as feather picking
Haloperidol	Avian	1-2	IM	q2-3w	E	1431	
Haloperidol	Avian	0.2-0.4	PO	QD	E	1431	
Haloperidol	Avian	0.1-0.15	NL	NL	E	1477	For self-mutilation
Haloperidol	Avian	0.2	PO	BID	E	1492	For birds < 1 kg for feather picking and self-mutilation, most effective in cockatoos
Haloperidol	Parrot, Quaker	0.08	PO	BID	E	1492	For feather picking and self-mutilation
Haloperidol	Psittacine	0.15	PO	BID	E	111	Control feather picking and mutilation, birds over 1 kg
Haloperidol	Psittacine	1-2	IM	q2-3w	E	111	Control feather picking and mutilation
Haloperidol	Psittacine	0.2	PO	BID	E	111	
Haloperidol	Psittacine	0.4	PO	QD	E	1240	For feather picking
Halothane	Avian	N/A	IH	PRN	E	1617	Induction, maintain with 1-1.5%
Halothane	Pigeon	N/A	IH	PRN	E	1432	1.5-3% maintenance
Heparin Sodium	Avian	40-50 U/kg	IV	Once	E	1151	Add aminophylline, dexamethasone Na phosphate, fluids, oxygen for polytetraflouroethylene gas poison
Heparin Sodium	Avian	0.1 ml of 1000U/ml	Nebulize	QD	G	1705	Add to 6.5 ml saline
Heparin Sodium	Avian	1.0 ml of 1000U/ml	Topical	QD	G	1705	Add aloe vera
Heparin Sodium	Swan	55	IV	Once	G	1707	Emaciated near death
Hepasan	Avian	1 ml/kg	PO	QD	G	1035	For liver disease
Hepasan	Psittacine	1 ml/kg	PO	QD	G	1722	Liver support
Hetastarch	Avian	10-15 ml/kg	IV	TID	E	1650	For emergency care, reduce crystalloid fluids
Hetastarch	Avian	10-15 ml/kg	IV	TID	E	1151	For hypoproteinemia

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Hetastarch	Hawk, Red-tailed	10 ml/kg	IV	NL	G	1626	Supportive therapy post-trauma
Hexyl Pyropheoborbide-a	Hornbill, Great	0.3	IV	q2m	G	1587	For squamous cell carcinoma
Hyaluronidase	Avian	150 U/L fluids	SC	NL	G	1103	Mix with fluids to aid in absorption
Hyaluronidase	Avian	1.5 KIU/L	Flush	NL	G	1719	Mix into nasal and sinus flushes
Hyaluronidase	Avian	1.5 KIU/L	Nebulize	NL	G	1719	Mix into nebulization fluid
Hyaluronidase	Avian	1.5 KIU/L	IV	NL	G	1719	For egg yolk stroke
Hydrocortisone Sodium Succinate	Avian	10	IV	NL	E	1470	For lead toxicity and severe infection with liver disease
Hydroxychloroquine Sulfate	Pigeon	4 tablets/L	Drink	NL	G	590	Change daily
Hydroxyzine HCl	Avian	2	PO	TID	G	1324	For pruritis
Hydroxyzine HCl	Psittacine	2	PO	TID	D	1446	For hypersensitivity-triggered feather picking
Hypericum	Avian	10 drops/kg	PO	TID	E	1205	For feather picking, anxiety
Imidacloprid	Raptor	7	IT-IV	NL	E	1359	For babesiosis
Imidocarb HCl	Raptor	7	NL	q3w	E	1359	For babesiosis
Imiquimod	Amazon, Blue-fronted	cream	Vent	q2-3d	B	1281	For cloacal papillomatosis, apply to cloacal surface
Indomethacin	Avian	0.4	IM	NL	E	1232	
Indomethacin	Chicken	2	PO	NL	A	772	Effective for 8-10 hours
Indomethacin, Copper	Avian	0.4	IM	QD	E	1492	For acute/chronic inflammation from trauma, infection or organ dysfunction
Insulin, NPH	Avian	0.01-0.1 U TD	Parenteral	NL	E	1650	For diabetes mellitus in birds larger than budgerigar
Insulin, NPH	Avian	0.07 U/kg	IM	QD	G	1325	For diabetes, monitor blood glucose
Insulin, NPH	Budgerigar	0.067-3.3 U/kg	IM	BID	E	1326	For diabetes
Insulin, NPH	Budgerigar	0.002 U TD	IM	PRN	G	58	
Insulin, NPH	Psittacine	0.01-0.1 U TD	IM	PRN	G	58, 88	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Insulin, Regular	Avian	0.0013 mg/kcal	IM	QD	E	1180	
Insulin, Zinc Suspension	Toucan, Toco	0.1-0.5 U TD	IM	PRN	G	58	
Interferon, Alpha	Avian	15 KIU/m²	SC	q2d	E	1470	Give 15, 000 Units/m² of body surface for lymphosarcoma
Interferon, Omega	Avian	1000 KIU TD	IM	q2d	B	1465	Add F10® for circovirus prevention and limited effectiveness therapy of 90 days
Interferon, Omega	Avian	1000 KIU TD	Parenteral	QD-BID	G	1268	Add F10® for circovirus therapy
Iodine, Lugol's	Avian	4 drops solution/L	Drink	NL	E	1186	Used to keep drinking water uncontaminated
Iodine, Lugol's	Raptor	4 drops/L 7% soln	Drink	QD	E	1612	
Iodophor	Avian	N/A	Topical	PRN	E	1151	Use 0.5-1% solution for wound debridement and flushing
Iodophor	Avian	N/A	Topical	NL	E	1240	For wounds, apply and wash off after 3 minutes, very safe
Iodophor	Raptor	N/A	Topical	NL	E	1400	For wounds, wash off within 5 minutes
Iohexol	Avian	700-800	IV	NL	E	1182	Use 70-80% iodine concentration for urography
Iohexol	Cockatiel	240	PO	NL	G	1623	
Iopamidol	Avian	0.8-1.2	IN	NL	G	1270	Use 20% concentration, sinus contrast study, flush with saline when completed
Ipronidazole	Avian	0.125 g/L	Drink	QD	E	111, 741	
Ipronidazole	Pigeon	0.250 g/L	Drink	QD	G	590	Change daily
Ipronidazole	Psittacine	0.232 g/L	PO	NL	E	111	
Iron Dextran	Anseriformes	10	IM	QW	D	1150	For hemapoiesis
Iron Dextran	Avian	10	IM	QW	G	62	
Iron Dextran	Psittacine	10	IM	QW-q10d	E	1240	Hemapoiesis
Iron Dextran	Raptor	10	IM	Once	E	1400	For iron-deficiency anemia
Isoflurane	Avian	N/A	IH	PRN	E	1483	Maintain 1.5-3%
Isoniazid	Amazon, Yellow-cheeked	30	NL	QD	G	713	Add ethambutol + rifampin, antibacterial
Isoniazid	Avian	30	PO	QD	D	1470	Add ethambutol + rifampin, antibacterial
Isoniazid	Avian	15	PO	BID	E	1473	Antiparasitic

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Isoxuprine HCl	Raptor	5-10	PO	QD	E	1359, 1400	For wing tip edema
Itraconazole	Amazon Parrot	10	PO	QD	A	16	Dissolve in 0.1N HCl, dilute with orange juice to tube feed
Itraconazole	Amazon, Blue-fronted	5-10	PO	BID	A	1739	Continue once daily for duration of therapy
Itraconazole	Anseriformes	5-10	PO	BID	E	1473	
Itraconazole	Anseriformes	10	PO	QD	G	708	
Itraconazole	Avian	10	PO	QD	A	577	
Itraconazole	Avian	5-10	NL	BID	E	1239	
Itraconazole	Bird, Aquatic	20	PO	QD	E	1503	For aspergillosis prophylaxis
Itraconazole	Bird, Aquatic	15	PO	QD	E	1559	For aspergillosis prophylaxis
Itraconazole	Chicken	10	PO	BID	A	1376	Add miconazole for epidermal *Aspergillus* and *Alternaria* cysts after surgical removal
Itraconazole	Cockatiel	5	PO	QD	D	1330	Dissolve 100 mg capsule 2 ml HCl + 18 ml orange juice for candidiasis
Itraconazole	Crane	10	PO	BID	B	1189	Add enrofloxacin + clotrimazole for aspergillosis
Itraconazole	Crane	5-10	PO	BID	E	1361	
Itraconazole	Emu	5-10	PO	QD	G	16	
Itraconazole	Falcon	10-20	PO	QD	G	746	For aspergillosis
Itraconazole	Gull	5	PO	QD	E	1357	
Itraconazole	Penguin	10	NL	BID	G	1353	Aspergillosis therapy long-term
Itraconazole	Pigeon	6	PO	BID	A	709	
Itraconazole	Pigeon	26	PO	BID	A	709	For respiratory disease, this dosage may be toxic
Itraconazole	Psittacine	10	NL	QD	A	1424	For aspergillosis
Itraconazole	Psittacine	5-10	PO	BID	D	1446	For cutaneous cryptococcosis or aspergillosis
Itraconazole	Raptor	5-10	NL	BID	E	1188	Continue QD for 4 months for asymptomatic cases of aspergillosis
Itraconazole	Raptor	10	PO	BID	G	742	Add amphotericin B or enilconazole + F10® for mild aspergillosis
Itraconazole	Ratite	6-10	PO	QD	G	1308	Reduce dosage if neurologic signs occur
Ivermectin	Anseriformes	0.2	PO-SC	Once	D	1150, 1190, 1240	Control nematodes and nasal or duck leeches
Ivermectin	Avian	0.2	IM	r10d	E	741, 924	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Ivermectin	Bird, Aquatic	0.4-0.8	NL	NL	E	1559	For parasites
Ivermectin	Budgerigar	0.2	Topical	NL	E	1240	Injectable form may be toxic
Ivermectin	Budgerigar	0.4	IM-PO	q10d	G	1309	
Ivermectin	Bustard, Houbara	0.3	PO	Once	G	932	
Ivermectin	Canary	0.2	PO	QW	G	861	For air sac mites and scaly leg mites
Ivermectin	Crane	0.2	IM-PO	q2w	E	629	Nematodes and mites
Ivermectin	Emu	0.2	SC	NL	G	1308	Effective against chandlerellosis
Ivermectin	Falcon	2-3	IM	Once	B	1427	For serratospiculiasis
Ivermectin	Falcon	1	SC	Once	B	1585	For capillariasis
Ivermectin	Passerine	0.2-0.4	PO-Topical	QW	E	1438	For mites
Ivermectin	Penguin	0.4-1	PO	NL	E	1478	For nematodes
Ivermectin	Pigeon	0.3	SC	NL	G	57	
Ivermectin	Pigeon	0.2	PO	NL	G	232	
Ivermectin	Pigeon	0.5-1	Parenteral-PO	NL	G	260	
Ivermectin	Psittacine	0.2	IM-PO-SC	q2-4w	D	1446	
Ivermectin	Raptor	0.4	SC	NL	G	57	
Ivermectin	Raptor	0.2	SC	Once	G	234	
Ivermectin	Raptor	0.2	IM	NL	G	1409	For capillariasis
Ivermectin	Ratite	0.2	SC	NL	G	1308	
Ivermectin	Stork, Saddle-billed	0.2	PO	QD	G	1645	For microfilaria
Kanamycin Sulfate	Anseriformes	20-40	IM	BID	G	739	For large waterfowl
Kanamycin Sulfate	Avian	10-20	IM	BID	E	565	
Kanamycin Sulfate	Ratite	20-40	IM	BID	G	739	
Kaolin + Pectin	Psittacine	2 ml/kg	PO	BID-QID	G	632	Neonate dosage
Kaolin + Pectin	Raptor	67 drops/kg	PO	TID	E	1612	For diarrhea
Ketamine HCl	Amazon Parrot	10-20	IM	PRN	E	243	Add xylazine
Ketamine HCl	Anseriformes	25	NL	PRN	D	1403	Add diazepam, supplement at 15 mg/kg PRN
Ketamine HCl	Anseriformes	10	IV	PRN	D	1503	Add medetomidine + midazolam, atipamezole + flumazenil reverses, good anesthesia 30 minutes
Ketamine HCl	Avian	5-25	NL	PRN	C	1392	
Ketamine HCl	Avian	10-30	IM	PRN	E	243	Add diazepam, higher dosage for smaller bird
Ketamine HCl	Avian	20	IM	PRN	E	704	Add midazolam
Ketamine HCl	Avian	5-10	IM	PRN	E	704	Add medetomidine
Ketamine HCl	Avian	10-25	IM	PRN	G	53	Add midazolam

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Ketamine HCl	Avian	10-50	IM	PRN	G	53	Add diazepam
Ketamine HCl	Bird, Aquatic	3-8	IM	PRN	E	1503	Add medetomidine
Ketamine HCl	Bird, Aquatic	2.5-5	IV	PRN	E	1503	Add xylazine
Ketamine HCl	Canary	100-200	IM	PRN	G	1328	
Ketamine HCl	Cassowary	2.2	IV	PRN	E	1533	Add xylazine
Ketamine HCl	Chicken	20-100	IM	PRN	G	1328	
Ketamine HCl	Chicken	4-5	Parenteral	PRN	G	1718	Add butorphanol + medetomidine, preanesthetic for isoflurane
Ketamine HCl	Cockatiel	25	IM	PRN	E	243	Add xylazine
Ketamine HCl	Cockatoo, Palm	5	IM-SC	PRN	E	924	Sedative
Ketamine HCl	Duck, Mallard	8.8	IM	PRN	B	764	Add medetomidine + midazolam, 33% mortality
Ketamine HCl	Duck, Pekin	20	IV	Once	B	718	
Ketamine HCl	Emu	3	NL	PRN	C	1392	Tranquilization
Ketamine HCl	Emu	2.2	IV	PRN	E	1533	Add xylazine
Ketamine HCl	Falcon, Peregrine	30	IV	PRN	D	1401	Add diazepam, supplement at 5 mg/kg IV PRN
Ketamine HCl	Finch	100-200	IM	PRN	G	1328	
Ketamine HCl	Galliformes	3	Parenteral	PRN	G	1718	Add butorphanol + medetomidine, preanesthetic for isoflurane
Ketamine HCl	Goose	5-10	IM-IV	PRN	B	1577	Add medetomidine
Ketamine HCl	Goose	20-50	IM	PRN	G	1328	
Ketamine HCl	Guinea Fowl	25	IM	PRN	B	553	Add xylazine
Ketamine HCl	Hawk	25-30	IM	PRN	E	243	Add xylazine
Ketamine HCl	Hawk	4-5	Parenteral	PRN	G	1718	Add butorphanol + medetomidine, preanesthetic for isoflurane, improves quality of anesthesia
Ketamine HCl	Hawk, Broad-winged	30-40	IV	PRN	B	1092	Combine with diazepam
Ketamine HCl	Heron	20	NL	PRN	G	1356	
Ketamine HCl	Ostrich	5	IV	PRN	B	555	Add xylazine + alphaxalone/alphadolone
Ketamine HCl	Ostrich	2	IM	PRN	B	1628	Add medetomidine
Ketamine HCl	Owl	10	IM-SC	PRN	E	924	Sedative
Ketamine HCl	Pelican	7	IM	PRN	G	595	
Ketamine HCl	Pigeon	20	IM	PRN	B	779	Follow with propofol
Ketamine HCl	Pigeon	5	IM	PRN	B	1588	Add medetomidine, usually moderate to heavy sedation
Ketamine HCl	Poultry	50	IM	PRN	E	4	
Ketamine HCl	Poultry	30	IM-SC	PRN	E	924	Sedative
Ketamine HCl	Psittacine	3-7	IM	PRN	B	1577	Add medetomidine

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Ketamine HCl	Psittacine	2-5	IV	PRN	B	1577	Add medetomidine
Ketamine HCl	Psittacine	10	NL	PRN	D	1401	Add diazepam, supplement at 5 mg/kg PRN
Ketamine HCl	Psittacine	3-7	IM	PRN	E	649	Add medetomidine
Ketamine HCl	Psittacine	2.5-7	IV	PRN	E	649	Add medetomidine
Ketamine HCl	Psittacine	20	IM-SC	PRN	E	924	Sedative
Ketamine HCl	Raptor	25-35	IM	PRN	B	1176	For laparoscopy
Ketamine HCl	Raptor	2-4	IV	PRN	B	1577	Add medetomidine
Ketamine HCl	Raptor	3-5	IM	PRN	B	1577	Add medetomidine
Ketamine HCl	Ratite	3-7	IM	PRN	E	4	Add medetomidine
Ketamine HCl	Ratite	2-4	IV	PRN	E	243	Proceed with xylazine
Ketamine HCl	Ratite	3-20	NL	PRN	E	1386	May add diazepam or xylazine if desired
Ketamine HCl	Rhea	6.6	IM	PRN	G	1628	Add butorphanol + medetomidine
Ketamine HCl	Spoonbill	20	NL	PRN	G	1356	
Ketamine HCl	Stork	20	NL	PRN	G	1356	
Ketamine HCl	Swan	4	IV	PRN	G	1291	Add medetomidine, gas anesthesia premed
Ketamine HCl	Swan, Mute	12.5	IV	PRN	E	1190	Add xylazine
Ketamine HCl	Vulture	10	IM	PRN	B	568	Add xylazine
Ketoconazole	Amazon Parrot	30	PO	BID	B	68	
Ketoconazole	Amazon, Yellow-naped	12.5	PO	BID	G	740	
Ketoconazole	Avian	10-30	PO	BID	D	1330	Dissolve 50 mg tablet 0.2 ml HCl + 0.8 ml water, mix with acid juice, lactulose, methylcellulose
Ketoconazole	Avian	10-30	PO	BID	D	1743	For systemic yeast
Ketoconazole	Bird, Aquatic	25	PO	BID	E	1503	For candidiasis and aspergillosis therapy not responding to nystatin
Ketoconazole	Canary	0.2 g/kg soft food	Feed	QD	D	1330	
Ketoconazole	Canary	0.2 g/L	Drink	QD	E	695	
Ketoconazole	Ostrich	10	PO	QD	G	401	
Ketoconazole	Passerine, Small	1 g/L	Drink	NL	E	1187	
Ketoconazole	Passerine, Small	0.2 g/kg food	Feed	Once	E	1187	
Ketoconazole	Pigeon	30	PO	BID	A	68	
Ketoconazole	Pigeon	3	PO	QD	E	704	Tables crushed and water suspension
Ketoconazole	Pigeon	3	Gavage	QD	E	1240	For resistant candidiasis, *Mucor* and *Penicillium*

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Ketoconazole	Psittacine	25	PO	BID	E	763	For candidiasis or adjunct for aspergillosis therapy, disguise taste in food
Ketoconazole	Psittacine	10	PO	BID	E	1240	For resistant candidiasis, *Mucor* and *Penicillium*
Ketoconazole	Raptor	15	PO	BID	E	1132	
Ketoconazole	Raptor	25	IM	BID	E	1240	For resistant candidiasis, *Mucor* and *Penicillium*
Ketoconazole	Raptor	60	PO	BID	E	1240	"
Ketoconazole	Raptor	25	PO	BID	E	1359	
Ketoconazole	Ratite	5-10	PO	QD	G	1309	
Ketoconazole	Swan	12.5	PO	BID	D	1330	Dissolve 50 mg tablet 0.2 ml HCl + 0.8 ml water, mix with acid juice, lactulose, methylcellulose
Ketoprofen	Anseriformes	1	IM	QD	D	1150	For pain relief or arthritis
Ketoprofen	Avian	2	IM	NL	D	1240, 1533	
Ketoprofen	Avian	1-4	IM	NL	E	1167	
Ketoprofen	Duck	5	IM	NL	B	1339	Effective 30 to 70 minutes, given during isoflurane anesthesia
Ketoprofen	Pigeon	1 mg TD	IM-SC	QD-BID	E	1240	Pain relief, arthritis, antiinflammatory
Ketoprofen	Psittacine	2	IM	NL	E	1240	"
Ketoprofen	Quail	2	PO	NL	B	1674	
Lactulose	Amazon Parrot	0..07 g TD	PO	BID-TID	G	1309	For hepatic encephalopathy
Lactulose	Avian	0.3 ml/kg	PO	NL	E	1492	For liver disease
Lactulose	Psittacine	200	PO	BID-TID	G	632	Neonate dosage
Lasalocid	Chicken	75	Feed	QD	B	958	For coccidial prophylaxis
Lasalocid	Galliformes	90-120 g/ton	Feed	QD	C	705	For coccidial prophylaxis
Leuprolide Acetate	Amazon, Yellow-naped	0.75-1	Parenteral	q2-3w	G	1033	Depot formulation
Leuprolide Acetate	Avian	0.1	IM	NL	D	1475	For feather picking triggering by excessive reproductive behavior
Leuprolide Acetate	Avian	0.5-1	IM	q2w	E	1151	After egg binding
Leuprolide Acetate	Avian	0.1-0.14	Parenteral	NL	E	1474	Treat chronic egg laying
Leuprolide Acetate	Avian	0.052-0.156	IM	NL	E	1570	Use 30-day depot formulation for egg chronic egg laying
Leuprolide Acetate	Avian	0.75	IM	q2w	G	994	For birds 300 g or less
Leuprolide Acetate	Avian	0.5	IM	q2w	G	994	For birds more than 300 g

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Leuprolide Acetate	Avian	0.75	IM	q2w	G	1278	Induce molt
Leuprolide Acetate	Psittacine	0.1	IM	QD	D	1446	For sexually-triggered feather picking
Leuprolide Acetate	Raptor	0.25	IM	q2-3w	E	1359	For cystic ovaries
Levamisole HCl	Anseriformes	25-50	SC	Once	D	1150, 1190	Control nematodes
Levamisole HCl	Avian	2	PO	QD	D	1470	For liver parasitic disease
Levamisole HCl	Avian	2	IM-SC	q2w	E	111	
Levamisole HCl	Avian	10-20	PO	q2w	E	704	
Levamisole HCl	Avian	8	IM-SC	r10d	E	741	Not for debilitated birds or lories, use 13.65% injectable
Levamisole HCl	Avian	10-20	PO-SC	q2w	E	1434	
Levamisole HCl	Avian	5	IM-SC	q2w	E	1473	
Levamisole HCl	Avian	0.8 g/L	Drink	QD	E	1479	For roundworms, cecal worms and hair worms
Levamisole HCl	Avian	40-50	PO	NL	E	1492	For most worms
Levamisole HCl	Crane	25	PO	q2w	E	1361	Chick dosage for intestinal strongyles, acarids and capillarids
Levamisole HCl	Crane	40	PO	q2w	E	1361	For intestinal strongyles, ascarids and capillarids
Levamisole HCl	Finch	40-50	PO	NL	E	1650	For most worms except tapeworm, particularly effective on acuaria in Australian finch
Levamisole HCl	Galliformes	40	PO	Once	E	1503	
Levamisole HCl	Galliformes	40	PO	Once	E	1526	
Levamisole HCl	Magpie	30	SC	NL	G	1609	
Levamisole HCl	Ostrich	30	PO	NL	B	526	Add resorantel
Levamisole HCl	Parakeet, Australian	15	Gavage	q10d	G	1309	For intestinal nematodes, use injectable
Levamisole HCl	Pheasant	25	SC	NL	G	678	Histomoniasis therapy
Levamisole HCl	Pigeon	15	IM	QD	E	704	
Levamisole HCl	Pigeon	0.18 g/L	Drink	QD	E	704	
Levamisole HCl	Pigeon	40	PO	Once	E	1503	
Levamisole HCl	Pigeon	40	PO	Once	E	1526	
Levamisole HCl	Pigeon	10-20	PO	Once	G	232	Repeat in 2 weeks if needed
Levamisole HCl	Pigeon	40	PO	Once	G	260	
Levamisole HCl	Poultry	18-36	PO	Once	G	1332	
Levamisole HCl	Poultry	1.25-2.5	PO-SC	NL	E	111	
Levamisole HCl	Psittacine	20	IM	r2w	E	763	Emesis common
Levamisole HCl	Psittacine	20-50	PO	QD	E	1240	Loft treatment for capillariasis and ascariasis, follow up with parenteral therapy

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Levamisole HCl	Psittacine	2-5	IM-SC	q10d	E	1240	Immunostimulant, low therapeutic index
Levamisole HCl	Quail	25	SC	NL	G	678	Histomoniasis therapy
Levamisole HCl	Quail	20	SC	NL	G	678	Anthelmintic
Levamisole HCl	Raptor	10-20	PO-SC	QD	E	1400	Narrow therapeutic margin
Levamisole HCl	Raptor	15	PO-SC	NL	E	1463	Anthelmintic
Levamisole HCl	Raptor	2	SC	q4-6d	D	1612	Immune stimulant
Levamisole HCl	Ratite	30	IM-PO	QM	G	1308	Begin at 1 month of age, juvenile dose for *Libyastrongylus douglassi*
Levamisole HCl	Ratite	30	IM-PO	q3m	G	1308	Adult dose for *Libyastrongylus douglassi*
Levothyroxine Sodium	Avian	20 ug/kg	PO	QD-BID	E	111	
Levothyroxine Sodium	Avian	1	IM-PO	NL	E	924	For severe feather disorders and lipomas
Levothyroxine Sodium	Avian	20-100 ug/kg	PO	BID	E	1431	Monitor levels to avoid thyrotoxicosis
Levothyroxine Sodium	Avian	20 ug/kg	PO	QD-BID	E	1473	
Levothyroxine Sodium	Avian	0.0008-0.0025 g/L	Drink	QD	E	1492	For goiter and hypothyroidism
Levothyroxine Sodium	Avian	0.02-0.04	PO	QD	G	58	
Levothyroxine Sodium	Avian	0.02	PO	QD-BID	G	88	
Levothyroxine Sodium	Budgerigar	0.004-0.008 g/L	Drink	QD	D	1446	For lipomatous growth and xanthomas
Levothyroxine Sodium	Budgerigar	0.003 g/L	Drink	QD	G	1309	For hypothyroidism, stir water and offer for 15 minutes, repeat daily
Levothyroxine Sodium	Psittacine	0.020-0.1	PO	BID	E	1240	For hypothyroidism
Levothyroxine Sodium	Raptor	1	PO	QD	E	1160	To induce molting
Lidocaine	Avian	2-3	SC	NL	B	1346	Local infiltration
Lidocaine	Raptor	2	SC	NL	E	1400	Dilute to 0.4%, infuse locally, use with caution in small birds
Lidocaine	Raptor	10	SC	NL	E	1463	Dilute to 0.2% for large bird local analgesia
Lincomycin HCl	Amazon Parrot	75	PO	BID	E	111, 1431	
Lincomycin HCl	Avian	50	PO	BID	E	1234	For bumblefoot
Lincomycin HCl	Avian	0.2 g/L	Drink	QD	E	1431	For infected foot lesions
Lincomycin HCl	Avian	100	IM	BID	E	1431	For infected foot lesions
Lincomycin HCl	Hawk	50	PO	BID	G	1110	Therapy prior to tendon surgical repair
Lincomycin HCl	Pigeon	25-50	IM	NL	G	260	
Lincomycin HCl	Pigeon	35-50	PO	QD	G	590	
Lincomycin HCl	Poultry	2.2 mg/kg food	Feed	QD	C	564	
Lincomycin HCl	Poultry	0.017 g/L	Drink	QD	C	564	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Lincomycin HCl	Psittacine	100	IM	BID	E	1240	For Gram positive bumblefoot, dermatitis and mycoplasmal respiratory infection.
Lincomycin HCl	Raptor	110	PO	QD	E	741	For skin disease
Lincomycin HCl + Spectinomycin HCl	Avian	50	PO	QD	E	565	
Lincomycin HCl + Spectinomycin HCl	Chicken	50	PO	QD	B	1065	
Lincomycin HCl + Spectinomycin HCl	Falcon	30	IM	TID	E	1027	Broad spectrum for mycoplasma and salmonella
Lincomycin HCl + Spectinomycin HCl	Pigeon	50	PO	QD	F	1061	
Lithium Carbonate	Avian	6-25	NL	BID	E	1477	For avian behavior problems, mood stabilizer and antipsychotic, no longer used in birds
LL-E19020	Poultry	0.01-50	Feed	QD	G	564	Improves feed efficiency
Maduramicin	Avian	7.5	PO	QD	E	1650	For *Plasmodium*
Maduramicin	Chicken	5	Feed	QD	B	958	For coccidial prophylaxis
Magnesium Sulfate	Anseriformes	500-1000	PO	QD	E	1240	Increase gut motility and aid passage of lead
Magnesium Sulfate	Bird, Aquatic	500-1000	PO	NL	E	1503	Precipitates lead and cathartic
Magnesium Sulfate	Budgerigar	53	IM	PRN	B	1084	Add chloral hydrate + pentobarbital sodium, reduce dose 15 to 20% in debilitated birds
Magnesium Sulfate	Canary	53	IM	PRN	B	1084	See above
Magnesium Sulfate	Chicken	53	IM	PRN	B	1084	See above
Magnesium Sulfate	Crow, Eastern	53	IM	PRN	B	1084	See above
Magnesium Sulfate	Eagle, Golden	40.3	IM	PRN	B	1086	See above
Magnesium Sulfate	Gull (Herring, laughing)	53	IM	PRN	B	1084	See above
Magnesium Sulfate	Hawk, Marsh	32.4	IM	PRN	B	1086	See above
Magnesium Sulfate	Ostrich	1.25-30 ml TD	PO	NL	G	401	
Magnesium Sulfate	Pigeon	53	IM	PRN	B	1084	Add chloral hydrate + pentobarbital sodium, reduce dose 15 to 20% in debilitated birds
Magnesium Sulfate	Raptor	250-1000	PO	QD	E	1400	For oral heavy metal poisoning

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Magnesium Sulfate	Raptor	500-1000	PO	QD	E	1240	Increase gut motility and aid passage of lead
Malathion	Avian	1 g/L	Topical	NL	E	1479	Remove birds and treat environment for flea, lice, mange and mite treatment and aid in tick control
Malathion	Avian	1 g/L	Dip	NL	E	1479	For flea, lice, mange and mite treatment and aid in tick control
Malathion	Raptor	5% mixture	Topical	q10d	G	1411	For chewing lice
Mannitol	Avian	0.5	IV	QD	E	111	Give slowly
Marbofloxacin	Buzzard, Common	2	IV	BID	A	778, 1428	
Marbofloxacin	Buzzard, Common	10	PO	QD	A	979	
Marbofloxacin	Chicken	2	PO	QD	A	765	
Marbofloxacin	Raptor	10	IM-PO	QD	E	1433	
Marbofloxacin	Raptor	15-20	IM-PO	QD	G	1068	For sinusitis
Mebendazole	Anseriformes	5-15	PO	QD	D	1,150,111	Control *Syngamus*
Mebendazole	Avian	25	PO	QD	E	1492, 1650	For all intestinal worms
Mebendazole	Canary	10	PO	BID	G	57	Avoid during breeding season
Mebendazole	Fowl, Domestic	0.06 g/kg food	Feed	QD	E	1051	
Mebendazole	Pigeon	5-6	PO	QD	G	260	
Mebendazole	Psittacine	25	PO	BID	E	111, 1473	
Mebendazole	Raptor	25	PO	BID	E	111	
Meclofenamic Acid	Avian	2.2	PO	QD	D	1533	
Meclofenamic Acid	Avian	2.2	PO	QD	G	201, 1320	
Meclofenamic Acid	Avian	2.2	PO	QD	E	1573	
Meclofenamic Acid	Bird, Aquatic	2.2	PO	QD	E	1503	
Medetomidine HCl	Amazon, Yellow-crowned	2	IM	PRN	B	1629	Sedation produced not reliable, reverse with atipamezole
Medetomidine HCl	Anseriformes	0.05	IV	PRN	D	1503	Add ketamine + midazolam
Medetomidine HCl	Anseriformes	0.6-0.85	IM	PRN	E	1231	Add ketamine
Medetomidine HCl	Anseriformes	0.06	Parenteral	PRN	G	1718	Add butorphanol + ketamine, preanesthetic for isoflurane
Medetomidine HCl	Avian	0.1	IM	NL	G	704, 1320	
Medetomidine HCl	Bird, Aquatic	0.025-0.075	IV	PRN	E	1503	Add ketamine, reverse with atipamezole
Medetomidine HCl	Bird, Aquatic	0.05-0.1	IM	PRN	E	1503	Add ketamine, reverse with atipamezole

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Medetomidine HCl	Cassowary, Double-wattled	0.26-0.31	IM	PRN	B	1229	For restraint of captive birds
Medetomidine HCl	Chicken	0.1	IM	PRN	E	1573	Sedation
Medetomidine HCl	Chicken	0.06-0.08	Parenteral	PRN	G	1718	Add butorphanol + ketamine, preanesthetic for isoflurane
Medetomidine HCl	Duck, Mallard	0.44	IM	PRN	B	764	Add ketamine + midazolam, 33% mortality
Medetomidine HCl	Goose	0.1-0.2	IM-IV	PRN	B	1577	Add ketamine
Medetomidine HCl	Hawk	0.06-0.08	Parenteral	PRN	G	1718	Add butorphanol + ketamine
Medetomidine HCl	Ostrich	0.08	IM	PRN	B	1628	Add ketamine
Medetomidine HCl	Ostrich	0.1	IM	PRN	E	1573	Sedation
Medetomidine HCl	Pigeon	0..08	IM	PRN	B	1588	Add ketamine, usually moderate to heavy sedation
Medetomidine HCl	Pigeon	0.2	IM	PRN	B	1588	Variable light sedation
Medetomidine HCl	Psittacine	0.075-0.15	IM	PRN	B	1577	Add ketamine
Medetomidine HCl	Psittacine	0.05-0.1	IV	PRN	B	1577	Add ketamine
Medetomidine HCl	Psittacine	0.06-0.085	IM	PRN	E	1240	Add ketamine
Medetomidine HCl	Raptor	0.1-0.3	IM	NL	E	1400	Can be reversed, combined with ketamine for anesthesia
Medetomidine HCl	Raptor	0.025-0.075	IV	PRN	B	1577	Add ketamine
Medetomidine HCl	Raptor	0.05-0.1	IM	PRN	B	1577	Add ketamine
Medetomidine HCl	Raptor	0.15-0.35	IM	PRN	E	1240	Add ketamine
Medetomidine HCl	Raptor	0.2	NL	PRN	E	1433	Add ketamine, reverse with atipamezole
Medetomidine HCl	Ratite	0.05-0.15	IM	PRN	E	4	Add ketamine
Medetomidine HCl	Rhea	0.73	IM	PRN	G	1628	Add butorphanol + ketamine
Medetomidine HCl	Swan	0.15	IV	PRN	G	1291	Add ketamine, gas anesthesia premed
Medroxyprogesterone Acetate							Lower dose for larger birds. Side effects: lethargy, inappetance, polydipsia, fatty liver
Medroxyprogesterone Acetate	Avian	25	IM-SC	QD	E	111	For 700 g bird
Medroxyprogesterone Acetate	Avian	40	IM-SC	QD	E	111	For 150 to 300 g bird
Medroxyprogesterone Acetate	Avian	50	IM-SC	QD	E	111	For <150 g bird
Medroxyprogesterone Acetate	Avian	30	IM-SC	QD	E	111	For 300 to 700 g bird
Medroxyprogesterone Acetate	Avian	5-25	IM-SC	QD	E	111	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Medroxyprogesterone Acetate	Avian	5-10	IM-SC	NL	E	704	For persistent egg laying
Medroxyprogesterone Acetate	Avian	2-10	PO-SC	QW	E	924	Repeat after 4 months for ovulation suppression
Medroxyprogesterone Acetate	Psittacine	25-50	IM-SC	q4-6w	E	1240	Lower dose for larger birds. Side effects: lethargy, inappetance, polydipsia, fatty liver
Mefloquine HCl	Passerine	30	PO	PRN	E	1438	Give BID on day 1, give daily on days 2 and 3 and once per week
Megestrol Acetate	Avian	2.5	NL	QD	E	1431	Then continue q3-4d for feather picking with hormonal etiology
Megestrol Acetate	Cockatoo, Rose-breasted	0.2 g/L	Drink	NL	G	1609	For self-mutilation
Megestrol Acetate	Cockatoo, Rose-breasted	0.2 g/kg food	Feed	Once	G	1609	For self-mutilation
Meloxicam	Avian	0.1	IM-PO	QD	E	1167, 1431	
Meloxicam	Avian	0.1-0.2	IM	QD	E	1431	Long course well tolerated
Meloxicam	Avian	0.1 - 1.0	PO	QD	G	1341	
Meloxicam	Avian	0.1	NL	QD	G	1484	
Meloxicam	Avian	0.1	PO	QD	G	1671, 1675	
Meloxicam	Avian	0.1	PO	QD	E	1431	Long course well tolerated
Meloxicam	Crane	0.5	PO	BID	G	1341	
Meloxicam	Raptor	0.1-0.2	IM-PO	QD	E	1400	For arthritis and other painful conditions
Meloxicam	Raptor	0.1	IM-PO	QD	E	1359, 1433	
Meperidine HCl	Raptor	3-5	NL	QID	G	1481	
Meperidine HCl	Raptor	3-5	NL	NL	G	1483, 1484	
Metformin (glucophage)	Avian	100 mg/L water	Drink	QD	G	T. Lightfoot, 2005	Hyperglycemia
Methocarbamol	Avian	50	IV	NL	G	201	
Methohexital Sodium	Chicken	5-10	IV	PRN	G	497	
Methohexital Sodium	Dove	5 g/L bait	Feed	Once	G	1763	Corn is usual bait
Methohexital Sodium	Duck	5-10	IV	PRN	G	498	
Methohexital Sodium	Goose	5-10	IV	PRN	G	498	
Methohexital Sodium	Poultry	4-8	IV	PRN	G	496	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Methoprene	Raptor	0.02 g/L	Topical	NL	E	1068	Add permethrin + piperonyl butoxide for ectoparasites
Methylprednisolone Acetate	Avian	0.5-1	IA-IM	NL	D	1533	
Methylprednisolone Acetate	Bird, Aquatic	0.5-1	IA-IM	NL	E	1503	
Methylprednisolone Acetate	Raptor	5	IM	QW	E	1463	
Methylprednisolone Sodium Succinate	Avian	0.5-1	IM-IV	NL	D	1554	For shock
Metoclopramide HCl	Avian	0.3	IM-IV-PO	NL	E	111	
Metoclopramide HCl	Avian	0.5	IM	BID-TID	E	1151	To improve motility, this dosage not for macaws
Metoclopramide HCl	Avian	0.5	IM-IV-PO	NL	E	1473	
Metoclopramide HCl	Avian	0.5	Parenteral	NL	E	1650	Increase gastric motility
Metoclopramide HCl	Hawk, Red-tailed	0.5	IM	NL	G	1626	Supportive therapy post-trauma
Metoclopramide HCl	Lorikeet, Rainbow	0.5	SC	BID	G	1620	For vomiting
Metoclopramide HCl	Macaw	0.1	IM-PO	NL	E	1151	To improve motility- Use low dose
Metoclopramide HCl	Ostrich	0.1	IV	NL	G	1255	Add psyllium for proventricular obstruction
Metoclopramide HCl	Psittacine	0.5	IM-IV-PO	TID	E	1240	Antiemetic and for gut stasis
Metoclopramide HCl	Psittacine	0.5	NL	BID	G	1263	For ileus
Metoclopramide HCl	Raptor	0.5-2	IM-SC	q4-6h	E	1359	For gastrointestinal stasis
Metomidate HCl	Avian	10	IM	PRN	E	1130	
Metomidate HCl	Duck, Mallard	17	PO	PRN	B	1095	
Metomidate HCl	Eagle, African Hawk	15.2	IM	PRN	G	1611	
Metomidate HCl	Eagle, Tawny	9.6	IM	PRN	G	1611	
Metomidate HCl	Hawk, African Harrier	14.4-16	IM	PRN	G	1611	
Metomidate HCl	Ostrich	15-20	IM	PRN	G	481	With or without azaperone for captive animal
Metomidate HCl	Owl, Barn	10.2	IM	PRN	G	1611	
Metomidate HCl	Raptor	10	IM	PRN	E	1463	Smaller dosages for vultures
Metomidate HCl	Raptor	5	IM	PRN	E	1463	For sedation
Metomidate HCl	Turkey	17 g/L whole corn	Feed	Once	E	1386	Safer than chloralose

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Metronidazole	Avian	25	PO	BID	D	1221	For clostridiosis
Metronidazole	Avian	25-50	PO	QD	E	704, 924	
Metronidazole	Avian	0.2 g/L	Drink	NL	E	1180	
Metronidazole	Avian	50	PO	NL	E	1434	
Metronidazole	Avian	5	IM	BID	E	1434	
Metronidazole	Avian	10-30	PO	BID	E	1650	For anaerobic infections
Metronidazole	Avian	10	IM	QD	E	1650	For anaerobic infections
Metronidazole	Avian	50	PO	QD	G	55	
Metronidazole	Avian	20	PO-SC	QD	C	705	For trichomoniasis
Metronidazole	Avian	10-30	PO	BID	D	1470	For liver parasitic disease
Metronidazole	Avian	10	IM	QD	D	1470	For liver parasitic disease
Metronidazole	Canary	0.1 g/kg food	Feed	Once	E	1187	
Metronidazole	Chicken	30	PO	BID	A	795	
Metronidazole	Falcon	50	PO	QD	E	1027	For trichomoniasis
Metronidazole	Ostrich	1.25 g/L	Drink	QD	E	627	Antihistomoniasis
Metronidazole	Passerine	0.1 g/L	Drink	QD	E	1437	Add drug to food at the same time
Metronidazole	Passerine	0.1 g/kg soft food	Feed	QD	E	1437	Add drug to drink at the same time
Metronidazole	Pigeon	200	PO	QD	E	704	
Metronidazole	Pigeon	200-250	PO	QD	G	260	
Metronidazole	Poultry	20	Drink	QD	C	705	
Metronidazole	Poultry	110	PO	BID	G	585	
Metronidazole	Psittacine	10-30	PO	BID	E	111, 1473	
Metronidazole	Psittacine	25	PO	BID	E	763	
Metronidazole	Raptor	30-50	PO	QD	E	1359	For trichomoniasis, may increase dosage to 100 mg/kg
Metronidazole	Raptor	50	PO	QD	E	1400, 1433	For trichomoniasis
Metronidazole	Raptor	100	PO	NL	E	1463	For trichomoniasis
Metronidazole	Raptor	50	PO	QD	G	61, 234	
Metronidazole	Raptor	10-40	PO	QD	G	94	Juvenile dosage
Metronidazole	Raptor	100	PO	QD	G	1068	
Metronidazole	Raptor	30-65	PO	QD	G	1409	For trichomoniasis
Metronidazole	Ratite	20-25	PO	BID	G	1308	For anaerobic infections
Metronidazole	Stork, Saddle-billed	42	PO	BID	G	1645	Add amikacin + enrofloxacin
Miconazole Nitrate	Amazon, Red Lored	2 mg	Nebulize	QD	G	700	Add acetylcysteine
Miconazole Nitrate	Avian	20	IV	TID	D	1330	
Miconazole Nitrate	Avian	10-20	IM	QD	E	565	
Miconazole Nitrate	Avian	20	IM	NL	E	924	For candidiasis, *Aspergillus, Mucor* and *Penicillium*

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Miconazole Nitrate	Avian	40	IV	NL	E	924	For candidiasis, *Aspergillus, Mucor and Penicillium*
Miconazole Nitrate	Falcon	cream	Topical	BID	G	1589	
Miconazole Nitrate	Psittacine	20	IM-IV	QD	E	704	
Miconazole Nitrate	Raptor	10	IM-IV	QD	E	704	
Midazolam HCl	Anseriformes	2	IV	PRN	D	1503, 1533	Add ketamine + medetomidine
Midazolam HCl	Anseriformes	1-2	IV	PRN	E	1503	"
Midazolam HCl	Avian	1-2	IM-IV	NL	E	1573	"
Midazolam HCl	Avian	1	IM	NL	G	201	
Midazolam HCl	Avian	1-2	IM-IV	NL	G	1320	Use with an analgesic for pain control
Midazolam HCl	Avian	4	IM	PRN	E	704	Add ketamine
Midazolam HCl	Avian	0.2	IM	PRN	E	1181, 1231	Add ketamine, smooth induction and recovery
Midazolam HCl	Duck, Mallard	4	IM	Once	G	1468	Pre-gas anesthesia
Midazolam HCl	Emu	0.4	IV	PRN	G	418	
Midazolam HCl	Ostrich	0.15	IV	PRN	G	418	
Midazolam HCl	Pheasant, Ring-necked	1	IM	PRN	G	1644	Anesthesia premed
Midazolam HCl	Pigeon	0.5	IM	PRN	B	1588	Add medetomidine, usually moderate to heavy sedation
Midazolam HCl	Pigeon	2	IM	PRN	G	1644	Add glycopyrrolate for anesthesia premed, reverse with flumazenil
Midazolam HCl	Psittacine	0.2	IM-SC	PRN	E	1240	Use in combination with ketamine
Midazolam HCl	Raptor	0.5-1	IM-IV	TID	E	1240, 1400	Control seizures
Midazolam HCl	Swan	0.1-1	NL	PRN	G	1291	May add butorphanol, premed for gas anesthesia, lower dosage if add butorphanol
Midazolam HCl	Turkey	0.1-1	NL	PRN	G	1291	"
Milbemycin Oxime	Galliformes	2	PO	QM	B	588	
Milbemycin Oxime	Galliformes	2	PO	NL	E	1492, 1650	For *Heterakis, Capillaria* and ascarids
Milk Thistle	Amazon Parrot	2 drops mixture TD	PO	BID	G	1488	Mixture is 5 drops extract per 7.5 ml lactulose, useful for treating birds on all seed diet
Mineral Oil	Avian	5 ml/kg	Gavage	Once	E	1309, 1554	To flush GI - laxative
Minocycline HCl	Budgerigar	5.5 g/kg seed	Feed	QD	A	731	Hulled medicated millet

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Minocycline HCl	Hawk, Red-shouldered	5	PO	BID	G	1179	Add levamisole + *Staphylococcus* toxoid for bumblefoot
Miporamicin	Poultry	0.1 g/kg	Feed	QD	G	564	
Monensin Sodium	Chicken	0.1 g/kg	Feed	QD	B	869	Preventive for coccidiosis
Monensin Sodium	Chicken	100	Feed	QD	B	958	For coccidial prophylaxis
Monensin Sodium	Chicken	100-120 g/ton	Feed	QD	C	705	For coccidiosis
Monensin Sodium	Crane	0.099 g/kg food	Feed	Once	E	111	
Monensin Sodium	Galliformes	0.099 g/kg food	Feed	QD	E	1473	
Monensin Sodium	Pigeon	1-1.2 g/kg feed	Feed	Once	E	1492	For coccidiosis
Monensin Sodium	Pigeon	1-1.2 g/kg food	Feed	QD	E	1650	For coccidia
Monensin Sodium	Turkey	90-100 g/ton	Feed	QD	C	705	For coccidiosis
Morphine Sulfate	Galliformes	2.5-3	IM-SC	NL	E	111, 1473	
Moxidectin	Avian	0.2-0.8	NL	Once	D	1221	For capillariasis
Moxidectin	Cardinal, Red-crested	0.2	PO	NL	G	1585	For capillariasis
Moxidectin	Falcon	1	PO	QW	G	1068	For *Serratospiculum*, *Capillaria*, *Physlaoptera* and *Acanthocephalae*
Moxidectin	Finch	2	PO	NL	E	1479	For lice, mites and nematodes
Moxidectin	Penguin	0.2	PO	NL	E	1478	For nematodes
Moxidectin	Pigeon	0.25 mg TD	PO	NL	D	1479	For lice, mites and nematodes
Moxidectin	Psittacine	0.2	Gavage	Once	D	1479	For lice, mites and nematodes
Moxidectin	Raptor	0.2	PO	QD	E	1359	For capillariasis
Moxidectin	Sparrowhawk	0.2	SC	NL	G	1585	For nematodiasis
Mullein	Avian	10 drops/kg	PO	TID	E	1205	Respiratory, wound support
Mycobacterium Vaccae Vaccine	Crane, Whooping	0.05 ml TD	ID	q2m	G	207	
Nalbuphine HCl	Guinea Fowl	1-2	IM	q3h	G	252	
Naloxone HCl	Avian	0.05-0.25	IM-IV	PRN	E	1573	
Naloxone HCl	Psittacine	0.05-0.25	IM-IV	NL	G	1320	Use slow IV
Naloxone HCl	Ratite	0.05-0.067	NL	PRN	G	418	
Naltrexone HCl	Avian	1.5	Drink	NL	E	704	Behavior modification, feather picking
Naltrexone HCl	Ostrich	300 mg TD	IV	PRN	B	521	Reverse carfentanil

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Naltrexone HCl	Psittacine	1.5	PO	BID	E	1240	Prevent self-mutilation
Nandrolone	Psittacine	0.4	IM-SC	q3w	E	1240	For chronic and debilitating disease, may cause liver disease
Nandrolone	Raptor	0.4	IM-SC	q3w	E	1240	"
Narasin	Chicken	70	Feed	QD	B	958	For coccidial prophylaxis
Narasin	Chicken	60-80 mg/kg food	Feed	QD	E	564	Add nicarbazin, 5-day slaughter withdrawal, prophylactic
Neomycin Sulfate	Avian	1.25 g/L	Drink	NL	E	111	
Neomycin Sulfate	Avian	10	PO	QD	E	565	
Neomycin Sulfate	Avian	20	PO	NL	E	924	Gram negative, poor absorption
Neomycin Sulfate	Chicken	11	Drink	BID	C	1307	For bacterial enteritis
Neomycin Sulfate	Chicken	.077-.154 g/kg food	Feed	QD	C	1307	For bacterial enteritis
Neomycin Sulfate	Duck	.077-.154 g/kg food	Feed	QD	C	1307	For bacterial enteritis
Neomycin Sulfate	Duck	11	Drink	BID	G	1307	For bacterial enteritis
Neomycin Sulfate	Passerine	0.2 g/kg soft food	Feed	QD	E	1437	Add drug to drink at the same time
Neomycin Sulfate	Passerine	0.2 g/L	Drink	QD	E	1437	Add drug to food at the same time
Neomycin Sulfate	Poultry	0.035-0.08 g/L	Drink	QD	C	564	Prophylactic
Neomycin Sulfate	Poultry	38.5-154 mg/kg food	Feed	QD	C	564	
Neomycin Sulfate	Poultry	11	PO	QD	C	564	
Neomycin Sulfate	Raptor	15	PO	NL	E	1463	For bacterial infections
Netobimin	Avian	0.035-0.08 g/L	Drink	QD	E	1492	For tapeworms, ascarids and Heterakis
Netobimin	Bird, Aquatic	0.35 g/L	Drink	QD	E	1503	
Netobimin	Raptor	20	PO	QD	B	1568	
Nicarbazin	Chicken	20-50 mg/kg food	Feed	QD	E	564	Add narasin, 5-day slaughter withdrawal, prophylactic
Niclosamide	Avian	50	PO	q2w	E	111	
Niclosamide	Avian	150-250	PO	NL	G	57	
Niclosamide	Avian	5 g/kg food	Feed	QD	G	57	
Niclosamide	Crane	250	NL	q2w	G	596	
Niclosamide	Falcon	200	PO	Once	E	1027	For tapeworms
Niclosamide	Finch	500	Feed	QW	E	111	
Niclosamide	Ostrich	100	PO	q6w	G	401	
Niclosamide	Ostrich	50	PO	NL	G	481	
Niclosamide	Pelican, Brown	103-160	PO	Once	B	1089	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Niclosamide	Pigeon	75	PO	NL	G	57	
Niclosamide	Raptor	156	PO	Once	D	1612	For cestodiasis
Niclosamide	Raptor	125	PO	NL	E	1068	For cestodes
Nifursol	Poultry	77 mg/kg food	Feed	QD	C	564	Prophylactic
Nifursol	Turkey	75 mg/kg food	Feed	QD	E	564	Nutritional use, 5-day slaughter withdrawal
Nifursol	Turkey	50 g/ton	Feed	QD	C	705	For histomoniasis
Nithiamide	Raptor	40	PO	NL	E	1463	For trichomoniasis
Nitrofurazone	Avian	1.25 ml/L	Drink	QD	E	741	For coccidial infections
Nitrofurazone	Chicken	55 mg/kg food	Feed	QD	E	564	5-day slaughter withdrawal, prophylactic
Nitrofurazone	Lorikeet	0.625 cc/L	Drink	NL	E	111	Dosage based on 9.2% powder
Nitrofurazone	Poultry	55 mg/kg food	Feed	QD	C	564	
Nitrofurazone	Psittacine	1.25 cc/L	Drink	NL	E	111	Dosage based on 9.2% powder
Nitromide	Chicken	250 mg/kg food	Feed	QD	E	564	Add sulfanitran + roxarsone, 5-day slaughter withdrawal
Nitroxinil	Galliformes	24	Drink	QD	C	706	For gapeworms
Nordihydroquaiaretic	Avian	2 drops/kg	PO	BID	E	1205	Use with caution in egg layers or w/ hepatic disease
Norfloxacin	Chicken	10	PO	BID	A	789	
Norfloxacin	Chicken	8	PO	QD	A	830	
Norfloxacin	Chicken	20-40	Drink	NL	A	840	For *Hemophilus paragallinarum* infection.
Norfloxacin	Goose	10	PO	BID	A	789	
Norfloxacin	Ostrich	30	Drink	QD	G	562	
Norfloxacin	Poultry	8	PO	QD	G	564	
Norfloxacin	Turkey	10	PO	QID	A	789	
Norfloxacin	Turkey	10	SC	QD	A	1020	
Nortriptyline HCl	Psittacine	0.016 g/L	Drink	NL	E	111	Control feather picking
Nosiheptide	Chicken	5-10 mg/kg	Feed	QD	G	564	Improves feed efficiency
Novobiocin Sodium	Anseriformes	385 mg/kg food	Feed	Once	G	597	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Novobiocin Sodium	Duck, Pekin	0.3 g/kg food	Feed	Once	B	984	Prophylaxis for *Pasteurella multocida* in ducklings.
Nystatin	Anseriformes	300 KIU/kg	PO	BID	D	1150	For candidiasis
Nystatin	Avian	200 KIU/kg	Feed	QD	E	704	Soft food
Nystatin	Avian	0.05 g/kg food	PO	NL	E	924	For candidiasis
Nystatin	Avian	300 KIU/kg	PO	BID	E	1221	For candidiasis
Nystatin	Avian	200-300 KIU/kg	PO	BID	G	1373	For candidiasis
Nystatin	Avian	100 KIU/L	Drink	QD	G	1744	For candidiasis
Nystatin	Finch	333 KIU/kg	PO	QD	E	1572	For candidiasis
Nystatin	Hummingbird	100 KIU/ml	Topical	q4-6h	G	599	For mouth lesions
Nystatin	Hummingbird	25 KIU/L Nectar	Drink	QD	G	599	
Nystatin	Ostrich	2500 KIU/kg food	Feed	Once	G	283	
Nystatin	Ostrich	12.5 KIU/kg	PO	QD	G	401	
Nystatin	Ostrich	0.24 g/kg feed	Feed	QD	G	1254	For candidiasis
Nystatin	Ostrich	20-50 KIU/kg	PO	QD	G	1254	
Nystatin	Passerine	100 KIU/L	Drink	QD	D	1330	Add to food also, upper GI Candidiasis
Nystatin	Pigeon	20-100 KIU	PO	QD	E	704	
Nystatin	Pigeon	100-150 KIU/kg	PO	BID	G	590	
Nystatin	Poultry	110 mg/kg food	Feed	QD	C	564	Half dosage is prophylactic
Nystatin	Psittacine	300 KIU/kg	PO-Topical	BID-TID	D	1330	Upper GI candidiasis
Nystatin	Psittacine	333 KIU/kg	PO	TID	E	763	For candidiasis, use lesser dosage to diet for neonates on antibiotic therapy
Nystatin	Raptor	100 KIU/kg	PO	TID	D	1612	
Nystatin	Raptor	1 KIU/kg	PO	BID	E	1132	
Nystatin	Raptor	20 KIU/kg	PO	BID	E	1188	
Nystatin	Ratite	300 KIU/kg	PO	NL	G	418	
Nystatin	Ratite	100 KIU TD	PO	TID	G	594	
Nystatin	Turkey	0.055-0.11 g/kg food	Feed	QD	C	1307	
Oleandomycin Phosphate	Avian	1.17 mg/kcal	PO	QD	E	1180	
Oleandomycin Phosphate	Avian	0.58 mg/kcal	IM	QD	E	1180	
Oleandomycin Phosphate	Pigeon	25	IM	QD	E	565	
Oleandomycin Phosphate	Pigeon	50	PO	QD	E	565	
Ormetoprim + Sulfadimethoxine	Chicken	0.2 g/kg food	Feed	QD	E	564	5-day slaughter withdrawal, prophylactic

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Ormetoprim + Sulfadimethoxine	Crane	0.41 g/kg food	Feed	QD	E	1361	For coccidiosis
Ormetoprim + Sulfadimethoxine	Duck	0.22 g/kg	Feed	Once	B	825	For *Pasteurella multocida* in ducklings
Ormetoprim + Sulfadimethoxine	Duck	0.2-0.8 g/kg food	Feed	Once	B	860	For riemerellosis and colibacillosis
Ormetoprim + Sulfadimethoxine	Poultry	0.3-0.5 g/L	Drink	QD	G	585	
Ormetoprim + Sulfadimethoxine	Turkey	0.44 g/kg	Feed	Once	B	846	Prophylaxis for *Pasteurella multocida*
Ormetoprim + Sulfadimethoxine	Turkey	0.1 g/kg food	Feed	QD	E	564	5-day slaughter withdrawal, prophylactic
Oxacillin Sodium	Psittacine	100	PO	TID	D	1446	For staphylococcal dermatitis
Oxfendazole	Avian	10	NL	q2w	D	1221	For capillariasis
Oxfendazole	Avian	40	PO	QD	D	1470	For liver parasitic disease
Oxfendazole	Avian	4	PO	QD	D	1479	For most intestinal worms including tapeworms
Oxfendazole	Avian	0.1-0.2 g/L	Drink	QD	D	1479	For most intestinal worms including tapeworms
Oxfendazole	Avian	10	PO	QD	E	1650	For most intestinal worms
Oxfendazole	Avian	0.1-0.2 g/L	Drink	QD	E	1650	For most intestinal worms
Oxfendazole	Ostrich	5	PO	q3w	G	401	Begin at age 4 months, alternate therapeutic agent
Oxfendazole	Pigeon	10-40	PO	Once	G	590	
Oxfendazole	Raptor	10	PO	QW	G	1068	
Oxytetracycline	Amazon Parrot	22-58	IM	QD-BID	A	266	
Oxytetracycline	Anseriformes	2.5 g/L	Drink	QD	E	1240	For chlamydophilosis
Oxytetracycline	Anseriformes	50 mg TD	PO	QD	E	1240	
Oxytetracycline	Avian	5000 mg/kg	Feed	QD	E	704	Add citric acid, therapeutic dosage
Oxytetracycline	Avian	50-100	IM	QD	E	1431	
Oxytetracycline	Avian	50	IM	QD-BID	E	1492	
Oxytetracycline	Avian	100	PO	QD-BID	E	1492	
Oxytetracycline	Bird, Aquatic	100	SC	QD	E	1503	For respiratory infections
Oxytetracycline	Budgerigar	2 g/L	Nebulize	q4-6h	A	876	For respiratory disease
Oxytetracycline	Chicken	2.5 g/L	Drink	NL	A	802	Add oxytetracycline feed also
Oxytetracycline	Ostrich	10	IM	QD	G	401	Double dosage for birds below 5 kg
Oxytetracycline	Owl	16	IM	QD	A	55	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Oxytetracycline	Pheasant	43	IM	QD	A	55	
Oxytetracycline	Pigeon	50	IM-SC	QD	A	1067	
Oxytetracycline	Pigeon	0.133-0.444 g/L	Drink	QD	E	704	
Oxytetracycline	Pigeon	100	IM	q2d	E	704	
Oxytetracycline	Pigeon	20	IM	q2d	E	1240	Add tetracycline for birds over 700 g
Oxytetracycline	Pigeon	80	IM	q2d	E	1240	Add tetracycline for birds under 400 g
Oxytetracycline	Poultry	0.0265-0.106 g/L	Drink	QD	C	564	
Oxytetracycline	Poultry	6.25-200 mg TD	PO	QD	C	564	
Oxytetracycline	Poultry	110-220 mg/kg food	Feed	QD	C	564	Add neomycin
Oxytetracycline	Poultry	0.06-0.25 g/L	Drink	QD	C	705	
Oxytetracycline	Psittacine	58	IM	QD	A	55	
Oxytetracycline	Psittacine	50	IM	QD	E	1240	
Oxytetracycline	Psittacine	50-100	SC	q2-3d	E	1365	For chlamydophilosis
Oxytetracycline	Raptor	25-50	IM-PO	TD	E	1240	IM injection may cause muscle necrosis
Oxytetracycline	Turkey	2.5 g/L	Drink	NL	A	802	Add oxytetracycline feed also
Oxytetracycline	Turkey	2500 ppm	Feed	Once	A	802	Add oxytetracycline drinking water also
Oxytetracycline	Turkey	220 mg/kg food	Feed	QD	E	564	3-day slaughter withdrawal, prophylactic
Oxytetracycline Amphoteric	Amazon Parrot	60	IM	QD	A	847	
Oxytetracycline Amphoteric	Amazon, Blue-fronted	75	SC	q3d	E	703	For chlamydophilosis
Oxytetracycline Amphoteric	Anseriformes	200	IM	QD	D	1150	For pasteurellosis and other bacteria
Oxytetracycline Amphoteric	Avian	50	SC	q3-5d	E	1648	For respiratory infections, may cause muscle necrosis
Oxytetracycline Amphoteric	Bird, Aquatic	50	SC	q3-5d	E	1503	For respiratory infections
Oxytetracycline Amphoteric	Cockatoo, Goffin	50-100	IM-SC	q3d	A	380	
Oxytetracycline Amphoteric	Macaw, Blue and Gold	50-75	SC	q2-3d	E	1170	Causes irritation at injection site
Oxytetracycline Amphoteric	Ostrich	20	IM	q3d	G	401	Double dosage for birds below 5 kg
Oxytetracycline Amphoteric	Psittacine	50-100	IM-SC	q2-3d	E	565	
Oxytetracycline Amphoteric	Raptor	50-200	IM	q3-5d	E	1240	For pasteurellosis
Oxytetracycline Amphoteric	Turkey	150	SC	q3d	A	727	Long-lasting formulation

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Oxytocin	Anseriformes	3-5 IU/kg	IM	NL	D	1150	Egg binding
Oxytocin	Avian	0.5-1 IU TD	IM	NL	E	924	Use with calcium and vitamin A for egg expulsion
Oxytocin	Avian	5 U/kg	IV-SC	Once	E	1151	To help with egg expulsion, add calcium, fluids and heat
Oxytocin	Avian	2 IU/kg	NL	NL	E	1431	For egg binding, give calcium borogluconate also
Oxytocin	Ostrich	5 IU TD	IV	Once	B	1257	Give 2 to 4 minutes before semen collection
Paromomycin Sulfate	Avian	100	PO	BID	E	1474	For neonate with cryptosporidiosis
Penetran	Avian		Topical	NL	G	1486	For rashes, ulceration, local irritation, trauma, bites, burns, etc.
Penetran	Avian	2.5 ml/L spray	Topical	NL	G	1103	For picking or itching
Penicillamine	Anseriformes	55	PO	BID	D	1150	For heavy metal poisoning
Penicillamine	Avian	55	PO	BID	D	1221	For lead or zinc toxicosis, may use in conjunction with edetate calcium disodium
Penicillamine	Avian	52	PO	QD	F	1236	For copper toxicosis
Penicillamine	Avian	55	PO	BID	G	87	Repeat after 3 to 5 days
Penicillamine	Psittacine	50-55	PO	BID	E	1240	Chelation agent for copper, zinc, mercury and lead
Penicillamine	Raptor	55	PO	BID	E	1240	For heavy metal poisoning
Penicillin	Anseriformes	50 KIU/kg	Parenteral	NL	G	597	
Penicillin	Poultry	44 KIU/kg	PO	QD	C	564	Add streptomycin
Penicillin	Poultry	55-110 mg/kg food	Feed	QD	C	564	Prophylactic
Penicillin	Poultry	375 KIU/L	Drink	QD	C	564	
Penicillin	Poultry	110 mg/kg food	Feed	QD	C	564	
Penicillin	Poultry	2.65 mg/kg food	Feed	QD	E	564	Add streptomycin, nutritional use, no slaughter withdrawal
Penicillin	Raptor	<100 KIU/kg	IM-IV	NL	E	1463	Use crystalline formulation for bacterial infections
Penicillin G Benzathine	Rhea	100 KIU TD	SC	QD	G	582	Add tylosin
Penicillin G Benzathine	Turkey	100	IM	QD	A	385	Add penicillin G procaine for *Pasteurella multocida*

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Penicillin G Potassium	Avian	66-133 KIU/kg	IM	NL	F	1209	
Penicillin G Potassium	Falcon, Lanner	20 KIU/kg	IM	QID	B	1175	Post-surgical treatment of bumblefoot
Penicillin G Procaine	Galliformes	100	IM	q2d	E	111, 1473	
Penicillin G Procaine	Turkey	100	IM	Once	A	385	Loading dose, add penicillin G with procaine benzathine for *Pasteurella multocida*
Pentobarbital + Chloral Hydrate + Magnesium Sulfate	Avian	160-180	IM	PRN	F	1209	Dosage base on combined mg of pentobarbital/chloral hydrate/magnesium sulfate
Pentobarbital Sodium	Avian	5	IM	PRN	E	1130	
Pentobarbital Sodium	Avian	50	IM	PRN	F	1209	
Pentobarbital Sodium	Budgerigar	24	IM	PRN	B	1084	Add chloral hydrate + magnesium sulfate, reduce dose 15 to 20% in debilitated birds
Pentobarbital Sodium	Canary	24	IM	PRN	B	1084	See above
Pentobarbital Sodium	Chicken	16.2	IM	PRN	B	1080	Add thiopental sodium
Pentobarbital Sodium	Cockatoo, Major Mitchell's	100 mg TD	IV	NL	G	1590	
Pentobarbital Sodium	Cowbird	100-150 g/kg food	Feed	Once	B	1094	
Pentobarbital Sodium	Crane, European	24	IM	PRN	B	1084	Add chloral hydrate + magnesium sulfate, reduce dose 15 to 20% in debilitated birds
Pentobarbital Sodium	Duck, Mallard	22	PO	PRN	B	1095	
Pentobarbital Sodium	Duck, Northern Pintail	390	IV	Once	B	1593	Precede with ketamine by 15 minutes
Pentobarbital Sodium	Eagle, Golden	18.2	IM	PRN	B	1086	Add chloral hydrate + magnesium sulfate, reduce dose 15 to 20% in debilitated birds, fledgling
Pentobarbital Sodium	Emu	13.3	IV	PRN	G	283	Premedicate with diazepam
Pentobarbital Sodium	Galliformes	50-100	PO	PRN	E	1130	Use tablets
Pentobarbital Sodium	Goose	50-100	PO	PRN	E	1130	Use tablets
Pentobarbital Sodium	Gull, California	24	IM	PRN	B	1084	Add chloral hydrate + magnesium sulfate, reduce dose 15 to 20% in debilitated birds
Pentobarbital Sodium	Hawk	14.7	IM	PRN	B	1086	"
Pentobarbital Sodium	Penguin	33	NL	Once	G	595	
Pentobarbital Sodium	Pigeon	24	IM	PRN	B	1084	Add chloral hydrate + magnesium sulfate, reduce dose 15 to 20% in debilitated birds

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Pentobarbital Sodium	Quail, Japanese	25-50 g/kg food	Feed	Once	B	1094	
Pentobarbital Sodium	Raptor	< 20	PO	PRN	E	1463	For sedation
Pentobarbital Sodium	Raptor	30	IM-IV	PRN	E	1463	Not usually recommended
Pentobarbital Sodium	Skua, McCormick's	47	IM-IV	PRN	B	1075	
Pentobarbital Sodium	Swan, Mute	25 mg TD	IV	PRN	G	1074	Adult
Pentobarbital Sodium	Turkey	16.2	IM	PRN	B	1080	Add thiopental sodium
Pentoxifylline	Avian	15	PO	BID-TID	G	1489	Add aloe vera for frostbite, vasodilator
Permethrin	Pigeon	dusting	Topical	NL	E	1240	For fleas and lice
Permethrin	Psittacine	dusting	Topical	NL	E	1240	Ectoparasite control
Phenobarbital	Avian	1-5	PO	BID	E	111, 1473	
Phenobarbital	Avian	3.5-7	NL	BID	D	1475	Control of seizures
Phenobarbital	Avian	0.048-0.080 g/L	Drink	PRN	G	58	
Phenobarbital	Psittacine	3	PO	BID	E	1240	For feather picking, may cause deep sedation and inability to perch
Phenobarbital	Raptor	< 30	PO	PRN	E	1463	For sedation
Phenobarbital	Raptor	20	PO	NL	D	1612	For neurologic signs with organochlorine toxicity
Phenylbutazone	Ostrich	2.2	PO	QD	G	710	
Phenylbutazone	Psittacine	3.5-7	PO	BID-TID	E	1473	
Phenylbutazone	Raptor	3.5-7	PO	BID-TID	E	111	
Phytonadione	Avian	2.5-5	Parenteral	BID-QID	E	1492	For anticoagulant rodenticide
Phytonadione	Raptor	0.2-2.5	IM-PO	QD	E	1433	For anticoagulant poisoning
Piperacillin Sodium	Amazon Parrot	75-100	IM	q4-6h	A	1473	
Piperacillin Sodium	Avian	4.31 mg/kcal	IM-IV	TID-QID	E	1180	Synergistic with aminoglycosides
Piperacillin Sodium	Avian	2.55 mg/kcal	IM-IV	q4-6h	E	1180	Synergistic with aminoglycosides
Piperacillin Sodium	Avian	10 g/L saline	Flush	BID-QID	E	1183	Nasal or sinus
Piperacillin Sodium	Avian	10 g/L saline	Nebulize	BID-QID	E	1185	10 to 30 minutes
Piperacillin Sodium	Avian	100-200	IM	TID-QID	E	1650	Synergistic with aminoglycosides
Piperacillin Sodium	Avian	150	IM	BID	G	55	
Piperacillin Sodium	Bird, Aquatic	100-200	IM	BID	E	1478	Post-trauma infections
Piperacillin Sodium	Budgerigar	200	IM	TID	A	411	
Piperacillin Sodium	Cockatoo, Citron-crested	90	Parenteral	BID	G	740	Add amikacin
Piperacillin Sodium	Crane	100	IM-IV-SC	BID	E	629	
Piperacillin Sodium	Duck, Ruddy	100	IM	BID	G	1468	Begin pre-operatively for tendon surgery

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Piperacillin Sodium	Falcon	200	IM	TID-QID	E	1027	Broad spectrum
Piperacillin Sodium	Gull	100	IM	BID	E	1357	
Piperacillin Sodium	Lorikeet, Rainbow	100	IM	BID	G	1620	
Piperacillin Sodium	Macaw	4 mg TD per egg	Parenteral	q4d	G	820	For embryonic death, begin first dose at day 14, use 26 ga needle
Piperacillin Sodium	Pigeon	100	IM	BID-TID	G	260	
Piperacillin Sodium	Pigeon	100-200	IM	BID	G	590	
Piperacillin Sodium	Psittacine	100	IM	BID	A	411	
Piperacillin Sodium	Psittacine	50-100	SC	TID	E	632	Neonate dosage
Piperacillin Sodium	Psittacine	100-200	IM-IV	BID-TID	E	1240, 1756	
Piperacillin Sodium	Raptor	100	IM-IV	BID	E	1433	
Piperacillin Sodium + Tazobactam Sodium	Avian	150-200	IM-IV	QD-BID	G	1279	Substitute for piperacillin sodium, DO NOT use aminoglycosides concurrently
Piperacillin Sodium + Tazobactam Sodium	Falcon, Peregrine	200	IM	BID	G	1426	
Piperazine	Anseriformes	45-200	PO	NL	E	111	
Piperazine	Avian	100-500	PO	q2w	E	1650	For roundworms
Piperazine	Crane	4-5.26 g/L	Drink	QD	G	596	Repeat in 2 weeks
Piperazine	Finch	0.3 g/L	Drink	NL	E	1572	
Piperazine	Galliformes	100-500	PO	q2w	E	111	
Piperazine	Pigeon	1.9 g/L	Drink	QD	C	704	For 30 birds
Piperazine	Pigeon	500 mg	Drink	TD	C	706	For capillaria
Piperazine	Pigeon	250 mg	Drink	TD	C	706	For ascaridiasis
Piperazine	Pigeon	12.5	Drink	QD	E	1240	Repeat every 10 to 14 days
Piperazine	Pigeon	35	PO	QD	G	260	
Piperazine	Poultry	1 g/L	Drink	QD	G	57	
Piperazine	Poultry	100-500	PO	q10d	G	1309	For ascarids
Piperazine	Psittacine	250	PO	NL	G	57	
Piperazine	Quail	30-50 mg TD	PO	NL	G	678	Anthelmintic
Piperazine	Raptor	100	PO	NL	E	57, 1463	Anthelmintic
Piperonyl Butoxide	Raptor	6.25 g/L	Topical	NL	E	1068	Add permethrin + methoprene for ectoparasites
Piroxicam	Crane	0.5	PO	BID	G	1671	
Policosanol	Amazon Parrot	1 mg TD	PO	BID	G	1273	For hyperlipidemia or hypercholesterolemia

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Policosanol	Amazon, Yellow-naped	2	PO	QD	G	1466	Mix with lactulose for hyperlipidemia of triglyceride and cholesterol
Polymyxin B Sulfate	Avian	10-15	IM-PO	NL	E	924	
Polymyxin B Sulfate	Avian	1000 mg/kcal	PO	BID	E	1180	
Polymyxin B Sulfate	Avian	3000 KIU/L	Drink	NL	E	1180	
Polymyxin B Sulfate	Avian	50 KIU/kg	PO	BID	E	565	
Polymyxin B Sulfate	Canary	50 KIU/L	Drink	QD	E	695	
Polymyxin B Sulfate	Canary	50 KIU/kg food	Feed	QD	E	695	
Polymyxin B Sulfate	Canary	500 KIU/L	Drink	NL	E	1187	
Polymyxin B Sulfate	Canary	500 KIU/kg food	Feed	Once	E	1187	
Polymyxin B Sulfate	Passerine	100 KIU/kg food	Feed	QD	E	1437	Add drug to drink at the same time
Polymyxin B Sulfate	Passerine	100 KIU/L	Drink	QD	E	1437	Add drug to food at the same time
Polymyxin B Sulfate	Passerine, Small	500 KIU/L	Drink	NL	E	1187	
Polymyxin B Sulfate	Passerine, Small	500 KIU/kg food	Feed	Once	E	1187	
Polysulfated Glycosaminoglycan	Crane, Demoiselle	50 mg TD	IA	Once	G	579	Musculosketal joint therapy
Polysulfated Glycosaminoglycan	Pheasant, Peacock	12.5 mg TD	IM	Once	G	579	Musculosketal joint therapy
Polysulfated Glycosaminoglycan	Vulture, King	20 mg TD	IM	QW	G	579	Musculosketal joint therapy
Potassium Bromide	Cockatoo, Umbrella	80	PO	QD	G	1691	For control of seizures
Potassium Bromide	Parrot, Grey	80	PO	QD	G	1274	Seizure control
Potassium Bromide	Pigeon	80	PO	QD	G	1274	Seizure control
Potassium Chloride	Avian	0.3	IV	NL	E	1473	
Potassium Chloride	Avian	0.1-0.3	IV	NL	E	111	
Potassium Permanganate	Falcon	14 g/m³	NL	Once	G	1029	Add formaldehyde for fumigation TOXIC
Pralidoxime Chloride	Avian	10-30	IM	QD	E	111	
Pralidoxime Chloride	Raptor	100	NL	BID-QID	E	1400	For organophosphate poisoning
Pralidoxime Chloride	Raptor	20	IM	PRN	G	50	Continue only if first dose produces response
Pralidoxime Iodide	Avian	10-100	Parenteral	NL	E	1492	Reduce dosage if used with atropine for organophosphates

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Pralidoxime Iodide	Avian	10-100	Parenteral	BID-TID	E	1650	Use lower dosage if used with atropine for organophosphate toxicity
Pralidoxime Iodide	Raptors	100	IM	Once	B	1093	For monocrotophos toxicity
Pralidoxime Mesylate	Anseriformes	100	IM	QID	D	1150	For organophosphate toxicity
Pralidoxime Mesylate	Psittacine	100	IM	QID	E	1240	
Praziquantel	Anseriformes	10-20	PO-SC	q10d	D	1150	Control cestodes
Praziquantel	Avian	10-20	PO	QD	D	1470	For liver parasitic disease
Praziquantel	Avian	9	IM	QD	D	1470	Continue PO for 11 days for liver parasitic disease
Praziquantel	Avian	10	PO	NL	E	1648	For tapeworms
Praziquantel	Bird, Aquatic	10	PO	NL	E	1503	For tapeworms
Praziquantel	Bustard	10	PO-SC	Once	E	1240	For cestodes and trematodes
Praziquantel	Finch	10-20	PO	q10-14d	E	1572	
Praziquantel	Finch	12 mg/kg	Feed	Once	E	1617	Crush tablet and mix with enough food for 1 day, scatter throughout aviary
Praziquantel	Ostrich	7.5	PO	NL	C	283	
Praziquantel	Ostrich	5	PO	NL	G	481	
Praziquantel	Passerine, Small	10	NL	NL	E	1187	For trematodes
Praziquantel	Penguin	10-20	PO	NL	E	1478	For cestodes
Praziquantel	Pigeon	11.4	SC	QM	E	704	
Praziquantel	Pigeon	10-20	PO	q2w	E	704	Tablet crushed in water suspension
Praziquantel	Pigeon	10	PO	NL	E	1192	For cestodes
Praziquantel	Psittacine	9	IM	NL	E	763	For tapeworms
Praziquantel	Psittacine	10-20	PO	Once	E	1240	For tapeworms repeat after 10 days
Praziquantel	Raptor	50	PO-SC	NL	E	1068	For trematodes and cestodes
Praziquantel	Raptor	30	NL	q10d	E	1132	For tapeworms and flukes
Praziquantel	Raptor	5-10	PO-SC	QD	E	1240	
Praziquantel	Raptor	10-50	PO	q2w	E	1359	For tapeworms
Prednisolone	Avian	2	PO	BID	E	1431	May predispose to mycotic infection
Prednisolone	Avian	6.7	PO	NL	E	1526	Use decreasing dosage for long-term therapy
Prednisolone	Psittacine	2	PO	BID	E	1240	Also analgesic
Prednisolone	Raptor	0.5-1	IM	Once	E	1400	
Prednisolone	Raptor	2-4	IM-IV	Once	E	1400	For shock

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Prednisolone Sodium Phosphate	Avian	0.5-1	IM-IV	NL	E	111	
Prednisolone Sodium Phosphate	Avian	2-4	IM-IV	NL	E	111	Immunosuppressive
Prednisolone Sodium Phosphate	Raptor	10-30	IM-IV	PRN	G	234	
Prednisolone Sodium Succinate	Avian	0.5-1	IM-IV	Once	E	1151	For neurologic emergencies
Prednisolone Sodium Succinate	Avian	10-20	IM-IV	NL	D	1554	For shock
Primaquine Phosphate	Avian	0.03	PO	QD	D	1470	For liver parasitic disease
Primaquine Phosphate	Avian	1	PO	QW	E	1479	
Primaquine Phosphate	Avian	1	PO	QW	E	1492	Add chloroquine for *Plasmodium*
Primaquine Phosphate	Avian	0.3-1	PO	TID	G	57	Usually in combination with chloroquine
Primaquine Phosphate	Falcon	0.75	NL	Once	E	1177	Add chloroquine
Primaquine Phosphate	Penguin	0.03	PO	QW	D	1470	For liver parasitic disease prophylaxis
Primaquine Phosphate	Penguin	0.03	PO	QD	E	111, 1473	
Primaquine Phosphate	Penguin	1	PO	QD	B	262	Add chloroquine phosphate
Primaquine Phosphate	Psittacine	1	PO	QD	E	1365	For sarcocystosis
Primaquine Phosphate	Raptor	1	PO	QD	G	61	Add chloroquine
Primaquine Phosphate	Raptor	1	PO	QW	G	94	Add chloroquine phosphate, juvenile dosage
Primidone	Amazon Parrot	125 mg TD	Drink	QD	G	1753	Long-term seizure control
Primidone	Raptor	125	PO	PRN	E	1463	For sedation
Probenecid	Avian	200	NL	q2d	E	704	For body weight > 200g, to decrease excretion rate of penicillin/cephalosporin during therapy
Probenecid	Macaw	125	PO	QID	G	45	Uricosiuric
Probiotic	Anseriformes	21 ml/kg food	Feed	Once	E	1240	For hand-rearing (6 hours old) and convalescing birds, Avipro Pediatric®
Probiotic	Anseriformes	5 scoops/L	Drink	NL	E	1240	Double dosage for stressed bird, Avipro®
Probiotic	Avian	5 cc/L	Feed	Once	E	111	Add to hand feeding formula

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Probiotic	Avian	1 pinch TD	Feed	QD	E	111	
Probiotic	Psittacine	21 ml/kg food	Feed	Once	E	1240	For hand-rearing (6 hours old) and convalescing birds, Avipro Pediatric®
Probiotic	Psittacine	5 scoops/L	Drink	NL	E	1240	Double dosage for stressed bird, Avipro®
Probiotic	Psittacine	5 ml/L food	Feed	Once	E	1473	Hand feeding formula
Probiotic	Psittacine	pinch TD	PO	QD	E	1473	
Probiotic	Ratite		PO	NL	G	418	
Proflavine	Avian	30 mg TD	Topical	QD-BID	E	1240	For wounds, stimulate granulation
Propentofylline	Avian	5	PO	QD	G	1240	Vasodilator for wing tip edema or dry gangrene
Propentofylline	Raptor	5	PO	BID	E	1400	Improve circulation (wing tip edema)
Propofol	Amazon, Hispaniolan	5	IV	PRN	B	1436	Maintain anesthesia by continuous infusion at 1 mg/kg per minute
Propofol	Anseriformes	8	IV	PRN	E	1190	Induction agent
Propofol	Avian	1.33-14	IV	PRN	E	1181	Short duration, smooth induction, high safety margin
Propofol	Duck, Mallard	10	IV	PRN	B	1358	Administer over 1 minute, follow with 4 mg/kg, respiratory depression
Propofol	Ostrich	3	IV	PRN	B	1628	Maintain with 0.2 mg/kg/min constant rate infusion
Propofol	Pigeon	4.1-8.6	IV	PRN	B	779	Follows ketamine
Propofol	Pigeon	14	IV	PRN	B	779	Preliminary to inhalation anesthesia
Propofol	Psittacine	1.33	IV	PRN	E	1240	For anesthesia induction
Propofol	Swan	6	IV	Once	G	1695	Induction for isoflurane
Propofol	Swan (Black, Mute)	10	IV	PRN	B	1291	Gas anesthesia premed
Propolis	Chicken	30 mg TD		Once	B	1632	Immune and growth stimulant
Propranolol HCl	Avian	0.2	IM	NL	E	111	
Propranolol HCl	Avian	0.04	IV	NL	E	111	
Prostaglandin E (See Dinoprostone)	Raptor	N/A	Topical	PRN	E	1400	For egg binding, use gel formulation
Psyllium Hydrophilic Mucilloid	Avian	2.5 ml/kg	PO	NL	G	87	Mix with syringeable food, bulking agent to remove GI materials

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Psyllium Hydrophilic Mucilloid	Bird, Aquatic	40 ml/L soft food	Feed	Once	E	1478	Add sodium + magnesium sulfate + mineral oil for lead poisoning, add bran
Pyrantel Pamoate	Anseriformes	7	PO	q2w	E	1358	For nematodes
Pyrantel Pamoate	Avian	4.5-25	PO	q2w	D	111,1221, 1473	For nematodiasis
Pyrantel Pamoate	Crane	4.5	PO	q10d	E	1361	For intestinal nematodes
Pyrantel Pamoate	Finch	4.5	PO	q10-14d	E	1572	
Pyrantel Pamoate	Passerine	100	PO	Once	G	1335	For intestinal nematodes
Pyrantel Pamoate	Pigeon	20-25	PO	QW-q2w	E	1364	
Pyrantel Pamoate	Psittacine	4.5	PO	q10d	E	1240	For nematodes
Pyrantel Pamoate	Psittacine	100	PO	NL	G	57, 1335	
Pyrantel Pamoate	Raptor	20	PO	Once	E	1240, 1400	For nematodes
Pyrantel Pamoate	Raptor	4.5	PO	q2w	G	94	Juvenile dosage
Pyrantel Pamoate	Ratite	5-7	PO	NL	G	418	
Pyrethrin	Crane	0.1% powder	Topical	QW-q2w	E	1361	For ectoparasites, apply lightly
Pyrethrin	Finch		Topical	QD	E	1572	Dust feathers
Pyrethrin	Raptor	0.5-2% mixture	Topical	q10d	G	1411	For chewing lice
Pyrimethamine	Anseriformes	0.25-0.5	PO	BID	D	1150	For sarcocystosis and toxoplasmosis
Pyrimethamine	Avian	0.5	PO	BID	D	1470	
Pyrimethamine	Avian	0.3	PO	NL	E	1650	For *Leucocytozoon, Plasmodium*
Pyrimethamine	Raptor	0.25-0.5	PO	BID	E	1400	For sarcocystosis, toxoplasmosis and other protozoal infections
Pyrimethamine	Raptor	0.5	PO	BID	E	1433	For leucocytozoonosis
Pyrimethamine + Sulfaquinoxaline	Anseriformes	0.06 g/L	Drink	QD	D	1150	2 days off, then repeat for coccidiosis
Pyrimethamine + Sulfaquinoxaline	Psittacine	0.06 g/L	Drink	QD	E	1240	Wait 2 days then repeat for coccidiosis
Quinacrine HCl	Avian	5-10	PO	QD	E	111	
Quinacrine HCl	Avian	240	NL	QD	E	1479	For *Plasmodium*
Quinacrine HCl	Avian	7.5	PO	QD	E	1650	For *Plasmodium*
Quinacrine HCl	Canary	240	PO	BID	G	57	
Quinacrine HCl	Pigeon	10 mg TD	PO	QD	G	47	
Quinacrine HCl	Psittacine	5-10	PO	QD	E	1473	
Red Clover	Avian	10 drops/kg	PO	QD	E	1435	Use low alcohol formulation for soft shelled egg

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Red Palm Oil	Avian	10-60 drops/kg	PO	QD	G	1727	May give over food
Rescue Remedy	Finch	2 drops TD	PO	Once	G	1723	For stress of handling or minor procedures
Reserpine	Raptor	2-4	PO	PRN	E	1463	For prolonged sedation
Resorantel	Ostrich	130	PO	NL	B	283,401, 524	Add fenbendazole or levamisole
Rifabutin	Avian	15	PO	QD	D	1470	Add ethambutol + enrofloxacin for mycobacteriosis
Rifabutin	Avian	1.15 mg/kcal	PO	QD	E	1180	Add ethambutol + azithromycin or clarithromycin for mycobacteriosis
Rifampin	Amazon, Yellow-cheeked	45	NL	QD	G	713	Add ethambutol + isoniazid
Rifampin	Avian	10-20	PO	BID	E	111, 1473	
Rifampin	Avian	15	PO	QD	G	1154	Add ciprofloxacin + ethambutol for mycobacteriosis
Rifampin	Bustard, Houbara	20	PO	BID	G	932	For bacterial diarrhea
Rifampin	Crane, Whooping	45	PO	QD	G	207	Add ethambutol
Rifampin	Psittacine	15	PO	BID	E	1240	For avian tuberculosis
Rifampin	Raptor	30	PO	TID	B	1178	Add IT and IV amphotericin B + flucytosine for aspergillosis
Rimantadine HCl	Poultry	0.1 g/L	Drink	NL	G	435	
Robenidine HCl	Chicken	33 mg/kg food	Feed	QD	E	564	7-day slaughter withdrawal, prophylactic
Robenidine HCl	Pheasant, Ring-necked	0.033 g/kg	Feed	QD	B	895	For coccidiosis
Ronidazole	Avian	0.323 mg/kcal	PO	QD	E	1180	For flagellates
Ronidazole	Avian	0.65 g/L	Drink	NL	E	1180	
Ronidazole	Avian	1 g/L	Drink	QD	E	1479	Treat 4 times yearly for trichomoniasis
Ronidazole	Avian	6-10	PO	QD	G	57	
Ronidazole	Budgerigar	1 g/L	Drink	QD	E	1479	Treat 4 times yearly for trichomoniasis
Ronidazole	Canary	0.4 g/L	Drink	QD	E	695	
Ronidazole	Canary	0.4 g/kg food	Feed	Once	E	1187	
Ronidazole	Finch	0.1 g/L	Drink	QD	E	1572	
Ronidazole	Finch	6-10	PO	QD	E	1572	
Ronidazole	Finch	0.05 g/kg food	Feed	QD	G	1334	For cochlosomiasis

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Ronidazole	Finch	0.05 g/L	Drink	QD	G	1334	For cochlosomiasis
Ronidazole	Passerine	0.04 g/L	Drink	QD	D	1438	For trichomoniasis, giardiasis and cochlosomiasis
Ronidazole	Pigeon	6-10	NL	QD	D	1221	For trichomoniasis
Ronidazole	Pigeon	0.1 g/L	Drink	QD	E	1192	For hexamitiasis, add trimethoprim or enrofloxacin in severe infections
Ronidazole	Pigeon	0.1-0.2 g/L	Drink	NL	E	1192	For treatment of trichomoniasis, give periodically for 2 to 3 days for prophylaxis
Ronidazole	Pigeon	12.5	Drink	QD	E	1240	For trichomoniasis treatment
Ronidazole	Pigeon	10	PO	NL	E	1432	"
Ronidazole	Pigeon	0.1 g/L	Drink	NL	E	1432	"
Roxarsone	Poultry	25-50 mg/kg food	Feed	QD	E	564	Nutritional use, no slaughter withdrawal
Salicylic Acid	Psittacine		Topical	q2w	D	1446	Add 3 g tannic acid + 3 g salicylic acid + ethyl alcohol for 100 ml of solution for dermatomycosis
Salinomycin	Chicken	0.06 g/kg	Feed	QD	B	869	Preventive for coccidiosis
Salinomycin	Chicken	0.075 g/kg	Feed	QD	B	869	Preventive for coccidiosis
Salinomycin	Chicken	60	Feed	QD	B	958	For coccidial prophylaxis
Salinomycin	Chicken	44-66 mg/kg food	Feed	QD	E	564	No slaughter withdrawal, prophylactic
Salinomycin	Pheasant, Ring-necked	60 ppm	Feed	Once	B	783	For coccidiosis
Salinomycin	Poultry	60 g/ton	Feed	QD	C	705	For coccidiosis
Sarafloxacin HCl	Chicken	10	PO	BID-TID	A	787	
Sarafloxacin HCl	Chicken	8	Drink	QD	B	855	Continuous or 4-hour pulse dose
Sarafloxacin HCl	Chicken	0.02 g/L	Drink	QD	B	864	For *Escherichia coli* infections
Sarafloxacin HCl	Turkey	0.05 g/L	Drink	NL	A	805	
Secobarbital Sodium	Blackbird, Red-winged	0.025-0.03	PO	PRN	G	1769	Add chloralose
Secobarbital Sodium	Dove	5 g/L bait	Feed	Once	G	1763	Bait is usually corn
Secobarbital Sodium	Duck, Mallard	25	PO	PRN	B	1095	
Selenium	Psittacine	0.06	NL	q3d-q2w	G	1613	
Semduramycin	Chicken		PO	QD	G	564	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Seromycin	Avian	10	PO	BID	G	1154	Add lamprene + ethambutol HCl, add ciprofloxacin or enrofloxacin or streptomycin for advanced cases
Silver Nitrate	Avian		Topical	q10d	E	1650	For cautery of papillomas
Silver, Colloidal	Avian	2 drops/kg	PO	QD	E	1435	Broad spectrum
Silver, Colloidal	Avian	40 drops/L	Drink	QD	E	1435	Broad spectrum
Sodium Bicarbonate	Avian	1 mEq/kg	IV-SC	PRN	G	1309	First dose IV, remainder SC, maximum total dose 4 mEq/kg for metabolic acidosis
Sodium Bicarbonate	Avian	1-4 mEq/kg	IV	NL	E	1473	Give slowly over 15 to 30 minutes, do not exceed 4 mEq/kg
Sodium Bicarbonate	Avian	5 mEq/kg	IO-IV	Once	G	1311	For cardiopulmonary resuscitation
Sodium Chloride	Avian	30 ml/kg	PO	TID-QID	E	1151	Use 0.9% solution
Sodium Chloride	Bird, Aquatic	10 g/L	Gavage	QD	D	1559	To maintain activity of salt gland of marine birds kept on freshwater
Sodium Chloride	Bird, Aquatic	100	Feed	QD	E	1501	For pelagic sea birds kept in fresh water
Sodium Chloride	Bird, Aquatic	100	PO	NL	E	1503	For procelliform birds kept in fresh water
Sodium Chloride	Penguin	150-1667	PO	QD	G	1353	Supplement fish diet
Sodium Chloride	Psittacine	50 ml/kg	IM-IV-SC	QD	E	1240	Use 0.9% solution
Sodium Chloride	Raptor	1-2% solution	Topical	NL	E	1400	1-2% solution for wounds, wash off within 5 minutes
Sodium Hypochlorite	Finch	30 ml/L	Topical	NL	E	1572	For salmonellosis environmental cleanup, use household bleach
Sodium Iodide	Avian	60	IM	PRN	G	54	
Sodium Iodide	Avian	67-200	IM	NL	F	1209	
Sodium Iodide	Budgerigar	2 mg TD	IM	PRN	G	85	
Sodium Iodide	Budgerigar	134-200	IM	QD	G	1212	For acute thyroid hyperplasia
Sodium Iodide	Budgerigar	67	IM	QD	G	1212	For chronic thyroid hyperplasia
Sodium Lactate Solution	Avian	50 ml/kg	IV	NL	E	111	Maintenance fluids
Sodium Lactate Solution	Avian	10-30 ml/kg	IV	NL	E	1650	Emergency care, give slowly, warm fluid

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Sodium Lactate Solution	Avian	25 ml/kg	IO-IV-SC	NL	G	1311	
Sodium Lactate Solution	Avian	50-100 ml/kg	IO-IV-SC	BID	G	1756	For hyperuricemia, give until level drops to normal
Sodium Lactate Solution	Hawk, Red-tailed	20 ml/kg	IV	NL	G	1626	Supportive therapy post-trauma
Sodium Lactate Solution	Lory, Red	50 ml/kg	SC	NL	G	1621	Intra-operative fluids
Sodium Lactate Solution	Psittacine	50-100 ml/kg	IO-IP-IV-SC	NL	E	1688	
Sodium Sulfate	Avian	2000	PO	QD	E	111	Large birds, give slurry
Sodium Sulfate	Avian	500-1000	PO	NL	E	1554	For botulism to flush gastrointestinal tract
Sodium Sulfate	Avian	125-250	PO	NL	G	1746	For removal of metals from GI tract
Somatostatin	Toucan, Sulfur-breasted	0.003	SC	BID	G	589	
Spectinomycin HCl	Avian	30	PO	QD	E	565	
Spectinomycin HCl	Avian	1 g/L	Drink	QD	E	704	
Spectinomycin HCl	Avian	10-45	IM	NL	E	704	
Spectinomycin HCl	Avian	0.58 mg/kcal	IM-SC	TID	E	1180	
Spectinomycin HCl	Avian	1 g/L	Drink	NL	E	1180	
Spectinomycin HCl	Canary	0.2-0.4 g/L	Drink	NL	E	1187	
Spectinomycin HCl	Chicken	5 mg TD	SC	Once	B	848	Day-old neonates
Spectinomycin HCl	Chicken	2 g/L	Drink	NL	C	1307	For *Mycoplasma gallisepticum* in broilers
Spectinomycin HCl	Chicken	0.5 g/L	Drink	NL	C	1307	For improved weight gain in broilers
Spectinomycin HCl	Duck	5	SC	Once	B	841	For *E. coli* and *Salmonella*
Spectinomycin HCl	Finch	0.2-0.4 g/L	Drink	QD	E	1572	
Spectinomycin HCl	Finch	0.4 g/kg food	Feed	QD	E	1572	
Spectinomycin HCl	Passerine	0.2-0.4 g/L	Drink	QD	E	1437	Add drug to food at the same time
Spectinomycin HCl	Passerine	0.4 g/kg soft food	Feed	QD	E	1437	Add drug to drink at the same time
Spectinomycin HCl	Passerine, Small	0.4 g/kg food	Feed	Once	E	1187	
Spectinomycin HCl	Passerine, Small	0.2-0.4 g/L	Drink	NL	E	1187	
Spectinomycin HCl	Pigeon	25	IM-SC	TID	E	565, 1061	
Spectinomycin HCl	Pigeon	30	PO	QD	F	1061	
Spectinomycin HCl	Pigeon	0.15-0.25 g/L	Drink	NL	G	260	
Spectinomycin HCl	Poultry	0.264-0.53 g/L	Drink	QD	C	564	
Spectinomycin HCl	Poultry	0.132 g/L	Drink	QD	C	564	Prophylactic
Spectinomycin HCl	Poultry	2.5-20 mg TD	SC	QD	C	564	Chick dosage
Spectinomycin HCl	Psittacine	10-30	IM	BID-TID	E	1240	Gram negative enteritis
Spectinomycin HCl	Psittacine	35	IN	NL	E	1240	Give 1/3 each nare, remainder IM

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Spectinomycin HCl	Raptor	50	IM	BID	G	234	
Spectinomycin HCl	Turkey	12.5-25 mg TD	SC	NL	C	1307	Dilute 1:4 normal saline for newly hatched chicks
Spectinomycin HCl	Turkey	10 mg TD	SC	NL	C	1307	Inject in base of the neck of 1 to 3-day-old poults for airsacculitis
Spiramycin	Avian	250	PO	QD	E	565	
Spiramycin	Avian	100	IM-PO	NL	E	924	
Spiramycin	Avian	2 g/L	Drink	NL	E	1180	
Spiramycin	Avian	0.58 mg/kcal	IM	QD	E	1180	
Spiramycin	Avian	1.08 mg/kcal	PO	QD	E	1180	
Spiramycin	Canary	0.2-0.4 g/L	Drink	QD	E	695	
Spiramycin	Canary	400 mg/kg food	Feed	QD	E	695	
Spiramycin	Passerine, Small	0.2-0.4 g/L	Drink	NL	E	1187	
Spiramycin	Passerine, Small	0.4 g/kg food	Feed	Once	E	1187	
Spiramycin	Pigeon	25	IM	QD	E	565, 1061	
Spiramycin	Pigeon	50	PO	QD	F	1061	
Spiramycin	Poultry	0.4 g/L	Drink	QD	C	564	
Spiramycin	Raptor	20	IM	NL	E	1463	For bacterial infections
Stanozolol	Avian	25-50	IM	q3d-QW	E	111, 1209, 1240	
Stanozolol	Avian	25-50	IM	q3-4d	E	1473	
Stanozolol	Avian	25-50	IM	q3d-QW	G	1309	Anabolic agent
Stanozolol	Psittacine	0.016 g/L	Drink	NL	E	763	For debilitation, anorexia and anemia
Stanozolol	Psittacine	0.5-1	Parenteral	NL	E	763	For debilitation, anorexia and anemia
Streptomycin Sulfate	Avian	10-30	IM	BID-TID	E	111	
Streptomycin Sulfate	Avian	33	IM	BID-TID	E	741	Used in large birds and poultry
Streptomycin Sulfate	Avian	15	IM-PO	NL	E	924	Poor spectrum
Streptomycin Sulfate	Avian	0.65 mg/kcal	IM	BID-TID	E	1180	
Streptomycin Sulfate	Avian	1.28-4.67 mg/kcal	PO	QD	E	1180	
Streptomycin Sulfate	Avian	10-30	IM	BID-TID	E	1470	
Streptomycin Sulfate	Avian	30	IM	BID	G	55, 1154	Add ethambutol + rifampin for mycobacteriosis
Streptomycin Sulfate	Avian	10	IM	BID	G	819	Add rifampin + ethambutol for avian tuberculosis
Streptomycin Sulfate	Chicken	25-50	IM	QD	A	320	
Streptomycin Sulfate	Chicken	50-100	PO	QD	E	565	
Streptomycin Sulfate	Pheasant	220 mg/kg food	Feed	QD	G	678	
Streptomycin Sulfate	Pheasant	55-110 mg/kg food	Feed	QD	G	678	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Streptomycin Sulfate	Pigeon	100-200	PO	QD	E	565, 1061	
Streptomycin Sulfate	Pigeon	25-50	IM	QD	F	1061	
Streptomycin Sulfate	Poultry	55	PO	QD	C	564	
Streptomycin Sulfate	Poultry	0.068-0.1 g/L	Drink	QD	C	564	
Streptomycin Sulfate	Poultry	13.2 mg/kg food	Feed	QD	E	564	Add penicillin, nutritional use, no slaughter withdrawal
Streptomycin Sulfate	Quail	55-110 mg/kg food	Feed	QD	G	678	
Streptomycin Sulfate	Raptor	15	PO	NL	E	1463	May give with kaolin or sulfonamide for bacterial infections
Succimer	Avian	25-35	PO	BID	D	1221, 1470	Preferred oral drug for lead toxicosis
Succimer	Avian	30	PO	QD	E	1120	For zinc toxicoses
Succimer	Avian	30	PO	BID	E	1151	Metal chelating agent
Succimer	Avian	25-35	PO	BID	E	1236	Give 5 days per week for lead toxicosis
Succimer	Avian	15-35	PO	BID	G	1337	For lead poisoning
Succimer	Bird, Aquatic	25-35	PO	BID	D	1478	
Succimer	Psittacine	30	NL	NL	G	1710	For zinc toxicity
Succimer	Raptor	25	PO	BID	E	1359	After 2 days off, repeat over 3-5 weeks for lead toxicity
Sucralfate	Avian	25	PO	TID	E	1492	Mix with water for GI bleeding
Sucralfate	Lorikeet, Rainbow	24	PO	BID	G	1620	For intestinal ulcers
Sucralfate	Psittacine	25	PO	TID	E	111	
Sucrose	Raptor	< 5 ml/kg 5% soln	PO	PRN	E	1400	Mild purgative, may stimulate appetite
Sulfachlorpyridazine	Avian	0.175 g/L	Drink	QD	E	1240	Antibacterial,* use 5-10 days
Sulfachlorpyridazine	Avian	0.125 ml powder	Drink	QD	G	1309	Antibacterial*
							*Vetisulid Oral Powder® is 560 mg/ml volume for flock treatment for E. coli
Sulfachlorpyridazine	Avian	59 g/L	Drink	QD	E	1479	Use 5 days per week, reduces shedding of *Atoxoplasma*
Sulfachlorpyridazine	Avian	0.4 g/L	Drink	QD	G	57	Repeat after 2 days, antiparasitic
Sulfachlorpyridazine	Canary	0.15 g/L	Drink	QD-q2d	E	1187	Give 5 days per week until molting complete in juveniles with atoxopasmosis
Sulfachlorpyridazine	Canary	0.3 g/L	Drink	QD-q2d	E	1187	Give 5 days per week for coccidiosis
Sulfachlorpyridazine	Canary	0.15-.0.3 g/L	Drink	NL	E	1187	
Sulfachlorpyridazine	Passerine	0.15 g/L	Drink	QD	G	1334	Give 5 days of each week, continue until after molting

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Sulfachlorpyridazine	Passerine, Small	0.15-.0.3 g/L	Drink	NL	E	1187	
Sulfachlorpyridazine	Pigeon	0.3 g/L	Drink	QD	G	260	
Sulfachlorpyridazine	Pigeon	0.3 g/L	Drink	QD	G	260	
Sulfadiazine, Silver	Psittacine	1% cream formula	Topical	NL	D	1448	Post-surgical wound care
Sulfadiazine, Sulfamerazine, Sulfamethazine	Penguin, African	300-400	Feed	BID	D	1614	Precede with sulfamethazine injectable, give dose in fish for malaria
Sulfadimethoxine	Avian	24	IM	NL	F	1209	
Sulfadimethoxine	Avian	25	IM-PO	NL	E	924	
Sulfadimethoxine	Chicken	0.5 g/L	Drink	NL	B	826	For *Pasteurella multocida* and *Hemophilus gallinarum*
Sulfadimethoxine	Crane	50	PO	QD	E	1361	For coccidiosis
Sulfadimethoxine	Falcon	25 (50 loading dose)	PO	QD	E	1027	For coccidiosis, do not deprive of water
Sulfadimethoxine	Gull	25	PO	BID	E	1357	coccidiosis
Sulfadimethoxine	Pigeon	25	PO	BID	G	590	
Sulfadimethoxine	Pigeon	250	IM	BID	A	992	For coccidiosis
Sulfadimethoxine	Pigeon	0.5 g/L	Drink	QD	E	1432	For coccidiosis and atoxoplasmosis
Sulfadimethoxine	Pigeon	0.19-0.25 g/L	Drink	QD	G	260	Loading dose 375 mg/L
Sulfadimethoxine	Poultry	0.25-0.5 g/L	Drink	QD	C	564	
Sulfadimethoxine	Raptor	25	NL	QD	E	1132	Double dose for first day for coccidiosis
Sulfadimethoxine	Raptor	25-50	PO	QD	G	94	Juvenile dosage, repeat after 2 days
Sulfadoxine + Pyrimethamine	Avian	1	PO	QW	E	1479	
Sulfadoxine + Pyrimethamine	Avian	0.3	PO	NL	E	1650	Dosage based on pyrimethamine for *Leucocytozoon* and *Plasmodium*
Sulfadoxine + Pyrimethamine	Psittacine	0.5	PO	BID	E	1365	For sarcocystosis
Sulfamerazine	Avian	2 g/L	Drink	QD	E	924	Repeat after 2 days for coccidiosis
Sulfamethazine	Avian	30	IM-SC	NL	E	704	
Sulfamethazine	Avian	0.2 g/L	Drink	QD	E	704	Repeat after 2 days
Sulfamethazine	Avian	50	PO	NL	E	704	
Sulfamethazine	Avian	1 g/L	Drink	NL	E	741	For coccidiosis plus mild respiratory and enteric diseases
Sulfamethazine	Avian	1 g/L	Drink	NL	E	741	For coccidiosis plus mild respiratory and enteric diseases

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Sulfamethazine	Avian	0.15 g/L	Drink	NL	E	1180	
Sulfamethazine	Avian	0.125 g/L	Drink	QD	G	57	Use liquid formulation, wait 2 days then repeat
Sulfamethazine	Budgerigar	7	PO	QD	G	57	Wait 2 days then repeat
Sulfamethazine	Canary	0.15 g/L	Drink	NL	E	1187	
Sulfamethazine	Canary	0.15 g/L	Drink	QD	E	695	
Sulfamethazine	Chicken	1 g/L	Drink	QD	E	564	12-day slaughter withdrawal
Sulfamethazine	Passerine	0.15 g/L	Drink	QD	E	1437	
Sulfamethazine	Passerine, Small	0.15 g/L	Drink	NL	E	1187	
Sulfamethazine	Penguin, African	83-125	IM	BID	D	1614	Switch to sulfadiazine + sulfamerazine + sulfamethazine when able to give orally for malaria
Sulfamethazine	Pigeon	2 g/L	Drink	QD	E	704	Repeat 1 to 2 times at 2-day intervals
Sulfamethazine	Pigeon	2 g/L	Drink	QD	E	704	Repeat after 5 days
Sulfamethazine	Pigeon	2 g/L	Drink	QD	E	1240	Wait 2 days then repeat for 3 treatment cycles for coccidiosis, perhaps toxoplasmosis
Sulfamethazine	Pigeon	3.33-6.66 g/L	Drink	QD	E	1240	For coccidiosis, perhaps Toxoplasmosis
Sulfamethazine	Pigeon	3.33-6.66 g/L	Drink	QD	E	1432	
Sulfamethazine	Pigeon	1.1 g/L	Drink	QD	G	232	
Sulfamethazine	Pigeon	1.1 g/L	Drink	QD	G	260	Loading dose 375 mg/L, treat with B vitamins for 5 days then repeat
Sulfamethazine	Poultry	0.19-0.25 g/L	PO	QD	C	564	
Sulfamethazine	Poultry	1 g/L	Drink	QD	C	564	
Sulfamethazine	Raptor	1 g/L	Drink	QD	E	1612	Repeat after 2 days
Sulfanitran	Chicken	300 mg/kg food	Feed	QD	E	564	Add nitromide + roxarsone, 5-day slaughter withdrawal, prophylactic
Sulfanitran	Poultry	300 mg/kg food	Feed	QD	E	564	Add nitromide + roxarsone, nutritional use, no slaughter withdrawal
Sulfaquinoxaline	Anseriformes	500 mg/kg food	Feed	Once	G	597	
Sulfaquinoxaline	Chicken	0.17 g/L	Drink	QD	E	564	10-day slaughter withdrawal
Sulfaquinoxaline	Duck	0.25 g/kg	Feed	Once	B	825	For *Riemerella anatipestifer* in ducklings
Sulfaquinoxaline	Lory	100	PO	QD	G	57	
Sulfaquinoxaline	Pigeon	100	PO	QD	G	57	
Sulfaquinoxaline	Poultry	0.397 g/L	Drink	QD	C	564	
Sulfaquinoxaline	Raptor	0.034 g/L	Drink	QD	E	1612	Repeat after 2 days
Sulfaquinoxaline	Turkey	0.17 g/L	Drink	QD	E	564	10-day slaughter withdrawal

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Sulfaquinoxaline + Diaveridine	Avian	0.1 g/kg feed	Feed	QD	E	1492	For coccidiosis
Sulfaquinoxaline + Diaveridine	Avian	0.1 g/L	Drink	QD	E	1492	For coccidiosis
Sulfaquinoxaline + Diaveridine	Avian	0.1 g/kg food	Feed	QD	E	1650	For coccidiosis
Sulfaquinoxaline + Diaveridine	Avian	0.1 g/L	Drink	QD	E	1650	For coccidiosis
Sulfaquinoxaline + Diaveridine	Chicken	0.5 g/kg feed	Feed	q4d	E	1479	Coccidial preventive for market broilers
Sulfaquinoxaline + Diaveridine	Chicken	0.55 g/kg feed	Feed	QD	E	1479	Coccidial preventive for replacement/breeder 0 to 8-week old broilers
Sulfaquinoxaline + Diaveridine	Chicken	0.33 g/kg feed	Feed	QD	E	1479	Coccidial preventive for replacement/breeder 8 to 12-week old broilers
Sulfaquinoxaline + Diaveridine	Poultry	0.56 g/L	Drink	QD	E	1479	Repeat after 3 days, coccidiosis therapy
Sulfaquinoxaline + Diaveridine	Poultry	0.56 g/L	Drink	q4d	E	1479	Coccidial preventive
Sulfathiazole Sodium	Poultry	1 g/L	Drink	QD	C	564	
Sulfatroxazole	Pigeon	50	PO	BID	A	565	Add trimethoprim
Sulfonamide	Avian	0.045 g/L	Drink	QD	E	924	Repeat after 2 days for coccidiosis
Tamoxifen Citrate	Galliformes	40	NL	NL	B	677	Induce molting
Tannic Acid	Avian	3 g	Topical	PRN	E	1240	Add aspirin + ethyl alcohol to prepare 100 ml for fungal dermatitis
Tannic Acid	Psittacine		Topical	q2w	D	1446	Add 3 g tannic acid + 3 g salicylic acid + ethyl alcohol for 100 ml of solution for dermatomycosis
Tea	Ramphastid	Dilute solution	Drink	QD	G	1470	Prevent hemochromatosis by limiting iron uptake, no citrus in diet
Terbinafine	Avian	10-15	PO	QD-BID	E	1668	May combine with itraconazole for systemic mycosis

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Terbinafine	Avian	10-15	PO	QD-BID	G	1338	For mycosis
Terbinafine	Parrot, Grey Congo	1g/L aqueous	Nebulize	TID	G	1743	For respiratory aspergillosis
Terbinafine	Raptor	30	PO	QD	E	1359	Prophylactic
Testosterone	Avian	10	IM	QW	E	924	For feather disorders
Testosterone	Avian	8	IM	QW	E	1473	
Testosterone	Canary	2.5	IM	QW	D	1441	For baldness
Testosterone Cypionate	Psittacine	8	IM	QW	E	1240	Use with great care
Tetanus Antitoxin	Falcon, Saker	250 IU	IM	QD	G	822	Loading dose of 125 IU each IV and IM in lesion for localized tetanus
Tetracycline	Avian	0.031-0.124 g/L	Drink	NL	E	111	
Tetracycline	Avian	200-250	PO	BID	E	111	
Tetracycline	Avian	1.25 ml/L	Drink	NL	E	741	
Tetracycline	Galliformes	30-60	Drink	QD	E	704	
Tetracycline	Pheasant	0.04-0.12 g/L	Feed	QD	G	678	
Tetracycline	Pheasant	55-110 mg/kg food	Feed	QD	G	678	
Tetracycline	Pigeon	220 mg/kg food	Drink	QD	E	704	50%, 2.5 g per teaspoon
Tetracycline	Pigeon	0.549 g/L	Drink	QD	E	1240	For chlamydophilosis
Tetracycline	Pigeon	50 mg TD	PO	QD	G	47	Remove grit from diet during treatment
Tetracycline	Poultry	60	Drink	QD	C	705	
Tetracycline	Psittacine	50	PO	TID	E	565	
Tetracycline	Psittacine	1 g/L soft food	Feed	Once	E	763	May add long-lasting oxytetracycline
Tetracycline	Quail	220 mg/kg food	Feed	QD	G	678	
Tetracycline	Quail	55-110 mg/kg food	Feed	QD	G	678	
Thiabendazole	Anseriformes	550	NL	NL	G	1325	For thorny headed worms
Thiabendazole	Avian	100	PO	QD	E	111	For *Syngamus*
Thiabendazole	Avian	250-500	PO	q2w	E	111	For ascarids
Thiabendazole	Avian	200	PO	q10d	E	924	For *Syngamus, Amidostomum*
Thiabendazole	Avian	100	PO	QD	G	1309	For *Syngamus trachei*
Thiabendazole	Avian	250-500	PO	q10d	G	1309	For ascarids
Thiabendazole	Chicken	5 g/kg food	Feed	QD	G	1325	For ascarids, *Capillaria* and gapeworms
Thiabendazole	Chicken	44	PO	Once	G	1325	For Ascarids, *Capillaria* and gapeworms
Thiabendazole	Crane	100	PO	QW-q2w	E	1361	For intestinal strongyles, ascarids
Thiabendazole	Falcon	100	PO	Once	G	1325	For ascarids, *Capillaria* and gapeworms
Thiabendazole	Pigeon	44	PO	Once	G	1325	For Ascarids, *Capillaria* and gapeworms
Thiabendazole	Raptor	100-200	PO	BID	E	1400	Control of nematodes, may interfere with egg laying

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Thiabendazole	Ratite	50	NL	NL	G	418	
Thiabendazole	Turkey	44	PO	Once	G	1325	For ascarids, *Capillaria* and gapeworms
Thiamine	Anseriformes	25 mg/kg fish	Feed	Once	E	1190	
Thiamine	Anseriformes	25	Feed	QD	G	46	Supplement for fish eaters
Thiamine	Avian	1-2	IM-PO	Once	E	1650	For hypovitaminosis B1
Thiamine	Bird, Piscivorous	0.2 g/kg fish	Feed	QD	E	1554	For fish diet
Thiamine	Bird, Piscivorous	1-2	PO	QD	E	1554	For fish diet
Thiamine	Crane	1-2	PO	QD	E	111	
Thiamine	Duck	100 µg/L	Drink	QD	E	1358	For stargazing and weight loss in ducklings
Thiamine	Eagle, Bald	1-3	NL	QW	E	1188	If eating frozen fish
Thiamine	Osprey	1-3	NL	QW	E	1188	If eating frozen fish
Thiamine	Raptor	1-2	PO	QD	E	111	
Thiamine	Raptor	2	PO	QW	E	1068	While feeding frozen fish
Thiamine	Raptor	0.2-0.25 g/kg fish	Feed	QD	E	1132	For fish diets
Thiamine	Raptor	25	IM	NL	E	1433	
Thiamine	Raptor	1	IM	NL	E	1463	
Thiopental Sodium	Chicken	16.2	IM	PRN	B	1080	Add pentobarbital sodium
Thiopental Sodium	Chicken	10-12.5	NL	PRN	B	1126	
Thiopental Sodium	Duck, Mallard	54	PO	PRN	B	1095	
Thiopental Sodium	Turkey	16.2	IM	PRN	B	1080	Add pentobarbital sodium
Thyroid Stimulating Hormone	Amazon Parrot	1 IU TD	IM	Once	B	88	
Thyroid Stimulating Hormone	Cockatiel	0.1 U TD	IM	Once	B	88	
Thyroid Stimulating Hormone	Parrot, Grey	1 U TD	IM	NL	B	533	
Thyroid Stimulating Hormone	Pigeon	1 U TD	IM	Once	B	88	
Thyroid Stimulating Hormone	Psittacine	1-2 IU/kg	IM	NL	E	1473	
Tiamulin	Avian	25-50	PO	QD	E	565	
Tiamulin	Avian	1.08 mg/kcal	PO	QD	E	1180	
Tiamulin	Avian	25	PO	QD	E	1492	For mycoplasmosis
Tiamulin	Chicken	0.25 g/L	Drink	NL	B	857	For *Mycoplasma*
Tiamulin	Galliformes	25-30	Drink	QD	E	704	Use 1/2 strength for prophylaxis

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Tiamulin	Pigeon	0.225 g/L	Drink	QD	E	704	
Ticarcillin	Avian	150-200	IM-IV	TID-QID	E	111	
Ticarcillin	Avian	5.1 mg/kcal	IM-IV	q2-4h	E	1180	Synergistic with aminoglycosides
Ticarcillin	Raptors	200	IM	TID	E	1027	
Ticarcillin	Pigeon	200	IM-IV	BID-TID	G	590	
Ticarcillin	Psittacine	200	IM	BID-QID	E	565	
Tiletamine + Zolazepam	Albatross	2	IV	PRN	E	1617	Induction for gas anesthesia
Tiletamine + Zolazepam	Avian	5-10	IM	PRN	E	1181, 1231	Good immobilization
Tiletamine + Zolazepam	Avian	2	IV	PRN	E	1617	Induction for gas anesthesia
Tiletamine + Zolazepam	Budgerigar	15-20	IM	PRN	E	243	
Tiletamine + Zolazepam	Budgerigar	20-22	IM	PRN	G	518	
Tiletamine + Zolazepam	Cassowary	2-3	IV	PRN	E	1533	
Tiletamine + Zolazepam	Chicken	30	IM	Once	B	1737	
Tiletamine + Zolazepam	Cockatoo, G Sulfur-crested	2.64-25.2	IM	PRN	G	518	
Tiletamine + Zolazepam	Dove, Ring-necked	50-75	IM	PRN	B	1393	
Tiletamine + Zolazepam	Duck	5-10	IM	PRN	E	243	
Tiletamine + Zolazepam	Eagle, Bald	15	NL	PRN	D	1401	Supplement with 15 mg/kg ketamine
Tiletamine + Zolazepam	Egret, Cattle	2	IV	PRN	E	1617	Induction for gas anesthesia
Tiletamine + Zolazepam	Emu	2-3	IV	PRN	E	1533	
Tiletamine + Zolazepam	Emu	5-10	IM	PRN	E	1617	Induction for gas anesthesia
Tiletamine + Zolazepam	Flamingo	22	IM	PRN	G	518	
Tiletamine + Zolazepam	Flamingo, Chilean	6.6	IM	PRN	B	1393	
Tiletamine + Zolazepam	Gannet	2	IV	PRN	E	1617	Induction for gas anesthesia
Tiletamine + Zolazepam	Goose, Lesser Magellan	6.6-8.8	IM	PRN	B	1393	
Tiletamine + Zolazepam	Goose, White-fronted	2.7	IM	PRN	B	1393	
Tiletamine + Zolazepam	Hawk	15	NL	PRN	D	1401	May cause salivation and poor immobilization
Tiletamine + Zolazepam	Hornbill, Rhinoceros	28.7	IM	PRN	G	518	
Tiletamine + Zolazepam	Ibis, Wood	11	IM	PRN	G	518	
Tiletamine + Zolazepam	Jabirus	2	IV	PRN	E	1617	Induction for gas anesthesia
Tiletamine + Zolazepam	Macaw, Scarlet	4.4-11	IM	PRN	B	1393	
Tiletamine + Zolazepam	Osprey	9.26-17.6	IM	PRN	G	518	
Tiletamine + Zolazepam	Ostrich	5-20	IM	PRN	B	522	Chick dosage
Tiletamine + Zolazepam	Ostrich	4-5	IM	PRN	B	1393	
Tiletamine + Zolazepam	Ostrich	5-10	IM	PRN	G	481	Add diazepam just prior to recovery for captive animal

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Tiletamine + Zolazepam	Owl	10	NL	PRN	D	1401	Supplement with 10 mg/kg ketamine PRN
Tiletamine + Zolazepam	Parakeet, Ring-necked	26	IM	PRN	B	1393	
Tiletamine + Zolazepam	Parrot, Patagonian	11	IM	PRN	G	518	
Tiletamine + Zolazepam	Partridge, Crested Wood	10	IM	PRN	B	1393	
Tiletamine + Zolazepam	Pea Fowl	11.3	IM	PRN	G	518	
Tiletamine + Zolazepam	Pelican	2	IV	PRN	E	1617	Induction for gas anesthesia
Tiletamine + Zolazepam	Pheasant, Golden	16.6	IM	PRN	B	1393	
Tiletamine + Zolazepam	Pigeon	40-60	IM	PRN	B	1393	
Tiletamine + Zolazepam	Pigeon	30-60	IM	Once	B	1737	
Tiletamine + Zolazepam	Plover	17.6	IM	PRN	G	518	
Tiletamine + Zolazepam	Psittacine	10	NL	PRN	D	1401	
Tiletamine + Zolazepam	Psittacine	5-10	IM	PRN	E	1240	Provides good immobilization
Tiletamine + Zolazepam	Raptor	10-20	IM	NL	D	1400	
Tiletamine + Zolazepam	Ratite	2-5	IM	PRN	G	418	Supplement with ketamine PRN
Tiletamine + Zolazepam	Stork	2-5	IM	NL	E	243	Sedation
Tiletamine + Zolazepam	Swan, Black	6.6	NL	PRN	D	1401	Supplement with 6.6 mg/kg ketamine PRN
Tiletamine + Zolazepam	Teal, Blue-winged	22-35	IM	PRN	B	1393	
Tiletamine + Zolazepam	Woodcock	44	IM	PRN	G	518	
Tilmicosin	Chicken	0.075 g/L	Drink	NL	A	804	For *Mycoplasma*
Tilmicosin	Chicken	0.25-0.5 g/L	Drink	NL	B	768	For *Mycoplasma*
Tilmicosin	Chicken	0.1-0.3 g/L	Drink	NL	B	798	
Tinidazole	Avian	50	PO	NL	G	57	
Tobramycin	Avian	10	IM	BID-TID	E	565	
Tobramycin	Avian	5	IM	BID-TID	E	704	
Tobramycin	Avian	5 g/L	Nebulize	TID	E	741	For sinusitis or airsacculitis
Tobramycin	Crane	2.5-5	IM	BID	E	1473	
Tobramycin	Parrot, Grey	10	IM	TID	E	741	Possible polyuria
Tobramycin	Pheasant	2.5-5	IM	BID	E	1473	
Tobramycin	Psittacine	2.5-5	IM	BID	E	111	
Tobramycin	Psittacine	5-10	IT	QD	E	741	Add carbenicillin for pneumonia
Tobramycin	Raptor	2.5-5	IM	BID	E	1240	For resistant *Pseudomonas*
Tobramycin	Raptor	5-10	IM-IV	BID	E	1400	Less nephrotoxic than other aminoglycosides
Tobramycin	Raptor	10	IM	BID	G	234	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Tolazoline HCl	Avian	15	IV	PRN	E	1320	Reverse xylazine +detomidine + medetomidine
Tolazoline HCl	Ostrich	1	IV	PRN	G	481	Reverse xylazine
Tolazoline HCl	Raptor	15	IV	PRN	G	201	
Toltrazuril	Anseriformes	0.0125 g/L	Drink	QD	D	1150	For coccidiosis
Toltrazuril	Avian	7	PO	QD	D	1221	Antiprotozoal
Toltrazuril	Avian	0.15 g/L	Drink	NL	E	1180	
Toltrazuril	Avian	0.090 mg/kcal	PO	QD	E	1180	Coccidiostatic
Toltrazuril	Avian	7-10	PO	QD	E	1554	For coccidiosis
Toltrazuril	Chicken	6	PO	QD	E	1479	May repeat after 5 days for treatment and control of *Eimeria* coccidiosis
Toltrazuril	Chicken	25	PO	QD	G	564	
Toltrazuril	Falcon	25	PO	QW	B	1134	For *Caryospora* infestation
Toltrazuril	Kestrel, European	25	PO	QD	B	1131	For *Caryospora* infection
Toltrazuril	Kiwi	25	PO	Once	G	1223	For coccidiosis
Toltrazuril	Mynah, Bali	12.5	PO	QD	D	1438	For coccidiosis
Toltrazuril	Turkey	0.025 g/L	Drink	QD	B	1025	
Tribromoethanol	Bird, Seed-eater	2 g/kg grain	Feed	Once	E	4	Add chloralose
Tribromoethanol	Bird, Seed-eater	0.025 g/L	Feed	Once	E	4	
Tribromoethanol	Booby	125	PO	PRN	B	1081	Add amylene hydrate
Tribromoethanol	Cormorant	125	PO	PRN	B	1081	Add amylene hydrate
Tribromoethanol	Duck, Mallard	100-158	PO	PRN	B	1095	
Tribromoethanol	Frigatebird, Magnificent	125	PO	PRN	B	1081	Add amylene hydrate
Tribromoethanol	Turkey	40-44 g/L bait	Feed	Once	G	1767	Corn is usual bait
Trichlorfon	Raptors	1.5 g/L (0.15%)	Topical	NL	G	1408	Spray for mites
Tricide	Avian	5 g/L	Topical	NL	G	1278	Spray on wounds and dermatitis
Tricide	Avian	5 g/L	Flush	NL	G	1278	Sinus flush, add antibiotic to solution, potentiates antibiotics amikacin, neomycin, enrofloxacin
Trimethoprim + Sulfa	Anseriformes	0.048 g/L	Drink	QD	E	1240	For nephritis, septicemia, hepatitis. For *E. coli, Pasteurella, Proteus, Salmonella, Listeria*
Trimethoprim + Sulfa	Avian	25	IM	BID	E	1434	
Trimethoprim + Sulfa	Avian	50	IM	QD	E	1434	
Trimethoprim + Sulfa	Avian	30-60	PO-SC	BID	E	1483	Emergency therapy

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Trimethoprim + Sulfa	Avian	50-100	PO	BID	G	1319	
Trimethoprim + Sulfa	Avian	0.15-0.4 g/L	Drink	NL	E	1180	Dosage based on trimethoprim
Trimethoprim + Sulfa	Avian	20-50	PO	BID	E	1431	For coccidiosis
Trimethoprim + Sulfa	Avian	1 g/L	Drink	QD	E	1431	For coccidiosis
Trimethoprim + Sulfa	Avian	10	IM	BID	E	1431	For coccidiosis
Trimethoprim + Sulfa	Bustard	8-30	IM	BID	E	1240	For nephritis, septicemia, hepatitis. For *E. coli, Pasteurella, Proteus, Salmonella, Listeria*
Trimethoprim + Sulfa	Canary	0.05-0.1 g/L	Drink	QD	E	695	Dosage based on trimethoprim
Trimethoprim + Sulfa	Canary	0.1 g/kg food	Feed	QD	E	695	Dosage based on trimethoprim
Trimethoprim + Sulfa	Canary	0.2 g/kg food	Feed	Once	E	1187	Dosage based on trimethoprim only
Trimethoprim + Sulfa	Canary	0.15-0.4 g/L	Drink	NL	E	1187	Dosage based on trimethoprim only
Trimethoprim + Sulfadiazine	Chicken	46-62	Drink	QD	A	1023	
Trimethoprim + Sulfachlorpyridazine	Goose	0.3-0.7 g/kg	Feed	Once	A	776	For *Pasteurella multocida,* trimethoprim to sulfa ratio 1:5
Trimethoprim + Sulfa	Ostrich	15	IM	QD	G	401	
Trimethoprim + Sulfa	Passerine	0.2 g/L	Drink	QD	E	1437	Dosage based on trimethoprim, add drug to food at the same time
Trimethoprim + Sulfa	Passerine	0.2 g/kg	Feed	QD	E	1437	Dosage based on trimethoprim, add drug to drink at the same time
Trimethoprim + Sulfa	Penguin, African	110	PO	BID	G	128	
Trimethoprim + Sulfa	Pigeon	120	PO	QD	E	704	
Trimethoprim + Sulfa	Poultry	55	PO	BID	G	585	
Trimethoprim + Sulfa	Poultry	0.3-0.5 g/L	Drink	QD	G	585	
Trimethoprim + Sulfa	Psittacine	20	PO	BID-TID	E	1240	For nephritis, septicemia, hepatitis. For *E. coli, Pasteurella, Proteus, Salmonella, Listeria*
Trimethoprim + Sulfa	Psittacine	8	IM	BID	E	1240	For nephritis, septicemia, hepatitis. For *E. coli, Pasteurella, Proteus, Salmonella, Listeria*
Trimethoprim + Sulfa	Psittacine	16-100	PO	BID-TID	E	1756	Use lower dosage for birds under 300 g for bacterial nephritis
Trimethoprim + Sulfa	Psittacine	52.8	IM	QD-BID	G	1309	For respiratory and enteric infections
Trimethoprim + Sulfa	Raptor	30	SC	BID	E	1240	For nephritis, septicemia, hepatitis. *For E. coli, Pasteurella, Proteus, Salmonella, Listeria*
Trimethoprim + Sulfa	Raptor	20-50	PO	BID	E	1433	Do not use on dehydrated birds
Trimethoprim + Sulfa	Raptor	60	PO	BID	E	1400	For coccidiosis, repeat after 2 days
Trimethoprim + Sulfa	Raptor	17.5	PO	QD	G	94	Juvenile dosage

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Trimethoprim + Sulfa	Raptor	50	IM	BID	G	234	
Trimethoprim + Sulfa	Ratite	30-50	IM	BID	G	1308	For toxoplasmosis
Trimethoprim + Sulfamethoxazole	Pigeon	60	PO	QD	A	968	
Trimethoprim + Sulfamethoxazole	Psittacine	16-24	PO	BID-TID	A	1473	
Trimethoprim + Sulfamethoxazole	Psittacine	8	IM	BID	A	1473	
Trimethoprim + Sulfatroxazole	Pigeon	60	PO	BID	A	968	
Tubocurarine Cl	Raptor	3 g/L	Ophthalmic	NL	E	111	Every 5 minutes
Tylosin	Anseriformes	20-30	IM	TID	D	1150	For mycoplasmosis
Tylosin	Anseriformes	20	PO	TID	D	1150	For mycoplasmosis
Tylosin	Anseriformes	10 g/L	Flush	QD	D	1150	For mycoplasmosis, use 10 ml per bird
Tylosin	Avian	0.75 g/L	Drink	NL	E	1180	
Tylosin	Avian	0.86 mg/kcal	IM	TID-QID	E	1180	
Tylosin	Avian	1.08 mg/kcal	PO	QD	E	1180	
Tylosin	Avian	10 g/L saline	Flush	BID	E	1183	Nasal or sinus
Tylosin	Avian	10 g/L saline	Nebulize	BID	E	1185	10 to 60 minutes
Tylosin	Avian	15	IM	TID	E	1240	For birds > 1 kg for upper respiratory infection for *Mycoplasma, Pasteurella, Chlamydophila*
Tylosin	Avian	10	IM	BID	E	1617	For Mycoplasma
Tylosin	Avian	10 g/L	Nebulize	TID-QID	E	1650	Add dimethyl sulfoxide, adjunct to parenteral therapy for air sacculitis
Tylosin	Avian	10-40	IM	BID	E	1650	May use with lincomycin + spectinomycin for mycoplasmosis
Tylosin	Bird, Aquatic	10	IM	BID	E	1503	For *Mycoplasma*
Tylosin	Bustard, Houbara	20	IM	TID	G	932	For *Mycoplasma*
Tylosin	Canary	0.25-0.4 g/L	Drink	QD	E	695	
Tylosin	Canary	0.4 g/kg food	Feed	Once	E	1187	
Tylosin	Chicken	0.5 g/L	Drink	NL	A	838	For *Mycoplasma gallisepticum*, less effective than danofloxacin
Tylosin	Chicken	0.25 g/L	Drink	QD	B	1016	Add colistin for chronic respiratory disease with E. coli
Tylosin	Cockatiel	Mix powder 1:10	Ophthalmic	BID-TID	E	111	Mix powder with sterile water 1 to 10

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Tylosin	Cockatiel	1:10 dilution water	Topical	QD-TID	E	741	Eye spray, powder 250 g/8.8 oz, conjunctivitis, may use with tylosin in water
Tylosin	Crane	15	SC	TID	A	596	
Tylosin	Emu	15-25	IM	TID-QID	E	1473	
Tylosin	Falcon	30	IM	TID	E	1027	For *Mycoplasma*
Tylosin	Finch	0.4 g/kg food	Feed	QD	E	1572	
Tylosin	Finch	0.25-0.4 g/L	Drink	QD	E	1572	
Tylosin	Finch, House	1 g/L	Drink	QD	D	1439	Add ciprofloxacin for mycoplasmosis
Tylosin	Finch, House	0.3 g/L	Drink	QD	D	1439	Preventive for mycoplasmosis
Tylosin	Ostrich	11.4	PO	TID	G	467	
Tylosin	Passerine	0.25-0.4 g/L	Drink	QD	E	1437	Add drug to food at the same time
Tylosin	Passerine, Small	0.25-0.4 g/L	Drink	NL	E	1187	
Tylosin	Pigeon	25	IM	QID	A	847	
Tylosin	Pigeon	15-25	IM	TID-QID	E	111	
Tylosin	Pigeon	50	PO	QD	E	704	
Tylosin	Pigeon	0.5 g/L	Drink	QD	E	704	
Tylosin	Pigeon	15-25	IM	TID-QID	E	1473	
Tylosin	Psittacine	Mix powder 1:10	Ophthalmic	BID-TID	E	111	Mix powder with sterile water 1 to 10
Tylosin	Psittacine	40	IM	NL	E	763	Initial *Mycoplasma* therapy, used in combination with aminoglycosides
Tylosin	Psittacine	20-40	IM	TID	E	1240	
Tylosin	Quail	15-25	IM	TID-QID	E	1473	
Tylosin	Raptor	15	IM	QD	D	1612	For chronic air sacculitis
Tylosin	Raptor	20	IM	BID	G	234	
Tylosin	Rhea	12.5 mg TD	Parenteral	Once	G	582	Into distended sinus
Tylosin	Rhea	50 mg TD	SC	QD	G	582	Add penicillin G benzathine
Urate Oxidase	Hawk, Red-tailed	100-200 KIU/kg	NL	NL	A	1761	Uricolytic
Urate Oxidase	Pigeon	20-600 KIU/kg	NL	NL	A	1761	Uricolytic
Valnemulin	Chicken	0.5 g/L	Drink	QD	B	873	For *Mycoplasma*
Vasotocin, Arginine	Sparrow, House	0.0004-0.0016	IV	PRN	B	1759	Dosage is dosage per minute, used for antidiuresis
Vecuronium Bromide	Avian	1 drop 4mg/ml soln	Ophthalmic	q15min	E	1233	

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Vecuronium Bromide	Cockatoo	0.8 g/L	Ophthalmic	NL	G	218	
Vincristine Sulfate	Avian	0.75 mg/m^2 surface	IO	QW	E	1470	For lymphosarcoma - Check curent literature
Vinegar, Apple Cider	Avian	15 ml/L	Drink	QD	D	1330	For low-grade candidiasis
Vinegar, Apple Cider	Avian	60-120 ml/L	Drink	QD	E	1205	For dysbiosis, *Clostridium*, GI tract bacteriosis
Vinegar, Apple Cider	Avian	60-120 ml/L	Drink	QD	E	1487	For dysbiosis, candidiasis, chronic bacterial diarrhea, Gram negative infections, foul smell feces
Virginiamycin	Poultry	22 mg/kg food	Feed	QD	C	564	
Vitamin A	Amazon, Red Lored	20 KIU/kg	IM	Once	G	700	
Vitamin A	Avian	< 50 KIU/kg	NL	Once	E	1481	For clinical deficiency
Vitamin A	Raptor	< 20 KIU/kg	IM	QW	E	1400	For hypovitaminosis A and epithelial regeneration (bumblefoot)
Vitamin B Complex	Amazon, Yellow-naped	1	Parenteral	NL	G	1697	Dosage based on thiamine
Vitamin B Complex	Avian	1-3	IM	QW	E	1470	After hemorrhage with liver disease
Vitamin B Complex	Avian	10	IM	NL	E	1650	Dosage based on thiamine for emergency care
Vitamin B Complex	Bird, Aquatic	10-30	IM-SC	QW	E	1503	For debilitation, anemia, appetite stimulant, neurological disorders
Vitamin B Complex	Hawk, Red-tailed	10	PO	Once	G	1644	Dosage based on thiamine for debilitation
Vitamin B Complex	Psittacine	10-30 (thiamine dose)	IM	q2d	E	1240	Dosage based on thiamine, neuromuscular disease, hepatic disorders, thiamine-responsive seizures
Vitamin B Complex	Raptor	10-30	IM	QW	E	1240	Dosage based on thiamine, stimulate appetite
Vitamin D	Avian	6.6 KIU/kg	IM	Once	G	58	Add calcium gluconate + oxytocin
Vitamin E	Avian	40	IM-PO	NL	E	924	Antioxidant, metal toxicity
Vitamin E	Avian	0.05-0.1	Parenteral	QW	E	1650	For hypovitaminosis E
Vitamin E	Bird, Aquatic	0.1 KIU/kg oil fish	Feed	Once	E	1478	
Vitamin E	Ostrich	0.2-0.3 KIU/kg	IM	NL	G	93	Juvenile dose

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Vitamin E + Selenium	Amazon, Red Lored	1 mg Se, 25 mg E	IM	Once	G	700	
Vitamin E + Selenium	Avian	0.05-0.1	IM	q2w	E	111	
Vitamin E + Selenium	Ostrich	4-100 KIU TD	IM	QM	G	401	
Vitamin E + Selenium	Raptor		SC	q3d	E	1240	0.05 mg selenium + 3.4 IU vitamin E total dose for muscular weakness, capture myopathy, deficiency
Vitamin K	Amazon, Yellow-naped	0.2	Parenteral	NL	G	1697	
Vitamin K	Avian	0.2-2.2	IM	q4-8h	G	87	Follow with daily dose for 1 to 4 weeks
Vitamin K	Avian	0.2-2.5	IM	PRN	E	1151	For hematochezia
Vitamin K	Avian	0.025-2.5	NL	BID	F	58	Give every 2 to 3 weeks
Vitamin K	Avian	0.2-2.5	Parenteral	NL	G	246	
Vitamin K	Lory, Chattering	0.2	IM	BID	G	42	
Vitamin K	Psittacine	0.25-0.5	IM	q2d-QW	E	632	Neonate dosage
Vitamins A, D, E	Avian	36.3 KIU/kg	IM	QW	E	111	Give 33 KIU vitamin A + 3.3 KIU vitamin D per kg
Vitamins A, D, E	Pigeon	33 KIU Vit A/kg	IM	QW	G	590	Dosage based on Vitamin A
Vitamins A, D, E	Psittacine	33-66 KIU/kg	IM	QW	E	1240	For deficiencies, reproductive disorders and bone healing
Xylazine HCl	Amazon Parrot	1-2	IM	PRN	E	243	Add ketamine
Xylazine HCl	Amazon Parrot	1-2 mg TD	IV	PRN	G	1609	Add ketamine, mix together in syringe, may start with half dose
Xylazine HCl	Avian	4	IM	PRN	E	704	Add ketamine
Xylazine HCl	Avian	2.2	IM	PRN	E	1181	Add ketamine, reverse with atipamezole, prolonged recovery if not reversed
Xylazine HCl	Avian	2.2	IV	PRN	E	1431	Add ketamine, good for short surgical procedures, must reverse
Xylazine HCl	Budgerigar	6.5	IM	PRN	E	243	Add ketamine
Xylazine HCl	Cassowary	0.25	IV	PRN	E	1617	Add ketamine, use yohimbine to shorten recovery
Xylazine HCl	Cassowary	1	IM	PRN	E	1617	Add ketamine, use yohimbine to shorten recovery
Xylazine HCl	Cockatiel	2.5	IM	PRN	E	243	Add ketamine
Xylazine HCl	Crane	1	NL	PRN	E	1189	Add ketamine for anesthesia
Xylazine HCl	Emu	0.25	IV	PRN	E	1617	Add ketamine, use yohimbine to shorten recovery
Xylazine HCl	Falcon	2	IM	PRN	E	243	Add ketamine

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Xylazine HCl	Hawk	2.2	NL	PRN	G	1388	Add ketamine
Xylazine HCl	Ostrich	0.66	IM	PRN	B	447	Add acepromazine + etorphine
Xylazine HCl	Ostrich	150 mg TD	IM	PRN	G	283	Add carfentanil
Xylazine HCl	Parrot, Grey	1.5	IM	PRN	E	243	Add ketamine
Xylazine HCl	Pigeon	10	IM	PRN	G	1481	Add butorphanol + ketamine for surgery
Xylazine HCl	Psittacine	1-2.2	IM-IV	PRN	E	1240	Use in combination with ketamine 1:3 or 1:5, reverse with yohimbine
Xylazine HCl	Psittacine	1-10	IM	PRN	E	1573	Sedation for small birds at high dosages
Xylazine HCl	Psittacine	1-10	IM	PRN	G	201	Sedation, small psittacine
Xylazine HCl	Raptor	0.5	IV	PRN	D	1401	Add ketamine
Xylazine HCl	Raptor	3	IM	PRN	E	4	Add ketamine
Xylazine HCl	Raptor	1-2.2	IM-IV	PRN	E	1240	Use in combination with ketamine 1:3 or 1:5, reverse with yohimbine
Xylazine HCl	Raptor	2	IV	PRN	G	1174	Add ketamine for diurnal raptors, lasts 1 hour
Xylazine HCl	Raptor	0.25-0.5	IV	PRN	G	1174	Add ketamine, lower dosage regimen produces less respiratory disturbance
Xylazine HCl	Ratite	2.2	IM	PRN	E	4	Add ketamine
Xylazine HCl	Ratite	1-2.2	IM	PRN	E	4	Immobilization
Xylazine HCl	Ratite	0.5-1	IM	PRN	E	243	Add ketamine after 15 minutes
Xylazine HCl	Ratite	1-2.2	Parenteral	PRN	G	418	Heavy sedation
Xylazine HCl	Stork	0.2-0.4	IM	PRN	E	243	Sedation
Xylazine HCl	Swan (Black, Mute)	2 mg TD	IV	PRN	G	1291	Add ketamine, give 3/4 dose initially, remainder if needed, gas anesthesia premed
Xylazine HCl	Swan, Mute	0.28	IV	PRN	E	1190	Add ketamine
Yeast Cell Derivatives	Avian	N/A	Topical	PRN	E	111	Promote skin healing, Preparation H®
Yohimbine HCl	Avian	1	IO-IV	PRN	E	243	
Yohimbine HCl	Avian	0.1-0.2	IV	PRN	E	1181	Reverse xylazine
Yohimbine HCl	Avian	0.1	NL	PRN	E	1231	Reversal of xylazine
Yohimbine HCl	Avian	0.125-1	NL	PRN	E	1481	Reverse xylazine
Yohimbine HCl	Budgerigar	0.11-0.27	IM	Once	G	83	
Yohimbine HCl	Cassowary	0.2	IV	PRN	E	1533	Shorten ketamine + xylazine anesthesia
Yohimbine HCl	Emu	0.2	IV	PRN	E	1617	To shorten xylazine recovery
Yohimbine HCl	Guinea Fowl	1	IV	PRN	B	553	
Yohimbine HCl	Guinea Fowl	0.15	NL	PRN	D	1401	Antagonize ketamine/xylazine anesthesia

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

DRUG NAME	SPECIES	DOSAGE (mg/kg unless otherwise stated)	ROUTE	DOSAGE INTERVAL	C.I.**	REFERENCE	COMMENTS (see references for duration, precautions and contraindications)
Yohimbine HCl	Hawk, Red-tailed	0.1	NL	PRN	D	1401	Antagonize ketamine/xylazine anesthesia
Yohimbine HCl	Ostrich	0.125	NL	PRN	D	1401	Antagonist for xylazine
Yohimbine HCl	Ostrich	12.5 mg TD	IV	PRN	G	283	
Yohimbine HCl	Ostrich	0.125	IV	PRN	G	481	Reverse xylazine
Yohimbine HCl	Psittacine	0.1-0.2	IV	PRN	E	1240	Reverse xylazine
Yohimbine HCl	Raptor	0.1-0.2	IV	PRN	E	1240	Reverse xylazine
Yohimbine HCl	Raptor	0.1	IV	PRN	G	201	
Yohimbine HCl	Ratite	0.125	IV	PRN	G	418	
Yunnan Paiyao	Avian	1 ml/kg	PO	QD	E	1435	Stock solution 2 capsules per 15 ml lactulose
Yunnan Paiyao	Avian	1.25-2.5 ml TD	PO	BID	G	1112	Mix with 3 (cockatiel) to 10 (macaw) ml water, use 1 day pre-operative, also use as topical flush
Zeranol	Ostrich	12 mg TD	SC	NL	C	283	Implant growth promotion
Zinc Methionine	Raptor	25	PO	QD	E	1359	Antioxidant therapy, administer during lead chelation therapy

**A = Pharmacokinetic research; B = Clinical efficacy trial; C = Manufacturer's recommendation; D = Published with reference; E = Published without reference; F = Extrapolation from other species; G = Anecdotal

Abbreviations

Abbreviation	Translation
<	less than
>	greater than
>=	greater than or equal
Bath	1 day or more in therapeutic liquid
BID	two times daily
BID-q2d	two times daily to every two days
BID-QID	two to four times daily
BID-TID	two to three times daily
cm^2	square centimeter
cm^3	cubic centimeter
d	day
d > w	days to weeks
Dip	less than 1 day in therapeutic liquid
doses	number of doses to give
Drink	drinking water
Epidural	epidural injection
Feed	include in feed
Flow	constant flow
Flush	flush
Fog	fog the environment
g	gram
ga	gauge
GI	gastrointestinal
gr	grain
h	hour
IA	intraarticular
IA-IM	intraarticular or intramuscular
ICa	intracardiac
ICa-IP-IV	intracardiac or intraperitoneal or intravenous
ICr	intracranial
ID	intradermal
IH	inhalation
IM	intramuscular
IM-IN-IO-IV	intramuscular or intranasal or intraosseous or intravenous
IM-IN-IV	intramuscular or intranasal or intravenous
IM-IO-IT-IV	intramuscular or intraosseous or intratracheal or intravenous
IM-IO-IV	intramuscular or intraosseous or intravenous
IM-IP	intramuscular or intraperitoneal
IM-IP-IV	intramuscular or intraperitoneal or intravenous
IM-IP-IV-SC	intramuscular or intravenous or intraperitoneal or subcutaneous
IM-IP-PO	intramuscular or intraperitoneal or by mouth
IM-IP-PO-SC	intramuscular or intraperitoneal or by mouth or subcutaneous
IM-IP-SC	intramuscular or intraperitoneal or subcutaneous
IM-IV	intramuscular or intravenous
IM-IV-PO	intramuscular or intravenous or by mouth
IM-IV-PO-SC	intramuscular or intravenous or by mouth or subcutaneous
IM-IV-SC	intramuscular or intravenous or subcutaneous
IM-PO	intramuscular or by mouth
IM-PO-SC	intramuscular or by mouth or subcutaneous
IM-SC	intramuscular or subcutaneous
IM-Topical	intramuscular or topical

Abbreviation	Translation
IN	intranasal
indef	indefinite
IO	intraosseous
IO-IP-IT-IV	intraosseous or intraperitoneal or intratracheal or intravenous
IO-IP-IV-SC	intraosseous or intraperitoneal or intravenous or subcutaneous
IO-IT-IV	intraosseous or intratracheal or intravenous
IO-IV	intraosseous or intravenous
IO-IV-SC	intraosseous or intravenous or subcutaneous
IP	intraperitoneal or intracoelomic or intrapleuroperitoneal
IP-IM	intraperitoneal or intramuscular
IP-IV	intraperitoneal or intravenous
IP-IV-PO	intraperitoneal or intravenous or by mouth
IP-IV-SC	intraperitoneal or intravenous or subcutaneous
IP-PO	intraperitoneal or by mouth
IP-SC	intraperitoneal or subcutaneous
IT	intratracheal
IT-IV	intratracheal or intravenous
IT-IV-PO	intratracheal, intravenous and orally
IU	International Units
IUt	intrauterine
IV	intravenous
IV-PO	intravenous or by mouth
IV-PO-SC	intravenous or by mouth or subcutaneous
IV-SC	intravenous or subcutaneous
kcal	kilocalories
kg	kilogram
KHz	kilohertz
KIU	thousand International Units
L	liter
life	lifelong therapy
long	long therapy regimen
m	month
m^2	square meter
m^3	cubic meter
MD	maximum dose
mEq	milliequivalent
mg	milligram
min	minute
MIU	million International Units
ml	milliliter
mm^2	square millimeter
mmol	millimoles
mol	molar solution
months	for several months
N	normal solution
nebulize	nebulization
NL	not listed
once	give single dose
ophthalmic	in the eye
oz	ounce
Parenteral-PO	parenteral or by mouth
PO	by mouth
PO-SC	by mouth or subcutaneous
PO-SC-Topical	by mouth or subcutaneous or topical

Abbreviation	Translation
PO-Topical	by mouth or apply topically
ppm	parts per million
ppt	parts per thousand
PRN	as needed
q10-14d	every ten to fourteen days
q10d	every ten days
q1-2h	every one to two hours
q1-4h	every one to four hours
q15min	every fifteen minutes
q2-3d	every two days to three days
q2-3h	every two to three hours
q2-3w	every two to three weeks
q2-4d	every two days to four days
q2-4h	every two to four hours
q2-4w	every two weeks to four weeks
q2-5d	every two days to five days
q2-6h	every two to six hours
q2d	every two days
q2d-QW	every two days to every week
q2h	every two hours
q2m	every two months
q2w	every two weeks
q30min	every thirty minutes
q33h	every thirty three hours
q3-4d	every three to four days
q3-4h	every three to four hours
q3-4w	every three to four weeks
q3-5d	every three to five days
q36h	every thirty six hours
q3-6h	every three to six hours
q3-6w	every three to six weeks
q3-7d	every three to seven days
q3-8h	every three to eight hours
q3d	every three days
q3d-q2w	every 3 days to two weeks
q3d-QW	every three days to every week
q3h	every three hours
q3m	every three months
q3w	every three weeks
q4-5d	every four to five days
q4-5h	every four to five hours
q4-6d	every four to six days
q4-6h	every four to six hours
q4-6m	every four to six months
q4-6w	every four to six weeks
q4-8h	every four to eight hours
q4d	every four days
q4d-QW	every four days to weekly
q4h	every four hours
q4m	every four months
q5d	every five days
q5d-QW	every five days to every week

Abbreviation	Translation
q5h	every five hours
q5min	every five minutes
q60h	every sixty hours
q6d	every six days
q6m	every six months
q6m-QA	every six months to every year
q6w	every six weeks
q7w	every seven weeks
QA	every year
QD	every day
QD-BID	every day to two times daily
QD-q2d	every day or every two days
QD-q3d	every day to every three days
QD-QID	every day to four times daily
QD-QW	every day to every week
QD-TID	every day to three times daily
QH	every hour
QID	four times daily
QM	every month
QM-q2m	every month to every two months
qow	every other week
QS	add sufficient quantity
QW	every week
QW-q10d	every week to ten days
QW-q2w	ever week to every two weeks
QW-q3w	every week to every three weeks
r10d	repeat in ten days
r2m	repeat in two months
r2w	repeat in two weeks
r6m	repeat in six months
r7-10d	repeat in seven to ten days
ra	repeat annually
rm	repeat in one month
rw	repeat in one week
SC	subcutaneous
SCJ	subconjunctival
SC-Topical	subcutaneous or topical
sec	second
short	short therapy regimen
slowly	administer slowly
soln	solution
TD	total dose
TID	three times daily
TID-q2d	three times daily to every two days
TID-QID	three to four times daily
topical	apply topically
vapor	vapor in enclosure
vent	anus, rectum or cloaca
w	week
weeks	for several weeks
y	year
µg	microgram

References

4. Fowler M: Restraint And Handling of Wild And Domestic Animals. Iowa State University Press Ames 2 1995.
6. Kollias G: Diets feeding practices and nutritional problems in psittacine birds. Vet Med 90 29-39 1995.
16. Orosz S: Fungal and mycobacterial diseases. Proc N Am Vet Conf Eastern States Vet Assoc Gainesville FL 569-70 1995.
30. Miller P, Langenberg J, et al: *Pseudomonas aeruginosa* associated corneal ulcers in captive cranes. J Am Assoc Zoo Vet 25 449-54 1995.
31. Law J, Tully T, et al: Verminous encephalitis apparently caused by the Filaroid nematode in emus. Avian Dis 37 597-601 1993.
32. Ramsay E, Vulliet R: Pharmacokinetic properties of gentamicin and amikacin in the cockatiel. Avian Dis 37 628-634 1993.
40. Coleman C and Oliver R Lymphosarcoma in a blue and gold macaw and a mature canary J Assoc Avian Vet 8 64-8 1994.
41. Wack R, Kramer L, et al: Cardiomegaly and endocardial fibrosis in a secretary bird. J Assoc Avian Vet 8 76-80 1994.
42. Orcutt C, Bartick T: Mucormycotic meningoencephalitis and pneumonia in a chattering lory. J Assoc Avian Vet 8 85-9 1994.
43. Ramsay E, Grindlinger H: Treatment of obsessive behavior with clomipramine J Assoc Avian Vet. 8 9-15 1994.
44. Oglesbee B, Oglesbee M: Feather dystrophy in a cockatiel. J Assoc Avian Vet 8 16-20 1994.
45. Clyde V, Kollias G: Diet associated gout in juvenile macaws. Proc Am Assoc Zoo Vet 90 1989.
46. Pokras M: Captive management of aquatic birds. AAV Today 2 24-33 1988.
47. Marx D: Preventive medicine for pigeons AAV Today 2 41-43 1988.
48. Dustin L: Clinical conditions in pet canary practice. J Assoc Avian Vet 4 80-2 1990.
49. Haneveld M, Dorrestein G: Sudden high mortality in canaries. J Assoc Avian Vet 4 82 1990.
50. Porter S and Snead S Pesticide poisoning in birds of prey. J Assoc Avian Vet 4 84-5 1990.
51. Degernes L, Talbot B, et.al. Raptor foot care. J Assoc Avian Vet 4 93-5 1990.
52. Rode J, Bartholow S, et al: Ventilation through an air sac canula during tracheal obstruction in ducks. J Assoc Avian Vet 4 98-102 1990.
53. Wheeler C: Avian anesthetics analgesics and tranquilizers. Bauck, L: Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 2 7-12 1993.
54. Huff D: Fluid therapy and nutritional therapeutics. Bauck, L: Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 2 13-16 1993.
55. Bauck L, Hoefer H: Avian antimicrobial therapy. Bauck, L: Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 2 17-22 1993.
56. Prus S: Avian antifungal therapy. Bauck, L: Semin Avian Exotic Pet Med WB Saunders, Philadelphia 2 30-32 1993.
57. Marshal R: Avian anthelmintics and antiprotozoals. Bauck, L: Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 2 33-41 1993.
58. Worrell A: Therapy of noninfectious avian disorders. Bauck, L: Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 2 42-7 1993.
61. Porter S: Veterinary care of native wildlife. Proc N Am Vet Conf Eastern States Vet Assoc, Gainesville, FL 693-6 1995.
62. Tseng F: Medical management of oiled wildlife. Proc N Am Vet Conf Eastern States Vet Assoc Gainesville, FL 697-8 1995.
68. Kollias G, Palgut J, et al: The use of ketoconazole in birds: preliminary pharmacokinetic and clinical applications. Proc Assoc Avian Vet AAV, Miami 103 1986.
80. Harris D: Care of the critically ill patient. Jenkins, J: Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 3 175-9 1994.
83. Jenkins J: Postoperative care. Murray, M: Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 2 97-102 1993.
85. Bauck L: Nutritional problems in pet birds. Rosenthal, K: Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 4 3-8 1995.
86. Degernes L: Toxicities in waterfowl. Rosenthal, K: Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 4 15-22 1995.
87. LaBonde J: Toxicity in pet avian patients. Rosenthal, K: Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 4 23-31 1995.
88. Rae M Endocrine disease in pet birds. Rosenthal, K: Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 4 32-8 1995.
94. Joseph V: Raptor pediatrics. Speer B: Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 2 142-51 1993.
96. Clipsham R: Rhamphorthotics and surgical corrections of maxillofacial defects. Redig, P: Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 3 92-99 1994.
109. van Reeth K, Vercruysse J: Efficacy of toltrazuril against experimental Eimeria in racing pigeons. Avian Dis 37 218-21 1993.
110. Greth A, Gerlach H, et.al: Pharmacokinetics of doxycycline after parenteral administration in the houbara bustard, Avian Dis 37 31-6 1993.
111. Ritchie B, Harrison G: Formulary. Ritchie B, Harrison G, et al: Avian Medicine: Principles and Application, Wingers Publishing, Lake Worth FL 457-78 1994.
112. Gronwell R, Brown M, et al: Pharmacokinetics of amikacin in African grey parrots, Am J Vet Res 50 250-2 1989.
123. Page C, Schmidt R, et al: Antemortem diagnosis and treatment of sarcocystosis in two species of psittacines, J Zoo Wildl Med AAZV 23 77-85 1992.
124. Frank T, Carpenter J: Coronaviral enteritis in an ostrich, J Zoo Wildl Med AAZV 23 103-7 1992.
125. Sacre B, Oppenheim Y, et.al. Presumptive histiocytic sarcoma in a great horned owl, J Zoo Wildl Med AAZV 23 113-21 1992.
127. Garner M: Suspected lead toxicosis in captive goshawk, J Am Vet Med Assoc 199 1069-70 1991.
128. Cook R: Clinical challenge case one, J Zoo Wildl Med AAZV 23 135-6 1992.
134. Balanger L, Romano J, et al: Nodular typhlitis of pheasants caused by heterakis J Zoo Wildl Med AAZV 23 249-53 1992.
145. Mainka S, Dierenfeld E, et al: Circulating tocopherol following intramuscular or oral vitamin E administration, J Zoo Wildl Med AAZV 25 229-32 1994.
153. Aguilar R, Stiles J, et al: Surgical synechiotomy in an adult bald eagle, J Zoo Wildl Med AAZV 24 63-7 1993.
168. Isaza R, Budsberg S, et al: Disposition of oral ciprofloxacin in red tailed hawks on single oral dose, J Zoo Wildl Med AAZV 24 508-14 1993.
173. Hannon D, Swaim S, et al: Full thickness mesh skin grafts in two great horned owls, J Zoo Wildl Med AAZV 24 539-52 1993.
179. Dorrestein G, Verburg E: Pharmacokinetics of Baytril in homing pigeons after different administration routes, Proc Conf Avian Dis Munich 207-9 1988.
201. Clyde V: Avian analgesia. Junge R: Proc Am Assoc Zoo Vet 125-7 1994.
203. Gamble K, Falzone C, et al: Proventricular ascaridiasis in lappet-faced and African vultures. Junge R: Proc Am Assoc Zoo Vet 149-50 1994.
207. Snyder S, Richard M: Treatment of avian tuberculosis in a whooping crane. Junge, R: Proc Am Assoc Zoo Vet 167-9 1994.
230. Jakoby JR, Gylstorff I: Comparative investigations on chemotherapeutic psittacosis control, Berl Munch Tieraerztl Wschr 96 261-4 1983.
232. Keymer I: Pigeons. Beynon P, Cooper J: Manual of Exotic Pets, Iowa State Univ. Press, Ames 180-202 1991.
233. Scullion F: Wild birds. Beynon P, Cooper J: Manual of Exotic Pets Iowa State Univ. Press, Ames 203-11 1991.
234. Forbes N: Birds of prey. Beynon P, Cooper J: Manual of Exotic Pets Iowa State Univ. Press Ames 212-20 1991.
252. Heard D: Principles and techniques of anesthesia and analgesia for exotic practice. Quesenberry K, Hillyer E: Vet Clinic NA Sm Anim Prac WB Saunders, Philadelphia 23:6 1301-27 1993.
260. Harlin R: Pigeons. Roston M, Marx K: Proc Mid-Atlantic States Assoc Avian Vet MASAAV Blacksburg, VA 16 69-82 1995.
262. Graczyk T, Shaw M, et al: Hematological characteristics of avian malaria cases in African black foot penguins, J Parasitol 80 302-8 1994.
266. Teare J, Schwark W, et al: Pharmacokinetics of a long acting oxytetracycline preparation on pheasants, owls and parrots, Am J Vet Res 46 2639-43 1985.
283. Huchzermeyer F: Ostrich Diseases Agricultural Research Council Onderstepoort 1994.
320. Smith H: Serum levels of penicillin dihydrostreptomycin chloramphenicol aureomycin and terramycin in chickens, J Comp Pathol Therap 64 225 1954.
340. Loomis M, Wright J: Treatment of iron storage disease in Bali mynah. Junge R: Proc Am Assoc Zoo Vet AAZV, St. Louis 28 1993.
341. Norton T, Greiner E, et al: Medical aspects of Bali mynah. Junge, R: Proc Am Assoc Zoo Vet AAZV, St. Louis 29-44 1993.
354. Flammer K, Aucoin D, et al: Intramuscular and oral disposition of enrofloxacin in African grey parrots, J Vet Pharmacol Therap 14 359-66 1991.
379. Dorrestein GM: Bioavailability of doxycycline injectables in pigeons. Ffriis C, et al: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Acta Vet, Scand, Copenhagen 5th 291-2 1991.
380. Flammer K, Aucoin D, et al: Potential use of long acting injectable oxytetracyclines for treatment of chlamydiosis, Avian Dis 34 228-34 1990.
385. Hirsh D, Knox S, et al: Pharmacokinetics of penicillin G in the turkey Am J Vet Res 39 1219-21 1978.
394. Clark C, Thomas J, et al: Plasma concentrations of chloramphenicol in birds, Am J Vet Res 43 1249-53 1982.
399. Locke D, Bush M, et al: Pharmacokinetics and tissue concentrations of tylosin in selected avian species, Am J Vet Res 43 1807-10 1982.
401. Foggin C: Veterinary problems of ostriches. Hallam M, Topaz: Introduction to Practical Ostrich Farming, MG Hallam Harare 61-96 1992.
411. Hochleithner M, Georgopo Aulos: Experiences with piperacillin in Psittaciformes, Proc Conf Avian Dis, Munich 157 1988.
418. Jensen J, Johnson J, et al: Husbandry & Medical Management of Ostriches, Emus, & Rheas, Wildl & Exotic Anim Telconsult, College Station 1992.
435. Cross G Antiviral therapy. Fudge A: Semin Avian Exotic Pet Med WB Saunders, Philadelphia 4 96-101 1995.
457. Bush M, Locke D, et al: Pharmacokinetics of cephalothin and cephalexin in selected avian species, Am J Vet Res 42 1014-7 1981.
458. Custer R, Bush M, et al: Pharmacokinetics of gentamicin in blood plasma of quail, pheasants and cranes, Am J Vet Res 40 892-5 1979.
467. Hoover J, Lochner F, et al: Quill mites in an ostrich with rhinitis sinusitis and air sacculitis, Comp Anim Practice 3:2 23-6 1988.
493. Dorrestein G, VanGogh H, et al: Bioavailability and pharmacokinetics of ampicillin and amoxicillin from tablets, capsules... pigeons, J Vet Pharmacol Therap 9 394-401 1986.
508. Dein F, Monard D, et al: Pharmacokinetics of chloram-

phenicol in Chinese spot billed ducks, J Vet Pharmacol Therap 3 161-8 1980.
531. Onderka D, Doornenbal E: Mycotic dermatitis in ostriches Can Vet J 33 72-6 1992.
544. Devriese L, Dutta G: Effects of erythromycin inactivating lactobacillus crop flora on blood levels, J Vet Pharmacol Therap 7 49-53 1984.
562. Perelman B, Kuttin E: Zygomycosis in ostriches, Avian Pathol 21 675-80 1992.
564. Tanner A: Antimicrobial drug use in poultry Prescott J, Baggot J: Antimicrobial Therapy in Veterinary Medicine Iowa State Univ Press, Ames 2 507-23 1993.
565. Dorrestein GM: Antimicrobial drug use in pet birds. Prescott J, Baggot J: Antimicrobial Therapy in Veterinary Medicine, Iowa State Univ. Press, Ames 2 490-507 1993.
567. Ensley P, Janssen D: A preliminary study comparing the pharmacokinetics of ampicillin given orally and intramuscularly, J Zoo Anim Med 12 42-7 1981.
577. Orosz S: Personal communication extending findings of the previous reference, Proc Assoc Avian Vet AAV Reno 13 1994.
578. Tully T and Morris M A treatment protocol for non-responsive arthritis in companion birds, Proc Assoc Avian Vet AAV Reno 45-9 1994.
579. Suedmeyer W Use of Adequan in articular diseases of avian species, J Assoc Avian Vet 7 105 1993.
580. van der Heyden N Update on avian mycobacteriosis Proc Assoc Avian Vet AAV Reno 53-61 1994.
582. Phalen D, Wigle W: Sinusitis in five rheas: response to treatment ,Proc Assoc Avian Vet AAV, Reno 147-50 1994.
583. Doolen M: Antimicrobial therapy including a focus on psittacosis. Roston M, Marx K: Proc Mid-Atlantic States Assoc Avian Vet MASAAV, Blacksburg, VA 16 34-41 1995.
585. Harlin R: Backyard Poultry. Roston M, Marx K: Proc Mid-Atlantic States Assoc Avian Vet MASAAV Blacksburg VA 16 65-8 1995.
588. Hedberg G, Bennett R: Preliminary studies on the use of milbemycin oxime in Galliformes, Proc Assoc Avian Vet AAV, Reno 261-4 1994.
589. Kahler J: Somatostatin treatment for diabetes mellitus in a sulfur breasted toucan, Proc Assoc Avian Vet AAV, Reno 269-73 1994.
590. Rupiper D, Ehrenberg M: Introduction to pigeon practice Proc Assoc Avian Vet AAV, Reno 203-11 1994.
594. Bruning D, Hyde E: Ratites. Fowler M: Zoo and Wild Animal Medicine, WB Saunders, Philadelphia 2 277-91 1986.
595. Stoskopf M, Stoskopf S: Aquatic birds. Fowler M: Zoo and Wild Animal Medicine, WB Saunders, Philadelphia 2 293-313 1986.
596. Carpenter J: Cranes. Fowler M: Zoo and Wild Animal Medicine, WB Saunders, Philadelphia 2 315-26 1986.
599. Ingram K: Hummingbirds and miscellaneous orders. Fowler M: Zoo and Wild Animal Medicine, WB Saunders, Philadelphia 2 448-56 1986.
625. Page C: Current reptilian anesthesia procedures. Fowler M: Zoo and Wild Animal Medicine, Current Therapy, WB Saunders, Philadelphia 3 140-3 1993.
626. Redig P: Avian aspergillosis. Fowler M: Zoo and Wild Animal Medicine, Current Therapy, WB Saunders, Philadelphia 3 178-81 1993.
627. Jensen J: Diseases of bird groups. Fowler M: Zoo and Wild Animal Medicine, Current Therapy, WB Saunders, Philadelphia 3 200-3 1993.
628. Hicks K: Ostrich reproduction. Fowler M: Zoo and Wild Animal Medicine, Current Therapy, WB Saunders, Philadelphia 3 203-6 1993.
629. Carpenter J: Infectious and parasitic diseases of cranes. Fowler M: Zoo and Wild Animal Medicine, Current Therapy, WB Saunders, Philadelphia 3 229-37 1993.
632. Joyner K: Psittacine incubation and pediatrics. Fowler M: Zoo and Wild Animal Medicine, Current Therapy, WB Saunders, Philadelphia 3 247-60 1993.
674. Flammer K: An overview of antifungal therapy in birds, Proc Annu Conf Assoc Avian Vet AAV, Nashville 1-4 1993.
675. Millam J, Finney H: Leuprolide acetate can reversibly prevent egg laying in cockatiels, Proc Annu Conf Assoc Avian Vet AAV, Nashville 46 1993.
676. Turner R: Trexan use in feather picking in avian species Proc Annu Conf Assoc Avian Vet AAV, Nashville 116-8 1993.
677. Hines R, Kolattukuty P, et al: Pharmacological induction of molt and gonadal involution in birds, Proc Annu Conf Assoc Avian Vet AAV, Nashville 127-34 1993.
678. Olsen G: Common infectious and parasitic diseases of quail and pheasants, Proc Annu Conf Assoc Avian Vet AAV, Nashville 146-50 1993.
692. Gronwall R, Brown MP, et al: Pharmacokinetics of amikacin in African grey parrots, Am J Vet Res 52:2 250-2 1989.
693. Lumeij JT: Psittacine antimicrobial therapy, Antimicrobial Therapy in Caged Birds and Exotic Pets, Veterinary Learning Systems, Trenton, NJ 38-48 1995.
695. Dorrestein GM: Infectious diseases and their therapy in Passeriformes, Antimicrobial Therapy in Caged Birds and Exotic Pets, Veterinary Learning Systems, Trenton, NJ 11-27 1995.
697. Flammer K: Antimicrobial therapy. Ritchie B, Harrison G, et al: Avian Medicine: Principles and Application, Wingers Publishing, Lake Worth 434-56 1994.
700. Baumgartner R, Hoop RK, et al: Atypical nocardiosis in a red-lored Amazon parrot (*Amazona autumnalis autumnalis*), J Assoc Avian Vet 8:3 125-7 1994.
701. Millam JR, Finney HL: Leuprolide acetate can reversibly prevent egg laying in cockatiels (*Nymphicus hollandicus*), Zoo Biol 13:2 149-55 1994.
702. Goldsmith T: The use of azithromycin in psittacines, J Assoc Avian Vet 8:3 135 1994.
703. Smith K, et al: Compendium of measures to control Chlamydophila psittaci infection among human and pet birds, www.avma.org /pubhlth/psittacosis.asp 12/16 2003.
704. Bishop YM: Guidance on prescribing, Veterinary Formulary RPSGB and BVA, London 3 3-80 1996.
705. Bishop YM: Drugs used in the treatment of bacterial, fungal, viral and protozoal infections, Veterinary Formulary RPSGB and BVA, London 3 83-128 1996.
706. Bishop YM: Drugs used in the treatment and control of parasitic infections, Veterinary Formulary BPSGB and BVA, London 3 129-179 1996.
707. Orosz SE, Frazier DL: Antifungal agents: A review of their pharmacology and therapeutic indications, J Assoc Avian Vet 9:1 8-18 1995.
708. Forbes NA: Diagnosis of avian aspergillosis and treatment with itraconazole, Vet Rec 128 263 1991.
709. Lumeij JT, et al: Plasma and tissue concentrations of itraconazole in racing pigeons, J Assoc Avian Vet 9 32-5 1995.
710. Tully TN, et al: A subchondral cyst in the distal tibiotarsal bone of an ostrich, J Assoc Avian Vet 9 41-4 1995.
711. Westerhoff I: Treatment of tracheal obstruction in psittacine birds using a suction technique: …study, J Assoc Avian Vet 9 45-9 1995.
713. Tully TN, et al: What is your diagnosis? J Assoc Avian Vet 9 57-8 1995.
714. Bird JE, Miller KW, et al: Pharmacokinetics of gentamicin in birds of prey, Am J Vet Res 44:7 1245-7 1983.
715. Pedersoli WM, Ravis WR, et al: Pharmacokinetics of single-dose intravenous or intramuscular administration of gentamicin in rooster, Am J Vet Res 51:2 286-9 1990.
716. Flammer K, Clark CH, et al: Adverse affects of gentamicin in scarlet macaws and galahs, Am J Vet Res 51:3 404-7 1990.
718. Ludders JW, Rode J, et al: Effects of ketamine, xylazine and a combination of ketamine and xylazine in Pekin ducks, Am J Vet Res 50:2 245-9 1989.
727. Skeeles JK, Swafford WS, et al: Studies on the use of long-acting oxytetracycline in turkeys: Serum levels and tissue residues, Avian Dis 29:1 145-9 1985.
728. Flammer K, Aucoin DP, et al: Plasma concentrations of enrofloxacin in African grey parrots treated with medicated water, Avian Dis 34 1017-22 1990.
730. Prus SE, Clubb SL, et al: Doxycycline plasma concentrations in macaws fed a medicated corn diet, Avian Dis 36 480-3 1992.
731. Schacter J, Bankowski RA, et al: Measurement of tetracycline levels in parakeets Avian Dis 28:1 295-302 1984.
732. Panigrahy B, Harmon BC, et al: Hemorrhagic disease in canaries (*Serinius canarius*), Avian Dis 28 536-41 1984.
733. Dorrestein GM, Van Gogh H, et al: Comparative study of ampicillin and amoxicillin after intravenous, intramuscular and oral, Res Vet Sci 42 343-8 1987.
734. Brisbin Jr IL, Wagner CK: Some health problems associated with the maintenance of American kestrels in captivity, Intl Zoo Yearbook 10 29-30 1970.
736. Flammer K: Use of amikacin in birds, Proc Int Conf Zoo Avian Med AAZV and AAV, Oahu 195-7 1987.
738. Luthgen W, Bretschneider J, et al: Prophylaxis and therapy of psittacosis in lovebirds, cockatiels and other problematic birds, Dtsch Tierarztl Wochenschr 86 426-9 1979.
739. Fudge AM: Avian antimicrobial therapy Proc Annu Conf Assoc Avian Vet AAV 162-83 1983.
740. Rosskopf WJ, Woerpel RW, et al: Successful treatment of aspergillosis in two psittacine birds: Esophageal and respiratory tract, Proc Assoc Avian Vet AAV, Miami 119-28 1986.
741. Clubb SL: Therapeutics in avian medicine. Harrison GJ: Vet Clinic NA Sm Anim Prac, WB Saunders, Philadelphia 14:2 345-61 1984.
742. Bailey T: Aspergillosis: Therapy and Prevention in Zoo Animals with Emphasis on Raptors, Falco Newsletter Middle East Falcon Research Group 20 18-22 2002.
743. Biasia I, Giovanardi AA: Fungal Infections. Fowler ME, Cubas ZS: Biology, Medicine and Surgery of South American Wild Animals, Iowa State University Press, Ames, IA 163-5 2001.
745. Oglesbee B: Mycotic Diseases, Internationalen Symposium Erkrankungen Zoo und Wildtiere Institut Zoo Wildtierforschung, Berlin 37 323-31 1995.
746. Verwoerd D: Aerosol use of a novel disinfectant, … preventing and treating aspergillosis in falcons, Falco Newsletter Middle East Falcon Research Group 17 15-7 2001.
747. Jensen J, Westerman E: Amikacin pharmacokinetics in ostrich (*Struthio camelus*). Cambre RC Proc Am Assoc Zoo Vet 238-42 1990.
748. Flammer K: An update on psittacine antimicrobial pharmacokinetics, Proc Assoc Avian Vet AAV, Phoenix 218-20 1990.
749. Flammer K: New advances in avian therapeutics Proc Assoc Avian Vet, New Orleans 14-8 1992.
750. Krautwald ME, Pieper K, et al: Further experiences with the use of Baytril pet birds, Proc Assoc Avian Vet, Phoenix 226-36 1990.
751. Dorrestein GM, Van Gogh H, Rinzema JD: Pharmacokinetic aspects of penicillins, aminoglycosides and chloramphenicol in birds …mammals, Vet Q 6:4 216-24 1984.
758. Locke D, Bush M: Tylosin

aerosol therapy in quail and pigeons, J Zoo Anim Med 15 67-72 1984.
759. Bush M, Locke D, et al: Gentamicin tissue concentration in various avian species following recommended dosage therapy, Am J Vet Res 42:12 2114-6 1981.
762. Anadon A, Martinez-Larranga MR, et al: Pharmacokinetics of doxycycline in broiler chickens, Avian Pathol 23 79-90 1994.
763. McDonald SE: Summary of medications for use in psittacine birds, J Assoc Avian Vet 3 120-6 1989.
765. Anadon A, Martinez-Larranga MR, et al: Pharmacokinetic characteristics and tissue residues for marbofloxacin…in broiler chickens, Am J Vet Res 63 927-33 2002.
778. Garcia-Montijano M, Waxman S, et al: The disposition of marbofloxacin in Eurasian buzzards after intravenous administration, J Vet Pharmacol Therap 24 155-7 2001.
779. Fitzgzgerald G, Cooper JE Preliminary studies of the use of propofol in the domestic pigeon, Res Vet Sci 49 334-8 1990.
787. Ding HZ, Zeng ZL, et al: Pharmacokinetics of sarafloxacin in pigs and broilers following IV, IM and oral single doses, J Vet Pharmacol Therap 24 303-8 2001.
788. Tell L, Harrenstein L, et al: Pharmacokinetics of ceftiofur sodium in exotic and domestic avian species, J Vet Pharmacol Therap 21 85-91 1998.
789. Laczay P, Semjen G, et al: Comparative studies on the pharmacokinetics of norfloxacin in chickens, turkeys and geese, J Vet Pharmacol Therap 21 161-4 1998.
790. Bailey TA, Sheen RS, et al: Pharmacokinetics of enrofloxacin after IV, IM and oral administration in a houbara bustard, J Vet Pharmacol Therap 21 288-97 1998.
795. Cybulski W, Larrson P, et al: Disposition of metronidazole in hens and quails: pharmacokinetics and autoradiography, J Vet Pharmacol Therap 19 352-8 1996.
815. Bailey TA, Sheen RS, et al: Pharmacokinetics of enrofloxacin after IV, IM and oral administration to houbara bustards, J Vet Pharmacol Therap 20 Suppl 204-5 1997.
818. Clipsham R: New treatment for tapeworms, J Assoc Avian Vet 3 132 1989.
819. Rosskopf WJ: Clinical use of selected therapeutics, J Assoc Avian Vet 3 127-8 1989.
820. McDonald SE: Injecting eggs with antibiotics, J Assoc Avian Vet 3:1 9 1989.
821. Bond M: Intradermal injections for pox eye lesions, J Assoc Avian Vet 3 132 1989.
822. Oaks JL Treatment of a falcon for localized tetanus J Assoc Avian Vet 3 140-2 1989.
823. McCluggage DM Self Assessment Test J Assoc Avian Vet 3 168, 173 1989.
830. Anadon A, Martinez-Larranga MR, et al: Pharmacokinetics of norfloxacin and metabolites in broiler chickens, Am J Vet Res 53:11 2084-9 1992.
835. Vaden SL, Cullen JM, Riviere JE: Pharmacokinetics of cyclosporine in woodchucks and Pekin ducks, J Vet Pharmacol Therap 18 30-3 1995.
841. Freed M, Clarke JP, et al: Effect of spectinomycin on *Escherichia coli* infection in 1-day-old ducklings, Avian Dis 37 763-6 1993.
842. Kempf I, Gesbert F et al: Dose titration study of enrofloxacin against respiratory colibacillosis in Muscovy ducks, Avian Dis 39 480-8 1995.
843. Bradley FA, Bickford AA, Walker RL: Efficacy of miconazole nitrate against favus in oriental breed chickens, Avian Dis 39 900-1 1995.
847. Bush M Pharmacokinetics of selected antibiotics in avian species (a review), Proc Symp Avian Dis Fifth 53-9 1983.
848. Vernimb GD, Bachman H, Panitz E: Effect of gentamicin on early mortality and later performance of chickens, Avian Dis 20:4 706-13 1976.
849. Turbahn A, de Jackel SC, et al: Dose response study of enrofloxacin against Riemerella anatipestifer in ducklings, Avian Pathol 26 791-802 1997.
851. Hamdy AH, Kratzer DD, et al: Effect of a single injection of lincomycin and spectinomycin on early chick mortality, Avian Dis 23:1 164-73 1979.
853. Intorre L, Mengozzi G, et al: The plasma kinetics and tissue distribution of enrofloxacin and ciprofloxacin in the Muscovy duck, Vet Res Comm 21 127-36 1997.
854. Froyman R, Sheer M, De Jong A: Serum kinetics of enrofloxacin in Pekin ducks during drinking water medication and Baytril 10%. Lees, P: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Blackwell Scientific Publ, Edinburgh 6th 236-7 1994.
855. Charleston B, Gate JJ, et al: Comparison of the efficacies of three fluoroquinolone antimicrobial agents…chickens, Antimicrobial Agents Chemother 42 83-7 1998.
856. Jordan FTW, Gilbert S, et al: Effects of Baytril, tylosin and tiamulin on avian mycoplasmas, Avian Pathol 18 659-73 1989.
861. Rosskopf WJ, Woerpel RW: Practical avian therapeutics and dosages of commonly used medications. Rosskopf WJ, Woerpel RW: Vet Clinic NA Sm Anim Prac WB Saunders, Philadelphia 21:6 1265-71 1991.
862. Barbour EK, Hamadeh S, et al: Evaluation of an enrofloxacin-treatment program against *Mycoplasma gallisepticum* in broilers, Preventive Veterinary Medicine 35 91-9 1998.
863. Sumano LH, Ocampo CL, et al: Effectiveness of two fluoroquinolones for the treatment of chronic respiratory disease outbreak, British Poultry Sciences 39 42-6 1998.
864. McCabe MW, Rippel RH, Miller RE: Efficacy of sarafloxacin for the control of mortality associated with *E. coli* infections, Proc Western Poultry Disease Conf, Sacramento 42nd 75 1993.
865. Glisson JR The efficacy of enrofloxacin for the treatment of colibacillosis…and fowl cholera in turkeys, Proc Western Poultry Disease Conf, Cancun 45th 222-4 1996.
866. Dorrestein GM and Verburg E Pharmacokinetics of enrofloxacin in homingpigeons, Proc Intl Cong Eur Assoc Vet Pharm Toxicol Univ of Veterinary Science, Budapest 4th 27-30 1988.
869. Migaki TT, Chappel LR, Babcock WE: Anticoccidial efficacy of…salinomycin, in comparison to monensin and lasalocid in battery trials, Proc Western Poultry Disease Conf, Davis, CA 27th 40-2 1978.
870. Galvan G, Huerta A, del Oro M: Efficacy of tiamulin in the treatment of mycoplasmosis in chickens in Mexico, Proc Western Poultry Disease Conf, Davis, CA 27th 80-2 1978.
872. Hamdy AH, Saif YM: Efficacy of various concentrations of lincomycin spectinomycin against mycoplasma in turkey poults, Proc Western Poultry Disease Conf, Davis, CA 26th 115-8 1977.
873. Jordan FTW, Forrrester CA, et al: In vitro and in vivo…valnemulin, tiamulin, tylosin, enrofloxacin, lincomycin/spectinomycin, Avian Dis 42 738-45 1998.
874. Droual R, Bickford AA, et al: Favus in a backyard flock of game chickens Avian Dis 35 625-30 1991.
876. Dyer DC, van Alstine WG: Antibiotic aerosolization: tissue and plasma oxytetracycline concentration in parakeets, Avian Dis 31 677-9 1987.
877. Jordan FTW, Knight D: The minimum inhibitory concentration of kitasamycin, tylosin and tiamulin for Mycoplasma, Avian Pathol 13 151-62 1984.
878. Law JM, Tully TN, Stewart TB: Verminous encephalitis apparently caused by the filaroid nematode *Chandlerella quicali* in emus, Avian and Exotic Animal Medicine Symposium 37 597-601 1993.
879. Ramsey EC, Vulliet R: Pharmacokinetic properties of gentamicin and amikacin in the cockatiel, Avian Dis 37 628-34 1993.
880. Pedersoli WM, Ravis WR, et al: Pharmacokinetics of single doses of gentamicin given intravenously and intramuscularly to turkeys, J Vet Pharmacol Therap 12 124-32 1989.
881. Cracknell VC, Andreotis J, et al: An evaluation of apramycin soluble powder for the treatment of naturally acquired *E. coli* infections, J Vet Pharmacol Therap 9 273-9 1986.
882. Hamlin RL, Stalnaker PS: Basis for use of digoxin in small birds, J Vet Pharmacol Therap 10 354-6 1987.
883. Maldonado A: Reporte Preeliminar: Digitalizacion en pollos de engorda com metodo preventivo el sindrome ascitico, Proc Western Poultry Disease Conf 35th 1986.
888. Baert L, van Poucke S, et al: Pharmacokinetics and anthelmintic efficacy of febantel in the racing pigeon, J Vet Pharmacol Therap 16 223-31 1993.
890. Schriemer TR, Paulissen JB, Dame KJ: Evaluation of ceftiofur sodium for control of terminal bacterial infections in broiler chickens, Proc World's Poultry Cong, Amsterdam 19th:1 427 1992.
891. Vanparijs O, Hermans L, van de Flaes L: Anticoccidial efficacy of diclazuril in game birds Proc World's Poultry Cong, Amsterdam 19th:1 394-7 1992.
893. Tanner AC, Davidson JN: Efficacy of danofloxacin against induced colisepticemia in broiler chickens, Proc World's Poultry Cong, Amsterdam 19th:3 84-5 1992.
894. Schneider KH, Scheller W: Effect of exogenous testosterone and gonadotropic hormones on ejaculate production by ganders, Proc World's Poultry Cong, Amsterdam 19th:3 307 1992.
895. Ruff MD, Chute MB, Wilkins GC: Control of coccidiosis in ring-necked pheasants, Proc World's Poultry Cong, Amsterdam 19th:3 337-41 1992.
899. Flinchum G: Infraorbital Sinus Flushes in Birds, Exotic DVM 1.1 18-22 1999.
905. Lierz M: Phallus Amputation in Pet Ducks, Exotic DVM 1.3 51-4 1999.
912. Harrison GJ: Rescue remedy for birds, Exotic DVM 1.4 40 1999.
914. Schoemaker NJ: Selected Dermatologic Conditions in Exotic Pets, Exotic DVM 1.5 5-11 1999.
916. Dorrestein GM: Enrofloxacin in pet avian and exotic animal therapy, Proc Int Baytril Symp Bonn 1st 63-70 1992.
918. Redmann T, Glunder G, et al: Attempted therapy with enrofloxacin of a flock of laying hens with pullorum-salmonellosis, Dtsch Tierarztl Wochenschr 96 137-8 1989.
919. Dorrestein GM, Verburg E: Pharmacokinetics of Baytril in homing pigeons after different administration routes, Proc Symp Avian Dis, Munich 6th 132-43 1988.
921. Linderstruth H: Feldversuch zur wirksamkeits- und vertraglichkeitsprufung von Baytril bei importierten Psittaciden..Vet Med Diss, Giessen 1992.
922. Uyttenbroek E, Gevaert D, Devriese LA: Effect of different chemotherapeutics on experimental salmonellosis in pigeons, Vlaams Diergeneesk Tijdschr 58 51-4 1989.
923. Schmahl C: Therapieversuche bei der spontanen infektion von Brief- und Rasstauben mit Salmonella typhimurium, Proc Tagung Vogelkrankheiten. Munich 7th 148-53 1990.
924. Krautwald-Junghans ME: Avian therapeutics. Proc Eur Chap Assoc Avian Vet. Vienna 1st 30-9 1991.
925. Bougerol C: Antibiotherapie chez les oiseaux, Rec Med Vet 166 361-6 1990.
932. Ostrowski S, Greth A, Mikaelian

I: Pathology and Veterinary Care. Saint Jalme M, van Heezik Y: Propagation of the Houbara bustard Kegan Paul International, London 70-96 1995.
936. Scheer M: Concentrations of active ingredient in the serum and in tissues after oral and parenteral…Baytril, Vet Med Rev 2 104-18 1987.
939. Zantop D: Medetomidine in birds, Exotic DVM 1.5 34 1999.
943. Harcourt-Brown NH: Treatment of bumblefoot in an eagle, Exotic DVM 1.6 5-11 2000.
951. Lindenstruth H: Feldversuch zur wirksamkeits- und vertraglichkeitsprufung von Baytril bei importierten psittaciden. Vet Med Diss, Giessen 1992.
956. Bauditz R: Enrofloxacin - clinical evaluation in several animal species. Simon F, et al: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Univ of Veterinary Scien, Budapest 4th 21-6 1988.
957. Vlaminck K, Vanparijs O, et al: Clazuril, anticoccidial agent for pigeons. Simon F, et al: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Univ. of Veterinary Scien, Budapest 4th 177-9 1988.
958. Varga I, Laczay P, Voros G: Anticoccidial efficacy of maduramicin in batter, floor pen and field trials. Simon F, et al: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Univ of Veterinary Scien, Budapest 4th 181-6 1988.
962. Meyer KF, Eddie B: Chemotherapy of natural psittacosis and ornithosis field trials of tetracyclines, Antibiot Chemother 5 289-9 1955.
966. Dorrestein GM, Van Gogh H, et al: Clinical pharmacology and pharmacokinetics of flumequine IV, IM and oral administration in pigeons, J Vet Pharmacol Therap 6 281-92 1983.
968. Dorrestein, GM, Rinzema JD, et al: Clinical pharmacology and pharmacokinetics of trimethoprim and selected sulfonamides in the pigeon, Dissertation Proefschrift Utrecht 139-69 1987.
978. Herrenstien LA: Disposition of enrofloxacin in red-tailed hawks and great horned owls. Soback S, McKellar QA: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Blackwell Science Ltd, Jerusalem 8th B49 2000.
979. Waxman S, Garcia-Montijano M, et al: Pharmacokinetic behavior of marbofloxacin after oral administration in *Buteo buteo*. Soback S, McKellar QA: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Blackwell Science Ltd, Jerusalem 8th B51 2000.
983. Webb AI, French RA, Gaskin JM: The efficacy and safety of fenbendazole in gamebirds. Soback S, McKellar QA: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Blackwell Science Ltd. Jerusalem 8th H27 2000.
987. Bauditz R: Results of clinical studies with Baytril in poultry, Vet Med Rev 2 130-6 1987.
990. TerHune TN, Wages DP, et al: Clinical evaluation of efficacy of danofloxacin treatment of *E. coli* airsacculitis, Proc Western Poultry Disease Conference, Acapulco 40th 270 1991.
991. Krieg J, Kosters J: Pharmacokinetics of amoxycillin in healthy and specifically compromised adult pigeons, Proc Conf Avian Dis, Munich 5th 90-6 1986.
992. Bruch JV, Aufinger P, Jakoby JR: Studies of pharmacokinetics and efficacy of sulphadimethoxine to…adult pigeons, Proc Conf Avian Dis, Munich 5th 97-106 1986.
994. Zantop D: Using leuprolide acetate to manage common avian reproductive problems, Exotic DVM 2.3 70 2000.
1006. Aerts R, Verheijen F, et al: Efficacy of dosage schemes of Paracillin SP in *E. coli* challenged broilers. Lees P: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Blackwell Scientific Publ, Edinburgh 6th 94-5 1994.
1009. Semjen G, Magyar T, Laczay P: Efficacy of doxycycline in the treatment of experimentally induced fowl cholera in broiler chickens. Lees P: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Blackwell Scientific Publ, Edinburgh 6th 200-1 1994.
1015. Pashov D, Deyanov M, et al: Efficacy of some formulations containing tylosin against avian borreliosis. Lees P: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Blackwell Scientific Publ, Edinburgh 6th 223-4 1994.
1016. Rolinski Z, Furmaga R, et al: Laboratory, pharmacological and clinical studies on a combination of colistin and tylosin. Lees P: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Blackwell Scientific Publ, Edinburgh 6th 224-5 1994.
1020. Gulkarov A, Ziv G: Some pharmacokinetic features of norfloxacin nicotinate in turkeys. Lees P: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Blackwell Scientific Publ, Edinburgh 6th 235 1994.
1021. Delaporte J, Froyman R, et al: Distribution of enrofloxacin within hatching eggs during …treatment with Baytril 10% in turkeys. Lees P: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Blackwell Scientific Publ, Edinburgh 6th 235-6 1994.
1023. Bousquet E, Barrowman P, et al: pharmacokinetics of a trimethoprim/sulfadiazine combination administered drinking water to broilers. Lees P: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Blackwell Scientific Publ, Edinburgh 6th 241 1994.
1024. de Backer P, Vermeersch H, et al: Pharmacokinetics and anthelmintic efficacy of febantel in racing pigeons. Lees P: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Blackwell Scientific Publ, Edinburgh 6th 248-9 1994.
1025. Pashov D, Koinarski V: Comparative studies of anticoccidial efficacy sulfadimidine, trimethoprim and toltrazuril in turkey. Lees P: Proc Intl Cong Eur Assoc Vet Pharm Toxicol Blackwell Scientific Publ, Edinburgh 6th 264 1994.
1027. Remple JD, Riddle SK: Pharmacological Considerations. Weaver JD, Cade TJ: Falcon Propagation The Peregrine Fund, Boise, Idaho, 2nd Ed 77-85 1991.
1028. Sandfort C, Dierenfeld E, Lee J: Nutrition. Weaver JD, Cade TJ: Falcon Propagation The Peregrine Fund, Boise, Idaho, 2nd Ed 81-2 1991.
1029. Heck WR, Konkel D: Incubation and rearing Weaver JD, Cade TJ: Falcon Propagation The Peregrine Fund, Boise, Idaho, 2nd Ed 34-76 1991.
1033. Zantop DW: Treatment of Bile Duct Carcinoma in Birds with Carboplatin, Exotic DVM 2.3 76-8 2000.
1034. Lierz M: Use of inhalation chamber for aspergillosis therapy, Exotic DVM 2.3 79-80 2000.
1035. Hochleithner D, Hochleithner M: Innovative approach to liver and kidney diseases in psittacine birds, Exotic DVM 2.3 84-7 2000.
1051. McKellar QA, Scott EW: The benzimidazole anthelmintic agents, J Vet Pharmacol Therap 13 223-47 1990.
1056. Pedersoli WM, Askins D, et al: Pharmacokinetics of gentamicin in chickens after single and repeated administration, Pharmacologist 28 204 1986.
1058. El-Gammal AA, Ravis WR, et al: Pharmacokinetics and intramuscular bioavailability of amikacin in chickens following dosing, J Vet Pharmacol Therap 15 133-42 1992.
1060. Norton TM, Kollias GV, et al: Acyclovir (Zovirax) pharmacokinetics in Quaker parakeets, J Vet Pharmacol Therap 15 252-8 1992.
1061. Zwijnenberg RJG, Vulto AG, et al: Evaluation of antibiotics for racing pigeons available in the Netherlands, J Vet Pharmacol Therap 15 364-78 1992.
1062. Gylstorff I, Jakoby JR, Gerbermann H: Investigations of the efficacy different medicated feed to parrots infected with *Chlamydia psittaci*, Berl Munch Tieraerztl Wschr 97 91-9 1984.
1065. Oostindjer AJ, Frankenhuis MT, et al: Compendium Behandelingsadviezen Vogelziekten Gezondheidsdienst voor Plumvee Doorn 5th 1987.
1066. Dorrestein GM, van der Hage MH, Zwart P: Examination, diagnosis and therapy of diseases of aviary birds, Proc Eur Symp Bird Dis, Beerse 123-54 1987.
1067. McDonald SE Practical avian therapeutics Proc Assoc Avian Vet Houston 7-19 1988.
1068. Samour J: Management of raptors. Harrison G, Lightfoot T (eds): Clinical Avian Medicine, 914-956 2005.
1070. Arnstein P, Cohen DH, et al: Medication of pigeons with chlortetracycline in feed, J Am Vet Med Assoc 9 921-4 1964.
1072. Reid BL, Hulett BJ: Aspirin stimulates egg production, Feedstuffs 36 32 1964.
1077. Arnstein P, Buchanan WG, et al: Chlortetracycline chemotherapy for nectar-feeding psittacine birds, J Am Vet Med Assoc 154 190-1 1969.
1079. Arnstein P, Eddie B, et al: Control of psittacosis by group chemotherapy of infected parrots, Am J Vet Res 29 2213-27 1968.
1087. Bush M: External fixation of avian fractures, J Am Vet Med Assoc 171 943-6 1977.
1088. Kennedy S, Crisler JP, et al: Lead poisoning in sand hill cranes, J Am Vet Med Assoc 171 955-8 1977.
1089. Courtney CH, Forrester DJ, White FH: Anthelmintic treatment of brown pelicans, J Am Vet Med Assoc 171 991-2 1977.
1090. Stoskopf MD, Beier J: Avian malaria in African black-footed penguins, J Am Vet Med Assoc 175 944-7 1979.
1091. Ensley PK, Davis CE, et al: Cryptococcosis in a male Beccari's crowned pigeon, J Am Vet Med Assoc 175 992-4 1979.
1093. Shlosberg A: Treatment of monocrotophos-poisoned birds of prey with pralidoxime iodide, J Am Vet Med Assoc 169 989-90 1976.
1096. Ward FP: Thiamine deficiency in a peregrine falcon, J Am Vet Med Assoc 159 599-601 1971.
1110 Harcourt-Brown NH: Tendon Repair in the Avian Pelvic Limb, Exotic DVM 2.5 28-33 2000.
1112. Flinchum G, Harrison G: Yunnan Paiyan for surgical support, Exotic DVM 2.6 10 2001.
1114. Samour JH, Naldo JL: Medical management of avian pox lesions on the cere of captive falcon, Exotic DVM 2.6 14-5 2001.
1119. Rodriguez-Quiros J: Bone grafting in birds, Exotic DVM 3.1 17-23 2001.
1120. Richardson JA, Murphy LA, et al: Managing pet bird toxicoses, Exotic DVM 3.1 23-7 2001.
1124. Samour J, Naldo J: Trichomoniasis infections in captive falcons, Exotic DVM 3.2 10-1 2001.
1127. Bailey TA, Samour JH, et al: Analysis of amoxycillin in bustard plasma by highperformance liquid chromatography, J Vet Pharmacol Therap 19 313-5 1996.
1128. Bailey T: On long-acting amoxicillin in birds Personal notes or communications, BirdMed 3-18 2003.
1131. Klueh PN: Investigations on therapy and prophylaxis of Caryospora-infections in falcons with toltrazuril… Dissertation Tierarzlichen Hochschule, Hanover 1994.
1132. Arent L, Martell M: Care and Management of Captive Raptors, Raptor Center Univ, Minnesota, St Paul 1996.
1133. Forbes NA: Falcon herpesvirus in the UK, Falco, Newsletter Middle East Falcon Research Group 17 2001.
1134. Forbes NA: Control of endemic *Caryospora* species infestation of captive raptors, Falco, Newsletter Middle East Falcon Research Group 16 2000.
1135. McKinney P: Amyloidosis in falcons in the United Arab

Emirates Falco, Newsletter Middle East Falcon Research Group 16 2000.
1139. Dorrestein GM, Buitelaar MN, Wiggelinkhuizen JM: Pharmakokinetishce aspectin von antibiotika bei volierevogeln und tauben, Proc Symp Avian Dis, Munich 2nd 1981.
1140. Lawrence K: Therapeutics. Price CJ: Manual of Parrots, Budgerigars & Other Psittacine Birds, BSAVA West Sussex 171-87 1988.
1142. Lawrence K: Facts and data to help the treatment of smaller domestic pets. Evans J: Henston Veterinary Vade Mecum Henston, London 1986.
1143. Clark CH: Pharmacology of antibiotics. Harrison GJ, Harrison LR: Clinical Avian Medicine and Surgery, WB Saunders, Philadelphia 319-26 1986.
1150. Flinchum GB: Management of waterfowl. Harrison G, Lightfoot T (eds): Clinical Avian Medicine, 830-848 2005.
1151. Harrison GJ, Flinchum GB: Emergency and critical care. Harrison G, Lightfoot T (eds): Clinical Avian Medicine, 213-232 2005.
1152. Pees M, Straub J, Krautwald-Junghanns ME: Evaluating and treating the cardiovascular system. Harrison G, Lightfoot T (eds): Clinical Avian Medicine, 379-410 2005.
1154. Pollock CG: Implications of mycobacteria in clinical disorders. Harrison G, Lightfoot T (eds): Clinical Avian Medicine, 681-690 2005.
1156. Forbes NA: Epidemic and endemic chlamydiosis in a collection of raptors. Samour JH: Proc Specialist Group Middle East Falcon Research Group MEFRG, Abu Dhabi 3-8 1995.
1157. Verwoerd D: Observations on the use of azithromycin in falcons Falco, Newsletter Middle East Falcon Research Group 16 Letters 2000.
1158. Samour J: Serratospiculiasis in birds of prey, Falco, Newsletter Middle East Falcon Research Group 16 Letters 2000.
1159. Lierz M, Remple D: Endoparasitenbefall bei grossfalken in Dubai Kleintier, Praxis 42 757-68 1997.
1160. Baronetzky-Mercier A, Seidel B, Goltenberg R & HG: Klos Krankheiten der Zoo und Wildtiere Blackwell Berlin 443-65 1995.
1167. Bradley T: Recognizing pain in exotic animals Exotic DVM 3.3 21-6 2001.
1169. Papich MG: Drug therapy and selection in exotic medicine Compend Contin Educ Prac Vet 25, Suppl 15-20 2003.
1170. Flammer K: Common bacterial infections and antibiotic use in companion birds, Compend Contin Educ Prac Vet 20, Supp 3A 34-48 1998.
1175. Riddle KE: Surgical treatment of bumblefoot in raptors. Cooper JE, Greenwood AG: Proc Intl Symp of Diseases of Birds of Prey, Chiron Publications, West Yorkshire 1980 67-73 1981.
1177. Remple JD: Avian malaria with comments on *Haemosporidia* in large falcons. Cooper JE, Greenwood AG: Proc Intl Symp of Diseases of Birds of Prey, Chiron Publications, West Yorkshire 1980 107-10 1981.
1178. Redig PT: Aspergillosis in raptors. Cooper JE, Greenwood AG, Proc Intl Symp of Diseases of Birds of Prey, Chiron Publications, West Yorkshire 1980 117-22 1981.
1179. Satterfield WC, O'Rourke KI: Immunological considerations in the management of bumblefoot. Cooper JE, Greenwood AG: Proc Intl Symp of Diseases of Birds of Prey, Chiron Publications, West Yorkshire 1980 123-9 1981.
1180. Dorrestein GM: Quick reference for drug dosing. Tully TN, Lawton MPC, et al: Avian Medicine, Butterworth Heinemann, Oxford 386-90 2000.
1183. Dorrestein GM: Nursing the sick bird. Tully TN, Lawton MPC, et al: Avian Medicine, Butterworth Heinemann, Oxford 74-111 2000.
1184. Gaggemeier B, Henke J, et al: Lihtersachunjen en schmerzlinderung unit buprenorphine bei hanstauben, Proc Conf Avian Dis, Munich 12th 21 2000.
1185. Rupley AE: Manual of Avian Practice, WB Saunders, Philadelphia 1997.
1186. Harcourt-Brown NH: Psittacine birds. Tully TN, Lawton MPC, et al: Avian Medicine, Butterworth Heinemann, Oxford 112-43 2000.
1187. Dorrestein GM: Passerines and exotic softbills. Tully TN, Lawton MPC, et al: Avian Medicine, Butterworth Heinemann, Oxford 144-79 2000.
1188. Redig PT, Ackermann J: Raptors. Tully TN, Lawton MPC, et al: Avian Medicine, Butterworth Heinemann Oxford, 180-214 2000.
1189. Olsen G: Cranes. Tully TN, Lawton MPC, et al: Avian Medicine, Butterworth Heinemann Oxford, 215-27 2000.
1190. Routh A , Sanderson S: Waterfowl. Tully TN, Lawton MPC, et al: Avian Medicine, Butterworth Heinemann Oxford, 234-65 2000.
1191. Worell A: Rhamphistids. Tully TN, Lawton MPC, et al: Avian Medicine, Butterworth Heinemann Oxford, 296-311 2000.
1192. de Herdt P, Devriese L: Pigeons. Tully TN, Lawton MPC, et al: Avian Medicine, Butterworth Heinemann Oxford, 313-38 2000.
1197. Bailey T: Aerosol therapy in birds using a novel disinfectant, Exotic DVM 3.4 17 2001.
1198. Stanford M: Use of F10 in psittacines, Exotic DVM 3.4 18 2001.
1199. Kelleher S: Nebulization recipe Exotic DVM 3.4 6 2001.
1204. Flinchum G: Corticosteroids may have some use in birds Exotic DVM 3.5 43 2001.
1205. McCluggage D: Overview of nutriceutical and herbal therapies for birds, Exotic DVM 3.6 8-11 2002.
1208. Devriese LA: Ziekten van Siervogels en Duiven Rijksuniversiteit Gent 3rd 1986.
1212. Lafeber TJ: Other diseases of budgerigars. Kirk RW: Current Veterinary Therapy Small Animal, WB Saunders, Philadelphia III 376-9 1968.
1217. Vickers JH: Respiratory diseases in primates. Kirk RW: Current Veterinary Therapy Small Animal, WB Saunders, Philadelphia III 398-400 1968.
1221. Gelis S: Evaluating and treating the gastrointestinal system. Harrison G, Lightfoot T (eds): Clinical Avian Medicine, 411-440 2005.
1223. Jakob-Hoff R, Bucham B, Boyland M: Kiwi coccidia - North Island survey Kokako 6 3-5 1999.
1224. Doneley B: Management of captive ratites. Harrison G, Lightfoot T (eds): Clinical Avian Medicine, 957-990 2005.
1226. Black DG: Common medical and surgical conditions of ostriches, Proceedings 2789 Basics of Avian Medicine, Univ of Sydney, Sydney 243-58 1996.
1233. Kellner SJ: Eye and eyelid injuries. Samour J: Avian Medicine, Mosby, London 120-3 2000.
1234. Harcourt-Brown NH: Bumblefoot. Samour J: Avian Medicine, Mosby, London 126-31 2000.
1236. Samour J: Toxicology. Samour J: Avian Medicine, Mosby, London 180-93 2000.
1238. Zwart P Bacterial diseases Samour, J Avian Medicine Mosby London 254-64 2000.
1239. Redig P: Fungal diseases - Aspergillosis. Samour J: Avian Medicine, Mosby, London 275-87 2000.
1240. Samour J: Pharmaceutics commonly used in avian medicine. Samour J: Avian Medicine, Mosby, London 388-418 2000.
1254. Perelman B, Kuttin ES: Mycotic infections in ostriches. Deeming DC: Improving Our Understanding of Ratites, Farming Environment, Ratite Conf, Oxon, UK 47-9 1996.
1257. Irons PC, Bertschinger HJ, et al: Semen collection and evaluation in the ostrich. Deeming DC: Improving Our Understanding of Ratites, Farming Environment, Ratite Conf, Oxon, UK 157-9 1996.
1259. Macwhirter P, Pyke D, Wayne J: Use of carboplatin in the treatment of renal adenocarcinoma in a budgerigar, Exotic DVM 4.2 10-2 2002.
1263. Doneley B: Acute pancreatitis in parrots, Exotic DVM 4.3 13-6 2002.
1268. Stanford M: ICE presentation and discussion, International Conference on Exotics, Zoological Education Network, Palm Beach, FL 2003.
1269. Johnson-Delaney C: ICE presentation and discussion, International Conference on Exotics, Zoological Education Network, Palm Beach, FL 2003.
1273. Flinchum G: ICE presentation and discussion, International Conference on Exotics, Zoological Education Network, Palm Beach, FL 2003.
1274. Bowles H: ICE presentation and discussion, International Conference on Exotics, Zoological Education Network, Palm Beach, FL Supp 16 2003.
1278. Zantop D: ICE presentation and discussion, International Conference on Exotics, Zoological Education Network, Palm Beach, FL 2003.
1279. Lennox A: ICE presentation and discussion, International Conference on Exotics, Zoological Education Network, Palm Beach, FL 2003.
1280. Cubas ZS, Godoy SN: Hemochromatosis in toucans, Exotic DVM 4.3 27-8 2002.
1281. Lennox AM: The use of Aldara (imiquimod) for the treatment of cloacal papillomatosis in psittacines, Exotic DVM 4.3 34-5 2002.
1296. Finkelstein AD, DiGesualdo CL: Wound management in a pheasant, Exotic DVM 4.5 10 2002.
1297. Stanford M: Use of Doxirobe Gel®, Exotic DVM 4.5 11 2002.
1302. Stanford M: Further uses of F10 in exotic practice, Exotic DVM 4.5 19 2002.
1306. Oglesbee B, Bishop C: Avian infectious disease. Birchard S, Sherding R: Manual of Small Animal Practice, WB Saunders, Philadelphia 1257-70 1994.
1307. Plumb DC: Veterinary Drug Handbook, Iowa State Press Ames, IA 4th 2002.
1308. Jenson J: Current ratite therapy, Vet Clinic NA Food Anim Prac, WB Saunders, Philadelphia 16 1998.
1309. Clubb SL: Therapeutics: Individual and flock treatment regimens. Harrison GJ, Harrison LR: Clinical Avian Medicine and Surgery, WB Saunders, Philadelphia 327-56 1986.
1311. Rupiper DJ, Carpenter JW, Mashimo TY: Formulary. Olsen GH, Orosz SE: Manual of Avian Medicine, Mosby, St. Louis, MO 553-89 2000.
1313. Tully TN: Formulary. Altman RB, Clubb SL, et al: Avian Medicine and Surgery, WB Saunders, Philadelphia 671-88 1997.
1314. Joseph V: Preventive health programs for falconry birds, Proc Annu Conf Assoc Avian Vet 171-8 1995.
1316. Kasper A: Rehabilitation of California towhees Proc Annu Conf Assoc Avian Vet 83-90 1997.
1317. Moore DM, Rice RL: Exotic animal formulary. Holt KK, et al: Veterinary Values, Veterinary Medicine Publishing, Lenexa, KS 5 159-245 1998.
1318. Clubb SL, Johnston DE: Bristol Veterinary Handbook of Antimicrobial Therapy, Veterinary Learning Systems, Trenton, NJ 2 188-99 1987.
1319. Hoeffer H: Antimicrobials in pet birds Bonagura, J Current Veterinary Therapy Small Animal, WB Saunders, Philadelphia XII 1278-83 1995.

1320. Clyde V, Paul-Murphy J: Avian analgesia. Bonagura J: Current Veterinary Therapy Small Animal, WB Saunders, Philadelphia XIII 1126-8 2000.
1323. McConnell VC, Hughey T: Formulary. Univ of Georgia Vet Hospital, Athens, GA 1987.
1324. Hillyer E: Avian dermatology. Birchard S, Sherding,R: Manual of Small Animal Practice ,WB Saunders, Philadelphia 1271-81 1994.
1325. Stunkard JM: Diagnosis, Treatment and Husbandry of Pet Birds, Stunkard Publishing Edgewater, MD 1984.
1326. Lothrop CD, Harrison GJ, et al: Miscellaneous diseases Harrison GJ, Harrison LR: Clinical Avian Medicine and Surgery, WB Saunders, Philadelphia 535-6 1986.
1329. Tully T: Psittacine therapeutics, Vet Clinic NA Exotic Anim Prac, WB Saunders, Philadelphia 3 59-90 2000.
1330. Velasco MC: Candidiasis and cryptococcosis in Birds, Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 9 75-81 2000.
1332. Flammer K: Oropharyngeal diseases in caged birds. Kirk RW: Current Veterinary Therapy Small Animal, WB Saunders, Philadelphia IX 699-702 1986.
1334. Sandmeier P, Coutteel P: Management of canaries, finches and mynahs. Harrison G, Lightfoot T (eds): Clinical Avian Medicine, 878-914 2005.
1335. Roberson EL: Antinematodal agents. Booth NH, McDonald LE: Veterinary Pharmacology and Therapeutics, Iowa State University Press, Ames, IA 6 882-927 1988.
1336. Marshall R: Avian anthelmintics and antiprotozoals, Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 2 33-41 1993.
1337. Calvert C, Mieurs K: CVT update. Bonagura J: Current Veterinary Therapy Small Animal, WB Saunders, Philadelphia XIII 756-60 2000.
1338. Dalhausen B, Lindstrom J, et al: The use of terbinafine in the treatment of avian fungal disease, Proc Assoc Avian Vet, Portland 35-9 2000.
1341. Paul-Murphy J, Ludders J: Avian analgesia. Heard DJ: Vet Clinic NA Exotic Anim Prac, WB Saunders Philadelphia 4 35-45 2001.
1347. Machin KL, Livingston A: Assessment of the analgesic effects of ketoprofen in duck anesthetized with isoflurane, Am J Vet Res 63 821-6 2002.
1348. Phalen DN Implications of macrorhabdosis in clinical disorders. Harrison G, Lightfoot T (eds): Clinical Avian Medicine, 705-710 2005.
1349. Filippich JL, Herdrikz JK: Prevalence of megabacteria in budgerigar colonies, Aust Vet J 76 92-5 1998.
1350. Phalen DN, Tomaszewski D, Davis A: Investigation into the detection, treatment and pathogenicity of avian gastric yeast, Proc Assoc Avian Vet 49-51 2002.
1352. Phalen DN: Implications of viruses in clinical disorders. Harrison G, Lightfoot T (eds): Clinical Avian Medicine, 721-746 2005.
1353. Cranfield MR: Sphenisciformes (Penguins). Fowler ME, Miller ER: Zoo and Wild Animal Medicine, Saunders, St. Louis, MO 5 103-10 2003.
1354. Stoskopf MD, Beall FB: The husbandry and medicine of captive penguins, Proc Am Assoc Zoo Vet Washington DC 81-91 1982.
1355. Cranfield MR, Shaw M, et al: A review and update of avian malaria in the African penguin, Proc Am Assoc Zoo Vet, S Padre Is, TX 1990.
1357. Carpenter JW, Mashima TY, Rupier DJ: Raptor Biomedicine, Greystone Publications, Manhattan, KS 1996.
1358. Kearns KS: Anseriformes (Waterfowl, Screamers) Zoo and Wild Animal Medicine 141-9 2003.
1359. Redig PT: Falconiformes (Vultures, Hawks, Falcons, Secretary Bird), Zoo and Wild Animal Medicine 150-61 2003.
1361. Carpenter JW: Gruiformes (Cranes, Limpkins, Rails, Gallinules, Coots, Bustards), Zoo and Wild Animal Medicine 171-80 2003.
1363. Olsen GH, Carpenter JW et al: Cranes. Altman RB, Clubb SL, et al: Avian Medicine and Surgery, WB Saunders Philadelphia 973-91 1997.
1364. Schultz DJ: Columbiformes (Pigeons, Doves), Zoo and Wild Animal Medicine 180-7 2003.
1365. Lamberski N: Psittaciformes (Parrots, Macaws, Lories), Zoo and Wild Animal Medicine 187-210 2003.
1366. Flammer K, Whitt-Smith D: Plasma concentrations of enrofloxacin in psittacine birds offered water medicated with 200 mg/l, J Avian Med Surg 16(4) 287-90 2002.
1367. Crosta L: Alloplastic and heteroplastic bill prostheses in 2 Ramphistidae birds, J Avian Med Surg 16(3) 218-22 2002.
1368. Ryan TP: Grit impaction in 2 neonatal African grey parrots, J Avian Med Surg 16(3) 230-3 2002.
1369. Lamberski N, Theon AP: Concurrent irradiation and intratumoral chemotherapy with cisplatin for treatment of a fibrosarcoma, J Avian Med Surg 16(3) 234-8 2002.
1370. Heinz K, Tully TN: What is your diagnosis? J Avian Med Surg 16(3) 245-6 2002.
1372. Samour JH, Naldo JL: Diagnosis and therapeutic management of candidiasis in falcons in Saudi Arabia, J Avian Med Surg 16(2) 129-32 2002.
1373. Bauck L: Mycoses. Ritchie B, Harrison G, et al: Avian Medicine: Principles and Application: Wingers Publishing, Lake Worth, FL 997-1006 1994.
1374. Oglesbee BL: Mycotic diseases. Altman RB, Clubb SL, et al: Avian Medicine and Surgery, WB Saunders, Philadelphia 323-31 1997.
1375. Boydell IP, Forbes NA: Diseases of the head (including the eyes). Beynon PH, Forbes NA: Manual of Raptors, Pigeons and Waterfowl, BSAVA, Cheltenham 140-6 1996.
1376. Suedmeyer WK, Bermudez AJ, Fales WH: Treatment of epidermal cysts associated with Aspergillus and Alternaria species in a chicken, J Avian Med Surg 16(2) 133-7 2002.
1377. Hernandez-Divers SJ: Endosurgical debridement and diode laser ablation of lung and air sac granulomas in psittacine birds, J Avian Med Surg 16(2) 138-45 2002.
1378. Mazet JAK, Newman SH, et al: Advances in oiled bird emergency medicine and management, J Avian Med Surg 16(2) 146-9 2002.
1379. Navarez J, Tully TN, et al: What is your diagnosis? J Avian Med Surg 16(2) 154-8 2002.
1380. Tellier LA, Lair S, Livingston A: Pharmacodynamics of flunixin and ketoprofen in mallard ducks, J Zoo Wildl Med 32 222-9 2001.
1381. Samour JH, Naldo J: Diagnosis and therapeutic management of lead toxicosis in falcons in Saudi Arabia, J Avian Med Surg 16(1) 16-20 2002.
1382. Bowles HL, Zantop DW: A novel surgical technique for luxation repair of the femorotibial joint in a monk parakeet, J Avian Med Surg 16(1) 34-8 2002.
1390. Africa-Avian Clark, RK, Jessup, DA: Wildlife Restraint Series IWVS Inc. Salinas, CA, Rev 1992 A7:1-2 1991.
1400. Cooper JE: Medicine and other agents used in treatment, including emergency anesthesia kit, Birds of Prey, Health and Disease, Blackwell Science, Oxford 3 271-7 2002.
1409. Smith SA: Parasites of birds of prey: their diagnosis and treatment, Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 5 35-7 1996.
1410. Krone O, Cooper JE: Parasitic diseases Birds of Prey, Health and Disease, Blackwell Science, Oxford 3 107-20 2002.
1411. Smith SA and Smith BJ Lice in birds of prey Comp Anim Practice 2 35-7 1988.
1412. Heidenreich M: Birds of Prey, Medicine and Management, Wissenschafts-Verlag, Oxford, UK 1997.
1413. Cawthorn RJ: Cyst forming coccidia in raptors: significant pathogens or not. Redig PT, Cooper JE, et al: Raptor Biomedicine, Univ Minnesota Press, Minneapolis 1993.
1415. Bogue G: Treating endoparasites and ectoparasites in birds of prey, Methods, J Anim Health Tech 3 10-3 1980.
1416. Stehle S: Behandlung von helminthosen bei Greifvogeln mit fenbendazol Kleintier, Praxis 22 261-8 1977.
1417. Lawrence K: Efficacy of fenbendazole against nematodes in captive birds, Vet Rec 122 433-4 1983.
1418. Santiago C, Mills PA, Kirkpatrick CE: Oral capillariasis in a red-tailed hawk: treatment with fenbendazole, J Am Vet Med Assoc 187 1205-6 1985.
1420. Di Somma A, Garlinzoni M: Trichomoniasis in high performance falcons, Proc Aust Assoc Avian Vet 251-4 2002.
1421. Ostrowski S, Shobrak M: Poisoning by acetylcholinesterase inhibiting pesticides in free-ranging raptors: a case report, Falco, Newsletter Middle East Falcon Research Group 20 8-9 2002.
1422. Papich MG, Adams, RA, et al: Veterinary Pharmacology and Therapeutics, Iowa State Univ Press, Ames, IA 918-32 1997.
1423. Jones MP, Orosz SE, et al: Pharmacokinetic disposition of itraconazole in redtailed hawks, J Avian Med Surg 14(1) 15-22 2000.
1424. Orosz SE, Schroeder EC, et al: The effects of formulation on the systemic availability of itraconazole in pigeons, J Avian Med Surg 10 255-62 1996.
1426. Verwoerd D: Case study: episodic epileptiform seizures in peregrine falcons in Dubai, UAE, Falco, Newsletter, Middle East Falcon Research Group 18 10-13 2001.
1427. Lierz M: Evaluation of the dosage of ivermectin in falcons, Vet Rec 148 596-600 2001.
1428. Garcia-Montijano M, Waxman S, et al: Pharmacokinetics of marbofloxacin in Eurasian buzzards, Falco, Newsletter, Middle East Falcon Research Group 19 20 2002.
1429. Barton N, Bailey T: Editorial comment, Falco, Newsletter Middle East Falcon Research Group 20 2002.
1431. Stanford M: Cage and aviary birds. Meredith A, Redrobe S: BSAVA Manual of Exotic Pets, BSAVA, Gloucester, UK 4 157-67 2002.
1432. Redrobe S: Pigeons. Meredith A, Redrobe S: BSAVA Manual of Exotic Pets, BSAVA, Gloucester, UK 4 168-78 2002.
1433. Chitty J: Birds of prey. Meredith A, Redrobe S: BSAVA Manual of Exotic Pets, BSAVA, Gloucester, UK 4 179-92 2002.
1434. Redrobe S: Avian medicine and surgery, www.vetnet.co.uk, VetNet 10/20/2003.
1435. McCluggage D and Higdon PL Holistic Care for Birds Howell Book House New York 1999.
1437. Dorrestein GM: Diagnostic approaches and management of diseases in captive passerines, Semin Avian Exotic Pet Med, Elsevier Science, Philadelphia 12 11-20 2003.
1438. Joseph V: Infectious and parasitic diseases of captive passerines, Semin Avian Exotic Pet Med, Elsevier Science, Philadelphia 12 21-8 2003.
1439. Massey JG: Diseases and medical management of wild Passeriformes, Semin Avian Exotic Pet Med, Elsevier Science, Philadelphia 12 29-36 2003.
1441. Rosskopf WJ: Common conditions and syndromes of canaries, finches, lories and lorikeets, lovebirds and

macaws, Semin Avian Exotic Pet Med, Elsevier Science, Philadelphia 12 131-43 2003.
1444. Szelinyi Z, Peczely P: Thyroxin induced moult in domestic hens, Acta Physiol, Hung 72 143-9 1988.
1445. Johnston MS, Ivey ES: Parathyroid and ultimobranchial glands: calcium metabolism in birds, Semin Avian Exotic Pet Med, Elsevier Science, Philadelphia 11 84-93 2002.
1446. Koski MA: Dermatologic disease in psittacine birds: an investigational approach, Semin Avian Exotic Pet Med, Elsevier Science, Philadelphia 11 105-24 2002.
1447. Oglesbee BL: Hypothyroidism in a scarlet macaw, J Am Vet Med Assoc 201 1599-1601 1992.
1457. Gerlach H: Megabacteriosis, Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 10 12-9 2001.
1462. Tell L, Silverman S, Wisner E: Imaging techniques for evaluating the head of birds, reptiles and small exotic mammals, Exotic DVM 5.2 31-7 2003.
1463. Taylor M: A wound packing technique for rabbit dental abscesses, Exotic DVM 5.3 28-31 2003.
1465. Stanford M: Recombinant omega interferon in combination with F10 nebulization for the treatment…circovirus, Exotic DVM 5.3 43-6 2003.
1466. Flinchum GB: Potential use of policosanol in the treatment of hyperlipidemia in pet birds, Exotic DVM 5.3 51-5 2003.
1468. Bowles HL: Repair of displaced flexor tendon in two ducks Exotic DVM 5.3 91-4 2003.
1469. Joseph V Aspergillosis in raptors Semin Avian Exotic Pet Med WB Saunders Philadelphia 9 66-74 2000.
1470. Redrobe S: Treatment of avian liver disease, Semin Avian Exotic Pet Med, WB Saunders, Philadelphia 9 136-45 2000.
1472. Bichnese EJ: Review of avian sarcystosis Proc Assoc Avian Vet 52-8 1992.
1473. Black, D: Medicating birds, Birds 2000: the KVF Jubb Refresher Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 334 23-39 2000.
1474. Gelis S: Avian reproduction and reproductive disorders, Birds 2000: the KVF Jubb Refresher Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 334 187-208 2000.
1475. Gill J: Feather picking, Birds 2000: the KVF Jubb Refresher Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 334 247-53 2000.
1476. Cannon M: Exotic avian diseases - their diagnosis and treatment, Birds 2000: the KVF Jubb Refresher Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 334 263-80 2000.
1477. Cannon M: Avian behavior, Birds 2000: the KVF Jubb Refresher Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 334 281-312 2000.
1478. Cannon M: Medicine and surgery of aquatic birds, Birds 2000: the KVF Jubb Refresher Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 334 313-50 2000.
1479. Madill DN: Parasitology, Birds 2000: the KVF Jubb Refresher Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 334 351-81 2000.
1480. Taylor M: Diagnostics 2: Hematology and biochemistry, Birds 2000: the KVF Jubb Refresher Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 334 385-95 2000.
1481. Taylor M: Avian anesthesia and analgesia, Birds 2000: the KVF Jubb Refresher Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 334 397-405 2000.
1483. Taylor M: Emergency and critical care, Birds 2000: the KVF Jubb Refresher Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 334 429-36 2000.
1484. Taylor M: Avian surgical principles, Birds 2000: the KVF Jubb Refresher Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 334 434-35 2000.
1485. Taylor M: Chlamydiosis: disease, diagnosis and treatment, Birds 2000: the KVF Jubb Refresher Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 334 447-53 2000.
1487. McCluggage D: Apple cider vinegar. O'Donaghue D: Exotic DVM Online Group, Zoological Education Network, Lake Worth, FL 10/14 2003.
1488. Flinchum G: Milk thistle seed vs. dandelion root for treatment of liver disease. O'Donaghue D: Exotic DVM Online Group, Zoological Education Network, Lake Worth, FL 10/14 2003.
1489. Wellehan JFX: Frostbite in birds: pathophysiology and treatment, Compend Contin Educ Prac Vet 25 776-81 2003.
1490. Machin KL, Tellier LA, et al: Pharmacodynamics of flunixin and ketoprofen in mallard ducks, J Zoo Wildl Med 32 821-6 2001.
1491. Orosz SE, Jones MP, et al: Pharmacokinetics of amoxicillin plus clavulanic acid in blue-fronted Amazon parrots, J Avian Med Surg 14 107-12 2000.
1492. Doneley B: Control and Therapy of Diseases of Birds, Post Graduate Fdn Vet Science, Sydney NSW A-21 1996.
1501. Vogelnest L: Triage and treatment of oiled seabirds, Marine Wildlife: The Fabian Fay Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 335 167-78 2000.
1503. Vogelnest L: Veterinary management of seabirds, Marine Wildlife: The Fabian Fay Course for Veterinarians, Post Graduate Fdn Vet Science, Sydney NSW 335 199-236 2000.
1516. Byme RF, Davis C, et al: Prescribing for birds, Bishop Y Veterinary Formulary Pharmaceutical Press, London 5 43-56 2001.
1519. Cannon M: Orthopedic surgery in small exotic pets avian, Internal Medicine: Small Companion Animals, Post Graduate Fdn Vet Science, Sydney NSW 306 285-305 1998.
1526. Vogelnest L: Veterinary management of oiled fauna, Wildlife in Australia - Healthcare & Mangement, Post Graduate Fdn Vet Science, Sydney NSW 327 55-80 1999.
1533. Vogelnest L: Chemical restraint of Australian native fauna, Wildlife in Australia - Healthcare & Mangement, Post Graduate Fdn Vet Science, Sydney NSW 327 149-88 1999.
1549. Bellamy T: First aid for native fauna, Wildlife in Australia - Healthcare & Mangement, Post Graduate Fdn Vet Science, Sydney NSW 327 267-71 1999.
1554. Rose K: Common diseases of urban wildlife, Wildlife in Australia - Healthcare & Mangement, Post Graduate Fdn Vet Science, Sydney NSW 327 365-430 1999.
1559. Cannon M: Medicine and surgery of aquatic birds, Wildlife in Australia - Healthcare & Mangement, Post Graduate Fdn Vet Science, Sydney NSW 327 527-68 1999.
1560. Cannon M: Emerging avian diseases - their diagnosis and treatment, Wildlife in Australia - Healthcare & Mangement, Post Graduate Fdn Vet Science, Sydney NSW 327 569-86 1999.
1568. Punch P: A retrospective study of the success of medical and surgical treatment of wild Australian raptors, Aust Vet J 79 747-52 2001.
1571. Phalen DN: Avian mycobacteriosis. Bonagura JD: Current Veterinary Therapy Small Animal, WB Saunders, Philadelphia XIII 1116-8 2000.
1572. Steinohrt LA: Diagnosis and treatment of common diseases of finches. Bonagura JD: Current Veterinary Therapy Small Animal, WB Saunders, Philadelphia XIII 1119-23 2000.
1573. Clyde VL, Paul-Murphy J: Avian analgesia. Bonagura JD: Current Veterinary Therapy Small Animal, WB Saunders, Philadelphia XIII 1126-8 2000.
1575. David S: Ibuprofen, Private communication 3/26 2002.
1585. Samour JH, Naldo J: Serratospiculiasis in captive falcons in the Middle East: A review, J Avian Med Surg 15(1) 2-9 2001.
1587. Suedmeyer WK, McCaw D, Turnquist, S: Attempted photodynamic therapy of squamous cell carcinoma in the casque of a great hornbill, J Avian Med Surg 15(1) 44-49 2001.
1589. Abrams GA, Paul-Murphy J, et al: Aspergillus blepharitis and dermatitis in peregrine falcon-gyrfalcon hybrid, J Avian Med Surg 15(2) 114-120 2001.
1591. Doneley B: Cutaneous cryptococcosis in an African grey parrot, Proc Aust Assoc Avian Vet 247 1994.
1592. Tully TN, Oliver JL: What is your diagnosis? J Avian Med Surg 15(2) 147-8 2001.
1593. Sherrill J, Ware LH, et al: Contrast radiography with positive-pressure insufflation in northern pintails, J Avian Med Surg 15(3) 178-86 2001.
1609. Perry RA: Rehabilitation of birds, Australian Wildlife: John Keep Refresher Course, Post Graduate Comm Vet Science, Sydney NSW 104 115-210 1988.
1612. Jakob-Hoff RM: The husbandry and diseases of raptors, Australian Wildlife: John Keep Refresher Course, Post Graduate Comm Vet Science, Sydney NSW 104 403-51 1988.
1613. Gill J: Parrot diseases and nutrition, Australian Wildlife: John Keep Refresher Course, Post Graduate Comm Vet Science, Sydney NSW 104 585-610 1988.
1614. Harrigan KE: Causes of mortality in Little penguins, Australian Wildlife: John Keep Refresher Course, Post Graduate Comm Vet Science, Sydney NSW 104 705-16 1988.
1617. Vogelnest L: Veterinary care of wild Australian native birds II - medicine, Wildlife: TG Hungerford Refresher Course for Veterinarians, Post Graduate Comm Vet Science, Sydney NSW 233 141-89 1994.
1618. Cannon M: Avian orthopaedics, Wildlife: TG Hungerford Refresher Course for Veterinarians, Post Graduate Comm Vet Science, Sydney NSW 233 373-93 1994.
1620. Ferrell ST, Tell L: Clostridium tertium infection in a rainbow lorikeet with enteritis, J Avian Med Surg 15(3) 204-8 2001.
1621. Langlois I, Jones MP: Ventral abdominal hernia associated with hepatic lipidosis in a red lory, J Avian Med Surg 15(3) 216-22 2001.
1622. Tully TN, Porthouse K, Taylor W: What is your diagnosis? J Avian Med Surg 15(3) 223-5 2001.
1624. Wellehan JFX, Zens MS, et al: Diagnosis and treatment of conjunctivitis in house finches associated mycoplasmosis in Minnesota, J Wildl Dis 37 245-51 2001.
1625. Flammer K, Whit-Smith D, Papich M: Plasma concentrations of doxycycline in selected psittacine birds when administered in water, J Avian Med Surg 15(4) 276-82 2001.
1626. Jones R, Redig PT: Autogenous callus for repair of a humeral cortical defect in a red-tailed hawk, J Avian Med Surg 15(4) 302-9 2001.
1631. Itoh N, Kikuchi N, Hiramune T: Antimicrobial effects of amikacin therapy on experimentally induce *Salmonella typhimurium* infection, Proc World Veterinary Congress World Veterinary Association, Yokohama, Japan XXV 149 1995.
1632. Hegazi AG, Abd El Haey FK, Turker S: Evidence of chicken

immune status after propolis administration, Proc World Veterinary Congress World Veterinary Association, Yokohama, Japan XXV 159 1995.
1633. Fukomoto Y, Daimaru H, et al: Acceleration of wound healing of chitosan in zoo and wild animals, Proc World Veterinary Congress World Veterinary Association, Yokohama, Japan XXV 111 1995.
1638. Powers LV, Flammer K, Papich M: Preliminary investigation of doxycycline plasma concentration in cockatiels after administration, J Avian Med Surg 14(1) 23-30 2000.
1639. Flammer K, Aucoin DP, Whitt D: Preliminary report on the use of doxycycline medicated feed in psittacine birds, Proc Assoc Avian Vet AAV, Chicago 1-4 1991.
1640. Dorrestein GM: Doxycycline formulations for avian use, Proc Assoc Avian Vet AAV, Chicago 5-7 1991.
1644. Gentz EJ, Linn KA: Use of a dorsal cervical single pedicle advancement flap in 3 birds with cranial skin defects, J Avian Med Surg 14(1) 31-6 2000.
1645. Echols MS, Craig TM, Speer BL: Heartworm infection in 2 saddle-billed storks, J Avian Med Surg 14(1) 42-7 2000.
1646. Miller S, Fleetwood J, Williams J: What is your diagnosis? J Avian Med Surg 14(1) 54-8 2000.
1647. Gibbons P, Horton S: What is your diagnosis? J Avian Med Surg 14(1) 60-4 2000.
1648. Vogelnest L: Birds and Reptiles Urban Wildlife, Post Graduate Comm Vet Science, Sydney NSW 204 1-48 1992.
1650. Doneley B: Control & Therapy of Diseases of Birds, TG Hungerford Course, Post Grad Foundation Vet Sci, Sydney NSW A 21 1996.
1653. Meyer KF, Eddie B, et al: Chemotherapy in the control of psittacosis in parakeets. Richardson JH: Progress in psittacosis research and control, Rudgers Univ Press, New Brunswick 163 1958.
1656. Gaggermeier B, Henke J, et al: Investigation on analgesia in domestic pigeons using buprenorphine and butorphanol, Proc Eur Chap Assoc Avian Vet, Munich 6 70-74 2001.
1668. Dahlhausen RB: Implications of mycoses in clinical disorders. Harrison G, Lightfoot T (eds): Clinical Avian Medicine, 691-704 2005.
1669. Doneley B: Semen collection in the ostrich, Ostrich Odyssey, Australian Ostrich Assoc 1996.
1671. Mansour A, Khachaturian H, et al: Anatomy of CNS opioid receptors, TINS 11 308-14 1988.
1675. Woolf CJ, Chong MS: Preemptive analgesia - treating postoperative pain by preventing the establishment…sensitization, Anesth Analg 77 362-79 1993.
1682. Ness RD: Integrative therapies. Harrison G, Lightfoot T (eds): Clinical Avian Medicine, 343-364 2005.
1683. Filippich LJ: First aid in birds. Hand SJ: Care and Handling of Australian Native Animals, Surrey Beatty and Sons Chipping Norton Revised 53-62 1997.
1691. Bowles HL: Management with potassium bromide of seizures of an undetermined origin in an umbrella cockatoo, Exotic DVM 5.4 7-8 2003.
1694. Levine BS: Review of antibiotic-impregnated polymethylmethacrylate beads in avian and exotic pets, Exotic DVM 5.4 11-4 2003.
1695. Gardner G, Brown C, et al: Selected topics in captive swan medicine and surgery, Exotic DVM 5.4 33-8 2003.
1697. Harrison GJ: Amazon upper respiratory case report, Avian Examiner 1-12 3-4 1998.
1698. Hudelson S: Use of Spartrix, Avian Examiner 1-12 4 1998.
1699. Hudelson S: Comment on prostaglandins, Avian Examiner 1-12 6 1998.
1700. Cornelissen H: Treatment of hemochromatosis, Avian Examiner 1-12 11 1998.
1702. Lightfoot T: HCG for feather picking, Avian Examiner 1-12 16 1998.
1703. Harrison GJ: Suspected polyomavirus in finches, Avian Examiner 1-12 16 1998.
1704. McCluggage D: Nutritional therapy for African greys, Avian Examiner 1-12 23 1998.
1705. Harrison GJ: Home remedies, Avian Examiner 1-12 25 1998.
1707. Harrison GJ: More on heparin, Avian Examiner 1-12 27 1998.
1708. Harrison GJ: Results with Hetastarch, Avian Examiner 1-12 27 1998.
1714. Flinchum G: Convulsing cockatiels, canary, Avian Examiner 1-12 35 1998.
1715. Zantop D: Cisapride use in birds, Avian Examiner 1-12 35 1998.
1716. Gerlach H: Immune stimulators in birds, Avian Examiner 1-12 36 1998.
1720. Flinchum G: More on glyburide for cockatiel PU/PD, Avian Examiner 17 2 1999.
1721. Zantop D: Using leuprolide acetate to manage common avian reproductive problems, Avian Examiner 18 2 1999.
1722. Hochleithner C, Hochleithner M: Treatment of liver disease in psittacine birds, Avian Examiner 18 2-3 1999.
1723. Graham H, Vlamis G: Bach flower remedies for birds, Avian Examiner 18 4 1999.
1724. Flinchum G: Corticosteroid use in birds, Avian Examiner 20 2 2001.
1725. Harrison G: Uses of Avizyme, Avian Examiner 21 2 2002.
1726 Doneley B: Treatment of diabetes mellitus in a galah, Avian Examiner 21 7 2002.
1727. What's new, Avian Examiner 23 5 2003.
1728. Kramer MH: Diagnosing avian cryptosporidium, Avian Examiner 24 4 2003.
1729. Sandmeier P: Use of fipronil in canaries, Avian Examiner 25 3 2003.
1731. Morelli C, Finkelstein A: Common parasitic diseases of passerines, Exotic DVM 5.6 11-12 2004.
1732. Dorrestein GM: Passerines and softbill therapeutics, Vet Clinic NA Exotic Anim Prac, WB Saunders, Philadelphia 3 35-57 2000.
1736. Dorrestein GM, Zwart P, van der Hage MH: Pet Bird Nutrition. Rubel A, Baumgartner R: Proc Eur Chap Assoc Avian Vet, Vienna 1 10-19 1991.
1737. Trah M: Tilest - ein neues narkotikum auch für dir vogelpraxis? Kleintier Praxis 35 413-6 1990.
1738. McDonald SE: A selection of disease syndromes in psittacine birds. Rubel A, Baumgartner R: Proc Eur Chap Assoc Avian Vet, Vienna 1 46-73 1991.
1739. Orosz SE, et al: Pharmacokinetic properties of itraconazole in Blue-fronted Amazon Parrots (*Amazona aestiva aestiva*), J Avian Med Surg 10 168-73 1996.
1740. Phalen D: Respiratory medicine of cage and aviary birds, Vet Clinic NA Exotic Anim Prac 3 423-52 2000.
1741. Orosz SE, et al: The effects of formulation on the systemic availability of itraconazole in pigeons, J Avian Med Surg 9 255-62 1995.
1742. Joseph V, Pappagianis D, Reavill DR: Clotrimazole nebulization for the treatment of respiratory aspergillosis, Proc Annu Conf Assoc Avian Vet 301-6 1994.
1743. Dahlhausen RD, Lindstrom JG, Radabaugh CS: The use of terbinafine hydrochloride in the treatment of avian fungal disease, Proc Annu Conf Assoc Avian Vet 35-9 2000.
1744. Rupiper DJ: Diseases that affect race performance of homing pigeons. Part II: bacterial, fungal, parasitic, J Avian Med Surg 12 138-48 1998.
1747. Hoogesteijn AL, et al: Oral treatment of avian lead intoxication with meso-2,3-dimercaptosuccinic acid, J Zoo Anim Med 34 82-7 2003.
1748. Cannon M: Avian cryptococcosis, a case report. Proc Aust Assoc Avian Vet, Noosa QLD 1-5 1999.
1750. Dahlhausen B, Aldred MD, Colaizzi E: Resolution of clinical proventricular dilatation disease by cyclooxygenase 2 inhibition, Proc Assoc Avian Vet 9-12 2002.
1752. Clippinger TL, Platt SR: Seizures. Olsen GH, Orosz SE: Manual of Avian Medicine Mosby, St. Louis, MO 170-82 2000.
1753. Harrison GJ: Long term primidone use in an Amazon, AAV Newsletter, AAV 4 89 1982.
1756. Echols MS: Evaluating and treating the kidneys. Harrison G, Lightfoot T (eds): Clinical Avian Medicine, 451-492 2005.
1757. Dennis PM, Bennett RA: Ureterotomy for removal of two ureteroliths in a parrot, J Am Vet Med Assoc 217 865-8 2000.
1758. Lumeij JT: Pathophysiology, diagnosis and treatment of renal disease in birds of prey. Lumeij JT, et al: Raptor Biomedicine, Zoological Education Network, Lake Worth, FL III 169-78 2000.
1759. Goecke CS, Goldstein DL: Renal glomerular and tubular effects of antidiuretic hormone and two antidiuretic analogues …sparrow, Physiol Zool 70 283-91 1997.

CHAPTER

10

Integrative Therapies

ROBERT D. NESS, DVM

Greg J. Harrison

Ed. Note: According to the American Holistic Veterinary Medicine Association, "...the word 'holistic' means taking in the whole picture of the patient – the environment, the disease pattern, the relationship of pet with owner – and developing a treatment protocol using a wide range of therapies for healing the patient." This includes integrating conventional protocols with possible complementary and alternative therapies – whatever are the most efficacious, least invasive, least expensive and least harmful paths to cure. The chapter presented here is a brief introduction to selected integrative therapies in order to familiarize the avian practitioner with those that have been used in pet bird practice and to offer options for possible further study.

Integrative therapies constitute a very wide range of disciplines from around the world. Many of these therapies can be utilized to treat pet birds, although none was specifically developed for avian species. Because birds have not been domesticated, remaining genetically and evolutionarily close to their wild counterparts, they tend to be very responsive to natural therapies. Certain modalities, such as chiropractic and acupuncture, must be modified for differences in avian anatomy and physiology. Others can easily be extrapolated to pet birds from human or other mammals with only slight adjustments. Some examples include homeopathy, flower essences, nutriceuticals and many herbs. Other therapies, such as diffusion aromatherapy, must be used with caution to avoid toxic reactions. Integrative therapy in birds has existed for centuries in poultry medicine through acupuncture and herbal therapy in China.

Many terms have been used to describe these forms of treatment, including integrative therapy, alternative therapy, holistic care and complementary medicine. Each of these terms has specific implications and none of them is entirely accurate. Alternative therapy suggests another way to do the same thing. Complementary implies that it augments conventional therapy. Holistic refers to treatment of the whole patient in a complete approach, but usually infers that it is separate from conventional therapy. Integrative therapy involves the integration of a variety of modalities into a more complete healthcare system. This term is most appropriately applied when the varying therapies are used in conjunction with conventional Western therapy.

Several integrative modalities are described below, with specific indications for birds. This is not intended to be an exhaustive list of therapies for birds or serve as a

complete description of these therapies. Rather, this is an introduction into the wide array of holistic modalities and their potential implications for pet birds. Further training and education is recommended prior to widespread implementation of these therapies in practice. Veterinary certification programs are available for some of these modalities, including animal chiropractic and veterinary acupuncture. A list of resources and programs is provided in Table 10.6.

Integrative Modalities

ANIMAL CHIROPRACTIC

The practice of chiropractic is credited to D.D. Palmer during the mid-1890s.[27] D.D. Palmer's son, B.J. Palmer, further developed the practice through research and clinical practice. Although the Palmers are known as the founders of current chiropractic care, adjustments have been used for thousands of years. B.J. Palmer established the first chiropractic school in Davenport, Iowa, known as Palmer Chiropractic College.

Chiropractic is defined as "that science and art which utilizes the inherent recuperative powers of the body and deals with the relationship between the nervous system and the spinal column, including its immediate articulations, and the role of this relationship in the restoration and maintenance of health."[27] Because all functions of the body are innervated and controlled by nerves, the implications of chiropractic care in health management are enormous. Not only can chiropractic therapy treat a stiff neck or back pain, it may be useful in many systemic and metabolic disorders.

Chiropractic therapy is directed at the release of fixations and subluxations of the spine. The term subluxation is used to describe a misaligned vertebra that is unable to properly move in relation to adjacent vertebrae. This can be either a structural or functional malalignment, which may not be obvious on radiograph or conventional physical examination. These subluxations are corrected by a precise manipulation of the spine known as an adjustment. An adjustment involves the application of a high-velocity, low-amplitude manual force to release fixations without damage to the motor unit.[27] A motor unit is defined as two adjacent vertebrae and the associated structures between them, including ligaments, blood vessels, nerves, joints and muscles. The adjustment must be specific in regard to the force and angle applied to the specific vertebral joint.

In general, any vertebrate is a potential chiropractic patient, including birds. The avian skeletal system is unique from those of mammals in several ways. Birds are bipedal with modified forelimbs as wings. Many of the long bones in their limbs are pneumatic, allowing for extension of the air sac system as well as less weight for flying. The air sacs also may perfuse certain segments of the vertebral spine. The avian spine is divided into cervical, thoracic, synsacral, free-caudal and pygostyle (fused-caudal) sections.[20] The number of cervical vertebrae varies with species, with budgerigars having 11 and Amazon parrots having 12. The last cervical vertebrae and first 3 thoracic vertebrae are fused in Galliformes. The number of thoracic vertebrae varies from 3 to 10, depending on species. Ribs are present on both cervical and thoracic vertebrae. A large portion of the avian spine is fused into the synsacrum, including the lower thoracic, lumbar, sacral and caudal spine. There are 10 to 23 synsacral vertebrae and 5 to 9 caudal vertebrae. The ilium and ischium are fused together and to the synsacrum. The pubic bones are unfused, except in ratites.

Chiropractic care can be used in a variety of avian cases from trauma to reproductive conditions. Traumatic injury to the cervical vertebrae is a sequela to flying into a wall or window. Torticollis and localized feather picking also can be potential chiropractic cases. Adjustment of the thoracic spine may correct certain respiratory or digestive disturbances with underlying neurologic or neuromuscular origin. Dystocia can be the result of an abnormal egg, metabolic disturbance or abnormal pelvic anatomy. The latter etiologies may be assisted by chiropractic adjustment.[22]

VETERINARY ACUPUNCTURE

Acupuncture has been used for at least 5000 years in China, which is considered the site of origin. Early acupuncture needles were made from stone and fish bones. About 500 A.D., the practice of acupuncture spread to Japan and Korea, which established their own forms. By the 6th century, acupuncture had spread throughout Asia. By the 17th century, it was found in Europe, and finally arrived in North America during the 19th and 20th centuries. It was not until 1971 that acupuncture made its way into American culture. This was the result of a New York Times journalist being treated with acupuncture while on assignment in China. He had his appendix removed and was treated with acupuncture for postoperative pain. Over the past 30 years, acupuncture has slowly become more mainstream in American culture.

Veterinary acupuncture also has a long history. Evidence of elephant acupuncture dates back about 3000 years in Sri Lanka. The Chinese Chou Dynasty dating back to 1066 to 221 B.C. recorded several veterinary applications. The father of Chinese veterinary medicine is Shun

Yang (Pao Lo), who was the first full-time practitioner of Chinese veterinary medicine in 430 B.C.[31] Veterinary acupuncture has developed in various parts of the world, especially in Asia, over the past 2000 years. In 1974, the National Association of Veterinary Acupuncture (NAVA) was established as the first veterinary acupuncture association in the West, but was active for only 5 years.[10] Later in 1974, the International Veterinary Acupuncture Society (IVAS) was established and has since become a core association for veterinary acupuncture in the United States and the world. Since 1998, three other teaching organizations in the USA have offered training in veterinary acupuncture.

Acupuncture is one part of the holistic health system known as Traditional Chinese Medicine (TCM). Other TCM components include proper nutrition, exercise, herbal remedies and appropriate lifestyle. The main premise of TCM is that we are all part of nature, and health is achieved by establishing balance with the natural world. This balance of nature is characterized by the Chinese concept of Yin-Yang, which is the balance between such things as light and dark, wet and dry and hot and cold.

Acupuncture involves the placement of fine needles into specific points on the body to elicit a physiologic and energetic response along energetic pathways known as meridians. Meridians are interconnected energetic pathways that run throughout the body. These pathways carry the body's Qi (vital life force or energy). The presence of Qi is what defines the existence of life. The placement of acupuncture needles into points along these meridians enables the body to restore itself to homeostasis by affecting the Qi flow.

The physiologic effects of acupuncture are being studied and verified by scientific methods. The anatomic locations of acupuncture points coincide with sites of an increased density of nerve endings, small capillary beds and mast cell aggregation. As a result, a measurable physiologic effect in beta-endorphin release, stimulation of circulation and decrease in inflammation results from acupuncture stimulation. In pain control, experiments have shown a modification in neural impulse transmission from the spinal cord to the brain after acupuncture. This effect is known as "gate control" theory, which proposes that acupuncture can block the action of pain fibers in the spinal cord.[26]

A variety of acupuncture techniques exist. The use of the different techniques depends on the species and general cooperation of the patient, type and severity of the condition being treated, and personal preference of the acupuncturist. Traditional dry needling is commonly used in mammals but is more difficult in birds. Most juvenile and some adult parrots, columbiformes, waterfowl and poultry readily accept dry needles. The use of 36- or 38-gauge by 15-mm needles is appropriate for parrots, poultry and other larger birds. Plastic Seirin #5 needles can be made lightweight and better balanced by cutting off the plastic handle for better retention of the needle in birds. Smaller birds may require Sooji Chim hand needles (Korean gauge 8-mm length).

An effective alternative to traditional needling is aquapuncture. This technique involves the injection of cyanocobalamin (vitamin B_{12}) or saline into the acupoint using a 27- to 29-gauge hypodermic needle and 0.5- to 1-ml syringe. Medium to large size parrots receive up to 0.10 ml per site, while smaller birds get as little as 0.01 ml per site. The aquapuncture technique has the added advantage of providing a longer lasting effect at the site.

Another technique for potential use in birds is laser therapy. Low-intensity, cold laser lights are effective in penetrating the thin skin of birds to stimulate the shallow acupoints on birds. Disadvantages of laser therapy include the lack of specificity for acupoint stimulation in areas where multiple points are close together and lack of stimulation of deeper acupoints. Gold beads or wire implants have been used in birds for chronic cases requiring much longer periods of stimulation. Acupuncture techniques seldom used in pet birds include electroacupuncture and moxibustion, since birds are Yang by nature and both of these are strong Yang-stimulating techniques.[24]

The clinical applications of veterinary acupuncture include everything from pain management to treatment of systemic diseases. Acupuncture is effective for many chronic disorders such as allergies, arthritis, urinary incontinence and reproductive disorders. Typically, acupuncture is combined with Chinese herbs and proper nutrition to achieve the greatest effect.

Acupuncture and Traditional Chinese Veterinary Medicine (TCVM) have been developing over the past 20 years in pet bird medicine. Historically, the use of acupuncture on birds in China was primarily restricted to poultry, which was fairly limited due to the lack of economic benefit in treatment of individual birds. Rather, the administration of herbal treatments was more common for flock treatment.[7] However, the use of acupuncture in pet birds has gained some popularity in the USA in recent years, especially for the treatment of feather picking.[1,28] Despite the lack of historical documentation, acupuncture can be beneficial in the treatment of many pet bird conditions.

The use of acupuncture in birds poses various challenges from their anatomic differences and physiologic

characteristics. Birds have a high metabolic rate and relatively high body temperature (42.4° C) with rapid heart and respiratory rates. They have hollow bodies with air sacs, pneumatic bones and hollow feather shafts. As compared to mammals, birds are relatively dry, possessing minimal moisturizing glands. These characteristics make birds Yang by nature.[24] As a result, birds have a tendency toward a relative or true Yang excess when they are sick. The stress and anxiety inherent in the restraint of birds also must be considered when using acupuncture. In addition, because birds instinctively mask signs of disease, they must be thoroughly examined to reveal their true status prior to selection of the acupuncture points.

Acupuncture points are commonly extrapolated from one species to another, and special points are commonly described for individual species. Avian acupuncture employs the same techniques to locate and describe acupuncture points. Transpositional points from mammals constitute the majority of the acupoints in birds, and these may be of TCM origin or special points defined in other species. Special TCM points for poultry without a mammalian counterpart also are used in pet birds, including Gu Duan (end of thigh), used for drooping wings, and Bei Ji (back of the body spine), which is a grouping of three points used to treat respiratory disease. A few points that have been specifically described for pet birds include some of the back Shu points, which do not correspond to the mammalian counterparts because of the fused synsacrum. Detailed descriptions and indications of specific avian acupoints are defined in the listed references.[24]

Certain disorders in TCVM are more frequently seen in birds. In general, these include Liver Yin deficiency, Heart Yin deficiency, stagnant Liver Qi, Kidney Yin deficiency, Blood deficiency, Lung Yin deficiency and Lung Dryness. Kidney Essence deficiency is common in cockatiels and budgerigars that have been inbred for generations. External pathologic factors, described as Wind-Damp and Damp-Heat, are common in the Western diagnosis of microbial infections.[16]

Acupuncture can be effective in the treatment of many conventional Western conditions diagnosed in pet birds. Bacterial infections are commonly diagnosed in birds and are described as Damp-Heat or other pathogenic Heat conditions in TCVM. Conjunctivitis can be treated with local points and specific meridian points for Liver/Kidney Yin deficiency. Sinusitis is often the result of a Wind-Cold or Wind-Heat condition, based on the characteristics of the discharge. Various other TCVM conditions can present with sinusitis as a clinical sign. Identification and specific treatment of underlying factors is just as important in TCVM as in conventional therapy. Crop stasis is thought of as a problem with the Stomach or Liver meridian. The TCVM diagnosis of egg binding is a Kidney Qi condition. Kidney disease is not treated as directly with TCVM in birds as in mammals because the kidney association point is not available, yielding to the use of various kidney meridian points based on the TCVM diagnosis. Applying the basic concepts of TCVM to establish a diagnosis and treat accordingly is more effective than applying a standard set of procedures to a conventional diagnosis. As a point of reference, Table 10.1 has a list of common acupoints used in the treatment of common conditions in pet birds. In addition, Figs 10.1-10.3 illustrate the position of these points.[16]

HERBAL THERAPY

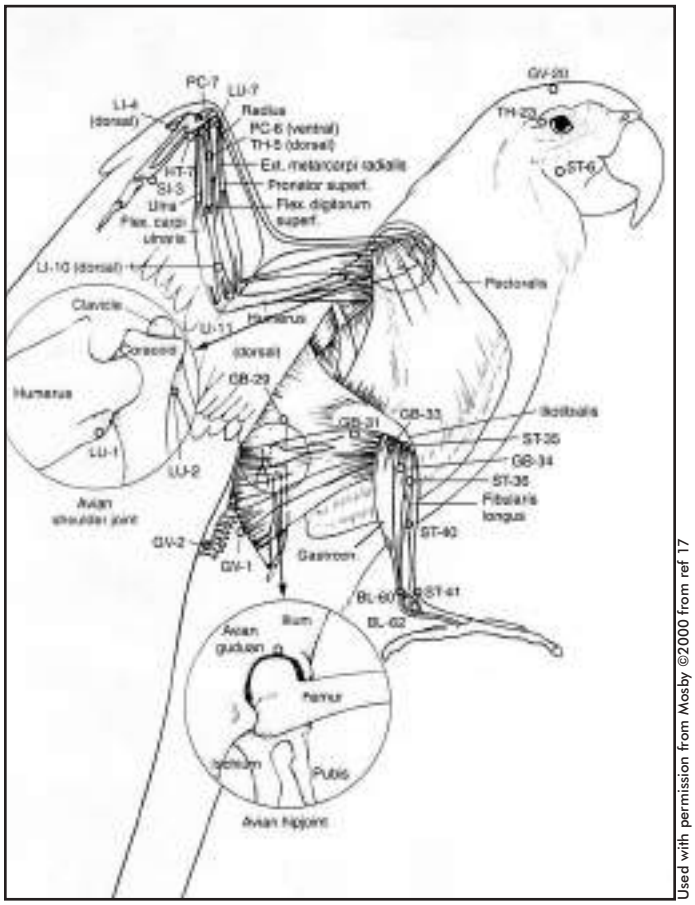
The use of specific herbs for medicinal purposes dates back thousands of years. Several herbs are mentioned in the Bible, and archeologists have documented herb use back to prehistoric times. Herbs are used around the world, including Western herbs from North America, Ayurvedic herbs from India and traditional Chinese herbs.

Approximately 25% of our conventional drugs are derived from plants. Conventional drugs typically contain a single active constituent from the plant, whereas herbs provide a broader and more balanced effect on the body through the synergistic actions of the herbal components. Herbs are best prescribed to treat the entire individual and not only the clinical signs. Herbal blends and formulations combine the benefits of multiple herbs, which typically produce a synergistic action while minimizing the potential toxic effects of a single herb. Herbs provide many unique qualities that are very limited in conventional medicine, such as anticancer, antiviral and immunoregulation properties.

Currently, herbal products are not regulated or controlled. Therefore, practitioners and clients must remain cautious in administering a product without evaluating the company and verifying that the active component of the herb or plant actually is in the formulation. Product labels can bear the name of an herb or plant substance as long as some portion of it is present in the formulation, but it does not always imply that the medicinally active constituent is included. Standardized extracts are available for certain herbs through concentrating the active ingredients, resulting in more of a plant drug than an herbal medicine.[29] Standardizing alters the physical and energetic nature of the herb. This process also eliminates the synergistic effects of the myriad chemical components in the plant. For some herbs such as milk thistle, standardization is advantageous, since the specific active constituent is clearly known and purified in the process.

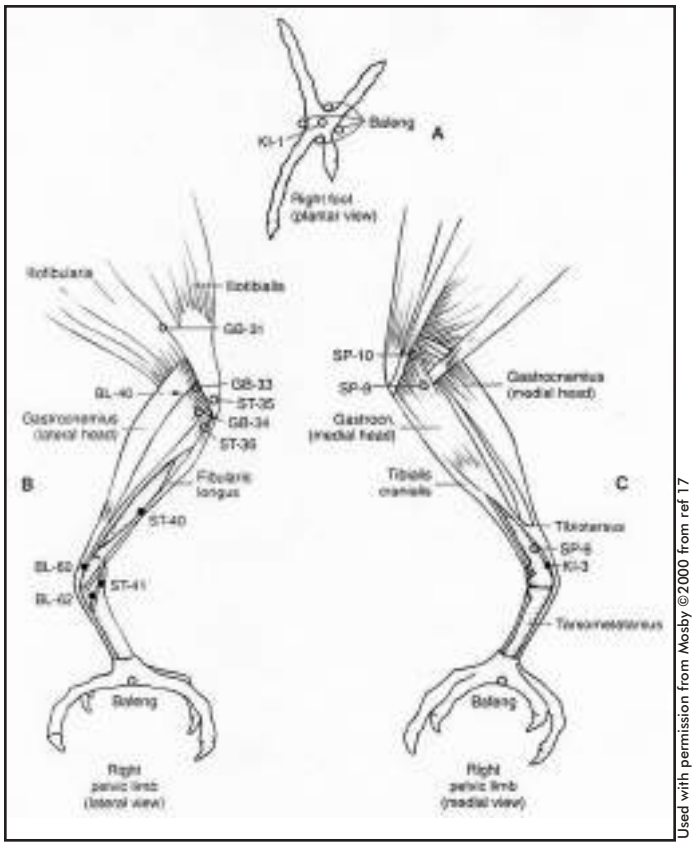
Table 10.1 | Acupuncture Points in Birds

Acupoint	Name	Characteristics	Indication / Action
LU-1	Avian Fei Tang	Alarm point for Lung	Acute resp. disease, fever, wing weakness
LU-2	Avian Yi Gen		Cold, fever, tracheitis, ptosis of wing
LU-7	Lie Que	Master point for head & neck	Neck stiffness, resp. disease, weak carpus
LU-9	Tai Yuan	Tonification for LU channel	Regulate Lung Qi, clear Lung & Liver heat
LI-4	He Gu	Master point for face & mouth	Facial swelling, eye pain, egg binding, diarrhea
LI-10	Shou San Li	Tonifies Qi	Abdominal pain, Bi syndrome of wing
LI-11	Qu Chi	He Sea point, tonification point	Pain of elbow, abdominal pain & regurgitation
ST-6	Jian Che		Facial paresis & swelling, neck pain & stiffness
ST-35	Avian Xi Gai		Inflamed, painful & swollen knee, leg weakness
ST-36	Zu San Li	Master point upper abdomen	Tonifies Qi & Blood, raises Yang, body strength
ST-37	Shang Ju Xu	Lower He-Sea point of LI	Chronic diarrhea & loose stool, remove LI damp
ST-40	Feng Long	Connecting point	Resp. disease, muscle atrophy & weak, mental
ST-41	Avian Gou Qian	Tonification point	Bi syndrome in hock & digits, pharyngitis
SP-6	San Yin Jiao	"Three Yin meeting" of leg	Egg binding, cloacal prolapse, leg paralysis/pain
SP-9	Yin Ling Quan	Sea & Water point	Distended abdomen, edema, diarrhea, knee pain
SP-10	Xue Hai	Sea of blood	Blood disorders, urticaria, pain in medial thigh
SP-11	Avian Kua Nei	Blood letting point	Inflammation, swelling or poor mobility in legs
HT-7	Shen Men	"Mind door," source point	Mental disorder, epilepsy, feather picking
SI-3	Hou Xi	Tonification point, opening of GV	Neck rigidity, wing contracture, back problems
BL-11	Avian Xin Shu	Assn. point for Heart	Mental disorders, irritability, epilepsy
BL-12	Avian Fei Shu	Assn. point for Lung	Respiratory disease/infection, fever
BL-13	Avian Wei Shu	Assn. point for Stomach	Crop disease, vomiting/regurg., indigestion
BL-14	Avian Pi Shu	Assn. point for Spleen	Maldigestion, indigestion, diarrhea, vomiting
BL-15	Xiao Chang Shu	Assn. point for Small Intestine	Lower abdominal pain, diarrhea
BL-16	Avian Gan Shu	Assn. point for Liver	Hepatopathy, conjunctivitis, inflamed cloaca
BL-17	Avian Xin Shu	Assn. point for Large Intestine	Pain in lower back, LI & cloacal disorders
BL-40	Avian Xi Wan	Master point of back & legs	Inflammation, pain, & swelling of feet & knee
BL-60	Kun Lun	Expels Wind & clears Heat	Pain in back, shoulder & wing, egg binding
BL-62	Shen Mai	Eliminate interior Wind	Epilepsy, mental confusion, pain in back & legs
KI-1	Avian Jiaodi	Foot base, sedation point	Calms mind, tonifies Yin, remove Yin-heat
KI-3	Tai Xi	Greater stream, source point	Infertility, sore throat, back pain, insomnia
KI-6	Zhao Hai	Nourishes Yin, cools blood,	Calms mind, anxiety, soft-shelled eggs, Yin def.
PC-6	Nei Guan	Inner gate, master chest/lung	Mental disorder, pain, epilepsy, fever
PC-7	Da Ling	Source point, sedation point	Gastric pain, regurgitation, panic, epilepsy
TH-4	Yang Chi	Yang pond, source point	Pain in wing & shoulder, kidney disease
TH-5	Wai Guan	Outer gate, connecting point	Motor problems in wing, behavior problems
TH-10	Tian Jing	Heavenly well	Mood swings, Bi syndrome of wing, damp-heat
TH-23	Avian Yan Jiao	Eye correct	Eye problems, expels wind, pain relief
GB-13	Benshen	Point of Yang Linking Vessel	Emotional problems, epilepsy, calming point
GB-29	Ju Liao	Removes channel obstructions	Pain & paralysis of back & legs
GB-31	Avian Kua Wai	Expels Wind - relieves itching	Inflammation, swelling & difficult leg movement
GB-34	Xi Yang Guan	Relaxes Sinews	Pain & swelling of knee (Bi syndrome)
GV-1	Avian Hou Hai	Regulates GV & CV	Loss of appetite, mental depression, prolapse
GV-2	Avian Wai Gen	Extinguishes interior Wind	Diarrhea, mental depression, cloacal prolapse
GV-12	Avian Bei Ji	TCVM poultry point	All respiratory diseases
GV-13	Avian Bei Ji	Similar to Tao Dao, clear heat	All respiratory diseases
GV-14	Avian Bei Ji	GV, BI, GB & ST meeting point	All respiratory diseases
GV-20	Avian Guan Ji	Meeting point of all Yang channel	Mental stress, depression, cloacal prolapse
GV-24	Shenting Du	Meeting point of GV & ST	Severe anxiety & fear (calms mind)
Ba Feng	Avian Jiao Pan	TCVM poultry bleeding point	Relax Sinew, pain & infection in feet
Gu Duan	Avian Gu Duan	TCVM poultry point	Ptosis of wing (poultry), Bi syndrome of pelvis



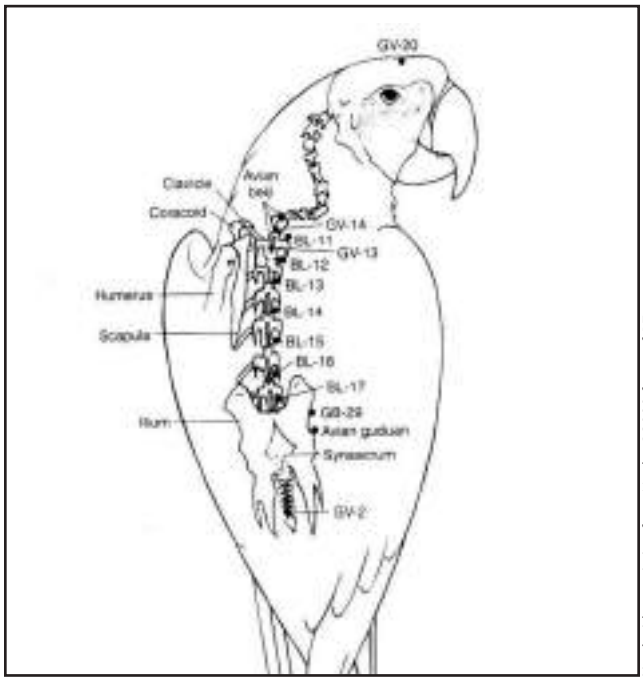
Used with permission from Mosby ©2000 from ref 17

Fig 10.1 | Acupuncture points of birds, lateral view.



Used with permission from Mosby ©2000 from ref 17

Fig 10.3 | Acupuncture points of the avian leg and foot.



Used with permission from Mosby ©2000 from ref 17

Fig 10.2 | Acupuncture points of birds, dorsal view.

Other factors that affect the potency and medicinally active components of the herb include the method and time of harvest, the parts and preparation of the plant that are included and the handling and processing of the finished product. Only well-known and respected herbal companies should be considered when purchasing herbal products. Whenever possible, fresh herbs or vegetable glycerin-based extracts should be used.

Herbs are effective in the treatment of many conditions in birds. Herbal remedies are much more effective than conventional therapy in treating metabolic conditions such as liver and kidney diseases. Herbs are an excellent alternative to antibiotics in the treatment of infectious diseases, with wider antibacterial effects in addition to various antifungal and antiviral actions. Many of these herbal remedies also support the immune system to assist in the full recovery of the patient. Some herbal formulations serve as detoxification agents, antioxidants and anticancer therapies. Table 10.2 lists several common herbal remedies with potential indications in avian therapy.[18]

Liver disease is a common diagnosis in pet birds. Hepatic lipidosis is often the result of poor nutrition, typically sunflower seed-based diets. Other chronic conditions leading to hepatic disease in birds include repeated aflatoxin exposure, heavy metal toxicity and *Chlamydophila* spp. Hepatic fibrosis and cirrhosis are potential sequelae to these conditions. However, conventional therapy falls short in treatment of these liver diseases. Certain herbs have been used for centuries in the treatment of liver disease in people, and these can

Table 10.2 | Herbal Remedies for Birds

Herbal Remedy	Scientific Name	Healing Properties	Indications
Astragalus	*Astragalus membranaceous*	Immune system booster, especially on the digestive tract	Enteritis and diarrhea, pancreatic disorders, respiratory disease, uric acid excretion
Burdock Root	*Arctium lappa*	Nutritive liver tonic, blood cleansing, gallbladder stimulant and diuretic	Chronic liver disease, environmental toxins, skin disease and irritation
Chamomile	*Matricaria recutita*	Analgesic, calming, anti-inflammatory and symptom relief of GI disorders	Calming effects, eg, feather-picking birds
Chaparral	*Larrea tridentata*	Anticancer, antioxidant, analgesic, antiseptic and anti-arthritic	Cancer treatment with red clover Do not use in egg laying, hand-feeding, or liver-diseased birds
Dandelion	*Taraxacum officinale*	Potent diuretic (leaf) and increase bile production and excretion (root)	Diuresis - ascites and respiratory fluid, liver disease - increase bile flow
Echinacea	*Echinacea*	Immune booster (especially against viruses)	Early course of infection, chronic sinusitis viral or candida infections, PBFD support
Elderberry	*Sambucus nigra*	Anti-inflammatory, alterative and antiviral	Viral infection, viral skin disorders, acute rhinitis and sinusitis
Essiac	Combination of herbs - Indian rhubarb, Slippery elm, Burdock root & Sheepshead sorrel	Anticancer formulation and lessens pain of cancer	Pain, cancer
Feverfew	*Tanacetum parthenium*	Analgesic and lowers fever	Relieves non-specific pain and inflammation, especially of GI tract
Ginseng	*Panax*	Potent immune and energy booster (use less than 2 weeks)	Anemia, immune deficiencies, diarrhea, chronic enteritis, cystic ovaries, cancer
Hawthorn Berry	*Crataegus*	Cardiac supportive, lowers blood pressure	Heart disease, supports heart in several aspects
Marshmallow	*Althea officinalis*	Antitussive, soothing of membranes and emollient	Cloacaliths and uroliths, mycobacteria, GI inflammation, feather picking
Milk Thistle	*Silybum marianum*	Hepatoprotective, hepatoregenerative and potent antioxidant	Hepatitis, hepatic lipidosis, cirrhosis, bile duct inflammation, hepatic toxicosis
Mullein	*Verbascum thapsus*	Emollient, antitussive, antispasmodic, expectorant and vulnerary properties	Self-induced trauma, ear infections, respiratory disorder, diarrhea
Olive Leaf	*Olea europaea*	Antimicrobial (bacterial, viral and fungal) and diuretic	Virtually any infection (potent effect) ascites - promote fluid excretion
Red Clover	*Trifolium pratense*	Anticancer, blood cleansing, diuretic, tonic, nutritive, estrogenic	Cancer therapy (with other herbs) supports debilitated patients
St. John's Wort	*Hypericum perforatum*	Sedative, antidepressive effects, anti-inflammatory and astringent	Pruritic or painful feather picking, chronic viral infection, anxiety
Valerian Root	*Valeriana officinalis*	Tranquilizer and sedative	Nervousness, convulsions/epilepsy, pain relief, insomnia

be extrapolated for use in birds and other pets. Some of the herbs that support and protect the liver include milk thistle (*Silybum marianum*), dandelion (*Taraxacum officinale*), Oregon grape (*Mahonia* spp.), burdock root (*Arctium lappa*) and licorice root (*Glycyrrhiza glabra*).[23]

NUTRICEUTICAL SUPPLEMENTS

Nutriceuticals are micronutrients, macronutrients and other nutritional supplements that are used as therapeutic agents. Examples include vitamins and minerals, probiotics, digestive enzymes and antioxidants. This is the clinical application of nutrition in the treatment of disease and metabolic disorders. It is commonly stated that malnutrition is the underlying cause of many of the disease syndromes encountered in birds and exotic pets. Significant advances have been made in avian nutrition with the advent of formulated diets, but it is only the beginning. Specific nutritional requirements have not been established for the various bird species commonly kept as pets, therefore current recommendations and diets are based on anecdotal experience and limited nutritional studies. The primary diet of most pet bird species should be an organic formulated diet, with limited portions of fresh organic fruit, vegetables and rice. Seeds and nuts should be considered treat items and fed in limited proportions because they are breeding stimulants (see Chapter 4, Nutritional Considerations).

The recommended diet varies according to species, age, health status and activity level. In addition, certain nutritional supplements may be indicated in the face of disease or metabolic challenges to further complement an otherwise balanced diet.

Nutriceuticals are used for various digestive disorders and other metabolic conditions in pet birds.[18] Some of the commonly used supplements are aloe juice, apple cider vinegar, probiotics[a] and digestive enzymes. *Aloe vera* (Fig 10.4) provides an effective boost to the immune system, a soothing anti-inflammatory effect on the GI tract and is an excellent source of vitamins, minerals and amino acids. Aloe can be administered orally in the form of a gel or juice at the dose of 1 drop per 100 g body weight 3 to 6 times daily or in the drinking water at the rate of 2 ml per 4 ounces of drinking water. Apple cider vinegar is an


Greg J. Harrison

Fig 10.4 | Aloe is a hardy plant grown in the full sun. It is often the best source of bioactivity. The gel from inside the plant's leaves is used for wounds. For internal consumption, the pointed edges are removed and the entire leaf is chopped up and offered, or further ground and gavaged.

acidifier of the intestinal tract and entire body. Specific avian indications for apple cider vinegar (organic, non-pasteurized) include chronic bacterial or yeast infections, chronic diarrhea or foul stools and proventricular dilation disease support. It is dosed at 1 to 2 tablespoons per 8 ounces of drinking water, as the only water source for 2 weeks. Probiotics are supplements of beneficial bacteria[a] given to reestablish the normal bacterial flora in the digestive tract. They may be administered to birds after antibiotic therapy or severe GI disturbances.

Digestive enzymes are beneficial in birds with pancreatic disease or primary digestive disorders leading to maldigestion. The classic essential enzymes provided in most formulations include protease, lipase and amylase. These may be combined with other specific enzymes or herbs, depending on the condition.

Supplements used in the treatment of inflammatory conditions and arthritis include glucosamine, methyl sulfonal methane (MSM) and proanthocyanidins.[18] Proanthocyanidins are a group of strong antioxidants that scavenge destructive free radicals and include grape seed extract, pine bark extract, bilberry and citrus bioflavonoids. These substances provide excellent antioxidant effects that reduce inflammation, improve cellular integrity and eliminate free radicals from the body. Glucosamine sulfate is the preferred and most effective form of glucosamine products. It has reparative effects on arthritis. Some formulations of glucosamine contain chondroitin or MSM for further joint support, but glucosamine is shown to be effective alone. MSM is a sulfur-based supplement that is proposed to have anti-inflammatory effects on joints and generally supports healthy tissue and cells. Sulfur is suspected to be an important mineral for the body to prevent degradation of tissues at the cellular level.

HOMEOPATHY

Homeopathy as practiced today is credited to Dr. Samuel Hahnemann, a German medical doctor from the mid-1800s. The governing principle of homeopathic medicine is *"similia similibus curantur"* or "like cures like." This concept is based on using a very diluted form of a substance to treat a condition or group of symptoms, which in its full strength would cause the same set of symptoms in the patient. These remedies are made from plants, minerals, drugs, viruses, bacteria or animal substances. Homeopathic remedies work on the deep energetic level of the patient to undermine the constitutional cause of the disease, rather than mask its symptoms.[4]

Homeopathy is very effective in pet birds.[13] Birds are highly energetic beings and thus are particularly responsive to energetic therapies. In choosing an appropriate homeopathic remedy, the practitioner must be thoroughly acquainted with the Western medical examination, conventional diagnosis, particular behavior characteristics and situational conditions of the avian patient. The mental and emotional disturbances may be difficult to discern, because most bird owners do not fully understand the normal behavior and nature of their pet. Evaluating the bird in its own environment, either personally or by videotape, is invaluable in evaluating these aspects of the diagnosis.

Because most pet birds do not visit the veterinary clinic until they are quite ill, allopathic medications (antibiotics or antifungals) may be required to get the patient through the crisis before treating with the homeopathic remedy. Due to the critical nature of clinically sick birds in practice, the practitioner may not have the opportunity to try a second remedy if the first is ineffective. The first indication of a remedy failure in these cases may be death of the patient. Initial supportive care with allopathic and other holistic therapies to stabilize the critical patient either before or in addition to the homeopathic remedy is recommended by the author.

The practice of homeopathy involves matching the patient's symptoms with an appropriate remedy. The first step involves making a list of the clinical signs from an evaluation and thorough history of the patient. This list is then used to look up rubrics, or lists of potential remedies, for each clinical sign from a homeopathic repertory. The rubrics are compared for overlapping remedies, which are selected as possible treatments. These are then compared in a homeopathic *materia medica*, which describes all the symptoms potentially treated with that remedy. The remedy that matches the

symptoms or clinical signs most accurately is selected as the first remedy of choice. The most detailed repertories and *materia medica* are based on human symptoms and responses, however, limited veterinary references exist and continue to be developed. An avian homeopathic repertory (Table 10.3) has been compiled[16] and a simplified *materia medica* (Table 10.4) summarized.[3] The potency and frequency are selected based on the severity of the condition and the characteristics of the patient.

Homeopathic remedies are made by serial dilutions of toxic substances that, if used in full strength, would cause symptoms similar to those being treated. Substances are diluted serially, either in 1/10 (X potency) or 1/100 (C potency) stages. Therefore, a 30C potency is a tincture of the homeopathic substance diluted 1/100, 30 times.

Table 10.3 | Avian Homeopathic Repertory

Location	Condition	Treatment
Beak	Cere, brown hypertrophy	Arnica montana, graphites, lycopodium clavatum, pulsatilla pratensis
	Easily cracked	Antimonium crudum, natrum muriaticum, silicea
	Dryness	Silicea, thuja occidentalis
	Exfoliation	Arsenicum album, graphites
	Overgrown, distorted	Calcarea carbonica, graphites, silicea, sulphur, thuja occidentalis
	Chronic liver disease	Argentum nitricum, carbo vegetabilis, chelidonium, calcarea carbonica, graphites, kali carbonicum, lycopodium clavatum, mercurius solubilis, natrum muriaticum, nux vomica, phosphorus, silicea, sulfur, thuja occidentalis
Extremeties	Arthritis	Amazons - ruta graveolens
		Budgerigars - bryonia alba, kali iodatum, rhododendron chrysanthum, rhus toxicodendron, sulphur, urtica urens
		General - aconitum nepellus, arnica montana, belladonna, bryonia alba, ferrum phosphoricum, kali iodatum, ledum palustre, lithium carbonicum, lycopodium clavatum, merdurius solubilis, natrum muriaticum, rhododendron chrysanthum, rhus toxicodendron, ruta graveolens, silicea, sulphur, tuberculinum avium
		Cold ameliorates - kali sulphuricum, ledum palustre, pulsatilla pratensis, sulphur
	Broken bones	Symphytum
		Bruising, associated with - arnica montana
Feet	Red and ulcerated	Sulphur
	Paralysis	Argentum nitricum, cocculus indicus, gelsemium sempervirens, hypericum perfoliatum, kali carbonicum, plumbum metallicum
	Splay-legged	Calcarea carbonica, calcarea fluorica, calcarea phosphorica, fluoricum acidum, gelsemium sempervirens, phosphorus, silicea
Feathers	Beak and feather disease	Hypericum, sulphur
	Bronzing of	Arsenicum album, nux vomica, sulphur
	Growth, none	Arsenicum album, nux vomica, selenium
	Grooming disorders (plucking or chewing)	Arnica montana, arsenicum album, calcarea carbonica, folliculinum, ignatia amara, natrum muriaticum, nux vomica, phosphoricum acidum, sepia, silicea, sulphur, thallium, tuberculinum avium, veratrum album
		African Greys - arsenicum album, natrum muriaticum
		Separation anxiety, with - natrum muriaticum
		Amazon parrots, in - nux voica, sepia, sulphur, veratrum album
		Females - aconitum napellus, apis mellifica, calcaria carbonica, chamomilla, lycopodium clavatum, pulsatilla pratensis, silicea, sulphur
		Males - apis mellifica, camphora officinarum, cantharis, conium maculatum, nux vomica, staphisagria, tuberculinum avium
	Aggression, general	Nux vomica, tuberculinum avium
		Cockatoos, in - arnica montana, arsenicum album, chamomilla, ignatia, natrum muriaticum, nux vomica, sepia
		Males, in - nux vomica
		Females, in - pulsatilla pratensis, silicea
		Folliculitis, secondary to - hepar sulphuris, hypericum perforatum, kali bichromicum, mercurius solubilis, sarsaparilla, staphisagria, sulphur
		Frantic - belladonna, stramonium, veratrum album Macaws, in - nux vomica, tuberculinum avium Males, in - nux vomica Sexual - nux vomica, sepia
Female	Binding, egg	Calcarea carbonica, kali carbonicum, pulsatillapratensis
		Blood on eggs, with - pulsatilla pratensis
		Soft-shelled eggs - calcarea carbonica, kali carbonica
	Egg laying	Kali carbonica, lycopodium clavatum, pulsatilla pratensis, sepia
		Soft-shelled eggs - kali carbonica
		Stopping of - sepia
	Infertility	Natrum muriaticum, sepia, silicea
	Ovarian cysts	Arsenicum album, belladonna
	Oviduct	Kali carbonica, pulsatilla pratensis, sepia

Table 10.3 | Avian Homeopathic Repertory (continued)

Location	Description	Treatment
Generalities	Abscesses, Granulomas	Tuberculinum avium
	Anemia	Calcarea carbonicum, ferrum metallicum, plumbum metallicum, sulfur
	Anesthesia, slow to recover	Acetic acid, carbo vegetabilis, phosphoric acid
		Ailments from - acetic acid, carbo vegetabilis, hepar sulphurous, phosphorus, phosphoric acid
	Cancer	Calcarea carbonica, carcinosinum, graphites, lycopodium clavatum, nitricum acidum, phosphorus, silicea, sulphur, thuja occidentalis
		Budgerigars, in - calcarea carbonica, carcinosinum, graphites, lycopodium
		Familial history of - carcinosinum, lycopodium clavatum
	Candida albicans infection	Calcarea carbonica, calcarea phosphorica, china officinalis, helonias dioica, lycopodium clavatum, medorrhinum, pulsatilla pratensis, natrum phosphoricum, nitricum acidum, sepia, thuja occidentalis
	Emaciation	Arsenicum album, calcaria carbonica, calcaria phosphorica, iodium, natrum muriaticum, nux vomica, lycopodium clavatum, pulsatilla pratensis, phosphorus, sepia, silicea, sulphur, tuberculinum bovinum
		Appetite ravenous with - baryta carbonica, baryta iodata, calcaria carbonica, calcaria phosphorica, causticum hahnemanni, china officinalis, cina, iodium, lycopodium clavatum, natrum muriaticum, nux vomica, silicea, sulphur
	Exposure to tobacco smoke, ailments	Gelsemium sempervirens, nux vomica, tabacum
	Lead poisoning	Alum, aurum metallicum, causticum, lycopodium clavatum, mercurius solubilis
	Pyemia	Arsenicum album, calcaria carbonica, hippozaenium, lachesis, pyrogenium
	Sepsis	Arsenicum album, arsenicum iodatum, baptisia tinctoria, china officinalis, crotalus horidus, echinacea angustifolia, lachesis
	Trauma	Aconium napellus, arnica montana, hepar sulphuris calcareum, rhus toxocodendron, ruta graveolens, symphytum officinale
		Head, with seizures - belladonna
		Neurologic symptoms, with - hypericum perforatum
	Vaccinations, acute reactions	Aconitum napellus, apis mellifica, belladonna, thuja occidentalis
		Ailments after - aconitum napellus, apis mellifica, belladonna, mercurius solubilis, phosphorus, silicea, sulphur, thuja occidentalis
	Weakness, unable to rise due to severe illness	Carbo vegetabilis
	Zinc poisoning	Aurum metallicum, mercurius solubilis
Heart	Cardiomyopathy	Crataegus oxyacantha et monogyna, digitalis purpurea
	Cyanosis	Digitalis purpurea
	Heart, general	Crataegus oxyacantha et monogyna, digitalis purpurea, rhus toxocodendron
Liver	Liver disease, general	Nux vomica, lycopodium clavatum, phosphorus
	Fatty liver disease	Calcaria carbonica, carbo vegetabilis, chelidonium majus, kali bichromica, kali carbonica, lyssinum (hydrophobinum), lycopodium clavatum, mercurius solubilis, nux vomica, phosphorus, picricum acidum, sulphur
Mind	Agitated, overstimulated	Lachesis, stramonium, veratrum album
		Outlet, without - ignatia amara, lachesis, nux vomica
	Aggression	Nux vomica, pulsatilla pratensis
	Anger	Arsenicum album, chamomilla, ignatia amara, lycopodium clavatum, nitricum acidum, nux vomica
		Underlying - nux vomica
		Violent - aconitum napellus, lycopodium clavatum, nitricum acidum, pulsatilla pratensis
	Anxiety	Aconitum napellus, argentum nitricum, arsenicum album, belladonna, calcaria carbonica, calcaria phosphorica, cannabis indica, carboneum vegetabilis, conium maculatum, euphasia officinalis, hyoscyamus niger, ignatia amara, kali carbonicum, kali nitricum, lachesis, lycopodium clavatum, mercurius solubilis, natrum muriaticum, nitricum acidum, phosphorus, pulsatilla pratensis, sepia, silicia, sulphur, thuja occidentalis, veratrum album
	Cowardliness	Gelsemium sempervirens, lycopodium clavatum
	Dependent on others	Baryta carbonica, pulsatilla pratensis
	Fatigue, mental, from inability to adapt to new surroundings	Conium maculatum, kali phosphoricum, picricum acidum
	Fear, violently throwing self around cage	Aconitum napellus, belladonna, lycopodium clavatum, nux vomica, stramonium, veratrum album
	Grief	Causticum hahnemanni, natrum muriaticum
	Irritability	Kali sulphuricum, natrum muriaticum, nitricum acidum, nux vomica, phosphorus, sepia
		Idle, while - calcarea carbonica
	Jealous, bites owner when others approach	Calcarea sulphuricum, hyoscyamus niger, lachesis, lycopodium clavatum, nux vomica, pulsatilla pratensis, stramonium
	Rigid, unable to adapt to captivity	Calcarea carbonicum, kali carbonicum
	Sensitivity	Gelsemium sempervirens, natrum muriaticum, pulsatilla pratensis, silicia
	Timid	Kali sulphuricum, pulsatilla pratensis

Table 10.3 | Avian Homeopathic Repertory (continued)

Location	Description	Condition
Mouth	Pharynx	Inflamed, chronic - graphites, sulphur
		Choanae, elongated - phosphorus
		Choanal papillae, eroded - phosphorus
Nerves	Ataxia	Arsenicum album, calcaria carbonica, nux vomica, phosphorus, plumbum metallicum, silicea, stramonium, zinc
	Nerves, general	Rhus toxicodendron, hypericum perforatum
	Paralysis	Argentum nitricum, cocculus indicus, gelsemium sempervirens, hypericum perforatum, kali carbonicum, lachesis, phosphorus, plumbum metallicum, zinc
		Renal tumors, with - hypericum perforatum, lycopodium clavatum
	Seizures	Aconitum napellus, belladonna, calcaria carbonica, ignatia amara, lycopodium clavatum, silicia
		Status epilepticus - aconitum napellus, belladonna
	Weakness	Iodium, nux vomica, plumbum metallicum, silicea, zinc, zinc phosphoricum
Nose	Catarrh	Graphites
	Colds, get easily	Graphites
	Coryza	Graphites
		Dry, obstructed - phosphorus
	Pharyngitis	Nux vomica, phosphorus, sulphur
	Sinusitis	Arsenicum album, bryonia alba, hepar sulphuris, calcareum, kali bichromicum, kali nitricum, lycopodium clavatum, mercurius solubilis, natrum muriaticum, nux vomica, phosphorus, pulsatilla pratensis, silicea
Pediatrics	Infantile behavior	Baryta carbonica
	Separation anxiety	Nux vomica
	Slow development	Calcaria carbonica
	Stunted growth, in chicks	Baryta carbonica
Respiratory	Chronic colds	Graphites
	Chronic upper respiratory infection	Arsenicum album, graphites, sulphur

Table 10.4 | Avian Homeopathic *Materia Medica*

Materia	Indication	Usage Comments
Acetic acid (glacial)	Antidote for vaporized anesthetics.	It can liquefy catarrh, which causes desperate gasping for breath. Antidotes include aconite, ignatia and opium. It must be neutralized before use of other medications - aconite is the antidotes for it. Not compatible with Arnica, Lachesis, Mercuris and Causticum.
Aconitum napellus (monkshood)	For effects of shock from injury, with fear; physical and mental restlessness.	First aid for skin injuries from cat scratches and tears, inflammation. Diarrhea during very hot weather. Useful where there is redness of skin and bird is very restless and frightened. State of collapse, where heat and fear are present.
Alumen (common potash alum)	Useful in cases of diarrhea, especially when bird is eating well and will not cease eating to produce a dropping.	Antidote for lead poisoning and other mercurials.
Apis Mellifica (honey bee)	Most cases of swelling, especially from bee stings.	Useful in cases of reddened eyes with surrounding swelling.
Argentum nitricum (nitrate of silver)	Loss of balance and coordination of mind and body.	Trembling in affected parts. Legs are withered, bird agitated. Ocular ulcers and abundant discharge.
Arnica (leopard's bane)	First aid for any injuries from blows, with bruising or danger of concussion.	Use for concussion. Sprains or strains respond well. Useful before and after surgery. For broken skin with bruising, do not apply directly to wound, but dose internally. Okay to use as ointment to bruises if skin is intact. Causes of diarrhea caused by accident or shock from surgery respond well. Useful for problems caused by old injuries. Aconite is complementary.
Arsenicum album (arsenic trioxide)	Bird is restless and prepared to bite. Body temperature is normal and eyes bright.	Useful in cases of food poisoning, often caused by bad meat; usually with green-stained vent feathers. For red, swollen legs, but not as puffy as for Apis. Gradual weight loss from impaired nutrition. Ill effects from fright. Paralysis with atrophy of legs. Putrid odor from discharges. Ailments during varying weather conditions.
Aurum metallum (gold)	Cases where bird is quiet and ready to give up and die. Sometimes is glassy eyed.	Knees weak, worse in cold weather; usually remedy for winter complaints. Antidote for lead poisoning.

Table 10.4 | Avian Homeopathic *Materia Medica* (continued)

Materia	Indication	Usage and Comments
Belladonna (deadly nightshade)	Bird is restless, unnaturally glaring eyes; convulsive movements; aversion to water; changeable attitudes. It attacks one moment and hides the next.	Useful for swollen joints; tottering gait. Cold legs and feet with jerking limbs. Wants to stand up and will not lie down.
Bellis perennis (daisy)	Use for results of accidents with nerve injuries.	Lameness from strains and sprains; sore joints and muscular stiffness. First remedy in injuries to deeper tissues and after major surgery.
Calcarea carbonica ostrearum (carbonate of lime)	Useful for abscesses in deep muscles.	For relapses during convalescence. Helps blood to clot. Eyes sensitive to light. Bird hides head in corner. Swollen eyelids. Extreme difficulty in breathing.
Calcarea Suphurica (plaster of paris)	Follows Ruta well in cases of leg stiffness.	Useful for inflammation with thick yellow discharge. Diarrhea with blood.
Calendula officinalis (marigold)	Great healer of wounds.	Stops bleeding and aids in formation of healthy tissue. Applied topically as tincture or cream or taken internally as tablet. Useful for lacerations.
Carbo vegetabilis (vegetable charcoal)	Bird is usually slow, quiet and cold. Eyes partially closed.	Used for food poisoning caused by fish.
Dulcamara (bittersweet)	Ailments caused by damp.	Recurrent rheumatism during wet weather. Birds that look ill during cold, wet weather, but no specific cause. Stiff legs; drooping wings; any weakness; chills - during wet weather.
Euphrasia (eyebright)	Use for red, sore eyes; ocular discharge.	
Gelsemium (yellow jasmine)	Bird is tired; weakness or paralysis; chilliness.	No fear of handling, and fatigued after slightest movement. Negative response to fear or fright.
Hamamelis virginica (witch hazel)	Stops bleeding.	Ideal after surgery - superior to morphine for pain. Great value in open, painful wounds. Bruised soreness of affected parts.
Hepar sulphuris calcareum (Hahnemann's calcium sulphide)	Use for suppuration with pain; unhealthy skin.	Use on sensitive ulcers and abscesses that bleed easily.
Hypercal	Useful for wounds.	Cleansing, healing and pain removing.
Hypericum (St. John's wort)	Useful for injuries involving nerves, especially toes and claws.	Injured nerves after predator attack. Relieves pain after surgery. Paralysis of legs due to mechanical spinal injury (higher potency).
Ignatia (St. Ignatius' bean)	For grief and loss of mate. For fear.	Very nervous birds; ideal for female birds that are quick, but submissive. Rapid characteristic change from quiet to panic. Fluctuating signs between appearing ill and healthy. Useful for injuries of the spine.
Ipecacuanha (ipecac root)	For upset stomach, where bird is hot.	Respiratory trouble; congestion in chest or throat; gasping for breath. General weakness of body, eyes partially closed.
Lachesis (bushmaster)	Dirty and infected wounds; sepsis; risk of gangrene.	Dark appearance of wounds.
Lathyrus (chick pea)	Paralysis without pain.	Legs dangle when picked up. Cold limbs. Slow recovery of nerve function. Cannot lift feet off ground, yet cannot lower hocks to ground.
Ledum (marsh tea)	Use for puncture wounds, especially if wound is cold.	Useful as antitetanus. Bottom of feet painful, reluctant to stand on them.
Lycopodium (club moss)	Ailments that develop slowly.	Functional powers weakening with failure of digestive function, with liver disturbance.
Manganum aceticum (manganese acetate)	Progressive paralysis with wasting of limbs.	Feeble and staggering gait; leans forward while walking, so falls onto beak. Swelling of joints; sore feet. Worse in cold weather.
Mercurius hydrargyrum (quick silver)	Indicated in weight loss; feather loss; tremors, great prostration; sensitivity to heat.	Ulceration of mouth and throat; abscesses; foul-smelling excretions; tendency for pus formation, usually greenish, thin and streaked with blood. Antidote for mercury poisoning.
Natrum muriatum (chloride of sodium)	Indicated for weakness and weariness.	Ill effects of fright. Easily irritable. Complements Ignatia.
Opium-papaver somniferum (dried latex of poppy)	Drowsy stupor. Painfulness. Lack of reaction to stimuli. Warm to hot bodied.	Does not respond to indicated remedies. Birds tuck their heads under their wings and refuse to wake up, poorly responsive.
Oxalicum acidum (sorrel acid - oxalic acid)	For short, jerky breathing, with constriction.	Paralysis due to spinal injury.
Petrolrum (crude rock oil)	Antidote for oil pollution, especially oiled birds that have digested oil off feathers.	
Plumbum metallicum (lead)	Lead poisoning, especially paralysis of wing.	Progressive muscle atrophy, excessive and rapid emaciation. Anemia. Do not give many doses of this remedy.

Table 10.4 | Avian Homeopathic *Materia Medica* (continued)

Materia	Indication	Usage and Comments
Phosphoricum acidum (phosphoric acid)	Loss of vital fluids, after diarrhea or blood loss.	Listlessness. Dyspnea. Effects of shock; gives up on life.
Psorinum (scabies vesicle)	Bird is cold and poor response to indicated remedy.	Foul odor to secretions. Single dose of 30C or 200C usually sufficient, followed by indicated remedy.
Pulsatilla (artificial sepsin - pyrogen)	Usually indicated in female changeable in characteristics.	Signs improve when outside; little to no thirst; worse from heat. Limbs are painful; stiffness in legs; swollen veins in wings; red, inflamed and swollen feet. Bird wants to sit or lie down.
Pyrogenium (artificial sepsin - pyrogen)	Food poisoning, with offensive brown-black diarrhea.	Offensive discharge; pain and burning in affected areas. Patient is restless. Great antiseptic.
Rhus Toxicodendrom (poison ivy)	Rheumatic pains are worse when limbs are kept still; bird stiff until it gets moving.	Ailments from strains; getting wet while hot. Rheumatism in cold weather. Limbs stiff, paralyzed; hot, painful swelling of joints. Worse in cold air.
Ruta graveolens (rue bitter wort)	Strained limbs, usually after Arnica stops working.	Ideal for stiff legs and/or wings.
Silicea (silica pure flint)	Promotes suppuration; brings abscesses to a "head."	Bird is cold and tired. Slow recovery after respiratory problems. Loss of strength in legs; bottom of feet are sore.
Scirrhinum	Specific for cancer.	Use with care.
Sulphur (sublimated sulphur)	Used with Aconite in cases of collapse where Aconite is indicated.	Used with Ipecac in cases of collapse. Complaints that relapse. Birds are lazy, but snappy; thin and weak; good appetite. Helps paralyzed legs after use of Rhus tox.
Symphytum (comfrey)	Healing of broken bones, tendons and sinews.	Increases strength and rate of healing. Helps heal injured eyes.
Urtica urens (stinging nettle)	For burns and scalds.	Used in tincture, cream or tablet.
Zincum Metallicum (zinc)	Lameness and weakness with twitching of various muscles.	Sensitive to noise; lethargic; cold feet. Works well with Manganese acetate.

Succession is carried out at each stage to release the curative energy of the substance to imprint on the memory of the water at the energetic level as well as remove the toxic and harmful effects of the substance.[4] The end result is a homeopathic substance that contains only the energetic signature of the toxic substance, but no physical amount of the substance itself.

FLOWER ESSENCE THERAPY

Healing with flower essences proposes similar principles to homeopathy. Both forms of therapy are based on curing the patient by restoring the body's energy pattern and vibrational characteristics. The underlying premise is that all life forms possess an innate vibrational energy force that is disrupted by conditions and circumstances of our environment, leading to disease and illness.[8] These disruptions are further related to emotional and behavioral specifics, which can be characterized and treated with the vital energy or essence of certain flowers. The aroma or essence of a flower naturally elicits an emotional response, similar to the way music affects an individual's mood.

Dr. Edward Bach is credited for the development of the first 100-year-old therapeutic system of flower essence therapy.[2] Dr. Bach was a distinguished British physician in the early 1900s with a strong influence from Hahnemann and the concepts of homeopathy. Dr. Bach developed various vaccines during his tenure in immunology, and then developed some of the first nosodes, oral homeopathic vaccines. In his clinical experience, Dr. Bach realized the importance of the mental and emotional states of mind in the recovery from illness. In 1930, he embarked on a quest to develop a treatment method that did not depend on the destruction or alteration of one living thing to benefit another, which ultimately lead to the discovery of his first twelve healing herbs with a natural affinity to mental traits. In all, 38 healing remedies were identified, which he believed would remedy all the negative states of mind that afflict mankind.

A variety of other flower essences have been described in the past 30 years. In the 1970s, Richard Katz and Patricia Kaminski developed the California Flower Essences. Ian White described the Australian bush flower essences in the 1980s, influenced by the Australian Aborigines' traditional knowledge and experience with native plants. Other flower essence lines include the Alaskan (1980s), Bailey in Britain and Celestial Remedies (1990s), to name a few.[8] All of these flower essence lines are based on the same premises described by Dr. Bach.

Flower essences can be very effective in the treatment of clinical and behavioral issues of birds and other pets. The underlying premise in using flower essences to treat

conditions in birds is the presence of an emotional component to the problem. These formulations act on the energetic signature of different emotions that produce the outward behaviors. It is commonly accepted that emotional and psychological stress can lead to physical illness; therefore, the flower essences can be incorporated into a holistic treatment plan.

In general, birds are more emotional than most other animals.[2] The stress and anxiety experienced by birds during treatment may be more detrimental than the disease condition itself; therefore, the use of a flower essence prior to and during a veterinary exam and treatment may significantly improve the chances of survival. Birds also respond quickly to the remedies, probably due to their sensitive emotional natures. Most formulations of flower essence are based in brandy, which can be harmful to the patient if given directly. Therefore, these remedies should be diluted in spring water before they are administered to birds at the rate of 10 to 12 drops per ounce of water.

Birds present with a number of medical conditions that have an emotional or behavioral basis. Feather picking is by far the most common and frustrating of these conditions. A more progressive and intense manifestation is self-mutilation, as exhibited in Moluccan cockatoos chewing into the pectoral muscles of their chests. Biting and screaming are other undesirable behaviors that are merely displaced natural behaviors, which can be modified with flower essence remedies. Birds that suffer from a physical loss of a companion, physical injury or medical illness can be supported with these remedies as part of their therapy.

The choice of remedies is individualized for each patient. Oversimplification by using a single remedy for a particular problem is much less effective than thoroughly evaluating the patient and formulating a remedy of various flower essences. Of course, there are certain remedies[a] that are commonly effective for a particular condition such as the stress and anxiety of a veterinary visit or treatment. Rescue Remedy is a classical formulation of five flower remedies, consisting of star-of-Bethlehem, rock-rose, impatiens, cherry plum and clematis. This remedy can be sprayed in the exam room, sprayed directly onto the bird or given directly by mouth.[15] A list of the classic Bach Flower Essences and their basic uses is summarized in Table 10.5.[2,8] Extensive repertories of other flower essences exist for man[11] and animals.[5]

AROMATHERAPY

The therapeutic application of aromatic essential oils is known as aromatherapy. The administration of the oils by diffusion or aerosolization is most common, but topical and oral applications also are effective routes for some formulations. The essential oils act on the underlying vibrational energy of the patient to restore the energetic imbalance causing the disease or condition. By increasing the vital force of the patient, aromatherapy strengthens the natural immune system and promotes self-healing.[9]

Birds are extremely susceptible to any aerosolized agents, including essential oils used in aromatherapy. Therefore, care must be taken not to overwhelm the bird's respiratory system with too strong a treatment. An electric aromatic diffuser can be used in a well-ventilated room for 5-minute intervals, several times daily for certain conditions. The scent of the essential oils should be barely detectable, or else it is too strong for the bird's respiratory system. Some aromatic agents may inherently be too strong or noxious for use around birds, therefore, these should be used with caution. This therapy is used less commonly in birds, except for cases of stress reduction, due to the potential respiratory risks.

Some conditions in pet birds respond well to aromatherapy, including certain respiratory ailments and stress and anxiety issues. A respiratory essential oil blend for diffusion consists of eucalyptus (50%), pine (25%), tea tree (10%), and niaouli or cajeput (15%).[9] This blend is diffused near the cage several times daily for 5-minute intervals. A 15-ml essential oil blend for stress and anxiety is composed of lavender (10 ml), marjoram (4 ml) and neroli (1 ml).[9] This is diffused for 5 minutes near the cage, repeated 4 to 5 times daily. A diffusion of lavender, bergamot or ylang ylang in the exam room provides a calming and relaxing effect on the patient, client and doctor.[23] The electric aromatic diffuser can be turned on in the waiting room or exam room for 5 to 10 minutes every 3 to 4 hours during the day.

ENERGY THERAPY

Various forms of energy therapies have developed through the ages in many cultures. Some of the currently practiced energy therapies include Reiki, therapeutic touch and pranic healing. These healing practices involve directing the ability to consciously modulate the energies of a living being. These healing practices involve the healer or practitioner serving as a conduit for the universal energy to stabilize or balance the patient's innate energy field.

Therapeutic touch is an example of this type of therapy that is practiced throughout the world. Dolores Krieger and Dora Kunz developed this practice based on the following four basic scientific premises:[12] 1) Humans and animals are physically open energy systems. This implies that the transfer of energy between living things is a natural and continuous process; 2) Humans and animals are bilaterally symmetrical, implying a pattern to the underlying energy field; 3) Illness is an imbalance in an individual's

Table 10.5 | Flower Essences

Essence	Remedies	Restores
Agrimony	Concealed distress	Inner peace
Aspen	Fear of unknown, apprehension	Courage
Beech	Intolerance, bad temper	Tolerance, flexibility
Centaury	Submissiveness, compliance	Assertiveness, resistance
Cerato	Lack of confidence	Confidence
Cherry Plum	Uncontrolled behavior, compulsiveness	Control
Chestnut Bud	Learning difficulty, repetitive behavior	Ability to learn
Chicory	Possessiveness, attention seeking	Normal caring & protectiveness
Clematis	Absentmindedness	Alertness
Crab Apple	Uncleanliness, infection, poisoning	Cleanliness, dignity
Elm	Inadequacy, overwhelmed	Competence
Gentian	Discouragement, setback	Perseverance
Gorse	Hopelessness, despair	Endurance
Heather	Loneliness, inattentiveness	Quiet composure
Holly	Malice, intense dislike	Harmlessness
Honeysuckle	Homesick, inability to cope with present conditions	Adjust to present circumstances
Hornbeam	Weakness, unresponsiveness	Vitality
Impatiens	Impatient, irritability	Patience
Larch	Hesitancy, loss of confidence	Confidence
Mimulus	Fear of known things, nervousness	Courage
Mustard	Depression, gloominess	Serenity
Oak	Lack of resilience in normal strong bird	Resilience
Olive	Fatigue & exhaustion	Strength
Pine	Guilt & contriteness	Positive attitude
Red Chestnut	Overprotectiveness, overconcern	Confidence, trust
Rockrose	Terror, hysteria	Courage, calm
Rock Water	Rigidity, repression, inflexibility	Flexibility, spontaneity, gentleness
Scleranthus	Imbalance, uncertainty	Stability, balance
Star-of-Bethlehem	Mental, emotional & physical shock	Mental, emotional & physical calmness
Sweet Chestnut	Extreme mental & physical distress	Endurance
Vervain	Impulsiveness, hyperactivity	Restraint
Vine	Dominance, territoriality	Positive leadership abilities
Walnut	Difficulty coping with change	Adaptability
Water Violet	Indifference, aloofness, reserve	Social contact
White Chestnut	Restlessness, sleepiness, preoccupation	Ability to rest
Wild Oat	Lack of direction	Direction
Wild Rose	Resignation, apathy	Will to live
Willow	Maliciousness, spitefulness	Good temper

energy field, with healing being achieved by balancing this energy field; and 4) Humans have natural abilities to transform and transcend their conditions of living. Other forms of energy therapy and healing arts are based on similar assumptions and current scientific premises.

Therapeutic touch and other energy therapies produce certain consistent and reliable results.[12] The first response experienced within a few minutes of a treatment is relaxation. Clinically, there is a significant reduction or elimination of pain. Healing responses tend to be accelerated, presumably by boosting the patient's immune system. Psychosomatic illnesses are alleviated through the effects on the patient's autonomic nervous system.

The success of these therapies is based on the learned skills and techniques combined with intentionality. Proper centering and focus is critical in assessing the patient's energy field for subtle changes and asymmetry. With practice and deliberate intent, the healing practitioner can balance the patient's energy. This technique is very useful in calming and soothing nervous or distressed patients; it also helps ease the induction and recovery from anesthesia. The pain and discomfort from trauma or illness, as well as boosting the body's innate healing response, can be relieved through this energy modulation.

Integrative Therapies for Common Avian Conditions

FEATHER PICKING

Feather picking is a clinical sign of a multitude of potential diseases and disorders and not a diagnosis in itself. The underlying causes include systemic or metabolic disorders (see Chapter 4, Nutritional Considerations, Section II Nutritional Disorders), infectious diseases, allergies, parasites and psychogenic disturbances. Most cases are psychogenic in nature, but they should have a complete diagnostic analysis to rule out other contributing factors. Treatments for feather-picking disorders vary considerably but should be based on the individual assessment with an attempt to address the underlying factors as well as the psychogenic manifestations.

Many integrative therapies can be utilized to complement the conventional approach to treating a feather-picking bird. Some of these address the psychogenic component of the problem, including Bach flower essences, herbal therapy and aromatherapy.[21] Other modalities address the deeper energetic components, such as acupuncture and homeopathy. Some treatments are directed at stabilizing the nutritional or metabolic imbalances, such as nutriceuticals and antioxidant therapy.

Acupuncture is reported as a viable option for the treatment of feather picking in pet birds.[1,7,16,24,28] The conventional diagnosis of feather picking is often attributed to the social/emotional well-being, psychological status or stressful environmental conditions of the bird. This

determination relates to a TCVM diagnosis as a Shen disturbance (deficient Heart Blood), Phlegm and Heat disturbance of the Heart, Heat invading the Pericardium, or excess Liver Yang.[28] Certain acupuncture points are routinely used in birds with feather-picking issues, but the final selection of points should be based on a complete assessment of the patient and TCVM diagnosis. Some of the routine points include SP-6, ST-36, LI-4, LI-11, PC-6 and HT-7. SP-6 is used to tonify and strengthen Yin. ST-36 is the master point of the abdomen and used to treat deficiencies, dispel Cold and tonify Qi and Blood. LI-4 helps to expel Wind Heat and to release the Exterior, tonify defensive Qi and calm the spirit. LI-11 is used to clear Heat, resolve Dampness, and regulate Nutritive Qi and Blood. PC-6 can calm the mind, regulate Heart Qi and relieve irritability due to stagnation of Liver Qi. HT-7 is useful in calming the mind, quieting the spirit, improving thinking and regulating other emotional issues. Other potential points used for feather-picking cases include GV-20, LIV-3, GB-34, SP-10, SP-11, BL-12 and BL-15. **Table 10.1** lists the common avian acupuncture points and general indications for use. The treatment regimen is dependent on each case but is usually started at once to twice weekly for the first several weeks and gradually reduced, based on the patient's response. Each session should begin with an assessment of the patient's condition and response, with adjustment of the selected acupuncture points as indicated.

Homeopathy is useful at addressing the underlying energetics of the feather-grooming problem but must be individualized for each patient.[19] A homeopathic remedy is prescribed based on the full assessment of the patient; however, certain remedies commonly surface as primary choices. Some of these include aconitum napellus, apis mellifica, arnica montana, arsenica album, belladonna, ignatia, natrum muriaticum, nux vomica, pulsatilla pratensis, psorinum, sepia, staphisagria, stramonium, tuberculinum avium and veratrum album.[19] An avian homeopathic repertory has been summarized in **Table 10.3**. In general, the study of homeopathic remedies for mental problems will reveal the proper remedy. Each type of bird has general personality characteristics that can influence the choice of remedy. For instance, cockatoos are very social birds, thus requiring a remedy that addresses social issues when separated from the flock. Alternately, Amazon parrots and macaws are more likely to be suffering from internal systemic diseases, requiring a remedy to address those problems as well. The homeopathic remedy, potency or frequency may have to be adjusted based on the patient's response and discontinued upon resolution of the problem.

Western herbs are helpful in treating the psychological issues and calming the feather-picking patient.[19] Saint John's wort has many properties that make it particularly useful in the treatment of feather picking. Numerous scientific studies have determined that Saint John's wort is an effective antidepressant.[29] In addition, it serves as a nerve tonic and speeds wound healing, which is useful in soothing and repairing the damaged feather follicles. Saint John's wort has good antibacterial and antiviral properties, beneficial in cases of infectious folliculitis or systemic diseases. Several other herbs are sedative and soothing in nature, including valerian root, passionflower and kava kava.[29] These may be effective individually or in combination for the management of very nervous and hyperactive patients.

Bach Flower Essence is excellent at addressing the underlying emotional and behavioral issues that often serve as the root of the feather-picking condition.[8,19] A combination of three to five different essences may be indicated for an individual case. Some essences address the underlying issue of fear or anxiety, such as aspen, agrimony, cherry plum, mimulus and rockrose. Others are useful when birds are picking at certain times or situations, such as red chestnut or heather for when the bird picks when left alone or scleranthus for when picking occurs during breeding season. Mustard or gorse may be indicated if the bird is feather picking out of depression. Walnut is helpful when feather picking begins after a move or other change in the environment. Wild oat can be given if the bird is simply over-preening, while agrimony is a better choice for self-mutilation. Chicory is a good choice when the picking is used to get attention, whereas cherry plum is used when the picking seems to be compulsive in nature. Most flower essence formulas also should contain a combination remedy[b] of impatiens, clematis, rockrose, cherry plum and star-of-Bethlehem for stress and anxiety. This formula also is used to calm patients for veterinary examinations, aid in recovery after surgery and treat shock and distress during severe illness or injury. **Table 10.5** contains a summary of the flower essences and general indications.

A variety of nutriceuticals can be added to balance the nutritional deficiencies and resolve underlying metabolic conditions leading to feather picking.[1,19] *Aloe vera* has anti-inflammatory and vulnerary effects, which assist healing of damaged and irritated skin and feather follicles in feather-picking cases. Aloe also provides a multitude of nutrients for healthy feather condition, including natural vitamins, minerals and amino acids. Antioxidants are often beneficial in treating chronic feather-picking cases by scavenging free radicals and supporting the healing process of damaged skin and feathers. Potent antioxidants, also called oligomeric proanthocyanidins, include grape seed extract, pycnogenol, bilberry and citrus bioflavonoids. These are available in various forms, either individually or

in combination, and can be added to the food or water as a general supplement. A potent amino acid supplement, 5-hydroxy L-tryptophan, has been suggested for feather-grooming problems.[19] Being very potent, only a few grains should be added to the food daily. Omega-3 fatty acids have been suggested for a variety of veterinary conditions including feather disorders. The benefits of omega-3 fatty acids pertinent to feather-picking disorders include treatment of seborrhea and pruritus as well as mood stabilization.[1] These fatty acid supplements also help reduce inflammation by modifying the arachidonic acid cascade.

DIGESTIVE DISORDERS

The digestive tract of birds is often disrupted by infectious and metabolic conditions. This can include anything from sour crop caused by *Candida albicans* to cloacal papillomas. Whenever possible, the underlying cause of the gastrointestinal (GI) disorder also must be identified and rectified. Many of these disturbances, however, can be stabilized with the use of nutriceuticals and herbal formulations. Several aspects of the digestive process can be addressed with these supplements, including soothing and protecting the GI mucosa, balancing the microbial population and providing nutritive support.

The GI mucosa is easily inflamed and irritated by invading pathogens or foreign agents. As a result, the inflamed mucosa will be less effective in the absorption of nutrients and proper digestion. Several Western herbs are highly effective in soothing the inflamed GI mucosa.[29] Slippery elm bark has a soothing, protecting and lubricating effect on the GI tract. In addition, it serves as an astringent and nutritive. The tannin constituents tighten the digestive mucosa to relieve inflammation and prevent further fluid loss in the intestines. The mucilage constituents help lubricate the digestive tract and facilitate the removal of waste material. Slippery elm is effective in many digestive conditions on several levels. Marshmallow root provides a soothing, lubricating and protective barrier to mucosal surfaces through its mucilage component. This makes marshmallow beneficial in cases of GI ulceration or irritation. Marshmallow is best taken internally as a tea or low-alcohol tincture. Licorice root is an excellent anti-inflammatory and demulcent herb. It is good at healing GI ulcerations and reducing the gastric acid secretions while producing anti-inflammatory effects similar to corticosteroids.

Several nutriceutical products have beneficial nutritive and supportive effects on the digestive tract. *Aloe vera* is an excellent nutritive and anti-inflammatory agent. *Aloe vera* is effective in treating inflammatory bowel disorders and constipation. The active constituents of *Aloe vera* include barbaloin and isobarbaloin. Aloe has purgative, cholagogue, anti-inflammatory, vulnerary and anthelmintic effects on the GI tract.[14] *Aloe vera* juice can be dosed orally at 1 to 2 drops per 100 g body weight or added to the drinking water at the rate of 2 ml per 4 ounces of drinking water. Apple cider vinegar is an excellent acidifier to the intestinal tract and general nutritive.[19] Indications for use include chronic diarrhea, dysbiosis, candidiasis and chronic bacterial enteritis. Apple cider vinegar can be added to the drinking water at the rate of 1 to 2 tablespoons per 8 ounces of water for up to 2 weeks. A line of rice-based intestinal support products[c] is commercially available. Rice is highly digestible and gluten free, thereby being a good hypoallergenic whole-grain product. Certain protein fractions of rice support gastrointestinal secretory function and repair of mucosal cells.[14] Therefore, these rice-based products are well suited for management of gastrointestinal inflammatory and allergy disorders, including chronic vomiting, chronic diarrhea, dysbiosis, food allergy and gram-negative enteritis. One rice-based product, Ultraclear[c], is dosed at 1 g per kg body weight, given 3 times daily.

The digestive system of birds has evolved with plant enzymes for proper digestion.[19] As more cooked and processed foods are fed to pet birds, fewer digestive enzymes are found in the diet because cooking inactivates the plant enzymes. Animal source digestive enzymes, such as pancreatic enzymes, are less effective in birds, because they are inactive in acidic environments such as the bird's crop and proventriculus. Therefore, pet birds should be provided plant sources of digestive enzymes[d], which are stable and active over a wide pH range. A source of natural enzymes produced by *Saccharomyces cerevisiae* in fermentation vats has empirically shown to have beneficial effects in birds (G.J. Harrison, personal communication, 2003).

A wide variety of digestive disorders in birds, including bulky stools, intestinal gas, undigested food in feces, slow crop emptying, chronic bacterial enteritis, weight loss and chronic immunosuppression, benefit from the addition of digestive enzymes.

Probiotics are microbial supplements given to reestablish a balanced gastrointestinal microflora. These products generally contain various species of *Lactobacillus* and *Bifidobacterium*, which are intended to repopulate the patient's intestinal tract with beneficial bacteria. Probiotics are indicated after chronic digestive disease or extended or excessive use of antibiotics, where the normal bacterial flora would be disrupted. Avian-specific products[a] are recommended; however, limited benefit may result from mammalian products or active yogurt culture.

LIVER DISEASE

Liver disease is a common diagnosis in birds. Severe liver damage in birds is commonly seen in a variety of chronic conditions ranging from nutritional disorders to psittacosis. The usual presentation is elongation and bruising of the beak and toenails. Hepatic lipidosis is common in pet birds that become obese due to their sedentary life and malnutritive seed diets. Therefore, nutritional management is crucial in managing liver disorders in birds. Certain hepatic tumors are common in budgerigars, and bile duct carcinomas are found in Amazon parrots. Iron storage disease is seen in mynahs and toucans (see Chapter 15, Evaluating and Treating the Liver).

Several Western herbs have liver-protective and liver-supportive properties. Some of them actually stimulate the regeneration of the liver cells. Herbs often perform more effectively as a synergistic formula rather than as the individual herbs. Some of the more common hepatotonic herbs are listed below with their associated benefits and indications.[23]

Milk thistle seed (*Silybum marianum*) has been used for 2000 years to treat a wide variety of liver diseases. It is used for treatment of cirrhosis, hepatitis and various forms of hepatotoxicity. Milk thistle is indicated whenever the liver has been damaged or is at risk for damage. Studies have shown that the chemical component, silymarin, has hepatoprotective properties. It has been found to serve as an antioxidant, decrease free radicals and increase hepatocyte synthesis. Other studies have shown that silymarin inhibits cytochrome P-450 enzymes in liver microsomes. As a result, milk thistle should not be used with drugs metabolized by the P-450 enzyme. There are no other known drug or herbal interactions. Elevations in liver enzymes and bile acids are possible during the first few days of using milk thistle (G.J. Harrison, personal communication, 2003).

Dandelion root (*Taraxacum officinale*) acts by gently and safely stimulating the liver into an increased state of efficiency. The improved liver function resultantly improves digestion, increases the elimination of waste from the blood and body and reduces the burden on the kidneys and immune system. The root has been used for centuries to treat jaundice. This herb makes an excellent adjunctive therapy with gentle liver support and general nutritive properties.

Oregon grape (*Mahonia* spp.) is a stronger and faster acting hepatic stimulant than dandelion root. It is indicated in cases suggestive of a deficient liver, such as poor protein digestion, constipation, or poor skin and feather condition. Since this herb stimulates bile production, its use should be avoided in suspected cases of bile duct occlusion. Because of its strong stimulatory effects, it should be used with caution in animals with preexisting liver damage.

Burdock root (*Arctium lappa*) has been used for thousands of years for a variety of ailments such as eczema, allergies, constipation and toxicity. Burdock root is very cleansing to the blood and stimulating to the liver. It possesses strong antioxidant and nutritive properties. The fresh root has broad antibacterial effects and antitumor action.

Licorice (*Glycyrrhiza glabra*) neutralizes liver toxins. Scientific articles credit a specific licorice derivative known as glycyrrhizin for successfully treating chronic hepatitis.[29] Licorice also has been shown to increase the production of interferon, which is commonly used to treat hepatitis B. Licorice also is used in combination with other herbs as a potentiator, to strengthen the effects of the herbal formula.

In addition, some Chinese herbal formulations have very beneficial effects on the liver. The herbal treatment should be based on a TCVM diagnosis. However, herbal formulations containing bupleurum and gardenia are particularly useful in treating most liver conditions in birds.[14] Coptis and Scute Combination (*Huang Lian Jie Du Tang*) is indicated in cases of viral or bacterial hepatitis with elevated white blood cell counts.[15] Another useful TCVM approach is acupuncture, with acupoints being selected by the TCVM diagnosis.

Liver detoxification is crucial in the management of many primary and secondary hepatic diseases. Certain treatments that are very effective in this detoxification process include herbal formulations and antioxidant therapy and/or rice-based intestinal products such as Ultraclear Plus[e]. These products are very useful in the management of gastrointestinal inflammation and allergy, as well as detoxification of the liver.[14] Oligomeric proanthocyanidins (OPC) are powerful antioxidants that scavenge free radicals, thus preventing further cellular degeneration. These products are very useful in chronic and degenerative diseases that involve cellular decay and degeneration. Examples of OPC antioxidants include pycnogenol, grape seed extract, bilberry and citrus bioflavonoids. Herbal formulations for liver detoxification include those described above as well as other herbs that address the individual patient needs.

Liver regeneration is often overlooked in the treatment of liver disease by conventional means. However, several holistic products are available to facilitate liver regeneration. Milk thistle is effective in regenerating hepatocytes in addition to the other benefits discussed previously. Specific products with liver-regenerative potential are

commercially available.[14] Livaplex[f] is a liver glandular combined with other liver-supportive nutrients. Lipogen[g] is indicated in cases of hepatic lipidosis.

RENAL DISEASE

Kidney disease is frequently diagnosed in pet birds but seldom specifically treated. Conventional treatments often attempt to correct only the clinical signs, such as lowering the elevated uric acid level in gout cases or reducing urine output in cases of polyuria. Specific diagnosis of the renal disorder is important in developing an effective treatment plan. Certain natural supplements and holistic remedies can be generalized to support renal function and healing of the kidney (see Chapter 16, Evaluating and Treating the Kidneys).

Omega-3 fatty acids (*n*-3 FA) have several renal benefits.[6] The supplementation of *n*-3 FA can increase renal blood flow and nephron glomerular filtration rate, lower systemic arterial blood pressure and reduce hyperlipidemia. Specifically, the *n*-3 FA supplements reduce total triglycerides and very low-density lipoprotein concentrations; *n*-3 FA reduce inflammation by modulating the arachidonic acid cascade. In addition, *n*-3 FA decrease plasma viscosity. Sources of omega-3 and omega-6 fatty acids include fish oils, flax seed, borage and pumpkin oils. The specific dosing for *n*-3 FA has not been established, but it is suggested that the ratio of *n*-6 to *n*-3 FA may be of greater importance in the overall analysis. One recommendation for dosing suggests mixing flax seed oil with 4 parts corn oil and dosing at 0.10 to 0.20 ml per kg body weight.[1]

Various Western herbal remedies have positive effects on the kidneys.[29] Dandelion leaf is a potent natural diuretic and excellent nutritive herb. The leaves provide a wide range of vitamins and minerals including potassium, which is commonly lost with mainstream diuresis. Couch grass serves as an excellent tonic and disinfectant of the urinary tract. It is a soothing, anti-inflammatory demulcent and saponin-based diuretic with mild antimicrobial effects. Couch grass is a specific remedy for chronic or acute cases of cystitis and urethritis in mammals. Herbs with good antimicrobial effects and an affinity for the kidneys include echinacea, Oregon grape and thyme. Marshmallow root is a very safe and gentle mucilage, providing a soothing and protective barrier to mucosal surfaces, including the urinary tract.

Chinese herbal products formulated as Kidney Yin/Yang/Qi tonics are beneficial for certain renal diseases. Classic formulations include Six Flavor Tea, Eight Flavor Tea, Rehmannia 6 and Rehmannia 8. Other Chinese herbal formulations may be indicated, depending on the TCVM diagnosis and concurrent problems. Acupuncture also may be beneficial in managing the patient, based on the TCVM diagnosis.

Specific nutriceutical products that are marketed for mammals may be useful in birds with similar conditions. These products often include kidney glandulars, vitamins, herbs and enzymes. Arginase is the enzyme necessary for the detoxification of arginine from the kidney.[14] Kidney-supportive products are commercially available.[h,i,j]

Recommended dietary changes include lower protein diets, but debate continues regarding the degree of protein restriction that is beneficial and safe.[6] Cases implicating protein as a contributing factor in renal diseases such as gout consistently have very high protein levels. Most importantly is a proper balancing of the diet with normal protein levels and natural, organic foods (see Chapter 4, Nutritional Considerations).

EGG BINDING

Egg binding is failure of the oviduct to pass an egg into the cloaca. In a TCVM perspective, this would be failure of the Kidney Qi to warm the lower burner, thus weakening the oviduct.[16] If the egg is stuck due to lack of lubrication in the oviduct, the TCVM assessment would be failure of the Spleen to transport and move fluids where needed, and failure in production of Qi for egg laying. Egg binding is diagnosed as interior Cold deficiency with a build up of phlegm. Potential acupuncture points include SP-6, ST-36, GV-20 and PC-6. SP-6 is chosen to strengthen the Spleen, tonify the Kidneys and calm the mind. ST-36 is the Master point of the abdomen. In addition, ST-36 tonifies the Spleen, strengthens the body and tonifies Qi. GV-20 is used to lift the spirit, clear the mind and tonify Yang. PC-6 calms the mind and tonifies the uterus. Other acupoints are selected on an individual basis, depending on the overall condition of the patient.

Chiropractic adjustment of the pelvis and synsacrum of the hen may be necessary for the proper passage of an egg. Improper nerve innervation of the oviduct can result in poor oviduct contractions with slowing or blockage of the egg-laying process. In addition, the pelvis may not widen properly due to erratic nerve innervation or hormonal imbalances. Relief of the fixations and subluxations will aid in proper nerve innervation of the reproductive tract and ease the egg-laying process.

Proper vitamin and calcium levels are critical in the laying of eggs. Low blood calcium can affect various aspects of egg laying. Calcium is necessary for the production of the eggshell, resulting in soft-shelled eggs when deficient. Calcium also is required for the normal contraction of muscles, including the smooth muscle in the wall of the oviduct. In addition, calcium must be balanced

with phosphorus and vitamin D_3 for optimal utilization and absorption. An excellent source of calcium is calcium lactate, which can be powdered onto soft foods.[19] Commercial broad-spectrum vitamin and mineral supplements formulated for birds may also be appropriate sources of calcium and related minerals (see Chapter 4, Nutritional Considerations: Section I, Nutrition and Dietary Supplementation, Chapter 5, Calcium Metabolism and Chapter 19, Endocrine Considerations).

IMMUNE DEFICIENCY AND CHRONIC INFECTIONS

Many diseases in birds, as in any other species, begin with a suppressed immune system. The weakened immune system can be the result of physical or psychological conditions. The physical causes include poor genetic constitution, malnutrition, toxic agents or infectious debilitation. Psychological sources may be environmental stress, lack of socialization, sexual frustration and abuse or neglect. The course of disease when exposed to an infectious agent is dependent on the stability of the immune system.

Nutriceutical supplements can boost the immune system by providing proper nutritional support and immune-strengthening components. Apple cider vinegar and *Aloe vera* are two examples of nutritional supplements. Antioxidants boost the immune system by clearing free radicals that otherwise would cause tissue degradation and immune suppression. These products can simply be added to the drinking water or food on a daily basis. *Aloe vera* and most antioxidants can be given as continuous daily supplements, whereas apple cider vinegar is best administered for up to 2 weeks or as needed.

Several Western herbs are effective immune boosters. Certain herbs specifically boost the patient's immune system, such as *Spirulina* sp. and pau d arco. Other herbs support the immune system through their antimicrobial properties. Herbs with antimicrobial effects include *echinacea* spp, goldenseal, Oregon grape and olive leaf.[29] These are often used in combination with other herbs as formulations. Certain Chinese herbs possess immune-stimulating properties and antimicrobial effects, including *Huang Lian Jie Du Tang* (Coptis and Scute) and *Yin Qiao San* (Lonicera and Forsythia Formula).[15]

The energetic modalities such as acupuncture and homeopathy have strong immune-modulating effects. These therapies treat at the deep energetic level of the patient to boost its innate immune response. As a result, the immune-stabilizing effects are stronger and longer lasting (see Table 10.1 for specific acupuncture points and Table 10.4 for homeopathic remedies in birds).

NEOPLASIA

Tumors and various cancers are prevalent in certain species of pet birds. Budgerigars have the highest incidence of neoplasia in pet psittacines. Various types of tumors and cancers have been reported in many avian species. Therapeutic options are often very limited when approached conventionally. However, many of these cancers can be managed with nutriceuticals, herbal remedies and other integrative therapies.

The general life-style and environment play a vital role in the risk of developing cancer in pets as well as people.[25] A well-balanced and positive emotional environment helps set the tone for health. Toxic conditions, such as smoke, chemical products, strong aerosolized sprays and odors, should be avoided. Exposure to cooking in coated cookware or plastics should be minimized. Provide fresh, clean air in a well-ventilated room and natural unfiltered sunlight. Encourage the bird to fly and/or otherwise exercise.

Good nutrition with a wide variety of healthy foods and a basis of an organic formulated diet should be provided. A variety of organic foods should be offered, including whole grains, raw fruits and vegetables. Foods high in essential vitamins and minerals are often the same as those with good antioxidant and anticancer properties. These include certain vegetables such as asparagus, tomatoes, bell pepper, turnips, kale, cauliflower and broccoli. Certain fruits also provide excellent nutrients, including apples, cranberries, pomegranate, cherry, fig, grapes and mango. Foods rich in fiber and antioxidants are recommended to fight off cancer and support the body's immune system.[29] Excellent foods to include in a diet for cancer patients include *Spirulina* spp, kelp, garlic, onions, tumeric and parsley.[25] Foods to avoid include white flour, sugar, meats, fats and dairy products. The diet also should minimize salt intake and excessive vitamin and mineral supplementation.

Herbs assist the body in fighting cancer by providing tonic support of organs and systems. Herbal formulations that support the liver, kidneys and lymphatics help strengthen the immune system by cleansing the body of toxins and metabolic waste products.[29] The classic Essiac and Hoxsey formulas were designed for this purpose. These formulations likely have changed over the years, but still contain an array of alterative and cholagogue herbs primed to cleanse the body, improve digestion and eliminate waste products.

Some notable individual herbs with strong anticancer effects include red clover, burdock root and dandelion root. Red clover is an effective anticancer herb; it inhibits the activity of carcinogenic compounds, improves blood

structure and strengthens lymphatic functions.[29] Liver-supportive herbs useful in fighting cancers include yellow dock and milk thistle. Yellow dock serves as a strong liver stimulant, while milk thistle protects the liver from harmful by-products of the cancer or cancer therapy. Diuretic herbs like dandelion leaf and nettle aid in removal of systemic waste through the kidneys and urinary tract. Herbs that assist toxic waste removal by soothing and protecting the mucus membranes of the urinary and digestive tracts include slippery elm, marshmallow, flaxseed, psyllium and plantain. Finally, immunostimulant herbs that strengthen the cancer patient's immune response include astragalus and garlic.

Anticancer herbal formulations should be individualized for each cancer patient, based on their specific cancer and the debilitating effects on the body. A typical herbal formulation usually contains three to five herbs. An example of a general tonic anticancer support formulation consists of 2 parts red clover, 1 part astragalus, 1 part dandelion root and 1 part garlic.[29] This formula, which can easily be adjusted for the particular patient or condition, provides good systemic support, immunostimulation and cleansing properties.

Nutriceuticals are common among the anticancer remedies on the market for pets and people. Inositol with IP-6[k] is a classic example of a nutriceutical product shown

Table 10.6 | Holistic Resources and Organizations

Professional Holistic Organizations

Academy of Veterinary Homeopathy (AVH)
751 NE 168th Street
North Miami, FL 33162-2427
Phone: 1-305-652-5372
Fax: 1-305-653-7244
E-mail: webmaster@acadvethom.org

The American Academy of Veterinary Acupuncture (AAVA)
PO Box 419
Hygiene, CO 80533-0419
Phone: 1-303-772-6726
E-mail: AAVAoffice@aol.com

American Holistic Veterinary Medical Association (AHVMA)
2218 Old Emmorton Road
Bel Air, MD 21014
Phone: 1-410-569-0795
Fax: 1-410-569-2346
E-mail: Office@AHVMA.org

American Veterinary Chiropractic Association (AVCA)
623 Main Street
Hillsdale, IL 61257
Phone: 1-309-658-2920
Fax: 1-309-658-2622
E-mail: AmVetChiro@aol.com

International Veterinary Acupuncture Society (IVAS)
PO Box 271395
Fort Collins, CO 80527
Phone: 1-970-266-0666
Fax: 1-970-266-0777
E-mail: IVASOffice@aol.com

Certification Programs

Veterinary Acupuncture

IVAS
PO Box 271395
Fort Collins, CO 80527
Phone: 1-970-266-0666
Fax: 1-970-266-0777
E-mail: IVASOffice@aol.com

Chi-Institute
9708 West Hwy 318
Reddick, FL 32686
Phone: 1-352-591-5385
Fax: 1-352-591-2854
E-mail: admin@chi-institute.com

Colorado State University
College of Veterinary Medicine & Biomedical Sciences
1601 Campus Delivery
Fort Collins, CO 80523-1601
Phone: 1-970-491-7051
Fax: 1-970-491-2250
E-mail: cvmbsweb@colostate.edu
Web site: www.cvmbs.colostate.edu

Animal Chiropractic

Options for Animals
Animal Chiropractic Center
623 Main St.
Hillsdale, Illinois 61257
Phone: 1-309-658-2920
Fax: 1-309-658-2622
Web site: www.animalchiro.com

Healing Oasis Wellness Center
2555 Wisconsin St.
Sturtevant, WI 53177
Phone: 1-262-878-9549
Fax: 1-262-886-6460
Web site: www.thehealingoasis.com

Internet Resources

Complementary and Alternative Veterinary Medicine
www.AltVetMed.com

Veterinary Medicine Internet Resources
www.holisticmed.com/www/veterinary.html

Bach Flower Remedies Internet Resources
www.holisticmed.com/www/bach.html

Botanical/Herbal Medicine Resources
www.rosenthal.hs.columbia.edu/Botanicals.html

Herbal Database
www.herbmed.org

to fight cancer by stimulation of the body's natural killer cell activity.[30] Antioxidants are popular additions to holistic cancer therapies because of the stabilizing effects on the patient's tissues produced through the scavenging of free radicals. Antioxidants can be in the gentle form of vitamin C or the very potent form of proanthocyanidins such as grape seed extract or pycnogenol. Certain mushrooms also have shown potential in the treatment of cancer, including Reishi, Maitake and Shiitake.[25] Coenzyme Q10 and N,N Dimethylglycine (DMG) are examples of immune-stimulating nutriceuticals with potential use in cancer therapy.[14] These products also are effective in the treatment of chronic diseases, which debilitate the body's innate healing process.

Products Mentioned in the Text

a. Parrot-specific Lactobacillus (Munich), www.janezek.de
b. Rescue Remedy, Bach Flower Remedies Ltd, Nelson Bach USA Ltd, Wilmington, MA 01887, USA
c. Ultraclear, Metagenics, 1152 Ensell Rd, Lake Zurich, IL 60047, USA, www.metagenics.com
d. Avian Enzyme, HBD International Inc, Brentwood, TN, USA, www.harrisonsbirdfoods.com
e. Ultraclear Plus, Metagenics, 1152 Ensell Rd, Lake Zurich, IL 60047, USA, www.metagenics.com
f. Livaplex, Standard Process, PO Box 904, Palmyra, WI 53156, USA, www.standardprocess.com
g. Lipogen, Metagenics, 1152 Ensell Rd, Lake Zurich, IL 60047, USA, www.metagenics.com
h. Arginex, Standard Process, PO Box 904, Palmyra, WI 53156, USA, www.standardprocess.com
i. Renatrophin, Standard Process, PO Box 904, Palmyra, WI 53156, USA, www.standardprocess.com
j. Renagen, Metagenics, 1152 Ensell Rd, Lake Zurich, IL 60047, USA, www.metagenics.com
k. Cellular Forte, Phytopharmica, Integrative Therapeutics, 9775 SW Commerce Circle, Suite A-6, Wilsonville, OR 97070, USA, www.phytopharmica.com

References and Suggested Reading

1. Baillie JW: Alternative therapy ideas for feather picking. Proc Assoc Avian Vet, 2001, pp 191-196.
2. Ball S, Howard J: Bach Flower Remedies for Animals. Saffron Walden Essex, England, The CW Daniel Co Ltd, 1999.
3. Chapman BM: Homeopathic Treatment for Birds. Saffron Walden Essex, England, The CW Daniel Co Ltd, 1991.
4. Day C: The Homeopathic Treatment of Small Animals, Principles and Practice. Saffron Walden Essex, England, The CW Daniel Co Ltd, 1998.
5. Devi L: Flower Essences for Animals. Hillsboro, OR, Beyond Words Publishing, 2000.
6. Echols S: Management of specific avian renal diseases. Proc Assoc Avian Vet, 1999, pp 101-108.
7. Ferguson B: Basic avian acupuncture. Exotic DVM 4(3): 31-34, 2002.
8. Graham H, Vlamis G: Bach Flower Remedies for Animals. Tallahassee, FL, Findhorn Press, 1999.
9. Grosjean N: Veterinary Aromatherapy. Saffron Walden Essex, England, The CW Daniel Co Ltd, 1994.
10. Jaggar DH, Robinson NG: History of veterinary acupuncture. *In* Schoen AM (ed): Veterinary Acupuncture: Ancient Art to Modern Medicine 2nd ed. St. Louis, MO, Mosby, 2001, pp 3-17.
11. Kaminski P, Katz R: Flower Essence Repertory. Nevada City, CA, Flower Essence Society, 1998.
12. Kreiger D: Accepting Your Power to Heal, The Personal Practice of Therapeutic Touch. Santa Fe, NM, Bear & Co Publishing, 1993.
13. McCluggage DM: Avian holistic veterinary medical care. Proc Amer Holistic Vet Med Assoc, 1995.
14. McCluggage DM: Avian holistic medicine: Acupuncture, nutriceuticals, and botanical medicine. Proc Assoc Avian Vet, 1997, pp 519-533.
15. McCluggage DM: Traditional Chinese medicine in birds. Proc Assoc Avian Vet, 1997, pp 535-541.
16. McCluggage DM: Holistic medicine in exotic species practice. *In* Schoen AM, Wynn SG (eds): Complementary and Alternative Medicine, Principles and Practice. St. Louis, MO, Mosby, 1998, pp 649-663.
17. McCluggage DM: Acupuncture for the avian patient. *In* Schoen AM (ed): Veterinary Acupuncture: Ancient Art to Modern Medicine 2nd ed. St. Louis, MO, Mosby, 2001, pp 307-332.
18. McCluggage DM: Overview of nutriceutical and herbal therapies in birds. Exotic DVM 3(6):8-11, 2002.
19. McCluggage DM, Higdon PL: Holistic Care for Birds. New York, Howell Book House, 1999.
20. McMillan MC: Imaging techniques. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l Inc, 1999, pp 249-250.
21. Ness RD: Alternatives for the anxious pet. Exotic DVM 3(6):12, 2002.
22. Ness RD: Chiropractic care for exotic pets. Exotic DVM 2(1):15-18, 2000.
23. Ness RD: Herbal treatment for liver disease. Exotic DVM 3(6):15-16, 2002.
24. Partington M: Avian acupuncture. *In* Schoen AM (ed): Veterinary Acupuncture: Ancient Art to Modern Medicine. St. Louis, MO, Mosby, 1994, pp 291-314.
25. Pitchford P: Healing with Whole Foods: Oriental Traditions and Modern Nutrition. Berkeley, CA, North Atlantic Books, 1993.
26. Steiss JE: The neurophysiologic basis of acupuncture. *In* Schoen AM (ed): Veterinary Acupuncture: Ancient Art to Modern Medicine 2nd ed. St. Louis, MO, Mosby, 2001, pp 27-46.
27. Willoughby S: Chiropractic Care. *In* Schoen AM, Wynn SG (eds): Complementary and Alternative Medicine, Principles and Practice. St. Louis, MO, Mosby, 1998, pp 185-200.
28. Worell AB, Farber WL: The use of acupuncture in the treatment of feather picking in psittacines. Proc Assoc Avian Vet, 1993, pp 1-6.
29. Wulff-Tilford ML, Tilford GL: Herbs for Pets. Irving, CA, BowTie Press, 1999.
30. Wynn SG: Emerging Therapies, Using Herbs and Nutraceutical Supplements for Small Animals. Lakewood, CO, AAHA Press, 1999.
31. Xie H: Traditional Chinese Veterinary Medicine. Beijing, China, Beijing Agricultural University Press, 1994.

CHAPTER
11

Low-risk Pest Management

DIANA POST, VMD

Greg J. Harrison

Information in this chapter is provided to help ensure that wherever birds are being maintained, pest problems can be dealt with effectively and potentially hazardous conditions can be avoided.

The Environmental Protection Agency's (EPA) classification of pesticides as "low risk" refers to the potential effects in humans, not companion animals or wildlife. This should be kept in mind when a chemical pesticide is being promoted as a "low-risk" alternative. Check with Rachel Carson Council (RCC),[8,16] Biointegral Resource Center (BIRC)[13] or other organizations for non-chemical alternatives that can represent low risks to pets, wildlife and the ecosystem, as well as to people.

Most veterinarians have traditionally used pesticides to control the parasite problems of their patients. However, many of them have adopted a hands-off approach toward routine environmental pest control for ants, cockroaches, rodents, etc, leaving this to the specialized domain of certified exterminators and trusting them to use the products properly. A recent incident serves as a warning to animal health care professionals to reevaluate this practice. The incident happened in January 2003 following use of the intensely toxic rodenticide zinc phosphide. The fatal poisoning of two valuable red pandas at the National Zoo resulted in part from failure of veterinary medical personnel to give sufficiently critical oversight to rodent control procedures.

A promising outcome of the zinc phosphide incident has been the National Zoo's adoption of Integrated Pest Management (IPM) procedures for its animal facilities. IPM in principle involves greater reliance on a variety of low-risk pest management practices and the use of toxic chemicals only as a last resort. Pest problems where companion birds reside can be controlled with similar strategies.

Those in charge of keeping animals healthy cannot afford to delegate pest control measures without first making certain that the intended procedures provide the lowest possible risk to non-target individuals. In the future, with this greater awareness, we are confident that veterinarians will be able to promote low-risk pest management methods for use on premises where animals are under their care. This can happen only if veterinarians understand certain concepts and how they work.

Justifications for avoiding use of chemical pesticides near companion birds can be similar to those given by the United States Environmental Protection Agency (USEPA) for avoiding them around another vulnerable population — children: "Chemical pesticides…may cause a range of harm, such as cancer, (as well as) acute or chronic injury to…(respiratory), nervous, reproductive, endocrine and immune systems…"[22] There are additional reasons for bird owners to avoid chemical pesticides. Tests for acute toxicity of pesticide products are performed on rodents. Since birds are generally more sensitive to chemical pesticides than most mammals including rodents, the required tests may not be predictive for toxicity in birds.

Sensitivities to chemicals can vary widely among avian species and it is not possible to designate a single representative bird species to serve as the surrogate for all others in testing pesticide hazards.[9] Due to birds' increased vulnerability, they have been used to provide early warnings of toxic environmental conditions potentially harmful to those less sensitive, for example, the canaries were used to protect coal miners from asphyxiation due to methane gases that replace oxygen and tend to collect in coal mines.

Sentinel birds can suffer serious adverse consequences in the process of protecting people.[12,19] If environments are safe for birds, however, then people sharing homes with them will likely benefit as well.

A CASE HISTORY

Before it was banned in 2000 for indoor application, the organophosphate insecticide chlorpyrifos was widely used. Chlorpyrifos treatments for cockroaches in a home-based aviary resulted in the eventual loss of the entire breeding bird population of 15 pairs and their offspring. The toxic nature of the chemical as well as actions by the parties involved contributed to this unfortunate outcome. The applicator called the pesticide "safe" and did not appear to know its potential danger to birds, although the owner specifically questioned him about it. The aviary owner accepted the exterminator's assurances of safety and failed to get a second opinion from those knowledgeable in pesticide toxicity to make certain that the pest control methods used truly afforded the lowest possible risk to birds. In an ironic twist, the owner was able to collect financial compensation for theoretical damage to his own health that the birds' deaths had indicated.[12]

COULD CHEMICAL PESTICIDES IN USE TODAY RESULT IN BIRD INJURIES?

Although chlorpyrifos and diazinon are no longer available for indoor use in the USA, neurotoxic chemicals (including cholinesterase inhibitors, pyrethroids as well as newer entities such as fipronil) are registered for indoor use and can contact a pet bird through diffusing from application sites such as rugs, furniture, the skin of a dog or cat, and bait stations, even if the bird is removed during the actual application process. Two newer insecticides, fipronil and imidacloprid, have been associated with adverse reactions in people and pets when used in flea control programs (J.H. Gainer, personal communication, 2003). Both insecticides are registered for additional uses in and around the home.

By persisting indoors for months or years on rugs or furniture, certain chemical pesticides may present the potential for adverse effects. When applied outdoors, these same chemicals, plus others registered for outdoor use only, can drift indoors through open windows, contacting a caged bird kept nearby, or they can gain access from air intake ducts or be carried indoors on shoes. Birds and/or their environments may be treated with cholinesterase inhibitors such as the insecticide carbaryl (recommended for mite control) or outdated remedies such as the toxic substance paradichlorobenzene (found in moth crystals) used around cages. Further, the effects of multiple chemical exposures can be additive or even synergistic, with greater likelihood of adverse reactions occurring as a result.

Due to concern over West Nile virus, chemical pesticides may be broadcast from an aircraft or from a land-based vehicle in an effort to reduce mosquitoes. Pet birds (and other sensitive individuals) can be protected from contact with these sprays by closing windows and air intake ducts when the application occurs. If possible, a pet owner should obtain information from local governments, professional pest control companies, landscapers or neighbors who apply pesticides as to time, place and nature of the pesticide product being sprayed close to the home. Greater surveillance of marketed products is needed to collect adverse reaction information. This is especially important for products used in the home and applied to pet animals, such as those products with fipronil and imidacloprid as active ingredients.

INDOOR AIR QUALITY AND PESTICIDES

We have much to learn about the persistence of chemical pesticides indoors. However, what we do know raises troubling concerns even for those chemical agents considered to be less acutely hazardous to birds and people — the pyrethroids. Since the recent banning of chlorpyrifos and diazinon for indoor use, chemicals of the pyrethroid class have become more popular. The indoor half-life of long-lasting pyrethroids such as permethrin, deltamethrin, cypermethrin and cyfluthrin may be up to 10 years.[17] Pyrethroids are synthetic versions of the naturally occurring pyrethrins. The latter are much less persistent and less acutely toxic, but they can still evoke allergic reactions.[6] A study reported that a percentage of the human population in contact with pyrethroid-treated carpets had vague signs of discomfort (headaches, respiratory disorders, burning eyes, dizziness, tiredness, pain of muscles, bones and joints). In a significant number of subjects, these adverse effects regressed when the carpeting was removed. A scientist working on the study has questioned, "…whether the indoor use of pyrethroids is safe enough to avoid adverse health effects."[17] Could birds be harmed due to presence of pyrethroids indoors? Perhaps yes, but we do not know with certainty. However, we do know that a number of alternative methods are available in place of synthetic chemical pesticides for management of pests, both indoors and out.

General Principles and Examples

Approaches to management of indoor and outdoor pests can be very different. Indoors, it is frequently possible to exclude the pest and employ low-risk products when necessary. Biological controls are rarely used indoors. Outdoors, exclusion is less common and biological controls are routinely used, especially by organic farmers and landscapers using biosustainable methods. Soils not designed to grow plants or crops need more pesticides.

BEFORE ANY CONTROL ACTION IS TAKEN

Accurate identification of the "pest" in question is the first step to successful management, especially when dealing with an insect. Entomologists at institutions, local extension service employees or professional pest control personnel can be useful here. Ants, cockroaches, moths, etc, may vary by species in their preferred habitats, food choices and life cycles. Accurate identification should help with selecting effective management methods, deciding whether the organism in question is a true pest, a non-threatening, neutral insect visitor or even a beneficial insect (distinguishing the latter can be especially important for outdoor pest control); see The Importance of Identifying the Pest and Lessons from a Lacewing below.

INDOOR PEST MANAGEMENT STRATEGIES

Indoor — Primary Strategy

After determining that a pest is present and there is a problem, the primary strategy involves preventing the pest from having access to a life-support system consisting of water, food and indoor habitat. Frequently, these measures are the only ones required to significantly reduce or eliminate problem pest populations. They also are prerequisites to further management actions;[5] see each pest for particulars.

Indoor — Secondary Strategy

Once the primary strategy has been implemented, further action may be needed. Secondary control methods change the pests' habitat while presenting low risks to people, pets and the environment;[5] see each pest for particulars.

OUTDOOR PEST MANAGEMENT STRATEGIES

Outdoor — Primary Strategy

Many lawn and garden pests can be avoided by taking into account the local growing conditions, choosing naturally disease-resistant plants that are also non-invasive native varieties, as well as by using organic fertilizer and compost. Awareness of soil conditions and knowledge of native plant species can help guide landscaping decisions.[21] Exclusion is used less frequently than for indoor pests, but if needed it can be accomplished with row covers, netting or fences to keep insects, birds and even deer away from plants.

Outdoor — Secondary Strategy

When pests are found at unacceptable levels and intervention is needed, the secondary strategy makes use of mechanical and biological controls. There are astonishing biological controls (most suitable for outdoors only) for use against pest insects. For example, beneficial insects such as lady beetles (ladybugs) obligingly prey upon aphids and other pests. Females of the non-stinging Brachonid wasps deposit eggs in the body of pests, resulting in the growth of wasp larvae at the expense of the pest. Such insects can be invited to a garden through selective plantings and/or they can be purchased from suppliers (see Suggested Reading for *"If You Plant It,*

Table 11.1 | Biological Control Resources

Refer to these resources for more information on biological control products and low-risk pest control methods:

1. Rachel Carson Council, Inc (RCC)
   www.RachelCarsonCouncil.com
   E-mail: rccouncil@aol.com
   Phone: 301-593-7507
2. Biointegral Resource Center (BIRC)
   www.birc.org
   E-mail: birc@igc.org
   Phone: 510-524-2567
3. Gardens Alive!
   www.GardensAlive.com
   E-mail:
   gardner@GardensAlive.com
   Phone: 812-537-8650

They Will Come" and Table 11.1). Bacteria, fungi, viruses and nematodes also are available for selective pest control. Birds can act as biological controls for insects; so can turtles, amphibians and even fish. For some large herbivores such as deer, contraception can be an effective control method. For mosquito management outdoors, see the discussion below on Mosquitoes.

EXAMPLES OF INSECT IDENTIFICATION

The Importance of Identifying the Pest

A family with two small children moved into a country house where they encountered a large number of tiny red insects that the mother believed to be fleas. Instead of spraying the house, as advised by a local exterminator, the mother contacted RCC for advice. RCC loaned her a yellow/green intermittent light flea trap that she used to obtain specimens of the insects. Her local USDA extension agent identified the insects as red clover mites, non-biting insects. Using her vacuum cleaner, as recommended by RCC, she was able to rid the house of the mites without resorting to the chemical spray.[8]

Lessons from a Lacewing

Environmentalist Anna Edey described how she became an expert in recognizing beneficial insects: "One time I saw a little cluster of 10 to 15 very fine hairs about one-half inch long protruding straight up from a hibiscus leaf. Each hair had a tiny pinhead-sized egg…at the end of it…I decided it looked like a virulent fungus…I nipped off the leaf and discarded it. Later I found out that I had destroyed the eggs of one of the most valuable beneficial insects, the green lacewing."[16]

Indoor Low-Risk Pest Management

ANTS

Ants (Fig 11.1) are one of the most abundant social insect species on earth. They can become pests in buildings as they search for water, food or places to establish

Greg J. Harrison

Fig 11.1 | Ants following a crack in the cement to a food source. Ants leave a chemical trail that soap and water can erase.

colonies. Carpenter ants can cause considerable damage in moist areas by excavating galleries and establishing residences in wall voids. Ants living outdoors, however, like spiders, can be considered as non-threatening or beneficial through helping to control insects including fleas, termites and fly larvae. Denying access to ants seeking shelter indoors, as well as eliminating access to water and food, can significantly reduce ant populations along with those of other insects.

Preventing Indoor Access

Prevent access by having a sweep on the door where it touches the floor that is in continuous contact with the floor (vertical strips of plastic are not effective at keeping ants out); caulking cracks in the walls and around pipes can prevent ants from coming indoors. In an emergency, duct tape can be used to seal up an ant access area until it can be caulked.[13] It is important to be sure that the caulk does not contain volatile components that may be irritating to chemically sensitive people and pets.

Preventing Water Access

Ants are attracted to moisture, so any leaks in ceilings or walls and conditions that predispose to moisture should be promptly repaired. These can come from water pipes, downspouts, damaged roofing tiles as well as structures that do not allow for proper ventilation.

Preventing Food Access

Ants are attracted to and consume both sugar and protein, so spilled food should be removed quickly and the area wiped with a soapy water solution. Pet food dishes can be placed in a moat/dish (for example a shallow pie pan) of soapy water to prevent ant access. The soap is important to reduce surface tension so the ants drown.[13]

Food should be stored in ant-proof containers. These consist of a glass jar with a rubber seal or a plastic container with a tight-fitting lid. Ants sometimes get into the refrigerator if the seal around the door is faulty.

Kitchen areas should be kept clean and dry with frequent sweeping, vacuuming and washing up of spills. If water accumulates in a pan under the refrigerator, the pan should be regularly emptied and cleaned.

Dishes can be soaked in soapy water if immediate washing is not possible. Food containers should be rinsed of all food residues before discarding. If possible, trash should be stored outside until collected.

Water, food and droppings in birdcages can attract ants. Birds can be protected by placing a circle of sticky material such as double-sided tape completely around the birdcage stand, since ants do not like to cross sticky material.[5] The same type of sticky material also can be placed around the chain or rope from which the birdcage is suspended. Since birds can become immobilized on sticky tape, it should be placed where birds will not routinely encounter it. Sticky barriers also can be used outdoors to discourage ants, although wild birds can be at greater risk from this placement, especially if they feed on insects caught on the sticky tape. Alternatively, the birdcage stand can be placed in a moat of soapy water.

Ants seem to find extruded polystyrene foam insulation (also known as "blue board") to be ideal for locating colonies. This could be important to those considering remodeling where ants could be a potential problem.

Changing Habitat — Indoor

If ants continue to be present indoors, a non-volatile pesticide can be applied in such a way that children and pets will not be able to come in contact with it. For example, diatomaceous earth or silica aerogel, either alone or in conjunction with boric acid powder, can be placed in floor cracks and crevices and behind cabinets — sites usually accessible only to ants. Dust products should not be inhaled. Contracting with a professional for any applications or employing safety equipment if the homeowner uses them can minimize problems. Diatomaceous earth should not be the kind used in swimming pool filters; it has been polished and therefore is not effective and presents a serious respiratory hazard. Alternatively, bait stations or gel with a pesticide and an ant attractant can be applied in areas not accessible to pets or people, but where ants will pick it up and carry it to the colony. Pesticide bait stations for ants can be placed around the floor if they are not accessible to children and pets. Pesticide concentrations in most bait stations are low enough not to kill the individual ants until they have returned to the colony and shared the bait with other members. It is important to avoid using bait stations in the same vicinity as pesticides with pyrethrins acting as repellents (see Suggested Reading "What's Wrong with My Bait?").

Baits may take longer to establish control, especially if the active ingredient is an insect growth regulator. Low-risk baits should be placed where they will not come in contact with people or pets, or where a curious bird cannot find one and chew on it. Ants respond to either sugar or protein baits and they may alternate their preferences.

Ideally, the pesticide chemicals in baits should not be volatile, as that would present a hazard to sensitive people or pets. Since new formulations may not follow this rule, monitoring for adverse signs associated with placement of baits is recommended. The chemical fipronil is believed to adversely affect chemically sensitive people when present in bait stations (W. Currie, personal communication, 2002).

An innovative device, the ant guard, has successfully prevented, among other things, fire ants from gaining access to nursing home patients in their beds. In appearance it resembles the metal collar-like rat guard used to block rats from crawling up ships mooring ropes. These initial ant guards have had a chemical deterrent in the form of the pyrethroid insecticide, permethrin, painted on the inside of the collar. If the chemical component could consist of a very low-risk repellent or toxicant instead of permethrin, then it is very likely that ant guards could enjoy a host of additional uses.[7]

Carpenter Ants

A pest control professional probably should be consulted for carpenter ants, the large ants that take up residence in wall voids and woodwork but regularly travel to the outside. Frequently, individuals can be seen traveling along the outside wall from the ground, or up a shrub that is touching the outside wall, and entering the wall through cracks. These ants often remove wood and other substances that pile up in small mounds near the entry sites. These access points should be closed when the colony is treated. Indoors, the colony can be located by listening with a stethoscope for the characteristic noise made by the ants. The most easily heard sounds made by carpenter ants are intermittent clicks. The chewing sounds that they produce are softer and harder to detect. These sounds can be generated during day or nighttime hours (J. Ward, personal communication, 2004). Once its parameters are identified, the colony can be treated with boric acid powder and/or diatomaceous earth, blown into the wall void through a series of strategically placed holes drilled on the inside wall of the area they occupy. Afterward, a professional should fill in the holes.


Greg J. Harrison

Fig 11.2 | Palmetto bugs (roaches) are common in the tropics around dense foliage. Food sources can encourage them indoors in any setting.


Greg J. Harrison

Fig 11.3 | The small (German) roach is much harder to eliminate than its larger cousin. After eliminating food sources, a thorough (steam) cleaning and sealing of cracks is a must.

Following the wall treatment, baits can be used until all signs of the carpenter ants are gone.

COCKROACHES

Cockroaches (Fig 11.2, 11.3) like buildings because they provide all the essential elements needed for survival including shelter (sometimes called harborage), moisture and food. Cockroaches are strongly attracted to water. Although they can survive without food, they must have frequent access to water.[4] They prefer shelter where they can touch the area above them with their antennae, such as areas under cabinets.[4] Cockroach problems almost always indicate the presence of excessive moisture and poor sanitation, as well as access to harborage or shelter. Reducing access to indoors, to moisture and food can prevent infestations. Otherwise, cockroach populations are very difficult to manage because small residual populations can survive in even the most sanitary of environments. Further, residual populations can explode into major problems. The use of chemical pesticide applications can never be regarded as a substitute for either prevention or good sanitation practices. Pesticidal suppression of cockroach populations without a change in the environmental conditions that support them only gives a false and temporary sense of security and may result in chemical resistance in pest populations.[5] Cockroach food can consist of that eaten by people and pets, as well as soiled paper and glue.[13]

Preventing Indoor Access

In a home or apartment, good barrier maintenance is mandatory if cockroaches are to be denied access. This requires elimination of the holes and cracks that cockroaches use to gain entry. Before sealing see Boric Acid below. Access can be denied to cockroaches and other insects through the following measures: installing tight-fitting windows, doors, screens and door sweeps; caulking all exterior and interior cracks and holes in foundations, walls, sills, sinks, gas, water and electrical lines; covering prong holes in electrical outlets at all times; screening air and ventilation vents, open sewer lines and drains. Stainless steel baskets can be used in sink and floor drains to prevent entry of cockroaches from sewers.[13] Even the tiniest crevices must be attended to since an adult roach can hide in cracks as small as one-sixteenth inch and young nymphs can get through a void even smaller.

Preventing Water Access

Leaking faucets and pipes should be repaired. If possible, standing water should be eliminated or covered. Screens should be placed on fish tanks.[13]

Preventing Food Access

Foods should be stored in insect-proof jars with rubber gaskets or tight-fitting lids and/or be kept in the refrigerator. Food and grease should be removed each day from stoves and counters. (A fingerprint left on a countertop by someone who has eaten fried chicken can feed a roach for several days.[5]) Thorough cleanup of food particles on and under tables and counters should be done as soon as possible after meals. Dishes not promptly washed should be immersed in soapy water. Pet food, caches of crackers, nuts or even cough drops should be stored in the refrigerator between meals and dry food should be stored in plastic containers with snap-on lids.[13] Garbage should be placed in a plastic container with a tight snap-on lid.[13] Refrigerators can pose weak links in cockroach management programs because they provide heat, harborage around coils, a constant water supply and hiding places that are difficult to treat.[4]

School for Shaft Squatters

A school cafeteria was being switched to low-risk pest

management and all the appropriate steps had been taken to restrict access, remove sources of water and food, and treat known harborage, but cockroaches were still present. Finally, the pest management manager checked the hollow steel tube legs of the cafeteria tables and found a previously overlooked cockroach harborage. When this site was treated the cockroach problem resolved (W. Currie, personal communication, 2003).

Advantages of Cleaning

Regular vacuuming reduces cockroaches' food sources. Cleaning out clutter reduces nesting and breeding sites, as well as possible food sources such as glue and soiled paper.

Changing Habitat

At the first sign of a cockroach problem, sticky traps can be placed to detect the pests' preferred shelter sites and later for monitoring to determine whether control efforts have succeeded. The traps should be non-toxic and placed along the edges of walls or counters where roaches normally travel.[13] Cracks or crevices in floors or walls can be treated with boric acid before being closed by caulking or other means. See Boric Acid below.

FLEAS

The flea usually infesting USA homes is the cat flea, *Ctenocephalides felis*. Adult fleas most commonly obtain blood meals from mammalian hosts including dogs and people as well as cats. These organisms are parasites requiring a blood meal to reproduce. Fleas spend part of their life cycle on the host animal, but can develop off the host so long as they have access to droppings from the adult flea, since the droppings contain partially digested blood and serve as a food source for immature fleas. Fleas can reproduce indoors where the pet sleeps or rests, as well as outdoors during the warmer months in shaded areas such as crawl spaces under houses. Since fleas are parasites, they do not depend on non-living sources of food to survive.

Preventing Indoor Access

Animals coming into the house for the first time or after being away at a kennel or in transit should be inspected and, if necessary, bathed or vacuumed for fleas before being given access to indoors. This helps prevent seeding of the premises. If people have visited a known flea-infested area, they should change clothes or vacuum their clothes upon entering the house. The car and pet carrier should be vacuumed and washed if necessary. Once the vacuum has been used it needs to be cleaned and treated for fleas or they will escape. Residents can check for fleas indoors by walking around with white socks and looking for small, black, moving forms. Flea problems in cats can be avoided by keeping them indoors or allowing access to a deck, screened or otherwise not in contact with the ground, to prevent the cats' picking up fleas from infested ground sites.

Changing Habitat — Indoors

Washing pet bedding in hot water and drying in a dryer where possible along with vacuuming and disposing of the vacuum contents so that the eggs do not continue to develop inside the vacuum bag can remove fleas. Frequent cleaning of non-carpeted floor areas and anywhere the pet sleeps will help reduce numbers of developing fleas. Furniture and rugs can be shampooed in cases of heavy flea infestation.

A light trap for fleas can be used to locate high population levels and measure control methods. Light traps are cardboard box-like stationary devices consisting of a light source to attract the adult fleas, as well as a sticky surface, which fleas encounter and on which they become caught when they jump toward the light.

Use of a fine-toothed comb (32 teeth per inch) is recommended when combing a pet for fleas. It is best to comb pets on a white sheet to catch any eggs or larvae removed by the flea comb. Fleas should be dropped in soapy water after removing them. When combing is completed, the sheet should be picked up by the corners and taken to be washed. Cats and dogs can be bathed using flea shampoo and, before they are completely dry, combed to remove fleas while the fleas are still immobilized from the bathing procedure. Washing flea-infested clothing or other material in hot water and drying in a dryer will destroy all stages in the flea life cycle. Dogs and cats can be treated with an oral flea control medication, Program,[d] that prevents fleas from developing.

Changing Habitat — Outdoors

If a pet goes outdoors, grass and vegetation should be kept short in places where the pet sleeps or rests. Sunlight can inhibit flea development. If possible, pets and wildlife should be prohibited from having access to areas such as porch crawl spaces, which are permanently shaded and ideal for flea development. A strain of the nematode *Steinernema feltiae* can attack larval stages of fleas[20] (A. Pye, personal communication, 2003). The nematodes are sprayed outdoors on the soil where they can take up residence and help reduce numbers of viable fleas. Products containing this nematode species are commercially available[a] (see Table 11.1 for Gardens Alive!, another resource for this organism). Nematodes are not intended for use as indoor flea controls.

INSECT INFESTATION OF FOOD

Larvae of beetles and moths and grain mites can infest stored grain or birdseed. Some insects lay their eggs in the grains in the field and therefore are present as eggs in seeds and even flour. The heat associated with processing the grains usually eliminates the eggs from pellitized and extruded products. Many of these insects can crawl down the threads of an ordinary screw-cap jar, or penetrate a single paper or cloth bag, so it is necessary to use a glass jar with a rubber gasket or containers with tight-fitting lids to exclude them. For storage, bulk seed should be divided into 5- and 10-lb. containers so only small amounts will be lost if some of it becomes contaminated. One solution is to use 5-gallon buckets of the type used by bakeries. These buckets usually have tight-fitting plastic or metal lids.[13] Food should be stored in a cool and dry environment. Most pests will not infest products if the humidity is below 6%.[13] An inexpensive instrument can measure the humidity in the storage area. Alternatively, grain and seeds can be stored in the freezer. Food spills should be promptly cleaned up.

Preventing Indoor Access

At the time of purchase and/or when they are first brought into the home, foodstuffs should be inspected for possible mite infestation in the form of web-like structures on or around the material.[13]

Changing Habitat

Bacillus thuringiensis (*B.t.*) is a commercially available bacterial species that causes disease in certain insects by acting as a stomach poison. *B.t.* can be applied to stored grains for protection against the larval form of the Indianmeal moth. These insects seldom feed below a surface layer more than 4 inches deep in a grain bin, so *B.t.* can be effective if applied to the surface.[13] See Low-Risk Agents below.

Parasitoids such as the tiny *Trichogramma* can kill the egg stages of the moth. This approach could be very helpful for controlling long-standing infestations in the home or aviary that have proved resistant to the normal physical or sanitary methods already described.[13]

All stages of the Indianmeal moth are killed by freezing at 0° F (-17° C) for 4 days. A pheromone trap for monitoring is commercially available. Once infestation is found, the material can either be placed in a freezer or heated to kill pests. The material should then be discarded. Seriously infested products with webs are often secondarily contaminated with toxic levels of molds. Therefore, they should not be fed after freezing or heating.

Greg J. Harrison

Fig 11.4 | Spiders are often misunderstood and killed. Moving them, removing their webs and reducing the insect attractions that cause spiders to be attracted contributes to insect control in the long run.

CLOTHES MOTHS

The larvae of clothes moths attack various types of household material including blankets, upholstery and carpets. Clothes moth larvae avoid light and require material stained with food or sweat to provide the proper nutrients to grow. These moths do not attack clean clothes or fabrics stored in sealed containers. Carcasses of dead mice in wall voids can attract moths. Unoccupied bird or bat nests in attics can be a source of clothes moths and other insects.

Infestation by clothes moths can be managed chiefly by prevention. Clothes should be thoroughly cleaned before storing in tight containers. Vacuuming around the home and furniture is a way of controlling moths. Exposure to heat is a direct way to kill clothes moths. If the temperature can be maintained at or above 100° F (37° C) for several hours to days, all stages of the insect can be killed. Attics can often reach such temperatures in the summer. Extremely cold temperatures (sealed bags in a freezer) for several days also can control clothes moths. Traps to monitor for the presence of clothes moths can be hung in the closet as an early warning device.[13]

YELLOW JACKETS

Yellow jackets are a form of wasp that usually nest in the ground, but they can at times nest in walls of homes. Indoors they can actually excavate through plaster and in rare cases penetrate into adjacent rooms. Professional control using low-risk methods and performed at night to minimize disturbing the insects can be effective. When proper precautions are taken so as to not endanger residents, injecting a spray of pyrethrins and diatomaceous earth and then closing the access to the outdoors can be effective in eliminating the colony from a wall void. See also Traps to follow.

SPIDERS

The presence of spiders can indicate that there are flies in the house. Spiders are useful and should be removed to outdoors whenever possible (Fig 11.4). Spiders can be removed to the outside mechanically with a glass and a piece of stiff cardboard or by using a vacuum that will usually kill most spiders. Once the flies have been controlled and the spiders removed mechanically or by vacuum, they can be kept out by the same exclusion methods as those used for cockroaches and ants, and by caulking and using screens and door sweeps. There is usually no need to use a chemical spray.

MICE

Adult house mice can weigh from 0.5 to 1.0 oz (15 to 30 g). House mice breed year-round under optimum indoor conditions. Outdoors, these mice are seasonal breeders in the spring and fall. They are active primarily at night but also can be seen moving about during the day. Mice thoroughly investigate any change in their territory.[4] They can squeeze through openings slightly more than one-quarter inch in diameter. They prefer cereals to other food items. Management and prevention of infestations by house mice is a three-part process: mouse-proofing, sanitation and population reduction indoors with traps. When a mouse population already exists, some kind of lethal control is necessary. Otherwise, the reproductive capability of the mice and their remarkable ability to find food in almost any habitat will keep their populations steady or increasing.[4] The practice of using poisons on mice may lead to dead mice in wall voids, resulting in a moth infestation that can spread to clothing. Clothes moths are often attracted to the carcasses of dead animals. Other insects and bacteria can result from poisoned, decomposing mice; and pets, including pet birds, can be at risk from feeding on the dead mice or the rodenticide used for rodent control.

Preventing Indoor Access

Mice are found around human housing, especially in rural habitats. During the autumn and winter, they readily move into buildings that are not made rodent-proof. All holes must be sealed to limit the movement of mice into and through a building. Holes in foundation walls for pipes, utility lines and vents should be plugged with quarter-inch hardware cloth, copper mesh or steel wool.[4] Doors and windows should fit tightly and be caulked if necessary.

Preventing Food Access

Practicing good sanitation is a way to reduce the food supply. It enhances the effectiveness of traps and makes detection of a mouse infestation easier. Bulk foods should be stored in mouse-proof containers or rooms. Stored materials should be kept away from walls and off the floor.

If corn gluten herbicide is being used, it should be stored over the winter in a container that mice cannot penetrate. Any birdseed should be stored in tight containers or kept under refrigeration.

Changing Habitat — Indoors

Types of Traps

When used correctly, snap traps are very effective for controlling mice. Traps must be set in the right places, in high numbers and in the right position (see below) or mice will avoid them.[4] If used properly, snap traps inflict less suffering than do glue traps and possibly the so-called humane box traps, since if animals are released in a distant territory they will likely be attacked as aliens by resident mice.

Setting Traps

The territory of mice rarely extends farther than 30 feet from the nest, and more often is about 10 feet. If mice are sighted throughout a building it usually means there are numerous locations where traps should be set. Traps should be placed wherever there are obvious signs of mice. Probably the biggest mistake made when trapping mice is not using enough traps.[4] Traps should be set every 3 to 6 feet in prime mouse habitat. They should be set in a three-dimensional sphere about 10 feet in diameter around the signs.

Baits

Choosing good baits for mice and rats increases the effectiveness of mousetraps. Peanut butter (the chunky kind) is considered one of the best baits; food baits should be fresh to be effective. A cotton ball, which females like to use for nest material, also can be used. The cotton should be tied securely to the trigger.

Outdoor Low-Risk Pest Management

WEED MANAGEMENT

Synthetic chemical pesticides do not need to be routinely used against most types of weeds.[11] For poison ivy, digging out the root and/or applying a 9% solution of vinegar (used for pickling) are effective. For other broadleaf weeds, corn gluten meal (Fig 11.5) is an effective pre-emergent herbicide and can be used in spring and fall. Herbicidal soap is available for spot-treating weeds. On paved surfaces, boiling water, steam, a flamer or other heat source can be used to remove unwanted vegetation.

Greg J. Harrison

Fig 11.5 | Corn gluten meal serves as a pre-emergence herbicide and then deteriorates into a soil nutrient supply source.

INSECT PESTS OF THE LAWN AND GARDEN

In many instances, the need for chemical insecticides can be replaced with management strategies and natural enemies of pests. Biological controls are commercially available. Beneficial insects also can be encouraged by providing certain plants in the garden needed by the insects for stages of their life cycles when they are not preying on pests (see Table 11.1 for RCC Web site for further details and information on grubs in the lawn; see also reference 16 and Suggested Reading "If You Plant It They Will Come").

MOSQUITOES

Mosquito Management

In most naturally occurring streams or even backyard ponds, living organisms such as fish, amphibians and insects feed on mosquito larvae and prevent the emergence of adults. Biological control of adults includes birds and bats.[3] In artificially occurring standing water, the mechanical emptying of containers every 4 to 5 days is the best prevention for adult mosquito emergence. When water cannot be changed due to high volume and/or there is not a system with naturally occurring biological controls, *B.t.* preparations (see below) can be used to kill off the developing mosquito larvae in standing water, as will dish soap or a thin coating of olive oil.

It is most important to clean out rain gutters so that they empty freely after a storm. Habitat should be provided for wildlife such as birds, bats and dragonflies (Figs 11.6, 11.7) that act as biological controls by consuming adult mosquitoes. For special outdoor occasions, there are mosquito-trapping devices[b], some quite successful, that generate carbon dioxide, heat and synthetic attractants.

Greg J. Harrison

Fig 11.6 | The beneficial dragonfly acts as flying insect control by consuming large numbers of pest insects.

Greg J. Harrison

Fig 11.7 | Swarms of dragonflies are seen seasonally during the height of the pest insect season.

Effective natural mosquito repellents include garlic oil, catnip and botanicals such as mint, citronella, geraniol, linalool, elemol (from the Osage orange tree), etc, used singly or in combination. There also is the synthetic chemical repellent DEET (best used at 30% concentration or less, applied to clothing, not to skin).

Mosquito repellent products can be sorted into three categories according to where they are applied: those placed on the clothing or skin; those dispersed into the air near people by a battery-powered device; and those applied to the ground area-wide, as granules or liquid or as liquid sprayed at ground level. More research is needed on the effectiveness of repellents in various delivery systems, even for active ingredients of the natural, non-synthetic type. The area repellent products require more research into their potential environmental hazards. Could they, for example, adversely affect beneficial insects and other life forms? See Garlic Juice Products below.

Low-Risk Agents

BACILLUS THURINGIENSIS (B.t.)

B.t is a spore-forming bacteria named for a town in Germany. Its insecticidal properties were first found in 1911 in diseased flour moths. *B.t.* contains a protein (endotoxin) that is toxic for many caterpillars and other insect larvae. Varieties have been discovered that affect a relatively narrow range of hosts, specifically mosquitoes, as well as certain moths and beetles. *B.t.* products in general lack toxicity for non-target species and the natural insect enemies of pests. *B.t* has been granted an exemption from establishing a tolerance for use on food crops due to its low toxicity for humans when taken by mouth.[13]

However, spray preparations of *B.t.* products have apparently been associated with adverse reactions when inhaled by certain sensitive individuals. Although in most cases *B.t.*would not be recommended for home use indoors against moth infestation, it is available commercially for that situation as well as for outdoor use against mosquito larvae, gypsy moth larvae and other garden pests.[13] *B.t.* preparations used for mosquito control include dunks and granules intended for use in standing water.

BORIC ACID

Boric acid is a crystalline material derived from borax, (a combination of sodium, boron and oxygen mined from the earth). It acts as a stomach poison when ingested by the cockroach or ant causing the insect to starve, which can take 5 to 10 days. It can be toxic to mammals when ingested in high doses. Boric acid does not represent a volatile danger in its powder form. It is considered "virtually vaporless."[13] It must be kept away from food, children and pets including pet birds to prevent oral consumption. Those applying boric acid powder should wear a dust mask, gloves, long sleeves and eye protection. It is considered a low-risk alternative when used indoors and placed in areas not accessible to pets or people, provided that precautions are taken and/or the formulation does not include another active ingredient or inert component that may volatilize and induce adverse effects through inhalation or dermal contact. If boric acid is applied to cracks, crevices and wall voids, the areas should subsequently be sealed by caulking or closed off by another means so that it will not present a problem to non-target individuals.[13] Boric acid remains active for months and cockroaches appear not to have developed any resistance after 50 years of use. Boric acid can be toxic under conditions of high exposure, so it is important to limit access.

CORN GLUTEN MEAL

The pre-emergent weed suppression qualities of corn gluten meal (CGM) were discovered in 1987. CGM, a waste product of corn milling that has been used in animal feed, is 60% protein and 10% nitrogen by weight. Protein is the active ingredient in CGM. It inhibits the growth of roots at the time of a plant's germination.

Timing of the application of this pre-emergent herbicide is very important. The precise moment in the year most advantageous for CGM weed control varies with growing conditions. The local cooperative extension service can be very helpful with advice on when best to apply CGM. For example, in the Northeast, to manage crabgrass in a lawn situation in the spring, CGM should be applied at the time of the crocus bloom and before the forsythia bloom takes place. If applied later, after the weeds have already germinated, the nitrogen in CGM could help the emerging weeds thrive (M. Talbot, personal communication, 2004). See Table 11.1 for Gardens Alive!, a resource for CGM. The use of CGM as an herbicide is patented by Iowa State University.

DIATOMACEOUS EARTH

Diatomaceous earth is obtained from the fossilized silica shell remains of diatoms, marine plankton. It has abrasive, sorptive and desiccant properties. It can be used alone or in conjunction with boric acid and/or pyrethrins (naturally occurring pesticides from chrysanthemums — see below). As with any dust preparation, a mask, goggles and gloves should be worn by the applicator to protect the eyes, respiratory system and skin.[13]

NEMATODES

Nematodes are tiny worms. Most species live in the soil and a few species are internal parasites of higher organisms including man. When fleas are replicating outdoors, certain species of nematodes may be effective in inhibiting their development[a].

PARASITOIDS

See Clothes Moths.

PYRETHRINS

Pyrethrins are short-lived biological pesticides isolated from chrysanthemums. They act as neurotoxic agents and have the ability to paralyze insects. They can be used in conjunction with diatomaceous earth or silica aerogel (see below). They are usually formulated with the agent piperonyl butoxide to prevent metabolism of the pyrethrin. Untoward allergic-type reactions in people or pets may take place when pyrethrins are used. Pyrethrins have very low acute toxicity for most

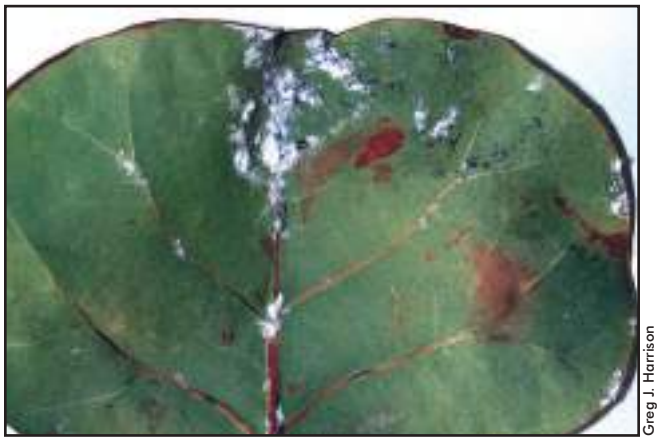

Greg J. Harrison

Fig 11.8 | Leaf mold on this sea grape plant is a result of mites and the soil in the pot is not supporting healthy plant growth. Repotting and an application of liquid dish soap are considered an effective control.

mammals and birds, but they are highly toxic to fish.[2]

SILICA AEROGEL

Silica aerogel is an amorphous, non-abrasive, chemically inert, dust-like material. It results from the reaction of sodium silicate and sulfuric acid. Silica aerogels can absorb water and oil. When used as insecticide, it is believed to absorb the waxy protective coating on an insect's cuticle — an action that can result in dehydration and death of the insect. Silica aerogel particles are capable of irritating human lungs and eyes, so a dust mask, goggles and gloves should be worn during application.[13]

SOAPY WATER

A soap solution reduces the surface tension of water; as a result, insects are unable to float and they drown. Some plant pests are deterred by the use of soapy water applications (Fig 11.8). (See Ants - Preventing Access to Food).

GARLIC JUICE PRODUCTS

Neither the author nor the editors have any experience with garlic products. A veterinarian in the Netherlands states that one product[c] is very effective in controlling mosquitoes in the yard (J. Hooimeijer, personal communication, 2003). The smell is a potential problem for people who are sensitive to garlic. Originally garlic was developed for control of pest insects on organic vegetable crops. The farmers using the product found mosquitoes were no longer a problem in the fields for rather long periods of time after its use on vegetables.

TRAPS

A variety of traps are commercially available including the following:

- A yellow jacket outdoor trap for use away from the area where people are gathering, consisting of one-way ports and baited with cat food, sugar-containing cola drinks or meat. After yellow jackets enter the trap, placing the trap in the sun will kill them. This should be used only during the late summer when yellow jackets are aggressive toward people and their food. Traps can be purchased from local hardware stores.
- Ant and cockroach indoor traps have attractants and insecticides at sufficiently low concentrations for ants and cockroaches to carry the poison back to members of the colony without endangering other life forms.
- A mosquito outdoor trap[b] uses propane to generate carbon dioxide and heat. In addition, certain models may contain specific attractants such as octanol. These traps are designed to lure and kill only insects that seek a blood meal. They spare the non-biting forms.
- Moth traps use pheromones.

Products Mentioned in the Text

a. Scan Mask, Biologic Co, www.biologicco.com
b. Mosquito Magnet, www.mosquitomagnet.com
c. Garlic Spray, www.garlicbarrier.com/mosquitobarrier.html
d. Program - Lufenaron - Novartis

References

1. Bichsel P, Lyman R: Pyrethroid toxicity in felines: Prognosis good to guarded. DVM Magazine, June 2003.
2. Briggs, SAB and the staff of Rachel Carson Council: Basic Guide to Pesticides Their Characteristics and Hazards. Hemisphere Press, 1992.
3. Christensen B: In defense of bats. Letter, JAVMA, v.222, #10, May 15, 2003.
4. Currie W: How Integrated Pest Management Can Reduce Your Risk From Exposure to Pests and to Highly Toxic Pesticides. Proc Seminar Three—Ants, Cockroaches, Rodents and Turf. University of the District of Columbia, Washington, D.C.,1992.
5. Currie W: Integrated Pest Management Procedures Manual. International Pest Management Institute, August 2002.
6. Gainer JH, et al: Adverse effects in human beings, dogs and cats associated with the use of flea and tick products. Pesticides, People and Nature 1(2):135-142, 1999.
7. Grossman, Joel ESA 2002 Annual Meeting Highlights-Part Two. *In* The IPM Practitioner, V. XXV, #5/6, May/June 2003.
8. Health-Based Integrated Pest Management (IPM) for Indoors. Rachel Carson Council, Inc, 2000.
9. Hoffman DJ: Wildlife Toxicity Testing. In Handbook of Ecotox 2nd ed. CRC Press, 2003.
10. Katz H: Li'l Hummer, Non-toxic Pest Control. IPM Practitioner, XVI, #4, April 1994.
11. Lerner J: Garden upkeep is good for the mind, body and soil. The Washington Post, April 19, 2003.
12. Mohan R: Dursban Toxicosis in a Pet Bird Breeding Operation. Proc Assoc Avian Vet, 1990, pp 112-114.
13. Olkowski W, Daar S, Olkowski H: Common Sense Pest Control: Least-toxic solutions for your home, garden, pets and community. The Taunton Press, 1991.
14. Payne P, Dryden M: Feline flea control. *In* Clinical Edge, Thomson Veterinary Healthcare Communications (under a grant from Pfizer Animal Health), 2003.
15. Post D: The Other Road to Flea Control. Rachel Carson Council, Inc, 1994.
16. Post D, Collins M: EcoHeal IPM for Outdoors. Rachel Carson Council, Inc, 2000.
17. Prohl A, et al: Activities of an Environmental Analysis Van in the German Federal State Schleswig-Holstein. Environ Health Perspectives, V. 105, #8, Aug 1997.
18. Quarles W: New Flea Control Products—Blessing or Curse? Common Sense Pest Quarterly, VXIII, #2, Spring 1997.
19. Ramelmeier J, Post D: A Silent Killer of Birds in Our Care: Heat-Generated PTFE Fumes. Rachel Carson Council, Inc, 2002.
20. Silverman J, et al: Infection of the cat flea *Ctenocephalides felis* (Bouche) by *Neoplectana carpocapsae Weiser*. J Nematology 14:394-397, 1982.
21. Talbot M: Principles of Ecological Landscaping; A Primer on Environmentally Sensitive Design, Installation and Care of Our Lawns and Landscapes. Michael Talbot & Assoc, Inc, 2003.
22. United States Environmental Protection Agency: Region 8 Pesticides/Toxic Chemicals & Children, 2001.

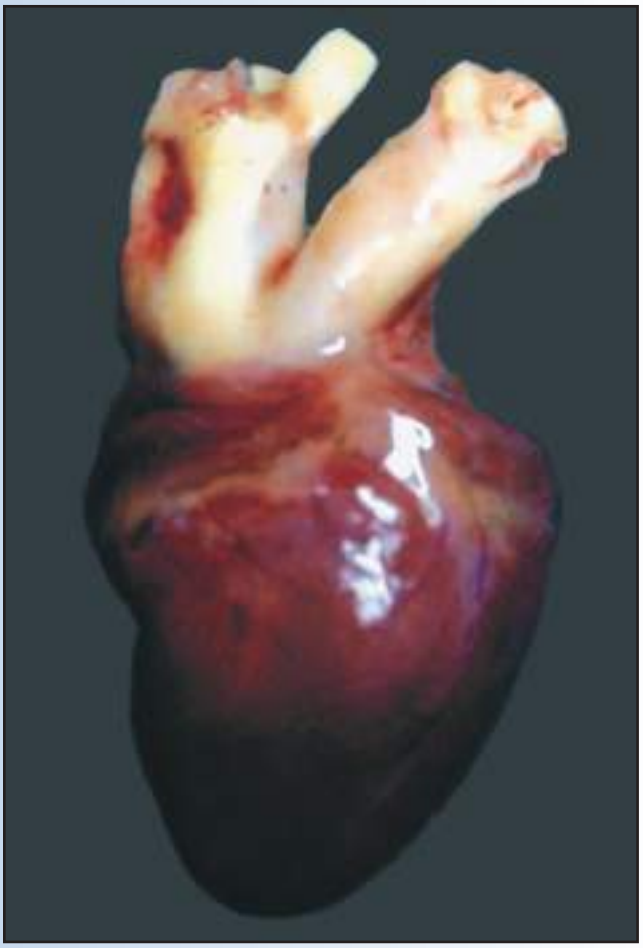
Suggested Reading

1. **If You Plant It They Will Come**
   This brochure available from RCC lists vegetation designed to attract and nurture beneficial insects.
2. **Questionnaire for Interviewing Potential Pest Control Specialists**
   This handout available from RCC was designed to help individuals find pest control specialists that favor low risk methods of pest control.
3. **What's Wrong with My Bait?**
   This article from the BIRC publication Common Sense Pest Control provides hints on how to use baits safely and successfully. It is available from BIRC or RCC. See Table 11.1 for contact information.

CHAPTER
12

Evaluating and Treating the Cardiovascular System

MICHAEL PEES, Dr med vet, German vet spec for birds/reptiles;
MARIA-ELISABETH KRAUTWALD-JUNGHANNS, Prof Dr med vet, Dr habil, Dipl ECAMS, German vet spec for birds/reptiles;
JENS STRAUB, Dr med vet, German vet spec for birds/reptiles



Anatomical Considerations

The avian heart is located within the cranial part of the thoracoabdominal cavity parallel to the spine in close proximity to the sternum. No diaphragm is present and the apex of the heart is surrounded by the liver lobes. The pericardium covers the heart and normally encloses a small quantity of fluid within the pericardial space.

The anatomy of the avian heart is similar to the mammalian heart, although some morphological peculiarities exist. As in mammals, it is four chambered and functionally divided into the right and the left sides each consisting of an atrium and a ventricle. The right ventricle is sickle-moon shaped and surrounds the left ventricle. The wall thickness of the ventricles changes from the base to the apex of the heart (for measurements see Table 12.6). The triangular right atrioventricular (AV) valve is muscular and unlike the right atrioventricular valve in the mammalian heart, does not contain *chordae tendinae*. Contraction assists emptying of the right ventricle during systole. In some species, the right cranial and the caudal *V. cava* form a *Sinus venosus* that is separate from the right atrium. In contrast, the ascending aorta curves to the right in birds rather than the left side of the body in mammals.[2,25,26,41,44,45,53]

Diagnostics

Diagnosis of cardiovascular diseases in cage and aviary birds is complicated by several factors. Only a few systematic investigations on post mortem diagnosis of cardiac diseases have been conducted.[4,24] All descriptions of ante mortem diagnostics are case reports, based on individual experience. Documented normal reference values are rare. Clinical signs of cardiac disease in birds are often nonspecific and may be accompanied by other concurrent disease conditions disguising the clinical picture. Diagnostic techniques in living birds are limited by the size of the patients and high heart rates. Fortunately, within recent years, modern imaging techniques (like echocardiography) have been evaluated in birds and initial reference values for the assessment of cardiac function are now available. Since these techniques require special equipment and experience, they are not commonly performed in practice. Nevertheless, the practitioner should be aware of the diagnostic potential of ultrasound and other imaging techniques in avian cardiac disease.

Birds with acute circulatory problems have to be handled as emergency cases. Handling should be kept to a minimum and diagnostic procedures be carefully selected. The bird should be maintained in an upright position to prevent circulatory failure. Echocardiography may therefore be less stressful in these patients than radiographic examination.

CLINICAL EXAMINATION

The clinical diagnosis of cardiovascular disease in living birds can be difficult. There is no palpable pulse in birds. In addition, auscultation, an important standard technique in mammals, is difficult to interpret in birds.

Birds suffering from cardiac disease are often presented to the veterinarian with a history of weakness and lethargy. In some cases, cardiovascular failure can be suspected on the basis of bluish discoloration of the periorbital skin (especially in African grey parrots) and abdominal distension. Nonspecific symptoms like dyspnea and exercise intolerance may also lead to a tentative diagnosis of a cardiac problem and provide an indication for further diagnostic procedures.

STANDARD RADIOLOGY

Radiology is a commonly performed and well established imaging technique in avian medicine. It may (often by chance) disclose signs of cardiovascular disease and indicate the need for further diagnostics, especially echocardiography. Position, size and shape of the heart and other internal organs as well as the radiodensity of the large vessels should be assessed.

Alterations of cardiac shape and size are often seen as an enlargement of the heart silhouette. This can be caused by different etiologies (eg, hypertrophy, dilatation, pericardial effusion, aneurysm, inflammation or neoplasia). Radiographic differentiation between these etiologies is difficult. In cases of an existing pericardial effusion, the conventional radiograph may reveal cardiomegaly with an irregular cardiac silhouette.

An increased radiodensity of the large heart vessels can be seen on the ventrodorsal projection as roundish shadows superimposed on the base of the heart and in the lateral projections as an enlarged and radiodense aortic shadow. These findings may indicate atherosclerosis, whereas their lack does not prove the absence of pathological alterations.

Furthermore, radiographs may reveal secondary changes in other organs, such as increased radiodensity of the lungs, enlargement of the hepatic silhouette, or ascites in the case of congestive heart failure (see Fig 12.10). Ascites can also mask the radiographic detail of the thoracocoelomic cavity and the air sac shadows may be decreased and/or displaced.

In birds, measurements of the length and width of the cardiac silhouette on radiographs are limited. Initial examinations have been performed in Canada geese and psittacines.[12,46] Measuring the length of the cardiac silhouette is often complicated by superimposition of the apex of the heart on the liver on both the lateral and ventrodorsal projections or the ventral aspect of the heart on the sternum on the lateral projection. Measurements of the width of the cardiac silhouette (see "h" in Fig 12.1) should be performed on the ventrodorsal radiograph with exact superimposition of the keel and the spine. To assess the width of the cardiac silhouette, the measured value should be taken at the level of the maximum width of the cardiac silhouette, and compared to the maximum width of the thorax (see "t" in Fig 12.1). The sternal length should be taken on the radiograph or on the patient using a standard sliding calliper. In medium sized psittacines (approximately 200 to 500 g) the width of the cardiac silhouette should be approximately 36 to 41% of the length of the sternum or 51 to 61% of the width of the thorax respectively. There is a strong correlation between the width of the cardiac silhouette and the width of the thorax.[46] In Canadian geese the width of the cardiac silhouette is 47 to 57% of the width of the thorax.[12] This study also showed that the movements of the thorax due to respiration are not as important as might be expected.

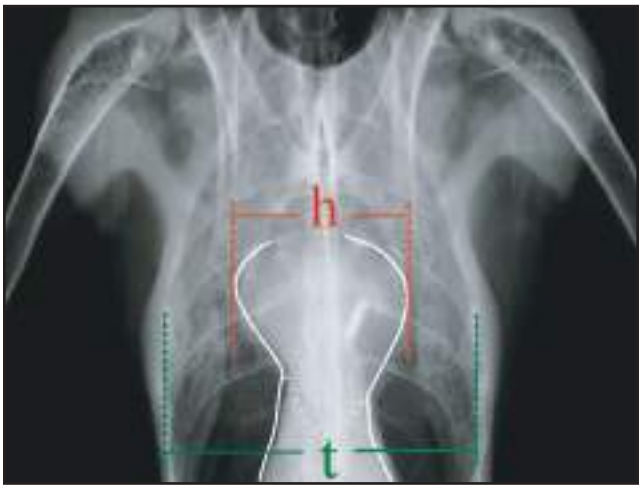


Fig 12.1 | Schematic illustration of the distances to be measured for the evaluation of the avian heart on ventrodorsal radiographs (h = maximum width of the heart size, t = width of the thorax at the level of the maximum width of the cardiac silhouette (microchip can be seen in the left pectoral muscle).

ANGIOCARDIOGRAPHY

Although few reports about the use of angiocardiography in birds exist, the authors' initial experiences show a high potential value for its use in diagnosing cardiac problems. Angiocardiography cannot replace echocardiography but it might give additional information, especially in birds with insufficient detail demonstrated on echocardiographic examination. Examination of the heart vessels is another indication for angiocardiography. In one report, an aneurysm of the right coronary artery was demonstrated in a white cockatoo (*Cacatua alba*) using angiocardiography.[52]

Angiocardiography should be performed under anaesthesia. A venous catheter is placed within the jugular or basilic vein. Iodinated contrast media (eg, Iopamidol, 510 mg/ml adequate 250 mg iodine per ml) is used according to mammalian angiocardiography, but the dosage in birds (2-4 ml/kg of pamidol ie, 1,020-2,040 mg body mass) is twice as high as commonly used in mammals. Administration rate in medium sized birds should be approximately 1 to 2 ml of contrast medium per second.

Due to the rapid heart rate in birds, assessment of contractility may be difficult to evaluate, but hypertrophy (ventricles), dilatation (ventricles, atria), stenosis (vessels, valves) and aneurysm (vessels) can be detected (see Fig 12.2).

ECHOCARDIOGRAPHY

In veterinary medicine, echocardiographic examination has become a very important diagnostic tool, and is indicated for assessment of cardiac function and the structure of the heart. For a long time, due to anatomical peculiarities (especially the position of the airsacs),

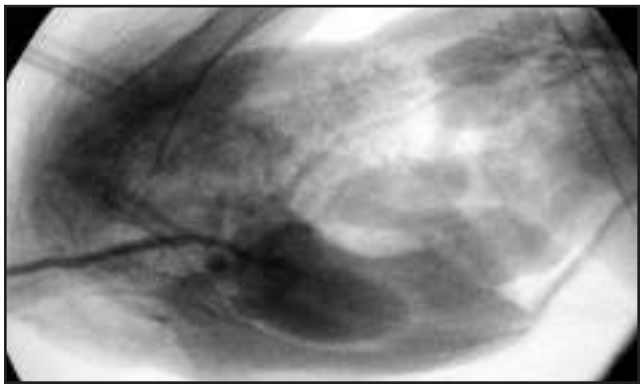


Fig 12.2 | Angiocardiography: filling of the heart in a common buzzard (*Buteo buteo*).

echocardiography has been considered inaccurate and difficult in birds. Bone and air block the passage of ultrasound waves. Standardized views and protocols for mammals recommended by the American College of Veterinary Internal Medicine[50] cannot be used in birds and comparison of the measurements with those in mammal or human medicine are not valid.[15] However, in recent years, case reports regarding the use of echocardiography for diagnosis of cardiac disease in birds have demonstrated the potential of this diagnostic procedure.[29,30,47,52] Initial studies have been conducted and standardized protocols for echocardiographic examination in avian patients have been established.[15,34,46,47]

Improved technology has produced ultrasound equipment that is effective and affordable for avian echocardiography. Parameters of these machines should include
1) minimum 100 frames/second
2) Doppler function
3) microcurved or phased array probes
4) minimum 7.5 MHz frequency

On avian patients that are tolerant of restraint, echocardiography can be performed awake; stress-sensitive birds should be anesthetized.

Because the food-filled gastrointestinal tract might be interposed between the scanner and the heart ideally the GI-tract should be empty. Therefore it is recommended to fast the bird before ultrasound examination. Granivorous birds should be fasted for about 2 (small psittacines) to 12 (pigeons) hours. Raptors should generally be fasted for a longer period.

Echocardiographic measurements should be done in relation to the patient's size. Two parameters should be determined: first, the bird's weight (although this might be misleading in seriously ill birds that are emaciated), and second, the length of the sternum, taken on the radiograph or on the patient using a standard sliding calliper. Simultaneous ECG recording and triggering is

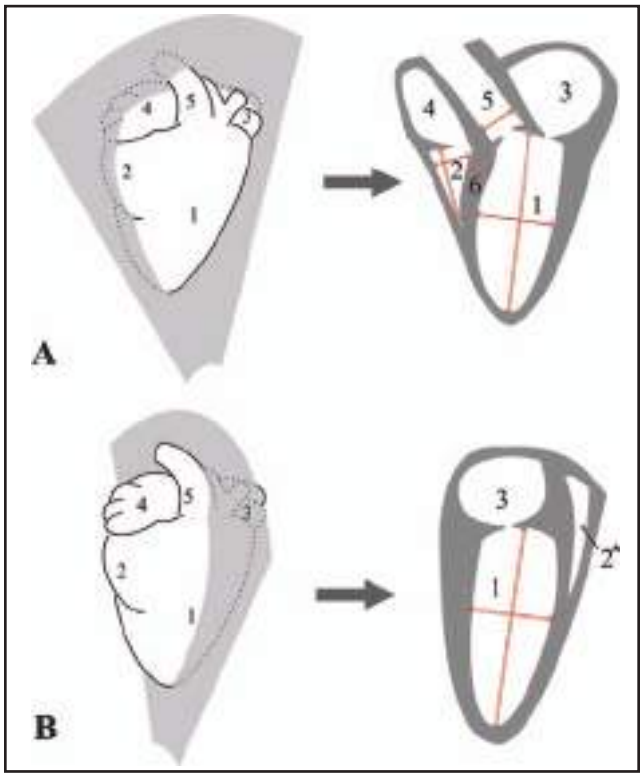


Fig 12.3 | Two-dimensional echocardiography, schematic views: ventromedian approach, horizontal (a) and vertical (b) view. Important measurement points, values see Tables 12.1 and 12.2. (1 = left ventricle, 2 = right ventricle, 3 = left atrium, 4 = right atrium, 5 = aortic root, 6 = interventricular septum, * = no assessment possible in B view)

recommended to allow measurements at defined stages of the heart cycle. For the examination, birds should be held in an upright position in order to minimize stress and its influence on the cardiovascular system.

Because of the avian anatomy, suitable echocardiographic windows to the heart are limited. There are two possibilities: the ventromedian and the parasternal approach.[15] In psittacines and raptors, the ventromedian approach is routinely used. With this approach the homogenous liver tissue serves as an acoustic window. The scanner is placed on the midline directly behind the sternum and the beam plane is directed craniodorsally. In birds with sufficient space between the last ribs and the pelvis (eg, pigeons), the parasternal approach can be used. The transducer is placed on the right side of the body, since in the left part of the thoracoabdominal cavity, the grit-filled ventriculus is present and often makes visualization of the heart impossible. The probe is placed behind the last ribs and the plane is directed craniomedially.

With the ventromedian approach, two longitudinal (comparable to "long axis" in mammal echocardiography) views of the heart are obtained (Fig 12.3). The vertical view (corresponding to the "two-chamber view") shows the heart lying on the inner surface of the sternum. The horizontal view ("four-chamber view") is produced by a counter-clockwise 90 degree rotation of the scanner.

B-mode-echocardiography (2-D-echocardiography)

Due to the fact that only long axis views are possible with the ventromedian approach, M-Mode echo can not be used in birds to evaluate contractility and wall thickness. Therefore, measurements have to be taken on the 2-D-echocardiograph.

Longitudinal Vertical View ("Two Chamber View")

The vertical view shows the left ventricle and the left atrium (which is often not completely distinguishable from the surrounding tissue) as well as the left atrioventricular valve. Sometimes border areas of the right ventricle may also be seen next to the total reflexion of the sternum (Fig 12.4) but cannot be used for assessment. In this view, artifacts caused by reflexions of the sternal bone may produce a second "heart" lying outside the thoracoabdominal cavity (mirror picture artifact).

Longitudinal Horizontal View ("Four Chamber View")

In the four-chamber view, both ventricles, the interventricular septum, both atria, the atrioventricular valves and the aortic root with the aortic valves can be visualized (see Fig 12.4). In this view it is difficult to outline the borders of the atria.

Measurements in 2-D-echocardiography

Measurements have to be taken from 2-D images, since there is no possibility for a suitable cross section for M-Mode technique. Additionally, the images are of very small structures. Therefore the interpretation of measurements is limited and small changes in size may not be detected with the current techniques.

To measure specific cardiac parameters, the position of the transducer must be adjusted until the maximum expansion of the chamber is visible. Measuring points are demonstrated in Fig 12.3. All parameters should be measured in systole and diastole at the point of the widest distance using the inner edge method.[56] Measurements of the left ventricle are possible in both views, but are easier to obtain in the vertical view. In psittacines, there is a strong correlation between the sternal length/the body mass and the length of the left ventricle (LVL); the expected length can be estimated with the formula LVL = 0.5 + 0.33 x sternal length.[32] Measuring the length of the right ventricle is more difficult because it is visible as an acute-angled triangle. Additionally, the muscular right atrioventricular valve is — due to its motility

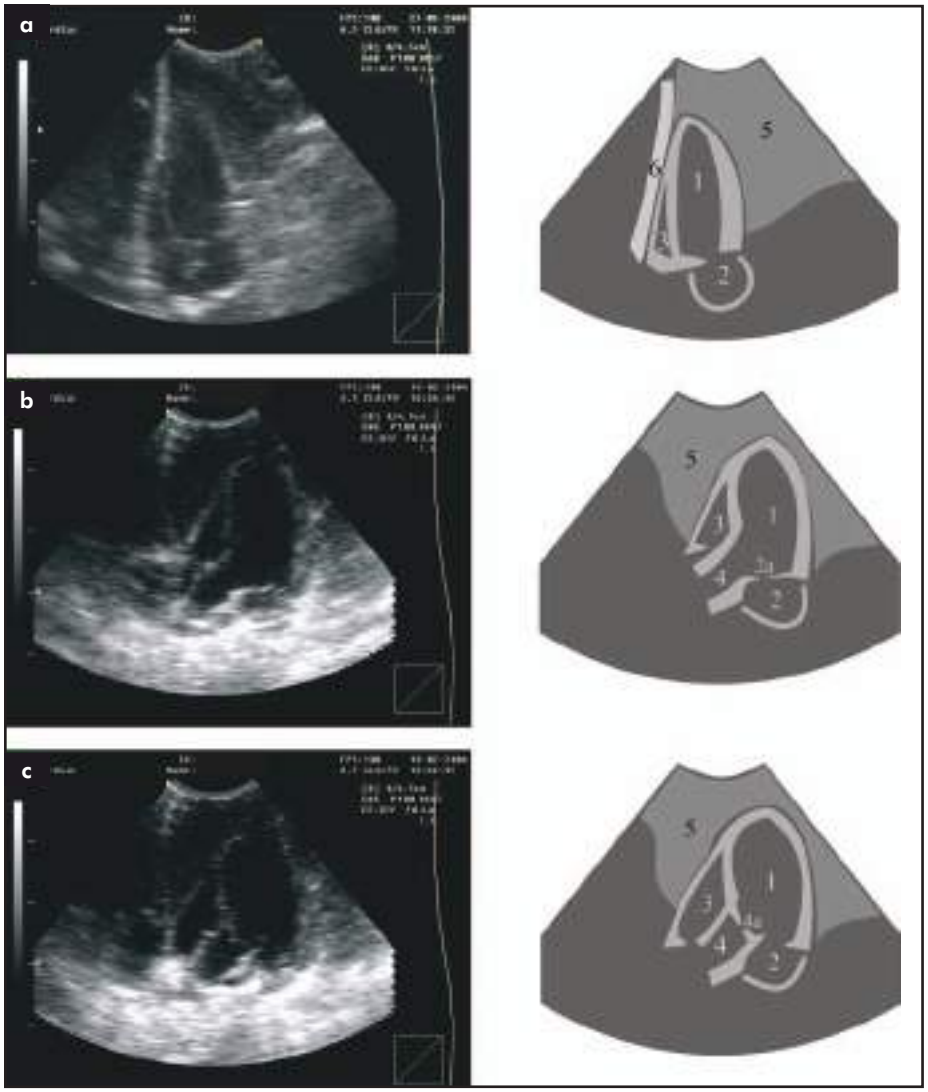


Fig 12.4 | Two dimensional echocardiography, normal hearts. a: vertical view, blue fronted Amazon (*Amazona aestiva*), b: horizontal view, systole, carrion crow (*Corvus corone*), c: horizontal view, diastole, carrion crow (*Corvus corone*). (1 = left ventricle, 2 = left atrium, 2a = left atrioventricular valves, 3 = right ventricle, 4 = aortic root, 4a = aortic valves, 5 = liver tissue, 6 = sternal reflexion).

— an imprecise margin for right ventricular measurement. The outer walls of the heart are often difficult to distinguish from the surrounding tissue. The thickness of the interventricular septum may give information about ventricular hypertrophy. Measurements of the atria are imprecise and therefore of limited use. The importance of atrial contraction for blood flow into the ventricles is limited, therefore assessment may not be important.[42]

Calculations for Functional Assessment (Tables 12.1-12.3)

On the basis of the measurements obtained, the fractional shortening (FS) can be calculated. This is one functional parameter for evaluating the capability of ventricular contraction. It is calculated as the relative reduction of the width of the transverse left ventricular diameter during systole in relation to the diastolic diameter (%FS = (diastole - systole) 100/diastole). The values are different than those in mammals, which might be due to anatomical peculiarities (see above). Contractility of the right ventricle is significantly higher than the value found for the left one.

The relationship of the width of the ventricle to the length can give information about dilatation. For psittacines, a relatively constant value (systolic 0.33, diastolic 0.40) (see Table 12.3) has been noted.[32,33,34]

Volume calculations, based on simplified models of the chamber volume of the left ventricle, have not yet been documented as diagnostic.[34]

Preliminary reference values for morphometry and assessment of cardiac function have been established for psittacines,[32,33,34] birds of prey[3] and pigeons[15] (Tables 12.1-12.3).

Doppler-echocardiography

To date, there only a few reports of Doppler echocardiography in birds. The principles are the same as in mammalian echocardiography, but specific problems exist. The frame rate when color Doppler mode is used is significantly lower in most ultrasound devices. This makes it difficult to assess blood flow. Also, pulsed-wave Doppler has limitations due to the high velocity of flow in the avian heart.

Color-Doppler can be used for the detection of valvular insufficiencies (reported in an Indian Hill Mynah Bird).[39] It is also helpful for the positioning of the gate for spectral Doppler-analysis to determine the degree of valvular insufficiency. In one case, the use of color Doppler for diagnosis of an aneurysm in a coronary artery has been reported.[52] With the standard ventromedian approach, the diastolic inflow into the ventricles is displayed red (flowing towards the transducer) and the systolic outflow blue (flowing away from the scanner) (see Fig 12.5).

Table 12.1 | Longitudinal and Transverse Diameter of the Left Ventricle in Systole and Diastole (mm)*

	LVLS^{V}	LVLD^{V}	LVTS^{V}	LVTD^{V}	LVLS^{H}	LVLD^{H}	LVTS^{H}	LVTD^{H}
Psittacus erithacus erithacus	22.2 ± 1.9	23.9 ± 1.9	7.0 ± 1.1	9.1 ± 1.5	22.5 ± 1.9	24.0 ± 1.9	6.8 ± 1.0	8.6 ± 1.0
Psittacus erithacus timneh	18.4 ± 1.9	19.5 ± 1.9	6.9 ± 0.8	8.9 ± 1.4	17.9 ± 2.4	18.6 ± 3.0	5.9 ± 0.2	7.6 ± 1.3
Amazona spp.	20.7 ± 1.5	21.8 ± 1.9	6.7 ± 1.1	8.7 ± 1.2	21.1 ± 2.3	22.1 ± 2.2	6.7 ± 1.2	8.4 ± 1.0
Cacatua spp.	18.9 ± 1.7	19.4 ± 1.8	6.6 ± 1.7	8.8 ± 1.8	19.0 ± 1.3	19.9 ± 1.6	6.4 ± 1.7	8.3 ± 1.5
Poicephalus s. senegalus	14.5 ± 1.1	15.1 ± 1.0	5.2 ± 0.9	6.9 ± 1.0	14.4 ± 1.2	15.1 ± 2.0	4.6 ± 0.3	5.9 ± 0.5
Diurnal raptors (male)	17.7 ± 1.2	19.3 ± 1.6	6.6 ± 0.9	7.5 ± 1.0	14.7 ± 2.0	16.5 ± 1.8	6.1 ± 0.8	7.4 ± 1.0
Diurnal raptors (female)	18.2 ± 4.7	20.1 ± 5.2	7.7 ± 1.8	8.9 ± 2.1	14.7 ± 4.5	16.3 ± 4.5	6.8 ± 1.7	8.3 ± 1.8
Pigeons (parasternal approach)	—	—	—	—	17.9 ± 1.0	20.1 ± 1.4	5.2 ± 0.4	7.4 ± 0.6

*Psittaciformes according to Pees,[32,33,34] birds of prey according to Boskovic,[3] pigeons according to Krautwald-Junghanns[15]
V = vertical view; H = horizontal view
LVLS = left ventricle, longitudinal diameter systole; LVLD = left ventricle, longitudinal diameter diastole;
LVTS = left ventricle, transverse diameter systole; LVTD = left ventricle, transverse diameter diastole

Table 12.2 | Longitudinal and Transverse Diameters of the Right Ventricle in Systole and Diastole (mm)*

	RVLS^{H}	RVLD^{H}	RVTS^{H}	RVTD^{H}	IVSS^{H}	IVSD^{H}	AOS^{H}	AOD^{H}
Psittacus erithacus erithacus	9.2 ± 1.4	11.5 ± 1.9	2.8 ± 0.9	4.8 ± 1.1	2.9 ± 0.5	2.5 ± 0.3	3.6 ± 0.4	4.0 ± 0.6
Amazona spp.	9.4 ± 1.8	10.3 ± 1.3	3.1 ± 0.7	5.2 ± 1.3	2.2 ± 0.1	2.1 ± 0.4	3.0 ± 0.5	3.4 ± 0.6
Cacatua spp.	10.3 ± 1.2	11.3 ± 2.3	2.3 ± 0.0	3.5 ± 0.5	1.9 ± 0.3	1.7 ± 0.4	—	—
Poicephalus s. senegalus	7.5 ± 1.1	7.6 ± 0.2	2.5 ± 0.4	3.3 ± 0.3	1.9 ± 0.3	1.7 ± 0.2	2.5 ± 0.3	2.4 ± 0.0
Diurnal raptors (male)	12.6 ± 1.9	13.8 ± 1.8	2.1 ± 0.5	2.5 ± 0.7	1.8 ± 0.4	1.9 ± 0.4	—	2.7 ± 0.4
Diurnal raptors (female)	13.0 ± 4,6	14.2 ± 4.2	2.2 ± 0.8	2.5 ± 1.1	2.0 ± 0.8	2.0 ± 0.7	—	2.9 ± 0.4
Pigeons (parasternal approach)	—	9.9 ± 0.8	—	4.0 ± 0.5	3.8 ± 0.1	3.3 ± 0.2	—	3.0 ± 0.1

*Mean value ± standard deviation (Psittaciformes according to Pees,[32,33,34] birds of prey according to Boskovic[3], pigeons according to Krautwald-Junghanns[15])
H = horizontal view
RVLS = right ventricle, longitudinal diameter systole; RVLD = right ventricle, longitudinal diameter diastole; RVTS = right ventricle, transverse diameter systole; RVTD = right ventricle, transverse diameter diastole; IVSS = interventricular septum, thickness systole; IVSD = interventricular septum, thickness diastole; AOS = aorta, diameter systole; AOD = aorta, diameter diastole

Table 12.3 | Calculated Parameters in Psittacines*

	Relation Width to Length of the Ventricles				Fraction Shortening %	
	LVS^{V}	LVD^{V}	RVS^{H}	RVD^{H}	LVT^{H}	RVT^{H}
Psittacus erithacus erithacus	0.32 ± 0.05	0.39 ± 0.07	0.31 ± 0.08	0.43 ± 0.1	22.6 ± 4.4	40.8 ± 11.9
Amazona spp.	0.32 ± 0.04	0.40 ± 0.05	0.35 ± 0.11	0.51 ± 0.13	22.8 ± 4.2	34.1 ± 3.7
Cacatua spp.	0.35 ± 0.08	0.45 ± 0.09	0.23 ± 0.03	0.32 ± 0.07	25.6 ± 7.0	33.3 ± 10.3
Poicephalus s. senegalus	0.36 ±0.06	0.46 ± 0.07	0.30 ± 0.07	0.44 ± 0.04	24.9 ± 3.1	37.1
All examined psittacines	0.33 ± 0.05	0.4 ± 0.07	0.31 ± 0.08	0.43 ± 0.1	23.1 ± 4.6	39.6 ± 11.4

*Mean value ± standard deviation (Pees[32,33,34])
V = vertical view; H = horizontal view
LVS = left ventricle systole; LVD = left ventricle diastole; RVS = right ventricle systole;
RVD = right ventricle diastole; LVT = left ventricle, transverse diameter; RVT = right ventricle, transverse diameter

Spectral Doppler-echocardiography is the standard method for noninvasive blood flow velocity measurements in mammalian medicine. Signals are displayed as two-dimensional time versus velocity graphs. Two different types of spectral Doppler are of interest: pulsed-wave (PW) and continuous-wave (CW). PW Doppler is limited to velocities lower than 2 m/second but allows measurements within an operator-specified sample volume or "gate". Diastolic inflow into the left and the right ventricle, as well as systolic aortic outflow, have been measured in psittacines and raptors using PW Doppler (Fig 12.6).[5,43,47,49] For the mitral inflow, two peaks could be recorded (E-wave corresponding to ventricular filling, A-wave to atrial contraction); E:A was positive for macaws and African grey parrots, whereas in cockatoos it was negative.[5] Blood flow within the pulmonary artery could only be differentiated in a few cases.[5] Measurements are given in Table 12.4.

CW Dopplers are useful for the detection of very high blood flow velocities but do not allow the measurement of the flow velocity at a certain point. No studies regarding the use of CW Dopplers in birds have been published so far.

Birds under stress significantly increase their intracardial blood flow velocity. It is recommended that Doppler-echocardiography be performed under anesthesia in these patients.

ELECTROCARDIOGRAPHY (ECG)

Although electrocardiography was the first

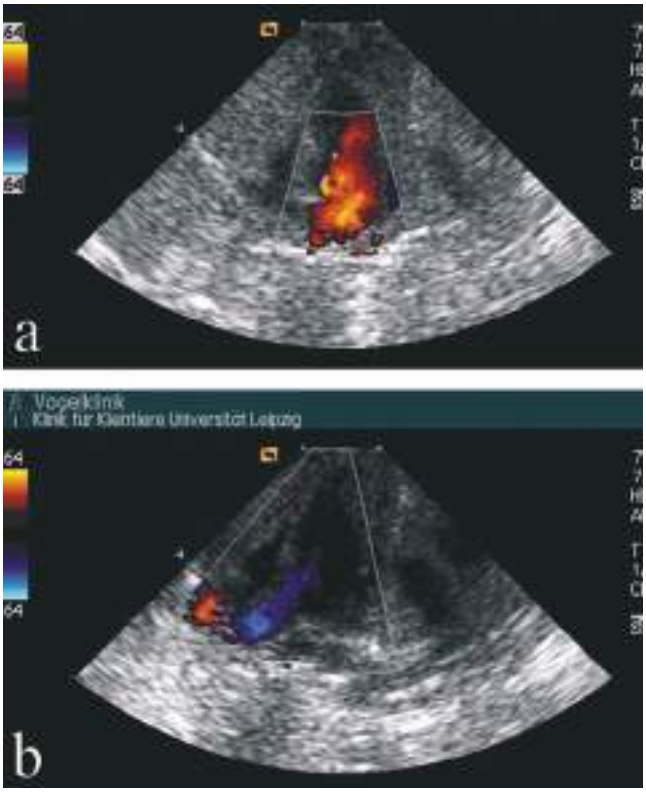
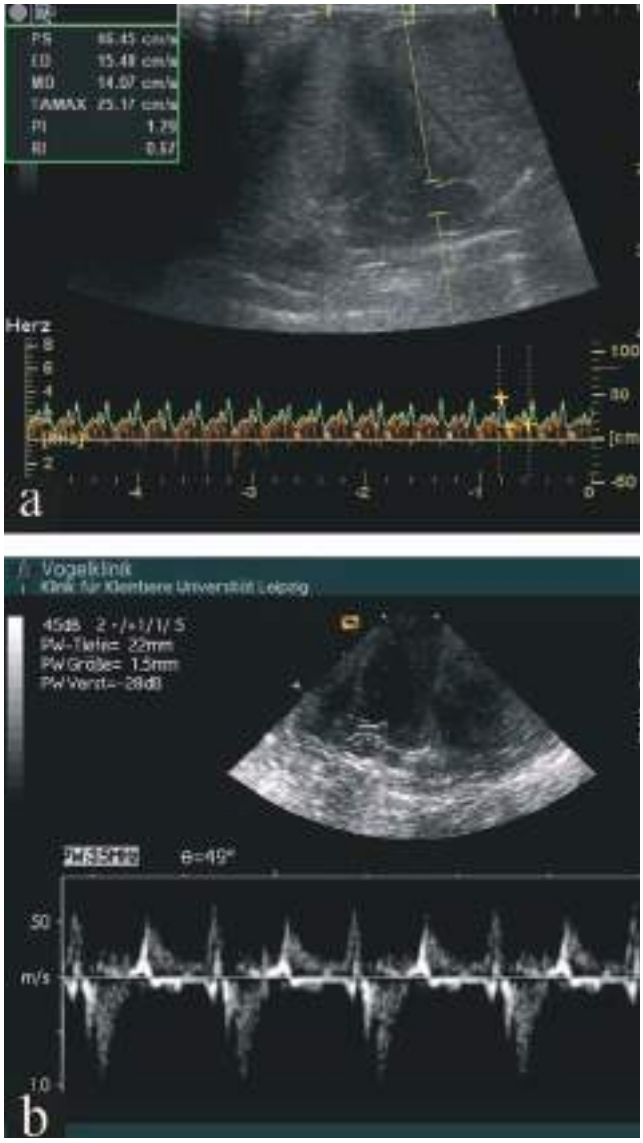


Fig 12.5 | Color-Doppler echocardiography, carrion crow (*Corvus corone*): **a)** Filling of the left ventricle (red inflow). **b)** Systolic outflow into the aorta (blue). Red colored areas are caused by fluid moving towards the transducer, blue areas show movement away from the transducer.

described technique for ante mortem diagnosis of cardiac diseases, it is used less frequently in birds compared to mammals. Difficulties may occur with the connection of the leads to the skin and stress may cause alterations of the recorded ECG. Additionally, no reference values have been published for many of the bird species.

The main indication for ECG is diagnosis and control of arrhythmias and conduction disorders. It may also be valuable for detecting enlargement of the ventricles and metabolic disorders.[38] ECG can be used for monitoring cardiac function during anesthesia and for recording cardiac stages (systole, diastole) while performing other imaging techniques (eg, echocardiography). Another indication for performing ECG is monitoring cardiac disease therapy.

There are many reports on how to perform ECGs in birds. Some authors recommend examination under anesthesia, others prefer recording the ECG in the awake bird. Anesthesia may induce alterations in the ECG. With isoflurane anesthesia, arrhythmias have been described, such as second- and third degree AV-block, sinus arrest, T-wave depression and atrial premature contraction.[1]



I. Kiefer

Fig 12.6 | Pulsed wave Doppler echocardiography: **a)** Diastolic inflow into the left ventricle, channel-billed toucan (*Ramphastos vitellinus*). **b)** Systolic aortic blood flow, carrion crow (*Corvus corone*).

Table 12.4 | Doppler Derived Intracardial Blood Flow Velocities in Birds*

Species	Diastolic Inflow Left Ventricle (m/s)	Diastolic Inflow Right Ventricle (m/s)	Systolic Outflow Aortic Root (m/s)
Amazona spp.[A,43,48,49]	0.18 ± 0.03	0.22 ± 0.05	0.83 ± 0.08
Cacatua galerita[A,5]	0.32 ± 0.15	—	0.78 ± 0.19
Psittacus erithacus[A,5]	0.39 ± 0.06	—	0.89 ± 0.13
Ara sp.[A,5]	0.54 ± 0.07	—	0.81 ± 0.16
Buteo buteo[A,43,48,49]	0.14 ± 0.01	0.14 ± 0.02	1.18 ± 0.05
Buteo buteo[C,43,48,49]	0.22 ± 0.03	0.19 ± 0.03	1.36 ± 0.16
Parabuteo unicinctus[C,43,48,49]	0.19 ± 0.03	0.21 ± 0.03	1.09 ± 0.17
Tyto alba[C,43,48,49]	0.2 ± 0.03	0.22 ± 0.06	1.08 ± 0.12
Falco spp.[C,43,48,49]	0.21 ± 0.03	0.21 ± 0.04	0.95 ± 0.07
Falco spp.[C,43,48,49]	0.28 ± 0.05	0.27 ± 0.05	1.25 ± 0.09

*Straub;[43,48,49] Carrani[5]
A = anesthetized
C = accustomed to handling, conscious

For the clinical application of an ECG in avian patients, six leads, as commonly performed in mammals, can be used. Due to the high cardiac heart rate, electrocardiographs in avian medicine must be able to run at a paper speed of at least 100 mm/second. Leads are attached on the right wing (RA), left wing (LA) and left leg (LL), and the right leg is connected to the ground (Lumeij and Ritchie, 1994). If the ECG is used to monitor anesthesia or to trigger echocardiographic images, bipolar leads (following the electrical heart axis; with one attached cranially to the sternum and slightly paramedian on the right side of the body, and the other one attached caudally to the sternum, slightly on the left side) may be sufficient.

Several techniques for attachment of the leads to the skin have been described (subcutaneous needles, alligator clips, specially constructed feather clips). In the authors' experience, alligator clips can be effectively used in birds, when clipped on the proximal part of the rachis of the feathers. By using alligator clips without sharp teeth there is no damage to the feathers. To get electrical contact with the skin, water soluble ECG gel or small amounts of alcohol are applied between the skin and clip.

The avian ECG is quite different from the mammalian ECG. A schematic lead II-ECG-complex typical for a healthy bird is shown in Fig 12.7. Reference values exist for racing pigeons,[21] Amazons and African grey parrots[23] and some macaw species.[6] A complete overview of the electrophysiologic evaluation of the avian heart and the clinical use of the ECG in birds is given by Lumeij and Ritchie.[22]

POST MORTEM MORPHOMETRY

Whilst reproducible measurements of the thickness of the atrial myocardium are nearly impossible, the thickness of the ventricular myocardium is readily detectable. Due to the huge number of different species that are seen in avian practice, the establishment of reference values for each species is impossible. Fortunately, current investigations could prove that relative values (as a %) rather than absolute values (in mm) are of significance in the assessment of the avian myocardium.

Dissection of the heart under standardized conditions is a basic requirement for performing measurements of the ventricular myocardium thickness at necropsy. The heart should be placed on its left side on a cutting board. A longitudinal cut running through the apex of the heart, the uppermost region of the right ventricular wall and the center of the interventricular septum has to be performed. All blood clots must be removed and the mass of the heart should be determined. Measurements of the thickness of the myocardium at three regions of the left

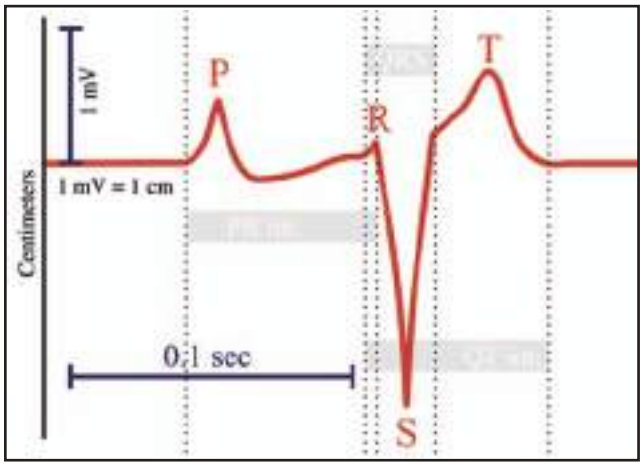


Fig 12.7 | Schematic view of a normal ECG-complex, lead II, in a healthy macaw. Measurement points following Lumeij.[22]

and the right ventricular free wall as well as the interventricular septum should be made using precision callipers. Additionally, measurements of the length of the left ventricle should be ascertained (Fig 12.8).

To date, the amount of scientific data concerning the morphometry of the avian heart is limited. Scientific studies have been performed in two psittacine species (*Melopsittacus undulatus, Alisterus scapularis*) and the common buzzard (*Buteo buteo*).[44,45] Interestingly, by comparing the relative values (thickness of the myocardium in relation to the length of the sternum and the length of the left side of the interventricular septum, respectively) of these different species, it is speculated that the hearts of most avian species follow a set morphological pattern. The myocardium of the ventricles as well as the interventricular septum shows changes of the thickness from the base to the apex of the heart. Whilst the thickness of the left ventricular free wall decreases in the direction of the apex the interventricular septum and the right ventricular free wall become thicker from the base to the middle region and then decrease in thickness towards the apex (Fig 12.8 and Table 12.6).

Therapy of Cardiovascular Diseases

The therapy of avian cardiac disease is still in its infancy. Only a few scientific studies exist, and many drugs routinely used in mammals have not as yet been tested in caged and aviary birds. Overdosing may cause severe side effects; and the pharmacodynamics of drugs in birds is often different due to physiological differences.

Nevertheless, the principles of cardiac therapy are the same as in mammal medicine. Stabilization of the

Table 12.5 | Reference Values for Electrocardiograms in Selected Avian Species*

Parameter	*Columbia* sp.[21]	*Psittacus erithacus*[22]	*Amazon* sp.[22]	*Macaw* sp.[23] Small sp.	Large sp.
	Inner limits for $P_{2.5}$ and $P_{97.5}$ with a probability of 90%$_t$			Mean value ± standard deviation	
Heart rate (1/min)	160-300	340-600	340-600	389 ± 85	275 ± 72
Heart axis (°)	(-83) - (-99)	(-79) - (-103)	(-90) - (-107)	-97 ± 5	-98 ± 8
P-wave duration (s)[M]	0.015-0.02	0.012-0.018	0.008-0.017	0.016 ± 0.002	0.019 ± 0.002
P-wave amplitude (mV)[M]	0.4-0.6	0.25-0.55	0.25-0.60	0.34 ± 0.11	0.24 ± 0.05
PR interval duration (s)[M]	0.045-0.07	0.04-0.055	0.042-0.055	0.050 ± 0.010	0.053 ± 0.009
QRS complex duration (s)[M]	0.013-0.016	0.01-0.016	0.01-0.015	0.017 ± 0.002	0.018 ± 0.002
R amplitude (mV)[M]	0-0.5	0-0.2	0-0.65	0.04 ± 0	0.08 ± 0.04
S amplitude (mV)[M]	1.5-2.8	0.9-2.2	0.7-2.3	0.624 ± 0.234 (QRS-ampl.)	0.624 ± 0.234 (QRS-ampl.)
T amplitude (mV)[M]	0.3-0.8	0.18-0.6	0.3-0.8	0.4 ± 0.09	0.25 ± 0.1
QT Interval Duration					
Anaesthetized (s)[M]	—	0.039-0.07	0.038-0.055	0.081 ± 0.006	0.104 ± 0.018
Unanaesthetized (s)[M]	0.06-0.075	0.048-0.08	0.05-0.095	—	—

*Lumeij and Stokhof;[21] Nap;[23] Casares[6]
M = measurements taken in lead II

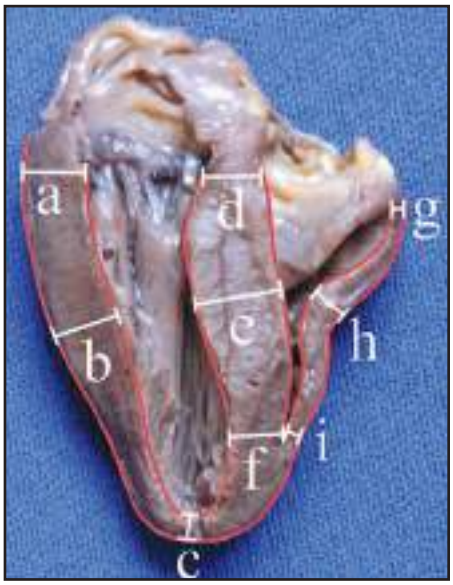


Fig 12.8 | Heart of a common buzzard (*Buteo buteo*). Illustration of the measuring points for the post-mortem evaluation of the myocardial thickness: a to c = left ventricular free wall, a = basal, b = middle, c = apical; d to f = interventricular septum, d = basal, e = middle, f = apical; g to i = right ventricular free wall, g = basal, h = middle, i = apical; distance d to c = length of the left ventricle close to the interventricular septum.

Table 12.6 | Approximate Thickness of the Ventricular Myocardium in Relation to the Length of the Sternum and the Length of the Left Ventricle in *Melopsittacus undulatus*, *Alisterus s. scapularis* and *Buteo buteo*

Myocardium	% of Sternal Length			% of the Length of Interventricular Septum*		
	Basal (Point a)	Middle (Point b)	Apical (Point c)	Basal (Point a)	Middle (Point b)	Apical (Point c)
Left ventricle	7-10%	7-10%	2-4%	20-30%	20-30%	5-10%
Interventricular septum	5.5-7.5%	6.5-9.0%	5-7%	12-22%	16-30%	12-21%
Right ventricle	1.5-2.7%	1.8-3.0%	1.3-2.5%	4.5-8.0%	5.0-8.5%	3.5-6.5%

Straub[44,45]
*Measured on the left side of the interventricular septum points a, b, c, etc. are ref. in Fig 12.8.

patient is essential, and besides symptomatic therapy, the underlying disease has to be diagnosed and treated.

Because cardiac disease in birds is often not diagnosed until the heart decompensates, the veterinarian commonly has to deal with advanced changes. Due to weakness, emaciation and high-grade circulatory problems, these birds are often presented as emergencies. Stress during handling, examination and treatment may be fatal.

ACCOMPANYING THERAPY

Accompanying therapy is essential to maintain circulatory stability and organ function. Liver and kidney function may be affected by circulatory problems. The liver may be congested, renal blood flow may be reduced, and the resulting increase of toxic metabolites and urinary excreted substances may affect the general condition of the bird. The function of the lung can be reduced as a direct result from left heart failure causing pulmonary congestion. Fibrotic lungs will increase the work load of the right ventricle. In addition, reduced air sac volume can lead to dyspnea and hypoxemia damaging the heart.

Cardiac therapy's goals are to reduce congestion (diuretics, ACE inhibitors) and improve cardiac output (ACE inhibitors) which can improve renal/hepatic function. The following supportive measures should also be taken:

- Incubation in a warm environment (80° F (25° C) or above), with sufficient air humidity (60% relative humidity or above) in order to improve circulation and to reduce energy loss.
- Reduction of stress: handling should be reduced to a minimum. Positioning of the patient in ventrodorsal recumbency (eg, for radiology) is a risk and should be considered carefully. Diagnostic procedures in an upright position should be performed (eg, echocardiography). The environment should be calm and temporarily darkened.
- Fluid administration is essential to prevent circulatory collapse and shock and to prevent dehydration due to increased diuresis due to therapy with ACE-inhibitors, and furosemide. Therapy should be

started intravenously or via intraosseus canula. Maintenance administration may be subcutaneous or (preferably) orally. Careful monitoring must be done so the patient is not volume overloaded which would increase congestion.
- Addition of electrolytes, vitamins, amino acids and buffer solution may also be indicated.
- Fluid aspiration from the thoracoabdominal cavity is easily performed in avian patients (if possible, do it ultrasound-guided) and helps to reduce dyspnea due to ascites. For pericardiocentesis, see pericardial effusion.

DRUGS FOR CARDIAC THERAPY
(see Table 12.7)

Heart Glycosides (Digoxin)

Heart glycosides have a positive inotropic effect on the contraction of cardiac muscle. Also relaxation of cardiac muscle will be improved and the heart rate will decrease. These effects lead to a reduction of oxygen demand and an improvement in circulation of the cardiac vessels. Therefore digoxin works best for cardiac diseases that involve volume overloads (insufficient valves), decreased contractility (dilatative cardiomyopathy), or supraventricular tachycardia. The increased cardiac function decreases ascites and edema. To date, there are only two scientific studies on the use of glycosides in pet birds. Pharmacokinetics of digoxin has been examined in sparrows (*Passer domesticus*), budgerigars (*Melopsittacus undulatus*), and quaker (*Myiopsitta monachus*).[11,54]

The therapeutic margin of cardiac glycosides is small, and half-life varies greatly between species. Overdosing may lead to accumulation of cardiac glycosides, and side effects include arrythmias. Contraindications are ventricular tachycardia, second and third degree atrioventricular heart block, hypercalcemia, potassium deficiency and stenotic valves. Cardiac side effects are documented with the combination of glycosides and ketoconazole (therapy of aspergillosis) in humans.

In the authors' experience, cardiac glycosides are useful for emergencies. Chronic administration may be problematic due to difficulties in controlling the side effects and plasma levels. An initial recommended dose of digoxin is 0.02 to 0.05 mg/kg (20 to 50 μg/kg) q 12 h. Maintenance should be dosed carefully at 0.01 mg/kg (10 μg/kg) q 12 h.[11,54]

Angiotensin Converting Enzymes (ACE-) Inhibitors (Enalapril)

Angiotensin II is responsible for the constriction of arterial and venous vessels and the retention of sodium and water by the kidneys. The inhibition of this hormone leads to diuresis (and indirectly to an improvement of renal function), and a decrease of blood pressure. The effect on the heart is a decreased pre- and afterload so that cardiac workload is eased and the cardiac muscle cells may be able to recover.

Although scientific studies regarding the pharmacokinetics of enalapril in birds are lacking, clinical experiences with this drug exist. The results indicate that its use in birds with cardiovascular disease may be beneficial. Empirical dosage used for treatment of birds is 5 mg/kg/day, with reduction to 1 mg/kg/day following improvement of cardiac function. Tolerance is much better when compared to cardiac glycosides and long term administration is possible. Observed side effects after high-dose therapy (5 mg/kg/day) were an increase of PCV and signs of dehydration. These effects were not present when the dose was reduced to 1 mg/kg BID orally.[29,47]

Diuretics (Furosemide)

Indications for furosemide are pulmonary edema, ascites, and pericardial effusion as well as an increased pre- and afterload. The recommended dosage used in birds is 0.15 to 2.0 mg/kg/day PO/IM.[36] Long term administration may lead to a potassium deficiency and therefore cause heart arrhythmias. Since there is a risk of dehydration, especially in small birds, additional careful fluid administration is essential.

In the authors' experience, the main use for diuretics is in the initial therapy of cardiac failure with fluid accumulation (ascites, pericardial effusion) in combination with ACE-inhibitors or glycosides.

Antiarrhythmics (ß-blockers)

A protective effect against the development of atherosclerotic plaques has been demonstrated using oxprenolol in poultry (2 mg/kg/day).[27] Other uses, such as for supraventricular or ventricular arrhythmias, have not been described.

Before using antiarrhythmics, it is important to exclude metabolic causes for the arrhythmia (eg, potassium deficiency, see diuretics). The safety margin of these drugs is small, and the half-life is normally very short, but the clinical effects can last longer, especially if cardiac failure is present.

Calcium Sensitizers (Pimobendan)

Calcium sensitizers are substances with a positive inotropic effect. The pharmacokinetic mechanism is unknown and there are no scientific reports of the use of calcium sensitizers in birds.

Table 12.7| Cardiac Medications in Birds*

Drug	Indication	Application / Dose	Remarks / Side effects
Digoxin	Systolic myocardial failure	PO 0.02-0.05 mg/kg BM BID initially, following 0.01 mg/kg BM BID	• Low therapeutic index • Overdosing: bradycardia, arrhythmia, diarrhea, vomitus
Enalapril	Myocardial failure, increased pre-/afterload	PO 2.5 mg/kg BM BID initially, after one week 1 mg/kg BM BID	• High therapeutic index • Side effect: dehydration
Furosemide	Pericardial effusion, edema, increased pre-/afterload	PO, IM 0.15-2.0 mg/kg BM SID/BID	• Risk of dehydration esp. in smaller birds (lorikeets) • Side effect: arrhythmia caused by potassium deficiency
Oxprenolol	Cardioprotection, prevention from arteriosclerotic plaques (poultry)	PO 2 mg/kg BM SID	• Possible side effects: hypotonia, arrhythmia, tachycardia, AV-block
g-Strophantin (only available in EU)	Circulatory system stimulant	PO drop-wise, to effect IV, IM 1 ml/kg BM	• In case of long-time overdosage, heart hypertrophy especially in smaller birds possible
Atropine	Conduction disturbances, bradycardia	IM 0.01-0.1 mg/kg BM SID	• Only short-time • Overdosing: arrhythmia, gastrointestinal stasis
Etilefrine (only available in EU)	Hypotonia	PO drop-wise, to effect	• Possible side effects: tachycardia, arrhythmia, hypertonia

*Hamlin and Stalnaker;[11] Wilson;[54] Lumeij and Ritchie;[22] Ritchie and Harrison;[36] Pees;[29] Krautwald-Junghanns and Kummerfeld.[19]
SID = 1x/day, BID = 2x/day, PO = orally, IM = intramuscular, BM = body mass

Pathology of Cardiovascular Diseases

Caged birds, in comparison with free living birds, are frequently compromised by restricted exercise, nutritional deficiencies and abnormal climactic conditions. Combined with the bird's natural physiologically high blood pressure, the risk factor for cardiovascular disease in pet birds is significant.

Recent post mortem studies show the frequency of cardiac pathology in pet birds. According to these studies, heart disease occurs commonly in avian species. Oglesbee and Oglesbee[24] found gross and histological evidence for cardiac disease in 26 of 269 psittacine birds. A study of 107 psittacines submitted for routine necropsy found macroscopic lesions of the heart and/or the large vessels in more than one third of the birds[4,20,42] (Table 12.8). In 99% of these 107 birds, at least low-grade histologic changes were present (predominantly inflammatory mixed-cellular infiltration, bacterial infiltration, and/or fat cell accumulation within the myocardium).

Diseases of the heart may occur as the result of congenital, infectious, toxic or idiopathic etiologies. No data currently exist on age-related cardiovascular diseases in pet birds. A variety of cardiac abnormalities occur secondarily as acquired disease and/or compensation/decompensation due to other organ failure (ie, lung and liver), neoplasia or systemic infections.[16,17,20]

CAUSES OF (CONGESTIVE) HEART FAILURE

Heart failure is the clinical syndrome resulting from abnormalities of cardiac function (eg, myocardial failure, valvular regurgitation).

Table 12.8 | Incidence of Selected Cardiac and Vascular Diseases in 107 Psittacine Birds. Macroscopic Findings (Findings in 39 Birds (36.4 %)*

	Findings	Cases
Pericardium	Pericarditis	16 (14.9%)
	Pericardial effusion	6 (5.6%)
Myocardium	Hypertrophy/dilatation	16 (14.9%)
	Petechial bleeding	6 (5.6%)
Endocardium	Endocarditis *valvularis*	1 (0.9%)
Aorta/A. *pulmonalis*	Yellow discoloration/hardening	11 (10.3%)

*Braun;[4] Krautwald-Junghanns[20]

The syndrome "congestive heart failure (CHF)" results from inadequate cardiac output and an increased preload. Fluid retention is caused by elevated venous and capillary pressures. In left heart failure, an increase of the pressure in the left atrium and the pulmonary veins leads to pulmonary edema. In birds with right heart failure, ascites, liver congestion and hydropericardium are commonly seen.

CONGENITAL DISEASES

Due to the high physiological load of the avian heart, congenital cardiovascular disorders often lead to early embryonic or fledgling death. They are rarely presented as clinical diseases in birds, ante mortem diagnosis is rare and the cause is normally found only at necropsy. In one case, evidence for a congenital defect of the muscular right atrioventricular valve could be found in a 35-year-old Amazon diagnosed with dilatation of the right ventricle.[30]

PERICARDIAL DISEASES (PERICARDITIS, PERICARDIAL EFFUSION)

Pericardial changes frequently found in birds include inflammation (pericarditis) and effusion (hydropericardium, hemopericardium).

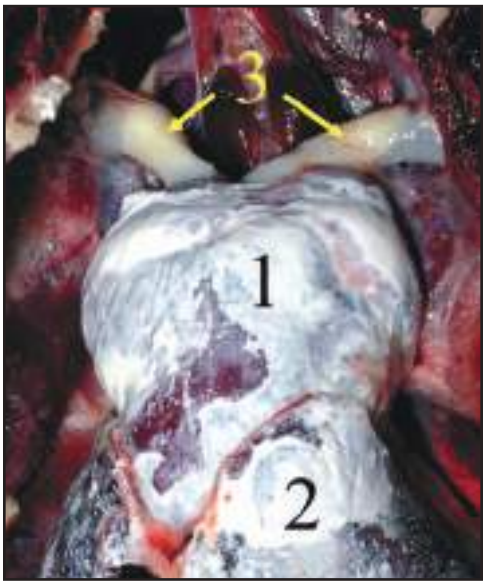


Fig 12.9 | Necropsy, blue fronted Amazon (*Amazona aestiva aestiva*), pericarditis urica. Uric acid deposits can be seen on pericardium (1) that covers the heart and the serosa of the liver (2). The yellowish discoloration of the large vessels (3) is an indication for arteriosclerosis.

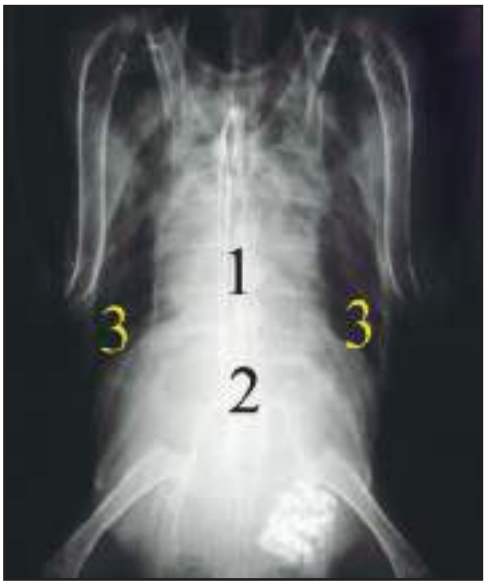


Fig 12.10 | Radiograph, ventrodorsal view, African grey parrot (*Psittacus erithacus*), tentative diagnosis of cardiac disease (corresponding to Fig 12.11). The heart (1) and the liver (2) shadow are enlarged. Detail recognition in the thoracoabdominal cavity is reduced; the air sacs (3) are compressed.

Inflammation of the pericardium may develop in the process of infectious diseases of surrounding tissue. Common causes are generalized trichomonas infections in pigeons, mycotic infections originating from the respiratory tract, and bacterial infections including mycobacteriosis. Deposits of uric acid in the pericardium may be seen with visceral gout (Fig 12.9).

Hydropericardium is the cardiac change most frequently diagnosed ante mortem. It may develop with infectious exudative pericarditis, but can also occur due to congestion in cardiac failure, metabolic disorders (protein deficiency) or as an idiopathic syndrome. Hemopericardium is generally found following trauma or sclerosis with subsequent rupture of vessels, and is almost always fatal.

Clinical symptoms in birds with pericarditis are often nonspecific. Diagnosis is usually confirmed at necropsy. Radiography may reveal cardiomegaly with an irregular cardiac silhouette. Echocardiography is of limited use for diagnosis if there is no pericardial effusion. Endoscopy is the most reliable ante mortem diagnostic aid, although there is an increased risk during anaesthesia (see Chapter 24, Diagnostic Value of Endoscopy and Biopsy). In cases of acute visceral gout, plasma uric acid levels may be increased.

Common symptoms in birds with pericardial effusion are abdominal distension, dyspnea and exercise intolerance. Hydropericardium is often combined with congestion of the liver and ascites. The typical radiographic findings are enlargement of the heart and liver shadow as well as loss of detail (Fig 12.10). ECG may show low voltage,[22] but allows only a tentative diagnosis (adipose tissue and space-requiring processes like tumors can cause low voltage on the ECG as well).[29] The only reliable ante mortem diagnostic test for pericardial effusion is echocardiography. Hydropericardium can be visualized as an anechoic area between the myocardium and the pericardium (Fig 12.11). For further examinations (cytology, microbiology) an ultrasound guided aspirate from the pericardial space may be taken. In most cases, pericardial effusion is non-infectious and clear. Nevertheless, inflammatory cells may indicate infection, and culture may help to identify bacterial or fungal causes.

Treatment of exudative pericarditis, in addition to treating the underlying condition, may include administration of furosemide or another diuretic. Treatment for visceral gout is generally unrewarding (see Chapter 16, Evaluating and Treating the Kidneys). For treatment of hydropericardium due to congestive heart failure, diuretics in combination with ACE inhibitors are indicated.[29,47] Pericardial effusion may result in diastolic heart failure, ie, in an insufficient filling of the ventricles in diastole due to compression of the atria by the fluid in the pericardial cavity. This is a contraindication for the use of glycosides. An ultrasound guided pericardiocentesis may be performed to remove fluid from the pericardial space. Repeated echocardiography can be used to evaluate success of the therapy.

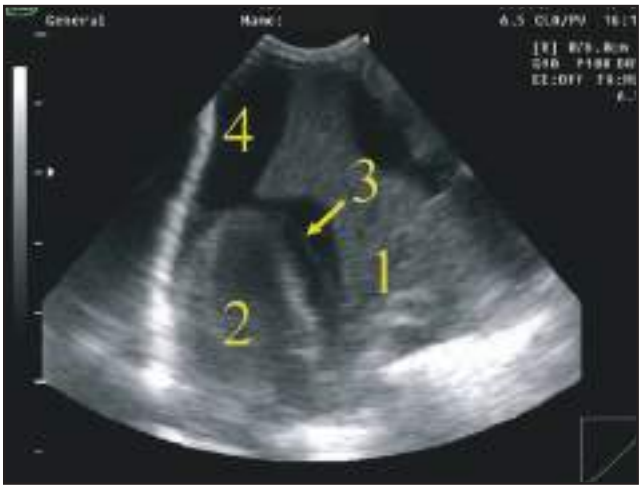


Fig 12.11 | 2-D-Echocardiography, African grey parrot (*Psittacus erithacus*), pericardial effusion and ascites (4) (corresponding to Fig 12.10). The pericardial effusion (1) can be seen as an anechoic area between the heart (2) and the pericardium (3).

MYOCARDIAL DISEASES (MYOCARDIAL FAILURE)

A decreased myocardial contractility (myocardial failure) may be primary, idiopathic (dilatative cardiomyopathy), or secondary, as a result of systemic diseases of infectious (myocarditis), toxic, metabolic (lipomatosis cordis, arteriosclerosis) and neoplastic origin. Frequently, an increased workload (eg, due to arteriosclerosis and pulmonary hypertension) leads to hypertrophy of the ventricles and can eventually result in decompensation and dilatation. Valvular insufficiencies can also lead to ventricular dilatation and eventually failure.[30] In psittacines, myocarditis is described as a complication in neuropathic proventricular dilatation disease.[40,51] Iron deposition in the myocardium combined with dilatation of the ventricles occurs in mynah birds with iron storage disease.[9] After prolonged transport of wild birds, myocardial necrosis may be seen as neurogenic arrhythmias as a consequence of metabolic disturbances.

Mostly myocardial failure is right-sided. Systemic lung diseases (ie, chronic mycosis) frequently result in dilatation of the right ventricle (including the right, muscular AV valve) and the right atrium and therefore the venous part of the circulatory system. Isolated left-sided myocardial failure is rare, since the resulting pulmonary congestion also affects the right ventricle and leads to a secondarily increased afterload and eventually right-sided CHF.

Although one case report suggests that myocardial compensation of valvular insufficiencies may occur for years, decompensation and development of clinical signs seem to develop more quickly in birds than they do in mammals.[30]

Clinical signs of myocardial failure of any cause can present as generalized weakness and as respiratory impairment. Hepatic congestion and ascites often cause abdominal distension.

Radiographs may demonstrate a consolidated, enlarged and sharply delineated shadow of the entire heart and liver. The appearance of the lung fields may demonstrate homogenous or non-homogenous increased opacity of the honeycomb pattern of the lungs which indicates pulmonary edema.

In birds with dilatative cardiomyopathy, the ECG may show an increased R-wave and a negative P-wave. The heart axis can change to between 0° and -170°. Arrhythmias are often seen with cardiomyopathy and myocarditis.[22]

In 2-D echocardiography, altered wall thicknesses and diameters of the ventricles as well as decreased contractility can be seen (measurements see Tables 12.1-12.3). In birds with right heart failure, the right ventricle is often as large as the left (Fig 12.12). Accompanying common findings are hydropericardium, ascites and congested liver parenchyma (see dilated vessels, Fig 12.13).

In case of suspected cardiomyopathy, blood-chemistry evaluation should include AST, CK, uric acid, LDH and electrolytes.

In addition to treatment of the underlying cardiac disease, supportive care including cage rest and diet change is important. Diuretics are indicated when edema (eg, pulmonary edema) or ascites are present. In the authors' experience, a combination of glycosides, ACE-inhibitors and diuretics seems to be the most effective initial therapy for cases of acute heart failure. For long-term therapy, ACE-inhibitors are preferable.[29,47]

ENDOCARDIAL DISEASES (ENDOCARDITIS, VALVULAR INSUFFICIENCY, VALVULAR STENOSIS)

Alterations of the endocardium, in particular the valves, may be idiopathic.[30] However, these changes are found more frequently secondary to infections with streptococci, staphylococci, *Pasteurella multocida* or *E. coli*.[20,22,35]

Independent of the etiology, functional damage of the AV valves leads to regurgitation and to left heart failure with pulmonary edema (small circulatory cycle) and/or right heart disease with liver congestion (large circulatory cycle) and can cause general cardiac failure. Thickening of the right AV valve is normally caused by muscular hypertrophy. It may be a consequence of valvular insufficiency or due to hypertrophy of the whole right ventricle (see Fig 12.12). This alteration occurs

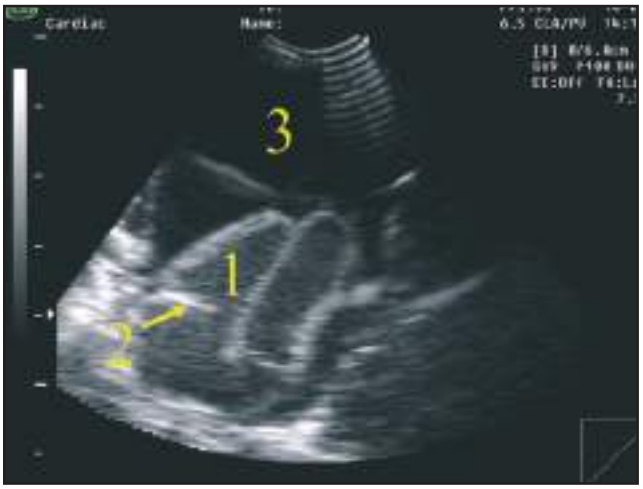


Fig 12.12 | 2-D-Echocardiography, yellow-crowned Amazon (*Amazona ochrocephala*), right heart failure. The right ventricle (1) is as large as the left one, the muscular right atrioventricular valve (2) is clearly thickened (3 = ascites).

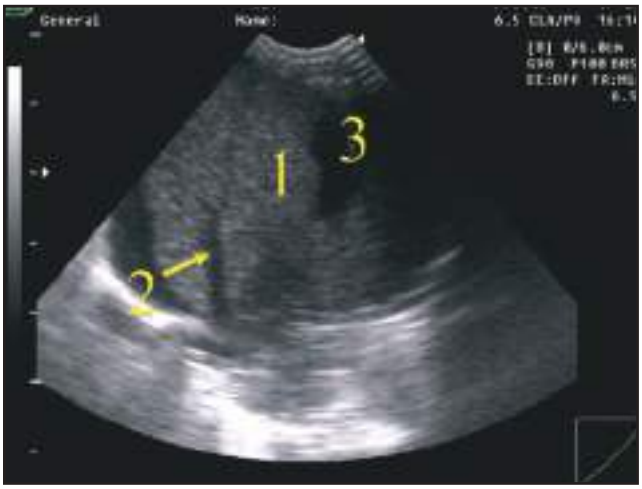


Fig 12.13 | 2-D-Echocardiography, African grey parrot (*Psittacus erithacus*). Liver (1) congestion and ascites (3). Dilated vessels (2) can be demonstrated in the liver tissue.

more often than alterations of the left AV valve. Chronic inflammation of the semilunar valves can also result in stenosis.[20]

Clinically, nonspecific symptoms (weakness, dyspnea, distended abdomen) may be noticed.

Radiographic investigation gives information about cardiac size and congestion of organs (see Myocardial Diseases section) but radiographic diagnosis of endocardial alterations is not possible.

2-D-Echocardiography can show accompanying myocardial thickening and changes of the valves, especially thickening of the right atrioventricular valve (see Fig 12.12).[30,35] Doppler echocardiography is the imaging tool of choice for demonstration of valvular regurgitation in mammals. Although initial examinations have been performed in birds (see Echocardiography), little has been published about the use of Doppler echocardiography in case of suspected valvular damage. In an Indian hill mynah, color flow and spectral Doppler have been used for demonstration of mitral regurgitation.[37,39]

A good prognosis may be given in cases of acute endocarditis with aggressive treatment of the causal agent (Myocardial Diseases section).

In patients with valvular regurgitation, glycosides can be used, whereas in case of stenosis, these drugs are contraindicated. A decrease in the heart rate to a normal range and decreased congestion may be interpreted as therapeutic success.

ARTERIOSCLEROSIS

Arteriosclerosis is the most frequently described pathologic change of the vessels in psittacine birds.[4,10] Different etiological factors are discussed; the most frequent causes are hyperlipidemia, endothelial inflammation, toxins, immune complexes, hypertonia and/or stress factors. Age and nutritional deficiencies over many years, as well as lack of exercise seem to play a role in the development of arteriosclerosis.[13] Psittacines commonly affected by arteriosclerosis are amazons (especially blue fronted Amazons), African grey parrots and cockatoos.[4,13,20]

Macroscopic changes include arterial wall thickening, intimal roughening, induration and yellowish discoloration.[10] Calcification can cause plaque-like or diffuse hardening of the larger arteries like the aorta and brachiocephalic trunk (Fig 12.14). Arteriosclerosis cannot be diagnosed by gross findings alone, but requires histological examination, especially in earlier stages.[10,20]

A diagnosis of arteriosclerosis is often made at necropsy. Clinical findings are usually absent or may only lead to a tentative diagnosis. Possible symptoms include lethargy, neurological signs (tremor, paralysis of the legs), decreased exercise tolerance and dyspnea.[8,13,14] Acute death may occur.

Radiologically, an increased radiodensity and widening of the aorta may be seen in advanced cases, sometimes in connection with left atrial or left ventricular enlargement. Echocardiography may be useful for diagnosis of the arteriosclerotic processes of the large vessels close to the heart, but no systematic studies have been done to date. In a white cockatoo, an aneurysm of a coronary artery associated with arteriosclerosis has been diagnosed by echocardiography.[52]

Effective therapy for arteriosclerosis is not known for birds. In poultry, ß-blockers (eg, oxprenolol) proved to have a protective effect against the development of

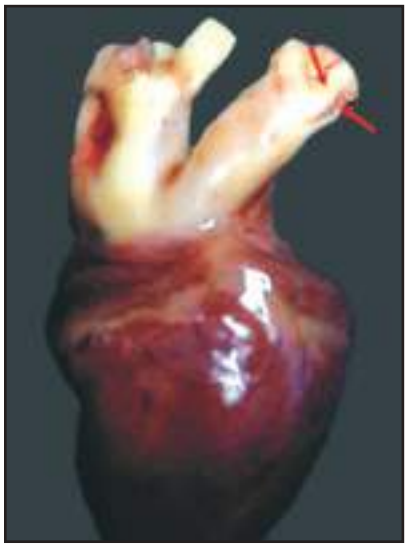
arteriosclerotic plaques. Unfortunately it is not possible to remove plaques which are already present.[27,28] Diuretics may be indicated as well as ACE-inhibitors if heart failure occurs.[7] Balanced food supply and sufficient opportunities to exercise are important preventive factors. Proper flight exercise and diet will prevent obesity, a major contributor to this cardiac disease.

CIRCULATORY DISTURBANCES (CIRCULATORY COLLAPSE, SHOCK)

Physiologically, the avian circulatory system has a large load capacity. Unexpected deaths, after catching small birds, can sometimes be attributed to asphyxia (strong pressure on the sternum). Long restraint of fractious birds may lead to circulatory collapse, especially in birds with dyspnea, ascites (resulting in displacement of the air sacs) or cardiac failure. Hypovolemic, septic (toxic-infectious) and neurogenic shock as well as anaesthetic complications may also be a cause. Overcrowding of carriers or transporting in temperatures near or above 100° F (38° C) may lead to circulatory collapse.

Clinical symptoms of circulatory collapse start as respiratory signs with spread wings/ legs and tachypnea/ dyspnea. This is followed rapidly by respiratory arrest, convulsions with opisthotonus, and a loss of consciousness.

Further diagnostics should only be done after stabilization of the patient. If possible, the bird should be kept upright for examination (eg, circulatory risk during positioning for radiology). The treatment for shock is always handled as an emergency situation. The bird must immediately be brought to a calm and darkened area. A volume substitution with warmed (100-103° F) lactated



S. Braun

Fig 12.14 | Necropsy, African grey parrot (*Psittacus erithacus*), with arteriosclerosis. Thickening and discoloration of the wall of the large vessels (arrows).

Ringer's solution (LRS) or half-strength LRS + 2.5% dextrose (IV, intraosseus cannula, for maintenance also SC) is indicated. Infusions with sodium bicarbonate solution should be given in case of metabolic acidosis, recommended dosage is 1 mEq/kg (=1 ml/kg), in intervals of 15 to 30 minutes up to a maximum of 4 mEq/kg.[55] Additionally, oxygen supply is indicated. If faced with respiratory arrest administration of doxapram (respiratory stimulant, orally dropwise to effect) may be useful. Oral /IV/IM administration of g-strophantin (see Table 12.7) may successfully stabilize patients presenting in circulatory failure. Sympathomimetics (etilifrine) may be administered orally to increase the stroke volume in case of heart failure (in particular, in case of cardiogenic shock).

Parts of this chapter are copied or paraphrased from Proc Assoc Avian Vet, 2001, pp 225-330. Permission granted by AAV 2003.

References and Suggested Reading

1. Aguilar RF, Smith VE, Ogburn P, Redig PT (1995) Arrhythmias associated with isoflurane anaesthesia in bald eagles (*Haliaeetus leucocephalus*). J Zoo Wildl Med 26, 508-516.
2. Bezuidenhout AJ (1983) The valva atrioventricularis dextra of the avian heart. Zentralbl Vet Med C Anat Histol Embryol 12, 104-108.
3. Boskovic M, Krautwald-Junghanns ME, Failing K, et al. (1999). Möglichkeiten und Grenzen echokardiographischer Untersuchungen bei Tag- und Nachtgreifvögeln (Accipitriformes, Falconiformes, Strigiformes). Tierärztliche Praxis 27, 334-341.
4. Braun S, Krautwald-Junghanns ME, Straub J. (2002) Zur Art und Häufigkeit von Herzerkrankungen bei in Deutschland in Gefangenschaft gehaltenen Papageienvögeln. Deutsche Tierärztliche Wochenschrift 109, 253-292.
5. Carrani F, Gelli D, Salvadori M, Aloisi M (2003). A preliminary echocardiographic initial approach to diastolic and systolic function in medium and large parrots. Proc 7th Europ AAV Conf, Puerto de la Cruz, 145-149.
6. Casares M., Enders F. Montaya JA. (1999). Electrocardiography in some macaw species (genus *Anodorhynchus* and Ara). Proceedings of the 5th European AAV meeting, Pisa, 158-163.
7. Chamontin B, Amar J (2001). Prevention in cardiovascular pathology. Therapy 56, 119-124.
8. Dorrestein GM, Zwart P, Borst GHA, Poelma FG, Buitelaar MN (1977). Ziekte-en doodsoorzaken van vogels. Tijdschr Diergeneesk. 102, 437-447.
9. Dorrestein GM, Van Der Hage MH (1988). Veterinary problems in mynah birds. Proc Assoc Avian Vet, 263-274.
10. Fricke C, Dorrestein GM, Straub J, Krautwald-Junghanns ME (2003). Macroscopic and microscopic changes in blood vessels of psittaciformes. Proc 7th Europ AAV Conf, Puerto de la Cruz, 137-144.
11. Hamlin RL, Stalnaker PS (1987). Basis for use of digoxin in small birds. J Vet Pharmacol Therap 10, 354-356.
12. Hanley SH, Helen GM, Torrey S, et al. (1997). Establishing cardiac measurement standards in three avian species. J Avian Med Surg 11,15-19.
13. Johnson HJ, Phalen DN, Kondik VH, Tippit T, Graham DL (1992). Atherosclerosis in psittacine birds. Proc Assoc Avian Vet, New Orleans, 87-93.
14. Kempeneers P (1987). Atherosklerose bij de papegaai. Utrecht, Utrecht University.
15. Krautwald-Junghanns ME, Schulz M, Hagner D, Failing K and Redmann T (1995). Transcoelomic two-dimensional echocardiography in the avian patient. J Avian Med Surg 9,19-31.
16. Krautwald-Junghanns ME, Straub J, Pees M, Braun S (2001a). Anatomy and pathology of the psittacine heart. Proceedings of the 4th Scientific ECAMS Meeting, Munich, 14-15.
17. Krautwald-Junghanns ME, Straub J (2001b). Avian Cardiology: Part 1. Proceedings of the Assoc Avian Vet, Orlando, 323-330.

18. Krautwald-Junghanns ME, Pees M, Schütterle N (2001c). Echokardiographische Untersuchungen an unsedierten Brieftauben (*Columbia livia f. domestica*) unter besonderer Berücksichtigung des Trainingszustandes. Berliner und Münchner Tierärztliche Wochenschrift 114, 1-4
19. Krautwald-Junghanns ME, Kummerfield N (2003). Arzneimittelverzeichnis. *In:* Kaleta EF, Krautwald-Junghanns ME: Kompendium der Ziervogelkrankheiten. Schluetersche Verlagsanstalt, Hannover.
20. Krautwald-Junghanns ME, Braun S, Pees M, Straub J, Valerius HP (2004). Investigations on the anatomy and pathology of the psittacine heart. J Avian Med Surg, 18,2-11.
21. Lumeij JT, Stokhof AA (1985). Electrocardiogram of the racing pigeon (*Columbia livia forma domestica*). Res Vet Sci 38, 275-278.
22. Lumeij JT, Ritchie BW (1994). Cardiovascular System. *In:* Ritchie BW, Harrsion GJ, Harrsion LR (eds): Avian Medicine, Principles and Application, Brentwood, TN, HBD Int'l Inc, 694-722.
23. Nap AMP, Lumeij JT, Stokhof AA(1992). Electrocardiogram of the African Grey (*Psittacus erithacus*) and Amazon (*Amazona* sp.) parrot. Avian Pathol 21, 45-53.
24. Oglesbee BL, Oglesbee MJ (1998). Results of post-mortem examination of psittacine birds with cardiac disease: 26 cases (1991-1995). JAVMA 212, 1737-1742.
25. Orosz S (1997). Anatomy of the cardiovascular system. *In:* Altman RB, Clubb SL, Dorrestein GM, Quesenberry K (eds): Avian medicine and surgery. WB Saunders Co, Philadelphia, 489-490.
26. Owen R (1866). Anatomy of vertebrates. Vol II. Birds and mammals. Longmans, Green, and Co., London.
27. Pauletto P, Pessina AC. Pagnan A, Thiene G, Semplicini A, Vescovo G, Mazzucato A, Scannapieco G, Angelini A, Dal Palu C (1985). Evidence of reduced atherosclerotic lesions in broad breasted white turkeys treated with oxprenolol. Artery 12, 220-233.
28. Pauletto P, Vescovo G, Scannapieco G, Pessina AC, Dal Palu C. (1990). Cardioprotection by beta-blockers: molecular and structural aspects in experimental hypertension. Drugs Exp Clin Res. 16,123-128.
29. Pees M, Straub J, Krautwald-Junghanns ME (2000). Pericardial effusion in birds: Demonstration of clinical cases. Proceedings of the Assoc Avian Vet, Portland, 189-191.
30. Pees M, Straub J, Krautwald-Junghanns ME (2001). Insufficiency of the muscular right atrioventricular valve in the heart of a blue-fronted Amazon (*Amazona aestiva aestiva*). Vet Rec 148, 540-543.
31. Pees M, Straub J, Krautwald-Junghanns ME (2003a). Echokardiographische Untersuchungen bei Psittaciformes, Teil 1: Durchführung und gattungsspezifische Unterschiede. Tierärztl Praxis 31(K): 126-31.
32. Pees M, Straub J, Krautwald-Junghanns ME (2003b). Echokardiographische Untersuchungen bei Psittaciformes, Teil 2: Sonografisch ermittelte Messwerte zur Herzanatomie und errechnete Parameter der Herzfunktion. Tierärztl Praxis 31(K), 180-187, 2003.
33. Pees M, Straub J, Krautwald-Junghanns ME (2003c). Echocardiographical examinations of healthy psittacine birds under special consideration of the African Grey parrot (*Psittacus erithacus erithacus*). Proc 7th Europ AAV Conf, Puerto de la Cruz, 161-167.
34. Pees M, Straub J, Krautwald-Junghanns ME (2004). Echocardiographical examinations of psittacine birds with special reference to the African grey parrot. Vet Rec, 155,73-76.
35. Randolph J, Moise S, Graham D, Murphy C (1984). Bacterial endocarditis and thromboembolism of a pelvic limb in an emu. J Am Vet Med Assoc 11, 1409-1410.
36. Ritchie BW, Harrison GJ (1994). Formulary. *In:* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine, Principles and Application, Brentwood, TN, HBD Int'l Inc, 457-478.
37. Rosenthal K and Stamoulis M (1992). Congestive heart failure due to mitral regurgitation in an Indian hill mynah bird. Proc of the Assoc Avian Vet, New Orleans, 171-173.
38. Rosenthal K, Miller M (1997). Cardiac disease. *In:* Altman RB, Clubb SL, Dorrestein GM, Quesenberry K (eds): Avian medicine and surgery. WB Saunders Co, Philadelphia, 491-500.
39. Rosenthal K and Stamoulis M (1993). Diagnosis of congestive heart failure in an Indian hill mynah bird (*Gracula religiosa*). J Assoc Avian Vet 1, 27-30.
40. Shivaprasad HL, Barr BC, Woods LW, Daft BM, Moore JD, Kinde H, Anderson ML, Drougal R (1995). Spectrum of lesions (pathology) of proventricular dilatation syndrome. Proc Assoc Avian Vet, California, 505-506.
41. Smith FM, West NH, Jones DR (2000). The cardiovascular system. *In:* Avian Physiology, 5th edition (GC Whittow ed) Academic Press.
42. Straub J, Pees M, Krautwald-Junghanns ME (2000). Heart diseases in birds: Rare or just not diagnosed? Proc Assoc Avian Vet, Portland, 285-289.
43. Straub J, Pees M, Schumacher J, Gompf RE, Baade H, Krautwald-Junghanns ME (2001). Doppler-echocardiography in birds. Proceedings of the 6th European AAV Main Conference, Munich, 92-94.
44. Straub J, Valerius KP, Pees M, Krautwald-Junghanns ME (2002a). Morphometry of the heart of budgerigars (*Melopsittacus*), Alisterus parrots (*Alisterus s. scapularis*) and common buzzards (*Buteo buteo*). Research in Veterinary Science 72, 147-151.
45. Straub J, Valerius KP, Pees M, Krautwald-Junghanns ME (2002b). Untersuchungen zur Myokarddicke bei Wellensittichen (*Melopsittacus undulatus*) und Königssittichen (*Alisterus s. scapularis*). Berliner und Münchener Tierärztliche Wochenschrift 115, 440-444.
46. Straub J, Pees M, Krautwald-Junghanns ME (2002c). Measurement of the cardiac silhouette in psittacines. Journal of the American Veterinary Medical Association 221, 76-79.
47. Straub J, Pees M, Enders F, Krautwald-Junghanns ME (2003a). Pericardiocentesis and the use of enalapril in a Fischer's lovebird (*Agapornis fischeri*). Vet Rec 152, 24-26.
48. Straub J, Forbes N, Pees M, Krautwald-Junghanns ME (2003b). Effect of handling-induced stress on the results of spectral Doppler-echocardiography in falcons. Res Vet Sci 74,119-122.
49. Straub J, Forbes N, Pees M, Krautwald-Junghanns ME (2004). Pulsed-wave Doppler derived velocity of diastolic ventricular inflow and systolic aortic outflow in diurnal and nocturnal raptors. Veterinary Record 154,145-147.
50. Thomas WP, Gaber CE, Jacobs GJ, Kaplan PM, Lombard CW, Moise NS, Moses BL (1993). Recommendations for standards in transthoracic two-dimensional echocardiography in the dog and cat. J Vet Int Med 7, 247-252.
51. Vice C, Cazayoux A (1992). Myocarditis as a component of psittacine proventricular dilatation syndrome in a Patagonian conure. Avian Diseases 35, 1117-1119.
52. Vink-Nooteboom M, Schoemaker NJ, Kik MJL, Lumeij JT, Wolvekamp WTC (1998). Clinical diagnosis of aneurysm of the right coronary artery in a white cockatoo (*Cacatua alba*). J Small Anim Pract 39: 533-537.
53. West NH, Langille BL, Jones DR (1981). Cardiovascular system. *In:* King, AS, McLelland J (eds): Form and Function in Birds (Vol. 2). Academic Press, London, New York, 235-251.
54. Wilson RC, Zenoble RD, Horton CR, and Ramsey DT (1989). Single dose digoxin pharmacokinetics in quaker conure. J Zoo Wildl Med 20, 432-434.
55. Wilson H (2003). Avian Emergency and Critical Care. Proceedings of the Assoc Avian Vet, Pittsburgh, 261-268.
56. Wyatt HL, Heng MK, Meerbaum S (1979). Cross-sectional echocardiography I. Analysis of mathematic models for quantifying mass of the left ventricles in dogs. Circulation 60, 1104-1113.

CHAPTER
13

Integument

ROBERT E. SCHMIDT, DVM, PhD, Dipl ACVP;
TERESA L. LIGHTFOOT, DVM, Dipl ABVP-Avian


Bob Doneley

Fig 13.1a | Mustached parakeet with facial dermatitis resulting in feather loss and replacement. Pin feathers predominate in the affected area.


Greg J. Harrison

Fig 13.1b | A burn from administrating scalding hot food. Microwaved food is often the source.

Skin and feather problems are common disorders in pet avian species (Fig 13.1a). The skin has limited responses to insults. A variety of causes will lead to similar clinical signs and possibly similar gross and histologic changes. The clinician's challenge is to use available diagnostic methods to determine an etiology and rational therapeutic approach.

Birds often present with feather loss or picking. The appearance of the skin may vary from grossly normal to severely inflamed and/or necrotic (Fig 13.1b). In assessing gross morphologic changes, the effect of self-trauma must be considered. Although the lesions may be due to a primary problem within the skin and/or feathers, a variety of internal disorders, as well as behavioral problems, can also result in external lesions.

History

Arriving at a meaningful diagnosis requires a logical process that considers the differential diagnostic possibilities. History is essential. A complete history should include information on the bird's environment, changes in routine and diet. (See Chapter 4, Nutritional Considerations and Chapter 6, Maximizing Information from the Physical Examination for more specific information).

A description of the physical surroundings of the bird is needed, including such things as temperature and humidity, which can influence normal molting and which may play a part in clinical disease syndromes. The conditions of other birds in the household/aviary should also be determined. References are available for feather anatomy review.[6]

Physical Examination

Refer to Chapter 6, Maximizing Information from the Physical Examination for the description of a complete physical examination. During the physical examination, specific dermatologic lesions should be examined and classified. Examination includes the distribution of lesions, presence or absence of pruritus, relative conditions of the skin and feathers and presence of plaques, ulcers and exudates. The association of individual lesions with specific conditions is not as well documented in birds as it is in domestic pets. Notation of these dermatologic abnormalities will aid in both the clinical description to accompany biopsy submissions and in tracking by the practitioner of the course of the disease. A simple anatomic illustration, such as is used in dog and cat medicine can be valuable in recording these lesions. See Chapter 6, Maximizing Information from the Physical Examination for an example of this stamp.

DIAGNOSTICS

Evaluation of systemic illness and organ function via a complete blood count (see Chapter 22, Diagnostic Value of Hematology) and serum biochemistries (see Chapter 23, Diagnostic Value of Biochemistry) should be performed. Specific tests for syndromes such as PBFD circovirus may be indicated (see Chapter 32, Implications of Viruses in Clinical Disorders). An evaluation for nutritional deficiency or toxicities should be made from the dietary history (see Chapters 4, Nutritional Considerations and Chapter 6, Maximizing Information from the Physical Examination).

Several diagnostic procedures are available in order to gain information about skin lesions (Tables 13.1, 13.2). Scrapings may reveal the presence of mites, but in some lesions the mites are deep within the subcutis and will be missed by superficial scraping. Impression smears can give an indication of inflammation vs. neoplasia. Bacteria and fungi are also seen in impression smears, but their significance may be difficult to determine. Feather pulp smears potentially provide information concerning inflammatory processes within the pulp. Care must be taken not to confuse melanin granules with bacteria. Melanin granules will be uniform with tapered ends and will not be stained, having a natural brown-black color.

Culture is important but must be done correctly or the significance of the isolate is questionable. If folliculitis is suspected, aspiration of the follicle by sterile needle and syringe is necessary.

Skin and feather biopsy is an important tool, but its effectiveness is compromised by the lack of clinical history and description in many submissions. The presence of an overwhelming microbial population can be diagnostic, although the sensitivity of the organism to various antimicrobials cannot be determined from histopathology. In the absence of a definitive etiologic agent, allergy, self-trauma or endocrinopathy may be suggested from the biopsy.

Pulling of feathers and submission for histopathology may lead to a diagnosis in some cases, but if the feathers are normal the possibility of primary skin disease cannot be ruled out.

Because skin disease can reflect internal disease, appropriate laboratory tests or radiographic examination may be indicated in cases where a thorough examination has ruled out primary disease of the skin or feathers. (See Chapter 15, Evaluating and Treating the Liver and Chapter 4, Nutritional Considerations).

Table 13.1 | Diagnostics for Avian Skin Lesions

- Skin scraping
- Gram's stain of superficial skin scrape
- Culture of skin scrape
- Gram's stain of follicle
- Culture of follicle
- CBC
- Biopsy of affected skin/feather follicles
- Serum biochemistries, including bile acids
- Consider radiographs
- Viral DNA test

Table 13.2 | Therapy Pending Diagnostic Results

Clinical Presentations	Therapy
Feather loss or skin abnormality with no self-trauma	• Dietary assessment and correction • Environmental assessment and correction • Preliminary therapy based on Gram's stain results, if applicable • Viral profiling with supportive care
Pruritus	• Topical therapy (see Chapter 9, Therapeutic Agents) • Systemic antipruritic/tricyclic antidepressant (eg, diphenhydramine, amitriptyline, see Chapter 9, Therapeutic Agents) • Dietary assessment and correction • Environmental assessment and correction • Preliminary therapy based on Gram's stain results, if applicable • Behavior consultation
Self-mutilation	• Barrier to mutilation (Elizabethan collar or modified extension collar) • Antibiotics for systemic infection • Topical antibiotic/antifungal (eg, 1% silver sulfadiazine cream[a]) • Preliminary therapy based on Gram's stain results, if applicable • Consider psychotropic medications (see Chapter 3, Concepts in Behavior and Chapter 9, Therapeutic Agents) • Behavior consultation

Congenital and Acquired Malformations

Occasional feather cysts are seen in all species. In some

Courtesy Exotic DVM

Fig 13.2 | Feather cyst containing concentrically laminated keratin that must be differentiated from caseous exudate.

canaries there is an apparent inherited predisposition that is associated with color. Neoplasia has recently been found in the formation of feather follicle cysts in canaries.

Grossly, feather cysts present as an oval or elongated swelling of the feather follicle with accumulation of yellow-white material (keratin) (Fig 13.2). The gross lesions must be differentiated from follicular infections. The causes of acquired feather cyst formation are usually not determined but can include infection, trauma or any condition that interferes with normal growth of the implicated feather.

Resection of a feather follicle cyst is indicated in the presence of self-trauma or recurrent infection. (See Chapter 35, Surgical Resolution of Soft Tissue Disorders for this procedure).

Congenital or developmental beak abnormalities are encountered with some frequency. Improper incubation or feeding techniques have been implicated but have not been documented as causative. The two most common presentations are mandibular prognathism and scissors beak. (See Chapter 14, Evaluating and Treating the Gastrointestinal System for correction of beak deformities).

Abnormalities of the beak or claws can be a reflection of abnormalities of the underlying bone. They can also result from trauma, infection or neoplasia interfering with growth of the germinal epithelium of the beak or claw keratin. The result can be asynchronous growth or incomplete keratinization. Vitamin deficiencies that cause problems in domestic poultry are not well documented in pet avian species. Hepatopathy has been linked to beak and nail deformities in psittacines, but whether this is a direct result of the hepatic insufficiency or a sequela to nutritional disease is not well documented.

See Chapter 6, Maximizing Information from the Physical Examination and Chapter 15, Evaluating and Treating the Liver for photos of feather, beak and nail deformities.

Infectious Diseases

PARASITIC

The primary parasitic skin disease is mite infestation. Several different types of mites are found affecting both feathered and unfeathered skin. Most of these parasites are present in the superficial portion of the skin, which is usually hyperkeratotic and acanthotic, leading to gross thickening, irregularity and flaking. Severe and/or chronic infestation of the cere can result in malformation of the beak (Fig 13.3a).

Mites are usually superficial and can be demonstrated by skin scraping. Some species of mite and some individual cases will require deep scrapings or biopsy to identify.

Knemidocoptes spp. is most prevalent in budgerigars and passerines. The presentation in budgerigars is usually a pronounced hyperkeratosis of the cere and adjacent tissue. Occasionally the vent and legs of budgerigars will be affected (Fig 13.3b). A fine pinhole appearance of affected tissue on the cere is typical with this mite infestation. Clinical disease seems to require some degree of immune compromise.

Passerines with *Knemidocoptes* spp. generally present with "tassel foot." This hyperkeratosis of the legs is often accompanied in chronic cases with a curling and overgrowth of the nails.

Ivermectin has been utilized topically, orally and via injection for the treatment of mites, including *Knemidocoptes* spp. (See Chapter 9, Therapeutic Agents). In budgerigars that are otherwise clinically healthy, the infestation commonly clears, although recurrence is possible. Passerines with *Knemidocoptes* spp. infestation often improve but may not clear with ivermectin therapy. This may be due to secondary staphylococcal or mycotic infections.

Lice are uncommon in well cared for pet birds. See Chapter 6, Maximizing Information from the Physical Examination for photos. Unless the infestation is severe, gross lesions are not seen. Treatment with ivermectin is generally effective, although pyrethrin and carbaryl powders are also used successfully.

MYCOTIC

Folliculitis due to dermatophytes appears to be less common in birds than its counterpart in mammals, based on biopsy material. When present, there may be gross swelling of follicles with variable hyperkeratosis and crust formation (Fig 13.4). A variable amount of necrotic debris may be seen.

Courtesy Exotic DVM

Fig 13.3a | Roughened, inflamed cere and face due to *Knemidocoptes* spp. mite infestation.

Greg J. Harrison

Fig 13.3b | A close up view of a *Knemidocoptes* spp. mite infestation showing the characteristic pin-point tunnels in the skin that can be used to make the diagnosis.

Courtesy Exotic DVM

Fig 13.4 | Swelling of follicles in a bird with dermatomycosis.

Teresa Lightfoot

Fig 13.5 | Amazon with *Malassezia* spp. facial dermatitis.

Recent research indicates that *Malassezia* spp., *Aspergillus* spp. and other fungi may play a role in some cases of dermatitis or feather picking. Clinical reports of improvement in feather plucking following nebulization with antifungal agents for respiratory disease lend credence to this possibility. (M. Stanford, personal communication, August, 2001). Further research is needed to determine whether fungal infection or sensitivity to *Aspergillus* spp. may play a role in dermatitis and feather picking.

Malassezia spp. is occasionally found as an etiologic agent, generally documented on cytology or histopathology, for feather loss and dermatitis. Treatment is largely anecdotal and follows the sensitivities of this organism noted in other species. Oral fluconazole and topical clotrimazole or chlorhexidine spray have been used with good results. This may be an under-reported syndrome related to feather destructive behavior (Fig 13.5).

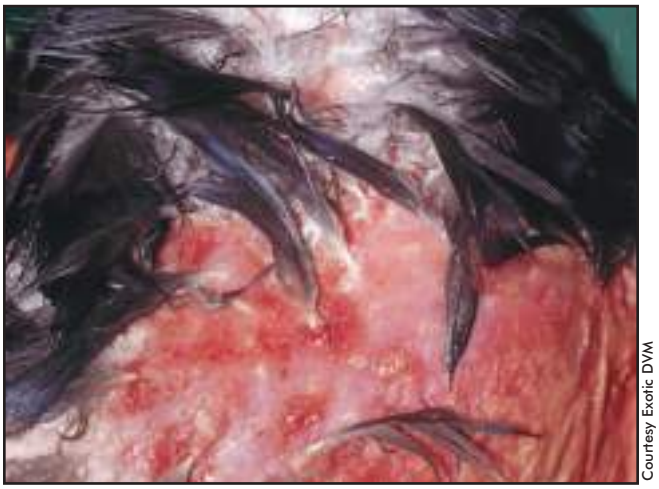
Saprophytic fungi have been noted to cause black discoloration of feathers in birds. The prevalence of this type of fungal growth is unknown but it seems most likely to occur in birds with marginal hygiene and/or health.

BACTERIAL

Two primary forms of bacterial skin disease are commonly seen. Folliculitis is often associated with *Staphylococcus* spp. Grossly there is swelling of the perifollicular skin with a variable amount of reddening. The lesion must be differentiated from mycotic folliculitis.

Generalized bacterial dermatitis (pyoderma) is usually intensely pruritic leading to self trauma that results in a more severe superficial lesion. Reddening, exudation and crust formation are associated with necrosis (Fig 13.6). The necrosis may extend through the epidermis into the dermis in severe cases. Bacteria, usually gram-positive cocci, may or may not be present in samples taken for microscopic examination.

Long-term antibiotic therapy is often needed in these cases. A positive culture and sensitivity will allow the selection of the appropriate antibiotic. A Gram's stain performed at the time of culture may improve interpretation of the culture results. In the absence of a positive culture, treatment may be selected based on the common sensitivities of the class of organisms identified in



Courtesy Exotic DVM

Fig 13.6 | Generalized bacterial dermatitis leading to necrosis, reddening and crust formation.

Courtesy Exotic DVM

Fig 13.7 | Localized periocular inflammation and minimal feather loss in a lovebird with circovirus infection.

the Gram's stain. Treatment failures are often the result of either continued self-trauma or insufficient length of antibiotic therapy.

A specialized form of bacterial dermatitis is severe chronic-active pododermatitis. (See Bumblefoot/ Pododermatitis under Non-Infectious Diseases).

Focal granulomatous dermatitis due to mycobacterial infection is also seen. Clinically, the lesion presents as a lump or multiple lumps that histologically are comprised of large macrophages and a variable number of heterophils and plasma cells. Acid-fast bacteria are found in the macrophages.

VIRAL

Circovirus

Psittacine beak and feather disease virus (PBFDV) is one of several avian circoviruses. This virus is enzootic in many species of free-ranging Australian parrots and has also been found in free-ranging African parrots.

PBFDV in nestlings is acute in onset and generalized so that it affects all growing feathers. Acutely affected birds may die within 2 months of the onset of disease. The chronic form of disease is generally seen in older birds when these birds go through their first molt. Dystrophic feathers replace normal ones during the molt. Powderdown feathers may be the first affected in cockatoos (*Cacatua* spp.).

Currently, PBFDV in the United States is most commonly seen in lovebirds (*Agapornis* spp.), budgerigars, lories, lorikeets, *Eclectus* spp. and African grey parrots (*Psittacus erythacus*). Feather lesions in lovebirds are usually not as severe as in cockatoos and may be localized (Fig 13.7). Some lovebirds show no signs of disease.

One study was conducted on 32 peach-faced (rose-faced) lovebirds (*Apapornis roseicollis*) with skin and feather problems.[6] Birds with chronic ulcerative dermatitis (CUD), the feather-less syndrome (FLS) or polyfolliculitis (PF) were screened for avian polyomavirus (APV) and psittacine beak and feather disease (PBFD). Of the birds with CUD, greater than fifty percent were positive for APV, and approximately 20% were positive for PBFDV. Of the birds with FLS, 16% were positive for APV and 65% were PBFD positive. All birds with PF were negative for APV and PBFD. The history of all of these birds also indicated malnutrition (Harrison/Gerlach, personal communication).

A generalized feather disease is seen in African grey parrots infected with circovirus, but often the disease is confined to the tail feathers, or there may be no feather involvement at all. African grey parrots may show ectopic red feathers; however, this abnormal coloration may also be caused by nutritional factors.

Eclectus parrots do not show typical feather lesions of PBFDV, but affected birds may have a delayed molt and old, poor quality feathering. An older age of onset of clinical signs of circovirus has been noted in *Eclectus* spp.

Infection in cockatoos leads to deformed feathers, feather loss and variable skin lesions. Beak lesions are less common than feather changes but are a prominent feature of this disease in some species of *Cacatua*. Variable necrosis and loss of keratin can be seen. Secondary candidiasis of the beak is common in affected cockatoos (Fig 13.8).

Necrosis and annular constriction of the base of the feather shaft and hemorrhage in the feather pulp are noted. There may be severe shedding of affected feathers. Affected feathers are stunted and may have thickened, hyperkeratotic sheaths, pulp hemorrhage, annular


Courtesy Exotic DVM

Fig 13.8 | Severe feather loss and dystrophy as well as beak necrosis in a cockatoo with circovirus infection.


Courtesy Exotic DVM

Fig 13.9 | Detail of feathers from Fig 13.8.


Judith St. Leger

Fig 13.10 | Columbiforme circovirus.


Judith St. Leger

Fig 13.11 | Columbiforme circovirus.


Judith St. Leger

Fig 13.12 | Columbiforme circovirus.


Judith St. Leger

Fig 13.13 | Columbiforme circovirus.

constrictions of the calamus, curling or stress lines on the vanes (Fig 13.9). Discoloration of feathers may be the initial sign in some birds. As mentioned above, African grey parrots may develop red feathers, and yellow feathers have been seen to replace green feathers in other species of parrots.

Gross lesions of circovirus infection are usually not seen in non-psittacine birds; however, feather dystrophy similar to that seen in psittacines has been reported in pigeons, doves and finches (Figs 13.10-13.13).

Polyoma/Papilloma Virus

Papilloma virus can cause proliferative skin lesions that are multiple and may superficially resemble mite infestation (Fig 13.14). This has been confirmed in African grey parrots. The lesions are fronds of hyperplastic epithelial cells supported by a vascular stroma. Radiosurgery, electrocautery and cryosurgery have all been utilized to resect the papillomas and to attempt to stimulate an immune response.

Teresa Lightfoot

Fig 13.14 | Viral-induced papillomas on the face of an African grey parrot.

Polyomavirus was originally reported as a disease of budgerigars with feather loss. Primary feathers may appear abnormal. Polyomavirus infection is also seen in other psittacine species and grossly there may be dermal/follicular hemorrhage. See Chapter 32, Implications of Viruses in Clinical Disorders.

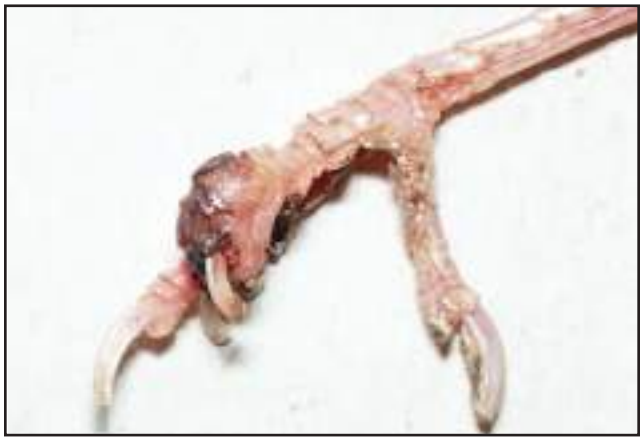
Poxvirus

This is an ubiquitous viral infection seen in all avian species. Fortunately the pox virus is relatively species-specific. Lesions are common on the head face and feet, but can also be present in other locations (Fig 13.15). The lesions are proliferative and may have rough or smooth surfaces depending on chronicity, self-trauma and the degree of secondary bacterial infection. In some cases much of the superficial portion of the lesion can be comprised of necrotic debris and crusts associated with bacterial or yeast infection, and care must be taken to ensure that any material removed for biopsy or cytology contains epidermal tissue (Fig 13.16). If no epidermis is present the correct diagnosis will probably not be made. Impression smears will contain epithelial cells with ballooning degeneration and cytoplasmic inclusion bodies (Fig 13.17).

The severity and location of lesions will dictate whether euthanasia is indicated or if treatment should be

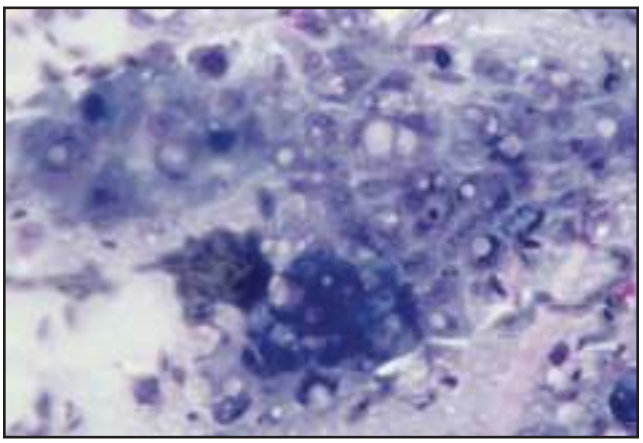
Courtesy Exotic DVM

Fig 13.15 | Facial lesions due to poxvirus infection in a canary.



Courtesy Exotic DVM

Fig 13.16 | Typical poxvirus-induced lesions of the leg and toes in the canary from Fig 13.15.



Courtesy Exotic DVM

Fig 13.17 | Impression smear of proliferative epidermis in poxvirus infection. Note ballooning degeneration and cytoplasmic inclusion bodies.

Courtesy Exotic DVM

Fig 13.18 | Depigmented, proliferative lesion (arrow) associated with cytomegalic herpesvirus infection of the skin of a blue and gold macaw.

Courtesy Exotic DVM

Fig 13.19 | Color change in feathers secondary to nutritional problems, possibly a carotene deficiency.

Courtesy Exotic DVM

Fig 13.20 | Stress bars in growing feathers. This is a nonspecific change that can be associated with a variety of insults during feather formation.

attempted. Despite supportive care, permanent deformity of eyelid margins and other facial tissue is common.

Herpesvirus

In cases of systemic herpes infection there is occasionally involvement of the epidermis of the skin or feather leading to necrosis and inclusion body formation. Since the generalized disease is usually catastrophic, little attention is paid to what may be grossly minimal skin lesions. In some psittacines, particularly macaws and cockatoos, proliferative lesions of the lower legs and feet have been described due to a herpes virus infection. Solitary or multiple proliferative nodules or plaques are more common in *Cacatua* spp., while depigmentation is more often encountered in macaws (Fig 13.18). The presence of these lesions in susceptible species should lead to herpes virus infection being included in the differential diagnosis.

Non-Infectious Disease

NUTRITIONAL/METABOLIC

A number of specific and non-specific nutritional problems can result in poor feather quality and skin disease. This may be the most common cause of primary feather abnormalities. See Chapter 6, Maximizing Information from the Physical Examination.

Depigmentation or altered pigmentation, improper molting and poor quality feathers can be seen (Figs 13.19, 13.20) (see Chapter 4, Nutritional Considerations). Gross changes are rarely specific. These lesions are not inflammatory, but poor nutrition can predispose the bird to skin infections and subsequent inflammation.

Metabolic disease could also result from failure of proper nutrient metabolism even though nutrition is adequate. Gastro-intestinal, hepatic and pancreatic diseases are potential underlying causes. The diagnostic approach to chronic non-inflammatory skin disease should include examination and laboratory testing to rule out disease processes in internal organs.

PHYSICAL/ENVIRONMENTAL AGENTS

Trauma, burns, excessive cold and other physical factors often cause skin lesions, and although the cause may be obvious, histories are occasionally not obtained (Tables 13.3-13.5). Gross changes include loss of feathers, varying degrees of hemorrhage, necrosis, and superficial crust formation. Severe necrosis and sloughing of epidermis and possibly portions of dermis can be seen in injuries due to both heat and cold. Discoloration of the lesions is variable. Traumatic injuries are characterized by variable amounts of hemorrhage, edema and inflammation, depending on severity of the insult and time elapsed prior to examination (Figs 13.21, 13.22).

Beak trauma is a common presentation in psittacines. Injury from a bite from another bird is the most frequent cause. Damage from cage wires or cage equipment is also common.

Treatment and prognosis depend entirely on the severity of the injury. If proper beak occlusion is maintained, then treatment can be limited to prevention of infection. Topical and systemic antibiotics are warranted if the injury sustained is extensive or deep.

A hemostatic matrix such as Surgicell®[d] can be used to both stop bleeding and to provide a slow release of antibiotic. Antibiotics that are used in polymethyl methacrylate applications should provide a selection that is not tissue toxic and has good bioavailability (see

Courtesy Exotic DVM

Fig 13.21 | Subcutaneous hemorrhage secondary to trauma.

Courtesy Exotic DVM

Fig 13.22 | Severe edema of the subcutis following trauma.

Table 13.3 | Thermal Burn Treatment Protocol

- Stabilization of patient first:
  1. Fluids, electrolytes
  2. Treat for potential septicemia/endotoxemia
- Topical treatment if weight bearing surfaces affected (ie, plantar surfaces of feet)
- Bandaging if potential exists for self-mutilation of affected area

Table 13.4 | Treatment of Band Injuries
(Figs 13.23-13.25)

- Removal of band with minimal additional tissue damage (ie, Veterinary Specialty Products band cutters)[b]
- Assessment of distal foot for viability.
- Hydroscopic dressing to preserve tissue, vascularity and innervation (ie, Biodres®)[c]
- Antibiotics as indicated for prevention of infection; both topical and systemic
- Prevention of self trauma and frequent reassessment for continued viability and absence of infection.

Table 13.5 | Broken Blood Feather Treatment

- Keep quiet and confined to allow blood pressure to lower and bleeding to stop.
- Apply a styptic powder to the broken feather area or twist off and apply to the tip.
- If occurs in the hospital, observing the bird for a few hours after powdering is often indicated to prevent excitation and subsequent bleeding in transport.
- When danger of hemorrhage is no longer present, assess feather damage.
- May trim end of feather to decrease movement or pain.
- Pulling of affected feather may result in follicular damage and abnormal growth of subsequent feather.
- Imping may be indicated for cases of chronic/repeat trauma.
- Long-term treatment for recurrent blood feather trauma,
  1. Wing trim should be redesigned (see Chapter 1, Clinical Practice).
  2. Nutrition should be assessed (see Chapter 4, Nutritional Considerations).

Chapter 9, Therapeutic Agents).

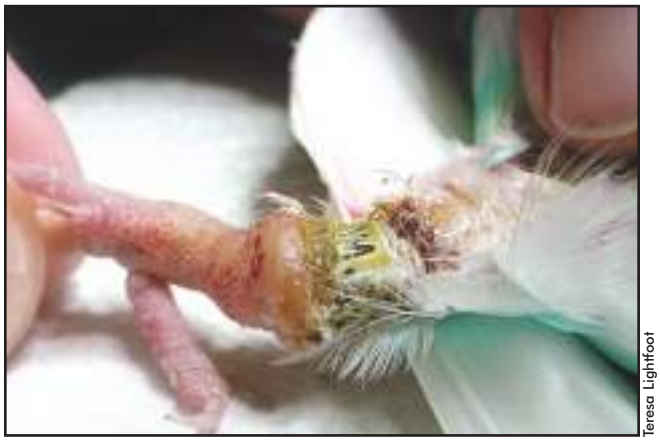
More extensive trauma that either involves the occlusal surfaces or the growth plates of the beak warrants a guarded prognosis (see Chapter 14, Evaluating and Treating the Gastrointestinal System). Attention must be paid to adequate supportive care including analgesia, and maintenance of fluid and caloric intake.

BUMBLEFOOT/PODODERMATITIS: DECUBITAL SORES

Plantar decubital ulceration is common in older, obese and nutritionally deficient psittacines. See photos and classification of fat deposition in Chapter 6, Maximizing Information from the Physical Examination. Amazons, budgerigars and cockatiels are over-represented in the current population. Vitamin A deficiency weakens the epithelium of affected birds (see Chapter 4, Nutritional Considerations). Obesity and inactivity produce excessive pressure on plantar surfaces. Subsequent erosions and then ulcers occur. Localized staphylococcal infection is a common sequela (Fig 13.26).

Presentation may be subclinical and encountered on a routine annual examination. Correction of the underlying predisposing factors will often reverse this disease process. Perches must be altered in diameter and texture. The application of Vetrap®[e] or a similar product to the perch provides both padding and change in diameter when the material is wrapped at varying intervals and thicknesses. Diet should be corrected to decrease caloric intake and increase general nutritional balance, with emphasis on replacement of Vitamin A precursors (see Chapter 4, Nutritional Considerations).

More advanced cases of decubital ulceration require additional therapy. Systemic infection may be involved, and a complete blood count should be performed. Bandaging of the feet with the application of topical

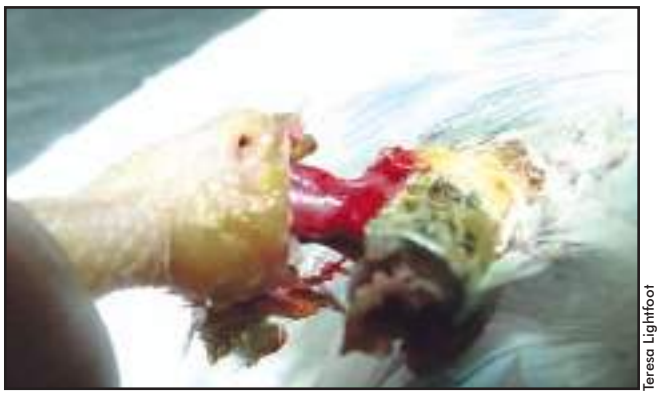


Teresa Lightfoot

Fig 13.23 | Leg band injury. Aluminum breeder band is embedded in skin and underlying tissue.

Teresa Lightfoot

Fig 13.24 | Leg band injury. Removal with the appropriate equipment is necessary to prevent fracturing the leg. In this case, band cutters by Veterinary Specialty Products, Boca Raton, FL, USA, were used.



Teresa Lightfoot

Fig 13.25 | Leg band injury. Although minimal viable tissue remains beneath the removed band, the innervation and circulation to the foot are still intact. Frequent bandage changes allowed this area to granulate and amputation was avoided. However, many band injuries of this severity will require amputation and the owners should be so forewarned.

Courtesy Exotic DVM

Fig 13.26 | Bacterial pododermatitis. This lesion usually develops following pressure necrosis with a subsequent bacterial infection.

antibiotic and sufficient padding to reduce and better distribute pressure on the plantar surfaces is required in many cases. Pain relief in the form of NSAIDs (nonsteroidal antiinflamatory drugs) or synthetic opioids may be needed (Table 13.6) (see Chapter 9, Therapeutic Agents). Debridement should be approached cautiously, since significant bleeding can occur from the decubitus.

Table 13.6 | Treatment of Decubital Sores (Bumblefoot)

- Topical antimicrobials
- Hydrophilic dressings
- Padded foot bandages
- Anti-inflammatory/analgesics (ie, butorphanol/meloxicam)
- Systemic antibiotics when indicated
- Consider use of antibiotic impregnated matrix
- Debridement and suturing of more extensive lesions
- Long-term treatment requires owner compliance
  1. Alter/pad perches
  2. Exercise
  3. Assess and alter diet with particular attention to correcting obesity and providing adequate Vitamin A precursors

(See Chapter 34, Surgical Resolution of Orthopedic Disorders and Chapter 4, Nutritional Considerations).

When osteomyelitis is involved, the prognosis for recovery decreases dramatically. If systemic infection and pain can be controlled, therapy can be approached as above. The owner must be forewarned that the therapy will be of long duration and the prognosis is guarded. Ethical considerations arise when the degree of affectation is such that the bird can not stand without severe pain.

Endocrinopathies

Endocrine disorders can lead to generalized feather loss and abnormal feathering. There is usually no specific pattern or features that grossly indicate endocrine disor-

Courtesy Exotic DVM

Fig 13.27 | Excessive fat deposits in the skin of a bird with hypothyroidism.

ders. To confirm a diagnosis of endocrine related skin disease, appropriate clinical laboratory testing is necessary. Confirmation can also result from finding appropriate endocrine gland lesions at necropsy (Fig 13.27). Although currently Thyroid Stimulating Hormone (TSH) for avian thyroid stimulation assays is not commercially available, research has shown that a 2 to 4 fold increase in circulating T_4 is a normal response in birds to administration of TSH. Interpretation of a baseline T_4 level has limitations as it does in domestic pet medicine, but may be useful diagnostically (see Chapter 19, Endocrine Considerations).

HYPERSENSITIVITY

Allergic skin disease in birds is occasionally reported, but is not well documented, and confirmation can be difficult. Gross changes include feather loss (often self-induced), reddening and occasionally, surface exudates. Some of the gross lesions may be secondary to self trauma.

Periocular and occasionally periaural pruritic, hyperkeratotic lesions are observed seasonally in outdoor birds in the southeastern US. When a biopsy is performed and these birds are housed indoors pending receipt of histopathology results, the lesion generally clears. Both pollen and insect sensitivity have been theorized.

Definitive diagnosis of allergic skin disease is difficult. Food elimination has lead to improvement in some cases (see Chapter 4, Nutritional Considerations: Section II, Nutritional Disorders) and successful treatment with anti-inflammatory drugs is presumptive evidence of allergy. The greatly diminished response of the avian patient to histamine administration has hindered the development of avian skin testing methods. Recent research has established positive and negative controls and preliminary standards for this testing.[8] Diagnostic skin testing for avian patients may be of great benefit in separating this category of disease from other conditions.

According to Patricia MacWhirter, DVM (personal communication, December 2003) an early researcher into avian intradermal testing: "Intradermal skin testing can be carried out in birds using the apteria on either side of the sternum. A statistically significant difference has been found in the occurrence of positive intradermal skin test reactions to Aspergillus, sunflower, house dust mites (*D. pteronyssinus* and *D. farinae*) and/or maize (corn) in a variety of psittacine species showing evidence of feather plucking, feather chewing or self injurious behavior compared with normal birds. This suggests that allergy may play a role in the occurrence of these syndromes. However, response to treatment by attempted avoidance of the suspected allergen(s) or the use of vaccines has to date often not been successful. Skin testing can be problematic to carry out because of the need for fresh allergens and accurate injection, the small area of bare skin available and difficulties in getting consistent results with positive controls. While promising, the technique is probably best suited to specialist dermatology practices and more research is needed before it can be routinely recommended." See Chapter 4, Nutritional Considerations.

Chronic Internal Disease

In many cases of chronic internal disease, including infectious, degenerative and neoplastic conditions, there is poor feather quality and loss of feathers.

NEOPLASIA

(See Chapter 20, Overview of Tumors).

Epithelial

Epithelial tumors originate in the surface epithelium, follicular epithelium or the uropygial glands. The uropygial gland may become abscessed as a result of occlusion of the papilla. This condition is treated much like an anal sac abscess in a dog, with debridement, reestablishment of patency of the duct and antibiotics as indicated. In some cases, neoplasia of the gland may underlie the infected state. Uropygial gland tumors can be either adenomas or carcinomas, and gross differentiation is difficult. Both will present as swellings that may be secondarily inflamed in some cases. Adenomas are usually well circumscribed and encapsulated with carcinomas being less differentiated and more infiltrative into surrounding tissue. See Chapter 35, Surgical Resolution of Soft Tissue Disorders for surgical considerations.

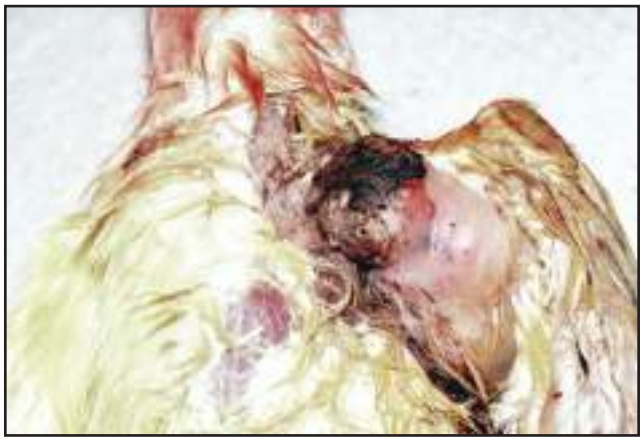

Courtesy Exotic DVM

Fig 13.28 | Aggressive squamous cell carcinoma with loss of normal skin and severe secondary inflammation.


Courtesy Exotic DVM

Fig 13.29 | Large mass typical of subcutaneous lipoma.

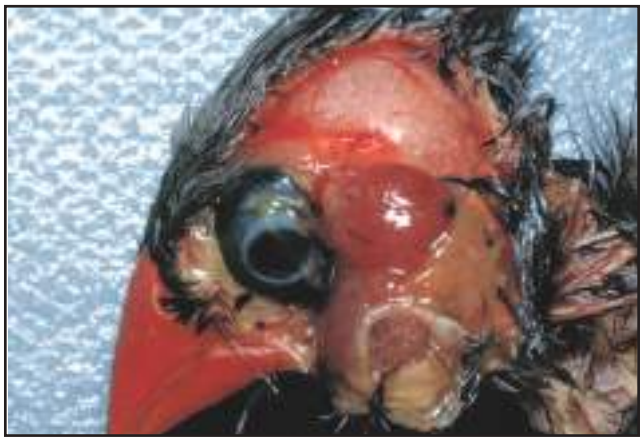

Courtesy Exotic DVM

Fig 13.30 | Circumscribed red mass consistent with hemangioma.


Courtesy Exotic DVM

Fig 13.31 | Deeply located fibrosarcoma replacing soft tissue and bone.

Papillomas of the skin are not common and may be virally induced in African grey parrots (see previous discussion).

Squamous cell carcinomas are often ulcerated and hemorrhagic as well as infiltrative (Fig 13.28). They may involve any portion of the skin and no particular site predilection has been identified. In some cases there is no obvious ulceration or inflammation in the early stages. Metastasis is not common, but occurs, particularly in chronic cases. This neoplasia often appears grossly as a delayed or non-healing cutaneous infection, and diagnosis is therefore often delayed.

Basal cell tumors often originate in feather cysts, and although expansile, are usually benign.

Mesenchymal tumors include those of vascular, fibrous, adipose and connective tissue origin. These tumors originate in the dermis or subcutis but may expand to involve the epidermis with secondary ulceration. Gross differentiation can be difficult with malignant tumors. Lipomas are common and have the gross appearance of a mass of normal fat (Fig 13.29). Hemangiomas are often dark red and hemorrhagic. They must be differentiated from melanomas (Fig 13.30).

Fibromas and fibrosarcomas may both be seen but the later are more common. They present as nodular masses that may be ulcerative and infiltrative into deep tissues (Fig 13.31).

Dermal lymphosarcoma may present as a diffuse thickening of the skin with loss of feathers. This condition can be misdiagnosed as chronic resistant inflammation unless biopsied.

Melanocytic Tumors

Melanoma has been diagnosed in several psittacine birds. The tumor is not common and is usually malignant. These tumors often occur on the face and may involve the beak. They are brown-black, raised masses with poorly defined margins (Fig 13.32).

Mast cell tumors have only been reported in chickens and owls.

Granular cell tumors are infrequent in birds, and are seen primarily in psittacine birds, particularly Amazon

Courtesy Exotic DVM

Fig 13.32 | *Eclectus* spp. female with malignant melanoma of the face and cere.

Courtesy Exotic DVM

Fig 13.33 | Xanthoma that has replaced much of the wing. This is a common location for the condition.

Teresa Lightfoot

Fig 13.34 | Seven year old male Eclectus with xanthoma. This bird had been feather picking for 5 years. Hormonal manipulation and psychotropic drugs had temporarily decreased his plucking, but were not curative. When the xanthoma developed, dietary change to an organic formula resulted in resolution of the xanthoma and decreased feather destructive behavior.

Teresa Lightfoot

Fig 13.35 | Same Eclectus after 9 months of dietary correction, with no additional therapy.

parrots. They are small smooth nodules. (See Chapter 20, Overview of Tumors).

Non-neoplastic Proliferative Lesions

Xanthomatosis is a condition of uncertain etiology. Xanthomas are seen most commonly in cockatiels and budgerigars and usually are present on the wing as a variable-sized, yellow mass (Fig 13.33). Alternate common presentation sites include the sterno-pubic area and the keel. Surgical resection may be necessary in advanced cases and in those where the affected area is traumatized. In some species and some cases, nutritional therapy has been reported as successful. Feeding a balanced diet with increased Vitamin A precursors is the predominant dietary change initiated in the therapy of affected birds (Figs 13.34, 13.35).

Feather Destructive Behavior

Various degrees of feather destructive behavior, from over-preening to feather plucking and self-mutilation, are commonly encountered in avian practice. Based on skin biopsies, many of these cases have an underlying lesion that would account for pruritus and self-trauma. In some birds there is no evidence of skin or systemic disease or condition and these cases are considered behavioral problems after other causes have been ruled out. Since self-trauma can lead to lesions, histologic changes must be carefully assessed before a diagnosis of behavioral feather picking is made. In addition to complete physical and laboratory examination, history is very important for a proper diagnosis of this condition. (See Chapter 3, Concepts in Behavior and Chapter 4, Nutritional Considerations).

Skin Conditions

Several syndromes with no identified etiologies are commonly recognized by practitioners. These include chronic ulcerative dermatitis, Quaker (Monk) parakeet (*Myiopsitta monachus*) mutilation, and Amazon foot necrosis.

CHRONIC ULCERATIVE DERMATITIS

Chronic ulcerative dermatitis (CUD) is commonly reported in lovebirds and presents as self-trauma. The affected area is usually the patagium or neck and back. A linear lesion is generally encountered, and the bird often presents with either a chronic scarified area or with an acutely lacerated and hemorrhagic wound. As discussed under viruses, recent research on a small population indicates that polyoma virus, circovirus or both may be involved in this syndrome.[6] The finding of a viral etiology in some cases of chronic ulcerative dermatitis in lovebirds would be consistent with reports of flock outbreaks of this condition. Other cases seem to occur in isolated individuals. Antibiotics are often clinically useful in controlling what is likely a secondary bacterial infection. Elizabethan collaring may be necessary to prevent self-mutilation and blood loss. Even when the primary lesion is healed, scar tissue often restricts movement and recurrence of self-mutilation is the rule. Some practitioners have associated Omega-3 fatty acid supplementation with clinical improvement. Use of psychotropic drugs and/or antihistamines has been reported with equivocal results.

QUAKER MUTILATION SYNDROME

A syndrome in Quaker (Monk) parakeets has been noted for many years in which sudden and aggressive self-mutilation is encountered (Table 13.7). Feather destructive behavior does not seem to be a precursor to this syndrome. The mutilation is often directed at the neck and chest area. Self trauma can include fatal damage to the crop and the jugular vein. With no etiology yet determined, treatment is limited to providing a mechanical barrier to the self-trauma and supportive care. Due to the severity and chronicity of this syndrome, euthanasia is often elected. Increased submissions for pathology may identify an etiology. Theories of potential etiologies and/or associated conditions include: viral, obesity, hepatic lipidosis, pancreatic insufficiency and lipemia (M. Rae, personal communication, 2002).

AMAZON FOOT NECROSIS

Amazon foot necrosis has historically been more prevalent on the west coast of the USA than in other areas.

Teresa Lightfoot

Fig 13.36 | Bandaging for Amazon foot necrosis. Topical antimicrobial agents and hydrophilic bandage material may aid in healing. Bandaging both feet, even if only one is affected, tends to divert the patient's attention and prevent removal of the bandaging.

Table 13.7 | Treatment Protocol for Quaker Mutilation Syndrome

- E. collar often necessary to prevent severe/fatal self-mutilation.
- Be aware that self-mutilation may be displaced to an accessible body part.
- Diazepam or other anti-anxiety/anti-psychotic drug
- Antibiotic for secondary infection.
- Wound dressing as needed.
- Owners should be informed of guarded prognosis.

The potential for a contact dermatitis would suggest that prior to handling these birds the owners wash their hands to rid them of residual nicotine, hand lotions, etc. Inhalant hypersensitivity has been theorized. Nutritional deficiencies or toxicities and hormonal influences have also been suggested. A recurrence and seasonality is commonly reported (Fig 13.36).

POLYFOLLICULITIS

Follicular malformations and dystrophy are occasionally seen. The most recognized has been called "polyfolliculitis." This is a misnomer as in many cases there is no inflammation. The condition is seen in budgerigars, cockatiels and lovebirds and presents as multiple feather shafts from a single follicle. Feathers are thick and short and may have retained sheaths. Grossly they present as fluctuant subcutaneous swellings that contain slightly viscid fluid.

Calcinosis circumscripta is an unusual condition in birds. It presents as nodular lesions that may have a white, chalky appearance grossly.

OTHER SKIN CONDITIONS

Occasionally severe inflammation is seen associated with collagen necrosis. A severe granulocytic response is present, and many of these cells may be eosinophils, however they are difficult to distinguish from heterophils

histologically. The lesion is similar to idiopathic collagenolytic inflammation seen in several mammalian species.

Autoimmune skin disease has not been documented in birds, but several cases with intraepidermal pustule formation and acantholysis have been seen. Unfortunately these few cases were lost to follow-up.

In many skin diagnoses there are inflammatory lesions whose exact etiology cannot be determined. Based on the pattern and type of inflammation a tentative diagnosis may be made, but until many more cases with complete histories and follow-up information become available, many lesions will have obscure origins.

Products Mentioned in Text

a. Silvadene, Marion Labs, Inc., Kansas City MO
b. Veterinary Specialty Products Bandcutter, PO Box 812005, Boca Raton, FL, USA, 33481, 1-800-362-8138, www.vet-products.com
c. BioDres, DVM Pharmaceuticals, Miami, FL, USA, www.dvmpharmaceuticals.com/about_dvm.html
d. Surgicell®, Johnson & Johnson's, www.jnjgateway.com
e. Vetrap - 3M Animal Care Products, St. Paul, MN, USA, www.3m.com

References and Suggested Reading

1. Andre JP, Delverdier M, Cabanie D, Bartel G. 1993. Malignant melanoma in an African grey parrot. JAAV 7: 83-85.
2. Brush, AH 1993. The origin of feathers: A novel approach. Avian Biology, Farner, DS, et. al. Eds. Vol. IX. New York, Academic Press, pp 121-11162.
3. Chitty J, A novel disinfectant in psittacine respiratory disease, AAV Proceedings 2002, p 25-27.
4. Clubb SL, Herron A, Feather discoloration due to saprophytic fungal growth, Proc Annual AAV, 1998, p 71-76.
5. Cooper JE, Harrison GJ: Dermatology. *In*: Ritchie BW, Harrison GJ, Harrison LR, (eds): Avian Medicine: Principles and Application. Brentwood, TN: HBD Int'l, Inc, p 613-621.
6. Cornelissen JMM, Gerlach H, Miller H, et al: An investigation into the possible role of circo and avian polyoma virus infections in the etiology of three distinct skin and feather problems (CUD, FLS, PF) in the rose-faced lovebird (*Agapornis roseicollis*). Proc Euro Col Avian Med and Surg, 2001, p 3-5, Munich, Germany.
7. Ferrer, L, Ramis, A, Fernandex, J, Majo, N 1997. Granulomatous dermatitis caused by a *Mycobacterium genavense* in 2 psittacine birds. Vet. Dermatol. 8: 213-219.
8. Foil C, Daigle J, Heatley J, Daigle J, Tully TN: Intradermal skin testing in Amazon parrots: establishing a protocol, Proc Annual Conf AAV, 2001, p 103-105.
9. Garcia A, Latimer KS, Niagro, FD, Norton, TM, et al. 1993. Avian polyomavirus infection in three black-bellied seed crackers (*Pyrenestes ostrinus*). JAAV 7: 79-82.
10. Grahm DL, 1985. The avian integument. Proc. AAV, Boulder, pp 33-52.
11. Hadley NF, 1991. Integumental lipids of plants and animals-comparative function and biochemistry. Advances in Lipid Res. 24: 303-320.
12. Jacobson ER, Mladinich CR, Clubb S, Sundberg FP, Lancaster WD. 1983. A papilloma-like virus infection in an African Grey parrot. JAVMA 183: 1307-1308.
13. Latimer KS. 1994. Oncology. *In*: Ritchie BW, Harrison GJ, Harrison LR, eds. Avian Medicine: Principles and Application. Brentwood, TN: HBD Int'l, Inc, pp 640-672.
14. Latimer KS, Niagro FD, Rakich PM, Campagnoli, RP, et al, 1992. Comparison of DNA dot-blot hybridization immunoperoxidase staining and routine histopathology in the diagnosis of psittacine beak and feather disease in paraffin-embedded cutaneous tissues. JAAV 6:165-168.
15. McDonald, SE, Lowenstine, LJ, Ardans, AA. 1981 Avian pox in blue-fronted Amazon parrots. JAVMA 179: 1218-1222.
16. Macwhirter P, Mueller R, Gill J, Ongoing research report: Allergen testing as a part of diagnostic protocol in self-mutilating psittaciformes. Proc AAV Annual Conf 1999, p. 125-129.
17. Pass, DA. 1989. Pathology of the avian integument: A review. Avian Path. 18: 1-72.
18. Patnaik, AK. 1993. Histologic and immunohistochemical studies of granular cell tumors in 7 dogs, 3 cats, one horse and one bird. Vet Pathol 30: 176-185.
19. Pizarro M, Villegas P, Rodriques A, Rowland GN. 1994. Filariasis (*Pelecitus* spp) in the cervical subcutaneous tissue of a pigeon with trichomoniasis. Avian Dis. 38: 385-389.
20. Pye GW, Carpenter JW, Goggin, JM, Bacmeister, C. 1999. Metastatic squamous cell carcinoma in a salmon-crested cockatoo (*Cacatua moluccensis*). J Avian Med. Surg. 13: 192-200.
21. Quist CF, Latimer KS, Goldade SL, Rivera A, Dein FJ. 1999. Granular cell tumor in an endangered Puerto Rican Amazon parrot (*Amazona vittata*). Avian Path. 28: 345-348.
22. Raidal SR. 1995. Viral skin diseases of birds. Sem. Avian Exot. Pet. Med. 4: 77-82.
23. Raidal SR, Riddoch PA. 1997. A feather disese in Senegal doves (*Streptopelia senegalensis*) morphologically similar to psittacine beak and feather disease. Avian Path. 26: 829-836.
24. Ramis, A, Latimer, KS, Niagro FD, Campagnoli, RP, et al. 1994. Diagnosis of psittacine beak and feather disease (PBFD) viral infection, avian polyomavirus infection, adenovirus infection and herpesvirus infection in psittacine tissues using DNA in-situ hybridization. Avian Path. 23: 643-657.
25. Ramis A, Latimer KS, Gilbert X, Campagnoli R. 1998. A concurrent outbreak of psittacine beak and feather disease virus and avian polyomavirus infection in budgerigars (*Melopsittacus undulatus*). Avian Path. 27: 43-50.
26. Rece RL. 1992. Observations on naturally occurring neoplasms in birds in the state of Victoria, Australia. Avian Path. 21: 3-32.
27. Ritchie BW, Harrison GJ, Harrison LR, (eds) Avian Medicine: Principles and Application. Brentwood, TN: HBD Int'l, Inc, 1997.
28. Ritchie BW, Niagro FD, Lukert PD, Latimer KS. et al. 1989. A review of psittacine beak and feather disease. JAAV 3: 143-150.
29. Samour J, Naldo J, Therapeutic Management of Pox Lesions on the Cere of Captive Falcons, Proceedings of the AAV Annual Conf, p. 233-235, 2002.
30a. Schmidt RE. Pathologic aspects of the skin and feathers. Chap. 26A in: Diseases of cage and aviary birds. Rosskopf WJ Jr, Woerpel, RW (Eds) 3rd Ed. Baltimore, Williams & Wilkins. pp 387-396.
30b. Schmidt RE: Pictorial guide to selected avian skin desseases. Exotic DVM 4(1): 27-32, 2002.
31. Schmidt RE. 1992. Morphologic diagnosis of avian neoplasms. Sem. Avian Exot. Pet Med. 1: 73-79.
32. Spearman RFC, Hardy J. 1989. Integument. Chapt. 1 *In*: Form and function in birds. King, AS, McLelland, J (Eds). Vol. 3. New York, Academic Press. pp1-52.
33. St. Leger J: Feather Dystrophy Associated with circovirus infection in columbiformes. In Proc of the 47th Western Poultry Disease Conference. March 8-10, 1998.
34. Tell LA, Woods LW, Mathews KG. 1997. Basal-cell carcinoma in a blue-fronted Amazon parrot (*Amazona aestiva*). Avian Dis. 41: 755-759.
35. Trinkaus K, Wenisch S, Leiser R, Gravendyck M, Kaleta EF. 1998. Psittacine beak and feather disease infected cells show a pattern of apoptosis in psittacine skin. Avian Path: 27 551-561.
36. Tsai SS, Chang SF, Chi YC, Cher RS, et al. 1997. Unusual lesions associated with avian poxvirus infection in rosy-faced Lovebirds (*Agapornis roseicollis*). Avian Path. 26: 75-82.
37. Van Sant F, Impression cytology: New insights into avian skin flora. Proc Annual Conf AAV, 1999, p. 139-141.
38. Welle K, Application of Imping feathers in psittacine birds. AAV Proc 1998.
39. Wheeldon DB, Culbertson MR Jr. 1982. Feather folliculoma in the canary (*Serinus canarius*). Vet Pathol 19: 204-206.
40. Woods LW, Latimer KS. 2000. Circovirus infection of nonpsittacine birds. J. Avian Med Surg 14: 154-163.

CHAPTER
14

Evaluating and Treating the Gastrointestinal System

STACEY GELIS, BSc, BVSc (Hons), MACVSc (Avian Health)

Greg J. Harrison

The avian gastrointestinal tract (GIT) has undergone a multitude of changes during evolution to become a unique anatomical and physiological structure when compared to other animal orders. On the one hand it has evolved to take advantage of the physical and chemical characteristics of a wide variety of food types.[1] On the other hand, it has had to do so within the limitations of the requirements for flight.[2] To this end, birds have evolved a lightweight beak and muscular ventriculus, which replaces the heavy bone, muscular and dental structure characteristic of reptiles and mammals. The ventriculus and small intestine are the heaviest structures within the gastrointestinal tract and are located near the bird's centre of gravity within the abdomen. The overall length of the GIT is also less than that of a comparable mammal, another weight-saving flight adaptation. Interestingly, these characteristics are still shared with the flightless species such as ratites and penguins. In addition, the actual digestive process needs to be rapid to support the high metabolic rate typical of flighted birds.[3]

Gastrointestinal adaptations to the wide range of ecological niches that birds occupy mean that birds can take advantage of a huge variety of foodstuffs. The GIT hence shows the greatest degree of diversity of all the organ systems between different avian taxa. However, the pressures of convergent evolution have also meant that many distantly related species have developed a similar gastrointestinal anatomy to take advantage of particular food niches.[3,4] Examples of these will be presented in the discussion of each section of the GIT.

The avian GIT also has the capacity to accommodate changes which occur during the life cycle of a bird and also which occur due to seasonal environmental conditions and hence differing available foodstuffs over the course of a year or years.

Anatomy and Physiology of the Digestive Tract

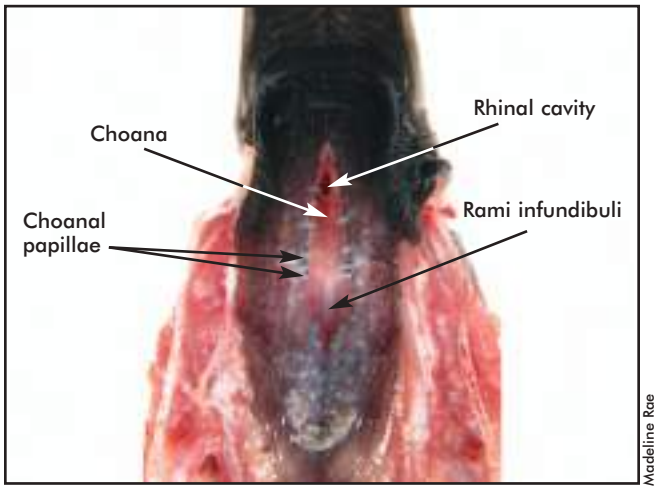
The avian gastrointestinal tract is a double-ended open tube (as is also seen in mammals) that begins at the beak and finishes at the vent. In sequential order it is composed of a mouth, esophagus, crop, proventriculus, ventriculus (gizzard), intestine, ceca, rectum and cloaca. Some of these structures may be vestigial or even lost during the evolution of some species. The progress of food through the tract follows a specific digestive sequence including premoistening and softening, acidifying, grinding, hydrolyzing, emulsifying and propulsion of the end products.[1] This propulsion is not always in a unidirectional pattern as will be outlined later.

BEAK, MOUTH, TONGUE AND PHARYNX

The beak or bill is the avian substitute for teeth and lips and forms the entrance to the oral cavity. It is used for grasping and processing foods, as well as for climbing and various behavioral functions such as biting, preening and displaying. It consists of the mandibular bones, the premaxilla and maxilla and their horny covering, the rhamphotheca. The upper bill covering is known as the rhinotheca, which covers the premaxillary bones and partly covers the maxillary bones. It is usually composed of hard keratin, although in waterfowl only the tip is hard, and in shorebirds the entire bill is relatively soft.[2] The keratin layer covering the lower bill or mandible is known as the gnathotheca. Both keratin layers are continually lost by wear and replaced by new growth. Beak shape is influenced by the location and rate of wear and hence regrowth, which is in large part determined by diet. This may subtly change over time as food types change. For example, the anterior of the outer edges of the beak, the tomia, may be sharp in some species to assist in cutting seed coats.[1] Other anatomical characteristics further facilitate the feeding process. The lower beak is loosely attached to the skull, allowing for a large gape. The size of the gape determines the maximum size of food particles that can be swallowed. This is particularly important in fruit-eating species such as toucans. Psittacines typically have a very powerful beak. The rhinotheca is broad with a curved rostral tip, giving the typical hooked appearance. The gnathotheca has a blunt chisel-shaped rostrum that pushes against a prominent ridge found on the undersurface of the rhinotheca. Psittacines also have developed a prokinetic maxilla that allows them to move their mandible and maxilla independently. This allows an increased gape of the beak, an improved ability to position food items in the beak, as well as providing flexion and shock absorption associated with seed and nut cracking and also with some behaviors such as pecking.[5] The strength generated by these structures is exemplified by the Hyacinth macaw's ability to crack palm nuts in order to extract the kernel. Pigeons, on the other hand, have a typical seedeater's bill being mildly conical.[6] It is also not as keratinized as in psittacines. Raptors tend to also have curved, hook-like bills but lack the prokinetic maxilla.[6] Their bill is adapted to tearing and shredding meat. "Darwin's Finches" on the Galapagos Islands best exemplify the variability in beak shape brought about by the need to adapt to changing environments. Here, the species are all similarly colored, but are separated on the basis of bill shape and feeding habits. Each has evolved to its own ecological niche, avoiding interspecific competition.[7] The avian bill is often endowed with sensitive nerve endings, particularly in species that use the bill to probe for food. Examples include waders, diving ducks and woodpeckers.[3]

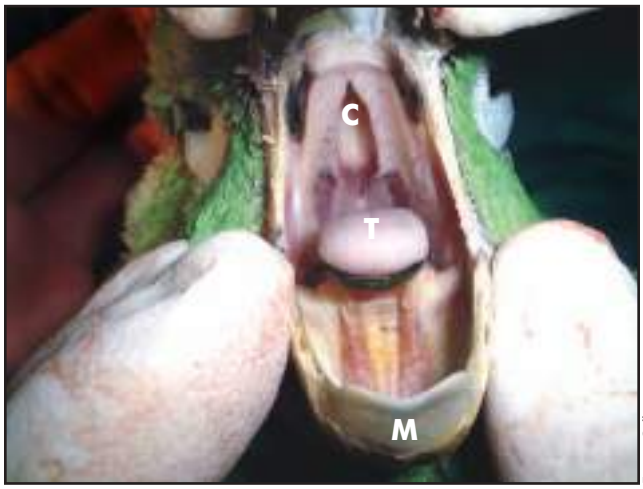
Unlike mammals, birds do not have a soft palate or a pharyngeal isthmus, nor do they have a sharp demarcation between the mouth and pharynx.[2] Instead, they have a combined oropharynx (Figs 14.1a,b). A longitudinal fissure, the choana, which connects the oral and nasal cavities, splits the palate. The choana is variably developed. In pigeons it is narrow and lacks papillae. In falcons it forms a narrow "V"- shape with few papillae. In psittacines, it forms a wide "V" and is bordered by caudally-pointed sensory papillae.[6] The choanal slit closes during swallowing. The infundibular cleft is located at the caudal edge of the choana and is the caudal opening of the left and right pharyngotympanic (Eustachian) tubes (*Rami infundibuli* Fig 14.1a) from the middle ear.[6] The palate forms a roof over the anterior part of the oral cavity. In finches, canaries, budgerigars and cockatiels it contains two ridges that assist in the removal of husks from seeds before ingestion.[1]

The tongue (Fig 14.1b) originates from the floor of the oropharynx and is mobilized by the hyoid apparatus and its multiple articulating bones and musculature. It functions to collect, manipulate and swallow food. Again, great species diversity exists in tongue development. The tongues of passerines and pigeons tend to be smooth, short and simple. Psittacines are unique amongst birds in having additional striated muscles in the anterior regions of their tongues that are independ-



Madeline Rae

Fig 14.1a | Ventral dorsal view of the palate area of the orapharnyx.



Espen Odberg

Fig 14.1b | Frontal view of orapharnyx of an Amazon parrot. T=tongue, M=mandible, C=choanal slit. Note the reduction of papillae.

ent of the hyoid apparatus and permit added flexibility and manipulative capabilities.[1] The typical psittacine tongue is thick and muscular and its maneuverability allows for the extraction of seeds and nuts from their husks, cones or pods. Lories and lorikeets have relatively long tongues that end in fine papillae that aid in the harvesting of pollens and the collection of nectar from flowers by capillary action.[8,9] In birds of prey the tongue is rasp-like, with a roughened tip and many small caudally pointed papillae near the base.[6,10] In ducks and other waterfowl which strain food particles, the rostral part of the tongue is scoop-like and has a double row of overlapping bristles on its lateral borders. These bristles work with the beak lamellae to filter particles.[2] It is interesting to note that birds have poor taste sensitivity compared to humans. For example, parrots have approximately 350 taste receptors compared with 9000 in humans[11], and chickens have up to 300 taste buds.[12] These are mostly located on the palate near salivary glands and on the posterior tongue. However, the beak, tongue and oral cavity have many touch receptors that make the mouth an important sensory area.[1]

The laryngeal mound lies immediately behind the tongue in most species and contains the glottis, the opening to the trachea. In most species the glottis lies directly under the caudal portion of the choana or just caudal to the choana in raptors. The laryngeal mound contains rows of caudally directed papillae that assist in the propulsion of food towards the esophagus during swallowing.[2] Birds lack an epiglottis.

Salivary Glands

There is great species variability in the number and distribution of salivary glands.[2] Granivorous species such as some parrots, pigeons, chickens and finches have a large number of glands to assist in swallowing the dry feeds they ingest. These glands are located in the roof, cheeks and floor of the oropharynx. Raptors have less developed salivary glands and piscivorous (fish-eating) species have poorly developed glands, or lack them all together. This is presumed to be related to the lubricated nature of the food they ingest. The content of the saliva produced also varies between species. The salivary glands of house sparrows secrete significant amounts of amylase whereas those of chickens and turkeys secrete little amylase.[13]

Esophagus and Crop

The esophagus is a thin-walled distensible tube that delivers food from the oropharynx to the proventriculus. It allows birds to swallow their food items whole. In birds the esophagus is divided into a cervical and a thoracic region. In the budgerigar, it lies dorsal to the trachea in the anterior regions of the neck and then runs along the right side.[14] The esophagus' distensibility is facilitated by a number of longitudinal folds. These folds are large and extensive in owls (Strigiformes) and species that swallow whole prey items, or those that store large amounts of food material such as gulls (*Larus* spp.). By contrast, parrots exhibit minimal esophageal fold development and possess a relatively narrow esophagus.[6] Mucus-secreting glands are present in the esophageal mucosa of most birds, particularly in the thoracic esophagus. These glands are actually absent from the cervical esophagus of budgerigars.

The avian esophageal wall consists of a mucosa, submucosa, a muscular tunic and a serosal layer. It generally contains only smooth muscle cells with a circular muscle layer predominating.[10] Peristaltic contractions of inner circular and outer longitudinal muscles propel food posteriorly through the esophagus.[1]

The crop or ingluvies is an expansion of the cervical

esophagus that functions as a food storage organ.[1,2,6] It mostly lies on the right side of the neck and when distended may also lie on the left side and will rest on the furcula. The crop has varying degrees of development in different species. In its simplest form, it is merely a spindle-shaped enlargement of the cervical esophagus. This arrangement is seen in ducks (*Anas* spp.) and owls.[6] Parrots have well-developed crops that lie at the caudal cervical esophagus. A prominent right pouch and a small left pouch typifies parrots. Pigeon crops have a more complicated structure. Both right and left lateral pouches or diverticulae are well-developed. The lateral pouches produce a holocrine secretion from the crop epithelium — "crop milk" — which is fed to the squabs during the breeding season.[15] It is produced in response to prolactin. It contains 12.4% protein, 8.6% lipids, 1.37% ash and 74% water.[2] Therefore it is mainly a protein and fatty acid source for these chicks, and is devoid of carbohydrate and calcium. Both males and females produce crop milk. Sheets of striated muscle that attach to the crop adventitia support the large crop. Parrot and pigeon crops possess a functional sphincter at the junction of the crop and the thoracic esophagus. This helps to form and regulate the boluses of food being propelled to the proventriculus.[6] It should be noted that birds lack the true upper and lower esophageal sphincters found in mammals.[1] Some granivorous species such as the European goldfinch (*Carduelis carduelis*) lack a true crop but have a very expandable esophageal pouch that can store food items.[1] Gulls, penguins and ostriches lack a crop but have a very distensible esophagus.[2]

The crop's storage function allows birds to ingest and store feed in the evening before roosting, thus providing for overnight energy needs. It also allows birds to rapidly ingest food items in a short period of time, and then take refuge in safe cover where the meal can be digested at a more leisurely rate. The crop also acts to soften ingested food by holding swallowed water and by contributing mucus to the saliva. Enzymes within the food or microbes present in the crop may further contribute to digestion.[16,17] Any glucose released in the crop can be absorbed by the crop mucosa, but this is of minimal importance.[2]

The crop is particularly well-developed in chicks to store food fed by the parents. The parents of altricial chicks premoisten and soften food in their crops and esophagus before regurgitating it to their chicks. The crop also provides an important immunological function in pigeons feeding squabs.[15]

THE AVIAN STOMACH: PROVENTRICULUS AND VENTRICULUS

The avian stomach consists of 2 distinct structures, the first part being the proventriculus or glandular stomach and the second structure is called the ventriculus (gizzard) or muscular stomach.

The relative size and shape of these structures is based on diet and is hence quite variable. In carnivorous and piscivorous species both structures are very distensible and may be difficult to differentiate grossly. This is due to the soft nature of their diet. In birds that eat hard food items, the proventriculus is relatively thin-walled and glandular. The ventriculus is muscular, thick-walled and powerful. The intermediate zone connects the two.[2,6] This gastric arrangement is typical of granivores, omnivores, insectivores and herbivores and hence most of the commonly found species in captivity.

The proventriculus is confluent with the esophagus cranially but has its own distinctly different structure. It lacks ridges, except in carnivorous and piscivorous species, and is lined with a mucous membrane. Its epithelium contains two principal types of glands that make up most of the thickness of the proventricular wall.[2] The first of these, the tubular glands, secrete mucus. The second type, the gastric glands, secrete hydrochloric acid and pepsin. This provides an acidic environment for digestion. Typically, the fasted chicken has a pH of 2.6, whilst that of a pigeon is 2.1.[6] Nectarivorous parrots have gland-free spaces between the longitudinal rows of glands. This allows for distension of the glandular stomach that may be an adaptation to pollen digestion.[16,18]

The proventriculus contains two muscular layers, the innermost circular layer and the outer longitudinal layer. The outer longitudinal layer is poorly developed or absent in parrots, waterfowl and some passerines. In these birds the myenteric plexus is located immediately under the serosal layer rather than between the two muscle layers.[2]

The intermediate zone between the proventriculus and ventriculus is aglandular and lacks folds. In parrots and pigeons, it closes tightly during ventricular contractions to segregate the ventriculus from the proventriculus.[6]

The ventriculus or gizzard has evolved to mechanically break food down. Hence it is best developed in species that ingest hard foods such as granivores,[1,2,6] and also in insectivores that need to break down the hard exoskeletons of their prey.[18] It is also the location where hydrochloric acid and pepsin can further chemically break food particles down. It consists of two pairs of opposing muscles. The caudoventral and craniodorsal thin muscles line the caudal and cranial sac of the gizzard respectively. The cranioventral and caudodorsal thick muscles are responsible for the powerful grinding

contractions seen in the gizzard.[16] The asymmetrical arrangement of these four muscles provides mixing and grinding actions during contractions.[1] The ventriculus is lined by the koilin, a cuticle layer, which acts as a grinding surface and protects the underlying mucosa from the acid and pepsin produced by the proventriculus. The koilin is made up of a combination of proteinaceous rod-like projections produced by the deep tubular glands lining the gizzard, together with desquamated epithelial cells that form a matrix.[1,2,6] The hardened composite frequently has raised areas and distinct longitudinal and transverse grooves that aid in mechanical breakdown of foods. It is thickest in species with well-developed, muscular stomachs. It is continuously worn and replaced in many species, but in falcons it may occasionally be sloughed and shed. The koilin lining may be green, brown or yellow in color due to bile staining caused by ventricular reflux from the small intestine. This is a normal finding.[6]

The gizzard is separated from the small intestine by a small pyloric fold that regulates the passage of food into the small intestine by slowing the movement of large particles.[19] In lorikeets and honeyeaters (both nectarivorous species), the proventricular and pyloric openings of the gizzard lie in a median plane, which is thought to allow rapid passage of ingesta.[8]

It is interesting to note that the size of the gizzard can change with diet within the same species, being thicker and larger when dry seeds are eaten and softer and lighter in summer when fruits are eaten.[1]

The role of grit in avian digestion is an interesting one. Insoluble grit may lodge in the gizzard and add to the maceration of the food, particularly in species that do not dehusk the seed before swallowing it, eg, pigeons, and galliforms like quail. It is controversial whether birds deliberately seek insoluble grit to aid in digestion or whether its ingestion is incidental to eating digestible foods or soils containing minerals and trace elements.[6] Grit is absent from the stomachs of nectarivorous birds, which also have poorly developed gizzards.[18]

GASTRIC MOTILITY

Food passes through the proventriculus very quickly where it is coated with hydrochloric acid and pepsin, with little enzymatic digestion. Digestion is controlled by the vagus nerve and by the hormones gastrin, secretin, cholecystokinin and pancreatic polypeptides. The food is propelled into the ventriculus where most of the mechanical digestion occurs by a combination of coordinated muscle contractions and the action of grit. The exact process varies with species. In turkeys and parrots, for example, the muscles contract in a clockwise direction around the ventriculus.[6,20] The paired thin muscles contract first and the isthmus closes, segregating the ventriculus from the proventriculus. As these muscles reach maximum contraction, the pylorus opens allowing digesta to pass into the duodenum. The thin muscles then relax as the thick muscles contract. This coincides with the closing of the pylorus and the beginning of peristaltic contractions along the duodenum. The isthmus may also open to allow the passage of ingesta back into the proventriculus for the addition of fresh acid and pepsin, allowing additional time for the breakup of large lipid globules and the breakdown of proteins. In particular, lipid is retained in the anterior region of the tract and digested more slowly than are protein or carbohydrates.[21,22] This cycle of contraction occurs as a seamless movement that gives the appearance that the gizzard is flipping.[6] This coordinated complex of contractions is controlled intrinsically by the myenteric plexus.

Raptors have a more simple stomach arrangement that has to produce the pellets, indigestible contents of fur, bone, teeth, feathers, claws, as well as perform the normal digestive function. Neck extension and head pumping assist the peristaltic propulsion of food into the proventriculus. The stomach fills with digestive juices over the next hour and vigorous, high frequency waves of contractions occur in a clockwise direction from the isthmus to the pylorus. This is followed by a 7- to 9-hour period of chemical digestion where forceful proventricular contractions occur at low frequency. By the end of this period, digestion is complete. Next, a short phase of paired contractions removes any further liquid from the indigestible pellet. This is followed by a further 5- to 6-hour phase of pellet compaction after which it is expelled by retroperistalsis. The timing of pellet ejection varies with the species.

INTESTINES AND PANCREAS

The small intestine is the main site for enzymatic digestion and nutrient absorption in the avian gut. It is less differentiated between species than are the more proximal regions of the gastrointestinal tract. The duodenum arises from the pylorus and forms a loop that encircles the bulk of the pancreas. The pancreas is trilobed in most species with the third lobe or splenic pancreas sometimes not being directly attached to the other two lobes. In budgerigars, the three pancreatic lobes are each drained by a separate duct. Two of these ducts empty into the distal duodenal loop adjacent to the bile duct whilst the other duct empties into the opposite side of the duodenum.[14] In pigeons, all three pancreatic ducts empty into the distal duodenum.[6] The exocrine pancreas contains enzymes similar to those found in mammals such as amylase, lipases, trypsin and chymotrypsin,

carboxypeptidases A, B and C, deoxyribonucleases, ribonucleases and elastases.[1] It also produces bicarbonate that buffers the intestinal pH. It is also important to remember that the intestinal wall mucosa also produces amylase, maltase, sucrase, enterokinase, lipases and peptidases and so contributes to enzymatic digestion.[6] These enzymes are produced in response to duodenal distension, hydrochloric acid, vagal stimulation, cholecystokinin, secretin and vasoactive intestinal peptide.[2,23] Birds have not been shown to possess any intestinal lactases so they should not be fed significant quantities of lactose-containing foods.[24] Amylase levels are actually highest in the jejunum, but the jejunum and ileum cannot be readily differentiated from the duodenum in birds. In general, the jejunum is thought to begin just after the ascending duodenal loop begins to turn back on itself, where the jejunal branches of the cranial mesenteric artery begin. The ileum is thought to begin at the vitelline (Meckel's) diverticulum and end at the recto-cecal junction.[6] There is great variation in jejunal and ileal anatomy in different species.

Nectarivorous and insectivorous birds have shorter intestines than do similar sized granivorous or herbivorous species.[25] This is believed to be due to the highly digestible nature of their diet.

The intestinal epithelium contains villi, microvilli and crypts. The villi's increased surface area allows efficient absorption of nutrients and their rich capillary system enables transport of these nutrients to the portal blood system. A thick layer of mucus produced by goblet cells in the epithelium protects the intestinal epithelium from the digestive juices and from physical abrasion, particularly anteriorly near the gizzard. Two muscle layers surround the intestine, the inner circular and outer longitudinal layers that allow mixing and propulsion of the digesta through the intestinal tract.

The avian duodenum is unique in its ability to exhibit both normograde and retroperistalsis.[6] These retrograde peristaltic waves bring the digesta back towards and into the ventriculus, as is evidenced by the presence of bile staining in the ventricular koilin. These waves are powerful and visibly distinct from the normal peristaltic waves. They occur every 15 to 20 minutes in the turkey[20] and up to once a minute in parrots on a moderate fat diet.[6]

The liver also empties into the distal duodenum via the bile ducts. Its primary digestive function is the production of bile acids and salts that assist in the emulsification of fats, allowing their digestion by lipases. These acids and salts, together with cholesterol and phospholipids, are secreted into the bile canaliculi that drain into the bile duct. Gall bladders are present in raptors and waterfowl, but are absent in many psittacines and pigeons.[6,26]

The ceca are important in fermentation of vegetable matter and in water balance and are hence most developed in chickens, ratites and ducks.[27] They are absent or vestigial in parrots and small insectivorous passerines, appearing histologically as a nodule of lymphatic tissue at the small intestinal-rectal junction. In the domestic pigeon they are entirely lymphatic in structure and are called the cecal tonsils. They are, however, well developed in herbivorous or omnivorous passerines.[16]

The avian rectum or colon is found between the ileocecal junction and the cloacal coprodeum. Except in the ostrich, it is very short and has a smaller relative diameter than the mammalian large intestine and is structurally dissimilar, being similar to the small intestine except for having shorter villi that are richer in lymphoid follicles. The avian rectum exhibits marked retroperistalsis, carrying urine from the urodeum and coprodeum into the colon up to the ceca.[6] This allows for further water resorption in the colon and hence aids in water conservation. In the pigeon the rectum enters the coprodeum from the right side, whereas in the parrot it enters from the left side at a 60 to 90° angle.[6]

CLOACA

The avian cloaca is a three-chambered structure that is responsible for the terminal deposition of digestive, urinary and reproductive products. It is much wider than the rectum. The first, most proximal chamber, is the coprodeum into which the rectum empties. It is the largest chamber of the psittacine cloaca and has a flat, vascular, avillous mucosa, covered by columnar epithelium and an extensive branching vascular pattern.[6,28] It is separated from the second chamber, the urodeum by an encircling sphincter-like ridge, the coprodeal fold. This fold can completely close off the coprodeum from the other chambers of the cloaca, preventing contamination of eggs or semen during egg laying or ejaculation.

The urodeum is the smallest cloacal chamber in psittacines, columbiforms and falconiforms. It receives the ureters and also the oviduct in females and the ductus deferens in males. The ureters enter the urodeum on either side of the dorsal midline, and in pigeons and parrots these openings are simple. In females, the oviduct has a rosette-like opening on the left dorso-lateral wall. It is smaller and less prominent in juveniles and hence difficult to visualize in these birds.[28] A membranous tissue may occlude this opening in females that have not yet laid in species such as the ostrich.[29] In males, the ductus deferens enters the urodeum on symmetrical, raised papillae located on the left and right dorsolateral walls. It is separated distally from the proctodeum by the uroproctodeal fold. The urodeal mucosa is smoother and less vascular than that of the coprodeum.

The urodeum exhibits retroperistalsis, pushing urates and urine cranially into the coprodeum and rectum where water and solutes are further resorbed, thus maximizing water conservation.[6,21,30] This retroperistalsis explains why urates and feces are sometimes intertwined when passed from the cloaca.

The proctodeum is the final cloacal chamber and is slightly larger than the urodeum in most species. The uroproctodeal fold is more developed dorsally and gradually loses prominence ventrally. This chamber is the most frequent site of papillomas in psittacines. It also gives rise to the Bursa of Fabricius on the dorsal midline just caudal to the uroproctodeal fold. The bursa is most prominent in the juvenile bird where its lymphoid tissue is responsible for the production of B-lymphocytes. In mature birds the lymphoid tissue involutes but the bursa's opening and chamber frequently persist and can be viewed during cloacoscopy. The timing of bursal involution is usually between 2 and 6 months of age, but varies between species.[6]

The cloacal blood supply is via the pudendal artery and vein. Innervation is via the pudendal nerve that follows the ureters to the dorsal cloacal wall where the cloacal ganglia are found.[28] These are important surgical landmarks.

The final structure of the gastrointestinal tract is the vent, a transverse opening in the ventrocaudal body wall through which body wastes and reproductive products are expelled. It is demarcated by lips dorsally and ventrally and is surrounded by voluntary muscles that form a sphincter. This provides birds with some control over defecation. For example hens that are incubating may pass a large urofeces in the morning when nest changeover occurs. Some psittacines can also be toilet trained to defecate on command. The act of defecation involves the partial eversion of the vent lips, resulting in the formation of a circular orifice through which feces, urates and urine can be expelled.[1]

Cloacal "sucking" has been noted in psittacines in juveniles and breeding females, where material is brought in from the outside via the vent lips under negative pressure.[28,31] In chicks, this is thought to have an immune stimulation function by exposing the B cells in the bursa to external antigens. In breeding females it is thought to facilitate sperm transport and hence fertilization in species where the male lacks a phallus.[31] See Chapter 18, Evaluating and Treating the Reproductive System for further discussion of the vent.

Gastrointestinal Diseases

DISORDERS OF THE BEAK

Deformities

Deformities of the bill of young birds, both congenital and acquired, have been described. Congenital deformities have been mostly described in poultry and waterfowl. Some of these are part of a more generalized problem, such as Micromelic Syndrome of white Pekin ducklings, an autosomal recessive mutation which causes a short maxilla, reduced overall size, shortened limbs, cervical subcutaneous edema and abnormal feathering.[32] Others are specific to the beak such as variations in the shape and curvature causing malocclusion, often leading to the maxilla being caught inside the lower mandible (prognathism) (Fig 14.2).[33] "Scissor-beak" is a condition where the upper beak rhinotheca is bent to one side, resulting in the overgrowth of the gnathotheca in psittacine chicks (Fig 14.4a). The condition becomes progressively worse due to the continued forces applied during the bird's normal beak usage and as the chick grows.[34,35] It has also been noted in other avian species such as ostriches,[36] softbills and passerines[32] where either the mandible or maxilla may deviate. Multiple potential etiologies have been described including heredity, incubation problems, malnutrition, infectious sinusitis, viral diseases and trauma.[36,37,38*]

In young psittacines, incorrect hand feeding techniques may result in bruising of the rictus on one side of the beak, leading to uneven growth and scissor beak.[38,39] **Eds. Note: In most psittacine rearing facilities establishing a formulated diet program for adults and youngsters has eliminated these problems completely. Facial bones are not as malleable in properly fed parents or their offspring.* Treatment involves altering the forces that direct the rostral growth of the affected part of the beak. Surgical techniques to achieve this have been described.[38,40] In addition, revision of incubation and chick feeding practices and nutrition may be warranted.

Mandibular compression has also been described in young macaws.[35] Prognathism, where the upper beak tucks into the lower beak, is another congenital beak deformity sometimes seen in chicks, particularly cockatoos.[35,41] The etiology of this condition is unknown. If attended to early, it can be corrected with physiotherapy by applying traction rostrally to the maxillary beak several times daily. If the maxillia is calcified, physiotherapy and beak trimming may help.[42] In more severe cases, dental acrylic prostheses (Fig 14.3) can be applied to the tip of the maxillary beak to force it to stretch out over the mandible[41] or KE wires and rubber bands or cable ties can be used in a modified Doyle technique (Figs 14.4a-f, 14.5a-f).[40]


Greg J. Harrison

Fig 14.2 | Baby umbrella cockatoo with mandibular prognathism.


Greg J. Harrison

Fig 14.3 | Acrylic applied to beak tip to allow new growth pressure to overcome the prognathic condition. A metal transverse pin made from a hypodermic needle is often implanted to help the acrylic maintain adhesion to the beak.

Crusty scab-like lesions at the commisures of the beak have been associated with biotin and pantothenic acid deficiencies in gallinaceous birds and ostriches.[36,43] Vitamin D and calcium deficiencies have resulted in soft beaks due to insufficient mineralization in many species.[33,44] Malnutrition and hypovitaminosis A were associated with significant beak deformities in hand-reared African grey parrot (*Psittacus erithacus*) chicks (see Chapter 5, Calcium Metabolism).[44] These were seen as significant grooved ridges and indentations of the rhinotheca and gnathotheca. Birds with rhinothecal overgrowth characterized by intralaminal hemorrhages have often been diagnosed with previous or current liver disease and malnutrition.[44] Assessing liver function, correcting the diet and trimming the beak as required are all useful management tools.

Traumatic Lesions

Traumatic lesions to beaks are amongst the most commonly seen problems. These frequently occur as a result of intra- or interspecific aggression, parent birds that mutilate chicks, accidents or predator attack. Iatrogenic causes have also been described from incorrect handling, use of mouth specula or incorrect beak trimming.[39] It should be remembered that the avian beak lacks a subcutis, and hence the thin dermal fibrovascular stroma is directly apposed to the periosteum. Therefore pressure necrosis of parts of the rhamphotheca and gnathotheca can lead to permanent beak defects.[46] Immediate treatment for trauma cases involves stopping hemorrhage, counteracting shock via fluid therapy, providing analgesia and preventing infection. Nutritional support then needs to be provided.[39,47] The decision as to how to best manage the injuries needs to be made early, to decide if indeed the bird is salvageable or if the owner is prepared to accept the care and cost involved with long-term management. Loss of the distal third of the bill has potential for regeneration, at least in psittacines,[48] but not in other species such as ostriches (*Struthio camelus*) in which beak growth stops in adulthood.[37] Trimming and reshaping using a mild grinding tool, such as an electric motor drill, can treat minor distal fractures. Rhamphothecal fractures can be stabilized with tissue glues such as cyanoacrylate (Figs 14.6a-i).[49,50] More serious fractures may require surgery with pins, wires, sutures, plating or acrylic remodeling techniques, depending on species, patient size, nature and location of the injury or fracture (Fig 14.7).[37,47,51,52] It should be noted that fractures and avulsions of the upper rhamphotheca are the most challenging due to the kinetic nature of the maxilla (in psittacines), the forces exerted and the presence of small bones.[47] Damage to the germinative layer of the rhamphotheca or gnathotheca or of the underlying bone means that the affected area will not regenerate keratin (Fig 14.8).[50,52] If the associated beak structure has been avulsed and if the damage is great enough, the entire mandible or maxilla may be lost and not regenerate. Acrylic prosthetics have been used as a temporary means of restoring beak function and appearance until new keratin growth occurs.[52] In permanent injuries, these prosthetic beaks need to be remodeled, replaced or reapplied on a regular basis, as they invariably work loose. In cranes, this is every 3 to 6 months.[53] Natural prosthetic devices have been successfully used in toucans utilizing the beaks from dead birds of the same or similar species. The surgeon should instruct the owner that these prostheses are also, like the acrylic or metal repairs, only temporary.[47]

Palatine bone luxation has been described in blue-and-gold macaws (*Ara ararauna*) following trauma in which


Greg J. Harrison

Fig 14.4a | Scissor beak in an umbrella cockatoo.


Greg J. Harrison

Fig 14.4b | Bands used for beak orthodonture. A electrician's black cable tie and an orthodonture rubber band are shown.


Greg J. Harrison

Fig 14.4c | The final shape of the transverse sinal pin for a beak traction technique.


Greg J. Harrison

Fig 14.4d | Band applied to beak with enough pressure on the transverse sinal pins to correct the deviation.

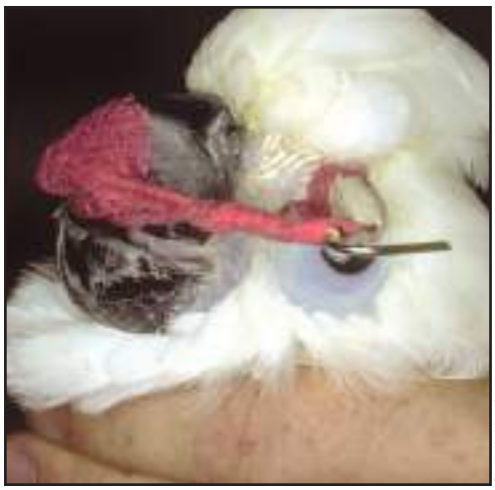

Greg J. Harrison

Fig 14.4e| Bandage material is placed over the rubber bands to avoid removal or becoming snagged.


Greg J. Harrison

Fig 14.4f | Several weeks after correction and removal of appliance. Traction was applied for 2 weeks.

the palatine bones became hooked onto the interorbital septum.[40,54] The luxation was reduced under general anesthesia by using digital pressure on the maxilla both directly and via an intramedullary pin placed through the infraorbital sinuses.[54]

Infectious Causes of Beak Malformation

Various infectious disease processes can involve the beak. Psittacine circovirus disease (Psittacine Beak and Feather Disease) infects numerous psittacine species, both wild and captive, where it can cause beak lesions as part of the chronic presentation of the disease, particularly in young cockatoos.[55] Affected beaks typically become abnormally elongated and may develop transverse or longitudinal fractures.[56,57] In some cases only the tips may be fractured. There may be necrosis of the palate and ulcers of the mouth. As the disease progresses, the beak may fracture and avulse, exposing the underlying bone, which can be very painful. Secondary bacterial and fungal infections may complicate the infection and cause life-threatening disease. Diagnosis is by PCR or HA (hemagglutination assay)[55,56] for presence of the virus or HI (hemagglutination-inhibition) for antibody levels.[56] Histopathology of affected tissues is also useful. Severely affected birds are usually euthanized, as there is no specific treatment for this disease except for supportive care which includes maximizing hygiene, providing soft foods, treatment of any secondary infections and immunostimulation.

Poxvirus infections are seen on the unfeathered regions of many avian species, but less commonly in psittacines.[57] This *Avipoxvirus* classically causes raised lesions which may or may not become necrotic and then secondarily infected. These may be found on the beak or at the beak/skin margin and also in the oropharynx. The beak and mouth may become painful and disfigured, and the bird may show reluctance to eat. Transmission requires direct contact with open wounds or inoculation via an insect vector. In one case, the basal layers of the beak epidermis were infected causing sloughing of the keratinized layers.[57] Although species-specific strains are transmitted by mosquitoes, cross-species infections may occur,

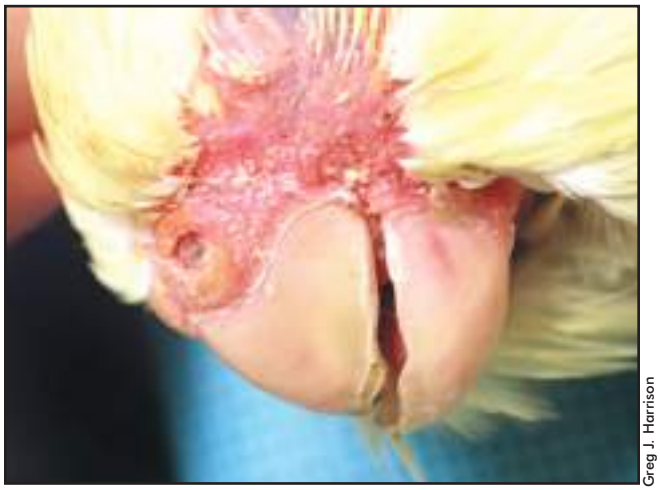

Greg J. Harrison

Fig 14.5a | Young cockatiel that was having its beak ground as a first step in the therapy for prognathism.


Greg J. Harrison

Fig 14.5b | A hypodermic needle is used to pre-drill a hole in the frontal bones. Then a stainless steel pin is placed transversely through the frontal sinus.

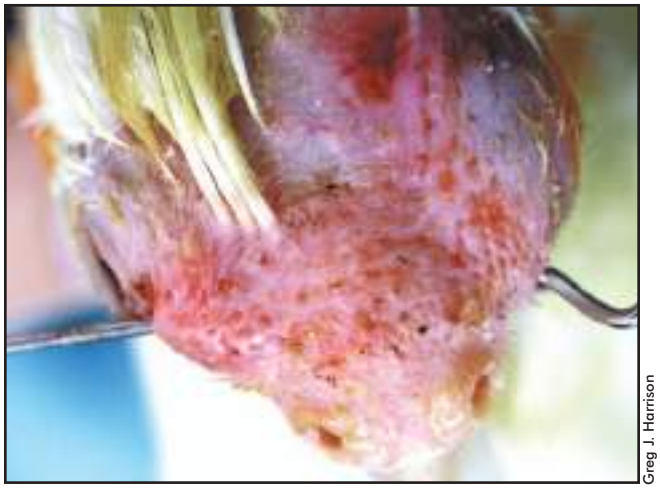

Greg J. Harrison

Fig 14.5c | The first hook is bent into one end.

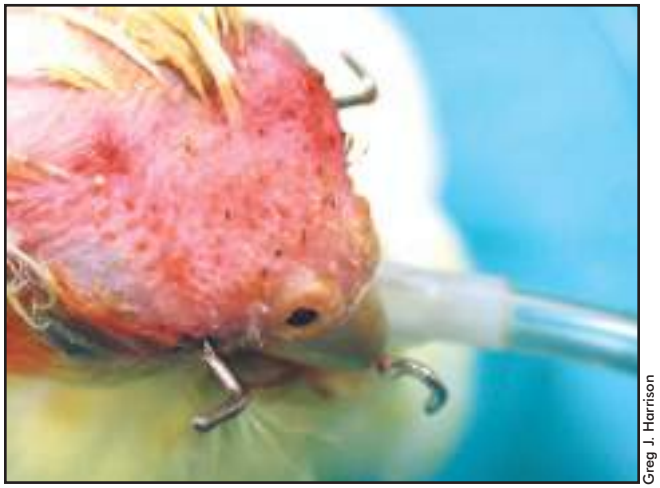

Greg J. Harrison

Fig 14.5d | A second bend is made in the transverse pin and a second S-shaped pin is formed and inserted in the distal rhinotheca.


Greg J. Harrison

Fig 14.5e | An orthodonture rubber band is placed around the left dorsal transverse sinus pin's hook and the ventral S-pin. A hemostat is placed around the tensed rubber band and stainless steel suture is placed to keep the traction on the rubber band once the hemostat is removed. The unused portion of the band is cut off.


Greg J. Harrison

Fig 14.5f | The finished traction device is in place. The extra length of the rubber bands have been cut off just above the stainless steel suture retention knot on the rubber bands. A plastic protective collar has been placed on the bird.


Greg J. Harrison

Fig 14.6a | A sun conure has been bitten by a larger bird. The walls of the rhinotheca are fractured and compressed into the maxillary sinus diverticulum of the infraorbital sinus.


Greg J. Harrison

Fig 14.6b | An electric motor drill has a small shank shim replacing the larger variety. This allows the use of a small dental burr to hone out the damaged rhinotheca and bone seen in 14.6a.


Greg J. Harrison

Fig 14.6c | A dental burr is used to hone out the damaged tissue around the edges of the depressed slab of rhinotheca and bone.


Greg J. Harrison

Fig 14.6d | Microsurgical forceps grasp the slab and bone and remove it from the site to avoid a sequestration.


Greg J. Harrison

Fig 14.6e | The rhinal cavity mucosa was not penetrated so an absorbable layer of calcium hydroxide[a] is applied as a bed for the regrowth of the bone, periosteum and rhinotheca. This layer is dried. The warm air from the electric motor can hasten this step.


Greg J. Harrison

Fig 14.6f | A thin layer of cyanoacrylic solvent[b] is layered over the calcium layer.

Greg J. Harrison

Fig 14.6g | Powdered dental acrylic resin[c] is sprinkled over the solvent layer, and the powder liquifies.

Greg J. Harrison

Fig 14.6i | The kit used to perform the procedure, 14.6 a-h.

Greg J. Harrison

Fig 14.8 | This gray-cheeked conure had a proximal traumatic maxillary amputation, involving the germinal area, and will likely not regrow. The conure is shown several months post-injury.

Greg J. Harrison

Fig 14.6h | The process has been repeated on both sides of the conure's damaged maxilla. In a couple of weeks the acrylic will dehisce and the underlying tissue continue to heal with no further attention. No antibiotics or antifungals were used pre- or post-operatively.

Greg J. Harrison

Fig 14.7 | A fractured mandibular symphysis has been repaired using a pair of S-shaped hooks like those used in Fig. 14.5d,e. One is placed on each side of the fracture site and the impaction bands applied. A layer of acrylic helps hold the pins and protects the fracture site.

usually with less pathogenic consequences.[58] Diagnosis is by viral culture or histopathological demonstration of proliferated epithelial cells with intracytoplasmic inclusions or Bollinger bodies. No specific antiviral treatment exists but lesions can be topically debrided and treated with antimicrobials for secondary infections as deemed necessary. Systemic antibiotics and fluid therapy along with supplemental vitamin A may aid recovery.

Disinfection with lipid solvents (eg, quaternary ammonium compounds, sodium hypochlorite) and exclusion of potential insect vectors will help to stop further spread of the infection. In outbreak situations, euthanasia of severely affected birds may be carried out. Vaccines are also available for some strains.[59]

Avian polyomavirus (APV) has been seen to cause tubu-

lar elongation of the lower mandible in Gouldian finch (*Erythrura gouldiae*) juveniles that have survived outbreaks of the disease.[60] These birds also exhibited delayed fledging and did not grow well. Other passerine infections with this virus have been associated with outbreaks of sudden death.[61] It has been postulated but not proven that recovered passerines may in fact become persistently infected and may shed the virus intermittently. Vertical transmission of the virus through the egg has also been postulated.[61]

Parvovirus infection in ducklings reportedly caused stunted beaks with protruding tongues in survivors.[62]

Primary bacterial and fungal infections of the beak are usually associated with trauma. They may cause necrosis, inflammation, hemorrhage, hyperkeratosis and the accumulation of necrotic debris with or without malodor. Cytology, culture and sensitivity are required to diagnose these infections.

Cryptococcosis has been associated with proliferative masses causing disruption of the nares, rhamphotheca and deeper beak and sinus structures in several psittacine species.[63,65] In some cases these lesions may be mistaken for neoplasms. They are characterized by gelatinous exudates which, when stained, contain large oval budding yeasts (4 to 7 μm) surrounded by a capsule 2 to 4 times the diameter of the cell.[64,65] Gram's stain, India ink, and Wright's stain have all been used to diagnose this infection cytologically. It can also be easily cultured on Sabouraud- dextrose agar. There is usually little surrounding inflammation, restricted to mild numbers of epithelioid macrophages, multinucleated giant cells and heterophils. *Cryptococcus neoformans* var. *neoformans* has a worldwide distribution, grows poorly at temperatures over 40° C and hence rarely causes problems in birds. It is commonly found in pigeon droppings. *C. neoformans* var. *gattii* is restricted to river red gums (*Eucalyptus camaldulensis*) and forest red gums (*E. tereticornis*) and grows poorly above 37° C and is most commonly identified in avian infections.[63] Treatments such as fluconazole orally at 8 mg/kg/day for at least two months, ketoconazole at 2 mg/kg BID per os gradually increased to 25 mg/kg bid per os and surgery to debulk the proliferative masses have all been suggested.[63-65] However, recurrence of lesions weeks to months after treatment is common, with early detection and aggressive therapy most likely to yield favorable results. Cryptococcosis is a potential zoonotic infection so public health issues need to be considered before treatment is instituted.

Fungal infections causing beak necrosis in Gouldian finches (*Erythrura gouldiae*) have also been described.[60] Affected birds had rhamphothecas that were characterized by a flaky white or yellow appearance. Fungal hyphae were detected in the beak matrix but no species identification was presented.

Bob Doneley

Fig 14.9 | Knemidocoptes mites and gross over-growth of the beak.

Knemidocoptes spp. mites can cause proliferation and inflammation of the psittacine beak (Fig 14.9) and are commonly seen in budgerigars, particularly young or immunosuppressed birds. Close inspection reveals the characteristic honeycomb patterning resulting from the mites tunneling into the skin. In chronic lesions, the germinal layer of the rhinothecal and gnathothecal epithelium can be so disrupted that permanent beak deformities result. Diagnosis is by way of skin scrapings. Treatment is simple with ivermectin/moxidectin, topical ectoparaciticides or even paraffin oil over the lesion that suffocates the mites being successful. Spiruroid (*Oxyspirura* spp.) infections in cranes have also been linked to beak deformities, as has trichomoniasis in cockatiels.[44]

Mycotoxins from *Fusarium* spp. in moldy food have resulted in beak deformities in poultry.[32]

Neoplasia

A number of neoplasms involving the beak have been described. Fibrosarcomas are considered the most common neoplasms of the beak, whilst squamous cell carcinomas and malignant melanomas are also seen. They cause distortion of the beak and surrounding tissue. Cytology of fine needle aspirates or histopathology on biopsy specimens provide a diagnosis, give information as to the likelihood of success with surgical debulking or chemotherapy and provide a prognosis for the patient.[57,66]

DISEASES OF THE OROPHARYNX AND ITS STRUCTURES

Diseases of the oropharynx are characterized by anorexia, dysphagia, halitosis, gaping, rubbing of the

beak or more generalized signs of ill thrift such as lethargy and disheveled plumage. Direct visual examination of the oral cavity under illumination will reveal most lesions, especially if magnification is used. This can be done with the patient awake or under general anesthesia. Further magnification in difficult to examine places can be achieved via endoscopy. Offending lesions can be swabbed or biopsied and the material obtained can be stained on slides, cultured or sent for cytological or histological examination.

Infectious Causes

Various viral infections have been found to infect the avian oropharynx. As mentioned previously, poxvirus can cause proliferative caseous lesions in the mouth and esophagus. Pigeon herpesvirus (PHV-1) can cause mucosal ulceration and diphtheritic membrane formation in the oropharynx, cere or beak commissure as part of the overall infection.[44,67] It affects young birds and the immunosuppressed most severely and should be suspected in flocks that suffer repeated bouts of trichomoniasis that are difficult to control. Spread is via fecal and pharyngeal secretions, and latent carriers are important reservoirs of infection.[67] Diagnosis is presumptively based on the presence of basophilic and eosinophilic intranuclear inclusion bodies seen on histology or cytology of affected tissue, particularly epithelial cells. Virus isolation and neutralizing antibody techniques are also available.

Abscesses and micro abscesses, plaques and granulomas are consistent with a number of diseases including viral, bacterial, yeast and parasitic infections, hypovitaminosis A and even chemical burns. Bacterial infections in the mouth can be caused by a variety of bacteria. Some of the more frequently isolated pathogens include *Staphylococcus* spp., *E. coli*, *Klebsiella* spp., *Pseudomonas aeruginosa* and other gram-negative bacteria.[37,44,68] The lesions may be localized or cause a generalized stomatitis and are usually secondary to oropharyngeal trauma, other infectious diseases or other causes of immunosuppression. Treatment should include systemic antibiotics based on culture and sensitivity results, local debridement, supportive care, identification and, where possible, correction of underlying immunosuppressive factors.

Mycobacterial granulomas may sometimes be seen in the mouth, though they are more commonly associated with lesions in the intestinal tract and liver and other intracoelomic organs.[69] Fine needle aspirates of lesions and acid-fast staining may reveal the presence of the mycobacterial organisms within macrophages. Where possible, cultures to speciate the type of mycobacteria should be carried out, although recently PCR testing that gives more rapid results has become available.[70] Hematology is characterized by a very high leukocytosis, often with a monocytosis. *Mycobacterium genavense* has recently been recognized as the causative agent in many avian infections that were previously attributed to *M. avium* subsp *avium*.[70,71] *M. tuberculosis* has only infrequently been responsible for disease in birds, even though it is the primary cause of tuberculosis in people.[72] Mycobacteriosis is a chronic debilitating disease with the potential for zoonotic spread, particularly in the immunosuppressed person.[72] Therefore the decision to treat or to euthanize is an important consideration. Multiple drug therapy is essential if treatment is to be attempted due to the high level of resistance to any single antimicrobial. Several treatment modalities based on human trials have been suggested.[73,74] Currently the combination of clarithromycin, ethambutol and rifabutin is the treatment of choice in humans and has been used with enrofloxacin in psittacines with success (see Chapter 28, Implication of Mycobacteria in Clinical Disorders).[74]

Candidiasis is a very common cause of stomatitis in birds, particularly in young, immunosuppressed birds, those on antibiotics and in lorikeets because of the high sugar content of some nectar mixes. The causative agent is usually *Candida albicans*, although other species may be involved. It is opportunistic and can be a primary or secondary pathogen. It causes white oral plaques with a caseous exudate. It is easily cultured and, when smears of lesions are made, the characteristic budding spores may be seen. Gram's stain, Diff-Quik and new methylene blue stains may help visualization. Histopathology is required to confirm that the yeast is causing the infection, however the presence of large numbers of budding yeasts or the presence of hyphal forms is suggestive. Treatment may be topical and/or systemic. Mild infections may respond to oral nystatin at 300,000 IU/Kg orally twice daily and/or topical chlorhexidine or oral miconazole formulations. More severe infections may require systemic antifungal therapy such as ketoconazole (10 to 30 mg/kg orally twice daily), fluconazole (20 mg/kg orally every 48 hours),[75] flucytosine at 250 mg/kg orally twice daily for 14 to 17 days or itraconazole (10 mg/kg orally twice daily for 21 days).[76]

Parasites are another cause of oropharyngeal pathology. *Capillaria* spp. are the most common nematode in the upper gastrointestinal tract. They may cause oral inflammatory masses, diphtheritic oral lesions or hemorrhagic inflammation of the commisure of the beak.[75] They are more commonly found in the small intestine.[77] They parasitize most species of birds including psittacines, passerines, columbiforms, gallinaceous birds and raptors.[77,78] Affected birds exhibit head flicking, dysphagia, weight loss and diarrhea. The adult parasites are very thin and can be difficult to see, but may be found in smears of lesions, as may their characteristic bi-opercu-

lated ova. Ova may also be detected upon fecal floatation, but they are intermittent shedders of ova and produce less than most ascarids. Their life cycle can be direct or indirect using earthworms as intermediate hosts.[77] They can be quite resistant to anthelmintics so high doses may need to be instituted. Examples include benzimidazoles (fenbendazole 100 mg/kg once or 25 mg/kg daily for 5 days, oxfendazole 10 mg/kg,) levamisole (40 mg/kg, beware of narrow safety margin); moxidectin 200 μg/kg (used up to 800 μg/kg by this author).[77,78] Ivermectin at standard doses (200 μg/kg) has been ineffective.[78] Benzimidazole or levamisole treatments should be repeated in 14 days. Environmental hygiene to prevent reinfection and removing potential intermediate hosts are all important control measures.

Spiruroids have been diagnosed in raptors, corvids and other species, and may cause raised granulomatous reactions in the mouth and crop. The worms, or their thick-walled embryonated eggs, may be found in oral, crop or fecal samples. Treatments include oral dosing with moxidectin[77] and/or manual removal of adults. *Contracaecum* spp. have been associated with severe oral infections in young piscivorous birds, particularly pelicans.[44] In birds of prey, *Synhimanthus falconis* has been reported in the oropharynx[44] and *Serratospiculum amaculatum*, a parasite of the air sacs, can cause diphtheritic lesions of the oropharynx, which need to be differentiated from those caused by trichomoniasis.[77,79,80] Their eggs can be found in oral mucus or in feces.

Trichomoniasis is commonly found in pigeons, budgerigars and raptors and is occasionally seen in other species such as cockatiels, Amazon parrots, conures, canaries and zebra finches.[44,79] The causative organism, usually *Trichomonas gallinae*, can exist as different strains with different pathogenicities. In pigeons and raptors, white or yellow caseated plaques may be seen in the oral cavity. These usually extend to the crop and esophagus and may go as far as the proventriculus. These plaques may need to be differentiated from other diseases such as candidiasis and poxvirus infection. Budgies usually show no oral lesions. Affected birds usually exhibit regurgitation, dysphagia, weight loss, listlessness, palpable mucous in the oropharynx and crop and, in severe cases, vomiting blood and death. In pigeons the disease may be generalized, infecting the liver, umbilicus and cloaca, especially in squabs. Diagnosis is via wet mount examination of oral lesions or crop fluid, revealing the motile flagellated organism under high power magnification. Warming samples increases protozoan activity. The life cycle is by direct oral contact between birds, and spread through common drinking water is also important. Raptors are thought to acquire infection through ingestion of infected pigeons. However, freezing carcasses has proven not to work (S. Hudelson, personal communication, 2004). Carrier states exist and are thought to be responsible for reinfecting flock mates. Such birds should be culled. Treatments suggested include ronidazole (6 to 10 mg/kg once daily for 7 to 14 days), dimetridazole (100 to 400 mg/L drinking water), metronidazole (20 to 50 mg/kg twice daily) and carnidazole (20 to 30 mg/kg once).[75,77,78,81] A dose of 50 mg of carnidazole has been reported to be most effective (S. Hudelson, personal communication, 2004). Note that dimetridazole has a low safety margin and should be avoided in hot weather, during breeding or when racing pigeons. Diphtheritic plaques may need to be removed by debridement. Antibiotics for secondary infections may also be required.[68] Regular monitoring of flocks for infection is recommended in pigeons and budgerigars.

Nutritional Causes

Hypovitaminosis A can lead to squamous metaplasia of the oropharyngeal epithelium, particularly glandular epithelium, leading to plaque and granuloma formation.[44,75,82] In psittacines, this typically involves the submandibular or lingual salivary glands. Sometimes affected birds exhibit a subcutaneous swelling caudal to the mandible. The choanal papillae are often shortened and stunted. If severely damaged, the choanal papillae fail to regenerate, so caution is warranted when using this sign for diagnosis. Affected birds typically have a history of being fed a predominately seed-based diet. Dietary correction via parenteral vitamin A supplementation or use of reputable formulated diets is needed. Some granulomas may be excised surgically. Secondary bacterial infections may be found and should be treated as required. In gallinaceous birds, lesions are confined to the mucous glands of the pharynx and their ducts. Keratinization of the glandular epithelium causes blockage of duct openings, hence secretions and necrotic debris accumulate. These appear as small white hyperkeratotic lesions (see Chapter 4, Nutritional Considerations).

Traumatic Causes

Traumatic injuries can occur in the oropharynx due to fighting or accidental trauma. Injuries of the psittacine tongue are common due to its frequent use as a probing and sensing organ. Tongues are very vascular, and in psittacines well muscled, so control of bleeding is a first priority. This may involve the use of electrocautery or suturing. The birds may easily remove sutures. Some authors have found the need to wire the beak closed to prevent suture removal.[75] In this case a pharyngostomy tube may need to be placed until the wound heals. Caustic injuries can be caused by the ingestion of certain

chemicals, for example silver nitrate cautery sticks, access to excessively hot foods or ingestion of trichotecenes, especially T2 toxin.[44] These often cause anorexia and dysphagia due to the pain experienced. Analgesia may be of added benefit in these cases, as well as supportive care (pharyngostomy feeding, fluids, antibiotics) until healing is complete. Foreign bodies including wire, wood and plastic have also been known to cause penetrating wounds.

Neoplastic Causes

Neoplastic diseases of the oral cavity have been documented and include epithelial and mesenchymal tumors.[57] The most common problem seen in new world psittacines is oral papillomatosis. Lesions range from mild mucosal roughening to overt verrucous masses. They can also be found in the crop, esophagus, proventriculus, and cloaca and have been associated with bile duct carcinomas. Severe lesions can ulcerate, hemorrhage or cause gastrointestinal or distal reproductive tract obstruction. Their exact cause is unknown. Although histologically the lesions appear similar to those of mammalian papillomaviruses, there is no immunohistochemical or DNA evidence to support the presence of an avian papilloma virus. Instead, herpesvirus is consistently being detected in these papillomatous lesions.[83] True parrot papillomavirus has been reported in only one African grey parrot.[84] Squamous cell carcinomas, fibrosarcomas and lymphosarcomas have also been reported.[85] They can be quite painful and cause inappetence. Diagnosis is based on biopsy and histological examination.

DISEASES OF THE ESOPHAGUS AND CROP

Many of the diseases found in the oropharynx are, not surprisingly, also found in the esophagus and crop. The esophagus can be injured as a result of tube-feeding (Figs 14.10a-c).

Infections

Viral infections such as poxvirus and herpesvirus have been reported. Proventricular dilatation disease (PDD), of suspected viral origin, may also affect the crop but will be more fully discussed later. Bacterial infections, both primary and secondary, are commonly seen with crop diseases. In mild infections, there may be bacterial growth and mucosal colonization with little inflammatory response. Severe infections, however, are characterized by hemorrhage, necrosis and sometimes fibrinopurulent exudates of the mucosal surface.[57]

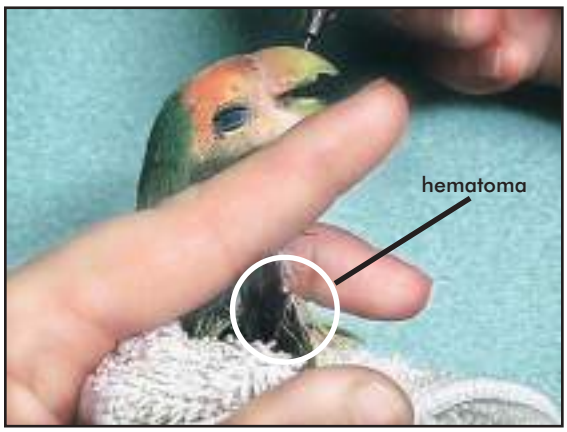
Typically, crop motility is impaired with delayed crop emptying and regurgitation. Birds can quickly become

Greg J. Harrison

Fig 14.10a | Tubefeeding a sick bird is a frequent event in an avian veterinary facility. The round ball tipped needles can make the job easier for one person.

Greg J. Harrison

Fig 14.10b | The clear delicate esophagus can be palpated for the tube presence or visualized by wetting the right ventral-lateral cervical area. The ball is easily seen through the transparent esophagus and skin of the neck in small thin birds.

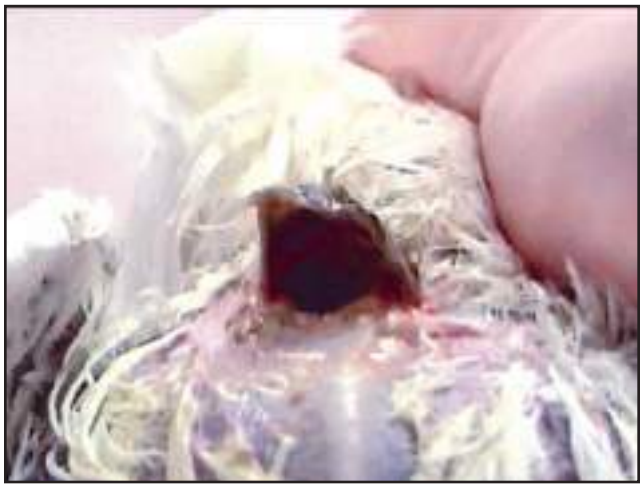


Greg J. Harrison

Fig 14.10c | A similar feeding tube in a lovebird with a hemotoma in the crop area from trauma resulting from bruising that can occur from improper restraint and flailing when a bird is being tubed. Although this is very uncommon, it is even less likely to occur if a speculum and a soft catheter are used.

Greg J. Harrison

Fig 14.11a | A crop burn, usually in a handfeeding baby that is fed scalding food heated by a microwave oven. The area is soft and friable and is best allowed to form a scab prior to any surgery.



Greg J. Harrison

Fig 14.11b | Several days after becoming a dark, hard scab the burnt area will open allowing food to fall from the crop. Topical acrylic glues can expand the time the scab is retained and often can be kept in place long enough for the damage to completely heal, avoiding surgery completely.

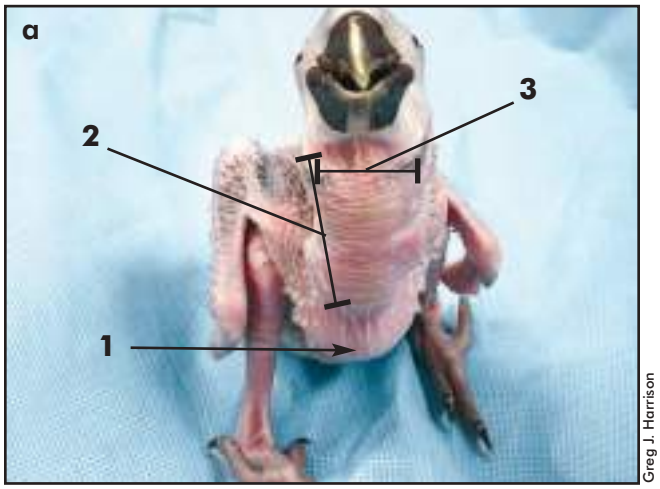
listless, toxemic, dehydrated and, if untreated, die. Yeast infections may also be involved, and the organisms are typical of *Candida* spp. infections. *Candida* spp. has also been described as a primary crop pathogen in some species, namely lovebirds (*Agapornis* spp.) and cockatiels.[86] As well as the clinical signs listed above, a palpably thickened crop wall may be found. Causative organisms can be diagnosed via examination of wet and stained smears obtained via crop washes and culture and sensitivity. Treatment involves the use of oral topical and/or systemic antifungals, crop washes, antiemetics and fluid therapy. Predisposing management factors need to be addressed, particularly in hand-reared birds.

A large number of endoparasites are known to infect the crop. The most familiar parasitic disease of the crop is trichomoniasis, which was discussed previously. A host of nematodes and trematodes can also be found in the crop and esophagus. As well as *Capillaria* spp. already mentioned, *Echinura uncinata*, *Gongylonema ingluvicola* (quail and gallinaceous birds) and *Dispharynx nasuata* have all been found to invade the crop esophageal mucosa.[44,77]

Noninfectious Diseases

Primary non-infectious diseases of this region include crop burns, foreign body penetration, lacerations and impactions, hypovitaminosis A and ingluvioliths. Excessively hot feeding formulas may lead to full thickness burns of the crop and skin in hand-reared chicks and occasionally in adults.[44,85] The burns usually occur in the anteroventral region of the crop. Initially the area may appear just dry, but subsequently reddening and edema may occur, followed by blistering and often necrosis (Figs 14.11a,b). This process may take several days. The dehisced wound, which is externally visible, allows food to fall out of the crop as the chick is fed. Treatment involves addressing any dehydration and infection problems. It may be best to manage this condition medically for the first 3 to 5 days to allow the burn to scab over and fully fistulate before surgery is attempted. Premature surgical closure usually results in wound dehiscence due to continued necrosis of the surrounding skin that may not have been noticeable at the time of initial surgery. Surgery involves anesthesia and debridement of the affected area, removing all dead and discolored tissue, and closure of the deficit in at least two layers, ensuring that the crop and skin are closed separately. Beware of reduced crop capacity immediately post-surgery, until healing and crop expansion can occur (see Chapter 35, Surgical Resolution of Soft Tissue Disorders).[87] Other causes of crop trauma such as forceful use of feeding/medicating tubes, bite wounds or foreign bodies can be similarly treated. Caution — food traumatically placed subcutaneously or within the neck structures must be flushed and debrided immediately in the event feeding has caused a rent — let heal by secondary intention healing (Figs 14.12a-f).

Ingluvial foreign bodies include food items, grasses, wood, metal and plastic items usually accidentally ingested by birds. In hand-reared chicks, plastic feeding tubes can come loose and be accidentally swallowed during the vigorous head pumping which birds may do during feeding. Diagnosing these problems is via history, crop/esophageal palpation, radiography (both plain and contrast) or endoscopy. The offending items can be removed manually, endoscopically or surgically via an ingluviotomy. Impactions of the crop frequently occur as a result of a sudden change of food. In ducks and poultry



Greg J. Harrison

Greg J. Harrison

Fig 14.12a,b | A baby macaw an hour after having hand-feeding formula traumatically introduced into the cervical neck tissues. This injury occurs from using a syringe for feeding. Macaws "pump" hard and if the head is not controlled the syringe tip can penetrate the pharyngeal tissues resulting in food being deposited into the cervical tissue. Edema and some hemorrhage also adds to the swelling. The food must be flushed out within hours, or septicemia may be rapid and overwhelming. 1. A normal crop with some food present. 2. Vertical swelling from food deposited into tissues. 3. Horizontal swelling of tissues.

Greg J. Harrison

Fig 14.12c | An incision over the food swelling — avoiding the esophagus, crop and vascular structures of the cervical region.

Greg J. Harrison

Fig 14.12d | The incision has reached the pocketed food and it spills out.

Greg J. Harrison

Fig 14.12e | A cotton-tipped wood applicator enters the oral penetration wound and exits the neck incision. The cotton tip is grasped with a forcep and the forcep is pulled up and out through the mouth. A cut rubber band is grasped by the forcep and pulled out of the incision and tied in place to act as a seton to allow flushing for 2 days.

Greg J. Harrison

Fig 14.12f | Rubber band seton tied in place. Flushing of the wound QID with Normosol®, and a tissue disinfectant is performed. Antibiotics and antifungals are administered in a hand feeding formula that is fed via a silicone tube to assure the food gets into the crop.

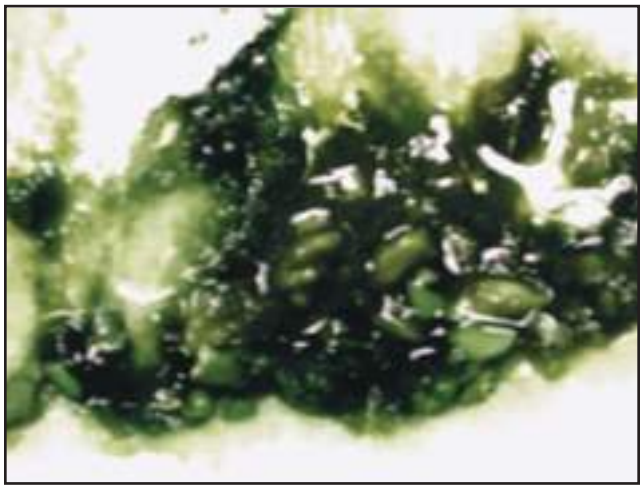
it has been associated with sudden access to lush grasses and sprouted grains.[44] In caged birds, *ad libitum* supply of grit has been associated with crop impactions and, on occasions, this has resulted from the overzealous feeding of grit to chicks.[88] In raptors it is associated with the sudden availability of roughage in a previously low-roughage diet. Diagnosis and treatment are as described for foreign bodies.

Ingluvioliths are various mineral concretions that occasionally develop in the crops of some birds, particularly budgerigars. Calculi consisting of urates surrounding seed husks, potassium phosphate, oxalate and cystine have been described. The exact cause of these is not known, but it is speculated that birds that have experienced periods of starvation may have been forced to eat seed husks and urates.[44] In some instances these calculi become large enough that they need to be removed. This can be achieved via endoscopy or ingluviotomy.

As well as the neoplasms mentioned in the oropharynx, the crop and esophagus could suffer from tumors of smooth muscle origin such as leiomyomas and leiomyosarcomas. Although asymptomatic when small, they can become very large, necrotic and hemorrhagic. They are characterized microscopically by interlacing bundles of fusiform cells with moderate amounts of cytoplasm.[57] Carcinomas of the submucosal glands also occur. They are often large, sometimes necrotic and hemorrhagic, and involve much of the esophageal or crop wall with invasion into surrounding tissue.[57]

Crop Stasis

Crop stasis or "sour crop" is a clinical sign of disease, and not a disease in itself. Clinical signs include regurgitation, delayed crop emptying, a sour odor, inappetence, dehydration, anorexia and listlessness.[87] "Sour crop" is usually complicated by bacterial and or fungal infection that may be primary, but is more often secondary. Crop stasis is most often seen in hand-reared chicks and results from poor management. Food fed at the wrong temperature or consistency, not allowing the crop to empty between feedings, poor hygiene, incorrect incubation temperatures and humidity and concurrent disease are all examples of possible causes of crop stasis in chicks. In adults, the condition can result from various crop infections, systemic or metabolic disease, heavy metal toxicity, foreign body ingestion or even PDD. Normal psittacine crop floras include few gram-positive bacteria and scant non-budding yeasts. Treatment of this condition involves identifying and treating the underlying disease, as well as crop flushes with mild antiseptic solutions, antimicrobial therapy as appropriate and supportive fluid therapy. See Chapter 7 Emergency and Critical Care.



Greg J. Harrison

Fig 14.13 | The passing of whole seeds is characteristic of gastrointestinal disease. Many causes such as lead, parasites, PDD and pancreatitis are possible.

DISEASES OF THE PROVENTRICULUS AND VENTRICULUS

Diseases of the proventriculus and ventriculus can have varying clinical signs ranging from regurgitation, weight loss, appetite changes (either anorexia or polyphagia), undigested seed in the feces (Fig 14.13) and lethargy. Most diseases of these organs produce similar clinical signs that make their identification more challenging.

Infectious

Perhaps the most common gastrointestinal disease is proventricular dilatation disease (PDD). The suspected cause is a virus. It has been diagnosed in over 50 species of psittacines, but also in several other avian species including Canada geese, canaries, weavers, toucans, spoonbills and honeycreepers.[89] It is characterized by a lymphoplasmacytic infiltration of peripheral and central nerve tissue. It commonly affects the myenteric plexuses supplying the gastrointestinal tract, resulting in atrophy of the smooth muscles of the crop, proventriculus, ventriculus or small intestine. This causes delayed gastrointestinal motility and organ dilatation. It can also affect the Purkinje cells of the heart, the adrenal medulla, the brain and the spinal cord. The lesions can be very segmental which may explain the variation in clinical signs seen.[90]

Gastrointestinal clinical signs include progressive weight loss, regurgitation, crop impaction, passage of undigested food and eventually death, usually within 12 months. An 80 to 120 nm enveloped virus has occasionally been isolated from infected birds and has been used experimentally to induce infection in some birds.[89] A virus is certainly suspected but not proven. The virus itself may not be the cause of the disease; however the inflammatory response may be. However, its exact identity

is unknown at the time of writing. Diagnosis is based on finding lymphoplasmacytic infiltrates in ganglia and associated nerves of the myenteric plexus of the gastrointestinal tract.[90,91] However, given the segmental nature of the disease, it is difficult to know which area of myenteric plexus to biopsy. Thus, a positive biopsy is diagnostic but a negative biopsy does not rule out the disease.[90] The crop is considered the safest area from which biopsies can be taken. Biopsy sections should contain blood vessels, as these are most likely to contain nerve tissue. The proventriculus has a thin wall and acid secreting glands which make it a risky choice for biopsy. Similarly, ventricular biopsies are not recommended, as there is real risk of damaging the myenteric plexus.

Large amounts of the suspect virus are shed by infected birds and transmission is proposed to occur via the fecal-oral route.[91] The virus is fragile in the environment and, hence, hygiene and management are vital in preventing spread of infection. The virus's environmental fragility may explain why epornitics occur more commonly in indoor aviaries rather than outdoor collections. Although many birds eventually die from this disease, some chronically infected parrots have been detected which have lived for years, intermittently shedding the virus. These may be reservoirs for reinfection in aviary situations. Although there is no cure for the disease, celecoxib, a Cox-2 NSAID, has been used to improve clinical signs by decreasing the inflammatory reaction around affected nerves.[92] The dose given was 10 mg/kg orally every 24 hours for 6 to 24 weeks. Improvement in clinical signs occurred in 7 to 14 days. Treatment was ceased once birds resumed normal body weight, condition and diet. The longest survivor reported was a blue and gold macaw (*Ara ararauna*) that finished therapy two years previously and remained in normal physical condition, eating a normal diet and had no radiographic signs of PDD and was negative on biopsy. However, no comment was made as to whether virus particles continued to be shed in its stools.

Macrorhabdus, previously known as avian gastric yeast (AGY) or megabacteria, have been reported to infect a large range of pet and wild birds (see Chapter 30, Implications of Macrorhabdus in Clinical Disorders).

Bacterial infections in the proventriculus and ventriculus can be primary but are usually secondary to other immunosuppressive or disease states. The organisms most often associated with disease are gram negative and include *E. coli*, *Klebsiella* spp., *Salmonella* spp. and *Enterobacter* spp.[85] These often affect the intestine, thus giving clinical signs including diarrhea, maldigestion/malabsorption, anorexia and weight loss. Diagnosis is based on culture, with fecal cultures often taken. Fecal Gram's stain may also show gram-negative overgrowth. *Candida* spp. infiltrating the proventricular and ventricular wall have also been documented, particularly in finches.[60,76] Treatments as outlined earlier, optimal nutrition, hygiene and correcting predisposing stressors are all important management tools.

A number of nematodes have been diagnosed in the avian proventriculus. *Echinura uncinata*, *Gongylonema* spp., *Cyrnea* spp., *Tetrameres* spp. and *Dyspharynx nasuata* have all been found. Ventricular parasites include *Amidostomum* spp., *Cheilospirura* spp., *Epomidiostomum* spp. and *Acuaria* spp.[44,77] Of these, *Acuaria* spp. appear to be the most commonly encountered. They are commonly found to infect finches, but galliforms are also susceptible. These "gizzard worms" are fine and hair-like and burrow just under the koilin lining of the ventriculus, impairing gizzard function and digestion of food. Affected birds usually exhibit ill thrift, may have undigested seed in the droppings and die. Secondary bacterial infections further complicate the infection. The life cycle is indirect, so removal of insects from the environment is important. Anthelmintics such as ivermectin, moxidectin, benzimidazoles and levamisole have all been used with varying degrees of success.[77]

Cryptosporidiosis is a protozoal disease usually seen to infect the intestine of immunosuppressed animals, including birds. In finches, however, it has a predilection for the proventriculus, where it causes necrosis and hyperplasia of glandular epithelial cells.[113] Finch isolates are different genetically from other species and may represent a unique species of *Cryptosporidium*.[114] Affected birds show decreasing body weight and yellowish droppings which may contain undigested seed. Azithromycin, roxithromycin, toltrazuril and paromomycin have met with some success as treatments.[113] Underlying immunosuppressive diseases or environmental stressors need to be identified and corrected.

Non-infectious

Proventricular or ventricular foreign bodies are more commonly encountered in ratites, galliforms and waterfowl, but are also seen in psittacines and other species. Psittacine chicks which ingest indigestible fabric fibers or bedding material such as ground corncob, kitty litter, crushed nut shells, shredded paper, styrofoam, grit, plastic, rubber or wood shavings may develop proventricular/ventricular impactions.[44,75] In older parrots, these same items plus other cage or household items may be ingested. In flightless birds, and in particular ostriches, exposure of birds to a new substrate may predispose

them to proventricular/ventricular impaction. Other illnesses may lead to a depressed appetite or pica.[115] Affected birds classically have poor appetites, pass scant feces, exhibit regurgitation especially if force-fed, and are depressed and lethargic. Diagnosis is via radiography, endoscopy, palpation (in larger species) or exploratory laparotomy. Where nails or other ferric compounds are suspected, this author has also seen metal detectors successfully used. Fiber will only show up on gastroscopy (Fig 14.15).

Treatment depends on the severity of the impaction, or the nature of the foreign body ingested. If the bird is bright, the impaction is not complete and the offending item is capable of being passed by the bird, then medical treatment may be adequate. This may include supportive fluid therapy and antibiotics, the administration of psyllium (beware in cockatiels) or paraffin liquid and force-feeding with easily assimilated high-energy soft foods. Metoclopramide may help stimulate small intestinal motility and thus assist in ventricular/proventricular emptying. Prevention of access to the offending items is also necessary. Attempts should be made to identify underlying disease states that have a bearing on the final outcome.

Some items may be able to be removed endoscopically. This can be done through the mouth or via an ingluviotomy. In the ostrich, a technique of proventricular flushing is described.[116] With the bird held firmly above the tarsus, it is turned upside down, its glottis closed and a hose connected to a steady stream of water is passed from the mouth to the proventriculus. Massaging the proventriculus helps to loosen the impaction. The loosened material often passes out the mouth with the hose *in situ*. This is repeated until clean water passes out the mouth. Needless to say, this procedure requires considerable manpower but is useful where general anesthesia is not an option and medical therapy has failed. In many cases, however, surgery is indicated to relieve the impaction or remove the foreign body. These techniques have been described elsewhere.[115,117]

Heavy metal toxicities can also lead to gastrointestinal, renal and CNS signs as part of their pathophysiology. Psittacines in particular fall victim to inadvertent acute lead and zinc intoxication due to their curious nature and penchant for chewing any object they may find. Items varying from galvanized cage wire to paint, curtain weights, stained glass windows, jewelry, coins, wine bottle foil, toys and mirror backing are possible sources for these heavy metals.[118] Waterfowl ingest lead shot whilst feeding, mistaking it for gravel.[119] Falcons ingest lead by eating prey that has been shot. The ingested heavy metal pieces are acted on by the acidic content of the proventriculus and macerated by the ventriculus leading to rapid absorption. The mucosal linings become very irritated, and in severe cases, the ventricular koilin may be damaged. Pancreatic damage may also be a result of zinc toxicosis. Affected birds may show variable clinical signs which include inappetence, decreased fecal volume, regurgitation and vomiting, ileus, green diarrhea, polyuria, polydypsia, CNS signs (particularly with lead poisoning) and feather picking.[118,120] Lead poisoning in waterfowl causes weight loss, limb and neck weakness, and bright green feces.[119] A tentative diagnosis is based on a suggestive history, clinical signs and the presence of radiodense particles within the gastrointestinal tract on survey radiographs. However, the client is often unaware of exposure to heavy metals, the clinical signs are nonspecific and the heavy metal is not visible on radiographs. Definitive diagnosis is based on the presence of elevated blood lead or zinc levels.[90,118, 121,122] Elevated levels of amylase, CPK and uric acid may be found with zinc intoxication.[120] The acute cases respond well to chelation therapy with edetate calcium disodium (EDTA) at 30 to 50 mg/kg once to four times daily by intramuscular or intravenous injection, depending on the severity of signs and amount of heavy metal ingested.[90,120-122] Other chelating agents such as penicillamine given orally at 55 mg/kg twice daily can be used in conjunction with CaEDTA.[118] Succimer is the preferred oral lead chelator at 25 to 35 mg/kg orally twice daily.[81,118] Concurrent parenteral fluid therapy is essential for rehydration and assisting excretion of the metals. Where gastrointestinal function permits, gavaging of high energy/electrolyte fluids and lubricants such as mineral oil, peanut butter, psyllium, magnesium sulphate or sodium sulphate have all been suggested. These products have been found to be ineffective in waterfowl.[118] Antibiotics for secondary bacterial infections can be given and antiemetics and intestinal prokinetic agents (eg, metoclopramide) may also be helpful. If possible, the offending particles should be removed via endoscopy or ingluviotomy/gastroscopy. Particles that have a ferrous or iron base can also be removed by inserting a feeding catheter equipped with powerful neodymium-ferro-borium alloy magnets (Fig 14.14).[44] Pure lead, zinc, or many of their alloys cannot be removed with a magnet. It should be remembered that lead is not normally found in animals, as it is not involved in any normal biochemical pathway.[118,122] It also accumulates in the body over time. Zinc on the other hand is an essential trace metal and so needed in low levels. It is not stored in the body over time but is excreted.[118,121] Chronic zinc toxicities are thus due to continued or repeated exposure.

Ulceration of the proventriculus occurs occasionally in pet birds but more commonly in flightless birds secondary to foreign body ingestion or disease states. No common

Greg J. Harrison

Fig 14.14 | A set of magnets installed in the end of a rubber catheter that have attached to copper coated ferrous shot. This device can be used to remove ferrous metals, some of which can be galvanized and thus contain zinc and/or lead in toxic amounts.

Greg J. Harrison

Fig 14.15 | Nylon fiber retrieved from the proventriculus of a cockatiel on a seed diet that was allowed to pull apart a nylon carpet.

etiologic agent has been identified, but a link has been suggested between chronically stressful environments and the occurrence of proventricular ulcers.[90] Ulceration may also be secondary to zinc toxicity.[85,121] Clinical signs are non- specific but may include anorexia, regurgitation, gastrointestinal pain, lethargy and melena. Once an ulcer perforates, most birds will die from sepsis and shock within 6 to twelve hours.[90] Thus, early detection of an ulcer is imperative but can be difficult. Gastroscopy should be considered in any patient with persistent signs of gastrointestinal pain or melena. Some of the human rapid tests for melena are useful for detecting the presence of digested blood in the feces of seed eating birds.*

**Eds. Note: some debate continues on the specificity of such tests.*

There is a report of proventricular obstruction in an adult male eclectus parrot caused by a tubular diverticulum of the ventriculus.[123] This diverticulum consisted of dysplastic koilin and smooth muscle and caused complete obstruction of proventricular outflow. No inflammation, organisms or neoplastic changes were associated with the lesion. The cause was undetermined.

Neoplasms of the proventriculus and ventriculus are seen in a number of species, particularly in budgerigars and grey-cheeked (*Brotogeris pyrrhopterus*) and Amazon parrots. Proventricular carcinomas are most commonly found at the isthmus and are usually flat rather than nodular.[124] They are invasive, often extending through the muscular layers and may reach the ventricular wall and serosa.[57,125] However, they rarely metastasize.[125] Clinical signs may include anorexia, regurgitation, weight loss, maldigestion and melena. Papillomas may occur as discussed previously and smooth muscle tumors are uncommon.[57]

Surgery of the gastrointestinal tract is discussed in Chapter 35, Surgical Resolution of Soft Tissue Disorders.

DISEASES OF THE INTESTINES AND PANCREAS

Intestinal tract disorders usually manifest themselves clinically as changes in the color, bulk and nature of the feces produced. Thus, diarrhea, maldigestion, voluminous droppings and/or melena may be evident in affected birds. Anorexia, depression and weight loss often accompany these enteric signs.

Infectious

The majority of intestinal pathology is at least in part attributable to infectious agents. By far the most common cause of diarrhea in pet birds is due to bacterial infections, although these are seen less commonly in adult raptors.[57,78,90] Gram-negative bacteria are most commonly implicated. Enterobacteriacae are most frequently isolated including *E. coli*, *Salmonella* spp., *Klebsiella* sp., *Yersinia* sp., *Pseudomonas aeruginosa* and *Proteus* sp.[90] They can be both primary and secondary pathogens. The gross lesions induced in the affected intestine include redness, exudation and occasionally ulceration. Histologically, necrosis, fibrin deposition and predominately heterophilic infiltrates are noted,[57] although the bacteria may not always be present in all lesions.

Gram-positive bacteria have also been responsible for intestinal disease. *Enterococcus hirae* has caused enteritis and septicemia in 10 psittacine species.[126] *Campylobacter* spp. especially *C. jejuni* has been associated with yellowish diarrhea and enteritis in many avian species including psittacines,[85] passerines, waterfowl, galliformes[107] and ostriches.[127] Affected birds, which are often young, exhibit lethargy, anorexia, diarrhea and emaciation. Erythromycin and tetracyclines are the frontline

treatments for this bacterium.[107] Clostridial infections have also been diagnosed in many avian species and are known for their ability to produce potent toxins.[107] *Clostridium perfringens* can cause necrotic enteritis and foul-smelling feces in psittacines, ostriches,[127] and other species. *C. tertium* has been reported in a cockatoo with megacolon and chronic, foul-smelling diarrhea. This resulted in severe dilation of the colon characterized by a lymphoplasmacytic inflammatory reaction. The sporulated form of this bacterium has a "safety-pin" appearance that is quite visible under Gram's stain (see Chapter 4, Nutritional Considerations, Section II, Nutritional Disorders). Anaerobic culture yields a definitive diagnosis. Treatment involves the use of metronidazole (25 mg/kg orally twice daily)[75] or clindamycin (100 mg/kg orally once daily).[81,107]

Mycobacteriosis is typically a chronic wasting infection in birds, primarily affecting the gastrointestinal tract rather than the respiratory tract, as is the case with mammals. Most avian species are susceptible. Waterfowl, flightless birds, grey-cheeked parakeets, older Amazon and Pionus parrots, budgerigars, siskins, Gouldian finches, toucans and pigeons are particularly susceptible.[69,75] It is particularly a problem where birds are congregated. The intestine appears to be the primary site affected. The submucosa becomes infiltrated with large numbers of histiocytes that contain many acid-fast organisms. This affects the bowel's ability to digest and absorb ingesta. Other granulomatous lesions may be found in the liver and spleen, the bone marrow and the respiratory tract. In other cases, only skin lesions are noted. The course of this disease may take years.

Primary mycotic intestinal infections are rare, but secondary invasion by *Candida* spp. or *Zygomycetes* spp. are sometimes seen.[57]

Viral diseases can cause severe disease to the intestine. PDD can cause segmental damage to the intestinal smooth muscle, nerves and ganglia.

The clinical picture of paramyxoviruses (PMV) can include diarrhea and melena. The pathogenicity, types of lesions and clinical signs seen depend on the serotype and strain of the virus and the host's susceptibility. PMV-1, which causes Newcastle Disease, has caused gross hemorrhage (due to vasculitis of the intestinal wall) and necrosis of submucosal lymphoid tissue in the intestines of some birds.[57]

Adenoviruses also cause hemorrhagic enteritis in psittacines, American kestrels and turkeys, and a greenish diarrhea in pigeons and galliforms.[57,128] In affected psittacines, gross necrosis and hemorrhage are noted. Histologically, inflammation is variable, thrombosis of intestinal capillaries is evident, and the enterocytes contain large basophilic intranuclear inclusions.[57]

The intestine is the primary site of infection by various protozoal agents. Coccidia are some of the most widespread and well-known agents, consisting of several genera affecting a wide range of birds. Coccidia's pathogenicity may range from inapparent infections to severe hemorrhagic diarrhea and death. *Eimeria* is most common in pigeons and galliformes, whereas *Isospora* is primarily found in psittacines and passerines.[129] Both species have direct life cycles, with transmission occurring via ingestion of sporulated oocysts in fecal-contaminated food or water.[129] Disease is often precipitated by stress. Diagnosis is via detection of large numbers of oocysts in the fecal wet smears or floatation. Antiprotozoal treatments include toltrazuril (7 mg/kg orally every 24 hours), sulfa-based drugs or amprolium.[77,81] Cryptosporidiosis has been diagnosed in over 30 avian species and is considered an uncommon disease of the young and immunosuppressed **(Figs 14.16a-c)**.[130] The only confirmed speciation in a non-galliformes was the detection of *Cryptosporidium meleagridis* in an Indian ring-neck parrot chick presented with diarrhea and delayed crop emptying.[131] Treatment has been covered previously. Microsporidia, primarily *Encephalitozoon hellem,* has been diagnosed in lovebirds, budgerigars, Amazon and eclectus parrots[132] and Gouldian finches.[133] Infection has been linked to concurrent disease, especially circovirus infections,[132] and other causes of immunosuppression.

Flagellated protozoa are also recognized as causes of enteritis. Giardiasis has been diagnosed in a variety of psittacines, poultry, waterfowl, finches and toucans.[77] Clinical signs vary from inapparent infections to weight loss, failure to thrive, diarrhea[75] or even feather picking in cockatiels in the USA.[134] Diagnosis is via direct fresh fecal examination for the presence of the pear-shaped trophozoites, trichrome fecal staining or ELISA testing. Treatments used successfully include ronidazole, metronidazole, dimetridazole and fenbendazole.[75,77,134] *Hexamita*/*Spironucleus* spp. occasionally cause enteric signs of variable pathogenicity in galliforms, pigeons and parrots.[77, 134] They can be recognized by their cigar shape and rapid motility and are more difficult to clear than are giardia.[134] *Cochlosoma* spp. cause lethargy and moist bulky droppings, as well as dehydration and death in young Gouldian finches being fostered under Bengalese finches. Adult birds are unaffected, although they may have bulkier stools. The Bengalese finches act as an asymptomatic carrier, as do a range of other finches, but not adult Gouldian finches.[135] The organism is characterized by its six anterior flagella and a helicoidal anterior ventral sucker; it is diagnosed on wet mounts of fresh fecal samples. Transmission is via the fecal-oral oral route, and the

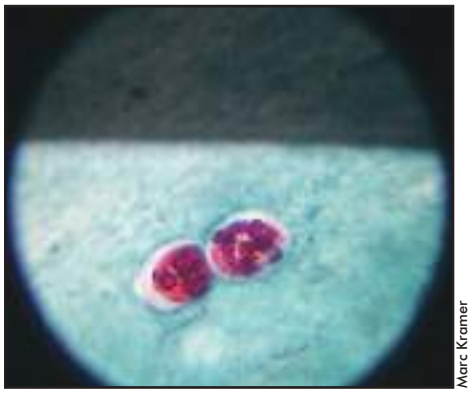

Marc Kramer

Fig 14.16a | Acid fast stain of a fresh stool specimen showing a form of cryptosporidium.


Marc Kramer

Fig 14.16b | Another form of cryptosporidium from the stool. Acid fast stain.

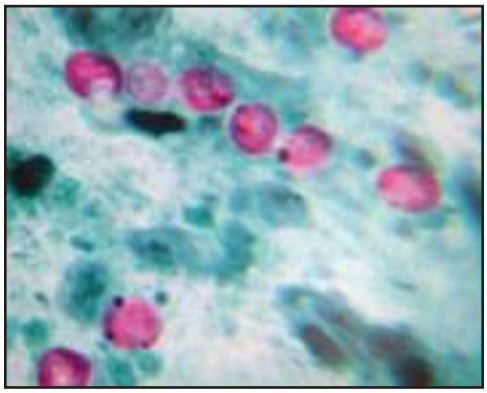

Marc Kramer

Fig 14.16c | A necropsy specimen had a proventricular scraping acid fast stained. The tissue forms of cryptosporidia are shown.

organism appears to be very sensitive to antiprotozoal drugs such as ronidazole and metronidazole.[135]

Nematode parasites are commonly diagnosed in Australian psittacines, although other psittacines have also been infected.[129,136] Pigeons, galliforms, waterfowl and toucans are just some of the other species susceptible. Nematodes are particularly a problem in wild-caught birds or those housed in planted aviaries that favor the parasite's life cycle. Ascarids and *Capillaria* spp. are most commonly diagnosed (Fig 14.17). They cause ill thrift, weight loss, diarrhea and death. Transmission is either direct by ingestion of embryonated eggs or indirect via ingestion of an intermediate host, depending on the parasite species. Diagnosis is via fecal floatation and identification of the offending eggs. Treatments include benzimidazoles, levamisole, ivermectin, moxidectin and pyrantel (4.5 to 25 mg/kg per os repeated in 14 days).[81,136] *Capillaria* spp. can be particularly difficult to eradicate, and high doses of anthelmintics may need to be given. Beware of toxicities associated with high doses. For example, Columbiformes appear susceptible to toxicosis after treatment with fenbendazole or albendazole at 50 to 100 mg/kg.[137] Secondary bacterial infections may also need to be addressed, and supportive care such as warmth, fluid therapy, nutritional support and intestinal lubricants and laxatives may all be helpful. Environmental control is paramount. Avoiding contact with contaminated feces and providing a dry environment to stop embryonation of eggs are all important. Decreasing environmental load and reinfection of an aviary by suitable housing and quarantining of new birds are all recommended.

Cestode infections can cause problems in the avian intestinal tract. Since their life cycles are largely indirect and involve an intermediate host such as an insect, mollusk or arthropod, cestodes are more a problem of birds with access to the ground.[77,129] They are most common in insectivorous finches and parrots of wild stock, particularly cockatoos, African greys and eclectus parrots.[129] Infections may cause diarrhea, ill thrift and death, particularly in finches.[138] Diagnosis is via the presence of proglottids in the feces. These may rupture, releasing the eggs. Microscopically, the eggs contain the hexacanth larvae with six hooks on the oncosphere. In some birds, the cestode can be visible, protruding from the cloaca after defecation. However, proglottids may not always be shed or may not rupture, so infections can be missed. Praziquantel (10 to 30 mg/kg orally, repeated in 14 days)[77,81,138] appears to be the most effective cestocide. Avoidance of exposure to intermediate hosts is important in control.

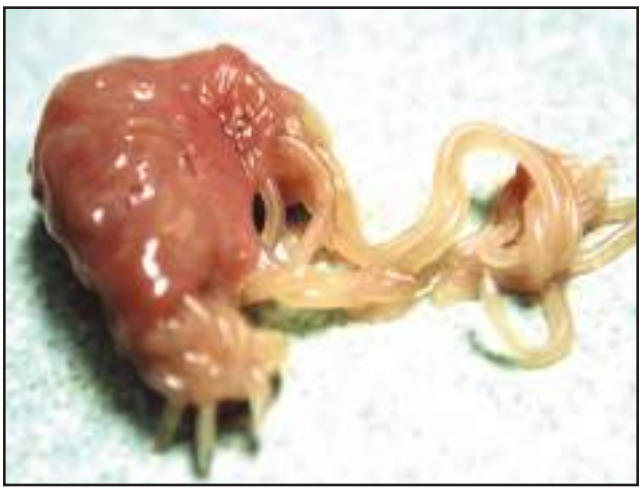

Gwen Flinchum

Fig 14.17 | A lethal intestinal nematode obstruction in this Quaker parakeet is an unusual finding in captive raised pet birds in the USA. To compound the issue, a fecal exam for parasites was negative.

Intussusception

Intussusception of the distal small intestine is less common in birds than in mammals and mostly occurs in gallinaceous birds secondary to enteritis.[44] As the proximal segment telescopes into the distal segment, blood flow is impaired and intestinal necrosis follows. Rectal intus-


Greg J. Harrison

Fig 14.18a | A malnourished female budgerigar with rhinal discharge over a hyperkeratotic cere.


Greg J. Harrison

Fig 14.18b | Same budgerigar in (a) showing swollen abdomen.


Greg J. Harrison

Fig 14.18c | The swelling was caused by massive fecal retention. A celomic mass, usually a tumor, is involved in such an obstruction. Changing the angle of the vent allowed 5-10 cc of feces to be removed, temporarily giving the bird and the owner time to decide on the final fate.


Greg J. Harrison

Fig 14.18d | An intestinal obstruction resulting from auto-obstruction in a parrot due to a necrotic intestinal lining slough. The cause was never determined. The necrotic section passed on day 3 of tube feeding and fluids, and the bird recovered.

susception may result in the rectum telescoping onto itself or into the coprodeum, where it may protrude from the vent lips. Both of these are medical and surgical emergencies, which may involve resection of the offending piece of bowel using magnification, and very fine sutures. (6-0 to 8-0).[90]

Ileus

Ileus or intestinal hypomotility/amotility can be caused by both physical obstructions (in the intestinal lumen, wall or as a result of external extra-intestinal compression) and by poor motor function. Examples of the former include foreign bodies, neoplasia, heavy parasite burdens, granulomas, strictures and various torsions and adhesions. Paralytic ileus can be caused by enteritides, PDD, peritonitis, lead toxicity and thrombosis of splanchnic vessels.[44] The impaired section of bowel dilates with intestinal fluid and gas. The bird becomes dehydrated. Ischemic necrosis of the intestinal wall leads to further fluid and protein loss. Gram-negative bacteria proliferate and produce endotoxins that can result in shock. Death can occur within 24 to 48 hours. Depending on how acutely the bird is affected, clinical signs can vary from vomiting, diarrhea, depression, listlessness, anorexia, decreased fecal output and emaciation.[44,90] In affected birds, abdominal palpation is resented.[37,115] Plain and contrast radiography may reveal dilated gas-filled bowel loops and identify the location of any obstructions. Supportive therapy with fluids, antibiotics, particularly for anaerobes, and analgesia are all recommended. Corrective surgery may then be performed to either remove or relieve any obstructions or to resect any debilitated sections of bowel.

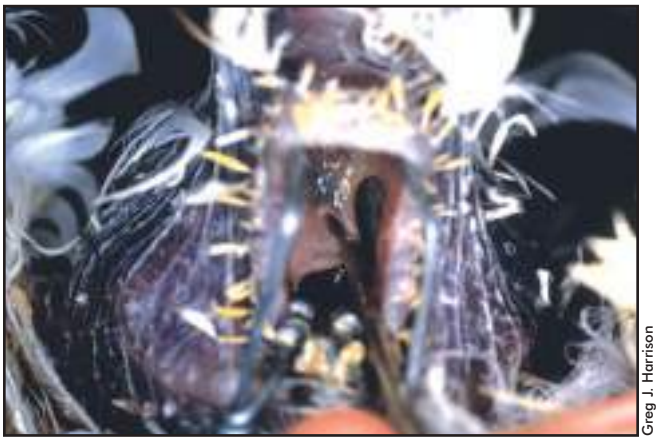
Neoplasia

Primary intestinal neoplasms include carcinomas, papillomas, smooth muscle tumors and lymphosacoma.[57,85] Lymphosarcoma presents as diffuse or nodular thickening that may be mistaken for other conditions such as mycobacteriosis. Leiomyomas and leiomyosarcomas present as firm, red-brown masses within the intestinal wall and can only be distinguished from one another histologically (Figs 14.18a-d).[57]

DISEASES OF THE CLOACA

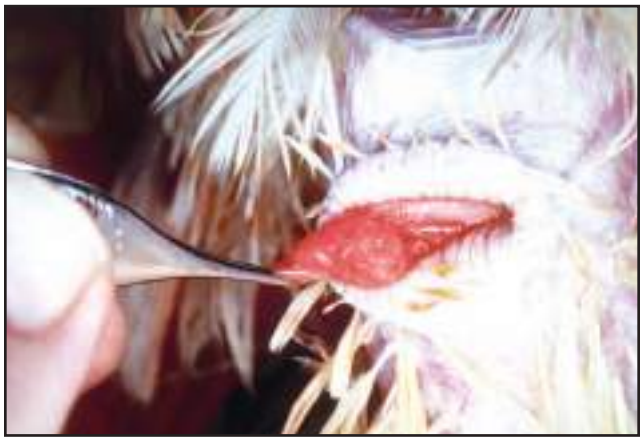
Infectious

Speculums allow complete observation of the cloaca (Fig 14.19). Internal Papillomatous Disease (IPD) can be responsible for the irregular, cobblestone mucosal lesions seen in the psittacine proctodeum (Figs 14.20). IPD is one of the most common cloacal masses seen in



Greg J. Harrison

Fig 14.19 | A cloacal speculum can make observation of this complex structure much more understandable. The probe on the left side is in the vagina of a female cockatoo with PBFD. The fold containing the vaginal orifice separates the urodeum from the proctodeum. The bird is in dorsal-ventral position.



Espen Odberg

Fig 14.20 | Gastrointestinal disorders often involve papillomatosis. Any case of loose or smelly stool in a susceptible species should be investigated.

birds, particularly South American species. Moderate to severe lesions may lead to partial proctodeal obstruction.[31] Affected birds typically present with tenesmus, bloody droppings, malodorous feces, flatulence and staining of the vent and tail feathers with urofeces. Definitive diagnosis is via biopsy, but affected lesions will usually blanch when dilute acetic acid is applied to them. Various treatments have been suggested. These include sharp surgical excision, electrosurgery, silver nitrate cautery, cryosurgery, laser surgery and mucosal stripping.[139] All procedures carry the risk of causing iatrogenic traumatic cloacitis. Recurrences of the papillomas are common. Some lesions spontaneously regress but may recur. An empirical report of a commercial pepper diet allowing regression, as long as birds were fed the diet, has been reported (G. Harrison, personal communication, 2000) (see Chapter 32, Implications of Viruses in Clinical Disorders).

Bacterial cloacitis is rare in most birds but can create significant pathology when it occurs. It can occur as a result of localized trauma (such as cloacoliths, chronic cloacal prolapse), coexisting disease (such as internal papillomatous disease) or nutritional deficiencies. *Candida* spp. have most commonly been isolated from the proctodeum and vent lips but *Trichosporon begielli* has been found in one immunocompromised macaw.[31] These infections should be treated with appropriate antimicrobials both topically and systemically, and any underlying causes need to be corrected.

Cloacoliths are firm aggregations of urates that collect in the cloaca. They will at times also contain fecal material. They are often the result of iatrogenic intervention for other cloacal disease (eg, surgery or during forceful cloacal examination or sampling). The exact pathogenesis of cloacolith formation is unknown but is believed to involve impaired defecation with retention of urates that may cause dehydration. This may solidify and chemically alter the urate mass, causing it to form a solid structure. Gentle removal, application of topical cleaning, antibiotic and anti-inflammatory agents, systemic antibiotics and regular monitoring of the affected area may be required. In chronic cases, recovery can be slow and characterized by repeated recurrences.

Cloacal Prolapses

Cloacal prolapses are not uncommon in birds and can take one of several forms. Oviductal prolapse occurs in egg-laying females that strain excessively to lay due to uterine or egg-related factors. These often need to be surgically repaired, which may involve a hysterectomy if the oviduct damage is severe. Endoscopy may need to be performed to differentiate oviductal from rectal prolapses. Idiopathic coprodeal prolapse is seen in male cockatoos in particular and less commonly in other psittacines. The exact cause is unknown, but it is suspected that affected birds have never been fully weaned, are bonded to their human companions and interpret their owner's behavior such as petting as sexually stimulating. This is distinct from the overt masturbation exhibited by some cock birds in the presence of the owner. Various surgical techniques have been described, including cloacopexy of the ventral cloaca to the abdominal wall and ventplasty. Hormonal investigations and chemical and surgical neutering are all being evaluated.[31] Behavioral modification may be appropriate. A true intestinal prolapse can occur if a rent from the cloaca or rectum is opened into the abdomen (Fig 14.21).

Phallic Prolapses

Phallic prolapses have been described in waterfowl and

Greg J. Harrison

Fig 14.21 | A massive intestinal prolapse contains the congested pancreas. This can only occur from a fistula from the cloaca or rectum into the peritoneal cavity.

ostriches and result from excessive sexual stimulation, particularly in younger males, as well as other causes of phallic trauma and underlying systemic disease.[139] Cleaning and replacement of the phallus in the ostrich so that the tip is resting in the dorsal cloacal sulcus and sexual rest are the treatments of choice. Occasionally stay sutures across the vent may be required to keep the phallus in place. Treatment with antibiotics and anti-inflammatories may be necessary if the phallus has been traumatized.[139]

Neoplasia

Cloacal carcinomas are infiltrative tumors leading to thickening of the cloacal wall. Smooth muscle cloacal tumors are infrequently reported.[57]

Diseases of the Exocrine Pancreas

Although diseases affecting the pancreas will often impair both endocrine and exocrine function, only the exocrine effects will be discussed here.

Pancreatitis occurs when the digestive enzymes such as trypsin, protease and phospholipase are prematurely activated within the gland and begin to digest it. The resultant damage to the cell wall leads to the release and activation of these enzymes into the ducts and extracellularly. Free radicals are produced causing further damage. The initiating cause of this autodigestion can be difficult to pinpoint, but several factors have been recognized.[140] Viral infections such as Paramyxovirus type 3, herpesvirus, polyomavirus, adenovirus and avian influenza A can cause necrosis and variable inflammation. PMV-3 can also cause chronic pancreatitis leading to a firm and irregular pancreas. Histologically, a variably lymphoplasmocytic inflammation can be seen, with the formation of lymphoid follicles evident. *Neophema* spp. seem particularly susceptible to this form of the disease.[75] A number of bacterial agents have also been associated with pancreatitis. Non-infectious causes include obesity associated with fatty diets or high fat meals, zinc toxicosis and secondary damage from egg yolk peritonitis.[141] Birds on all seed diets seem particularly prone to pancreatitis. Psittacines, particularly quaker parakeets, may die suddenly from acute pancreatic necrosis.[57] The pancreas from affected birds appears pale and firm and may exhibit variable degrees of hemorrhage. There is often necrosis of adjacent fat. Histologically, the lesions include coagulation necrosis of pancreatic acini, intralobular hemorrhage and necrotic foci in mesenteric adipose tissue.[57] Zinc toxicity targets the pancreas, causing vacuolation and degeneration of acinar cells. Grossly, the pancreas may appear normal or exhibit mild parenchymal mottling.[57]

Diagnosis of pancreatitis can be difficult. Clinical signs are non-specific but may reflect gastrointestinal dysfunction and pain. Vomiting, diarrhea, anorexia, lethargy, ileus, weight loss, polyuria and polydypsia and abdominal distension are some of the signs noted.[140,141] Signs of abdominal pain include kicking, feather plucking (especially around the abdomen), falling off the perch, wide-based stance, sudden flight attempts, aggression and obsessive chewing. Measurement of blood amylase levels has been described, but absolute normal values are yet undetermined for most species. However, amylase levels above 1,100 IU/dl are considered elevated.[140,141] Increases of only 2 to 3 fold may be attributed to extra-pancreatic causes such as gastrointestinal disease (eg, small intestinal obstruction), renal disease or glucocorticoid administration. Thus, interpretation of blood levels needs to be done with care. Pancreatic biopsy is the method of choice for diagnosing pancreatic disease. This can be achieved via laparotomy or endoscopically through the right thoracic air sac. A histological examination may also shed light on likely effective treatments and prognosis.

Treatments for pancreatitis are based on mammalian strategies.[140,141] Fluid therapy to improve pancreatic perfusion is important. Converting birds onto low-fat pelleted diets is preferred to withholding food due to the high metabolic caloric requirements of most birds. Analgesia (eg, butorphanol, carprofen) to counteract abdominal pain, intestinal motility stimulants such as metoclopramide or cisapride to counteract intestinal ileus and parenteral antibiotic therapy are all of value in dealing with this disease. Any underlying causes should also be treated (eg, zinc toxicosis). Some workers have used omega-3 fatty acids for their lipid stabilizing and anti-inflammatory properties.[140,141] In life-threatening cases, plasma transfusions may help by replacing protease inhibitors and thus stopping further pancreatic damage.[141]

Pancreatic enzyme therapy may help stop pain by inhibiting the endogenous production of pancreatic enzymes; it is useful in treating pancreatic insufficiency,

which may follow a bout of acute pancreatitis.[141] Exocrine pancreatic insufficiency manifests in the production of pale voluminous feces. It can result from any chronic inflammatory process that may affect the pancreas, including those listed as causing acute pancreatitis.

Pancreatic neoplasms can be either benign or malignant. Birds suffering from IPD seem to have a high incidence of pancreatic adenocarcinomas[142] that may present as masses that have caused adhesions between viscera and peritonitis.[57]

Products mentioned in text

a. Hypo-Cal.® Calcium hydroxide. Ellman International, Inc., Hewlett, NY www.ellman.com.

b. Temp-Plus® Liquid. Ellman International, Inc., Hewlett, NY www.ellman.com.

c. Temp-Plus Resin.® Ellman International, Inc., Hewlett, NY www.ellman.com.

References and Suggested Reading

1. Klasing KC: Avian gastrointestinal anatomy and physiology. Semin Avian Exotic Pet Med 8:42-50, 1999.
2. Denbow DM: Gastrointestinal anatomy and physiology. *In* Whittow GC (ed): Sturkie's Avian Physiology. Academic, San Diego, CA, pp 299-325, 2000.
3. Freethy R: How Birds Work: A Guide to Bird Biology. Blandford Press. Dorset UK, pp 102-111, 1982.
4. King AS, McLelland J: Birds: Their Structure and Function. London, UK, Bailliere Tindall, 1984.
5. Bock WJ: An approach to the functional analysis of bill shape. Auk 83:10-51, 1966.
6. Taylor M: Anatomy and physiology of the gastrointestinal tract for the avian practitioner. *In* Birds 2000. Post Grad Found in Vet Sci, Uni of Sydney, Aus. Proc 334:107-113.
7. Lack D: Darwin's Finches. Cambridge University Press. Cambridge, UK, 1947.
8. Richardson KC, Wooler RD: Adaptations of the alimentary tracts of some Australian lorikeets to a diet of pollen and nectar. Aust J Zool 38:581-586, 1990.
9. Churchill DM, Christensen P: Observations on pollen harvesting by brush-tongued lorikeets. Aust J Zool 18:427-437, 1970.
10. McLelland J. Digestive System. *In* King AS, McLelland J (eds): Form and Function in Birds. Academic Press, London, pp 69-181, 1979.
11. Berkhoudt H: Special sense organs: Structure and function of the avian taste receptors. *In* King AS, McLelland J (eds): Form and Function in Birds, Vol 3. New York, NY Academic, pp 462-496, 1985.
12. Ganchrow D, Ganchrow JR: Number and distribution of taste buds in the oral cavity of hatchling chicks. Physiol Behav 34:889-894, 1985.
13. Jerrett SA, Goodge WR: Evidence for amylase in avian salivary glands. J Morphol 139:27-46, 1973.
14. Evans HE: Anatomy of the budgerigar and other birds. *In* Rosskopf WJ, Woerpel RW (eds): Diseases of Cage and Aviary Birds, 3rd Ed. London, UK, Williams & Wilkins, pp79-162, 1996.
15. Horseman ND, Buntin JD: Regulation of pigeon crop-milk secretion and parental behaviors by prolactin. Annu Rev Nutr 15:213-238, 1995.
16. Ziswiler V, Farner DS: Digestion and the digestive system. *In* Farner DS, King JR (eds.) Avian Biology II. London, England: Academic Press, pp 343-430, 1972.
17. Soedarmo D, Kare MR, Wasserman RH: Observations on the removal of sugar from the mouth and crop of the chicken. Poult Sci 40:123-141, 1961.
18. Gartrell BD: The nutritional, morphologic, and physiologic bases of nectarivory in Australian birds. J Avian Med Surg 14(2):85-94, 2000.
19. Vergara P, Ferrando C, Jimenez M, et al: Factors determining gastrointestinal transit time of several markers in the domestic fowl. Quart J Exp Physiol 74:867-874, 1989.
20. Duke GE: Recent studies on regulation of gastrointestinal motility in turkeys. Poultry Sci 71:1-8, 1992.
21. Duke GE: Gastrointestinal motility and its regulation. Poul Sci 61:1245-1256, 1989.
22. Duke GE, Place AR, Jones B: Gastric emptying and gastrointestinal motility in Leach's Storm-Petrels chicks (*Oceanodroma leucchorboa*). Auk 106:80-85, 1989.
23. Hill KJ: The physiology of digestion. *In* Bell DJ, Freeman BM (eds): Physiology and Biochemistry of the Domestic Fowl. London, UK, Academic, pp 1-49, 1971.
24. Sell JL, Angel CR, Piquer FJ, et al: Developmental patterns of selected characteristics of the gastrointestinal tract of young turkeys. Poult Sci 70:1200-1205, 1991.
25. Richardson KC, Wooller RD: The structures of the gastrointestinal tracts of honeyeaters and other small birds in relation to their diets. Aust J Zool 6;34:119-124, 1986.
26. Waine JC: Post -mortem examination technique. *In* Beynon (ed): Manual of Raptors, Pigeons and Waterfowl. BSAVA, Gloucestershire, UK, p 103, 1996.
27. Clench MH, Mathias JR: The avian cecum- a review. Wilson Bull 107:93-121, 1995.
28. Taylor M, Murray M: A diagnostic approach to the avian cloaca. Proc Assoc Avian Vet 1999, pp 301-304.
29. Black DG: Cloacal prolapse in immature ostrich hens. *In* Cross, GM (ed): Proc Aust Committee of Assoc Avian Vets 1992 Annual Conference, pp 114-116.
30. Klasing KC: Comparative Avian Nutrition. Wallingford, UK, CAB International, 1998.
31. Taylor M, Murray MJ: The psittacine cloaca: A clinical review. Proc Assoc Avian Vet, Monterey, CA, 2002, pp 265-269.
32. Cannon M: Diagnosis, differential diagnosis & treatment of swellings of structures other than the abdomen (Lumps & Bumps). Proc Assoc Avian Vet Aust Comm, Echuca, VIC 2000 pp 43-61.
33. Perry RA, Gill J, Cross GM: Disorders of the avian integument. Vet Clin North Am Small Anim Pract 21:1302-1327, 1991.
34. Speer B L: Non-infectious diseases. *In* Abramson J, Speer BL, Thomsen JB: The Large Macaws. Raintree Publications, pp 323-324, 1995.
35. Romagnano A: Avian pediatrics. Proc Assoc Avian Vet, Monterey, CA, 2002, pp 287-296.
36. Doneley RJ: Eye, beak and neck problems in ostriches. Ostrich Odyssey '95.Aust Ost Assoc ACT AUS pp 81-83, 1995.
37. Black D: Ostrich examination-what to look for! Ostrich Odyssey. Post Grad Com in Vet Sci Uni of Sydney, pp 99-111, 1995.
38. Speer BL: Trans-sinus pinning technique to address scissors-beak deformities in psittacine species. Proc Assoc Avian Vet Aust Comm, Gold Coast, QLD, pp 283-290, 2002.
39. Olsen GH: Problems of the bill and oropharynx. *In* Olsen GH, Orosz SE (eds): Manual of Avian Medicine. St. Louis, MO, Mosby Inc, pp 359-368, 2000.
40. Martin H, Ritchie BW: Orthopedic surgical techniques. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing Inc. pp 1165-1169, 1994.
41. Flammer K, Clubb SL: Neonatology. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing Inc., pp 805-838, 1994.
42. Altman RB, Forbes NA: Self-assessment Color Review of Avian Medicine. Iowa State University Press. Ames, Iowa, pp 99-100, 1998.
43. Austic RF, Scott ML: Nutritional diseases. In Calnek B, et al (eds): Diseases of Poultry. Ames, Iowa State University Press, pp 45-71, 1991.
44. Lumeij JT: Gastroenterology. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing Inc., pp 482-521, 1994.
45. Carpino MR, Phalen D: Beak deformities associated with malnutrition in hand fed pediatric African Grey Parrots or "Ruffles with ridges syndrome". Proc Assoc Avian Vet, Reno, 1997, pp 393-396.
46. Graham DL: The Morphological bases of common alimentary tract disorders unique to birds. Proc Assoc Avian Vet Aust Comm, O'Reilly's, QLD, 1996, pp 67-76.
47. Crosta L: Alloplastic and heteroplastic bill prostheses in 2 Rhamphastidae birds. J Avian Med Surg 16(3):218-222, 2002.
48. Harrison GJ: Disorders of the integument. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, PA, WB Saunders, pp 509-524, 1986.
49. Molnar L, Ptacek M: Traumatic injuries of beak and talon of captive raptors. Proc Assoc Avian Vet Europ Comm 2001, Munich, pp 246-247.
50. Boydell IP, Forbes NA: Disease of the head (including the eyes). *In* Beynon PH (ed): Manual of Raptors, Pigeons and Waterfowl. Cloucestershire UK, BSAVA Ltd., pp 140-146, 1996.
51. Clipsham R: Rhamphorthotics and surgical corrections of maxillofacial defects. Semin Avian Exotic Pet Med, 3:92-99, 1994.
52. Altman RB: Acrylic beak repair. Proc Assoc Avian Vet Aust Comm, O'Reilly's, QLD, 1996, pp 137-139.
53. Olsen GH, Carpenter JW, Langenberg JA: Medicine and surgery. *In* Ellis DH, Gee GF, Mirande CM (eds): Cranes: Their Biology, Husbandry and Conservation. Blaine, WA:Hancock House Publishers, pp 168-174, 1996.
54. Foerster SH, Gilson SD, Bennett RA: Surgical correction of palatine bone luxation in a blue-and-gold macaw (*Ara ararauna*). J Avian Med Surg 14(2):118-121, 2000.
55. Ritchie B: Circoviridae. *In* Avian Viruses, Function and Control. Lake Worth, FL, Wingers Publishing, pp 223-252, 1995.
56. Raidal SR, Sabine M, Cross GM: Laboratory diagnosis of psittacine beak and feather disease by haemagglutination and haemagglutination inhibition. Aust Vet J 70:133-137, 1993.
57. Schmidt RE: Pathology of gas-

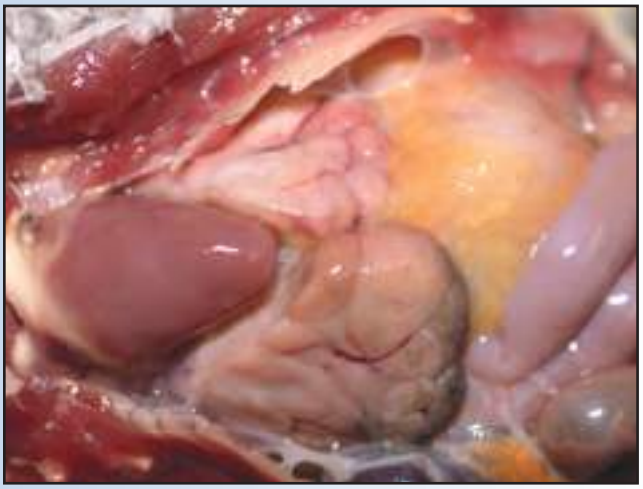
trointestinal diseases of psittacine birds. Semin Avian Exotic Pet Med 2:75-82, 1999.
58. Malley AD, Whitbread TJ: The integument. *In* Beynon (ed): Manual of Raptors, Pigeons and Waterfowl. BSAVA Gloucestershire, UK, pp 129-139, 1996.
59. Ritchie B: The poxviridae. *In* Avian Viruses, Function and Control. Lake Worth, FL, Wingers Publishing, pp 285-311, 1995.
60. Bauck L, Brash M: Survey of diseases of the Lady Gouldian finch. Proc Assoc Avian Vet 1999, New Orleans, LA, pp 204-212.
61. Ritchie BW: Papovaviridae. *In* Avian Viruses: Function and Control. Lake Worth, FL, Wingers Publishing, pp 136-179, 1995.
62. Lu YS, Lin DF, Lee YL, et al: Infectious bill atrophy syndrome caused by a parvovirus in a co-outbreak with duck viral hepatitis in ducklings in Taiwan. Avian Diseases 37:591.
63. Doneley R: Cutaneous cryptococcus in an African grey parrot. Proc Assoc Avian Vet Aust Comm, Currumbin, QLD, 1994, p 247.
64. Cannon M: Avian cryptococcosis-a case report. Proc Assoc Avian Vet Aust Comm, Noosa, QLD, 1999, pp 1-5.
65. Raidal SR, Butler R: Chronic rhinosinusitis and rhamphothecal destruction in a Major Mitchell's cockatoo (*Cacatua leadbeateri*) due to *Cryptococcus neoformans var gattii*. J Avian Med Surg 15(2):121-125, 2001.
66. Bauck L: A clinical approach to neoplasia in the pet bird. Semin Avian Exotic Pet Med 1:65-72, 1992.
67. Ritchie BW: Herpesviridae. *In* Avian Viruses: Function and Control. Lake Worth, FL, Wingers Publishing, pp 171-222, 1995.
68. Samour JH: *Pseudomonas aeruginosa stomatitis* as a sequel to trichomoniasis in captive saker falcons (*Falco cherrug*). J Avian Med Surg. 14(2):113-117, 2000.
69. Van Der Heyden N: Clinical manifestations of mycobacteriosis in pet birds. Semin Avian Exotic Pet Med 6:18-24, 1997.
70. Mendenhall MK, Ford SL, Emerson CL, et al: Brief communications: Detection and differentiation of *Mycobacterium avium* and *Mycobacterium genavense* by polymerase chain reaction and restriction enzyme digestion analysis. J Vet Diagn Invest, 12:57-65, 2000.
71. Antinoff N, Kiehn TE, Bottger EC: Mycobacteriosis caused by *Mycobacterium genavens*e in a psittacine bird. Proc Assoc Avian Vet, Tampa, FL, 1996, pp 169-170.
72. Hoefer HL, Liu SK, Kiehn TE et al: Systemic *Mycobacterium tuberculosis* in a Green Winged Macaw. Proc Assoc Avian Vet, Tampa, FL, 1996, pp 167-168.
73. VanDerHeyden N: New strategies in the treatment of avian mycobacteriosis. Semin Avian Exotic Pet Med, 6:25-33, 1997.
74. Lennox A: Successful treatment of mycobacteriosis in three psittacine birds. Proc Assoc Avian Vet, Monterey, CA, 2002, pp 111-113.
75. Morrisey JK: Gastrointestinal diseases of psittacine birds: Semin Avian Exotic Pet Med, 2:66-74, 1996.
76. Suedmeyer WK, Haynes N, Roberts D. Clinical management of endoventricular mycoses in a group of African finches. Proc Assoc Avian Vet, Reno, 1997, pp 225-227.
77. Madill DN: Parasitology. *In* Birds 2000. Post Grad Found in Vet Science. Uni of Sydney, Aus, pp 351-381, 2000.
78. Forbes NA: Chronic weight loss, vomiting and dysphagia. *In* Manual of Raptors, Pigeons and Waterfowl. BSAVA. Gloucestershire UK, pp 189-196, 1996.
79. Murphy J: Psittacine trichomoniasis. Proc Assoc Avian Vet, New Orleans, 1992, pp 21-24.
80. Ward FP: A clinical evaluation of parasites of birds of prey. J Zoo Anim Med, 6:3-8, 1975.
81. Rupiper DJ, Carpenter JW, Mashima TY: Formulary. *In* Manual of Avian Medicine. St. Louis, MO, Mosby Inc, 2000.
82. McDonald D: Vitamins and supplementation requirements. Proc Assoc Avian Vet Aust Comm, Gold Coast, QLD, 2002, pp 9-16.
83. Styles DK, Phalen DN, Tomaszewski EK: Elucidating the etiology of avian mucosal papillomatosis in psittacine birds. Proc Assoc Avian Vet, Monterey CA, 2002, pp 175-178.
84. Latimer KS, Niagro FD, Rakich PM, et al: Investigations of parrot papilloma virus in cloacal and oral papillomas of psittacine birds. Vet Clin Pathol 26:158-163, 1997.
85. Hoefer HL: Diseases of the gastrointestinal tract. *In* Altman R, Clubb S, Dorrestein GM, et al (eds): Avian Medicine and Surgery. Philadelphia, PA, Saunders, p 421, 1997.
86. Rosskopf W: Digestive system disorders. *In* Rosskopf W, Woerpel R (eds): Diseases of Cage and Aviary Birds, 3rd Ed. Baltimore MD, Williams & Wilkins, pp 436-448, 1996.
87. Gelis S: Avian reproduction and reproductive disorders. *In* Birds 2000. Post Grad Found in Vet Science. Uni of Sydney, Aust, pp 187-205, 2000.
88. Ryan TP: Grit impaction in 2 neonatal African grey parrots. J Avian Med Surg 16(3):230-233, 2002.
89. Ritchie BW, Gregory CR, Latimer K, et al: Progress in preventing PDD, polyomavirus, and PBFD virus. Proc Assoc Avian Vet, St Paul, 1998, pp 25-40.
90. Taylor M: Disorders of the avian digestive system. *In* Birds 2000. Post Grad Found in Vet Science. Uni of Sydney, Aust, pp 407-416, 2000.
91. Gregory CR, Ritchie BW, Latimer KS et al: Progress in understanding proventricular dilatation disease. Proc Assoc Avian Vet, Portland, 2000, pp 269-275.
92. Dahlhausen B, Aldred MD, Colaizzi E: Resolution of clinical proventricular dilatation disease by cyclooxygenase 2 inhibition. Proc Assoc Avian Vet, Monterey, CA, 2002, pp 9-12.
93. Gerlach H: Going light in budgerigars (*Melopsittacus undulatus*). Proc Assoc Avian Vet, Miami, FL, 1986, pp 247-250.
94. Filippich L, O'Boyle D, et al: Megabacteria in birds in Australia. Aust Vet Pract. 23(2):71, 1993.
95. Hargreaves R: A fungus commonly found in the proventriculus of small birds. 30th Western Poultry Disease Conference and the 215th Poultry Health Symposium, Cooperative Extension Service, Uni of California, Davis,CA 1981.
96. Greenacre C B, Wilson GH, Graham JE: The many faces of megabacterium. Proc Assoc Avian Vet, Portland, 2000, pp 193-196.
97. Huchzermeyer F, Henton M, et al: High mortality associated with megabacteriosis of proventriculus and gizzard in ostrich chicks. Vet Rec 133:143, 1993.
98. Miller R, Sullivan N: A retrospective analysis of ostrich diseases in Queensland and New South Wales (1992-1994). Ostrich Odyssey 1994.
99. Filipich LJ, Parker MG: Megabacteria in wild birds in Australia. Aust Vet Pract 24(2):84, 1994.
100. Macwhirter P: Megabacteria in birds. Control and Therapy. 183:760, 1995.
101. Henderson GM, Gulland FMD and Hawkey CA: Hematological findings in budgerigars with megabacteria and trichomonas infections associated with "going light". Vet Rec 123:492-494, 1988.
102. Tomaszewski B, Snowden K, Phalen D. Rethinking megabacteria. Proc Assoc Avian Vet (Research Forum), Portland, OR, 2000.
103. Phalen DN, Tomaszewski E, Davis A: Investigation into the detection, treatment and pathogenicity of avian gastric yeast. Proc Assoc Avian Vet. Monterey, CA 2002, pp 49-51.
104. Baker JR: Clinical and pathological aspects of "going light" in exhibition budgerigars (*Melopsittacus undulatus*). Vet Rec 16:406-408, 1985.
105. Bredhauer MG: Megabacteria and proventricular/ventricular disease in Australian birds. Cross GM (ed) Proc Assoc Avian Vet Aust Comm, O'Reilly's QLD 1996, pp 27-33.
106. Taylor M, Dobson H, Hunter DB, et al: The functional diagnosis of avian gastrointestinal disease- an update. Proc Assoc Avian Vet, New Orleans, 1999, pp 85-86.
107. Gerlach H: Bacteria. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Applications, Lake Worth, FL, Wingers Publishing, pp 482-521, 1994.
108. Filippich LJ, Parker MG: Megabacteria and proventricular/ventricular disease in psittacines and passerines. Proc Assoc Avian Vet, Currumbin, QLD, 1994, pp 287-293.
109. Gerlach H. Megabacteriosis. Semin Avian Exotic Pet Med 2001, 10:12-19.
110. Moore RP, Snowden KF, Phalen DN: Diagnosis, treatment and prevention of megabacteriosis in the budgerigar (*Melopsittacus undulatus*). Proc Assoc Avian Vet Aust Comm, Hobart, TAS, 2000, pp 95-101.
111. Filippich LJ: Megabacteria and proventricular disease in birds. Proc Assoc Avian Vet Aust Comm, 1992, pp 1-12.
112. Lublin A, Mechani S, Malkinson M, et al: A five-year survey of megabacteriosis in birds of Israel and a biological control trial. Proc Assoc AviaVet, St Paul, 1998, pp 241-245.
113. Gelis S, Raidal S: Cryptosporidiosis in finches. Proc Assoc Avian Vet Aust Com. Echuca, VIC, 2000, pp 327-330.
114. Sulaiman IM, Morgan UM, Thompson RC et al: Phylogenetic relationships of cryptosporidium parasites based on the 70-kilodalon heat shock protein (HSP 70) gene. Appl Environ Microbiol. Aug, 65(8):3386-91.
115. Black D. Common surgical procedures. *In* Ostrich Odyssey. Post Grad Comm Vet Sci, pp 117-119, 1993.
116. Putter G: A non-surgical method of treatment of proventricular impaction in ostriches. Proc Assoc Avian Vet, Tampa, FL, 1996, pp 131-134.
117. Bennett RA: Surgery of the avian gastrointestinal tract. Proc Assoc Avian Vet Aust Comm, Hobart, TAS, 2001, pp 47-51.
118. Atkinson R: Heavy metal poisoning in psittacines and waterfowl. Proc Assoc Avian Vet 1995, pp 443-446.
119. Forbes NA: Nervous diseases. *In* Manual of Raptors, Pigeons and Waterfowl. BSAVA. Gloucestershire UK, pp 320-321, 1996.
120. Samour JH, Naldo J: Diagnosis and therapeutic management of lead toxicosis in falcons in Saudi Arabia. J Avian Med Surg 16(1):16-20, 2002.
121. Van Sant F: Zinc and parrots: more than you ever wanted to know. Proc Assoc Avian Vet, St Paul, 1998, pp 305-312.
122. Rosenwax A: Utilisation of blood lead levels. Proc Assoc Avian Vet Aust Comm, Noosa, QLD, 1999, pp 163-169.
123. De Voe R, Degernes L, Karli K: Dysplastic koilin causing obstruction in an Eclectus parrot (*Eclectus roratus*). Proc Assoc Avian Vet, Monterey, CA, 2002, pp 107-109.
124. Schmidt RE: Morphologic diagnosis of avian neoplasms. Semin Avian Exotic Pet Med 1:73-79, 1992.
125. Graham DL: The morphological bases of common alimentary tract disorders unique to birds. Proc Assoc Avian Vet Aust Comm, O'Reilly's, QLD, 1996, pp 67-76.
126. Deriese LA, Chiens K, Dehedt P, et al: *Enterococcus hirae* infections in psittacine birds-epidemiologic, pathological and bacteriological observations. Avian Pathol 24:523-531, 1995.
127. Button K: Enteritis in Australian ostrich chicks. Ostrich Odyssey '95. Proc Fifth Aust Ostrich Assoc Conf Gold Coast Aust, 1995, pp 105-110.
128. Ritchie BW: Adenoviridae. *In*

Ritchie BW: Avian Viruses: Function and Control. Lake Worth, FL, Wingers Publishing, pp 313-333, 1995.
129: Greiner EC, Ritchie BW: Parasites. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers, pp 1007-1029, 1994.
130. O'Donoghue PJ: *Cryptosporidium* and cryptosporidiosis in man and animals, Int J Parasitol, 25:139-195, 1995.
131. Morgan UM, Xiao L, Limor, J et al: *Cryptosporidium meleagridis* in an Indian ring-necked parrot (*Psittacula krameri*). Aust Vet Journ, 78(3):182-183, 2000.
132. Snowden K, Barton C, Pulpurampil N, et al: Avian microsporidiosis. Proc Assoc Avian Vet Aust Comm, Canberra, ACT, 1998, pp 31-33.
133. Carlisle MS, Snowden K, Gill J, et al. Microsporidiosis in a Gouldian finch (*Erythrura* [*Chloebia*] *gouldiae*. Aust Vet J 80:41-44, 2002.
134. Fudge A: Cockatiel disorders seen in the USA. Proc Assoc Avian Vet Aust Comm, Hobart, TAS, 2001, pp 83-92.
135. Filippich LJ, O'Donoghue PJ: *Cochlosoma* infection in finches. Proc Assoc Avian Vet Aust Comm, Dubbo, NSW 1995, pp 101-104.
136. Scott RJ: Ascaridiasis and gastrointestinal stasis in a hyacinth macaw. AAV Newsletter and Clinical Forum. June-August 2002, pp 7-10.
137. Howard LL, Papendick R, Stalis IH, et al: Fenbendazole and albendazole toxicity in pigeons and doves. J Avian Med Surg 16(3):203-210, 2002.
138. Filippich LJ: The effectiveness of anthelmintics in finches. Proc Assoc Avian Vet Aust Comm.Currumbin QLD 1994, pp 141-143.
139. Black D: Troubleshooting reproductive problems. *In* Ostrich Odyssey '95. Proc Fifth Aust Ostrich Assoc Conf Gold Coast, QLD, 1995, pp 99-104.
140. Speer B: A clinical look at the avian pancreas in health and disease. Proc Assoc Avian Vet, St Paul, 1998, pp 57-64.
141. Doneley B. Acute pancreatitis in psittacine birds. Proc Assoc Avian Vet Aust Comm.Echuca VIC 2000, pp 261-267.
142. Kennedy FA, Sattler-Augustin S, Mahler JR, et al: Oropharyngeal and cloacal papillomas in two macaws with neoplasia with hepatic metastasis. J Avian Med Surg 10:89-95, 1996.

CHAPTER
15

Evaluating and Treating the Liver

MANFRED HOCHLEITHNER, Dr med vet, Dipl ECAMS;
CLAUDIA HOCHLEITHNER, Dr med vet; LORI DANIELLE HARRISON, DVM



Hepatic dysfunction is among the most common medical problems seen in companion birds.[7,16] In many cases, the involvement of the liver in the overall disease process is not obvious. This chapter reviews the basic pathophysiology, diagnosis, clinical signs and the current therapies for liver disease.

The Role and Function of the Liver

The liver is the largest organ in the body, but its relative size varies among species. The liver performs many critical functions that maintain homeostasis. Liver functions include synthesis of cholesterol and bile acids and the generation and utilization of glycogen. It also is the site for metabolism of various substances in preparation for their excretion from the body via the bile or urine. It is the site of production of plasma proteins, albumin, fibrinogen, lipoproteins and a variety of alpha and beta globulins. The liver produces all clotting factors. The liver filters from the blood infectious agents and foreign materials that have been absorbed by the intestines. This helps prevent these materials from gaining access to the systemic circulation. Energy is produced by oxidative phosphorylation and beta-oxidation of fatty acids in hepatic mitochondria. This energy is used to sustain the activity of the liver and to provide a reservoir of glycogen for the body.

HEPATIC DYSFUNCTION

Hepatic dysfunction occurs after severe injury or repeated significant insults. The liver has considerable functional reserve and regenerative capacity. Only lesions that affect the majority of hepatic parenchyma are likely to produce the signs of hepatic failure. Focal lesions rarely destroy sufficient parenchyma to deplete the liver's

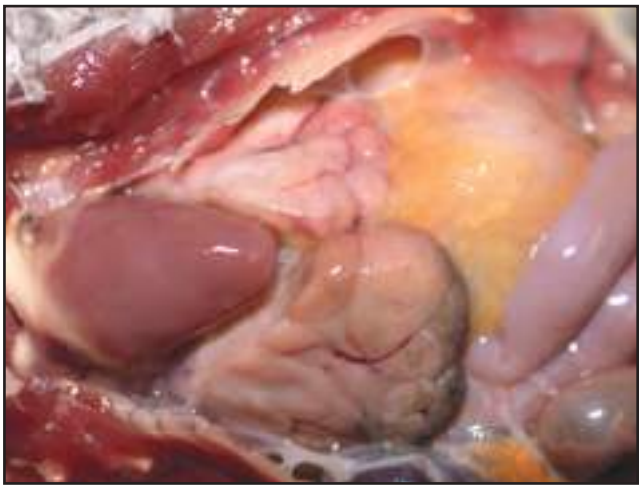


Fig 15.1 | The marked change in color and liver texture are characteristic of cirrhosis.

reserve. The term hepatic failure implies loss of adequate function from either acute or chronic damage, however, usually not all functions are lost at the same time.

METABOLIC DISTURBANCES AS A RESULT OF HEPATOPATHY

Decreased hepatic function can be manifested by a variety of metabolic disturbances. The type and duration of the disorder may influence the nature of the metabolic perturbation. Coagulopathies, hypoalbuminemia, cutaneous manifestations and increased resistance in the blood flow through the liver due to fibrosis are common.

RESPONSE OF THE LIVER

The destruction of hepatic parenchyma results in regeneration, fibrosis and/or biliary hyperplasia. The liver can rapidly and efficiently regenerate lost hepatic mass. Extensive hepatic necrosis is usually followed by parenchymal regeneration without scarring, as long as the normal extra-cellular matrix remains intact. Chronic hepatic injury most commonly manifests as fibrosis. Early fibrosis may respond to treatment or removal of the source of injury, but more advanced fibrosis is generally irreversible with the therapies currently available. Hepatic fibrosis can be produced by a variety of hepatic injuries. The hepatic stellate cells change from the typical lipid-storing cells to cells with a myofibroblastic appearance that subsequently develop the ability to synthesize collagen, leading to hepatic fibrosis. Stellate cells can be activated by various cytokines produced either by inflammatory cells that infiltrate damaged hepatic parenchyma or by constituent cells of the liver (Kupffer cells, endothelial cells, hepatocytes). Damage to the extracellular matrix also stimulates activation of hepatic stellate cells, as do various toxins. End-stage liver disease or cirrhosis as a result of chronic fibrosis is characterized by loss of normal hepatic architecture due to nodular regeneration of parenchyma, fibrosis and, often, biliary duct hyperplasia (Fig 15.1). Often, clinical manifestations of hepatic failure do not occur until the end stage. The cause of the hepatic damage that leads to end stage liver failure frequently cannot be determined by the time signs of hepatic failure are observed.

Diagnosis and Clinical Signs of Liver Disease

Clinical signs of hepatic failure in birds are variable and can range from mild inappetence and inactivity to acute hemorrhage and death. A large percent of liver tissue must be affected before any obvious clinical signs are observed because of the liver's large functional reserve (Table 15.1).

All birds on a poor diet (seeds, poor-quality formulated diets) should be presumed to have decreased functional hepatic mass (see The Improper Diet Cascade in Chapter 4, Nutritional Considerations: Section II, Nutritional Disorders). The liver's ability to regenerate may mask underlying liver disease in its early stages.

There are some clinical signs that are highly suggestive of hepatic failure, however, none are pathognomonic. Hepatic failure is associated with yellow or green discolorations of the urates and/or the feces, which may result from biliverdinuria or bilirubinuria (Fig 15.2). Feathers develop a glossy black color due to the exposure of melanin from total loss of normal green or blue pigment. Hepatomegaly and/or ascites may cause dyspnea. Weight loss, poor feathering, diarrhea, overgrowth and bruising of the beak and nails, bruising or bleeding of the skin

Table 15.1 | **Clinical Signs of Liver Disease**

Clinical Sign	Non-specific	More Specific
Anorexia	✓	
Lethargy	✓	
Weight loss	✓	
Weakness	✓	
Diarrhea	✓	
Polyuria	✓	
Polydipsia	✓	
Poor feathers	✓	
Dyspnea	✓	
Green or yellow urates		✓
Abdominal swelling		✓
Ascites		✓
Coagulopathies		✓
Melena		✓
Abnormal beak/nails		✓
Malcolored feathers		✓

Fig 15.2 | Yellow urates. See Chapter 23, Diagnostic Value of Biochemistry, for a discussion of biliverdin (green) versus bilirubin (yellow).

and prolonged clotting time may be associated with hepatic failure. Clinical disorders of any type that frequently recur or are resistant to diagnosis and therapy often involve liver malfunction. Diagnostic testing is used to support the diagnosis of liver disease (Table 15.2).

CLINICAL PATHOLOGY

Analytes such as alanine aminotransferase (ALT), lactate dehydrogenase (LDH) and serum alkaline phosphatase (SAP) are not sensitive or specific for liver disorders in birds[7,11] (see Chapter 23, Diagnostic Value of Biochemistry). Aspartate amino transaminase (AST) is sensitive but not specific for hepatocyte necrosis in birds because AST also is released from damaged muscle tissue. AST levels should be examined in conjunction with creatine phosphokinase (CPK). If only AST is elevated, liver damage is more likely.

Elevations of bile acids are often consistent with hepatic insufficiency and decreased liver function.[16] Therefore, bile acid assays are useful in determining chronic, long-standing and non-inflammatory states of decreased hepatic function that may not be reflected in hepatic enzyme elevation. Since food (protein) does not affect bile acids in psittacines, pre- and postprandial sampling is unnecessary. Species differences may exist. See Chapter 23, Diagnostic Value of Biochemistry.

An elevated white blood cell count (WBC) is common in bacterial and fungal infections, while a depressed WBC is more common in toxic and viral disorders. Lowered serum total solids, hypoalbuminemia, hyperglobulinemia, and hypokalemia also are indicative of hepatic compromise.

Biliverdin is the most important bile pigment in birds; therefore, icterus is not a typical clinical sign of hepatic failure. Carotene pigments from the diet may be responsible for yellow skin and plasma color.[14] A study in broilers with gross hepatic bile duct lesions traced the yellow color of the pericardium and carcass to bilirubin.[19] In some cases, bacterial reduction may be responsible for the degradation of avian biliverdin to bilirubin. One practitioner has found elevated bilirubin levels in cockatiels with yellow urine, urates and feathers that were normally white, but turning yellow to gold (G. Harrison, personal communication). Other liver function tests are available, but currently their use in the practical setting is limited. These tests include clearance of indocyanine green and bromsulphalein.

Table 15.2 | Diagnostic Approach to Assess Liver Disease in Birds

History	
Baseline testing	Complete blood count/chemistries/urinalysis
Non-invasive imaging	Radiology and ultrasound
Screening for infectious diseases	See Chapter 21, Preventive Medicine and Screening
Liver function testing	Bile acids
Abdominocentesis	Culture, cytology, chemistries
Coagulation testing	See Chapter 23, Diagnostic Value of Biochemistry
Liver biopsy	Culture, cytology, histopathology

Table 15.3 | Radiographic Findings

Common Radiographic Findings	Interpretation	Comments
Hepatomegaly	Hepatic lipidosis, cardiac-portal hypertension, infection (viral, bacterial, fungal)	The liver shadow of neonates can appear bigger because the proventriculus in neonates is relatively larger.
Normal hepatic size	Early stage of hepatopathies	Normal size may indicate normal liver.
Microhepatia	End stage cirrhosis, congenital small liver, emaciation, chronic malnutrition	Species predilection exists: umbrella, Moluccan and palm cockatoos, macaws.

Table 15.4 | Hepatomegaly

Common Causes in Neonates	Common Causes in Adults
Normal neonate	Obesity
Hepatic lipidosis	Hepatic lipidosis
Hepatic hematoma	Bacterial hepatitis: *Chlamydophila* spp., *Mycobacterium* spp.
Viral hepatitis	Lymphoma
Lymphoma	Hepatic adenocarcinoma
	Fungal hepatitis
	Viral hepatitis: herpes
	Iron storage disease
	Ovarian disorders

RADIOLOGY

Hepatomegaly and microhepatia are common findings in birds. Great variations appear among psittacine species but also between individuals. Baseline radiographs of each individual bird should be taken as a part of the routine examination and may be used for comparison in subsequent years (Tables 15.3, 15.4).

15_Liver.qxd 8/23/2005 5:16 PM Page 444

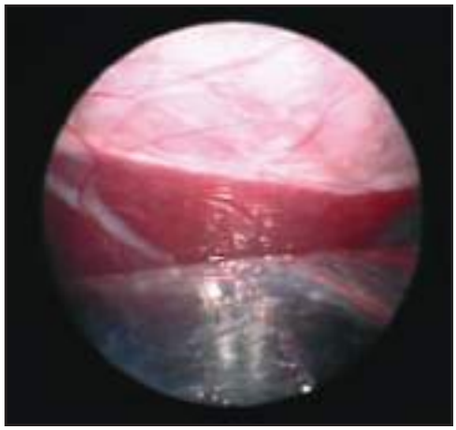


Fig 15.3 | The normal liver with a sharp border and dark brown mahogany color as seen through the membranes of the air sacs.

ULTRASOUND

This test provides information concerning the size and structure of the liver. In most cases, it is not stressful. To prevent hypothermia and ensure good contact of the probe with the bird, warmed lubricating jelly should be used.

ENDOSCOPIC EXAMINATION AND BIOPSY

Liver biopsies are frequently utilized to establish a definitive diagnosis of the pathophysiology of liver disease (Fig 15.3). The risk to the patient in order to gain this information must be considered. Coagulopathies associated with hepatopathies increase the risk of severe bleeding. These authors find very few situations in which liver biopsies are advantageous to the individual avian patient. Many infectious agents responsible for hepatopathy can be diagnosed via serology. Viral infections that are not readily diagnosed via serology are often of short duration. When a diagnosis has been made, treatment for many pathological conditions is purely supportive. Liver biopsy will not change the prognosis in most cases of hepatic dysfunction in parrots. Exceptions would include infection with *Mycobacterium* spp., various neoplasias and conditions unresponsive to therapy. In an aviary situation, hepatic biopsy or submission of necropsy specimens can be a significant means of identifying and eliminating disease. The risk of loss of an individual may benefit the entire flock and to justify the procedure.

A retrospective study of liver biopsies of 71 birds was performed in a zoological collection. Important diagnostic findings included the identification of iron storage disease in mynahs and toucans; presumptive *Atoxoplasma* hepatitis in mynahs; chronic active hepatitis associated with intestinal coccidial infections; and acid-fast organisms or severe amyloidosis in ducks and geese suspected of harboring mycobacteria.[17] The information was useful, but the 4.2% death rate would not be acceptable in pet bird practice (see Chapter 24, Diagnostic Value of Endoscopy and Biopsy).

Non-infectious Diseases of the Liver

The liver's function as a primary organ of filtration makes it susceptible to a myriad of environmental toxins and irritants (Table 15.5).

HEMATOMA

Hepatic hematoma may present similarly to or in conjunction with pediatric hepatic lipidosis. Hepatic hematoma often occurs in young birds because they have a prominent abdomen, which provides very little keel protection for the underlying organs. In obese birds, the liver is more friable and extends farther into the unprotected region of the caudal abdomen. The hematoma may be visible through the abdominal skin. The extent of anemia and the bird's physical condition will dictate treatment decisions. Parenteral fluids must be given with care. Homologous blood or plasma transfusions can be beneficial using guidelines as in mammals. The ultimate treatment is husbandry and the appropriate amounts of a proper diet.

HEPATIC LIPIDOSIS

Lipids are normally transported to the liver from the gastrointestinal tract and adipose tissue in the form of chylomicrons and free fatty acids, respectively. Within hepatocytes, free fatty acids are esterified to triglycerides that are complexed with apoproteins to form low-density lipoproteins, which are released into plasma as a readily available energy source. Hepatic lipidosis, or fatty liver, occurs when the rate of triglyceride accumulation within hepatocytes exceeds either their rate of metabolic degradation or their release as lipoproteins. Hepatic lipidosis is not a specific disease entity but can occur as a sequel to a variety of perturbations of normal lipid metabolism. Excessive dietary intake of fat or increased mobilization of triglycerides from adipose tissue subsequent to increased demand, such as in starvation or endocrine abnormalities, may be responsible (see Chapter 4, Nutritional Considerations: Section II, Nutritional Disorders). Abnormal hepatocyte function can lead to an accumulation of triglycerides due to decreased energy for oxidation of fatty acids. Excessive dietary intake of carbohydrates can result in the increased synthesis of fatty acids and excessive triglyceride formation. Hepatotoxins or drugs can impair secretion of lipoprotein from the liver.

Table 15.5 | Causes of Non-infectious Liver Disease

- Congenital
- Traumatic: hematoma
- Hepatic lipidosis: pediatric versus adult
- Amyloidosis
- Iron storage disease
- Toxins
- Malnutrition

Decreased apoprotein synthesis can occur subsequent to decreased production and export of lipoprotein from hepatocytes. Deficiencies of the amino acids biotin, choline and methionine have been shown to cause hepatic lipidosis in mammals and some species of birds. More than one defect can occur in any given hepatic disorder. Hepatic lipidosis is most commonly seen in the neonate and the obese, malnourished adult bird. See Chapter 19, Endocrine Considerations.

Hepatic Lipidosis (*Pediatric*): The liver in neonates is typically larger relative to the total body weight than in adult birds. Baby birds affected with hepatic lipidosis present with a common history of being handfed with a commercial formula to which the owners have added a high-fat supplement. The higher fat content of macaw hand-feeding formulas is also implicated in this syndrome when these formulas are inappropriately used on other species (especially cockatoos). Affected babies are often grossly overweight for their age and exhibit severe respiratory distress. These birds must be handled gently and minimally to avoid exacerbation of the condition. Cool oxygenation is the best first step. The enlarged liver reduces lung and air sac capacity. These neonates usually present when the stress of feeding and breathing at the same time has exceeded their oxygen reserves. General nutritional changes that are required include reducing the quantity of food per feeding, adjusting the fat type and content and adding lactulose to the formula. Oral or parenteral fluid supplementation, when tolerated, should be added to keep the initially hyperthermic bird hydrated and to detoxify the body. When possible, further diagnostic testing should be pursued to check for concurrent infection or other diseases (Table 15.6).

Hepatic Lipidosis (*Adult Bird*): A second group that is predisposed to hepatic lipidosis is the obese adult bird being fed a high-fat diet, which is common in birds with ovarian disorders and hypercholesterolemia. This syndrome can occur in many species, but seems most common in *Amazona* spp. Long-standing malnutrition in an obese bird contributes to the cause of this syndrome. Septicemia can occur secondarily. The most common clinical presentation is an anorectic bird with a quiet demeanor, although other signs of hepatic dysfunction

Table 15.6 | Treatment for Pediatric Hepatic Lipidosis

- Cool oxygenation: place the bird in an oxygen chamber at 21° C
- Non-lactated fluids when stable
- Antibiotics that do not require hepatic transformation or elimination if bacterial infection warrants
- Hand-feed small amounts with increased frequency
- Metabolic aids: lactulose[a], milk thistle[b], policosanol[c]

Table 15.7 | Treatment of Adult Hepatic Lipidosis

- Fluid therapy guideline: 60-90 ml/kg of non-lactated fluids every 24 hours
- Nutritional support
- Metabolic aids: lactulose[a], milk thistle[b], policosanol[c], psyllium[g], Ayurvedic herbs,[h] SAMe
- Treatment of secondary infections

may be present. Complete blood count and serum biochemistries help confirm the diagnosis and direct treatment. The serum is often very lipemic in spite of fasting. An elevated WBC is often observed. Serum chemistries may be normal or may demonstrate an increase in levels of bile acids, AST, LDH, cholesterol and triglycerides.

Sick birds' caloric needs are 2 to 3 times normal. Diets too low in fat cannot provide sufficient calories. Start the patient on a 100% carbohydrate diet[d], add metabolic detoxifier[e] then slowly convert to a sick bird support formula or a hand-feeding mixture[f] with adequate caloric content. Further supportive care includes fluid therapy, metabolic aids (lactulose[a], milk thistle[b], psyllium[g], policosanol[c]) and antibiotics if needed (Table 15.7). Improvement of nutritional status is critical for complete recovery and prevention of recurrence (see Chapter 7, Emergency and Critical Care).

AMYLOIDOSIS

Amyloidosis is a term used for various diseases that lead to the deposition of proteins in internal organs. The proteins are composed of beta-pleated sheets of non-branching fibrils. The physical properties of amyloid are responsible for its birefringence and apple green appearance in Congo red-stained sections viewed under polarized light. In primary amyloidosis, amyloid light chain protein is derived from immunoglobulin light chains synthesized by plasma cells in affected tissues. Primary amyloidosis may be localized or systemic.[18] In secondary amyloidosis, the most common type seen in veterinary medicine, the liver synthesizes serum amyloid-associated protein, which is the precursor to amyloid-associated fibrils. Secondary amyloidosis occurs as a consequence of prolonged inflammation resulting from chronic infection, tissue destruction, stress or chronic antigenic stimulation.[18] The

15_Liver.qxd 8/23/2005 5:16 PM Page 446

third type, inherited or familial amyloidosis, has been described only in some mammalian species.

Regardless of the cause, amyloid accumulates in the intercellular spaces and impairs the normal access of plasma to hepatocytes. Amyloid deposits can produce varying degrees of hepatomegaly, and extensive accumulations cause the liver to appear pale. In severe cases, affected birds may have clinical signs of either hepatic dysfunction or failure. While hepatic amyloidosis is usually fatal, a case was described in a falcon with hepatomegaly, ascites, leucocytosis, elevated AST, bile acids and iron levels. Abdominocentesis was performed and a milk thistle derivative was administered for a month. The bird survived for over 3 years.[18] Resolution of the primary cause is the goal in preventing the progression of this condition.

IRON STORAGE DISEASE

Pathological storage of iron in the liver (iron storage disease) has to be differentiated from hemosiderosis. Hemosiderosis is defined as the excessive accumulation of iron in hepatocytes without the alteration of normal tissue morphology.[13] High-risk species for iron storage disease include ramphastids (toucans), mynah birds, starlings and birds of paradise. High amounts of dietary iron seem to be the main cause, although complete pathogenesis is unknown. Most of the susceptible species live in a naturally iron-poor nutritional environment.[4,21] The mynah was found to have high intestinal absorption and transfer capacity of iron leading to high retention levels[4] (see Chapter 4, Nutritional Considerations). Dyspnea, hepatomegaly, ascites and sudden death are the most common clinical presentations.

Symptomatic therapy to stabilize the bird's respiratory problems should be followed by long-term therapy based on weekly phlebotomy of 1 to 2% of body weight. Dietary sources of iron such as grapes and raisins should be eliminated, and birds should be maintained on a low-iron (20-50 ppm) formulated diet.[k] Therapy with a chelating agent[i], has been described as useful (Table 15.8).[2] Items that enhance iron uptake, such as vitamin C, should be avoided. Iron uptake interference by dietary chemicals such as tannin may provide natural protection from excessive iron absorption.[25b]

TOXINS

The liver is the most common site for toxic injuries. The liver receives the major amount of its blood supply from the portal vein, which drains blood from the gastrointestinal tract. Therefore, ingested toxic substances including plants, aflatoxins and bacterial products, as well as metals, minerals, pharmaceuticals and other chemicals absorbed into the portal blood are transported to the liver. The liver possesses enzymes capable of metabolizing a variety of endogenous and exogenous substances for elimination from the body. This metabolic process may alter some substances such that they become more toxic. Toxin production may result in necrosis of hepatocytes, which may be replaced by fibrotic cells or infiltrated with lipids. This process can be self-perpetuating, even if the inciting agent is no longer present.[9]

Table 15.8 | Treatment for Iron Storage Disease

Initial	Ongoing
Oxygenation	Phlebotomy
Diuretics	Low-iron diet[k]
	Defroxamine[2,j]

Table 15.9 | Infectious Diseases of the Liver

Type	Disease
Bacterial	*E. coli*, *Salmonella* spp., *Klebsiella* spp., *Chlamydophila* spp., *Mycobacterium* spp., *Mycoplasma* spp.
Viral	Herpesvirus, polyomavirus, adenovirus, reovirus
Fungal	*Aspergillus* spp., *Candida* spp.
Protozoan	*Atoxoplasma* spp., *Histomonas* spp., *Trichomonas* spp., *Leucocytozoan* spp.
Nematodes	
Trematodes	

Infectious Diseases of the Liver

The pathogenesis, diagnosis and treatment of the individual infectious diseases are beyond the scope of this chapter. See Chapter 28, Implications of Mycobacteria in Clinical Disorders; Chapter 29, Implications of Mycoses in Clinical Disorders; and Chapter 32, Implications of Viruses in Clinical Disorders. The reader is referred to other references for more details on infectious diseases that affect the liver (Table 15.9).

Therapy for Liver Disease

This section reviews anecdotal and research-based therapy for hepatobiliary disease in birds; the mechanism of action, potential indications, efficacy as reported in humans and side effects. The liver can regenerate lost hepatic mass rapidly and efficiently. The therapy for hepatic dysfunction is directed at regeneration. Potentially hepatotoxic drugs should be avoided. High-quality nutrients are the best source of support for the regeneration of liver cells. The specific etiologic agent of liver disease is often undetermined. Clinical objectives for treatment of liver disease can be divided into supportive care and pharmacologic therapy.

SUPPORTIVE CARE

Supportive care for birds with hepatic damage includes reducing stress, fluid therapy, nutritional support and treatment of encephalopathy, ascites, coagulopathies and gastrointestinal ulceration. Proper caging options range from a cool, oxygenated incubator in a low-stress area to the bird's normal cage in its household environment. Lowering perches and padding can protect birds with coagulopathies. If infectious disease is suspected, then quarantine would be warranted.

Fluid therapy will flush toxins and toxic by-products through the metabolic pathways and from cells, organs and blood (see Chapter 7, Emergency and Critical Care). The selection of fluids for replacement therapy may be based on serum biochemistry results and physical exam findings and should be devoid of lactate. The volume to be administered will vary, depending on existing fluid deficits and continuing loss. Administration of colloids[j] may be effective in severe non-responsive hypoalbuminemia, however, clotting times should be monitored. Conspecific plasma or whole blood may be a better option.

Nutritional support with gavage-feeding should be provided three to four times daily for the anorectic patient. Calculate the metabolized energy requirements to determine the volume of food to use (see Chapter 4, Nutritional Considerations). A high-quality balanced formula with appropriate levels of vitamins and minerals and devoid of potential hepatotoxic agents, such as pesticides and preservatives, should be provided.

Hepatic Encephalopathy

The goal of therapy for hepatic encephalopathy is to restore normal neurologic function. In mammalian medicine, this is accomplished by dietary protein restriction, lactulose[a] therapy and antimicrobials. Lactulose[a] is a synthetic non-absorbable disaccharide commonly used in mammalian medicine for the treatment of elevated blood ammonia levels seen in hepatic encephalopathy. It is fermented in the gut by bacteria into acetic acid and lactic acid, which reduces the pH. The acidification causes ammonia (NH_3) to migrate from the blood into the colon where it is trapped as an ammonium ion (NH_4^+) and expelled with the feces. These acids also increase osmotic pressure drawing water into the bowel, causing a laxative effect. Lactulose[a] has minimal side effects and is therefore considered safe to administer to all birds during gavage-feeding.

Ascites

Removal of a large volume of coelomic fluid can cause severe protein loss. Removal is indicated if the ascites is associated with respiratory embarrassment or anorexia. Diuretics may be useful in controlling fluid retention. Dietary sodium restriction also is recommended, but the efficacy is unknown.

Coagulopathies

Cholestasis can cause impaired production of coagulation factors and antithrombin III as well as vitamin K malabsorption. Normal avian values are not available. Coagulopathies may be observed as petechia, hemorrhage or melena. Blood component therapy may be indicated. Vitamin K_1 can be administered for hemorrhage associated with vitamin K deficiency.

Gastrointestinal Ulceration

The empiric indications for the use of H2 blockers include nausea, inappetence and melena. Famotidine or ranitidine are recommended, since cimetidine and omeprazole are involved in cytochrome P450 inhibition and potential drug interactions in mammals.[23] Sucralfate may enhance healing of ulcers in patients with impaired mucosal blood flow, but this agent may bind and prevent absorption of other drugs.

PHARMACOLOGIC THERAPY

The proper choice of therapy depends on a definitive diagnosis. Technical or financial restrictions often preclude obtaining a definitive diagnosis in avian veterinary medicine. Therapeutic agents used are chosen for their anti-inflammatory, antifibrotic, hepatoprotectant, antimicrobial, diuretic, procoagulant, antacid or bivalent chelating actions. The pharmacologic therapies the authors recommend for hepatic failure in birds have not been validated by controlled studies. The personal experiences of the authors as well as anecdotal reports from colleagues support their use in therapy.

IMMUNOSUPPRESSANTS

The use of glucocorticoids in avian medicine is controversial. They cause immunosuppression by a variety of pathways. Glucocorticoids may exacerbate an underlying infection, increase hepatic lipid deposition, derange adrenal function and may be contraindicated unless a definitive diagnosis is determined (see Stress in Chapter 19, Endocrine Considerations). They are the treatment of choice in humans with autoimmune hepatitis.

Azathioprine is a thiopurine analogue metabolized in the liver to 6-mercaptopurine. Its metabolites inhibit the proliferation of rapidly dividing cells and modify T-lymphocyte function. Azathioprine is indicated when glucocorticoids are not controlling the immune response or are not tolerated by the patient. No data exist on the efficiency of conversion of azathioprine to its active metabolites in

15_Liver.qxd 8/23/2005 5:16 PM Page 448

hepatic insufficiency in birds. Serious side effects include dose-dependent bone marrow suppression, pancreatitis and idiosyncratic hepatotoxicity in mammals.

ANTIFIBROTICS

Colchicine is indicated when there is evidence of fibroplasia or bridging fibrosis. Colchicine helps prevent fibrosis by a variety of inhibitory actions. It also may protect the liver via stabilization of hepatocytes' plasma membranes. Most common side effects are associated with gastrointestinal upset, but in humans a peripheral neuropathy and bone marrow suppression have been reported. Formulations of colchicine containing probenecid can inhibit biliary and renal excretion of many drugs. Elemental zinc and glucocorticoids also have antifibrotic properties, but should be used with caution.

CHELATING AGENTS

Chelating agents are used to chelate bivalent metals such as zinc, lead, copper and sometimes iron. Copper induced liver disease is not a common problem in avian medicine.

HEPATOPROTECTANTS

Hepatoprotectants comprise a varied group of compounds that may protect hepatocytes from injury caused by free radicals, bile salts, drugs, environmental toxins and other insults.

Ursodiol, ursodeoxycholic acid, is a hydrophilic bile acid that competes with other bile acids for absorption in the ileum, and shifts the bile acid profile in favor of less toxic hydrophilic forms. It is suspected to reduce hepatocellular injury and fibrosis, modulate immune response and act indirectly as an antioxidant by preventing bile acid-induced peroxidation. Ursodiol is indicated in mammals for chronic active hepatitis, cholangiohepatitis, and disorders involving cholestasis or elevated bile acids. No efficacy studies have been performed with birds. GI upset has been reported rarely in humans. Ursodiol is contraindicated in bile duct obstruction because it is choleretic.

Vitamin E is a potent antioxidant. Studies indicate that it protects against bile salt-induced oxidant injury in-vitro.[29] It is indicated as an empirical therapy for inflammatory hepatopathies. The side effects are minimal unless there is a massive overdose or if it used with selenium (see Chapter 4, Nutritional Considerations).

S-adenosylmethionine (SAMe) is an indirect precursor of the antioxidant glutathione[20] (see Chapter 4, Nutritional Considerations: Section II, Nutritional Disorders). There are no side effects reported in veterinary medicine. Neurological side effects were reported in humans taking tricyclic antidepressants.[12] Early empirical results of SAMe use in birds with elevated cholesterol and fatty disorders are encouraging.

Milk thistle[b] (*Silybum marianum:* silymarin) is an extract from the seeds of milk thistle. It is thought to have antioxidant effects via scavenging of reactive oxygen radicals, and to have anti-inflammatory effects via inhibition of 5-lipoxygenase.[3] Side effects of GI upset and allergic rashes are rare in humans. It is a nutritional supplement, not a pharmaceutical. See Chapter 10, Integrative Therapies for more information.

Policosanol[c] is a blend of compounds isolated from natural plant waxes. It decreases blood triglyceride and reduces low-density lipoprotein cholesterol, inhibits abnormal platelet aggregation, protects against low-density lipoprotein oxidation, suppresses arterial inflammatory factors and increases beneficial high-density lipoprotein cholesterol.

Monitoring Parameters

The best test for monitoring the treatment of liver disease is the test(s) that was used to confirm the diagnosis in the first place. If hepatic lipidosis was diagnosed in an obese Amazon by history and clinical signs, hepatomegaly noted on radiographs and an elevated serum bile acid assay, then repeat radiographs and serum bile acid assay would be indicated. Repeat biopsies would give the most definitive answer, but are not always best for the individual patient.

Products Mentioned in the Text

a. Lactulose, Cephulac, Marion Merrell Doug, Kansas City, MO, USA
b. Milk Thistle, *Silybum marianum*, Nature's Answer, Hauppauge, NY, USA
c. Policosanol, Mountain States Health Products, Inc, Lyons Co, USA, 1-800-647-0074
d. Ultrafuel, Malt dextran and fructose, Twin Laboratories, Inc, Ronkonkoma, NY 11779, USA
e. Ultra Clear Plus, Ultra Balance Medical Foods, 5800 Soundview Dr, Gig Harbor, WA, 98335, USA, www.ultrabalance.com
f. Juvenile Hand-Feeding Formula, HBD International, Inc, 7108 Crossroads Blvd., Suite 325, Brentwood, TN 37127, USA, 1-800-346-0269, www.harrisonsbirdfoods.com
g. Metamucil, Proctor and Gamble Pharmaceuticals, Cincinnati, OH, USA
h. Ayurvedic herbs, Hepasan, Indian Herbs Research and Supply Co Ltd, Institut fur Veterinarpharanakologic und — toxikologie, Winterhurstrasse 260, 80547 Zurich, Schueiz, http:/www.vetpharm.unezh.ch
i. Desferal, Novartis Pharmaceuticals Corp, East Hanover, NJ, USA
j. Hetastarch, DuPont Pharmaceuticals, Wilmington, DE, USA
k. Harrison's Bird Foods, HBD International, Inc, 7108 Crossroads Blvd., Suite 325, Brentwood, TN 37127, USA, 1-800-346-0269 www.harrisons-birdfoods.com

References and Suggested Reading

1. Conn HO, Leevy CM: Comparison of lactulose and neomycin in the treatment of chronic portal-systemic encephalopathy. Gastroenterology 72:573-583, 1977.
2. Cornelissen H, Ducatella R, Roels R: Successful treatment of a channel-billed toucan with iron storage disease by chelation therapy: Sequential monitoring of the iron content of the liver during treatment period by quantitative chemical and image analysis. J Avian Med Surg 9:131-137, 1995.
3. Dehmlow C, Murawski N, de Groot H: Scavenging of reactive oxygen species and inhibition of arachidonic acid metabolism by silibinin in human cells. Life Sci 58:1591-1600, 1996.
4. Dorrenstein GM, et al: Hemochromatosis/iron storage: New developments. Proc Assoc Avian Vet, 2000, pp 233-237.
5. Flinchum GB: Potential use of policosanol in the treatment of hyperlipidemia in pet birds. Exotic DVM 5(3): 19-23.
6. Flora K, et al: Milk thistle (*Silybum marianum*) for the therapy of liver disease. Amer J Gastroenterology 93(2):139-142, 1998.
7. Fudge A: Avian liver and gastrointestinal testing. *In* Fudge A (ed): Laboratory Medicine: Avian and Exotic Pets. Philadelphia, WB Saunders Co, 2000, pp 47-55.
8. Garner MM, et al: Three psittacine fatalities due to an unidentified sporozoan parasite infection in a closed psittacine aviary. Proc Assoc Avian Vet, 2001, p 56.
9. Hardy RM: Diseases of the liver and their treatment. *In* Ettinger SJ (ed): Textbook of Veterinary Internal Medicine Vol. II, Philadelphia, WB Saunders Co, 1989, pp 1479-1527.
10. Hess L: Possible complications associated with topical corticosteroid use in birds. Proc Assoc Avian Vet, 2001, pp 29-32.
11. Hochleithner M: Biochemistries. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD International, Inc., 1999, pp 223-245.
12. Irulea LM, et al: Toxic interaction of S-adenosylmethionine and clomipramine. Amer J Psychiatr 150:522, 1993.
13. Jacobs A.: Iron metabolism, deficiency and overload. *In* Scott BR (ed): Price's Textbook of the Practice of Medicine. Oxford, Oxford University Press, 1978, pp 1118-1129.
14. Lin GL, Cornelius CE: Bilirubin and biliverdin excretion by the chicken. Amer J Phys 226:881-885, 1974.
15. Lothrop C, et al: Miscellaneous Diseases. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1986, p 526.
16. Lumeij S: Hepatology. *In* Ritchie BW, Harrison GJ, Harrison LR, (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD International, Inc., 1999, pp 522-536.
17. McGavin MD, Carton WW, Zachary JF (eds): Thomson's Special Veterinary Pathology 3rd ed. St. Louis, Mosby, 2001, pp 81-124.
18. McKinney PA: Amyloidosis in Falconiformes. Proc Assoc Avian Vet Australian Com, 2002, pp 257-264.
19. Mizobe M, et al: High performance liquid chromatographic analysis of bilirubin and biliverdin from jaundiced broilers. J Vet Med Sci, 59(8):677-680, 1997.
20. Osman E, Owen JS, Burroughs AK: Review article: S-Adenosyl-L-Methionine — A new therapeutic agent in liver disease? Aliment Pharmacol Ther 7:21-28, 1993.
21. Otten BA, et al: Mineral content of food items commonly ingested by keel-billed toucans (*Ramphastos sulfuratus*). J Avian Med Surg 15(3):194-196, 2001.
22. Pepping J: Milk thistle: *Silybum marianum*. Amer J Health Syst Pharm, 56:1195-97, 1999.
23. Reynolds JC: The clinical importance of drug interactions with antiulcer therapy. J Clin Gastroenterol Suppl 2:S54-S63, 1990.
24. Ritchie BW: Diagnosis and management of common problems in companion birds. Proc Assoc Avian Vet, 2001, pp 99-119.

25a. Ritchie BW, Harrison GJ: Formulary. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD International, Inc., 1999, p 465.

25b. Seibels B, et al: Effective use of tea to limit dietary iron available to starlings (*Sturnus vulgaris*). J Zoo Wildl Med 2003; 34:314-316.

26. Spalding MG, et al: Hepatic encephalopathy associated with hemochromatosis in a toco toucan. J Am Vet Assoc 189(9):1122-23, 1986.
27. Stanford M: Effect of dietary change on fecal Gram's stains in the African grey parrot. Exotic DVM, 4(6):12, 2003.
28. Stone EG, Redig PT: Preliminary evaluation of hetastarch for the management of hypoproteinemia and hypovolemia. Proc Assoc Avian Vet, 1994, pp 197-199.
29. Twedt D, et al: Vitamin E protects against oxidative damage of bile acids in isolated hepatocytes (abstract). Proc 16th ACVIM Forum, 1998, p 705.
30. Weber MA, et al: Benefits and complications of liver biopsy in birds. Proc Assoc Avian Vet, 2001, pp 211-213.

15_Liver.qxd 8/23/2005 5:16 PM Page 450

Clinical Avian Medicine

VOLUME II

GREG J. HARRISON, DVM
Diplomate Emeritus American Board of Veterinary Practitioners (Avian)
Diplomate European College of Avian Medicine and Surgery (n.p.)
Palm Beach, Florida

TERESA L. LIGHTFOOT, BS, DVM
Diplomate American Board of Veterinary Practitioners (Avian)
Florida Veterinary Specialists
Tampa, Florida

Spix Publishing, Inc.,
Palm Beach, Florida

Library of Congress Control Number: 2005922214

Clinical Avian Medicine (2-volume set) ISBN: 00-9754994-0-8

Production: Zoological Education Network

Cover Design: Jean Coffinberry

Cover photos: Peter Coutteel (canaries), Jan Hooimeijer (racing pigeons), Lorenzo Crosta (little blue penguins), Gwen Flinchum (black swans), Jaime Samour (saker falcon), E. Albertini (great grey owl), Auckland Zoo (North Island brown kiwi), Greg Harrison (Lady Gouldian finches)

Proofreading: K. Stormy Hudelson, DVM, Dipl ABVP-Avian, Paul Hudelson

Indexing: Ann Truesdale, DVM

Administrative Assistants: Lesley Flahie, Patty Wolf

Technical Assistance: Chris Lane

Printing and Binding: Four Colour Imports

Advertising and Sales: HBD Intl Inc. www.harrisonsbirdfoods.com

©2006 Spix Publishing, Inc., Palm Beach, Florida
All rights reserved. No part of this book may be reproduced or used in any form or by any means, electronic or mechanical, including photocopying, or by any information storage or retrieval system, without permission in writing from the publisher. Address any inquiries to Spix Publishing Inc, 3610 South Ocean Blvd #601, Palm Beach, FL 33480-5877.

Last digit is the print number: 9 8 7 6 5 4 3 2 1

Clinical Avian Medicine

CONTENTS VOLUME II (Chapters 1-15 - Volume I)

Appendices

Index

Note to the Reader

The National Research Council (NRC) has not established standards for nutritional products fed to birds; therefore, claims of nutritional completeness can not legally or ethically be made. Human medical textbooks and those of domestic animals have established normal laboratory values, recommended treatment regimes and nutritional disease states — all based on individuals meeting minimum standards of nutrition. These are not available in avian medicine.

Disclaimer

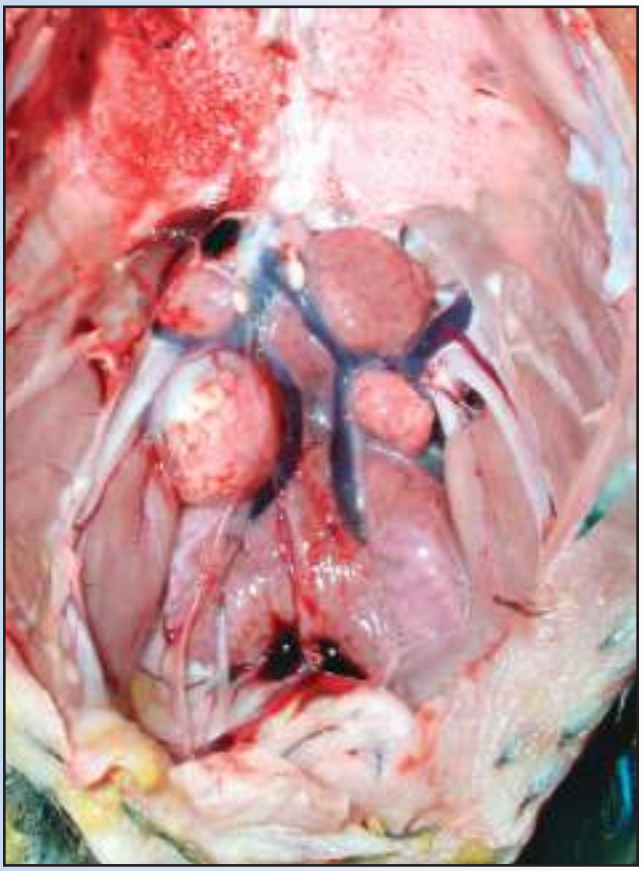
The publisher, editors, authors, reviewers and distributors involved assume no legal responsibility for use and make no claims or warranties regarding results that may be obtained from information in this text, nor necessarily endorse the procedures, medications, medication dosages or their uses as offered in this work. The above parties shall not be liable to any party for any damages, nor considered negligent for any misstatement or error obtained in this text.

CHAPTER

16

Evaluating and Treating the Kidneys

M. SCOTT ECHOLS, DVM, Dipl ABVP-Avian



Renal diseases and their various classifications are well documented in the avian literature. The precise pathogenesis of avian renal disease, however, is not nearly as well described as it is in mammals. Renal disease has been shown to be fairly common in avian species. In studied poultry, as much as 29.6% of all disease conditions had abnormal pathology associated with or attributable to renal disorders.[20,214,251] Amyloidosis, urate nephrosis and gout were the most common diseases associated with mortality in a 4-year retrospective study conducted at a waterfowl park in Ontario, Canada.[210] Thirty-seven percent of all avian cases presenting with renal tissue for histopathological examination, included over a 15-month period at the Schubot Exotic Bird Health Center, had one or more histologically identified kidney lesions.[187] Nine of 75 pheasants (*Phasianus colchicus*) died with nephritis, one or both ureters impacted, and visceral gout in another comprehensive study on avian mortality.[186] These case reports and retrospective studies support the conclusion that renal diseases are relatively common and are clinically significant in multiple avian species.

When compared to mammalian counterparts, the avian urogenital system has many structural and functional differences, which have been described previously.[77,90,118,181,187,227] Differences including gross anatomy, renal portal blood flow and protein waste elimination should be considered when reviewing this chapter, as findings obtained from mammalian studies may not necessarily be applicable to birds. By better understanding the pathophysiology of renal disease, practitioners will be able to better diagnose, clinically evaluate and treat kidney disorders in birds.

Part one of this two-part chapter will combine mammalian and avian literature to help describe the pathogenesis and progression of renal disease. Various forms of specific kidney disorders that have been reported in birds are described. Discussions of treatments will be deferred to the second half of this chapter.

Part two will focus on methods of diagnosis and management of specific avian renal diseases. Many of the diagnostics and treatments discussed are rationalized and based on avian renal anatomy, physiology and an understanding of the pathophysiology behind kidney disease, all of which are covered in the first half of the chapter.

PART 1: Pathophysiology, Pathogenesis and Classification of Avian Renal Disease

ANATOMY

Kidneys

The avian renal system is quite unique among vertebrate kidneys. In-depth discussions of gross and microscopic avian kidney anatomy have been covered elsewhere as referenced, but pertinent features will be discussed here.[27,38,39,90,113,141] In general, avian kidneys comprise 1 to 2.6% of body weight compared to an average of 0.5% of body weight in mammals.[77] Kidney mass also is relatively larger in those birds with active salt glands.[77,110] At least in Pekin ducks (*Anas platyrhynchos*), females have more and larger nephrons and bigger kidneys relative to body mass.[15] Finally, the left kidney in laying hens tends to be heavier and have a higher rate of renal portal blood flow than the right.[248]

Birds have paired kidneys located within a cavity formed by the ventral surface of the synsacrum. The kidneys extend from the caudal edge of the lungs to the caudal synsacrum.[36] An abdominal air sac diverticulum extends between the synsacrum and kidney. Normal bird kidneys are surrounded by air.[36] In most birds, the kidney is composed of three divisions: cranial, middle and caudal. Figs 16.1a,b demonstrate basic gross renal anatomy. The middle and caudal renal divisions of most passerines are fused, while the caudal renal divisions are connected across the midline in herons, puffins and penguins.[36] Additional variations in gross renal anatomy can be found in other avian species.

Within each division are numerous renal lobules, each containing a cortex and a cone-shaped medulla (medullary cone). Avian medullary cones have no inner and outer regions as described in most mammalian kidneys.[40]

One of the most unique features of avian kidneys is the presence of two types of nephrons, with and without a loop of Henle.[38] The loop of Henle allows for urine concentration and is the primary reason that birds and mammals are the only classes of vertebrates that can consistently produce hyperosmotic urine.[38,39] In birds, only about 10 to 30% of the nephrons are of the mammalian type.[40,77,141,248] Most avian nephrons are loopless ("reptilian" type) and stay within the cortex.[141] The looped nephrons ("mammalian" type) extend from the cortex into the discrete medullary areas known as medullary cones. Since birds have primarily "reptilian" type nephrons, which produce isoosmotic urine, urine concentration is limited.

Neurovascular System (Including the Renal Portal System)

The vascular system surrounding avian kidneys is quite complex and is one of the main reasons that renal surgery is difficult in birds. Another unique feature of avian kidneys is the presence of an arterial and venous, or dual, afferent blood supply.[141] (See Figs 16.1a-e for anatomy of the gross renal neurovascular system). The arterial afferent blood supply to the kidneys is as follows. The cranial renal division is supplied by the cranial renal arteries, which branch off the aorta. The glomeruli and postglomerular tubular network of the middle and caudal renal divisions are supplied by the middle and caudal renal arteries, which branch off the ischiadic or external iliac arteries.[77,141]

The renal portal system forms the second afferent blood supply, which is venous, to the kidneys.[141] The external iliac, ischiadic and caudal mesenteric veins supply blood to the renal portal system.[248] A ring is formed on the ventral side of the kidneys by the cranial and caudal portal veins, which branch off the external iliac and common iliac veins.[141]

The renal portal system works by either directing blood to or shunting it past the kidneys as directed by the renal portal valve. For example, venous blood from the limbs is shunted straight to the caudal vena cava when the renal portal valve, within the common iliac vein, is open.[141] The opposite is true when the renal portal valve is closed, as venous flow from the legs is directed to the afferent venous system of the kidneys.[129] This, of course, means that blood may pass through the kidneys prior to any other organ. Additional shunting either to the caudal mesenteric vein (caudally) or internal vertebral sinus

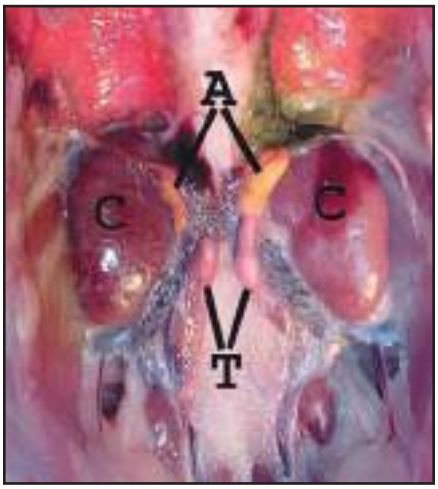


Fig 16.1a | Gross renal anatomy of a normal immature male red-tailed hawk (*Buteo jamaicensis*) that died from head trauma. Note the testes (T), adrenal glands (A) and cranial renal divisions (C). This hawk has a moderate amount of fat covering the middle and caudal renal divisions.

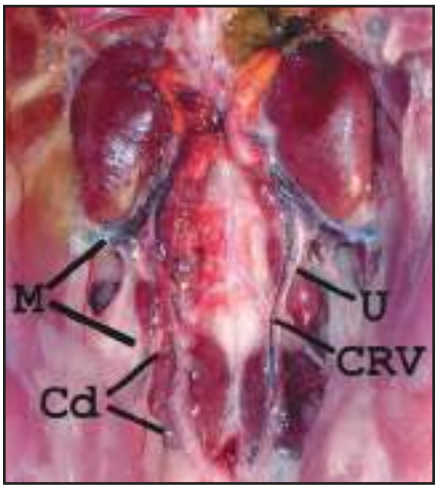


Fig 16.1b | The excess ventral perirenal fat has been removed. The middle (M) and caudal (Cd) renal divisions are now visible. The immature ductus deferens runs alongside the ureter (U) and caudal renal vein (CRV), but is not distinguishable in this young bird.

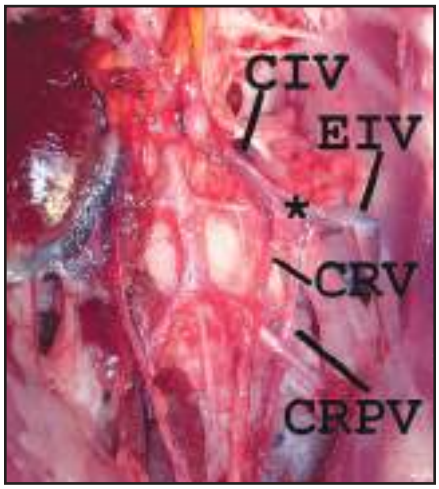


Fig 16.1c | The left kidney, ductus deferens and ureter have been removed, leaving the large vascular structures intact. The external iliac (EIV), caudal renal portal (CRPV), caudal renal (CRV) and common iliac (CIV) veins and the approximate location of the renal portal valve (*) are identified.

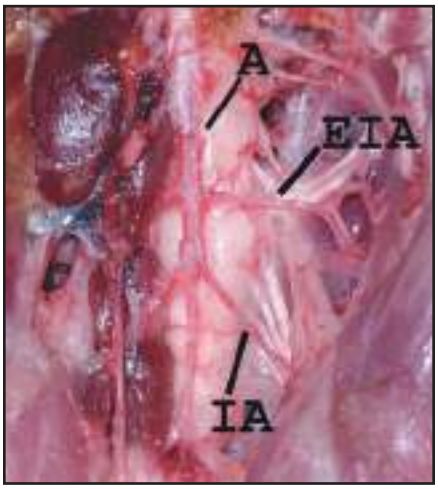


Fig 16.1d | The venous system has been removed to demonstrate the aorta (A), external iliac (EIA) and ischiadic (ischiatic) (IA) arteries.

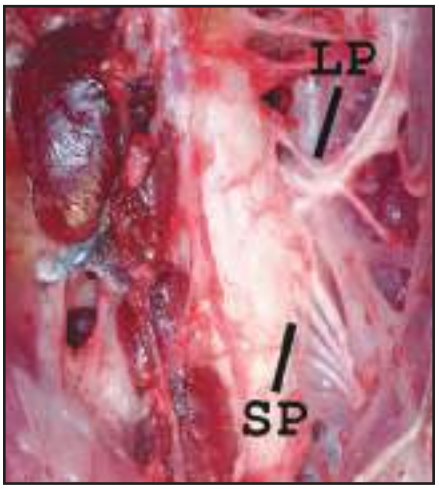


Fig 16.1e | The left renal, vascular, reproductive and endocrine systems have been removed, revealing the overlying renal fossa of the synsacrum and lumbar (LP) and sacral plexi (SP).

(cranially), again bypassing the kidneys, also may occur once blood has entered the renal portal ring.

By route of the afferent caudal and cranial renal portal vein branches, blood is delivered to the peritubular capillary network.[141] While virtually all renal arterioles terminate in the glomerular capillary beds, renal portal blood flow does not.[248] This system allows only arterial blood into the glomeruli and both postglomerular arterial and renal portal vein venous blood to the renal tubules. Blood is ultimately drained out of the kidneys via the centrolobular to the cranial and caudal to the renal and finally common iliac veins just proximal to the renal portal valve.[141]

This unique system accounts for some clinical concerns.[141] First is the fact that blood can be directed from the lower limbs straight into the renal parenchyma. This may increase the effect of nephrotoxic drugs and/or enhance elimination by taking the compound directly to the kidneys. Some drugs eliminated by tubular secretion and given into the leg may enter the renal portal system and be eliminated without ever entering systemic circulation.[223] As a general rule, parenteral drugs probably should be given in the cranial half of the body. Because some of the venous afferent blood comes from the caudal mesenteric vein (*v. coccygeomesentericae*), which drains the lower intestines, alimentary tract disease may ascend into and have an effect upon the kidneys. As

addressed under Part 1: General Mechanisms and Consequences of Renal Injury, Gastrointestinal Complications, the effect of lower intestinal disease upon the kidneys should be considered and treatment such as antibiotics for colitis instituted.

Hemodynamics

Studied birds have an impressive ability to maintain renal blood flow, even with severe hemodynamic alterations. Chickens seem to be able to "autoregulate" (keep constant) glomerular filtration rate when the arterial blood pressure range is within 60 to 110 mmHg.[73] Arterial pressures below this "autoregulatory" range result in decreased glomerular filtration rate until urine flow ceases at pressures below 50 mmHg.[73] Also of interest is that renal perfusion does not decrease in chickens (*Gallus domesticus*) until nearly 50% of the blood volume has been removed.[23] Despite severe hemorrhage, birds are able to maintain their blood pressure, suggesting other compensatory mechanisms such as extravascular fluid mobilization are utilized to ensure normal renal blood flow.[23]

Local Renal Neurologic System

The lumbar and sacral nerve plexi are closely associated with the kidneys. The lumbosacral plexus is formed by the ventral rami of about eight spinal nerves.[180] From these rami, the first three form the lumbar plexus, which produces the femoral and obturator nerves. In turn, these nerves provide innervation to the stifle extensors and leg adductors.[180] The lumbar nerve plexus forms dorsal to the cranial renal division and exits the pelvis cranial to the hip joint.[59]

The sacral plexus is formed by the caudal five to six spinal nerve ventral rami.[180] These nerves go on to supply innervation to the lower leg and some of the proximal leg muscles. The sacral plexus runs through the middle renal division parenchyma and exits the pelvis via the ischiadic foramen.[59,180]

Pressure on the nerve plexi can result in non-weight-bearing lameness.[180] This is the reason why some birds with renal diseases, especially those that cause renomegaly such as cancer, result in one-leg lameness in clinically affected birds. Other causes of one-leg lameness in birds include egg-laying disorders, bumblefoot, testicular cancer and trauma, and should be considered before making a diagnosis of renal disease.

Salt Glands

Salt glands are present in almost all birds, but have important functional significance in waterfowl, marine birds, and some raptors and desert avian species.[15,34,200,213] Birds have limited ability to produce hypertonic urine. As a compensatory mechanism, the extrarenal salt glands allow birds to adapt to brackish and saline environments and maintain normal electrolyte balance. There is likely an intimate association between renal function and extrarenal NaCl excretion.[110]

In chickens, the gland appears vestigial, but its anatomical features have been studied. The (supraorbital) salt gland is approximately 2 cm long and 2.5 mm in diameter. The caudal portion is located above the orbit, adjacent to the frontal bone, while the rostral extent is in the lateral wall of the nasal cavity, next to the dorsal and medial turbinates.[200] The salt gland's draining duct crosses under the nasal cavity and opens from the nasal septum, adjacent to the rostral part of the ventral turbinate, into the nasal cavity.[200] Fluid is then removed by shaking movements of the head or by passively dripping from the tip of the beak.[213] Similar features also have been noted in the turkey nasal salt gland.[200]

The salt glands function by providing an extrarenal pathway for the excretion of sodium chloride when the bird must consume salt quantities greater than its relative ability of renal clearance.[34,110] In some birds, the secreted sodium chloride can reach 10 times plasma concentrations.[213] The salt glands may remove more than 20% of the sodium chloride delivered by blood and have been considered one of the most efficient ion-transporting organs in the animal kingdom.[213] One reference notes that active salt glands can remove 60 to 88% of sodium and chloride eliminated by the bird's body.[77] Salt encrustation may be noted around the nares of dehydrated, heat-stressed birds and represents a gross manifestation of the gland's function.[141]

The gland size depends on the bird's salt consumption, and a hyperplastic response is considered normal in some species.[213] By adding high levels of sodium to the drinking water, salt gland hyperplasia can be induced in aquatic birds, but not in chickens.[200] In general, birds exposed to little salt have small salt glands. Once a bird is exposed to high salt loads, there is a rapid and profound hyperplasia and hypertrophy response that results in a greatly enhanced salt-secretory capacity within 1 to 7 days.[213]

Diseases of the salt glands are rarely described. This may imply that salt glands either are infrequently evaluated or are truly uncommonly affected by disease conditions. One study found that domestic ducks induced with plumbism had high concentrations of lead in the salt glands.[34] The authors hypothesized that in ducks, salt glands are involved in the elimination of lead. Also, lead toxicity results in obvious renal impairment and possibly damages the salt glands, making it difficult for wild waterfowl to adapt to different saline environments.[34] High cadmium intake significantly increased salt gland

mass in Pekin ducks (*Anas platyrhynchos*) and, combined with the toxic renal effects, was believed to adversely affect osmoregulation.[15] Salt gland enlargement from hyperplasia and inflammation are noted incidentally in range-reared tom turkeys.[200] Reported clinical signs are mild and consist of localized or unilateral swelling above the eye.[200]

PHYSIOLOGY

Roles of the Avian Kidney

Undoubtedly, the kidneys play numerous vital roles in birds. One primary role of the kidney is elimination of metabolic wastes. The kidneys also aid the liver in detoxification.[248] Because the kidneys are responsible for eliminating numerous metabolites, tissue concentrations of antibiotics (apramycin and ciprofloxacin) and toxins (lead and cadmium) are often highest in renal tissue.[3,7,10,178] As a result, various compounds are best identified and quantified in the kidney tissue.

Renal regulation of water via electrolyte (Na^+, K^+, Cl^-) balance is essential to maintaining intra- and extracellular fluid volumes and osmolalities.[248] By regulating fluid volume, the kidneys also regulate blood pressure. Arginine vasotocin is likely the primary mediator in response to dehydration, but norepinephrine, aldosterone, rennin, angiotensin II and prolactin also may each have an effect on avian kidneys and osmoregulation.[27,88,89,96,131,204,205]

The avian kidney has other endocrine functions and it is likely that future studies will elucidate more roles of this complex organ. One function of the kidney is the production of the active form of vitamin D (1,25- $[OH]_2D_3$) via the renal enzyme ($25[OH]D_3$)-1-hydroxylase.[66,244] Parathyroid hormone also has been shown to have a profound effect on renal excretion patterns of calcium and phosphate in birds.[61,70] As a result, the kidney is partly responsible for mineral metabolism.[248] The avian kidney also is the target organ for numerous growth factors, the functions of which are not yet known.[57] In addition to production in the liver, chick kidneys secrete apolipoproteins and are believed to contribute to the plasma lipoprotein pool.[232] This may be a functional response to the lipids coming from the terminal ileum, via the renal portal system, that contributes to production of lipoproteins.[232]

Fluid Regulation

Fluid regulation in birds is complex, as is true in many other animals. Birds have the ability to absorb and secrete various electrolytes and nutrients, which have some effect on fluid regulation. These osmoregulatory mechanisms are covered in depth in other references.[38,39,90,223,248] As mentioned previously, birds have the ability to produce concentrated urine, but because avian nephrons are primarily loopless, urine concentration within the kidney is limited. In birds, the process for concentrating urine is believed to be similar to that in mammals, but in avian species, sodium chloride acts as the major solute, not urea or potassium. The end effect is that sodium chloride does not have as much osmotic force as does urea, further limiting the concentration of avian urine.[223] Although birds inhabiting arid environments generally produce more concentrated urine than those from the tropics, many exceptions exist.[38,39] This leads to other water conservation methods that are variable between species.

In response to dehydration in birds, glomerular filtration and urine flow rate are consistently decreased while solute concentration increases. In studied birds, arginine vasotocin is the natural avian antidiuretic hormone and is believed to be the primary mediator in response to dehydration.[88,89,204] Increased plasma osmolarity is likely the major stimulus for release of arginine vasotocin.[131] Arginine vasotocin acts by controlling tubular water permeability, and thus the concentrating capacity of the avian kidney.[33]

As a component of water and possibly protein conservation, birds have the ability to absorb significant amounts of excreted (renal) water in the colon and ceca.[28] This system also allows some birds to regulate electrolyte loss through the urine.[248] The ureters empty into the urodeum where reverse peristaltic waves of the cloaca cause a reflux of urine into the cloaca and ceca, which are sites of water reabsorption.[28,220,236]

The amount of water reabsorbed is highly variable among different avian species. Rock ptarmigan (*Lagopus mutus*) ceca play a minimal role in digestion, but account for 98% of water absorbed in the hindgut.[252] In domestic turkeys (*Meleagris gallopavo*), 20 to 40% of the urine is refluxed into the ceca, from which 80% of all fluids that enter are absorbed. It has been shown that 77% of the water, 72% of sodium and 82% of potassium from ureteral urine in Gambel's quail (*Callipepla gambelii*) is subsequently reabsorbed in the ceca, lower intestine, cloaca and rectum.[28,252] In domestic fowl, it has been estimated that 13 to 28 ml/kg body weight per day of fluid is absorbed in the cloaca.[248]

Cecectomized birds often have changes in fluid regulation. Cecectomized Gambel's quail and great horned owls (*Bubo virginianus*) temporarily drank more water than controls. Water intake gradually returned to preoperative levels, suggesting a compensatory response either in the intestines or kidneys.[252] Cecectomized great horned owls and chickens also have shown a transient increase in water excretion (in the droppings).[220,252]

While the lower intestines appear to play a significant role in water and possibly electrolyte reabsorption, the ceca apparently do not have an obligatory role in osmoregulation in some species.[252] Additionally, many birds have no functional or anatomic ceca.

No studies were found that demonstrate the effect of lower intestinal disease on osmoregulation. Regardless, diseases such as typhlitis and colitis may adversely affect water and electrolyte balance beyond simple intestinal fluid loss, and should be a consideration when treating affected birds. Aside from nephrotoxic drugs such as aminoglycosides, no studies were found that show a clear correlation between antibiotic use for treatment of colitis/typhlitis and altered osmoregulation.

Uricotelism

Uricotelism is simply the excretion of uric acid as the end product of nitrogen metabolism. Birds lack carbamyl phosphate synthetase, an enzyme needed to synthesize urea from amino acid nitrogen.[99] While birds produce very little urea, the avian urea cycle is important, but is primarily related to renal detoxification processes and not nitrogenous waste excretion.[248] In birds, xanthine dehydrogenase is the terminal enzyme of purine metabolism and ultimately produces uric acid as the end product of nitrogen metabolism.[106,132] This is an adaptation that allows birds to minimize urinary water loss. Because uric acid is osmotically inactive, little water is required to excrete this nitrogenous waste.[248] The true advantage of water conservation in adult birds is debatable, though. The real advantage of uricotelism may simply be the storage of nitrogenous waste in eggs where a water-soluble product such as urea may prove toxic to the developing embryo.[248]

GENERAL MECHANISMS AND CONSEQUENCES OF RENAL INJURY

Initiation of Renal Disease

Proposed mechanisms of the process of initiation of renal injury and perpetuation of disease are complex, but have been described in mammals. These "mammalian" mechanisms may or may not apply directly to birds, but help form the basis on which some treatments are considered (see Part 2: Treatment, Nutritional Supplementation and Non-steroidal Anti-inflammatories). For this reason, some of the inflammatory cascade that occurs with renal disease is described.

The products resulting from the arachidonic acid cascade have effects throughout the body. For the purposes of this discussion, the cyclo-oxygenase pathway of the arachidonic acid cascade will be briefly covered.

In studied species, the renal medulla and papilla are a rich source of the group of enzymes collectively called prostaglandin synthetases.[62] The action of the prostaglandin synthetase cyclo-oxygenase upon arachidonic acid results in the formation of numerous prostaglandins (PE_2, PGF_{2a} and PGD_2) and thromboxanes (thromboxane A_2 [TXA] and thromboxane B_2), all of which have varying actions on cells. In response to renal ischemia and vasoconstriction, prostaglandin and thromboxane production is altered (primarily increased). These "alterations" subsequently result in varying effects on the body and kidney including changes in renal vascular resistance, blood flow, recruitment of inflammatory cells and other physiologic effects. Non-steroidal anti-inflammatories act to inhibit prostaglandin synthetase and represent another method by which to "alter" these arachidonic acid by-products and their subsequent actions.[62]

Specifically, TXA production, secondary to toxic or ischemic injury, is considered the main cause of renal vasoconstriction associated with acute renal failure and is believed to play a pathogenic role in many forms of kidney disease.[92,93,95] Thromboxane A_2, again an eicosanoid derived from the action of cyclooxygenase on arachidonic acid, is produced by many mammalian cells including glomerular epithelial and mesangial cells, renal medulla tubular cells and especially platelets.[91-95]

In mammals, TXA causes mesangial cell contraction and is a potent vasoconstrictor. Both of these actions can result in decreased glomerular filtration rate (GFR).[31,91,94,95] Renal vasoconstriction decreases GFR and delivery of oxygen and nutrients to tubular cells, resulting in renal damage.[95] Thromboxane A_2 also promotes platelet aggregation and may be partially responsible for hemostatic abnormalities noted with renal disease.[91,190] As histologic progression of renal disease continues when TXA is inhibited, it is possible that TXA only helps initiate kidney pathology.[93]

The above-described outcomes of increased TXA production serve only to show some of the possible negative effects of one by-product created as a result of renal injury. Management of these negative effects may be needed, especially when a clearly identified cause such as bacteria in the kidney parenchyma is not found. This then brings up the reasoning behind using products such as omega-3 fatty acids and low-dose NSAIDs (non-steroidal anti-inflammatory drugs) when managing some forms of renal disease.

Brief Review of Selected Potential Consequences of Renal Disease

Kidneys are dynamic organs and are directly or indirectly associated with multiple body systems. As a result,

renal disorders can lead to or be caused from multiple other disease processes. Some processes, such as hypertension, hypercoagulability and the nephrotic syndrome, are well described in mammalian renal disease, but are never or rarely discussed in the avian literature.

Hemostatic Abnormalities

Abnormalities of hemostasis are noted with some forms of renal disease and may lead to additional kidney or systemic disease. Platelet aggregation and activation occur secondary to complement activated antigen-antibody interactions and renal endothelial damage.[23,86,91,94] Activated platelets may then release vasoactive and inflammatory products (including TXA), growth stimulation factors and facilitate the coagulation cascade.[91,94] These reactions can result in glomerular damage via glomerular basement membrane thickening and, potentially, hyalinization and sclerosis.[94]

Fibrinous renal vessel thrombi have been noted in red-faced lovebirds (*Agapornis pullarius*) with membranous glomerulopathy and in chickens with *Erysipelothrix rhusiopathiae* sepsis. However, thrombus formation has been suggested to be rare in birds compared with mammals.[86,212] Using multiple staining methods, it could not be confirmed that fibrin-like thrombi noted histologically in various psittacine birds with polyomavirus-associated glomerulopathy were truly composed of fibrin.[189]

Gastrointestinal Complications

Gastrointestinal ulcerations are reported in some animals with uremia and advanced renal disease, but are rarely mentioned concurrently in clinical reports of birds with kidney disorders.[26] In chickens, gizzard erosions have been associated with naturally occurring urolithiasis.[148] Due to the overall lack of reports in the reviewed literature, it is unlikely that birds with renal disease develop gastrointestinal ulcers.

Intestinal inflammation may lead to renal disease. In humans, inflammatory bowel disease (IBD) can be related to renal disorders.[183] In humans, those with IBD have a 10 to 100 times greater risk of developing nephrolithiasis compared with other hospitalized patients.[183] Human IBD patients also may have an increased risk of glomerulonephritis and tubulointerstitial nephritis.[183] The avian coccygeomesenteric vein drains the mesentery of the hindgut into the hepatic portal and/or the renal portal vein.[226] Colitis may serve as a source of infectious agents, toxins and inflammatory products to the avian kidney if blood flow draining the colon is diverted into the renal vasculature. As a result, antibiotic therapy should be considered in all cases of colitis, especially when renal disease is suspected or confirmed.

Abnormal Lipid Metabolism

Aberrant lipid metabolism as evidenced by increased serum total cholesterol, low-density lipoproteins and triglycerides has been noted in humans, cats and dogs with renal disease.[31,179] In rats, lipid accumulation is known to stimulate glomerular mesangial cell and excess matrix production known as glomerulosclerosis.[31] Hyperlipidemia has been associated with glomerulosclerosis and/or loss of renal function in rats, guinea pigs, rabbits and dogs.[190] Glomerulosclerosis is histologically similar to atherosclerosis and may share a common pathogenesis.[190] Although scarcely noted in the avian literature, abnormal lipid intake, production and/or metabolism may be associated with renal disease in birds, as described below.

High-cholesterol diets actually may induce renal disease in birds. Pigeons supplemented with dietary cholesterol (0.2%, 0.4% and 0.5% of the diet) had a high incidence of end-stage renal disease, atherosclerosis and increased mortality rate compared with controls.[121] Although specific data was not presented, pigeon mortality was influenced largely by the degree and duration of hypercholesterolemia.[121] The implication herein is that diets high in cholesterol may lead to renal disease, at least in pigeons.

Gout

Renal disease may lead to numerous other conditions including gout, which can further damage the kidneys or additional body systems.[214] Gout reportedly may be caused by reduced excretion of urates or by increased dietary protein (although this has been disputed as discussed under Part 2: Treatment, Dietary Modification).[236] Dehydration and many forms of renal disease including obstructed ureters and general kidney damage can result in decreased uric acid elimination. As blood levels of uric acid rise and exceed the solubility of sodium urate in plasma (hyperuricemia), monosodium urate crystal precipitation is initiated.[236] It has been concluded that gout may not prove to be a nutritional disease in birds except under unusual circumstances such as deficiency of vitamin A.[207]

Visceral gout results secondarily from elevated plasma uric acid levels and its resultant deposition on visceral organs[9] **(Fig 16.2)**. During visceral gout, urate depositions are commonly found on the pericardium, liver and spleen.[134] Additionally, uric acid deposits are noted histologically within the lamina propria of the proventriculus, ventriculus and sometimes intestine and within the kidney, but can be found on or in any tissue. Visceral gout may appear as a white coating when on the capsular surface of affected tissue. Visceral gout has been associated with multiple forms of renal pathology.[9,214] Experimentally, visceral gout has been induced in chickens fed excessive

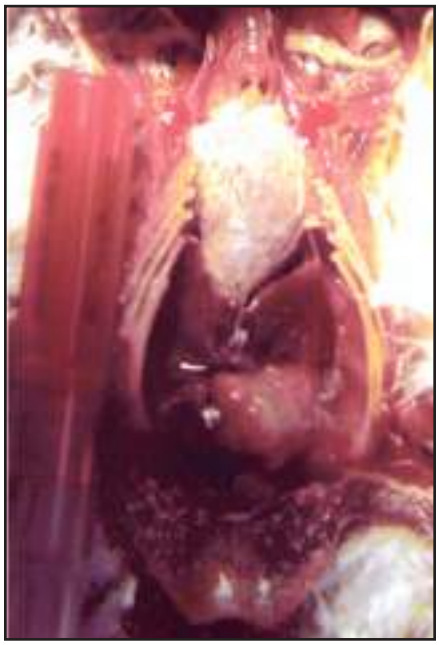
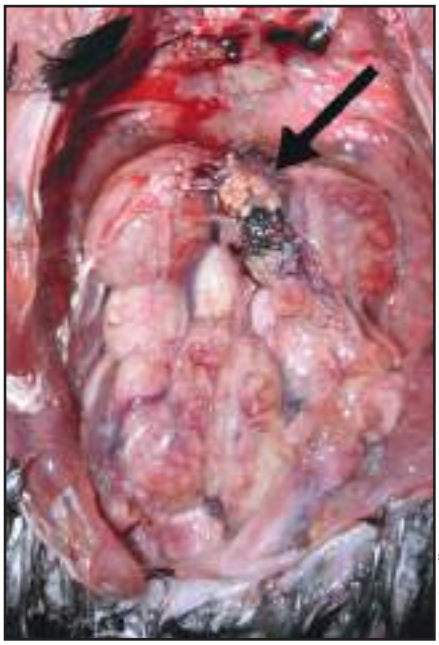


Fig 16.2 | An adult cockatiel (*Nymphicus hollandicus*) with visceral gout. Note the whitish deposits encasing the heart. The bird died with renal failure due to chronic renal fibrosis and interstitial nephritis.

Fig 16.3 | An adult budgerigar (*Melopsittacus undulatus*) with articular gout secondary to renal carcinoma. The postmortem picture shows subcutaneous "gouty" deposits over the dorsal tarsometatarsus and ventral phalanges. The skin has been teased open with a needle.



Drury Reavill

Fig 16.4 | An adult female black lory (*Chalcopsitta atra*) with severe renomegaly and visceral and articular gout. This female recently laid two eggs, but the ovary (arrow) was quiescent at the time of death. Histologically, renal tubular necrosis with urate stasis and multiple acute "gout tophi" were noted. The etiology was not defined.

dietary calcium and a diet deficient in vitamin A, administered various nephrotoxic agents, and following ureteral ligation and urolithiasis.[9,214]

Articular gout results from the accumulation of urates in the synovial capsules and tendon sheaths of the joints[9] (Fig 16.3). Diffuse urate deposits on visceral surfaces do not occur in articular gout.[214] However, visceral and articular gout can be present in the same bird (Fig 16.4). Gross lesions typically consist of soft swellings on the feet at the metatarsophalangeal and interphalangeal joints.[214] These swellings appear to be painful, as noted in clinical cases. Spontaneous articular gout in birds without underlying renal pathology is relatively uncommon and appears to have a hereditary basis, at least in chickens.[214]

Continuing Damage

Once renal damage occurs, persistent and progressive kidney damage is likely to occur, even if the initial insult is treated and "cured."[114] In humans, 50 to 60% of children with pyelonephritis develop irreversible lesions of the renal parenchyma.[13] Although no refereed literature describes the post-treatment progression of renal lesions in living avian patients, the author reported repeated kidney biopsies in numerous birds in an effort to help evaluate their clinical progression.[64] Repeat biopsies have shown that in birds with histologic confirmation of various kidney diseases, some mild renal lesions persist, even if the patient is clinically normal or improved.[64] When repeating kidney biopsies, the author has noticed no increase in scarring (gross or histologic lesions) or other abnormalities at the prior surgery sites, suggesting some treated birds have good regenerative and/or healing properties. Although these repeat biopsies are encouraging, the long-term health of these patients' kidneys is still unknown.

GENERAL RENAL DISEASE CATEGORIES

Nephritis

Nephritis is simply inflammation of the kidney and may involve the interstitium, tubules and/or the glomerulus (although "glomerulonephritis" is typically reserved for glomerular lesions). While "pyelonephritis" has been described in birds, this term is technically incorrect, as avian species lack a renal pelvis.[116,214] Nephritis is a nonspecific description, but some histological patterns and (especially) identification of infectious organisms help define the etiology.

Glomerulopathies

In the literature reviewed for this chapter, glomerular disease has been loosely termed "glomerulonephritis," but unless inflammation is specifically present, the term "glomerulopathy" would be more appropriate. Glomeru-

lonephritis describes inflammation of the glomerulus, usually considered mediated by the deposition of immune complexes or antiglomerular basement membrane antibodies.[22,94,239] A more accurate description of glomerular lesions, based on light and electron microscopy and immunohistochemistry, helps define the actual type of glomerulopathy present.

Glomerular disease is the most important cause of end-stage renal disease in humans worldwide and of chronic renal insufficiency/failure in dogs.[109,237,239] Proteinuria is the hallmark sign of glomerulonephritis in mammals prior to the onset of clinical renal insufficiency.[94] However, chicken leukocytes lack proteolytic enzymes that would potentially damage the glomerular basement membrane (and allow protein leakage) and birds may, in fact, not develop pathologic proteinuria with glomerulopathies.[22] In one study, no pathologic proteinuria was found in chickens with experimental autoimmune glomerulonephritis.[21] As noted below, glomerulopathies are well documented in avian species, but numerous differences exist when comparing this disease in birds and mammals.

The cause of glomerulopathies is generally assumed to be immune-mediated, but the inciting etiology is often unknown. Membranous nephropathy, the most common cause of nephrotic syndrome in humans, is usually idiopathic and specific etiologies are identified in only 20% of cases.[196] With few exceptions, the causes of glomerulopathies in birds are poorly studied. Polyomavirus infection is associated with membranous glomerulopathy in psittacines.[86,189] Glomerular pathology has been noted in chickens with various septic conditions and naturally occurring multicentric histiocytosis.[102,219] Glomerulopathies also can be induced experimentally in chickens by intravenous fungal injections, *Plasmodium gallinaceum* infections and by feeding aflatoxin.[158,214] Grossly normal 6- to 7-week-old broiler chickens at slaughter have been diagnosed with proliferative glomerulonephritis of unknown etiology.[193] Proliferative glomerulopathy can be induced in pigeons fed diets high in cholesterol.[121] It has been suggested that because of the extensive (dual) renal blood supply, severe chronic glomerulonephritis may persist without any clinical manifestation in birds.[214] It has been further suggested that avian glomerulonephritis may be present in far more birds than it is currently diagnosed.[214]

Although humorally mediated immunity is frequently discussed as the etiology of glomerulopathies, research has strongly suggested that cell-mediated immunity plays an important role in producing glomerular disease in chickens and other animals.[22,109,237] Under experimental conditions, cyclophosphamide bursectomized (humorally deficient) chickens develop glomerulonephritis. Although gross histologic lesions are similar, bursectomized chickens develop no IgG glomerular basement membrane deposits compared to controls when glomerulonephritis is induced in both groups.[22] These and other findings support the conclusion that cell-mediated immunity or some other non-humoral immune response is responsible for inducing glomerulonephritis in chickens.[22,237] Interestingly, in the above described study, even birds with massive mesangial enlargement maintained normal glomerular filtration.[22] Due to the small centrally oriented avian glomerular mesangium, the capillary loops were only slightly displaced to the periphery without compromising function.[22] Given our current knowledge regarding the differences between avian and mammalian species, renal biopsy is the best way to definitively diagnose glomerular (and other) kidney diseases in birds (see Part 2: Diagnostic Tests, Biopsy).

Infectious Diseases

Bacterial

Certain patterns may be expected with bacterial nephritis. Chickens experimentally infected with *E. coli* (*E. coli* $0_1K_{67}[B_{12}]$), *Staphylococcus aureus* and *Actinomyces pyogenes* developed a fairly consistent pattern and progression of renal disease.[219]

Birds inoculated subcutaneously developed more severe renal lesions and these lesions were noted earlier than those exposed to bacteria per os. Additionally, lesions were more severe in birds infected with *E. coli* and *S. aureus* compared to the slight reaction induced from *A. pyogenes*. Gross renal changes included congestion, enlargement and hemorrhagic foci. Although specific timelines were not given in regard to lesion development, bird kidneys were histologically examined at 4, 7, 10, 14 and 21 days postinoculation. The early-stage lesions consisted of acute interstitial nephritis (mainly lymphocytes, plasma cells and macrophages), prominent congestion and hemorrhage. The lesions progressed to nephrotoxic nephritis and included tubular epithelial cell degeneration and necrosis with the formation of hyaline casts and eosinophilic material. Later histology showed decreased congestion, persistence of mononuclear cells, introduction of connective tissue running around hyperplastic tubules and glomerular lesions.[219]

Certain renal histologic characteristics, with or without organisms present, may suggest an ascending or hematogenous bacterial infection in the avian kidney. The typical lesions suggestive of bacterial nephritis include tubular dilatation and impaction with inflammatory cells.[214] As nephritis becomes chronic, tubular necrosis, cyst formation, distortion and interstitial fibrosis with mononuclear cell infiltration become evident.[214]

Using sterile collection and culture methods, bacterial nephritis is definitively diagnosed by recovering bacterial organisms from affected kidneys. Light microscopic identification of bacteria within renal tissue may be difficult, as has been noted in dogs and swine with renal disease.[26,54] In a *Coturnix* quail processing plant outbreak, *Erysipelothrix rhusiopathiae* was cultured from multiple organs.[169] While the kidneys were swollen and congested, no organisms were specifically noted histologically, which emphasizes the importance of tissue culture.[169] Specifically, *Escherichia coli* has been identified in chickens as a cause of bacterial nephritis (pyelonephritis).[116] As a component of systemic paratyphus, *Salmonella typhimurium* var. *Copenhagen* was identified in kidney tissue and most frequently caused interstitial nephritis in a study of 78 experimentally infected pigeons.[87] The same organism also was recovered from kidney tissue, as a component of systemic salmonellosis, in pigeons from a large production colony.[121] As is likely true of most viral and fungal renal diseases, bacterial nephritis is often a component of systemic infection and multiple organs may be involved.[187] In summary, any septicemia can potentially result in kidney infection and inflammation (Fig 16.5).

Viral

Viruses perhaps have the most varied effect on avian kidneys. Numerous viruses may infect and affect avian kidneys (Table 16.1). Histologic patterns are highly variable, as some viruses, such as pheasant coronavirus-associated nephritis, directly affect the kidneys while others, like psittacine herpesvirus and polyomavirus, damage renal tissue as part of a more systemic process.[126,186,202]

Other viruses may cause minimal to no renal disease, but can be identified in the avian kidney because of viremia and/or viral replication and transmission through the urinary tract. For example, the reovirus that causes viral arthritis of chickens infects the kidneys within a few days of inoculation, but causes minimal, if any, renal lesions.[174] Some viral infections such as the West Nile virus are best identified in the kidney, and provide an additional reason to save extra renal tissue (frozen and/or formalinized) for later testing[124] (Fig 16.6).

Parasitic

Renal Coccidia

Primary and secondary renal parasites have been noted throughout the avian literature and some contribute to significant morbidity and mortality[18,19,43,69,76,82,83,133,160,175,177,194,217,218,236,253] (Table 16.2). Renal coccidiosis, found predominately in some waterfowl and marine species, is the most frequently reported avian renal parasite in those

Table 16.1 | Viruses Known to Infect or Affect Avian Kidneys

Virus	Common Name	Renal Lesions	Reference(s)
Adenovirus	New gosling virus enteritis virus	Renal hemorrhage	44
	Hydropericardium syndrome in broiler chickens	Renal hemorrhage, tubular nephrosis	2
Astrovirus	Duck astrovirus (aka duck hepatitis II)	Swollen congested kidneys	202
Coronavirus	Infectious bronchitis virus	Interstitial nephritis, urolithiasis, visceral gout and renomegaly	202, 231, 243
Enterovirus	Avian nephritis virus	Renal disease of young chickens and turkeys	202
Herpesvirus	Marek's disease	Renal lymphoma, renal masses	202
	Psittacine herpesvirus	Renomegaly	202
	Pigeon herpesvirus-1	Renal necrosis	202
Orthovirus	Influenza A of ratites	Renomegaly and green urate-filled ureters	202
Paramyxovirus	Pigeon paramyxovirus-1	Renomegaly, lymphoplasmacytic nephritis	202
Polyomavirus	Hemorrhagic nephritis enteritis of geese	Nephritis	101
	Avian polyomavirus	Basophilic and amphophilic mesangial cell intranuclear inclusion bodies, minimal lesions	126
	Psittacine polyomavirus	Membranous glomerulopathy	202
	Passeriforme polyomavirus	Renomegaly and perirenal hemorrhage	202
Reovirus	Viral arthritis or tenosynovitis of chickens	None to minimal inflammation	174
Retrovirus	Avian Leukosis/lymphoid leukosis	Cancer-nephroblastomas, renal lymphoma/adenoma/carcinoma, leukemia	202
	Reticuloendotheliosis virus	Renal tumors	202
Togavirus	West Nile virus	Neptritis (Fig 16.6)	124
	Avian viral serositis (EEE)	Pale kidneys	202
	Chukar alphavirus (EEE and WEE)	Urate-distended kidneys	202
	Turkey alphavirus (EEE)	Renal necrosis	202
	Guinea fowl alphavirus (EEE)	Renomegaly	202
	Crane alphavirus (EEE)	Necrotic nephritis and visceral gout	202
	Emu WEE	Renomegaly, necrotic nephritis	202

This table should serve only as an example of the large variety of viruses known to be associated with the avian kidney.
EEE = eastern equine encephalitis WEE = western equine encephalitis

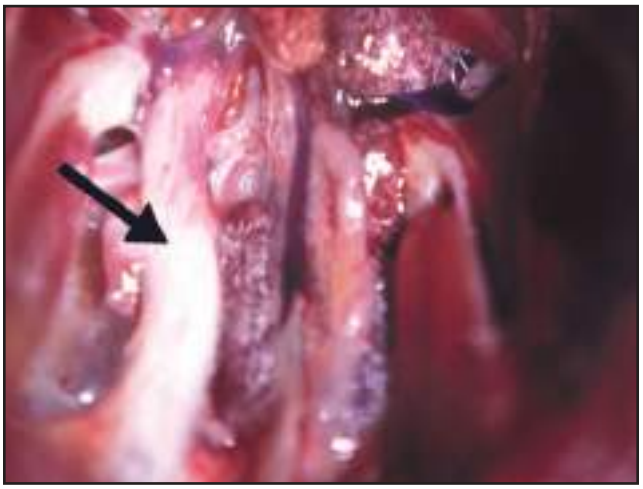


Fig 16.5 | A cloacal carcinoma, right ureteral obstruction (arrow) and *Streptococcus* sp. nephritis in an adult female *Amazona* sp. parrot. The *Streptococcus* sp. isolated from the kidney and heart blood was resistant to enrofloxacin, with which this bird was being treated chronically for cloacal straining.

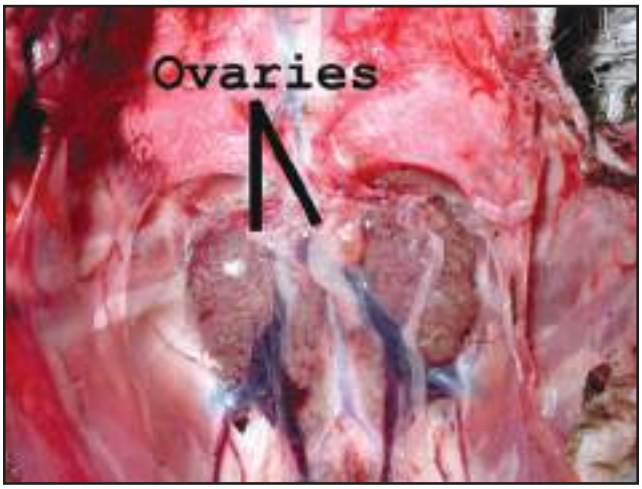


Fig 16.6 | West Nile virus-associated nephritis in an immature female Swainson's hawk (*Buteo swainsoni*). Note the moderate deposition of fat indicating the bird was in good overall body condition prior to acute death. The kidneys are pale. Two ovaries also are present as indicated by the lines.

Table 16.2 | Reported Incidence of Renal Coccidia in Various Avian Species

Affected Avian Species	*Eimeria* Species	Associated with Morbidity/Mortality	Reference(s)
Bufflehead (*Bucephala albeola*)	Unidentified	N/A	84
Canvasback (*Aythya valisineria*)	Unidentified	N/A	84
Duck, long-tailed (*Clangula hymenalis*)	*E. somateriae*	N/A	253
Duck, mallard (*Anas platyrhynchos*)	*E. boschadis*	N/A	253
Eider, common (*Somateria mollissima*)	*E. truncata*, *E. somateriae*	Mortality in ducklings (*E. somateriae*)	217, 253
Gadwall (*Anas strepera*)	Unidentified	N/A	84
Goldeneye, common (*Bucephala clangula*)	Unidentified	N/A	84
Goose, bar-headed (*Anser indicus*)	*E. truncata*	N/A	253
Goose, Canada (*Branta canadensis*)	*E. truncata*	N/A	253
Goose, domestic (*Anser domestica*)	*E. truncata*	Mortality in goslings	83, 84, 177
Goose, greater snow (*Chen caerulescens*)	*E. truncata*	N/A	253
Goose, graylag (*Anser anser anser*)	*E. truncata*	Mortality in goslings	177, 253
Goose, lesser snow (*Chen caerulescens caerulescens*)	Unidentified	Mild morbidity	83
Goose, Ross's (*Chen rossii*)	*E. truncata*	N/A	253
Gull, black-headed (*Larus ridibundus*)	*E. renicola*	N/A	82
Gull, herring (*Larus argentatus*)	*E. wobeseri, E. goelandi*	Incidental finding, nestlings	82
Loon, common (*Gavia immer*)	*E. gaviae*	Inconclusive	82, 160
Oldsquaw (*Clangula hyemalis*)	*E. somateriae*	Unlikely	76
Owl, great-horned (*Bubo virginianus*)	Unidentified	N/A	253
Penguin, little (*Eudyptula minor*)	Unidentified	Mortality	176
Pintail, northern (*Anas acuta*)	Unidentified	N/A	84
Puffin, Atlantic (*Fratercula arctica*)	*E. fraterculae*	Incidental findings, nestlings	133
Redhead (*Aythya americana*)	Unidentified	N/A	84
Scaup, lesser (*Aythya affinis*)	Unidentified	N/A	84
Shearwater, Cory's (*Calonectris diomedea*)	Unidentified	N/A	253
Shearwater, short-tailed (*Puffinus tenuirostris*)	Unidentified	N/A	253
Shoveler, northern (*Anas clypeata*)	Unidentified	N/A	84
Swan, mute (*Cygnus olor*)	*E. christianseni*	N/A	253
Swan, whistling (*Cygnus columbianus*)	Unidentified	N/A	84
Teal, blue-winged (*Anas discors*)	Unidentified	N/A	84
Teal, green-winged (*Anas crecca*)	Unidentified	N/A	84
Widgeon, American (*Anas americana*)	Unidentified	N/A	84
Woodcock (*Scolopax minor*)	Unidentified	N/A	253

N/A = not available

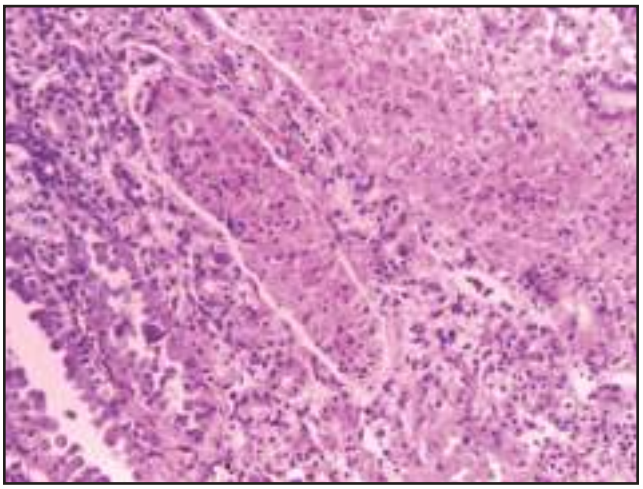

Robert E. Schmidt

Fig 16.7a | Renal coccidiosis in a screamer (*Chauna* sp.). Note the numerous oocysts and various developing stages of the parasite throughout the renal tubules.

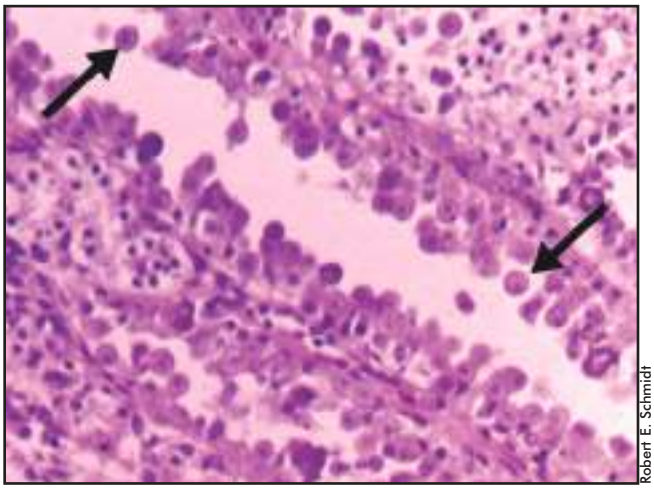

Robert E. Schmidt

Fig 16.7b | Close up of a renal tubule with numerous oocysts being released into the lumen (arrows).

species and has been clearly associated with disease in some cases.[76,82-84,133,160,176,177,217,253] Reports of various other parasitic diseases affecting the kidneys are noted, but their significance is not well established.

Several renal coccidia species have been identified and primarily include *Eimeria truncata*, *E. somateriae*, *E. christianseni*, *E. boschadis*, *E. gaviae*, *E. fraterculae*, *E. goelandi* and *E. wobeseri*.[82,83,133,160] Disease has ranged from mild histologic changes found incidentally (most species) to acute renal failure and death, such as in juvenile eiders (*Somateria mollissima*) and domestic geese (*Anser domestica*).[76,83,217] Flock mortality in domestic geese due to *E. truncata* has been reported to be as high as 87%.[76]

Renal *Eimeria* spp. oocysts are passed in feces via the ureter and sporulate rapidly in the environment.[217] Affected birds typically breed in large colonies or are otherwise under crowded conditions, which likely favors transmission of this parasite.[177,217] The prepatent period appears to range between species and has included 5 to 21 days.[83] Although transmission between different avian species is not clear, one study suggested that renal coccidia of geese do not infect ducks.[83]

The clinical gross and histologic abnormalities noted with renal coccidiosis seem to be fairly consistent across affected species. Most clinically affected species are young birds.[177,217] Clinically affected birds are typically emaciated and may have diarrhea with or without blood.[83,160,177,217] It should be kept in mind that many reported birds are wild and also have had intestinal parasites that may contribute to the described clinical signs. Grossly, the kidneys are often enlarged with white to yellowish nodules containing urates and/or oocysts.[76,83,160,217]

Cytologic smears of renal tissue and ureters often contain different endogenous stages of coccidian oocysts.[160,177,217] The renal tubules are parasitized and histologic lesions vary from mild dilatation to severe tubular destruction with associated degrees of inflammatory cell infiltrate (unusually mononuclear).[76,83,160,177,217] The tubules are often distended with endogenous developmental stages (micro- and macrogamonts, macrogametes) and maturing *Eimeria* spp. oocysts[217] (Figs 16.7a,b). In severe cases tubular nephrosis, necrosis and interstitial nephritis, potentially causing significant renal dysfunction, may be noted.[217]

Sarcocystis

Numerous other parasites have been noted in the kidneys of birds, but oftentimes association with disease is not clear. Canaries (*Serinus canaria*) experimentally infected with *Sarcocystis falcatula* developed mild multifocal interstitial renal infiltrates and glomerular hypertrophy with mesangial hyperplasia that modestly progressed with duration of infection.[218] While precystic merogony was primarily noted in the pulmonary tissue, infected canaries had low levels of merogony in the kidney and other tissues. Similarly infected pigeons developed no renal lesions.[218] Sarcocystis organisms also have been noted histologically in the renal parenchyma of cockatiels, but again the significance is unclear (T. Lightfoot, personal communication, 2003).

Microsporidia

Microsporidia (*Encephalitozoon* spp.) have been reported in numerous avian species with variable effects on the kidney. A psittacine beak and feather virus-positive eclectus parrot (*Eclectus roratus*) had heavily parasitized (*Encephalitozoon hellem*) kidney cells with associated renal tubular distension. As has been noted in other reported cases, renal cellular reaction was minimal

in the eclectus. Similar histological lesions and parasite morphology and locations (liver, kidney, intestines) also have been reported in three species of lovebirds, budgerigars (*Melopsittacus undulatus*) and a double-yellow headed Amazon parrot (*Amazona ochrocephala*).[175,192,194] The author also has seen renal microsporidiosis in a canary (*Serinus canaria*) that presented for acute illness and died shortly thereafter. Histology confirmed that numerous microsporidial organisms (not further defined) were present in the renal tubules and were associated with tubular necrosis. Other histologic lesions were minimal to mild, placing renal failure as the likely cause of death. Although it is not clear what role the kidney plays in disease, some believe that *E. bellem* is an avian and human pathogen, and may be primarily found in immunocompromised individuals.[194]

Cryptosporidia

Urinary tract cryptosporidiosis also has been noted in multiple bird species with varying associated disease. Although renal cryptosporidiosis is infrequently reported, it has been directly associated with kidney lesions in a 4-month-old black-throated finch (*Poephila cincta*), an 8-week-old Sonnerat's junglefowl (*Gallus sonneratii*), 4-month-old pullets and adult laying hens.[19,85,170,199,236] Four-day-old chickens co-infected with Marek's disease virus also have been studied.[1] Clinical signs ranged from acute death (finch and junglefowl) to thinning, depression, leg weakness and respiratory distress (4-month-old pullets and 4-day-old chicks) to slightly increased morbidity and mortality (adult chickens).[1,19,85,170,199,236] Pulmonary cryptosporidiosis also was a common feature of the pullets.[170]

Similarities were noted among gross and microscopic findings. The affected black-throated finch and Sonnerat's junglefowl had pale and swollen kidneys, and all birds had some degree of tubular epithelial tissue change with organism colonization.[1,19,85,170,199,236] The finch, adult layers, pullets and chicks also had interstitial nephritis, while the junglefowl had no inflammatory response.[1,19,85,170,199,236] Although no organisms were specifically found in the kidneys, a diamond firetail finch (*Stagnopleura bella*) with proventricular cryptosporidiosis also had similar tubular lesions in addition to multifocal amyloidosis (kidney included), severe chronic urate nephrosis, and protein and cellular tubular casts.[19]

Increased incidence of visceral gout, 1 to 2% higher than expected mortality, and numerous stages of *Cryptosporidium* sp. organisms within the epithelial cells lining the renal collecting tubules and ureters (of histologically evaluated kidneys) were found in egg-laying chickens from a production facility. Visceral gout was likely caused by the partial ureteral obstruction resulting from heavy diffuse lymphoplasmacytic infiltration in the wall of the ureter and (parasitized) epithelial wall hyperplasia.[236] Regarding the experimentally infected chicks, the authors concluded that *Cryptosporidium baileyi* can be highly pathogenic, and induce mortality and urinary tract infections in chickens infected with Marek's disease virus (an immunosuppressive effect).[1] Several authors have hypothesized that urinary tract *Cryptosporidium* infection originates in the cloaca and retrogrades into the kidneys via the ureters.[170,236] Although relatively uncommon, urinary tract cryptosporidiosis and associated disease seem to be primarily a concern in chickens, especially those with concurrent immunosuppressive illness.

Flukes

Scattered reports of renal flukes are noted in the literature. Spindle-shaped eggs, belonging to the blood fluke *Dendritobilharzia anatinarum*, were identified in kidney tissue pressed between glass slides in mallards (*Anas platyrhynchos*). The birds died from severe enteritis associated with blood fluke eggs, but no renal histology was described.[43] Eggs of other schistosomes may occasionally cause granulomatous ureteritis in waterfowl.[188] Parasites of the genus *Renicola* also may parasitize the renal tubules of several waterfowl species.[188,225] The renicolid flukes appear to have an indirect life cycle, and likely first infect mollusks and then mature in the renal tubules of susceptible species.[225] Eucotylid renal flukes may reside in the dilated ducts of the renal medulla of pigeon and passerine kidneys. They seldom cause problems and their eggs may be found in the feces and confused with other fluke eggs.[98] Clinical descriptions of affected animals are poorly described.

Miscellaneous Parasites

Other parasitic diseases also may be found incidentally in the kidneys of birds. Visceral larval migrans lesions consisting of a granulomatous reaction surrounding intact or degenerate *Baylisascaris procyonis* larvae in the renal (and other tissue) parenchyma of the house sparrow (*Passer domesticus*) were noted in one study. As most of the mixed species of birds had neural larval migrans only, the renal lesions seemed comparatively uncommon.[69] Chickens and pigeons have been experimentally infected with *Toxoplasma gondii* oocysts and evaluated for disease. While infected chickens developed no clinical signs and minimal evidence of infectivity, pigeons showed rapidly progressive disease (diarrhea, trembling, incoordination, death) and toxoplasma organisms in the kidney and other tissues. The authors stressed the importance of the pigeon crop in shedding the organisms with no emphasis on the kidneys.[18] It is probable that other parasites can affect the avian kidney and should be kept as an unlikely or rare differential diagnosis for renal disease.

Fungal

Fungal nephritis is uncommonly reported in birds. One chicken with renal and pulmonary cryptosporidiosis had *Aspergillus* sp. lesions in the lungs, air sacs, thoracic walls and kidneys.[170] In a separate study of 4-day-old chicks co-infected with *Cryptosporidium baileyi* and Marek's disease virus, one bird had necrotic renal aspergillosis.[1] Fungal nephritis, caused by *Aspergillus flavus-oryzae* group, was the only lesion seen in a moribund grey-headed albatross.[235] While focal coagulative necrosis, fibrous tissue and pronounced cellular reaction consisting of macrophages and multinucleated giant cells surrounding occasional fungal hyphae were noted, the lesions spared most of the renal tissue and did not account for the bird's poor condition.[235] Given the close association between the air sacs and kidneys, direct extension from the respiratory system (rather than primary renal invasion) is the likely cause of the necrotic fungal lesions in the kidneys.

Nephrosis

Nephrosis is a non-specific histopathologic change characterized as any degenerative, non-inflammatory lesion of the kidney, from cloudy swelling to necrosis, whatever the cause.[214] **(Figs 16.8, 16.9)** This is a microscopic diagnosis that cannot be made with gross observation. Due to its role in elimination, the avian kidney is vulnerable to the effects of many chemical toxins.[214] Inflammatory changes may develop, especially if the condition persists, and may confuse the diagnosis.[214] It was noted that tubular lesions may be reversible if the noxious substance is removed, provided the pathologic changes are not too advanced.[214] Causes of avian nephrosis have included avian malaria and hemoglobinuria, adenovirus infections, *Clostridium welchii* enterotoxemia, and lead, zinc, cadmium, calcium, aminoglycosides, phenoxyacid, sodium, ochratoxin A, ethylene glycol, 2,4-D, cadmium and 3-chloro-p-toluidine (avicide) toxicities.[2,14,15,16,30,55,72,125,154,161,178,214,224,242] This list is incomplete and serves only to emphasize the diversity of potential avian nephrosis-inducing agents. Although many toxins have been shown to induce nephrosis and other kidney diseases, renal lesions caused by specific toxicities are difficult to prove outside of a controlled study.

Hypertonic solutions also may cause a specific osmotic nephrosis in birds.[214] Hypertonic sucrose solutions (concentration not recorded) given intravenously have caused extensive vacuolation of the proximal convoluted tubules in birds.[214] Similar renal findings have been noted in other animals and man when injected with hypertonic sugar solutions and dextran intravenously.[214]

Selected Toxic and Nutritional Diseases

Also see Chapter 4, Nutritional Considerations: Sections I and II.

Vitamin D Intoxication

Vitamin D intoxication has been discussed in birds.[181,187] Vitamin D is converted in the liver to 25-hydroxycholecalciferol and then further hydroxylated to 1,25-dihydroxycholecalciferol in the kidney. Avian macrophages have the capacity to convert vitamin D to its active form 1,25-dihydroxycholecalciferol.[119] It is 1,25-dihydroxycholecalciferol that enhances the intestinal absorption of calcium and phosphate.[208,211]

As a result of excessive calcium uptake, visceral calcinosis, nephrocalcinosis, visceral gout and urate nephrosis are considered frequent complications of vitamin D intoxication in birds.[187] Symptoms of hypervitaminosis D include hypercalcemia, anorexia, nausea, polyuria, polydipsia, demineralization of bones, disorientation, painful joints and muscle weakness.[211] In normal animals experimentally subjected to hypervitaminosis D, 25-hydroxycholecalciferol, and not 1,25-dihydroxycholcalciferol, increase in the serum.[208] Chicks fed *Cestrum diurnum* leaves, which contain an analog of 1,25-dihydroxycholecalciferol, develop nephrocalcinosis and hypercalcemia, but the ultrastructural lesions are different than is noted with vitamin D toxicity.[208]

Hypervitaminosis D & A may occur when feeding developing birds vitamin D containing supplements **(Fig 16.10)**. A 3.5-month-old blue and gold macaw (*Ara ararauna*) and 5.5-month-old salmon-crested cockatoo (*Cacatua moluccensis*) from the same household developed polyuria, polydipsia and anorexia after being fed a diet (including supplements) with excessive vitamins A and D_3 and of calcium.[211] The cockatoo was hypercalcemic and had radiographic evidence of renomegaly. Hypercalcemia, hyperphosphatemia, hyperuricemia and elevated plasma creatine kinase were noted in the macaw. The calculated levels of vitamins A (119,000 IU/kg feed) and D_3 (26,790 IU/kg feed) were over 20 times the recommended levels (5000 IU/kg feed and 1000 IU/kg feed, respectively). Vitamin D_3 is considered toxic at 4 to 10 times the recommended amount. The cockatoo died 6 days after presentation and had chronic interstitial nephritis and calcifications in the kidney, proventriculus and lung. The macaw improved gradually and became disease free after discontinuing the supplemental vitamins and minerals. Hypercalcemia was attributed to oversupplementation with calcium and the vitamin mixture.

It has been suggested that African grey parrots (*Psittacus erithacus*) may be susceptible to hypervitaminosis D,[211] although no reviewed papers support this statement. Any bird species can potentially be susceptible to hypervitaminosis D.

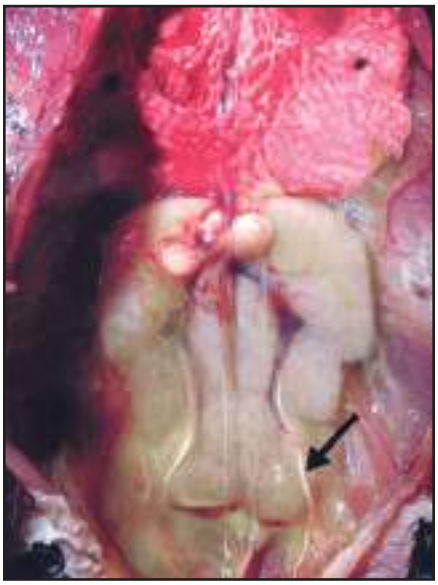
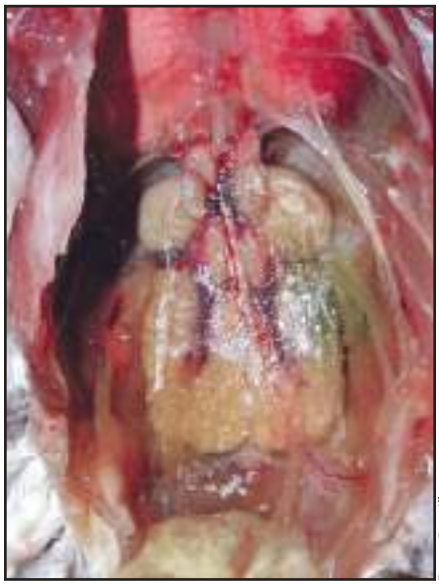


Fig 16.8 | A young adult male canary (*Serinus canaria*) with tubulonecrosis, mineralization and urate stasis of unknown etiology. Note the pale swollen kidneys with almost indistinct renal divisions. Urates are seen in both ureters (arrow points to left ureter).



Drury Reavill

Fig 16.9 | An adult male cockatiel (*Nymphicus hollandicus*) with severe renal tubular mineralization and necrosis. All renal divisions are pale. The etiology is undefined.

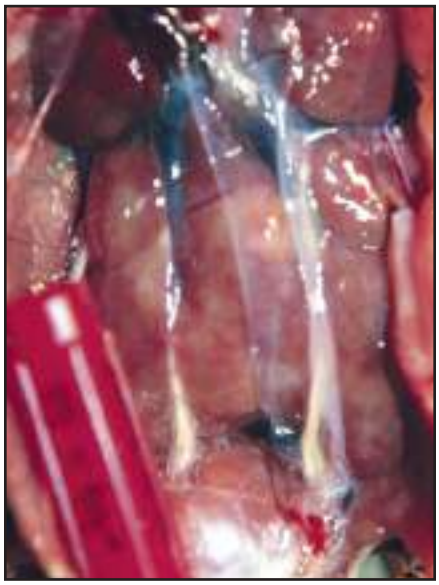


Fig 16.10 | A young blue and gold macaw (*Ara ararauna*) with suspected vitamin D_3 toxicity from excess supplementation in the diet. All renal divisions are severely swollen and indistinguishable from each other. The primary histologic lesion was nephrosis.

Hypercalcinosis

High calcium intake also has been directly correlated with renal disease in birds. Broiler chicks fed 3.27% calcium in the diet for 15 weeks, starting at 18 days old, developed numerous renal lesions throughout the study.[41] Nephrosis was noted by 7 weeks and progressed to nephritis (10 weeks), visceral gout (11 weeks) and replacement of the kidney parenchyma with urate granulomas (12 weeks).[41] In two separate studies, some growing chickens fed 3% calcium and 0.38% and 0.4% phosphorous, respectively, developed renal lesions such as nephritis, and ureteral and collecting duct occlusion due to probable calcium urate salts.[162] Limestone sand substrate (13.48% calcium and 0.02% phosphorous) was associated with rickets and nephrocalcinosis in young ostriches. Clinically affected birds returned to normal and no new cases developed once the substrate was changed to acid-washed sand (0.03% calcium and 0.02% phosphorous).[162]

In a study involving young and adult budgerigars (*Melopsittacus undulatus*), increasing dietary calcium levels were shown to be more renal toxic than was excess vitamin D_3 (D. Phalen, personal communication, 2003). Parent birds were fed diets containing 0.3%, 0.7% and 1.5% dietary calcium with a range of 500, 1000, 1500 and 3000 IU of vitamin D_3 per kg/feed. The adults subsequently fed the young the same diet. When fed a diet containing 3000 IU of vitamin D_3 per kg/feed, there was a questionably increased mortality rate only in the birds receiving 1.5% dietary calcium. However, there was a clear correlation with mild and severe metastatic (renal) mineralization in birds fed 0.7% and 1.5% calcium, respectively. The young birds fed 0.7% and 1.5% calcium died by 24 to 32 days old and never fledged (32 to 35 days). Growth rate and hatchability were poor only in the groups fed 1.5% calcium. While only a few adults died by 5 months on diets containing 1.5% calcium, most had metastatic renal mineralization when fed 0.7% calcium. Birds fed 0.3% calcium had no evidence of metastatic mineralization, and had good hatchability and growth rates (D. Phalen, personal communication, 2003). This study suggests that some species, such as budgerigars, may be very sensitive to dietary calcium levels and that supplementation should be used cautiously.

Hypovitaminosis A

Hypovitaminosis A also may lead to renal disease in avian patients. In birds with hypovitaminosis A, the ureters and renal collecting ducts may undergo metaplasia, changing the normal double-layered epithelium to keratinized stratified squamous tissue.[214] These epithelial changes can result in decreased mucin production and excessive keratin leading to plug formation and ureteral obstruction.[214] The consequential (secondary) lesions include renal tubular dilatation and necrosis, tophus formation and interstitial fibrosis.[214] Nephrosis, nephritis, visceral gout and severe replacement of the kidney parenchyma by urate granulomas were noted in broiler chicks fed vitamin A-deficient diets for 15 weeks starting at 18 days old.[41] See Chapter 4, Nutritional

Considerations: Section II, Nutritional Disorders, for Hypervitaminosis A.

High-Cholesterol Diets

Cholesterol supplemented in the feed can induce significant renal disease in pigeons.[121] Crystalline cholesterol and 10% lard were added to the diets of these pigeons under experimental conditions. The kidneys of some affected birds are firm, diffusely off-white, have an irregular capsular surface and may be enlarged up to 3 times their normal size. All renal components are susceptible and lesions may include tubular degeneration and dilatation, glomerular hypercellularity and hypertrophy (proliferative glomerulopathy), periglomerular fibrosis, lipid-laden cells within the glomeruli and multifocal, acute intersitial nephritis.[121] Since only mortality and necropsy results were reported, clinical information such as diagnosis and management/treatment were not provided. However, this does bring up the potential complication of feeding some birds high-cholesterol foods.

High-Protein Diets

High-protein diets have been associated with renal disease in birds, but only under specific conditions. Compared to a low-protein diet group, pigeons fed a high-protein diet had an observed increase in drinking rates and urine production.[153] Unfortunately, too little information was present to draw any conclusions relating dietary protein to renal disease. It has been shown that feeding 18-day-old broiler chicks a 42.28% protein diet for 15 weeks did induce multiple renal abnormalities (primarily nephrosis and visceral gout).[41] Extraordinarily high protein levels in the diet of genetically predisposed chickens have been shown to cause gout, but a direct relationship with renal disease has not been established. A more detailed discussion of the effects of dietary protein and hyperuricemia are discussed under Part 2: Serum or Plasma-based Biochemistries, Uric Acid, and Part 2: Dietary Modification, Protein.

Diets high in urea also have been linked to nephritis outbreaks in poultry.[42] Fish meal adulterated with urea was linked to high (6-8%) mortality in two separate farms. Clinically affected birds had gross lesions that ranged from pale nephromegaly and hepatosplenomegaly to urolithiasis and visceral gout. Histologic lesions ranged from interstitial, perivascular and pericapsular nephritis to proliferative glomerulopathy, and severe tubular and glomerular atrophy and fibrosis in severe cases. The disease was termed "nephritis-nephrosis syndrome in poultry" and was eliminated when the urea-adulterated feed was replaced with a different balanced diet.[42] See Chapter 4, Nutritional Considerations for more on protein levels in birds.

"Diet-Induced Renal Disease of Color Variety Psittacine Birds"

Although not formally entered into the veterinary literature, there appears to be a form of renal disease induced by feeding predominately pelletized diets to various color variety psittacine birds (M.S. Echols, unpublished data). All affected birds observed by the author have been color variety cockatiels (*Nymphicus hollandicus*), lovebirds (*Agapornis* spp.), budgerigars and parrotlets (*Forpus* spp.) and have eaten a predominately commercial pelletized diet. As most of the major brands of commercial pelletized diets have been involved, there appears to be no predilection toward any one manufacturer's product. With the exception of a history of predominately commercial pelletized diet, affected birds do not display any characteristics pathognomonic for "diet-induced renal disease." Of the birds with suspected "diet-induced renal disease," in which the kidneys have been histopathologically examined (pre- and postmortem), lesions have been limited to non-specific tubular nephrosis and were reversible after feeding a non-pelletized diet for 1 to 3 months. The diet should be converted to one appropriate for the species being treated.

Mycotoxic Nephropathy

Mycotoxic nephropathy, due primarily to ochratoxin A, has been reported in chickens and ducks.[63,149,224] Ochratoxin A is produced by several species of *Aspergillus* and *Penicillium*.[149] Ochratoxicosis occurs primarily because of ochratoxin A buildup in chick feed stored under conditions of excessive moisture, and has been identified from moldy feed, rice, groundnuts and foods prepared from these materials.[149,157,224] Ochratoxicosis causes liver and kidney damage, and specifically induces degeneration and vacuolation of hepatic cells and distension, enlargement and hypertrophy of renal proximal convoluted tubules, respectively.[63] Because of the multiple potential sources of the toxin, it is reasonable to assume that multiple avian species, other than chickens and ducks, can be exposed to and damaged from ochratoxin.

Other mycotoxins also have been closely correlated with renal disease in birds. Oosporein, a toxic pigment produced by *Chaetomium trilaterale*, *C. aureum* and several other species of filamentous fungi, is considered to be primarily a renal toxin.[184,185] The importance of oosporein is that the toxic isolates have been found in various agricultural commodities such as animal feeds, cereal grains and food products. Moldy corn in particular, growing *C. trilaterale*, may yield high concentrations of oosporein toxin. In studied young broiler chickens and turkey poults, oosporein toxicosis is dose-dependent and can cause dehydration, stunted growth, pale nephromegaly and death, and appears to severely affect uric acid secretion leading to hyperuricemia and visceral

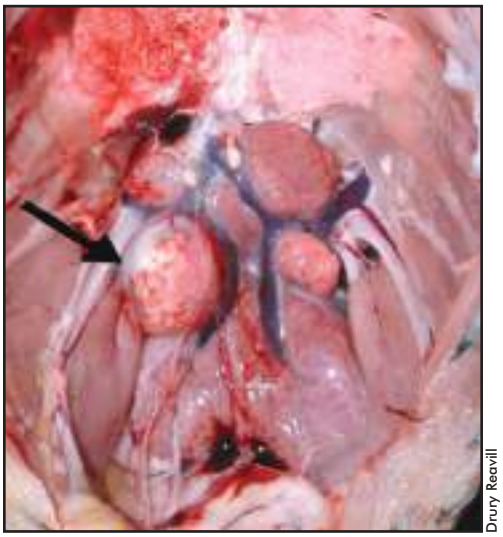

Drury Reavill

Fig 16.11a | A young male hyacinth macaw (*Anodorhynchus hyacinthinus*) has a large renal cyst (arrow) deforming the right middle renal division.

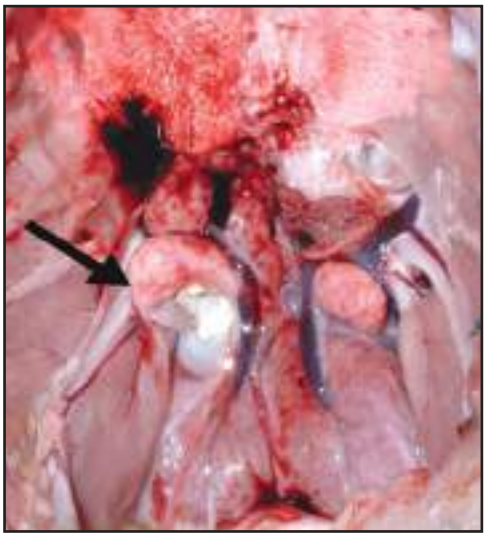

Drury Reavill

Fig 16.11b | The cyst (arrow) is opened, revealing a white, pasty interior.

and articular gout.[184] Although still severely affected, turkey poults seemed to tolerate higher doses of oosporein before toxicosis was apparent than did broilers, bringing up the issue of physiological differences between these two species.[185] Sterigmatocystin (STG) is produced by multiple fungal species and has caused acute liver and renal disease and death in 10- to 12-day-old leghorn chicks.[222] Chicks given intraperitoneal STG developed tubular nephrosis and hepatic necrosis and died within 21 hours of injection.[222]

Lead Nephropathy

Lead toxicity is the most common cause of metal poisoning in waterfowl and affects a wide variety of other bird species.[151] Although neurological and gastrointestinal clinical signs are usually seen, lead can have severe effects on avian kidneys. Renal lesions may include proximal tubular necrosis and degeneration (nephrosis), visceral gout and, in some birds, acid-fast intranuclear inclusion bodies.[55] Kidney, liver and brain tissue concentrations of 3 to 6 ppm wet weight are suggestive and greater than 6 ppm is diagnostic for lead poisoning.[125] Also see Chapter 31, Implications of Toxic Substances in Clinical Disorders and Chapter 17, Evaluating and Treating the Nervous System.

Congenital and Hereditary Defects

Multiple congenital renal defects are reported in birds.[214,238] Heritable renal diseases such as X-linked hereditary nephritis in Samoyed dogs and Alport's syndrome in humans are discussed in many mammals, but are poorly described in the current avian literature.[100] In some large poultry flocks, up to 20% of the necropsied birds have had evidence of "faulty kidneys" considered to be congenital in nature.[238] Reported renal abnormalities include complete or partial kidney agenesis, ureteral dilatation, structural glomerular changes and predilection toward hyperuricemia (due to presumed proximal tubule defects).[9,214,238] Renal cysts are occasionally seen and may be congenital or acquired[188] (Figs 16.11a,b). Polycystic renal disease has been noted in chickens, pigeons and a bald eagle (*Haliaeetus leucocephalus*).[228] Renal agenesis is the most frequently described inherited defect and has been attributed to a simple recessive gene with variable penetrance in brown leghorn chickens.[214] With partial renal agenesis, the cranial renal division is most likely affected.[214] Although birds usually die with neurological signs or massive interrenal hemorrhage, emus (*Dromiceius novaebollandiae*) with inherited neuronal storage disease (gangliosidosis) develop unusual large vacuoles in the renal tubular epithelial cells of the proximal convoluted tubules.[17] Congenital renal diseases have been reported in chickens, pigeons, quail, a canary and a mandarin duck but likely exist in numerous other species.[187,214,238]

Fatty Associated Diseases

Lipids are not histologically evident in normal avian renal tissue, but may be noted under certain pathologic circumstances.[214] Fasting (water and food) may result in reversible lipid deposition within the renal tubular epithelium.[214] Defects in lipid metabolism or storage also may account for renal tubule cell lipidosis.[187]

The now rare fatty liver and kidney syndrome of broiler flocks and turkeys (due to biotin deficiency) can cause heavy lipid accumulation within the proximal convoluted tubules.[214,247] At necropsy, the liver, kidneys and

sometimes other organs are often pale and swollen with deposition of sudanophilic lipid droplets.[247]

A fatty liver-kidney syndrome also has been reported in merlins (*Falco columbarius*).[48,72] Only captive birds have been affected.[48] Most affected merlins have been approximately 5% above normal body weight and fed a diet predominately of day-old chicks for several months prior to death. Most affected merlins died suddenly either while eating or with the keeper. A few became lethargic a few hours before death.[48] As is seen in broiler chicks, merlins with fatty liver-kidney syndrome develop excess fat in the liver, kidneys and spleen. One-day-old (feeder) chicks contain appreciable avidin, which may bind dietary biotin, in turn leading to (a theoretical) biotin deficiency. Biotin and other deficiencies, high-fat diet, hepatic anoxia and various toxic agents, have been proposed as causes of fatty liver-kidney syndrome of merlins, but a definitive etiology has not been confirmed.[72]

Neoplasia

The avian kidney, just as with other animal tissue, is susceptible to neoplastic conditions. Nephroblastomas are the most commonly reported avian renal tumor.[214] Nephroblastomas and renal adenocarcinomas comprise the majority of kidney tumors in budgerigars (*Melopsittacus undulatus*).[173,187] Renal carcinomas are the most frequently reported tumor of the urinary system in non-domestic free-ranging and captive birds.[130] Malignant renal tumors are more commonly seen in males than females and are more commonly observed in psittacine than passerine species.[79] In one study of 74 budgerigars suspected of having coelomic tumors, one-legged lameness and abdominal enlargement were the primary clinical signs. In the same study, 47 birds (63.5%) had renal tumors and were diagnosed most commonly within 5 years of age.[173]

Lymphoid, myeloid and erythroleukemias, lymphoma, ovarian, liver and oviductal adenocarcinomas, hemangioma, lipoma, histiocytic cell sarcoma, neurofibroma, granulosa cell tumor, cystadenoma with bone, squamous cell carcinoma, unclassified carcinoma and osteogenic sarcoma have all been reported either as primary or secondary renal neoplasms in birds.[121,193,214,241]

Like other cancers, there are likely many causes of renal tumors in birds, but there is little information regarding definitive etiologies. Avian leukosis virus (ALV) can induce renal tumors in chickens. While ALV has been found in budgerigars with renal tumors, a definitive association has not been made.[173]

A common presentation with renal cancer is unilateral to bilateral leg weakness or paralysis and slight ataxia.[241] Other clinical signs may vary, but often include diarrhea, dyspnea, abdominal distension and weight loss.[130,241]

The lumbar plexus lies dorsal to the cranial renal division, while the sacral plexus runs through the middle division parenchyma.[180] Because of this close association, any parenchymal inflammation or pressure on or from within the kidney can potentially result in nerve dysfunction and resultant lameness. Additional neoplastic extension to the overlying spinal column also may result in nerve dysfunction. Peripheral neural compression should result in peripheral neuropathy with eventual loss of the withdrawal reflex, not seen with most spinal cord lesions.[79] In addition to lameness and muscle atrophy, ipsilateral osteopenia was noted in a cockatiel (*Nymphicus hollandicus*) with a renal adenocarcinoma.[79]

Unfortunately, avian renal tumors carry a poor prognosis. In reported cases of renal cancer, most birds lived less than 3 months following diagnosis.[79] It has been stated in reference to budgerigar renal tumors that the course of the disease may take weeks to several months.[241]

Urolithiasis and Ureteral Obstructive Disease

In birds, urolithiasis refers to the formation of large urate "stones" in the ureters, is primarily seen in pullets and caged laying hens, and can result in increased mortality and decreased egg production.[50] Urolithiasis has been reported primarily in the poultry literature on numerous occasions, but is rarely described in other avian species.[20,42,214,251]

Common findings include atrophic ipsilateral renal tissue, a normal to hypertrophic (compensatory) contralateral kidney and a dilated ureter obstructed with one or more urate stones.[20,214,251] Histologic lesions noted with urolithiasis have included glomerular nephritis, tubular nephrosis, ureteritis and pyelonephritis with interstitial mononuclear infiltrates.[50] One study noted that virtually every cull hen or out-of-production hen examined at affected layer complexes (sites with high incidences of urolithiasis) had gross kidney lesions and kidney stones.[50] In birds, ureteral obstruction (as may occur with ureteroliths, cloacal masses, urodeal fold thickening, etc) may cause a postobstructive form of renal disease. Simple ligation of a bird's ureter results in ipsilateral renal atrophy and this result is similarly expected with urolithiasis.[214] Naturally occurring ureteroliths in chickens are known to contain uric acid, urates, calcium and ammonia.[148] These statements suggest that the kidney should be closely evaluated (eg, via biopsy) when urolithiasis is present.

The cause of urolithiasis in poultry flocks has not been definitely identified.[214] However, it is known that coronavirus-associated nephritis in pheasants can induce inter-

stitial nephritis, tubular dilatation, ureteral impaction and subsequent visceral gout.[186] In addition to infectious bronchitis virus infection (IBV — a coronavirus), other proposed causes of urolithiasis in poultry include water deprivation, excess dietary calcium and nutritional electrolyte imbalances.[50] One group reported that by changing the form of calcium from small particle size to flakes, adding additional phosphorous and by modifying the IBV vaccination protocol, the investigators were able to significantly reduce the incidence of urolithiasis in a previously affected layer flock. However, they could not determine which management change resulted in the beneficial effect.[50]

Urolithiasis in psittacine species is rare, but has been reported. A 21-year-old male double-yellow headed Amazon parrot (*Amazona ochrocephala*) with a lifelong history of straining to void and chronic intermittent vomiting for a "few years" was diagnosed with septic ureteral fluid and ureterolithiasis.[56] Dorsocaudal coelomic radiodense opacities were noted on screening radiographs, but the diagnosis was ultimately made via exploratory celiotomy. Multiple surgeries were required to remove the stones. A kidney biopsy was not collected and a relationship to renal disease could not be made. The ureteroliths were composed of monosodium uric acid crystals and proteinaceous material mixed randomly or forming irregular laminae. Although the bird had dry, flaky skin, a urate-pasted vent, dull feathers and heterophilic (28,840 cells/μl) leukocytosis (32,000 cells/μl), the authors concluded that the clinical signs associated with ureterolithiasis in this bird were non-specific and may result in delayed diagnosis.[56] The cause was not determined.

Amyloidosis

Amyloidosis is occasionally noted in association with avian renal disease. Amyloid deposits are often related to chronic inflammatory disease and usually found systemically, but can affect specific tissues.[29] Typically, amyloid presents histologically as amorphous, eosinophilic, homogenous material that stains red-orange with Congo red and bright green when examined under polarized light. Amyloidosis is most frequently noted in captive Anseriformes (geese, ducks, swans), Gruiformes (cranes) and Phoenicopteridae (flamingos), but also has been reported in numerous other species.[127,201] See Chapter 15, Evaluating and Treating the Liver for a discussion of amyloidosis.

There are a few reports of amyloidosis involving the kidneys of birds. Multifocal amyloidosis was noted in a diamond firetail finch (*Stagnopleura bella*) with proventricular cryptosporidiosis, and was found specifically in the glomeruli and interstitial tissue around the tubules.[19] Numerous laying Japanese quail with systemic amyloidosis had amyloid deposits in the renal tubules and no to minimal deposition in the glomeruli.[171] While some of the birds had concurrent inflammatory diseases such as egg yolk peritonitis, the etiology of the amyloidosis was not determined.[171] Four days after acute onset illness, a roseate flamingo (*Phoenicopterus ruber*) died with necrogranulomatous and septic air sacculitis, perihepatic serositis and hepatic capsulitis, hemosiderosis, atherosclerosis and systemic amyloidosis.[29] The renal amyloid involvement was severe, resulting in a marked glomerulopathy and was likely the cause of death.[29] Amyloid was found within the connective tissue of mycobacterial tubercles found on the kidney surface of a hooded merganser (*Lophodytes cucullatus*). No details were given regarding the premortem disposition of the bird.[209] The author has noted renal amyloidosis in pet geese. These birds presented in end-stage renal failure and necropsy showed severe renal amyloidosis. The underlying cause was never elucidated.

Renal Hemorrhage

Renal hemorrhage is sporadically reported in the literature and may exist predominantly as a secondary finding. Sudden death syndrome (SDS), also known as "perirenal hemorrhage syndrome," is the main cause of death in heavy turkey flocks from 8 to 14 weeks of age.[24] Primarily male turkeys in good body condition die acutely with SDS and typically have characteristic postmortem lesions including perirenal hemorrhage and organ congestion including the lungs, spleen and liver.[24,75,129] One group noted that most affected birds had hypertrophic cardiomyopathy and proposed that acute congestive heart failure was the cause of death and severe passive congestion accounted for the perirenal hemorrhage.[129] The cause is still unknown, but other theories include severe lactic acidosis and limited cardiac capacity, noted in predisposed turkeys, as contributing factors.[24]

An adenovirus, new gosling viral enteritis virus (NGVEV), has been shown to cause renal hemorrhage and hyperemia 4 days postinfection in newly hatched goslings.[44] Renal tubular and ureteral epithelial cell degeneration and intestinal glandular epithelial cell necrosis and sloughing also were consistently seen in the goslings infected with the rapidly progressive NGVEV.[44]

Hydropericardium syndrome of broiler chickens is a contagious disease caused by an adenovirus and can result in grossly swollen kidneys with extensive renal hemorrhage and hydropericardium.[2] Three- to six-week-old broilers are typically affected and mortality ranges from 10 to 60%. Renal tubular nephrosis and necrosis within the liver, spleen and bursa of Fabricius may be seen microscopically.[2]

Other causes of renal hemorrhage also may be seen. Simple trauma, such as from an animal bite or endoscopic biopsy, may result in renal hemorrhage. If the renal capsule is left intact, a subcapsular hematoma may form, increasing the renal size and possibly placing pressure on the neighboring nerve plexi.[134] Renal petechial hemorrhage resulting from *Clostridium perfringens* toxemia was reported in a rock partridge (*Alectoris graeca*).[134]

Metabolic Renal Disease

Metabolic renal disease includes dehydration, diabetes mellitus, amyloidosis, gout and lipidosis, the latter three of which have already been discussed. Diabetes mellitus has been noted in a variety of birds and is seen with polyuric, polydipsic glucosuria and hyperglycemia.[228] Descriptions of the gross and microscopic effects of diabetes mellitus on avian kidney tissue were not found.

One of the more common metabolic derangements associated with renal disease is dehydration. In chickens, dehydration has been associated with nephrosis characterized by tubular dilatation, with or without proteinaceous casts, epithelial necrosis and rare urate granulomas or casts.[198] Food restriction during dehydration may lessen the nephrosis lesions.[198]

Gross Renal Changes

Gross renal changes including masses, discolorations, and size and shape alteration are non-specific and should be cautiously interpreted.

Differential diagnoses for renomegaly include neoplasia, inflammation (including infectious and non-infectious diseases), cystic formation, ureteral obstruction, toxic changes, metabolic disorders (including dehydration, gout, lipidosis) and congenital abnormalities.[79] Also, non-pathological increase in kidney size has been noted in chickens fed certain dietary precursors such as inosine that increase plasma uric acid levels.[215] In these chickens, the renal enlargement was likely due to the increase in processing of uric acid in the kidney.[215] Renal and ureteral calculi also may be noted.

Postmortem Renal Change

Renal postmortem changes are noted in chickens as soon as 22 minutes following death at 37° C (98.6° F).[163] Early renal postmortem changes occur in the proximal tubular epithelium, followed by collecting tubule epithelium and glomerular nuclei.[163] Even with cooling to 4° C (39.2° F), proximal tubular changes can be observed within 45 minutes of death. The early postmortem proximal tubular changes can be confused with antemortem proximal tubular degeneration and should be interpreted with caution.[163] In effort to decrease postmortem changes, perform a necropsy and fix tissues as soon after death as possible.

PART 2: A Review of Diagnosis and Management

HISTORY AND PHYSICAL EXAMINATION

A historical review of a bird's environment, diet, source, exposure to infectious agents and toxins, genetics and behavior becomes important for both diagnosis and management of avian renal disease. Environmental factors can include exposure to known aerosolized, ingested or topical toxins. Adverse conditions that might lead to dehydration or other stresses also may be identified. The diet should reflect what is appropriate for that species, and the history should include any additional dietary supplementation or changes. Understanding the bird's origin, whether from a specific aviary, store, quarantine station, the wild, etc, may suggest the possibility of problems seen in other avian species from the same source. Known exposure to infectious agents (and again, toxins) is especially important, as definitive diagnosis of bacterial, viral, parasitic, fungal and toxic agents is not always possible without cultures, special stains, electron microscopy, in situ DNA hybridization, PCR probes or other diagnostics. Genetic problems are poorly described in birds, but with intense inbreeding, development of mutations or conservation breeding efforts from an extremely limited gene pool, it is reasonable to assume that hereditary defects will become more common. Behavioral changes including depression, anorexia, anuria, oliguria, polyuria, polydipsia, feather picking over the synsacrum, self-mutilation, seizures and others may be associated with renal disease and should be noted in the history.[181]

Most physical examination abnormalities associated with avian renal disease are non-specific, but there are some key findings that tend to warrant further investigation. It is highly likely that a bird with articular gout has had or currently has some form of renal disease. For this reason, consider renal biopsy in some birds with articular gout to help rule out or specifically identify kidney disease. Not all birds afflicted with articular gout, however, have renal disease. Unilateral leg lameness or paresis may accompany renal disease. This is particularly true if kidney disease causes inflammation or compression on the lumbar and/or sacral nerve plexus that is so intimately associated with the dorsal renal parenchyma. Birds with renal disease also may exhibit dehydration,

generalized weakness, regurgitation and decreased muscle mass with or without historical anorexia, all of which are non-specific signs.[166]

DIAGNOSTIC TESTS

Multiple diagnostic tests are available to help clinicians identify and define multiple disease processes in birds. As diagnostic technology improves, so will our ability to accurately diagnose diseases in birds. The tests listed below are ones that are most frequently discussed or used in diagnosing renal disease in birds. See **Table 16.3** for reported selected plasma-based diagnostics sometimes used in diagnosing renal disease in birds. Many diagnostics such as fecal floatation, which help diagnose renal coccidiosis, are not discussed, but should be included in a minimum database when evaluating sick birds. Some new or unfamiliar diagnostics also are introduced.

Considering all the diagnostic tests available, the author has noticed a pattern of laboratory abnormalities that is often strongly correlated with many forms of renal disease in birds. This includes persistently elevated uric acid (at least two consecutive tests on a well-hydrated and fasted bird), elevated creatinine phosphokinase (CPK), mild anemia and a relative heterophilia with or without a total heterophilia. Elevated CPK is a very non-specific indicator of multiple types of tissue damage and is not mentioned further. Using the currently available diagnostics, the actual type and degree of renal disease can be confirmed only with a kidney biopsy.

Complete Blood Count (CBC)

Some non-specific CBC changes may be associated with avian renal disease. A marked (relative) heterophilia was noted in two chickens with urolithiasis, but no total white blood cell count was given.[20] Heterophilia, monocytosis, lymphopenia and normocytic-normochromic anemia were noted in broiler chicks with various forms of histologically confirmed renal disease, but specific details were not given.[41] In a different study in chickens, clinically affected birds with histologically identified nephritis had significant heterophilic leukocytosis when compared to "normal" birds.[42] The author has reported that many pet birds (geese, doves, various psittacine birds) with different forms of renal disease have demonstrated a mild to marked relative heterophilia with a normal total white blood count.[47,64,65] These changes are non-specific, however, and can be seen in healthy birds under stress alone.[221]

Serum or Plasma-based Biochemistries

Selected plasma biochemistries may provide several useful clues toward renal disease in avian patients. Although many serum and plasma-based tests may be "abnormal" in birds with renal disease, only specific diagnostics are covered.

Table 16.3 | Selected Plasma-based Diagnostics in Birds

Diagnostic Test	Species	Normal Range	Reference(s)
Uric Acid	Pigeon	94-518 µmol/L, 225-574 µmol/L	87, 138, 141
	Peregrine falcon	253-996 µmol/L, 4.3-16.7 mg/dl	
Urea	Pigeon	0.36-0.64 mmol/L, 0.27-0.94 mmol/L	87, 138, 141
	Peregrine falcon	0.8-2.9 mmol/L, 2.2-7.0 mg/dl	
Creatinine	Pigeon	23.7-32.3 µmol/L, 20-56 µmol/L	87, 141
	Peregrine falcon	24-64 µmol/L, 0.27-0.72 mg/dl	
Urea/Uric Acid	Pigeons	1-3	138, 141
	Peregrine falcon	1.7-6.4	
Osmolality (mOsm/kg H_2O)	Pigeon	299.4-312.6	138, 141
	Peregrine Falcon	322-356	

Reference values for the pigeon (*Columba livia domestica*) and peregrine falcon (*Falco peregrinus*) are included. These reported values are highlighted because of their potential use in identifying renal disease and dehydration.

Uric Acid

Plasma uric acid can be useful as a screening tool for advanced renal disease. With the exception of gastrointestinal uricolysis, uric acid and its salts (urate) are the end product of nitrogen metabolism in birds.[9,60,132,214,246] Elevated uric acid has been correlated with histologically confirmed severe renal disease in chickens (tubular nephrosis and interstitial nephritis).[224] In a separate study involving dehydrated chickens, increased serum uric acid was associated with histologic renal lesions.[198] Broilers given oosporein (renal toxin), developed visceral and/or articular gout, swollen, pale kidneys and had a 48% increase of uric acid over control birds.[184] In a similar study with oosporein in turkey poults, intoxicated birds had dose-dependent increases in uric acid (over controls) ranging from 76 to 140%.[185] It was noted that fasting hyperuricemia (>16.7 mg/dl [>1000 µmol/L]) in peregrine falcons (*Falco peregrinus*) indicates renal failure.[141]

Uric acid is produced and secreted in the avian liver, kidney and pancreas.[46,106] Although produced predominately in the liver, at least 17% of the uric acid found in chicken urine may be synthesized in the kidney.[46] Specifically, nephrogenic uric acid synthesis may increase when plasma purine precursors are elevated.[46] These findings suggest the avian kidney has an important role in the synthesis, in addition to elimination, of uric acid, especially when increased precursors are available.[46] Precursors, including body proteins degraded because of poor nutritional status, have been suggested as a cause of elevated uric acid and should be considered in birds with hyperuricemia.[155]

An interesting secondary role of uric acid in birds is its antioxidant capability. In chickens, it has been clearly shown that plasma uric acid concentrations are inversely correlated with oxidative activity.[215] It has been stated that uric acid constitutes one of the most important antioxidants in birds and is directly linked to their longevity.[215]

Uric acid is cleared mainly via tubular secretion and is largely independent of glomerular filtration, water resorption and urine flow rate.[9,140,141,187,195] Blood uric acid levels are mildly affected by a bird's hydration status, but rather reflect the functional capacity of the renal proximal tubules.[187] However, in a study with dehydrated chickens, uric acid levels increased after 24 to 48 hours of water restriction, but only in those birds allowed free access to food.[198] Serum uric acid levels actually dropped within 24 hours in birds denied food and water.[198] It has been estimated that renal function must be below 30% of its original capacity before hyperuricemia develops.[166] Suggested normal avian uric acid levels range from less than 1 to 10 mg/dl (59.48-594.8 μmol/L).[214]

Hyperuricemia is defined as "any plasma uric acid concentration higher than the calculated limit of solubility of sodium urate in plasma." In bird plasma, this theoretical limit of solubility of sodium urate is estimated to be 600 μmol/L (10.8 mg/dl).[143]

In chickens, the uric acid renal tubule transport system does not appear to become saturated until plasma uric acid levels exceed 60 mg/dl (3569 μmol/L)[9] which demonstrates the lack of clarity in the literature and experimental dosages.[9] Chickens genetically predisposed to hyperuricemia and fed high-protein (60%) diets develop an elevated steady state of plasma uric acid (10-60 mg/dl {59.48-3569 μmol/L}) in order to excrete their daily loads of this by-product.[9] The increased basal plasma uric acid made the affected chickens susceptible to articular gout formation.[9] One group suggested that these chickens genetically predisposed to gout had a defective uric acid transport mechanism at the peritubular membrane.[184]

Uric acid represents 80% or more of the nitrogen excreted by birds.[9,214] Therefore, a significant increase in the proportion of nitrogen excreted as uric acid is not likely, even with increased dietary protein consumption. At least in chickens, hyperuricemia is likely due to reduced renal tubular secretion of uric acid and not excessive production as can occur in humans.[9,214] These findings imply that renal tubular diseases are likely responsible for hyperuricemia, and uric acid abnormalities may not be evident until very high-protein diets are fed. Specifically in chickens, dysfunctional proximal convoluted tubules result in reduced urate secretion and can lead to hyperuricemia if severe.[214]

In birds of prey, uric acid production is directly related to the amount of protein consumed and transient rises are noted following high-protein meals.[140,143] Peregrine falcons (*Falco peregrinus*) and red-tailed hawks (*Buteo jamaicensis*) are reported to have a "significant" postprandial increase in plasma uric acid concentration (hyperuricemia) for up to 12 hours after ingesting a natural meal.[140,143] The significant postprandial uric acid increase noted in peregrine falcons was up to 32 mg/dl (reported as 1881 μmol/L) between 3 and 8 hours after being fed.[143] It has been stated that significant postprandial increases in both urea and uric acid persist for up to 15 hours in peregrine falcons.[143] It was not clear why these birds of prey did not develop gout lesions, but the authors recommended a 24-hour fast prior to evaluating serum uric acid in peregrine falcons.[143] The authors further recommend that a 24-hour fast should be considered for all carnivorous avian species prior to blood uric acid testing. Almost identical findings of postprandial hyperuricemia were noted in blackfooted penguins (*Spheniscus demersus*) and represent another species that should be fasted before measuring uric acid levels.[122]

Uric acid production following a high-protein meal has been studied in various psittacine birds. In one study with African grey parrots (*Psittacus erithacus* sp.), plasma uric acid concentrations showed a positive correlation with dietary protein consumption.[105] However, even though the fed protein level was as high as 30%, plasma uric acid levels remained within normal ranges.[105] In cockatiels fed 11, 20, 35 and 70% protein for 11 months, serum uric acid increased linearly with dietary protein levels.[123] However, the serum uric acid level was significantly greater only in birds fed 70% protein diets. Because no histologic or gross renal lesions were found at necropsy, the authors concluded that the rise of uric acid was related to dietary protein concentration and not kidney damage.[123] It was found that feeding diets containing 13.5, 18.2 and 24.6% protein for up to 24 weeks had no effect on serum uric acid levels in parakeets.[8]

In consideration of the above-described causes of elevations in uric acid, this single biochemistry value can help identify significant renal disease. The author prefers to repeat (fasting) uric acid levels on well-hydrated birds before a suggestion of renal disease is made. In birds with suspect renal disease that have a single laboratory value of hyperuricemia, the author will often give a total of 100 ml/kg SQ, SID to BID of isotonic fluids for 2 days and then recheck the uric acid level. In the author's experience, birds with persistent hyperuricemia after fluid therapy and/or fasting have some form of renal disease.

Urea

Unlike mammals, urea in birds is produced only in small

amounts (by renal mitochondrial breakdown of arginine) and does not serve as the end product of protein metabolism.[187] Plasma urea in birds is excreted by glomerular filtration and, unlike uric acid, blood urea concentrations are more significantly affected by the bird's hydration status.[138,140,187] During normal hydration, filtered urea is 100% excreted but is 99% reabsorbed in the tubules during dehydration.[138,141] Plasma urea also has been shown to significantly increase in peregrine falcons for up to 15 hours postmeal.[143] In studied cockatiels, serum urea levels increased linearly with dietary protein levels (11, 20, 35 and 70%).[123] Separate studies involving the domestic fowl and pigeons demonstrated decreased urea elimination and/or increased blood urea levels (6.5- to 15.3-fold increase in pigeons) in dehydrated birds.[138,187] It has been shown that plasma urea nitrogen increased in a dose-dependent fashion (in turkeys) at every level of dietary oosporein (nephrotoxin).[185] These intoxicated turkey poults also were showing signs of dehydration.[185] It has been proposed that plasma urea is the single most useful indicator of prerenal (dehydration) causes of kidney failure in birds.[140]

The urea:creatinine and urea:uric acid ratios can be used to better define pre- and postrenal azotemia. Because reabsorption of urea is disproportionally higher than both creatinine and uric acid, these ratios should be high during dehydration and ureteral obstruction.[138] The formulas for these ratios are listed below:

$$\text{Urea:creatinine} = \frac{\text{urea (mmol/L)} \times 1000}{\text{creatinine } (\mu\text{mol/L})}$$

$$\text{Urea:uric acid} = \frac{\text{urea (mmol/L)} \times 1000}{\text{uric acid } (\mu\text{mol/L})}$$

Creatinine

Birds produce little creatinine from its precursor, creatine.[166] Creatinine is eliminated by tubular secretion but clearance is variable.[81] Clinically, creatinine may be elevated in pet birds by feeding high-protein diets.[81] It was shown that plasma creatinine also will increase significantly in dehydrated pigeons.[138] The relationship between creatine and creatinine in birds with renal disease is poorly understood, and differentiation does not appear to be useful clinically.[81,166,181]

Proteins

Although hypoproteinemia has been noted as being associated with renal failure, few studies have evaluated serum protein levels in birds with renal disease.[141] Biochemically determined low serum protein has been noted in chickens with advanced tubular nephrosis and interstitial nephritis.[224] In two chicken flocks with spontaneously occurring urolithiasis, plasma protein level changes (method of determination not disclosed) were not significantly associated with renal disease.[20,251] While affected birds developed articular and/or visceral gout, gross renal changes and death, broilers intoxicated with oosporein (fungal nephrotoxin) had, with the exception of one group, no significant changes in plasma protein (biuret method) over the normal (control) birds.[184] A single group of broilers receiving a midrange amount of oosporein had a statistically significant rise in plasma protein over controls. The cause for this single discrepancy was not determined.[184] In a similar study using oosporein-intoxicated turkey poults, statistically significant decreased albumin:total protein was noted at all levels of intoxication over controls, but total protein remained unchanged and albumin was not significantly decreased until the highest levels of the toxin were given.[185] These few studies show a couple of important facts: there is limited information properly associating plasma proteins with renal disease, and differing species may have dissimilar plasma protein levels under similar disease conditions. As discussed under Part 2: Electrophoresis, Plasma Protein Electrophoresis, protein levels should be evaluated electrophoretically (in addition to the more common biochemical methods).

PLASMA ELECTROLYTES

The effect of renal disease on plasma electrolytes is poorly studied in birds. Hyperkalemia and hyperphosphatemia have been loosely associated with renal failure, but studies are limited in birds.[141] No significant associations between renal disease and plasma sodium, potassium, calcium, magnesium, chloride and phosphate levels were noted in birds from two chicken flocks with spontaneously occurring urolithiasis.[20,251] Specific sample collection/storage was not discussed and the authors conceded that their handling of the samples might have affected the results.[20,251] Dehydrated chickens allowed free access to food developed significantly elevated serum sodium and phosphorous by 24 hours and after 24 hours, respectively, but maintained normal potassium levels.[198] Histologically, these chickens had mild renal tubular dilatation.[198] Turkey poults intoxicated with oosporein (nephrotoxin) developed significantly decreased plasma potassium and phosphorous, and had no changes in sodium compared to controls.[185] As the avian kidney is responsible for electrolyte regulation, it is reasonable to assume that electrolyte disorders can be present in birds with renal disease.

MICROBIOLOGIC ANALYSIS

Microbiologic assays may be useful in identifying infectious causes of avian renal disease. Bacteria may enter the renal system either hematogenously, ascending from the ureters and cloaca, or as an extension of surrounding organ infection.[187] The avian coccygeomesenteric vein drains the mesentery of the hindgut into the

hepatic portal and/or the renal portal vein.[226] It is conceivable that colitis may serve as a hematogenous source of infectious agents, toxins and inflammatory products to the kidney if blood flow draining the colon is diverted into the renal vasculature. For this reason, collection of a cloacal or fecal microbial culture is a rational portion of the supportive laboratory database in birds with suspected renal disease. Severe ulcerative colitis caused by *Salmonella* infection resulted in ascending bacterial nephritis in four African grey parrots.[187]

Bacterial nephritis in birds is often a component of systemic infection and multiple organs may be involved.[187] In one study, 50% of birds with systemic bacterial infections had kidney involvement, suggesting that any bacterial septicemia can potentially result in nephritis.[187] Identification of bacteria within renal tissue may be difficult, as has been noted in dogs and swine with renal disease putatively associated with a bacterial etiology.[26] Blood cultures are an appropriate consideration if septicemia is suspected. Prior to blood collection, the skin over the venipuncture site is aseptically prepared by thorough cleaning with alcohol and organic iodine (as with surgical preparation).[58,107] The jugular and basilic veins are described as appropriate blood collection sites in septicemic birds.[25,58] Using aseptic techniques, renal biopsy specimens also can be sampled for microbial cultures. The cause of infectious nephritis in birds is not limited to bacteria, and various culture methods and other diagnostic procedures also may be useful for identifying fungal, viral and parasitic organisms.

URINALYSIS

Biochemical and cytological sediment analysis of avian urine has been advocated as potentially useful in diagnosing avian renal disease.[128,166,181,188,228] In birds, hematuria may be noted with renal disease, but should be carefully differentiated from bleeding originating from the gastrointestinal and reproductive tracts.[181] Hemoglobinuria, as noted in *Amazona* spp. parrots with lead intoxication and in other species with differing disorders, may or may not be related to renal disease. Toxic, neoplastic, bacterial and viral nephropathies may be more frequently seen associated with hematuria in birds.[181] White blood cells were seen in 45% of urine sediment from pigeons with paratyphus, many of which had intersitial nephritis.[87] Sediment analysis should be a part of an avian urinalysis and specific cellular urinary components have been discussed.[128,188,228]

Several significant factors complicate interpreting avian urinalysis. First, urine is mixed with feces in the cloaca. The one possible exception is the ostrich, which appears to eliminate urinary waste separate from the feces.[168] Second, in many species ureteral urine is refluxed orad into the lower intestines to the ceca, where water and sometimes electrolyte reabsorption takes place.[252] Additionally, diseases of the lower intestine may alter urine production and composition. Gastrointestinal bleeding, inflammation, normal and abnormal organisms, etc, may end up in a "urinalysis" harvested from a dropping, giving the false impression that red and white blood cells and/or infectious agents, respectively, came from the urinary tract. In short, the "urine" present in a dropping is not the same urine produced from the kidneys. Urinalysis results should be carefully interpreted.

Collection

True urine can be collected in birds only with some difficulty. Once emptied of feces, specially designed cannulas can be inserted into the cloaca for collection of ureteral urine. One group used a Foley catheter to occlude the rectum but not the ureters and successfully collected ureteral urine in chickens.[21] Small closed-end cannulas constructed from micropipette tips were used to collect ureteral urine from house (*Passer domesticus*) and song sparrows (*Melospiza melodia*).[38] The opening of the closed-end cannula was placed over the ureteral orifices.[38] A similar design was used in house sparrows to make cloacal cannulas from PE-240 tubing with a hole cut near the sealed end.[89] The sealed end prevented intestinal fluids from contaminating the urine once the cannula was in place.[89] Under local anesthesia, 1.5-ml microcentrifuge tubes were sutured into the cloacas of chickens to allow collection of ureteral urine.[205] Cyanoacrylate was used to glue cannulas over the ureteral orifices of chickens.[73] Several obvious drawbacks include restraint or sedation of the patient while urine is slowly produced, and the cannulation itself may induce diuresis.[249,252] Clearly, there are numerous methods, with varying degrees of difficulty, used to collect ureteral urine.

Casts

Urinary casts represent cellular and/or acellular material sloughed from the inner lining of various renal tubules. This material is generally in the shape (or a "cast") of the tubule from which it originated. Casts are sometimes noted on histologic sections. Protein and cellular casts were histologically noted in an Australian diamond firetail finch (*Stagnopleura bella*) with *Cryptosporidium* sp. and multifocal amyloidosis.[19] Albuminous casts in renal tubules of pigeons infected with virulent *Trichomonas gallinae* were noted.[172] Hyaline casts were identified in kidney sections of birds experimentally infected with infectious bursal disease (Gumboro disease).[214] Eosinophilic granular casts have been found within the renal tubules of turkeys afflicted with salt toxicosis.[242] Eosinophilic tubular casts, possibly containing myoglobin, in an ostrich with acute muscle necrosis and anuric renal fail-

ure have been reported.[187] A rhea with hemoglobinuric nephrosis developed eosinophilic casts in the renal collecting tubules.[16] Both hyaline and granular tubular casts were present in racing pigeons infected with avian paramyxovirus type 1.[11] Granular, hyaline and albuminous casts were seen in the renal tubules of chickens experimentally infected with several pathogenic bacteria.[219]

Identifying casts in urine is reported as highly significant, a sign of renal disease and/or can be a non-specific indicator of tubular renal disease in birds.[128,134] With that stated, the papers cited above describe histological sections with no discussion of casts in the urine. The author disagrees that urinary casts are highly significant or a definite sign of renal disease, as there is little information correlating casts found in a urinalysis with any type of renal disease in birds. However, casts should be noted and may have correlation with some forms of avian renal disease. Epithelial casts were found in 2 out of 35 ostrich urine samples, but no correlation was made with any renal parameters.[168] Epithelial casts were noted in 20% of urine samples from *Salmonella typhimurium*-infected pigeons.[87] Although many birds did have histologically confirmed renal disease, no correlation was made between those pigeons with kidney lesions and those with urinary casts. The large variety of "types" of casts reported also suggests that an inconsistent naming system exists within the current literature.

Urine Chemistries and Electrolytes

Standard mammalian dipsticks may be used, but not all components are applicable to avian urine.[166] Chicken urine reportedly contains non-uric acid chromogen.[9] Non-protein chromogens are known to interfere with refractometric and chemical measurement of plasma proteins and also may apply to avian urine sampling.[81]

Few studies even mention test strips used in avian urinalyses. One study evaluated commercial urine dipsticks[a] on normal urine of 35 ostriches.[168] Because ostriches can eliminate urinary waste separate from feces, these values may not apply to most other birds. In the study, 31/35 (89%) and 35/35 (100%) of the urine samples were positive for nitrite and protein, respectively. The urine chemistry strips were negative for glucose, urobilinogen, bilirubin and ketones in all ostriches.[168] No association with renal disease was made. Using the dipsticks, nitrite and protein also were positive in 90% (18/20) and 50% (10/20), respectively, of the ureteral urine samples from pigeons with paratyphus.[87] The same strips identified blood in all samples, which correlated to red blood cells seen in only 45% of urine sediments.[87] If the cells in the urine had been lysed, the strips would be positive and the cytology was negative in this study. Urine strips also may detect undigested hemoglobin found in the excrement of the bird, especially carnivorous species with short digestion times, and give a positive result.[134] Myoglobinuria also may cause positive reactions and can be distinguished from hemoglobinuria only by spectrophotometry.[134] Finally, porphyrinuria, as seen with lead-poisoned Amazon parrots (*Amazona* spp.), may result in red-colored urine visually mimicking hemoglobinuria.[134] Because of the inconsistent results and limited critical studies noted in the literature, difficulty in obtaining ureteral urine and clinical experience, it is the author's opinion that the currently available chemistry strips have limited value in an avian urinalysis.

Urine electrolytes and chemistries can be collected, but there is limited information on their interpretation. It has been suggested that because renal intracellular enzymes are likely voided in the urine, urinary chemistries might be useful in detecting kidney damage.[141] Urine sodium and potassium were measured, and insignificantly changed, in house sparrows undergoing trials with the antidiuretic arginine vasotocin.[89] One study noted that in normal and dehydrated starlings (*Sturnus vulgaris*), cloacal urine contained significantly higher concentrations of magnesium, phosphate, potassium and total osmolality than found in ureteral samples.[204] This study supports the recommendation that ureteral samples must be collected to obtain a "true" evaluation of avian urine, again making urinary chemistry evaluation impractical in a clinical setting.

One renal enzyme, N-acetyl-ß-D-glucosaminidase (NAG), has been successfully evaluated in the urine of chickens as a marker for kidney damage.[74] In mammals and chickens, NAG is a renal tubular enzyme. In humans, urinary NAG has been suggested for use as an early predictor of renal tubular damage and may be a good non-invasive indicator of disease progression.[49] Elevated urinary (ureteral urine), but not plasma, NAG was noted at 40 days of excessive vitamin D_3 supplementation in chickens.[74] Although the information is limited, further studies may show that NAG, and possibly other urinary enzymes, may become useful as early markers of renal disease in birds.

Osmolality and Specific Gravity

Avian urine is typically isosmotic because the predominant reptilian-type nephrons cannot concentrate urine beyond plasma osmolality.[141] In normal birds, urine osmolality can maximally be increased to 2.0 to 2.5 times that of plasma osmolality.[27,28,40,141] Even this number is high for some species, as emus (*Dromiceius novaehollandiae*) are reported to have maximal urine to plasma osmotic ratio of only 1.4 to 1.5.[216] This is minimal in comparison to some mammals that can concentrate urine osmolality 25 to 30 times that of plasma.[27,28,40]

There is limited information on urine specific gravity or osmolality in avian health or disease. The reported average (refractometrically determined) urine specific gravity of ostriches (*Struthio camelus*) is 1.02 with a range of 1.01 to 1.05.[168] Consistent polyuria and hyposthenuria (60% had specific gravity below 1.007) was noted in *Salmonella typhimurium*-infected pigeons, many of which had interstitial nephritis.[87] In a separate evaluation, urine osmolality significantly increased up to 3 times control levels in postflight and dehydrated pigeons.[88] The author has used urine specific gravity diagnostically as discussed below under Water Deprivation Testing.

Urine pH

Urine pH is highly variable in birds. The urine pH may be acid (down to 4.7) in egg-laying female birds during calcium deposition.[77] Once the egg is laid or calcium is no longer being deposited, urinary pH may climb to 8.0. Male birds have an approximate urine pH of 6.4. Hypoxia, as noted in diving ducks, may drop urine pH to 4.7.[77] Normal ostriches have a urine pH range of 6.1 to 9.1, with a mean of 7.6.[168]

ELECTROPHORESIS

Plasma Protein Electrophoresis

Properly determined hypoalbuminemia (via plasma electrophoresis) is not reported in confirmed active cases of avian renal disease. However, it is possible that birds may develop low albumin/protein with some kidney disorders. Biochemically determined hypoalbuminemia has been noted in some active avian renal disease cases.[64,65,185]

The literature states that as the currently available biochemical tests likely do not accurately report avian albumin levels, serum/plasma protein electrophoresis is necessary to properly quantitate blood proteins and should be performed if hypoalbuminemia is suspected.[81,139,142] Decreased albumin and elevated betaglobulins and alpha$_2$ macroglobulin, as recorded with serum electrophoresis, have been reported with avian nephritis.[47,51] However, there are no controlled studies to support the above statements that correlate protein electrophoresis abnormalities with any renal pathology in birds.

With the above stated, one study showed that an analyzer using the biuret and bromocresol green dye-binding methodologies for total protein and albumin determination, respectively, had good agreement between whole blood and plasma samples.[115] On the contrary, there was poor correlation between the results from the studied analyzer and samples evaluated via electrophoresis used at two major reference laboratories. Due to the discrepancies, the authors concluded that neither reference laboratory using electrophoresis served as the "gold standard" for total protein and albumin determination.[115]

These very limited studies suggest inconsistencies in the "gold standard" method of serum/plasma total protein and albumin determination, and question the true value of these diagnostics in birds with renal disease. Regardless, it is the author's opinion that monitoring serum and/or plasma protein levels has diagnostic value in birds, even if not necessarily used in renal disease cases. The author recommends consistently using one of the common biochemical methods of protein determination and comparing those results to electrophoresis, the goal being to become familiar with test results from one or two diagnostic methods and correlating those results to (histologically) confirmed disease.

Urinary Protein Electrophoresis

In mammals, proteinuria is broken down into preglomerular, glomerular and postglomerular urinary protein loss. Preglomerular proteinuria occurs when large amounts of small molecular weight proteins (immunoglobulin fragments, hemoglobin and myoglobin) that readily pass through normal glomerular walls are lost in the urine.[137] Glomerular proteinuria occurs when diseased glomerular membranes allow large proteins (albumin, immunoglobulins, some coagulation proteins/antithrombin III) to pass.[137,245] Postglomerular proteinuria results from normal genital secretions as well as urogenital infections, trauma and neoplasia.[137] Although uncommon in mammals, defects resulting in proximal renal tubular protein resorption result in (postglomerular) tubular proteinuria.[137,245]

Avian urine normally contains a large amount of protein (average of 5 mg/ml up to 15 mg/ml), especially when compared to that of mammals (<0.09 mg/ml in dogs and humans).[27,111] Amino acids are freely filtered at the glomerulus, but normally are almost completely reabsorbed by the renal tubules in birds.[60] Because uric acid is poorly water soluble, very little avian ureteral urine is required to eliminate this protein waste. Instead, proteinuria is likely necessary to maintain the excreted uric acid-containing spheres in a colloidal suspension, preventing aggregation and renal tubular blockage.[28,111] Within the proximal tubule, uric acid is bound to a protein to solubilize the waste product and prevent crystal formation.[215] The reflux of urine into the cloaca may be a mechanism to recover some of the urinary protein, as cloacally voided fluid contains very little protein compared to ureteral samples.[28]

Serum albumin, among other proteins, is found in both the liquid urine and uric acid spheres in chickens.[111] In the normal junglefowl (*Gallus gallus*), the urinary pro-

teins (averaged 2.01 mg/ml urine) identified closely matched the plasma proteins. This led to the conclusion that protein is passed through a glomerular filtration barrier differently than occurs with most mammals.[111] There are, however, differences in concentrations of plasma and urinary proteins suggesting differential filtration and/or absorption of some proteins by renal tubules.[111]

Pathologic proteinuria is poorly described in birds. In one study, control chickens and those with experimentally induced autoimmune glomerulonephritis produced urinary protein (measured via 3% sulfosalicylic acid with a bovine serum albumin standard) at 5 mg/24 h.[21] Test birds developed no abnormal proteinuria, but were considered moderately proteinuric after given IV colloidal carbon (3 to 8 times increase in proteinuria). Colloidal carbon induces proteinuria in other species, but the mechanism is not clear.[21] As discussed under Part 1: General Renal Disease Categories, Glomerulopathies, birds may not be capable of developing pathologic proteinuria with glomerular disease as is recognized in mammals. However, it is possible that pathologic proteinuria develops more slowly in birds compared with mammals, and as a result has not been frequently discussed or evaluated in clinical cases.[86] If pathologic proteinuria is suspected, urine protein electrophoresis should be used to differentiate protein type and size.[94,245] If performed, it would be beneficial to compare urinary protein levels from a sick patient with samples from a healthy member of the same species. Finally, ureteral urine should be collected to rule out any effects from protein absorption or from other proteins present in the lower intestine. In a normal clinical setting, these collection requirements and limited studies make meaningful urinary protein interpretation in birds impractical.

IMAGING

Radiography

Plain and contrast radiography, nuclear scintigraphy, ultrasound, magnetic resonance imaging and computed tomography can be used to "image" the avian kidneys.[108,134,151,166,181,206] The avian kidney lies in a fossae created by the ventral surface of the synsacrum.[79,152] With bone dorsal and air sacs surrounding ventrally, imaging of the avian renal system is difficult with some techniques. Indirect methods such as positive contrast radiography of the alimentary tract may be helpful in outlining renal masses.[141]

A lateral view is the best method to radiographically view the kidneys[141] (Fig 16.12). As viewed with a lateral radiograph, the absence of the normal dorsal diverticulum of the abdominal air sac (dorsal to the kidney and ventral to the synsacrum) may indicate renal enlargement.[166,187] Improper positioning can artifactually change the appearance of this air-filled diverticulum.[166] Because the renal silhouettes are superimposed on a lateral view of the abdomen, an oblique view also may be used to distinguish each kidney.[79] Renal density and gross size changes may indicate renal disease.[166,181] Radiographically visible renomegaly was noted in a salmon-crested cockatoo with chronic interstitial nephritis and calcification as the result of hypervitaminosis D_3.[211] Nephrocalcinosis was detected radiographically in ostriches and appeared as multiple radio-opacities throughout the renal parenchyma.[162]

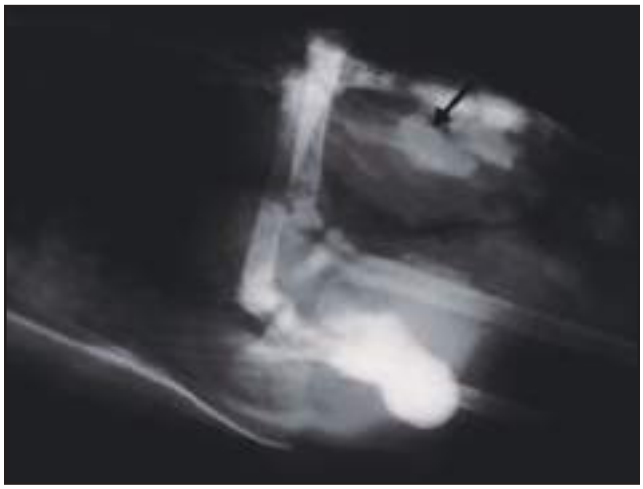


Fig 16.12 | Lateral radiograph of an adult domestic goose with renal fibrosis and mineralization. Note the mineralized kidney tissue (arrow).

Ultrasound

Due to the presence of surrounding air sacs (ventrally) and bone (dorsally and laterally), ultrasonographic imaging of normal avian kidneys is difficult.[108] In one study of 386 mixed bird species that underwent ultrasonographic evaluation of the urogenital tract, abnormalities such as renal cysts (6), cancer (12) and inflammatory nephromegaly (11) were identified in only 29 patients. The authors concluded that sonographic imaging of the normal kidney was not possible.[108] Some disease conditions that either obliterate the air sacs or result in fluid accumulation in the coelomic cavity may actually improve renal ultrasonographic imaging.[152] In these abnormal situations, ultrasonography can serve as a non-invasive and safe means to evaluate coelomic structures such as the kidneys.

Intravenous Excretory Urography

Intravenous excretory urography has been described in birds as a method to gain information on kidney size, shape and function.[141] Use of organic iodine compounds given IV in the basilic vein has been reported. The

organic iodine can be visualized radiographically in the heart and pulmonary artery within 10 seconds, and outlining the kidneys and ureters 20 to 50 seconds later. After 2 to 5 minutes, the cloaca will be outlined. This technique should not be used in birds with severe renal compromise.[141]

It is the author's opinion that intravenous excretory urography may have some limited uses in a clinical setting as demonstrated in the case report below. A water-soluble iodinated contrast agent[b] was successfully used to evaluate the ureters post-ureterotomy in a double-yellow headed Amazon parrot (*Amazona ochrocephala*).[56] The agent was dosed at 400 mg/kg and given in the right medial metatarsal vein. Radiographic images were taken at 1, 2, 7 and 10 minutes postinjection. Ureter peristaltic movement and size were successfully evaluated using this technique.[56]

Renal Scintigraphy

Avian renal scintigraphy has been described.[150] The radioisotopes ^{99m}Tc-dimercaptosuccinic acid (^{99m}Tc-DMSA) and ^{99m}Tc-diethylenetriamine pentaacetic acid (^{99m}Tc-DTPA) were used in domestic pigeons. The tested birds were given nephrotoxic doses of gentamicin at 15 mg/kg IM q 12 h for 6 days. The birds were divided into two groups and renal scintigraphy using a mean of 41.8 MBq of intraosseous ^{99m}Tc-DMSA or 42.8 MBq of intraosseous ^{99m}Tc-DTPA was performed on the last day of gentamicin toxicosis and again 2 days later. Pre and post-gentamicin-treated kidneys were biopsied and confirmed normal histology pre-treatment and significant renal damage post-treatment. Uric acid was measured and interestingly did not significantly correlate with renal histology or scintigraphy findings. The authors reported 'decreased renal radiopharmaceutical uptake for ^{99m}Tc-DMSA and ^{99m}Tc-DTPA indicated nephrotoxicosis'. More specifically, scintigraphy using ^{99m}Tc-DTPA correlated well with renal histologic grades. While scintigraphy using ^{99m}Tc-DMSA did not correlate well with renal histologic grades it may be used to demarcate neoplasms, cysts and other physical alterations to the renal parenchyma. While renal scintigraphy can be performed at facilities that routinely provide nuclear medicine procedures, obvious drawbacks include cost and the need to confine birds for 12 to 24 hours until the radiopharmaceutical used has degraded.[150]

WATER DEPRIVATION TESTING

Water deprivation testing is considered when attempting to rule out unknown causes of polyuria/polydipsia (PU/PD) including central and nephrogenic diabetes insipidus and psychogenic polydipsia. There are numerous causes of PU/PD in birds that first must be ruled out using a complete historical, physical and laboratory evaluation. Some of the many causes of PU/PD in birds include organic (liver, kidney, intestine and cardiac), endocrine (diabetes mellitus) and metabolic (hypercalcemia) diseases.

A water deprivation test is carefully performed using a simple cage. The bird is weighed and blood and urine are collected. Evaluate the packed cell volume (PCV), total solids and osmolality of blood, and specific gravity and osmolality of urine. In one report of an African grey parrot (*Psittacus erithacus erithacus*) undergoing a water deprivation test, the authors evaluated plasma sodium, potassium and osmolality in addition to the above listed urine parameters.[144]

Place the avian patient in a cage with no food or water for the duration of the test. Evaluate both blood and urine parameters every 3 to 24 hours for 12 to 48 hours, depending on the species and physical condition of the bird. The reported African grey parrot was evaluated every 24 hours.[144] As a normal response some birds such as European starlings may become distressed within 24 hours of water deprivation, which should be considered when interpreting the results.[204] On the other hand, pigeons deprived of water for 36 hours had little change in plasma osmolality, demonstrating the variable responses to dehydration in differing species.[88] As a general rule, smaller birds should be evaluated more frequently.

The bird's behavior and laboratory results give a presumptive diagnosis. Birds with psychogenic polydipsia should tolerate this test well and develop more concentrated urine (increased osmolality and specific gravity) and an increase in PCV, total solids and plasma osmolality, all consistent with dehydration. This was the pattern seen in the African grey parrot and subsequent treatment with water restriction proved curative.[144] These individual values should all be carefully interpreted as noted in a study of dehydrated starlings where the hematocrit remained unchanged (compared with hydrated birds) and was not a reliable indicator of hydration.[204]

Birds with central (lack of production of arginine vasotocin [AVT]) or nephrogenic (inadequate response to AVT) diabetes insipidus should have different results than those with psychogenic causes. Birds with diabetes insipidus become dehydrated (as supported by plasma variables) but maintain dilute urine (low specific gravity and osmolality). Normal house sparrows given arginine vasotocin (0.4 ng/kg per minute to 1.6 ng/kg per minute) had a significant drop in urine flow rate (50.2 to 28.9% of normal, respectively) and increased urine osmolality (150.1 to 196% of normal, respectively).[89] A similar response would be expected in other normal birds of different species.

A strain of chickens with hereditary diabetes insipidus has been described.[33] These polyuric chickens produced low osmolality urine and maintained high circulating levels of AVT. The vital functions of these chickens became impaired after 48 hours of water deprivation. When given AVT, additional to their high circulating levels, these birds had minimal response. Either the birds had improperly responding kidneys or the AVT was defective.[33]

In the author's experience with one male canary-winged parakeet (*Brotogeris versicolorus*) with suspected diabetes insipidus, the bird became panicked within 4 hours as he became rapidly dehydrated, but maintained excessive production of dilute urine. The canary-winged parakeet had normal plasma biochemistries, complete blood count, screening radiographs and renal biopsy (light microscopy), and had a history of severe PU/PD since weaning. A diagnosis beyond presumptive diabetes insipidus was not made, since AVT levels were not evaluated.

IDENTIFYING URIC ACID CRYSTALS

Gout results when uric acid precipitates out as a solid, chalky substance in joints (articular) or on tissue surfaces (visceral). Articular gout material may be recovered using fine needle aspiration. Uric acid crystals are easily confirmed using microscopy or the murexide test. Cytologically, "gouty" material typically presents as uric acid crystals surrounded by a pyogranulomatous infiltrate, usually without organisms. The needle-shaped crystals are easy to identify on direct and stained smears. To perform the murexide test, place a small amount of the suspect material on a slide and mix with nitric acid.[141] Use a flame to evaporate and/or dry the mixture. Once cool, add one drop of concentrated ammonia. If urates are present, a mauve color will appear.[141] Due to their water-soluble nature, urates will dissolve in formalin and, therefore, the crystalline form will not be seen on conventionally fixed tissue. However, urates can be seen in alcohol-fixed tissue using Gomori's methenamine silver impregnation technique.[141]

EVALUATING GLOMERULAR FILTRATION RATE

Glomerular filtration rate has been studied in chickens as a method to evaluate renal function. Glomerular filtration rate is considered the most reliable quantitative index of renal function, and is an important tool for the diagnosis and management of kidney disease of mammals.[156] Most methods of measuring glomerular filtration rate and effective renal plasma flow are difficult and time consuming.[197] As a result, determining glomerular filtration rate in birds is often limited to research situations.

In general, urine flow rate (UFR) is first calculated as the volume (of ureteral urine) collected per kilogram of body weight per minute. The urine to plasma concentration ratio of a (usually parenterally administered) marker substance such as inulin is multiplied by the urine flow rate. Glomerular filtration rate (milliliters per kilogram body weight per minute) can then be calculated by measuring the clearance of the marker substance.[73] The basic formula is as follows:

$$\text{Glomerular filtration rate} = \frac{\text{UFR} \times \text{urine marker substance concentration (inulin)}}{\text{plasma marker substance concentration (inulin)}}$$

The single injection, double isotope method, utilizing ^{3}H-inulin ([methoxy-^{3}H]-inulin) and ^{14}C-PAH (para-[glycyl-1-^{14}C]-aminohippuric acid), has been shown to be a simple, reliable and rapid method for evaluating renal function in chickens.[197] If needed, the specific procedures of evaluating glomerular filtration in birds can be reviewed in the literature.[73,88,90,131,197,204,249,250]

BIOPSY

When history, physical examination and/or laboratory abnormalities support the presence of renal disease, consider biopsy. Currently, the only way to definitively diagnose avian renal disease and specific pathologic patterns is with a kidney biopsy and histopathologic evaluation.[141] A renal biopsy is most frequently performed during endoscopic examination of the coelomic cavity and, specifically, the kidneys. Before a renal biopsy is performed, the cost:benefit of the surgical procedure versus conservative therapy must be considered, as many birds have compromised health, especially if they have kidney disease.

Several methods of renal biopsy, primarily via endoscopy, and detailed accounts of avian kidney anatomy and physiology have been previously discussed[78,134,165,167,181,227,229,233,234] (Figs 16.13-16.18). For the most part, renal tissues can be stored in 10% formalin for light microscopy. If available, additional tissue may be stored in glutaraldehyde (electron microscopy), culture media (organism recovery) and alcohol (visualizing uric acid crystals), or frozen (PCR studies).

Renal histologic lesions are rarely pathognomonic for a specific disease process. Many different diseases cause similar renal lesions. Additionally, different pathologists may make differing morphologic diagnoses on the same renal tissue.[239] The author encourages veterinarians to work with a pathologist familiar with normal and abnormal avian histology. Oftentimes, it is the pathologist's interpretation of a renal biopsy combined with the attending veterinarian's case familiarity that enables both parties to make a definitive diagnosis or build a reasonable differential diagnoses list compatible with the kidney lesions noted. This approach has a key role in the

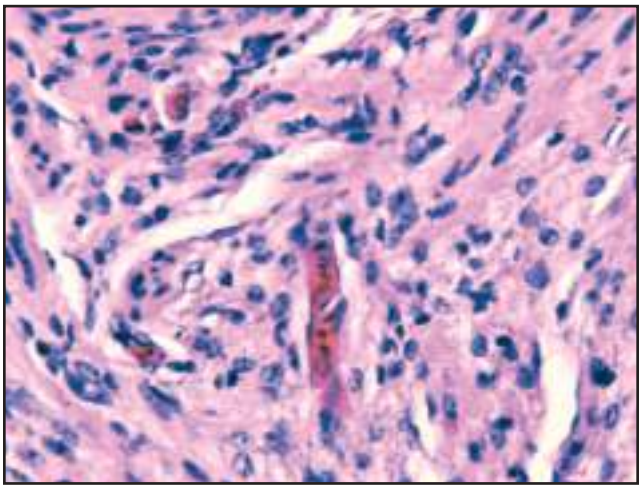


Fig 16.13 | An adult domestic goose with undifferentiated renal sarcoma. The renal architecture is destroyed and has been replaced by neoplastic spindle cells.

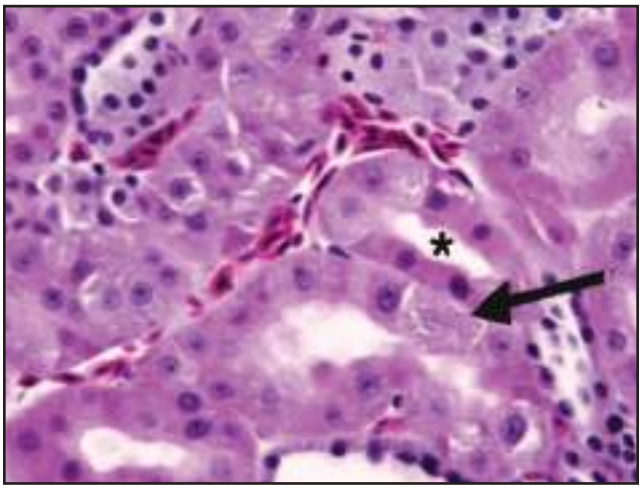


Fig 16.14 | Histologically normal renal tissue from an adult hyacinth macaw (*Anodorhynchus hyacinthinus*). Note the well-organized renal tubules, normal tubular lumen size (*) and lack of inflammatory cells. One tubular epithelial cell is undergoing degeneration (arrow), but the cells appear healthy otherwise.

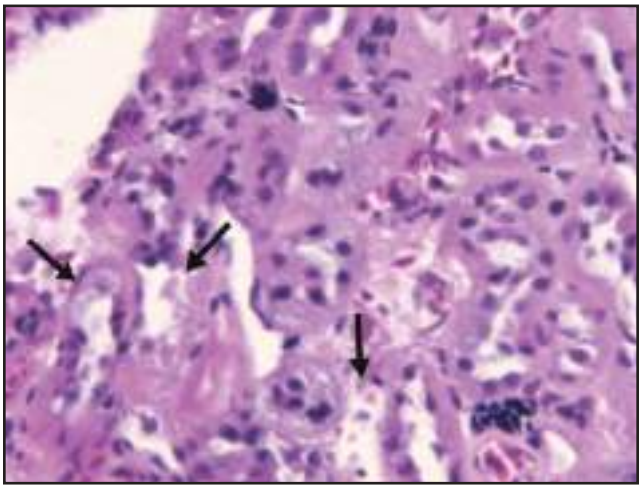


Fig 16.15 | A mitred conure (*Aratinga mitrata*) with mild nephrosis. Note the cellular disorganization and loss of tubular epithelial cell structure or degeneration (arrows).

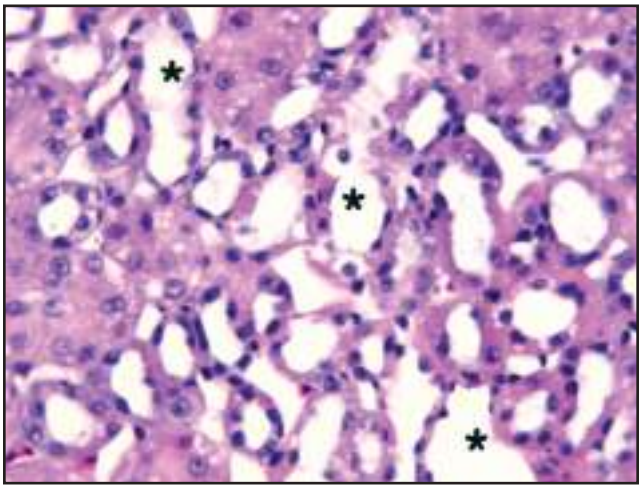


Fig 16.16 | An adult hyacinth macaw (*Anodorhynchus hyacinthinus*) with mild tubular dilatation 6 weeks post-treatment for histologically suspected bacterial nephritis. Note the multiple dilated renal tubules (*). Although there is no evidence of inflammation, tubular dilatation can be seen with bacterial infections and other diseases, and suggests that complete resolution has not been obtained.

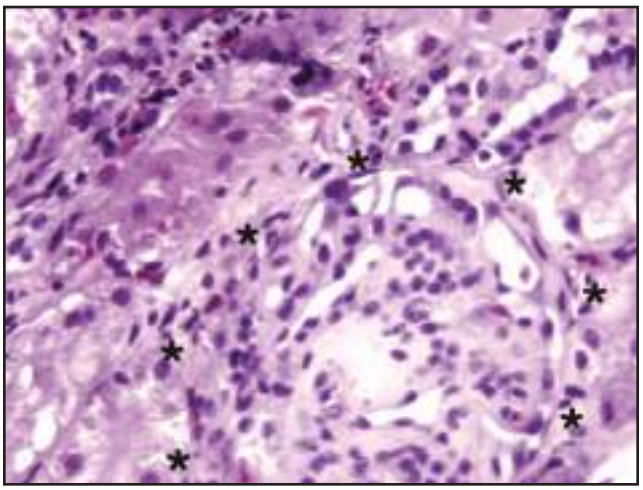


Fig 16.17 | A citron crested cockatoo (*Cacatua sulphurea* sp.) with membranous glomerulopathy of unknown etiology. Due to the significant mesangial enlargement, the mesangium has been pushed to the periphery of the glomerulus. The round glomerulus is outlined (*).

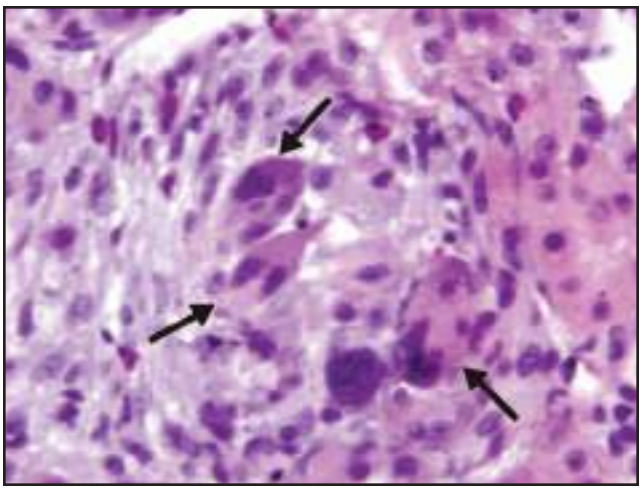


Fig 16.18 | An umbrella cockatoo (*Cacatua alba*) with granulomatous nephritis. Note the multinucleate giant cells within the renal interstitium (arrows).

formation of a viable therapeutic plan for the patient.

Treatment

THERAPEUTIC CONSIDERATIONS

Treatment options for renal disorders in birds depend upon the cause and type of kidney disease and secondary complications present. Most renal disease patients are medically managed, as kidney surgery is difficult and often not needed. Tables 16.4 and 16.5 list medications, and their possible indications, commonly used in renal disease patients.

Because of the location within the renal fossae, avian kidneys are difficult to surgically remove. The close associations with the lumbar and sacral plexuses and extensive vascular network surrounding the kidneys lead to the high probability of significant hemorrhage expected during surgery, and possible neurologic damage.[79] With that stated, focal therapeutic surgery (including endoscopic biopsy) for superficial renal lesions and the ureters may be useful in some cases. Given the concern of serious hemorrhage, most surgical renal disease cases are managed medically.

A few accounts of therapeutic renal surgery exist. Post-renal failure due to urolithiasis or some other obstruction of the ureters or cloaca may be noted. Cloacaliths and other masses within the cloaca may be easily removed, relieving a potential ureteral obstruction. Wideman and Laverty describe the effects of renal vein and ureter ligation on kidney function in domestic fowl.[250] Except for a small island of tissue adjacent to the testes and cranial renal artery, the cranial, and portions of the middle, renal divisions atrophied significantly without compromising overall kidney function.[250] Such a study is worth reviewing if considering renal division ablation or other similar radical procedures. Renal stones were successfully removed via extracorporeal shock wave lithotripsy in a Magellanic penguin (*Spheniscus magellanicus*).[146] Although multiple anesthetic procedures were required, ureteral stones were successfully removed from a 21-year-old male double-yellow headed Amazon parrot (*Amazona ochrocephala*).[56]

The author also has used minor surgery in articular gout cases. In effort to speed the removal of (stabilized) articular gout, make small incisions over the gouty lesions, which are often on the feet. Express the thick material out. Anesthesia is ideal as this can be quite painful. Also, this procedure tends to be bloody, and the feet often require minor bandaging to help prevent continued bleeding and secondary infection.

Another poorly explored area is renal cancer therapy. Clearly, as treatment options advance and are tested in avian species, renal cancer therapy will likely become more prevalent. For example, carboplatin at 5 mg/kg IV q 1 month was used to manage a renal adenocarcinoma (diagnosed at necropsy) in a budgerigar. The bird died approximately 3 months after initiating treatment, but temporarily did show improvement of clinical signs (decreased grip in one foot and lameness changed to almost normal perching, 1 month after starting therapy).[147] It was concluded that while carboplatin may be nephrotoxic in birds, this drug could possibly be useful in treating early renal tumors that have not progressed to renal failure.[147]

As a general note in any bird with organ dysfunction, patients should be monitored with routine physical and laboratory evaluation, especially when taking any medication(s) chronically. The intervals between recheck examinations will vary on the patient's condition and clinician's experience in handling the given case.

DIURESIS AND FLUID THERAPY

As in other animals with renal disease, maintaining hydration is important in birds with most kidney disorders. Acid-base and electrolyte disorders may likely be present in birds with renal disease. At this time, only general statements concerning diuresis and fluid therapy can be made.

Anuric and oliguric patients should be diuresed. Although mannitol and furosemide have been recommended to induce diuresis in birds, these drugs are poorly studied in avian species.[141,181] Mannitol (added to a solution containing inulin and para-amino hippuric acid) was used to induce diuresis in chickens at a dose of 2.5% given at a rate of 0.2 ml/kg per minute.[251] Furosemide given IV (1 mg/kg BID) along with SQ saline for 72 hours was used to successfully treat a red-tailed hawk (*Buteo jamaicensis*) with acute obstructive uric acid nephropathy.[141] Some birds, especially lories, may be sensitive to the effects of furosemide and its use should be judicious.[203] Furosemide also may cause increased urinary excretion of Na^+, K^+ and Cl^-.[248] If furosemide is used, electrolyte replacement may be needed. Clinically, providing parenteral fluids often induces diuresis in birds, even with most forms of renal disease.

Until acid-base and electrolyte disorders are better evaluated in birds with renal disease, balanced electrolyte solutions should be used to maintain hydration, replace fluid losses and/or induce diuresis as needed. The estimated daily fluid requirement for most birds is 40 to 60 ml/kg per day.[52,223] It has been recommended that 10% of the bird's body weight should be given in fluids when

Table 16.4 | Treatment Guide for Stable Avian Patients with Renal Disease

	Surgery	Fluid Therapy	Antibiotics	Allopurinol	Colchicine	Dietary Modification	Omega-3 Fatty Acids	Parenteral Vitamin A	Low-dose NSAIDs
Nondescript nephritis			+			+	+	+	+
Glomerulopathy							+++		++
Bacterial nephritis			+++				+		
Parasitic nephritis							+		
Nephrosis						++	++	+	
Fatty nephropathy						+++			
Neoplasia	+						++		
Urolithiasis	++					+	++	+	
Amyloidosis					+		+		
Renal fibrosis					+++		+		
Renal (visceral) gout		+++		+++	+++	+	++	+	
Articular gout	+	++		+++	+++	+	++	+	

Note: Most birds with visceral gout are likely in renal failure and usually require immediate medical attention. Fluid therapy, nutritional support and other appropriate supportive care may be required for any bird in poor condition, and treatment choices are based on the bird's health and attending clinician's experience.

NSAIDs = non-steroidal anti-inflammatory drugs
+ = occasionally indicated
++ = occasionally to often indicated
+++ = often indicated

Table 16.5 | Doses and Durations of Drugs Commonly Used in Psittacine Renal Disease Patients
(M.S. Echols, unpublished data)[37,78]

	Dose	Route	Duration	Potential Side Effects
Ceftazidime*	75-200 mg/kg BID-QID	IM, IV	4-6 weeks + for bacterial nephritis	—
Ceftiofur*	100 mg/kg TID	IM	4-6 weeks + for bacterial nephritis	—
Ciprofloxacin*	20-40 mg/kg BID	PO	4-6 weeks + for bacterial nephritis	—
Enrofloxacin*	10-30 mg/kg SID-BID	PO, IM	4-6 weeks + for bacterial nephritis	Muscle/tissue necrosis/irritation upon injection.
Piperacillin*	100-200 mg/kg BID-TID	IM, IV	6 weeks + for bacterial nephritis	—
TMP Sulfa*	16-100 mg/kg BID-TID	PO	6 weeks + for bacterial nephritis. Use lower dose for birds over 300 g.	May cause regurgitation. Use cautiously with dehydrated birds.
Allopurinol	10-30 mg/kg BID	PO	Use until hyperuricemia and/or physical signs of gout normalize. Use higher dose short-term (<4 weeks).	Renal toxicity noted in red-tailed hawks, but not psittacines.
Colchicine	0.04 mg/kg SID-BID	PO	Use until signs of hyperuricemia and/or histologic fibrosis normalize. Can be used with allopurinol and for 6-12 months.	—
Omega(Ω)-3 fatty acids	0.22 ml/kg of a supplement containing <6:1 (Ω-6:Ω-3 fatty acids)	PO	Use at least until laboratory and/or renal histologic abnormalities normalize. Can be given 6-12+ months.	—
Vitamin A	2000-5000 IU/kg once	IM	Use single dose in conjunction with diet modification. Repeat dose in 3 weeks if needed.	May lead to vitamin A toxicity if used chronically.
Aspirin	0.5-1.0 mg/kg SID-BID	PO	Use until evidence of glomerulopathy is gone or lab abnormalities have normalized. Can be given 6-12 months.	May lead to renal disease if overdosed. Do not use in dehydrated or moderate to severely compromised patients.

*Antibiotic choice should be based on culture and sensitivity (C&S) from histologically confirmed or suspected bacterial nephritis. Otherwise, base antibiotic choice on C&S results from a separate infected lesion, septicemic blood or cloacal cultures.

BID = twice daily
IM = intramuscular
IV = intravenously
PO = orally
SID = once daily
TID = three time daily
TMP = trimethoprim-sulfamethoxazole

For more complete dosing schedules in other species, see Chapter 9, Therapeutic Agents.

the patient is in renal failure.[141] Once a dose has been determined, warmed fluids are given with food (tube/syringe-fed), SQ, IV or IO. The IV and IO routes are most appropriate for critically ill patients.[223] While appropriate in many cases, subcutaneous fluids are not adequate to rehydrate patients with severe dehydration, shock or hypothermia.[223] Oral fluids are reserved for stable patients with mild dehydration that have normal gastrointestinal function, and are contraindicated in critically ill birds.[112]

Fluid therapy for critically ill birds should ideally be tailored to the bird's electrolyte status and/or overall condition and is ultimately decided by the attending clinician. The author typically diureses ill and severely hyperuricemic renal disease patients. While the definition is debatable, the author generally considers severe hyperuricemia to be present when one or more of the following conditions are met in clinically ill non-carnivorous and appropriately fasted carnivorous birds:

1. Uric acid levels exceed 30 mg/dl.

2. Uric acid levels are elevated (>10 mg/dl for most species) and rising over a period of several days (even if below 30 mg/dl).
3. There is evidence of rapidly progressive articular or visceral gout.

Depending on the patient's condition, the author will typically give 50 to 100 ml/kg of fluid BID via SQ, IV, IO or combination routes. Fluid therapy (combined with other medications if needed) is generally continued until the blood uric acid level drops to either normal or mildly elevated levels (10-20 mg/dl) and the bird is showing signs of improvement (eg, eating, more active). Lower amounts of parenteral fluids are given if overhydration is either suspected or a concern.

ANTIBIOTICS

Antibiotics are indicated in patients with known or suspected bacterial nephritis. Bacterial renal infections in birds may result from an ascending ureteritis, extension from local tissues (eg, peritonitis, oophoritis, salpingitis) and hematogenously.[187] Because of the renal portal system and possible shunting of blood from the intestines directly to the kidneys, alimentary tract organisms may contribute to kidney disease and should be considered when using antimicrobial therapy. Drug choices are based on an isolated renal organism (ie, identified during kidney biopsy sampling) or a suspected infectious agent (blood, ovarian, salpinx, or cloacal/fecal cultures and/or supportive histopathology). Clinical consideration regarding potential antimicrobial-induced toxicities is important.

The distribution, elimination and toxicities of many antimicrobials are poorly defined in most bird species, although an excellent review of antimicrobial use in birds with specific consideration toward the renal system is available.[78] Although mammalian literature warns of potential nephrotoxicity with amphotericin B, cephalosporin, fluoroquinolone, trimethoprim/sulfonamide and tetracycline use, only aminoglycosides have been consistently and definitively associated with renal disease in birds.[71,78,117,166] Those drugs with known potential nephrotoxicity should be cautiously used in birds with renal impairment. Until additional studies are completed in birds, antimicrobials that reach high concentrations in the renal tissue and urine without inducing toxicity should be chosen and cautiously used in kidney disease patients.

The ideal duration recommendable for treating renal infections has not been established in birds. In cats and dogs, greater than 4 to 6 weeks of antimicrobial use is generally recommended for treating bacterial kidney infections.[136] The author's clinical experience with bacterial nephritis suggests that response is best when a minimum of 6 weeks of antibiotic therapy is administered. These suggested guidelines are based on renal histopathologic evaluation supporting the presence of infectious nephritis, post-treatment resolution of clinical pathology abnormalities and improved follow-up kidney biopsy and histopathology in a small number of avian renal disease cases.[64,65] There are no controlled studies evaluating antibiotic therapy in active bacterial nephritis cases in birds.[64] Additionally, the author will generally treat concurrent colitis (based on culture and sensitivity results of fecal and/or cloacal cultures) for 5 to 7 days, or until signs abate, in renal disease patients.

MANAGING HYPERURICEMIA, RENAL FIBROSIS AND AMYLOIDOSIS

Allopurinol

Allopurinol's main action is to decrease uric acid production. Specifically, allopurinol inhibits xanthine oxidase, which is required to convert hypoxanthine to xanthine and subsequently to uric acid.[35,53,132,195] In chickens, xanthine dehydrogenase, closely related to xanthine oxidase, is the actual enzyme used in this pathway.[35,46,195] Allopurinol has been specifically shown to prevent renal synthesis of urates and allow the excretion of unchanged xanthine.[195] Regardless, both clinical and experimental data show decreased plasma/serum and/or urinary uric acid levels in birds treated with allopurinol.[46,53,68,132,215] Interestingly, allopurinol does not appear to affect pancreatic xanthine dehydrogenase activity, suggesting differing mechanisms of uric acid metabolism in the pancreas and kidney.[132]

Specifically in red-tailed hawks (*Buteo jamaicensis*), allopurinol has been shown to be toxic at 50 mg/kg PO SID with clinical signs of vomiting and laboratory-supported significant hyperuricemia and a renal function disorder.[145] The renal toxicity was even worse and included visceral gout when red-tailed hawks were given 100 mg/kg followed by 50 mg/kg of allopurinol. The toxic signs were attributed to oxypurinol, the active metabolite of allopurinol.[145] Allopurinol given at 25 mg/kg SID PO to red-tailed hawks was shown to be safe, but had no significant effect on plasma uric acid concentrations.[191c] The authors concluded that allopurinol has a very low therapeutic ratio, at best, in red-tailed hawks and that other means of controlling hyperuricemia, such as urate oxidase, in this species should be considered.[191c] With the exception of red-tailed hawks, allopurinol use is reported to be nontoxic in birds (in studied chickens), including chicks.[132,181,246] Although the long-term effects are not clear, allopurinol given to chickens increases oxidative activity by lowering plasma uric acid, an important avian antioxidant.[215]

The author uses allopurinol as a first-line drug to lower

uric acid when fluid therapy and diet modification alone are not sufficient or when hyperuricemia is severe. Clinical experiences suggest that allopurinol is safe to use at published doses in Psittaciformes and Columbiformes, even when used chronically (3-6+ months). Because of the noted toxicities in red-tailed hawks and until further studies are conducted, it is reasonable to assume that allopurinol should be used judiciously, if at all, in birds of prey.

Colchicine

Theoretically, colchicine can reduce serum uric acid levels in birds and be used to control hyperuricemia. In chicken livers, colchicine reversibly inhibits xanthine dehydrogenase (compared to a "pseudo-reversal" with allopurinol).[67,68] Colchicine prevents the progression of renal disease in humans with familial Mediterranean fever, a disease of recurring fever often complicated by amyloidosis.[179] In humans, colchicine is best known for its antigout activity.[191] In small animals, colchicine blocks the synthesis and secretion of serum amyloid A, and decreases the formation and increases the breakdown of collagen. For these reasons, colchicine has been used to treat amyloidosis and hepatic fibrosis, respectively.[191]

Clinical use of colchicine suggests possible benefit in reducing hyperuricemia in birds with renal disease.[64,65] The author also has used colchicine to reduce renal (and hepatic) fibrosis in birds, and has had good success based on pre- and post-treatment tissue biopsies (M.S. Echols, unpublished data). As such, the author uses colchicine as a second-line drug to reduce hyperuricemia and a primary medication for histologically confirmed tissue fibrosis. Allopurinol and colchicine are well tolerated when given together in most birds. If diagnosed antemortem, colchicine may be used in birds with amyloidosis. No controlled studies were found using colchicine in birds with renal disease.

Urate Oxidase

Urate oxidase also has been recently discussed as an alternative method to manage hyperuricemia in birds.[191b] At least in humans, urate oxidase is reported to degrade the excess of uric acid to allantoin, which the kidneys can clear more easily than uric acid. Urate oxidase also is very specific for urates and uric acid and does not interfere with the metabolism of purines as does allopurinol. In one study, urate oxidase was given (200 and 600 U/kg and 100 and 200 U/kg IM) to pigeons and red-tailed hawks, respectively. When compared to controls, all dosing regimens caused a significant decrease in plasma uric acid concentrations within 2 days of the first dose. The authors concluded that "urate oxidase is much more effective compared with allopurinol," but this promising drug needs further evaluation to better understand its use and potential long-term effects.[191b]

DIETARY MODIFICATION

As a general note, birds should be fed diets appropriate for their species. Supportive dietary therapy should always be considered in any anorectic patient. As is true with all sick birds, renal disease patients should be weighed routinely at regular intervals and monitored for weight loss.

Protein

The question of dietary protein restriction in the face of renal disease remains controversial. The current human and veterinary literature cites arguments for and against both restriction and supplementation of protein with renal disease patients.[94,95,137,239] The current human literature cites malnutrition (potentially from protein-restricted diets) as the most potent predictor of death in end-stage renal failure.[179] The resultant recommendation is that patients on protein-restricted diets should be well supervised and provided adequate calories.[179]

Although feeding 20% protein to chicks, including young cockatiels, has been recommended as a general level for normal development, excessive protein intake for birds with renal disease has not been determined.[207] Feeding diets consisting of 60 and 80% protein (2 separate studies) were required to induce articular gout in genetically predisposed chickens.[9] In a study using adult cockatiels, birds fed up to 70% protein for 11 months had no evidence of visceral or articular gout or significant renal lesions. This led the authors to the conclusion that, in cockatiels, high dietary protein levels are not associated with kidney dysfunction.[123] These experimental diets represent unnaturally high protein levels and do not serve as a realistic evaluation of the effect of diet on renal disease and/or gout in birds.

The management of hypoproteinemia also may be important in birds with renal disease. As mentioned under Part 2: Electrophoresis, Plasma Protein Electrophoresis, the identification of hypoproteinemia and association with renal disease in birds is unclear.

Until further research better defines the role of dietary protein needs in relation to renal disease, avian kidney disease patients should be fed a well-balanced diet appropriate for their respective species. If instituted, birds fed protein-restricted diets should be carefully monitored. No current studies evaluate the effect of low or high-protein diets in birds with naturally occurring renal disease were available at the time of writing. A safe recommendation is that birds with hyperuricemia and/or gout should not consume diets with protein levels greater

than what is considered normal for the given species.

NUTRITIONAL SUPPLEMENTATION

Treatment: Omega-3 Fatty Acids

Omega-3 fatty acids (*n*-3 FA) have gained popularity for their anti-inflammatory, lipid-stabilizing and antineoplastic effects, renal protective properties and other potential qualities.[12,190] The *n*-3 FA are polyunsaturated and are designated by their first carbon-carbon double bond occurring at the third carbon from the methyl group.[190] The *n*-3 FA are those rich in eicosapentaenoic (EPA), docosahexaenoic (DHA) and/or linolenic acid.[4,31] Flax seed and menhaden (cold-water plankton-feeding fish) oils contain predominately linolenic acid, and EPA and DHA, respectively, and therefore have different *n*-3 FA compositions.[4] DHA and EPA are more readily incorporated into biological tissues, but also carry greater potential to create metabolic oxidative stress than linolenic acid.[4] The clinical impact of the differences of the various *n*-3 FA has not been clearly defined.

Studies evaluating *n*-3 FA in mammals serve as the basis for potential treatment value in birds with selected renal disease. At this time, only anecdotal information exists regarding use of *n*-3 FA in birds with renal disease.

In mammals, *n*-3 FA can significantly reduce thromboxane A_2 (TXA) synthesis in platelets and glomerular cells, and increase production of vasodilatory prostaglandins.[95] *n*-3 FA partially substitute EPA and DHA for arachidonic acid in membrane phospholipid.[31,104,159] This pathway decreases the release of arachidonic acid and, subsequently, the cyclooxygenase-mediated synthesis of TXA.[31,95,190] In contrast, most animals readily convert omega-6 fatty acids (*n*-6 FA) to arachidonic acid and, subsequently, eicosanoids (prostaglandins, TXA).[31] As with arachidonic acid, EPA also serves as a substrate for the formation of vasodilatory prostaglandin/cyclins (PGI/PGE) and their respective products (PGI_2 /PGE_2 and PGI_3 /PGE_3), all of which have similar biologic potency.[95,190] These vasodilatory prostaglandin/cyclins increase renal blood flow and single nephron GFR.[31,190]

In humans and rats supplemented with *n*-3 FA for at least 4 to 6 weeks, single nephron GFR, plasma flow and renal blood flow increased and/or decreased renal vascular resistance occurred.[95] In a separate evaluation, dogs on a low-fat diet supplemented with *n*-3 FA had preserved renal function and structure when induced with renal disease.[31] Another study found that *n*-3 FA supplementation reduced glomerular capillary pressure and prevented deterioration of GFR in dogs with renal disease.[32] Compared with controls and thromboxane synthetase inhibitor-treated dogs, beagles supplemented with *n*-3 FA demonstrated increased renal production and excretion of PGE_2 and PGE_3, which was believed to have stabilized renal tubular lysosomal membranes.[95] These *n*-3 FA-supplemented dogs had decreased gentamicin-induced proximal tubular necrosis when compared to controls.[95]

Specific toxicities associated with *n*-3 FA supplementation are poorly described, but some potential adverse effects may occur. Chickens fed diets high in *n*-3 FA had reduced plasma and tissue vitamin E (the body's primary antioxidant) and plasma carotenoid levels due to lipid peroxidation.[4,5,230] Therefore, supplementing the diet with *n*-3 FA increases the requirements for dietary vitamin E.[4,45] As supported by clinical investigations, vitamin E supplementation should be considered with use of *n*-3 FA or any other polyunsaturated fatty acids.[182,230] Specifically, 160 mg/kg of vitamin E (dl-α-tocopherol acetate) was shown to prevent loss of α-tocopherol in tissues, and normalize or increase resistance to lipid peroxidation in chickens fed a commercial diet supplemented with 3% tuna oil (*n*-3 FA).[230]

Other potential side effects may be noted with *n*-3 FA supplementation in birds. Menhaden oil supplementation in laying chickens has been shown to contribute to hepatic lipidosis, likely via enhancing the lipogenic activity (along with estradiol) of the liver.[240] This study cautions the use of *n*-3 FA in reproductively active hens. In another study, chickens fed diets high in *n*-3 FA had no alteration in primary or secondary humoral response, but experienced a 50% reduction in antibody-dependent cell cytotoxicity (ADCC).[80] The concern presented therein was that reduction in ADCC-related immune functions might increase a patient's susceptibility to certain disease (Marek's).[80] The *n*-3 FA supplementation also may affect the ability of antigen-presenting cells to present antigen, again suggesting the potential for immune system alteration.[103] An increased incidence of infectious disease in birds has not definitively been associated with *n*-3 FA supplementation.

Although specific doses have not been established, some believe that the appropriate *n*-6 to *n*-3 FA ratio is more important to inhibiting eicosanoid synthesis from arachidonic acid than is the absolute amount of *n*-3 FA.[95] A dietary *n*-6 FA:*n*-3 FA ranging from 5:1 to 15:1 has been proposed as desirable for dogs and cats with renal disease.[31] Using the above dietary guideline, 2 to 4 weeks are required to see any initial effects of the dietary change in dogs and cats.[31] One study in chickens showed that maximal *n*-3 FA tissue (egg yolk) levels were obtained after 3 to 4 weeks of supplementation.[240] Long-term supplementation (3 to 6 months or more) is likely appropriate if *n*-3 FA are to be used.

The author has successfully used supplements containing

n-6 FA:*n*-3 FA of 4-5:1 to 1:3 combined with low-dose aspirin (0.5-1.0 mg/kg PO q 12 h) to manage histologically confirmed glomerulopathies in avian patients (M.S. Echols, unpublished data). Success was gauged on normalized hyperuricemia (4/4), improved clinical appearance (3/4) and repeat renal biopsy showing normal glomerular light microscopic histology (1/1) in an African grey parrot (*Psittacus erithacus erithacus*), citron-crested cockatoo (*Cacatua sulphurea citrinocristata*), red-lored Amazon parrot (*Amazona autumnalis*) and a ring-neck dove (*Streptopelia risoria*) (M.S. Echols, unpublished data). The author also has used a supplement[e] containing *n*-6 FA:*n*-3 FA of 1:3 (0.22 ml/kg body weight, PO, SID) alone to manage various forms of renal disease in mixed avian species with no recognized adverse side effects. Unfortunately, no clinical trials using fatty acids in avian renal disease were found, only anecdotal reports such as noted here.

Vitamin A

Parenteral vitamin A has been recommended in birds with renal disease.[141] Hypovitaminosis A is a reported cause of renal failure and results from metaplasia of the ureters leading to hyperkeratinization, decreased mucin production and impaction.[116,214] Vitamin A deficiency is discussed in more detail in Chapter 4, Nutritional Considerations. In birds with suspected hypovitaminosis A and renal disease, appropriate diet modification and short-term parenteral vitamin A are logical components of therapy. In such situations, the author gives a single IM vitamin A injection at the beginning of the therapy and recommends correcting the patient's diet to improve long-term nutritional status. The diet must be evaluated and the potential of hypervitaminosis A must be ruled out prior to parenteral vitamin A administration. See Chapter 4, Nutritional Considerations: Section II, Nutritional Disorders for more about hypervitaminosis A.

NON-STEROIDAL ANTI-INFLAMMATORY DRUGS

Non-steroidal anti-inflammatory drugs (NSAIDs) are frequently discussed for use in human and animal renal disease patients.[92,94,95,135,179] In general, NSAIDs such as aspirin and ibuprofen are non-specific cyclooxygenase inhibitors. Low doses of aspirin may actually inhibit platelet cyclooxygenase production, but allow beneficial (vasodilatory) prostacyclin formation and may be safe.[94] Consequently, low-dose aspirin therapy has been suggested to reduce platelet aggregation and subsequent thromboembolism, and to minimize glomerular inflammation for mammalian patients with some glomerulopathies.[94,137] More specific NSAIDs such as thromboxane synthetase inhibitors have been shown to attenuate renal dysfunction/damage as noted by one or more of the following: decreased proteinuria, enzymuria and tubular necrosis, and preserved renal blood flow and GFR in various animals with a variety of renal diseases.[92,94,95,135] Unfortunately, the beneficial effects of low-dose or specific NSAID therapy have not been studied in birds with renal disease.

Although there are limited avian studies, most NSAIDs are eliminated by renal clearance and should be used with caution, as they have been associated with a variety of renal lesions in birds and mammals.[6,120,137,179,196] Flunixin meglumine[d]-induced glomerular lesions in bobwhite quail (*Colinus virginianus*) that increased in severity proportionally with the dose. In this short study, no biochemical or electrolyte parameters were altered, but uric acid was not measured.[120] Aspirin has been associated with significant inhibition of prostaglandin synthesis (specifically prostaglandin $F_{2\alpha}$) in Japanese quail.[164] In this same experiment, aspirin was shown to induce liver enlargement resulting from hepatic lipid accumulation in *n*-6 FA-deficient Japanese quail.[164] Acetylsalicylic acid (aspirin) injected IV into Pekin ducks induced temporary diuresis lasting 30 minutes, which is in contrast to the antidiuretic effect seen in mammals, and had no effect on GFR or peripheral blood pressure.[97] Several *Gyps* spp. of vultures have died with renal failure and gout as a direct result of consuming diclofenac-treated livestock.[175b] The veterinary use of diclofenac has been specifically implicated in the decline of the critically endangered Oriental white-backed vulture (*Gyps bengalensis*) in Pakistan.[175b] These scattered studies serve only to point out potential varied effects of NSAIDs in birds.

Even with the noted toxicities and lack of therapeutic studies in birds, the author feels that low-dose aspirin, and possibly other NSAIDs, use can be beneficial in avian kidney disease patients. In the author's experience, low-dose aspirin (0.5-1.0 mg/kg PO q 12 h) combined with *n*-3 FA supplementation is safe and may be effective at reducing the severity of some forms of avian renal disease, especially glomerular disorders (M.S. Echols, unpublished data). Aspirin (and *n*-3 FA) therapy can be used chronically, and the author discontinues use once evidence of renal disease is gone or the disorder is satisfactorily managed.

TREATMENT SUMMARY

Treatments of avian renal disease should be individualized according to the patient's needs, accurate renal histologic diagnosis (if available), concurrent disorders and client considerations. Identified parasites are treated appropriately. If ova are identified in the urine, consider whether or not the eggs were actually released in the

intestines. Treatment of bacterial nephritis with appropriate antibiotics should be based, in part, on culture and sensitivity results when available. Otherwise, suspected bacterial-induced nephritis should be treated with broad-spectrum bacteriocidal antibiotics that reach high kidney concentrations and which are non-nephrotoxic. Antibacterials also should be considered when concurrent colitis is present. Removing known nephrotoxins and addressing secondary complications may best manage nephrosis. Such secondary complication of any renal disease may include dehydration, hyperuricemia, fibrosis, infectious diseases and anorexia. Dietary-induced renal diseases can be managed with diet change or supplementation, depending on the etiology. Antineoplastic treatment of certain avian renal tumors may be indicated and should be considered. Specifically identifying and managing underlying diseases that may be concurrently present may best control glomerulopathies. Confirmed glomerular disorders in birds without an obvious underlying disease may be managed in some cases with low-dose aspirin and *N*-3 FA supplementation. Nutritional management such as weight loss, providing a balanced diet and vitamin A supplementation also may be indicated.

Table 16.6 represents a quick treatment summary of some of the more common renal disease classifications.

Table 16.6 | Avian Renal Disease Treatment Summary

- **Nephrosis:** Parenteral vitamin A. Remove exposure to toxins if known. Consider *n*-3 FA supplementation.
- **Glomerulopathy:** If identifiable, remove/control any source of infection/inflammation. Give *n*-3 FA and low-dose aspirin until all signs of renal disease (hyperuricemia, histologic changes, etc) are gone. *n*-3 FA can be given chronically if needed.
- **Bacterial Nephritis:** Antibiotics for a minimum of 4 to 6 weeks.
- **"Diet-induced Renal Disease of Color Variety Psittacine Birds":** Discontinue pellets and change diet over to whole grains, seeds, fruits and vegetables as is appropriate for the species. If after 3 to 6 months all signs of renal disease are gone, pellets (<50% of total diet) can be cautiously added to the diet.
- **Renal Fibrosis:** Use colchicine until histologic fibrosis resolves. Otherwise, use colchicine for 6 to 12 months or until laboratory abnormalities normalize. The *n*-3 FA also may be beneficial.
- **Articular Gout:** Use colchicine and allopurinol together until all signs of gout and hyperuricemia have resolved. Consider diagnosing the cause of probable underlying renal disease and manage appropriately. Give vitamin A if hypovitaminosis A is suspected. Articular gout lesions also may be surgically opened and expressed to speed removal of uric acid crystal accumulation. *n*-3 FA may be beneficial. Use aggressive fluid therapy if articular or visceral gout is accumulating rapidly. See Chapter 4, Nutritional Considerations: Section 2, Nutritional Disorders.

Articular gout, although not a renal disease, also is included. With the possible exception of "diet-induced renal disease of color variety psittacine birds," the patient's diet should be modified as is appropriate for that avian species. Secondary infections, dehydration, unacceptable weight loss, etc, should be managed as needed. Combination therapy should be considered when two or more histologic renal lesions are present.

Prognosis

The World Health Organization classification of renal disease is based on distinct glomerular pathological findings and is used for prognosis, treatment and outcome.[109] Presently, no such classification system exists in avian medicine. In fact, there are limited studies that estimate the outcome of selected avian renal disorders. One such review noted that most birds live less than 3 months following a diagnosis of a renal neoplasm.[79] This may seem to offer a poor prognosis, but represents only one form of renal disease that is usually diagnosed late and with which there are few treatment options. Based on the author's experience, several forms of renal disease can be successfully managed and some resolved, giving a good prognosis for long-term health to the individual patient.

Clinicians are encouraged to thoroughly evaluate each avian renal disease patient individually from diagnosis through to management or completion of treatment. Consider renal biopsy as a viable tool for diagnosing and managing disease. Dr. Robert Schmidt states, "The problem is that clinical lab tests may indicate renal disease in birds, but several kidney disorders cause similar (lab) abnormalities. If you want a definitive diagnosis, biopsy the kidney" (R. Schmidt, personal communication, 2003). Treatment completion may have to be defined, in some cases, as return to normal renal histology by follow-up biopsy. Until renal diseases of birds are better understood, classified and treated, the short- and long-term prognoses can be estimated based only on the severity of kidney lesions at that time and secondary disorders of the patient.

Products Mentioned in the Text

a. Combur-9 Stix, Boehringer Mannheim www.burnsvet.com/home/default.asp

b. Renografin-76, Squib Diagnostics, Princeton, NJ

c. Optomega, USANA Health Sciences, Salt Lake City, UT, www.unitoday.net/USPSupplements

d. Banamine, Schering-Plough Animal Health, www.spah.com

References and Suggested Reading

1. Abbassi H, et al: Renal cryptosporidiosis (*Cryptosporidium baileyi*) in specific-pathogen-free chickens experimentally coinfected with Marek's disease virus. Avian Dis 43:738-744, 1999.
2. Abdul-Aziz m Hasan SY: Hydropericardium syndrome in broiler chickens: its contagious nature and pathology. Res Vet Sci 59:219-221, 1995.
3. Afifi NA, Ramadan A: Kinetic disposition, systemic bioavailability and tissue distribution of apramycin in broiler chickens. Res Vet Sci 62:249-252, 1997.
4. Allen PC, Danforth HD: Effects of dietary supplementation with *n*-3 fatty acid ethyl esters on coccidiosis in chickens. Poultry Sci 77:1631-1635, 1998.
5. Allen PC, Danforth HD, Levander OA: Diets high in *n*-3 fatty acids reduce cecal lesion scores in chickens infected with Eimeria tenella. Poult Sci 75:179-185, 1996.
6. Ambrus JL, Sridhar NR: Immunologic aspects of renal disease. JAMA 278:1938-1945, 1997.
7. Anadón A, et al: Pharmacokinetics and residues of ciprofloxacin and its metabolites in broiler chickens. Res Vet Sci 71:101-109, 2001.
8. Angel R, Ballam G: Dietary protein effect on parakeet plasma uric acid, reproduction, and growth. Proc Annu Conf Assoc Avian Vet, 1995, pp 27-32.
9. Austic RE, Cole RK: Impaired renal clearance of uric acid in chickens having hyperuricemia and articular gout. Amer J Physiol 223:525-530, 1972.
10. Bailey TA, et al: Lead toxicosis in captive houbara bustards (*Chlamydotis undulata maqueenii*). Vet Rec 137:193-194, 1995.
11. Barton JT, et al: Avian paramyxovirus type 1 infections in racing pigeons in California. I. Clinical signs, pathology, and serology. Avian Dis 36:463-468, 1992.
12. Bauer JE, et al: Effects of dietary fat and polyunsaturated fatty acids in dogs with naturally developing chronic renal failure. JAVMA 215(11):1588-1591, 1999.
13. Benador D, et al: Are younger children at highest risk of renal sequelae after pyelonephritis? The Lancet 349:17-19, 1997.
14. Bennett DC, et al: Suspected sodium toxicity in hand-reared great blue heron (*Ardea herodia*) chicks. Avian Dis 36:743-748, 1992.
15. Bennett DC, et al: Effect of cadmium on Pekin duck total body water, water flux, renal filtration, and salt gland function. J Tox Environ Health Part A 59:43-56, 2000.
16. Bermudez AJ, Hopkins BA: Hemoglobinuric nephrosis in a rhea (*Rbea americana*). Avian Dis 39:661-665, 1995.
17. Bermudez AJ, et al: Heritability and biochemistry of gangliosidosis in emus (*Dromaius novaehollandiae*). Avian Dis 41:838-849, 1997.
18. Biancifiori F, Rondini C, Grelloni V: Avian toxoplasmosis: experimental infection of chicken and pigeon. Comp Immun Microbiol Infect Dis 9(4):337-346, 1986.
19. Blagburn BL, et al: Cryptosporidium sp. infection in the proventriculus of an Australian diamond firetail finch (*Stagnopleura bella*: Passeriformes, Estrilididae). Avian Dis 34:1027-1030, 1990.
20. Blaxland JD, et al: An investigation of urolithiasis in two flocks of laying fowls. Avian Pathol 9:5-19, 1980.
21. Bolton WK, Tucker FL, Sturgill BC: Experimental autoimmune glomerulonephritis in chickens. J Clin Lab Immunol 3:179-184, 1980.
22. Bolton WK, Tucker FL, Sturgill BC: New avian model of experimental glomerulonephritis consistent with mediation by cellular immunity. Nonhumorally mediated glomerulonephritis in chickens. J Clin Invest 73:1263-1276, 1984.
23. Bottje WG, et al: Relationships between renal hemodynamics and plasma levels of arginine vasotocin and mesotocin during hemorrhage in the domestic fowl (*Gallus domesticus*). Comp Biochem Physiol 92A(3):423-427, 1989.
24. Boulianne M, et al: Effect of exercise on the cardiovascular and respiratory systems of heavy turkeys and relevance to sudden death syndrome. Avian Dis 37:83-97, 1993.
25. Bounous DI, Schaeffer DO, Roy A: Coagulase-negative Staphylococcus sp. septicemia in a lovebird. JAVMA 195:1120-1122, 1989.
26. Bourgault A, Drolet R: Spontaneous glomerulonephritis in swine. J Vet Diagn Invest 7:122-126, 1995.
27. Braun EJ: Comparative renal function in reptiles, birds and mammals. Sem Avian Exot Pet Med 7(2):62-71, 1998.
28. Braun EJ: Integration of renal and gastrointestinal function. J Exp Zool 283:495-499, 1999.
29. Brayton C: Amyloidosis, hemochromatosis, and atherosclerosis in a roseate flamingo (*Phoenicopterus ruber*). Ann NY Acad Sci 653:184-190, 1992.
30. Brown CS, et al: Lead poisoning in captive gentoo penguins (*Pygoscelis papua papua*). Proc Annu Conf Amer Assoc Zoo Vet, 1996, pp 298-301.
31. Brown SA, et al: Does modifying dietary lipids influence the progression of renal failure? Vet Clin North Amer Small Anim Pract 26:1277-1285, 1996.
32. Brown SA, Finco DR, Brown CA: Is there a role for dietary polyunsaturated fatty acid supplementation in canine renal disease? J Nutr 128:2765S-2767S, 1998.
33. Brummermann M, Braun EJ: Renal responses of roosters with diabetes insipidus to infusions of arginine vasotocin. Amer J Physiol 269:R57-R63, 1995.
34. Buggiania SS, Rindi S: Lead toxicosis and salt glands in domestic ducks. Bull Environ Contam Toxicol 24:152-155, 1980.
35. Cacini W: Comparative accumulation of uric acid and hypoxanthine by slices of avian renal cortex. J Pharm Exp Therapeutics 220:86-90, 1982.
36. Canny C: Gross anatomy and imaging of the avian and reptilian urinary system. Sem Avian Exot Pet Med 7(2):72-80, 1998.
37. Carpenter JW, Mashima TY, Rupiper: Birds: *In* Carpenter JW, Mashima TY, Rupiper (eds): Exotic Animal Formulary 2nd ed. Philadelphia, WB Saunders, 2001, pp 108-249.
38. Casotti G, Braun EJ: Renal anatomy in sparrows from different environments. J Morphol 243: 283-291, 2000.
39. Casotti G: Effects of season on kidney morphology in house sparrows. J Exp Biol 204: 1201-1206, 2001.
40. Casotti G, Lindberg KK, Braun EJ: Functional morphology of the avian medullary cone. Amer J Physiol Reg Integ Comp Physiol 279:R1722-R1730, 2000.
41. Chandra M: Hematologic changes in nephritis in poultry induced by diets high in protein, high in calcium, containing urea, or deficient in vitamin A. Poultry Sci 63:710-716, 1984.
42. Chandra M, et al: Clinicopathological, hematological, and biochemical studies in some outbreaks of nephritis in poultry. Avian Dis 29:590-600, 1985.
43. Cheatum EL: *Dendritobilharzia anatinarum n.* sp., a blood fluke from the mallard. J Parasit 27:165-170, 1941.
44. Cheng A-C, et al: Pathologic and pathological characteristics of new type gosling viral enteritis first observed in China. World J Gastroenterol 7(5):678-684, 2001.
45. Cherian G, Sim JS: Egg yolk polyunsaturated fatty acids and vitamin E content alters the tocopherol status of hatched chicks. Poultry Sci 76:1753-1759, 1997.
46. Chin TY, Quebbemann: Quantitation of renal uric acid synthesis in the chicken. Amer J Physiol 3:F446-F451, 1978.
47. Clubb, SL: Endoscopic renal biopsy. J Avian Med Surg 11:273-276, 1997.
48. Cooper JE, Forbes NA: Studies on morbidity and mortality in the merlin (*Falco columbarius*). Vet Rec 118:232-235, 1986.
49. Costigan MG, et al: Origin and significance of urinary N-acetyl-ß,D-glucosaminidase (NAG) in renal patients with proteinuria. Clin Chimica Acta 255:133-144, 1996.
50. Cowen BS, Wideman RF, Rothenbacher H: An outbreak of avian urolithiasis on a large commercial egg farm. Avian Dis 31: 392-397, 1987.
51. Cray C, Tatum LM: Applications of protein electrophoresis in avian diagnostics. J Avian Med Surg 12:4-10, 1998.
52. Curro TG: Anesthesia of pet birds. Sem Avian Exot Pet Med 7(1):10-21, 1998.
53. Czarnecki CM, Olivero DK, McVey AS: Plasma uric acid levels in ethanol-fed turkey poults treated with allopurinol. Comp Biochem Physiol 86C:63-65, 1987.
54. Dambach DM, et al: Morphologic, immunohistochemical, and ultrastructural characterization of a distinctive renal lesion in dogs putatively associated with Borrelia burgdorferi infection: 49 cases (1987-1992). Vet Pathol 34:85-96, 1997.
55. Degernes LA: Toxicities in waterfowl. Sem Avian Exotic Pet Med 4(1):15-22, 1995.
56. Dennis PM, Bennett RA: Ureterotomy for removal of two ureteroliths in a parrot. JAVMA 217(6):865-868, 2000.
57. Díaz-Ruiz C, Asbert M, Pérez-Tomás R: Immunochemical study of a transforming growth factor-α-related protein in the chicken kidney. Kidney International 49:1053-1063, 1996.
58. Dorrestein GM: Bacteriology. *In* Altman RB, et al (eds): Avian medicine and surgery. Philadelphia, WB Saunders, 1997, pp 255-280.
59. Dubbeldam JL: Systema nervosum periphericum. *In* Baumel JJ, King AS, Breazile JE, et al (eds): Handbook of Avian Anatomy: Nomina Anatomica Avium 2nd ed. Cambridge, MA, Nuttall Ornithological Club, 1993, pp 555-584.
60. Dudas PL, Renfro JL: Assessment of tissue-level kidney functions with primary cultures. Comp Biochem Physiol A 128:199-206, 2001.
61. Dudas PL, et al: Regulation of transepithelial phosphate transport by PTH in chicken proximal tubule epithelium. Amer J Physiol Reg Integ Comp Physiol 282(1):R139-R146, 2002.
62. Dunn MJ, Hood VL: Prostaglandins and the kidney. Amer J Physiol 233:169-184, 1977.
63. Dwivedi P, Burns RB: Pathology of ochratoxicosis A in young broiler chicks. Res Vet Sci 36:92-103, 1984.
64. Echols MS: Antemortem diagnosis and management of avian renal disease. Proc Annu Conf Assoc Avian Vet, 1998, pp 83-90.
65. Echols MS: Avian renal disease part II of II: Management. Proc Annu Conf Mid Atlantic States Assoc Avian Vet, 1999, pp 99-104.
66. Elaroussi MA, et al: Adaptation of the kidney during reproduction: role of estrogen in the regulation of responsiveness to parathyroid hormone. Poult Sci 72:1548-1556, 1993.
67. Escarmis C, Bozal J, Calvet F: Influencia de la colchicina y del alopurinol sobre la uricogenesis. I. Cinetica de sus acciones inhibidoras de la xantindeshidrogenasa hepatica. Revista Espanola De Fisiologia 26:109-120, 1970.
68. Escarmis C, Bozal J, Calvet F: Influencia de la colchicina y del alopurinol sobre la uricogenesis. II. Efectos de su administracion a polluelos y ratones. Revista Espanola De Fisiologia 26:121-130, 1970.
69. Evans RH: *Baylisascaris procyonis* (Nematoda: Ascarididae) larva migrans in free-ranging wildlife in Orange County, California. J Parasitol 88(2):299-301, 2002.
70. Feng JQ, Clark NB: Renal responses to parathyroid hormone in young chickens. Amer J Physiol 267:R295-R302, 1994.
71. Flammer K, et al: Adverse effects of gentamicin in scarlet macaws and galahs. AJVR 51:404-407, 1990.
72. Forbes NA, Cooper JE: Fatty liver-

kidney syndrome of merlins. *In* Redig PT, Cooper JE, Remple D, Hunter DB (eds): Raptor Biomedicine. Minneapolis, MN, University of Minnesota Press, 1993, pp 45-48.
73. Forman MF, Wideman RF: Renal responses of normal and preascitic broilers to systemic hypotension induced by unilateral pulmonary artery occlusion. Poultry Sci 78:1773-1785, 1999.
74. Forman MF, Beck MM, Kachman SD: N-acetyl-ß-D-glucosaminidase as a marker of renal damage in hens. Poultry Sci 75:1563-1568, 1996.
75. Frank RK, Newman J, Ruth GR: Lesions of perirenal hemorrhage syndrome in growing turkeys. Avian Dis 35:523-534, 1991.
76. Franson JC, Derksen DV: Renal coccidiosis in oldsquaws (*Clangula hyemalis*) from Alaska. J Wildl Dis 17(2):237-239, 1981.
77. Frazier DL, Jones MP, Orosz SE: Pharmacokinetic considerations of the renal system in birds: part I. Anatomic and physiologic principles of allometric scaling. J Avian Med Surg 9:92-103, 1995.
78. Frazier DL, Jones MP, Orosz SE: Pharmacokinetic considerations of the renal system in birds: part II. Review of drugs excreted by renal pathways. J Avian Med Surg 9:104-121, 1995.
79. Freeman KP, et al: Right leg muscle atrophy and osteopenia caused by renal adenocarcinoma in a cockatiel (*Melopsittacus undulatus*). Vet Radiol Ultrasound 40(2):144-147, 1999.
80. Fritsche KL, Cassity NA: Dietary *n*-3 fatty acids reduce antibody-dependent cell cytotoxicity and alter eicosanoid release by chicken immune cells. Poult Sci 71:1646-1657, 1992.
81. Fudge AM: Avian clinical pathology- hematology and chemistry: *In* Altman RB, et al (eds): Avian medicine and surgery. Philadelphia, WB Saunders, 1997, pp 142-157.
82. Gajadhar AA, Leighton FA: *Eimeria wobeseri* sp. n. and *Eimeria goelandi* sp. n. (Protozoa: Apicomplexa) in the kidneys of herring gulls (*Larus argentatus*). J Wildl Dis 24(3):538-546, 1988.
83. Gajadhar AA, Cawthorn RJ, Rainnie DJ: Experimental studies on the life cycle of a renal coccidium of lesser snow geese (*Anser c. caerulescens*). Can J Zool 60:2085-2092, 1982.
84. Gajadhar AA, et al: Prevalence of renal coccidia in wild waterfowl in Saskatchewan. Can J Zool 61:2631-2633, 1983.
85. Gardiner CH, Imes GD: Cryptosporidium sp. in the kidneys of a black-throated finch. JAVMA 185(11):1401-1402, 1984.
86. Gerlach H, et al: Membranous glomerulopathy as an indicator of avian polyomavirus infection in Psittaciformes. J Avian Med Surg 12:248-254, 1998.
87. Gevaert D, Nelis J, Verhaeghe B: Plasma chemistry and urine analysis in Salmonella-induced polyuria in racing pigeons (*Columbia livia*). Avian Pathol 20:379-386, 1991.
88. Giladi I, et al: Renal function and plasma levels of arginine vasotocin during free flight in pigeons. J Exp Biol 200:3203-3211, 1997.
89. Goecke CS, Goldstein DL: Renal glomerular and tubular effects of antidiuretic hormone and two antidiuretic analogues in house sparrows (*Passer domesticus*). Physiol Zool 70(3):283-291, 1997.
90. Goldstein DL, Skadhauge E: Renal and extrarenal regulation of body fluid composition. *In* Whittow GC (ed): Sturkie's Avian Physiology 5th ed. San Diego, Academic Press, 2000, pp 265-297.
91. Grauer GF, et al: Effects of a specific thromboxane synthetase inhibitor on development of experimental Dirofilaria immitis immune complex glomerulonephritis in the dog. J Vet Internal Med 2:192-200, 1998.
92. Grauer GF, et al: Effects of a thromboxane synthetase inhibitor on established immune complex glomerulonephritis in dogs. Amer J Vet Res 53:808-813, 1992.
93. Grauer GF, et al: Treatment of membranoproliferative glomerulonephritis and nephrotic syndrome in a dog with a thromboxane synthetase inhibitor. J Vet Internal Med 6:77-81, 1992.
94. Grauer GF: Glomerulonephritis. Sem Vet Med Surg 7:187-197, 1992.
95. Grauer GF, et al: Effects of dietary *n*-3 fatty acid supplementation versus thromboxane synthetase inhibition on gentamicin-induced nephrotoxicosis in healthy male dogs. Amer J Vet Res 57:948-956, 1996.
96. Gray DA, Frescura M: Urinary clearance of angiotensin II in Pekin ducks. Comp Biochem Physiol 115A(4):335-339, 1996.
97. Gray DA, et al: The effect of acetylsalicylic acid on renal function in the Pekin duck. Br J Pharmacol 82:329-338, 1984.
98. Greve JH: Gastrointestinal parasites. *In* Rosskopf WJ, Woerpel RW (eds): Diseases of Cage and Aviary Birds 3rd ed. Baltimore, MD, Williams and Wilkins, 1996, pp 613-619.
99. Griminger P: *In* Sturkie PD (ed): Avian Physiology 3rd ed. New York, NY, Springer-Verlag, 1976, pp 233-251.
100. Grodecki KM, et al: Treatment of X-linked hereditary nephritis in Samoyed dogs with angiotensin converting enzyme (ACE) inhibitor. J Comp Path 117:209-225, 1997.
101. Guerin J-L, et al: A novel polyomavirus (goose hemorrhagic polyomavirus) is the agent of hemorrhagic nephritis enteritis of geese. J Virology 74:4523-4529, 2000.
102. Hafner S, et al: Multicentric histiocytosis in young chickens. Gross and light microscopic pathology. Avian Dis 40:202-209, 1996.
103. Hall JA, et al: Effect of dietary *n*-6-to-*n*-3 fatty acid ratio on complete blood and total white blood cell counts, and T-cell subpopulations in aged dogs. Amer J Vet Res 60:319-327, 1999.
104. Hansen RA, et al: Duration of effects of dietary fish oil supplementation on serum eicosapentaenoic acid and docosahexaenoic acid concentrations in dogs. Amer J Vet Res 59:864-868, 1998.
105. Harper EJ, Skinner ND: Clinical nutrition of small psittacines and passerines. Sem Avian Exotic Pet Med 7:116-127, 1998.
106. Herzberg GR, et al: Uric acid synthesis by avian exocrine pancreas. Int J Biochem 23:545-548, 1991.
107. Hirsh DC: Optimizing laboratory diagnosis of infectious diseases. *In* Bonagura JD, Kirk RW (eds): Kirk's current veterinary therapy XII small animal practice. Philadelphia, WB Saunders, 1995, pp 261-266.
108. Hofbauer H, Krautwald-Junghanns M-E: Transcutaneous ultrasonography of the avian urogenital tract. Vet Radiol Ultrasound 40(1):58-64, 1999.
109. Hricik DE, Chung-Park M, Sedor JR: Glomerulonephritis. N Engl J Med 339:888-899, 1998.
110. Hughes MR: Responses of gull kidneys and salt glands to NaCl loading. Can J Physiol Pharmacol 73:1727-1732, 1995.
111. Janes DN, Braun EJ: Urinary protein excretion in red jungle fowl (*Gallus gallus*). Comp Biochem Physiol 118A:1273-1275, 1997.
112. Jenkins JR: Avian critical care and emergency medicine. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, PA, WB Saunders, 1997, pp 839-863.
113. Johnson OW, Mugaas JN: Some histological features of avian kidneys. Amer J Anat 127:423-436, 1970.
114. Johnson RJ: What mediates progressive glomerulosclerosis? The glomerular endothelium comes of age. Amer J Path 151:1179-1181, 1997.
115. Rosenthal KL, Johnston MS, Shofer FS. Assessment of the reliability of plasma electrophoresis in birds. AJVR 66:375-378, 2005.
116. Julian R: Water deprivation as a cause of renal disease of chickens. Avian Pathol 11:615-617, 1982.
117. Junge E, MacCoy DM. Amikacin therapy for *Pseudomonas* cellulites in an Amazon parrot. JAVMA 187:417-418, 1985.
118. King AS, McLelland J: Urinary system. *In* King AS, McLelland J (eds): Birds their structure and function 2nd ed. London, Bailliere Tindall, 1984, pp 175-186.
119. Klasing KC: Avian macrophages: regulators of local and systemic immune responses. Poult Sci 77:983-989, 1998.
120. Klein PN, Charmatz K, Langenberg J: The effect of flunixin meglumine (Banamine) on the renal function in northern bobwhite quail (*Colinus virginianus*): an avian model. Proc Annu Conf Assoc Rept Amphib Vet & Amer Assoc Zoo Vet, 1994, pp 128-131.
121. Klumpp SA, Wagner WD: Survey of the pathologic findings in a large production colony of pigeons, with special reference to pseudomembranous stomatitis and nephritis. Avian Dis 30:740-750, 1986.
122. Kolmstetter CM, Ramsay EC: Effects of feeding on plasma uric acid and urea concentrations in blackfooted penguins (*Spheniscus demersus*). J Avian Med Surg 14(3):177-179, 2000.
123. Koutsos EA, et al: Adult cockatiels (*Nymphicus hollandicus*) metabolically adapt to high protein diets. J Nutr 131:2014-2020, 2001.
124. Kramer LD, Bernard KA: West Nile virus infection in birds and mammals. Annals New York Acad Sci 951:84-93, 2001.
125. LaBonde J: Toxicity in pet avian patients. Sem Avian Exotic Pet Med 4(1):23-31, 1995.
126. Lafferty SL, et al: Avian polyomavirus infection and disease in a green aracaris (*Pteroglossus viridis*). Avian Dis 43:577-585, 1999.
127. Landman WJM: Amyloid arthropathy in chickens. Vet Quart 21:78-82, 1999.
128. Lane RA: Avian urinalysis a practical guide to analysis and interpretation. *In* Rosskopf WJ, Woerpel RW (eds): Diseases of Cage and Aviary Birds 3rd ed. Baltimore, Williams and Wilkins, 1996, 783-794.
129. Larochelle D, Morin M, Bernier G: Sudden death in turkeys with perirenal hemorrhage: pathological observations and possible pathogenesis of the disease. Avian Dis 36:114-124, 1992.
130. Latimer KS, et al. Metastatic renal carcinoma in an African grey parrot (*Psittacus erithacus erithacus*). J Vet Diagn Invest 8:261-264, 1996.
131. Leary AM, Roberts JR, Sharp PJ: The effect of infusion of hypertonic saline on glomerular filtration rate and arginine vasotocin, prolactin and aldosterone in the domestic chicken. J Comp Physiol B 168:313-321, 1998.
132. Lee PC, Fisher JR: Effect of allopurinol on the accumulation of xanthine dehydrogenase in liver and pancreas of chicks after hatching. Arch Biochem Biophysiol 148:277-281, 1972.
133. Leighton FA, Gajadhar AA: *Eimeria fraterculae* sp. n. in the kidneys of Atlantic puffins (*Fratercula arctica*) from Newfoundland, Canada: species description and lesions. J Wildl Dis 22(4):520-526, 1986.
134. Lierz M: Avian renal disease: pathogenesis, diagnosis, and therapy. Vet Clin Exot Anim 6:29-55, 2003.
135. Longhofer SL, et al: Effects of thromboxane synthetase inhibition on immune complex glomerulonephritis. Amer J Vet Res 52:480-487, 1991.
136. Lulich JP, Osborne CA: Bacterial infections of the urinary tract. *In* Ettinger SJ, Feldman EC (eds): Textbook of Veterinary Internal Medicine. Diseases of the dog and cat 4th ed. Philadelphia, PA, WB Saunders, 1995, pp 1775-1788.
137. Lulich JP, Osborne CA, Polzin DJ:

Diagnosis and long-term management of protein-losing glomerulonephropathy; a 5-year case-based approach. Vet Clin North Amer Small Anim Pract 26:1401-1416, 1996.
138. Lumeij JT: Plasma urea, creatinine and uric acid concentrations in response to dehydration in racing pigeons. Avian Pathol 16:377-382, 1987.
139. Lumeij JT: The diagnostic value of plasma proteins and non-protein nitrogen substances in birds. Vet Quarterly 9:262-268, 1987.
140. Lumeij JT: Avian plasma chemistry in health and disease. Proc Annu Conf Assoc Avian Vet, 1993, pp 20-26.
141. Lumeij JT: Pathophysiology, diagnosis and treatment of renal disorders in birds of prey. *In* Lumeij JT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network, Inc, 2000, pp169-178.
142. Lumeij JT, deBruijne JJ: Evaluation of the refractometric method for the determination of total protein in avian plasma or serum. Avian Pathol 14:441-444, 1985.
143. Lumeij JT, Remple JD: Plasma urea, creatinine and uric acid concentrations in relation to feeding in peregrine falcons (*Falco peregrinus*). Avian Pathol 20:79-83, 1991.
144. Lumeij JT, Westerhof I: The use of water deprivation test for the diagnosis of apparent psychogenic polydipsia in a socially deprived African grey parrot (*Psittacus erithacus erithacus*). Avian Pathol 17:875-878, 1988.
145. Lumeij JT, Sprang EPM, Redig PT: Further studies on allopurinol-induced hyperuricemia and visceral gout in red-tailed hawks (*Buteo jamaicensis*). Avian Pathol 27:390-393, 1998.
146. Machado C, et al: Disintegration of kidney stones by extracorporeal shockwave lithotripsy in a penguin. Proc 1st Internat Conf Zool Avian Med, 1987, pp 343-349.
147. Macwhirter P, Pyke D, Wayne J: Use of carboplatin in the treatment of renal adenocarcinoma in a budgerigar. Exotic DVM 4(2):11-12, 2002.
148. Mallinson ET, et al: Epizootiology, pathology, and microbiology of an outbreak of urolithiasis in chickens. Avian Dis 28:25-43, 1983.
149. Manning RO, Wyatt RD: Toxicity of *Aspergillus ochraceus* contaminated wheat and different chemical forms of ochratoxin A in broiler chicks. Poult Sci 63:458-465, 1984.
150. Marshall KL, Craig LE, Jones MP, Daniel GB. Quantitative renal scintigraphy in domestic pigeons (*Columba livia domestica)* exposed to toxic doses of gentamicin. AJVR 64:453-462, 2003.
151. Mateo R, et al: An epizootic of lead poisoning in greater flamingos (*Phoenicopterus ruber roseus*) in Spain. J Wildl Dis 33(1):131-134, 1997.
152. McMillan MC: Imaging of avian urogenital disorders. AAV Today 2:74-82, 1988.
153. McNabb FMA, McNabb RA, Steeves HR: Renal mucoid materials in pigeons fed high and low protein diets. The Auk 90:14-18, 1973.
154. Mitchell RR, et al: Health effects following chronic dosing with tungsten-iron and tungsten-polymer shot in adult game-farm mallards. J Wildl Dis 37(3):451-458, 2001.
155. Mitchell RR, et al: Hematological effects and metal residue concentrations following chronic dosing with tungsten-iron and tungsten-polymer shot in adult game-farm mallards. J Wildl Dis 37(3):459-467, 2001.
156. Miyamoto K: Evaluation of plasma clearance of inulin in clinically normal and partially nephrectomized cats. AJVR 62(8):1332-1335, 2001.
157. Mohiuddin SM, Warasi SMA, Reddy MV: Haematological and biochemical changes in experimental ochratoxicosis in broiler chicken. Indian Vet J 70:613-617, 1993.
158. Mollenhauer HH, et al: Ultrastructure of hepatic and renal lesions in chickens fed aflatoxin. Amer J Vet Res 50:771-777, 1989.
159. Mooney MA, et al: Evaluation of the effects of omega-3 fatty acid-containing diets on the inflammatory stage of wound healing in dogs. Amer J Vet Res 59:859-863, 1998.
160. Montgomery RD, Novilla MN, Shillinger RB: Renal coccidiosis caused by *Eimeria gavia* n. sp. in a common loon (*Gavia immer*). Avian Dis 22:809-814, 1978.
161. Morgulis MSFA, et al: Acute 2,4-dichlorophenoxyacetic acid intoxication in broiler chicks. Poultry Sci 77:509-515, 1998.
162. Morrow CJ, et al: Hypophosphatemic rickets and nephrocalcinosis in ostrich chicks brooded and reared on limestone sand. Vet Rec 140:531-532, 1997.
163. Munger LL, McGavin MD: Sequential postmortem changes in chicken kidney at 4, 20, or 37 C. Avian Dis 16:606-621, 1972.
164. Murai A, Furuse M, Okumura J: Involvement of (*n*-6) essential fatty acids and prostaglandins in liver lipid accumulation in Japanese quail. Amer J Vet Res 57:342-345, 1996.
165. Murray MJ: Diagnostic techniques in avian medicine. Sem Avian Exotic Pet Med 6:48-54, 1997.
166. Murray MJ, Taylor M: Avian renal disease: endoscopic applications. Sem Avian Exot Pet Med 8:115-121, 1999.
167. Murray MJ, Taylor M: The use of endoscopy and endoscopic biopsy as aids in the diagnosis of renal disease. Proc Annu Conf Assoc Avian Vet, 1997, pp 133-138.
168. Mushi EZ, Binta MG, Isa JW: Biochemical composition of urine from farmed ostriches (*Struthio camelus*) in Botswana. J S Afr Vet Assoc 72(1):46-48, 2001.
169. Mutalib A, Keirs R, Austin F: Erysipelas in quail and suspected erysipeloid in processing plant employees. Avian Dis 39:191-193, 1995.
170. Nakamura K, Abe F: Respiratory (especially pulmonary) and urinary infections of *Cryptosporidium* in layer chickens. Avian Pathol 17:73-711, 1988.
171. Nakamura K, et al: Systemic amyloidosis in laying Japanese quail. Avian Dis 42:209-214, 1998.
172. Narcisi EM, Sevoian M, Honigberg BM: Pathologic changes in pigeons infected with a virulent Trichomonas gallinae strain (Eiberg). Avian Dis 35:55-61, 1991.
173. Neuman U, Kummerfeld N: Neoplasms in budgerigars (*Melopsittacus undulatus*): clinical, pathomorphological and serological findings with special consideration of kidney tumours. Avian Pathol 12:353-362, 1983.
174. Ni Y, Kemp MC: A comparative study of avian reovirus pathogenicity: virus spread and replication and induction of lesions. Avian Dis 39:554-566, 1995.
175. Novilla MN, Kwapien RP: Microsporidian infection in the pied peach-faced lovebird (*Agapornis roseicollis*). Avian Dis 22(1):198-204, 1978.
175b. Oaks L, Gilbert M, Virani MZ, et al: Diclofenac residues as the cause of vulture population decline in Pakistan. Nature 427:630-633, 2004.
176. Obendorf DL, McColl K: Mortality in little penguins (*Eudyptula minor*) along the coast of Victoria, Australia. J Wildl Dis 16(2):251-259, 1980.
177. Oksanen A: Mortality associated with renal coccidiosis in juvenile wild greylag geese (*Anser anser anser*). J Wildl Dis 30(4):554-556, 1994.
178. Om A-S, Chung KW, Chung H-S: Effect of cadmium accumulation on renal tissue of broilers. Bull Environ Contam Toxicol 68:297-301, 2002.
179. Orth SR, Ritz E: The nephrotic syndrome. N Engl J Med 338:1202-1211, 1998.
180. Orosz S: Anatomy of the central nervous system. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 454-459.
181. Orosz S, Dorrestein GM, Speer BL: Urogenital disorders. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 614-644.
182. Öztürk-Ürek R, Bozkaya LA, Tarhan L: The effects of some antioxidant vitamin- and trace element-supplemented diets on activities of SOD, CAT, GSH-Px and LPO levels in chicken tissues. Cell Biochem Funct 19:125-132, 2001.
183. Pardi DS, et al: Renal and urologic complications on inflammatory bowel disease. Amer J Gastroenterology 93:504-514, 1998.
184. Pegram RA, Wyatt RD: Avian gout caused by oosporein, a mycotoxin produced by *Chaetomium trilaterale*. Poult Sci 60:2429-2440, 1981.
185. Pegram RA, Wyatt RD, Smith TL: Oosporein-toxicosis in the turkey poult. Avian Dis 26:47-59, 1982.
186. Pennycott TW: Causes of mortality and culling in adult pheasants. Vet Rec 146:273-278, 2000.
187. Phalen DN, Ambrus S, Graham DL: The avian urinary system: form, function, diseases. Proc Annu Conf Assoc Avian Vet, 1990, pp 44-57.
188. Phalen D: Avian renal disorders. *In* Fudge AM (ed): Laboratory Medicine Avian and Exotic Pets. Philadelphia, PA, WB Saunders, 2000, pp 61-68.
189. Phalen DN, Wilson VG, Graham DL: Characterization of the avian polyomavirus-associated glomerulopathy of nestling parrots. Avian Dis 40:140-149, 1996.
190. Plotnick AN: The role of omega-3 fatty acids in renal disorders. JAVMA 209:906-910, 1996.
191. Plumb DC (ed): Veterinary Drug Handbook. Ames, IA, Iowa State University Press, 1999.
191b. Poffers J, Lumeij JT, Redig PT: Investigations into the uricolytic properties of urate oxidase in a granivorous (*Columba livia domestica*) and in a carnivorous (*Buteo jamaicensis*) avian species. Avian Pathol 31:573-579, 2002.
191c. Poffers J, Lumeij JT, Timmermans-Sprang EPM, Redig PT: Further studies on the use of allopurinol to reduce plasma uric acid concentrations in the red-tailed hawk (*Buteo jamaicensis*) hyperuricemic model. Avian Pathol 31:567-572, 2002.
192. Poonacha KB, William PD, Stamper RD: Encephalitizoonosis in a parrot. JAVMA 186(7):700-702, 1985.
193. Puette M, Crowell WA, Hafner WS: Ultrastructural examination and cell count determinations of avian glomeruli from grossly normal and grossly swollen kidneys of broilers at slaughter. Avian Dis 38:515-522, 1994.
194. Pulparampil N, Graham D, Phalen D: *Encephalitozoon bellem* in two eclectus parrots (*Eclectus roratus*): Identification from archival tissues. J Eukaryot Microbiol 45(6):651-655, 1998.
195. Quebbemann AJ: Renal synthesis of uric acid. Amer J Physiol 224:1398-1402, 1973.
196. Radford MG, et al: Reversible membranous nephropathy associated with the use of nonsteroidal anti-inflammatory drugs. J Amer Med Assoc 276:466-469, 1996.
197. Radin MJ, Hoepf TM, Swayne DE: Use of a single injection solute-clearance method for determination of glomerular filtration rate and effective renal plasma flow in chickens. Lab Anim Sci 43(6):594-596, 1993.
198. Radin MJ, et al: Renal function and organic anion and cation transport during dehydration and/or food restriction in chickens. J Comp Physiol B 166:138-143, 1996.
199. Randall CJ: Renal and nasal cryptosporidiosis in a junglefowl (*Gallus sonneratii*). Vet Rec 119:130-131, 1986.
200. Riddell C, Roepke D: Inflam-

mation of the nasal gland in domestic turkeys. Avian Dis 35:982-985, 1991.
201. Rigdon RH: Occurrence and association of amyloid with diseases in birds and mammals including man: a review. Tex Rep Biol Med. 32:665-682, 1974.
202. Ritchie BW (ed): Avian Viruses Function and Control. Lake Worth, FL, Wingers Publishing, 1995.
203. Ritchie BW, Harrison GJ: Formulary. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 457-478.
204. Roberts JR, Dantzler WH: Glomerular filtration rate in conscious unrestrained starlings under dehydration. Amer J Physiol 256:R836-R839, 1989.
205. Roberts JR: The effect of acute or chronic administration of prolactin on renal function in feral chickens. J Comp Physiol B 168:25-31, 1998.
206. Romagnano A, et al: Magnetic resonance imaging of the brain and coelomic cavity of the domestic pigeon (*Columba livia domestica*). Vet Radiol Ultrasound 37:431-440, 1996.
207. Roudybush TE: Psittacine nutrition. Vet Clin N Amer Exot Anim Pract 2(1):111-1125, 1999.
208. Sarkar K, et al: The ultrastructure of nephrocalcinosis induced in chicks by Cestrum diurnum leaves. Vet Pathol 18:62-70, 1981.
209. Sato Y, et al: An occurrence of avian tuberculosis in hooded merganser (*Lophodytes cucullatus*). Avian Dis 40:941-944, 1996.
210. Schneider RR, et al: A descriptive study of mortality at the Kortright waterfowl park: 1982-1986. Can Vet J 29:911-914, 1988.
211. Schoemaker NJ, Lumeij JT, Beynen, AC: Polyuria and polydipsia due to vitamin and mineral oversupplementation of the diet of a salmon crested cockatoo (*Cacatua moluccensis*) and blue and gold macaw (*Ara ararauna*). Avian Pathol 26:201-209, 1997.
212. Shibatana M, et al: Disseminated intravascular coagulation in chickens inoculated with Erysipelothrix rhusiopathiae. J Comp Path 117:147-156, 1997.
213. Shuttleworth TJ, Hildebrandt J-P: Vertebrate salt glands: short- and long-term regulation of function. J Exp Zoo 283:689-701, 1999.
214. Siller WG: Renal pathology of the fowl- a review. Avian Pathol 10:187-262, 1981.
215. Simoyi MF, Van Dyke K, Klandorf H: Manipulation of plasma uric acid in broiler chicks and its effect on leukocyte oxidative activity. Amer J Physiol Reg Int Comp Physiol 282:R791-R796, 2002.
216. Skadhauge E, Maloney SK, Dawson TJ: Osmotic adaptation of the emu (*Dromaius novaehollandiae*). J Comp Physiol B 161:173-178, 1991.
217. Skirnisson K: Mortality associated with renal and intestinal coccidiosis in juvenile eiders in Iceland. Parassitologia 39:325-330, 1997.
218. Smith JH, Neill PJG, Dillard EA: Pathology of experimental *Sarcocystis falcatula* infections of canaries (*Serinus canarius*) and pigeons (*Columba livia*). J Parasitol 76(1):59-68, 1990.
219. Sokkar SM, Mohamed MA, Atawia M: Experimental induction of renal lesions in chickens. Berl Münch Tierärztl Wschr 111:161-163, 1998.
220. Son JH, Karasawa Y: Effects of caecal ligation and colostomy on water intake and excretion in chickens. Br Poultry Sci 42(1):130-133, 2001.
221. Speer BL, Kass PH: The influence of travel on hematology parameters in hyacinth macaws. Proc Annu Conf Assoc Avian Vet, 1995, pp 43-49.
222. Sreemannarayana O, Frohlich AA, Marquardt RR: Acute toxicity of sterigmatocystin to chicks. Mycopathologia 97:51-59, 1987.
223. Steinhort LA: Avian fluid therapy. JAMS 13:83-91, 1999.
224. Stoev SD, et al: Spontaneous mycotoxic nephropathy in Bulgarian chickens with unclarified mycotoxin aetiology. Vet Res 33:83-93, 2002.
225. Stunkard HW. Renicolid trematodes (*Digenea*) from the renal tubules of birds. Annales de Parasitologie 46:109-118, 1971.
226. Sturkie PD: Alimentary canal: anatomy, prehension, degludition, feeding, drinking, passage of ingesta, and motility. *In* Sturkie PD (ed): Avian Physiology 3rd ed. New York, Springer-Verlag, 1976, pp 186-195.
227. Sturkie PD: Kidneys, extrarenal salt excretion, and urine. *In* Sturkie, PD (ed): Avian Phsiology 4th ed. New York, Springer-Verlag, 1986, pp 359-382.
228. Styles DK, Phalen DN: Clinical avian urology. Sem Avian Exot Pet Med 7(2):104-113, 1998.
229. Suedmeyer WK, Bermudez A: A new approach to renal biopsy in birds. J Avian Med Surg 10(3):179-186, 1996.
230. Surai PF, Sparks NHC: Tissue-specific fatty acid and α-tocopherol profiles in male chickens depending on dietary tuna oil and vitamin E provision. Poul Sci 79:1132-1142, 2000.
231. Takaha N, Taira E, Taniura H, et al: Expression of gicerin in development, oncogenesis and regeneration of the chick kidney. Differentiation 58:313-320, 1995.
232. Tarugi P, et al: Secretion of apoB- and apoA-1-containing lipoproteins by chick kidney. J Lipid Res 39:731-743, 1998.
233. Taylor M: A new endoscopic system for the collection of diagnostic specimens in the bird. Proc Annu Conf Assoc Avian Vet, 1993, pp 83-86.
234. Taylor M: Biopsy techniques in avian medicine. Proc Annu Conf Assoc Avian Vet, 1995, pp 275-280.
235. Tham VL, Purcell DA, Schultz DJ: Fungal nephritis in a grey-headed albatross. J Wildl Dis 10:306-309, 1974.
236. Trampel DW, Pepper TM, Blagburn BL: Urinary tract cryptosporidiosis in commercial laying hens. Avian Dis 44:479-484, 2000.
237. Tucker FL, Strugill BC, Bolton WK: Ultrastructural studies of experimental autoimmune glomerulonephritis in normal and bursectomized chickens. Lab Invest 53:563-570, 1985.
238. Tudor DC: Congenital defects of poultry. World Poult Sci J 35:20-26, 1979.
239. Vaden SL, et al: The effects of cyclosporin versus standard care in dogs with naturally occurring glomerulonephritis. J Vet Internal Med 9:259-266, 1995.
240. Van Elswyk ME, et al: Dietary menhaden oil contributes to hepatic lipidosis in laying hens. Poult Sci 73: 653-662, 1994.
241. Van Toor AJ, Zwart P, Kaal GThF: Adenocarcinoma of the kidney in two budgerigars. Avian Pathol 13:145-150, 1984.
242. Wages DP, et al: Salt toxicosis in commercial turkeys. Avian Dis 39:158-161, 1995.
243. Wang HN, Wu QZ, Huang Y, Liu P: Isolation and identification of infectious bronchitis virus from chickens in Sichuan, China. Avian Dis 41:279-282, 1997.
244. Wark JD, et al: Regulation of 25-hydroxy-vitamin D-1a-hydroxylase in chick isolated renal tubules: effects of prostaglandin E2, frusemide and acetylsalicylic acid. Clinical Sci 61:53-59, 1981.
245. Waters CB, et al: Effects of glucocorticoid therapy on urine protein-to-creatinine ratios and renal morphology in dogs. J Vet Internal Med 11:172-177, 1997.
246. Weir E, Fisher JR: The effect of allopurinol on the excretion of oxypurines by the chick. Biochem Biophysiol Acta 222:556-557, 1970.
247. Whitehead CC, Siller WG: Experimentally induced fatty liver and kidney syndrome in the young turkey. Res Vet Sci 34:73-76, 1983.
248. Wideman RF: Avian kidney anatomy and physiology. CRC Critical Rev Poult Biol 1:133-176, 1988.
249. Wideman RF, et al: Acute heat acclimation and kidney function in broilers. Poult Sci 73:75-88, 1994.
250. Wideman RF, Laverty G: Kidney function in domestic fowl with chronic occlusion of the ureter and caudal renal veins. Poult Sci 65:2148-2155, 1986.
251. Wideman RF, Mallinson ET, Rothenbacher H: Kidney function of pullets and laying hens during outbreaks of urolithiasis. Poult Sci 62:1954-1970, 1983.
252. Williams JB, Braun EJ: Renal compensation for cecal loss in Gambel's quail (*Callipepla gambelii*). Comp Biochem Physiol 113A(4):333-341, 1996.
253. Wobeser G: Renal coccidiosis in mallard and pintail ducks. J Wildl Dis 10:249-255, 1974.

CHAPTER
17

Evaluating and Treating the Nervous System

SIMON R. PLATT, BVM&S, MRCVS, Dipl ACVIM (Neurology), Dipl ECVN, RCVS Specialist in Veterinary Neurology

The nervous system plays a role in nearly all body processes. Disease syndromes may affect the central nervous system (CNS), which includes the brain and spinal cord, and the peripheral nervous system, which includes cranial nerves, spinal cord nerve roots, spinal nerves, peripheral nerve branches and the neuromuscular junction. Evaluation of the nervous system of birds parallels that of mammals, with modifications specific to avian anatomy, behavior and physiology. An understanding of basic avian neuroanatomy is necessary to evaluate abnormal function of the nervous system.[15,16,62,87]

Suspicion of neurologic dysfunction arises from the history and physical examination. The signalment, presenting chief complaint, time course of clinical signs, and history may suggest the type of disease process or species-specific disorder. A complete neurologic examination is necessary to localize the anatomic distribution, to determine the severity of the disease process, and to assess the prognosis for patient recovery. A minimum database consisting of a complete blood count (CBC), serum or plasma biochemical analysis, and cytologic evaluation of choanal and cloacal swab samples is used to evaluate the contribution of other body systems to the observed clinical signs and physical abnormalities. If neuromuscular disease is suspected, electrophysiological tests are indicated, including electromyography and nerve conduction velocities, and these are becoming more commonly performed in birds. In recent years with the advancement of imaging technology and the improved availability for veterinary patients, scintigraphy, computed tomography and magnetic resonance imaging have been valuable diagnostic commodities in furthering our understanding of avian CNS disease. Only when a disease location and a pathologic process have been identified can appropriate treatment and prognosis be provided.

Anatomy and Physiology

Complete reviews of avian neuroanatomy are documented in other texts.[15,62,85,87] For a detailed description of the functional organization of the avian spinal cord, the reader is referred to an avian physiology text.[83] The general organization of the avian spinal cord resembles that of all other vertebrates. There are however, some specializations, which birds largely share with the phylogenetically related class of reptiles. Compared to the mammalian cord, the most outstanding deviations are the lack of a *cauda equina* and a *filum terminale* and the occurrence of a *sinus rhomboidalis* or *lumbosacralis*.[83] At the microscopic level there are some significant differences in the organization of cell groups and pathways. Birds differ from most other vertebrates in their locomotion due to bipedal walk and flight. It has to be kept in mind that specializations in the spinal cord may result from adaptations from this peculiar kind of locomotion.

Control of Eye and Head Movements

A sitting bird is able to survey a large part of its surroundings using both head and eye movements. Several types of eye movements can be distinguished such as saccades (fast flicks elicited by a sudden stimulus) and smooth pursuit (following a moving target).[33] Six muscles are responsible for all movements of the eye. Branches of the oculomotor, trochlear and abducens nerves innervate these extrinsic eye muscles. The two muscles that rotate the eye dorsally are innervated by contralateral centers, with the other muscles innervated by ipsilateral cell groups. These are stimulated by the vestibular centers.[37]

Control of Jaw and Tongue Movements

The jaw muscles have a visceral origin; therefore, the nerves innervating these muscles and the corresponding motor nuclei are considered visceromotor elements.[33] A complication in parrots is that they possess a so-called kinetic skull. This means that not only the lower jaw but also the upper jaw moves to open the beak.[33] Movement of the beak is achieved by four groups of muscles: two groups of beak openers and two groups of beak closers.

Movements of the tongue are caused by two groups of muscles. The extrinsic muscles are part of the visceral musculature, similar to the jaw muscles, and the intrinsic muscles have a somatic origin innervated by the hypoglossal nerve. Because the jaws and tongue move in close harmony during feeding and drinking, a premotor system is needed to coordinate the activity of the tongue and jaw motor centers.[33]

Control of Locomotion

The reticular formation in the brain stem also contains premotor neurons of the motor systems of the spinal cord, thus controlling flying and walking. The final affectors of wing and leg movement are the lower motor neurons of the brachial and lumbar plexuses, respectively. In-depth review of this area is covered in an avian physiology text.[15,33]

Control of Vocalization and Respiration

Birds use their syrinx, an organ at the transition of the trachea and bronchi, to produce sounds. Vocalization depends upon the control of a few small syringeal muscles and the precise regulation of the stream of (expiratory) air passing through the trachea. This requires a close coordination of the motor centers of the syringeal and respiratory muscles. The caudal part of the dorsal hypoglossal motor nucleus innervates the syringeal muscles, whereas the motor cells of the respiratory muscles are part of the motor system of the spinal cord. However, several cell groups in the cranial and caudal brainstem have a role in the control of respiration. In songbirds, these centers are all under direct telencephalic (cerebral) control.[120] See Chapter 19, Endocrine Considerations.

Control of Coordination

Birds possess a well-developed cerebellum but, as in mammals, its precise role in the control of motor activity is not well understood. However, there is little doubt that the ease and precision of motor performance depend upon an intact cerebellum. The axons of the Purkinje cells form the output of the cerebellar cortex; the central cerebellar nuclei and some vestibular nuclei are the targets of these fibers.[33] The Purkinje cells have an inhibitory effect on the activity of the central nuclei.

Neurological Examination and Lesion Localization

A neurological examination is easily integrated into a routine physical examination. The objectives of the neurological examination are to confirm if there is a neurological abnormality and to specifically localize the abnormality within the nervous system. In conjunction with the history, signalment, presenting complaint and the physical examination, the neurological lesion localization is a piece of a jigsaw, essential to creating a list of differential diagnoses for the disease. However, caution must be used, as some manipulations necessary for the neurological examination could exacerbate problems such as spinal cord disease.[80]

OBSERVATION

Observation of the bird is essential, as it allows evalua-

tion of the bird's mentation (level and content of consciousness), posture, attitude and gait. Changes in mentation are revealed by a history of personality change, change in awareness of surroundings and inappropriate behavioral responses.[26] Consciousness is a function of the brainstem (responsible for arousal) and the cerebral cortex (responsible for content and regulation).[26,40,101,117,119]

PALPATION

The bird's musculoskeletal system should be palpated for asymmetry, masses, tenderness, contour and tone. A mass effect, tenderness or contour change requires further investigation. The vertebral column should be palpated for deviations and pain, being cautious not to apply too much pressure if there is suspicion of an instability. Unilateral muscle mass loss or atrophy may indicate disuse if it is chronic, or a neurogenic loss if it is acute (within 7 to 10 days).[26]

CRANIAL NERVES (CNs)

Several differences exist between the neuroanatomy of avian and mammalian species.[15,26] Cranial nerves have specific functions and evaluation of these functions can help to precisely locate a neurological lesion due to their well-documented anatomy. The general functions and specific tests are summarized in Table 17.1.

Simplistically, cranial nerve dysfunction may indicate a CNS lesion (brainstem disease) or a peripheral lesion (affecting the cranial nerves after they have exited the brainstem and course through the skull). Evaluation of the cranial nerves should follow observation and palpation, with particular attention paid to normal functions of eye movement, head movement, blinking, jaw and tongue movement, and general symmetry of the head.

Initially an ophthalmic exam should be performed, which will assist with the evaluation of the optic (CN II), oculomotor (CN III), trochlear (CN IV), abducens (CN VI), and trigeminal (CN V) nerves.[15,16,62] The following tests are essential to the evaluation of cranial nerve function.

The Menace Response

How to Perform

Obscure the vision in one eye and make a slow, threatening hand gesture toward the other eye (Fig 17.1).

How to Interpret

This is a learned response, not a reflex, to a perceived threat, which evaluates CNs II and V (responsible for innervation of the *orbicularis oculi* muscle, which closes the eyelids), as well as the central visual pathways and the cerebellum.[62,87] Birds that are stoic or excited may not show a menace response even though nerve function is intact. Normal function is demonstrated by a blink. To localize the lesion, other cranial nerve tests would be required.

Table 17.1 | Cranial Nerves: Function and Applicable Tests

Cranial Nerve	Nerve Function	Applicable Tests
I. Olfactory	Smell	None applicable
II. Optic	Vision	i. Menace response ii. Pupillary light reflex
III. Oculomotor	Extrinsic and intrinsic ocular muscles/ upper eyelid muscle	i. Eyeball position ii. Menace response iii. Pupillary light reflex
IV. Trochlear	Extrinsic ocular muscles	i. Eyeball position
V. Trigeminal	Facial and beak sensation/beak movement	i. Palpebral reflex ii. Jaw palpation
VI. Abducens	Extrinsic ocular muscles and third eyelid muscle	i. Eyeball position
VII. Facial	Muscles of facial expression	None applicable
VIII. Vestibulocochlear	Hearing and balance	i. Startle ii. Oculocephalic reflexes
IX. Glossopharyngeal	Muscles of pharynx, larynx, crop and syrinx	i. Gag reflex
X. Vagus	Muscles of larynx, pharynx, esophagus and crop	i. Gag reflex
XI. Accessory	Superficial neck muscles	None applicable
VII. Hypoglossal	Muscles of tongue, trachea and syrinx	i. Tongue grab/ inspection

Fig 17.1 | The menace reaction is performed by making a threatening gesture with fingers or hand toward one eye and watching for a blinking action.

The Pupillary Light Reflex

How to Perform

Shine a bright light in each eye to evaluate the response of the pupil (Fig 17.2).

How to Interpret

This is a reflex. Light is sensed by CN II; parasympathetic

Fig 17.2 | The pupillary light response is performed by shining a light source into one eye at a time and monitoring the response of the pupil.

Fig 17.3 | The palpebral reflex is performed by touching the medial canthus of the palpebral fissures and watching for a blink in response.

fibers of CN III cause contraction of the iris muscle in normal birds with direct stimulation. There is no consensual response in birds due to the 100% decussation of the optic nerves at the optic chiasm.[26] However, some raptors have an incomplete bony septum between the globes and may respond to light reaching the contralateral retina, mimicking a consensual response.[82] The response may not be as marked as that seen in mammals, as birds have striated muscle in the iris, giving them some voluntary control over pupil size independent of light intensity. This reflex is best evaluated as soon as possible during the examination, as sympathetic tone in restrained birds may override constriction.

Evaluation of Strabismus

How to Perform

Observe the bird's head in a normal position for a deviation of one or both globes in the orbit(s).

How to Interpret

Cranial nerves III, IV and VI aid vision by maintaining the globe in a central position. Deviation of the globe from its central axis indicates dysfunction in one or more of these nerves: ventrolateral — CN III, dorsolateral — CN IV, and medial — CN VI. In contrast to mammals, the third eyelid in birds has striated muscle fibers, innervated by CN VI, that initiate movement of the nictitans across the cornea.[20,59,62,82,87] Prolapse of the third eyelid in birds does not directly indicate loss of sympathetic innervation, as found in Horner's syndrome in mammals.

The Palpebral Reflex

How to Perform

Touch the medial canthus of the normal eyelid and watch the response (Fig 17.3).

Greg J. Harrison

Fig 17.4 | A cockatiel (*Nymphicus hollandicus*) with eyelid paresis from infraorbital sinus infection demonstrates a cranial nerve V lesion.

How to Interpret

The normal eyelid should close. Cranial nerve V (trigeminal nerve) is responsible for facial sensation, whereas the motor response to facial sensory stimulation is generally provided by the facial nerve (CN VII). The eyelids are innervated by CN V in birds.[39,62,87] Eyelid sensation is provided by the ophthalmic branch (upper lid) and the maxillary branch (both lids) of CN V. Eyelid closure is provided by the mandibular branch (*orbicularis oculi* muscle) of CN V that, when damaged, may present as eyelid paresis (Fig 17.4).

Evaluation of Jaw/Beak Tone

How to Perform

Observe the bird for a dropped lower beak and/or an inability to eat with the beak. Assess the strength of the beak safely by manually opening the beak and evaluating the resistance to opening (Figs 17.5a,b).

How to Interpret

The mandibular branch of CN V provides motor func-

Fig 17.5a | The beak is assessed for strength and movement.

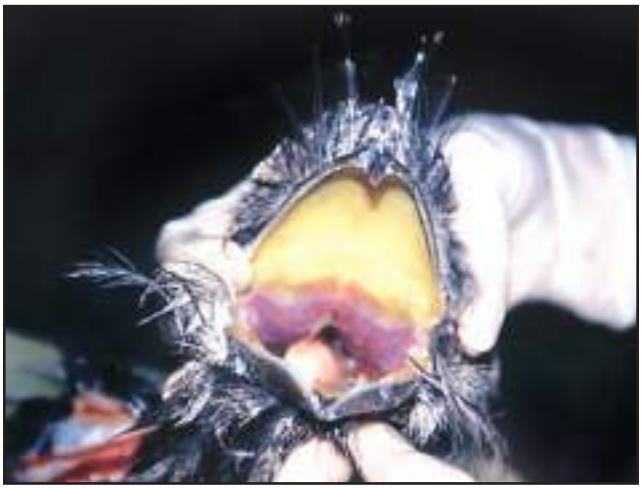


Fig 17.5b | The movement and symmetry of the tongue can be assessed by forcing open the beak or using a speculum.

tion to the jaw.[39,87] A dropped lower beak or the inability to chew indicates damage to CN V. Cranial nerve VII contributes to prehension by partial innervation of the muscles that open the jaw.

The Oculocephalic Reflex/Interpretation of Nystagmus

How to Perform

Move the head from side to side in a horizontal plane and observe the resulting movement of the eyes (Fig 17.6).

How to Interpret

In normal birds, a physiological nystagmus will be induced, with the fast phase in the direction of head movement. This reflex tests the integrity of CN VIII (vestibulocochlear nerve), which is the sensory arm of this reflex, and CNs III, IV and VI, which are responsible for the motor movement of the eyes. Clinical signs of peripheral vestibular disease are manifest after damage to the inner ear or vestibular branch of CN VIII, which effectively gives unbalanced input to the intact central vestibular system.[26] In the absence of head motion, spontaneous horizontal nystagmus is consistent with CN VIII damage, with the fast component away from the side of the lesion. Unilateral peripheral disease may cause a head tilt with circling toward the side of the lesion.

POSTURAL REACTIONS

The postural reactions are complex, requiring intact sensory and motor pathways throughout the nervous system, as well as unimpaired processing and integration in the brain.[26] The complexity of the postural reactions allows detection of minor deficits in any key component of the pathway. Postural deficits are seen caudal to or at the level of the lesion.[40] Additional testing must be performed to use the postural deficit to help localize the lesion within the pathway of the deficit.

How to Perform

A wing or leg is placed in an abnormal position and a correcting response by the bird is observed. Not all standard mammalian postural reactions can be done due to the modifications of the forelimbs in birds. However, knuckling the toes over while supporting the bird can be done to evaluate how long it takes for the bird to correct. Placing the bird's foot on its dorsal surface against a perch may be a more practical way to test proprioception in the limbs. Alternatively, a piece of paper may be placed under each foot and slowly moved sideways to see if the bird returns its foot to the standing position. Placing reactions of the wings are not evaluated routinely in birds. Placing reactions in the rear limbs may be helpful. Visual placing may be evaluated crudely by offering a perch for the patient to step onto (Fig 17.7). Tactile placing may be evaluated in birds such as ratites and raptors that allow hooding. A perch may be touched to the dorsum of the foot to initiate a step-up.

How to Interpret

Conscious proprioception is the patient's awareness of limb position and movement without visual information. The sensory branch of proprioception is carried from the skin, muscle and joints of the leg, through the spinal cord and brainstem to the sensory motor cortex where the brain responds by sending messages back to the lower motor neuron for motor function, resulting in a rapid correcting foot placement.[26] Ascending sensory pathways are located in the outermost regions of the spinal cord and are very sensitive to compression.[40,87] With minor spinal cord injury, proprioceptive deficits may be present because of disrupted sensory pathways, while motor function persists because the deeper motor tracts are unaffected. Both visual and tactile placing reactions require an intact motor cortex and intact motor pathways to the involved limb. A cortical lesion may

Fig 17.6 | By moving the head from side to side, the movements of the eyes can be evaluated for physiological nystagmus.

Table 17.2 | Clinical Signs Associated with Upper Motor Neuron (UMN) and Lower Motor Neuron (LMN) Diseases

Clinical Function	UMN Disease	LMN Disease
Motor function	Paresis or paralysis	Paresis or paralysis
Muscle tone	Normal to increased	Often reduced
Spinal reflexes	Normal to decreased	Decreased to absent
Muscle mass	Normal to decreased (disuse atrophy)	Dramatically decreased after 5-7 days (neurogenic atrophy)
Conscious proprioception	Often reduced to absent	Often reduced to absent

produce deficits in the contralateral limb, whereas a lower lesion produces deficits in the ipsilateral limb.

SPINAL REFLEXES

Completion of a reflex requires an intact sensory nerve that provides transmission to the spinal cord and an intact motor nerve that elicits function from the innervated muscle. The reflex arc itself does not involve the brain or the remainder of the spinal cord. Lesions in the motor arm of the reflex arc, termed lower motor neuron (LMN), may cause a decreased or absent reflex (hyporeflexia or areflexia). An exaggerated response (hyperreflexia) results from an interruption in proximal motor pathways that modulate the reflex, termed upper motor neuron (UMN). Lower motor neuron signs indicate damage to one or more components of the reflex arc. Upper motor neuron signs indicate damage anywhere between the reflex arc and the brain (Table 17.2).[39,62,87]

The Vent Sphincter Reflex

How to Perform

Pinch or prick the vent and watch for a wink-like contraction of the external sphincter muscles and a tail bob.

Fig 17.7 | Bringing the bird to a perch will assess the ability to place the limbs properly; this is one method of assessing proprioception. The bird also can be visually restricted using hooding.

Fig 17.8 | The pedal withdrawal can be evaluated by applying a pinching stimulus to the digits and watching for flexion and withdrawal of the joints and limb, respectively.

How to Interpret

This reflex reveals information regarding the pudendal plexus and caudal segments of the spinal cord. A flaccid, unresponsive vent and overdistended cloaca that is easily expressed indicate LMN damage to the pudendal nerve or its spinal roots. A hypertonic, hyperresponsive vent indicates UMN damage at any point cranial to the pudendal plexus.

The Pedal Flexor Reflex

How to Perform

Apply a pinch stimulus to the skin of each foot and evaluate the response of the ipsilateral and contralateral limb (Fig 17.8).

How to Interpret

This is a withdrawal reflex in which stimulation of sensory receptors in the feet elicits contraction of flexor muscle groups in the leg. Presence of a withdrawal

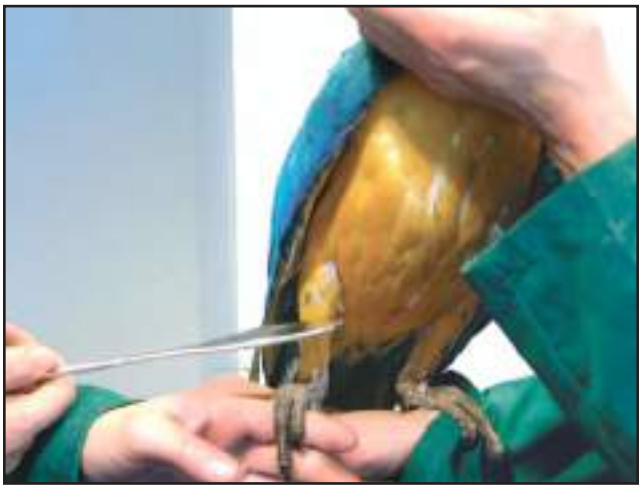


Fig 17.9 | With the limb allowed free movement, the patella reflex can be performed by applying a stretch stimulus to the patellar tendon.

Fig 17.10 | The wing withdrawal can be assessed by stretching each wing out and applying gentle pinching stimulus, watching a complete flexion and withdrawal.

reflex requires an intact ischiatic nerve (sensory and motor) and an intact spinal segment at the sacral plexus, but does not require transmission along the spinal cord to the brain. Absence of the withdrawal reflex in the leg denotes extensive lower motor neuron damage involving the sacral plexus or the ischiatic nerve. A space-occupying mass within the kidney or pelvic canal, such as a renal tumor or an egg, may impinge upon the lumbosacral plexus and its nerve, causing a hyporeflexic or areflexic withdrawal in the legs.[15]

The Patellar Reflex

How to Perform

A tap stimulus should be applied to the straight patellar tendon and the response of the limb should be evaluated (Fig 17.9). Certain birds, such as ostriches,[26] do not have a patella; in these species, the homologous straight quadriceps tendon is stimulated. Plexor size must be adapted to patient size for improved accuracy. Because of interference from the inguinal web, this reflex may be difficult to elicit in birds.[26]

How to Interpret

This is a myotatic (stretch) reflex that effectively stretches the *femorotibialis* muscle.[87] This stretch stimulates the femoral nerve (lumbosacral plexus), which generates muscular contraction to extend the stifle. Upper motor neuron lesions cause hyperreflexia and should be accompanied by weakness.[26] Disease in the lumbosacral plexus or peripheral muscle or nerve (LMN) causes hyporeflexia.

The Wing Withdrawal Reflex

How to Perform

A pinch stimulus to the major digit at the leading edge of the primary flight feathers generates flexion of the wing in the normal bird (Fig 17.10).

How to Interpret

This reflex tests the integrity of the reflex arc to the brachial plexus spinal segment. Absence of the wing withdrawal reflex in a wing indicates damage to the brachial plexus or its nerves. A positive crossed extensor reflex of a wing denotes damage cranial to the brachial plexus within the cervical spinal cord or the brain stem.

CUTANEOUS SENSATION AND PAIN

Cutaneous sensation testing provides information regarding the location and severity of a spinal cord or plexus lesion.[26] Birds do not have a *cutaneous trunci* muscle, so a panniculus response cannot be used to help localize a spinal cord lesion; however, the feather follicles contain sensory nerve fibers.[31]

Evaluation of nociception (deep pain perception) is reserved for those animals showing evidence of spinal cord disease based on abnormalities in gait, proprioception and spinal reflexes. Nociception requires cerebral perception of painful or injurious stimuli. It is important to remember that a withdrawal reflex is not an indicator of pain perception and may be elicited in an animal whose spinal cord has been transected cranial to the segment responsible for that reflex arc.[26,40] See Chapter 8, Pain Management.

Ancillary Neurological Tests

SURVEY RADIOGRAPHY AND MYELOGRAPHY

After a thorough clinical examination, selective or survey radiography may be indicated to evaluate body systems for

their contribution to clinical signs. As it is inexpensive and non-invasive, survey radiography may be one of the best screening tools to uncover a cause for neurologic disease.

Sedation or general anesthesia facilitates exact positioning to yield the best image of the desired area. Both lateral and ventrodorsal projections should be obtained, with the beam centered over the area of interest. The axial skeleton of the bird should be extended, and the spine should be made parallel to the cassette, supporting the bird's body with foam wedges if required. The most helpful spinal views include the following: cervical (lateral and ventrodorsal), thoracic (lateral), thoracolumbar (lateral and ventrodorsal), and lumbar (lateral and ventrodorsal). A fine screen and high-detail films[a] should be used for spinal studies in birds.

Myelography can be performed in birds with normal patient recovery and adds another diagnostic procedure to the armamentarium of avian veterinarians. Myelography is used to identify, characterize and localize spinal cord lesions to the extramedullary or intramedullary compartments. Myelography is indicated if precise localization of a spinal cord lesion is required, such as when surgical intervention is contemplated. A contrast medium is injected into the subarachnoid space and lack of opacification around the spinal cord indicates displacement of that cord segment, preventing contrast material flow and suggesting diffuse cord swelling or a space-occupying mass compressing the cord.

The synsacrum of birds is composed of the fused lumbar and sacral vertebrae and pelvis, and necessitates thoracolumbar rather than lumbar puncture of the subarachnoid space. Another structure unique to birds is the glycogen body, which bisects the spinal cord in the lumbosacral region ventral to the synsacrum and causes a narrowing of the subarachnoid space in this region.[51] It appears that injection of contrast medium into the subarachnoid space in the cerebellomedullary cistern cannot be consistently repeated and trauma to the spinal cord near the brainstem can result in death.[51]

A technique for administering a myelographic contrast agent, iohexol[b], in the thoracolumbar region has been described.[51] The region for injection is found by placing the thumb and middle finger on the bony prominences of the ileal crests, with the index finger used to palpate to the first indentation cranial to the synsacrum. The quantity of the contrast medium needed to produce a diagnostic myelogram is 0.88 ml/kg, 4 times the 0.22 ml/kg used in mammals in most institutions.[51] The use of a 27-gauge needle for this procedure is currently recommended,[51] and the administration of approximately 0.5 ml/minute has been described as safe.[51] Radiographs should be made within 10 minutes of the contrast administration. Seizures, the predominant side effect noted in mammalian myelography, have not been observed in birds, even at higher doses than those recommended above. However, this does not rule out the possibility of this occurring with iohexol myelography in birds.[51] There are some avian species in which myelography may not be possible. In duck, goose and swan skeletons, the thoracic and lumbar vertebrae have overlapping plates of bone that would prevent positioning a needle in the subarachnoid space. Potential thoracolumbar puncture sites do exist in psittacines and raptors, which are the most commonly seen patients in private practice and rehabilitation centers.

CEREBROSPINAL ANALYSIS

Cerebrospinal fluid (CSF) may confirm pathologic changes, determine the general nature of the pathologic process or reveal a specific cause of disease.[25,64] CSF is most useful in characterizing infectious, neoplastic or inflammatory disorders of the spinal cord or brain. Ideally, this procedure should be performed prior to myelography, as contrast will affect the analysis of the fluid.

In birds, the cerebellomedullary cistern is very small, and a large venous plexus exists just ventral to it.[62] There is less than a 1 mm space dorsal to the cervical spinal cord in which the tip of the needle could be placed. This can result in blood-contaminated samples, lack of samples and damage to the nervous tissue.[51] The damage, which can be focal and cause acute severe hemorrhage in the subarachnoid space, can result in postanesthetic recovery problems including cardiac arrest and death.[51] Presently, it is recommended to use a 27-gauge needle for this procedure.[30]

How to Perform

The patient is placed in lateral recumbency with the midline of the neck parallel to the tabletop. The caudodorsal skull region is aseptically prepared. The atlanto-occipital joint is flexed at a 30° angle, and a 27-gauge hypodermic needle is placed at dorsal midline, slightly caudal to the occipital protuberance, and is directed rostrally at a 45° angle to the horizontal axis of the head. The needle is advanced through the skin slowly at 1-mm intervals until a slight change in resistance is felt. Practically, this is not always easily recognized. However, the advantage of using a hypodermic needle with a translucent hub, rather than a spinal needle with a stilette, is that an immediate "flash" of fluid will appear in the hub of the needle once the dura has been penetrated. This limits the risk of advancing the needle into parenchyma. In general, 0.1 to 0.5 ml of fluid may be collected. In small mammals, 1 ml of CSF per 5 kg body weight may be removed safely, although this has not been confirmed in birds.[25]

Fig 17.11 | A barred owl (*Strix varia*) under general anesthesia for an electromyographic examination.

Fig 17.12 | A concentric needle is inserted into each muscle for evaluation of the presence of a spontaneous electrical activity.

How to Interpret

The small sample volume usually obtained precludes extensive biochemical analysis, bacterial or fungal culture, and antibody titer evaluation. Color and clarity of the CSF should be evaluated immediately. Gross visual examination of normal CSF reveals a clear and colorless fluid. Blood contamination may be iatrogenic or due to a pathologic process.[26] Turbidity indicates leukocytosis, high protein content or both. If the sample volume is sufficient, protein content may be evaluated in addition to cytological examination. High protein concentration indicates breakdown of the blood-brain barrier or intrathecal protein synthesis. Preparation of the CSF sample by cytospin centrifugation is recommended because of the small volume and low cellularity of fluid generally recovered.[26] If a cytocentrifuge is unavailable, the cells may be concentrated onto a slide by sedimentation.[25] The cell concentration, population and morphology are evaluated. Relative erythrocyte and leukocyte numbers and protein concentration should be compared with those found in the peripheral blood. Reference values for leukocyte numbers and protein concentration in CSF have not been established in birds. Subjective comparison of CSF values with those of peripheral blood and consideration of parameters used in domestic animals may aid interpretation.

Meningitis caused by infectious agents generally induces a moderate increase in cell numbers and an increase in protein concentration compared with peripheral blood.[3,26] Non-inflammatory conditions caused by degenerative disease or trauma cause a high protein concentration and a minimally increased cell count.[25,26]

ELECTRODIAGNOSTICS

The use of electrophysiological diagnostic techniques has become a cornerstone in the investigation of neuromuscular disease in animals. Electrodiagnostic evaluation of the neuromuscular system is used as an aid in the diagnosis of disease, to assess the progress or regression of the disease, and to assess the degree of recovery.[65,110] Skeletal and smooth muscle as well as the myoneural junction can be examined. Concentric needle electrodes are used to determine the spontaneous or evoked responses. They are usually 27-gauge, subcutaneous or longer, insulated needles with bare tips, which can be inserted into the muscle. A ground electrode must always be used. The patient usually has to be anesthetized for these procedures (Fig 17.11).

Electromyography

Electromyography (EMG) is the study of the electrical activity of muscle by insertion of a recording electrode into the muscle (Fig 17.12). It examines the integrity of the motor unit, which consists of the lower motor neuron and the muscle fibers that it innervates. Normal resting muscle does not show observable electrical activity once the electrode placement is stabilized and no audible signal is created.[110] Contraindications for electromyography include bleeding tendencies and unusual susceptibility to recurrent systemic infections.[61] Electrical activity demonstrated can be separated into three categories: insertional (associated with electrode movement), spontaneous (associated with resting muscle) and evoked (associated with electrical stimulation of nerves).[61]

Insertional

A brief burst of electrical activity accompanies needle insertion into a normally innervated muscle. This collection of monophasic and polyphasic potentials of variable amplitude and duration ends after the needle has stopped its movement. A prolonged discharge of these potentials is sometimes subjectively considered as abnormal, as it represents cellular hyperirritability. Insertional potentials become more noticeable on the

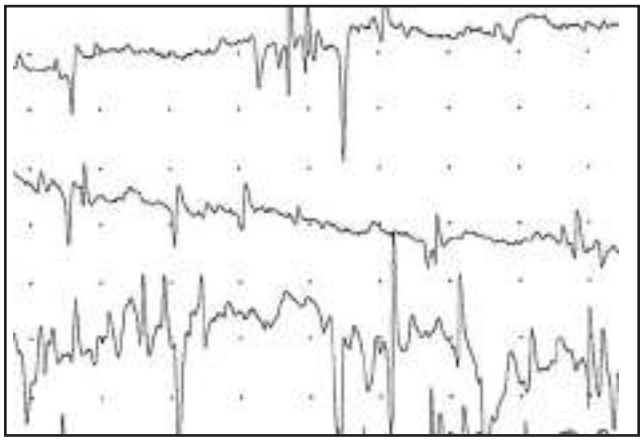


Fig 17.13 | Fibrillation potentials and positive sharp waves from a denervated muscle 5 days after brachial trauma.

fourth or fifth day after denervation and peak in intensity 8 to 10 days after denervation.[26] These may be decreased or absent in atrophied muscle.[110]

Spontaneous

Normally, muscles are said to be "electrically silent." Five to seven days after denervation in the dog, needle electromyography can demonstrate spontaneous, randomly occurring potentials.[26] These are the action potentials of several muscle fibers due to instability in the membrane potential and are termed fibrillation potentials. The time of onset of these potentials in birds has been poorly documented, but may be earlier than seen in the dog. Fibrillation potentials can be monophasic, biphasic and, rarely, triphasic in wave form with a usual amplitude of 20 to 300 μV and a duration of 0.5 to 5 msec (Fig 17.13), both of which decrease with cicatrization.[110] The presence of reproducible discharges in at least two different areas of muscle usually suggests lower motor neuron disease. These include diseases of the ventral horn cells, radiculopathies, plexopathies, myositis, and axonal mono- or polyneuropathies.[61] Positive sharp waves with a sawtooth appearance also are abnormal spontaneous potentials that are detected in motor unit disorders, and it has been suggested that they may precede fibrillation potentials by one or more days.[110] Complex repetitive discharges range from 50 μV to 1 mV in amplitude and up to 50 to 100 msec in duration, representing a group of muscle fibers firing in near synchrony.[61] These discharges typically begin suddenly and maintain a constant firing rate, ceasing abruptly and sounding like a machine gun.[61] These complex repetitive discharges appear as a "train" of identical discharges. These discharges may occur in a variety of myopathic conditions. Needle electromyography has been used in the cat, a species susceptible to organophosphorus-induced delayed neuropathy like the bird, to monitor progression and improvement.[1] This could not be repeated, though, in chickens.[107] Denervation is detectable on needle electromyography in birds. Fibrillation potentials and occasional complex repetitive discharges were seen in the wing muscles of two red-tailed hawks with traumatic brachial plexus injury. Motor unit action potentials observed in these same birds suggested that intact nerve fibers were present.[108] Fibrillation potentials and positive sharp waves were detected diffusely in limb musculature of a turkey vulture with a peripheral neuropathy associated with lead toxicosis.[92] Spontaneous EMG also has been used to determine prognosis and to direct treatment in seven wild birds with traumatic injuries involving peripheral nerves and musculoskeletal disease.[27] Widespread fibrillation potentials and lack of typical interference patterns suggested denervation in three of these seven birds that had muscle atrophy and wing paralysis or paresis.

Evoked

The M wave is an evoked potential resulting from the summation of motor unit potentials. The latency of response represents the time taken for impulse conduction from the point of stimulus to the nerve terminal, including a neuromuscular delay of about 0.5 msec, and the time taken for conduction from the end-plate region to the exploring electrode. The latency can be considered an adequate substitute for the nerve conduction velocity.[26] The amplitude of the evoked potential is determined by the number of muscle fibers activated by nerve stimulation. In a severed motor nerve, conduction stops abruptly after about 5 to 8 days.[26]

Nerve Conduction Studies

Nerve conduction studies have become a simple and reliable test of peripheral nerve function.[61] The conduction velocity is measured between the two stimulus points on the nerve, thereby eliminating the time for neuromuscular transmission and generation of muscle action potential. It is derived as the ratio between the distance between two stimulation cathodes and the corresponding latency difference.[61] The proximal stimulation site is a point just caudal to the greater trochanter of the femur, and the distal site is just caudal to the distal portion of the tibia (Fig 17.14). At each site, the cathode is placed distal to the anode. The indifferent electrode is placed subcutaneously over the midtarsometatarsus region. The active electrode is placed subcutaneously on the caudal portion of the hypotarsus just distal to the tarsometatarsal joint overlying the digital abductor muscles.[10] Similar electrode configurations are needed for evaluation of the wing nerves (Fig 17.15). The ground electrode is placed subcutaneously between the distal stimulation site and the recording site. This technique has been described in detail for two avian orders.[27]

Axonal damage or dysfunction will usually result in loss of amplitude, whereas demyelination leads to prolonga-

Fig 17.14 | Needle setup for evaluation of a tibial nerve conduction velocity in a barred owl.

Fig 17.15 | Needle setup for evaluation of an ulnar nerve conduction velocity in a rhea.

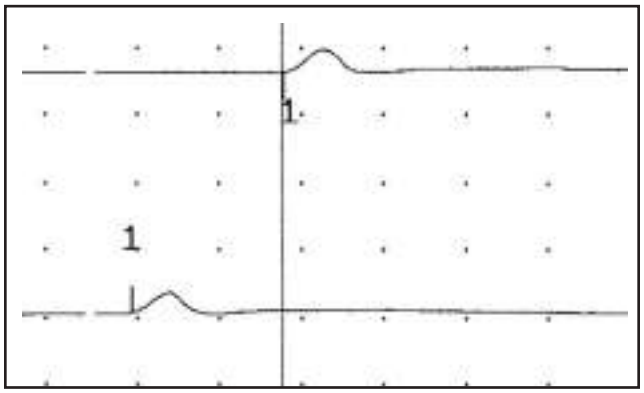


Fig 17.16 | A reduced nerve conduction velocity is demonstrated after stimulation of the proximal portions (top trace) of the tibial nerve and the distal portions (lower trace). The waveforms (M wave) demonstrated here are typical.

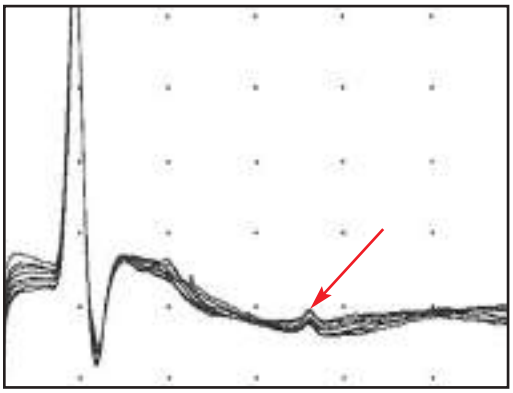


Fig 17.17 | An F wave (arrow) is demonstrated after the M wave and the stimulation artifact.

tion of conduction time (Fig 17.16).[61] Reference ranges for the motor nerve conduction velocities of the ulnar nerve and the tibial nerve in neurologically normal barred owls and rheas have been established.[27] Based upon these ranges, a turkey vulture with lead toxicosis was shown to have decreased sciatic-tibial nerve conduction velocity compatible with a demyelinating neuropathy.[92] Peripheral nerve histopathology confirmed this finding.

Effect of Temperature

A linear relation normally exists between motor nerve conduction velocity and temperature of the peripheral nerve segment within physiological limits.[67]

Effects of Age

Immature animals have slower motor nerve conduction velocity than do adults.[26]

The F Wave

Measurement of the F wave can help in the assessing of motor conduction along the most proximal segment of the nerve. This is because it results from the backfiring of antidromically activated ventral horn cells. It can supplement the conventional nerve conduction studies, especially in polyneuropathies where delay of the F wave can markedly exceed the normal range. It occurs after the M response, its initiating electrical stimulation having traveled toward the spinal cord before it returns to activate distal muscles (Fig 17.17). Studies of the F wave can help in the characterization of polyneuropathies and, more specifically, with those that are prominently proximal, such as radiculopathies.[61] Such studies have been attempted in normal birds, but as yet a reference range has not been established for clinical application.[27]

Repetitive Stimulation

Repeated supramaximal stimulation should create repeatable, identical action potentials in the normal patient. Any evident incremental or decremental response may have physiological significance. Any defect in the neuromuscular transmission will usually produce a maximal drop in amplitude between the first and second responses of a train, followed by further decline up to the fourth or fifth potential.[61] Examples of these disorders would be myasthenia gravis in humans and companion animals and botulism in all species. Repetitive stimulation has been performed in normal birds to

establish reference ranges for clinical application.[27] The techniques were easily performed and demonstrated 22% alteration in potential amplitude.[27]

MUSCLE BIOPSY

Muscle biopsy is indicated to confirm, define and possibly provide a cause for motor unit disease.[19,88] Biopsy techniques are well established in several mammalian species, but are not known for avian patients. The muscle selected for biopsy should be one that is clinically affected with the disease process. In acute disease, a severely affected muscle is selected. With chronic disease, a muscle demonstrating only moderate changes is the best choice. The muscle should be easily accessible and identifiable so that minimal postoperative discomfort is created. Ideally, the muscle chosen for biopsy would be one for which normal fiber type, distribution and size are known. This is not available for most avian patients. Studies of muscle fiber types have been done in chickens.[16]

SCINTIGRAPHY

Nuclear imaging or scintigraphy is a non-invasive method for evaluation of soft tissue and osseous structures associated with the nervous system.[112] The technique involves intravenous administration of a small amount of a gamma-emitting radionuclide alone or tagged to some other compound that will allow it to accumulate in certain tissues and exclude it from other tissues. A gamma camera is used to record the amount of radiation emitted from the body and to create images of the distribution of the radionuclide throughout the patient's body. Technetium-99m (^{99m}TC) is used most often, as it has a short half-life (approximately 6 hours) and emits pure gamma radiation. Because the level of radiation is extremely low, its use carries minimal risk for the patient and personnel involved with the procedure. Nuclear scans do not provide detailed anatomic imaging, but do have the potential to provide functional information when used with a digital image processor.

Scintigraphic imaging of the brain has been done in human medicine since 1960.[26] In recent years, cranial scintigraphic imaging has been replaced by computed tomography (CT) and magnetic resonance imaging (MRI), but is still useful where these modalities are considered too expensive or are not available. Bone scanning is particularly useful in identifying spinal abnormalities in birds.[26] Superimposition of osseous structures (ribs, synsacrum) in survey radiographs makes identification of spinal fractures and early vertebral osteomyelitis difficult. In a study of 12 birds with thoracic or pelvic limb paresis, bone scanning identified 100% of the lesions identified on survey radiographs.[75] In addition, several lesions not identified on survey radiographs, including fractures and early osteomyelitis, were identified by bone scanning. These pathologic lesions were confirmed by the results of necropsy. Basilic vein administration of the radionuclide was found to be superior to medial metatarsal vein administration. This is possibly because the renal portal system provided high uptake of radionuclide by the liver and kidney and obscured visualization of the spine after medial metatarsal vein administration.[75]

COMPUTED TOMOGRAPHY

Computed tomography imaging requires general anesthesia, uses ionizing radiation and is somewhat costly to perform. The CT scan uses x-rays and computer technology to create cross-sectional images of the patient.[54,68] Regions of the vertebral column and the skull are evaluated for abnormalities in CNS soft tissue or its protective skeleton. Computed tomography provides superior soft tissue imaging with no superimposition of structures compared with conventional radiography.[77,86] With contrast administration, soft tissue structures are better visualized, especially where there is increased blood flow.[86,99]

The greatest value of CT lies in its ability to reveal changes in bony tissues and to provide greater detail of osseous structures than conventional radiography. Slices are most commonly created every 2 to 3 mm in birds.[86] Although these slices may seem very close together, focal lesions may still be missed. Decreasing slice thickness to 2 mm decreases image quality, but may increase sensitivity for small lesions. Images also can be reformatted by the computer to provide images in configurations other than a transverse plane.

Computed tomography has been used to document topographic anatomy of the golden eagle and African grey parrot,[77,86] and it also has been used to specifically demonstrate brain and skull anatomy of the crested breed of the domestic duck (*Anas platyrhynchos*).[12] Computed tomography has been used with some success to locate intracranial lesions in birds, although it was only 80% sensitive.[58,99] A postmortem CT scan was used to identify spinal cord compression in a juvenile penguin (*Aptenodytes patagonicus*) that was euthanized because of an inability to stand despite 6 weeks of medical therapy;[38] radiographs at presentation had failed to identify any spinal abnormalities. Results of necropsy confirmed intervertebral disc rupture and vertebral body displacement.

MAGNETIC RESONANCE IMAGING

Magnetic resonance imaging (MRI) uses a pulsating external magnetic field and produces radiofrequency signals that are used to generate images.[26,112] Soft tissue con-

trast of the CNS is excellent with MRI, which also provides the spatial orientation of anatomic structures.[26,112] This modality also will help to differentiate central nervous system gray and white matter.

The advantages of MRI compared with CT include the ability to image the cranial brainstem, without artifact, in the caudal fossa of the skull, the increase in soft tissue detail, the creation of images in various planes and the lack of ionizing radiation.[98] However, MRI does not allow a satisfactory depiction of aerated bones in some avian skulls.[12] One main disadvantage of MRI besides the expense, as with CT, is the requirement for general anesthesia, as chemical sedation is not adequate to obtain satisfactory images. It also is necessary to obtain screening radiographs of birds before performing MRI evaluations to rule out the presence of metallic foreign bodies.[98] Avian patients will frequently chew on their cages, etc, and small metal fragments may contaminate commercial feed. Ferrous metal demonstrates susceptibility to the magnetic field and can move within the body.[98]

In a study of normal MRI brain anatomy of pigeons, imaging required approximately 20 minutes, and transverse images were created every 3 mm.[98] A 1.5 Tesla magnet and a human knee surface coil were used. Lesions may be missed because of this distance between slices. This imaging technique also was used in a clinical case in an attempt to identify the cause of seizure activity in a mealy Amazon parrot (*Amazona farinosa*).[98] Without contrast enhancement the lesions were not visualized; however, after the use of contrast material (gadopentate dimeglumine[c] 0.25 mmol/kg IV) perivascular infiltrates were identified, showing disruption of the blood-brain barrier. Because the contrast is eliminated through the kidneys, postcontrast uric acid levels have been compared to precontrast levels to monitor for potential renal toxicity in birds; however, no significant changes have been found.[98] Further, after contrast medium-enhanced MRI studies in birds, recovery has been uneventful and the birds have had normal excreta, appetites and attitudes.[98] Magnetic resonance imaging also has been used to evaluate the cranium of domestic ducks.[13] MRI images were recorded using a relatively large slice thickness of 5 mm, which can lead to difficulty in interpreting scans of small volumes.

Intracranial Disease

CLINICAL SIGNS

Intracranial CNS structures include the forebrain (telencephalon), thalamus (diencephalon), midbrain (mesencephalon), pons (metencephalon), medulla oblongata (myelencephalon) and the cerebellum. The midbrain, pons and medulla make up the brainstem and are associated with the majority of the cranial nerves. Disease affecting the intracranial CNS may be focal or diffuse. If it is focal, specific deficits may be identified relating to dysfunction of the area affected. The various syndromes and specific signs are described in Table 17.3.

Seizures

Definitions and Classifications

A seizure is a sudden, uncontrolled, transient alteration in behavior characterized by a change in motor activity, consciousness, sensation or autonomic function.[28] It is due to a synchronizing abnormal paroxysmal electrical discharge from the brain. *A generalized seizure* is manifested by symmetrical and synchronous clinical signs, which are classically tonic-clonic motions often accompanied by complete loss of consciousness.[28] *A focal*

Table 17.3 | Intracranial Lesion Localization and Clinical Signs[93]

Clinical Syndrome/ Lesion Localization	Characteristics of the Location	Specific Clinical Signs
Cerebrum	Damage to cerebrum affects intellectual, learned and sensory activities (vision, hearing, touch, pain)	i. Altered mental state ii. Seizures iii. Behavioral change iv. Pleurothotonus and adversion v. Head pressing vi. Central blindness
Diencephalon	Damage affects autonomic visceral functions and endocrine regulation	i. Altered mental state ii. Seizures iii. Behavioral change iv. Endocrine disturbances v. Abnormalities of temperature
Midbrain	Damage affects control of alert status, as well as CN III and CN IV function	i. Altered mental state ii. Opisthotonus iii. Contralateral paresis iv. Ipsilateral CN III dysfunction (mydriasis/ventrolateral strabismus) v. Contralateral CN IV dysfunction (dorsomedial strabismus)
Pons and Medulla	Damage affects multiple cranial nerves (V-XII) as well as cardiorespiratory centers	i. Altered mental state ii. Ipsilateral paresis iii. Irregular respiration iv. Ipsilateral CN V dysfunction (decreased beak strength, decreased palpebral reflex, decreased facial sensation) v. Ipsilateral CN VI dysfunction (third eyelid protrusion and medial strabismus) vi. Ipsilateral CN VII dysfunction (decreased tone in facial musculature, decreased taste, decreased lacrimation) vii. Ipsilateral CN VIII damage (ipsilateral deafness, vestibular dysfunction) viii. Ipsilateral CN IX-XII damage (dysphagia, regurgitation, tongue deviation, inspiratory dyspnea)
Cerebellum	Damage affects coordinating and reinforcing actions	i. Normal mental status and behavior ii. Normal strength iii. Opisthotonus iv. Dysmetric ataxia v. Head (intention) tremors

seizure is manifested initially by focal signs that may secondarily become generalized.

Treatment

1. Address the underlying cause.
2. Control seizure activity with diazepam (0.5-1.0 mg/kg IV or IO).[28] This can be repeated two to three times if necessary.
3. If diazepam does not stop the activity, administer phenobarbital (1-2 mg/kg IV/IO or IM q 6-12 h to effect).[28]
4. Gaseous anesthesia may be warranted in seizuring birds to allow diagnostic sampling and investigation.
5. Convert to oral phenobarbital maintenance therapy within 48 hours (1-10 mg/kg PO q 12 h is standard therapy).[28]
6. Primidone may be an alternative anticonvulsant, although its active compound is phenobarbital. Primidone use (125 mg/day in water source) has been reported in an Amazon parrot.[52]
7. Adjust to lowest dosage required to control seizure activity.

Head Tilt and Nystagmus

Definitions and Classifications

A head tilt is due to asymmetrical disease of the vestibular system (Fig 17.18). The vestibular system comprises the peripheral component, which is the vestibular portion of CN VIII arising in the inner ear from the semicircular canals and the central component, which is the nuclei of CN VIII in the medulla.[28] A further central component of this system is the flocculonodular lobe of the cerebellum. Head tilts are usually toward the side of the disease with peripheral vestibular dysfunction, but toward any side with central dysfunction. Nystagmus that is pathological is described as jerky eye movements with two obvious phases; slow and fast. The predominant direction of the eye movements, seen at rest, can be horizontal, rotational or vertical. Nystagmus also can be positional, which means that it is induced by a change in position of the head. With horizontal nystagmus, the fast phase is usually away from the side of the lesion. Vertical and positional nystagmus can indicate that the lesion is central rather than peripheral. All other directions can occur with both peripheral or central lesion localizations.

Treatment

1. Address the underlying cause and tailor therapy to the specific etiology.
2. Provide supportive care to maintain nutritional and fluid demands.

Tremors and Involuntary Movements

Definitions and Classifications

Head and or body tremors may occur with activity and appear worse with movement such as eating or drinking. These are called intention tremors and are the cardinal sign of cerebellar disease of any underlying etiology. Generalized body tremors that occur mainly at rest could result from a multifocal or diffuse lesion localization. This may be a disease affecting the meninges, nerve roots, peripheral nerves or muscles. Systemic diseases including electrolyte imbalances, liver and renal disease, and hypoglycemia can cause generalized tremors as can primary neurologic diseases.

Greg J. Harrison

Fig 17.18 | A lovebird (*Agapornis roseicollis*) demonstrates a right-sided head tilt caused by an ipsilateral loss of muscle tone due to peripheral vestibular disease.

Treatment

1. Address the underlying cause.
2. Attempt treatment with oral anticonvulsant therapy as above.
3. Attempt treatment with anti-inflammatory doses of glucocorticoids.
4. Provide supportive care to maintain nutritional and fluid demands.

Depression/Stupor/Coma

Definitions and Classifications

Depression represents a reduced mental state, although one which responds to external stimuli. Depression does not necessarily indicate primary neurologic disease. Stupor is a state of unconsciousness from which the bird can be roused by stimulation with a noxious stimulus (Fig 17.19). Coma is a state of unconsciousness from which the bird cannot be roused by noxious stimuli.

Treatment

1. Address the underlying cause.
2. Provide supportive care to maintain nutritional and fluid demands.
3. Address ventilatory needs.
4. Address excretion needs and keep clean.


Greg J. Harrison

Fig 17.19 | A painted bunting (*Passerina ciris*) with profound stupor due to pesticide toxicosis.

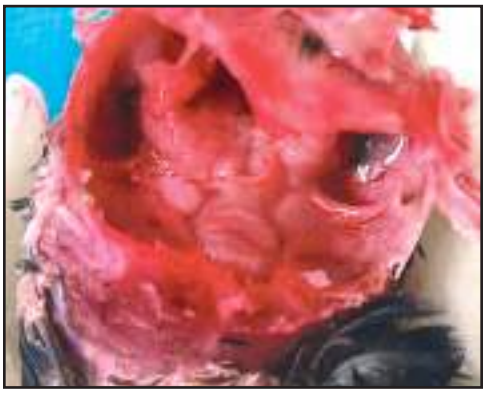

Greg J. Harrison

Fig 17.20 | A gross pathological specimen from an African grey parrot (*Psittacus erithacus*) with cerebral disease due to hydrocephalus.

DIFFERENTIAL DIAGNOSIS OF INTRACRANIAL DISEASE (Table 17.4)

Degenerative

Lysosomal Storage Disease

Gangliosidosis[18,60]

Neurolipofuscinosis[53]

Avian Vacuolar Myelinopathy[71]

This disease more commonly causes spinal and neuromuscular signs, but intracranial signs have been reported.

Anomalous

Hydrocephalus[100]

Dilated ventricles can be a congenital lesion as well as a postinflammatory consequence (Fig 17.20).

Metabolic

Hepatic Encephalopathy[15,29]

Clinical signs included depression, ataxia and blindness.

Hypoglycemia[15]

Seizure activity may occur when the glucose levels drop below 100 mg/dl.

Hypocalcemia

Serum calcium levels below 6.0 mg/dl with concentrations as low as 2.4 mg/dl have been reported to cause opisthotonus (Fig 17.21), tonic extension of the limbs and convulsions.[15]

Neoplasia[100,113]

In a retrospective study of 996 budgerigars (*Melopsittacus undulatus*), 14 (1%) intracranial tumors were detected.[113] Tumors of the ependyma (6), choroid plexus (1), adenohypophysis (6) and one unclassified tumor were found. In this study 87% of the birds were male, indicating a possible sex predisposition, as there were equal numbers of males and females in the study. Typically, neoplasia is a necropsy diagnosis, but it may be found antemortem with a CT or MRI scan. Glial cell tumors, choroid plexus papillomas, adenomas, adenocarcinomas and lymphosarcomas have all been reported as individual primary brain tumors.[15]

Intracranial Lipomas

Four birds in a flock of 125 purebred crested ducks (*Anas platyrhynchos*) had cerebellar signs of unknown

Table 17.4 | Differential Diagnosis of Intracranial Disease

Mechanism of Disease	Specific Diseases
Degenerative	i. Lysosomal storage disease - Gangliosidosis/Neurolipofuscinosis ii. Avian vacuolar myelinopathy
Anomalous	i. Hydrocephalus
Metabolic	i. Hepatic encephalopathy ii. Hypoglycemia iii. Hypocalcemia
Neoplastic	i. Glial tumors/Ependymoma/Choroid plexus papillomas/Adenomas/Adenocarcinomas/Lymphosarcoma/Lipomas
Nutritional	i. Pyridoxine deficiency ii. Vitamin E deficiency
Inflammatory	i. Viral encephalitis - Proventricular dilatation disease/Avian paramyxovirus type 1 (Newcastle disease)/Polyomavirus/WEE and EEE/West Nile virus/Avian influenza virus/Herpes (duck viral enteritis) ii. Bacterial encephalitis - *Salmonella/Pasteurella/Streptococcus/Enterococcus/Chlamydophila/Listeria* iii. Verminous encephalitis - *Chandlerella/Baylisascaris*/(fluke/*Schistosomiasis*) iv. Trichomonas/Protozoal encephalitis - Toxoplasmosis/Leucocytozoonosis/Sarcocystis v. Fungal encephalitis - Mucormycosis vi. Prion - Spongiform encephalopathy
Idiopathic	i. Idiopathic epilepsy
Toxicity	i. Lead ii. Zinc iii. Organophosphates and carbamates
Trauma	i. Head trauma
Vascular	i. Atherosclerosis


Greg J. Harrison

Fig 17.21 | A young cockatiel (*Nymphicus hollandicus*) demonstrates the marked dorsiflexion of the neck seen with opisthotonus.


Jan Hooimeijer

Fig 17.22 | A pigeon (*Columba livia*) with paramyxovirus infection of the central nervous system demonstrates profound torticollis of the neck.

etiology.[14] Gross necropsy of the euthanized ducks revealed yellow intracranial masses in the brain of each. Histologically, these masses were intracranial lipomas consisting of fatty tissue separated into lobules by connective tissue. These lipomas were similar to those recorded in other animals and humans.[14]

Nutritional

Pyridoxine (Vitamin B_6) Deficiency[15]

Vitamin E Deficiency (Nutritional Encephalomalacia)[6]

The recommended dietary level of vitamin E for domestic poultry is 10 to 25 IU/kg feed dry matter (DM), but this is not known for many other birds. Clinical signs are commonly seen in young chicks (2-6 weeks old) and include ataxia, head retraction and "cycling" with legs.[6] This has become known as crazy chick disease.[66] Gross and histological lesions of encephalomalacia are primarily found in the cerebellum, with evidence of ischemic necrosis of the cerebellar cortex and white matter, capillary thrombi, hemorrhages and malacia.

Inflammatory

Viral Encephalitis

Avian Paramyxovirus Type 1 (Newcastle Disease) causes a non-suppurative encephalomyelitis and has been reported to cause head shaking, head tremors, head tilt, circling and torticollis (Fig 17.22).[8,69] Infection is usually reported in juvenile wild birds, racing pigeons and domestic poultry.[57,69] The virus will commonly cause motor dysfunction and so is discussed below.

Avian Polyomavirus (APV), also known as budgerigar fledgling disease, is a member of the family Papovaviridae.[72] Polyomavirus infection in psittacine birds may be subclinical or may present as a fatal, acute, multisystemic disease.[72] Although observed more frequently in budgerigars (*Melopsittacus undulatus*), APV infection may result in death of both juvenile and adult non-budgerigar psittacine species.[72] Viral inclusions have been found in brain tissue, however, reports of virus-induced neurologic disease are rare.[72] Tremors of the head, neck and limbs, incoordination and ataxia have been described in nestling budgerigars with APV infection. Approximately 10% of susceptible neonates exhibited neurologic signs.[72] In addition, an adult Moluccan cockatoo (*Cacatua moluccensis*) with APV infection exhibited inability to perch properly and had a slight torticollis that progressed to a semicomatose state.[72] Neurologic deterioration was observed over a 2-day period following a 1-week history of illness. Body tremors and unsteadiness on the perch were signs documented in a Ducorps's cockatoo (*Cacatua ducorps*) with concurrent APV and psittacine beak and feather disease virus (PBFDV).[72] PBFDV (circovirus) infection is often associated with clinical evidence of acquired immunodeficiency, leading to a variety of secondary or opportunistic infections.[72]

Proventricular Dilation Disease (PDD) is a progressive, variably contagious and often fatal disease of psittacine birds, which is presumed to be caused by an unidentified neurotropic virus.[17] The disease was first recognized in macaws in the late 1970s in the USA and is now known to have a worldwide distribution affecting more than 50 species of psittacine birds.[17] Neurologic clinical signs include ataxia, tremors and seizures. Definitive diagnosis in live and dead birds is based on demonstration of characteristic lymphoplasmacytic infiltrates within autonomic nerves and ganglia at various levels of the digestive tract.[17,48]

Equine Encephalitis - Viral infections have been reported in commercial, domestic, and free-living flocks of passerines and Columbiformes for several decades.[96] Most infections are attributed to eastern equine encephalitis (EEE), but several outbreaks of western equine encephalitis (WEE) have been documented.[96] Many types of birds are natural hosts of EEE and WEE viruses.[96] The diseases are caused by an alpha virus in

the arbovirus group/family, and are transmitted through the bites of mosquitoes.

Specific signs of WEE and EEE have been vague. In 1991, the first confirmed outbreak of EEE was reported in ratites, specifically emus (*Dromiceius novaehollandiae*) in southeastern Louisiana.[96] This outbreak coincided with unseasonably heavy rainfall, an abundance of arthropod vectors and close proximity to reservoir host species.[96] Affected emus exhibit variable signs, becoming very drowsy and depressed, reluctant to stand up, trembling, often remaining sitting, and developing an S-shaped cervical curvature. Aroused sick birds are ataxic with leg weakness.[96] Both infections can progress to cause paralysis and death.[114]

Diagnosis is confirmed by isolation of the virus from the brains of birds that die. Serum antibody titers for EEE, WEE and Venezuelan equine encephalitis exposure can be evaluated by hemagglutination inhibition.[96] Recently, an immunohistochemistry technique for confirmatory diagnosis of EEE infection in birds has been developed.[121] Treatment has been supportive, consisting of oral electrolytes and broad-spectrum antibiotics.[96] Protection of commercial emu flocks may be attempted by a routine vaccination schedule for both EEE and WEE. Vaccine efficacy recently has been proven in emus, and it currently is the only protective action that exists, other than mosquito control.[96,114]

West Nile Virus (WNV) emerged in the northeastern USA in the summer of 1999 causing death to thousands of wild and zoo birds.[41,106] Previously this infection has been reported around the Mediterranean basin.[81] It is a mosquito-borne flavivirus that is endemic in Africa and Asia, occurring sporadically in temperate regions of Europe.[106] It has been shown to cause a diffuse encephalitis and has been recovered from the brains of birds in winter, long after the mosquito vectors ceased to be active, suggesting a prey-to-predator mode of transmission.[41] Live and inactivated vaccines for protection against WNV recently have been evaluated in young domestic geese with initial promising results.[78]

Avian Influenza Virus has been reported to cause 75 to 100% mortality in birds within 10 days of infection.[91] Depression and neurologic dysfunction were common clinical manifestations of the disease.[91] These signs include attenuated motor functions, such as paresis to paralysis, vestibular dysfunction as indicated by torticollis and nystagmus, and general behavior aberrations.[91]

Herpes (Duck Viral Enteritis) causes photophobia, ataxia, seizures and penile prolapse.[15]

Bunyavirus[109] rarely causes clinical disease after transmission of the virus from mosquitoes.

Bacterial Encephalitis

Listeria monocytogenes causes intracranial infections of birds resulting in opisthotonus, ataxia, and torticollis.[15]

Chlamydophila psittaci will occasionally cause neurologic signs in birds following acute respiratory or gastrointestinal disease.[15]

Abscesses, granulomas, encephalitis and meningitis has been reported to be due to *Salmonella typhimurium*,[53] *Pasteurella multocida* (fowl cholera),[50] Lancefield group D *Streptococcus* sp.,[24,47] and *Enterococcus* sp. (neonatal multifocal encephalomalacia with sepsis).[24,44]

Verminous Encephalitis

Chandlerella quiscali is a filariid nematode of grackles that has been reported to cause cerebrospinal nematodiasis in emus.[15] Clinical signs in the young include torticollis, ataxia, recumbency and death.

Baylisascaris procyonis - The common raccoon ascarid is known to cause life-threatening visceral, cerebral and ocular larva migrans in birds in North America.[23,32] Infected asymptomatic raccoons can harbor a large number of worms and shed large numbers of infective eggs into the environment.[23] Affected birds can develop ataxia and dysmetria that progresses to inability to stand, with severe torticollis and a head tilt.[23]

Trichomonas gallinae causes disease in the domestic pigeon and birds that feed on pigeons such as eagles, falcons and hawks; it has been responsible for avian enteric trichomoniasis.[89] Caseous masses in the roof of the mouth may extend to involve the brain.

Schistosomiasis causing granulomatous encephalitis has been reported in swans.[15]

Protozoal Encephalitis

Toxoplasma gondii, a cyst-forming coccidium, is not frequently identified as pathogenic in captive birds. Asymptomatic infection with this parasite is reportedly common.[74] Outbreaks of toxoplasmosis have been reported worldwide in chickens, and in passerine and psittacine birds and birds exhibited in zoos.[74,122] Naturally occurring infections have been diagnosed in chickens, ducks and many wild birds.[122] Because toxoplasmosis is a zoonotic disease, the source of the infection should be determined, if possible, to prevent infection of humans. Household cats should be tested. Common clinical signs of *T. gondii* infection in avian species include anorexia, blindness, head tilt, circling and ataxia.[74,122] Canaries (*Serinus canaria*) may be susceptible to a form of toxoplasmosis of the central nervous system and eyes that differs from the acute form seen in other species.[74]

Partridges (*Perdix perdix*) have been reported to be more susceptible to toxoplasmosis than other gallinaceous birds.[104]

Serological antibody titers have been reported (latex agglutination test) for infected birds, but have not been helpful. A single titer can rarely specifically diagnose the disease when the birds have been previously exposed.[42] Treatment of suspected cases and all in-contact birds with trimethoprim 0.08 g/ml H_2O and sulfadiazine 0.04 g/ml in water for 3 weeks has been advised, but eradication from a captive flock may be difficult.[122]

Trichomonas gallinae[89]

Leucocytozoonosis[95]

Sarcocystis spp. have been reported to cause toxoplasma-like infections of birds with a non-suppurative meningoencephalitis.[49] Infection has been reported in over 60 species of birds, with Old World psittacines apparently more susceptible (see Fig 17.25).[15]

This disease has been reported in 53 capercaillies (*Tetrao urogallus*) examined at necropsy in Sweden and one capercaillie from Finland.[34,49] Protozoa were mainly confined to the brain, but systemic disease is common.[34] Little is known about sarcocystosis of birds in general. In North America, *Sarcocystis falcatula* is the most pathogenic and well-defined species among birds, with schizonts persisting in the tissues for up to 5.5 months.[15,49] The predominant lesion in fatal *S. falcatula* infection is pneumonia, however, *Sarcocystis*-associated encephalitis has been described in a golden eagle (*Aquila chrysaetos*) and in a straw-necked ibis (*Carphibis spinicollis*) in the USA.[35,49]

Fungal Encephalitis

Mucormycosis is a rare infection in birds.[84] The order Mucorales includes a number of saprophytic fungi that have been mentioned as possible etiologic agents of meningoencephalitis in birds.[84]

Spongiform Encephalopathy

Three cases of a spongiform encephalopathy of unknown etiology have been reported in ostriches (*Struthio camelus*) from two zoos in northwestern Germany.[63] The birds were euthanized after showing progressive ataxia and uncoordinated feeding behavior. The lesions identified by light microscopy were similar to those of prion-induced transmissible encephalopathies in mammals and had gray matter vacuolation. However, a toxic or nutritional etiology could not be ruled out.[63]

Idiopathic

Epilepsy

Intermittent seizures with no other abnormality can indicate idiopathic disease, especially if there is a long history of seizures.[100] In chickens, epilepsy has been found to have genetic basis.[100] Idiopathic epilepsy also may be acquired as a result of a cerebral insult and residual brain damage.[100]

Toxicity

Lead[100]/Zinc[73]

Caged birds that eat their cage wire can become intoxicated if the wire is high in zinc content. A new wire fence syndrome has been reported in emus.[11] The birds gnaw away at their cage made of zinc material and eventually ingest so much that toxicity occurs. Some galvanized coatings contain as much as 99.9% zinc, but galvanized wire also may contain lead. The white rust associated with galvanized wire also is toxic.[11] Waterfowl may be exposed to elevated zinc concentrations through ingestion of contaminated vegetation and sediments due to the recently approved zinc-coated iron shot used for waterfowl hunting in the USA.[73]

Seizures can be a common clinical sign of zinc toxicity, in conjunction with paresis, polyuria/polydipsia, weight loss, anemia and gastrointestinal abnormalities. Sudden death has been reported in orange-bellied parrots (*Neophema chrysogaster*) due to zinc toxicoses, however, it was thought to be a result of the birds colliding with walls or structures in the aviary, causing head trauma.[55] Serum concentrations of zinc can be used to confirm the diagnosis and should be compared to normal birds as controls. Syringes with rubber stoppers should be avoided as this may be a source of zinc contamination.[11] In general, zinc levels greater than 2 ppm in emu are considered diagnostic for toxicity. Tissue levels can be evaluated postmortem in suspicious cases.[55] Published reference values of zinc levels in tissues of normal birds are as follows: macaws, 75 μg/g (tissue unspecified);[55] cockatiels (*Nymphicus hollandicus*), 59 μg/g in the kidneys, 72 μg/g in the liver and 94 μg/g in the pancreas;[55] goldeneye ducks (*Bucephala islandica*), 35.9 μg/g in the liver;[55] and peach-faced lovebirds (*Agapornis roseicollis*), 21 and 33 μg/g in the liver.[55] Treatment is discussed in Chapter 31, Implications of Toxins in Clinical Disorders.

Organophosphates, Carbamates and Pesticides

There are over 80 different registered organophosphates and carbamates in the USA, with both classes being acetylcholinesterase inhibitors that bind to and subsequently inactivate acetylcholinesterase, causing an accumulation of acetylcholine at the postsynaptic receptors.[15] Birds are 10 to 20 times more susceptible to these inhibitors than mammals. Acute intoxications can cause seizure activity. Exposure to carbamate insecticides can be detected using brain cholinesterase reactivation techniques.[5,56,105]

Johnathon Cracknell

Fig 17.23 | A rose-crowned fruit dove (*Ptilinopus regina*) is examined after head trauma, demonstrating superficial skin lesions.

Trauma

Head Trauma (Fig 17.23)[15,53]

Vascular

Atherosclerosis

Atherosclerosis occurs with some degree of frequency in birds.[15] Often this can cause acute death, but can cause sudden onset blindness, ataxia, paresis and seizures.[15] Clinical signs may be from the cerebrovascular accidents, and MRI or CT may be useful in their diagnosis.[15] See Chapter 12, Evaluating and Treating the Cardiovascular System.

Spinal and Neuromuscular Disease

CLINICAL SIGNS (Table 17.5)

Monoparesis/Monoplegia/Hemiparesis/Hemiplegia

Definitions and Classifications

Paresis indicates the presence of reduced motor function in the limbs. The suffix "-plegia" indicates the complete loss of motor function. The prefix mono- indicates the involvement of just one limb (Fig 17.24) and the prefix hemi- indicates the involvement of both limbs on one side of the body with normality on the contralateral side. Monoparesis sometimes may be expressed as a "clenched" claw (Fig 17.25).

Treatment

1. Specific treatment of the underlying etiology.
2. Skin care for recumbent birds or those that are dragging their limbs.
3. Physiotherapy.
4. Supportive care if unable to eat and drink.

Tetraparesis/Paraparesis/Paralysis

Definitions and Classifications

The prefix tetra- indicates the involvement of all four limbs. Tetraplegia is very rare due to the negative effect such a lesion would have on the respiratory capabilities of the patient. The prefix para- indicates the involvement of just the pelvic limbs, and paralysis or the equivalent paraplegia indicates complete loss of motor movement in the pelvic limbs (Fig 17.26).

Treatment

1. Specific treatment of the underlying etiology.
2. Skin care for recumbent birds or those that are dragging their limbs.
3. Physiotherapy.
4. Supportive care if unable to eat and drink.

Ataxia

Definitions and Classifications

Ataxia indicates an uncoordinated gait and does not necessitate an impairment of motor function, but there may be a concurrent paresis. Ataxia is often due to spinal disease, but may be due to either vestibular or cerebellar disease. There will be no motor dysfunction in the case of pure cerebellar ataxia.

Table 17.5 | Spinal Lesion Localization and Clinical Signs[93]

Clinical Syndrome/ Lesion Localization	Characteristics of the Location	Specific Clinical Signs
Cranial cervical spine	Damage to cranial cervical spinal cord reflects damage to peripheral white matter pathways	i. No cranial nerve deficits ii. UMN[a] wing, leg and vent signs iii. Possible neck pain iv. Very rare loss of pain perception
Cervicothoracic spine	Damage to the cervical intumescence produces clinical signs that reflect damage to the grey matter and brachial plexus[b]	i. No head signs ii. LMN[c] wing signs iii. UMN leg and vent signs unless brachial plexus lesion, only (ii)
Thoracolumbar spine	Damage to spine caudal to cervical intumescence[e] and cranial to lumbar intumescences reflects white matter lesions	i. No head or wing signs ii. UMN leg and vent signs iii. Possible kyphosis, spinal ± abdominal pain[d] iv. Variable loss of pain perception in limbs
Lumbosacral syndrome	A lesion in the lumbar intumescence[e] reflects damage to central gray matter and lumbosacral plexi[f]	i. No head or wing signs ii. LMN leg signs iii. LMN vent signs unless peripheral limb nerves only affected iv. Variable loss of sensation in legs and vent

a UMN = upper motor neuron. See Table 17.2.
b Brachial plexus includes the pectoral, medianoulnar, bicipital, ventral propatagial, axillary and radial peripheral nerves.
c LMN = lower motor neuron. See Table 17.2.
d A space-occupying mass within the kidney or pelvic canal may place pressure on lumbosacral plexus and its ischiatic nerve, mimicking lumbosacral syndrome.
e Lumbar intumescence encompasses lumbar, sacral and pudendal plexi.
f Lumbosacral plexi includes the femoral, obturator, ischiatic, pudendal, pelvic and coccygeal peripheral nerves.

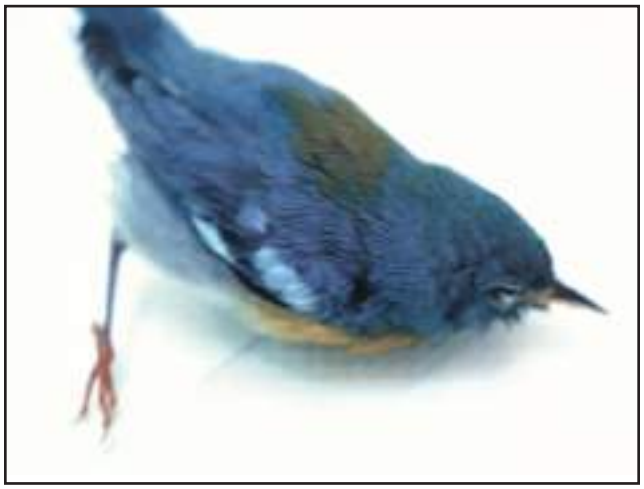

Greg J. Harrison

Fig 17.24 | A palm warbler (*Dendroica palmarum*) with pesticide toxicosis demonstrating incoordination.


Greg J. Harrison

Fig 17.26 | A cockatiel (*Nymphicus hollandicus*) with a spinal lesion of unknown etiology demonstrates a posture often seen with paraparesis.

Treatment

1. Specific treatment of the underlying etiology.
2. Skin care for recumbent birds or those that are dragging their limbs.
3. Physiotherapy.
4. Supportive care if unable to eat and drink.

DIFFERENTIAL DIAGNOSIS OF SPINAL AND NEUROMUSCULAR DISEASE (Table 17.6)

Degenerative

Psittacine Focal Symmetrical Poliomyelomalacia

This lesion has been documented in 15 adult birds in Australia, including five superb parrots, two budgerigars and one lovebird (*Agapornis* sp), all of which were from aviaries.[53] In caged birds, there was a history of one or more birds suddenly becoming uncoordinated, having difficulty perching, followed by posterior paralysis. Some birds could still fly. Others became tetraplegic within 2 or 3 days.[53]


Greg J. Harrison

Fig 17.25 | An African grey parrot (*Psittacus erithacus*) with suspected *Sarcocystis* infection of the central nervous system exhibits torticollis and bilateral clenched claws.

Table 17.6 | Differential Diagnosis of Spinal and Neuromuscular Disease

Mechanism of Disease	Specific Diseases
Degenerative	i. Psittacine focal symmetrical Poliomyelomalacia ii. Lysosomal storage disease - Gangliosidosis iii. Avian vacuolar myelinopathy iv. Lafora
Neoplastic	i. Abdominal neoplasia - ovarian pathology/renal masses
Nutritional	i. Vitamin E deficiency ii. Thiamine (vitamin B_1) deficiency iii. Riboflavin (vitamin B_2) deficiency iv. Pyridoxine (vitamin B_6) deficiency
Inflammatory	i. Viral - Proventricular dilatation disease/Marek's disease /Avian paramyxovirus type 1 (Newcastle disease)/Polioencephalomyelitis of rainbow lorikeets/Borna/Eastern and western equine encephalitis/West Nile virus/Picornavirus (duck viral hepatitis) ii. Fungal - *Aspergillus* spp.
Toxicity	i. Lead ii. Zinc iii. Tick Paralysis iv. Organophosphates, carbamates and insecticides v. Plant toxins vi. Botulism
Trauma	i. Peripheral nerve trauma ii. Spinal trauma
Vascular	i. Fibrocartilaginous embolic myelopathy

Lysosomal Storage Disease

Gangliosidoses are most commonly caused by deficient activity in one of the two enzymes necessary for ganglioside catabolism.[18] Specifically, an inherited defect in ß-galactosidase is associated with GM_1 gangliosidosis and a similar defect affecting the activity of ß-hexosaminidases is associated with GM_2 gangliosidosis.[18] The enzymatic defects lead to an accumulation of GM_1 or GM_2 respectively, within the neurons of the central and peripheral nervous system. Two related juvenile emus have been reported with severe, progressive, eventually fatal intraneuronal gangliosidosis.[18] Analysis of affected brain tissue demonstrated significant elevations in total ganglio-

sides with low levels of lymphocyte ß-galactosidase.[18]

Avian Vacuolar Myelinopathy (AVM)

AVM has been diagnosed in wild birds in the southeastern USA. It was first documented in bald eagles (*Haliaeetus leucocephalus*) in 1994 and also has been found in American coots (*Fulica americana*).[115] Bald eagles are frequently found dead, but have been noted to have difficulty flying and can crash into or overfly perches. Affected coots appear to fly or are wobbly in flight; they are uncoordinated on land and may swim in circles or on their backs.[71] On neurologic examination, the majority of coots have been documented with ataxia, decreased withdrawal reflexes, proprioceptive deficits and decreased vent responses.[71] Other signs seen in less than half of examined coots include beak and tongue weakness, head tremors, absent pupillary light responses, anisocoria, apparent blindness and nystagmus.[71]

Birds that die have no gross lesions of the nervous system, and some coots have been documented to recover from this disorder with supportive care.[71] Histologically, the disease is characterized by diffuse, spongy degeneration throughout the white matter of the CNS with the optic tectum most severely affected.[71] The etiology remains unknown.[115]

Lafora's Disease

Lafora's disease is a presumed inherited defect of carbohydrate metabolism first documented in humans.[103] This disturbance of the carbohydrate metabolism causes the formation of typical Lafora-bodies, which consist of polysaccharide complexes. Similar changes have recently been described in two cockatiels (*Nymphicus hollandicus*).[15]

Neoplasia

Primary spinal neoplasia is rare in birds. Compressive peripheral nerve trauma generally occurs secondary to an expanding mass that applies pressure to the nerve, because the pelvic nerves pass through the renal parenchyma.[15]

Nutritional

Vitamin E/Selenium Deficiency

Clinical signs associated with vitamin E and selenium deficiencies include tremors, ataxia, incoordination, reluctance to walk and recumbency.[15] This deficiency has been incriminated as the etiology of cockatiel paralysis syndrome. This condition appears to occur most frequently in lutino cockatiels, but similar syndromes have been reported in a variety of other species including blue and gold macaws, eclectus parrots and African grey parrots.[15] Supplementation with injectable and oral vitamin E is the recommended treatment, however, the patient may or may not respond, depending on the severity of damage.

Thiamine (Vitamin B_1) Deficiency

Clinical signs of thiamine deficiency include ataxia, ascending paralysis and opisthotonus.[15] A response to treatment provides a presumptive diagnosis, as affected birds generally respond within hours of oral or parenteral administration of vitamin B_1.[15]

Riboflavin (Vitamin B_2) Deficiency

Birds with this deficiency have weakness, atrophy of the leg muscles and are seen to walk on their hocks with their toes curled inward, although this does not always happen because death may occur first.[15,118] The condition has occasionally occurred in chickens, but since riboflavin has been added to poultry feed it has become quite rare.[118] Leg and wing paralysis has been reported in racing pigeons with this disorder.[118] The deficiency causes a demyelinating peripheral neuritis. Treatment involves administration of oral or parenteral riboflavin and diet correction.

Pyridoxine (Vitamin B_6) Deficiency

Deficiency of this vitamin causes characteristic jerky, nervous walking, progressing to running and flapping the wings.[15]

Inflammatory

Viral Disease

Proventricular Dilation Disease (PDD) has been reported to cause peripheral (sciatic, brachial and vagal) neuritis in psittacine birds.[17]

Marek's Disease Virus (MDV) in chickens is caused by an oncogenic herpesvirus and is characterized by lymphomas and paralysis.[43] Paralysis associated with MDV infection has usually been attributed to peripheral nerve lesions because gross enlargement of nerves with lymphoid infiltrates is a common feature of MD, and CNS damage has not been found consistently in paralyzed birds.[43] The onset of both lymphomas and paralysis usually occurs 4 to 12 weeks after infection with MDV.[43] A paralytic syndrome involving the brain also is induced by MDV and is now designated transient paralysis (TP).[43] Clinically, TP is characterized by the development of a flaccid paralysis 8 to 12 days after infection with an oncogenic MDV. This initially affects neck muscles and later tends to become generalized.[43] Most birds with TP recover completely within 24 to 48 hours, although a few birds that become severely affected may die within the same period of time.[43] A more acute and fatal form of TP has recently been described in young chickens.[123]

Once known as fowl paralysis and range paralysis, it was considered that gross enlargements of the peripheral nerves were a pathognomic lesion.[123] However, gross enlargement of peripheral nerves also can be induced by reticuloendotheliosis virus (REV).[7] Both REV and

subgroup J avian leucosis virus can induce lymphoid infiltrations in peripheral nerves, indicating that additional criteria may be needed for confirmation of etiology.[7]

Avian Paramyxovirus Type 1 (Newcastle Disease) is a variable disease with different susceptibilities between avian orders.[8] The disease is produced following infection and varies with virus pathotype; other factors include the species, age, immune status, general health of the bird and environmental conditions.[8] Clinical neurologic findings in affected birds include ataxia, torticollis, opisthotonus, head shaking and tremors, head tilt, leg paralysis progressing to lateral recumbency and blindness.[8] Some birds may survive for up to 18 months after developing CNS signs, but the CNS signs do not regress before death or euthanasia.[8] Definitive diagnosis requires virus isolation, demonstration of the viral antigen, or rising specific antibody titers.[8] A titer of 1:8 is usually considered evidence of exposure to this virus, and in unvaccinated birds when coupled with clinical signs is considered strong diagnostic evidence of infection.[8]

Suspected Viral Polioencephalomyelitis of Rainbow Lorikeets (*Trichoglossus haematodus*) has been documented in 35 rainbow lorikeets in Australia.[53] It affects adults at any time of the year and usually causes an inability to fly or perch. The birds are commonly alert and will eat, but are characteristically affected with clenched claws.[53] The cerebellum was affected in about half of the birds, and the lesions were largely restricted to the central cerebellar white matter and involved the roof nuclei.

Borna Disease Virus has been documented to cause a paretic condition in young ostriches in Israel.[4] Birds between 14 and 42 days old were affected. In a small number of cases, clinical signs of incoordination were seen 1 to 3 days before paresis supervened. At this stage of the disease, the birds could stand and move with difficulty if given external support.[4] Mortality can be remarkably high. About 1% of the ostriches seemed to recover if they were given intensive supportive care, however, they relapsed several months later.[4]

Eastern and Western Equine Encephalitis infections can cause weakness and paralysis of the legs.[96]

Picornavirus (Duck Viral Hepatitis) has been associated with neurologic signs in Galliformes, Anseriformes and Columbiformes of less than 28 days of age.[15] Clinical signs documented include depression, ataxia, paresis or paralysis, and severe but fine head and neck tremors.

West Nile Virus has been documented to cause paresis and paralysis in many avian species.[78]

Fungal Disease[21,22,46,102]

Toxicity

Lead

Lead toxicity, one of the most commonly recognized poisonings of companion and free-ranging birds, is a common cause of neurologic abnormalities and can be fatal.[92] Peripheral neuropathies caused by lead intoxication have been reported, but it is more common to see central nervous system abnormalities.[92] Various sources of lead have been documented, but the source of lead in any specific poisoning often remains difficult to determine. A classic source of lead exposure for birds is ingesting lead shot from the bottom of lakes in heavily hunted areas.[92] The combination of the grinding action of the ventriculus and its low pH (2.0-3.5) acts to solubilize ingested shot.[92] Raptors may eat carcasses containing lead shot and become intoxicated.[92] Inhalation of fumes from leaded gasoline is another possible route of exposure.[92] Lead poisoning as a result of lead shot embedded in soft tissues is rare because of the avascular fibrous tissue that develops around these foreign bodies.[92]

Clinically, lead toxicosis may be an acute or chronic problem, with clinical signs dependent on the amount and surface area of lead ingested.[92] Clinical signs in birds include behavioral changes, lethargy, anorexia, vomiting, diarrhea, ataxia, limb paresis or paralysis, seizures, anemia and emaciation.[92] Death may occur within 48 hours after the first appearance of clinical signs.[92]

Whole, unclotted blood is the sample of choice for determining lead concentrations because 90% of circulating lead is contained within RBCs.[92] Concentrations of >20 μg/dl lead in whole blood are suggestive of lead intoxication in psittacine birds, and concentrations >40 to 60 μg/dl are diagnostic of lead toxicosis.[92]

Zinc[73]

See Intracranial Disease above.

Tick Paralysis

Tick paralysis is a disease caused by neurotoxins associated with 60 species of hard and soft ticks in 10 genera.[76] Generally, the disease is considered a motor polyneuropathy characterized by a progressive, ascending, flaccid motor paralysis. Clinical signs include ataxia, paresis, paralysis, areflexia, hypotonus and respiratory failure.[76] Death is common if the tick is not removed.[76] The toxin is associated with the female tick's salivary glands and is probably secreted during feeding.[76] *Ixodes brunneus* has been associated with tick paralysis in birds.[76] All three stages of the tick feed exclusively on birds, particularly passerines and group-feeding species; at least 64 species of birds are known to be hosts for this tick.[76] Adult females are found on adult birds primarily in the colder

months of the year, with larvae and nymphs occasionally present at the same time, causing clinical signs during winter months.[76]

A definitive diagnosis of tick paralysis can be based only on dramatic clinical improvement and recovery of the host, usually within 24 to 72 hours following removal of the tick.

Organophosphates and Carbamates

Organophosphorus-induced delayed neurotoxicity (OPIDN) is a neurologic condition characterized clinically by the delayed onset of a progressively developing hindlimb ataxia and paralysis.[90,116] Two categories of OPIDN — Type I and Type II — have been identified,[116] and differ from each other in the length of the delay period prior to onset of symptoms, the type of resultant clinical signs and the extent of central nervous system involvement. Type I OPIDN has a longer delay period (typically 10-21 days) and affects only the spinal cord and brainstem, whereas type II OPIDN has a shorter delay period (4-7 days) and results in additional degeneration in the midbrain and forebrain. Widespread neuropathology follows both oral and injectable forms of the compounds.[116]

Plant Toxins

Nerium oleander[2] (See Chapter 31, Implications of Toxic Substances in Clinical Disorders.

Botulism (Limberneck)

Botulism in birds is usually the result of ingestion of the exotoxin of *Clostridium botulinum* type C. Occasionally *C. botulinum* type A and type E are involved.[15] This is an uncommon problem in companion birds but frequent in waterfowl.[15,45,70] The classic sign is limberneck resulting from paralysis of the cervical muscles.[15] Most birds exhibit hindlimb paresis first, which progresses to paralysis of the wings, followed by loss of control of the neck and head in the terminal stages.[15] Species' susceptibility and clinical manifestations vary, as has been evidenced in rehabilitation facility outbreaks.

Trauma

Spine Trauma/Peripheral Nerve Trauma

Trauma is fairly common in free-ranging as well as companion and aviary birds. The consequences, diagnosis and management of these traumas have been well discussed in other texts.[15]

Vascular

Fibrocartilaginous Embolization

Emboli may involve vessels of the spinal cord, leptomeninges or both. Fibrocartilaginous embolism (FCE) and ischemic myelopathy have been reported in several 15-week-old tom turkeys with peracute onsets of paresis and ataxia.[111] Recovery was noted in some affected birds suspected to have this disease, but cartilaginous emboli were found in the spinal cord vasculature accompanying myelomalacia in three turkeys that did not recover.[111] The articular cartilage of the vertebral body endplates are suggested to be the source of the emboli, as there were articular cartilage defects found in the affected birds. There is no known treatment in any species and diagnosis is by exclusion. Recovery is possible in some cases, but supportive care would be necessary.

Products Mentioned in the Text

a. Lanex fine screens/TML film, Kodak, Rochester, NY
b. Omnipaque 240, Winthrop Pharmaceuticals, New York, NY
c. Magnevist, Berlix Laboratories, Wayne, NJ

References and Suggested Reading

1. Abou-Donia MB, et al: Electromyographic, neuropathologic and functional correlates in the cat as the result of tri-o-cresyl phosphate delayed neurotoxicity. Toxicol Appl Pharmacol 83:126-141, 1986.
2. Alfonso HA, Sanchez LM: Intoxication due to *Nerium oleander* in geese. Vet Human Toxicol 36(1):47, 1994.
3. Armstrong DL, et al: Cerebrospinal nematodiasis in macaws due to *Baylisascaris procyonis*. J Zoo Wildl Med 20(3):354-359, 1989.
4. Ashash E, et al: Causes of losses including a Borna disease paralytic syndrome affecting young ostriches of one breeding organization over a five-year period (1989-1993). Avian Dis 40:240-245, 1996.
5. Augspurger T, et al: Mortality of passerines adjacent to a North Carolina corn field treated with granular carbofuran. J Wildl Dis 32(1):113-116, 1996.
6. Aye PP, et al: Encephalomalacia associated with vitamin E deficiency in commercially raised emus. Avian Dis 42:600-605, 1998.
7. Bacon LD, Witter RL, Silva RF: Characterization and experimental reproduction of peripheral neuropathy in white leghorn chickens. Avian Pathol 30:487-499, 2001.
8. Bailey TA, et al: Avian paramyxovirus type 1 infection in Houbara bustards (*Chlamydotis undulata MacqueenII*): Clinical and pathological findings. J Zoo Wildl Med 28(3):325-330, 1997.
9. Bagley RS, Wheeler SJ, James RL: Age-related effects on motor nerve conduction velocity in chickens. Am J Vet Res 53:1309-1311, 1992.
10. Bagley RS, Wheeler SJ, Gay JM: Effects of age on temperature related variation in motor nerve conduction velocity in healthy chickens. Am J Vet Res 56(6):819-821, 1995.
11. Baptiste K: New wire fence syndrome in emus. Proc MASAAV, 1998, p 208.
12. Bartels T, et al: The use of conventional radiography and computer-assisted tomography as instruments for demonstration of gross pathological lesions in the cranium and cerebrum in the crested breed of the domestic duck (*Anas platyrhynchos* f. dom.). Avian Pathol 29:101-108, 2000.
13. Bartels T, et al: Magnetic resonance imaging of intracranial tissue accumulations in domestic ducks (*Anas platyrhynchos* f. dom) with feather crests. Vet Radiol Ultrasound 42(3):254-258, 2001.
14. Bartels T, et al: Ataxia and disequilibrium in domestic ducks (*Anas platyrhynchos* f. dom.) with intracranial lipomas. Vet Pathol 39:396-399, 2002.
15. Bennet RA: Neurology. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 728-747.
16. Benzo CA: Nervous system. *In* Sturkie PD (ed): Avian Physiology 4th ed. New York, Springer-Verlag, 1986, pp 1-37.
17. Berhane Y, et al: Peripheral neuritis in psittacine birds with proventricular dilatation disease. Avian Pathol 30:563-570, 2001.
18. Bermudez AJ, et al: Gangliosidosis in emus (*Dromaius novaehollandiae*). Avian Dis 39:292-303, 1995.
19. Bicknese EJ: Review of avian sarcocystosis. Proc Assoc Avian Vet, 1993, pp 52-58.
20. Bravo H, Insunza O: The oculomotor nucleus, not the abducent, innervates the muscles which advance the nictitating membrane in birds. Acta Anat 122:99-104, 1985.
21. Brown PA, Redig PT: *Aspergillus* ELISA: A tool for detection and management. Proc Assoc Avian Vet, 1994, pp 295-300.
22. Bygrave AC: Leg paralysis in pheasant poults (*Phasianus colchicus*) due to spinal aspergillosis. Vet Rec 109:516, 1981.
23. Campbell GA, et al: Naturally occurring cerebral nematodiasis due to *Baylisascaris* larval migration in two black-and-white ruffed lemurs (*Varecia variegata variegata*) and suspected cases in three emus (*Dromaius novaehollandiae*). J Zoo Wildl Med 28(2):204-207, 1997.
24. Chamanza R, et al: *Enterococcus*-associated encephalomalacia in one-week-old chicks. Vet Rec 143:450-451, 1998.
25. Chrisman CL: Cerebrospinal fluid analysis. Vet Clin No Am Small Anim Pract 22(4):781-810, 1992.
26. Clippinger TL, Bennett RA, Platt SR: The avian neurologic examination and ancillary neurodiagnostic techniques. J Avian Med Surg 10(4):221-247, 1996.
27. Clippinger TL, et al: Electrodiagnostic evaluation of peripheral nerve function in rheas and barred owls. Amer J Vet Res 61(4):469-472, 2000.
28. Clippinger TL, Platt SR: Seizures. *In* Olsen GH, Orosz, SE (eds): Manual of Avian Medicine. St. Louis, MO, Mosby Inc, 2000, pp 170-182.
29. Coleman CW, Oliver R: Lymphosarcoma in a juvenile blue and gold macaw (*Ara ararauna*) and a mature canary (*Serinus canarius*). J Assoc Avian Vet 8(2):64-68, 1994.
30. Comunale JP, Han SS, Wang LC: Lumbar myelography: Side effects correlated with exam findings and needle size. Spine 19:1063-1065, 1994.
31. Cooper JE, Harrison GJ: Dermatology. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 607-639.
32. Cunningham CK, et al: Diagnosis and management of *Baylisascaris procyonis* infection in an infant with nonfatal meningoencephalitis. Clin Infect Dis 18:868-872, 1994.
33. Dubbeldam JL: Motor control system. *In* Sturkie PD (ed): Avian Physiology 5th ed. New York, Springer-Verlag, 2000, pp 83-99.
34. Dubey JP, Rudback E, Topper MJ: *Sarcocystis* in capercaillie (*Tetrao urogallus*) in Finland: Description of the parasite and lesions. J Parasitol 84(1):104-108, 1998.
35. Dubey JR, et al: Neural sarcocystosis in a straw-necked ibis (*Carphibis spinicollis*) associated with a *Sarcocystis* neurona-like organism and description of muscular sarcocysts of an unidentified *Sarcocystis* species. J Parasitol 87(6):1317-1322, 2001.
36. Dubey JR, et al: Disseminated toxoplasmosis in Magpie Geese (*Anseranas semipalmata*) with large numbers of tissue cysts in livers. J Parasitol 87(1):219-223, 2001.
37. du Lac S, Lisberger SG: Eye movements and brainstem neuronal responses evoked by cerebellar and vestibular stimulation in chicks. J Comp Physiol 171:629-638, 1992.
38. Emerson CL, et al: Ruptured intervertebral disc in a juvenile king penguin (*Aptenodytes Patagonia*). J Zoo Wildl Med 21(3):345-350, 1990.
39. Evans HE: Anatomy of the budgerigar. *In* Petrak ML: Diseases of Cage and Aviary Birds 2nd ed. Philadelphia, Lea and Febiger, 1982, pp 111-187.
40. Fenner WR: Diseases of the brain. *In* Ettinger SJ, Feldman EC (eds): Textbook of Veterinary Internal Medicine 4th ed. Philadelphia, WB Saunders Co, 1995, pp 578-629.
41. Garmendia AE, et al: Recovery and identification of West Nile virus from a hawk in winter. J Clin Microbiol 38(8):3110-3111, 2000.
42. Gibbens JC, Abraham EJ, MacKenzie G: Toxoplasmosis in canaries in Great Britain. Vet Rec 140:370-371, 1997.
43. Gimeno IM, Witter RL, Reed WM: Four distinct neurologic syndromes in Marek's disease: Effect of viral strain and pathotype. Avian Dis 43:721-737, 1999.
44. Goodwin MA, Brown J, Waltman WD: Neonatal multifocal encephalomalacia and other lesions in the brains of Georgia chicks: 1991-95. Avian Dis 40:894-899, 1996.
45. Goulden S: Botulism in water birds. Vet Rec 137(13):328, 1995.
46. Greenacre CB, Latimer KS, Ritchie BW: Leg paresis in a black palm cockatoo (*Probosciger aterrimus*) caused by aspergillosis. J Zoo Wildl Med 23(1):122-126, 1992.
47. Greenwood AG, Marshall J, Tinsley EGF: Vegetative endocarditis in a Waldrapp ibis. Avian Pathol 25:387-391, 1996.
48. Gregory CR, et al: A review of proventricular dilatation syndrome. J Assoc Avian Vet 8(2):69-75, 1994.
49. Gustafson K, et al: Meningoencephalitis in capercaillie (*Tetrao urogallus* L.) caused by a *Sarcocystis*-like organism. J Zoo Wildl Med 28(3):280-284, 1997.
50. Gustafson CR, et al: *Pasteurella multocida* infection involving cranial air spaces in white leghorn chickens. Avian Dis 42:413-417, 1998.
51. Harr KE, et al: A myelographic technique for avian species. Vet Radiol Ultrasound 38(3):187-192, 1997.
52. Harrison GJ: Long term primidone use in an Amazon. Assoc Avian Vet Newsletter 4:89, 1982.
53. Hartley WJ, Reece RL: Nervous diseases of Australian native and aviary birds. Aust Vet Prac 27(2):91-96, 1997.
54. Hathcock JT, Stickle RL: Principles and concepts of computed tomography. Vet Clin No Am Small Anim Pract 23(2):400-415, 1993.
55. Holz P, et al: Suspected zinc toxicosis as a cause of sudden death in orange-bellied parrots (*Neophema chrysogaster*). J Avian Med Surg 14(1):37-41, 2000.
56. Hunt KA, Hooper MJ, Edward EL: Carbofuran poisoning in herons: Cholinesterase reactivation techniques. J Wildl Dis 31(2):186-192, 1995.
57. Ibrahim RS, Moustafa FA, Mubarak M: Paramyxovirus infection in pigeons. Vet Med J 44(87):206-222, 2000.
58. Jenkins JR: Use of computed tomography (CT) in pet bird practice. Proc Assoc Avian Vet, 1991, pp 276-279.
59. Kern TJ, et al: Disorders of the third eyelid in birds: 17 cases. J Avian Med Surg 10(1):12-18, 1996.
60. Kim DY, Cho DY, Taylor HW: Lysosomal storage disease in an emu (*Dromaius novaehollandiae*). Vet Pathol 33:365-366, 1996.
61. Kimura J: Electrodiagnosis in diseases of nerve and muscle: Principles and practice 2nd ed. Philadelphia, FA Davis, 1993, pp 83-84.
62. King AS, McClelland J: Birds - Their Structure and Function 2nd ed. London, Bailliere Tindall, 1984, pp 237-315.
63. Kirkwood JK, Cunningham AA: Epidemiological observations on spongiform encephalopathies in captive wild animals in the British Isles. Vet Rec 135:296-303, 1994.
64. Klappenbach KM: Cerebral spinal fluid analysis in psittacines. Proc Assoc Avian Vet 1995, pp 39-42.
65. Klappenbach KM: Neuroelectrodiagnostics in psittacines. Proc Assoc Avian Vet 1995, pp 37-38.
66. Klein DR, Novilla MN, Watkins KL: Nutritional encephalomalacia in turkeys: Diagnosis and growth performance. Avian Dis 38:653-659, 1994.
67. Kornegay JN, et al: Motor nerve conduction velocity in normal chickens. Am J Vet Res 44:1537-1540, 1983.
68. Krautwald-Junghanns ME, Tellhelm B: Advances in radiography of birds. Sem Avian Exotic Pet Med 3(3):156-160, 1994.
69. Kuiken T, et al: Pathology of Newcastle disease in double-crested cormorants from Saskatchewan, with comparison of diagnostic methods. J Wildl Dis 35(1):8-23, 1999.
70. Kurtdede A, Sancak AA: Botulism in a long-legged buzzard (*Buteo rutinus*). Vet Rec 151(2):64, 2002.
71. Larsen RS, et al: Clinical features of avian vacuolar myelinopathy in American coots. J Am Vet Med Assoc 221(1):80-85, 2002.
72. Latimer KS, et al: Polyomavirus encephalopathy in a Ducorps's cockatoo (*Cacatua ducorpsii*) with psittacine beak and feather disease. J Vet Diagn Invest 8:291-295, 1996.

73. Levengood JM, et al: Influence of diet on the hematology and serum biochemistry of zinc-intoxicated mallards. J Wildl Dis 36(1), 2000.
74. Lindsay DS, et al: Central nervous system toxoplasmosis in roller canaries. Avian Dis 39:204-207, 1995.
75. Lung NP, Ackerman N: Scintigraphy as a tool in avian orthopedic diagnosis. Proc Am Assoc Zoo Vet, 1993, p 45.
76. Luttrell MP, Creekmore LH, Mertins JW: Avian tick paralysis caused by *Ixodes brunneus* in the Southeastern United States. J Wildl Dis 32(1):133-136, 1996.
77. Love N, Flammer K, Spaulding K: The normal computed tomographic (CT) anatomy of the African grey parrot (*Psittacus erithacus*): A pilot study. Proc Am Coll Vet Radiol, 1993, p 28.
78. Malikinson M, et al: Use of live and inactivated vaccines in the control of West Nile fever in domestic geese. Ann NY Acad Sci 951:251-261, 2001.
79. Mateo R, et al: Lead poisoning in a free ranging griffon vulture (*Gyps fulvus*). Vet Rec 140:47-48, 1997.
80. Moore MP: Approach to the patient with spinal disease. Vet Clin No Am Small Anim Pract 22(4):751-780, 1992.
81. Murgue B, et al: West Nile in the Mediterranean basin: 1950-2000. Ann NY Acad Sci 951:117-126, 2001.
82. Murphy CJ: Raptor ophthalmology. Compend Cont Educ Vet 9(3):241-258, 1987.
83. Necker R: Functional organization of the spinal cord. *In* Sturkie PD (ed): Avian Physiology 5th ed. New York, Springer-Verlag, 2000, pp 71-81.
84. Orcutt CJ, Bartrick TE: Mucormycotic meningoencephalitis and pneumonia in a chattering lory (*Lorius garrulus*). J Assoc Avian Vet 8(2):85-89, 1994.
85. Orosz SE, Ensley PK, Haynes CJ: Avian Surgical Anatomy-thoracic and Pelvic Limbs. Philadelphia, WB Saunders Co, 1992.
86. Orosz SE, Toal RL: Tomographic anatomy of the golden eagle (*Aquila chrysaetos*). J Zoo Wildl Med 23(1):39-46, 1992.
87. Orosz SE: Principles of avian clinical neuroanatomy. Sem Avian Exotic Pet Med 5(3):127-139, 1996.
88. Page CD, et al: Antemortem diagnosis and treatment of sarcocystosis in two species of psittacines. J Zoo Wildl Med 23(1):77-85, 1992.
89. Patton CS, Patton S: *Tetratrichomonas gallinarium* encephalitis in a mockingbird (*Mimus polyglottos*). J Vet Diagn Invest 8:133-137, 1996.
90. Pennycott TW: Diazinon poisoning in pigeons. Vet Rec 138(4):96, 1996.
91. Perkins LEL, Swayne DE: Pathobiology of A/Chicken/Hong Kong/220/97 (H5N1) avian influenza virus in seven Gallinaceous species. Vet Pathol 38:149-164, 2001.
92. Platt SR et al: Peripheral neuropathy in a turkey vulture with lead toxicosis. J Am Vet Med Assoc 214(8):1218-1220, 1999.
93. Platt SR, Clippinger TL: Neurologic Signs *In* Olsen GH, Orosz SE (eds): Manual of Avian Medicine. St. Louis, MO, Mosby Inc, 2000, pp 148-169.
94. Porter SL, Snead SE: Pesticide poisoning in birds of prey. J Assoc Avian Vet 4(2):84-85, 1990.
95. Raidal SR, Jaensch SM: Central nervous disease and blindness in Nankeen kestrels (*Falco cenchroides*) due to a novel Leucocytozoon-like infection. Avian Pathol 29:51-56, 2000.
96. Randolph KD, Vanhooser SL, Hoffman M: Western equine encephalitis virus in emus in Oklahoma. J Vet Diagn Invest 6:492-493, 1994.
97. Redig PT, Post GC, Concannon TM: Development of an ELISA assay for the detection of aspergillosis in avian species. Proc Assoc Avian Vet, 1986, pp 165-178.
98. Romagnano A, et al: Magnetic resonance imaging of the brain and coelomic cavity of the domestic pigeon (*Columba livia domestica*). Vet Radiol Ultrasound 37(6):431-440, 1996.
99. Rosenthal K, et al: Computerized tomography in 10 cases of avian intracranial disease. Proc Assoc Avian Vet, 1995, p 305.
100. Rosenthal K: Disorders of the avian nervous system. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1996, pp 461-474.
101. Rosskopf WJ, et al: Epilepsy in red-lored Amazons (*Amazona autumnalis*). Proc Assoc Avian Vet, 1985, pp 141-146.
102. Rosskopf WJ, Woerpel RW: An unusual case of spinal aspergillosis in a Mexican red-headed Amazon parrot (*Amazona viridigenalis*). Proc Assoc Avian Vet, 1995, pp 351-356.
103. Scope A: Lafora-like inclusion bodies in the brain of a peach-faced love bird (*Agapornis roseicollis*, Vieillot 1818). Wien Tierarztl Mschr 87:58-60, 2000.
104. Sedlak K, et al: High susceptibility of partridges (*Perdix perdix*) to toxoplasmosis compared with other gallinaceous birds. Avian Pathol 29:563-569, 2000.
105. Selvaraj J, et al: Pathology of endosulfan toxicity in chickens. Indian Vet J 77:665-668, 2000.
106. Senne DA, et al: Pathogenicity of West Nile virus in chickens. Avian Dis 44:642-649, 2000.
107. Shell L, Jortner BS, Ehrich M: Assessment of organophosphorous-induced delayed neuropathy in chickens using needle electromyography. J Toxicol Environ Health 24:21-33, 1988.
108. Shell L: Brachial plexus injury in two red-tailed hawks. J Wildl Dis 29(1):177-179, 1993.
109. Shivaprasad HL, et al: Turlock-like bunyavirus associated with encephalomyelitis and myocarditis in an ostrich chick. J Vet Diag Invest 14:363-370, 2002.
110. Sims MH: Clinical electrodiagnostic evaluation in exotic animal medicine. Sem Avian Exotic Pet Med 5(3):140-149, 1996.
111. Stedman NL, Brown TP, Rowland GN: Intravascular cartilaginous emboli in the spinal cord of turkeys. Avian Dis 42:423-428, 1998.
112. Stoskopf MK: Clinical imaging in zoological medicine: A review. J Zoo Wildl Med 20(4):396-412, 1989.
113. Suchy A, Weissenbock H, Schmidt P: Intracranial tumours in budgerigars. Avian Pathol 28:125-130, 1999.
114. Tengelsen LA, et al: Response to and efficacy of vaccination against eastern equine encephalomyelitis virus in emus. J Am Vet Med Assoc 218(9):1469-1473, 2001.
115. Thomas NJ, Meteyer CU, Sileo L: Epizootic vacuolar myelinopathy of the central nervous system in bald eagles (*Haliaeetus leucocephalus*) and American coots (*Fulica americana*). Vet Pathol 35:479-487, 1998.
116. Varghese RG, et al: Organophosphorus-induced delayed neurotoxicity: A comparative study of the effects of Tri-ortho-tolyl phosphate and triphenyl phosphite on the central nervous system of the Japanese quail. Neuro Tox 16(1):45-54, 1995.
117. Wack RF, Lindstrom JG, Graham DL: Internal hydrocephalus in an African grey parrot (*Psittacus erithacus timneh*). J Assoc Avian Vet 6:159-163, 1992.
118. Wada Y, Kondo H, Itakura C: Peripheral neuropathy of dietary riboflavin deficiency in racing pigeons. J Vet Med Sci 58(2): 161-163, 1996.
119. Walsh MT: Seizuring in pet birds. Proc Assoc Avian Vet, 1985, pp 121-128.
120. Wild JM: The avian nucleus retroambigualis: A nucleus for breathing, singing and calling. Brain Res 606:119-124, 1993.
121. Williams SM, et al: Diagnosis of eastern equine encephalitis by immunohistochemistry in two flocks of Michigan ring-necked pheasants. Avian Dis 44:1012-1016, 2000.
122. Williams SM, et al: Ocular and encephalic toxoplasmosis in canaries. Avian Dis 45:262-267, 2001.
123. Witter RL, et al: An acute form of transient paralysis induced by highly virulent strains of Marek's disease virus. Avian Dis 43:704-720, 1999.

CHAPTER
18

Evaluating and Treating the Reproductive System

HEATHER L. BOWLES, DVM, Dipl ABVP-Avian,
Certified in Veterinary Acupuncture (Chi Institute)

Reproductive Embryology, Anatomy and Physiology

FORMATION OF THE AVIAN GONADS AND REPRODUCTIVE ANATOMY

The avian gonads arise from more than one embryonic source. The medulla or core arises from the mesonephric ducts. The outer cortex arises from a thickening of peritoneum along the root of the dorsal mesentery within the primitive gonadal ridge. Mesodermal germ cells that arise from yolk-sac endoderm migrate into this gonadal ridge, forming the ovary. The cells are initially distributed equally to both sides. In the hen, these germ cells are then preferentially distributed to the left side, and migrate from the right to the left side as well.[58] Some avian species do in fact have 2 ovaries, including the brown kiwi and several raptor species. Sexual differentiation begins by day 5 in passerines and domestic fowl and by day 11 in raptor species. Differentiation of the ovary is characterized by development of the cortex, while the medulla develops into the testis.[30,58]

As the embryo develops, the germ cells undergo three phases of oogenesis. During the first phase, the oogonia actively divide for a defined time period and then stop at the first prophase of the first maturation division. During the second phase, the germ cells grow in size to become primary oocytes. This occurs approximately at the time of hatch in domestic fowl. During the third phase, oocytes complete the first maturation division to

become secondary oocytes. Completion of the second maturation period results in an ovum.[30,34,53,58]

The ovarian medulla consists of blood vessels arranged in irregular vascular zones, interstitial cells, autonomic nerve fibers and smooth muscle. The ova are located peripherally in the cortex of the ovary. The ovarian surface is covered by parietal peritoneum with an underlying layer of dense connective tissue, the tunica albuginea. The ovary is located just caudal to the adrenal gland, near the tip of the cranial division of the kidney. It lies deep to the abdominal air sac, which forms an ovulation pocket near the time of lay. This pocket is thought to help receive the ovulated ovum with its yolk into the oviductal opening. The pocket is suspended by a dorsal mesentery, the mesovarium. Vascular supply to the ovary is through the cranial renal artery, which has several short branches. There are often two ovarian veins that drain blood directly into the caudal vena cava. The ovary has a roughened, granular appearance due to follicular development. As the hen becomes sexually active, the follicles begin to grow in a hierarchal pattern. [30,34,53,58]

The oviduct enlarges to occupy the dorsal aspect of the left intestinal peritoneal portion of the coelomic cavity. Seasonal growth and differentiation of the reproductive tract is under hormonal control. In a mature hen that is not reproductively active, the ovary and oviduct appear similar to that of a juvenile bird: small with no active follicles. The oviduct develops from a thickening in the peritoneal epithelium between the degenerating pronephric ducts and the first mesonephric tubules. These thickenings invaginate to form a tubular structure. This process occurs bilaterally and symmetrically in both sexes, but regression and disappearance of the ducts occurs in the cock and a relative regression of the right duct occurs in the hen. A right oviduct may be present in some raptors. The opening of the oviductal lumen into the cloaca often appears near the time of production of the first egg.[30,34,53,58]

There is a period of time during fetal development when the testes are ambisexual due to extensive covering with a cortical crust. These cortical remnants normally disappear prior to hatch; however, they may remain and further complicate visual sex identification. The seminiferous tubules differentiate in the medullary portion of the gonad. The ductus deferens develops from the mesonephric duct, while the epididymis arises from the mesonephros. The epididymis and ductus deferens initially develop in both sexes, as does the oviduct. The male tubular structures normally regress in the female. However, they have been reported to persist in some passerines and domestic fowl, but are significantly smaller than the oviduct.[23,35,58]

AVIAN REPRODUCTIVE PHYSIOLOGY

Avian reproduction is guided by seasonal physiologic controls such as favorable climate, low predation risk, social interaction, and food and nest site availability. Internal physiologic processes work in combination with external factors to promote gonadal development. This annual cycle involves integration between environmental, physiologic and behavioral conditions. Biologic clocks control the release of hormones and other chemicals that regulate metabolism, reproduction and behavior. Photoperiod plays a key role in this system using environmental light, which stimulates neural receptors, and clock information from an internal circadian cycle. This allows the bird to measure day length. In most of our Neotropical psittacine species, birds normally start to nest when the rains begin, when food is the most available. In temperate zone species, reproduction is stimulated by photoperiod. The lengthening day in early spring stimulates gonadal development. Warmer temperature, rainfall and behavioral displays fine-tune the physiologic events of breeding and the resulting increased secretion of sex hormones.[23,47,55,58]

There is strong evidence that the pineal gland (*hypophysis cerebri*) is largely responsible for photoperiodic control. However, unlike reptiles, the avian pineal gland does not have primary light receptors, but they do have photoreceptors like cells in the brain and retinae. Birds do not monitor day length visually, but rather by means of special receptors in the hypothalamus. After direct stimulation of the photoreceptors, neurosecretory cells in the hypothalamus induce the release of neurohormones in the median eminence (neural portion) of the epiphysis (pituitary), which is linked to the midbrain. These neurohormones are then carried via the bloodstream to the anterior pituitary gland, inducing the synthesis and subsequent release of luteinizing hormone (LH) and follicle-stimulating hormone (FSH). Luteinizing hormone stimulates gonadal activity and in combination with FSH stimulates ovarian development and testicular spermatogenises.[23,37,54,55,70] *See Ed. Note on page 522.*

Each circadian cycle includes a limited time period of each day during which photoreceptors are particularly sensitive to light. This stimulates a series of physiologic reactions. As daylight length increases each year, so does the opportunity for light to stimulate these receptors during this limited time period. In addition, the length of time these receptors are affected increases with increasing daylight length (see Chapter 19, Endocrine Considerations).[23,37,55,70]

During the late summer after breeding season has ended, the shortened daylight length stimulates the main molt. Gonadal hormones and tissue size decreases dramatically. Although the days are still relatively long, there is a photorefractory period that does not stimulate gonadal hormone release and tissue growth. This photorefractory period is best developed in migratory temperate zone species and is weak to absent in most tropical zone species. This may represent an adaptation for scheduling a major molt and preparation for migration by terminating reproductive activity while days are still long. The shorter days of winter inhibit gonadal growth. This is necessary to restore photosensitivity during the spring, as gonadal tissue will not grow in response to increasing day length unless there has been a prior period of short daylight length.[34,35,58]

Birds have developed several mechanisms to reduce body weight, thereby conserving energy expended during flight. The reproductive tract (ovary, oviduct, testes and ductus deferens) is greatly reduced in size during the non-breeding season, and eggs are laid and incubated externally.[34,35,58]

Physiology of the Female

When reproductively active, the ovary enlarges and follicles form in a hierarchal manner. The primary or F1 follicle is the largest and first in line to be ovulated. There are several smaller follicles on the ovarian surface as well. A stalk that contains smooth muscle with a blood and nerve supply suspends each large follicle.[30,34,49,58] Gonadotropin secretion causes follicles to develop on the ovary in a hierarchal manner. As the breeding season approaches, follicles undergo a period of rapid development and growth. There is deposition of yolk protein and lipid from the liver; gonadotropins and steroid hormones regulate this. The primary oocyte is surrounded by six layers of tissues: the oocyte plasma membrane, perivitelline membrane, granulosa cells, basal lamina, and the theca interna and externa. These tissues have an endocrine role, providing communication between the ovary and oviduct with passage of each ovum. The nerve supply is both adrenergic and cholinergic. Ovulation occurs under the influence of several hormonal factors. Meiotic or reduction division occurs approximately 2 hours preovulation, while the primary oocyte is still within the follicle. This yields a secondary oocyte and first polar body, each with a haploid number of chromosomes. Most birds have a meridional band or stigma on large preovulatory follicles. This is where the oocyte breaks through the follicular wall during ovulation.[30,34,58]

Ovulation occurs at a relatively fixed time period after oviposition under several physiologic, neural and hormonal controls. Determinant layers such as budgerigars and crows lay a fixed number of eggs, while indeterminate layers such as domestic fowl and Japanese quail replace eggs that are lost. Continuous breeders lay throughout the year. A rate of lay is the number of eggs laid in a given time period. A sequence is a number of eggs laid on successive days, separated by pause days. A clutch is a number of eggs laid during a sequence. A longer sequence is associated with a shorter oviposition/ovulation cycle.[30,31,34,52]

During the non-breeding season, ovarian follicles normally undergo atresia. Atresia is a process of regression and resorption of a follicle. Two types of atresia have been described: bursting and invasion. Bursting atresia occurs when the follicular wall ruptures and the yolk is released into the coelomic cavity where it is usually absorbed without any harm to the bird. Invasion atresia involves granulosa and thecal cells invading the ovum and subsequent *in situ* yolk absorption. Early atresia is noted when a vesicular lesion appears on the follicular surface. This vesicular formation progresses until the entire follicle is covered. As the largest F1 follicle is absorbed, the other smaller follicles will progress in a similar manner. Small follicles may be covered with connective tissue, occasionally leaving a scar-like area. Large follicles may undergo cystic degeneration. If ovulation ceases suddenly, as may occur during trauma or stress, developing follicles may become hemorrhagic, resulting in regression of the developing follicles. Aflatoxicosis also may cause follicular atresia. Aging hens may exhibit permanent ovarian involution, which is believed to be a normal physiologic process.[30,31,34,73]

Knowledge of the oviduct and its different anatomic regions is important in discerning pathologic conditions of the oviduct and developing egg. The oviduct is divided into five parts: the infundibulum, magnum, isthmus, uterus or shell gland, and vagina. A mucosal layer of ciliated epithelium with unicellular mucous glands or goblet cells lines the wall of the oviduct. The submucosa has mucosal folds that vary in height, thickness and tubular glands. The muscular layer has an inner layer of circular smooth muscle and an outer layer of longitudinal smooth muscle.[30,31,34,73]

The infundibulum is divided into the proximal funnel portion and a distal tubular portion. The funnel portion is where fertilization occurs. It has a thin wall with low mucosal folds. This portion surrounds and engulfs the ovum during ovulation. At the beginning and end of the ovulatory cycle, the oviduct and ovary may not be synchronized, resulting in ectopic ovulation also referred to as internal laying. This yolk and ovum may be resorbed without incident or may lead to coelomitis. The exact mechanism by which coelomitis occurs after ectopic

ovulation is not well understood.[17,30,31,34,73]

The tubular portion of the infundibulum is thicker, with taller branching folds. Underneath these folds are branched, convoluted tubular glands that produce the chalaza, which are fibrous bands that suspend the yolk within the egg. A thin, dense layer of albumin is added to surround the yolk. In some species, sperm host glands maintain sperm for fertilization for a variable time period and are located within this portion of the infundibulum.[17,30,31,34,73]

Large mucosal folds that result from numerous tubular glands distinguish the magnum histologically. It is the longest portion of the oviduct. The majority of albumin as well as sodium, magnesium and calcium are added to the egg by these glands. The release of albumin may be controlled by mechanical, neural and endocrine factors.[17,30,31,34,73]

The isthmus follows and, in domestic fowl, is clearly delineated from the magnum by a narrow translucent band. This band is not present in psittacines. The isthmus is relatively short and the mucosal folds are less prominent. The tubular glands are unique in that they produce sulfur-containing proteins. These proteins are incorporated into the shell membranes that are produced in the isthmus. A small amount of albumin is added to the developing egg. Calcification is initiated in the isthmus.[17,30,31,34,73]

During passage of the egg through the oviduct, the majority of the time is spent in the uterus, or shell gland. There are two portions: a short, narrow region which the egg traverses rapidly, and a pouch-like region where the egg spends the majority of the time. The mucosal lining is characterized by a large number of leaf-like lamellar folds that press against the surface of the egg. This increases the surface area to improve efficiency of calcification and plumping. "Plumping," a process in which a large amount of water and solutes are added to the egg relatively quickly, occurs in the proximal short, narrow region of the oviduct. It is in the pouch-like region that calcification of the shell is completed.[17,19,30,31,34,73]

The vagina is separated from the uterus by the uterovaginal sphincter. In most species the egg passes rapidly through the vagina to exit into the urodeum. However, in some species the egg may remain for a longer time period to allow for hardening of the shell. There are sperm host glands at the uterovaginal sphincter for sperm storage. Sperm from multiple male birds may be stored and remain viable for long time periods. This may be up to several months in the turkey. This allows for the possibility of insemination of different ova of a single clutch by different males.[17,19,30,31,34,58,73]

Physiology of the Male

The testes are paired, ellipsoid- to bean-shaped organs that lie near the cranial pole of the kidney. The surface is covered with a fibrous tunic, the tunica albuginea. Each testicle is suspended by a short mesentery or mesorchium that protrudes into the intestinal peritoneal cavity and is partially surrounded medially by the abdominal air sac. The testes change in size and color in response to hormonal fluctuations that influence sexual activity. The inactive testicles are often white to yellow due to accumulation of lipid in the interstitial cells. In some species, the inactive testicles are black due to a large number of melanocytes. Active testicles are significantly larger and paler due to the increased volume in the seminiferous tubules. The increased size is a result of increased length and diameter of the seminiferous tubules, and numbers of Leydig or interstitial cells. Generally speaking, the increase in testicular size is a result of increasing serum concentrations of FSH and LH. These physiologic processes occur during the nuptial or culmination phase of the reproductive cycle.[35,38,52,73] *(Ed. Note: These results have not been verified since FSH was purified. Prior to the early 1990s, FSH was contaminated with LH [Etches, R, University of Guelph, 2003]).*

Birds do not have septa that divide the testicles and there are no mediastinal testes. Unlike mammals, the seminiferous tubules anastomose with each other. Each seminiferous tubule is composed of a lining of spermatogonia and sustentacular or Sertoli cells. The spermatogonia divide to form primary, then secondary spermatocytes. As these spermatocytes progress toward the lumen, they undergo a maturation process to become spermatids, which then mature into spermatozoa or sperm. This maturation process proceeds with the head of each sperm embedded in the sustentacular cells. The sustentacular cells extend the width of the epithelium to provide support for the developing spermatozoa. They are phagocytic and also may produce steroid hormones and bind testosterone.[35,38,58]

The spermatozoa detach from the Sertoli cells and travel down the seminiferous tubules when mature. In most species, these tubules converge into a smaller number of short, straight tubules that continue as the rete testes. The rete testes is a meshwork of tubules embedded in connective tissue, located dorsomedially to each testicle, adjacent to the epididymis. Both the rete testes and straight tubules are lined by sustentacular cells.[35,38,58]

The Leydig, or interstitial, cells are located between the seminiferous tubules. They are light-colored and stain eosinophilic with hematoxylin-eosin stain because of the large concentration of smooth endoplasmic reticulum and cholesterol. The smooth endoplasmic reticulum is

involved in the conversion of cholesterol to steroid hormones, with testosterone and androstenedione being the major androgens. These hormones stimulate the secondary sex characteristics, including courtship, coloration, song, and the development and maturation of the tubules, particularly the ductus deferens.[35,38,58]

The epididymis of the bird is concealed due to its dorsomedial location on the testicle and its small size compared to mammals. It is not divided into a head, body and tail, but is composed of several efferent ductules that drain the rete testes and straight tubules. Several efferent ductules drain into the main epididymal duct along its length. The epididymal duct is relatively short and straight, and is lined by non-ciliated pseudostratified columnar epithelium. The epithelium is secretory and provides some of the seminal fluid. Sperm may be stored in the epididymis or in the seminal glomus of more seasonal birds. Some species have an appendix epididymis that extends cranially into the adrenal gland. The efferent ductules of this tissue may secrete androgens following castration.[35,38,58]

The epididymis continues distally as the ductus deferens. The ductus deferens is closely associated with the ureter in the dorsomedial coelom. In passerines, each ductus elongates distally during the culmination phase of the reproductive cycle to form the cloacal promontory, which can project into the cloaca. This protrusion gives the male external cloaca a pillar-like prominence compared to a rounded profile in females; therefore, it can be used for sex determination during the breeding season. Birds with a seminal glomus use it as the main storage site for sperm. The ductus is composed of non-ciliated pseudostratified squamous epithelium and has less secretory function when compared with the epididymis. There are no accessory sex glands in birds.[35,38,58]

Avian semen is derived in part from the sustentacular cells and epithelial cells that line the reproductive tract. Lymph-like fluid is produced from lymphatic folds in the floor of the proctodeum. This fluid appears to be harmful to spermatozoa because of the presence of clotting factors and high concentrations of chlorine and calcium. Avian spermatozoa are either complex or simple. Complex sperm is found in passerines and simple sperm in other species. Similar to mammals, each spermatozoon is composed of an acrosome, head and tail. In simple spermatozoa, the acrosome is attached to the head only at its most rostral point. The head is long and slender, and the tail is long and moves in an undulating manner. In complex sperm, the entire sperm is spiral in appearance and moves by rotating along its longitudinal axis.[35,38,58]

See Chapter 14, Evaluating and Treating the Gastrointestinal System for a discussion of the cloaca.

The vent may be either a circular opening as in psittacines or a transverse slit as in Galliformes. The sphincter muscle surrounds the opening and has an outer circular and an inner transverse striated muscle layer. In addition, there is a transverse muscle originating on the pelvic bone and/or caudal vertebrae that interdigitates with the sphincter muscle surrounding the vent. Upon contraction of this transverse muscle, the vent is pulled ventrocranially, which is important during coitus. This muscular action allows the cloaca in the male bird to be directed over the female's cloaca. The levator muscle originates on the ventral tailhead and inserts ventrally onto the vent and/or the phallus. This muscle pulls the vent caudally after copulation and defecation.[15,36,73]

The phallus may be intromittent as in ratites and Anseriformes, non-intromittent as in Galliformes, or absent as in psittacines and passerines. The intromittent phallus may be found in two forms: one form lacks a ventral cavity, and is found in ostriches, kiwis and tinamous; the other form has a cavity and is found in emus, rheas, cassowaries and Anseriformes. The former type of phallus consists of paired fibrolymphatic bodies with a dorsal sulcus to deliver semen. It lies on the floor of the cloaca and partially everts during micturition and defecation. Tumescence occurs by increased lymphatic flow and stasis into an elastic vascular body within the distal end of the phallus. Those phalluses with a ventral cavity also are located on the cloacal floor, but are enclosed in a sac or cavity. The proximal portion stays within the cavity and does not become engorged, while the distal portion everts when engorged with lymphatic fluid.[15,36,73]

The non-intromittent phallus, as found in domestic fowl, is located on the floor near the lip of the vent. It consists of a median and two lateral phallic bodies. Lateral to the phallic bodies are lymphatic folds located on the ventrolateral floor of the proctodeum. A lymphatic meshwork connects these folds and phallic bodies. Tumescence is a result of lymphatic flow through these structures. The lymphatic folds and lateral phallic bodies accumulate a greater amount of fluid than the median phallic body, resulting in eversion of the phallus and creating a groove for delivery of semen. The phallus contacts the everted oviductal opening where semen is deposited.[15,36,73]

Reproductive Disorders

Avian reproductive disorders are a result of complex combinations of hormonal, physiologic and behavioral actions reacting to photoperiods, food availability and availability of nest sites.[30,70] Environmental influences in captivity may result in the induction of reproductive and hormonal activity in several ways. Artificial lighting may

interfere with the normal photoperiod and annual light cycles, resulting in abnormal cycling.[23] Food is typically available ad libitum and is often high-fat, calorically dense seed, or foods high in simple carbohydrates such as corn and fruit. These foods may actually stimulate reproduction. Most pet birds are not intended for breeding and do not have mates. In some environments, pet birds may select an abnormal mate such as their human cohabitants or cage furniture. There may be a genetic predisposition and lack of normal reproductive hormonal balance[30,34,37] (see Chapter 3, Concepts in Behavior: Section III, Pubescent and Adult Psittacine Behavior).

Reproductively driven birds may display instinctual territorial and mate-related behaviors. These behaviors may include, but are not limited to aggression, biting, masturbation and excessive vocalization. These "undesirable" behaviors may jeopardize their value as pets, diminishing the pet-human relationship and even result in these birds losing their homes.[30,34,37]

Reproduction is often not desired in pet birds. Egg production and hormonal cycling may lead to disease processes of the reproductive system or systemic, endocrine and metabolic disorders. Therefore, avian practitioners have sought medical and surgical methods to limit reproductive drive and hormone production.[8,56,59]

CHRONIC EGG LAYING

Chronic egg laying in pet birds occurs when a hen lays repeated clutches or larger than normal clutch size without regard to the presence of a mate or accurate breeding season. This process often physically exhausts the reproductive tract and is a serious metabolic drain, particularly on calcium stores, all of which may predispose the hen to egg binding, yolk coelomitis and osteoporosis. Commonly affected species include cockatiels, finches and lovebirds, however, any species may be affected. Diagnosis of chronic egg laying is based on history and physical examination. There typically is a history of the hen laying large numbers of eggs with or without a pause period in between clutches. A thorough history of the home environment will often reveal several reproductive stimuli and a "mate relationship" with a member of the household or owner. Physical exam may reveal normal findings, a palpable egg in the coelom, or other secondary disease conditions such as a pathologic fracture secondary to osteoporosis. Serum chemistries may reveal hypercalcemia, hypercholesterolemia and hyperglobulinemia supportive of an ovulating hen. There may be a hypocalcemia present if the hen's calcium stores are depleted, and particularly if she is consuming a low-calcium diet such as seed[9,31,33,65] (see Chapter 5, Calcium Metabolism).

Therapy for chronic egg laying focuses on stopping egg production while altering any predisposing stimuli and correcting any secondary diseases that may be present. Pharmacologic, behavioral, nutritional, environmental and surgical options are used alone or in combination depending on the needs of the individual patient. Pharmacologic options have included medroxyprogesterone acetate[a], levonorgestrel[b], human chorionic gonadotropin[c], norethindrone/mestranol[d], testosterone, leuprolide acetate[e] and tamoxifen[f] **(Table 18.1)**. Medroxyprogesterone acetate, though often effective, may cause serious side effects such as polyuria/polydipsia, obesity, lethargy, hepatic lipidosis, diabetes mellitus, hepatic cirrhosis and death.[9,31,63] Levonorgestrel, another synthetic progestin, has been evaluated only in Japanese quail (*Coturnix coturnix japonica*) and may carry the same side effects as medroxyprogesterone acetate.[72,77] Testosterone therapy interrupts the ovulatory cycle, but has variable results and is contraindicated in patients with liver disease.[73] Norethindrone/mestranol has caused severe hypertension in one Rouen duck (D. Zantop, personal communication, 2000). Human chorionic gonadotropin has demonstrated to be a safer alternative with significantly fewer side effects; however, it has not been consistently effective in managing these disorders and patients may become refractory to treatment.[34,44] Tamoxifen is a non-steroidal anti-inflammatory agent used as an estrogen blocker to treat women with breast cancer. Tamoxifen was administered to budgerigars presumed to be hens, but not actively laying for 38 to 46 weeks.[46] Leukopenia was a significant side effect that resolved after therapy was discontinued. An incidental finding in this study was the change in coloration of the hen's cere from brown to pink or blue. This change implies that tamoxifen does have some estrogen-blocking affects in birds. Leuprolide acetate is a long-acting gonadotropin-releasing hormone (GnRH) analog. A single injection in women and studied laboratory rodents results in an initial stimulation followed by a prolonged suppression of pituitary gonadotropins. In rats, this reduction in serum gonadotropin levels is achieved by reducing the number of pituitary GnRH receptors. Repeated monthly injections result in receptor down regulation of GnRH pituitary receptors, which causes a decreased secretion of gonadal steroid hormones. Therefore, tissues and functions that depend on these hormones for maintenance become quiescent, and diseases resulting from reproductive hormone production improve or resolve.[16,33,50,51,78]

Environmental stimuli should be altered, including decreasing the photoperiod to 8-10 hours of daylight per day. Nest sites, toys and other items toward which the bird has a sexual affinity should be removed from

Table 18.1 | Summary of Medical Therapy for Reproductive Disorders

Therapy	Dosages	Comments
Leuprolide Acetate[e]	700-800*[1] µg/kg IM for birds <300 g	Administered every 14 days; 3 doses are usually adequate
	500 µg/kg IM for birds >300 g	Stable in standard freezer 9 months
Human chorionic gonadotropin*[2,c]	250-500 IU/kg IM on days 1, 3 and 7, 500-1000 IU/kg IM	Stable in refrigerator 60 days. If a second egg is laid, repeat dose on day 3; if a third egg is laid, repeat dose on day 7
Levonorgestrel[h]	—	Not recommended
Medroxyprogesterone[a]	—	Not recommended
Tamoxifen[f]	2 mg/kg PO QD	Leukopenia
Arginine vasotocin	0.01-1.0 mg/kg IM	Stable in standard freezer
PGE (Prepidil Gel)[g]	0.025 ml/100 g 0.2 mg/kg applied topically	May freeze into aliquots and thaw prior to administration; relaxes uterovaginal sphincter while inducing uterine contractions
PGF2alpha (Lutalyse)	0.02-1.0 mg/kg IM	Does not relax utero-vaginal sphincter when inducing uterine contractions
	Topically	Applied to prolapsed uterine tissue to stop hemorrhage and shrink tissues

*[1] *Ed. Note: 1000 mg/kg has been used in recalcitrant cases.*
*[2] *Ed. Note: Should be administered with dexamethasone to avoid what appears to be immune-based response to HCG. Can be given with leuprolide acetate.*

the enclosure. Access to nesting environment or materials such as a box, other dark cavities, or shredded papers should be prohibited. In the event that a pet bird is showing nesting behavior and laying eggs in a designated site, removal of eggs from the "nest" should be avoided for the normal incubation period for each species to discourage the hen from laying further eggs to replace those removed. Any perceived or actual mate should be removed from the cage or room. In some species such as the cockatiel, visual and auditory separation from an actual or perceived mate may be necessary. A "one-person bird," which has a single household person who exclusively or primarily handles and cares for it, should potentially be viewed as having a "mate relationship" with that person. This may serve as a trigger for reproductively driven behaviors. Stimulatory petting such as rubbing the pelvis, dorsum and cloacal regions and kissing the beak should be avoided. Feeding calorically dense diets should be avoided, as mentioned previously. Interactive behaviors that simulate a "flock relationship" should be encouraged such as the bird being handled by several people in the household. The cage location and furniture (toys, perches, food dishes) should be changed and rotated periodically to discourage territorial behavior and limit reproductive drive in response to a perceived "nest site." Any nutritional problems should be corrected to improve the hen's dietary plane to reduce the severity of metabolic drain.[31,53,63] Dietary alteration and reduction of caloric intake does appear to anecdotally reduce or stop egg production. This nutritional effect is often achieved by converting the pet bird from a seed-based diet to a formulated one. The exact reason for this effect is unknown, but it is common practice in poultry to reduce feed intake to stop egg production and induce molting.[56] Surgical salpingohysterectomy may be elected or necessary if medical therapy is not successful and if there is no intent to breed the particular hen. Laparoscopic salpingohysterectomy may be performed as a preemptive measure on juvenile birds to prevent egg production and its associated diseases. Any secondary disease conditions also should be appropriately treated.[6,59,71]

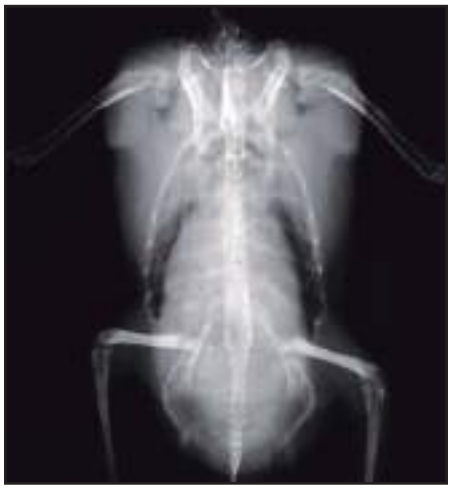
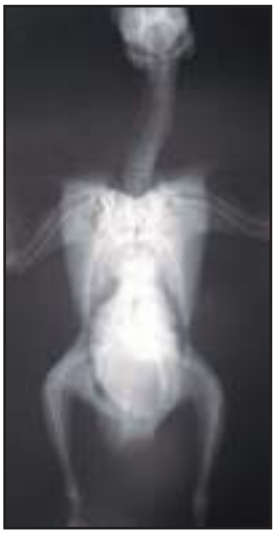


Fig 18.1 | Ventrodorsal radiograph of a budgerigar hen with polyostotic hyperostosis. Note the increased density of both femurs.

POLYOSTOTIC HYPEROSTOSIS

Polyostotic hyperostosis differs from physiologic osteomyelosclerosis in that the latter condition occurs in nonlaying hens and cocks as a result of pathologic conditions. Typically, radiographs reveal significantly increased bone density of the long bones and occasionally the vertebrae (Fig 18.1). The pathogenesis of polyostotic hyperostosis is still unclear. Many affected birds exhibit concurrent reproductive-associated activity or may suffer from reproductive-associated disease conditions. In addition, increased medullary bone density does appear to resolve radiographically with resolution of reproductive drive or disease. Hepatic disease may play a role in this condition due to the liver's role in the inactivation of estrogen. However, a recent study does not support this theory, stating that budgerigars affected by polyostotic hyperostosis had no evidence of estrogen secretion or other endocrine disease.[5,29,58,74]



Figs 18.2a,b | Left lateral and ventrodorsal radiographs of a 3-year-old egg-bound cockatiel. The hen had been consuming a seed diet and was unable to perch for several days prior to presentation. The bird was thin (68 g), dehydrated and hypocalcemic (6.2 mg/dl), and responded to supportive care and arginine vasotocin. Egg production was controlled after egg expulsion with leuprolide acetate (750 μg/kg IM every 14 days for 3 injections) and alteration of environmental stimuli. Diet was modified to include a 90% formulated diet.

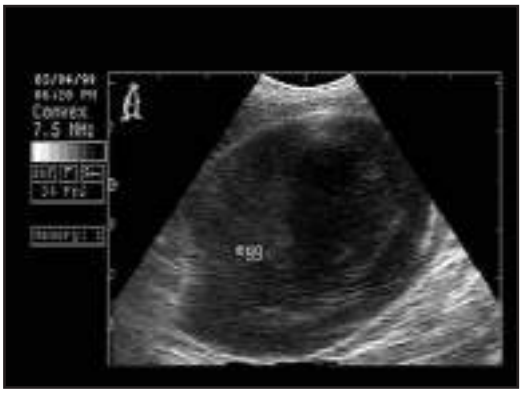


Fig 18.3 | Coelomic ultrasound of the same cockatiel. Note the well-calcified egg in normal presentation for oviposition (7.5-MHz probe).

EGG BINDING AND DYSTOCIA

Egg binding is defined as the failure of an egg to pass through the oviduct within a normal period of time. Most companion birds lay eggs at intervals of greater than 24 hours, and individuals may vary further. This variability may make it difficult to determine if there is a problem in the early stages of this disease. Dystocia involves the mechanical impedance to oviposition. The most common anatomic areas for this to occur are the distal uterus, vagina and vaginal-cloacal junction.[31,63,73]

Causes of egg binding may include chronic egg laying, oviductal muscle dysfunction secondary to excessive egg laying, calcium metabolic disease, vitamin E and selenium deficiencies, malnutrition, obesity, inadequate exercise and muscle strength, malformed eggs, mechanical tears or damage to the oviduct, oviductal infections, systemic disease, genetic predisposition and environmental stressors. Dystocia also may result when a developing egg in the distal oviduct obstructs the cloaca or causes oviductal tissue to prolapse. Oviductal torsion and oviductal or abdominal masses compressing the oviduct also may obstruct passage of an egg and result in dystocia. Breeding birds out of their natural season, egg-producing virginal hens and hens with a persistent right oviduct may be predisposed to egg binding or dystocia.[31,63,73]

Diagnosis

Cockatiels, lovebirds, canaries and finches are most commonly reported to be affected and seem to present with more severe clinical signs, possibly due to their small size. Clinical signs associated with egg binding and dystocia vary according to severity, size of the bird affected and degree of secondary complications. Common signs include acute depression, abdominal straining, persistent tail wagging, a wide stance, failure to perch, abdominal distension, dyspnea, and/or sudden death (Fig 18.3). An egg lodged in the pelvic canal may compress the pelvic blood vessels, kidneys and ischiatic nerves, causing circulatory disorders, lameness, paresis or paralysis. Pressure necrosis of the oviductal wall may occur. Dystocia may cause metabolic disturbances by interfering with normal defecation and micturition, and cause ileus and renal disease, respectively. The severity of the patient's condition can be estimated by the degree of depression and the length of time clinical signs have been present.[31,63,73]

Diagnosis of egg binding or dystocia in a severely compromised patient may be made based on history and physical examination alone, and the patient may not be stable enough to survive other diagnostic procedures. Rapid diagnosis and therapy are crucial for a successful outcome. Physical examination may reveal depression, lethargy, a thin or normal body condition, and dehydration. There may be dyspnea or an increased respiratory rate due to compression of the caudal thoracic and abdominal air sacs. The hen may not be able to perch, and may demonstrate pelvic limb paresis, paralysis or cyanosis. An egg typically, but not always, is palpable in the caudal abdomen. Cranially located, soft-shelled and non-shelled eggs may not be detected on abdominal palpation. Palpable eggs may be located within the oviduct or ectopically within the coelom, and careful abdominal palpation, cloacal examination, radiographs, coelomic ultrasound, laparoscopy and/or laparotomy may be required to determine the egg's position.[31,63,73]

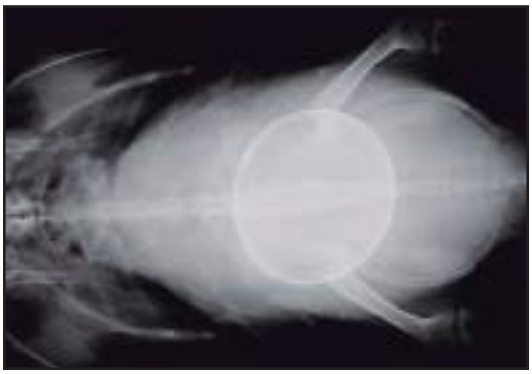


Fig 18.4a | Ventrodorsal radiograph of an eclectus hen with a history of depression and inappetence. Physical examination revealed a distended abdomen and palpable egg in the coelom. Radiographs revealed a calcified egg in the mid coelom.

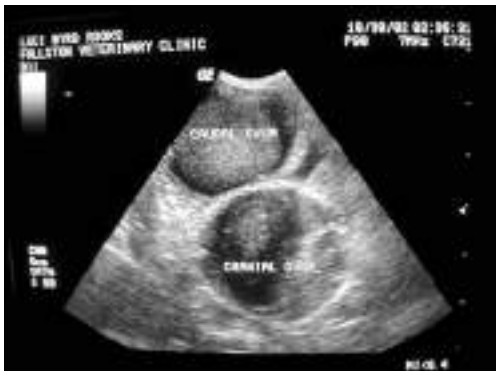


Fig 18.4b | Ultrasound of the same bird revealed a non shelled egg cranial to the shelled egg noted on the radiograph in Fig 18.4a.

Radiography and ultrasonography aid in evaluation of the position and characterization of the egg(s). There may be multiple eggs identified in the coelom due to an obstruction distally or secondary to motility disorders. Radiographs may reveal an egg in the coelom if the egg has a visible shell. The egg is typically located in the distal oviduct, in the region of the uterus (see Figs 18.2a,b, Fig 18.3). Osteomyelosclerosis of the femurs, tibiotarsi, radii, ulnas and/or spine may be visible, and a soft tissue density suggestive of an enlarged ovary in the region of the ovary may be noted, and is supportive of a reproductively active hen. A coelomic ultrasound will often reveal an egg and may identify soft-shelled or non-shelled egg(s) that may not be identifiable on radiographs (Figs 18.4a,b). Again, there may be several eggs visible within the coelom. Follicles may be visible on the ovary, indicating the potential for further ovulation and egg formation. A hematologic analysis and serum chemistries are useful to identify any predisposing and secondary diseases. A complete blood count may reveal a leukocytosis with a relative heterophilia if there is a concurrent inflammatory or infectious process. Serum chemistries may demonstrate elevated aminotransferase and creatinine phosphokinase due to skeletal muscle enzyme leakage from tissue damage, or as a result of reduced food consumption and a hypermetabolic state. Hypercholesterolemia, hyperglobulinemia are supportive of an ovulating hen. Elevated total and ionized calcium may be indicative of a cycling hen. Hypocalcemia may be observed if a hen has been consuming a calcium-poor diet or has been laying excessive numbers of eggs, resulting in depletion of calcium stores[31,40,60,73] (see Chapter 5, Calcium Metabolism).

Treatment

Therapy varies with history, severity of clinical signs and diagnostic test results. Supportive care should include elevated environmental temperature, parenteral calcium (only if indicated), fluid therapy and nutritional support. Broad-spectrum antibiotics are indicated if it is suspected that the integrity of the oviduct has been compromised. Analgesics are indicated if the patient appears to be in pain or if clinical knowledge of the patient's condition suggests that pain may be a part of the pathologic state. Supportive care alone is often enough to allow oviposition, although the hen should be monitored closely for deterioration of her condition, which may require further intervention.[40,63,73]

Prostaglandin and hormonal therapy may be used to induce oviductal contractions. This may result in expulsion of the egg if the contractility of the oviduct is sufficient to expel the egg, the uterus is intact, the egg is within the oviduct, and there is no obstruction such as a neoplastic mass, granuloma or egg adhered to the oviduct. Studies performed in poultry have found that prostaglandin E2 (PGE2) and prostaglandin F2alpha (PGF2alpha) bind at specific receptor sites in the uterus and vagina. The uterine myometrium appears to preferentially bind PGF2alpha because it contains low-affinity and some high-affinity binding sites for PGE2 and specific high-affinity for PGF2alpha. Prostaglandin F2alpha binds at the shell receptor sites to cause a time- and dose-dependent mobilization of cellular calcium in the presence of extracellular calcium, thereby causing uterine muscle contraction. It has been demonstrated in vitro that PGE2 is itself ineffective in calcium ion mobilization, but will enhance PGF2alpha-induced calcium mobilization. This suggests that PGE2 may potentiate the ability of PGF2alpha to cause uterine contraction. In the vagina, high-affinity binding sites for PGE2 predominate. It is possible that a high PGE2 concentration in the vagina is needed to saturate high-affinity binding sites and block PGF2alpha-binding sites. This allows for relaxation of the uterovaginal sphincter and vagina. Due to the fact that fewer PGE2 high-affinity binding sites are present in the uterus, they are not likely to interfere

with PGF2alpha binding and may potentiate the action of PGF2alpha.[27,28,63,67,68]

When an egg is present in the uterus, the administration of PGF2alpha and PGE2 will cause the concentration of arginine vasotocin (AVT) to increase in systemic circulation, however, PGF2alpha is the more potent stimulator of AVT release. It is suggested that prostaglandins stimulate uterine contractions, which in turn stimulate the release of AVT from the neurohypophysis, and that AVT probably acts synergistically with PGF2alpha to increase uterine contractions. Oxytocin and AVT appear to specifically affect the uterus, inducing contractions. It is important to note that PGF2alpha, oxytocin and AVT do not cause relaxation of the uterovaginal sphincter while inducing oviductal contractions. This may result in peristalsis of the egg, severe pain and/or rupture of the uterus. Prior to their use it should be determined if the utero- vaginal sphincter is open. In addition, prostaglandin and hormonal therapy does require adequate calcium to be effective. As many of these patients are severely hypocalcemic due to either malnutrition or chronic egg laying, supplemental calcium may be required prior to administration of these medications.[27,28,63,67,68]

PGE2 gel[g] may be applied to the uterovaginal sphincter at a dose of 0.1 ml per 100-g bird. PGE2 causes relaxation of the uterovaginal sphincter while causing oviductal contractions and may be applied topically, thereby decreasing the incidence of systemic side effects. These contractions may expel the egg within 15 minutes. Contact with PGE2 gel may cause altered menses and induce spontaneous abortion in women. Therefore, it is important to flush any excess from the cloaca after egg expulsion, and to caution staff and clients regarding contact with any stool and/or urine produced. Prostaglandin F2alpha, oxytocin and AVT also will cause powerful uterine contractions. Prostaglandin F2 alpha is administered parenterally, rather than locally, and is more likely to cause systemic reactions such as hypertension, bronchoconstriction and general smooth-muscle stimulation.[27,28,63,67,68]

If supportive care and medical therapy fail to induce oviposition, then manual manipulation may be necessary. Massaging the abdomen and vaginal opening may relax the vaginal sphincter and allow passage of the egg. It may be helpful to infuse lubricants into the cloaca to moisten the tissues. Careful digital pressure applied to the cranial portion of the egg and directed caudally may encourage movement through the distal oviduct and cloaca. Using a cloacal speculum, the vaginal opening of the oviduct can be dilated by inserting a blunt probe (eg, lubricated cotton-tipped swab) that is gently advanced in a twirling motion. Potential complications may include retroperistalsis of the egg out of the oviduct into an ectopic position within the coelom, rupture of the egg, oviductal trauma, oviductal laceration, oviductal avulsion, hemorrhage, and displacement of the egg or fragments into an ectopic position. If fertilization may have occurred and the egg may be fertile, it may be incubated if successfully removed intact.[31,63,73]

Fig 18.5 | Transabdominal ovocentesis and aspiration of egg contents in a 7-year-old egg-bound cockatiel. Patient was anesthetized with isoflurane by mask induction. A 22-gauge needle and 3-ml syringe were used to aspirate approximately 1 ml of egg contents. The shell was passed approximately 4 hours postovocentesis. Complete passage of the shell was confirmed by radiography.

Ovocentesis may be performed to facilitate passage of an egg. Aspiration may be performed through the cloacal opening if the egg is distally located, or transabdominally if the egg is more cranially positioned. The egg is manipulated so that it is visible through the cloaca and a needle is inserted into the egg through the cloaca. The contents of the egg are aspirated into a syringe, while the shell is manually collapsed and the pieces expelled through the cloaca. (see Chapter 7, Emergency and Critical Care and Chapter 24, Diagnostic Value of Endoscopy and Biopsy). If the egg cannot be visualized through the cloaca due to a more cranial location, transabdominal ovocentesis may be performed (Fig 18.5). The egg is manually placed directly against the abdominal wall so that other abdominal organs are displaced and not damaged during aspiration. A needle is inserted through the skin and abdominal wall into the egg. The egg contents are aspirated into the syringe while the egg is manually collapsed. The eggshell remnants are expelled through the cloaca, either naturally or with clinical assistance. It is important to confirm radiographically that these eggshell pieces have been completely expelled. If these pieces are not expelled within a reasonable amount of time, approximately 36 hours, it may be necessary to irrigate the oviduct through the cloaca or laparotomy approach. A salpingohysterectomy may be performed if egg remnants are retained and the hen is not required for breeding.

Some clinicians advocate flushing the uterus postoviposition with saline, chlorhexidine or iodine to remove any shell fragments and decrease the incidence of metritis. Oviductal rupture, resulting in an ectopic egg, shell fragments and yolk coelomitis, are possible complications of ovocentesis.[8,31,63,73] See Chapter 24, Diagnostic Value of Endoscopy and Biopsy.

Prostaglandin treatment, manual delivery and ovocentesis are contraindicated in cases of ectopic eggs, oviductal rupture, oviductal torsion and mechanical obstruction. Complications that may require surgical intervention include oviductal rupture with or without an ectopic egg, oviductal necrosis, oviductal torsion, abdominal hernia or if the condition is interfering with defecation and/or micturition. Medical therapy to reduce reproductive hormone levels and reproductive activity should be utilized to temporarily prevent further egg production. Surgical removal of an egg is required in cases of ectopic eggs and dystocia, including oviductal rupture, oviductal torsion, or mechanical obstruction, or if medical treatment is not successful. If surgical intervention is necessary, bacterial culture and sensitivity and histopathology should be performed on oviductal tissue samples. Salpingohysterectomy may be considered to prevent further reproductive complications, and any predisposing and secondary diseases should be corrected.[6,24,40,63,73]

OVIDUCTAL PROLAPSE

Oviductal prolapse may occur secondary to any condition that causes chronic, excessive abdominal straining such as normal physiologic hyperplasia, egg laying or dystocia. An intracoelomic space-occupying mass also may induce prolapse of the oviduct. Predisposing factors may include abnormal or soft-shelled eggs, malnutrition, obesity, salpingitis and cloacitis. Typically, the uterus protrudes through the cloaca, often with a partial prolapse of the vagina and cloaca (Fig 18.6).[31,63,73]

Rapid management is necessary to prevent necrosis of these tissues. Any egg that may be present should be removed, all exposed tissues cleaned, irrigated and kept well moistened to prevent desiccation. Topical anti-inflammatories such as dimethyl sulfoxide may be applied, any lacerations should be repaired and all tissues should be gently replaced. Temporary stay sutures may be indicated to aid in preventing recurrence, as prolapse of the oviduct may recur and repeated replacement is often required. Bacterial culture and sensitivity of the prolapsed tissue should be performed to aid in appropriate antibiotic therapy. Complete blood count, serum chemistries, radiographs, ultrasonography and laparoscopy should be included in a complete diagnostic evaluation to identify any predisposing and secondary disease conditions. Treatment should be directed at

Fig 18.6 | Uterine prolapse, including partial prolapse of the vagina and cloaca, in a 7-year-old cockatiel with a history of chronic egg laying. Samples of affected tissues were taken for bacterial culture and sensitivity. Prolapsed tissues were irrigated with sterile saline, dimethyl sulfoxide was applied and tissues were reduced manually. Therapy included supportive care, enrofloxacin (15 mg/kg PO q 12 h x 14 days), carprofen (2 mg/kg PO q 12 h x 3 days). Further egg production was controlled with leuprolide acetate (750 μg/kg IM every 14 days for three injections) and alteration of environmental stimuli.

clearing any bacterial infection and preventing further prolapse. It also is important to decrease reproductive hormone levels to prevent further egg formation, decrease the size of oviductal tissue and allow the reproductive tract to rest. Broad-spectrum antibiotics and anti-fungals should be initiated while bacterial and fungal cultures are pending.[8,31,63,69,73]

Salpingohysterectomy may be considered to prevent recurrence. Predisposing factors should be corrected to prevent recurrence and secondary diseases addressed (see Chapter 35, Surgical Resolution of Soft Tissue Disorders).

UTERINE TORSION

Uterine torsion is usually diagnosed in the later stages of the disease. Birds typically present with abdominal distension secondary to coelomitis. Early clinical signs may include depression and anorexia following recent oviposition. A complete blood count often demonstrates a leukocytosis with a relative heterophilia, and serum chemistries show an elevated aspartate transferase and creatinine kinase. Diagnosis is usually made at exploratory laparotomy or laparoscopy. Many times, severe vascular compromise and necrosis of the oviduct is found, which requires salpingohysterectomy.[1,6,24,71,73]

OVIDUCTAL IMPACTION

Oviductal impaction may occur following salpingitis, metritis or dystocia. Impactions may occur due to excess mucin or albumin, secondary to cystic hyperplasia of the

oviduct. Inspissated egg material also may cause obstruction. Clinical signs may be vague and can include cessation of egg production, broody behavior without egg production, weight loss, anorexia, depression, constipation, diarrhea, abdominal distension, and reluctance to walk or fly. A tentative diagnosis is made through history, physical examination and supporting diagnostic tests. A leukocytosis with or without a relative heterophilia may be noted. Serum chemistries may be supportive of an ovulating hen. Radiographs and coelomic ultrasound may demonstrate a soft tissue density in the region of the oviduct, displacement of other coelomic viscera, loss of coelomic visceral detail or coelomic fluid if there is a concurrent coelomitis. Definitive diagnosis of oviductal impaction is often made at laparoscopy or laparotomy, revealing an abnormal-appearing, enlarged oviduct with or without coelomitis and adhesions. In many cases, it is necessary to clean and repair or surgically remove the oviduct. Surgery may be complicated if coelomic fluid and/or adhesions are present.[6,24,31,40,63,69,73]

Bacterial culture and sensitivity should be performed on specimens from the affected oviduct, and histopathologic examination should be performed on biopsy samples. Treatment includes parenteral fluids, nutritional support, warmth and broad-spectrum antibiotics, pending culture and sensitivity results. Medical or surgical therapy to reduce reproductive hormone production and reproductive activity should be initiated, and environmental stimuli altered as discussed with chronic egg laying, to prevent recurrence.

SALPINGITIS AND METRITIS

Salpingitis is defined as inflammation of the oviduct either by an infectious or non-infectious etiology, the latter being far less commonly reported. It is generally seen associated with airsacculitis, liver disease, pneumonia, systemic infections, and ascending infections of the oviduct from the uterus or cloaca. Excessive abdominal fat has been associated with salpingitis in domestic fowl. Some of the most commonly identified pathogens are *Escherichia coli*, *Salmonella*, *Mycoplasma*, *Pasteurella* and *Streptococcus* spp. Newcastle disease also has been associated with salpingitis in several species. In ground-nesting species such as Anseriformes and emus, non-lactose-fermenting, gram-negative bacteria such as *Pseudomonas aeruginosa*, *Proteus mirabilis* and *Proteus vulgaris* are commonly identified. Noninfectious causes of salpingitis include trauma and inflammation secondary to oviposition disorders, malnutrition and foreign bodies. Salpingitis is most common in adult hens but may occasionally occur in young birds as well.[8,63,73,75]

Metritis is a localized infection or inflammatory process within the uterine portion of the oviduct. Metritis may occur secondary to dystocia, egg binding, oviductal impaction, systemic bacterial infection and ascending infection. Salpingitis and metritis may cause abnormal shell formation and impaired uterine contractions, and may cause infections in chicks and embryos including embryonic death. Fatalities are often associated with ovulation, egg binding or dystocia, oviductal rupture, coelomitis and septicemia.[8,63,73,75]

Clinical signs of salpingitis and metritis may be vague and difficult to detect initially. In pet birds, these include decreased egg production, infertility, abnormally shaped eggs and mild depression. More advanced cases may exhibit anorexia, lethargy, abdominal distension, oviductal rupture, coelomitis and septicemia. There may be a leukocytosis with a relative heterophilia. Serum chemistries may or may not be supportive of an ovulating hen. Radiographs and ultrasonography may reveal an enlarged oviduct. Laparoscopy may or may not identify inflammation of the serosal surface of the oviduct. The oviduct may be thin-walled, decreased in length or have vascular congestion. The lumen may contain fluid or fibrinous exudates. Definitive diagnosis of salpingitis and metritis is based on cytology, bacterial and fungal culture and sensitivity, and biopsy with histopathologic analysis of a specimen from the oviduct.[8,24,40,63,73]

Therapy of salpingitis and metritis is focused on correcting any underlying or contributing causes. Antibiotic therapy for identified or suspected bacterial organisms should be initiated pending results of bacterial culture and sensitivity. Pharmacologic treatment and husbandry-related intervention, as discussed with chronic egg laying, should be initiated to prevent further hormonal stimulation with subsequent egg production, which may perpetuate or contribute to this disease. There should be close follow-up including bacterial culture and sensitivity and fertility monitoring after treatment, as many cases are difficult to resolve completely. It is important to note that bacteria isolates from the cloaca are not equivalent to oviductal infectants, and cloacal bacterial cultures should be interpreted carefully. Severe refractory cases may require laparotomy to remove necrotic tissue and flushing of the oviduct with fluids and antibiotics. Patients suffering from severe salpingitis may require salpingohysterectomy, or this may be elected in milder cases to resolve disease and prevent recurrence if the hen is not intended for breeding.[6,63,71,73]

CYSTIC HYPERPLASIA OF THE OVIDUCT

Cystic hyperplasia of the oviduct may occur from improper formation of the left oviduct or secondary to an endocrine abnormality. Additionally, the vestigial right

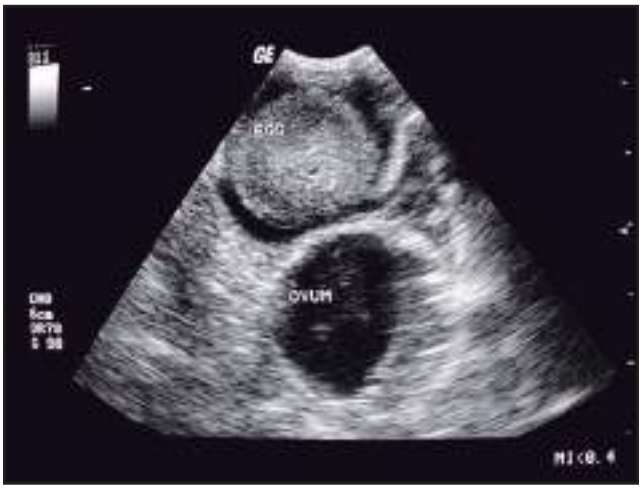


Fig 18.7 | Coelomic ultrasound. An ectopic egg and uterine laceration were noted on laparotomy, and a salpingohysterectomy performed. Note the calcified egg in the caudal coelom and the ovum located more cranially (7.5-MHz probe). This ovum could not be detected on the radiographs. The hen recovered fully.

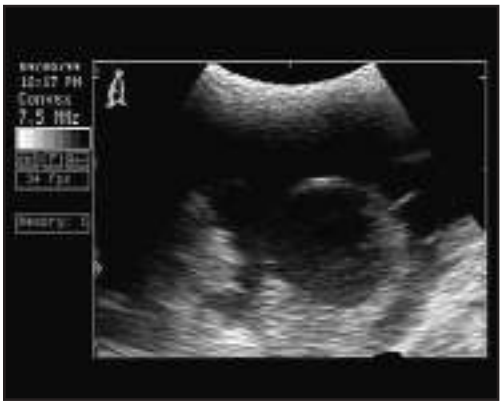


Fig 18.8 | Coelomic ultrasound of an 8-year-old African grey parrot. The patient had a history of depression and inappetence. Physical examination revealed abdominal distension. Note the coelomic fluid and large ovarian cyst (7.5-MHz probe).

oviduct may become cystic and the associated ovary often has cystic changes as well. Cystic hyperplasia often contributes to salpingitis and egg binding. Clinical signs may include depression, anorexia, abdominal distension, ascites and dyspnea. A tentative diagnosis is made through history, physical examination and supporting laboratory tests, similarly to that of salpingitis and metritis. Radiographs may demonstrate an enlarged soft tissue density in the region of the oviduct. Ultrasonography may reveal an enlarged oviduct that may be fluid-filled or have obvious cysts present, with or without concurrent ovarian follicles or cysts. Laparoscopy may show a dilated oviduct filled with a white or brown mucoid fluid. Definitive diagnosis requires laparotomy with biopsy, cytology, histopathology, and bacterial culture and sensitivity.[8,24,31,40,63,73]

Therapy to stop ovulation should be initiated due to increased risk of oviductal rupture during ovulation, oviposition and possible hormonal contribution to the cystic state of the oviduct. If bacterial infection is suspected or documented by cytology and bacterial culture and sensitivity, appropriate antibiotic treatment is indicated. Salpingohysterectomy may be required to resolve the current problem or prevent future recurrences and should be considered if the hen is not intended for breeding, as complete resolution with medical therapy alone may be difficult.[6,8,31,63,73]

OVIDUCTAL RUPTURE

Oviductal rupture may occur secondary to dystocia or oviductal disease. Prostaglandins, oxytocin, arginine vasotocin and ovocentesis may cause traumatic rupture of the oviduct. Clinical signs may include depression, anorexia, and abdominal distension secondary to coelomitis or the deposition of egg or oviductal contents. Radiographs and ultrasonography may reveal osteomyelosclerosis, polyostotic hyperostosis, a soft tissue density in the region of the ovary, ovarian follicles, an enlarged or cystic oviduct, a shelled or non-shelled egg, and coelomic fluid if a concurrent coelomitis is present (Figs 18.7, 18.8). Diagnosis is confirmed at laparoscopy or laparotomy. The laceration may be repaired, depending on the integrity of the tissue, or salpingohysterectomy may be performed as a therapeutic and preventive technique.[6,24,31,40,63,73]

ECTOPIC OVULATION

Ectopic ovulation may result from failure of the infundibulum to retrieve an ovulated ovum, reverse peristalsis of the oviduct or oviductal rupture. Ectopic ovulation does not necessarily result in coelomitis. Internal laying is actually a common occurrence in many avian species, and the ova are usually resorbed without any problems whatsoever. Reverse peristalsis may be triggered by obstruction of the oviduct, cystic hyperplasia, neoplasia, malnutrition, trauma and stress. The ectopic ova may be resorbed without incident or may induce a severe coelomitis.[30,31,56,63,73]

Clinical signs of ectopic ovulation may include transient or persistent depression, inappetence and abdominal distension, especially if there is an associated coelomitis. There may be a leukocytosis with a mature heterophilia. Serum chemistries may demonstrate an ovulating hen. Radiographs may reveal polyostotic hyperostosis and one or multiple eggs in the abdomen. It is important to

Greg J Harrison

Fig 18.9 | A cockatiel hen with a history of egg laying 3 months previous is showing depression and dyspnea. Note the severely distended abdomen. Diagnosis was cystic ovarian disease.

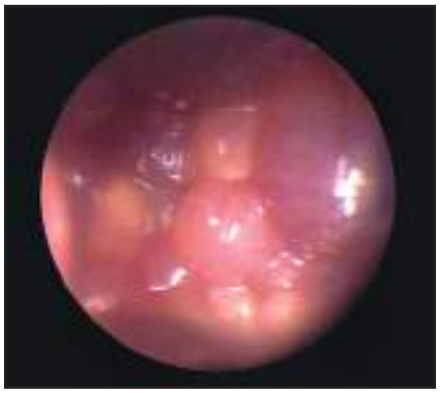


Fig 18.10 | Laparoscopic examination of the African grey hen in Fig 18.8. Note the two ovarian cysts and several smaller follicles.

note that an ectopic ova detected by ultrasound in the absence of clinical signs may resolve on its own with no treatment, and any medical or surgical intervention may be contraindicated. It may be difficult or impossible to determine if an egg is located within the oviduct or is ectopic without laparoscopy or laparotomy, depending on its location within the coelom. If the egg is not laid within a reasonable time period and/or the patient's condition is declining, a laparotomy is indicated as an exploratory procedure. Ectopic eggs are removed by laparotomy, and any oviductal tear should be surgically repaired or a salpingohysterectomy performed. Cytology, culture and sensitivity, and histopathology should be performed in cases of oviductal rupture, cystic hyperplasia and neoplasia.[8,31,40,63,73]

CYSTIC OVARIAN DISEASE

Ovarian cysts have been known to occur in several pet bird species including cockatiels, canaries, budgerigars, macaws, pheasants and domestic ducks. Cyst development may be caused by endocrine disorders, anatomic abnormalities on the ovary itself and pathologic conditions of the ovary. A thorough history may reveal current or previous egg production, with an abrupt halt. Owners may even report chronic reproductive behavior without egg production or impaired reproductive performance in breeding hens.[8,14,73]

Advanced cystic ovarian disease may cause depression, inappetence and weight loss. Abdominal distension, often due to secondary coelomitis, and related clinical signs may be noted as well (Fig 18.9). A leukocytosis with a relative heterophilia, as well as a peripheral hypercalcemia, hyperglobulinemia and hypercholesterolemia are common findings. Radiographs may demonstrate polyostotic hyperostosis, a soft tissue density in the area of the ovary and/or oviduct, coelomic fluid and displacement of coelomic viscera. Ultrasound may reveal a fluid-filled cyst(s) in the area of the ovary or simply coelomic fluid of an undetermined source. An ovarian cyst may be quite large and may actually fold onto itself as it grows. There may be normal ovarian follicles present as well (Fig 18.10). Abdominocentesis with cytology and bacterial culture and sensitivity should be performed in those patients suffering from associated coelomitis. It is beneficial and often necessary to perform a laparoscopic exam or celiotomy with ovarian biopsy, especially for those patients with cysts that do not resolve with medical therapy, as it is not uncommon for hens to develop ovarian cysts secondary to neoplasia and oophoritis. Laparoscopy will reveal an ovarian cyst(s), and the contents may be aspirated during this procedure. Cytology of fluid aspirated from these cysts is clear to straw-colored and of low cellularity. However, it is important to practice extreme caution during a laparoscopic exam and aspiration, as fluid from the cyst or coelom may gain access to the respiratory system through the entry hole in the abdominal air sac. Ovarian biopsy with cytologic analysis, histopathologic exam, and bacterial culture and sensitivity should be performed to identify any primary or secondary disease processes.[14,40,63,73]

Treatment goals include resolution of the cyst(s) and associated disease conditions such as coelomitis, oophoritis, ovarian granuloma and neoplasia. Abdominocentesis often improves related dyspnea if there is coelomic fluid compressing the air sacs. Pharmacologic, behavioral, environmental and dietary intervention to reduce ovarian activity are indicated as production of reproductive hormones may perpetuate ovarian cysts.[14] Aspiration of cysts, salpingohysterectomy and partial ovariectomy may be beneficial for complete resolution.[6,8,60,63,73] Cryosurgical destruction may be beneficial. Long-term resolution may be difficult and patients suffering from cystic ovarian disease should be regularly monitored for recurrence.[8,14,63,73,78]

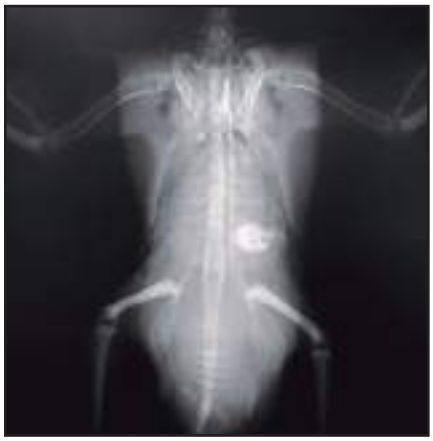


Fig 18.11 | Ventrodorsal radiograph of a cockatiel hen with reproductive-associated coelomitis. Note the severely enlarged fluid/soft tissue density, cranial displacement of the grit-filled ventriculus, obliteration/compression of caudal thoracic and abdominal air sacs, and increased density of both femurs.

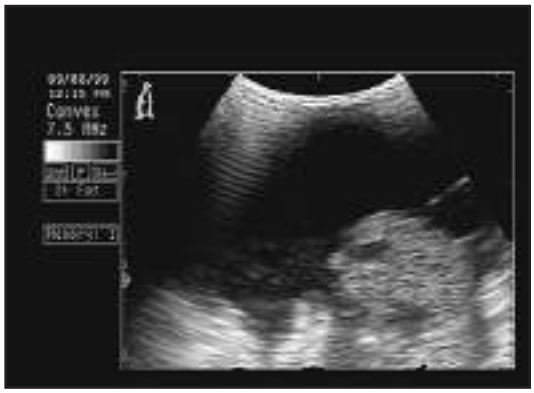


Fig 18.12 | Coelomic ultrasound of the cockatiel hen from Fig 18.11. Note the coelomic fluid and soft tissue density. Laparotomy revealed an oviductal granuloma, and the hen recovered fully in response to salpingohysterectomy and antibiotic therapy.

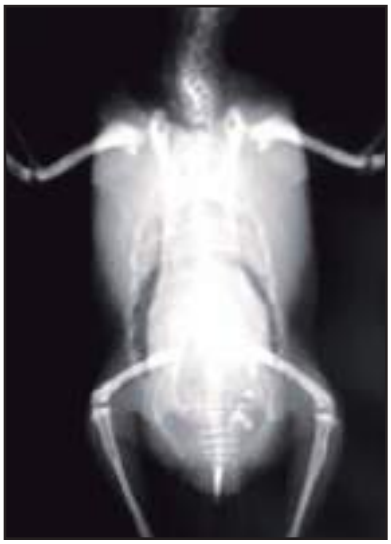
REPRODUCTIVE-ASSOCIATED COELOMITIS

Reproductive-associated coelomitis may encompass egg yolk coelomitis, (previous egg related peritonitis) ectopic ovulation-associated coelomitis and septic coelomitis. Coelomitis may be found in association with other diseases such as malnutrition, metabolic disorders and systemic infections. Cystic ovarian disease, salpingitis, metritis, cystic hyperplasia, oviductal rupture, oviductal and ovarian granulomas, septicemia, intestinal rupture and neoplasia may cause associated coelomitis as well.[8,31,73]

Tentative diagnosis of reproductive-associated coelomitis is made through history, physical examination and supporting laboratory tests. The hen may have a history of egg production, which may have abruptly stopped. Clinical signs may include transient or persistent depression, lethargy and inappetence. Patients with more advanced disease may suffer from weight loss, abdominal distension, and dyspnea associated with coelomic fluid and air sac compression (Fig 18.11). It is important to note that not all patients with coelomitis will have identifiable fluid present. Supportive diagnostic tests include a complete blood count, serum chemistries, radiographs, coelomic ultrasound and analysis of any fluid recovered from abdominocentesis including cytology, culture and sensitivity. A leukocytosis with a relative heterophilia, as well as a peripheral hypercalcemia, hyperglobulinemia and hypercholesterolemia compatible with pre- and immediate postovulation may be noted. Many birds will actually be hypocalcemic due to calcium depletion subsequent to malnutrition or chronic egg laying. Some birds may have egg yolk visible in their peripheral blood smears as well as above the buffy coat in separated blood samples and lipemia is common. Radiographs may demonstrate polyostotic hyperostosis, soft tissue density in the region of the ovary and/or oviduct, coelomic fluid, abdominal and caudal thoracic air sac compression, or even an obvious shelled or non-shelled egg, or egg remnants leading to oviductal granuloma (Fig 18.12). Contrast radiography may be helpful to illustrate organ displacement and locate any suspected space-occupying mass. Ultrasound may reveal coelomic fluid, ovarian follicle(s), ovarian cyst(s), an ovarian mass, and oviductal masses such as granuloma or neoplasia (Figs 18.13a,b). Cytology of coelomic fluid may demonstrate a septic or non-septic exudate, a transudate, or yolk or fat globules if such material is present. Bacterial culture and sensitivity should be performed on samples of coelomic fluid. Laparoscopy and/or laparotomy may be necessary to identify the causative etiology of coelomitis.[8,31,40,63,73]

Treatment of reproductive-associated coelomitis varies with type and severity of clinical signs. Many birds respond well to supportive care alone. Abdominocentesis is not only supportive of diagnosis but therapeutic as well, to relieve dyspnea due to air sac compression. Broad-spectrum antibiotics should be initiated in cases of suspected or confirmed infectious coelomitis while waiting for sensitivity results on fluid obtained from abdominocentesis. Corticosteroids may be indicated in cases where an infectious etiology has been excluded, but should be used judiciously due to potential serious side effects. Pharmacologic therapy may be used to stop further ovulation, reproductive hormone production and to reduce the size of the reproductive tract, which may perpetuate this condition.

Once the coelomic fluid has decreased and the patient is stable, it is beneficial to perform a laparoscopic



Figs 18.13a,b | Left lateral and ventrodorsal views of a 6-year-old male budgerigar with a Sertoli cell tumor. Note the soft tissue mass in the midcoelom and polyostotic hyperostosis of the long bones.

examination. This allows direct visualization of the ovary, oviduct and other organs to help confirm etiology such as a cyst, granuloma and/or tumor. Cytology and biopsy with histopathologic examination, and bacterial culture and sensitivity should be performed on abnormal tissue. A celiotomy with or without a salpingohysterectomy may be necessary to biopsy or remove a mass, cystic oviduct or remove inflammatory debris from the abdomen, particularly if medical therapy alone is not effective. It is important to note that during laparoscopy or laparotomy there is a risk of fluid gaining access to the respiratory system via the incision through the abdominal air sacs, and often there are significant adhesions between the oviduct and neighboring viscera due to chronic inflammation.[8,31,63,73]

OOPHORITIS

Inflammation of the ovary results from neoplastic, mechanical or infectious causes. Infectious oophoritis often occurs as a result of spread from adjacent organs or septicemia, and is frequently bacterial in origin. Clinical signs may be vague and include anorexia, weight loss, depression, cessation of egg production, egg binding and sudden death. A diagnosis of oophoritis is made through history, physical examination, radiography, ultrasonography, abdominocentesis with coelomic fluid analysis, laparoscopy, laparotomy, and biopsy of the ovary with bacterial culture and sensitivity and histopathologic analysis. Hematology may demonstrate a leukocytosis with a relative heterophilia. Radiographs and ultrasound may demonstrate an egg or an enlarged soft tissue density in the region of the ovary, ovarian follicle(s) and ovarian cyst(s), and there may be coelomic fluid present if there is a concurrent coelomitis. If coelomic fluid is present, abdominocentesis is beneficial both therapeutically and diagnostically. Cytologic analysis as well as bacterial culture and sensitivity should be performed on fluid recovered. Laparoscopy may demonstrate an enlarged, abnormal-appearing ovary, which may have associated hypervascularization. Persistent or chronic oophoritis may progress to granulomatous disease, which may be evident on ultrasound, laparoscopy and laparotomy. Definitive diagnosis is based on ovarian biopsy with histopathologic examination along with bacterial culture and sensitivity.[8,31,40,63,73]

Treatment of oophoritis includes broad-spectrum antibiotics, pending sensitivity results. Egg binding is handled as previously described. As discussed for chronic egg laying, pharmacologic therapy to temporarily stop ovulation should be initiated, as it does appear clinically that ovulation may perpetuate inflammation of the ovary. Laparoscopic exam, bacterial culture and sensitivity, and complete blood count should be repeated until culture results are negative and any leukocytosis has resolved. Reproductive performance and general condition should be carefully followed, as complete resolution may be difficult. Partial ovariectomy, usually performed with salpingohysterectomy, may be beneficial in refractory cases if the hen is not intended for breeding.[8,31,63,73]

OVARIAN AND OVIDUCTAL NEOPLASIA

Ovarian and oviductal neoplasia is most commonly seen in the budgerigar (*Melopsittacus undulatus*), cockatiel (*Nymphicus hollandicus)* and gallinaceous species. Clinical signs may include abdominal distension, coelomic fluid, lameness, dyspnea, depression, inappetence and chronic reproductive-associated behavior with or without egg production. Egg binding, oviductal impaction, ovarian cysts, abdominal hernia and coelomic fluid may be seen in conjunction with reproductive tract neoplasia. This fact demonstrates the extreme impor-

tance of a complete diagnostic work-up. Alteration of secondary sex characteristics such as a cere color change may occur as well. Diagnosis is supported by history and physical examination, demonstration of enlargement in the area of the ovary or oviduct on radiographs and ultrasound, and biopsy with histopathologic examination of abnormal tissues. Lymphomatosis, adenocarcinoma, leiomyosarcoma, leiomyomas, adenomas and granulosa cell tumors have been reported.[3,10,31,40,41,63,73] There have been anecdotal reports of treatment with chemotherapeutic drugs such as carboplatin (D. Zantop, personal communication, 2000); however, no consistent results have been documented to date. Prognosis for long-term recovery is grave, with no refereed reports of successful treatment. Salpingohysterectomy with partial or complete ovariectomy may have value in select patients.[31,63,73] Cryosurgical ablation of the ovary and anti-angiogenesis therapy may prove beneficial.

PARASITES

Ascarids and flukes have been reported to infect the oviduct from the cloaca by reverse peristalsis. Heavy infestation may cause soft-shelled and shell-less eggs, and may result in salpingitis. Anseriformes are most commonly affected. Ascarids and small flukes reportedly have been passed in eggs. Diagnosis is made by finding adult worms in the oviduct on celiotomy or necropsy, or by finding adult worms in eggs laid by affected hens. Fecal floatations should be performed, especially on ground-dwelling species, and prophylactic anthelmintic programs may be helpful in preventing severe infestations. If these parasites obstruct the oviduct, they require surgical removal or salpingohysterectomy.[73]

OVERPRODUCTION OF EGGS

Safe numbers for egg production for different species are not definitively documented. Nutrition and environmental conditions affect safe production levels. Free-ranging psittacines typically produce one to two clutches per year; however, many captive psittacines produce far more eggs than this. While many birds show no obvious side effects, chronically overproducing hens may develop reproductive tract disorders, as well as poor body condition and feather quality. To improve long-term health in producing birds, egg production should be limited to two clutches per year in birds that show any signs of poor health secondary to overproduction. It also is recommended that all birds receive some rest period each year to prevent reproductive disorders from developing.[73]

ORCHITIS

Infectious orchitis may occur from ascending infections, hematogenous spread or infected adjacent organs. Rarely, non-infectious causes are associated with inflammation of the testicles. Early clinical signs are vague and difficult to detect, and may include infertility, mild depression and decreased appetite. As the disease progresses, the patient may develop lethargy, inappetence and abdominal distension if a secondary coelomitis develops. Leukocytosis with a relative heterophilia may be noted. Testicular enlargement may be noted on radiographs and ultrasonography, which is a normal condition for a normal male. There also may be notable enlargement, inflammation and hypervascularization on laparoscopy. Definitive diagnosis is made by cytology, bacterial culture and sensitivity, and histopathologic examination of samples from affected testis. Therapy includes broad-spectrum antibiotics, pending sensitivity results.[12,31,73]

TESTICULAR NEOPLASIA

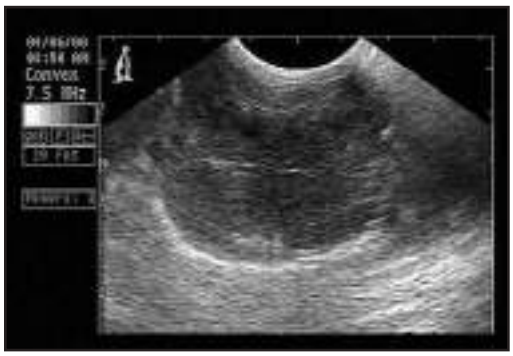
Testicular neoplasia has been commonly documented in the budgerigar (*Melopsittacus undulatus*) and is often unilateral. Clinical signs include abdominal distension and one-sided paresis, paralysis, or cyanosis and hypothermia of the pelvic limb due to compression of the ischiatic nerve and blood vessels. The disease is often advanced once clinical signs are evident. Definitive diagnosis is made by testicular biopsy and histopathologic examination.[13] Alterations of secondary sex characteristics such as cere color change from blue to brown may occur. Neoplasms reported include Sertoli cell tumor, seminoma, interstitial cell tumor and lymphosarcoma. Leiomyosarcoma and carcinoma have been reported to arise from the epididymis and ductus deferens.[3,10] Radiographs may reveal a soft tissue mass in the region of the testicles, air sac compression, and secondary sex changes such as polyostotic hyperostosis (see Figs 18.13a,b). Treatment includes orchiectomy. Cryosurgical ablation may be beneficial. Chemotherapy with carboplatin and O,P'-DDD (mitotane)[21] has been anecdotally reported if the tumor is deemed incompletely or non-resectable, or if the patient is not a good surgical candidate. However, no conclusive data have been reported to date regarding efficacy of chemotherapy.[31,73]

CLOACAL PAPILLOMAS

Cloacal papillomas have been noted in New World psittacine species with green-wing macaws over-represented. To date the cause is unknown, but a herpes virus etiology is strongly suspected[7,22,39,57,62] (see Chapter 32, Implications of Viruses in Clinical Disorders). Clinical signs may include infertility due to mechanical obstruction, hematochezia, and straining to urinate and defecate. Examination of the cloaca reveals one or several fleshy masses at the mucosal border. Inserting a lubricated swab into the cloaca to evert the tissue facilitates cloacal examination (Fig 18.14). In addition, white vinegar applied to

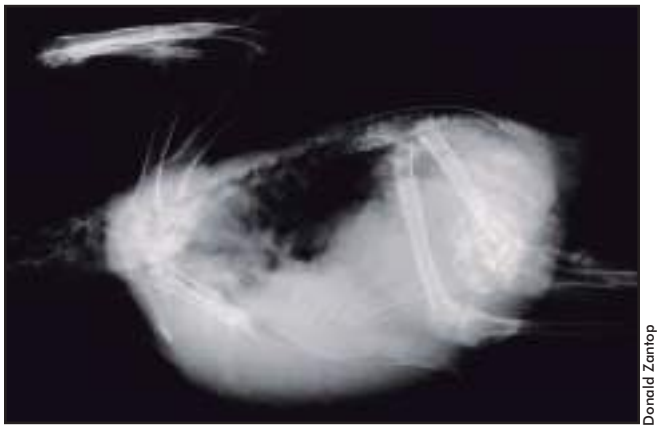
Donald Zantop

Fig 18.14 | A 22-year-old yellow-naped Amazon parrot with a history of depression, weakness and inappetence. Note the papillomatous masses at the mucocutaneous junction of the cloaca.

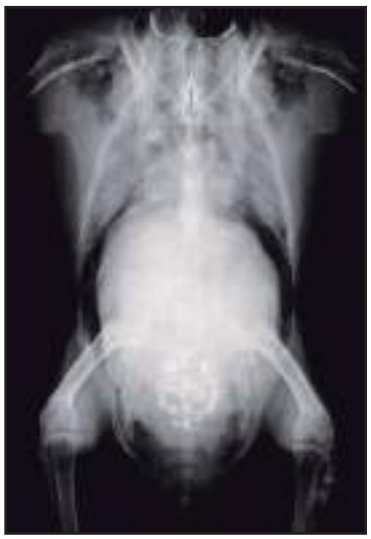


Donald Zantop

Fig 18.16 | Coelomic ultrasound of the same Amazon parrot. Note the severely enlarged liver with increased echogenicity. Liver biopsy and histopathologic examination revealed bile duct adenocarcinoma (7.5-MHz probe).



Donald Zantop



Donald Zantop

Figs 18.15a,b | Ventrodorsal and right lateral radiographs of the Amazon parrot in Fig 18.16. Note the severely enlarged liver and caudal displacement of the grit-filled ventriculus.

the cloacal wall will significantly blanch papillomatous tissue, further assisting in identification. A thorough endoscopic exam of the cloaca should be performed to rule out obstruction of the gastrointestinal, urinary and reproductive tracts. It is important to perform a full examination to detect oropharyngeal and laryngeal papillomas and hepatomegaly. It is equally as important to perform complete blood count, serum chemistries, bile acids evaluation, radiographs including contrast studies, coelomic ultrasound, and endoscopic exam of the coelom and cloaca. These tests are crucial to fully evaluate the patient, as papillomas may occur at any site of the gastrointestinal tract. Furthermore, bile duct and pancreatic adenocarcinoma are associated with papillomatous disease.[25,31,32,73,76] It is necessary to perform a liver and pancreatic biopsy for histopathologic examination to rule out early bile duct and pancreatic adenocarcinoma upon diagnosis of cloacal papilloma (Figs 18.15a,b, 18.16). Cytology, culture and sensitivity are beneficial to diagnose a secondary cloacitis. Histopathology of excised or biopsied tissue will confirm the diagnosis.[31,73]

Therapy includes cauterization of papillomas with silver nitrate, cryosurgery, cloacotomy and laser surgical removal.[18,61] Autogenous vaccines and cloacal mucosal stripping have not met with consistent success.[2,64] Imiquimod[h], an immune response modifier used in humans for the treatment of anogenital warts, has been shown to decrease lesion mass and tenesmus, but it did not cause remission of papillomatous tissue.[43] It is important when cauterizing papillomatous tissue with silver nitrate to flush the area profusely with saline when sufficient tissue is cauterized, otherwise normal cloacal tissue will be cauterized. Topical medications such as dimethyl sulfoxide and silver sulfadiazine cream should be applied to affected tissue after cauterization. Analgesics should be administered postoperatively as well as antibiotics if there is an associated cloacitis. Temporary spontaneous remission has been reported. Recurrence is extremely common and frequent re-examinations are necessary.[31,73] Spontaneous regression has been reported.

Carboplatin chemotherapy has been reported to benefit patients suffering from bile duct and pancreatic adenocarcinoma, and does appear to be well tolerated.[20,79]

Affected birds should be separated from the non-affected to prevent possible transmission.[8,31,73] It is beneficial to

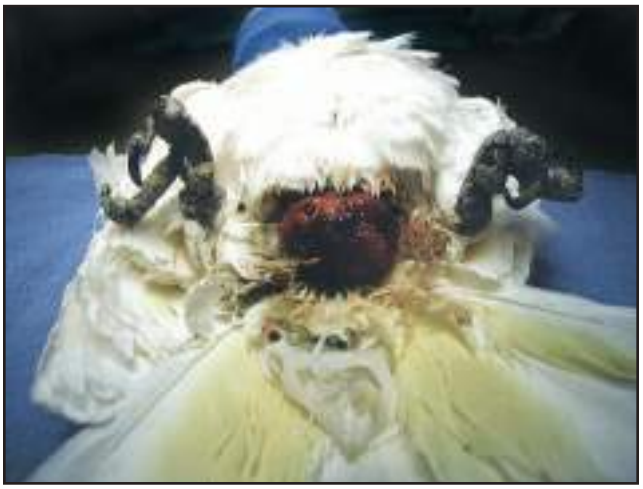


Fig 18.17 | Prolapsed cloaca in a 5-year-old male umbrella cockatoo. History included chronic masturbation and intermittent partial cloacal prolapse.

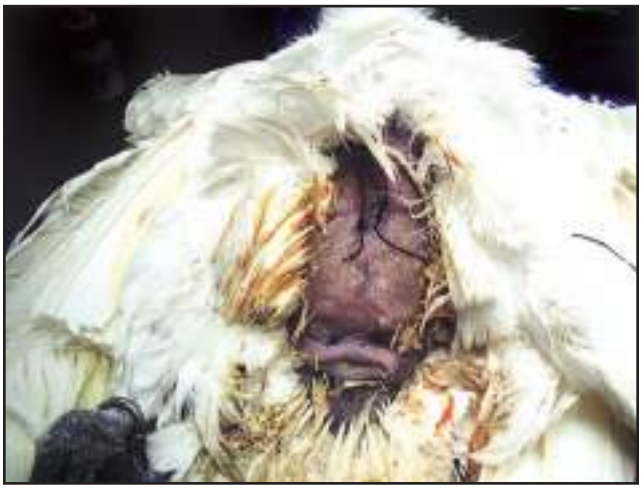


Fig 18.18 | The same umbrella cockatoo after manual cloacal reduction and cloacopexy.

perform a thorough cloacal examination, particularly on New World psittacines, during routine health examinations, as affected birds should be isolated to prevent potential spread to non-affected individuals. Healthy chicks have been raised from artificially incubated or fostered eggs from affected pairs.[31,73]

CLOACAL PROLAPSE

Cloacal prolapse may occur secondary to chronic straining from masturbation, egg laying, space-occupying abdominal masses, and inappropriate weaning and social behavior. Physical examination will reveal prolapsed tissue through the vent that may be intermittent or persistent (Fig 18.17). Careful cleaning, irrigation and lubrication of prolapsed tissue are a necessity. Affected tissue should be examined for necrosis, and any adhered egg should be removed. Cytology and bacterial culture and sensitivity should be performed on prolapsed tissue to aid in antibiotic therapy. A complete blood count, serum chemistries, radiographs, ultrasound and endoscopic exam of the coelom and cloaca are useful to determine any other predisposing cause.[31,73] Chronic reproductive-associated behavior and straining secondary to masturbation may respond to pharmacologic therapy such as leuprolide acetate or environmental manipulation to decrease reproductive stimuli. Cloacopexy and the use of temporary stay sutures may be helpful in temporary or permanent reduction. However, those procedures interfere with movement of the cloaca and may alter defecation and micturition[4,66] (Fig 18.18). Ventplasty may decrease the vent opening and prevent further prolapse if the vent has become flaccid (see Chapter 35, Surgical Resolution of Soft Tissue Disorders). Clomipramine hydrochloride and phenylpropanolamine administration has been anecdotally reported to contract the vent orifice and assist in the resolution of prolapse of the cloaca (D. Zantop, personal communication, 2003). Salpingohysterectomy with partial ovariectomy or orchiectomy may be beneficial in those patients refractory to medical therapy. Broad-spectrum antibiotics should be initiated, pending bacterial culture and sensitivity, because primary and secondary bacterial infections are common.[8,31,63] See Chapter 3, Concepts in Behavior.

CLOACITIS

Cloacitis may result from both infectious and non-infectious processes. Cloacal prolapse, cloacal papillomas, cloacoliths and bacterial infections may cause inflammation of these tissues. This may result in secondary urogenital and/or gastrointestinal disease due to the anatomic relationship to the cloaca. Cytology with bacterial culture and sensitivity should be performed. Appropriate antibiotics or anti-inflammatory therapy may be indicated. Dimethyl sulfoxide may be used to reduce inflammation with no systemic side effects, and swabbing the cloaca with petroleum jelly will prevent fecal and urate accumulation on the cloacal surface with subsequent irritation.[73]

CLOACOLITHIASIS

Cloacolithiasis is infrequently noted in pet birds. It may result from previous egg binding, infectious cloacitis, malnutrition or neurologic disease of the cloaca. Cloacoliths should be manually or surgically removed. Cloacal cytology with bacterial culture and sensitivity should be performed. The identification of anaerobic bacteria, notably *Clostridia* sp., is a common finding often associated with a fetid odor. Since routine aerobic cultures will not isolate these organisms, Gram's stains of the feces should be performed. Broad-spectrum antibiotics should be initiated, pending culture results.

Improved nutrition through diet change should be initiated. Patients should be monitored closely for recurrence, and prognosis for return to normal breeding performance is poor.[73]

CLOACAL NEOPLASIA

Cloacal carcinomas are infrequently reported in pet birds. An endoscopic examination of the cloaca should be performed, and the patency of openings into the urodeum, proctodeum and coprodeum should be noted. Definitive diagnosis is made from histopathologic analysis of biopsy samples. Neoplastic masses should be surgically removed, with caution not to damage the openings to the gastrointestinal, urinary and reproductive tracts.[3,41,42,73]

OTHER CLOACAL DISEASES

Other cloacal diseases include cloacal strictures and excessive vent feathering. Cloacal strictures may be gently manually dilated with the use of a speculum. Excessive feathering around the vent may cause infertility; prior to breeding season, these feathers should be removed by trimming or pulling.[73]

REPRODUCTIVE HORMONE-RELATED FEATHER PICKING

Birds pick their feathers for several different reasons. Avian veterinarians often cannot elicit a cause from a complete history, physical examination and diagnostic work-up. In some avian patients, history may reveal reproductive-related behaviors such as breeding, masturbating, regurgitation and failure to molt. Physical exam may demonstrate removal of one or several types of feathers, rough feather condition of any remaining feathers, and the patient may become reproductively stimulated during the examination. Serum chemistries panels may reveal elevated serum calcium in hens (provided they are not calcium deficient) and hypercholesterolemia. Radiography may demonstrate an enlarged gonad(s) and/or polyostotic hyperostosis. Ultrasonography and laparoscopy may reveal ovarian follicles, ovarian cysts, an enlarged oviduct or enlarged testicles. The diagnostic work-up must be extensive to rule out any other possible diseases. Serum estradiol, progesterone and testosterone level tests are available, although normal values are not known for every avian species. Pharmacologic treatment, behavior counseling and environmental changes to reduce reproductive drive and hormone levels as discussed with chronic egg laying are recommended. Response to treatment is important therapeutically and diagnostically. Treatment failures may be due to the presence of non-reproductive-related disease and concurrent disease. It is important to note that a full history and diagnostic work-up must be performed prior to establishing a diagnosis of reproductive-related feather disease. Many of these birds molt heavily after starting therapy and this should not be interpreted as a deleterious side effect.[8,11,48]

Products Mentioned in the Text

a. Depo-Provera, Upjohn C., Kalamazoo, MI, USA
b. Levonorgestrel, Sigma Chemical, St. Louis, MO, USA
c. HCG, Pregnyl Organon, Inc, West Orange, NJ, USA
d. Ortho-McNeal Pharmaceutical, Rantan, NJ, USA
e. Lupron Depot, TAP Pharmaceuticals Inc
f. Tamofen, Phone-Poulenc Rorer Canada Inc, Montreal Quebec, Canada
g. Prepidil, Pharmacia and Upjohn, Kalamazoo, MI, USA
h. Imiquimod Aldara 5%, 3M Pharmaceuticals

References and Suggested Reading

1. Altman RB: Soft tissue surgical procedures. *In* RB Altman, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, p 726.
2. Antinoff N, Hottinger HA: Treatment of a cloacal papilloma by mucosal stripping in an Amazon parrot. Proc Assoc Avian Vet, 2000, pp 97-100.
3. Antinoff N, et al: Smooth muscle neoplasia of suspected oviductal origin in the cloaca of a blue-fronted Amazon parrot (*Amazona aestiva*). J Avian Med Surg 11(4):268-272, 1997.
4. Avgeris S, Rigg D: Cloacopexy in a sulfur-crested cockatoo. J Am Anim Hosp Assoc 24(4):407-410, 1988.
5. Baumgartner R: Endocrinologic and pathologic findings in birds with polyostotic hyperostosis. J Avian Med Surg 9:251-254, 1995.
6. Bennett RA, Harrison, GJ: Soft tissue surgery. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 1125-1131.
7. Bonda M, Rose RC: Western blot immunoassay and immunohistology supporting a papillomavirus as the etiology of a cloacal papilloma/ adenomatous polyp in a hyacinth macaw. Proc Assoc Avian Vet, 1998, pp 49-54.
8. Bowles HL: Management of chronic egg-laying with leuprolide acetate. Proc Assoc Avian Vet, 2000, pp 105-108.
9. Bowles HL: Management of cystic ovarian disease with leuprolide acetate. Proc Assoc Avian Vet, 2000, pp 113-118.
10. Bowles HL: Diagnosis and management of female avian reproductive diseases. Proc Assoc Avian Vet, 2001, pp 349-58.
11. Burke WH, Attis YA: Molting single comb white leghorns with the use of the Lupron® depot formulation of leuprolide acetate. Poult Sci 73:1226-1232, 1994.
12. Crosta L: New perspectives in avicultural medicine, including testicular biopsy. Exotic DVM 3(3):75-76, 2001.
13. Crosta L, et al: Endoscopic testicular biopsy technique in Psittaciformes. J Avian Med Surg 16(2):106-110, 2002.
14. Degernes L: Chronic oviduct obstruction and salpingitis in an African grey parrot. Proc Assoc Avian Vet, 1992, pp 156-157.
15. Denbow DM: Gastrointestinal anatomy and physiology. *In* Whittow GC (ed): Sturkie's Avian Physiology 5th ed. San Diego, Academic Press, 2000, pp 304-305.
16. Dlugi RW, Bahr JM: Lupron® depot (leuprolide acetate for depot suspension) in the treatment of endometriosis: A randomized, placebo-controlled, double blind study. Fertility and Sterility 54:419-427, 1990.
17. Dorrestein GM: Physiology of the urogenital system. *In* RB Altman, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 622-625.
18. Dvorak L, Bennett, RA, Cranor K: Cloacotomy for excision of cloacal papillomas in catalina macaw. J Avian Med Surg 12(1):11-15, 1998.
19. Eastin WC, Spaziani E: On the control of calcium secretion in the avian shell gland (uterus). Biol Reprod 19:493-504, 1978.
20. Filippich LJ, et al: The pharmacokinetics of carboplatin in sulfur-crested cockatoos (*Cacatua galerita*). Proc Assoc Avian Vet, 2000, pp 281-284.
21. Flinchum G, Harrison GJ: Observations on the effect of adrenal-blocking drugs in terminally ill pet birds. Exotic DVM 2(3):71-75, 2000.
22. Goodwin M, McGee ED: Herpes-like virus associated with a cloacal papilloma in an orange-fronted conure (*Aratinga canicularis*). J Assoc Avian Vet 7(1):23-25, 1993.
23. Gwinner E, Hau M: The pineal gland, circadian rhythms, and photoperiodism. *In* Whittow GC (ed): Sturkie's Avian Physiology 2nd ed. San Diego, Academic Press, 2000, pp 557-568.
24. Harcourt-Brown NH: Torsion and displacement of the oviduct as a cause of egg-binding in four psittacine birds. J Avian Med Surg 10:262-267, 1996.
25. Hillyer EV, Moroff S, Hoefer H: Bile duct carcinomas in two out of ten Amazon parrots with cloa-

cal papillomas. J Assoc Avian Vet 5:91-95, 1991.
26. Hochleithner M: Biochemistries. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 223-245.
27. Hudelson KS: A review of the mechanisms of avian reproduction and their clinical applications, Sem Avian Exot Pet Med 5(4):189-1998, 1996.
28. Hudelson KS, Hudelson P: A brief review of the female avian reproductive cycle with special emphasis on the role of prostaglandins and clinical applications. J Avian Med Surg 10(2):6-74, 1996.
29. Hurwitz S: Calcium homeostasis in birds. *In* Aurbach GD, McCormick DB (eds): Vitamins and Hormones Vol 45. New York, Academic Press, 1989, pp 173-221.
30. Johnson AL: Reproduction in the female. *In* Whittow GC (ed): Sturkie's Avian Physiology 5th ed. San Diego, Academic Press, 2000, pp 569-596.
31. Joyner KL: Theriogenology. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 748-804.
32. Kennedy FA, et al: Oropharyngeal and cloacal papillomas in two macaws (*Ara* sp.) with neoplasia with hepatic metastasis. J Avian Med Surg 10(2):89-95, 1996.
33. Keymer IF: Disorders of the avian female reproductive system. Avian Pathol 9:405-419, 1980.
34. King AS, McLelland J: Female reproductive system. *In* Birds: Their Structure and Function, London, Balliere Tindall, 1984, pp 145-165.
35. King AS, McLelland J: Male reproductive system. *In* King AS, McLelland J (eds): Birds: Their Structure and Function. London, Balliere Tindall, 1984, pp 166-174.
36. King AS, McLelland J: Cloaca and vent. *In* King AS, McLelland J (eds): Birds: Their Structure and Function. London, Balliere Tindall, 1984, pp 187-198.
37. King AS, McLelland J: Endocrine system. *In* King AS, McLelland J (eds): Birds: Their Structure and Function. London, Balliere Tindall, 1984, pp 214-128.
38. Kirby JD, Froman DP: Reproduction in male birds. *In* Whittow GC (ed): Sturkie's Avian Physiology 5th ed. San Diego, Academic Press, 2000, pp 597-612.
39. Konrath JR, et al: Herpesviral, but no papovaviral sequences, are detected in cloacal papillomas of parrots. Arch Virol 146(10):1869-1880, 2002.
40. Krautwald-Junghanns ME, Enders F: Ultrasonography in birds. Sem Avian Exot Pet Med 3(3):140-146, 1994.
41. Latimer KS: Oncology: *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 641-672.
42. Leach MW: A survey of neoplasia in pet birds. Sem Avian Exot Pet Med 1(2):52-64, 1992.
43. Lennox AM: The use of Aldara® (imiquimod) for the treatment of cloacal papillomatosis in psittacines. Exotic DVM 4(3):35-35, 2002.
44. Lightfoot TL: Clinical use and preliminary data of chorionic gonadotropin administration in psittacines. Proc Assoc Avian Vet, 1996, pp 303-306.
45. Lombardo MP, et al: The beneficial sexually transmitted microbe hypothesis of avian copulation. Behav Ecol 10(3):333-337, 1999.
46. Lupu CA: Evaluation of side effects of tamoxifen in budgerigars. J Avian Med Surg 14:237-242, 2000.
47. Marshal AJ: Reproduction. *In* Marshall AJ (ed): Biology and Comparative Physiology of Birds Vol II. New York, Academic Press, 1961, pp 169-213.
48. Millam JR: Reproductive management of captive parrots. Vet Clin No Am Exot An Pract 2:93-110, 1999.
49. Millam JR: Reproductive physiology. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 12-28.
50. Millam JR, Finney HL: Leuprolide acetate can reversibly prevent egg laying in cockatiels. Proc Assoc Avian Vet, 1989, p 46.
51. Millam JR, Finney HL: Leuprolide acetate can reversibly prevent egg laying in cockatiels (*Nymphicus hollandicus*). Zoo Biol 13:149-155, 1994.
52. Millam JR, Zhang B, el Halawani ME: Egg production of cockatiels is influenced by the number of eggs in nest after incubation begins. Gen Comp Endocrinol 101:205-210, 1996.
53. Orosz S: Anatomy of the urogenital system. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 614-644.
54. Ottinger MA, Bakst MR: Endocrinology of the avian reproductive system. J Avian Med Surg 9(4):242-250, 1995.
55. Paster MB: Avian reproductive endocrinology. *In* Rosskopf WJ, Woerpel RW (eds): Vet Clin No Am Vol:1343-1360, 1991.
56. Peckman MC: Reproductive disorders. *In* Biester HE, Peckman MC (eds): Diseases of Poultry 5th ed. Ames, Iowa State University Press, 1965, pp 1201-1205.
57. Phalen DN, Tomaszewski, E, Wilson VG: Internal papillomatosis: A herpesviral connection. Proc Assoc Avian Vet, 1998, pp 45-48.
58. Pollock CG, Orosz SE: Avian reproductive anatomy, physiology, and endocrinology. Vet Clin No Am 5:441-474, 2002.
59. Pye GW, et al: Endoscopic salpingohysterectomy of juvenile cockatiels (*Nymphicus hollandicus*). J Avian Med Surg 15(2):90-94, 2001.
60. Quesenberry KE, Hillyer EV: Supportive care and emergency therapy. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application, Brentwood, TN, HBD Int'l, Inc, 1999, pp 382-416.
61. Ramsay E: Cryosurgery for papillomas. J Assoc Avian Vet 5(2):73, 1991.
62. Ritchie BW: Papovaviridae. *In* Ritchie BW: Avian Viruses: Function and Control, Brentwood, TN HBD Int'l, Inc, 1995, pp 127-170.
63. Romagnano A: Avian obstetrics. Sem Avian Exot Pet Med 5:180-188, 1996.
64. Rosskopf WJ: Vaccine therapy for papillomas. AAV Today 1(5):202, 1987.
65. Rosskopf WJ, Woerpel RW: Egg binding in caged and aviary birds. Mod Vet Pract 65:437-40, 1985.
66. Rosskopf WJ, Woerpel RW: Cloacal conditions in pet birds with cloacopexy update. Proc Assoc Avian Vet, 1989, pp 213-231.
67. Rosskopf WJ, Woerpel RW: Avian reproductive endocrinology. Vet Clin No Am Exot Anim Pract 4:93-110, 1999.
68. Saito N, Koike TI: Interrelationship between arginine vasotocin, prostaglandin, and uterine contractility in the control of oviposition in the hen. Gen Comp Endocrinol 67:342-347, 1987.
69. Saito N, Koike TI: Alterations in uterine contractility during oviposition cycle in the domestic hen. Biol Reprod 432:378-394, 1990.
70. Scanes CG: Introduction to endocrinology: Pituitary gland. *In* Whittow GC (ed): Sturkie's Avian Physiology 5th ed. San Diego, Academic Press, 2000, pp 437-460.
71. Smith RE: Hysterectomy to relieve reproductive disorders in birds. Avian Exotic Pract :40-43, 1985.
72. Speer B: Clinical reproductive avian medicine, Proc Assoc Avian Vet, 1995, pp 23-33.
73. Speer B: Diseases of the urogenital system. *In* RB Altman, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 633-644.
74. Stauber E: Polyostotic hyperostosis associated with oviductal tumor in a cockatiel. J Am Vet Med Assoc 196:939-940, 1990.
75. Taylor M: Endoscopic examination and biopsy techniques. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 327-354.
76. Taylor M: Examining the avian cloaca using saline infusion cloacoscopy. Exotic DVM 3(3):77-79, 2001.
77. Tell LT, et al: A comparison of the effects of slow release, injectable levonorgestrel and depot medroxyprogesterone acetate on egg production in Japanese quail (*Coturnix coturnix japonica*). J Avian Med Surg 13(1):23-31, 1999.
78. Zantop DW: Using leuprolide acetate to manage common avian reproductive problems. Exotic DVM 2:70, 2000.
79. Zantop DW: Treatment of bile duct carcinoma in birds with carboplatin. Exotic DVM 2(3): 76-78, 2000.

CHAPTER
19

Endocrine Considerations

K. STORMY HUDELSON, BS, DVM, Dipl ABVP-Avian;
PAUL M. HUDELSON, BA, MA

Greg J. Harrison

Fig 19.1 | The European stork is a perfect example of birds learning to modify stressors.

Stress

Many things can be stressful, including isolation,[20] weather change,[101] separation from mate, forced exercise,[95] fear,[68] starvation,[67] handling,[60] toxins and abnormal physical conditions, such as infection, trauma and pain.[93] Stress involves a cascade of physiologically adaptive responses. These responses can be demonstrated in altered behavior, such as aggression, escape attempts, vocalizations, changes in feeding and drinking, feather picking, repetitive movements and suppression of reproduction.[79] These varied physiological reactions do not constitute a uniform set of adaptive responses occurring in a predictable temporal fashion (Fig 19.1).[93] The most reliable response to stress is an elevation in circulating corticosterone levels.[93,60]

In a set of experiments, the temporal association of stress responses was determined. Juncos measured at handling and again in 30 minutes demonstrated a fourfold increase of corticosterone levels.[60] In chickens given a continuous infusion of adrenocorticotropic hormone (ACTH), the corticosterone response was within 2 hours. Other significant indicators of stress were elevation of glucose by 12 hours; increased liver weight along with increased hepatic lipid and decreased moisture by 18 hours; decreased relative weight of the spleen by 24 hours; elevated heterophil:lymphocyte ratio by 2 days; decreased body weight and relative weight of the bursa of Fabricius, as well as the weight of the thymus, by day 4; and decreased liver-soluble protein content by 12 days. Elevations in cholesterol, triglycerides, high-density lipoproteins, corticosterone and glucose were found to be reliable indicators of stress.[111] Polydipsia and polyuria were evident in chickens within 1 to 2 days of ACTH infusion.[94] Feed intake was slightly elevated over the

week of ACTH infusion; however, body weight was 20% lower 1 week postinfusion at the end of the experiment.[94] This was apparently due to a decrease in digestion of proteins, carbohydrates, dry matter and gross energy. Fat digestion was unaffected. Digestion was more affected than absorption and returned to control levels within 1 week. Post-ACTH infusion, as digestion returned to normal, absorption of nutrients was reduced. Losses of skeletal muscle from prolonged gluconeogenesis by catabolism of muscle protein from excess corticosteroids were not recovered by the end of the experiment (1 week).[94]

Migratory birds undergoing a prolonged fasting period did not demonstrate elevated corticosterone levels until fat stores were depleted to less than 5% of body mass. At that point, muscle catabolism occurred due to a moderate elevation of corticosteroids. Further secretion of corticosterone is inhibited when additional stressors are encountered. This may be a means to continue migration by using muscle protein for energy while finding a suitable landing area at which to feed.[67] Increased corticosterone levels coupled with decreased fat stores stimulate appetite and foraging.[79] In non-migratory birds forced to exercise, corticosterone levels were elevated proportionally as the intensity of the exercise increased.[95]

Corticosterone also affects various behaviors in birds. It can cause an inhibition of territoriality, rearing of young and behavior associated with breeding, without affecting luteinizing hormone or testosterone levels (see Table 19.2).[126] Severe stressors cause regression of the reproductive system in chicken hens.[106] Pharmacological doses of corticosterone can induce a preovulatory surge of luteinizing hormone and ovulation, whereas dexamethasone blocks ovulation, possibly because it suppresses corticosterone.[106]

Fear behaviors are seen with increased corticosterone levels; they may be attenuated with vitamin C supplementation.[68] Exogenous corticosterone induces protein catabolism and causes chickens to seek a high-protein diet.[68] Feather picking is associated with increased corticosterone levels.[68] This suggests that some feather-picking behavior may be a stress response to protein catabolism.

Glucocorticoids induce epinephrine synthesis while ACTH stimulates epinephrine and norepinephrine release.[78,127] These catecholamines augment, through β-adrenergic receptors, and suppress, through α-adrenergic receptors, the plasma corticosterone responses to ACTH.[96] Increased epinephrine stimulates glycogenolysis, gluconeogenesis and lipogenesis. Norepinephrine helps maintain blood pressure.

Stress-induced immunomodulation has both beneficial and deleterious effects. Exogenous glucocorticoids or increased circulating levels of corticosterone cause involution of the thymus, bursa of Fabricius and spleen.[35] With early destruction of the bursa, the bursal hormones are not produced. This leads to a future stress hyporesponsiveness.[24] The composition of white blood cells moves toward an overall decrease in leukocytes, except heterophils, leading to an increased heterophil:lymphocyte ratio.[45] This decrease in leukocytes is partly due to the cells remaining in lymphoid organs.[24] A decrease in lymphocytes causes an overall suppression in antibody production.[79] In addition, glucocorticoids decrease inducible cellular cytotoxicity, lymphoproliferation, T-cell immunity, interleukin-2 and γ-interferon production.[65] Taken together, these responses may increase susceptibility to viral and other infectious agents.[45]

Lymphocytes themselves secrete ACTH and therefore boost circulating corticosterone levels following antigenic challenge.[81] The increase in corticosterone appears to cause a shift to increased T-helper cells and decreased T-suppressor cells in the spleen.[81] Macrophages are activated as they phagocytize an antigen. The activated macrophages secrete interleukin-1. Interleukin-1 increases secretion of corticotropin-releasing factor and ACTH, and activates lymphocytes, which increase production of ACTH.[81,82] Prolonged stress prior to antigenic stimulation may blunt the ACTH and interleukin-1 response.[15] The increased glucocorticoids cause the redistribution of circulating T-cells to secondary lymphoid tissues where antigens may be sequestered.[81]

Some amount of corticosterone is important in the immune response. Corticosteroids cause enhanced lymphocyte activity and the production of antibodies. Corticosteroids and ACTH later act in a negative feedback manner to regulate and control the process of antibody production by inhibiting lymphocyte activities and reducing the responsiveness to stimuli.[82] There is a significantly lower corticosterone response postantigenic stimuli in chicken strains that have autoimmune thyroiditis or avian scleroderma (another autoimmune disease).[56] This lack of response would attenuate the negative feedback of corticosterones on ACTH and may allow for a more pronounced immune response causing the autoimmune disease.

The administration of dexamethasone has been shown to depress ACTH and corticosterone for more than 24 hours.[56] In addition, both dexamethasone and prednisone decrease body weight gain, the number of natural killer cells, the number of lymphocytes in the spleen, production of interleukin-2 and γ-interferon by T-cells of the spleen.[65] These steroids also cause splenic atrophy,

increased body fat deposition, decreased percentage of immunoglobulin-A and immunoglobulin-M-bearing B-cells, decreased lymphoproliferation and increased susceptibility to *Eimeria* spp.[65]

The use of dexamethasone may cause more problems by not allowing for the natural effects of corticosterone. If steroid administration could be beneficial to the patient, the use of corticosterone or ACTH may be a better choice. When chickens infected with *Streptococcus faecalis* were given corticosterone at 30 mg/kg in feed in combination with ampicillin, the chickens were able to increase weight better than without corticosterone. Ampicillin alone did not alter the course of disease. Corticosterone alone was as good as the combination of ampicillin and corticosterone in most cases.[48] Another study demonstrated better resistance to *Escherichia coli* challenge exposure when corticosterone was given at 40 mg/kg in feed.[49] Pericarditis was reduced from 78% to 7%. No antibiotics were given. Resistance to respiratory infections was improved in pigeons given corticosterone, because of improved tracheal mucociliary transport.[72] Realizing that simply restraining a bird can elevate endogenous corticosterone tremendously, it may not be necessary to even treat with corticosterone, but simply to catch the bird several times.[60]

On the other hand, corticosterone depresses resistance to viral and *Mycoplasma gallisepticum* infections in poultry.[46] When chickens were given drugs to block the adrenal gland's production of corticosterone, fewer deaths and more effective cell-mediated immunity were seen to Newcastle disease and *Mycoplasma gallisepticum* infections. Additionally, a rapid remission of Marek's tumors was demonstrated.[47] Corticosterone and dexamethasone may decrease inhibin levels; this in turn increases follicle-stimulating hormone, which may cause follicle abnormalities.[53] Handling birds could conceivably exacerbate these diseases. Clinically, the use of mitotane (o,p'-DDD) has shown promise in controlling some neoplasias, viral infections and feather picking (G. Harrison, personal communication, 2002).

Many clinical problems may be due to a stressful environment (i.e., fatty liver disease, feather picking and reproductive abnormalities). Many patients present with liver disease. In theses cases, the liver may not be able to metabolize natural or exogenous glucocorticoids. Appropriate nutrition is imperative to the health of our patients; however, nutrition and other therapies cannot override conditions brought on by chronic stress. The best therapy for many of the diseases we encounter should be directed toward the psychological well-being of our patients (Figs 19.2, 19.3 and Tables 19.1, 19.2).

Table 19.1 | Causes of Corticosterone Elevation

- Immobilization
- Protein restriction
- Starvation
- Fear
- Pain
- Increase in plasma osmolality
- Oviposition
- Prostaglandins
- Low 3,5,3'-triiodothyronine (T_3)
- Depressed liver metabolism of corticosterone
- Growth hormone
- Angiotensin II (See Fig 19.8)
- Prolactin
- Parathyroid hormone
- Catecholamines
- Serotonin
- Vasoactive intestinal peptide
- Activated immune cells
- Corticotropin-releasing factor
- Arginine vasotocin
- Mesotocin
- Adrenocorticotropin hormone (ACTH)
- Melatonin
- Progesterone
- Opioids
- Exercise

Table 19.2 | Effects of Corticosterone

Increases	Decreases
• Feeding behavior • Fat stores • Gastric transit time • Muscle catabolism • Glucose • Insulin (counteracted by down regulation of liver receptors, thus causing insulin resistance) • Involution of the thymus, bursa and spleen • Heterophil: Lymphocyte ratio • Trapping of leukocytes in secondary lymphoid organs • Susceptibility to viral infections, coccidiosis and *Mycoplasma* spp. • Resistance to some bacteria • Tracheal mucociliary clearance • Fearfulness • Oral stereotypic behavior (feather picking, polydipsia, pecking) • Protein eating • Sodium reabsorption in renal tubules • Level of sodium-linked water uptake across the intestinal and hindgut mucosa • Catecholamines • Cholesterol • Triglycerides • High-density lipoproteins • Liver size (due to hepatic lipogenesis) • Salt gland secretion of NaCl • Lipolysis from adipocytes • Free fatty acids • Luteinizing hormone • Glomerular filtration rate	• T_3 • Territoriality • Growth • Body weight • Circulating WBC • Lymphoproliferation • Interleukin-2 • γ-interferon • Inducible cellular cytotoxicity • Cell-mediated immunity • Antibody response • Potassium excretion in renal tubules • Absorption and digestion of nutrients

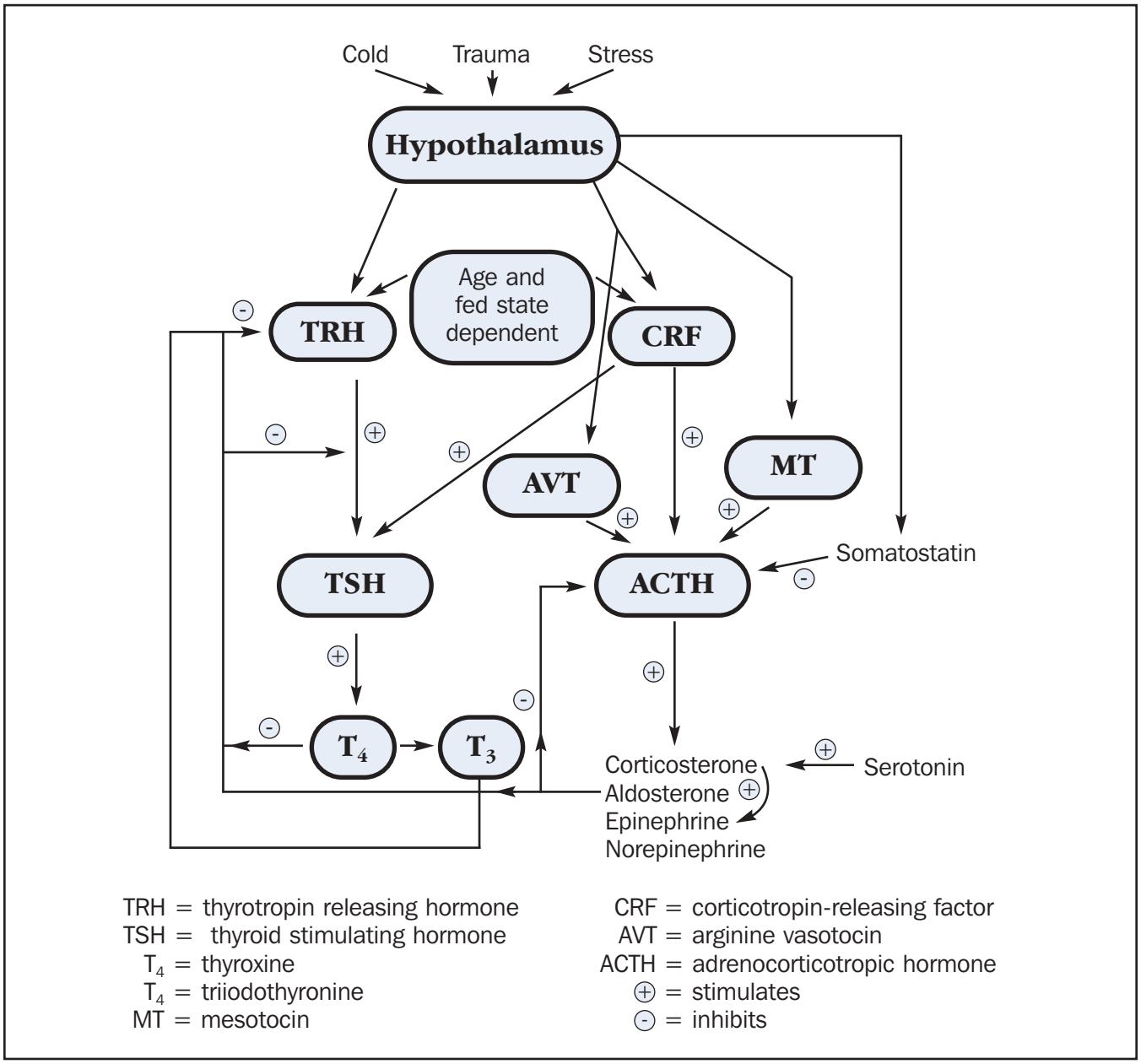


Fig 19.2 | Hypothalamo-Pituitary-Adrenal Axis Hormones

Growth

Growth primarily involves cell proliferation, but also may result from cell hypertrophy. The primary hormones involved with growth are: growth hormone (GH), triiodothyronine (T_3) and insulin-like growth factor-1 (IGF-1). Contradictions in the literature due to variable experimental designs are exacerbated by the different actions of various hormones, depending on age and nutritional status. Fig 19.4 demonstrates an overview of hormonal interactions. Tables 19.3-19.5 delineate what effects these hormones have on growth and body condition.

The reader should be aware that all available growth studies look at growth in poultry. There may be some undiscovered variations among other species of birds.

There are many hormones active during embryogenesis. Insulin and insulin-like growth factors are reported to stimulate embryonic metabolism, growth and differentiation.[41] Some growth factors appear to have more specific actions in embryogenesis, though many seem to have synergistic activity with IGF-1. Table 19.6 lists some of the growth factors and their respective actions.

As poultry moves into its rapidly growing phase at 3 to 4 weeks posthatch, GH and IGF-1 levels peak and then gradually decline to their minimum concentrations at about 8 weeks posthatch. During this period of rapid growth, administration of exogenous GH or IGF-1 demonstrates no effect on growth rate.[22,85] As endogenous nutrient partitioning is shifted toward greater fat deposition, as seen with increasing age and cessation of

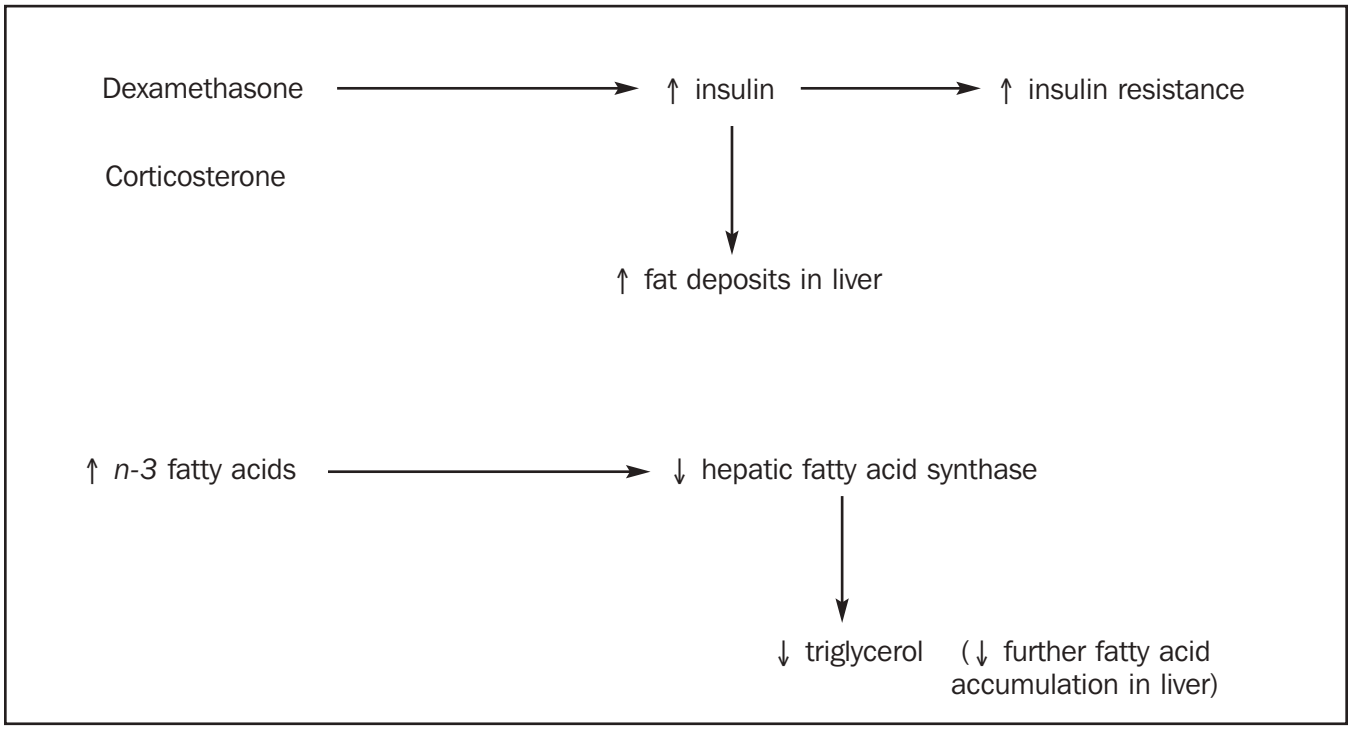


Fig 19.3 | Fatty Liver

growth, exogenous GH is effective in decreasing fat and increasing muscle formation.[85] GH in adult birds is a potent lipolytic hormone and also increases blood glucose levels.[22] There does not appear to be a diabetogenic effect, as seen in humans administered GH, possibly due to increased sensitivity of peripheral insulin receptors. GH also can block glucagon-induced lipolysis resulting in antilipolytic activity. Exogenous GH, in 8- to 9-week posthatch poultry, reduced feed intake and body weight gain.[119] The reduction of feed intake may be secondary to GH's reduction of neuropeptide Y protein, a potent appetite stimulant.[119]

In growing birds, GH decreases T_3 degradation resulting in elevated T_3 concentrations.[30] The effect of T_3 is developmentally regulated. Exogenous GH produces a hyperthyroid response in late embryogenesis, newly hatched and adult birds.[29,75] This effect disappears during the rapid growth phase, most likely due to masking by high endogenous GH and T_3 levels.[30,119] In mammals, GH must bind to two sites of the receptor, similar to antibody-antigen cross-bridging. Therefore, high levels of GH may block signal transduction.[37]

Growth hormone receptors and growth hormone-binding proteins also may play an important part in age-related actions. In young birds, growth hormone receptors are low, and GH and growth hormone-binding protein levels are high. Exogenous GH would have little to no effect. In adult chickens, growth hormone receptors are elevated and GH is low. In this case, exogenous GH may initially have stimulatory effects; however, at some point, the excess GH may be bound by increased levels of growth hormone-binding protein and GH-induced down regulation of growth hormone receptors.[53] Binding of GH may keep it from attaching to receptors, but also may prolong its half-life.[53]

β-adrenergic receptor agonists, epinephrine and norepinephrine also appear to have a significant role in growth and body composition. When clenbuterol (β-adrenergic receptor agonist) was given orally, growing chickens decreased their fat mass, while increasing their muscle mass, weight gain and gain-to-feed-intake ratio.[85] These changes could be from a direct effect on muscle and adipose tissue, modification of blood flow, central nervous system control of feed intake and/or action of other hormones (ie, T_3, corticosterone, insulin, GH).

The majority of information on growth in birds is still quite controversial, with many questions left unanswered. Those answers found in poultry may have little to offer other species of birds.

Thyroid

Thyroid hormones influence or are influenced by almost all physiological, environmental and nutritional parameters. When interpreting the literature, it is important to correlate the age of bird with the actual dose of drugs used in the experimental design to interpret the data correctly.

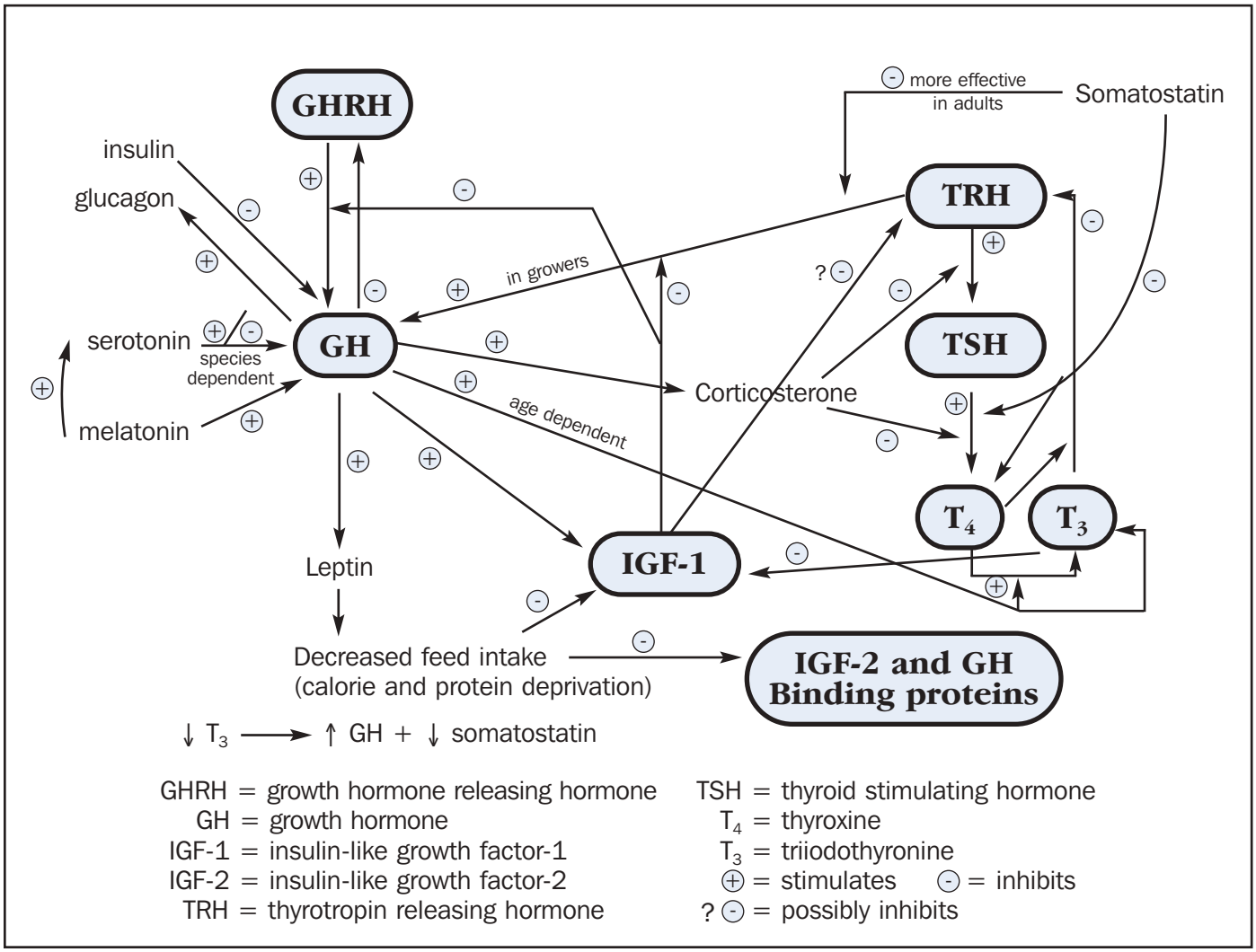


Fig 19.4 | Growth Hormone

Table 19.3 | Growth Hormone Effects

Increases	No Effect	Decreases
• Differentiation and proliferation of target cells (i.e., muscle) • Glucose • Fat in growing birds • Insulin-like growth factor-1 in selected muscles of adults • Exogenous GH: Fat birds between 3-5 weeks of age, lipolytic Other birds lipogenic Increase insulin and tryglycerides at 24 days of age Increase tibia length between 2-24 days of age In adult birds, large increase in T_3 • Large increase of T_3 at late embryo early hatch • Conversion of thyroxine (T_4) to triiodothyronine (T_3) • Moderate increase of T_3 at growth stage due to decreased degradation of T_3	• Exogenous GH 2-24 days post hatch - no effect on growth, feed intake, carcass protein, 8-9 weeks post- hatch - no effect on IGF-1 levels	• Fat in adults • Neuropeptide Y • Feed intake at 8-9 weeks post-hatch • Muscle growth at 8-9 weeks old • Hypothalamic epinephrine • Degradation of T_3

Table 19.4 | Effects of Insulin-like Growth Factor-1 (IGF-1)

Increases	No Effect	Decreases
• Metabolism, growth, differentiation, organogenesis of the embryo • Fat in growing poultry • Glucose uptake • Amino acid uptake and protein synthesis • Inhibition of protein degradation • Proliferation of turkey and chicken myoblasts • Peripheral tissue sensitivity to insulin • Exogenous IGF-1 increases heart weight and muscle breakdown in 3-week-old chickens	• Exogenous IGF-1 has no effect on growth or protein synthesis rate	• Insulin • Insulin:glucagon ratio • GH lipolysis in growing birds • Muscle growth in chickens greater than 8 weeks posthatch • Muscle weight in 3-week-old chickens • Glucose

Table 19.5 | Effects of Triiodothyronine (T_3) on Growth

Increases/Stimulates	Decreases	Low Levels of T_3
• Accretion of muscle protein during growth • Muscle mass and feather development in growers	• GH • IGF-1: may be age-dependent at low dose • Fat in growing birds • Muscle mass due to decreased protein synthesis in non-growing birds	• Increase GH • Decrease skeletal, muscle and feather development in growers • Decrease cardiac ventricle and pectoral mass in growers

Table 19.6 | Growth Factors Involved with Embryogenesis

Increases/Stimulates	Factors
Insulin-like Growth Factors (IGF)	• IGF-1 most important (see Table 19.4)
Epidermal Growth Factor (EGF)	• With IGF-1 and Fibroblast Growth Factor (FGF) stimulates proliferation of heart mesenchymal cells
Fibroblast Growth Factor (FGF)	See EGF Stimulates: • Muscle and bone development • Differentiation of blastoderm cells into erythropoietic cells • Proliferation of preadiposites • Proliferation of chondrocytes with transforming growth factor-β (TGF-β) • With IGF-1 proliferation of chicken myoblasts
Nerve Growth Factor (NGF)	• Neural development
Transforming Growth Factor-β (TGF-β)	Includes several TGF-βs, e.g.: • Inhibin/Activin subunits • Bone Morphogenic Protein-4 (BMP-4) • Glial Cell-Derived Nerve Growth Factor (GDNF) • Muellerian Inhibitory Substance (MIS) • Platelet-Derived Growth Factor (PDGF)
TGF-β	Decreases: • DNA synthesis and melanocyte formation in neural crest cells • Collagen production Stimulates: • Expression of fibronectin • Proliferation of chondrocytes with FGF and adiposites • Limb formation • Synthesis of non-collagen protein • Muscle cell differentiation
Inhibin	• Blocks release of follicle-stimulating hormone (FSH) • With activin may play an antagonistic role in paracrine regulation of embryonic gonadal steroidogenesis
Activin	See Inhibin • Stimulates release of FSH • Determines right and left asymmetry
Bone Morphogenic Protein-4 (BMP-4)	Stimulates: • Osteoinduction • Gastrulation • Neural crest cell apoptosis
Glial Cell-Derived Nerve Growth Factor (GDNF)	• Increase survival of motor neurons by reducing programmed apoptosis
Muellerian Inhibitory Substance (MIS)	• Muellerian duct regression in males
Platelet-Derived Growth Factor (FDGF)	• Stimulates transformation of chicken myoblasts • Inhibits differentiation

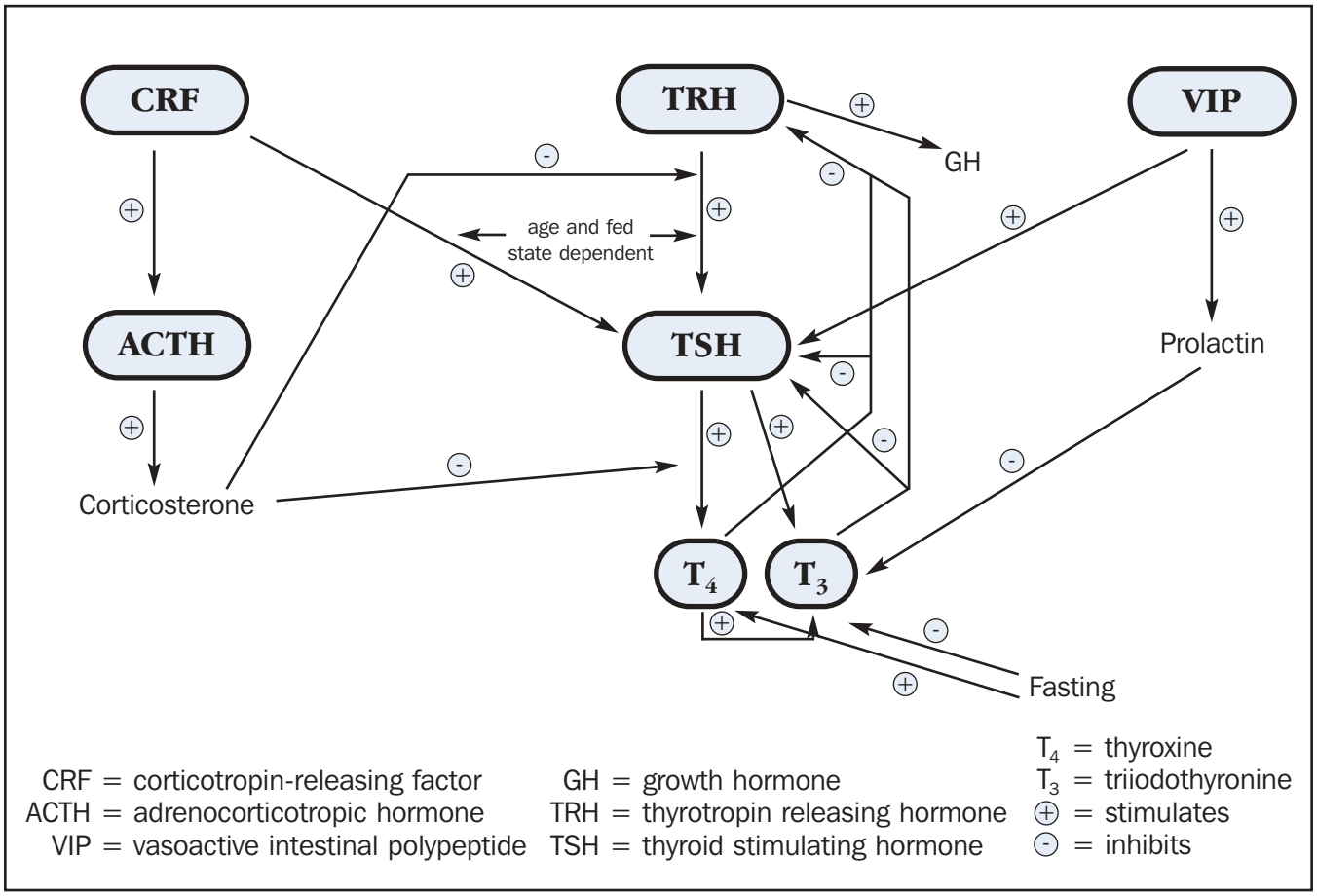


Fig 19.5 | Thyroid Hormones

In chickens of all ages, thyroid stimulating hormone (TSH) stimulates the release of predominately thyroxine (T_4) and lesser amounts of triiodothyronine (T_3). Age changes the mechanisms controlling TSH release and the peripheral productions of T_3. In the embryo, corticotropin releasing factor and thyrotropin releasing hormone may control TSH release.[105] Thyrotropin releasing hormone stimulates the release of growth hormone (GH), which inhibits degradation of T_3 through the growing phase (see Growth section, this chapter). Growing chickens respond differently to thyrotropin releasing hormone when fasted. During a 1-day fast, thyrotroph cells increase their sensitivity to thyrotropin releasing hormone and corticotropin releasing factor. However, these cells do not release TSH because fasting induces a decrease in thyrotropin releasing hormone secreted by the hypothalamus.[40] Fasting also increases corticosterone levels, which inhibit secretion of thyrotropin releasing hormone and, thus, TSH release. This was demonstrated in chickens with one injection of 40 μg of corticosterone.[40]

As chickens mature, they are less responsive to thyrotropin-releasing hormone effects on TSH. TSH appears to be more sensitive to circulating concentrations of T_3 than T_4.[105] Figs 19.2, 19.4 and 19.5 elucidate some of the pathways involved in thyroid regulation.

PHOTOSTIMULATION

Increasing day lengths induce photostimulated reproductive activity and postnuptial molt. It has been suggested that this onset of photostimulation combined with endogenous thyroid hormones organize the events of seasonality.[124] When American tree sparrows (*Spizella arborea*) were moved from short to long days, T_4 along with circulating luteinizing hormone increased early during photostimulation, peaking between weeks 1 and 2. This was followed by luteinizing hormone-releasing hormone and luteinizing hormone peaking between weeks 2 and 4. By week 6, gonad development was near maximum. Thyroid activity peaked during copulation.[25,97,123,124] The postnuptial molt began at week 9 and ended by week 18.[97] A series of experiments on intact and variably timed thyroidectomies concluded that thyroid-dependent gonadal growth was programmed during the first 7 days of photostimulation. Photorefractoriness and initiation of molt were programmed between 7 and 21 days of photostimulation.[53] T_3 failed to program birds for these events. This suggests that T_4 directly or non-T_3 metabolites are the responsible hormones.[124]

Cases

Most reports of thyroid conditions in birds involved those genetically designed for hypothyroidism (ie, chickens of the obese strain, and white carneaux pigeons). The only case of confirmed hypothyroidism in a psittacine was reported in a scarlet macaw. This report described contour feather loss, no indication of feather regrowth and excess subcutaneous fat. Skin biopsies demonstrated a catabolic state, a finding inconsistent with hypothyroid cases. A TSH stimulation test confirmed that the bird was hypothyroid. The bird responded to thyroid hormone replacement in the water at 0.015 mg/kg BID, which was later reduced to daily therapy.[89] It was not clear if this was a lifelong therapy or was discontinued after the resolution of signs. If the bird was taken off thyroid medication and remained euthyroid, it could have been due to a resolving thyroiditis, stressful environment and/or inadequate nutrition.

Goiter has often been associated with iodine-deficient budgerigars. Another incidence of goiters was reported in a goose flock of 2300 birds.[66] Though geese feeding on rapeseed meal develop goiters, rapeseed was not part of this flock's ration. The major pathological findings were increased relative thyroid weight, fat accumulation, and retarded growth and plumage development. One group of birds was treated with iodine and another group was not. After 55 days, both groups improved. A hunt for possible goitrogenic substance was not successful.[66] The two study groups were put in less dense environments. This may have resulted in less stress, therefore less circulating corticosterone and less suppression of thyroid activity. A recent report found a disproportional number of macaws with hyperplastic goiter. Seventy-five percent of these cases were in blue and gold macaws (*Ara arauna*). The cause was not determined.[104]

Avian muscular dystrophy in chickens has been treated by thyroidectomy. Maintenance of low T_3 plasma levels with exogenous T_3 replacement alleviated signs attributable to the disease. These chicks demonstrated a large increase in T_3 maximum binding capacity in dystrophic muscles.[73]

Cysts, adenomas and adenocarcinomas have been reported infrequently in several genera of birds. As intensive breeding and pollution with hormonally disruptive chemicals increase, thyroid disease may be reported more frequently.

TESTING

Most available thyroid tests are designed for humans or dogs. These tests are not usually able to detect the relatively low concentrations of T_4 in the avian patient.[43] When testing for thyroid hormones, it is important that the test be validated for birds. The necessity of incubating serum samples with proteolytic enzymes, and then precipitating with ethanol to avoid binding proteins in the serum has been demonstrated.[44] Without these extra steps, the test will be inaccurate.[43] Another source of difficulty is the lack of good reference ranges. With so many variables capable of altering thyroid levels, it is important that those birds used for reference ranges be fed, housed and cared for optimally.

T_4 blood levels fluctuate normally during a 24-hour cycle, generally being higher in the night and during a fast.[84] To measure T_4 activity more accurately, a TSH stimulation test is required. Recently, a stimulation test in several genera of birds has been developed using synthetic human thyroid stimulating hormone.[44] It was found that a 0-hour baseline blood sample, TSH at 1.0 IU/kg IM and a second blood sample in 6 hours gave consistent results.

Pancreas

ENDOCRINE-GLUCOSE REGULATION

Glucose regulation is the primary endocrine function of the pancreas. Effective glucose metabolism also maintains normal protein and lipid metabolism. Neural, retinal and adrenal tissues require glucose. The avian liver is the primary source of glucose to the plasma by its interaction with the pancreas. The pancreas releases the proper ratio of insulin and glucagon. The combination of hormones and absorbed nutrients work together in the liver to release products of digestion in dynamic response to the bird's needs. After a bird feeds, plasma glucose and amino acid levels increase causing insulin release from the pancreatic B-cells. Insulin aids hepatic enzymes in glycogenesis. In the liver, protein synthesis and lipogenesis produce approximately 95% of lipids through insulin stimulation. Thyroid hormones augment the process. Insulin is therefore an anabolic hormone.

In the fasting state, the liver is important for retrieving energy sources and making them available. With lower blood glucose concentrations, insulin levels are low and glucagon levels are high. At the liver, glucagon causes lipolysis, glycogenolysis and stimulates gluconeogenesis.[112] Glucagon is therefore a catabolic hormone. The glucagon insulin responses to food intake maintain stable blood glucose levels. A variety of species of birds demonstrate only small declines during prolonged (5 to 7 days) fasts.[114] We have seen emaciated birds with only slightly decreased blood glucose. A study in migrating garden warblers (*Sylvia borin*) found that glucose utilization rate is reduced as food deprivation continues to

about 7 days. These experiments suggest that during migration, a period of prolonged fasting, glucose utilization may be supplanted by oxidation of fatty acids.[113]

Adipose, kidney and muscle tissues each play a role in maintaining energy sources. The kidney can provide up to 30% of glucose by gluconeogenesis. Adipose tissue is important in providing lipids through lipolysis.[39] During a catabolic state, muscle tissue itself may be broken down (see Stress section, this chapter).

Cholecystokinin, glucagon and a mixture of absorbed amino acids strongly stimulate, whereas glucose only weakly stimulates insulin release. Insulin enhances protein synthesis and is required for embryonic myogenesis and muscle structural proteins in adult birds. At the level of the muscles and adipocytes, insulin increases glucose transport carriers. These proteins increase the transport of free glucose from the extracellular fluid across the cell membrane. Insulin aids transport of amino acids into cells. Insulin, or its hypoglycemic effect, causes glucagon release and results in an increase in free fatty acids. Therefore, insulin's overall effects are to remove glucose and amino acids from circulation, increase lipidemia and inhibit gluconeogenesis.

Free fatty acids and cholecystokinin stimulate glucagon release from the pancreatic A-cells. In contrast to other avian species, ducks also release glucagon in response to insulin and somatostatin. Glucose inhibits glucagon release. Glucagon suppresses leptin, a hormone that suppresses appetite, T_3 and T_4.[3,83]

Somatostatin controls the ratio of insulin to glucagon released from the pancreas. Islet cells appear to be contiguous with each other and share intra-islet extracellular fluid. Somatostatin from pancreatic D-cells inhibits secretion from all other islet cells. Glucagon is the most sensitive and pancreatic polypeptides are the least sensitive to somatostatin. At the level of the gut, somatostatin decreases intestinal absorption of glucose and lipids. This hormone's actions allow a dynamic regulation of gluco-homeostasis.

Absorbed nutrients stimulate D-cell somatostatin and A-cell glucagon activity. Increased glucose and amino acids stimulate B-cells (insulin) whereas glucose depresses A-cells (glucagon). F-cells (pancreatic peptides) are stimulated by cholecystokinin, secretin, gastrin and absorbed amino acids. In summary, A-cells stimulate B- and D-cells; D-cells inhibit A-, B- and F-cells. Pancreatic polypeptides from the F-cells are glycogenolytic without producing hyperglycemia. They also act to lower plasma glycerol, cholesterol, fatty acids and apolipoproteins, while increasing triglyceride levels.[105] Pancreatic polypeptides inhibit gut motility and gut, exocrine pancreatic and gallbladder secretions. They also are antilipolytic and induce satiety via the central nervous system.[55]

EXOCRINE

Exocrine pancreatic secretions include trypsinogen, chymotrypsinogen, trypsin inhibitor, procarboxypeptidase, amylase and lipase. These enzymes are released into the duodenum and, along with gut peptides, further digestion. Trypsinogen and chymotrypsinogen are secreted in an inactive form so as not to induce autodigestion. Enterokinase from the gut activates trypsinogen to trypsin. Trypsin, in turn, cleaves chymotrypsinogen to chymotrypsin.

Factors influencing exocrine pancreatic enzyme secretion include the following: feeding, distention of the proventriculus with peptones, gastrin-releasing peptides, increased cholecystokinin, vasoactive intestinal peptide, neurotensin, secretin, hydrochloric acid, vagus innervation and cholinergic agents.[23,59,74,100,118]

Cholecystokinin influences the enzymatic content of pancreatic secretions except amylase.[34,86] Secretin, and to a larger extent vasoactive intestinal peptide, increase the aqueous component of pancreatic juices. Neurotensin increases lipase and depresses amylase secretion. Ingestion of lipids causes neurotensin release from intestinal stores.[33] Diets rich in carbohydrates increase amylase, fats increase lipase, and protein increases chymotrypsin activity in pancreatic secretions.[19,64] The release of some of the islet hormones occurs before nutrients are even absorbed into the bloodstream, allowing for rapid adjustments to maintain homeostasis.

Exocrine Pancreatic Insufficiency

Exocrine pancreatic insufficiency in birds has been suggested by the observation of undigested food in the feces, voluminous foul-smelling droppings, weight loss and failure to thrive. With many cases of psittacine proventricular dilation presenting similarly, appropriate biopsies may be indicated to assist in diagnosis. Pancreatic insufficiency has been reported as a secondary effect of a pancreatic adenocarcinoma.[98] This yellow-naped Amazon (*Amazona ochrocephala auropalliata*) was presented with a 3-month history of weight loss and voluminous foul-smelling droppings. Confirmation of pancreatic insufficiency was made from fecal tests positive for neutral and split fats and negative for trypsin. In addition, no elevation in blood triglyceride levels occurred with oral corn oil administration until the oil was mixed with pancreatic enzymes. The pancreatic adenocarcinoma was found on necropsy.[98]

Another case in a macaw was reported with feces containing starch and fat. The bird's feces returned to nor-

mal with the addition of trypsin to the food. When trypsin was discontinued 6 weeks later, the feces remained normal.[90] A slightly acidic diet rich in peptones should increase pancreatic exocrine secretions. This may prove a useful therapeutic adjunct to the addition of pancreatic enzymes to the food.

Diabetes Mellitus and Pancreatitis

In most granivorous birds, hyperglycemia occurs with normal insulin and increased glucagon levels, however, some birds have been documented with low insulin levels.[90] Clinical signs attributable to hyperglycemia are severe polyuria, polydipsia, increased appetite, and weight loss.[90] Often there is a history of obesity and poor nutrition. Hyperglycemia may be secondary to other diseases and often resolves when the underlying disease is treated. Hyperglycemia in obese psittacines has responded to low-fat diets and weight loss. Obese Quaker parakeets (*Myiopsitta monachus*) have been found to have pancreatic necrosis.[71] Those that survive have pancreatic insufficiency. A number of viruses, such as herpes, pox and polyomavirus, may cause pancreatic pathology.[42] Birds on certain drugs (eg, medroxyprogesterone) have shown signs of hyperglycemia.

It has been suggested that an amylase greater than 2000 IU/L is diagnostic for pancreatitis. Lower values of amylase may be more indicative of renal disease. Chronic stress causes chronic hyperglycemia with increased insulin levels and resultant insulin resistance. To evaluate a bird with hyperglycemia it is important to keep environmental stressors to a minimum. Evaluating the home situation and diet may indicate stress and/or nutritional issues. If possible, glucagon, insulin and amylase should be evaluated along with evidence of persistent hyperglycemia. A urine dipstick in normal birds usually demonstrates negative or trace glucose. Radiographs and fecal Gram's stains may aid in the search for any underlying diseases.

Treatment

If insulin levels are depressed, the bird may respond to exogenous insulin. Insulin protocols are extrapolated from other species and are carried out in much the same way as in mammals. Somatostatin was tried in a sulfur-breasted toucan (*Ramphastus sulfuratus*) at 3 μg/kg SC BID. The bird improved clinically, but died acutely after 4 months. The body was not presented for necropsy. During therapy, the bird maintained high glucose (>1300 mg/dl) and glucagon (>3100 pg/ml) levels. It would be interesting to evaluate higher doses of somatostatin.[71] If glucagon is elevated, it would be interesting to try antiglucagon serum. When anti-insulin serum was used in chickens, glucose became extremely elevated.[107]

Oral medications may have promise. Glipizide, a drug that increases insulin release from β-cells, has been used successfully in some cases at 1 mg/kg PO BID (Echols, personal communication). Other oral medications that may be tried include thiazolidinediones, which enhance peripheral insulin sensitivity, and α glucosidase inhibitors, which delay glucose absorption from the gut. Chicks with dexamethasone-induced hypocorticalism demonstrated increased insulin sensitivity and attenuated glucagon responsiveness.[70]

Pineal Gland

The pineal gland is made of ependymal cells, photoreceptor-like cells and neurons.[17] The gland is located between the two hemispheres of the telencephalon and the cerebellum. This gland's photoreceptive-like cells are stimulated by light through pinopsen, a pineal-specific photoreceptive molecule, making the pineal a major component of the circadian pacemaking system.[91,117] The pineal's rhythmic synthesis and secretion of melatonin is regulated by neural signals from sympathetic nerves.[92] A second component of the circadian pacemaking system consists of a self-sustained oscillator, which is an area of the hypothalamic region possibly equivalent to the mammalian suprachiasmatic nuclei. The third component, at least in some galliforms and columbiforms, are the retinae of the eyes.[50,117] These three components work in concert to stabilize and amplify a self-sustaining circadian output. Light is the most powerful stimulus for the circadian rhythm and also can affect the system via the extraretinal/extrapineal photoreceptors located deep in the brain (Fig 19.6).[117] The relative contribution each part plays in keeping the rhythm differs between species and, at times, within the same individual. The predominant factor maintaining rhythm in the house sparrow is the pineal gland, in the pigeon it is both eyes and pineal gland, and in the Japanese quail it is the eyes.

Changes in the pacemaker are partly related to the rhythmic synthesis and release of melatonin. These changes may be important in environmental situations, such as mid-winter and summer in the high arctic where conditions are constant, and also in the ever-changing conditions encountered during migration.[50] In arctic mid-winter and mid-summer birds, and in migratory birds, there is a reduction in melatonin amplitudes. This reduction should result in a reduction in the degree of oscillation of the pacemaking system. A reduction in oscillation reduces the overall output of the pacemaker. This should facilitate adjustments to altered environmental conditions during migration, and in conditions that are more constant, it may enhance entrainability to weak Zeitgeber influences.[51]

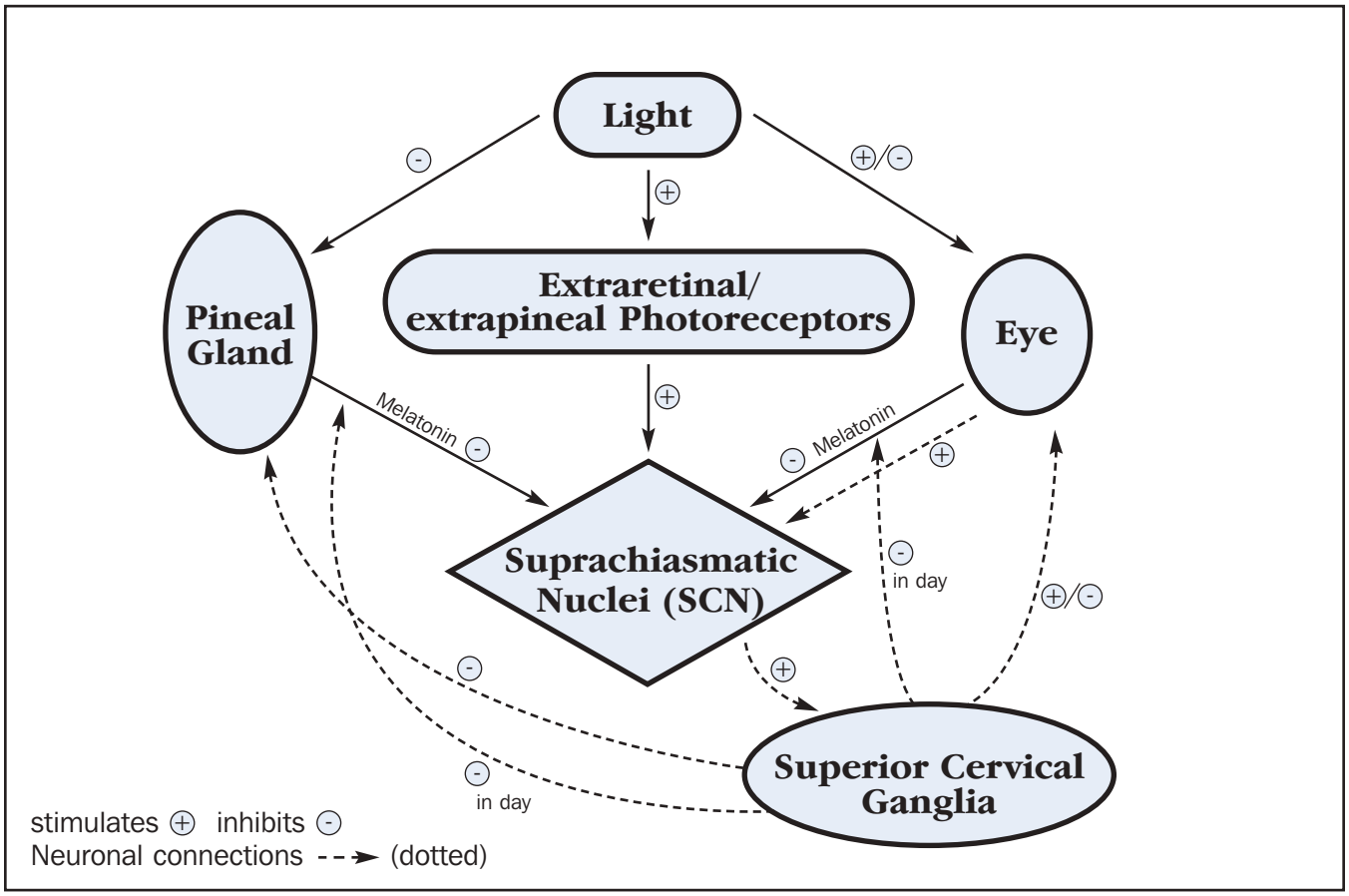


Fig 19.6 | Interactions of the Circadian Pacemaking System

Sometimes in constant conditions, the pacemakers are unable to sustain the persistent rhythm without periodic melatonin input from the eyes or pineal gland, or periodic neural input from the suprachiasmatic nuclei.[117] The most powerful stimuli of synchronization of the circadian rhythm is periodic alteration of light intensity.[2] Light affects melatonin synthesis by entrainment of circadian melatonin rhythms and acute inhibition of melatonin synthesis.[36] Light inhibition of melatonin causes sleep impairment in pigeons.[14]

Retinal photoreceptors appear to mediate light-induced suppression and photic entrainment in pigeons and Japanese quail.[36] Dopamine stimulates retinal melatonin synthesis. Dopamine has shown to have a rhythm of release opposite to melatonin, that is, increased levels in the day and decreased levels at night. Experiments have demonstrated that the interaction of dopamine and melatonin are necessary for maintaining the anti-phase relationship between the two rhythms.[36]

Melatonin also affects regulation of seasonal changes in immune function by enhancing cell-mediated immunity.[11] As discussed in the Song section in this chapter, melatonin also inhibits the neural plasticity of the song control system.[12,13,36] These seasonal regulations and the action of melatonin appear to be modified by the same central thyroid-dependent mechanism that controls the reproductive state in birds.[11]

Melatonin helps to time migration and the hatching of eggs, but does not fully coordinate reproductive activity with a favorable time of year.[11] In non-tropical birds, photoperiod is the predominant proximate factor. With increasing photoperiod in the spring, secretion of luteinizing hormone releasing hormone (LHRH) increases and results in gonadal maturation.[61] Breeding terminates while days are still long, due to induced photorefractoriness. The gonadal regression during this time is associated with a large decrease in LHRH. This suggests photoperiodic responses involve direct interactions between photoreceptors and LHRH neurons.[31]

Melatonin synthesis also has been demonstrated in Harderian glands, the gastrointestinal tract and retina. The extrapineal melatonin is only rarely released into the circulatory system. Presumably, it exerts its influence locally.[51,63] High concentrations of melatonin are found in enterochromaffin cells of the mucosal epithelium of the gastrointestinal tract.[63] Pinealectomized pigeons were found to have a 24-hour cycle of melatonin secretion in the gastrointestinal tract, with levels increased at night and decreased during the day.[63] Melatonin receptors have been found in a variety of tissues, which suggests

direct actions of melatonin on the function of different organ systems in response to internal and external stimuli.[102]

With the complex responses seen in birds due to duration and intensity of light, it seems paramount that the bird's natural photoperiod be accommodated. This is true for the home environment as well as the hospital. Night treatments should be done quickly, with an attempt not to shine light toward the head, because even a short duration of light exposure in the night can throw the natural circadian rhythm off. In 24-hour clinics, it is important to keep cages covered during the normal night duration.

THERMOGENESIS

The pineal gland influences thermoregulation. Pinealectomy performed on some species caused increased core body temperature and decreased tolerance to heat at night.[18] This effect returns to normal with the administration of melatonin. Exogenous melatonin in intact house sparrows decreased core body temperature by 4.7°C within 30 minutes.[16] Melatonin may be an effective antipyretic. Melatonin facilitates sleep and acts to synchronize the decrease in body temperature and metabolic rate in order to reduce energy expenditure during inactive periods.[14]

Another area of the brain that is involved in thermogenesis is the preoptic/anterior hypothalamic area (PO/AH) where warm- and cold-responsive neurons are found.[32] A number of neuroactive compounds have demonstrated effects in this area. When acetylcholine, dopamine, serotonin or norepinephrine were injected into the PO/AH of pigeons, the birds demonstrated peripheral vasodilation, inhibition of shivering and decreases in heat production.[58] Prostaglandin E_2 injected into the hypothalamus caused hyperthermia in pigeons and fowl, whereas prostaglandin $F_{2\alpha}$ caused hypothermia in fowl and hyperthermia in pigeons.[87] Arginine vasotocin reduces shivering, body temperature and oxygen consumption, whereas angiotensin-II has the opposite effects.[54] Thyroid hormones help to adjust the body temperature in response to the environment by increasing T_3 levels in cold and decreasing T_3 levels in warm surroundings.[26] This information implies that the hypothalamus is more an integrator of physiological inputs than a direct controller of thermogenesis.

It is clear that, under most situations, when a clinician is faced with a "sick" bird (excluding head trauma), keeping it warm is very important. If prostaglandins are administered to the bird or the bird's photoperiod is rearranged at this time, it would be prudent to monitor for changes in the patient's body temperature.

Song

Over the last decade, there has been an increased interest in the effects of hormones on behavior and changes in brain morphology. The complex learned behavior of singing is expressed differently in the different sexes of many species of birds. The current research suggests that steroid hormones can induce a neuroplasticity, which activates this behavior. This seasonal neuroplasticity is more dramatic in birds than in other vertebrates.

In many song birds, most notably the zebra finches (*Taniopygia guttata*) and canaries (*Serinus canaria*), a song control system (SCS) has been identified in the brain. The higher vocal center, a part of this system, appears to be unique to the songbird suborder.[4,21,88] This center is implicated in the learning of new song, as well as the production and perception of previously learned songs.[21,88] Several of the nuclei of the SCS contain high levels of androgen, estrogen, norepinephrine, and acetylcholine muscarinic and dopamine receptors along with acetylcholinesterase.[99] In addition, fibers containing neurotransmitters (catecholamines), or neuropeptides (vasoactive intestinal polypeptide, enkephalin, cholecystokinin, substance P and Metenkephalin) are present.[4,8,9]

Estrogenic metabolites of testosterone increase norepinephrine and dopamine turnover in the SCS, suggesting an interaction between the metabolites of testosterone and these catecholamines.[8,9] Androgen receptors in the SCS appear to be unique to song birds and the Anna's hummingbird (*Calypte anna*), a non-song bird.[38]

In the fall, when singing is not done to attract a mate, daylight is decreasing and sex hormone levels are low. In the song sparrow (*Melospiza melodia*), evidence suggests that the autumnal singing involves estrogen acting locally in the brain. The source of the neuroactive estrogen is unknown, but does not appear to be from testosterone of gonadal origin.[110] The administration of testosterone will increase song rate, whereas castration will reduce, but not always eliminate, male-typical song, depending on the species.[1] Castrated song sparrows in the wild sang at effective rates when territorial challenges occurred.[125] Testosterone influences song quality.[80] Studies indicate that it is the androgenic and estrogenic metabolites of testosterone, through testosterone's aromatization, that are required to completely restore the full rate of singing.[52] It appears that in zebra finches, estrogenic metabolites of testosterone selectively promote courtship song.[121]

Temperate zone wild male songbirds have seasonal peaks of testosterone associated with increasing daylight in the spring. At this time, increases in the volume of song control nuclei occur.[108,109] The song control nuclei

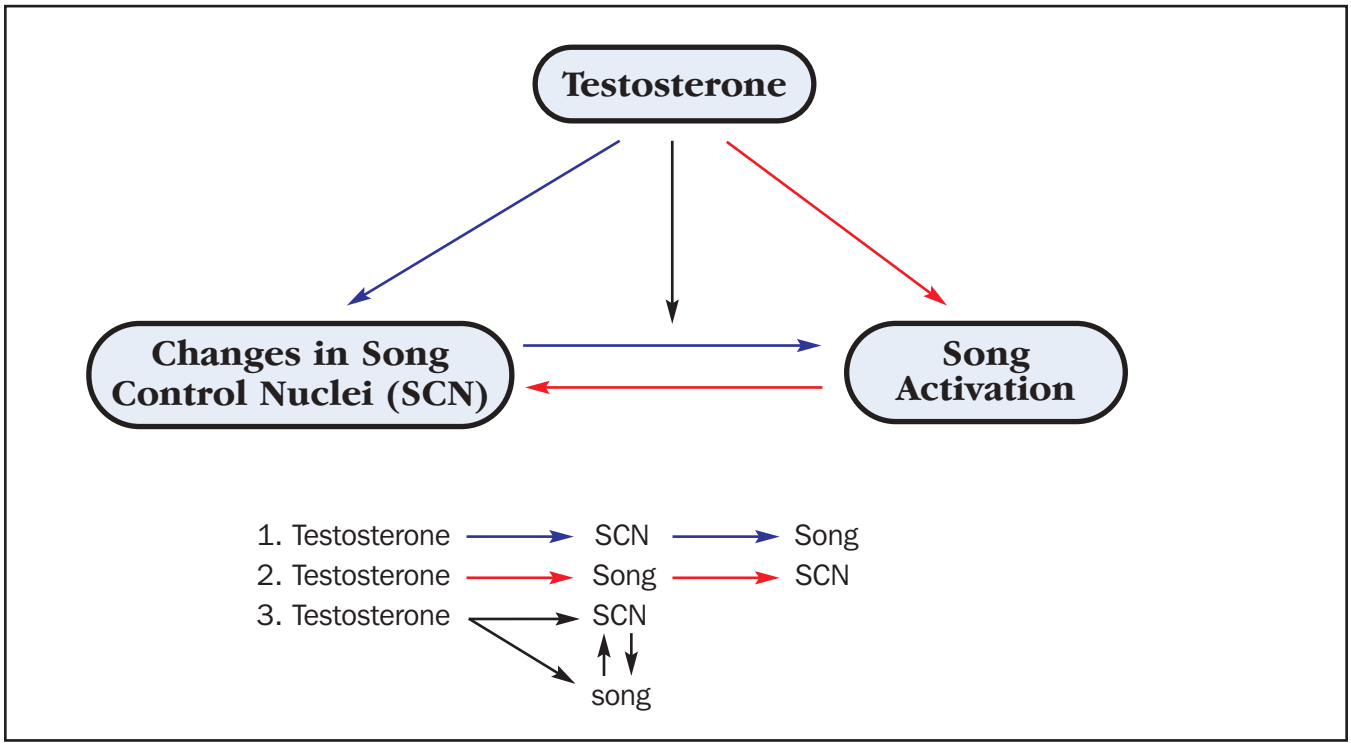


Fig 19.7 | Song Activation Models.

volume increase precedes the growth and development of the reproductive system.[116] Other environmental stimuli clearly play a role. Starlings in captivity, exposed to spring photoperiods, increased the volume of higher vocal center nuclei of the SCS, whereas males kept in outdoor aviaries in the spring had volume increases in the nuclei of most, if not all of the SCS.[10] It is possible that seasonal variation and/or steroid-induced changes in catecholaminergic afferent input into the SCS are important in regulating changes in the vocal learning or production of song, as well as the volume changes in nuclei of the SCS.

A significant amount of research indicates the importance of testosterone on the morphology of the SCS. Testosterone can restore SCS development in castrated males, corresponding to photoperiod changes in both gonad and SCS size.[1,108,109] Testosterone given to females results in male-typical song behavior and increase in the size of their song control systems.[5,115] There also is evidence that the SCS can grow in response to photoperiods independent of testosterone.[6] Recent data indicate that the volume of SCS nuclei correlates with song performance in some cases. It may be that high rates of singing could cause an increase in the SCS.[7] Brain-derived neurotrophic factor that promotes growth of the higher vocal center is released by singing activity. This information helps to explain the dissociation sometimes observed between steroid-induced and seasonal changes in the song system morphology and vocal behavior (Fig 19.7).

Melatonin also may play a role in song system morphology changes. The volume of the song control nuclei was studied in castrated starlings held under different photoperiods to induce reproductive states characteristic of different seasons. Long days are associated with larger volumes of the higher vocal center nuclei than are short days. Melatonin administration attenuated the long day-induced volume increases. The song system also was found to have high densities of melatonin receptors.[12,13] Long durations of melatonin secretion in late fall and winter subdue the effects of testosterone. With long days, melatonin secretion decreases rapidly and testosterone increases. It is apparent that the interaction between many hormones may fine-tune the activity of the SCS.

The SCS and behavior that induces singing are extremely complex. Before administering hormones to induce singing, it is imperative that we learn more about this system. More importantly, good nutrition and a good environment will allow the bird to sing when it is appropriate. A quiet bird out of breeding season may be a happy bird, with no need for male aggressive or territorial singing. Conversely, compulsive singing may be a sign of stress.

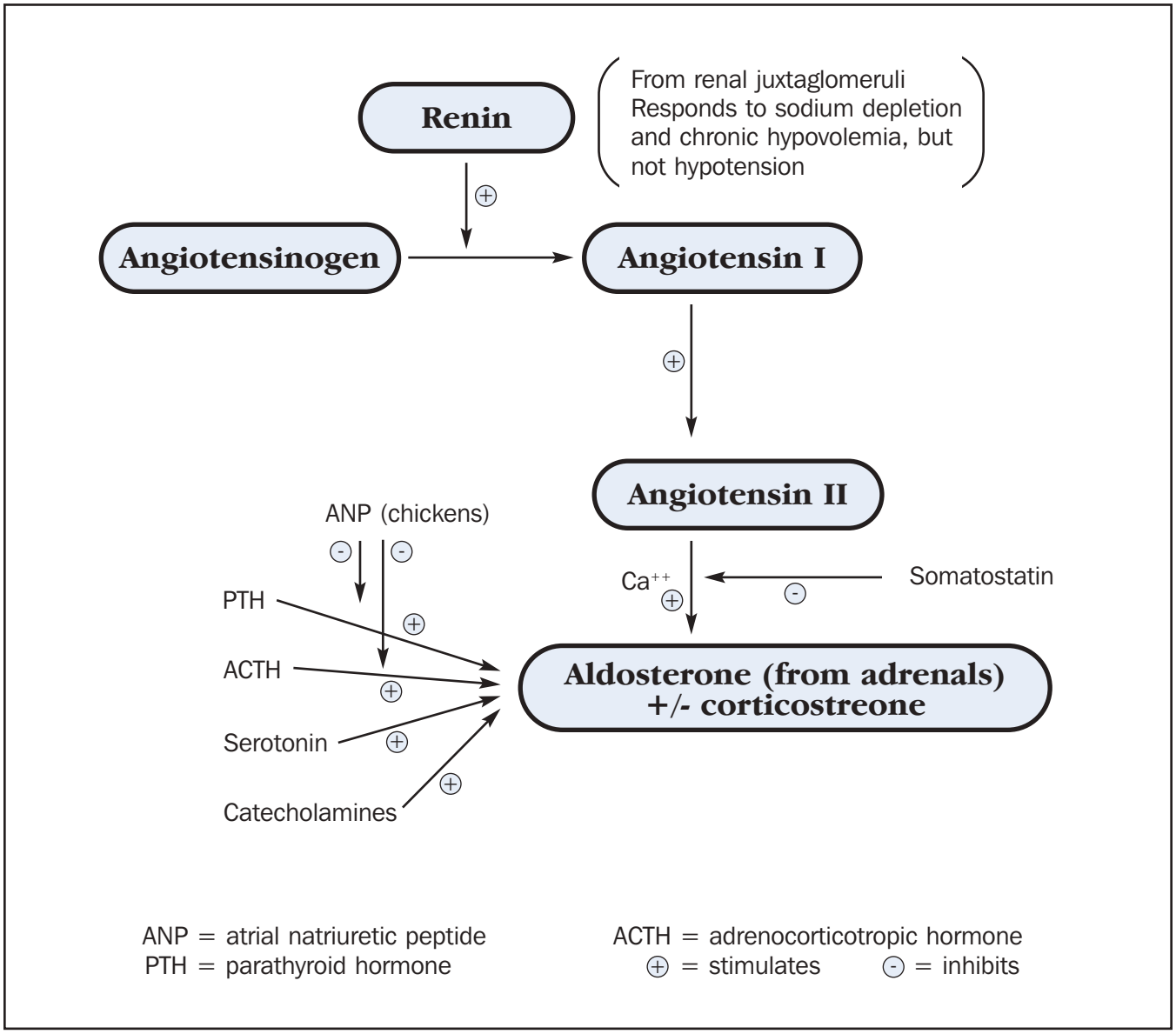


Fig 19.8 | Renin-Angiotensin System.

References and Suggested Reading

1. Arnold AP: The effects of castration and androgen replacement on song courtship, and aggression in Zebra finches. J Exp Zool 191:309-326, 1975.
2. Aschoff J (ed): Handbook of Behavioral Neurobiology, Volume 4: Biological Rhythms. New York, Plenum Press, 1991, pp 41-90.
3. Ashwell CM, et al: Hormonal regulation of leptin expression in boiler chickens. Amer J Physiol 276(1 Pt 2):R226-232, 1999.
4. Ball GF: Neurochemical specializations associated with vocal learning and production in songbirds and budgerigars. Brain Behav Evol 44:234-236, 1994.
5. Ball GF: Neuroendocrine basis of seasonal changes in vocal behavior among songbirds. *In* Hauser M, Konishi M (eds): The Design of Animal Communication. Cambridge, MA, MIT Press, pp 213-253, 1999.
6. Ball GF, Benard DJ, Wilson FE: Rapid effects of photoperiod on the volume of song control nuclei in American tree sparrows. The 5th meeting of the Society for Research on Biological Rhythms, Jacksonville, FL, 1996.
7. Ball GF, Riters LV, Balthazart J: Neuroendocrinology of song behavior and avian brain plasticity: Multiple sites of action of sex steroid hormones. Frontiers in Neuroendocrinology 23:137-178, 2002.
8. Barclay SR, Harding CF: Androstenedione modulation of monoamine levels and turnover in hypothalamic and vocal control nuclei in male zebra finch: Steroid effects on brain monoamines. Brain Res 459:333-343, 1988.
9. Barclay SR, Harding CF: Differential modulation of monoamine levels and turnover rates by estrogen and/or androgen in hypothalamic and vocal control nuclei of male zebra finches. Brain Res 523:251-262, 1990.
10. Benard DJ, Ball GF: Photoperiodic condition modulates the effects of testosterone on song control nuclei volumes in male European starlings. Gen Comp Endocrinol 105:276-283, 1997.
11. Bentley GE: Unraveling the enigma: the role of melatonin in seasonal processes in birds. Microsc Res Tech 53(1):63-71, 2001.
12. Bentley GE, Ball GF: Photoperiod-dependent and -independent regulation of melatonin receptors in the forebrain of songbirds. J Neuroendocrinol 12:745-752, 2000.
13. Bentley GE, Van't Hof TJ, Ball GF: Seasonal neuroplasticity in the songbird telencephalon: A role for melatonin. Proc Natl Acad Sci USA, 1999, 96 pp 4674-4679.
14. Berger RJ, Phillips NH: Constant light suppresses sleep and circadian rhythms in pigeons without consequent sleep rebound in darkness. Amer J Physiol 36(4):R945-R952, 1994.
15. Berkenbosch F, Wolvers DAW, Derijk R: Neuroendocrine and immunological mechanisms in stress-induced immunomodulation. J Steroid Biochem Molec Biol 40:639-647, 1991.
16. Binkley S: Pineal and melatonin: circadian rhythms and body temperatures in sparrows. *In* Sheving L, Halberg F, Pauly J (eds): Chronobiology. Tokyo, Japan, Igaku Shoin, 1974, pp 582-585.
17. Binkley S: The Pineal: Endocrine and Neuroendocrine Function. Englewood Cliffs, NJ, Prentice Hall, 1988.
18. Binkley S, Kluth E, Menaker M: Pineal function in sparrows: circadian rhythms and body temperature. Science 174:311-314, 1971.
19. Bird FH, Moreau GE: The effect of dietary protein levels in isocaloric diets on the composition of avian pancreatic juice. Poult Sci 57:1622-1628, 1978.
20. Bollweg G, Wei YX, Sparber SB: Behavioral and neuroendocrine assessment of ritanserin exposure in the developing chicken: Lack of toxicity at effective doses.

Pharmacol Biochem Behav 60:175-181, 1998.
21. Brenowitz EA: Comparative approaches to the avian song system. J Neurobiol 33:517-531, 1997.
22. Buyse J, Decuypere E: The role of the somatotrophic axis in the metabolism of the chicken. Domest Anim Endocrinol 17:245-255, 1999.
23. Campbell B, et al: Hormonal control of avian pancreas. Amer J Physiol 261:G16-G21, 1991.
24. Carsia RV, Harvey S: Adrenals. *In* Whittow GC (ed): Sturkie's Avian Physiology 5th Edition. New York, Academic Press, 2000.
25. Chauduri S, Maiti BR: Changes in thyroid activity during annual sexual cycle of a wild avian species, the tree pie *Dendrocitta vagabunda*. Eur J Morphol 34(4):321-325, 1996.
26. Cogburn LA, Freeman RM: Response surface of daily thyroid hormone rhythms in young chickens exposed to constant ambient temperature. Gen Comp Endocrinol 68:113-123, 1987.
27. Covasa M, Forbes JM: Selection of foods by broiler chickens following corticosterone administration. Br Poult Sci 36:489-501, 1995.
28. Dacke CG: The parathyroids, calcitonin, and vitamin D. *In* Whittow GC (ed): Sturkie's Avian Physiology 5th edition. New York, Academic Press, 2000, pp 473-488.
29. Darras VM, et al: Ontogeny of the effect of purified chicken growth hormone on the liver 5'-monodeiodination activity in the chicken: reversal of the activity after hatching. Gen Comp Endocrinol 77:212-220, 1990.
30. Darras VM, et al: Endogenous growth hormone controls high plasma levels of 3,3',5 triiodothyronine (T_3) in growing chickens by decreasing the T_3-degrading type III de-iodinate activity. Domest Anim Endocrinol 10:55-65, 1993.
31. Dawson A, et al: Photoperiodic control of seasonality in birds. J Biol Rhythms 16(4):365-380, 2001.
32. Dawson WR, Whittow GC: Thermoregulation. *In* Whittow GC (ed): Sturkie's Avian Physiology 5th edition. New York, Academic Press, 2000, pp 343-390.
33. DeGolier TF, et al: Neurotensin modulates the composition of pancreatic exocrine secretions in chickens. J Experimental Zool 283:455-462, 1999.
34. Dockray GJ: Comparison of the actions of porcine secretin in the cat and the turkey. J Physiol 244:625-637, 1975.
35. Donker RA, Beuving G: Effect of corticosterone infusion on plasma corticosterone concentration, antibody production, circulating leukocytes and growth in chicken lines selected for humoral immune responsiveness. Br Poult Sci 30:361-369, 1989.
36. Ebihara S, et al: In vivo microdialysis studies of pineal and ocular melatonin rhythms in birds. Biol Signals 6(4-6):233-240, 1997.
37. Fuh G, et al: Rational design of potent antagonists to the human growth hormone receptor. Science 256:1677-1680, 1992.
38. Gahr M: Neural song control system of hummingbirds: Comparison to swifts, vocal learning (songbirds) and nonlearning (suboscines) passerines, and vocal learning (budgerigars) and nonlearning (dove, owl, gull, quail, chicken) nonpasserines. J Comp Neurol 426:182-196, 2000.
39. George JC, John TM, Mitchell MA: Flight effects on plasma levels of lipid, glucagon and thyroid hormones in homing pigeons. Horm Metab Res 21(10):542-545, 1989.
40. Geris KL, et al: The Drop in plasma thyrotropin concentrations in fasted chickens is caused by an action at the level of the hypothalamus: role of corticosterone. Domest Anim Endocrinol 16(4):231-237, 1999.
41. Garb M, et al: Insulin and insulin-like growth factor-1 both stimulate metabolism, growth and differentiation in the postneurula chick embryo. Endocrinology 121:1477-1482, 1987.
42. Grahm DL, Heyer GW: Diseases of the exocrine pancreas in pet, exotic, and wild birds: a pathologist's perspective. Proc Annu Conf Assoc Avian Vet, 1992, pp 190-193.
43. Greenacre CB, et al: Validation of a novel high-sensitivity radioimmunoassay procedure for measurement of total thyroxine concentration in psittacine birds and snakes. Amer J Vet Res 62(11):1750-1754, 2001.
44. Greenacre CB, et al: The use of synthetic TSH to evaluate the thyroid gland. Proc Annu Conf Assoc Avian Vet, 2002, p 13.
45. Gross WB, Chickering W: Effects of fasting, water deprivation, and adrenal-blocking chemicals on resistance to *Escherichia coli* challenge. Poult Sci 66:270-272, 1987.
46. Gross WB: Effect of adrenal blocking chemical on viral and respiratory infections in chickens. Can J Vet Res 53:48-51, 1989.
47. Gross WB: Effect of adrenal blocking chemicals on the responses of chickens and turkeys to environmental stressors and ACTH. Avian Pathol 19:295-304, 1990.
48. Gross WB: Use of corticosterone and ampicillin for treatment of *Streptococcus faecalis* infection in chickens. Amer J Vet Res 52:1288-1291, 1991.
49. Gross WB: Effect of short-term exposure of chickens to corticosterone on resistance to challenge exposure with *Escherichia coli* and antibody response to sheep erythrocytes. Amer J Vet Res 53(3):291-299, 1992.
50. Gwinner E, Hau M, Heigl S: Melatonin: generation and modulation of avian circadian rhythms. Brain Res Bull 44(4):439-444, 1997.
51. Gwinner E, Brandstatter R: Complex bird clocks. Philos Trans R Soc Lond B Biol Sci 356(1415):1801-1810, 2001.
52. Harding CF, et al: Hormonal specificity and activation of social behavior in male red-winged blackbirds. Horm Behav 22:402-418, 1988.
53. Harvey S, Etches RJ (eds): Perspectives in Avian Endocrinology. Toronto, Society for Endocrinology, 1997.
54. Hassinen E, Pyörnilä A, Hissa R: Vasotocin and angiotensin II affect thermoregulation in the pigeon, *Columbia livia*. Comp Biochem Physiol A 107:545-551, 1994.
55. Hazelwood RL: Pancreas. *In* Whittow GC (ed): Sturkie's Avian Physiology 5th Edition. New York, Academic Press, 2000, pp 539-555.
56. Herold M, et al: Investigation of ACTH responses of chickens with autoimmune disease. Gen Comp Endocrinol 88:188-198, 1992.
57. Heryanto B, et al: Involvement of apoptosis and lysosomal hydrolase activity in the oviductal regression during induced molting in chickens: A cytochemical study for end labeling of fragmented DNA and acid phosphatase. Poult Sci 76:67-72, 1997.
58. Hissa R, Rautenberg W: Thermoregulatory effects of intrahypothalamic injections of neurotransmitters and their inhibitors in the pigeon. Comp Biochem Physiol 51:319-326, 1975.
59. Hokin LR, Hokin MR: Enzyme secretion and the incorporation of ^{32}P into phospholipids of pancreas slices. J Biol Chem 203:967-977, 1953.
60. Holberton RL, Able KP: Differential migration and an endocrine response to stress in wintering dark-eyed juncos (*Junco hyemalis*). Proc Royal Soc Lond 267:1889-1896, 2000.
61. Hudelson KS: A review of the mechanisms of avian reproduction and their clinical applications. Seminars in Avian and Exotic Pet Medicine 5(4):189-198, 1996.
62. Hudelson KS, Hudelson P: A brief review of the female avian reproductive cycle with special emphasis on the role of prostaglandins and clinical applications. J Avian Med Surg 10(2):67-74, 1996.
63. Heather G: The contribution of extrapineal sites of melatonin synthesis to circulating melatonin levels in higher vertebrates. Experientia 49:665-670, 1993.
64. Hulan HW, Bird FH: Effect of fat level in isonitrogenous diets on composition of avian pancreatic juice. J Nutr 102:459-468, 1972.
65. Isobe T, Lillehoj HS: Effects of corticosteroids on lymphocyte subpopulations and lymphokine secretion in chickens. Avian Diseases 36:590-596, 1992.
66. Ivanics E, et al: Massive goitre (struma parenchymatosa) in geese. Acta Veterinaria Hungarica 47(2):217-231, 1999.
67. Jenni L, et al: Regulation of protein breakdown and adrenocortical response to stress in birds during migratory flight. Amer J Physiol Regulatory Integrative Comp Physiol 278:R1182-R1189, 2000.
68. Jones RB: Fear and adaptability in poultry: Insights, implications and imperatives. World's Poult Sci J 52:131-174, 1996.
69. Jones RB, et al: Vitamin C supplementation and fear-reduction in Japanese quail: Short-term cumulative effects. Br Poult Sci 37:33-42, 1996.
70. Joseph J, Dandekar DS, Ramachandran AV: Dexamethasone-induced alterations in glucose tolerance and, insulin, glucagon and adrenaline responses during the first month in White Leghorn chicks. Br Poult Sci 37(3):665-676, 1996.
71. Kahler J: Sandostatin (synthetic somatostatin) treatment for diabetes mellitus in a sulfur-breasted toucan (*Ramphastus sulfuratus*). Proc Annu Conf Assoc Avian Vet, 1994, pp 296-273.
72. Kai H, et al: Influence of corticosterone on tracheal mucociliary transport in pigeons. Jpn J Pharmacol 52:496-499, 1990.
73. King DB, Entrikin RK: Thyroidal involvement in the expression of avian muscular dystrophy. Life Sciences 48:909-916, 1991.
74. Kokue E, Hayama T: Effects of starvation and feeding in the endocrine pancreas of chicken. Poult Sci 51:1366-1370, 1972.
75. Kühn ER, et al: Growth hormone and thyrotropin releasing hormone stimulate the peripheral conversion of thyroxine into triiodothyronine and the liver 5'-monodeiodinase activity in the adult chicken. IRCS Med Sci 14:479, 1986.
76. Lumeij JT: Relation of plasma calcium to total protein and albumin in African grey (*Psittacus erithacus*) and Amazon (*Amazona* spp) parrots. Avian Pathol 19:661-667, 1990.
77. Lumeij JT, Remple JD, Riddle KE: Relationship of plasma, total protein and albumin to total calcium in peregrine falcons (*Falco peregrinus*). Avian Pathol 22:183-188, 1993.
78. Mahata SK, Gnosh A: Role of splanchnic nerve on steroid-hormone-induced alteration of adrenomedullary catecholamines in untreated and reserpinized pigeon. J Comp Physiol B 161:598-601, 1991.
79. Maney DL, Wingfield JC: Central opioid control of feeding behavior in the white-crowned sparrow Zonotrichia leucophrys gambelii. Horm Behav 33:16-22, 1998.
80. Marler P, et al: The role of sex steroids in the acquisition and production of birdsong. Nature 336:770-772, 1988.
81. Mashaly MM, Trout JM, Hendricks GL III: The endocrine function of the immune cells in the initiation of humoral immunity. Poult Sci 72:1289-1293, 1993.
82. Mashaly MM, et al: The role of neuroendocrine immune interactions in the initiation of humoral immunity in chickens. Domest Anim Endocrinol 15(5):409-422, 1998.
83. McMurtry JP, et al: Metabolic responses of the turkey hen (*Meleagris gallopavo*) to an intravenous injection of chicken or porcine glucagon. Comp Biochem Physiol 114C(2):159-163, 1996.
84. McNabb FMA: Thyroids. *In* Whittow GC (ed): Sturkie's Avian Physiology, 5th edition. New York, Academic Press, 2000, pp 461-471.
85. Mersmann HJ: Overview of the effects of α-adrenergic receptor agonists on animal growth including mechanisms of action. J Anim

Sci 76:160-172, 1998.
86. Murai A, et al: Factors regulating amylase secretion from chicken pancreatic acini in vitro. Life Sci 66(7):585-591, 2000.
87. Nisticò G, Marley E: Central effects of prostaglandins E_2, A_1, and F_{2a} in adult fowls. Neuropharmacology 15:737-741, 1976.
88. Nottebohm F: Brain pathways for vocal learning in birds: A review of the first 10 years. *In* Sprague JM, Epstein AN (eds): Progress in Psychobiology and Physiological Psychology, Vol 9. New York, Academic Press, 1980, pp 85-214.
89. Oglesbee B: Hypothyroidism in a scarlet Macaw. JAVMA 201(10):1599-1601, 1992.
90. Oglesbee BL, Orosz S, Dorrestein GM: The Endocrine System. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, PA, WB Saunders Co, 1997, pp 475-488.
91. Okano T, Fukada Y: Photoreception and circadian clock system of the chicken pineal gland. Microsc Res Tech 53(1):72-80, 2001.
92. Okano T, et al: Molecular cloning heterotrimeric G-protein alpha subunits in chicken pineal gland. 44(suppl 1):S91-S92, 2001.
93. Puvadolpirod S, Thaxton JP: Model of physiological stress in chickens 1. Response parameters. Poult Sci 79:363-369, 2000.
94. Puvadolpirod S, Thaxton JP: Model of physiological stress in chickens 4. Digestion and metabolism. Poult Sci 79:383-390, 2000.
95. Rees A, Harvey S: Adrenocortical responses of pigeons (*Columba livia*) to treadwheel exercise. Gen Comp Endocrinol 65:117-120, 1987.
96. Rees A, Harvey S, Phillips JG: Adrenergic stimulation of adrenocortical secretion in immature fowl. Comp Biochem Physiol C 81:387-389, 1985.
97. Reinert BD, Wilson FE: The thyroid and the hypothalamus-pituitary-ovarian axis in American tree sparrows (*Spizella arborea*). Gen Comp Endocrinol 103(1):60-70, 1996.
98. Ritchey JW, Degernes LA, Brown TT Jr: Exocrine pancreatic insufficiency in a yellow-naped Amazon (*Amazona ochrocephala*) with pancreatic adenocarcinoma. Vet Pathol 34(1):55-7, 1997.
99. Ryan S, Arnold AP: Evidence for cholinergic participation in the control of bird song: acetylcholinesterase distribution and muscarinic receptor autoradiography in the zebra finch brain. J Comp Neurol 202:211-219, 1981.
100. Sahba MM, Morisset JA, Webster PD: Synthetic and secretory effects of cholecystokinin-pancreozymin on the pigeon pancreas. Proc Soc Exp Biol Med 134:728-732, 1970.
101. Saldanha CJ, Schlinger BA, Clayton NS: Rapid effects of corticosterone on cache recovery in mountain chickadees (*Parus gambelia*). Horm Behav 37:109-115, 2000.
102. Sat T: Sensory and endocrine characteristics of the avian pineal organ. Microsc Res Tech 53(1):2-11, 2001.
103. Savory CJ, Mann J'S: Is there a role for corticosterone in expression of abnormal behavior in restricted-fed fowls? Physiol Behav 62:7-13, 1997.
104. Schmidt RE, Ravel DR: Thyroid hyperplasia in birds. J Avian Med Surg 16(2):111-114, 2002.
105. Sharp PJ (ed): Avian Endocrinology. Bristol, UK, J Endocrinol, 1993.
106. Sharp PJ, Beuving G: The role of corticosterone in the ovulatory cycle of the hen. J Endocrinol 78:195-200, 1978.
107. Simon J, Derouet M, Gespach C: An anti-insulin serum, but not a glucagon antagonist, alters glycemia in fed chickens. Horm Metab Res 32(4):139-141, 2000.
108. Smith GT, et al: Seasonal changes in testosterone, neural attributes of song control nuclei, and song structure in wild songbirds. J Neurosis 17:6001-6010, 1997.
109. Smith TR: Seasonal plasticity in the song nuclei of wild rufous-side towhees. Brain Res 734:79-85, 1996.
110. Soma K, Tramontane AD, Wingfield JC: Oestrogen regulates male aggression in the non-breeding season. Proc R Soc Lond Biol 267:1089-1096, 2000.
111. Thaxton JP, Puvadolpirod S: Model of physiological stress in chickens 5. Quantitative evaluation. Poult Sci 79:391-395, 2000.
112. Totzke U, Hubinger A, Bairlein F: A Role for pancreatic hormones in the regulation of autumnal fat deposition of the garden warbler (*Sylvia borin*) Gen Comp Endocrinol 107:166-171, 1997.
113. Totzke U, Hubinger A, Bairlein F: Glucose utilization rate and pancreatic hormone response to oral glucose loads are influenced by the migratory condition and fasting in the garden warbler (*Sylvia borin*). J Endocrinol 158(2):191-196, 1998.
114. Totzke U, et al: Fasting increases the plasma glucagon response in the migratory garden warbler (*Sylvia borin*). Gen Comp Endocrinol 115:116-121, 1999.
115. Tramontin AD, Brenowitz EA: Seasonal plasticity in the adult brain. Trends Neurosci 23:251-258, 2000.
116. Tramontin AD, et al: Seasonal growth of song control nuclei precedes seasonal reproductive development in wild adult song sparrows. Gen Comp Endocrinol 122:1-9, 2001.
117. Underwood H, Steele CT, Zivkovic B: Circadian organization and the role of the pineal in birds. Microsc Res Tech 53(1):48-62, 2001.
118. Vaillant C, Dimaline R, Dockray GJ: The distribution and cellular origin of vasoactive intestinal polypeptide in the avian gastrointestinal tract and pancreas. Cell Tissue Res 211:511-523, 1980.
119. Vasilatos-Younken R, et al: New insights into the mechanism and actions of growth hormone (GH) in poultry. Domest Anim Endocrinol 17:181-190, 1999.
120. Verstappen FALM, Lumeij JT, Bronneberg RGG: Plasma chemistry reference values in ostriches. J Wildl Dis 38(1):154-159, 2002.
121. Walters MJ, Collado D, Harding CF: Oestrogenic modulation of singing in male zebra finches: Differential effects on directed and undirected songs. Anim Behav 42:445-452, 1991.
122. Wilson, FE: A test of the hypothesis that T_3 is the "seasonality" thyroid hormone in American tree sparrows (*Spizella arborea*): intracerebroventricular infusion of iopanoic acid, an inhibitor of T_3 synthesis and degradation. J Comp Physiol [B] 171(2):113-119, 2001.
123. Wilson FE, Reinert BD: The timing of thyroid-dependent programming in seasonally breeding male American tree sparrows (*Spizella arborea*). Gen Comp Endocrinol 103(1):82-92, 1996.
124. Wilson FE, Reinert BD: Long days and thyroxine program American tree sparrows for seasonality: evidence for temporal flexibility of the breeding season of euthyroid females. Gen Comp Endocrinol 113(1):136-145, 1999.
125. Wingfield JC: Regulation of territorial behavior in the sedentary song sparrow, *Melospiza melodia morphina*. Horm Behav 47:77-89, 1994.
126. Wingfield JC, Silverin B: Effects of corticosterone on territorial behavior of free-living male song sparrows *Melospiza melodia*. Horm Behav 20:405-417, 1986.
127. Zachariasen RD, Newcomer WS: Phenylethanolamine-N-methyl transferase activity in the avian adrenal following immobilization or adrenocorticotropin. Gen Comp Endocrinol 23:193-198, 1974.

CHAPTER
20

Overview of Tumors

Section I: Clinical Avian Neoplasia and Oncology

Section II: A Retrospective Study of Case Submissions to a Specialty Diagnostic Service

Overview of Tumors: Section I

Clinical Avian Neoplasia and Oncology

TERESA L. LIGHTFOOT, DVM, Dipl ABVP-Avian

Avian neoplasias encountered in practice include cancer of the skin, oral cavity, sinuses, liver, kidney, reproductive organs, bones, brain, vascular structures and connective tissue. External tumors may be detected by physical examination and can often be diagnosed by needle aspiration, wedge, punch or surgical biopsy. Internal neoplasias often require radiographs, ultrasound, endoscopy, and biopsy or exploratory surgery to identify, diagnose and determine the extent of the neoplastic processes.

Treatment of neoplastic processes in birds is poorly documented. Most reports of treatment protocols are either anecdotal or involve a single patient. Many reports are not published, but are to be found in avian veterinary discussion groups on the Internet.[35,36,37]

The presentation in this text of these anecdotal treatments is problematic. Failure to include preliminary information regarding efficacy and/or clinical response may reduce the practitioner's willingness and ability to recommend treatment. However, future studies may either reinforce these experimental protocols or they may demonstrate a lack of efficacy or serious side effects of these regimes. The ultimate decision will lie with the knowledgeable practitioner and the well-informed client.

To date, the treatment of avian neoplasia has mirrored treatment in other domestic species. Generally, solid tumors are best treated with surgical excision, while systemic neoplastic processes (ie, systemic lymphoma, metastatic conditions) are most effectively managed with use of systemic chemotherapy. Cases in which surgical excision is incomplete or impossible may benefit from alternative forms of local therapy, including external beam radiation (Cobalt 60 or linear accelerator), cryotherapy, photodynamic therapy or hand-held radiation applicators.

CUTANEOUS MASSES

These may be pseudo-neoplastic conditions such as xanthomas and lipomas, or neoplastic lesions.

Xanthomas

These are generally friable, yellow-colored, fatty-appearing masses that may be located anywhere on the body, but are often seen on the distal wing, in the sternopubic area and on the keel (see Chapter 13, Integument). The origin of xanthomas is unknown, however, dietary improvement, including sufficient vitamin A or vitamin A precursors, has been noted to be curative in less advanced cases. Xanthomas tend to be very vascular and surgical excision, when necessary, should be undertaken with due attention to hemostasis. Diffuse xanthomas may be amenable to cryotherapy, but attention must be paid to maintenance of the vascular supply.[35]

Lipomas

These occur most frequently in budgerigars and are usually located on the keel or in the sterno pubic area. Most early lipomas respond to modified diet therapy. Lipomas that cause clinical signs can be addressed via surgical excision. Malignant liposarcomas are rare in psittacines.[33]

Mucoepidermoid Carcinomas

Mucoepidermoid carcinomas are rarely reported in birds. In humans, these tumors demonstrate variable degrees of malignancy and surgical resection is often curative. Comparable grading of this type of neoplasia has not been established in the avian patient (Figs 20.1.1, 20.1.2).

Fibrosarcomas

These can occur anywhere on the body, but are most commonly seen in the oral cavity, associated with long bones, or in the abdominal cavity (Figs 20.1.3, 20.1.4).

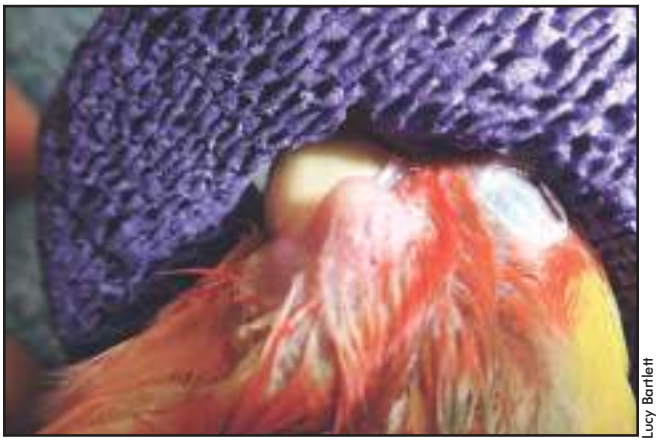

Lucy Bartlett

Fig 20.1.1 | Mucoepidermoid carcinoma.

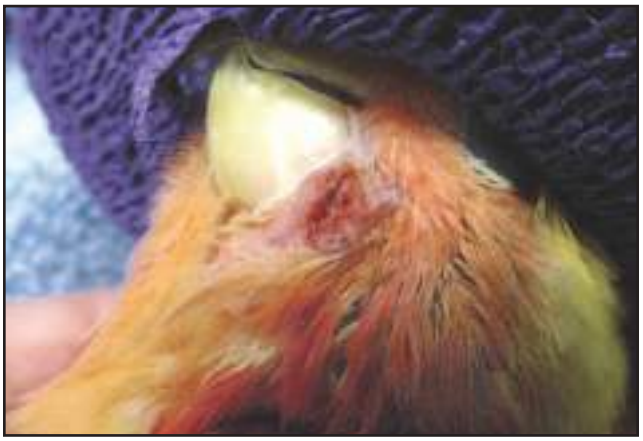

Lucy Bartlett

Fig 20.1.2 | Mucoepidermoid carcinoma after resection.

Fig 20.1.3 | Fibrosarcoma on the face of a budgerigar.

Fig 20.1.4 | Fibrosarcoma on the wing of a lovebird.

Fig 20.1.5 | Squamous cell carcinoma of the rhamphotheca, and papillomatosis in an older Timneh grey parrot.

Fibrosarcomas may be subcutaneous or more deeply located in underlying tissue, and often appear fixed and proliferative with a nodular, red surface. They tend to be locally invasive and often recur with conservative surgical excision. Therefore, additional local treatment in the form of radiation therapy is often indicated for providing long-term local control. As the metastatic rate in other domestic species ranges from 5 to 15%, local disease management is paramount, with metastatic control as a secondary concern. Surgical excision followed by both radiation and chemotherapy has been reported with some success in a few publications.[14] Strontium radiation therapy, although limited by depth of penetration, has been anecdotally reported as efficacious in several instances.[35]

Squamous Cell Carcinomas

These also may occur anywhere on the body, being most prevalent at mucocutaneous junctions of the head (Fig 20.1.5), on the distal wing and on the phalanges. The uropygial (preen) gland also may develop either adenocarcinoma or squamous cell carcinoma. (Note that *Amazona* spp. do not possess a preen gland). Squamous cell carcinomas tend to be extremely locally invasive and complete excision is rarely accomplished. Radiation therapy has been attempted with some success, however, squamous cell carcinoma appears to be an exceptionally radioresistant tumor and long-term control is rare. Anecdotal reports indicate that radioresistance may be even greater in birds than in mammals.[19,35] Strontium therapy when tumor depth is not a limiting factor has shown some promise in selected psittacine cases.[35] Distant metastasis is rare, therefore chemotherapy is not commonly utilized. Photodynamic therapy (PDT) has been attempted in two reported cases. One case of a squamous cell carcinoma in the beak of a hornbill showed a positive result in decreasing tumor size, but failure to eliminate the neoplasia.[31] The second case demonstrated a positive response to PDT after each treatment, but treatments were not able to be administered at regular intervals.[28]

NEOPLASIA OCCURRING IN THE MUSCULOSKELETAL SYSTEM

Theoretically these include the benign lesions such as chondroma and hemangioma, and malignant tumors including osteosarcoma, chondrosarcoma and leiomyosarcoma. Wide surgical resection or amputation are

Fig 20.1.6 | Chondroma on the leg of a budgerigar.

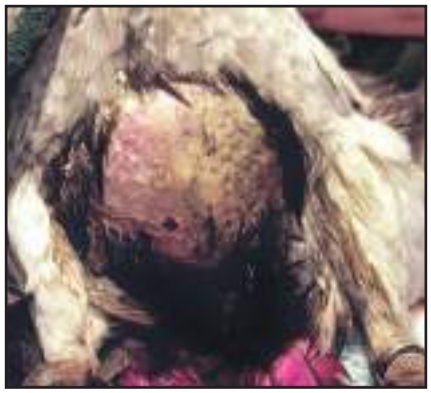


Fig 20.1.7 | Gross appearance of the abdomen in a 9-month-old African grey parrot with diffuse coelomic hemangioma.

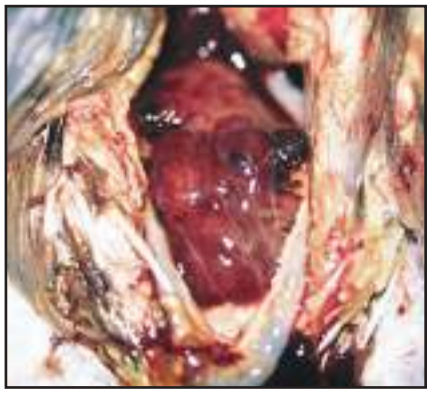


Fig 20.1.8 | The bird in Fig 20.1.11, showing hemangioma encompassing most abdominal viscera.

generally the suggested methods of treatment, as benign lesions are often cured with complete excision and a decrease in tumor burden can be accomplished in malignant lesions. As tumors such as osteosarcoma carry high metastatic rates, additional therapies may be indicated. Extrapolation from canine and feline oncology may suggest other modalities such as radiation therapy for additional local treatment and chemotherapy for systemic control.

Chondromas

Therapy for chondromas generally involves aggressive surgical excision of the affected area (Fig 20.1.6). Radiation and chemotherapy may be considered.

Osteosarcoma

Confirmation of osteosarcoma has rarely been reported in psittacines. Species and anatomic location predilections have not been noted in psittacines. Documentation of classifiable radiographic changes consistent with osteosarcoma is not available for birds.

A biopsy should be obtained from patients where radiographic bony lesions are present. Under inhalant anesthesia, a 22- to 20-gauge needle can be surgically introduced into the bone. A sufficient sample is usually obtained and subsequently retained in the hub of the needle. The sample can then be dislodged with smaller gauge wire and submitted. If a diagnosis of osteosarcoma is received, amputation with follow-up chemotherapy is the current recommended protocol extrapolated from canine medicine.

INTERNAL NEOPLASIA

Hemangiomas

These seem to occur more commonly than hemangiosarcomas in birds. Hemangioma may be internal or external and commonly appears as a red-purple, flat, firm lesion (see Chapter 13, Integument). Although histopathologically benign, in at least one case in this author's experience, hemangioma occurred in a juvenile African grey (*Psittacus erithacus*) and involved the coelomic cavity, small intestine, liver, lung, air sacs and pericardium. Complete surgical excision could not be accomplished and euthanasia was eventually required (Figs 20.1.7, 20.1.8). Treatment of a hemangiosarcoma with radiation therapy has been reported in one case.[9]

Internal Carcinomas

These are commonly reported in birds and include ovarian neoplasias (of various cell origins), renal carcinomas, hepatic adenocarcinoma, hepatobiliary and pancreatic adenocarcinoma (related to papillomas in Amazons), splenic and gastric carcinomas. Papillary carcinomas of air sac origin are locally invasive and may present as external masses. Anecdotal reports exist indicating intralesional carboplatin therapy may be useful in ovarian and renal adenocarcinoma, generally following surgical debulking and confirmation of the neoplasia on histopathology.[18,34] Bile duct carcinoma also has been treated with carboplatin successfully in one report.[38] Toxicity studies with cisplatin in cockatoos indicate that psittacine tolerance for this drug may be greater than that of mammals.[8]

Tamoxifen administration has not been evaluated for efficacy in cases of avian ovarian carcinoma, but anti-estrogenic activity was suggested and side effects were minimal in one drug trial.[17] GnRH agonists[a] have been effective empirically (dosed at 200-800 μg/kg), however, confirmation of neoplasia (as opposed to cystic ovarian disease) has not often been confirmed prior to therapy.[16,20]

Doxorubicin (adriamycin) is commonly employed in the treatment of carcinomas in human and canine patients. The limiting toxic effects of doxorubicin include myelosuppression and cardiac toxicity. To date, the degree to

which these concerns will apply to avian cancer patients has not been determined. Anecdotal reports of both toxicity and efficacy of doxorubicin in avian patients are currently inconclusive.[34] Dosages of 50-60 mg/m^2 have been used with no adverse reactions. In several cases there has been significant tumor regression (Goldsmith, Lightfoot, unpublished data, 2004).

Carcinomas, generally diagnosed at necropsy, are often found at the proventricular-ventricular junction. Death from this neoplasia may be due to hemorrhage, perforation and sepsis or endotoxic shock, or inanition and subsequent wasting. Metastasis to the lungs has been confirmed in one case report.[4]

Biliary and pancreatic carcinomas are frequently diagnosed in the genus *Amazona* and to a lesser degree, *Ara*, in conjunction with internal papillomatosis.[11,13] A recent connection to a herpesvirus has been identified (see Chapter 32, Implications of Viruses in Clinical Disorders). Carboplatin has been used in several cases with equivocal results, but with no apparent toxicity.[7,35,38]

Surgical excision is the treatment of choice with solitary lesions of hepatic cell carcinoma in other species, and is the only documented curative treatment in human medicine. Combinations of chemotherapy and radiation therapy have been used with equivocal results in people in an attempt to prevent or limit metastatic disease. In widely disseminated hepatic carcinoma, palliative chemotherapy is often employed. However, extrapolation from people would indicate that this type of cancer is highly resistant to chemotherapy. The most commonly employed chemotherapeutic agents in human medicine appear to be doxorubicin and 5-fluorouracil (5-FU), however, mean survival times do not appear to be statistically improved in patients with widely disseminated disease. The use of immunotherapy — including interferon, in conjunction with cisplatin, doxorubicin and 5-FU — has shown the most promise to date in human patients. Unfortunately, interferon is limited in its usefulness by cost and availability in veterinary medicine. The efficiency of radiation therapy for carcinomas and other neoplasias is largely unknown. However, tolerance of radiation therapy has been anecdotally reported as greater than anticipated.

Endocrine Neoplasia

Neoplasia of endocrine origin is not frequently reported in birds.

Pituitary Adenomas

These have been documented in multiple avian species, but are most prevalent in budgerigars and cockatiels. Affected animals may present with acute neurologic conditions (seizures/opisthotonos). They also may present with conditions related to the pituitary hormone(s) that are affected. Usually, this will be pronounced polydypsia and polyuria. Occasional presentations will be that of a retrobulbar mass and subsequent exophthalmia.[27] In human medicine, surgical resection and radiation therapy (if needed) are utilized for treatment. Size and monetary constraints make routine treatment by these methods unlikely in our small psittacine patients.

Lucy Bartlett

Fig 20.1.9 | Thymoma in a lovebird, intraoperatively.

Thyroid

Budgerigars that are iodine deficient may develop non-neoplastic thyroid hyperplasia that presents as a thyroid mass, often causing a change in the voice or a respiratory squeak.

Thyroid Tumors

These are not as common in birds as they are in domestic rabbits, but do occur (Fig 20.1.9). They may be intrathoracic or located in the area of the neck. In humans, classification according to cell type (medullary, cortical and mixed) is a prognostic indicator, with cortical tumors having the highest incidence of recurrence and malignancy. Thymoma and thyroid adenocarcinoma have been reported in several psittacine species. Surgical excision is the primary treatment recommendation. Adjuvant radiation and chemotherapy protocols are being utilized in human medicine. Cisplatin is used in many human chemotherapy protocols for thymomas and thymic carcinomas. Limited studies have shown that psittacines may be tolerant of the common side effects induced by cisplatin, and this agent may be useful in the treatment of these neoplasias.

Pancreatic Neoplasias

Infrequent accounts of primary pancreatic neoplasia of variable cell origin, not associated with internal papillomatosis, have been reported.[23]


Lori Harrison

Fig 20.1.10 | Retrobulbar lymphoma in a young African grey.

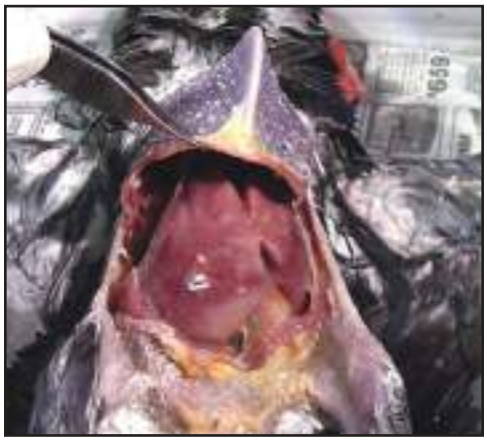

Lori Harrison

Fig 20.1.11 | Gross necropsy photo of the liver from the African grey in Fig 20.1.10. A fine-needle aspirate of the liver demonstrated that the lymphoma had spread to involve the hepatic parenchyma.

Respiratory Neoplasia

Primary respiratory neoplasia is uncommon in psittacines.[12] An exception seems to be an intrathoracic neoplasia reported in cockatiels. It is characterized by the inclusion of two cell types, having both mesenchymal and epithelial cell components (see Section II of this chapter). Few other primary pulmonary neoplasias have been reported in the literature.[2] Metastatic pulmonary neoplasia may occur, but it is not noted with the same frequency as is documented in dogs.[4]

Lymphoma/Lymphosarcoma

Numerous reports of exophthalmos in psittacines, particularly young African greys, have been diagnosed as retrobulbar lymphoma (Fig 20.1.10). Differential diagnoses for this condition are pituitary adenoma and hyperplasia or adenoma of the Harderian gland. Lymphoma may have many presentations in pet birds, much as it does in other companion animals (Fig 20.1.11). Chemotherapy is the treatment of choice for systemic disease, and surgery and radiation therapies have been successfully employed in cases of solitary lymphoma.[6,35] To date, no evidence of retroviral activity has been associated with psittacine lymphoma.

The clinician may find it useful to have access to current protocols for lymphoma that are utilized in canine medicine. Tracy LaDue, Diplomate ACVIM-Oncology and ACVR-Radiation Oncology, of Florida Veterinary Specialists in Tampa, Florida, US, has generously provided the following abbreviated outline of therapeutic options and chemotherapeutic agents. These protocols are NOT established for avian patients, but are provided to give the practitioner a point of reference when attempting to design potential therapeutic regimes for birds with lymphoma. Again, species differences in response may well exist. Some of the chemotherapeutic agents listed may be determined to be either ineffectual or contraindicated in birds. As documentation of these variables occurs, it is hoped that protocols can be developed that will produce more predictable results in the avian patient.

Chemotherapy Protocols for Canine Lymphoma*

(Current recommended canine dosages can be found in Plumb's Veterinary Handbook).

1. Oral Therapies
   a. Prednisolone and cyclophosphamide
   b. Lomustine (CCNU)
2. Injectable Therapy
   a. COP-L Protocol
      i. L-asparaginase injection
      ii. Oral prednisolone (tapering dose)
      iii. Oral weekly cyclophosphamide
      iv. Vincristine injections weekly IV
   b. Single-agent adriamycin injections q 3 weeks IV
   c. UW-Madison Cyclic Combination
      i. L-asparaginase
      ii. Vincristine
      iii. Cyclophosphamide
      iv. Adriamycin
      v. Prednisolone

Applicable testing (CBC, biochemistries, cardiac evaluation) should be performed to assess the initial and intratherapeutic health of the patient.

*It must be emphasized again that these protocols are designed for *canine* patients. Extrapolation to avian patients must be undertaken with the knowledge that efficacy and potential side effects have not been documented.

CHEMOTHERAPEUTIC AGENTS

Anticancer agents are typically broken into six categories based on their mechanism of action.

1. Alkylating agents such as cyclophosphamide and lomustine prevent cell replication by covalently binding to the nucleotide bases of the DNA molecule.
2. Antimetabolites will mimic purine, pyrimidine or metabolite precursors of the nucleotide bases, resulting in non-functional DNA.
3. Steroids such as prednisone and prednisolone cause lympholysis and suppress neutrophil function and antibody production.
4. Plant alkaloids such as vincristine bind to microtubules to prevent normal formation and function of the mitotic spindle. The antibiotics such as adriamycin intercalate between DNA base pairs to disrupt transcription and also cause oxygen free radical damage.
5. Miscellaneous drugs such as the Platinol compounds (cisplatin and carboplatin) also bind to bases of the DNA preventing replication, but have a bifunctional ability with double attachment to DNA strands.
6. L-asparaginase hydrolyzes asparagine to aspartic acid and ammonia, resulting in loss of an essential amino acid for cell function.

Most anticancer agents have associated vomiting, diarrhea and bone marrow suppression as sequelae. It is important to monitor patients for signs of dehydration or secondary infection as a result of chemotherapy administration. Some anticancer agents have particular toxicities known to that drug alone, such as sterile hemorrhagic cystitis due to cyclophosphamide metabolites in dogs and people. Such toxicities are not well reported in avian species and should be monitored for accordingly.

When confronted with a confirmed diagnosis of neoplasia, a current literature search is warranted due to the rapid advances and changes in treatment recommendations. Consultation with a veterinary oncologist will increase the likelihood of selecting an appropriate treatment regime and properly administering the chosen therapy.

Products Mentioned in Text

a. Depo-Lupron, Pharmaceuticals, Inc, Deerfield, IL USA, 800-622-2011

References and Recommended Reading

1. Altman RA, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997.
2. Andre JP, Delverdier M: Primary bronchial carcinoma with oseous metastasis in an African grey parrot (*Psittacus eritbacus*). J Avian Med Surg 13(3):180-186, 1999.
3. Burgmann PM: Common psittacine dermatologic diseases. Sem Avian Exotic Pet Med 4(4):169-183, 1995.
4. Campbell TW: Carcinoma of the ventriculus with metastasis to the lungs in a sulphur-crested cockatoo (*Cacatua galerita*), J Avian Med Surg 13(4):265-268, 1999.
5. Clyde VL, Orosz SE, Munson L: Severe hepatic fibrosis and bile duct hyperplasia in four Amazon parrots. J Avian Med Surg 10(4):252-257, 1996.
6. Coleman CW: Lymphoid neoplasia in pet birds: A review. J Avian Med Surg 9(1):3-7, 1995.
7. Degernes LA: Multicystic biliary adenocarcinoma in a blue-and-gold macaw (*Ara ararauna*). J Avian Med Surg 12(2):100-107, 1998.
8. Filippich LJ: Intravenous cisplatin administration in sulphur-crested cockatoos (*Cacatua galerita*): Clinical and pathologic observations. J Avian Med Surg 15(1):23-30, 2001.
9. Freeman KP: Radiation therapy for hemangiosarcoma in a budgerigar. J Avian Med Surg 13(1):40-44, 1999.
10. Hahn KA, Jones MP, Petersen MG: Metastatic pheochromocytoma in a parakeet. Avian Dis 41(3):751-4, 1997.
11. Hillyer EV, et al: Bile duct carcinoma in two out of ten Amazon parrots with cloacal papillomas. J Assoc Avian Vet 5(2):91-95, 1991.
12. Jones MP, Orosz SE: Pulmonary carcinoma with metastases in a Moluccan cockatoo (*Cacatua moluccensis*). J Avian Med Surg 15(2):107-113, 2001.
13. Kennedy FA, Sattler-Augustin S: Oropharyngeal and cloacal papillomas in two macaws (*Ara* spp.) with neoplasia with hepatic metastasis. J Avian Med Surg 10(2):89-95, 1996.
14. Lamberski N, Theon AP: Concurrent irradiation and intratumoral chemotherapy with cisplatin for treatment of a fibrosarcoma in a blue and gold macaw (*Ara ararauna*). J Avian Med Surg 16(3):234-238, 2002.
15. Lennox AM: The Use of Aldara (Imiquimod) for the Treatment of Cloacal Papillomatosis in Psittacines. Exotic DVM 4(3):34-35 July 2002.
16. Lightfoot TL: Avian Common Clinical Presentations: Neoplastic, Toxic, Viral and Miscellaneous. Proc Atlantic Coast Vet Conf, 2001.
17. Lupu CA: Evaluation of Side Effects of Tamoxifen in Budgerigars, (*Melopsittacus undulatus*). J Avian Med Surg 14(4):237-242 Dec 2000.
18. MacWhirter P: Use of Carboplatin in the Treatment of Renal Adenocarcinoma in a Budgerigar. Exotic DVM 4(2):11-12 May 2002.
19. Manucy TK, Bennett RA, Greenacre C: Squamous cell carcinoma of the mandibular beak in a Buffon's macaw (*Ara ambigua*). J Avian Med Surg 12(3):158-166, 1998.
20. Morrissey JK: Gastrointestinal Diseases of Psittacine Birds. Sem Avian Exotic Pet Med 8(2):66-74, 1999.
21. Orosz, SE, Ensley PK, Haynes CJ: Avian Surgical Anatomy. Philadelphia, WB Saunders Co, 1992.
22. Ottinger MA: Neuroendocrine Regulation of Reproduction in Birds and Clinical Applications of GnRH Analogues in Birds and Mammals. Sem Avian Exotic Pet Med 11(2):71-79 April, 2002.
23. Rae M: Endocrine Disease in Pet Birds. Semin Avian Exotic Pet Med 4(1):32-38 Jan 1995.
24. Ramos-Vara JA: Lymphosarcoma with plasmacytoid differentiation in a scarlet macaw (*Ara macao*). Avian Dis 41(2):499-504 Apr-Jun 1997.
25. Ritchie B, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, Inc, 1994.
26. Ritchey JW, Degernes LA, Brown TT Jr: Exocrine pancreatic insufficiency in a yellow-naped Amazon (*Amazona ochrocephala*) with pancreatic adenocarcinoma. Vet Pathol 34(1):55-7 1997.
27. Romagnano A, Mashima TY: Pituitary Adenoma in an Amazon Parrot. J Avian Med Surg 9(4):263-270, 1995.
28. Rosenthal K, et al: A Report of Photodynamic Therapy for Squamous Cell Carcinoma in a Cockatiel. Proc Ann Conf AAV 2001, pp 175-176.
29. Schmidt RE: Selected Neoplasia of the Avian Integument. Exotic DVM Vol 4(2):13-14, 2002.
30. Sturkie PD, Whittow GC (eds): Sturkie's Avian Physiology 5th ed. San Diego, Academic Press, Harcourt Brace and Co, 2000.
31. Suedmeyer WK: Attempted Photodynamic Therapy of Squamous Cell Carcinoma in the Casque of a Great Hornbill (*Buceros bicornis*). J Avian Med Surg 15(1):44-49, 2001.
32. Tell LA, Woods L, Mathews KG: Basal cell carcinoma in a blue-fronted amazon parrot (*Amazona aestiva*), Avian Dis 41(3):755-9 Jul-Sep 1997.
33. Tully T: Liposarcomas In A Monk Parakeet (*Myiopsitta monachus*). J Assoc Avian Vet 8(3):120-124 Fall 1994.
34. Watson CL, Lucroy MD: Primary Appendicular Bone Tumors in Dogs. Compend Contin Edu Pract Vet 24(2):128-138 Feb 2002.
35. www.vin.com, Member Search - Species: Bird, Subject: Neoplasia
36. www.exoticdvm.com
37. www.avianmedicine.net
38. Zantop DA: Treatment of bile duct carcinoma in birds with carboplatin. Exotic DVM 2(3):76-78, 2000.

Overview of Tumors: Section II

A Retrospective Study of Case Submissions to a Specialty Diagnostic Service

MICHAEL M. GARNER, DVM, Dipl ACVP

The occurrence of various types of avian neoplasia has been comprehensively reviewed.[6] This section documents the prevalence of neoplasms in 22 avian orders submitted to a specialty diagnostic service (Northwest ZooPath, Monroe, WA) from 1994 to 2002. Cases were selected based on histologic diagnosis. Cysts, hyperplastic processes, fibromatous polyps and poxvirus-related proliferative lesions were not included. Cases diagnosed as neoplastic based on cytology alone also were excluded. Although potentially reversible and not considered true neoplasms, adenomatous polyps and papillomas were included because of the known association of these lesions with concurrent neoplasia in psittacine birds.[4] Type, location, biological behavior and patient outcome are addressed. Apparent trends for particular types of neoplasms in some orders or species also are identified and discussed. For the purposes of this manuscript, prevalence refers to a given percentage within the study population, and the study population comprises the cases submitted to the service. The prevalence of these neoplasms in the populations from which these birds originated is not known.

Table 20.2.1 lists the tumor submissions by site and biological behavior. Skin was the most common site for tumor development, followed by alimentary tract, reproductive tract and liver. In all locations except alimentary tract, malignant tumors were more common than benign tumors; the large numbers of cloacal and oral papillomas and adenomatous polyps in psittacine birds account for this variation in behavior.

Table 20.2.2 summarizes total numbers of submissions and total numbers of tumors for each order. For the study period, 9574 avian samples were submitted, representing 22 orders; 557 neoplastic processes were identified, for an overall prevalence of 5.8%. The overall prevalence of neoplasia over the 7-year period was highest in Anseriformes (ducks, geese, swans), Galliformes (poultry, pheasants), Strigiformes (owls) and Cuculiformes (cuckoos, turacos).

Tables 20.2.3 and 20.2.4 list the tumor submissions by type and biological behavior. The most common types of tumors were cutaneous squamous cell carcinoma, multicentric lymphoma, cutaneous soft tissue sarcoma, biliary adenocarcinoma and ovarian/oviduct adenocarcinoma.

Neoplasia by Avian Order

PSITTACIFORMES

Order Psittaciformes (parrots and related species) had 3545 representatives and 220 neoplastic processes (prevalence = 6.2%) (see Table 20.2.2), slightly higher than the average prevalence for tumor submissions from other orders. Table 20.2.5 summarizes the most common presentations of neoplasms within this order. Trend criteria were based on total number of tumor types in a species (two or more), and percent of total for all tumors in a species (10% or greater). Using these criteria, numerous trends were observed within this order. For cockatiels (*Nymphicus hollandicus*), trends were identified in soft tissue sarcoma, squamous cell carcinoma, ovarian/oviduct adenocarcinoma, fibrosarcoma and seminoma. For Amazon parrots (*Amazona* spp.), trends were identified for squamous cell carcinoma, cloacal adenomatous polyp, cloacal papilloma and biliary adenocarcinoma. For macaws (*Ara* spp.), trends were identified for cloacal adenomatous polyp, cloacal papilloma and biliary adenocarcinoma. For cockatoos (*Cacatua* spp.), trends were identified for soft tissue sarcomas and cloacal adenomatous polyps. For budgerigars (*Melopsittacus undulatus*),

Table 20.2.1 | Tumor Submissions by Site and Biological Behavior

Location	Total	Malignant	Benign
Skin	120	92	28
Alimentary	67	32	35
Reproductive	64	59	5
Liver	54	45	9
Kidney	28	17	11
Respiratory	20	20	0
Intracoelomic	17	16	1
Pancreas	13	12	1
Endocrine	13	6	7
Uropygial gland	8	7	1
Musculoskeletal	7	7	0
Thymus	5	4	1
Conjunctiva	5	2	3
CNS	3	3	0
Spleen	2	1	1
Heart	2	2	0

Table 20.2.2 | Total Submissions and Prevalence of Neoplasia by Order

Order	Cases	Tumors	%
Anseriformes	1024	119	11.6
Strigiformes	131	13	9.9
Galliformes	783	74	9.4
Cuculiformes	62	5	8.1
Psittaciformes	3545	220	6.2
Columbiformes	294	17	5.8
Sphenisciformes	204	11	5.4
Phoenicopteriformes	265	13	4.9
Coraciiformes	192	9	4.7
Unknown	44	2	4.5
Gruiformes	249	11	4.4
Falconiformes	272	10	3.7
Ciconiiformes	307	11	3.6
Struthioniformes	111	3	2.7
Charadriiformes	240	6	2.5
Coliiformes	51	1	2.0
Piciformes	198	4	2.0
Passeriformes	1441	27	1.8
Pelecaniformes	58	1	1.7
Apodiformes	19	0	0.0
Procellariiformes	6	0	0.0
Gaviiformes	63	0	0.0
Caprimulgiformes	15	0	0.0
Totals	9574	557	5.8

Table 20.2.3 | Epithelial, Gonadal and Bimorphic Neoplasms[a]: Total Numbers, Biological Behavior and Patient Outcome

Tumor Type	Tumor #	Invasive Behavior	Lymphatic invasion	Meta-stasis	Death Due to Tumor	Death Due to Other	Excised	Lost to follow up
Malignant neoplasms								
Squamous cell carcinoma	48	48	1	4	16	0	3	29
Biliary adenocarcinoma	29	29	0	5	28	0	0	1
Ovarian/oviduct adenocarcinoma	28	28	2	9	19	2	0	7
Renal adenocarcinoma	16	16	0	1	15	0	0	1
Seminoma	15	15	0	0	5	8	3	3
Pancreatic adenocarcinoma	13	13	0	7	13	0	0	0
Intracoelomic adenocarcinoma	12	12	0	5	12	0	0	0
Hepatocellular carcinoma	11	11	0	3	10	1	0	0
Proventricular adenocarcinoma	10	10	0	4	10	0	0	0
Air sac adenocarcinoma	9	9	1	4	8	0	0	1
Pulmonary adenocarcinoma	6	6	0	0	6	0	0	0
Ventricular adenocarcinoma	5	5	1	1	5	0	0	0
Cloacal adenocarcinoma	4	4	0	0	1	0	0	3
Sertoli cell tumor	4	4	1	1	1	3	0	0
Bimorphic pulmonary tumor	4	4	0	1	4	0	0	0
Thyroid adenocarcinoma	3	3	0	0	3	0	0	0
Interrenal cell carcinoma	2	2	0	1	2	0	0	0
Nephroblastoma	2	2	0	0	1	1	0	0
Benign neoplasms								
Papilloma	21	0	0	0	0	2[b]	1	18
Adenomatous polyp	17	0	0	0	0	5[c]	1	11
Renal adenoma	12	0	0	0	6	6	0	0
Hepatoma	4	0	0	0	2	1	0	1
Thyroid adenoma	4	0	0	0	1	3	0	0
Interrenal cell adenoma[d]	3	0	0	0	0	3	0	0
Biliary adenoma/ cystadenoma	3	0	0	0	0	4	0	1
Granulosa cell tumor	2	0	0	0	1	1	0	0
Folliculoma	2	0	0	0	0	0	1	1

a. For all tumors represented two or more times in the study. Tumors represented only once not included.
b. One psittacine cloacal papilloma was associated with concurrent fatal biliary adenocarcinoma, and one psittacine ingluvial papilloma underwent transformation to fatal squamous cell carcinoma.
c. Four psittacine cloacal adenomatous polyps transformed locally to adenocarcinomas. Two psittacines with cloacal adenomatous polyps had concurrent fatal biliary adenocarcinoma, and two psittacines with cloacal adenomatous polyps had concurrent fatal pancreatic adenocarcinoma.
d. The function of interrenal cells (cells of the avian adrenal gland) is analogous to cortical cells of the mammalian adrenal gland.

Table 20.2.4 | Mesenchymal Neoplasms[a]: Biological Behavior and Patient Outcome

Tumor Type	Tumor #	Invasive Behavior	Lymphatic Invasion	Metastasis	Death Due to Tumor	Death Due to Other	Excised	Lost to follow up
Malignant tumors								
Lymphoma	40	40	0	36[b]	36	0	0	4
Soft tissue sarcoma[c]	36	33	1	4	15	2	6	13
Fibrosarcoma	19	19	0	0	6	2	1	10
Hemangiosarcoma	11	11	0	3	9	0	1	1
Osteosarcoma	6	6	0	1	5	0	1	1
Myelolipoma	5	5	0	0[d]	5	0	0	0
Nerve sheath	4	4	0	0	0	0	1	3
Melanoma	4	4	0	2	3	1	0	0
Thymoma	4	4	0	0	1	0	0	3
Liposarcoma	2	2	0	0	0	0	0	2
Lymphoproliferative disease	2	2	0	1*	2	0	0	0
Benign tumors								
Lipoma	16	0	0	0	0	0	3	13
Hemangioma	8	0	0	0	1	1	2	4
Myelolipoma	2	0	0	0	0	1	1	0

*multicentric

a. For all tumors represented two or more times in the study. Tumors represented only once not included.
b. Lymphoma is generally a multicentric process. In 35 birds, the tumor was considered multicentric rather than metastatic, based on the presence of neoplastic cells in at least two different tissue types. In only one dead bird with full tissue evaluation was lymphoma detected in only one tissue (thymus). The remaining four cases were single tissue biopsies that were lost to follow-up, but also were likely multicentric tumors. Two cases had apparent concurrent lymphoid leukemia, based on histologic evaluation.
c. Tumors were classified as soft tissue sarcomas if they were undifferentiated or had too much anaplasia to determine the cell of origin. Likely differentials were fibrosarcoma, leiomyosarcoma, rhabdomyosarcoma, synovial sarcoma, neurofibrosarcoma, myxosarcoma and amelanotic melanoma.
d. Intracoelomic myelolipomas were invasive and infiltrated many visceral tissues. It was difficult to determine if some visceral foci were extensions of the invasive process or represented metastatic lesions.

Table 20.2.5 | Prevalence of Most Common Types of Neoplasms in Psittacine Birds

	Species and Total # of Tumors							
Tumor Type and % of Total Psittacine Cases (220)	Cockatiel (39)	Amazon (30)	Macaw (28)	Cockatoo (25)	Budgerigar (25)	Lovebird (19)	African Grey (8)	Rosella (3)
Soft tissue sarcoma - 23 (10.5%)	6 (15%)	0	2 (7%)	4 (16%)	4 (16%)	4 (21%)	1 (12.5%)	0
Squamous cell carcinoma - 22 (10%)	7 (18%)	3 (10%)	1 (4%)	0	5 (20%)	0	2 (25%)	2 (67%)
Cloacal adenomatous polyp - 15 (6.8%)	0	4 (13%)	4 (14%)	4 (16%)	0	0	1 (12.5%)	0
Ovarian/oviduct adenocarcinoma - 13 (5.9%)	7 (18%)	0	1 (4%)	2 (8%)	0	1 (5%)	0	0
Cloacal papilloma - 12 (5.5%)	0	4 (13%)	7 (25%)	0	0	0	1 (12.5%)	0
Fibrosarcoma - 11 (5%)	2 (5%)	0	0	2 (8%)	4 (16%)	2 (11%)	0	0
Biliary adenocarcinoma - 11 (5%)	0	4 (13%)	3 (11%)	0	0	1 (5%)	0	0
Lymphoma - 8 (3.6%)	0	2 (7%)	1 (4%)	0	1 (4%)	3 (16%)	1 (12.5%)	0
Seminoma - 7 (3.2%)	3 (7.6%)	0	1 (4%)	0	1 (4%)	0	0	0
Renal adenocarcinoma - 6 (2.7%)	0	0	1 (4%)	1 (4%)	4 (16%)	0	0	0

trends were identified for soft tissue sarcoma, squamous cell carcinoma, fibrosarcoma and renal adenocarcinoma. Interestingly, although lipomas are recognized as a common tumor in budgerigars,[6,12] a trend was not identified in this analysis of submissions. This may be because clinicians easily recognize these tumors, thus biopsies are not routinely submitted. For lovebirds (*Agapornis* spp.), trends were identified for soft tissue sarcoma, fibrosarcoma and lymphoma. For African greys (*Psittacus erithacus*) and rosellas (*Platycercus* spp.), trends were identified for squamous cell carcinoma.

Cloacal Adenomatous Polyps and Papillomas

Cloacal adenomatous polyps were common in Amazon parrots and macaws, and also were seen in cockatoos (*Cacatua* spp.), an African grey, a thick-billed parrot (*Rhynchopsitta pachyrhyncha*) and a Patagonian conure (*Cyanoliseus patagonus*). This distribution is similar to that of retrospective studies of this condition.[4,14] Malignant transformation of cloacal "papillomas" has been described in psittacine birds.[6,14] In this study, four cloacal adenomatous polyps (two macaws, one amazon parrot and one cockatoo) underwent local transformation to adenocarcinoma, although no metastases were seen. Adenomatous polyps also were noted in the proventriculus of a cockatoo and on the eyelid of a cockatiel. Two cloacal adenomatous polyps were associated with concurrent biliary adenocarcinoma and two with concurrent pancreatic adenocarcinoma. Cloacal papillomas also were common in macaws and Amazon parrots, and one

was seen in an African grey. Concurrent or isolated oral/choanal papillomas were occasionally seen. Only one of the cloacal papillomas was associated with concurrent biliary adenocarcinoma, and none were associated with malignant transformation in the cloaca.

Proliferative lesions of the cloacal mucosa of psittacine birds are well recognized.[4,6,14] Although typically referred to as papillomas, there are different morphologic presentations: a papilliform proliferation of squamous mucosal epithelial cells resembling a typical papilloma; and an adenomatous proliferation of glandular and villous mucosa more typical of a polyp, perhaps best termed an adenomatous polyp. Although the morphologic features of cloacal adenomatous polyps and papillomas differ, it is possible that these represent different morphologic variants of the same disease process. Both have similar site and species predilections, and both are sometimes associated with concurrent pancreatic and biliary malignancies. However, the distinction between cloacal adenomatous polyps and papillomas may not be purely academic. Based on this collection of the two cloacal phenotypes, the adenomatous polyps appear to have the most potential for undergoing malignant transformation in the cloaca, and for being associated with concurrent pancreatic or biliary neoplasia. Cloacal papillomas have been associated with herpesvirus gene sequences.[5,13]

Bimorphic Pulmonary Neoplasms of Cockatiels

Four separate cases of an unusual malignant pulmonary neoplasm were seen in cockatiels. The birds presented with a history of dyspnea and usually had a radiographically apparent mass in the thoracic region of the coelomic cavity. Microscopically, the tumor involved the lung and spread by extension to serosal surfaces of viscera, especially heart and air sacs. One case of possible metastasis to the endocardial surface also was seen. The tumor appeared to arise within the pulmonary parenchyma, around the parabronchi. These neoplasms have unusual cell morphology and are characterized by densely cellular sheets of round to elongate cells with large vesicular to amphophilic smudged nuclei. The cells stain positively for mammalian vimentin, a mesenchymal cell marker, and have cytoplasmic intermediate filaments consistent with vimentin, suggesting that the cells are of mesenchymal origin. The cells stain negatively for mammalian epithelial pan cytokeratins; however, desmosomes, a feature of epithelial cells, are occasionally seen between adjacent cells. The cell of origin is poorly understood, but the tumors appear to be "bimorphic" with mesenchymal and epithelial cell components. The nuclear characteristics of the neoplastic cells resemble the inclusions caused by polyomavirus, but no virus has been detected by electron microscopy, in situ hybridization or PCR (M.M. Garner, unpublished data).

GALLIFORMES

Order Galliformes (chickens, turkeys, pheasants, peafowl) had 783 representatives and 74 tumors (9.5%) (see Table 20.2.2), considerably higher than the average for tumor submissions from other orders. Table 20.2.6 summarizes the most common presentations of neoplasms within this order. Trend criteria were based on total number of a tumor type in a species (two or more), and percent of total for all tumors in a species (10% or greater). Using these criteria, numerous trends were observed within this order. For chickens, trends were identified for lymphoma, ovarian/oviduct adenocarcinoma and squamous cell carcinoma. These tumors are well recognized in poultry.[2,10,11,16] Some of these tumors, particularly the lymphoid malignancies, may have been associated with or induced by viral infection.[2,10,11,16] The only identifiable trend in pheasants was for interrenal cell adenoma, an otherwise uncommon tumor in birds. For guinea fowl, trends were identified for squamous cell carcinoma, seminoma and hepatocellular carcinoma. The only trend identified in peafowl was lymphoma. For turkeys, trends were identified for ovarian/oviduct adenocarcinoma and lymphoma. Herpesviruses and retroviruses have been identified as important causes of neoplasia in gallinaceous birds, particularly chickens and turkeys,[2,10,11,16] and it is possible that viral oncogenesis was a factor in many of the cases within this order; however, serologic and other ancillary testing for viral causes was not routinely performed on case submissions, so viral status of affected birds was generally not known.

ANSERIFORMES

Order Anseriformes (ducks, geese, swans) had 1024 representatives and 119 tumors (11.6%) (see Table 20.2.2), considerably higher than the average for tumor submissions from other orders. All the birds were adults or aged and most tumors likely occurred spontaneously. Trend criteria were based on total number of a tumor type in a species (two or more), and percent of total for all tumors in a species (10% or greater). Using these criteria, only two trends were identified: lymphoma and biliary adenocarcinoma in ducks, although a broad spectrum of neoplastic processes was represented in this group.

PASSERIFORMES

Order Passeriformes (songbirds) had 1441 representatives and only 27 tumors (1.9%) (see Table 20.2.2), considerably below the average for other orders, suggesting that spontaneous neoplasia in passerine birds is relatively uncommon. All birds were adults, a broad spectrum of neoplastic processes was represented and tumors likely occurred spontaneously in most cases. One possible trend was observed: two mynahs had

Table 20.2.6 | Prevalence of Most Common Types of Neoplasms in Galliform Birds

Tumor Type and % of Total Galliform Cases (74)	Total # of Tumors per Species				
	Chicken[a] (37)	Pheasant (5)	Guinea fowl (8)	Peafowl (5)	Turkey (10)
Lymphoma - 12 (16.2%)	6 (16%)	0	0	2 (40%)	4 (40%)
Squamous cell carcinoma - 9 (12.1%)	4 (11%)	0	3 (38%)	1	0
Ovarian/oviduct adenocarcinoma - 8 (10.8%)	6 (16%)	0	0	0	2 (20%)
Renal Adenocarcinoma - 4 (5.4%)	2 (5%)	1 (20%)	0	0	1 (10%)
Pancreatic adenocarcinoma - 4 (5.4%)	3 (8%)	0	0	0	1 (10%)
Seminoma - 4 (5.4%)	0	0	2 (25%)	0	1 (10%)
Interrenal cell adenoma - 3 (4.1%)	0	2 (40%)	0	1 (20%)	0
Hepatocellular carcinoma - 3 (4.1%)	1	0	2 (25%)	0	0

a. Includes chickens, roosters, jungle fowl

hepatic malignancies and concurrent hemochromatosis, suggesting iron storage in the liver may precipitate malignant transformation in this species, as alluded to by other authors.[8,9]

PHOENICOPTERIDAE

Suborder Phoenicopteridae (flamingos) had 265 representatives and 13 tumors (4.9%) (see Table 20.2.2), suggesting that the overall prevalence of neoplasia in the family/suborder is about average. Interestingly, liver tumors accounted for slightly less than half of the tumor submissions (see Table 20.2.1), suggesting that hepatic neoplasia may be over-represented in captive flamingos. These birds typically store large amounts of iron in the liver[7,15] and all the flamingos with hepatic neoplasia in this study had iron deposition; however, no overt changes were noted morphologically in relation to the iron, such as cirrhosis seen in mynahs, toucans or birds of paradise,[3,7,15] so the significance of the iron deposition relative to the neoplasia is undetermined. Two flamingos had squamous cell carcinomas on the pads of the feet, and had previous and ongoing protracted episodes of bumblefoot, which may have predisposed to neoplastic transformation.

STRIGIFORMES

Order Strigiformes (owls) had 131 representatives and 13 tumors (9.9%) (see Table 20.2.2), suggesting that overall prevalence of neoplasia in this order may be relatively high compared to other orders in the study. Six of the owls were burrowing owls (*Athene cunicularia*), suggesting that these birds may have a higher than average prevalence of neoplasia. Three hepatocellular neoplasms were noted in this order, all in burrowing owls. Myelolipoma, an unusual neoplasm in birds, appears to be over-represented in owls, occurring in three cases in the study. All were intracoelomic neoplasms that were extensively invasive and of undetermined origin. Interestingly, the two affected snowy owls (*Nyctea scandiaca*) were pen mates for most of their lives and died from these tumors within months of each other. All three of the owls with myelolipomas were from the same zoo.

SPHENISCIFORMES

Order Sphenisciformes (penguins) had 204 representatives and 11 tumors (5.4%) (see Table 20.2.2), about average compared to submissions from other avian orders. Over half of the tumor submissions (6, 55%) were squamous cell carcinomas, occurring in four different species of penguins. These data suggest that, in general, penguins may be predisposed to development of this form of neoplasm.

CICONIIFORMES

Order Ciconiiformes (herons, storks, ibises, spoonbills, New World vultures) (see Table 20.2.2) had 307 representatives and 11 tumors (3.6%), indicating the overall incidence of neoplasia in this order was slightly below the submitted average. Eight of the tumor submissions were in roseate spoonbills (*Ajaia ajaja*) and seven of the tumors in this species were focal or multicentric renal adenomas, a tumor that was otherwise uncommonly encountered in avian submissions. These data indicate that roseate spoonbills may be predisposed to developing this form of neoplasia. Although benign, four of these tumors contributed directly to the cause of death.

MISCELLANEOUS ORDERS

Several orders had no apparent trends in neoplastic disease. These include Columbiformes (pigeons, doves), Gruiformes (cranes, related species), Falconiformes (eagles, hawks, falcons, Old World vultures), Charadriiformes (shorebirds), Coraciiformes (kingfishers, mot mots, hornbills), Cuculiformes (turacos, cuckoos), Piciformes (woodpeckers, toucans, barbets), Struthioniformes (ratites), Coliiformes (mousebirds) and Pelecaniformes (pelicans, cormorants). Two birds of undetermined species also had neoplastic processes. Four orders, Gaviiformes (grebes, loons), Procellariiformes (fulmars), Caprimulgiformes (tawny frogmouths) and Apodiformes (hummingbirds) were represented in low numbers and had no neoplastic processes (see Table 20.2.2).

References and Suggested Reading

1. Biggs PM: Lymphoproliferative disease of turkeys. *In* Calnek BW (ed): diseases of Poultry. Ames, Iowa State Univ Press, 1997, pp 485-489.
2. Calnek BW, Witter RL: Marek's Disease. *In* Calnek BW (ed): Diseases of Poultry. Ames, Iowa State Univ Press, 1997, pp 369-413.
3. Gosselin SJ, Kramer LW: Pathophysiology of excessive iron storage in mynah birds. J Am Med Assoc 183(11):1238-1240, 1983.
4. Graham DL: Internal papillomatous disease: A pathologist's view of cloacal papillomas – and then some! Proc Assoc Avian Vet, 1991, pp 141-143.
5. Johne R, et al: Herpesviral, but no papovaviral sequences, are detected in cloacal papillomas of parrots. Arch Virol 147(10):1869-1880, 2002.
6. Latimer KS: Oncology. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 640-672.
7. Lowenstine LJ, Munson L: Iron overload in the animal kingdom. *In* Fowler ME, Miller RE (eds): Zoo and Wild Animal Medicine. Current Therapy 4. Philadelphia, WB Saunders Co, 1999, pp 260-268.
8. Macwhirter P: Passeriformes. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 1172-1199.
9. Panigrahy B, Senne DA: Diseases of mynahs. J Am Vet Med Assoc 199(3):378-381, 1991.
10. Payne LN, Fadly AM: Leukosis/sarcoma group. *In* Calnek BW (ed): Diseases of Poultry. Ames, Iowa State Univ Press, 1997, pp 414-466.
11. Reece RL, et al: Common necropsy findings in captive birds in Victoria, Australia (1978-1987). J Zoo Wildl Med 23(3):301-312, 1992.
12. Reece RL: Tumors of unknown etiology. *In* Calnek BW (ed): Diseases of Poultry. Ames, IA, Iowa State Univ Press, 1997, pp 485-489.
13. Styles DK, Phalen DN, Tomaszewski EK: Elucidating the etiology of avian mucosal papillomatosis in psittacine birds. Proc Assoc Avian Vet, 2002, pp 175-178.
14. Sundberg JP, et al: Cloacal papillomas in psittacines. Am J Vet Res 47(4):928-32, 1986.
15. Ward RJ, et al: Hepatic iron overload in birds: Analytical and morphological studies. Avian Pathol 17:451-464, 1988.
16. Witter RL: Reticuloendotheliosis. *In* Calnek BW (ed): Diseases of Poultry. Ames, Iowa State Univ Press, 1997, pp 467-484.

CHAPTER
21

Preventive Medicine
and Screening

DAVID N. PHALEN, DVM, PhD, Dipl ABVP-Avian

We are all children at heart. When we want something, we want it now and are willing to trust in fate if it means that we can get it now. Many bird owners are like this. They want a bird, so they buy it. They don't necessarily spend the time to research the source of the bird, and often there is little money left over to make sure that their purchase is healthy. Many people buy birds and know very little about them, trusting in the information provided by their friends, the Internet or the pet store. As a result, husbandry-related diseases and infectious disease still remain a critical problem for bird owners and veterinarians.

Preventing the Introduction of Transmissible Diseases

EDUCATION

Preventive medicine is an essential element of avian medicine, aviculture and pet bird ownership. Preventive medicine begins with education. New bird owners should be provided with a basic understanding of nutrition, safe and adequate caging, household hazards, hygiene and bird behavior. Improper nutrition, poor caging, improper sanitation and improper or inadequate socialization can all lead to disease — physical and psychological — that can shorten the bird's life or result in significant lessening of its quality. Similarly, bird owners and potential bird owners must be educated to the risk factors that lead to the introduction of an infectious disease or the acquisition of a bird that is already sick.

The process of educating bird owners and potential bird owners about bird health-related issues works best when it is started before the bird is purchased. It begins at the grass roots level. Aviculturists, pet store owners and their employees have to educate themselves, assure that their stock is healthy and provide accurate information to their clients. Selling a healthy bird and providing the information to keep it healthy will result in a satisfied client and repeat business. Veterinarians also must be proactive. This means educating themselves, other veterinarians, clients and potential clients alike. Speaking to local bird clubs, contributing to newsletters and bird magazines, and being active in local and national avian veterinary organizations are all part of preventive medicine.

ACQUIRING BIRDS

The risk of disease drops dramatically if the person buying a bird researches potential sources of birds prior to purchase. It is reasonable for the buyer to ask a source for references and to request to see the facilities where the bird was raised. Aviculturists who are members of national organizations that promote bird breeding and education of their membership, such as the American Federation of Aviculture, are more likely to have a good preventive medicine plan in place. In the USA, aviculturists can become certified as having the basic elements of a preventive medicine program through the Model Avicultural Program. This or similar certification is a good indication that the aviculturalist is making efforts to produce healthy birds. It also is a positive sign when aviculturists or pet stores have an established relationship with an avian veterinarian. A great contribution to the health of avicultural birds in the USA was the cessation of indiscriminate importation of wild-caught birds. In other countries where the practice of wild bird importation persists, contagious diseases still flourish.

COMBINING DIFFERENT SPECIES OF BIRDS

Risk of disease transmission is increased when birds that originate from different parts of the world are combined. Aviculturists are strongly encouraged to focus on one group of closely related birds rather than raising a wide variety of unrelated species. If many species of birds are to be raised, separating them into different facilities by genus or at least continent of origin may reduce disease problems.

COMPONENTS OF THE PHYSICAL FACILITY[30]

Any multiple bird facility, including pet stores, should have specifically designated areas including a quarantine area for newly acquired birds, a separate hospital area for sick birds, the main facility for the breeding birds or pets, a kitchen, and if birds are being hand-raised, a nursery. A clean-up area separate from the kitchen is preferred. Obviously, pet bird owners and small hobbyists may only occasionally need a designated quarantine area, hospital or nursery, whereas large breeding operations may need these facilities all the time. Likewise, the actual physical structure of each component of the aviary will vary enormously. In the home, a bathroom or extra bedroom may serve as a hospital or quarantine area. Large breeding facilities, on the other hand, might have separate buildings for some of these components.

Other elements of aviary design can facilitate or reduce the chances of disease transmission. Outdoor aviaries are practical only in the warmer parts of the country, but they have a number of important advantages. Rain and wind naturally dilute pathogens, and freezing and direct sunlight also can inactivate some pathogens (Chapter 37, Management of Racing Pigeons). On the other hand, birds in outdoor aviaries are more likely to be attacked by raccoons and be exposed to sarcocystis from opossums. Parasites and mosquito-borne diseases also are a risk with outdoor aviaries. The chances of disease transmission can be reduced in indoor collections by making sure there is adequate ventilation and maximal separation of cages. If stocking density is high, especially if cages are stacked on top of each other, then the chances of disease transmission increase dramatically. Pacheco's disease and proventricular dilatation disease (PDD) are examples of diseases that are more likely to occur in an indoor aviary. Aspergillosis is a disease that is more likely to occur in climates with high humidity or indoor collections with poor ventilation.

Movement between these areas should be from the cleanest place to the dirtiest. The kitchen is the cleanest area, followed by the nursery, main collection of birds, hospital and quarantine area. Keeping the traffic flow through the facility to the minimum reduces the risk of disease transfer. The traffic flow can be analyzed by drawing a floor plan of a facility and using a pen to trace movement through it. The more complicated the movement pattern, the more chance for disease spread. It is often the case that a clean area has to be entered several times a day. In large facilities with multiple workers, designating individuals to work in specific areas can solve this problem. When this is not possible, changing clothes or washing up well between areas should be considered. Restricting access to the birds by the public and other bird owners also reduces the risk of disease transmission.

QUARANTINE

Quarantine will be effective only if the basic rules of quarantine are followed. The most important rule of

quarantine is the "all in and all out" rule. One or more birds are brought into quarantine initially and no new birds are allowed into quarantine until the first birds have left. The location of the quarantine facility or area also is important. Just keeping a bird in a separate cage is not good enough. Some degree of distance between the new bird and the previously acquired birds is necessary. A separate building is ideal, but the garage, basement or bedroom also are acceptable.

The proper duration of quarantine is a shifting target that will vary to some degree with each circumstance. The longer the quarantine period, the more likely it is that a disease problem will be recognized before a bird is introduced into the flock. Thirty days is generally the minimum quarantine period recommended for birds, but 45 to 60 days is safer. Some aviculturists who are particularly concerned about PDD may quarantine birds for 6 to 12 months. For the quarantine to be meaningful, it has to apply to all birds entering the collection, even those birds that originated in that facility and are now returning.

Applying quarantine procedures to all birds leaving and returning to the aviary can be onerous, especially for those who show their birds. One solution to this problem is to select birds that will be shown that season and isolate them from the rest of the flock during the show season. At the end of the show season, they can be quarantined for a designated period of time and screened for disease, if necessary, before being reintroduced into the collection.

NEW BIRD EXAMINATIONS

Having every new bird or group of birds acquired examined and possibly screened for disease by an avian veterinarian is a critical element to a preventive medicine program. The extent of testing that might be done with a new bird will vary considerably according to the circumstances. If a bird is going into a home where it will be the only bird, then testing is done to show that the bird is healthy. If the bird is going into a multiple-bird household or aviary, testing is designed not only to make sure that the bird is healthy, but also to determine as best as is possible if it is infected with diseases that could be introduced to a collection.

Preventing Diseases by Common Environmental Pathogens

SANITATION

All environments contain organisms that can potentially cause disease. These opportunistic organisms, however, cause disease only when they are allowed to reach high concentrations or when other husbandry practices are less than ideal. *Candida albicans*, *Aspergillus* spp., *Pseudomonas* spp. and members of the Enterobacteriaceae are examples of ubiquitous opportunistic pathogens. Diseases caused by these organisms can be greatly minimized with proper hygiene. Whenever possible, food containers should be located so that they are not contaminated with feces. Likewise, water sources should be designed and located to minimize contamination from feces and food dunked in the water. Uneaten perishable foods should be removed from the cage before they have a chance to spoil. Water and food bowls should be cleaned regularly and water sources such as automatic drip systems should be regularly flushed and disinfected. Cages should be designed so they are easily cleaned and fecal and other organic material buildup does not occur.

There are many misconceptions about sanitation and its importance. Many bird owners obsess about it. When considering the degree of sanitation necessary for a pet home or aviary, it should be remembered that birds do not come from a sterile environment and a sterile environment is not the goal. Many bird owners also obsess about transmitting viruses among birds. The best way to keep viruses out is to follow the above guidelines for preventing the introduction of these diseases. If a collection is not infected with viral disease(s), there is no potential for viral transmission within that closed collection.

Much time has been spent discussing which disinfectant is best. This focus on disinfectants ignores the most important part of sanitation, basic cleaning. Organic material must be removed first before any disinfectant can be effective. It is in the organic material that the bacteria can grow, parasite eggs are protected and viruses are at their highest concentration. In aviaries where transmissible diseases are not a problem, cleaning is usually all that is necessary. Studies have shown that one of the best ways to sanitize food and water bowls is to run them through the dishwasher. Syringes and other hand-feeding tools also are readily cleaned and for all practical purposes disinfected in the dishwasher. The dishwasher should be separate from the home kitchen dishwasher. If disinfectants are to be used, they should be used after a surface is cleaned. They work best if they are left in contact with the surface for as long as possible. It is difficult or impossible to disinfect organic material, therefore, it is impossible to disinfect dirt floors and very difficult to disinfect wood surfaces. Disinfectants may be toxic, however, if viral diseases are or have been a problem in an aviary, disinfecting food and water containers and environmental surfaces is indicated. Phenolic

disinfectants and bleach are effective against most viruses and are the only disinfectants that work against viruses that do not have an envelope. Quaternary ammonium and chlorhexidine-based disinfectants are effective only against enveloped viruses.

Testing

Examination and testing of birds has two goals. The first is to make sure the newly acquired bird is healthy and does not have an infectious or non-infectious disease. The second is to make sure the bird is not subclinically infected with a disease that could be transmitted to other birds in the owner's aviary. There are two different types of testing. The first types are non-specific tests that provide general information that suggests a bird is healthy or ill, but do not specifically identify the etiology. The second types of tests are very specific and permit the identification of specific infectious agents. Because we cannot test for all infectious agents, it is common to use both types of tests when evaluating a new bird.

NON-SPECIFIC ASSAYS

The most important diagnostic assays in the broadest manner of speaking are the history, examination of the bird in the cage and the physical exam (see Chapter 6, Maximizing Information from the Physical Examination). However, it is the information derived from this phase of the workup that will lead to the development of a testing plan, and will be important in the interpretation of the results of any diagnostics that may be done.

Common non-specific diagnostic assays that are often included in the new bird exam include the complete blood cell count (CBC), chemistry panel, fecal wet mount and float, and oral and cloacal Gram stains and culture. Plasma electrophoresis and even radiographs are included as part of the new bird exam by some veterinarians. Interpretation of these diagnostic assays is discussed in detail in other chapters and only a few comments will be made about these here.

It has been the experience of the author that the CBC is a very useful tool for screening the new bird. It rarely gives a definitive diagnosis, but if abnormalities in the CBC are found it is a strong indication of an underlying health problem. The plasma electrophoresis also has considerable value, as alterations in this assay are found early in the course of infectious diseases, often before the disease becomes patent.[6] Fecal wet mounts can reveal *Giardia spp.* infections and are the best way to detect infections with *Macrorhabdus ornithogaster* (megabacterium).[17]

The Gram stain and fecal cultures are commonly used screening tools that have a tremendous potential for abuse. It is generally assumed that parrots should have relatively few bacteria in swabs of their oral cavity and cloaca, and that the predominate bacteria present on these surfaces should be gram-positive rods and cocci. The presence of large numbers of gram-negative bacterial rods, clostridial spores or budding yeasts is considered to be abnormal. It has been the tendency of avian veterinarians to treat birds that have predominately gram-negative flora of these areas. Likewise, when *Pseudomonas* spp. or members of the Enterobacteriaceae such as *E. coli* are cultured, it has been common practice to treat these birds.

Over time, however, it has become clear that this is not the correct approach. The gastrointestinal flora is influenced by many factors. If the bird is not outwardly ill, the presence of the so-called "pathogenic bacteria" is more often a reflection of poor nutrition or other management problems than anything else and does not mean that these organisms are causing this particular bird a problem. In these instances, improving nutrition, hygiene or other management practices will result in the Gram stain becoming normal again without treatment (see Chapter 4, Nutritional Considerations).

The use of aerobic culturing to screen birds also can lead to false impressions. Many of the normal flora found in the bird's gut are anaerobes. When cultures are performed of cloacal swabs and feces, they are generally sent in for aerobic culture. Under these circumstances, only the facultative anerobes and the aerobic bacteria grow, and the culture becomes skewed toward the smaller numbers of *Pseudomonas* spp. and Enterobacteriaceae that may be present.

The fecal Gram stain should be evaluated in light of the other findings in the individual bird. If the Gram stain is abnormal but the bird is otherwise healthy, management problems may be the underlying cause of the abnormal GI flora. Appropriate corrections to diet and husbandry can then be initiated. After these changes have been implemented for an adequate period (several weeks is commonly employed), a fecal Gram stain should be reexamined. If the bird has diarrhea or is showing other signs of illness and the Gram stain is abnormal, a culture may be indicated. Pending culture results, treatment with appropriate antibiotic, anti-yeast or antifungal therapy should be initiated.

Salmonellosis is a problem that is rarely seen in parrots but is common in other species of birds. Currently, many facilities require that birds be screened for infection with *Salmonella* spp. prior to entry. It is fairly common for otherwise normal birds to have positive fecal cultures. This poses a significant problem, as these birds

Table 21.1 | Diagnostic Assays for Psittacosis+

Assay	Sample	Effective in These Species of Birds	Day Positive after Infection	Specifics
Serology*				
Elementary body agglutination	Serum or plasma	Parrots	Approx. 14 days	Negative with successful treatment
Complement fixation	Serum or plasma	All species of birds	Approx. 21 days	May remain positive with successful treatment
Solid phase ELISA	Serum or plasma	Parrots, others?	Unknown	Not available in the USA
PCR*	Whole blood and combined oral and cloacal swab	All species of birds	Oral 5 days, blood 10 days, cloaca 15 days	Rapidly negative after the onset of treatment

+ Commonly affected species include pigeons, doves and parrots (particularly budgerigars & cockatiels).
*A combination of the elementary body agglutination assay and PCR or the complement fixation assay and PCR is more sensitive than either test alone.

cannot be shipped and it is not known if they actually are a health risk to other animals. Little work has been done to determine the success of eliminating intestinal salmonellosis with antimicrobial treatment in exotic birds; it is known that this approach does not work in poultry. Serotyping *Salmonella* isolates may be of some benefit, as certain serotypes are more commonly associated with disease in birds than others. Serology has been an extremely useful tool for the eradication of *S. pullorum* in poultry. Serologic screening for salmonellosis in other species has potential value.[11]

SPECIFIC ASSAYS

In this day and age, we rely on two important types of diagnostic assays, serologic and polymerase chain reaction (PCR) assays. Serologic assays detect antibodies to specific organisms. PCR assays detect the DNA or RNA of targeted organisms. Serologic assays have the advantage of being generally inexpensive and able to be performed on an easily acquired sample (serum or plasma) that is inexpensively shipped to the laboratory. The disadvantages of serology are that these assays may not become positive until 2 to 3 weeks after infection and may remain positive after the infectious agent is no longer present. Other complicating factors related to serologic assays include non-specific substances in serum that can sometimes cause a virus-neutralizing assay to read positive at high concentrations of serum or plasma, and anti-complementary substances that can invalidate the complement fixation assay. Virus neutralization assays have the disadvantage of requiring that growing cells are always available, and these assays typically take 3 to 5 days to run. Because of the time it takes to set up these assays, they are generally performed only once a week. There are several serological assays that use a secondary antibody that is said to detect all avian immunoglobulins. This type of cross-reactivity is extremely unlikely and this type of assay cannot be recommended.[14]

PCR-based assays have the advantages of being extremely sensitive, able to detect pathogens in the early stages of infection, and do not require viable microorganisms to document their presence in the sample. The sensitivity of these assays also is one of their limitations. Contamination at the sampling site or in the laboratory can give false-positive results. Obtaining a sample that contains the organism and getting it to the laboratory before it degrades also can be a problem for some PCR-based assays. Likewise, inhibitory substances such as antibiotics and the contents of droppings can cause false-negative results.

Not all PCR assays are created equal. Different laboratories offer tests with differing levels of sensitivity. It is important to use a laboratory that has a long history of experience with avian samples.

There are limitations to the sensitivity and specificity of individual infectious agent testing. The practitioner must remember that assays exist for only a select number of the potentially pathologic avian agents. Testing only for specific agents may not yield a diagnosis for an individual sick bird, nor is this testing sufficient to declare an individual bird or a collection free of disease.

PSITTACOSIS

Infection with *Chlamydophila psittaci* is common in pet birds. Clinical signs vary from none to a mild respiratory disease to a severe multisystemic, often fatal, disease. Psittacosis is particularly important in avian medicine because it can spread widely before it is recognized and because it is a zoonotic and reportable disease. Clinical signs and traditional diagnostic assays such as hematology, clinical pathology and radiology, while helpful, are generally insufficient to specifically diagnose this disease.

Serology

The elementary body agglutination assay (EBA) is a tried-and-true serologic assay that has been used for the past 10 years. It detects anti-*Chlamydophila* IgM. It can detect infected birds within 15 days of infection and generally is positive by the time a bird is showing signs of illness (Table 21.1). Another advantage of this assay is that it becomes negative with successful treatment. It has a few minor limitations. Rarely, a bird may develop signs of disease before the agglutinating antibodies are produced. Also, rarely, a bird with chronic psittacosis may be EBA negative. The EBA works very well for psittacine birds, but may not work in all species, particularly doves

and pigeons. For doves and pigeons, the complement fixation assay (CF) is currently recommended.[10,20]

The CF test detects anti-*Chlamydophila* IgG that is present in blood a few days to a week after anti-*Chlamydophila* IgM. Therefore, there is a slight delay between when the EBA and the CF become positive. The CF also may stay positive for an extended period of time after a bird has been successfully treated. To the best of the author's knowledge at this time, only one laboratory offers the CF and EBA[‡]. The CF is a cumbersome test and is not routinely run, so there may be a delay in getting results.

A solid phase enzyme-linked immunoassay (ELISA)[a] is currently available, although not in the USA. This assay was found to compare favorably to the CF if the serum sample produced a spot as dark or darker than the positive control (J. Grimes, personal communication, 1995). Other serologic assays are offered, but have not been validated by peer-reviewed research.

PCR

PCR testing for psittacosis is another excellent way to identify infected birds. The organism can be detected in swabs of the oral cavity as early as 5 days after infection, in cloacal swabs by 10 days after infection and in the blood by 15 days after infection. The major disadvantage of the PCR is that birds that have been started on treatment before testing may be negative, and cloacal swabs that are heavily contaminated with feces may interfere with this assay. The sensitivity of this assay is improved if both blood and combined oral and cloacal swabs are tested.[1,8]

No test is 100% sensitive. Therefore, if the greatest degree of sensitivity is sought, the PCR and the EBA and egg inoculation culture or tissue culture could both be performed when screening parrots. PCR can be combined with the CF when screening doves and pigeons.

Chlamydophila psittaci infections can occur in almost any species of caged bird. The author recommends testing for this organism in most birds presented for new bird purchase examinations. The author especially recommends testing cockatiels (*Nymphicus hollandicus*) as they can carry this bacterium and not demonstrate clinical signs of disease. The few cases of human infections the author has observed have been acquired from an otherwise healthy pet cockatiel. Pigeons and doves are commonly infected with *C. psittaci* and should be routinely tested.

MYCOBACTERIOSIS

Several mycobacterial species, including *Mycobacterium avium*, *M. genavense*, *M. fortuitum* and *M. tuberculosis,* cause mycobacteriosis in birds. *Mycobacterium avium* and *M. genavense* cause the majority of avian infections. The time between infection and onset of clinical signs is long, possibly several months. As a result, infected but outwardly healthy birds may be introduced to a collection and infection disseminated widely before it is recognized. Mycobacteriosis is particularly common in captive populations of grey-cheeked parakeets (*Brotogeris pyrrhoptera*), canary-winged parakeets (*B. versicolorus*), *Pionus* spp., canaries (*Serinus canarius*), finches, the red siskin (*Carduelis cuculatta*) and waterfowl. Mycobacterial infections result in a multisystemic but slowly progressive disease that can present with many possible signs. Because signs are rarely specific and infection can remain inapparent for extended periods of time, ancillary diagnostic assays are needed.[33]

Detection of the Organism

Many mycobacterial infections colonize the intestinal lamina propria and mycobacteria can be shed in the feces. Acid-fast stains of the feces will reveal the organisms in some cases, but this assay has a very low sensitivity and is of limited value as a screening tool. A PCR assay[‡‡] is now being offered that can detect mycobacteria in the feces. The major limitation with this assay is that not all birds with avian tuberculosis are actively shedding the organism, or they shed the organism in small numbers or intermittently. A negative result with the PCR assay, therefore, does not rule out the possibility of infection. The technology associated with PCR diagnostics for avian mycobacteriosis is developing rapidly and this assay has significant long-term potential.[33]

Serology

Mounting evidence suggests that serology will be an important tool for detecting birds with mycobacteriosis. At the time of this writing, however, serologic assays for mycobacteriosis are still experimental. Successful serologic assays will have to be applicable to all species that are to be tested, and they will have to be able to detect infection with the multiple species of mycobacteria that infect birds. A complement fixation assay was developed to detect mycobacterial antibodies. This assay had the advantage that it did not require species-specific reagents. The complement fixation reaction, however, was very cumbersome and required reagents that had a short shelf life, making it impractical to use. Additionally, culture filtrate was used for this assay and it was necessary to run multiple tests with antigens from each specific serotype of *M. avium* in order to detect all of the infected birds.[18]

Indirect ELISAs have been used successfully to detect antimycobacterial antibodies in waterfowl and quail. The indirect ELISA, however, requires a specific secondary

antibody, limiting its usefulness to closely related species. A blocking ELISA has been developed that circumvents the need for a specific secondary antibody. This assay has been tested in canaries, white-winged wood ducks and quail with known or suspected *M. avium* infections. At this point, there has been good correlation between infected birds and birds that are positive with this assay. Protoplasmic antigen from one serotype of *M. avium* was found to cross-react with all other serotypes tested. However, early work suggests that antibodies to other species of mycobacteria can be detected only if their specific antigen is used.[3,4]

The immunological response of the host also may complicate the interpretation of serologic assays for mycobacteriosis. The complement fixation assay was used to screen ring-necked turtledoves for infection. The wild-type birds in this collection were all antibody positive, but the majority of the birds that had the white color mutation were seronegative. It is not known if the failure of an antibody response in these birds is limited to this particular color mutation or also may occur in other species and color mutations of birds[18] (see Chapter 28, Implications of Mycobacteria in Clinical Disorders).

MYCOPLASMOSIS

Mycoplasmosis is a common disease of pigeons and poultry, but occurs infrequently in companion birds with the possible exception of cockatiels. The disease in pigeons and companion birds is characterized by conjunctivitis and upper respiratory signs, and less frequently pneumonia. Distention of the infraorbital sinus with fluid or purulent material is common. PCR assays for *Mycoplasma* spp. have been developed and are offered by many diagnostic laboratories. Some of these assays will amplify DNA from all mycoplasmas, so that a *Mycoplasma* sp. infecting a parrot could be detected even if it was not one of the common poultry pathogens. The disadvantage of this assay is that just because mycoplasma is present in a lesion, it is not conclusive proof that it is the cause of disease.

ASPERGILLOSIS

Aspergillosis is an infection of the respiratory system that occurs sporadically in a wide range of birds (see Chapter 29, Implications of Mycoses in Clinical Disorders). Birds from cold and dry climates are highly susceptible to infection. Environments that are conducive to the environmental growth of *Aspergillus* spp. and environments that are poorly ventilated will result in an increased incidence of aspergillosis. Disease can be localized to the upper airways or the syrinx, or it may involve the air sacs and lungs. Respiratory signs are a common feature of this disease, but a bird may not manifest signs until the disease is advanced. Radiographs, endoscopy and biopsy, cytology and hematology are all valuable tools in the diagnosis of this disease. Even with all these assays, the diagnosis of aspergillosis is often a difficult one.

The diagnosis of aspergillosis has been most extensively studied in humans. Ancillary diagnostic assays used in people include PCR to detect *Aspergillus* DNA from blood, an ELISA to detect *Aspergillus* antigen and an ELISA to detect anti-*Aspergillus* antibody. These studies clearly indicate that even a combination of these three assays will not be adequate to detect many cases of aspergillosis.[4,5] The problem comes from the fact that most people who contract aspergillosis are immunocompromised. This also may be true in birds. If the infected person's immune system is adequate to contain the disease and the organism is localized in a walled-off granuloma, then these individuals are found to produce antibody. People with generalized disease are generally severely immunocompromised and they do not produce antibody. In these people, *Aspergillus* antigen and DNA are most likely to be found in the blood, but they are not when the lesion is encapsulated. If the pathophysiology of avian aspergillosis resembles that seen in humans, then none of these assays are likely to detect infection in most infected birds. A combination of these assays may be more specific, but false negatives are to be expected.

Serology

Extended efforts have been undertaken to develop a serologic assay for birds infected with *Aspergillus* spp. This work was pioneered at The Minnesota Raptor Center, which currently offers an ELISA assay for the detection of anti-*Aspergillus* antibodies‡‡‡. Differing secondary antibodies are used in this assay, depending on the species of bird to be tested. Using well-defined clinical case material, the assay has been validated for use in several species of Falconiformes, but as designed does not work in owls. Immune suppression appears to accompany aspergillosis in raptors. Prior to treatment, many raptors have little or no detectable antibody. Successful treatment results in a subsequent rise followed by a decline in antibody titers. A failure of antibody titers to rise with treatment or an increase in the titers without the expected decrease is considered to be a poor prognostic sign. Thus a medium to high positive antibody titer is highly suggestive of disease. However, negative to low positive results are inconclusive.[24] Similar results were found in penguins with aspergillosis.[25] Birds with high antibody titers, high beta and gamma globulins, and albumen concentrations above 1.8 g/dl had a favorable prognosis. In contrast, birds with low or undetectable antibody titers and albumin levels below 1.8 g/dl had a poor prognosis.

The accuracy of available serologic and antigen capture assays for the diagnosis of aspergillosis in parrots has been inadequately studied. In one study, a commercially available ELISA for anti-*Aspergillus* antibodies and antigen capture assay‡‡‡‡ was evaluated in seven birds with confirmed aspergillosis. Of these birds, only one was found to be weakly positive with serology and three birds were positive, two weakly, with the antigen detection assay, suggesting that either parrots in this study did not make anti-*Aspergillus* antibodies and had little circulating antigen, or that these assays were not sensitive.[13] A second study of ten birds found a higher percentage of sero- and antigen-positive birds; however, in neither study were non-infected birds tested, so the specificity of these assays remains to be determined.[16] Currently, the author does not recommend using any of the available *Aspergillus* assays for routine screening of parrots.

PSITTACINE BEAK AND FEATHER DISEASE VIRUS (PBFDV)

PBFDV is a common infection of wild birds in Australia. In the USA and possibly elsewhere, this virus is enzootic in many lovebird collections and also is seen to a lesser degree in budgerigar aviaries. Disease is seen in many species of parrots, including African grey parrots, lovebirds, budgerigars, lories, lorikeets, eclectus parrots and cockatoos. Infection also occurs in Neotropical parrots, but disease is rare and infections are transient in most cases.

The sequence of the PBFDVs genome varies up to 16% between isolates. This has diagnostic significance, as PCR primers have to be designed in the conserved region of this virus (ORF1) if they are to detect all variants of this virus.[3,37] Recently, a specific variant of PBFDV has been recognized in collections of lories in the USA. It also is reported to occur in lovebirds.[26] Its sequence and its relationship to previously published sequences of the PBFDV have not been reported. PCR primers derived from the ORF1 also can detect this variant. Primers also have been designed to differentiate it from other PBFDVs. Lories with this infection may remain viremic for 6 months or more without showing clinical signs. Lories that develop clinical signs often die, but some will recover.[31]

Serology

Birds that become infected with PBFDV but do not develop disease have high antibody titers. Birds that do develop disease have low antibody titers or no antibody at all. A hemagglutination assay has been developed to detect serum antibodies to PBFDV. Serum antibody has been detected within 1 to 2 weeks of exposure. This assay has been effectively used to study the prevalence of PBFDV infections in wild Australian parrots. Because PBFDV agglutinates only red blood cells from a few species of cockatoos, this assay is not practical outside of Australia.[23]

PCR

Birds become viremic 7 to 14 days after infection with PBFDV. If the birds are unable to mount an appropriate immune response they will remain viremic. If they do mount an appropriate immune response they cease to be viremic. Virus, however, may persist in the feathers and possibly the skin, so that these birds are a potential source of infection until their next molt. PCR is done on heparinized blood.[7] If multiple birds are to be sampled, care must be taken to prevent contamination between samples. In most circumstances, birds that are PCR positive and have clinical signs of disease will remain positive and are likely to die from their infection. Birds that are positive but are not showing signs of disease should be retested in 3 months. If they are negative at that time they are thought to be cured. Rarely, lories, lovebirds and occasionally other species of parrots will develop clinical disease, but will then recover and become virus negative.

It has been suggested that it is important to differentiate between the lory variant of PBFDV and the other variants. The author does not agree with this conclusion. Although the lory variant may behave somewhat differently than other PBFDV variants, it is still pathogenic, so the significance of a positive test is the same in a lory or any other parrot species, regardless of the variant.[31]

PBFDV infection in lovebirds (*Agapornis* spp.) may not follow the same patterns as seen in other parrots. PBFDV is widespread in commercial lovebird collections, but disease is rare. It is the author's impression that virus shedding may persist more than 3 months in birds that never show signs of disease.[26]

AVIAN POLYOMAVIRUS (APV)

APV is a common infection of a wide range of parrots. APV causes morbidity and mortality in nestling budgerigars (*Melopsittacus undulatus*), Indian ring-necked parakeets (*Psittacula krameri*), lovebirds and many parrots. Disease is less common in nestlings of other Old World parrots. Nestling budgerigars in aviaries with enzootic APV become viremic within 7 days of hatch and are serologically positive by 10 days after hatch. If they survive infection, fecal shedding may persist for 6 months or longer, but ceases at some point after the birds become sexually mature. Although they stop shedding virus, infected budgerigars will remain seropositive for life.[15,16] Nestling parrots of other species become viremic within 2 weeks of infection. They also develop virus-neutralizing antibody at

approximately 14 days postinfection. Antibody titers in most species of nestlings that survive infection are maintained for 10 or more years and possibly for life. Viremia persists for 6 to 8 weeks in most cases. Fecal shedding begins shortly after the onset of viremia, but persists for as long as 12 to 16 weeks.[21] In rare cases, viremia and fecal virus shedding may persist for more than 10 months.[7] The duration of viremia and virus shedding in adult birds infected with APV has been studied in only a limited number of birds. However, it appears that viremia and virus shedding occurs only briefly in mature birds or not at all.[21] Viremia and virus shedding also are significantly impacted by concurrent infections with PBFDV. Birds with co-infections appear to shed APV continuously and may never clear the virus.[16]

Serology

An excellent virus-neutralizing (VN) assay has been developed to detect antibodies that neutralize APV. In this assay, virus is first incubated with two-fold dilutions of serum or heparinized plasma. The virus-plasma mixtures are then incubated with chicken embryo fibroblasts, the fibroblasts are washed and the cells are monitored for cytopathic effects (CPE). If CPE do occur at the *highest* concentration of serum, then the bird did not have neutralizing antibody. If virus growth is inhibited and CPE do not occur, then the bird did have neutralizing antibody. In the author's hands, this assay takes 5 days to complete.[15]

Use of the APV VN

Parrots infected with APV may begin shedding virus prior to seroconversion and maintain high antibody titers many years after they stop shedding virus. Therefore, the serologic status of a bird is not a good indicator of virus shedding. Sensitive PCR assays should be used in place of serology to detect virus-shedding birds. The APV VN has some limited value in epidemiologic studies and could be used to determine if APV had ever been in a collection. Under these circumstances the immunization status of the birds should be considered. Nestlings do not produce virus-neutralizing antibody to the commercial APV vaccine. Therefore, antibody-positive nestlings have been infected with APV. Adult parrots do develop neutralizing antibody following immunization, but their antibody titers are typically low compared to those seen in birds that survived infection.[19]

Use of the APV PCR

Viremia may precede virus shedding and virus shedding continues after the cessation of viremia; therefore, combined oral and cloacal swabs and heparinized blood should be submitted for PCR analysis. If blood is tested alone, many virus-shedding birds will be missed. It appears that all species of parrots have the potential to become infected with and shed APV, so all birds that are going into an aviary where they might expose nestlings should be tested. Nestlings that survive an outbreak of APV are assumed to be shedding virus. Therefore, testing birds 4 months after the outbreak when virus shedding should have stopped, rather than immediately after the outbreak when virus shedding is expected, best uses the owner's resources.[21]

Testing birds older than 16 weeks that are going into a single-bird household is of questionable value. If they test positive and are not sick, they will shed transiently and stop shedding. If a bird is positive at 16 weeks, it has already been infected and will not generally become clinically ill. It will continue to shed for some time and it should be isolated from other birds.

The author seriously doubts that the veterinarian will be able to detect an infected bird that will subsequently come down with disease as, in his experience, the onset of viremia and the onset of disease occur very close together.

When the value of testing blood was first recognized, it was suggested that PCR of blood detected only fragments of DNA and that a positive did not reflect the true infection status of the bird. It also was suggested that immunized birds that were blood PCR positive were positive because of DNA present in the vaccine. Both these assumptions have been proven to be false. Therefore, if a bird is positive by blood PCR, it is infected with APV.[19] If they test positive and do not have APV disease, they will shed transiently and then stop shedding.

PSITTACID HERPESVIRUSES (PsHVs) OR PACHECO'S DISEASE VIRUSES

PsHVs are the causative agent of Pacheco's disease. Pacheco's disease occurs in sporadic outbreaks in newly formed and long-established parrot collections. Losses can range from a single bird up to hundreds of birds. Generally, birds that develop clinical Pacheco's disease die. There are four major genotypes of PsHVs. All are capable of causing Pacheco's disease and genotypes 1, 2 and 3 are capable of causing internal papillomas.[32,35,36] Outbreaks of Pacheco's disease occur when carrier birds expose naïve birds. The dynamics of each outbreak will depend on the genotype of the virus and the species of birds involved. In some collections, Pacheco's disease will not occur, but over time multiple birds will develop papillomas.

Macaws, Amazon parrots and some species of conures are most likely to be carriers of PsHVs. Infection prevalence appears to be higher in imported wild-caught birds. Infection also has been recognized in cockatoos

and African grey parrots, and under some circumstances it also may occur in lovebirds and cockatiels. The list of potential carrier species likely will grow as more is learned about these viruses. Any bird that survives an outbreak of Pacheco's disease should be considered infected until shown otherwise. Mounting evidence suggests that parent-to-offspring transmission occurs. The offspring may remain asymptomatic or develop internal papillomatosis, depending on the genotype of the virus. It appears that once a bird is infected they will be infected for life. This includes survivors of Pacheco's disease that were treated with acyclovir.

The key to preventing Pacheco's outbreaks and internal papillomatosis is keeping carrier birds out of the collection or, if they are already in the collection, isolating them from birds that are not infected. Studies to date show that PsHVs in carrier birds are present in significant concentrations in the mucous membranes of the cloaca and oral mucosa. Swabs of these surfaces can be tested by PCR. Virus also may be detected in blood, but concentrations of virus are low in the blood and in one study blood PCR was inconsistently positive, while mucosal swabs were more dependably positive. In rare individuals, birds have been identified that are only blood positive. The biological significance of this is not known; until it is, it is recommended that both blood and combined oral and cloacal swabs be used for PsHV PCR.[22]

PCR

Recently discovered sequence data has permitted the development of a single PCR assay that can detect all four genotypes of the PsHV (R. Dahlhausen, personal communication, 2003). Preliminary work with less ideal primer sets suggests that PCR of blood and a combined oral and cloacal swab will detect the majority of birds unapparently infected with PsHVs.

Serology

There are three major serogroups of the PsHV.[9] Serotype 1 contains genotypes 1 and 4, serotype 2 contains genotype 2 and serotype 3 contains genotype 3.[36] It is clear that many birds that are infected with PsHVs are seropositive. It still remains to be determined, however, if all birds infected with PsHVs will demonstrate positive serologic results. Preliminary evidence suggests that antibodies to one serotype inconsistently neutralize viruses of other serotypes.[22] Therefore, if serology is to be used to detect PsHV-infected birds, multiple assays using all three viruses or their antigens will have to be run.

PARAMYXOVIRUS 3 (PMV-3)

PMV-3 is a common cause of disease in the Australian grass parakeets (*Neophema* spp.). Clinical signs are variable and include central nervous system disease, respiratory signs, diarrhea and signs of pancreatic insufficiency.[28,29] In a recent report of an outbreak of PMV-3 in a pet store, the hemagglutination inhibition assay (HI) was found to be a sensitive means of detecting infected birds.[14] Others have tried serologic methods for detecting subclinical infections of PMV-3 in *Neophema* and other species with little success.[12,29] This discrepancy may be due to the duration of the infection at the time serology is performed. In a recent study with PMV-1, low levels of antibody were detected in African grey parrots. These antibodies were detectable with an experimental ELISA, but not with the HI. This assay is currently under development and may prove useful in the future.[14] In disease outbreaks where PMV-3 is suspected, submission of proper samples for histopathology is currently the most accurate method of confirmation.

Applied Preventive Medicine

TESTING NEWLY ACQUIRED BIRDS

The ultimate decision as to what type of testing should be done for a particular bird will depend on the specific details regarding the source of the bird, species of the bird, the aviary or home into which it is going, the resources of the owner and findings on physical examination. Non-specific assays such as CBC, oral and fecal Gram stain, protein electrophoresis, fecal wet mount and fecal floatation can be applied to all birds. *(Ed. Note: In some practitioners' experience, a negative fecal flotation has not correlated with the absence of intestinal parasites)*. Ascarids are commonly expelled from birds, especially those with previous exposure to warm, outdoor environments, following the administration of an appropriate anthelmintic. This occurs in birds with negative fecal flotations, and routine deworming may be advised in these situations (M. Wissman, personal communication, 2002).

Fecal and oral cultures are indicated if abnormalities are found on the Gram stains and birds show other evidence of illness. Chemistry panels are most likely to identify problems in older or unthrifty birds, but can be useful in detecting early disease or establishing baselines for future reference, even in clinically healthy individuals. Radiographs are relatively costly tests that can be used for screening. Generally, however, they are used only when there is some other indication of disease. Currently, the choice of serologic and PCR-based testing is best tailored to the species and background of the bird being examined (Table 21.2 and Table 21.1).

Table 21.2 | Diagnostic Tests Used to Screen for Specific Infectious Diseases

Infectious Agent	Assay	Sample for Testing	Species Commonly Infected	Sensitivity	Specificity
Mycobacteria	PCR	Feces	*Brotogeris* spp., canaries, finches, red siskins, waterfowl	Fair	Good
	Serology	Serum or plasma		Experimental	Experimental
M. ornithogaster	Wet mount	Feces	Budgerigars, finches, cockatiels, parrotlets, lovebirds, lories, other	Fair to poor	Good if many organisms present
PBFDV	PCR	Blood*	Old World parrots	Excellent	Excellent
APV	PCR	Blood and swabs**	Lovebirds, budgerigars, all parrots recently exposed to other birds	Excellent	Excellent
	Serology	Serum or plasma		Proof of previous or current infection	Does not reflect virus-shedding status
PsHV	PCR	Blood and swabs	Macaws, Amazon parrots, conures, others?	Excellent	Excellent
	Serology	Serum or plasma		Unknown	Unknown
PMV-3	HI+	Serum or plasma	*Neophema* spp., others?	Questionable in chronic infection	Good
	ELISA++	Serum or plasma		Experimental	Experimental

*Heparinized blood
**Combined oral and cloacal swab
+ HI: Hemoagglutination inhibition
++ ELISA: Enzyme-linked immunoassay

There is a saying: "Be careful for what you look for, because you may find it." This is particularly applicable to testing new birds. It is incumbent upon practitioners to know everything that they can about the tests they are using so that if one does come back positive, it can be properly interpreted. It also is important to correlate test results with the entire clinical picture. If the testing results don't make sense, then repeat those assays or have them performed by a different laboratory.

PREVENTIVE MEDICINE AND THE VETERINARY HOSPITAL

It is a common practice for veterinarians to board birds. There is no doubt that this is a valuable service to the veterinarian's clients, but it also poses challenges for the prevention of disease transmission. The greatest risk occurs if birds of uncertain infection status are housed in the same room. If all birds are screened for PBFDV, APV, PsHVs and *Chlamydophila psittaci* before they are allowed to board, then the risk of disease is diminished. There is no test for birds that have the etiologic agent of proventricular dilatation disease, however, so the transmission of this disease cannot be prevented. Other strategies for preventing disease transmission would be to keep birds in isolettes or to house birds separately in different parts of the hospital.

Veterinarians see sick birds and therefore will have birds with infectious diseases in their hospital. A protocol should be developed for every hospital for routine cleaning of the exam, treatment and hospital rooms and caging. Routine PCR testing of swabs of these environments can be used to determine if the cleaning is effective. Boarding birds should be housed separately from hospitalized birds.

PREVENTIVE MEDICINE AND BIRD MARTS

A common way for aviculturists to sell their birds is to bring them to bird marts or bird fairs that are sponsored by local bird clubs. These marts serve many valuable purposes. They provide an important outlet for the sale of birds and at the same time raise money for the sponsoring organizations. This money is used to help support the bird club and in many cases to fund research and conservation efforts. The bringing together of birds from multiple premises into a confined area and the handling of these birds by the general public, however, results in the ideal opportunity for disease spread, the most common of which is APV.

There are preventive measures that can be taken that will help to mitigate disease transmission at bird marts. The most important is to limit sale of birds to those that are completely weaned. Weaned birds will rarely, if ever, develop APV disease although they are still susceptible to infection. Birds that are taken to a bird mart but not sold should be quarantined away from the rest of the breeder's birds until they can be sold. A policy of not letting anyone handle birds unless they have bought them also will reduce the spread of disease. Finally, cages made of clear, hard plastic panels can be used to display birds that are for sale. Ideally, these cages would have a fan in the back that draws air out of the cage and a Hepa filter in the front to filter out potential pathogens. Even without these fans, cages made from clear plastic panels are much better than wire cages. If nestlings are allowed at bird marts, then they should be confined to brooders or cages made from this material and taken out to the car or hotel room for feeding. Nestlings that are not sold must go into quarantine after the show.

PREVENTIVE MEDICINE IN THE PET STORE

Pet store owners and managers who intend to sell birds first need to consider what market it is that they wish to reach. Budgerigars, cockatiels, lovebirds, canaries and finches appeal to one type of customer and come with their own significant disease problems. The larger species

of birds appeal to other types of customers. Combining these birds can lead to additional health problems.

It has been a common practice in the USA for individual producers of cockatiels, lovebirds, budgerigars and finches to sell their birds to buyers who combine birds from multiple sources and ship them to other sellers who distribute them to pet stores. This practice maximizes the potential for disease transmission. Cockatiels supplied in this manner have a high incidence of psittacosis. Similarly, lovebirds and budgerigars from these sources are commonly infected with APV, and lovebirds are commonly infected with the PBFDV. When infected birds are mixed with birds from clean collections, disease transmission is likely. When these birds come into a pet store, not only may they be unhealthy, but they also are an important source of infection for other parrots whose retail value may be much higher. The classic example of this is APV outbreaks that occur in nestling macaws, conures and eclectus parrots 2 weeks after they are brought into a pet store. The tendency in these circumstances is to blame the breeder who supplied the nestlings that died, but the problem lies with the budgerigars and lovebirds in the store that are actively shedding virus and that fatally exposed these birds after they entered the store.

It has been the author's experience that pet stores have healthier stock if they establish a relationship with one or more local breeders and buy birds directly from them. If the local breeder can see the possibility of a sustained market, they are more willing to spend money to verify that their flock does not contain the common diseases that can cause so many problems in the pet store. Aviaries that supply birds to stores can be screened for infectious diseases by environmental testing or testing a random selection of birds. The specific types of screening tests should be tailored to the type of birds being purchased, and this protocol is best done with the assistance of an avian veterinarian. Subsequently, if appropriate biosecurity measures are maintained, the pet store owners can feel assured that they are buying clean stock. This requires some initial investment, but this investment is spread out over many birds and is well worth it.

Other management techniques can be used to minimize the risk of disease. First and foremost, a relationship should be established with an avian veterinarian. The veterinarian's role is to provide advice that will help minimize the risk of disease, but at the same time will not result in huge expenditures. A general rule is that any change should increase the pet store owner's profit. If it does not, then another approach should be taken.

The two diseases that can substantially impact the pet store are APV and psittacosis. APV is predominately a disease of nestling parrots. Lovebirds and budgerigars from many sources may shed this virus. The risk of APV disease can be greatly reduced if stores buy only weaned birds. Alternately, some stores may choose not to sell the smaller species of birds. If nestlings are to be present in the store, all the sources of all birds brought into the store need to be screened for APV. Setting up a separate bird room that the public can look into but may enter only with supervision will help to keep customers from bringing disease into the store. If a customer wants to see a bird, they may be required to put on a clean smock and gloves and possibly even dip their feet in a foot bath before entry into the bird room. If economics require that nestlings that are still being hand-fed be purchased, an alternate approach to keeping them healthy is to raise and wean them in isolation away from the store.

Psittacosis is very common in cockatiels and can occur in any species of parrot. It can cause widespread disease in pet store birds, requires a long treatment period, is a reportable disease in most areas and is transmissible to people. All sources of birds should be tested for this disease.

Quarantine is another element of the preventive medicine that can provide important dividends to the pet store. All birds coming into the store should be isolated for some period of time before they are mixed with other birds in the store. If the incoming birds have been exposed to disease, it is likely that they will begin showing signs during the quarantine period. It has been common practice for some distributors to treat some species of birds with tetracyclines for variable lengths of time before they are sold. This can mask signs of psittacosis but may not cure the birds. Once the birds are off medication signs will often reappear. Money is a factor in any preventive health plan and a careful balance must be established between cost of preventive medicine and its benefits. Careful consideration should be taken so that all preventive measures have a clear economic benefit.

Finally, if preventive measures are undertaken, the public should be made aware of what is being done and birds should be sold as value-added products. For instance, if extensive efforts are undertaken to acquire polyomavirus-free birds, then these birds should be advertised as such. When the consumer sees that one store is concerned about infectious diseases and others do not place similar emphasis on them, the consumer will buy from that store, even if the cost may be somewhat higher.

IMMUNIZATION AND PREVENTIVE MEDICINE

Immunization for poxvirus, paramyxovirus-1 and salmonella can be important elements of disease control in rac-

ing pigeons. Poxvirus immunization also is advised for canaries that are raised outdoors. The current parrot herpesvirus vaccine in the USA is a monovalent vaccine. The exact serotype present in the current vaccine is not known at the time of this writing. It is expected that this vaccine will protect against the serotype from which it is derived. It is not known, however, if this vaccine will protect against other serotypes. In collections of birds where there is a high risk of Pacheco's disease, use of this vaccine may be indicated. A polyvalent vaccine that would protect against infection with the three common serotypes may someday be developed and could potentially protect against Pacheco's disease and internal papillomatosis.

The value of the equine West Nile virus vaccine in birds remains to be proven. The author was not aware of adverse reactions to the vaccine the first year that it was used. However, a hemolytic anemia has been reported in lories that were immunized a year after the first set of immunizations. This problem, however, has not been seen in another collection of birds immunized 2 years in a row. Given that the current information about the potential value and potential risks of the West Nile virus vaccine is minimal, it should probably be used only as a last resort. Screening in the enclosure of high-risk birds and other mosquito control programs may be the safest ways to prevent disease from the West Nile virus.

A discussion of the avian polyomavirus vaccine is included in Chapter 32, Implications of Viruses in Clinical Disorders. The author believes that management practices are critical to the control of avian polyomavirus, and that there are few circumstances where immunization would be helpful in its control.

Product Mentioned in the Text

a. Immunocomb, Biolage Laboratories, Kibbutz Baled, Israel

Resources

‡ Texas Veterinary Medical Diagnostic Laboratory, PO Drawer 3040, College Station, TX 77841, 979-845-3414

‡‡ Dr. Carlene Emerson, Department of Veterinary Microbiology and Pathology, College of Veterinary Medicine, Washington State University, Pullman, WA 99164-7040

‡‡‡ The Raptor Center, 1920 Fitch Ave, Saint Paul, MN 55108, 612-624-4969

‡‡‡‡ Avian and Wildlife Laboratory, University of Miami School of Medicine, Miami, FL

References and Suggested Reading

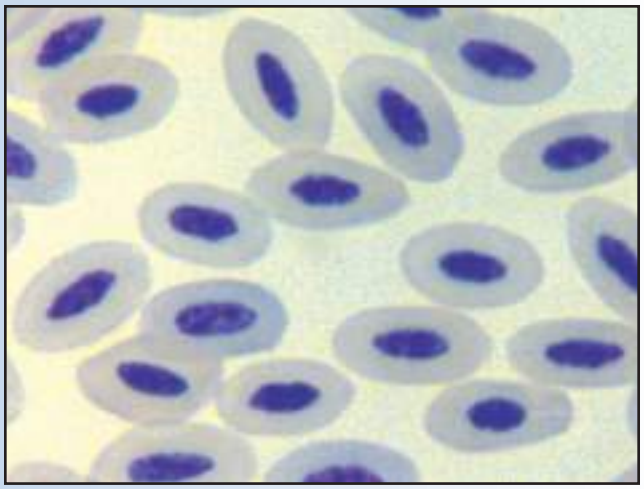
1. Andersen AA: Comparison of pharyngeal, fecal, and cloacal samples for the isolation of *Chlamydia psittaci* from experimentally infected cockatiels and turkeys. J Vet Diagn Invest 8:448-450, 1996.
2. Baghian A, et al: Production of a rabbit anti-cockatiel immunoglobulin G and characterization of its cross-reactivities with immunoglobulin G of other psittacine species. Avian Dis 43:48-54, 1999.
3. Bassami MR, et al: Genetic diversity of beak and feather disease virus detected in psittacine species in Australia. Virology 20:392-400, 2001.
4. Chan C, et al: Detection of antibodies specific to an antigenic cell wall galatomannoprotein for serodiagnosis of *Aspergillus fumigatus* aspergillosis. J Clin Micro 40:2041-2045, 2002.
5. Costa C, et al: Real-time PCR coupled with automated DNA extraction and detection of galactomannan antigen in serum by enzyme-liked immunosorbent assay for diagnosis of invasive aspergillosis. J Clin Micro 40:2224-2227, 2002.
6. Cray C: Plasma protein electrophoresis: An update. Proc Assoc Avian Vet, 1997, pp 209-211.
7. Dahlhausen B, Radabaugh CS: Update on psittacine beak and feather disease and avian polyomavirus testing. Proc Assoc Avian Vet, 1993, pp 5-7.
8. Dahlhausen B, Radabaugh CS: Detection of *Chlamydia psittaci* infection in pet birds using a molecular-based diagnostic assay. Proc Assoc Avian Vet, 1997, pp 191-198.
9. Gravendyck M, et al: Antigenic diversity of psittacine herpesvirus: Cluster analysis of antigenic differences obtained from cross-neutralization test. Avian Pathol 25:345-357, 1996.
10. Grimes JE: Evaluation and interpretation of serologic responses in psittacine birds to chlamydiosis and suggested complementary diagnostic procedures. J Avian Med Surg 10:75-83, 1996.
11. Grimes JE, Arzmendi F: *Salmonella typhimurium* agglutinins in exotic bird sera in the USA. J Vet Diagn Invest 7:270-274, 1995.
12. Grund C, Grimm F, Kosters J: Serological studies on persistent PMV-1 infection associated with PDD. Proc Assoc Avian Vet, 1999, pp 19-23.
13. Ivey ES: Serologic and plasma protein electrophoretic findings in 7 psittacine birds with aspergillosis. J Avian Med Surg 14:103-106, 2000.
14. Loudis BG: PMV-3 outbreak: Presentation, diagnosis and management. Proc Assoc Avian Vet, 1999, pp 223-227.
15. Phalen DN: Viruses. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 281-322.
16. Phalen DN: Avian polyomavirus: My thoughts. Am Fed Avicul Watchbird 25:28-39, 1998.
17. Phalen DN, Tomaszewski E, Davis A: Investigation into the detection, treatment, and pathogenicity of avian gastric yeast. Proc Assoc Avian Vet, 2002, pp. 49-51.
18. Phalen DN, et al: Serologic diagnosis of mycobacteria infections in birds: A preliminary report. Proc Assoc Avian Vet, 1995, pp 65-68.
19. Phalen DN, et al: Avian polyomavirus: More pieces to the puzzle. Proc Assoc Avian Vet, 1998, pp 151-156.
20. Phalen DN, et al: Diagnosis of *Chlamydia psittaci* infections in cockatiels and Columbiformes. Proc Assoc Avian Vet, 1999, pp 13-17.
21. Phalen DN, et al: Viremia, virus shedding, and antibody response during natural avian polyomavirus infection in parrots. J Am Vet Med Assoc 217:32-36, 2000.
22. Phalen DN, et al: Diagnosis of parrots infected with Pacheco's disease viruses. Proc Assoc Avian Vet, 2001, pp 87-89.
23. Raidal SR, Cross GM: The haemagglutination spectrum of psittacine beak and feather disease virus. Avian Pathol 23:621-630, 1994.
24. Redig PT, Orosz S, Cray C: The ELISA as a management guide for aspergillosis in raptors. Proc Assoc Avian Vet, 1994, pp 99-104.
25. Reidarson TH, McBain J: Serum protein electrophoresis and *Aspergillus* antibody titers as an aid to diagnosis of aspergillosis in penguins. Proc Assoc Avian Vet, 1995, pp 61-64.
26. Ritchie BW, et al: Documentation of a PBFD virus variant in lories. Proc Assoc Avian Vet, 2000, pp 263-268.
27. Romagnano A, et al: Aspergillosis testing: Comparison of serological data. Proc Assoc Avian Vet, 2002, pp 139-143.
28. Shihmanter EY, et al: Avian paramyxovirus serotype 3 isolated from captive birds in Israel: Clinical signs, pathology and antigenic characterization Avian Dis 42:418-422, 1998.
29. Speer BL: A clinical look at the avian pancreas in health and disease. Proc Assoc Avian Vet, 1998, pp 57-64.
30. Speer BL: Avicultural medical practice: The nuts and bolts. Proc Assoc Avian Vet, 1998, pp 347-355.
31. Strunk A, et al: Pathobiology and testing recommendations for psittacine circovirus 2 in lories. Proc Assoc Avian Vet, 2002, pp 45-47.
32. Styles D, Tomaszewski E, Phalen DN: Papillomas J Virol, submitted 2003.
33. Tell LA, Woods L, Cromie RL: Mycobacteriosis in birds. Rev Sci Tech Off Int Epiz 20:180-203, 2001.
34. Tell LA, et al: A multifaceted investigation into the diagnosis of mycobacterial infections in birds. Proc Assoc Avian Vet, 2002, pp 61-63.
35. Tomaszewski E, et al: Detection and heterogeneity of herpesviruses causing Pacheco's disease in parrots. J Clin Micro 39:533-538, 2001.
36. Tomaszewski E, Kaleta EF, Phalen DN: Molecular phylogeny of the psittacid herpesviruses causing Pacheco's disease: Correlation of genotype with phenotypic expression. J Virol, submitted 2003.
37. Ypelaar I, et al: A universal polymerase chain reaction for the detection of psittacine beak and feather disease virus. J Clin Micro 16:141-8, 1999.

CHAPTER

22

Diagnostic Value of Hematology

JAIME SAMOUR, MVZ, PhD, Dipl ECAMS



Hematology is the discipline of medical science that studies the blood and blood-forming tissues, and is currently considered an integral part of clinical laboratory diagnostics in avian medicine. Hematology assays seldom provide an etiological diagnosis, but they remain, nevertheless, indispensable diagnostic tools to evaluate health and disease in individuals, to monitor the progress of diseases, to evaluate the response to therapy and to offer a prognosis.

The routine collection and processing of blood samples allows the evaluation of the hematologic response to disease. In addition, the creation of hematology databases is important in establishing reference values for various avian species.

In the past 15 years, significant advances have been made in the use of hematology assays in the differential diagnosis of pathologic conditions in avian species. This appears to have developed parallel to other areas such as nutrition, wellness examinations, anesthesia, surgery and therapeutics.

The processing of hematology samples also has been enhanced in recent years. In the past, automatic analysis of avian blood samples was basically limited to total red cell counts using the cell counters[a] that were available and making manual adjustments of the thresholds and current aperture settings. More recently, the analysis of avian blood samples has received a significant boost with the advent of more comprehensive and accurate automatic analytical systems based on laser flow cytometry.[b] This methodology is based on the measurement of scattered laser light, which fluctuates with the size of the

Table 22.1 | Materials Needed for Blood Sample Collection

- Syringe, 1-ml or 3-ml, disposable
- Needle, 27-gauge to 23-gauge, bent, disposable
- Cotton
- Alcohol
- EDTA or heparin pediatric 0.5-ml or 1-ml blood tubes, or sodium citrate tubes

cell, the complexity of the cell (eg, overall shape, nucleus-to-cytoplasm ratio, granulation), and the size and shape of the nucleus after blood cells are exposed to a laser beam. The laser flow cytometry unit produces a graphic display containing a total optical white cell count, white cell differential count expressed in percentage, absolute values and total red cell count, hemoglobin measurement by the cyanmethemoglobin method, thrombocyte count, and white cell count by cell-lysing impedance measurement of cell nuclei.[14] However, the use of laser flow cytometric technology in avian species is not free from deficiencies.

There are certain pathological conditions in which the presence of enlarged thrombocytes (commonly referred to as megathrombocytes) in the blood film appear to be a characteristic hemoresponse. For instance, in the houbara bustard (*Chlamydotis undulata macqueenii*), the mean thrombocyte measurements in birds undergoing chronic inflammation (severe shoulder injury as a result of repeated crashing against the enclosure wall) were 9.22 ± 0.21 μm length and 8.10 ± 0.19 μm width compared with 5.47 ± 0.12 μm length and 4.96 ± 0.10 μm width in clinically normal birds.[9] The mean diameter of lymphocytes in clinically normal houbara bustards is 7.7 μm;[51] however, there are other species, such as the kori bustard (*Ardeotis kori*), in which the presence of large and small thrombocytes in the same blood film appears to be normal.[51] It is, therefore, probable that a sample containing megathrombocytes would yield a high lymphocyte count under an automatic analytical system, as it would be impossible for even a sophisticated unit to differentiate between lymphocytes and megathrombocytes. When dealing with such species, the software would require some adjustments in order to properly differentiate these cells. This would obviously imply the need to carry out extensive calibration based on repeated manual assessments on a significant number of samples.

Furthermore, in certain species it is relatively common to find large and small lymphocytes in the same blood film.[4,12,26,31,35] This phenomenon has been observed in many psittacine species. Clinically normal kori bustards, for instance, demonstrated a mean diameter for small lymphocytes of 7.2 ± 0.12 μm, whereas the mean diameter of large lymphocytes was 10.7 ± 0.16 μm.[31] Therefore, total white blood cell counts and differential white blood cell counts cannot be accepted as reliable in every clinical case and in every species if the values were estimated by laser flow cytometry. It is, therefore, highly recommended to re-evaluate these samples using manual methods. Clearly, the clinician must be fully familiar with the materials and methods of hematology analysis in order to assess and understand the results.

Blood Sample Collection

It is essential that blood samples be obtained from avian species by or under the supervision of a veterinarian who is experienced with avian venipuncture. The assistant, if one is used, also should be comfortable with restraint and handling techniques. The techniques used vary according to personal preferences and the species being handled. Materials needed for blood sample collection, ie, syringes, slides, tubes, should be labeled in advance and readily accessible (Table 22.1).

METHOD

The total blood volume in clinically normal birds is in the range of 6 to 11 ml per 100 g of body weight.[54] Thus, a bird weighing 250 g would have approximately 15 to 27.5 ml of blood, of which, in a clinically normal individual, up to 10% (1.5-2.7 ml) can be safely withdrawn without having any detrimental effect on the patient. However, 0.2 to 0.3 ml of blood is generally sufficient to carry out a comprehensive hematology examination in a bird.

In birds, blood samples are commonly collected using the right jugular vein (v. *jugularis dextra*), as this is generally larger than the left jugular vein in most avian species (Fig 22.1a). Other preferred sites include the basilic vein (Fig 22.1b) (v. *cutanea ulnaris superficialis*) and the caudal tibial vein (v. *metatarsalis plantaris superficialis*) (Fig 22.1c).

The methodology used for the collection of blood samples varies according to the species and the site selected (Fig 22.1d-h). For example, in long-legged birds such as large bustards, cranes and storks, the jugular or caudal tibial veins are very often used. In the author's opinion, blood samples should be collected from the heart or the occipital sinus only if these birds are under anesthesia and are to be euthanized. It is a poor practice to collect blood samples from clipped nails, as cell distribution and cell content is invariably affected.

The author prefers obtaining blood samples from most bird species from 200 to 4000 g using a basilic vein

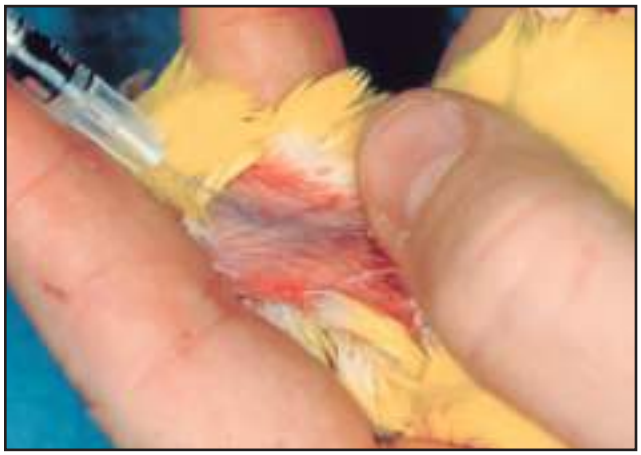


Fig 22.1a | Right jugular being blocked in a love bird and a syringe with a 27 ga needle that has been bent to allow venipuncture.

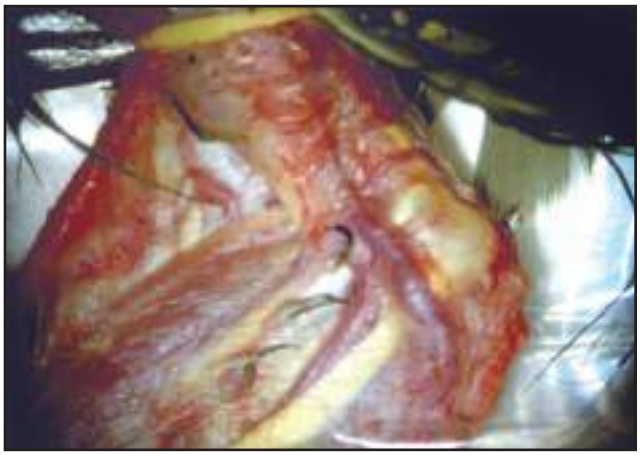


Fig 22.1b | The blocked basilic vein showing the torturous nature of the vein making cannulation with a hypodermic needle difficult at best.

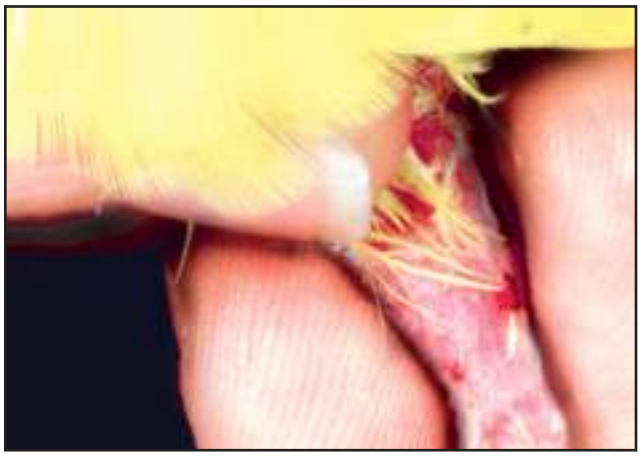


Fig 22.1c | Caudal tibial vein in a love bird.

while the bird is in dorsal recumbency, although most practitioners in the US dealing with psittacine species prefer jugular venipuncture. In most avian species, the optimal area for collecting a blood sample from a basilic vein is along the medial section of the vein. The preferred side is from the right wing if the practitioner is right-handed, while the left wing is the preferred side if the practitioner is left-handed. Venipuncture immediately above the elbow joint is not recommended, as hemostasis is difficult to achieve at this site in most cases. The application of digital pressure with the thumb at the proximal humerus would help in raising the vein, making it clearly visible running parallel to the external aspect of the humerus. After separating the feathers and preparing the site with an alcohol swab, the bent needle is gently inserted into the vein at an approximately 45° angle. The sample can now be collected, taking precaution not to exert high negative pressure while withdrawing with the syringe because this will invariably result in the collapse of the vein.

While withdrawing the sample, it is recommended to continue maintaining pressure on the proximal humerus to ensure a raised and well-defined vein. The method of approaching the basilic vein dorsally by entering under the adjacent tendon prevents hematoma formation because the underlying tissue exerts pressure on the venipuncture site when the needle is withdrawn. It is essential to avoid sudden movements that can alarm the bird and trigger a struggle, as this can easily lacerate the vein and result in a hematoma or, worse, a severe hemorrhage. After collection, a small ball of dry cotton should be placed over the venipuncture site and the wing closed to maintain pressure over the site for a few seconds. It is strongly advisable to check the venipuncture site before releasing the bird back to its enclosure to ensure no post-collection hemorrhage has occurred. Any sample that contains clots should be rejected, as the processing of such samples would invariably lead to imprecise and therefore misleading results.

STORAGE OF BLOOD SAMPLES

After collection, the needle should be removed and the blood gently deposited into a 0.5 to 1.0 ml commercially available pediatric blood storage tube containing the anticoagulant agent ethylenediamine tetra-acetic acid (EDTA) (1.5 mg/ml of blood) or lithium heparin (1.8 mg/ml of blood). Squirting the sample through the needle is a poor practice, as it may cause severe disruption to the fragile blood cells. For general hematology analysis, EDTA is the anticoagulant of choice, as it is not possible to estimate fibrinogen or to count white blood cells accurately in heparinized samples.

In some avian species, however, storing blood samples in tubes containing EDTA causes progressive red cell hemolysis and is not recommended; in these cases, it is preferable to use heparinized tubes. This is the case with some species of Corvidae such as the jackdaw

Figs 22.1d-h | Small Birds - Basilic Vein

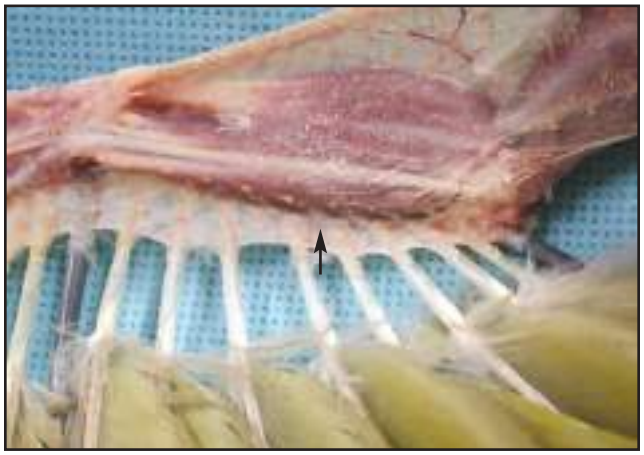


Fig 22.1d | The ventral surface of the wing of a cadaver with the covert feathers removed to show the location for superficial ulnaris vein lancing (arrow). Lancing the basilic vein in birds under 100 gms avoids subcutaneous hematomas and the possibility of death due to exsanguination. The site has a series of natural depressions over the vein that serve to allow the blood to pool. The depressions are formed by the insertion of the secondary feathers intermittently elevating and depressing the wing dermis just caudal to the border of the *flexor* carpi *ulnaris* muscle.

Fig 22.1f | Lancet with the cap removed and the 1 mm tip exposed.

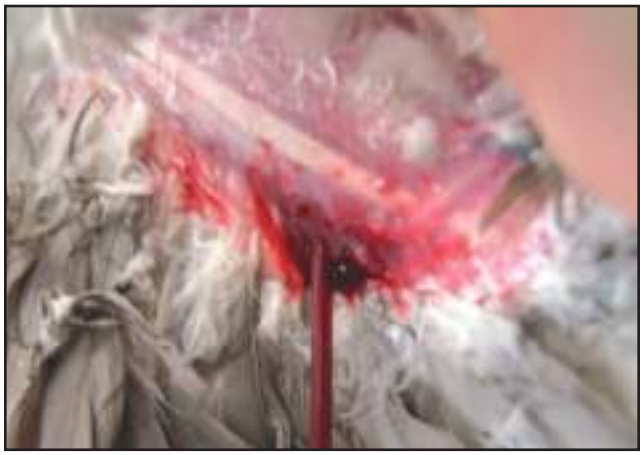


Fig 22.1h | The superficial ulnaris vein has been lanced and blood is being drawn into a micro capillary tube. A cotton ball is applied to the site until hemostasis is achieved.

Fig 22.1e | Lancet used in a finger stick technique for blood sampling for glucose analysis in humans. The lancet still has the metal tip in place in the plastic cap.

Fig 22.1g | The lancet readied at the site to lance the superficial ulnaris vein.

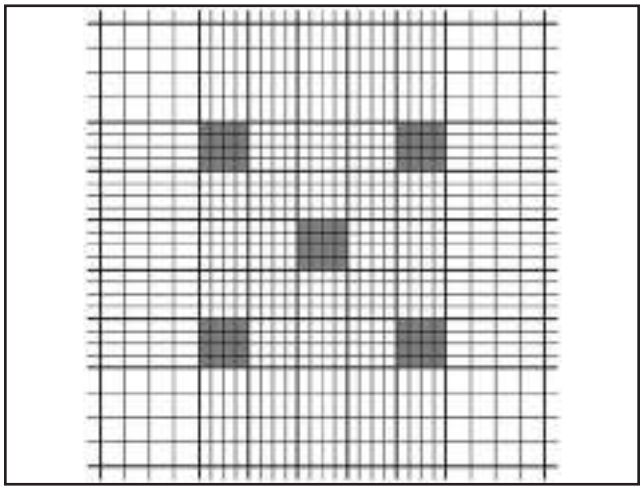


Fig 22.2a | The improved Neubauer counting chamber and the method for counting red blood cells. The total red blood cell count is performed by counting the number of cells contained in the 25 groups of 16 small squares (shaded) at the 4 corner squares and center square in the central area of the chamber. Closely ruled triple lines (illustrated in the drawing as thick lines) separate these squares.

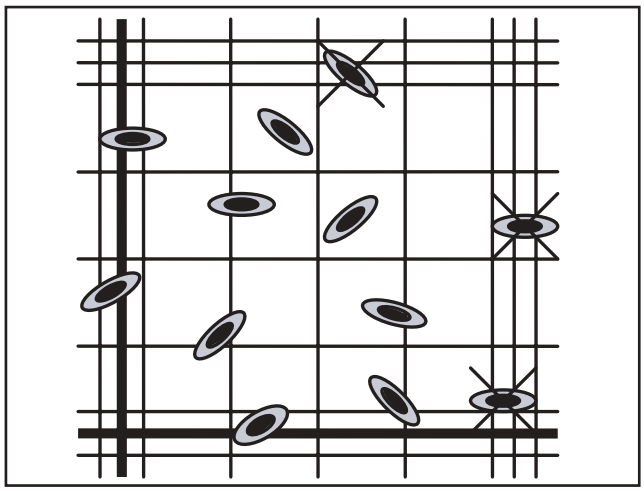


Fig 22.2b | The counting system. Count cells that touch the center triple line (seen here as a thick line) of the rules to the left and bottom; do not count cells that touch the center triple line of the rules to the right and top.

Table 22.2 | Special Considerations When Submitting Blood Samples to a Laboratory

- Seal the lid of the tube using waterproof tape in order to prevent any leakage.
- Wrap the tube using an absorbent packing material, eg, cotton, to soak up any potential leakage and to protect it from breakage. Fasten it securely with tape, preferably commercially available printed tape with the legend "Pathological specimen. Fragile handle with care" or similar tape.
- The tube should then be placed within two leak-proof plastic bags. Fasten the double wrapping securely with printed tape.
- Place submission form in a separate plastic bag.
- The package should then be placed within a postal-approved commercially available transport container made of aluminum, polystyrene, plastic or cardboard.
- Attach recipient and sender labels directly to the container using tape, or place container within a padded envelope and address it accordingly.

Table 22.3 | Priorities When Processing Hematology Samples

- Blood film (differential, white and red cell morphology, hemoparasites)
- Packed cell volume (PCV)
- White cell count (WBC)
- Hemoglobin (Hb)
- Red cell count (RBC)
- Fibrinogen

(*Corvus monedula*) and raven (*Corvus corax*); Gruidae such as the black-necked crowned crane (*Balearica pavonina*) and gray-necked crowned crane (*Balearica regulorum*); Cracidae such as the black curassow (*Crax alector*); Phasianidae such as the brush turkey (*Alectura lathami*); Bucerotidae such as the crowned hornbill (*Tockus alboterminatus*); and the ostrich (*Struthio camelus*).[3,25] Storing blood samples in sodium citrate tubes is recommended when sending samples to a commercial laboratory for processing using laser flow cytometry.[14,19]

Commercially available collection tubes usually have printed labels. A pencil or ballpoint pen is used to enter the date and identification of the bird, preferably prior to filling the tube with the collected blood sample. Always remember the rule of thumb in clinical pathology: label tubes, not lids.

TRANSPORTATION OF BLOOD SAMPLES

In avian practice, hematology samples are commonly sent to commercial laboratories for processing (Table 22.2). Therefore, it is essential to be familiar with and to submit samples in full compliance with current local mail and courier regulations.

Hematology Laboratory Analysis

Although the hematology laboratory analysis described in this chapter were developed primarily for testing human blood and are in full compliance with the recommendations of the International Committee for Standardization in Hematology,[34] these have been adapted and used successfully in avian hematology. Ideally, laboratory analysis should be carried out within 3 to 4 hours after collection. Many laboratories in the USA request that a smear be made immediately and sent along with the EDTA tube. If this is not possible, samples should be refrigerated at 8 to 12° C or within a suitable container for processing within 24 to 48 hours. Refrigerated samples are not ideal for hematology testing, as the cells invariably suffer some changes. Only an experienced hematologist would be able to differentiate these changes from true hemoresponses to particular medical disorders. Samples should not be exposed to extreme environmental conditions or excessive shaking, as this will affect the quality of the sample. Any form of mouth pipetting with a Thoma pipette or any other pipette with or without tubing is not acceptable within clinical laboratory practices.

The amount of blood available for testing from small birds (eg, <80 g) is very often limited, making it impossible to carry out a full range of analyses. The clinician should bear this in mind and request the analysis in order of priority (Table 22.3).

THE TOTAL RED BLOOD CELL COUNT (RBC X 10^{12}/L)

The total red blood cell count is in itself an important hematology assay, but it also is essential for the estimation of mean corpuscular volume (MCV) and mean corpuscular hemoglobin (MCH). Many laboratories prefer to estimate RBC using an automatic system, as this is more precise than manual methods. The materials, solutions and method described in Table 22.4, together with Fig 22.2a,b, apply to a manual technique.

HEMOGLOBIN ESTIMATION (Hb g/dl)

In avian species, estimation of hemoglobin is hampered by the presence of nuclei in the erythrocytes. Hemoglobin estimation relies on the colorimetric measurement of hemoglobin released after the lysing of the erythrocytes. Hemoglobin can be estimated using automatic methods or manual methods (Table 22.5). Commercial laboratories that estimate hemoglobin using an automatic hematology analyzer have to take into consideration the photometric interference of the free nuclei after lysing of the erythrocyte. In the manual method, it is essential to remove the nuclei from the preparation because its presence could yield unreliable results. The nuclei can be deposited by low-speed centrifugation, but because some hemoglobin remains attached to the nuclei, colorimetric readings are commonly low. This can be overcome by estimating hemoglobin as cyanmethemoglobin using alkaline Drabkin's cyanide-ferricyanide solution or as oxyhemoglobin using ammonia solution. In both cases, the estimation is carried out using a spectrophotometer at the absorbance reading of 540 nm. A calibration graph should be made using commercially available hemoglobin standards to express hemoglobin as oxyhemoglobin. Conversely, hemoglobin can be estimated directly as oxyhemoglobin using a commercially available hemoglobinometer. This is the preferred method used and recommended by the author.

PACKED CELL VOLUME ESTIMATION PCV % (HEMATOCRIT Hct L/L)

Packed cell volume (PCV) is an important hematologic assay because it provides an easy and objective way of estimating the number of erythrocytes in the sample. It also is essential for the calculation of the mean corpuscular volume (MCV) and mean corpuscular hemoglobin concentration (MCHC). In avian species, PCV is best estimated using the microhematocrit method described (Fig 22.3). The use of plain microcapillary tubes is preferable, since the same tube can be subsequently used to estimate fibrinogen.

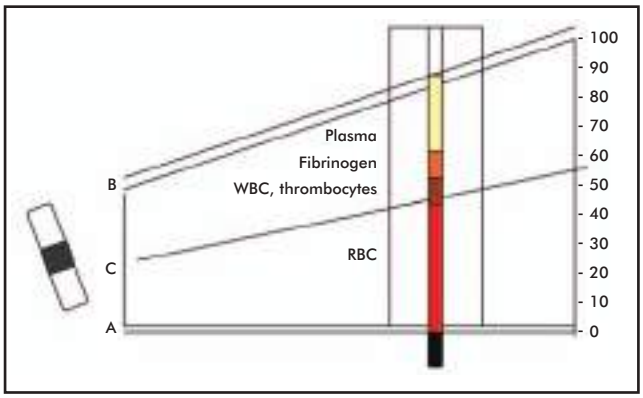


Fig 22.3 | Hematocrit reader and method for estimating packed cell volume. After centrifugation, position the capillary tube on the rack. Align tube (at the bottom, with the demarcation line between the sealing compound and the red blood cells) with line A. Slide the rack to the right or to the left, align the marginal meniscus at the top of the plasma column with line B. Position line C at the interface of the buffy layer and red cells, and read value on the scale.

Mean Corpuscular Values (Red Cell Absolute Values)

Mean corpuscular volume (MCV)

Mean corpuscular volume (MCV) is the expression of the average volume of individual erythrocytes calculated with the following formula:

MCV = (PCV x 10)/RBC = MCV femto liters (fl)

Mean corpuscular hemoglobin (MCH)

Mean corpuscular hemoglobin (MCH) is the expression of the average hemoglobin content of a single erythrocyte calculated with the following formula:

MCH = (Hb x 10)/RBC = MCH picogram (pg)

Mean corpuscular hemoglobin concentration (MCHC)

Mean corpuscular hemoglobin concentration (MCHC) is the expression of the volume within the erythrocyte occupied by the hemoglobin and is calculated with the following formula:

MCHC = (Hb x 100)/PCV = MCHC (g/L)

TOTAL WHITE BLOOD CELL COUNT (WBC X 10^{9}/L)

The total white blood cell count (WBC) is one of the most important hematology assays in the assessment of health and disease in an individual. The WBC also is useful because it is used together with the differential white cell count to calculate the absolute number of each white blood cell within a blood sample. The materials, solutions and method described below, Table 22.6 and Fig 22.4, apply to the technique.

THE BLOOD FILM

The examination of stained blood films is the single most important assay in hematology analysis. An adequately

Table 22.4 | Manual Total Red Blood Cell (RBC) Count Hematology Test

Materials and Equipment

- Automatic dispenser, 0-50 ml
- Disposable sample tube with lid, 5 ml
- Micropipette, 20 µl and corresponding tip
- Roller mixer
- Plain capillary tubes
- Improved Neubauer hemocytometer and coverslip
- Laboratory lens tissue
- Petri dish 8.5 cm diameter
- Filter paper 8.5 cm diameter
- Toothpick
- Distilled water
- Microscope, preferably with phase contrast capability

Test Systems

The Unopette 365851 system[c] is probably the most popular method used for manual red blood cell count in avian species. It uses 10 µl of whole blood in 1.9 ml of 0.85% saline, resulting in a 1:200 dilution. The two other commonly used systems are based on using either formol citrate solution (Dacie's fluid) or Natt and Herrick's solution, depending on whether the examination is carried out with or without phase contrast microscopy. Dacie's formol citrate solution is the least known diluting fluid, but one used and recommended by the author.

Working Solutions

1. BD Unopette 365851[c] red blood count manual hematology test

2. Natt and Herrick's solution
   (for use without phase contrast microscopy)

NaCl	3.88 g
Na_2SO_4	2.5 g
Na_2HPO_4 12 H_2O	2.91 g
KH_2PO_4	0.25 g
Formaldehyde 40%	7.5 ml
Methyl violet 2B	0.1 g
Distilled water	to 1000 ml

Note: Allow solution to stand overnight. Filter before use.

3. Formol citrate solution or Dacie's fluid
   (for use with phase contrast microscopy)

Formaldehyde 10%	10 ml
Trisodium citrate	31.3 g
Distilled water	100 ml

Note: Refrigerate at 8 to 12° C.

Method[c]

- Label sample tubes using a permanent marker.
- Use an automatic dispenser to transfer 4 ml of either formol citrate solution or Natt and Herrick's solution into sample tube.
- Wait for 5 minutes to allow working solution to reach room temperature.
- Aspirate 20 µl of whole blood from storage tube using micropipette, wipe side of pipette tip carefully using tissue and dispense on the side of sample tube to make a dilution of 1:200.
- Avoid touching the distal opening of the pipette tip with the tissue, as this will cause capillary shift of blood into the tissue.
- Avoid immersing the pipette tip into the diluting fluid. This is a poor laboratory practice.
- Place sample tube in roller mixer and wait for 3 minutes.
- Clean Neubauer hemocytometer and coverslip using a dry, lint-free cloth or laboratory lens tissue.
- Place coverslip onto hemocytometer and slide gently over it, making sure Newton's rings (colored interference pattern) appear on both sides of the contact surfaces.
- Withdraw a small aliquot of the diluted sample using a plain capillary tube.
- Fill up one side of the hemocytometer by touching gently the intersection between coverslip and hemocytometer with the loaded capillary tube. Avoid air bubbles and underfilling or overfilling.
- Place filter paper at the bottom of the Petri dish. Position two toothpicks on either side of the dish. Wet filter paper lightly with distilled water. Rest hemocytometer on toothpicks. Cover Petri dish. Leave for 5 minutes for the cells to settle down.
- The hemocytometer is now ready for use.
- Count cells contained in the four corner and central squares in the mid section of the hemocytometer. Following the "L" rule: count cells that touch the center triple line of the ruling on the left and the bottom sides; do not count cells that touch the center triple line of the ruling on the right and the top sides (see Figs 22.2a,b).
- Calculate red blood cell count using:
  $N/100 = RBC \times 10^{12}/L$

Note: N = number of cells counted in 160 small squares.

Table 22.5 | Hemoglobin Estimation

Materials and Equipment

- Automatic dispenser, 0-50 ml
- Disposable sample tube with lid, 5 ml
- Micropipette, 20 µl and tip
- Roller mixer
- Toothpicks
- Cuvette, 10 mm²
- Laboratory lens tissue
- Hemoglobinometer

Working Solution

Ammonia solution

Ammonia solution (0.88 specific gravity)	4 ml
Distilled water	to 1000 ml

Note: Refrigerate at 8 to 12° C.

Method

- Label sample tubes using a permanent marker.
- Use an automatic dispenser to transfer 4 ml of ammonia solution into sample tube.
- Wait for 5 minutes to allow working solution to reach room temperature.
- Aspirate 20 µl of whole blood from storage tube using micropipette, wipe side of pipette tip carefully using tissue and dispense on the side of sample tube.
- Avoid touching the distal opening of the pipette tip with the tissue, as this will cause capillary shift of blood into the tissue.
- Avoid immersing the pipette tip into the diluting fluid. This is a poor laboratory practice.
- Place sample tube in roller mixer and wait for 3 minutes.
- Decant approximately 3.5 ml of the diluted blood into cuvette.
- Remove cell nuclei jelly using toothpicks.
- Do not touch the clear reading walls of the cuvette with bare fingers.
- Clean clear reading walls of cuvette using laboratory lens tissue.
- Zero hemoglobinometer using ammonia solution as blank.
- Reading expressed as Hb g/dl.

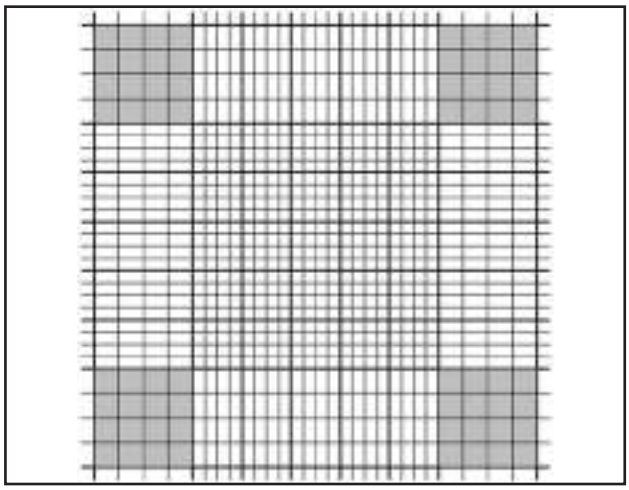


Fig 22.4 | Improved Neubauer counting chamber and the method for counting white blood cells. The total white blood cell count is performed by counting the number of cells contained in 4 groups (shaded areas) of 16 large squares at the four corner squares of the chamber. Closely ruled triple lines (illustrated in the drawing as thick lines) separate these squares.

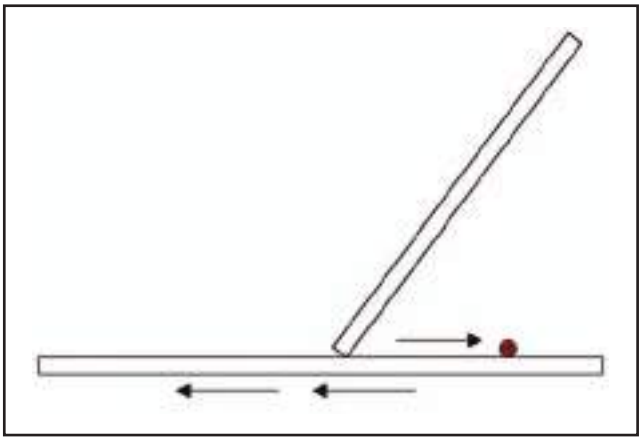


Fig 22.5 | The slide-to-slide technique. Move spreader slide backward to gently touch the drop of blood, allowing it to run across the edge of the slide. Move forward to make smear. Move slowly if blood runs slowly, move quickly if blood runs quickly.

prepared blood film provides the differential white blood cell count and absolute white blood cell count, the thrombocyte count and the hemoparasite examination.

Preparation of the Blood Film

Blood films can be made from a drop of fresh, non-anticoagulated blood directly from the tip of the syringe. Conversely, films can be made from blood stored in EDTA within 2 to 3 hours after collection. There are two generally accepted methods for the preparation of blood films in hematology: the slide-to-slide technique (Fig 22.5, Table 22.7) and the coverslip-to-slide technique (Fig 22.6, Table 22.8). A two cover-slip technique is not described here. The most popular method among avian clinicians is the coverslip-to-slide technique, as smudging of blood red cells is generally minimized.

Fixation and Staining of the Blood Film

It is commonly accepted that blood films can be prepared and be fixed and stained at a later date. This is incorrect; blood films should at least be fixed immediately after preparation, particularly if made in a hot and humid environment or under cold and freezing conditions. Blood films should not be exposed to direct sunlight, moisture of any kind or vapor from chemicals (formaldehyde in particular), as this would invariably affect cell morphology.

Table 22.6 | Total White Blood Cell (WBC) Count Hematology Test

Materials and Equipment

- Automatic dispenser, 0-50 ml
- Disposable sample tube with lid, 5 ml
- Micropipette, 100 µl and tip
- Roller mixer
- Plain capillary tubes
- Improved Neubauer hemocytometer and coverslip
- Laboratory lens tissue
- Petri dish, 8.5 cm diameter
- Filter paper, 8.5 cm diameter
- Toothpicks
- Distilled water
- Microscope, preferably with phase contrast capability

Test Systems

The Unopette 365877[d] system was originally developed for the estimation of eosinophils in human hematology, but it has proved useful for determining the total white cell count in avian species. This system uses 25 µl of whole blood into 0.775 ml of 1% Phloxine B diluent resulting in a 1:32 dilution, and is the system used by most practitioners in the USA.[3,19] The method described below is based on the use of ammonium oxalate solution, which is the method used and recommended by the author.

Working Solutions

1. BD Unopette 365877[d] eosinophil count manual hematology test[d]
2. Ammonium oxalate solution 1%

Ammonium oxalate	10 g
Distilled water	to 1000 ml

Note: Refrigerate at 8 to 12° C.

Method

- Label sample tubes using a permanent marker.
- Use an automatic dispenser to transfer 1.9 ml of 1% ammonium oxalate solution into sample tube.
- Wait for 5 minutes to allow working solution to reach room temperature.
- Aspirate 100 µl of whole blood from storage tube using micropipette, wipe side of pipette tip carefully using tissue and dispense on the side of sample tube.
- Avoid touching the distal opening of the pipette tip with the tissue, as this will cause capillary shift of blood into the tissue.
- Avoid immersing the pipette tip into the diluting fluid. This is a poor laboratory practice.
- Place sample tube in roller mixer and wait for 3 minutes.
- Clean Neubauer hemocytometer and coverslip using a laboratory lens tissue or dry, lint-free cloth.
- Place coverslip onto hemocytometer and slide gently over it, making sure Newton's rings (colored interference pattern) appear on both sides of the contact surfaces.
- Withdraw a small aliquot of the diluted sample using a plain capillary tube.
- Fill up one side of the hemocytometer by gently touching the intersection between coverslip and hemocytometer with the loaded capillary tube. Avoid air bubbles and underfilling or overfilling.
- Place filter paper at the bottom of the Petri dish. Position two toothpicks on either side of the dish. Lightly wet filter paper with distilled water. Rest hemocytometer on toothpicks. Cover Petri dish. Leave for 5 minutes for the cells to settle down.
- The hemocytometer is now ready for use.
- Count cells contained in the four outer large squares of the hemocytometer.
- Calculate total white blood cell count using:
  $N/20 = WBC \times 10^9/L$

Note: N = number of cells counted in 64 small squares.

Table 22.7 | Method for Slide-to-Slide Technique

- It is highly recommended to use one-end-frosted microscopic slides to easily note the ID of the sample on the slide using a pencil.
- Wipe slides clean with a lens tissue or lint-free cloth.
- Use a plain microcapillary tube to withdraw a small amount of fresh, non-anticoagulated blood directly from syringe tip or EDTA tube.
- Place a small drop of blood (2 µl) at one end of a slide.
- Select a spreader slide and position it in front of the drop of blood at about a 45° angle. The selected slide should be free from any indentation. To test this, pass the spreading edge over the edge of a fingernail.
- Gently move the spreader slide backward to touch the drop of blood and allow the blood to run across the edge of the slide.
- Gently drive the slide forward with a steady but firm movement to create a uniform smear.
- It is always a good practice to make two good-quality blood films.

Table 22.8 | Method for Coverslip-to-Slide Technique

The only significant difference between this method and the previous one consists of the following steps:

- Place a large rectangular coverslip over the drop of blood.
- Pull the coverslip and the slide in opposite directions in a steady but firm movement to create a uniform smear.

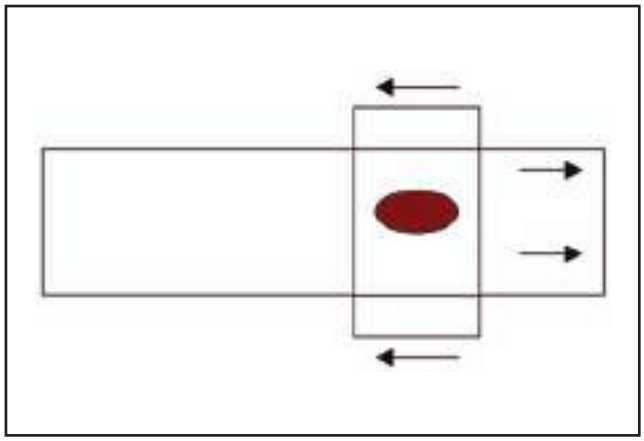


Fig 22.6 | The coverslip-to-slide technique. Place coverslip onto drop of blood. Apply gentle pressure downward. Move slide and coverslip in opposite directions to make smear.

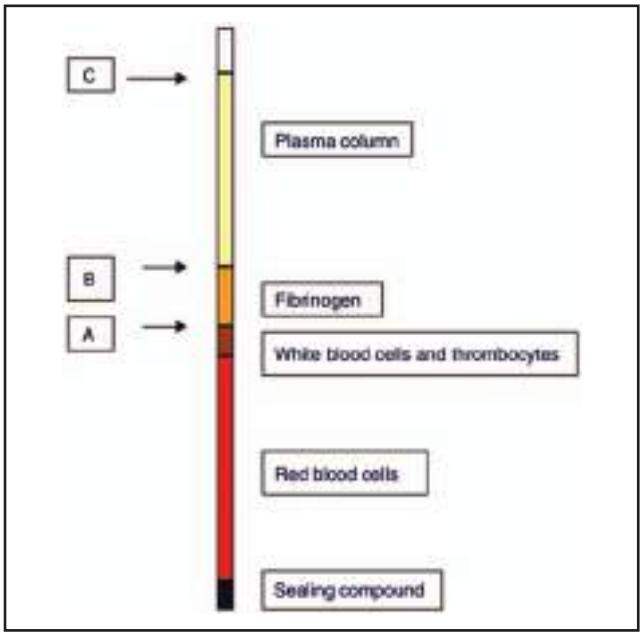


Fig 22.7 | A microcapillary tube after incubation and centrifugation, and the measurements necessary for the estimation of fibrinogen.

Fixation

In general, freshly prepared blood films should be immersed in absolute methanol within a Coplin jar for 5 to 10 minutes immediately after preparation. Fixed blood films can then be stored within commercially available slide storage boxes (eg, under field conditions) and be stained at a later date. Blood films also can be stained immediately after fixation.

The importance of adequate fixation of blood films from avian species cannot be overemphasized. The intracytoplasmic granules of the heterophils and basophils are water soluble; therefore, blood films should be adequately fixed before staining in order to preserve the integrity of these structures. A significant problem in avian hematology is the presence of smudged red cell nuclei as a consequence of hemolysis in poorly fixed blood films. This is one of the main reasons why clinicians and commercial laboratories are now inclined to use stains that are prepared in absolute methanol (eg, Wright-Giemsa stain, Leishman stain) and are used at full strength so films are fixed and stained at the same time. If absolute methanol within a Coplin jar is used for fixation in your laboratory, it must be replaced as soon as it begins showing chemical fatigue. This would depend on the number of slides fixed and the environmental conditions within the laboratory.

Staining

Most Romanovsky stains used for staining human and mammalian blood films are suitable for staining avian blood films. However, the results obtained with various stains may be slightly different, and the selection of stains is generally accepted as a matter of personal preference. Commonly used stains include Wright stain, Giemsa stain, Wright-Giemsa stain, Leishman stain, Wright-Leishman stain, May-Grünwald stain and May-Grünwald-Giemsa stain. In the author's opinion, rapid stains on their own, eg, Diff Quick and Rapid Diff, do not produce adequate quality for the differentiation of subtle blood cell structures and those of hematozoa. This is particularly important with respect to the morphological characteristics of the granulocytes.

Automatic slide stainers facilitate staining a relatively large number of blood films at the same time, producing consistent results and eliminating variations that may occur with manual techniques. However, this type of equipment is relatively expensive to purchase and maintain and is more appropriate for high-volume commercial laboratories.

It is important that clinicians or laboratory technicians recall the basic principles of hematology when staining blood films. The pH of the stains should be checked each time new stock is prepared. Some stains, particularly those prepared from powder, should be adequately filtered. Glassware should be properly washed, rinsed with distilled water and dried thoroughly before use. Many of the common artifacts on blood films are due to careless preparation and improper methodology.

The staining method currently used and recommended

Table 22.9 | Wright-Giemsa Staining Procedure

Working Stain

- 3 g Wright stain powder
- 0.3 g Giemsa stain powder
- 5 ml glycerol
- To 1000 ml absolute methanol (acetone free)
- Filter and store

Method

- Prepare thin blood smears.
- Place on staining rack.
- Flood smear with Wright-Giemsa stain, allow to stand for 3 minutes.
- Add equal amount of Sørensen's pH 6.5-6.8 buffer, depending on batch stain.
- Mix gently by blowing using a pipette until metallic green sheen forms on the surface, allow to stand for 6 minutes.
- Rinse with buffer, allowing to stand for 1 minute for differentiation.
- Wash copiously with buffer.
- Wipe the back of smear with tissue to remove excess stain.
- Prop in rack until dry.

by the author is a slightly modified technique[4] described in Table 22.9.

The placement of a coverslip using a commercially available mounting medium over the blood smear is optional. Additionally, the mounting of blood films offers several advantages such as preventing scratching during transport, protection against damage during excessive manipulation (eg, teaching material) and enhancing visualization for optimal examination and photography.

Morphologic and Staining Characteristics of Red Blood Cells, White Blood Cells and Thrombocytes

Normal red blood cells appear elliptical and have elliptical nuclei; the cytoplasm stains uniformly eosinophilic, and the nuclei is dark purple in color (modified Wright-Giemsa stain).

In general, the widely known "Romanovsky stains" contain blue azure that reacts with acid groups, including those of nucleic acids and proteins of the nucleus and cytoplasm and eosin Y, which has a particular affinity for basic groups of hemoglobin. When used in different avian species, the slight variations observed may be the result of true species diversity or simply variations in the materials and methods used from individual to individual or from laboratory to laboratory.

Adequate knowledge of the morphology and staining characteristics of the different blood cells is of the utmost importance for the differentiation and classification of those blood cells (Table 22.10 and Figs 22.8-22.43).

Table 22.10 | Morphologic and Staining Characteristics of Different Blood Cells

Blood Cell	Morphologic Characteristics	Staining Characteristics
Erythrocyte	**Mature cells**	
	Medium size, oval elongated shape, central oval elongated nucleus	Cytoplasm: uniform pale orange to red-pink; Nucleus: purple-red, condensed, clumped chromatin
	Immature cells	
	Smaller than mature cell, round to semi-oval, relatively larger nucleus	Polychromatic, cytoplasm pale to dark blue
Heterophil	Medium size, round shape, bilobed nucleus	Colorless cytoplasm, rod- to cigar-shaped brick red to pale blue granules
Eosinophil	Medium size, round shape, bilobed nucleus	Pale blue cytoplasm, round to oval brick red to pale blue granules
Basophil	Small size, round shape, unlobed nucleus	Pale blue cytoplasm, variable number of small, medium and large dark red-purple granules
Lymphocyte	Small to medium size, typically round to triangular shape, centrally positioned large round nucleus; in general, 25 cytoplasm:75 nucleus; ratio, coarsely condensed to highly condensed chromatin	Pale blue cytoplasm
Monocyte	Large size, typically round shape, eccentrically positioned kidney-shaped nucleus; in general 75 cytoplasm:25 nucleus ratio, cytoplasm lace-like appearance, often medium size vacuoles, coarsely condensed chromatin	Cytoplasm pale blue to pale gray
Thrombocyte	Small, oval to rectangular shape, nucleus oval to rectangular	Cytoplasm colorless to pale blue, large vacuoles, nucleus highly condensed dark purple-red chromatin

Differential White Blood Cell and Absolute White Blood Cell Count

For the differential white blood cell count and absolute white blood cell count, the film should be examined thoroughly under high-power magnification, under oil (1000x). The recommended topographic site is on the shoulder of the blood film. The *shoulder* is the edge of the oval-shaped end of a smear. This is the area where the blood cells are in one layer and are slightly segregated, thus facilitating examination.

In general terms, 100 white blood cells should be counted and classified according to the morphologic and staining characteristics. Counting is usually carried out using a commercially available manual or electronic differential cell counter. The differential white blood cell count is expressed as a percentage of the individual cell group. The percentage of each cell group is then converted into absolute numbers by reference to the total WBC using the following formula:

(Percentage of white blood cell counted x total WBC)/100 = absolute No. x 10^9/L

Thrombocyte Count

Thrombocytes are usually counted while performing the differential white blood cell count. Valid and reliable results cannot be obtained if there is evidence of thrombocyte clumping.

The absolute number of thrombocytes is estimated by using the following formula:

(No. of thrombocytes counted/100) x WBC = thrombocytes x 10^9/L

Figs 22.44-22.48 and Tables 22.12-22.18 are offered as references for interpreting hemotological findings.

FIBRINOGEN ESTIMATION (g/L)

Fibrinogen is a plasma protein essential for normal blood coagulation, but also is one of the acute reactive proteins that are detected in increased levels in association with medical disorders involving infection and inflammation (see Tables 22.11, 22.12 and 22.18).

Hemoparasite Examination

Hemoparasite examination is carried out on thin, good-quality blood films. Prior to a differential white cell count using high-power magnification (1000x), the blood film should be examined under low-power magnification (eg, 200x or 400x) in order to detect large extra cellular hemoparasites (eg, microfilariae), which could be missed if the film is examined only under high-power magnification. The examination under low-power magnification should concentrate on areas not commonly examined under high-power magnification, eg, head and tail of the blood smear. The blood film should be examined in full in a systematic way and following a consistent pathway.

A blood parasite quantitative assessment should be carried out, in certain cases and under certain circumstances, by examining 1000 red cells (in the case of intracytoplasmic parasitic forms) and determining the number of red cells containing hemoparasites (eg, *Haemoproteus* spp., *Babesia* spp.). The number is then expressed in percentage and this usually constitutes the degree of parasitemia.

If hemoparasites are observed during routine examination of the blood film under low- or high-power magnification, it is imperative to immediately prepare additional blood films. Ideally, a fresh blood sample should be obtained to prepare these new blood films from non-anticoagulated blood. This is of the utmost importance if a rare parasite is observed in the film. Blood films should be fixed but unstained when sending them to parasitologists, who have their own preferences for stains and staining procedures.

Table 22.11 | Fibrinogen Estimation

Materials and Equipment Needed for Fibrinogen Estimation

- Microcapillary tube rack
- Microhematocrit centrifuge
- Microhematocrit reader
- Water bath 56° C ± 1° C
- Microcapillary tube holder
- Microscope with measuring eyepiece and stage Vernier scale
- Timer

Method (following estimation of packed cell volume)

- Place microcapillary tube in tube rack.
- Place loaded rack in water bath at 56° C for 3 minutes (make sure the entire plasma column is immersed).
- Centrifuge microcapillary tubes again at 10,000 to 12,000 "g force" for 5 minutes.
- Place microcapillary tubes in tube holder and, using the microscope measuring eyepiece and the stage Vernier of the microscope, take reading at the upper and lower limits of the protein layer and at the upper limit of the plasma column (see Fig 22.7).
- Estimate fibrinogen with the following formula:
  (B - A)/(C - A) x 100 = fibrinogen g/L

Note: It is essential to perform this analysis on blood stored in EDTA because the analysis would be invalidated if performed on samples stored in heparin or on samples containing clots.

Unless specified otherwise, hematology images are from a saker falcon (*Falco cherrug*):

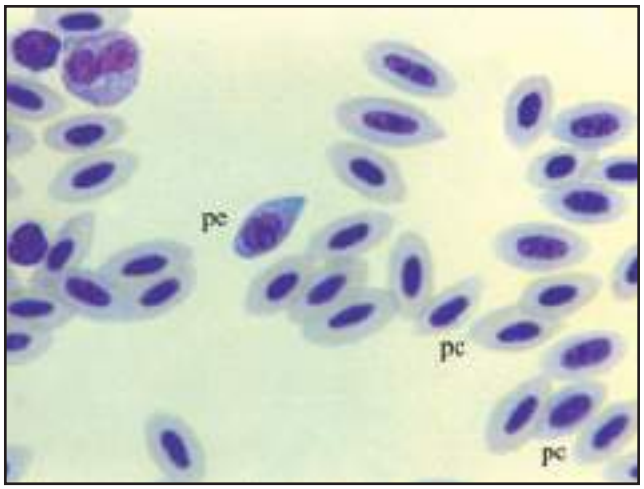


Fig 22.8 | Shown is a polychromatic erythroblast (pe) and poikilocytes (pc). The nuclear chromatin of the polychromatic erythroblast is clumped and the cytoplasm is highly basophilic (modified Wright-Giemsa stain).

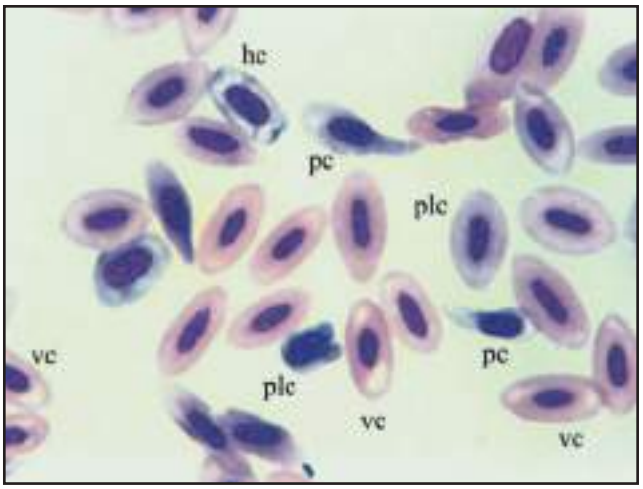


Fig 22.9 | The red cells in a bird with severe anemia show vacuolation (vc), hypochromia (hc) and polychromasia (plc). There are some poikilocytes (pc) in the smear (modified Wright-Giemsa stain).

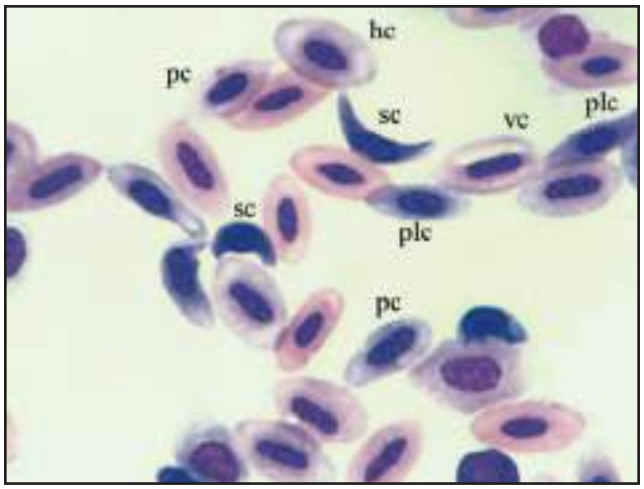


Fig 22.10 | The red cells in sickle cell anemia show sickling (sc), vacuolation (vc), hypochromia (hc) and polychromasia (plc). Some poikilocytes (pc) also are present (modified Wright-Giemsa stain).

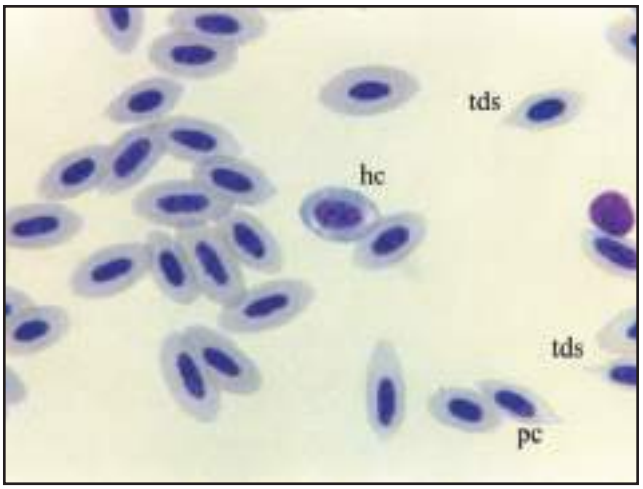


Fig 22.11 | Hypochromic (hc), teardrop-shaped red cells (tds) and poikilocytes (pc) are illustrated (modified Wright-Giemsa stain).

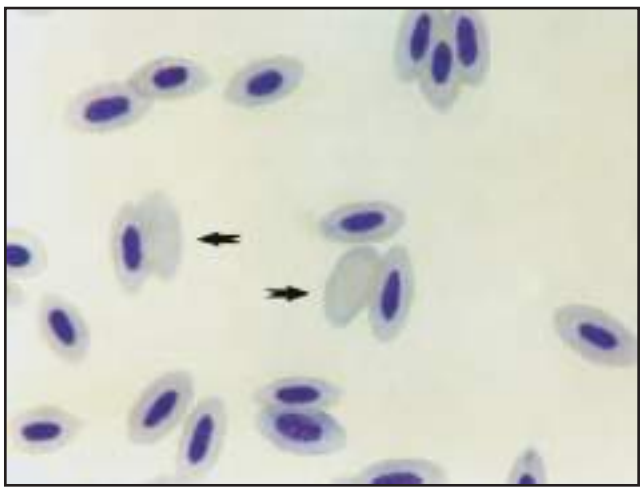


Fig 22.12 | Erythroplastid (arrows) forms (modified Wright-Giemsa stain).

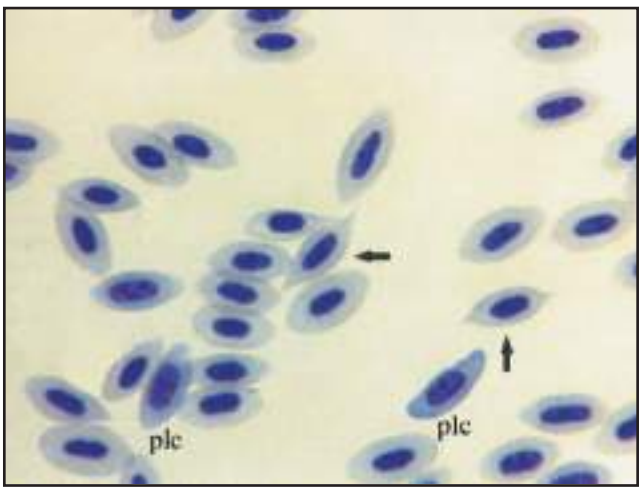


Fig 22.13 | Poikilocytes (arrows) are seen in metabolic defects and increased erythropoiesis. Polychromatic red cells (plc) are produced in response to severe blood loss. These are larger than normal cells (modified Wright-Giemsa stain).

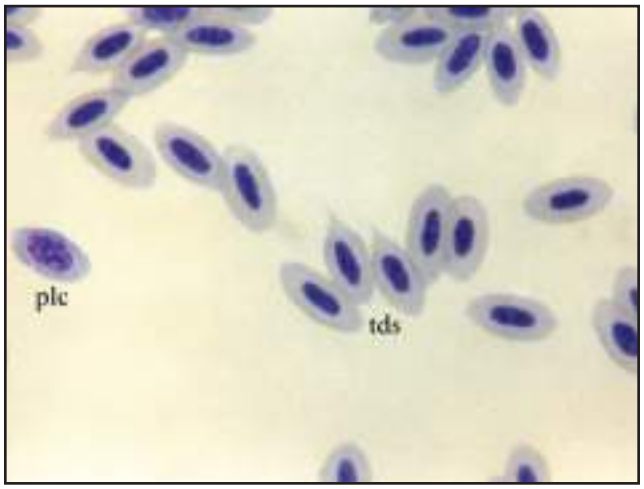


Fig 22.14 | Shown are teardrop-shaped red cells (tds) and polychromasia (plc). Teardrop-shaped cells are indications of toxicosis (May-Grünwald Giemsa stain).

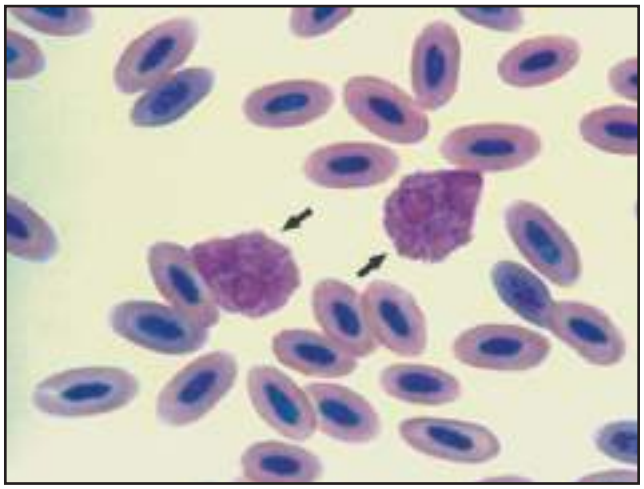


Fig 22.15 | Two normal heterophils (arrows). Heterophils are characterized by brick red, elongated intracytoplasmic granules and bilobed nuclei (modified Wright-Giemsa stain).

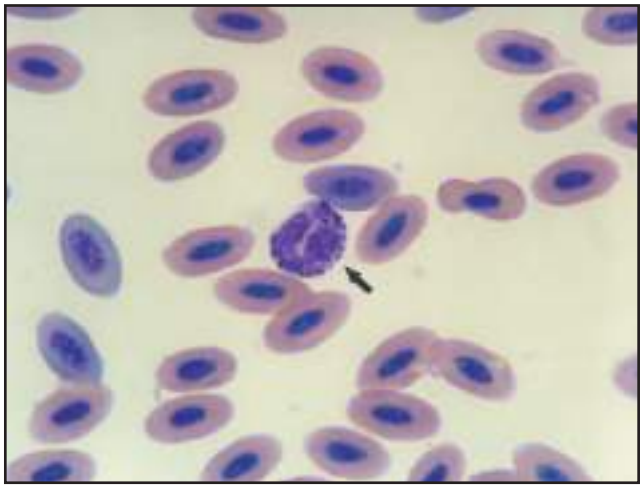


Fig 22.16 | In this eosinophil (arrow), note the numerous small and medium-sized, dark purple-colored granules located mainly in the periphery of the cytoplasm (modified Wright-Giemsa stain).

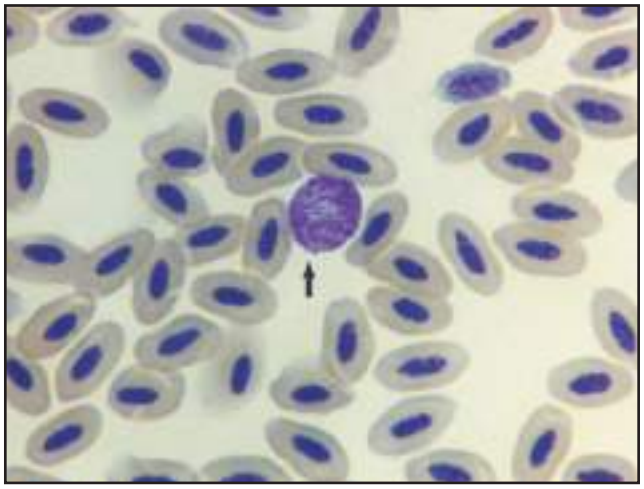


Fig 22.17 | An eosinophil (arrow) from an eclectus parrot (*Eclectus roratus*). Note the numerous small intracytoplasmic granules widespread across the cytoplasm. The granules stain dark purple in color (modified Wright-Giemsa stain).

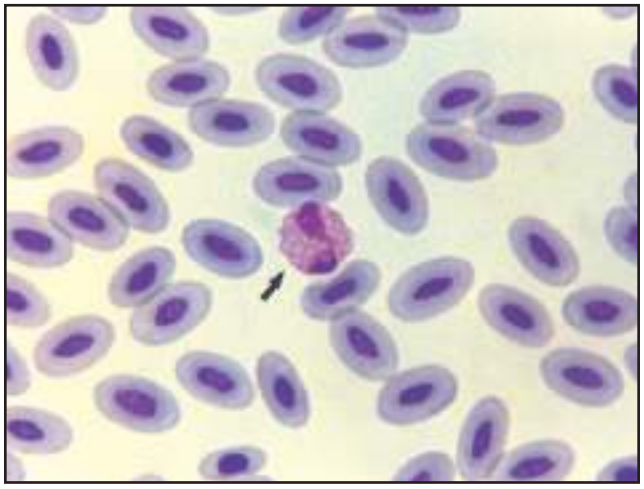


Fig 22.18 | An eosinophil (arrow) from a kori bustard (*Ardeotis kori*). Note the large, round, orange-colored granules characteristic of this species (May-Grünwald Giemsa stain).

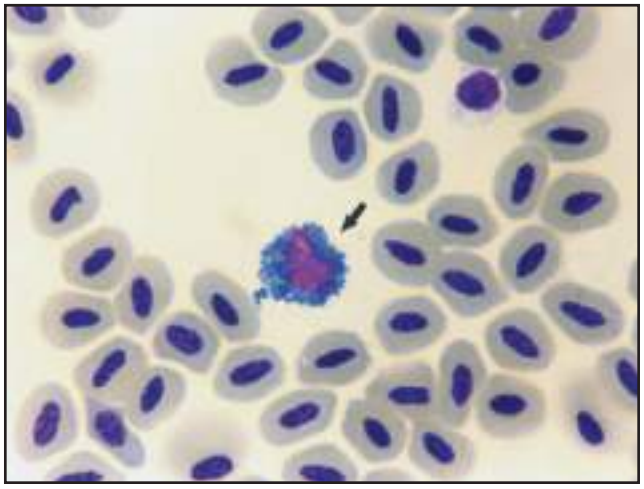


Fig 22.19 | A slightly disrupted eosinophil (arrow) from a lesser sulphur-crested cockatoo (*Cacatua sulphurea*). The medium-sized, round granules are blue in color (May-Grünwald Giemsa stain).

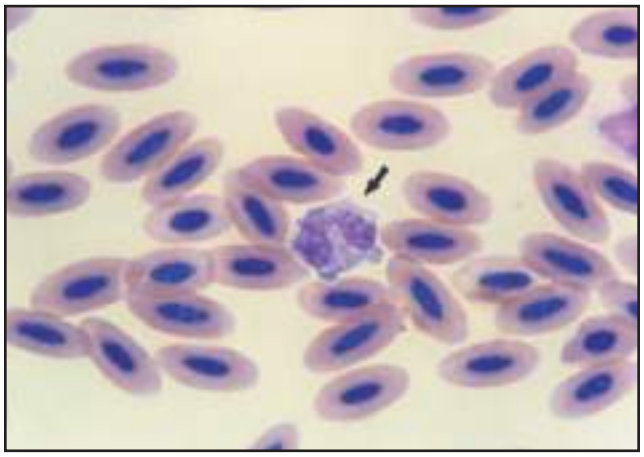


Fig 22.20 | In this eosinophil (arrow) from a saker falcon (*Falco cherrug*), the granules are not stained, giving the impression of numerous irregular vacuoles within the cytoplasm (May-Grünwald Giemsa stain).

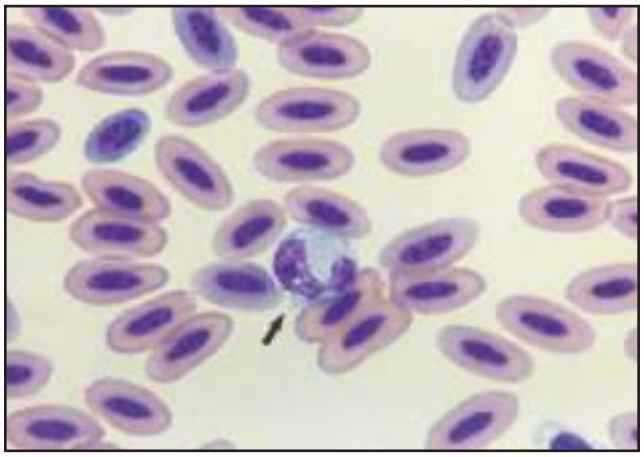


Fig 22.21 | In this eosinophil (arrow) is seen a similar-staining artifactual difference, as in the previous figure. The granules are not stained, giving the impression of numerous vacuoles within the cytoplasm (Diff Quik stain).

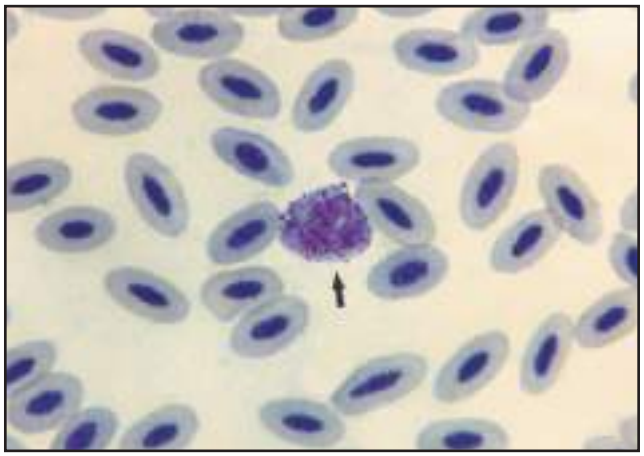


Fig 22.22 | These eosinophil (arrow) granules are well stained, irregular in shape and size, stained purple or dark purple (modified Wright-Giemsa stain). The author highly recommends the use of this stain for routine hematology.

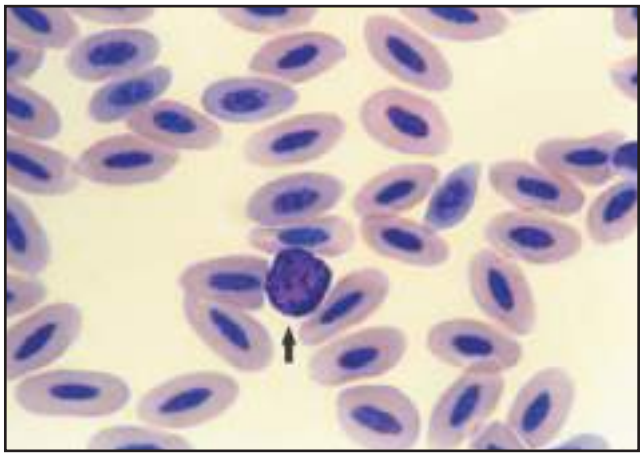


Fig 22.23 | A basophil (arrow) is characterized by the presence of large, round, dark purple granules widespread across the cytoplasm and an unlobed nucleus (modified Wright-Giemsa stain).

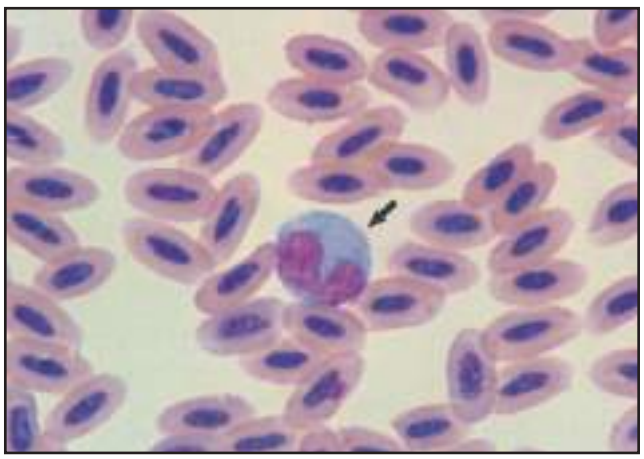


Fig 22.24 | A normal monocyte (arrow) is a relatively large cell with a kidney-shaped nucleus and abundant, slightly opaque, blue-gray, "lace-like" cytoplasm (modified Wright-Giemsa stain).

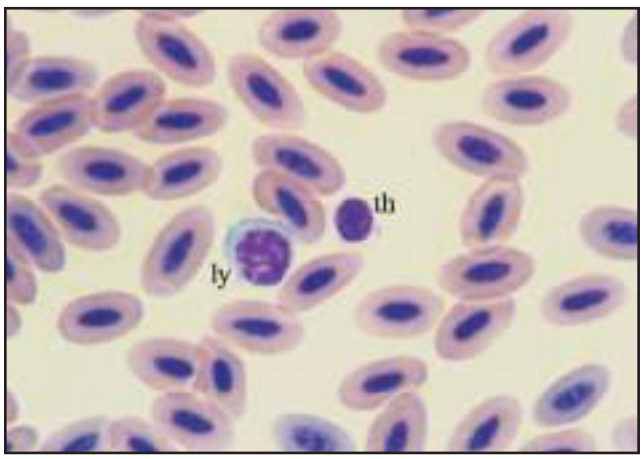


Fig 22.25 | A normal lymphocyte (ly) and a normal thrombocyte (th). Lymphocytes are regular round cells with a central or slightly eccentric nuclei, and with a varying amount of pale blue cytoplasm (modified Wright-Giemsa stain).

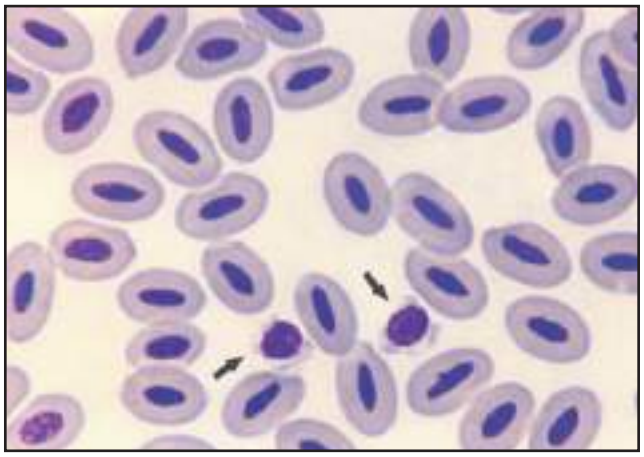


Fig 22.26 | Two normal thrombocytes (arrows) from a kori bustard (*Ardeotis kori*). Thrombocytes are round or irregular cells with completely dark purple and dense round or oval nuclei, and clear blue-gray cytoplasm. In some species, a few cytoplasmic projections can be observed. Sometimes it can be very difficult to differentiate between thrombocytes and small lymphocytes (May-Grünwald Giemsa stain).

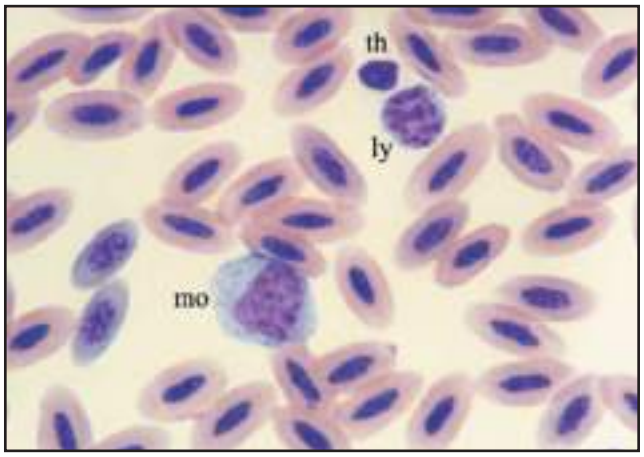


Fig 22.27 | Shown are a normal thrombocyte (th), normal lymphocyte (ly) and a normal monocyte (mo) for comparison of three different mononuclear cells. Thrombocytes and small lymphocytes can be very similar. In order to differentiate between them, the appearance of the nuclear chromatin has to be closely examined (modified Wright-Giemsa stain).

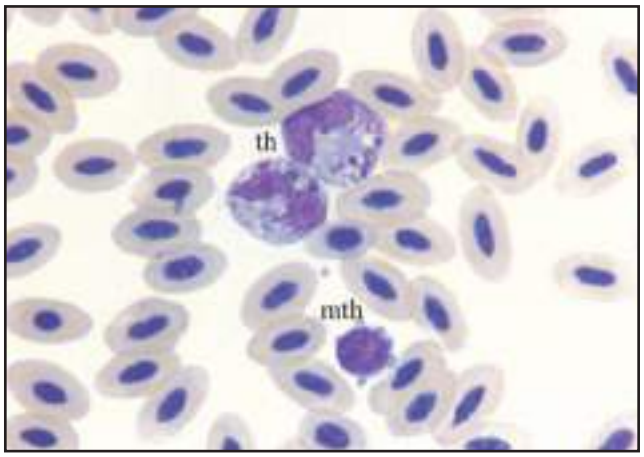


Fig 22.28 | Two toxic heterophils (th) and a megathrombocyte (mth). One of the heterophils shows a lack of lobulation of the nucleus (left shift); both show loss of granulation and the cytoplasm is stained basophilic. The megathrombocyte is significantly larger than a normal thrombocyte. The cytoplasm is basophilic, the nucleus cytoplasm ratio is increased and it has scalloped cytoplasmic margins (modified Wright-Giemsa stain).

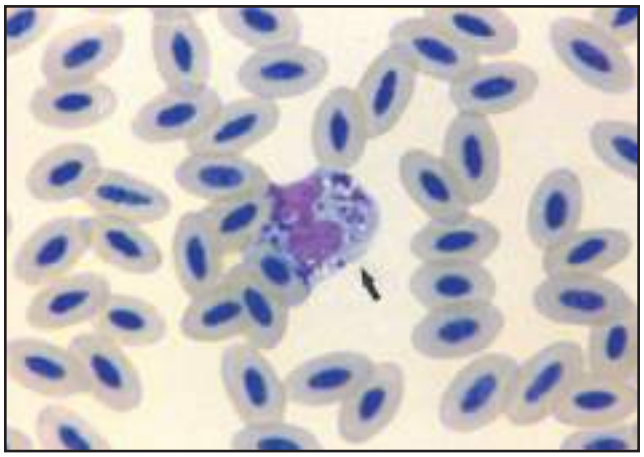


Fig 22.29 | A toxic heterophil (arrow) showing loss of nuclear lobulation (left shift) and loss of cytoplasmic granulation. The granules are round, large and stained dark purple, and the cytoplasm is basophilic (modified Wright-Giemsa stain).

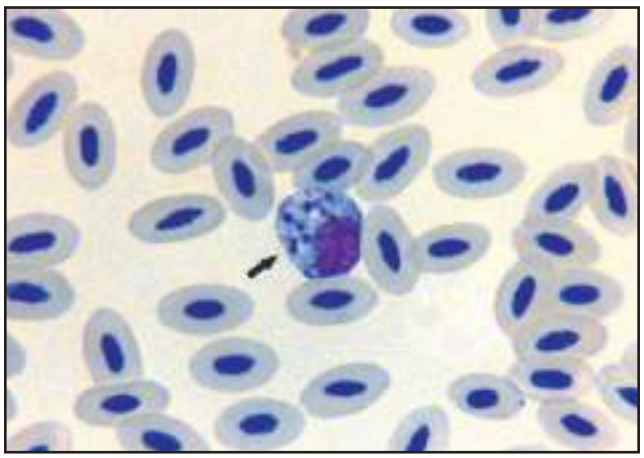


Fig 22.30 | A toxic heterophil (arrow) with a lack of nuclear lobulation (left shift) and loss of cytoplasmic granulation. Only a few large, round, dark purple granules are present and the cytoplasm is basophilic (modified Wright-Giemsa stain).

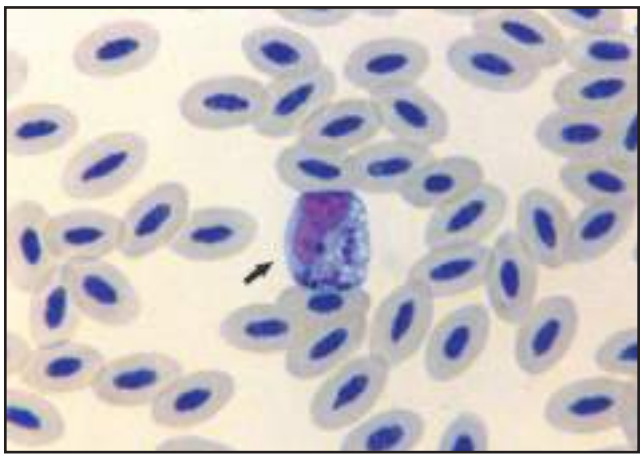


Fig 22.31 | A toxic heterophil (arrow). The heterophil shows loss of nuclear lobulation (left shift) and loss of cytoplasmic granulation. There are very few large, dark purple granules and the cytoplasm is basophilic (modified Wright-Giemsa stain).

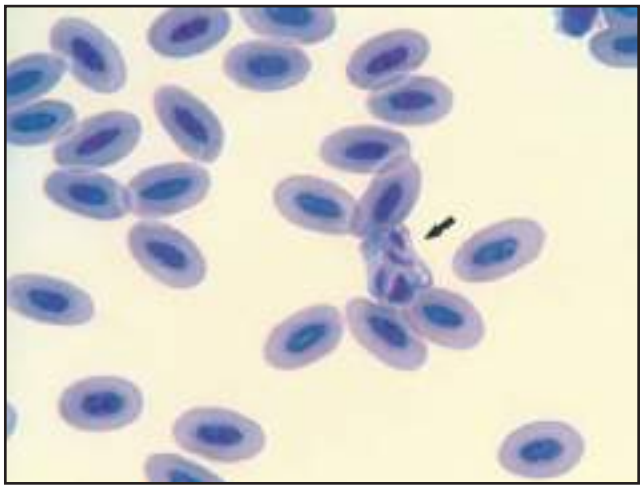


Fig 22.32 | A toxic heterophil (arrow). The nucleus is segmented into several fragments (right shift); the granules are not stained, giving the impression of numerous vacuoles within the cytoplasm (May-Grünwald Giemsa stain).

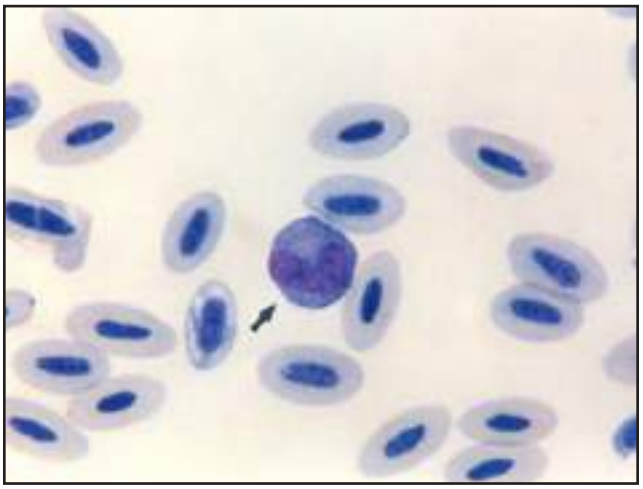


Fig 22.33 | A reactive monocyte (arrow). The cytoplasm is basophilic and the nuclear chromatin is coarse (modified Wright-Giemsa stain).

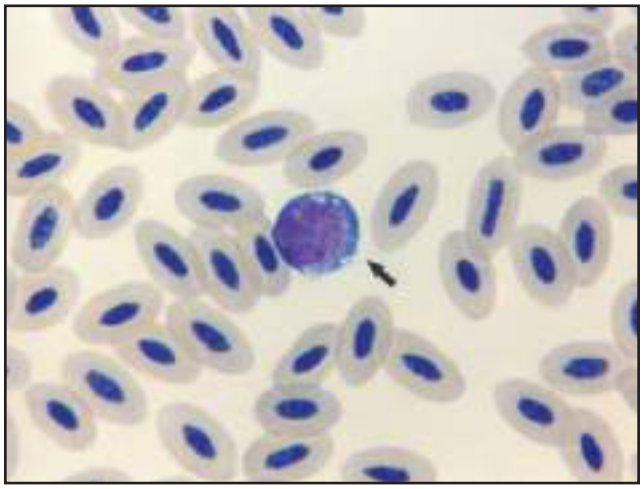


Fig 22.34 | A toxic monocyte (arrow). The nuclear/cytoplasm ratio is increased, the cytoplasm stains basophilic and there are numerous vacuoles within the cytoplasm (modified Wright-Giemsa stain).

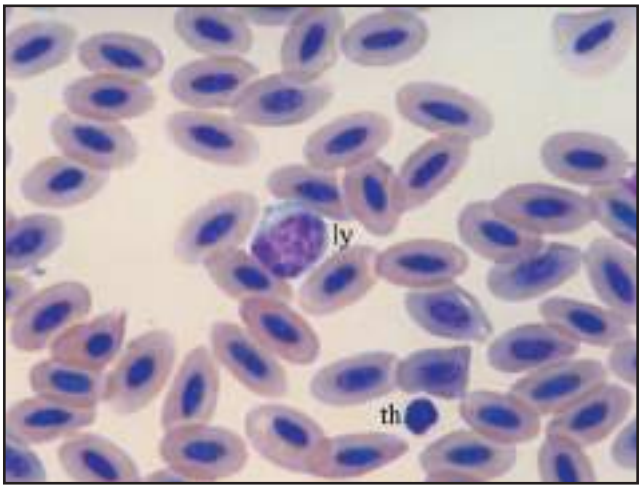


Fig 22.35 | A normal lymphocyte (ly) and a normal thrombocyte (th) (modified Wright-Giemsa stain).

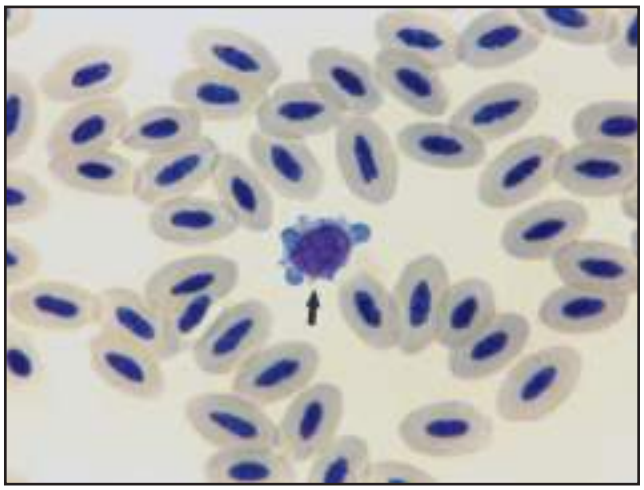


Fig 22.36 | A megathrombocyte (arrow). Megathrombocytes are larger than normal thrombocytes and can be confused with small lymphocytes. The cytoplasm of megathrombocyte stains basophilic and the nuclear chromatin is coarser (modified Wright-Giemsa stain).

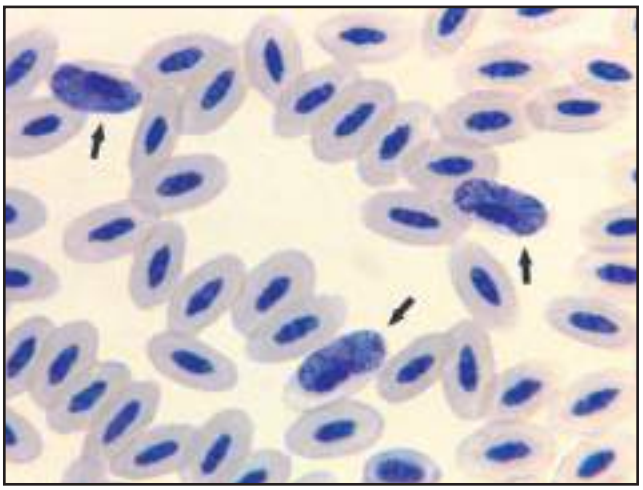


Fig 22.37 | *Haemoproteus tinnunculi* (arrows) (modified Wright-Giemsa stain).

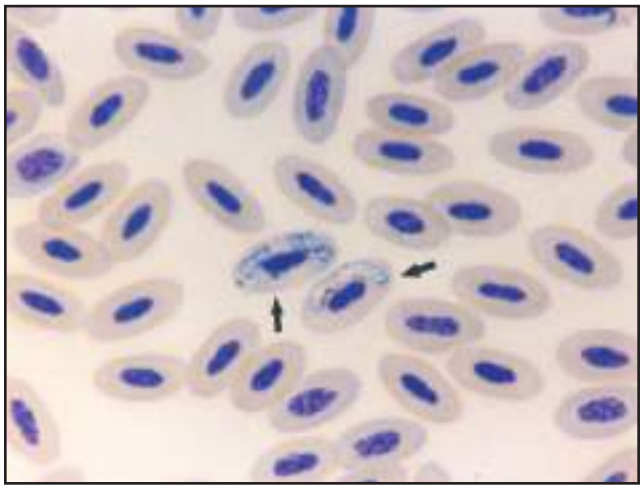


Fig 22.38 | *Haemoproteus psittaci* (arrows) from a green-winged macaw (*Ara chloroptera*).

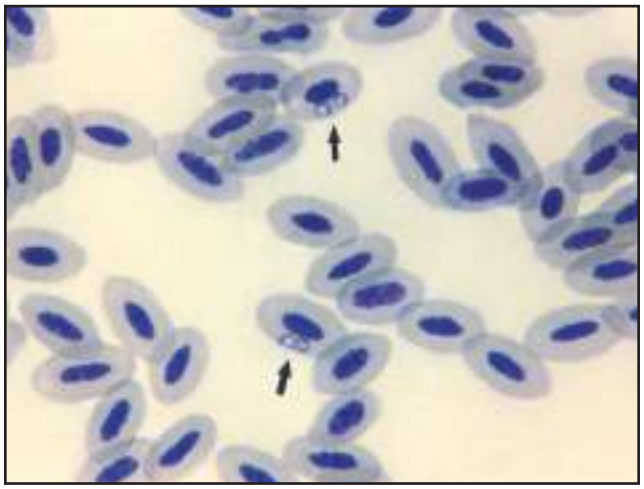


Fig 22.39 | *Babesia shortti* (arrows) (modified Wright-Giemsa stain).

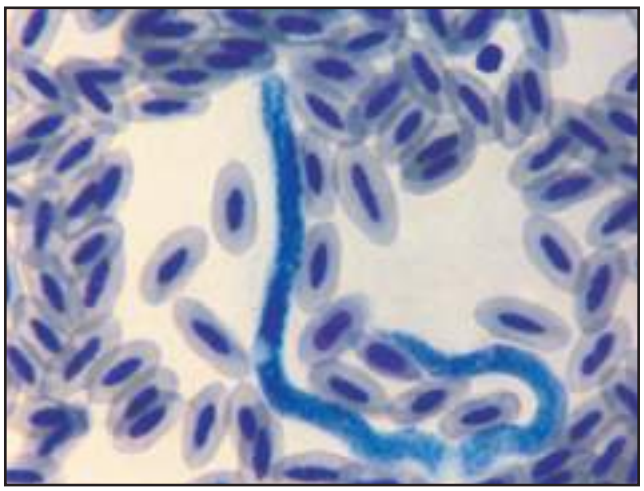


Fig 22.40 | *Microfilaria* sp. (modified Wright-Giemsa stain).

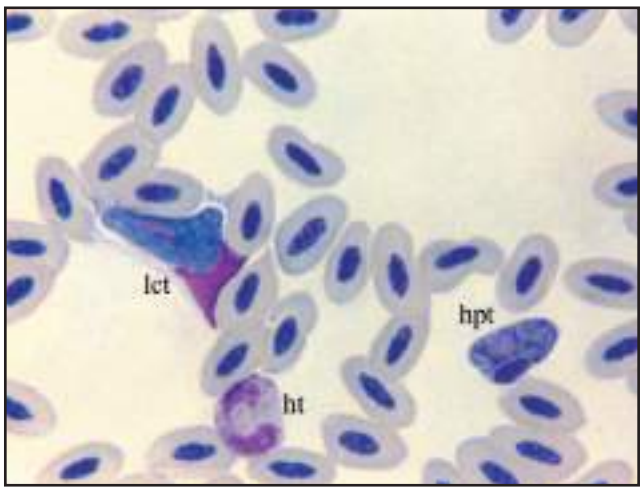
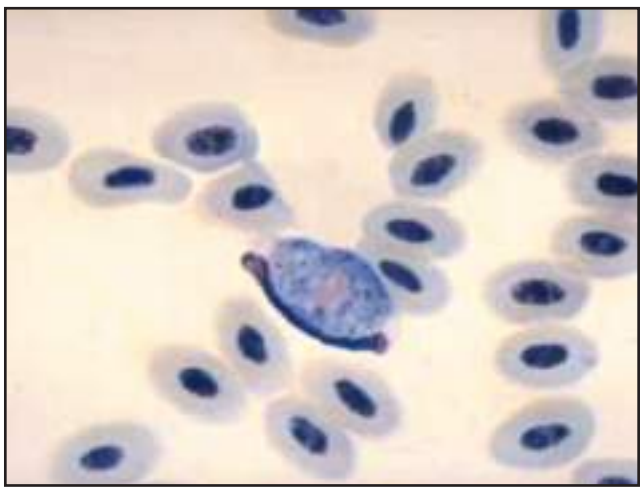
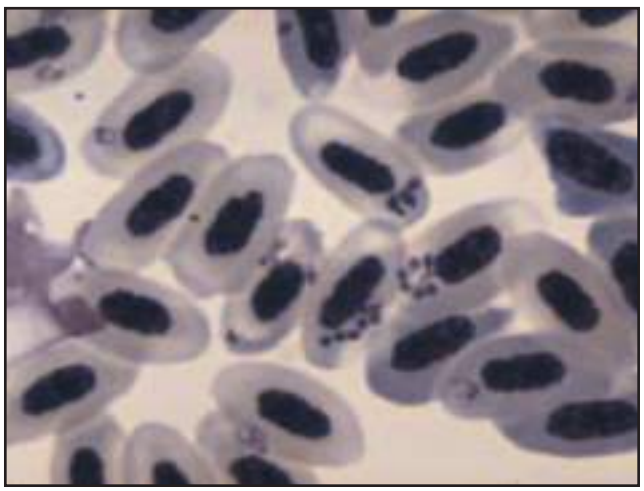


Fig 22.41 | *Leucocytozoon toddi* (lct), *Haemoproteus tinnunculi* (hpt) and normal heterophil (ht) (modified Wright-Giemsa stain).



Kendall Harr

Fig 22.42 | *Leucocytozoon simondi* from a Canada goose (*Branta canadensis*).



M. Griener

Fig 22.43 | *Plasmodium vaughani* schizont from a robin (*Turdus migratorius*).

Age-related Hematologic Changes

Age-related hematologic findings in kori bustard (*Ardeotis kori*) chicks during their growth and development are presented (Figs 22.44-22.48). Blood samples were collected from 16 clinically normal chicks at 1-month intervals. The tenth sampling was obtained at 15 months of age.

The following is a collection of hematology values and an interpretation guide for the avian veterinarian:

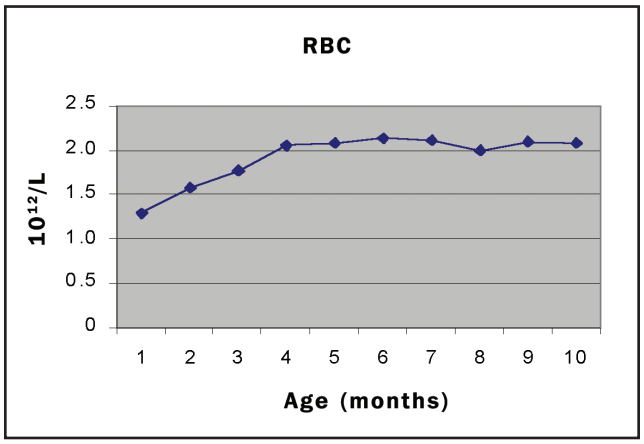


Fig 22.44 | The RBC increased steadily for the first 4 months from 1.28 ± 0.06 x 10^{12}/L at 1 month of age, increasing gradually up to the age of 4 months to 2.06 ± 0.08 x 10^{12}/L. After this time, the RBC remained fairly constant. The RBC value at the age of 12 to 15 months was 2.08 ± 0.06 x 10^{12}/L.

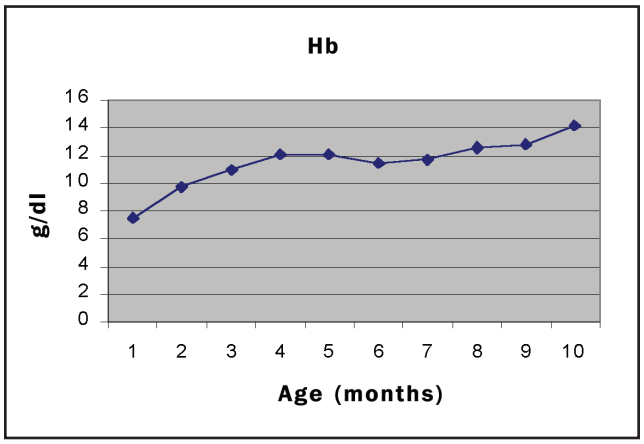


Fig 22.45 | The HB value followed a similar pattern as for RBC, with a value of 7.5 ± 0.2 g/dl at the age of 1 month, increasing to 12.1 ± 0.3 g/dl at 4 months of age. This value remained fairly constant until the age of 12 months, when it increased to 14.2 ± 0.4 g/dl.

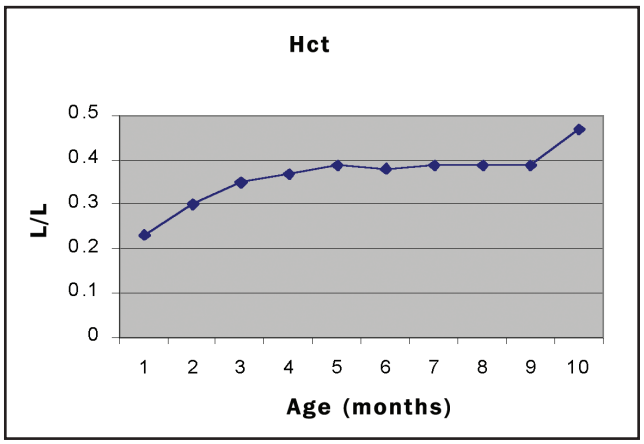


Fig 22.46 | The Hct value continued to increase steadily from 0.23 ± 0.7 L/L at 1 month of age to 0.399 ± 0.9 L/L at 5 months of age and remained fairly constant until the age of 12 to 15 months, when the value increased to 0.47 ± 0.9 L/L.

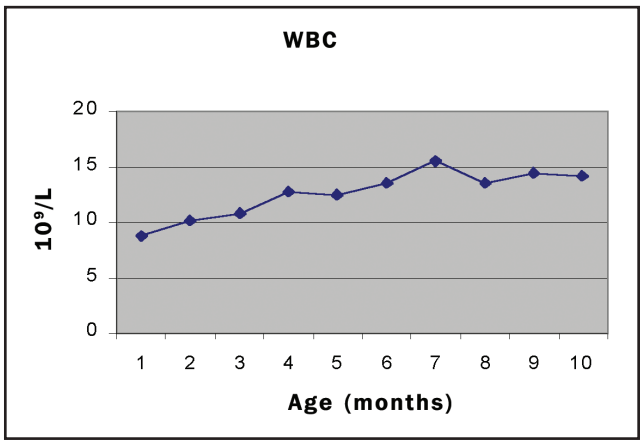


Fig 22.47 | The WBC count at 1 month of age was 8.78 ± 0.45 x 10^9/L, increasing to 15.6 ± 0.7 x 10^9/L at 7 months, then decreasing slightly to 14.5 ± 0.5 x 10^9/L at 9 months of age.

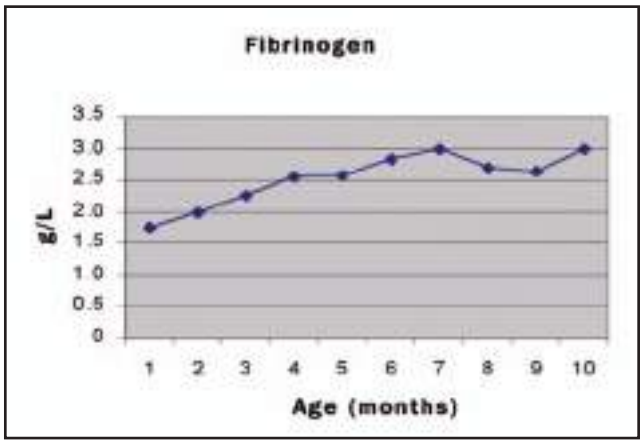


Fig 22.48 | The fibrinogen value was 1.76 ± 0.18 g/L at 1 month of age, increasing steadily to 3.0 ± 0.2 g/L at the age of 7 months.

Hemoresponses

WBC, DIFFERENTIAL WHITE BLOOD CELL COUNT

Table 22.12 | Evaluating the RBC, HB, PCV and Red Cell Indices

Hematologic Findings	Possible Causes
Polycythemia: Increased packed cell volume (PCV) or hematocrit (Hct) and red blood cell count (RBC)	**Absolute:** Primary polycythemia Polycythemia vera Secondary polycythemia, reaction to hypoxia Physiological: Adaptation to high altitudes Pathological: Chronic circulatory or respiratory disease (ie, COPD or asthma of macaws), iron storage disease, rickets Hypoxic increase in erythropoietin production Non-hypoxic, autonomous increase in erythropoietin production
	Relative: Dehydration, different etiologies
PCV >56% or Hct >0.56 L/L	Dehydration in most birds, relatively normal in small (<100 g) psittacine and passerine birds, especially cockatiels
Anemia: Decreased PCV or Hct and RBC	**Absolute:** Hemorrhage (trauma, coagulation disorders, ectoparasitism, endoparasitism); increased red cell destruction (hemoparasites, some bacterial infections, autoimmune hemolytic anemia); decreased red cell production (nutritional deficiencies, chronic infection, chronic renal disease, avian leukoses, toxicosis)
	Relative: Overhydration
Low hemoglobin (Hb) value: (eg, <11.0 g/dl)	Anemia in adult birds
Low mean corpuscular hemoglobin concentration (MCHC) value: (eg, <29.0 g/dl)	Possible iron and other element deficiency

Table 22.13 | Evaluating the Leukocytosis/Toxic Heterophilia with Left Shift

Monocyte Count	Possible Causes
Normal	**Infectious** *Acute:* Gram-negative septicemias. Tuberculosis (granulomas in Falconiformes and Galliformes, but not in Psittaciformes, in these species exclusive accumulation of epithelioid cells [Gerlach, personal communication, 2002]), coligranulomatosis, salmonellosis, yersiniosis and pasteurellosis
Monocytosis	Fungal: Aspergillosis, severe candidiasis Parasitic: Trichomonaisis, capillariaiasis, maggot infestations Miscellaneous: Foreign body inhalation pneumonia, focal peritonitis, chronic ulcerative lesions, old open wounds *Acute and chronic:* Pox and herpesvirus infections, chlamydophilosis *Chronic:* Granulomatous or purulent infections/ infestations
Monocytopenia	**Non-infectious** Maggot infestation, burns, lead/smoke intoxication, egg yolk peritonitis

Table 22.14 | Evaluating the Leukocytosis/Toxic Heterophilia with Left Shift (cont)

WBC and Differential	Monocyte Count	Humoral Cellular Response to Continued Presence of Pathogen	Prognosis
Normal WBC/toxic heterophils and/or reactive lymphocytes	Normal monocyte count - acute	Additional abnormalities-immature cells (left shift), anemia, bone marrow damage, excessive demand	Poor
	Monocytosis - chronic anemia due to bone marrow damage = depression/aplastic anemia	No additional abnormalities	Excellent

Note: Causes for bone marrow suppression and anemia include infections such as viral, bacterial endotoxins in gram-negative septicemia, neoplastic, toxic such as lead toxicosis, metabolic such as high estrogen levels, emaciation.

Table 22.15 | Evaluating Leukocytosis/Heterophilia/ Normal Heterophils

Monocyte Count	Serial Sampling	Possible Causes of Hemogram Changes
Monocytosis	WBC reduced	Healing of soft tissue damage or bone fractures without the complications of severe infection or necrosis, relative cell numbers depend on the degree of chronicity
Normal monocyte count	WBC stays elevated	Acute or chronic inflammation without severe tissue necrosis
	Return to normal within 24-72 hours in the absence of stressor	Stress

Note: Monocytes are slow-reacting cells of the immune system, still changing when other values are already close to normal levels.

Table 22.16 | Evaluating Lymphocytosis with Reactive Lymphocytes (seen rarely in species with a strong heterophilic leukogram)

Hematologic Findings	Possible Causes
Premature lymphoid cells, mitotic figures, anemia	Lymphoid leukosis; marked lymphocytosis with or without immature cells indicates lymphocytic leukemia, while marked lymphocytosis with predominantly mature, small lymphocytes with scalloped cell margins indicates lymphoid neoplasia
Blood parasites with or without lymphocytosis and/or anemia	*Haemoproteus* and *Leucocytozoon* spp. usually without manifesting clinical signs with the exception of young birds; *Babesia, Plasmodium* spp. may cause life-threatening condition with severe anemia and in some cases lymphocytosis
Monocytosis	Strong chronic stimulation of the immune system, eg, chronic inflammation, chronic viremias (leukopenia, lymphocytosis in chronic viral antigen exposure), chronic aspergillosis, immune-mediated diseases

Table 22.17 | Evaluating Leukopenia

Hematologic Findings	Possible Causes
Toxic heterophils, immature cells, anemia, monocytosis, reactive lymphocytes	Final stage of immune response, severe bone marrow damage, gram-negative septicemia, infected wounds, circovirus in psittacines, marked loss of skin, eg, burns, marked tissue necrosis, smoke intoxication. Grave prognosis.
Normal morphology, relative heterophilia, absolute lymphopenia	Initial stage of stress
Reactive lymphocytes, relative lymphocytosis, progressive or intermittent leukopenia	Acute viral infection (toxic heterophils in pox and herpesvirus infections)

Table 22.18 | Hematological Reference Values for Selected Avian Species

Hematology Assay	Egyptian Vulture[25] *(Neophron percnopterus)* n = 4	Common Buzzard[25] *(Buteo buteo)* n = 6	Golden Eagle[25] *(Aquila chrysaetos)* n = 4	Saker Falcon[50] *(Falco cherrug)* n = 25	Barn Owl[25] *(Tyto alba)* n = 10
RBC x 10^{12}/L	2.3 (1.9-2.6)	2.4 (2.2-2.7)	2.4 (1.9-2.7)	2.65 (2.0-3.9)	2.7 (2.2-3.0)
Hb g/dl	14.8 (13.3-16.5)	12.9 (11.6-14.6)	13.8 (12.1-15.2)	15.3 (13.3-21.2)	14.2 (12.7-16.4)
PCV %	43 (37-46)	38 (34-42)	41 (35-47)	47 (42-53)	46 (42-51)
MCV	190 (183-206)	159 (151-171)	174 (160-184)	183.1 (135.8-219.5)	176 (145-216)
MCH	67.7 (65.2-72.9)	53.8 (48.8-57.5)	58.9 (56.3-62.7)	60.7 (50.6-78.9)	51.1 (44.9-60.7)
MCHC	35.2 (35.0-35.5)	33.9 (31.4-36.0)	34.0 (32.3-35.9)	60.7 (50.6-78.9)	31.8 (28.9-34.9)
WBC x 10^9/L	7.6 (4.7-10.6)	9.1 (4.6-13.9)	13.1 (11.7-14.7)	33.2 (28.3-40)	16.6 (11.5-22.3)
Heterophils x 10^9/L	4.0 (1.2-5.5)	5.5 (2.3-8.8)	10.4 (9.5-12.7)	4.1 (2.1-5.9)	8.9 (5.2-12.5)
Eosinophils x 10^9/L	0.3-1.4	0.1-3.1	0.2-0.6	0	0
Basophils x 10^9/L	0	0.0-0.6	0.0-0.2	0	0
Lymphocytes x 10^9/L	2.5 (1.5-3.4)	1.7 (1.1-2.4)	2.2 (1.6-3.2)	1.3 (0.5-2.2)	5.0 (2.5-7.5)
Monocytes x 10^9/L	0.0-0.4	0	0	0.2 (0-0.6)	0
Thrombocytes x 10^9/L	13 (6-15)	27 (18-36)	14 (4-21)	0.41 (0.17-0.76)	33 (14-58)
Fibrinogen g/L	1.6 (1.0-1.9)	2.3 (1.3-3.3)	2.9 (2.0-4.1)	2.8 (1.7-4.7)	2.7 (1.9-3.3)

Hematology Assay	Crowned Crane[25] *(Balearica regulorum)* n = 33	Greater Flamingo[25] *(Phoenicopterus ruber)* n = 9	Rosy Flamingo[43] *(Phoenicopterus ruber ruber)* n = 25	White Stork[25] *(Ciconia ciconia)* n = 16	Kori Bustard[31] *(Ardeotis kori)* n = 28
RBC x 10^{12}/L	2.8 (2.4-3.1)	2.6 (2.3-2.8)	1.4 (1.1-1.8)	2.4 (2.1-2.7)	2.3 (1.74-2.95)
Hb g/dl	15.6 (11.9-18.8)	17.3 (15.9-19.6)	13.4 (9.2-17.6)	15.8 (14.4-17.7)	14.1 (11.9-15.9)
PCV %	47 (44-52)	50 (47-57)	47.8 (37.9-57.8)	45 (41-48)	47 (39.5-52.5)
MCV	171 (156-182)	193 (170-207)	326.6 (234.3-419.0)	189 (172-195)	208.5 (161.9-275.4)
MCH	64.3 (59.8-70.2)	66.2 (57.6-70.0)	91.5 (57.8-125.3)	67.2 (60.2-69.9)	62.4 (48-84.6)
MCHC	36.2 (34.5-39.2)	34.4 (33.5-35.2)	28.1 (20.4-35.8)	35.3 (31-36.9)	30.0 (29.7-34.9)
WBC x 10^9/L	11.1 (6.3-15.6)	2.4 (0.9-3.4)	8.7 (1.5-15.8)	10.8 (7-14.3)	7.3 (3.0-12.8)
Heterophils x 10^9/L	8.2 (4.1-13.3)	1.2 (0.2-3.0)	3.9 (1.0-11.4)	9.2 (5.1-14.9)	3.9 (0.9-9.25)
Eosinophils x 10^9/L	(0.0-1.3)	(0.0-0.4)	(0.0-0.3)	(0.0-0.7)	0.3 (0.0-1.1)
Basophils x 10^9/L	(0.1-0.8)	(0.0-0.4)	(0.0-0.8)	(0.0-0.5)	0.2 (0.0-0.8)
Lymphocytes x 10^9/L	1.6 (0.6-2.7)	0.9 (0.4-1.6)	5.2 (0.8-9.6)	0.8 (0.2-1.6)	2.2 (0.41-5.4)
Monocytes x 10^9/L	(0.0-0.3)	(0.0-0.2)	0.5 (0-1.8)	(0.0-0.3)	0.6 (0.0-1.5)
Thrombocytes x 10^9/L	3.6 (5-18)	4 (2-7)	—	19 (8-32)	5.5 (1.49-18.0)
Fibrinogen g/L	—	—	—	2.3 (1.7-3.2)	2.42 (1.42-4.5)

Table 22.18 | Hematological Reference Values for Selected Avian Species (continued)

Hematology Assay	Black-footed Penguin[25] *(Spheniscus demersus)* n = 57	Humboldt Penguin[55] *(Spheniscus humboldti)* n = 14	African Grey Parrot[25] *(Psittacus erithacus)* n = 11	Greater Sulphur-crested Cockatoo[25] *(Cacatua galerita)* n = 25	Scarlet Macaw[25] *(Ara macao)* n = 7
RBC x 10^{12}/L	1.74 (1.32-2.12)	1.8	3.3 (3.0-3.6)	2.7 (2.4-3.0)	3 (2.7-3.5)
Hb g/dl	16.8 (13.4-19.5)	15.0	15.5 (14.2-17.0)	15.7 (13.8-17.1)	16.8 (14.8-18.9)
PCV %	44 (36-51)	43	48 (43-51)	45 (41-49)	48 (46-52)
MCV	254 (232-273)	238.1	145 (137-155)	165.0 (145-187)	160 (143-175)
MCH	95.1 (87.2-104.3)	83.3	47.2 (41.9-52.8)	57.6 (53.8-60.6)	57.6 (51.1-64.2)
MCHC	37.8 (35.4-40)	34.8	32.5 (28.9-34)	34.9 (33.3-37.6)	35.9 (32.6-38.5)
WBC x 10^9/L	9.3 (3.5-16.3)	13.0	7.0 (3.3-10.3)	6.4 (1.4-10.7)	10.2 (6.4-15.4)
Heterophils x 10^9/L	8.1 (5.0-12.3)	8.0	4.9 (1.8-7.3)	3.7 (1-6.6)	8.0 (4.9-12.8)
Eosinophils x 10^9/L	(0.0-0.2)	1.1	0	(0.0-0.2)	0
Basophils x 10^9/L	(0.0-0.3)	0	(0.0-0.8)	(0.0-0.9)	(0.0-0.8)
Lymphocytes x 10^9/L	3.1 (0.8-5.2)	2.8	1.4 (0.7-2.1)	1.9 (1.0-3.6)	1.6 (1.2-2.2)
Monocytes x 10^9/L	0	0.6	(0.0-0.3)	(0.0-0.2)	0
Thrombocytes x 10^9/L	11 (5-19)	18.3	22.0 (11-42)	13.0 (7-24)	22 (17-30)
Fibrinogen g/L	2.9 (2.2-3.7)	—	2.2 (1.5-2.8)	1.4 (0.9-2.0)	1.7 (1.0-2.2)

Hematology Assay	Kea[25] *(Nestor notabilis)* n = 8	Fisher's Lovebird[250] *(Agapornis fischeri)*	Nicobar Pigeon[47] *(Caloenas nicobarica)* n = 16	Common Crowned Pigeon[47] *(Goura cristata)* n = 9	Brown Pelican[25] *(Pelecanus occidentalis)* n = 5
RBC x 10^{12}/L	2.6 (2.3-3.1)	4.5 (3.8-5.3)	3.4 (2.6-4.3)	2.31 (1.95-2.6)	2.7 (2.6-2.8)
Hb g/dl	13.4 (10.6-16.9)	15.3 (13.0-17.7)	17 (12.7-19.7)	12.3 (10.6-14.7)	14.5 (14.3-14.8)
PCV %	40 (34-46)	53 (45-61)	50.7 (45-56)	37.6 (33.8-42)	46 (43-49)
MCV	154 (137-186)	124.5 (108-141)	149.8 (127.6-168.5)	158.7 (142.9-175.0)	168 (166-173)
MCH	51.2 (41.6-68.1)	34.5 (29.3-39.8)	50 (41.3-57.6)	50.8 (44.2-57.3)	53.4 (51.2-56.8)
MCHC	33.2 (30.4-37.0)	29 (25.7-32.3)	33.5 (28.3-36.1)	31.9 (27.9-38)	31.7 (30.4-32.9)
WBC x 10^9/L	16 (12.1-22.6)	3.5 (0.6-6.4)	4.23 (2-8.2)	17.7 (11.7-25.1)	11.9 (6.6-19.4)
Heterophils x 10^9/L	13.8 (9.4-20.1)	2.5 (0.1-4.9)	5.2 (4.2-7.1)	6.6 (5.5-7.8)	6.7 (4.0-9.5)
Eosinophils x 10^9/L	(0.0-0.5)	0.15 (0.0-0.3)	3.7 (2.7-5.1)	0.2 (0.1-0.5)	(0.0-0.2)
Basophils x 10^9/L	(0.0-0.6)	0.2 (0.0-0.4)	0	(0.0-0.1)	(0.0-.0.2)
Lymphocytes x 10^9/L	1.9 (1.1-2.7)	2.3 (0.6-4.1)	3.7 (2.7-5.1)	3.0 (1.8-4.0)	4.0 (2.5-7.0)
Monocytes x 10^9/L	0	0.2 (0.0-0.3)	2.1 (1-5)	(0.0-0.02)	(0.0-0.2)
Thrombocytes x 10^9/L	16 (11-24)	15 (5-25)	—	—	27.5 (17-38)
Fibrinogen g/L	1.5 (1.1-1.8)	2.45 (0.9-4.0)	—	—	2.9 (2.6-3.1)

Hematology Assay	Ostrich[52] *(Struthio camelus)*	Domestic Fowl[20] *(Gallus domesticus)*	Wood Duck[45] *(Aix sponsa)* n = 15	Bar-Headed Goose[20] *(Anser indicus)*	Stone Curlew[52] *(Burhinus oedicnemus)* n = 18
RBC x 10^{12}/L	1.7	3.2 (2.5-3.9)	2.79	(2.5-3.2)	2.86 (2.59-3.27)
Hb g/dl	12.2	12.6 (10.2-15.1)	14.95	(12.2-17.2)	14.4 (12.2-16.6)
PCV %	32	39.5 (30-49)	45.5	(43-56)	47 (44-58)
MCV	174	119.5 (104-135)	164.2	(155-187)	167.3 (149.9-196.2)
MCH	61	37.9 (32.0-43.9)	54.0	(47.8-60.7)	50.7 (43.7-57.1)
MCHC	33	33.2 (30.2-36.2)	32.9	(28.5-33.9)	30.3 (27.7-35.5)
WBC x 10^9/L	5.5	5.7 (1.9-9.5)	2.3	(3.1-12.0)	7.88 (2.45-12.6)
Heterophils x 10^9/L	6.2	4.0 (0.5-7.6)	8.4	(0.8-8.3)	5.99 (0.9-11.5)
Eosinophils x 10^9/L	0	0.9 (0.0-1.8)	0.5	(0.0-0.5)	0.6 (0.0-2.7)
Basophils x 10^9/L	0	0.5 (0.0-1.0)	0.4	(0.0-0.8)	0.19 (0.0-0.8)
Lymphocytes x 10^9/L	3.4	2.7 (1.2-4.2)	13.2	(0.5-4.2)	0.5 (0.2-1.3)
Monocytes x 10^9/L	0.2	0.5 (0.0-1.0)	1.0	(0.0-1.2)	0.4 (0.0-0.9)
Thrombocytes x 10^9/L	—	18 (3-33)	—	(8-29)	8.9 (3.4-18.2)
Fibrinogen g/L	—	2.7 (1.3-4.1)	—	(1.9-4.8)	3.3 (2.1-4.1)

Products Mentioned in the Text

a. Coulter Counter ZF, Beckman Coulter Inc, Fullerton, CA, USA www.beckmancoulter.com
b. Cell Dyn 3500, Abbott Laboratories, Abbott Park, IL, USA www.abbottdiagnostics.com
c. BD Unopette 365851 red blood count manual hematology test, Becton Dickinson Co, Franklin Lakes, NJ, USA www.bd.com
d. BD Unopette 365877 eosinophil count manual hematology test, Becton Dickinson Co, Franklin Lakes, NJ, USA

Dedication

This chapter is dedicated to Dr. Christine M. Hawkey.

Acknowledgments

The author thanks HRH Prince Fahad bin Sultan bin Abdulaziz Al Saud for his support to the advancement of falcon medicine, and to Mr. Basil Al Abbasi, Director General, for his continuing interest in the clinical and research program of the Falcon Specialist Hospital and Research Institute; to Shinto K. John; Generoso Quiambao; Dr. Jesus Naldo.

References and Suggested Reading

1. Alonso JA, et al: Hematology and blood chemistry of free-living young great bustards (*Otis tarda*). Comp Biochem Physiol 97A:611-614, 1990.
2. Averbeck C: Hematology and blood chemistry of healthy and clinically abnormal great black-backed gulls (*Larus marinus*) and herring gulls (*Larus argentatus*). Avian Pathol 21:215-223, 1992.
3. Campbell TW: Hematology. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, 1994, pp 176-199.
4. Campbell TW: Avian Hematology and Cytology. Ames, Iowa State University Press, 1995, pp 3-19.
5. Clubb SL, et al: Hematologic and serum biochemical reference intervals in juvenile eclectus parrots. J Assoc Avian Vet 4:218-225, 1990.
6. Clubb SL, et al: Hematologic and serum biochemical reference intervals in juvenile cockatoos. J Assoc Avian Vet 5:16-21, 1991.
7. Clubb SL, et al: Hematologic and serum biochemical reference intervals in juvenile macaws (*Ara* sp). J Assoc Avian Vet 5:154-162, 1991.
8. Dacie JV, Lewis SM: Practical Hematology 8th ed. Edinburgh, Churchill Livingstone, 1995.
9. D'Aloia M-A, et al: Hemopathological responses to chronic inflammation in the houbara bustard (*Chlamydotis undulata macqueenii*). Com Haem Int 4:203-206, 1994.
10. D'Aloia M-A, et al: Normal hematology and age-related findings in rufous-crested bustards (*Eupodotis ruficrista*). Com Haem Int 5:10-12, 1995.
11. D'Aloia M-A, et al: Normal hematology of the white bellied (*Eupodotis senegalensis*), little black (*Eupodotis afra*) and Heuglin's (*Neotis heuglinii*) bustards. Com Haem Int 6:46-49, 1996.
12. Dein FJ: Hematology. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1986, pp 174-191.
13. Dorrestein GM: Cytology and haemocytology. *In* Beynon PH, Forbes NA, Lawton MPC (eds): Manual of Psittacine Birds. Cheltenham, Glos, Br Small Anim Vet Assoc, 1996, pp 38-48.
14. Fudge AM: Clinical application of laser flow cytometry to avian hematology analysis, Proc Assoc Avian Vet, 1995, pp 17-18.
15. Fudge AM: Clinical hematology and chemistry of ratites. *In* Tully TN, Shane SM (eds): Ratites: Management, Medicine, and Surgery. Malabar, FL, Krieger Publishing, 1996, pp 105-114.
16. Fudge AM: Avian clinical pathology: Hematology and chemistry. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 142-157.
17. Fudge AM: Problem-oriented approach to blood panel interpretation. Proc Assoc Avian Vet, 1998, pp 285-299.
18. Fudge AM: Avian cytology and hematology. Proc Assoc Avian Vet, 1998, pp 357-369.
19. Fudge AM (ed): Laboratory Medicine: Avian and Exotic Pets. Philadelphia, WB Saunders Co, 2000, pp 1-8.
20. Gulland FMD, Hawkey CM: Avian hematology. Vet Annual 30:126-136, 1990.
21. Harris DJ: Clinical tests. *In* Tully TN, Lawton MPC, Dorrestein GM (eds): Avian Medicine. Oxford, Butterworth Heinemann, 2000, pp 43-51.
22. Hauska H, Gerlach H: The development of the red blood cell pattern of growing parrot nestlings. Proc Assoc Avian Vet, 1995, pp 178-182.
23. Hauska H, Gerlach H: The development of the white blood cell pattern of growing parrot nestlings. Proc Assoc Avian Vet, 1995, pp 183-186.
24. Hauska H, Redig PT: Morphological changes in the white hemogram of raptors. Proc Assoc Avian Vet, 1997, pp 205-208.
25. Hawkey CM, Samour JH: The value of clinical hematology in exotic birds. *In* Jacobson ER, Kollias GV Jr (eds): Contemporary Issues in Small Animal Practice. London, Churchill Livingstone, 1988, pp 109-142.
26. Hawkey C, et al: Normal and clinical hematology of captive cranes (Gruiformes). Avian Pathol 12:73-84, 1983.
27. Hawkey C, et al: Haematological changes in domestic fowl (*Gallus gallus*) and cranes (Gruiformes) with *Mycobacterium avium* infection. Avian Pathol 19:223-234, 1990.
28. Hernandez M: Raptor clinical hematology. Proc Assoc Avian Vet, 1991, pp 420-433.
29. Hernandez M, Martin S, Fores P: Clinical hematology and blood chemistry values for the common buzzard (*Buteo buteo*). J Rapt Res 24(4):113-119, 1990.
30. Howlett JC: Clinical and diagnostic procedures. *In* Samour JH (ed): Avian Medicine. London, Harcourt Publishers Ltd, 2000, pp 28-50.
31. Howlett JC, et al: Normal hematology of captive adult kori bustards (*Ardeotis kori*). Com Haem Int 5:102-105, 1995.
32. Howlett JC, et al: *Haemoproteus* in the houbara bustard (*Chlamydotis undulata macqueenii*) and the rufous-crested bustard (*Eupodotis ruficrista*) in the United Arab Emirates. Avian Pathol 25:4-55, 1996.
33. Howlett JC, et al: Age-related hematology changes in captive-reared kori bustards (*Ardeotis kori*). Com Haem Int 8:26-30, 1998.
34. International Council for Standardization in Hematology: Guidelines for the evaluation of blood cell analyzers including those used for differential leucocyte and reticulocyte counting and cell marker applications. Clin and Lab Haem 16:157, 1994.
35. Jain CJ: Essentials of Veterinary Hematology. Philadelphia, Lea and Febiger, 1993, pp 19-53.
36. Jennings IB: Hematology. *In* Beynon PH, Forbes NA, Harcourt-Brown NH (eds): Manual of Raptors, Pigeons and Waterfowl. Cheltenham, Glos, Brit Small Anim Vet Assoc, 1996, pp 68-78.
37. Jimenez A, et al: Clinical hematology of the great bustard (*Otis tarda*). Avian Pathol 20:675-680, 1991.
38. Lane RA: Avian hematology. *In* Rosskopf W, Woerpel R (eds): Diseases of Cage and Aviary Birds 3rd ed. Baltimore, Lea and Febiger, 1996, pp 739-772.
39. Lind PJ, et al: Morphology of the avian eosinophil in raptors. J Assoc Avian Vet 4:33-38, 1990.
40. Maxwell MH: Avian blood leucocyte responses to stress. World Poult Sci J 49:34-43, 1993.
41. Maxwell MH, Robertson GW: The avian basophilic leukocyte: A review. World Poult Sci J 51:307-325, 1995.
42. Maxwell MH, Hocking PM, Robertson GW: Differential leucocyte response to various degrees of food restriction in broilers, turkeys and ducks. Br Poult Sci 33:177-187, 1992.
43. Merritt EL, Fritz CL, Ramsay EC: Hematologic and serum biochemical values in captive American flamingos (*Phoenicopterus ruber ruber*). J Avian Med Surg 10(3):163-167, 1996.
44. Mikaelian I: Variations circannuales des parametres hematologiques de l'outarde houbara (*Chlamydotis undulata*). Professional thesis (in French). Ecole Nationale Veterinaire de Lyon, Université Claude Bernard, Lyon France, 1993.
45. Mulley RC: Hematology of the wood duck (*Chenonetta jubata*). J Wildl Dis 16(2):271-273, 1980.
46. Palomeque J, Pinto D, Viscor G: Hematologic and blood chemistry values of the Masai ostrich (*Struthio camelus*). J Wildl Dis 27(1):34-40, 1991.
47. Peinado VI, et al: Hematology and plasma chemistry in endangered pigeons. J Zoo Wildl Med 23(1):65-71, 1992.
48. Pendl H: Avian hematology for practitioners. Proc Assoc Avian Vet, 2001, pp 387-400.
49. Samour JH, Peirce M: Babesia shortti infection in a saker falcon (*Falco cherrug altaicus*). Vet Rec 139:167-168, 1996.
50. Samour JH, D'Aloia M-A, Howlett JC: Normal hematology of the saker falcon (*Falco cherrug*). Com Haem Int 6:50-52, 1996.
51. Samour JH, et al: Normal hematology of the houbara bustard (*Chlamydotis undulata macqueenii*). Com Haem Int 4:198-202, 1994.
52. Samour JH, et al: Normal hematology and blood chemistry of captive adult stone curlews (*Burhinus oedicnemus*). Com Haem Int 8:219-224, 1998.
53. Stewart JS: Husbandry, medical and surgical management of ratites. Proc Assoc Avian Vet, 1989, pp 119-122.
54. Sturkie PD: Avian Physiology 2nd ed. London, Bailliére, Tindall and Cassell, 1965.
55. VanderHeyden N: Evaluation and interpretation of the avian hemogram. Sem Avian Exotic Pet Med 3(1):5-13, 1994.
56. Villouta G, Hargreaves R, Riveros V: Haematological and clinical biochemistry findings in captive Humboldt penguins (*Spheniscus humboldti*). Avian Pathol 26:851-858, 1997.

CHAPTER
23

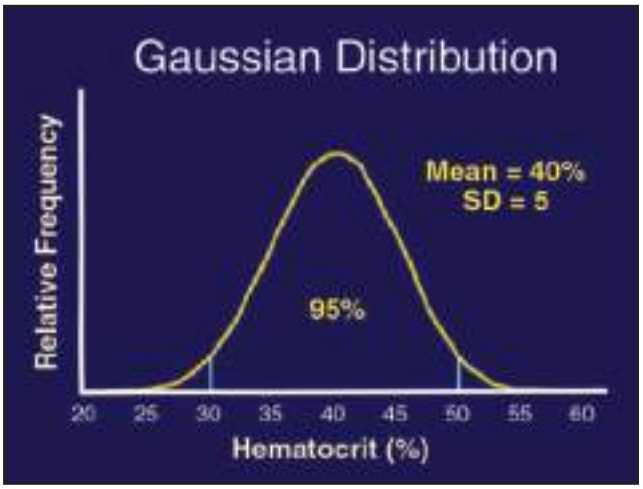
Diagnostic Value of Biochemistry

KENDAL E. HARR, DVM, MS, Dipl ACVP

Fig 23.1 | A normal serum sample on the left and a lipemic sample on right.

Clinical chemistry, along with hematology and physical examination, is the cornerstone of medical diagnosis of disease in any species. Plasma biochemistry is especially important in avian species, which frequently show minimal overt clinical signs of disease, even when seriously ill. Veterinarians, therefore, need accurate and useful biochemical analysis to successfully diagnose and treat avian species. The Clinical Laboratory Improvement Amendments (CLIA) are the federal laws and regulations that govern human diagnostic laboratories. No such governing policy exists in veterinary diagnostic medicine. This results in variable methodology, protocol, and most importantly, quality control, between veterinary laboratories. It falls to the veterinary clinician to ensure that the diagnostic laboratory being used maintains a high standard of quality control and that results are accurate. The clinician should develop a working relationship with the laboratory of choice and must continually monitor results for possible inaccuracy and error. Feedback to the laboratory must occur to ensure that the laboratory personnel are aware of any errors and can correct them. The laboratory used should be familiar with handling avian samples. Modified techniques in the laboratory, such as the use of 10-μl microhematocrit tubes, pediatric sample cups and dilution, can extend the sample and allow more data to be collected, especially from the smaller avian patient. When choosing a laboratory, the clinician also should consider its location, transport issues and delayed processing of the sample. All of these can cause artifactual change in the sample and decrease ability to diagnose disease.

Concentrations of analytes represent a steady state of input, consumption and partitioning. The body is always in flux. Generally, pathologic conditions cause either an increased or decreased concentration of various analytes. However, if both input and consumption are



J. Harvey

Fig 23.2 | 2.5% of the population is eliminated on each side of the curve to generate a 95% reference interval. This increases the likelihood that diseased patients are flagged as abnormal. It also means that it is quite likely that a healthy patient will have one analyte that is mildly abnormal.

decreased or increased at the same time, the concentration may remain in the normal reference interval even though the patient is very ill. For example, the globulin fraction may remain within the normal reference interval in a bird with marked enteritis because of increased production of acute phase inflammatory proteins and antibodies with concurrent loss of protein through a compromised gastrointestinal tract. Interpretation of clinical chemistries must therefore be done on a case-by-case basis with knowledge of species-specific physiology.

In comparison to domestic species, it can be a challenge to simply ascertain normal reference intervals for avian patients. Reference intervals for biochemical analytes are highly dependent on the machines, reagents and methods used, and may vary significantly between different laboratories. According to CLIA, each human diagnostic laboratory must establish reference intervals for each methodology validated within that laboratory in order to create a diagnostic range that can be used medically. A minimum of 100 healthy individuals is sampled. A 95% reference interval is created for normally distributed analytes using the formula, mean +/- 1.96 standard deviations. Therefore, the normal medical reference interval used by clinicians excludes 2.5% of normal individuals at the high and low ends of the range (Fig. 23.2). Biochemical reference intervals for common species of psittacines (parrots), passerines (canaries and finches), and galliformes (turkeys and chickens) have been established by specialized laboratories, eg, California Avian Laboratory, Citrus Heights, CA. Each laboratory analyzing avian samples should develop species-specific and methodology-specific reference intervals. These are still lacking in some university and private laboratories.

Reference intervals generated by a laboratory represent population reference intervals that are broader than the reference interval generated when repeatedly sampling an individual. Some individuals will regularly have concentrations in the low end of the population's range and some individuals will regularly have concentrations in the high end of the population's range. Therefore, an individual may remain within the population reference range even though there are abnormalities in the patient.

In addition to accurate normal reference intervals, the clinician must have knowledge of the sensitivity/specificity and positive/negative predictive value of a test's ability to diagnose diseases specific to that species. Although a great deal is now known regarding avian medicine, research into the diagnostic application, sensitivity, specificity, and positive and negative predictive values of biochemical analytes is still needed.

MEASURES OF THE ACCURACY OF A TEST

Accuracy is how close the test approximates the true value in the body. Precision measures how far from the mean or average of replicate measurements a particular measurement lies (Fig. 23.3). Most laboratory techniques were designed for use in human medicine and are modified for use in birds. Assessment of analytic accuracy and precision of a technique is very important when assessing different mammalian species, not to mention different kingdoms of animals. Any instrument error such as old bulbs, slight variation in machine temperature, variation in reaction time or degraded reagents can cause decreased accuracy and precision.

Sensitivity, specificity and predictive values are the measures of diagnostic accuracy. In medicine, sensitivity is the likelihood that a diseased patient will have a positive test result in a population of individuals with the disease. Sensitivity is a measure of false negative values. To help remember the relationships note that the letter "N" is present in sensitivity and false negative. Specificity is the likelihood that a patient without the disease has a test value that remains within the reference interval in a population of healthy individuals. Specificity is a measure of false positive values. Note that the letter "P" is present in specificity and false positive. The predictive value of a test is determined by its measurement in a population of healthy and sick individuals. A positive test result is measured when the disease is present (positive predictive value) and a negative test result is measured when the disease is not present (negative predictive value). These can be mathematically determined using the formulas in Table 23.2. Tests that are highly sensitive frequently have a low specificity and vice versa. This does

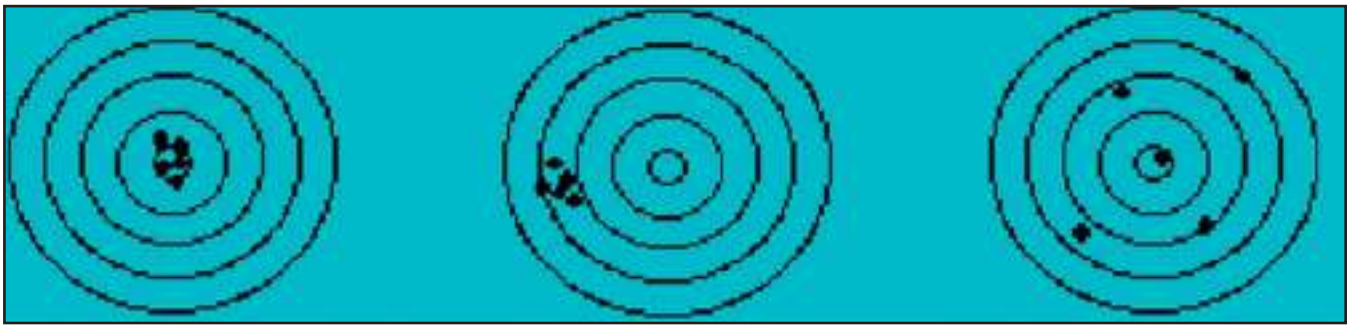


Fig 23.3 | Accuracy vs. Precision. When values are both precise and accurate, they are a tight cluster in the bull's eye. The precise values are all similar, but are some distance from the actual value. The accurate values are all within the third circle, with one almost approximating the actual value, but are scattered around the bull's eye.

Table 23.1 | Statistical Analysis of a Diagnostic Test

Formulas for the calculation of diagnostic sensitivity, diagnostic specificity, positive predictive value and negative predictive value. TP = true positive, TN = true negative, FP = false positive, FN = false negative.

- Diagnostic sensitivity = $\frac{TP}{TP+FN}$
- Diagnostic specificity = $\frac{TN}{TN+FP}$
- Positive predictive value = $\frac{TP}{TP+FP}$
- Negative predictive value = $\frac{TN}{TN+FN}$

not mean that the test is worthless. It simply means that it may have to be used with other tests to assess organ function and disease.

Sample Handling

HANDLING

In the USA, many exotic animal practitioners perform venipuncture using a syringe that has been coated with injectable sodium heparin to prevent clot formation. Experienced avian veterinarians working with experienced restrainers, who minimize trauma to the vessel wall, can collect a high-quality sample without the addition of sodium heparin. Reducing the amount of sodium heparin in the sample is desirable. If most of the heparin is expelled, it will minimally affect the sample. However, the amount of heparin actually retained may vary among samples. Any droplets remaining may cause dilutional effects as well as interfere with some analytical tests such as sodium and albumin. Samples for biochemical analysis should be placed into a lithium heparin microtainer rather than a red-topped tube to avoid variable clotting time and gelling of serum. Anticoagulant tubes must be filled to the appropriate volume. A plasma separator also can be used to increase plasma sample volume, though these tubes tend to be slightly more expensive.

Avian plasma samples are frequently yellow due to carotenoid pigments; rarely, in severe disease states, avian plasma may be truly icteric from bilirubin.[39,49] Pink or red plasma is usually indicative of hemolysis, though dyes from food should be ruled out. Green-tinged plasma is rarely observed, may be caused by biliverdin and is usually indicative of liver failure.[23,31] When working with smaller species of birds, tuberculin or insulin syringes are frequently used, however, not all of these syringes have detachable needles. Avian red blood cells are larger and deteriorate more quickly than mammalian erythrocytes. This can make accurate analysis difficult with ideal sample handling. Ejecting blood through a 25-gauge or smaller needle can cause moderate to marked hemolysis that will invalidate many biochemical analysis.[48] To avert this, attached needles can easily be cut from the syringe using a pair of large veterinary nail clippers or scissors before expelling the blood from the syringe.

ANTICOAGULANT

Prior to collection, the appropriate sample container is labeled with the names of the owner, patient and the signalment. Color-coded, rubber stoppered, evacuated tubes are well standardized. Green-topped tubes contain heparin, which should be used for plasma chemistry analysis in birds. Lithium heparin is the recommended anticoagulant, as sodium or potassium heparin can falsely increase the electrolyte values and skew anion gap and acid base analysis. Ammonium heparin should not be used, as it significantly increases ammonia and BUN concentrations. Heparin inhibits coagulation by binding to antithrombin III and greatly accelerates the inhibition of thrombin (factor II) by antithrombin III. Factors VII (proconvertin) and X (Stuart Prower factor) also appear to be inhibited by the heparin-antithrombin III complex.

The disadvantage of heparin is that leukocytes do not stain as well, and platelets and white blood cells clump much more than they do in blood collected in ethylene-diaminetetra-acetic acid (EDTA). This leads to decreased

accuracy and precision in the complete blood count (CBC) (see Chapter 22, Diagnostic Value of Hematology). Purple or lavender-topped tubes contain EDTA. Blue-topped tubes contain citrate and are used to harvest plasma for coagulation analysis. Both citrate and EDTA prevent coagulation by chelation of calcium (factor IV), an electrolyte essential to coagulation. Neither purple- nor blue-topped tubes are recommended for chemistry analysis because chelation of ions interferes with most reactions.

Red-topped tubes lack anticoagulant and are used to harvest serum required in antibody, hormone, and other protein analysis. At least 25% of avian serum samples will form a proteinaceous gel when separated, significantly decreasing sample volume and occasionally completely preventing biochemical analysis. Additionally, time to clot formation in avian species is variable, due in part to greater dependence on the extrinsic coagulation cascade. The use of heparinized plasma therefore decreases variability in time to sample separation and improves the chance of obtaining an adequate sample volume.

HEMOLYSIS

Hemolysis directly interferes with spectrophotometric absorbance readings and alters the pH of enzymatic reactions. Constituents that are found in higher concentrations within erythrocytes than in serum will be increased, eg, aspartate aminotransferase (AST) and, potentially, potassium. Alteration in the enzymatic reactions may appear randomly and cannot be predicted. Hemolysis can and should be monitored visually. Any sample that is more than very light pink should not be used diagnostically. Additionally, if the sample is analyzed by an automated hematology analyzer, a mean cell hemoglobin concentration (MCHC) that is greater than the reference range is an indication of possible hemolysis.

Artifactual change can vary between methods employed. Technical support and literature should be reviewed for each machine. Hemolysis in the sample falsely decreases bile acids measurement by the colorimetric assay, while radioimmunoassay (RIA) is unaffected. For many methods, hemolysis falsely increases alanine aminotransferase (ALT), aspartate aminotransferase (AST), lactate dehydrogenase (LDH), creatinine, calcium, albumin, potassium, amylase, creatine kinase (CK), hemoglobin, and MCHC. A false decrease in triglycerides can occur. Glucose, magnesium, phosphorus, cholesterol, alkaline phosphatase and lipase can be either increased or decreased depending on the methodology.[48]

LIPEMIA

Lipemia may be present in postprandial samples, but also may be indicative of underlying disease such as hypothyroidism, diabetes mellitus, hyperadrenocorticism, pancreatitis, or a primary lipid/lipoprotein disorder. Lipemia causes refraction of light and therefore causes error in many spectrophotometric and all refractometric methods (see Fig. 23.1).

Lipid can be partially cleared by ultracentrifugation or precipitating agents (polyethylene glycol, liposol, lipoclear). These techniques and clearing agents may themselves induce artifact. Additionally, the removal of lipid from a sample may in itself induce an artifact in analytes of interest. For example, lipoproteins bind bile acids, which would be discarded along with the lipid following ultracentrifugation. This may be one factor that contributes to the occasional measurement of decreased postprandial values in comparison to fasted values. The scattering of light due to lipemia will falsely increase the postprandial bile acid measurement. Varying technique can therefore significantly alter bile acid values. This underscores the importance of contact with your laboratory to determine which technique is used and that the techniques used are appropriate.

Again, technical support and literature should be reviewed for each machine. Wet chemistry analyzers are generally more impacted by lipemia than dry chemistry analyzers. Electrolytes measured by ion-specific electrodes are not affected by lipemia, but electrolytes measured by flame photometry are decreased.

Lipemia falsely increases all of the liver enzymes, alkaline phosphatase, hemoglobin, MCHC, bile acids, total bilirubin, glucose, calcium and phosphorous. Total protein measured by a refractometer is falsely increased, but the biuret method is minimally affected even by severe lipemia. BUN and gamma glutamyl transferase (GGT) may be increased or decreased depending on the methodology. Albumin is generally decreased using bromcresol green methodology.

Age

In general, non-protein nitrogen concentrations are lower in young, growing animals, as most nitrogen is being consumed by growth. In neonatal eclectus parrots *(Eclectus roratus)*, macaws and cockatoos, albumin, globulin and AST also have been found to be lower than in adults. This is likely due to decreased production of these analytes by the neonatal liver, combined with increased utilization in the tissues that is needed for growth. Additionally, it was found that calcium, sodium and chloride were decreased in chicks in comparison to adults. Alkaline phosphatase, a compound produced in

osteoblasts, is found in higher concentrations in growing animals. Blood phosphorus and potassium also are found in increased concentrations in young birds due partially to increased concentration of growth hormone, and mobilization for muscle and bone growth.[11,12,13]

Analytes

The following descriptions of analytes contain method, physiology and diagnostic value sections. The method sections are designed for practitioners who are running some values in their practice and, therefore, need to be familiar with the method that they are using to analyze plasma biochemical values. Different methods frequently will produce different results. The International Federation of Clinical Chemists (IFCC) has standardized some test methods and these should be used. Analyzer manufacturers may sell alternate methods at reduced prices to veterinarians, with the knowledge that they do not meet current standards. The veterinarian should be aware of the appropriate method to use and the limitations of interpretation in a species. Some artifacts and drug interactions are discussed in the method sections, though these should not be considered to be complete listings of those interactions. Product specification sheets for the methodology as well as technical support should be used as necessary. The sections on physiology discuss the function of the analyte in the body. The sections on diagnostic value discuss clinical utility in birds. See also the Differential Diagnoses in Table 23.2.

Acetoacetate, Acetone (Ketones)

Method

Common urine test strips present in most practices use the Rothera test in which alkaline nitroprusside turns purple in the presence of acetoacetate and, to a lesser extent, acetone. A third, relatively acutely produced ketone, 3-hydroxybutyrate, is not measured by this reaction. False negatives may occur if the patient is producing only 3-hydroxybutyrate. If diabetes is suspected and ketones are not measured, the urine should be rechecked in 48 hours. Some drug interactions may produce false positives, including penicillamine, levodopa and phenylketones.

Physiology

Decreased glucose availability to the tissues results in increased lipase activity in adipose tissue that catalyzes long-chain fatty acids. These are catabolyzed to acetyl CoA that is metabolized to the ketones: 3-hydroxybutyrate, acetoacetate and acetone. These compounds are excreted in urine and can be qualitatively measured in stressful physiologic or pathologic disease states.

Diagnostic Value

Healthy birds do not have ketones in their urine unless they have undergone strenuous activity, eg, migration. Measurement of ketone bodies in the urine is indicative of diabetes mellitus in most birds.

Albumin

Method

Most veterinary laboratories measure albumin using the dye bromcresol green (bcg), which has not been validated in companion avian species. Bromcresol green non-specifically binds protein. Binding of bcg causes increased color in the sample, which correlates with a higher reported albumin concentration. It has been demonstrated in dogs and humans that heparin can cause false increases in albumin concentration due to binding of fibrinogen.[59] Avian albumin is markedly different in structure than mammalian albumin and binds bcg with decreased affinity. Comparison of gel electrophoresis and bcg have revealed that bcg results in lower concentrations reported than actually exist in the patient.[58] This error is caused in part by use of human albumin standards and controls, which have different binding affinity for the dye than does avian albumin. This error in measurement may result in serious errors when assessing hypoproteinemic syndromes such as liver failure, protein-losing nephropathy and protein-losing enteropathy. At this time gel electrophoresis is the recommended method of albumin determination in avian species.[15,40,60] Bromcresol purple (bcp) also is commonly used in human laboratories and has different protein binding affinity for albumin.[2,5,64] Bromcresol purple may result in more accurate avian albumin measurement and better diagnostic acuity. Further study is needed.

Physiology

Albumin, a small, approximately 65-kD protein, is the most abundant protein found in plasma, most extravascular body fluid, CSF and urine. Albumin's synthesis by the liver is primarily controlled by plasma oncotic pressure. Albumin's main function is the maintenance of colloid oncotic pressure in the intravascular and extravascular spaces. Albumin also functions as a carrier protein to transport a large number of compounds including calcium and administered drugs. Albumin levels are lower in chicks than in adults.

Diagnostic Value

Accurate assessment of albumin enables the practitioner to assess hypoproteinemic disease such as liver failure, protein-losing nephropathy and protein-losing enteropathy. This diagnosis may lead to alternate fluid therapy such as colloid (hetastarch) administration to prevent edema, which is rarely seen in birds, and ascites. A true increase in albumin is pathognomonic for dehydration

Table 23.2 | Differential Diagnoses Based on Chemistry Abnormalities*

Albumin Increased	• Dehydration - generally accompanied by increased globulin and total protein • Reproductive - mild increase observed in females during egg formation
Albumin Decreased	• Liver failure - Cirrhosis/fibrosis - Neoplasia - Portosystemic shunt - Amyloidosis • Renal loss - Glomerulonephritis/sclerosis • Intestinal - Malabsorption/maldigestion · Mycobacterial disease · Endoparasites • Malnutrition (severe) • Exudative skin disease - Burns - Large wounds - Vasculitis - Frostbite • External blood loss (subacute to chronic) • Inflammatory disease state (seen with increased globulins) - Septicemia - Viremia • Neonates - normally lower than adults • Polyuria/Polydipsia
Alkaline Phosphatase Increased	• Bone isoenzyme - Trauma - Growth - Osteosarcoma - Osteomyelitis • No hepatic-associated increase currently documented
Ammonia Increased	• Hepatic disease/failure - Cirrhosis - Neoplasia - Polyoma • Artifactual - Hemolysis
Amylase Increased	• Pancreatic - Inflammation/Infection - Neoplasia - Necrosis - Pancreatic duct obstruction (eg, egg binding) • Zinc toxicity • Enteritis • Renal disease (decreased filtration) (mild to moderate increase)
Anion Gap Increased	• Respiratory acidosis - Anoxia (anesthetic induced, other) - Respiratory disease (chlamydia, aspergillosis, etc) • Hyperglobulinemia (reproductive, inflammatory) • Metabolic acidosis (increased lactic acid and other unmeasured anions) - Renal failure - Gastrointestinal bicarbonate loss - Hypoperfusion/reperfusion - Shock • Diabetic ketoacidosis • Toxic - Ethylene glycol (mammals) - Others in birds
Aspartate Aminotransferase (AST) Increased	• Muscle damage - Seizures - Trauma - Capture myopathy (exertional rhabdomyolysis) - Intramuscular injection • Hepatic damage - Drugs (cephalosporins, metronidazole, trimethoprim sulfa, dexamethasone) - Hemochromatosis (iron storage disease) - Endocrine disease (diabetes mellitus, hyperthyroidism) - Hypoxia (cardiopulmonary in origin) - Lipidosis (severe) - Inflammation/Infection
Aspartate Aminotransferase (AST) (cont.) Increased	• Hepatic damage (continued) - Infection [(bacterial, mycobacteriosis, chlamydophilosis, polyoma virus, Pacheco's disease - herpes virus, adenovirus, reovirus, duck virus hepatitis, *Plasmodium*, *Trichomonas*, *Histomonas* (turkeys), *Leucocytozoon* (ducks and geese), *Atoxoplasma*] - Toxic [(aflatoxin/mycotoxin, cottonseed (*Gossypium* sp.), *Crotalaria* sp., oleander (*Nerium* sp.), rapeseed (*Brassica napus*), ragwort (*Senecio jacobea*), castor bean (*Ricinus communis*)] - Neoplasia · Primary · Secondary - Artifactual · Erythrocyte leakage
Bicarbonate (CO_2) Increased	• Compensated respiratory acidosis - Respiratory disease - Drugs (anesthetics) • Small bowel obstruction • Gastric vomiting - Obstruction - Lead poisoning
Bicarbonate (CO_2) Decreased	• Metabolic acidosis - Dehydration - Renal failure • Respiratory alkalosis - Tachypnea/panting - Excess anesthetic ventilation • Artifactual - Delayed analysis
Bile Acids Increased	• Impaired liver function - Lipidosis - Biliary Stasis - Infection (see AST) - Inflammation - Neoplasia - Toxic (see AST) - Hemochromatosis - Cirrhosis/Fibrosis
Blood Urea Nitrogen Increased	• Prerenal azotemia - Dehydration (pigeons) - Postprandial (some raptors) - GI hemorrhage • Renal Failure
Blood Urea Nitrogen Decreased (Unlikely but can be measured in some species)	• Liver failure • Neonates • Diuresis - Iatrogenic - Physiologic - Pathologic
Calcium Increased	• Reproductive (may be marked) - Physiologic increase in females - Pathologic • Hypervitaminosis D • Primary hyperparathyroidism • Renal secondary hyperparathyroidism • Nutritional secondary hyperparathyroidism • Neoplasia - Lymphoma - Osteosarcoma • Osteomyelitis • Granulomatous disease
Calcium Decreased	• Nutritional - Excess dietary phosphorus (seed) - Hypovitaminosis D - Dietary deficiency (severe) • Chronic egg laying - Egg-bound hen • Hypomagnesemia • Hypoparathyroidism • Pancreatitis • Malabsorption • Alkalosis

Table 23.2 | Differential Diagnoses Based on Chemistry Abnormalities* (continued)

Chloride Increased	• Dehydration • Metabolic acidosis
Chloride Decreased	• Gastric vomiting - Obstruction - Lead poisoning • Metabolic alkalosis
Cholesterol Increased	• Reproductive - egg formation (cystic ovaries) • Nutritional/postprandial • Cholestasis • Obesity • Endocrine - Diabetes mellitus - Hypothyroidism - Hyperestrogenism • Nephrotic syndrome
Cholesterol Decreased	• Intestinal - Malabsorption/maldigestion • Liver failure • Starvation
Creatine Kinase Increased	• Muscle damage - Intramuscular injection - Seizures - Capture myopathy (exertional rhabdomyolysis) - Myositis · Sarcocystis · Toxoplasma · Other parasitic · Bacterial - Hyperthermia - Hypothermia - Vitamin E/Se deficiency - Trauma · Surgical - Ischemia
Fibrinogen Increased	• Bacterial infection • Other inflammation
Fibrinogen Decreased	• Liver failure • Coagulopathy
Gamma Glutamyltransferase (GGT) Increased	• Liver compromise - Cholestasis · Intrahepatic · Extrahepatic - Neoplasia · Biliary carcinoma • Other biliary compromise
Gamma Glutamyltransferase (GGT) Decreased	• Artifactual hemolysis
Globulin Increased	• Dehydration (concurrent increase in albumin) • Inflammation • Egg formation
Globulin Decreased	• Neonatal • Immunodeficiency • Blood loss (subacute to chronic) • Protein-losing enteropathy
Glucose Increased	• Endocrine - Diabetes mellitus • Pancreatitis • Stress • Drugs - Glucocorticoids - Progesterone
Glucose Decreased	• Liver failure • Starvation in small birds • Neoplasia • Septicemia
Iron Increased	• Hemochromatosis • Inflammation • Artifactual - Hemolysis
Iron Decreased	• Chronic blood loss • Nutritional dietary deficiency* **Ed. Note: Unlikely on formulated diet, but possible on a seed/fruit.*

Lipase Increased	• Enteritis • Pancreatitis • Pancreatic neoplasia • Renal disease (decreased loss)
Phosphorus Increased	• Renal disease • Neonates • Reproductive - Egg formation (concurrent rise in calcium) • Nutritional - Hypervitaminosis D - Increase dietary phosphorus • Toxic - Jasmine ingestion • Neoplasia - Osteosarcoma • Inflammation - Osteomyelitis • Artifactual hemolysis/delayed serum separation • Primary hyperparathyroidism • Nutritional secondary hyperparathyroidism • Neoplasia: PTH-like hormone
Phosphorus Decreased	• Diabetic ketoacidosis • Dietary deficiency
Potassium Increased	• Renal failure • Diabetic ketoacidosis • Severe muscle/tissue damage • Dehydration • Drugs - ACE inhibitors - Potassium-sparing diuretics • Artifactual - Collection in potassium heparin
Potassium Decreased	• Alkalosis • Drugs - Penicillins - Amphotericin B - Loop diuretics - Insulin therapy • Gastrointestinal loss • Renal disease (chronic)
Sodium Increased	• Gastric vomiting (water loss) • Intestinal fluid loss • Renal failure • Dehydration
Sodium Decreased	• Diabetes mellitus • Gastric vomiting • Intestinal sodium loss - Endoparasitism • Burns • Chronic effusions - Egg yolk • Psychogenic polydipsia • Renal failure (chronic) • Artifactual - Hyperlipidemia
Total Protein Increased	• Dehydration (albumin and globulin) • Artifactual - Hemolysis
Total Protein Decreased	• Hemorrhage (chronic) • Intestinal loss • Liver failure • Renal loss • Immune suppression
Uric Acid Increased	• Renal disease • Postprandial (carnivores) • Dehydration (severe)
Uric Acid Decreased	• Liver failure • Starvation

and would indicate the need for administration of IV crystalloid fluids.

Alkaline Phosphatase (ALP)

Method

Numerous methods have been developed to determine ALP activity. The IFCC recommended method uses 4-nitrophenyl phosphate (4-NPP) and 2A2M1P as a phosphate acceptor buffer at 37° C and absorbance at 405 nm. ALP catalyzes the hydrolysis of 4-NPP, forming phosphate and free 4-nitrophenol (4-NP) in an acidic solution. Alkalinization causes conversion of colorless 4-NP to 4-nitrophenoxide ion, which is an intense yellow color. As veterinary laboratories may employ different methods, normal reference intervals may be markedly different. Caution should be used when assessing a patient using reference intervals from a textbook. Laboratory-specific reference intervals should be generated.

Physiology

Alkaline phosphatase is a glycoprotein dimer with subunit masses ranging from 40 to 83 kD. The protein's exact function is unknown. Mammalian and avian isozymes of alkaline phosphatase have been identified in cell membranes in the liver (biliary epithelium), kidney, intestine, bone (osteoblasts), as well as a steroid-induced form in dogs. Isoenzymes from osteoblasts, duodenum and kidney have predominated in studies involving pigeons and domestic fowl.[28,43,45] Very low levels of alkaline phosphatase have been identified in the liver of pigeons and psittacines. Alkaline phosphatase levels are higher in chicks than in adults.

Diagnostic Value

In mammals, alkaline phosphatase is of particular interest in two specific disease states: biliary disease frequently associated with cholestasis and bone disease associated with increased osteoblastic activity. ALP does not increase with simple hepatocellular damage. In avian species at this time, marked increases in ALP have been associated only with increased osteoblastic activity including traumatic, neoplastic and infectious disease states. Further investigation into specific cholestatic and biliary disease such as biliary carcinoma is warranted to assess the sensitivity and specificity of ALP in these biliary diseases.

Ammonia

Method

Both enzymatic and chemical methods are used to measure ammonia. Enzymatic assay with glutamate dehydrogenase is the most frequently used method.[52] Glutamate dehydrogenase catalyzes the conversion of ammonium ion and 2-oxoglutarate to glutamate and water. This reaction oxidizes Nicoti Adenine Dinuleotide Hydrogen (NADH) to Nicotinamide Adenine Dinucleotide (NAD), which can be optically measured at 340 nm.

Meticulous precautions must be taken in sample handling to prevent false increases in ammonia concentration. Samples must be drawn cleanly, using an evacuated tube, and processed immediately for accurate results. Poor venipuncture technique or increased exposure to air may result in increased ammonia levels. Probing for a vein causes tissue damage that may elevate ammonia levels. Drawing blood into a syringe and transfer of that blood to a microtainer, or partial filling of an evacuated tube allows subsequent entry of air that may cause elevation of ammonia levels. Serum samples and ammonium heparin may cause falsely elevated levels. Production of ammonia by deamination of amino acids in the blood will occur once the specimen has been drawn. At 0° C, delays exceeding 15 minutes between blood sampling and centrifugation can increase ammonia concentrations.

Ammonia analysis are available on many dry chemistry analyzers used in practice. Machines-specific reference ranges should be established, as different methodologies will produce different reference intervals.

Physiology

The major source of ammonia is the gastrointestinal tract. It is derived from the hydrolysis of glutamine in the small and large intestine and from the action of bacterial proteases, ureases, and amine oxidases on digested food in the colon. Ammonia is converted to the less toxic uric acid and urea in the liver.

Diagnostic Value

Though plasma ammonia has not been validated in healthy or ill birds, some clinicians have observed up to a fourfold increase in blood ammonia values in birds with liver failure.

Amylase

Method

The three broad classifications of alpha-amylase (endoamylase) assays are saccharogenic, amyloclastic and chromogenic digestion of starch to glucose or maltose. The most commonly used assay in veterinary laboratories is the chromogenic alpha-amylase assay, which is the most appropriate assay in the canine.[35] This assay detects the release of dyes bound to synthetic starch substrates that are released as the starch is digested by amylase.

Physiology

Alpha-amylases are calcium-dependent metalloenzymes that catalyze hydrolysis of complex carbohydrates at

internal binding sites. The predominant sites of production are the pancreas and the duodenum. Trypsin in the small intestine degrades the enzyme, though some amylase frequently is still detectable in the feces. Urinary clearance of this small, 55- to 60-kD protein also has been documented in mammals.

Diagnostic Value

Urine-to-serum ratios are used in human medicine to diagnose acute pancreatitis.[26] Though this assay is frequently used for the diagnosis of pancreatitis in humans, it has decreased specificity and sensitivity in the dog. Validation of this assay for diagnosis of pancreatitis in birds is needed.

Anion Gap

Method

This number is calculated from the following formula:

Cations - Anions or
(Sodium + Potassium) - (Chloride + Bicarbonate)

Physiology

The gap is generally around 15 mEq/L (mmol/L) in most species with some variation, and represents unmeasured anions such as phosphate, sulfate, lactate, ketones, and drugs such as salicylates and ethylene glycol metabolites. If bicarbonate is not available, the total carbon dioxide value can be substituted. Generally, an increased anion gap indicates acidosis.

Diagnostic Value

Anion gap facilitates assessment of metabolic and respiratory acidosis. An increased anion gap should incite a search for the cause of increased numbers of unmeasured anions.

Acidemia causes an extracellular potassium shift as hydrogen ions enter the cells; the more chronic the disorder, the greater the intracellular potassium depletion. When correcting the acidosis, potassium moves back into the cell creating a potentially life-threatening hypokalemia. Accurate assessment of the acid base status of patients with chronic respiratory disease is essential to provide appropriate fluid therapy and electrolyte replacement.

Aspartate Aminotransferase (AST)

Method

The IFCC has standardized this reaction to some extent by limiting it to the rate-limiting reaction of L-aspartate and 2-oxoglutarate catalyzed by AST to form oxaloacetate and L-glutamate. Oxaloacetate is then reacted with NADH in the presence of malate dehydrogenase to form L-malate and NAD. Pyridoxal-5'-phosphate is a required coenzyme in the reaction and the IFCC recommends addition of this coenzyme in the reaction. The conversion of NADH to NAD can be optically measured at 340 nm. Reference intervals may vary slightly with variation of reagent concentration. AST is present in the cytosol of erythrocytes and extended red cell exposure can cause increased plasma AST concentration.

Physiology

The aminotransferases, including AST (formerly glutamate oxaloacetate transaminases, GOT) and alanine aminotransferase (ALT) (formerly glutamate pyruvate transaminases, GPT), are a group of enzymes that catalyze the interconversion of amino acids by transfer of amino groups. A variety of tissues, predominately liver and muscle, contain high aspartate aminotransferase. The mitochondrial and cytosolic isoenzymes of AST are approximately 90 kD in size.

Diagnostic Value

AST is not specific for hepatocellular damage, but is highly sensitive in detecting hepatocellular damage caused by ethylene glycol in pigeons.[40] Plasma AST activity returned to normal within 100 hours after doxycycline-induced muscle trauma in pigeons. AST activity is currently considered to be a very sensitive but nonspecific indicator of hepatocellular disease in other avian species as well, and is used with the muscle-specific enzyme creatine kinase (CK) to differentiate between liver and muscle damage.[16,30] ALT also is not liver-specific in birds. Prolonged postinjection increases in ALT decrease the diagnostic utility of this enzyme in the diagnosis of liver disease. ALT is therefore frequently omitted from avian chemistry panels.

Bicarbonate

Method

A common reaction used to measure bicarbonate is based upon phosphoenolpyruvate carboxylase (PEPC) utilizing bicarbonate present in the sample to produce oxaloacetate and phosphate. Malate dehydrogenase then catalyzes the reduction of oxaloacetate to malate, and the oxidation of NADH to NAD. NADH oxidation can be measured optically at 340 nm. Extended exposure of the sample to air (under-filled vials), late separation from the cell fraction or a dehydrated sample can introduce significant error in this measurement.

Physiology

Approximately 90% of carbon dioxide present in serum is in the form of bicarbonate. Therefore, measurement of total CO_2 is frequently used in place of bicarbonate measurement. This is different than pCO_2, which represents the remaining small percentage actually present in the gaseous form. The combination of water and CO_2 forms the weak acid carbonate, H_2CO_3, and its dissociated

Table 23.3 | Acid Base Imbalances and the Body's Compensation

	[H+]	pH	Imbalance	Compensation
Respiratory acidosis	↑	↓	↑ pCO_2	↑ $[HCO_3^-]$
Metabolic acidosis	↑	↓	↓ $[HCO_3^-]$	↓ pCO_2
Respiratory alkalosis	↓	↑	↓ pCO_2	↓ $[HCO_3^-]$
Metabolic alkalosis	↓	↑	↑ $[HCO_3^-]$	↑ pCO_2

forms, bicarbonate and hydronium ion, comprise one of the main buffering systems in animals. The Henderson Hasselbach equation, $pH = 6.1 + \log (HCO_3^- / H_2CO_3)$ where 6.1 = pK for the HCO_3^-/H_2CO_3 buffer pair, is used to quantitatively analyze buffering by carbonic acid.

Diagnostic Value

The measurement of bicarbonate, usually in conjunction with sodium, potassium and chloride, is used in the assessment of acid-base balance resulting from metabolic or respiratory disease. Respiratory acidemia is a common sequela in birds that have respiratory compromise or are anesthetized. Unfortunately, it is currently rarely assessed or treated. See the Anion Gap section for information and Table 23.3 for summaries of acid base assessment.

Bile Acids (BA)

Method

Radioimmunoassay (RIA) and enzymatic assay are the two commonly used methods for bile acid determination. Liquid chromatography also can be used in research settings. RIAs measure non-sulfated, conjugated bile acids.[29] Though less affected by hemolysis, RIA, an antibody-based assay, will measure only an unspecified proportion of bile acids in different species. The enzymatic BA method, validated for canine, feline and human samples, measures the 3-alpha-hydroxyl group present in most BAs. This test would be expected to best approximate total BA concentration in most avian species. The value generated by RIA is generally lower than the enzymatic measurement.

The pre- and postprandial sampling used in dogs and cats would likely be ideal for birds as well. However, the crop has varying emptying times in different species, and crop stasis is common in sick birds such that standardization of postprandial sampling is impossible. If possible, a fasted sample is preferred to eliminate random postprandial increases in BA concentration. Fasting is not necessary in species that do not have a gall bladder, such as pigeons, ostriches, and most parrots.[48]

Physiology

Bile acids are a group of amphipathic salts that act as detergent molecules both to facilitate hepatic excretion of cholesterol and to solubilize lipids for intestinal absorption. They promote formation of polymolecular aggregates known as micelles, which contain hydrophobic lipid in the center and have a hydrophilic outer surface. Bile acids are absorbed in the distal small intestine into the plasma and recycled from the blood by hepatocytes (enterohepatic circulation).

Diagnostic Value

Bile acids are used to assess liver function.[29,30,43] The clinician should be aware that RIA methodologies will generally produce significantly lower numbers than enzymatic methods. Laboratories should be questioned to determine which methodology they are using. Generally, using the enzymatic method, >75 μmol/L suggests hepatic insufficiency while >100 μmol/L is diagnostic for decreased liver function. Amazon parrots normally have slightly higher BA concentration than do other companion avian species.[29] Decreased bile acids may occur as a result of compromised intestinal absorption.

Bilirubin/Biliverdin

Method

The most commonly used method for bilirubin measurement are based on the diazo reaction, first developed by Ehrlich in 1883. Diazotized sulfanilic acid (diazo reagent) reacts with bilirubin to produce two azodipyrroles, which are reddish purple at neutral pH and blue at low or high pH values. The fraction of bilirubin that reacts with sulfanilic acid in the absence of alcohol is direct bilirubin (conjugated). Total bilirubin is determined after the addition of alcohol, and indirect bilirubin (unconjugated) is determined by subtracting direct bilirubin from total bilirubin.

At this time, biliverdin is measured only by high performance liquid chromatography (HPLC) for both clinical and research purposes.

Physiology

Bilirubin is the metabolic breakdown product of heme derived primarily from senescent erythrocytes. There are three types of bilirubin: unconjugated, conjugated and a fraction irreversibly bound to protein. The unconjugated portion of bilirubin is the most clinically important fraction, as this is most likely to cause tissue damage. Birds have heme oxygenase, which converts the protoporphyrin in heme to biliverdin;[4] however, birds and reptiles have significantly decreased hepatic production of biliverdin reductase that converts biliverdin to bilirubin. Bacteria in the intestine may produce biliverdin reductase and bilirubin may be absorbed from the GI tract. Additionally, though significantly decreased, hepatic biliverdin reductase is still present in some birds.[49,61] Bilirubin and biliverdin are detoxified via the glucuronic acid pathway in the liver and excreted in bile.

Diagnostic Value

Cholestasis and liver failure generally result in increased concentrations of biliverdin in birds. Bilirubin has been previously dismissed as unhelpful in the diagnosis of liver disease. However, there are reports of increased bilirubin in severe disease states.[49] Diagnostic sensitivity and specificity should be further assessed.

Blood Urea Nitrogen (BUN)

Method

There are numerous enzymatic, chemical and electrochemical methods for measurement of urea with good specificity for the compound. Reference intervals will vary with the methodology used. BUN levels are normally low in birds and may be below the detectable limit of some (but not all) assays used in the laboratory.

Physiology

During protein catabolism, nitrogen in amino acids is converted to urea in the liver by the action of the urea cycle enzymes. Though birds are predominately uricotelic, with urea being a minor component of nitrogen excretion, many still have functional hepatic enzyme action to drive the urea cycle. Additionally, bacteria in the gut may produce urea as well as ammonia, which can be absorbed from the intestinal lumen. The majority of urea is excreted through the kidneys, with some excreted through the GI tract in bile and through the skin. Urea is highly diffusible and, in addition to initial glomerular filtration, it moves passively through the renal tubules.

Diagnostic Value

Prerenal azotemia may be observed in dehydrated birds.[36,37] In penguins, it appears that BUN is not elevated postprandially, as was uric acid.[34] On the other hand, peregrine falcons (*Falco peregrinus*) had elevated BUN and uric acid when sampled 8 hours postprandially.[44] Renal disease also has been shown to cause azotemia.[40] BUN and uric acid may be used together — as separate pieces of the puzzle with history, physical exam, urinalysis and other more invasive diagnostic tests — to adequately assess prerenal versus renal disease. Using decreased BUN concentration as an indicator of liver failure has not been assessed in avian species, but may be possible in some species such as cockatoos.

Calcium (Ionized/Free)

Method

Ion-selective analyzers[b] capable of providing immediate whole-blood determinations of free calcium, electrolytes and blood gases are widely available. Calibration solutions, samples and wash solutions are pumped through a measuring cell containing calcium ion-selective, reference and pH electrodes. Sensitive potentiometers measure the voltage differential between electrodes, while the microprocessor calibrates the system and calculates calcium concentration and pH. Most of these units function at 37° C and so any significant temperature differential will make them inaccurate. These units must be maintained with regular calibration and assessment of controls.

Physiology

The ionized or free fraction of calcium is the freely diffusible, biologically active fraction. It is generally very tightly regulated by all species. It has been shown to increase mildly during active reproductive cycles in oviparous species.[33] Regulation of plasma calcium is achieved by interactions of parathyroid hormone, active vitamin D and calcitonin.

Diagnostic Value

A general reference interval is 1.0 to 1.3 mmol/L used in avian species at University of California Davis. Free calcium concentration in normal laying hens was found to be 1.3 to 1.6 mmol/L.[33] Species-specific values should be generated within the laboratory. There has been little work currently published on ionized calcium in disease in birds, but it will likely aid in differentiation of pathologic states such as renal disease, egg binding and malnutrition. Low ionized calcium in a symptomatic, possibly seizuring animal is an indication for well-monitored, intravenous calcium administration (see Chapter 5, Calcium Metabolism).

Calcium (Total)

Method

Photometric measurement of total calcium is generally used in veterinary diagnostic laboratories, though atomic absorption methods also may be used in research facilities. The two most common dyes used to bind calcium are o-cresolphthalein complexone (CPC) and arsenazo III. The sample is acidified to release protein-bound and complexed calcium. In alkaline buffered solution, CPC forms a red chromatophore when bound to calcium that can be measured at 580 nm. High magnesium concentration, lipemia and hemolysis will increase and invalidate results.

Physiology

The vast majority of calcium is stored in the skeleton as hydroxyapatite. In blood, a large portion of calcium is free, generally a smaller portion is protein bound and the smallest fraction is complexed to anions. Oviparous species have remarkable variability of the protein-bound and complexed portions due to estrogen-induced transport of calcium-bound yolk proteins to the ovary.[56]

Diagnostic Value

Calcium concentrations are dependent on the reproductive cycle, sex and possibly season; separate reference intervals for each of these variables should be established for accurate clinical evaluation of calcium values.

Absorption, excretion and compartmentalization all affect increases and decreases in plasma calcium. Disease states affecting the reproductive, renal, and digestive tract, as well as severe nutritive disorders may change calcium concentration.

The relationship between plasma total calcium concentration and total protein and albumin concentrations has been evaluated in several bird species.[3,38,46,62] Though correlations between calcium, total protein and albumin have been found in some species, they differ markedly between species. There are significant species differences in protein-calcium correlations such that generalized adjustment formulae will not be helpful in a clinical setting where many different species are evaluated. Total plasma calcium concentration, even if corrected for the effects of protein binding, does not provide information regarding ionized calcium concentration, the physiologically active fraction.

In oviparous species, increased phosphorus and calcium concentrations are observed during egg formation in females. Generally, these occur together and the calcium:phosphorus ratio stays above one in healthy individuals. If the calcium:phosphorus ratio is below one, renal disease should be investigated.

Chloride - See Electrolytes

Cholesterol

Method

There are numerous methods for lipid and cholesterol determination, with significant laboratory variation. Enzymatic methods are most commonly used in veterinary laboratories. Generally, cholesterol ester is reacted with water in the presence of cholesteryl ester hydrolase to form whole cholesterol and fatty acid. Cholesterol then reacts with oxygen in the presence of cholesterol oxidase to form cholest-4-en-3-one and hydrogen peroxide. Hydrogen peroxide is then measured in a peroxidase-catalyzed reaction that forms a dye that can be measured at approximately 500 nm.

Physiology

This 27-carbon, steroid alcohol is found in some concentration in almost all cells and body fluids in animals, and at a much lower concentration as phytosterols in plants. There is no cholesterol in plants. Cholesterol enters the intestine from three sources, the diet, bile and intestinal secretions, and sloughed cells. Cholesterol is metabolized by pancreatic secretions, intestinal secretions, and bile to micelles and then to chylomicrons that are absorbed into lacteals across intestinal villi. Transport of cholesterol in blood occurs via lipoproteins as high-density, intermediate-density, low-density, and very low-density lipoproteins. Higher density lipoproteins have increased concentrations of cholesterol. Cholesterol is synthesized and degraded in the liver (see Chapter 4, Nutritional Considerations, Section II Nutritional Disorders).

Diagnostic Value

Cholesterol levels may be altered in a normal bird due to oviparity, as well as postprandially. It should be noted that cholesterol can be elevated in oviparous reproductively active females before eggs can be visualized on a radiograph and may be accompanied by hyperostosis. In captive psitticines, cystic ovarian disease is a common syndrome that presents with the above mentioned clinical and laboratory findings in the absence of egg-laying (see Chapter 18, Evaluating and Treating The Reproductive System). When separated by plasma gel electrophoresis, the lipoproteins that transport cholesterol migrate predominately to the alpha and beta globulin regions. Cholesterol levels are rarely diagnostic but may be useful in determination of various disease syndromes (Table 23.3). HDL and LDL cholesterols are being investigated. Preliminary studies in birds show similar trends as those seen in humans (Stanford, 2004).

Creatine Kinase (CK)

Method

Bioluminescent methods are most sensitive and use the reaction of ATP (adenine triphosphate) with luciferin/luciferase. Spectrophotometric methods are used most commonly in veterinary laboratories, ATP also is used as the rate-limiting component of the reaction. CK catalyzes the reaction between creatine phosphate and ADP (adenine diphosphate) to form creatine and ATP at a pH of 6.7 and the reverse reaction at a pH of 9. This is coupled to a hexokinase-catalyzed reaction to form NADPH (reduced form of NADP). This conversion of NADP (nicotinamide adenine dinucleotide phosphate) to NADPH can be measured at 340 nm. Excess magnesium, manganese, calcium, zinc, copper, citrate, nitrate, iodide, bromide, malonate and L-thyroxine are inhibitory to the reaction and result in decreased values.

The addition of adenosine-5-monophosphate (AMP), N acetylcysteine, EDTA and diadenosine pentaphosphate (Ap5A) has been advocated to decrease changes in accuracy due to the above compounds. Addition of these components will change the value reported and result in significant laboratory variation.

Physiology

CK is a magnesium-dependent dimeric enzyme that catalyzes the reaction of ADP and creatine phosphate (CrP) to ATP and creatine in skeletal, cardiac and smooth muscle, as well as brain. It is present in the cytosol and mitochondria of myocytes.

Diagnostic Value

CK is increased for a short, <72-hour time period following myocyte damage or necrosis in birds.[47] Consideration should be given to the fact that all muscular structures in the body may be involved in CK elevation, including skeletal muscle, cardiac muscle and, less likely, muscle of the gastrointestinal tract. In mammalian species, hypothyroid patients frequently have marked (three- to five fold) increase in CK. This has not been reported in avian species at this time, but may exist.

Electrolytes - Chloride, Potassium, Sodium

Methods

The most common method for measuring electrolytes in veterinary laboratories is the ion-specific electrode (ISE). An ion-specific membrane is reacted either directly with plasma and serum or indirectly with a large volume of diluent, and then exposed to the membrane. The potentiometer measures the change in electromotive force (charge) in comparison to a reference electrode. The microprocessor then compares the unknown sample to a standard curve created from calibrators.

This method is very different from the spectrophotometric methods generally used in smaller machines in practitioner's offices. In this reaction, a specific ionophore changes color upon binding to the electrolyte. These dyes are measured by a spectrophotometer. Actual values that are generated by the two methods may be dramatically different and result in some difference between in-house and laboratory-generated reference intervals.

ISE is less affected by hemolysis; however, hemolysis will result in significant differences in potassium concentration (generally increased in most species) due to release from intracellular erythrocyte stores.

Physiology

Water homeostasis in any organism is a fundamental dynamic function. The four major electrolytes' primary functions are: maintenance of osmotic pressure, electroneutrality and water distribution. In addition to the main negatively charged ions (anions), bicarbonate and chloride, that are used to calculate anion gap, phosphates, sulfates, lactate, trace elements and proteins, all contribute to electrical neutrality and partitioning of water. The positively charged ions (cations) are predominately sodium and potassium, with contribution from divalent ions such as calcium and magnesium. The balance of cations and anions maintains pH and regulates nervous, cardiac and muscular function. Anions and cations also are essential cofactors in numerous enzymatic reactions.

Diagnostic Value

Any disease that disturbs water homeostasis will disturb electrolyte distribution and, therefore, plasma concentration. The success of electrolyte replacement therapy, fluid therapy or any attempt to restore nervous, muscular or cardiac function is completely dependent upon accurate assessment of electrolyte abnormalities.

Fibrinogen

Method

Heat precipitation is the most common method used in private practice settings and also is used commonly in commercial laboratories. It is the least accurate method for measuring fibrinogen in mammals, and is generally considered an estimate and not a true measurement. It is likely more inaccurate in birds. Protein is measured by refractometer in non-heated plasma and compared to a protein measurement in plasma heated to 57° C for 3 minutes. The difference in plasma protein represents the precipitated fibrinogen. This is obviously not specific to fibrinogen, as any refractile compound that precipitates will cause error. Birds, in comparison to mammals, frequently have increased quantities of refractile compounds, such as glucose and lipid, which will increase the error of this method.

In veterinary diagnostic labs, a modification of the thrombin test is used to more accurately measure fibrinogen. A known, relatively dilute concentration of thrombin is mixed with fibrinogen. Thrombin enzymatically cleaves fibrinogen to form fibrin, the final step in the coagulation cascade. The fibrin formation rate is proportional to fibrinogen concentration and can be calculated from tables. The tables were derived in humans, and though they work well in small animals, they markedly underestimate fibrinogen concentration in horses. The modified thrombin test has not been validated in avian species.

Physiology

Fibrinogen (coagulation factor I) is an acute-phase protein, made by the liver, that is digested by thrombin to form fibrin. Fibrin, when crosslinked, forms the backbone of the platelet clot.

Diagnostic Value

Fibrinogen is generally increased in non-specific inflammatory states. Fibrinogen concentration was increased above the reference interval in 77% of 89 birds, representing 20 species, with confirmed bacterial disease.[24] Decreased fibrinogen may be indicative of end-stage liver failure, but is most commonly detected in mammals with

disseminated intravascular coagulation (DIC). DIC is not commonly evaluated in birds at this time. Further investigation is warranted.

Galactose Clearance (GEC)

Method

Methods to determine GEC and blood galactose concentration in cockatoos have been described.[30] The birds were administered 0.5 g/kg of sterile galactose intravenously. Single-point galactose concentrations were best correlated with galactose clearance when sampled at 80 minutes postadministration. Blood samples were deproteinized within 90 minutes of collection by the addition of 1 ml of 0.3 M perchloric acid to a 0.2 ml aliquot of blood. After centrifugation at 1500 g, the clear supernatant was stored at -20° C for preservation until galactose was measured using a commercial ultraviolet method coenzyme assay kit[c]. Galactose clearance was calculated using the formula GEC (g/min)= (M-U)($t_{c=0}$ +7) where M is the amount of galactose injected, U is the amount of galactose excreted in urine, and $t_{c=0}$ is the extrapolated time when concentration equals zero. A standard value of 6% urinary excretion was used throughout, as the author determined previously in cockatoos. At 80 minutes, the normal reference intervals were 0.05 to 0.55 g/L for single-point galactose concentration and 0.86 to 1.52 g/min galactose clearance.

Physiology

Galactose is a monosaccharide isomer of glucose that is converted to glucose in the liver through gluconeogenesis pathways for use as energy. Approximately 90% of circulating galactose is filtered from the blood by a healthy mammalian liver during the first pass effect. As hepatic function decreases, the portion of galactose filtered decreases and so the concentration of galactose measured would increase in disease states.

Diagnostic Value

Galactose clearance and galactose single-point concentrations were evaluated during a prospective study on the effects of partial hepatectomy in cockatoos.[30] In the study, galactose clearance appeared to be a more sensitive indicator of hepatic insufficiency than plasma enzyme activities or BA levels, and were able to detect an 18% loss of hepatic mass. Though single-point concentration was never increased in animals with 18% hepatectomy, it is likely that this value would increase with more significant liver dysfunction. The authors concluded that GEC has the potential to be a simple, sensitive method of screening birds for decreased hepatic function.

Gamma Glutamyltransferase (GGT)

Method

The IFCC reference method uses L-gamma-glutamyl-3 carboxy-4-nitroanilide as the glutamyl donor and glycylglycine as the acceptor in a solution of hydrochloric acid. The nitrobenozoate produced is measured at 410 nm. Other methods are still in use and may produce different values, therefore reference intervals may vary from those values stated in available texts. Lipemia may increase or decrease GGT, depending on methodology used.

Physiology

GGT catalyzes the transfer of the gamma glutamyl group from a donor peptide to an acceptor compound. It is present in serum and in low levels in the cell membrane of all cells except muscle in mammals. It may be involved in glutathione metabolism and detoxification. The primary source of plasma GGT is the biliary system, while significant levels of GGT of renal epithelium origin are found in the urine.

Diagnostic Value

Significant increases in GGT are due to obstruction of or damage to the biliary tree including neoplasia, inflammation or cholelithiasis (stones). Hepatocyte damage alone will not significantly change plasma GGT concentration. In mammals, GGT is a more sensitive indicator of biliary damage than alkaline phosphatase. GGT has previously been thought to be insensitive in the diagnosis of "liver disease," which is likely due to the fact that it will not become elevated in cases where the biliary tree is not compromised. Increased plasma GGT activity was found in the majority of pigeons with experimentally induced liver disease.[47] Marked increases in GGT activity in birds with bile duct carcinoma also have been reported.[54]

Although reference intervals have not been established, GGT values of 0 to 10 U/L are considered normal at the Schubot Exotic Bird Health Center (College Station, Texas, USA). GGT values appear slightly higher in older Amazon parrots, which may have GGT values up to 16 U/L without other evidence of liver disease. These GGT values are higher than the reported reference intervals for GGT[47] of <3 or 4 U/L in most species except Amazon parrots, which had a high normal value of 10 U/L.[40] Differences in methodologies for measuring GGT may account for the marked differences in reference intervals.

There are numerous reports of birds with bile duct carcinoma or cholangiocarcinoma in which no concurrent increase in GGT activity was reported.[14,18,27,51] It is possible that GGT was not measured, since GGT activity would be expected to increase in these hepatic diseases. The authors did not indicate if GGT had been analyzed in these cases. The clinical utility of GGT in the diagnosis of biliary conditions in birds has not been adequately evaluated.

Globulin

Method

Globulin concentration is calculated by subtracting albumin from total protein. Any error in albumin or total protein measurement will cause error in globulin concentration. Globulins can be separated by plasma gel electrophoresis. When the exact size of the protein of interest is unknown, as is commonly the case in veterinary medicine, the proteins are classified in the alpha, beta or gamma globulin fraction, dependent upon where they band on the gel.

Physiology

Any plasma protein that is not albumin or, in birds, transthyretin (pre-albumin) is classified as a globulin. Plasma globulins that have been identified in the banding pattern of birds are alpha-1-antitrypsin (alpha-1 globulin fraction); alpha-2-macroglobulin (alpha-2 globulin fraction); fibrinogen, beta-lipoprotein, transferrin, complement, and vitellogenin (beta-globulin fraction); and immunoglobulins and complement degradation products (gamma globulin fraction).[15] In oviparous females, vitellogenin and other proteins used in egg formation can increase dramatically during reproductive activity. Thus, the globulin fraction increases more than the albumin fraction, causing the Albumin:Globulin (A:G) ratio to decrease physiologically in some female birds.

Diagnostic Value

In addition to egg formation causing increased globulins, inflammatory disease states frequently result in increased globulins. Acute-phase proteins such as alpha-2-macroglobulin are produced in the liver in response to inflammatory cytokines. Generally in inflammatory states, these acute-phase proteins and immunoglobulins increase while albumin, a negative acute-phase protein, decreases. This results in a decreased A:G ratio. Gel electrophoresis allows the practitioner to examine the banding pattern and determine if the decreased A:G ratio is due to acute or chronic inflammation or egg formation. Banding patterns cannot be used to diagnose specific disease such as aspergillosis or chlamydophila infection.

Decreased globulins result from decreased production such as in liver failure, or increased loss, most commonly due to protein-losing enteropathy.

Glucose

Method

There are several methodologies used to measure glucose that vary between the types of machines used. The normally high blood glucose level of many avian species may fall above the linear range of measurement of some handheld glucometers. The practitioner should know the linear limit of their glucometer, usually found in the manufacturer's insert. Results above that limit should be written as >linear limit (#). It should be noted that many small handheld units guarantee only 20% coefficient of variation, which means that the number obtained can vary as much as 20% from the actual glucose concentration in the sample.

Most veterinary laboratories use the hexokinase method on a wet reagent analyzer, where NAD is measured at 340 nm after two reactions using hexokinase and glucose-6-phosphate dehydrogenase. This method has an upper limit of 1000 mg/ml in undiluted samples and 2000 mg/ml with automatic dilution. It is therefore the preferred method in avian species.

Lipemia and hemolysis can both increase the measured plasma glucose concentration.

Physiology

There are species differences in the way that birds regulate blood glucose. The insulin content of the pancreas of granivorous species is about one-sixth that of the mammalian pancreas, while glucagon content is about 2 to 5 times greater.[25] Pancreatectomy induces hypoglycemic crisis in granivorous birds, but produces diabetes mellitus in carnivorous birds.[40] The finding suggests that while glucagon predominates in granivorous birds, insulin may predominate in carnivorous birds. Although diabetes mellitus in psittacines is attributed to increased glucagon secretion, there have been reports of decreased insulin concentration in a diabetic African gray *(Psittacus erithacus)* in comparison with a normal bird[9] and positive responses to insulin therapy. It is therefore possible that either glucagonemia or hypoinsulinemia are responsible for diabetes in psittacines and other species.

Stress hyperglycemia is induced in birds by high levels of endogenous or exogenous glucocorticoids.

Diagnostic Value

Stress hyperglycemia should be ruled out prior to a diagnosis of diabetes mellitus. Measurement of insulin and glucagon levels in comparison to a control bird should be attempted to determine etiology of diabetes on a case-by-case basis. Etiologies of hypoglycemia should be ruled out using additional diagnostics. Consider the use of a carbohydrate absorption test (xylene or glucose challenge) to rule out underlying malabsorption/maldigestion.

Iron

Method

Care must be taken to ensure that anticoagulant collection tubes do not contain iron, EDTA, oxalate or citrate that bind iron. Some iron methods require serum and

cannot assay plasma. Check with the laboratory prior to submission. There are a variety of methods, including coulometry, colorimetry and atomic absorption spectrophotometry, that are used to measure iron. Colometry involves applying a voltage to a reaction and measuring the amount of energy needed to drive the reaction. It can be performed as a titration with another ion and is generally quite accurate in the measurement of iron. Atomic absorption is unreliable due to matrix interference in serum. Through modification of the serum iron methods, total iron-binding capacity and serum transferrin also can be determined. Ferritin, the storage form of iron that is generally measured to assess total iron stores in mammals, is measured using antibody-based ELISA and RIA techniques that do not cross-react in avian species. Ferritin, therefore, cannot be measured at this time.

Physiology

All living organisms, except possibly *Lactobacillus* spp., require iron.[57] Aside from meat-based products, most ingested iron is in the less bioavailable ferric form. The ferric form (Fe^{3+}) can be reduced to the bioavailable ferrous form (Fe^{2+}) by intestinal bacteria. Free iron can catalyze free radical formation from oxygen and nitrogen, and can therefore cause marked cellular and tissue damage. For this reason, plasma and intracellular iron are protein bound. The majority of iron in the body is bound in hemoglobin; however, iron also is bound to transferrin for transport in the plasma, ferritin and hemosiderin for cellular storage, and myoglobin in muscle. Prior to egg laying, iron levels will increase 2 to 3 times normal in some species.[28]

Diagnostic Value

Serum chemistry results in birds with hemochromatosis may include increased liver enzyme activity, usually AST, which is believed to be due to iron-induced hepatocellular damage.[40] A few anecdotal case reports describe increased serum iron concentration in sick versus control birds.[40] Other studies found no significant correlation between serum chemistry values, serum iron concentration, total iron-binding capacity and unsaturated iron-binding capacity with hepatic iron accumulation, as assessed by histopathology and iron quantification.[66] However, the reference values used in one study were considerably higher than those used in domestic mammals and human beings. Additionally, birds with possible inflammation (eg, leukocytosis, heterophilia and monocytosis) were included in the study.[66]

Although serum ferritin concentration correlates significantly with non-heme iron in the liver and spleen of dogs, cats, horses and pigs, this correlation has not been explored in birds due to the species-specificity of antibody recognition and binding in ferritin ELISAs and RIAs.[21] Additionally, percentage iron saturation has not been evaluated in birds. Further study of iron status in companion avian species may have the clinical benefit of eliminating invasive liver biopsies as a screening modality for diagnosing hemosiderosis.

Lipase

Method

Many lipase methods including titrimetric, turbidimetric, spectrophotometric, fluorometric and immunological techniques have been described, with no one method recognized as a gold standard. Differences in laboratory values are likely due to differences in methodologies. Be cautious when using reference intervals from the literature to assess a patient.

Physiology

Lipase hydrolyzes glycerol esters of emulsified, long-chain fatty acids.[55] Most lipase produced in mammals is produced in the pancreas, however activity also is seen in gastrointestinal mucosa, leukocytes and adipocytes. Tissue enzyme contributions have not been investigated in birds.

Diagnostic Value

In mammals, lipase concentration in plasma and effusive fluid is most commonly used to investigate pancreatic disorders, usually pancreatitis. It is generally more useful in acute forms of the disease, as more chronic lesions are associated with increased parenchymal destruction that results in lower levels of lipase. Renal disease can result in mildly increased plasma levels due to decreased clearance, but increases are generally not as dramatic as with pancreatitic disease.

Phosphorous

Method

In the most common method used, phosphate reacts with ammonium molybdate to form a phosphomolybdate complex. The colorless phosphomolybdate can be measured photometrically at 340 nm or can be reduced to molybdenum blue and measured at 600 to 700 nm. Measurement in the 340-nm range is more likely to be affected by hemolysis, icterus and lipemia.

Common detergents are frequently contaminated with phosphate and it should be ensured that phosphate is measured using only new, unwashed equipment. Delayed plasma separation or hemolysis will significantly alter plasma phosphorus levels.

Physiology

Inorganic and organic phosphorus both have numerous vital roles in the body. Inorganic phosphate is complexed with calcium to form hydroxyapatite in beak and bone. This structural form also functions as a phosphate storage compartment. The dissolution of phosphoric

acid in plasma is an important buffering system that complements the carbonic acid buffering system. Chemical energy in all cells depends on the high energy bond in ATP and (guanosine triphosphate) GTP (both triple phosphate molecules). Organic phosphate is an essential component in phospholipid membranes and nucleic acids, and is critical for several important enzyme systems.

Diagnostic Value

In oviparous species, increased phosphorus and calcium concentrations are observed during egg formation in females. Generally, these occur together and the calcium:phosphorus ratio stays above one in healthy individuals. If the calcium:phosphorus ratio is below one, renal or other disease (ie, primary or secondary hyperparathyroidism) should be investigated. Both hyperphosphatemia and hypophosphatemia have been associated with renal disease in birds. This electrolyte flux may represent acute and chronic forms of renal disease as in mammals. Alkalosis will cause flux of phosphate into the cell and an apparent hypophosphatemia. Some acidosis, such as diabetic ketoacidosis, results in catabolism of phosphorylated compounds in the cell with excretion of phosphate at the kidney and whole-body phosphorus depletion.

Total Protein

Method

There have been several articles written on refractometer versus biuret analysis of total protein.[1,41,42,50] The biuret method is a more specific chemical reaction where peptide bonds are reacted with cupric ions to form a colored product measured spectrophotometrically at 540 nm. The total protein value determined using a refractometer is frequently inaccurate in companion avian species due to interference by high concentrations of other light-refractive compounds in plasma, such as chromagens, lipids and glucose. Studies in chickens, turkeys and ducks have shown good correlation between protein concentrations obtained by refractometer[d] and biuret methods.[1,50] These species tend to have lower blood glucose values than most psittacines and smaller birds. Correlation studies in avian species with high blood glucose levels, such as pigeons, have shown marked discrepancies between refractometer and biuret methods, however, a different brand of refractometer[e] was used, that may have contributed to the difference in results.[41,42] There is marked variation in normal blood glucose levels in avian species. The biuret method is the most accurate method to quantify total protein in the clinical setting, where samples from many different species may be evaluated.

Physiology

The body contains thousands of proteins each having one or more functions. An increase in plasma proteins may be observed in egg-laying females.

Diagnostic Value

Though not adding information about specific disease etiology, total protein is important information used to establish supportive care. Decreased total protein may indicate the need for colloid (hetastarch) supplementation and incite a search for an underlying protein-losing nephropathy, enteropathy or liver failure. In cases of increased total protein, reproductive activity should first be ruled out in females and then dehydration or an underlying inflammatory disease state should be investigated.

Uric Acid

Method

The most commonly used method is the uricase method where uric acid is catalyzed by uricase to allantoin. The decrease in concentration of uric acid is measured at approximately 285 nm.

Physiology

Uric acid is the major nitrogenous waste product of birds. It is hypothesized that this has evolved due to oviparity.[40] Embryonic and fetal development occur within a closed compartment, the egg, that lacks diffusion of nutrients and waste. Uric acid is relatively inert and substantially less toxic than ammonia or urea, thus ensuring a viable hatchling. Uric acid (an oxidized form of the purine, hypoxanthine) is synthesized predominantly in the liver from purine metabolism, with a small amount of synthesis occurring in the renal tubules. Approximately 90% of uric acid is secreted in the proximal convoluted tubules in the normal bird.[20] This percentage can be markedly altered in pathologic conditions. Uric acid is passed to the cloaca and then may be retropulsed to the rectum and ceca, where it may be broken down by bacteria and reabsorbed.[7,8]

Diagnostic Value

Due to active renal tubular secretion, blood uric acid levels are not notably affected by dehydration until GFR is decreased to the point that uric acid is not moved through the tubules, which may occur in severe dehydration. Raptors and penguins have higher reference values for uric acid, and marked increases in plasma uric acid concentration may be observed postprandially.[34,44] Therefore, sampling of carnivorous birds should be performed after a 24-hour fast. Fasting also will decrease the likelihood of lipemia, which is frequently observed in postprandial samples. Contamination of the blood sample with trace amounts of urates from the skin of birds may lead to extreme elevations in uric acid measurements. Questionable samples should therefore be redrawn and the testing repeated. Once the above etiologies are ruled

out, renal disease should be assessed with urinalysis, radiography, and/or renal biopsy (see Chapter 16, Evaluating and Treating the Kidneys).

Urinalysis

Osmoregulation is accomplished by contributions from the kidneys, intestinal tract, salt glands and, to some extent, the skin and respiratory tract.[19,20] Urine can be actively retropulsed from the urodeum to the coprodeum of the cloaca and then to the rectum and potentially the large intestine, where water can be reabsorbed and electrolytes can be modified. This results in a change in the specific gravity, electrolyte concentrations and bacterial contamination of urine.

Urinalysis is indicated when there is azotemia, uratemia, polyuria/polydipsia, hematuric abnormal urates, or genitourinary masses. Birds with renal pathology will frequently have polyuria, resulting in a urine sample of adequate volume for analysis. Avian urine is generally collected free catch by removal of cage paper and thorough cleaning of the cage surface. A needle and syringe or capillary tube can be used to aspirate urine from the cage bottom and minimize fecal contamination. Ureteral catheterization has been performed, but, requires anesthesia and is difficult.[65]

Normal urine has a clear fluid component. There is variation in normal urine volume among species that are adapted to different food sources and environments.[10,19] Specific gravity in most clinically normal birds has been reported as 1.005 to 1.020, and avian urine is generally acidic.[6] Normal urine sediment is generally composed of uric acid precipitates and crystals, sloughed squamous epithelial cells, <3 WBC/40x field and <3 RBC/40x field, and low quantities of predominantly gram-positive bacteria. Bacteria present in normal samples are attributed to fecal contamination.

The majority of uric acid in avian urine exists as a white to light yellow colloidal suspension made up of small, spherical conglomerates (urates) that range in diameter from 0.5 to 15 μm.[6] The urate precipitate is composed of uric acid, sodium and/or potassium, and protein. The precipitate is not measured in the specific gravity of the urine supernatant, and therefore urine specific gravity is lower in birds and reptiles than in mammals. Any protein not reabsorbed in the proximal tubule is generally precipitated with uric acid. Assuming there is no fecal contamination, normal avian urine should not contain protein that can be detected on a urine dipstick.[32]

Needle-shaped uric acid crystals also may be observed in normal urine. Uric acid crystals polarize and can be tested chemically using the murexide test. A drop of concentrated nitric acid is added to the crystals and heated to evaporation. A drop of ammonia is then added. If uric acid is present, the liquid will turn a mauve color. Adding several drops of sodium hydroxide to a urine sample will dissolve uric acid crystals. This can facilitate the identification of casts, bacteria and cells.

Biliverdinuria, grossly apparent as green urates, is not a normal finding and is most commonly caused by bile stasis due to liver compromise. It also may be seen in birds with hemolytic anemia. Biliverdinuria associated with nephrosis has been described histologically.[53] The clinical significance of the described nephrosis is unknown. Prior to diagnosing biliverdinuria, fecal contamination should be assessed by measuring urobilinogen with a urine dipstick. A positive urobilinogen result supports fecal contamination.

Hemoglobinuria has been documented in heavy metal poisoning, specifically lead toxicosis, in Amazon, Electus and African grey parrots secondary to intravascular hemolysis.[17] Ketonuria is not observed in normal birds. Ketones have been found in the urine of migratory birds, but otherwise support a diagnosis of diabetes mellitus.[9,63] (See acetoacetate, acetone in the Analytes section).

Even in free catch samples, culture and sensitivity are indicated when bacterial infection (eg, pyuria) is suspected based on clinical presentation or urinalysis results. Renal biopsy and culture also can be performed if inflammation or infection is believed to involve the kidney (see Chapter 16, Evaluating and Treating the Kidneys).

Products Mentioned in the Text

a. Vacutainer tubes, Becton Dickinson, Franklin Lakes, NJ, USA
b. I-STAT, Heska Corporation, Fort Collins, CO, USA
c. Lactose/D-galactose assay kit, Boehringer Mannheim, Mannheim, Germany
d. AO Goldberg Refractometer, American Optical Corporation, Buffalo, NY, USA
e. Atago Refractometer, Atago Corporation, Atago Ltd, Tokyo, Japan

References and Suggested Reading

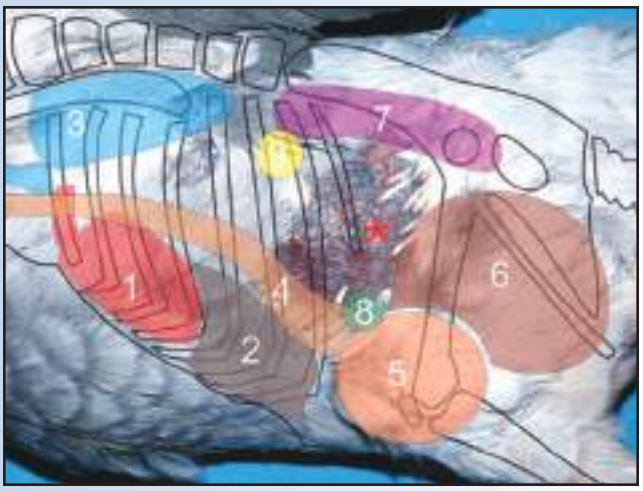
1. Andreasen CB, et al: Determination of chicken and turkey plasma and serum protein conce628nt628rations by refractometry and the biuret method. Avian Dis 33:93-96, 1989.
2. Assink HA, et al: The introduction of bromocresol purple for the determination of serum albumin on SMAC and ACA, and the standardization procedure. J Clin Chem Clin Biochem 22(10):685-692, 1984.
3. Bailey TA, et al: Age-related plasma chemistry findings in the buff-crested bustard *(Eupodotis ruficrista gindiana)*. Zentralbl Veterinarmed 45B:635-640, 1998.
4. Bonkovsky HL, Healey JF, Pohl J: Purification and characterization of heme oxygenase from chick liver. Comparison of the avian and mammalian enzymes. Eur J Biochem 189(1):155-166, 1990.
5. Brackeen GL, Dover JS, Long CL: Serum albumin. Differences in assay specificity. Nutr Clin Pract 4(6):203-205, 1989.

6. Braun EJ: Comparative renal function in birds, reptiles, and mammals. Sem Avian Exotic Pet Med 7:62-71, 1998.
7. Braun EJ: Integration of renal and gastrointestinal function. J Exp Zool 283(4-5):495-499, 1999.
8. Braun EJ, Campbell CE: Uric acid decomposition in the lower gastrointestinal tract. J Exp Zool Suppl 3:70-74, 1989.
9. Candeletta SC, et al: Diabetes mellitus associated with chronic lymphocytic pancreatitis in an African Grey Parrot. J Avian Med Surg 7:39-43, 1993.
10. Casotti G, Braun EJ: Renal anatomy in sparrows from different environments. J Morphol 243(3):283-291, 2000.
11. Clubb S, Schubot R, Joyner K: Hematological and serum biochemistry reference intervals in juvenile cockatoos. J Avian Med Surg 5:5-16, 1991.
12. Clubb S, Schubot R, Joyner K: Hematological and serum biochemistry reference intervals in juvenile eclectus parrots *(Eclectus roratus)*. J Avian Med Surg 4:218-225, 1991.
13. Clubb S, Schubot R, Joyner K: Hematological and serum biochemistry reference intervals in juvenile macaws. J Avian Med Surg 5:154-162, 1991.
14. Coleman CW: Bile duct carcinoma and cloacal prolapse in an orange-winged Amazon parrot *(Amazona amazonica)*. J Avian Med Surg, 1991, pp 87-89.
15. Cray C, Tatum LM: Applications of protein electrophoresis in avian diagnostics. J Avian Med Surg 12:4-10, 1998.
16. Dabbert CB, Powell KC: Serum enzymes as indicators of capture myopathy in mallards *(Anas platyrhynchos)*. J Wildl Dis 29(2):304-309, 1993.
17. Dumonceaux G, Harrison GJ: Toxins. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, Wingers Publishing, 1994, pp 1030-1052.
18. Elangbam CS, Panciera RJ: Cholangiocarcinoma in a blue-fronted Amazon parrot *(Amazona estiva)*. Avian Dis 1988, pp 594-596.
19. Goldstein DL, Braun EJ: Lower intestinal modification of ureteral urine in hydrated house sparrows. Am J Physiol 250(1 Pt 2):R89-R95, 1986.
20. Goldstein DL, Skadhauge E: Renal and extrarenal regulation of body fluid composition. *In* Whittow GC (ed): Sturkie's Avian Physiology. San Diego, Academic Press, 2000, pp 265-298.
21. Gonzalez D: Comparative study of ferritins from dove *(Columba oena)* and chicken *(Gallus domesticus)* livers. Comp Biochem Physiol 76:568, 1983.
22. Harr KE: Clinical chemistry of companion avian species: a review. Vet Clin Pathol 31(3):140-151, 2002.
23. Harrison GJ, Ritchie BW: Making distinctions in the physical examination. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 144-175.
24. Hawkey C, Hart MG: An analysis of the incidence of hyperfibrinogenemia in birds with bacterial infections. Avian Pathol 17:427-432, 1988.
25. Hazelwood RL: Pancreas. *In* Whittow GC (ed): Sturkie's Avian Physiology. San Diego, Academic Press, 2000, pp 539-556.
26. Henderson AR, Moss DW: Enzymes. *In* Burtis CA, Ashwood ER (eds): Tietz Fundamentals of Clinical Chemistry. Philadelphia, WB Saunders, 2001, pp 352-389.
27. Hillyer EV, et al: Bile duct carcinoma in two out of ten Amazon parrots with cloacal papillomas. J Avian Med Surg, 1991, pp 87-89.
28. Hochleithner M: Biochemistries. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 223-245.
29. Hoeffer H: Bile acid testing in psittacine birds. Sem Avian Exotic Pet Med 3:33-37, 1994.
30. Jaensch MJ, Cullen L, Raidal SR: Assessment of liver function in galahs/cockatoos *(Eolophus roseicapillus)* after partial hepatectomy: a comparison of plasma enzyme concentrations, serum bile acid levels, and galactose clearance tests. J Avian Med Surg 14:164-171, 2000.
31. James SB, Raphael BL, Clippinger T: Diagnosis and treatment of hepatic lipidosis in a barred owl *(Strix varia)*. J Avian Med Surg 14:268-272, 2002.
32. Janes DN, Braun EJ: Urinary protein excretion in red jungle fowl *(Gallus gallus)*. Comp Biochem Physiol A Physiol 118(4):1273-1275, 1997.
33. Kolling K, Hofmeier A, Merkenschlager M: Ionized calcium in the blood of domestic chickens: dependence on the laying cycle. Zentralbl Veterinarmed 39A:115-120, 1992.
34. Kolmstetter CM, Ramsay ER: Effects of feeding on plasma uric acid and urea concentrations in blackfooted penguins *(Sphenicus demersus)*. J Avian Med Surg 14:177-179, 2000.
35. Kramer JW, Hoffmann WE: Clinical Enzymology. *In* Kaneko JJ, Harvey JW, Bruss ML (eds): Clinical Biochemistry of Domestic Animals. San Diego, Academic Press, 1997, pp 303-325.
36. Lumeij JT: Plasma urea, creatinine, and uric acid concentrations in response to dehydration in racing pigeons *(Columba livia domestica)*. Avian Pathol 16:377-382, 1987.
37. Lumeij JT: The diagnostic value of plasma proteins and non-protein nitrogen substances in birds. Vet Q 9(3):262-268, 1987.
38. Lumeij JT: Relation of plasma calcium to toal protein and albumin in African grey *(Psittacus erithacus)* and Amazon (*Amazona* sp.) parrots. Avian Pathol 19:661-667, 1990.
39. Lumeij JT: Hepatology. In Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 522-537.
40. Lumeij JT: Avian Clinical Biochemistry. *In* Kaneko JJ, Harvey JW, Bruss ML (eds): Clinical Biochemistry of Domestic Animals. San Diego, Academic Press, 1998, pp 857-884.
41. Lumeij JT, de Bruijne JJ: Evaluation of refractometric method for the determination of total protein in avian plasma or serum. Avian Pathol 14:441-444, 1985.
42. Lumeij JT, Maclean B: Total protein determination in pigeon plasma and serum: comparison of refractometric methods with biuret method. J Avian Med Surg 10:150-152, 1996.
43. Lumeij JT, Remple JD: Plasma bile acid concentrations in response to feeding in peregrine falcons *(Falco peregrinus)*. Avian Dis 36(4):1060-1062, 1992.
44. Lumeij JT, Remple JD: Plasma urea, creatinine, and uric acid concentrations in relation to feeding in peregrine falcons *(Falco peregrinus)*. Avian Pathol 20:79-83, 2002.
45. Lumeij JT, Westerhof I: Blood chemistry for the diagnosis of hepatobiliary disease in birds. A review. Vet Q 9(3):255-261, 1987.
46. Lumeij JT, Remple JD, Riddle KE: Relationship of plasma total protein and albumin to total calcium in peregrine falcons *(Falco peregrinus)*. Avian Pathol 22:183-188, 1993.
47. Lumeij JT, et al: Changes in plasma chemistry after drug-induced liver disease or muscle necrosis in racing pigeons *(Columba livia domestica)*. Avian Pathol 17:865-874, 1988.
48. Meyer DJ, Harvey JW: Veterinary Laboratory Medicine: Interpretation and Diagnosis. Philadelphia, WB Saunders, 1992.
49. Mizobe M, et al: High-performance liquid chromatographic analysis of bilirubin and biliverdin from jaundiced broilers. J Vet Med Sci 59(8):677-680, 1997.
50. Morgan GW, Thaxton P, Eden FW: Estimation of protein content in the plasma of young chickens by a refractometric method. Poult Sci 54:1312-1314, 1975.
51. Munson L, Clyde VL, Orosz SE: Severe hepatic fibrosis and bile duct hyperplasia in four Amazon parrots. J Avian Med and Surg 10:252-257, 1996.
52. Newman DJ and Price CP: Nonprotein nitrogen metabolites. *In* Burtis CA, Ashwood ER (eds): Tietz Fundamentals of Clinical Chemistry. Philadelphia, WB Saunders, 2001, pp 414-426.
53. Phalen DN: The avian urinary system: form, function, diseases. Proc Assoc Avian Vet, 1990, pp 44-57.
54. Phalen DN, Homco L, Jaeger L: Investigation into the etiologic agent of internal papillomatosis of parrots and ultrasonographic and serum chemical changes in Amazon parrots with bile duct carcinomas. Proc Assoc Avian Vet, 1997, pp53-56.
55. Sayari A, Mejdoub H, Gargouri Y: Characterization of turkey pancreatic lipase. Biochimie 82(2):153-159, 2000.
56. Simkiss K: Calcium metabolism in the laying bird. *In* Simkiss K (ed): Calcium in Reproductive Physiology: A Comparative Study of Vertebrates. New York, Reinhold Publishing, 1967, pp 155-197.
57. Smith JE: Iron metabolism and its disorders. *In* Kaneko JJ, Harvey JW, Bruss ML (eds): Clinical Biochemistry of Domestic Animals. San Diego, Academic Press, 1998, pp 223-240.
58. Spano JS, et al: Comparative albumin determinations in ducks, chickens, and turkeys by electrophoretic and dye-binding methods. Am J Vet Res 49(3):325-326, 1988.
59. Stokol T, Tarrant JM, Scarlett JM: Overestimation of canine albumin concentration with the bromcresol green method in heparinized plasma samples. Vet Clin Pathol 30(4):170-176, 2001.
60. Tatum LM, et al: Protein electrophoresis as a diagnostic and prognostic tool in raptor medicine. J Zoo Wildl Med 31(4):497-502, 2000.
61. Vajro P, Thaler MM, Blanckaert N: Bile pigment composition and bilirubin esterification in the developing chick. Pediatr Res 38(3):349-355, 1995.
62. Verstappen FA, Lumeij JT, Bronneberg RG: Plasma chemistry reference values in ostriches. J Wildl Dis 38(1):154-159, 2002.
63. Wallner-Pendletom EA, Rogers D, Epple A: Diabetes mellitus in a red-tailed hawk *(Buteo jamaicensis)*. Avian Pathol 22:631-635, 1993.
64. Walsh RL: A comparison of dye binding methods for albumin determination: the effects of abnormal sera, reaction times, acute phase reactants and albumin standards. Clin Biochem 16(3):178-181, 1983.
65. Wideman RF, Jr., Braun EJ: Ureteral urine collection from anesthetized domestic fowl. Lab Anim Sci 32(3):298-301, 1982.
66. Worell AB: Further investigation in Ramphastids concerning hemochromatosis. Proc Assoc Avian Vet, 1993, pp 98-107.

CHAPTER
24

Diagnostic Value of Endoscopy and Biopsy

MICHAEL LIERZ, Dr med vet, MRCVS, Certified Specialist Poultry-Avian Medicine, Certified Species Conservation



M. Lierz, modified by Michael Pees

Endoscopy has been used in avian medicine since the 1970s, primarily for determining gender in birds that are not sexually dimorphic. With the advent of acceptable inhalation anesthetics, the safety of this procedure has increased while the stress to the patient has decreased. Unless otherwise noted, the descriptions in this chapter are of psittacine patients and related procedures using rigid endoscopes (Fig 24.1).

Endoscopes are fiberoptic probes that utilize magnification to facilitate visual inspection of internal body structures. Endoscopy can complement imaging modalities such as radiography and ultrasonography. Direct visualization of internal structures by the endoscope affords numerous advantages as a diagnostic tool (Tables 24.1-24.4). There are instances where the use of a semi-rigid or a flexible endoscope is advantageous. Rigid endoscopes are available in a variety of viewing angles (Figs 24.3a-b).

While most endoscopic procedures are minimally invasive, they do require anesthesia for restraint and pain management. Endoscopic visualization performed by an experienced practitioner carries minimal risk. However, the risk increases when more invasive procedures (biopsy, surgery) are performed (Table 24.5). In order to minimize the risk to the patient and maximize the diagnostic information, the practitioner should become proficient with endoscopic location and visualization of internal structures. Cadavers are excellent training tools, as they allow immediate comparison between what is visualized endoscopically and the actual organs or gross necropsy (see Chapter 26, Diagnostic Value of Necropsy).

Table 24.1 | Characteristics of Endoscopes
(see Figs 24.1, 24.2a-c)

Endoscopes	Comments
Rigid	• Best choice for primary scope • 2.7 mm diameter, 30° viewing angle, 19 cm length recommended • 1.9 mm diameter provides acceptable imaging for most patients and procedures • 4.0 mm provides best imaging but is too large for some avian patients • Quality varies between manufacturers (Fig 24.1)
Flexible	• Recommended for a second scope • Preferred for evaluating proventriculus, ventriculus and oviduct • 3 mm diameter is most useful, has three ports to accommodate the lens, fiberoptic light bundle, instruments • Quality differs between manufacturers (Figs 24.2a-c)
Semi-rigid[a]	• 1.2 mm diameter ideal for small patients, small orifices • See Chapter 1, Clinical Practice, Fig 1.13

Note: Diameters of scopes do not include sheaths and their working channels.

Table 24.2 | Light Sources

Light source	Comments
Halogen: xenon	• Gives the most light
Computed flash generator	• Necessary for 35-mm slide documentation
Light cable	• Must be compatible with the light source
Otoscope handle	• Portable scopes are available that attach to otoscopes • Less expensive, most provide adequate illumination

Table 24.3 | Lenses for Rigid Endoscopes

Lense	Comments
Single lens	Lower-quality image
	Less expensive
Rod lens	Best-quality image
	More expensive

Table 24.4 | Viewing Angles for Rigid Endoscopes
(see Fig 24.3a-e)

Angle	Comments
0°	• Natural straightforward orientation • Good for examination of the ear canal, oral cavity, crop, trachea • Superior for photodocumentation
30°	• Forward oblique angle • Allows visualization of a larger area by rotating the scope

Table 24.5 | Surgical Equipment Used during Endoscopy

- Small, curved mosquito hemostats
- Scalpel, #15 blade
- Small scissors
- Tissue-handling forceps
- Needle holder
- Suture

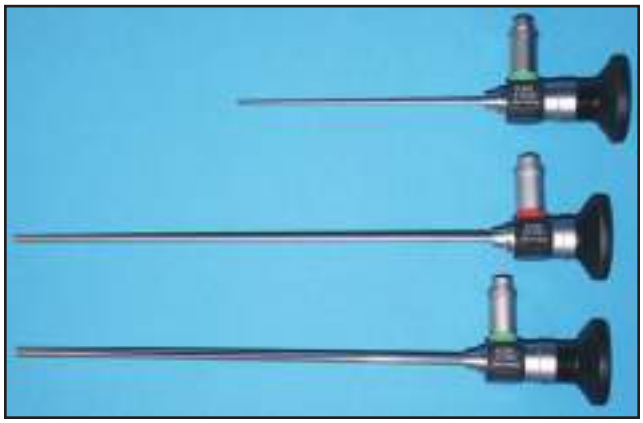
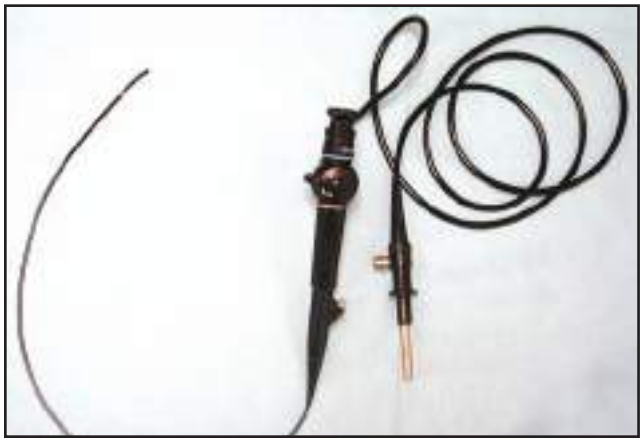


Fig 24.1 | Rigid endoscopes most commonly used in avian endoscopy. Top to bottom: 1.9 mm 0°, 2.7 mm 30°, 4 mm 0°.

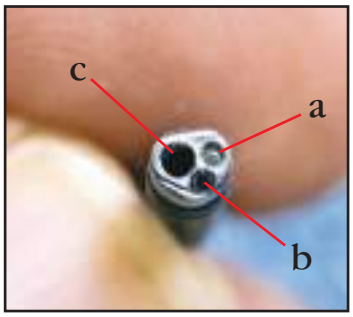


Greg J. Harrison

Fig 24.2a | A flexible endoscope allows more maneuverability in viewing lungs and the gastrointestinal system.

Greg J. Harrison

Fig 24.2b | The 3-mm measurement of a small, flexible endoscope.



Greg J. Harrison

Fig 24.2c | The fiberoptic bundle (a) for light and visualization, the air or flushing port (b), and an instrument port (c) of a 3-mm flexible endoscope.

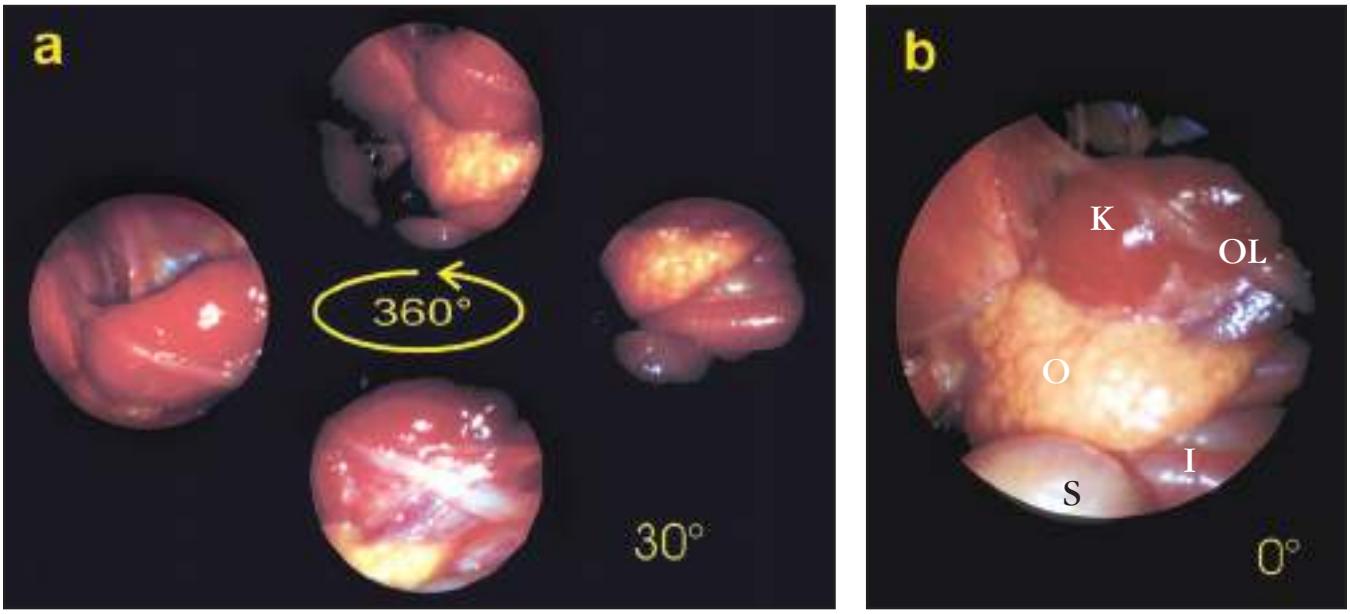


Fig 24.3 | a) The view as seen through a 2.7-mm 30° scope. Turning the scope around its optical axis, a panoramic view is possible. **b)** Kidney (K), ovary (O), Ovarian ligament (OL) Intestine (I) and Spleen (S) seen with a 4-mm 0° scope. Only a straightforward view of the organs is possible.

Fig 24.4 | Basic equipment for endoscopic-guided multiple-entry surgery in birds. Top to bottom: monopolar sling, scissor, grasping forceps, bipolar forceps for coagulations, trocars.

Table 24.6 | Endoscopic Accessories (see Figs 24.5a-b)

Accessory	Comments
Flexible biopsy forceps	• Must match working channel of scope to allow use of forceps without obstructing view • 1.8 mm recommended • Elliptical cup (deeper cuts) • Round cup (recommended for testicular, kidney biopsy)
Flexible grasping forceps	• Removal of foreign bodies, granulomas
Infusion/aspiration needle[a]	• 22-gauge flexible needle inside Teflon catheter[b] • Can be used in the working channel • Good for aspirating fluid from cysts and direct application of medications (Figs 24.5a,b)
Software	• Systems are available to store endoscopic images in medical records
Endoscopic-guided surgery equipment	• See Fig 24.4
Cleaning and sterilization	• Follow manufacturer's instructions • Adequate time between patients is necessary to properly sterilize all equipment

Most of the body can be examined using endoscopy. Apart from the visual assessment, tissue sampling is possible using the working channel and biopsy tools (Table 24.6). A sterile sheath is particularly valuable for microbiological sampling (especially of the deeper respiratory system). The sheath prevents contamination of the sample.

New surgical procedures using multiple endoscopes, multiple sites and radical new instruments are being developed (Fig 24.4). The use of a flexible needle can have multiple applications (Figs 24.5a,b).

Preparation and Contraindications

Duration of pre-endoscopic fasting will parallel that of presurgical fasting for similar procedures. Longer fasts may be required to facilitate visualization of abdominal organs. General anesthetic techniques and requirements are discussed in Chapter 33, Updates in Anesthesia and Monitoring.

Birds with bleeding dyscrasias are at heightened surgical risk, especially when an organ biopsy is performed. The

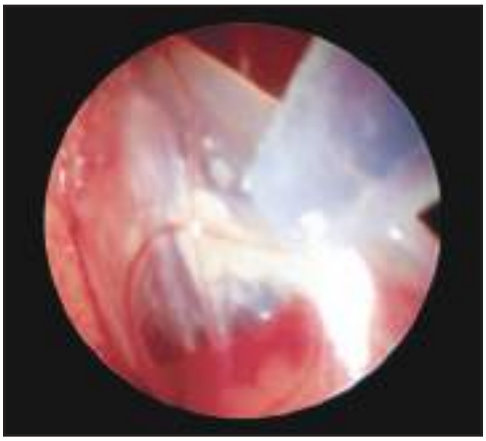


Fig 24.5a | The Teflon tube of the flexible needle is an excellent tool for the endoscopic-guided application of medicine directly to lesions such as this air sac granuloma.

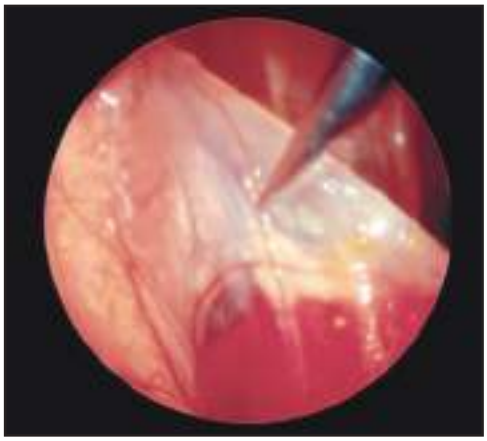


Fig 24.5b | The flexible needle can be used for aspiration, biopsies, penetrations and application of medicine. Here, it is ready to penetrate a granuloma for a needle aspirate.

presence of cystic structures within the coelom, organomegaly or the presence of any fluid will complicate the procedure and increase the risk to the patient. Fluid should be carefully drained or reduced with diuretics prior to endoscopy. Obesity often reduces the view in the body cavity, increasing the risk of organ damage.

Coelomic Laparoscopy Studies

LEFT LATERAL APPROACH

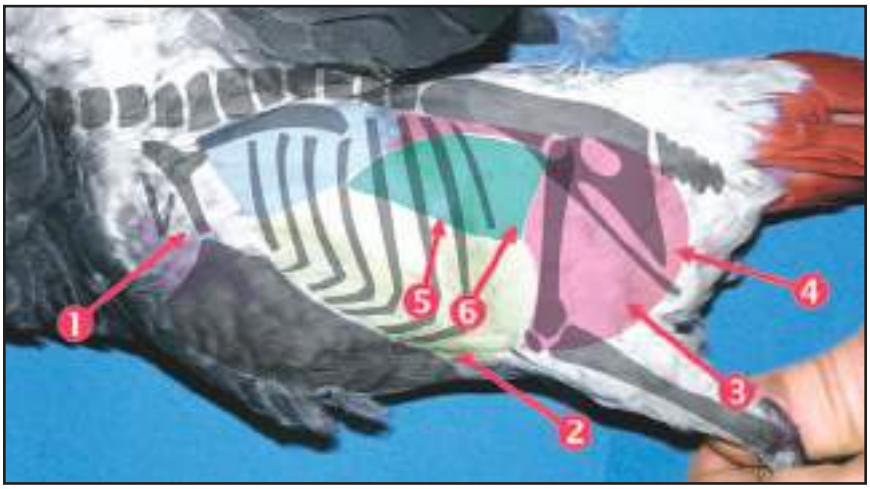
The endoscopic approach to the coelomic cavity depends on the diagnostic goal of the procedure (Fig 24.6) and the results of previous imaging studies. The approach caudal to the last rib is ideal for exploration of the entire coelom (Fig 24.7). Due to the presence of an ovary in most avian species on only the left side, approach from the left is generally utilized to allow visualization of female reproductive structures (see the section on gonads later in this chapter). The anesthetized bird is placed on its right side, with the left wing extended dorso cranially. The left leg may be pulled either cranially or caudally. The following description is for the approach with the leg extended caudally (Fig 24.8).

Orientation to the surgical site is provided in Figs 24.9-24.11. A small incision is made in the skin, followed by a caudal reflection of the muscle layers with curved forceps. Penetration into the air sac is accompanied by a palpable and sometimes audible pop. The use of blunt instruments for this penetration cannot be overemphasized. Sharp instruments may damage underlying tissue. The caudal thoracic air sac is most often penetrated, however, the abdominal or cranial thoracic air sacs also may be entered (Figs 24.12a,b). Some clinicians prefer to enter between the ribs. This is best accomplished by using a curved mosquito hemostat to elevate the ribs prior to penetration. However, using this technique the intercostal muscle is damaged, which is a disadvantage. The direction of penetration should be toward the cranial rib (remaining laterally positioned) to avoid the liver. The scope is stabilized with the tips of the forefinger and thumb while the hand rests on the table (Fig 24.13).

The scope is advanced between the legs of the forceps. The air sacs will then be visible. Clear air sacs allow visualization of the gonad, the adrenal gland, and the cranial division of the kidney, through the abdominal air sac (Fig 24.14). Cranial to this triad is the left lung. In larger birds, the scope may be advanced through the ostium between the lung and air sac to examine the bronchi (Figs 24.15-24.17). The medial lung, heart and liver can be seen as the scope advances cranially into the cranial thoracic air sac.

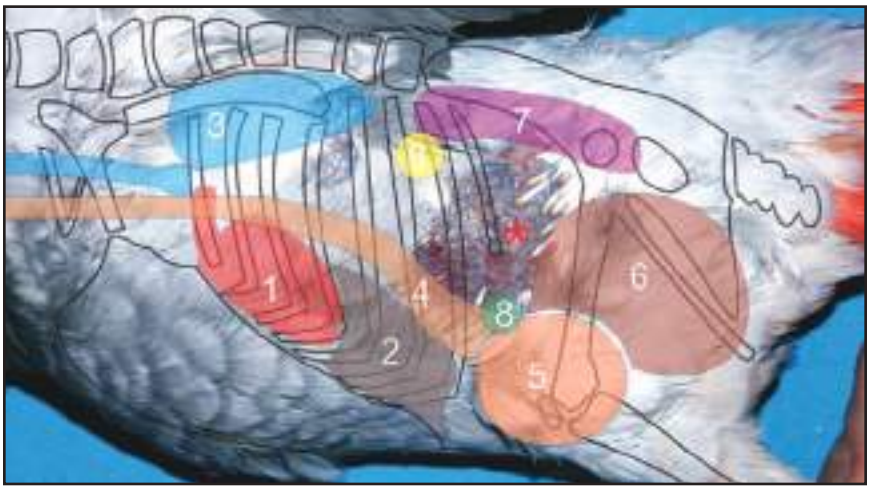
The ureter, uterus, and ductus deferens are seen ventral to the kidney, and intestinal loops come into view if the scope is directed ventrally from the original entry site. The proventriculus/ventriculus, liver and often the spleen may be visualized when the scope is directed cranioventrally. The punctured air sacs close rapidly and heal uneventfully. The skin incision can be closed with sutures or tissue glue.

A similar approach to the coelom is made by entering caudal to the pelvic limb, which is pulled cranially. The incision is made caudal to the last rib. With this approach, there is less chance of entering the cranial thoracic air sac.



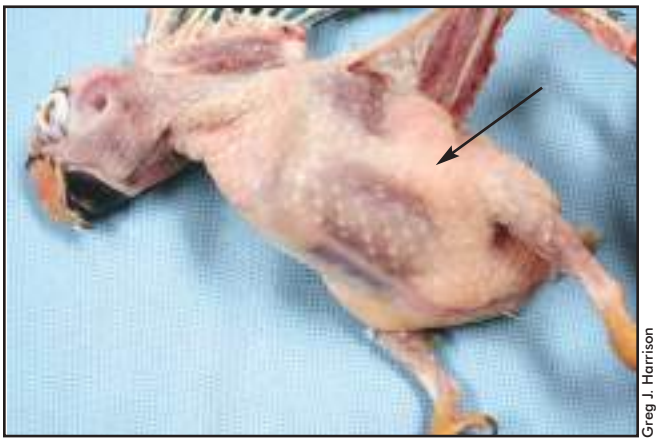
M. Lierz, modified by Michael Pees

Fig 24.6 | The various endoscopic entry sites overlying the artist's rendition of bones with colored areas representing the various air sacs encountered in endoscopy. Blue represents the intraclavicular, yellow the cranial thoracic, green the caudal thoracic and orange the abdominal air sacs.



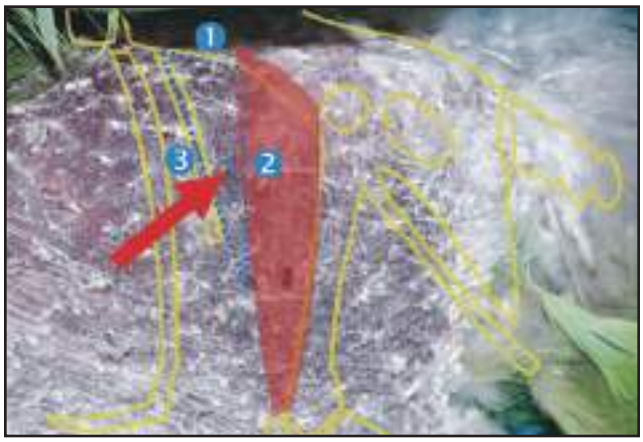
M. Lierz, modified by Michael Pees

Fig 24.7 | An artist's rendition of the organs encountered in endoscopy from the left lateral aspect. 1) heart 2) liver 3) trachea and lungs 4) proventriculus 5) ventriculus 6) intestines 7) kidney 8) spleen 9) adrenal-gonad area. The red star is a typical entry location.



Greg J. Harrison

Fig 24.8 | Right lateral recumbency, left leg caudal. This bird is malpositioned; not being in a true lateral, which is critical for organ relationships. Secondly, it can be seen that in this blue crowned conure the area in front of the leg has a thick fat pad (arrow) that has to be penetrated to reach the musculature.



M. Lierz, modified by Michael Pees

Fig 24.9 | The middle of a triangle formed by the spine (1), m. iliotibialis (2) and last rib (3); the red arrow represents the most common lateral point of entry for laparoscopy.

Fig 24.10 | The muscle iliotibialis (a) overlies the point of entry, demonstrated on a dead bird with skin removed.

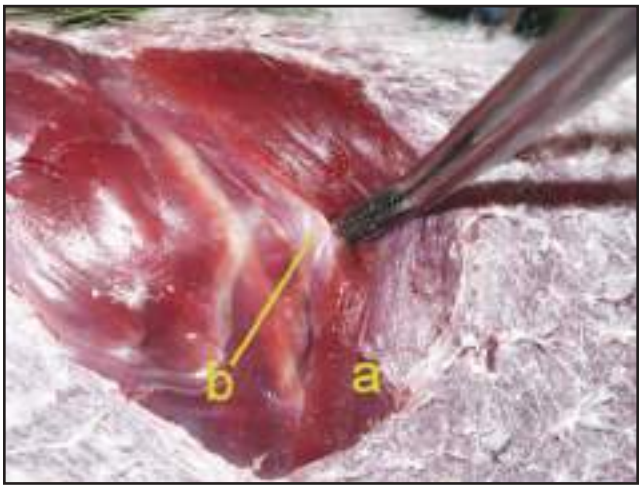
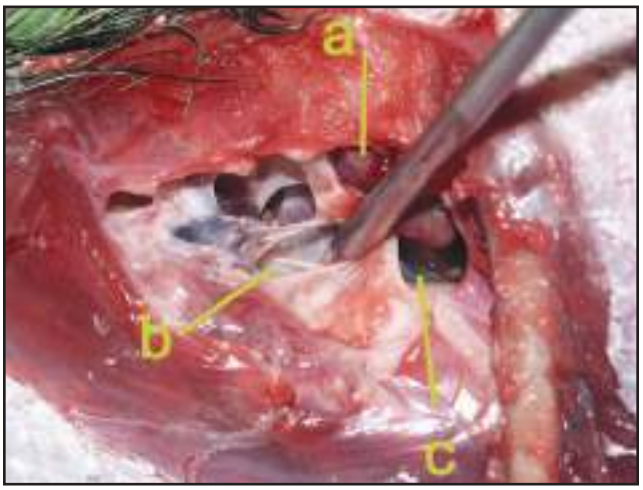
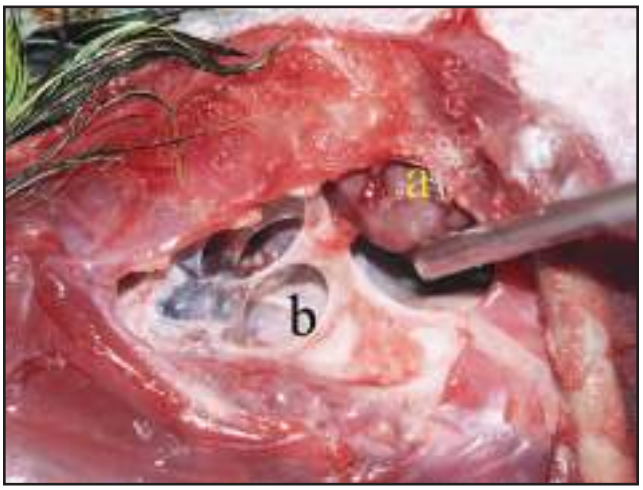


Fig 24.11 | Using a curved forceps, the *m. iliotibialis* (a) is reflected caudally and the underlying fascia is penetrated caudal to the last rib (b), as is demonstrated here on a dead bird with skin removed.





Figs 24.12a,b | Entering the body cavity caudal to the last rib usually places the scope into the caudal thoracic air sac (b). Changing the route of penetration slightly, the scope is guided into the abdominal air sac (c) where the kidney (a) is located. One must penetrate the confluent wall of the medial aspect of the caudal thoracic air sac and the left lateral wall of the abdominal air sac.

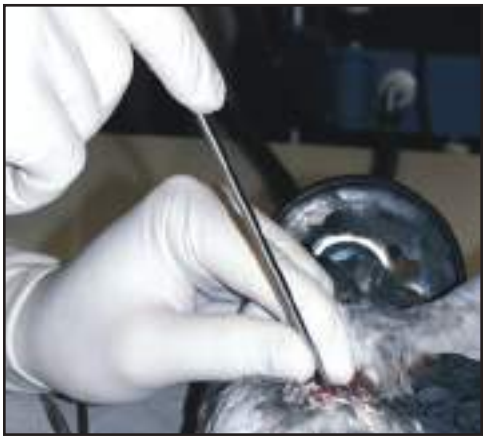


Fig 24.13 | Correct anchoring of the tip of the scope. The hand should always be in contact with the bird while the wrist is rested on the table.

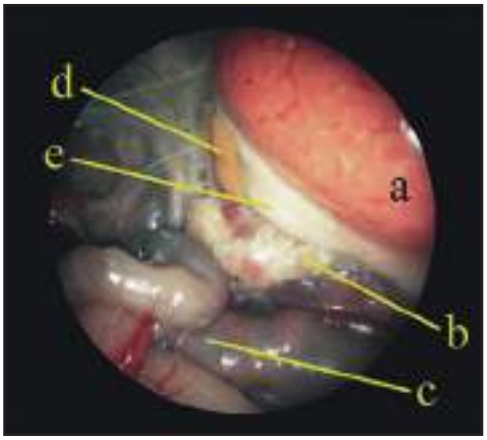


Fig 24.14 | View into the abdominal air sac. Kidney (a), ovary (b), intestine (c), adrenal gland (d), ureter and oviduct (e).

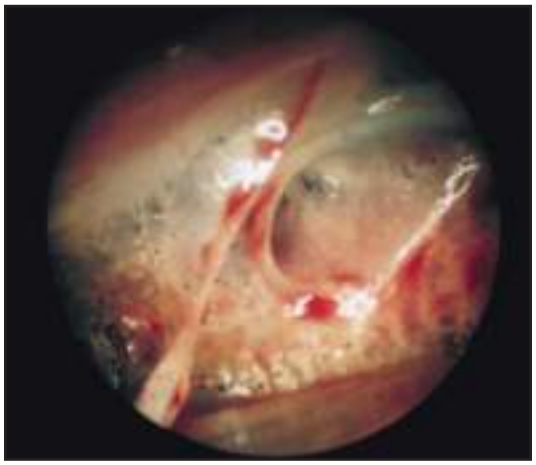


Fig 24.15 | The opening from the caudal thoracic air sac into the lung (aperta) allows retrograde endoscopy of internal structures of the lung.

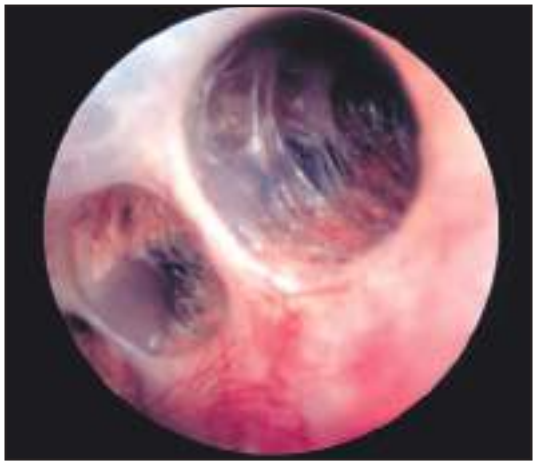


Fig 24.16 | Lung tissue viewed from the caudal thoracic air sac. The bronchi and lung parenchyma are clearly visible.

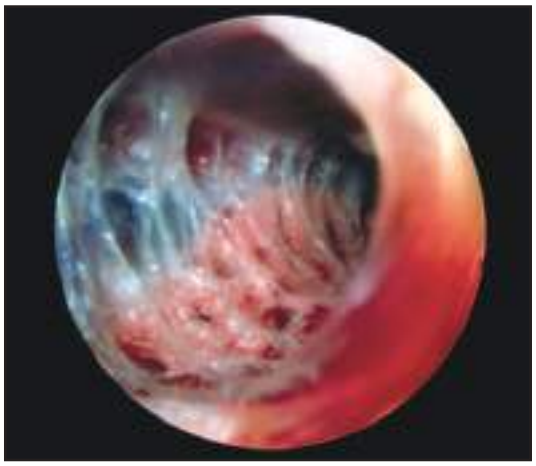


Fig 24.17 | The endoscope has been advanced into the horizontal secondary bronchi that branches from the left or right primary bronchi. The honeycomb structure of the lungs is seen. This view cannot be appreciated via the trachea due to the diameter restrictions of the syrinx, the fragility of the primary bronchi and the acute angle at the origin of the secondary bronchi.

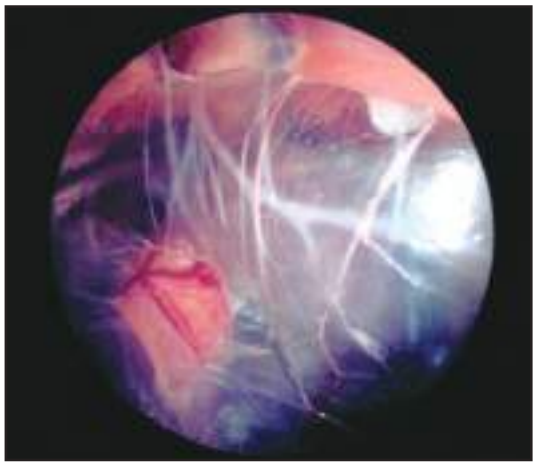


Fig 24.18 | The ideal air sac is transparent. Minor blood and lymph vessels are commonly visible in pet and aviary birds.

VENTRAL MIDLINE APPROACH

The bird is positioned in dorsal recumbency and a ventral midline approach to the coelom is made. A layer of fat may be present just under the linea alba in the area directly caudal to the sternum. Care must be taken on smaller birds not to inadvertently penetrate the duodenum or pancreas. The duodenum, pancreas and central liver can be examined from this approach.

Air Sacs

Normally the air sacs are transparent, although a few vessels may be present (Fig 24.18). Fatty infiltrates may be noted during routine examination without associated pathology. Opacity and small vessels in the wall of an air sac are early signs of inflammation (Fig 24.19). Other abnormalities of the air sacs include increased vascularity, thickened walls and granulomas (Fig 24.20). These changes may be due to infectious processes, or to inhalation of respiratory irritants (ie, smoke, volatile chemicals). In some cases, a definitive diagnosis can be made from visualization, cytology and/or biopsy of air sac lesions (Figs 24.21a-c). Removal or debulking such lesions has been described using laser and radiosurgery via the endoscope.

Lungs and Bronchi

The lungs are dark pink with a prominent reticular pattern. Within the lungs, the anastamosing bronchi are visible (see Figs 24.16, 24.17). Pneumonia will obscure the normally well-defined parenchymal pattern of the lung. A yellow discoloration of the lung tissue is often noted with pneumonia (Fig 24.22). Anthracosis (focal black

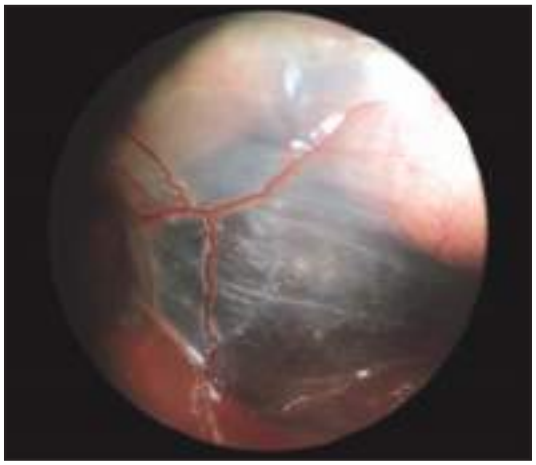


Fig 24.19 | Prominent vessels in the air sac, opacity or small granulomas are signs of infections and/or irritation from environmental contaminants (smoke, volatile chemicals).

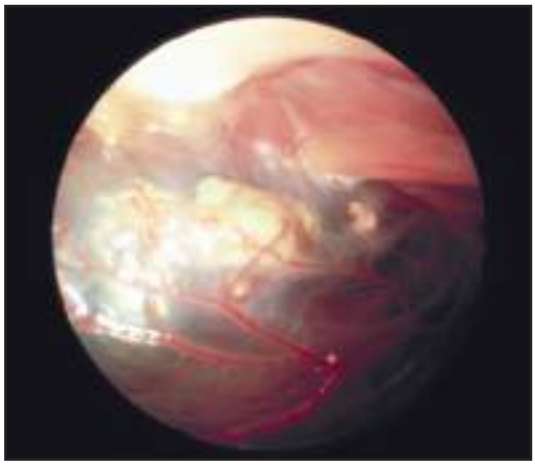


Fig 24.20 | Granulomas are forming in this case of air sacculitis.

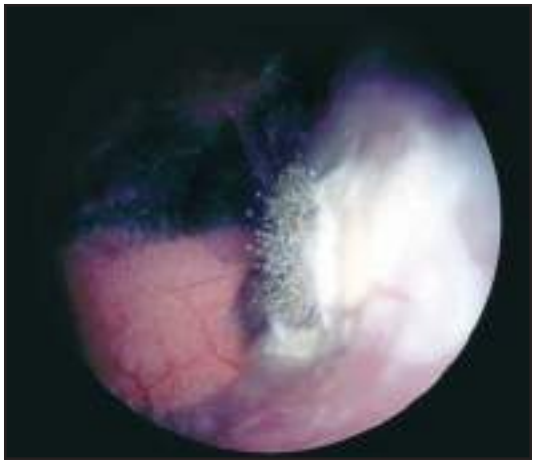


Fig 24.21a | Fruiting aspergilloma in the air sac. This presentation carries a guarded prognosis.

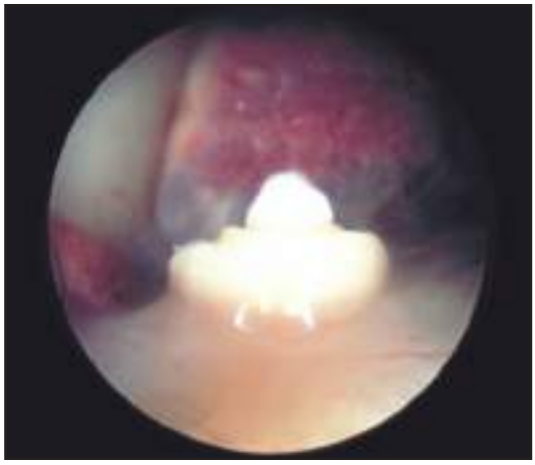


Fig 24.21b | Active aspergilloma in an air sac with fluid exudate.

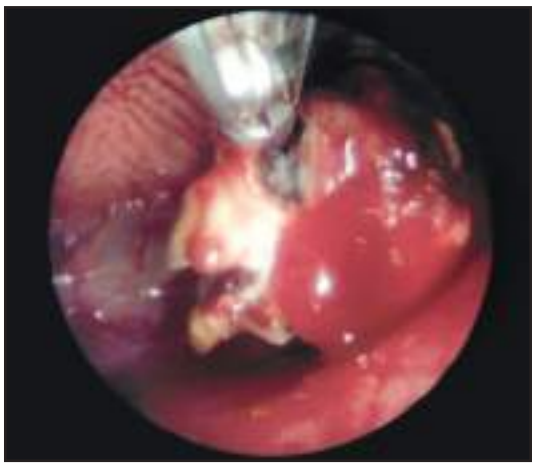


Fig 24.21c | Removal of an old aspergilloma using a biopsy forceps.

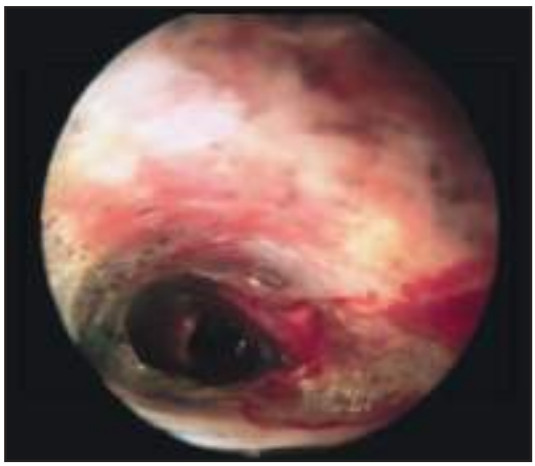


Fig 24.22 | Internal lung tissue of a bird with dyspnea, viewed from the caudal thoracic air sac. Yellow areas and the loss of the typical lung parenchyma are signs of pneumonia. A biopsy to aid in specific diagnosis and treatment is highly recommended. The black spots are soot (anthracosis) and can be found in birds from smokers or cities.

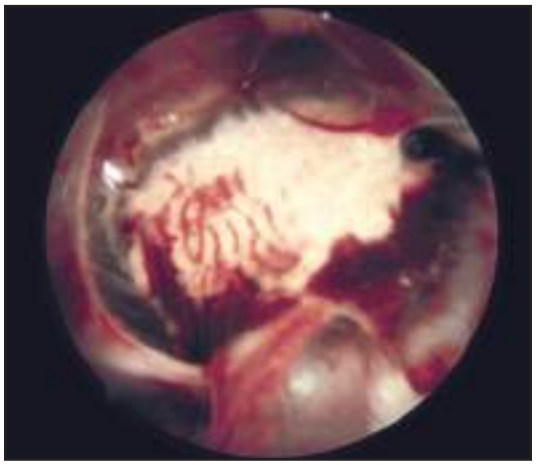


Fig 24.23 | Post-traumatic hemorrhage of the lung.

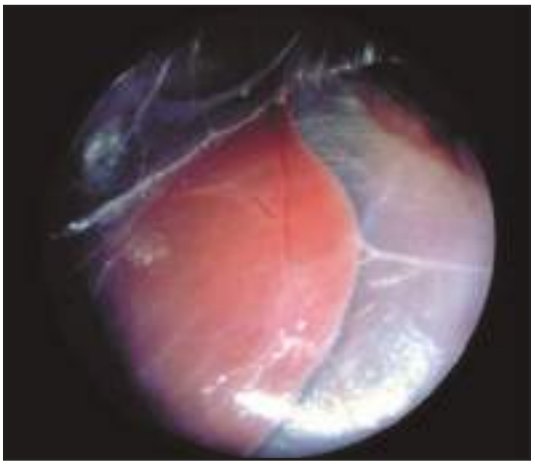


Fig 24.24 | The normal liver is a brown-red homogeneous color with a sharp border.

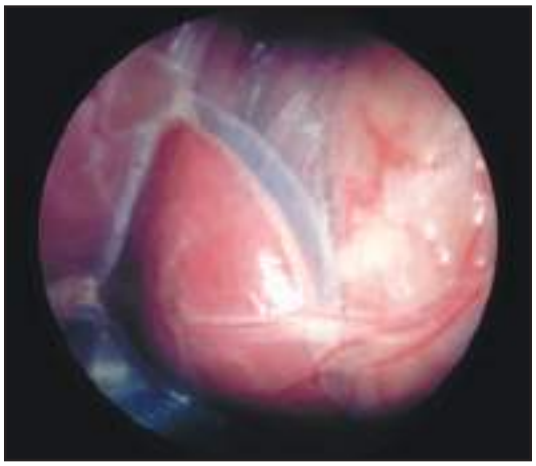


Fig 24.25 | A rounded liver border indicates an enlarged liver. A liver biopsy is often indicated to identify the nature of the hepatomegaly.

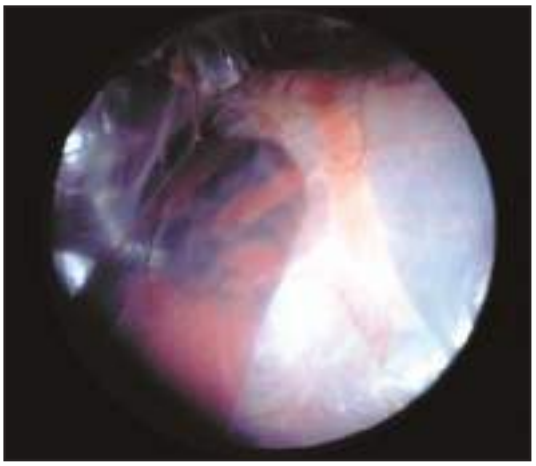


Fig 24.26 | Hematoma of the liver after trauma.

spots) is regularly found in birds from cities, industrial areas or the homes of smokers (Fig 24.22). Bleeding from trauma can be diagnosed by endoscopic examination (Fig 24.23).

Proventriculus, Ventriculus — Serosal Examination

The proventriculus is an elongated, usually white organ located in the ventral coelom, surrounded by the abdominal air sac and the liver. The surface appearance and size of the proventriculus are diagnostically important. An enlarged proventriculus with a glossy surface might indicate proventricular dilatation disease (PDD). Focal bleeding may indicate foreign bodies or infections. The ventriculus cannot always be visualized. In birds with a muscular ventriculus, abnormalities are seldom discernible endoscopically (see Chapter 26, Diagnostic Value of Necropsy).

Liver

The liver is a large organ of uniform brownish red color. The liver border tapers to an edge (Fig 24.24). A rounded liver border is not normal and may indicate infection or hepatic lipidosis (Fig 24.25). The liver color changes to yellow with fatty liver. Focal bleeding in the liver appears bright red, while hemosiderosis appears dark red that over time can turn black in color (Fig 24.26). Multiple white foci represent necrosis, abscesses or neoplasia (Figs 24.27, 24.28). Pseudomembranous infiltrates of the liver capsule and air sacs may also be due to infection, inflammation or neoplasia (Fig 24.29). Liver biopsies offer very valuable diagnostic information (see Chapter 15, Evaluating and Treating the Liver).

Heart and Pericardium

The lateral approach through the caudal thoracic air sac into the cranial thoracic air sac allows the visualization of the heart and pericardium. Pericardial effusions can be drained utilizing this approach. The normal pericardium is transparent (Fig 24.30a). A milky pericardium is the result of pericarditis. The presence of fat at the heart

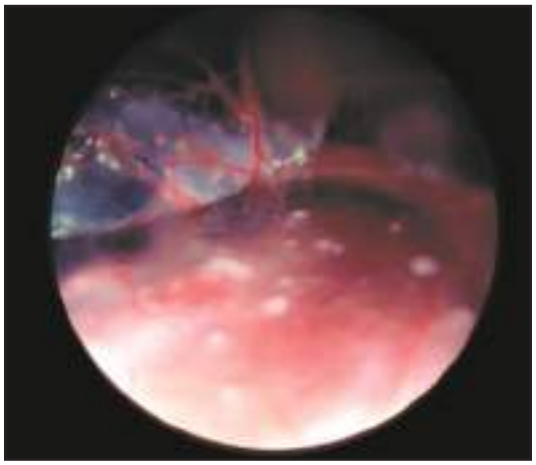


Fig 24.27 | White-yellow spots on the liver, diagnosed as abscesses. Neoplasias have a similar appearance.

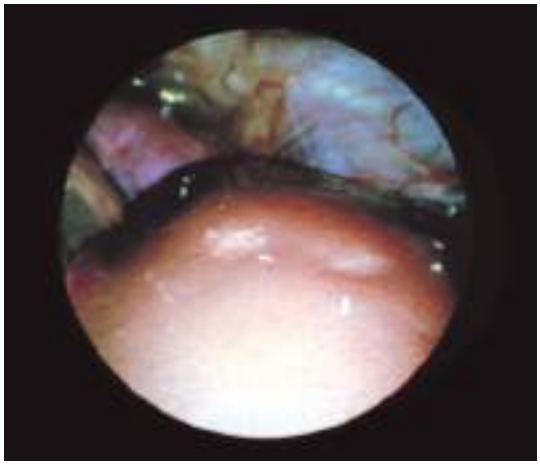


Fig 24.28 | Diffuse necrosis of the liver (histomoniasis).

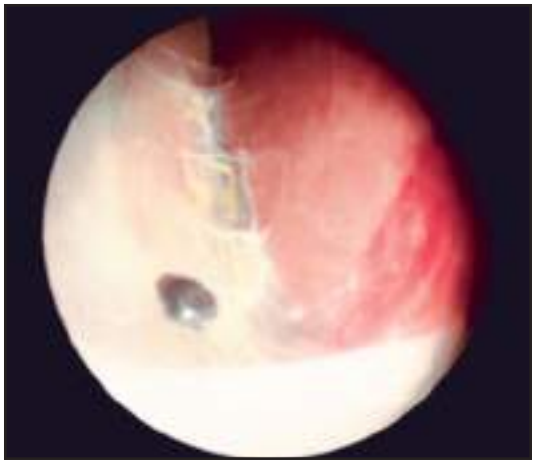


Fig 24.29 | Yellow, caseated material present on the liver and air sac due to a bacterial infection.

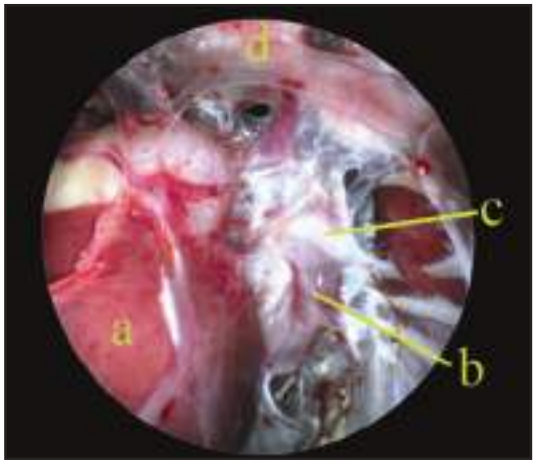


Fig 24.30a | Liver (a), heart pericardium (b), heart fat (c), lung (d) as seen in a normal bird.

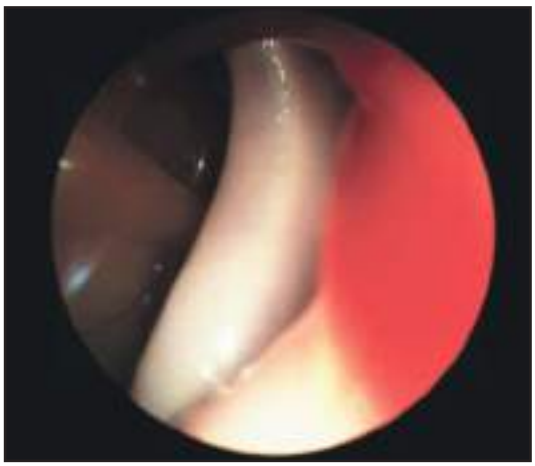


Fig 24.30b | The aorta branching into the carotid artery is seen lying between the costal musculature, the base of the heart and the returning jugular vein.

base and heart apex is normal. An absence of fat is a sign of starvation or chronic disease. The main heart vessels are visible at the heart base as thick white tubes with regular pulses (Fig 24.30b). The cardiac nerve supply can be found emanating from the thoracic vertebrae.

Kidney

The avian kidney is divided into three divisions. The adrenal gland and gonad are present at the cranial pole of the kidney (Fig 24.31). The ureter can be seen and, in most cases, traced to the cloaca. The kidney is brown-red-orange. Star-shaped collecting tubules filled with urates are often visible on the surface. These structures become hidden in swollen kidneys (Fig 24.32). Yellow to white deposits on the surface of the kidney are often uric acid crystals and may indicate renal gout (Fig 24.33). These foci might occur due to dehydration as well. After rehydration the foci are eliminated, while in the case of gout the foci are still present. Obesity can make the kidney appear diffusely yellow. Abscesses or cysts may appear as large yellow spots (Figs 24.34, 24.35). Neoplasias or other gross abnormalities should be biopsied; assuming that the patient's condition is sufficiently stable (see Chapter 16, Evaluating and Treating the Kidneys).

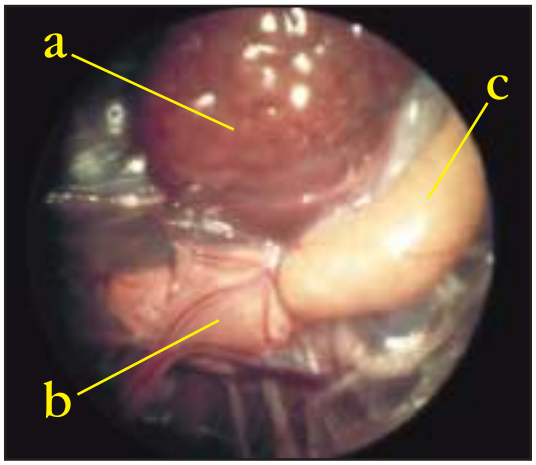


Fig 24.31 | Kidney (a), adrenal gland (b) and testicle (c). Apart from the clearly visible testicles, the absence of a ligament crossing the cranial pole of the kidney represents a male bird. The lumpy nature of the kidney surface is normal for swans.

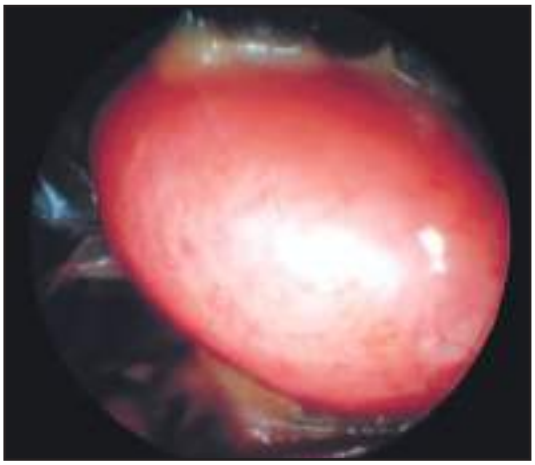


Fig 24.32 | A swollen kidney as seen in acute nephritis. Note the lack of predominate stellate vasculature pattern associated with the renal glomeruli.

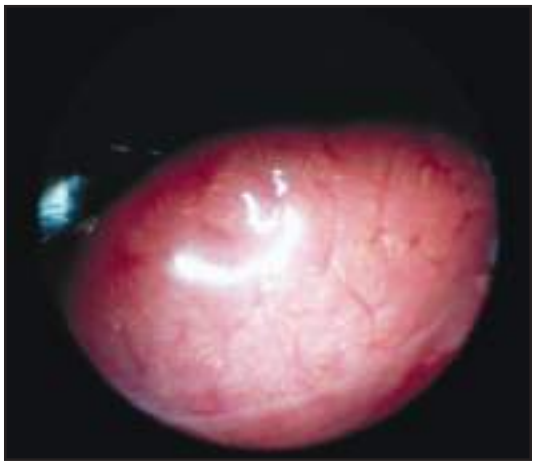


Fig 24.33 | Uric acid deposits within the kidney. If this situation remains after several applications of intravenous fluids, renal gout is likely.

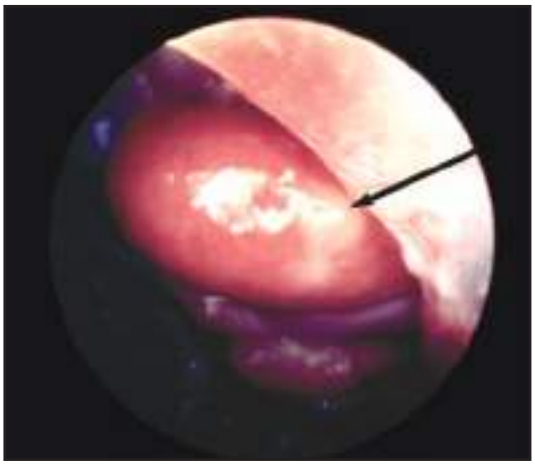


Fig 24.34 | Renal cyst. Abscess or neoplasia are possible differential diagnoses.

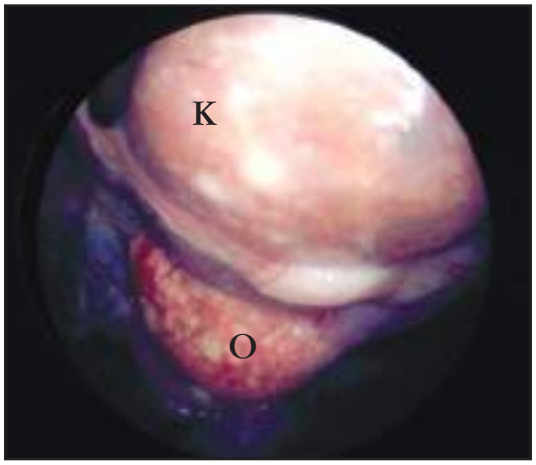


Fig 24.35 | Apart from the color changes of this kidney (K), multiple yellow foci are visible. The typical renal structure is no longer detectable. Histological examination of a renal biopsy showed a pyelonephritis. The ovary (O) has many involuted follicles.

Gonads

The left lateral approach is best for viewing gonads because hens generally lack a right ovary. Right ovaries may be present in juvenile birds, especially in accipiters. Gonads are present ventral to the cranial poles of the kidneys.

DNA methods are available for sexing most monomorphic avian species and are less invasive than surgically sexing. Endoscopic sexing has the advantage of allowing direct visualization and evaluation of the gonads and other organs. Sexual function can be estimated and any damage from sterilization or castration can be observed. The normal appearance of the gonads varies between species. The right side should be examined if the gonads are not clearly observed or discernible as to either ovary or testes, or if presumed abnormalities are present. Gonads increase in size during sexual activity (Figs 24.36-24.38). Gonads can be small due to stress, malnutrition,

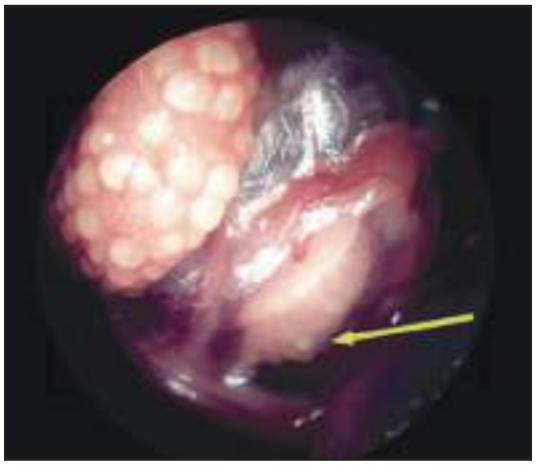


Fig 24.36 | In juvenile birds, the rudimentary ovary on the right side still might be visible, sometimes showing single follicles (arrow). The normal left ovary is at the 9-11 position.

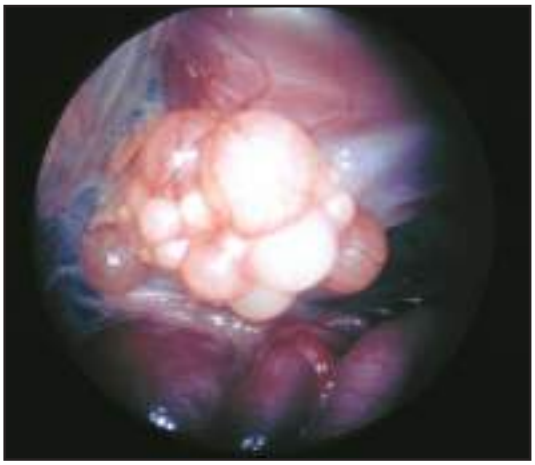


Fig 24.37 | Secondary or tertiary follicles dramatically increase in size during the reproduction cycle. Pathological alterations such as inflammation or neoplasias might lead to similar findings. A detailed anamnesis indicating sexual display behavior may indicate an active ovary. If a large follicle is very close to the tip of the endoscope, the follicle can be easily confused with a testicle.

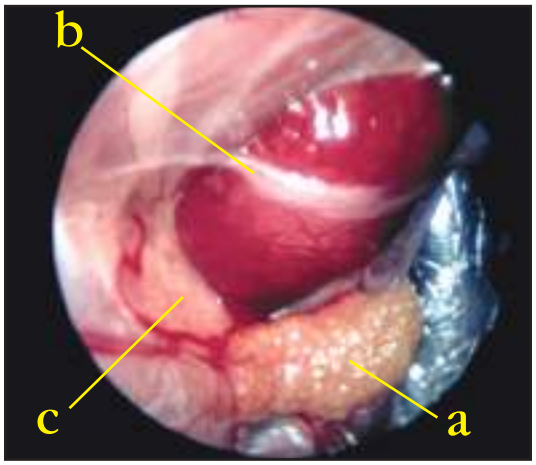


Fig 24.38 | A cluster of follicles makes identification of the ovary (a) easy. More important is the detection of the suspensory ligament (b) of the ovary. It crosses the cranial pole of the kidney. Apart from sexing, evaluation of the ligament is important to judge the possible breeding performance of the bird. Adrenal gland (c).

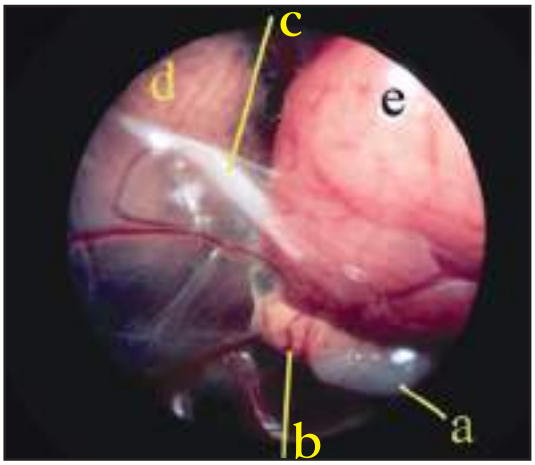


Fig 24.39 | In juvenile birds, the ovary (a) might be difficult to detect. Only the suspensory ligament (c) at the cranial pole of the kidney (e) characterizes the female bird. Lung (d), adrenal (b). Small ovarian follicles are not an accurate estimation of age or reproductive ability.

lack of nesting stimuli or immaturity.

Female Birds

The *ligamentum dorsale oviductus* (suspensory ligament) from the ovary crosses the cranial pole of the kidney coursing toward the uterus (Fig 24.38). Lacking visualization of a well-defined gonad, this ligament is the main evidence for sexing the bird as a female. The ovaries can be difficult to detect in juvenile birds (Fig 24.39). When examining breeding birds this ligament must be carefully assessed. In cases where this ligament is damaged or absent, the breeding performance of the bird is questionable (Fig 24.40). If this ligament is cut in juvenile birds, they will not lay eggs. A large uterus may indicate previous egg laying or pathology. Inactive ovaries may be flat with a cobblestone appearance, while active ovaries may appear as a cluster of spheres. The size and number of visible follicles will vary with the age and reproductive status of the hen. Immature ovaries are sometimes difficult to distinguish from testicles. The ovary is generally an off-white, yellowish color, but pigmentation (usually black) occurs in some species (Fig 24.41). In addition, the entire uterus should be evaluated.

Male Birds

In male birds there is no *ligamentum dorsale oviductus*

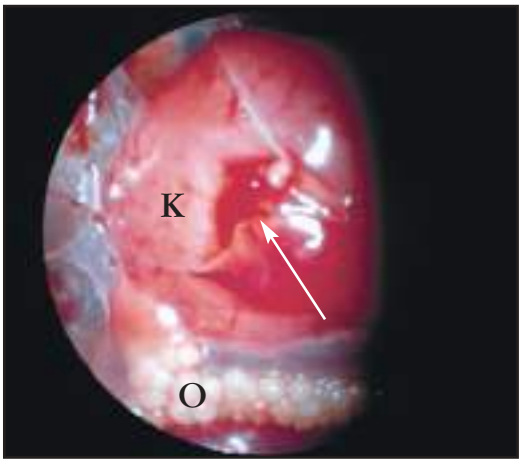


Fig 24.40 | The ovary (O) is clearly visible and the suspensory ligament is missing. This bird cannot be recommended for breeding. The kidney (K) has just been biopsied (arrow).

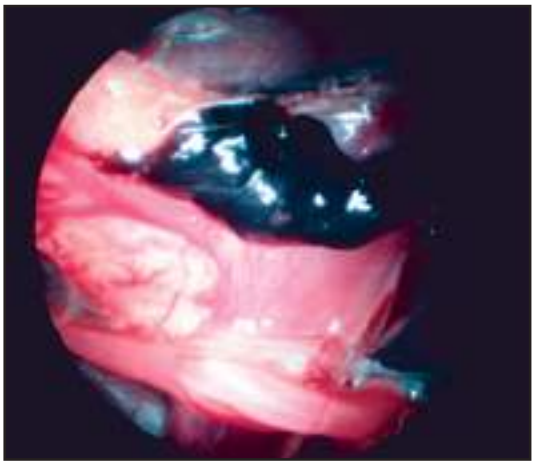


Fig 24.41 | Some avian species have melanistic gonads, as is seen in this ovary.

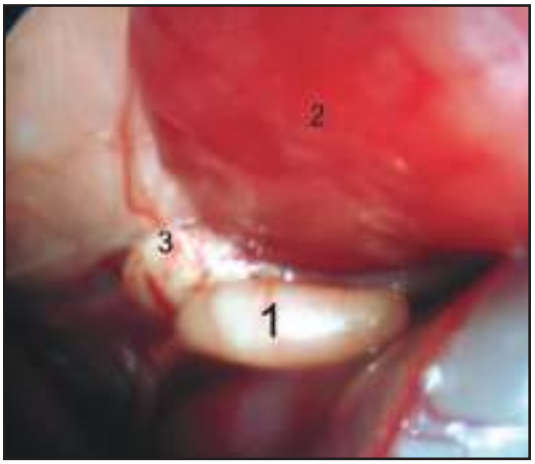


Fig 24.42a | The teste (1) is at the cranial pole of the kidney (2) and close to the adrenal gland (3).

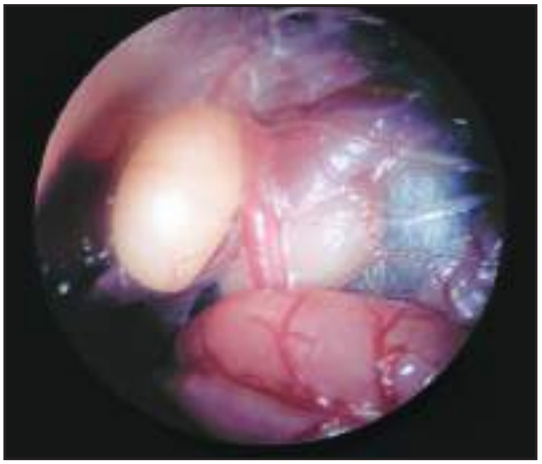


Fig 24.42b | Both paired teste are pictured here. With increased size of the left testicle or opacity of air sacs, the right testicle may be obscured from view.

(Figs 24.42a,b). The paired testicles are normally oval shaped with one to three faint vessels crossing the surface. In birds with clear air sacs, both the left and right testicles may be visualized from the left lateral approach. In some species the testicles are pigmented (eg, Cacatua, some macaws and wading birds). The torturous course of the ductus deferens makes it distinguishable from the ureter (Fig 24.43). The size of the testicles, epididymides and ductus deferens vary with the species, size, age and breeding condition of the individual bird (Fig 24.44). The breeding potential of a male bird normally cannot be assessed by visual observation of the male anatomy. If a reproductive problem is suspected, a testicular biopsy is suggested (Fig 24.45).

Adrenal Gland

Adrenal glands vary in color, size and shape (see Fig 24.42a). They may be confused with immature or inactive gonads. If the gonads are well-developed, the adrenal glands may be obscured. The adrenal glands are usually located immediately cranial to the gonads. Changes in size or increased vascularity of the adrenal glands may indicate stress or disease (Fig 24.46).

Intestine

Visible pathologic changes of the intestinal serosal surface are uncommon. Coelomic filarial worms are a rare finding in captive bred psittacines, but are regularly seen in birds of prey (Figs 24.47, 24.48). The intestine has a smooth surface covered with many vessels. The generally reddish-gray color varies according to the intestinal fluid. White foci may be a sign of previous penetration by endoparasites. Both thinning and thickening of the intestinal wall are signs of enteritis. Thinning can be appreciated endoscopically from the visibility of intestinal contents. Necrosis of the intestine wall might be visible in cases of clostridiosis or coccidiosis. Enlarged ceca filled with caseous yellow material could indicate histomoniasis.

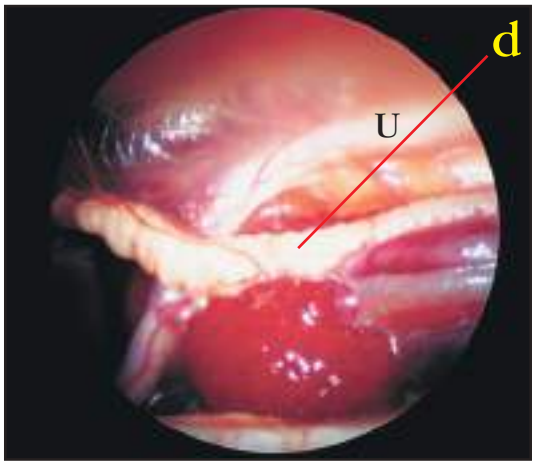


Fig 24.43 | Ductus deferens (d) and ureter (U) in a sexually active male.

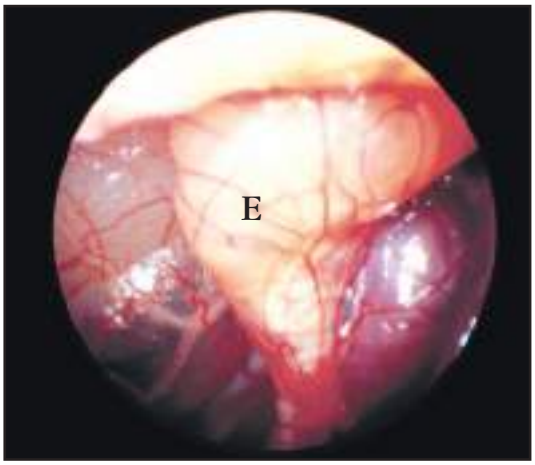


Fig 24.44 | Epididymis (E) of a sexually active male.

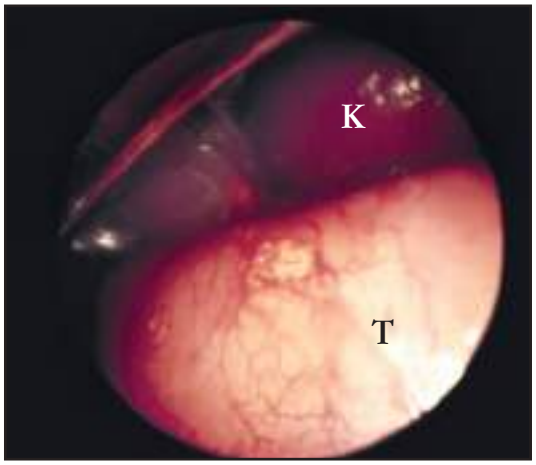


Fig 24.45 | A testicle (T) with a structural abnormality that would indicate the need for a biopsy. Kidney (K).

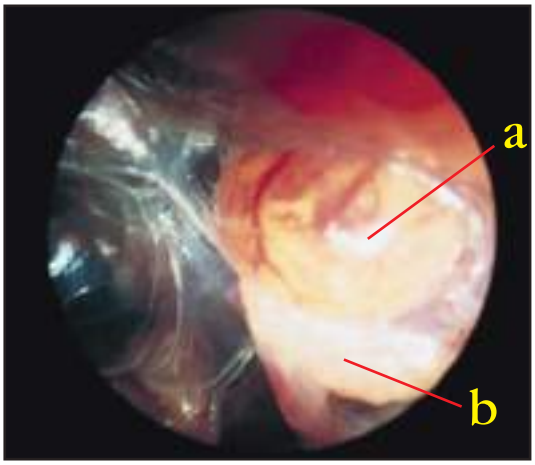


Fig 24.46 | Enlarged adrenal glands (a) appear in cases of stress or diseases. Juvenile ovary (b).

Pancreas

The pancreas lies within the duodenal loop (Fig 24.49). The pancreas is a white-yellow color with a homogeneous matrix. Color changes, glassy appearance or an uneven surface often accompany pathologies and may warrant biopsy (see Chapter 26, Diagnostic Value of Necropsy).

Spleen

Psittacine spleens are round, purplish and often speckled (Fig 24.50). The spleen is located at the dorsal aspect of the proventricular/ventricular junction on the right side from a left lateral approach. Splenomegaly (immune response), yellow appearance (fatty spleen) and multiple white foci (necrosis) are possible pathological alterations (Figs 24.51, 24.52). Chlamydophilosis and other bacterial diseases would be included in the differential. The spleen can be biopsied utilizing the same precautions as in mammals.

Tracheoscopy Studies

TRACHEA AND THYROID GLAND

The trachea and thyroid gland can be approached via the cervical branch of the cervicocephalic air sac, the clavicular air sac or through the coelomic cavity. The thyroid gland is visible as an elliptical pink structure attached to the trachea near the syrinx (Fig 24.53). Alterations in size or a shiny appearance are considered abnormal and a biopsy may be indicated.

ENDO-TRACHEAL EXAMINATION

Endoscopic examination of the tracheal lumen is accomplished by extending the patient's neck and gently advancing the scope through the larynx and down the trachea (Fig 24.54). Unless the procedure is very rapid, an air sac breathing tube is necessary (see Chapter 33, Updates in Anesthesia and Monitoring). Many endoscopes are equipped with a protective sheath. Removal

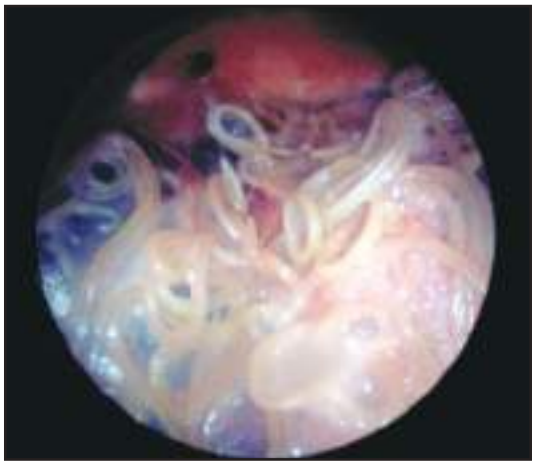


Fig 24.47 | *Serratospiculum* sp. in the air sac in a falcon.

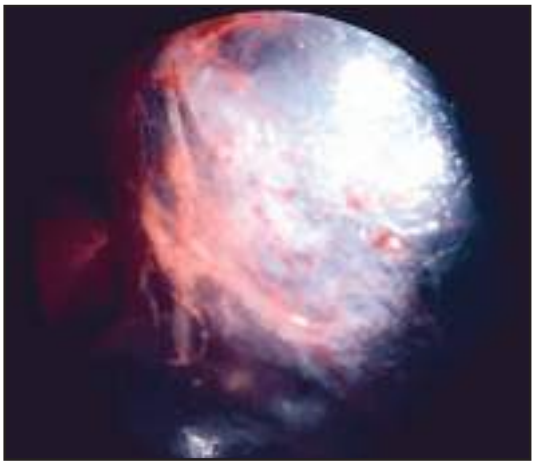


Fig 24.48 | The same bird as Fig 24.47 12 days after treatment with ivermectin. The dead worm can easily be confused with a bacterial infection.

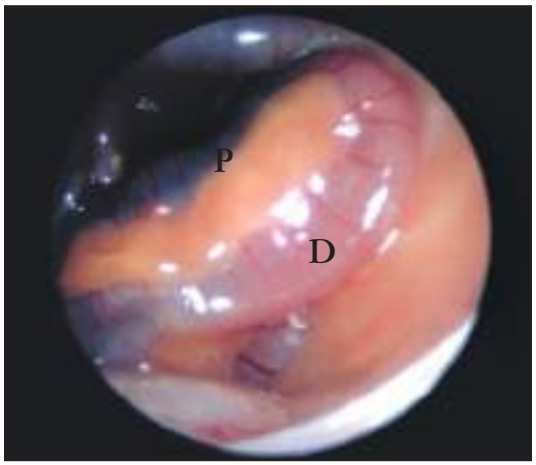


Fig 24.49 | The pancreas (P) identified in the duodenal loop (D). The pancreas should have a homogeneous structure and color.

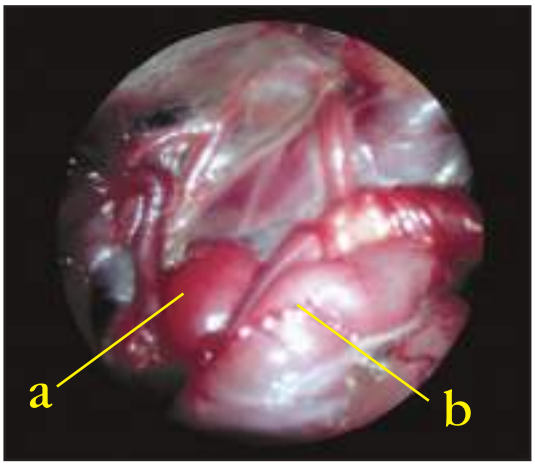


Fig 24.50 | Using the left lateral approach, the spleen (a) is accessible from the abdominal air sac by pushing the proventriculus in the caudal-ventral direction to expose the right side of the proventriculus. The psittacine spleen is round and similar in color (red-brown) to the kidney and liver. Intestine (b).

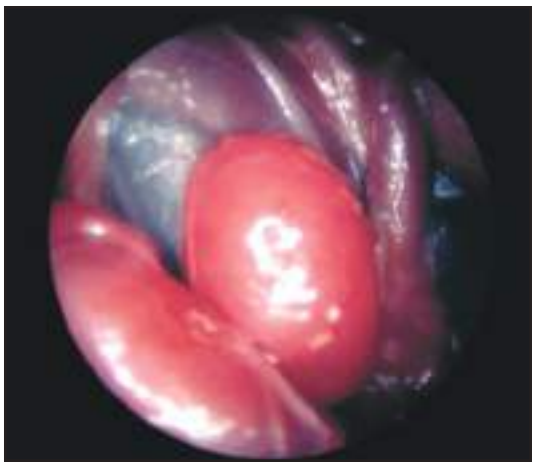


Fig 24.51 | Enlarged spleen of a bird with psittacosis.

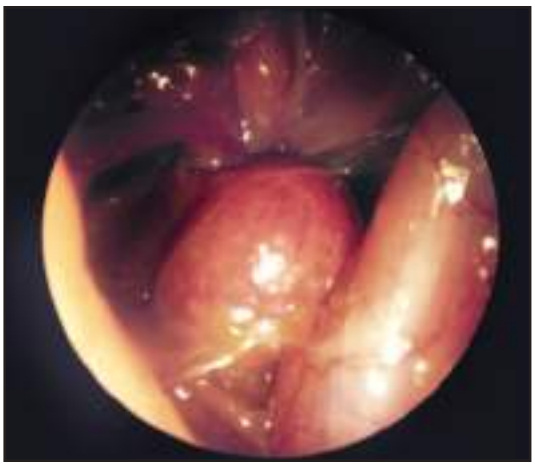


Fig 24.52 | Pale color changes and enlargement of a spleen.

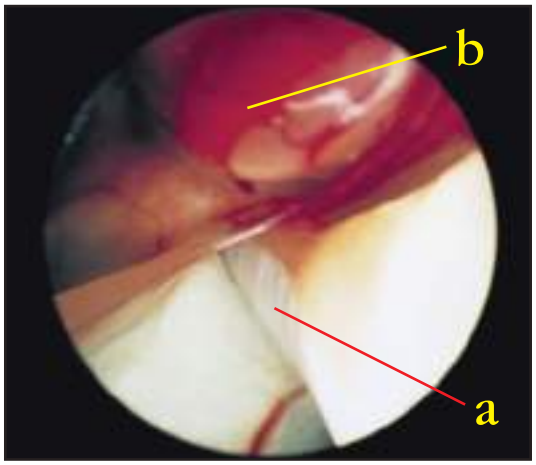


Fig 24.53 | The thyroid gland on the carotid artery adjacent to the trachea as seen from the interclavicular air sac. A laparoscopic approach may be used by pushing the scope cranial, passing over the heart ventrally and following the trachea (a). The thyroid gland (b) can be visualized.

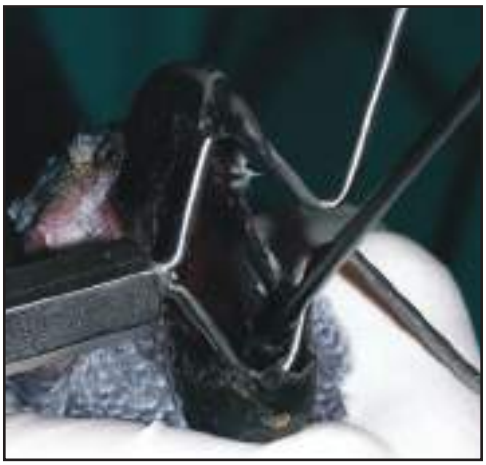


Fig 24.54 | Performing a tracheobronchoscopy. The neck of the bird must be fully extended. A beak speculum allows a better view and is safer for the scope in case the bird wakes up and attempts to bite the instrument. Anesthesia can be delivered via an air sac tube.

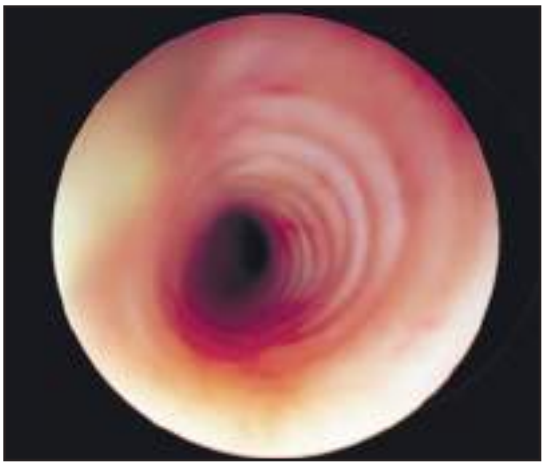


Fig 24.55a | Normal trachea.

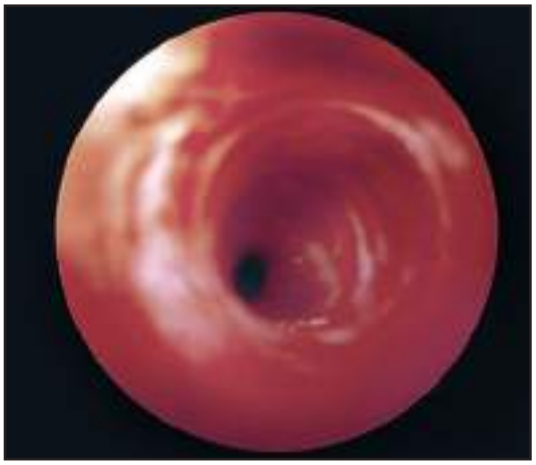


Fig 24.55b | A case of severe tracheitis. The tracheal rings appear distorted due to the mucosal swelling, sloughing and hemorrhage.

of this sheath will decrease the scope's diameter and enable its introduction into the trachea of smaller birds. Unfortunately, this also increases the chance for damage to the endoscope. An unsheathed 1.2-mm scope[a] can allow visualization of the trachea of birds the size of canaries and finches (Fig 24.58). Without the sheath, the tracheal lumen can be examined, but no instruments can be introduced into the visual field in such a small patient. Evaluation should be made of the tracheal color, and mucosal texture. The mucosa of the trachea and the bronchi are light pink and glistening. The tracheal rings are clearly visible (Fig 24.55a). In cases of tracheitis, the mucosa becomes red and swollen, making the rings less obvious (Fig 24.55b). Tracheal exudates, when present, should be collected for cytology and culture. Possible abnormalities of the trachea include strictures, tumors, inflammation, parasites, fungal granulomas and foreign bodies (Figs 24.56, 24.57). The tracheal diameter typically decreases toward the syrinx. The narrowed diameter and the tracheal bifurcation into the main stem bronchi make this area particularly prone to fungal granulomas and foreign bodies. Some degree of post-examination hyperemia of the trachea is normal.

Acute dyspnea warrants endoscopic tracheal examination once the patient is stabilized. Hemorrhage of the tracheal mucosa may be seen with polytetrafluoroethylene toxicosis. More pronounced pathology would be expected in the lung parenchyma with this condition.

Respiratory parasites often can be visualized with the endoscope (Figs 24.57, 24.59). If mites are suspected and not visualized, swabbing the scope onto a sterile slide and examining the slide microscopically may reveal tracheal mites.

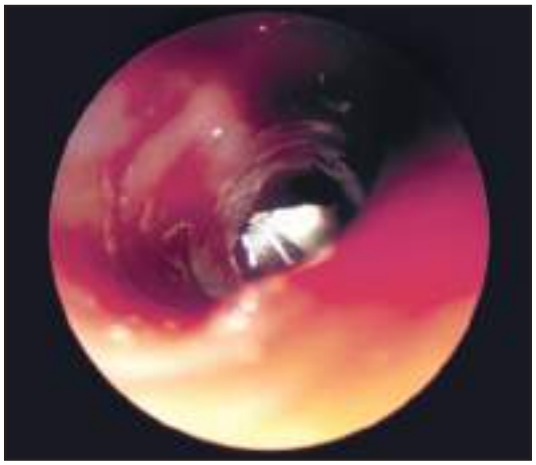


Fig 24.56 | Foreign body (seed) in a trachea. The mucosa is irritated and swollen.

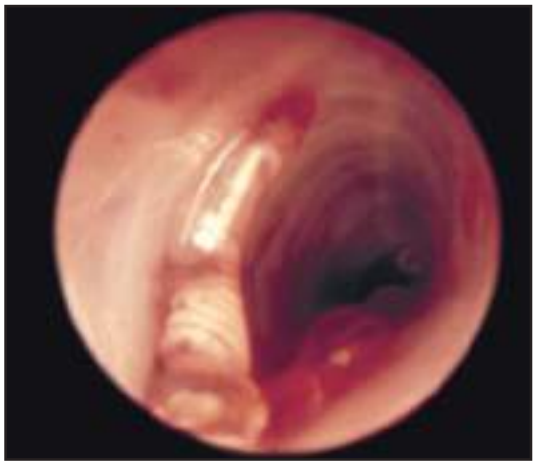
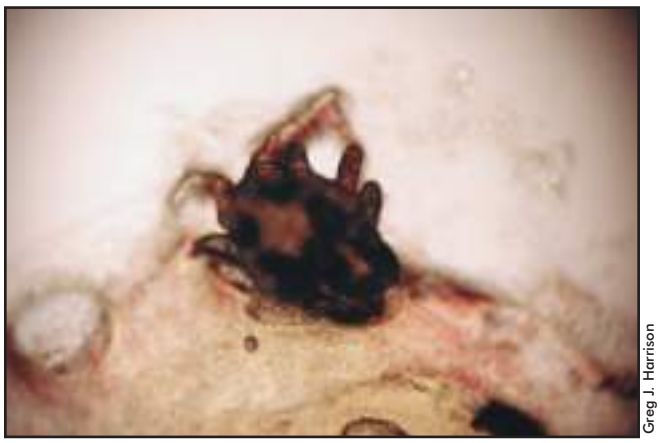


Fig 24.57 | *Syngamus trachea* in the trachea.

Greg J. Harrison

Fig 24.58 | Endoscopy of a canary trachea with a 1.2 mm semiflexible endoscope[a].



Greg J. Harrison

Fig 24.59 | A canary tracheal mite under 100x. This mite was obtained from the endoscope tip. The mites in the trachea were not visualized during endoscopy.

Pharyngoscopy and Upper GI Studies

OROPHARYNX

Sufficient anesthesia or restraint is necessary to prevent damage to the endoscope by the bird's beak. In addition, the use of a beak speculum is advantageous. While under anesthesia, the bird is held in a ventral recumbency position and the neck is fully extended (Fig 24.61). This position allows endoscopic examination of the inner surface of the beak, oral cavity, choana, rhinal cavity, tongue and larynx. The shape of the tongue differs from species to species. The infundibular cleft should be free of swelling and debris. Check the entire oral cavity for signs of pathology (Table 24.7).

RHINAL CAVITY

The rhinal cavity can be entered from the choana and the turbinates examined. The operculum prevents the passage of a scope through the nares. The points used to perform an infraorbital flushing technique also can be entered endoscopically.

ESOPHAGUS, CROP, PROVENTRICULUS, VENTRICULUS - MUCOSAL EXAMINATION

Examination of the esophagus, crop and proventriculus via the oral cavity is a common procedure (Fig 24.62). As the esophagus, crop (Fig 24.63) and proventriculus are hollow organs, insufflation is necessary for visualization.

Table 24.7 | Common Oral Pathology in Psittacines

- Squamous metaplasia
- Keratinized debris
- Blunting of choanal papillae
- Plaques
- Fungal or yeast colonization
- Parasites: trichomonads
- Bacterial infection
- Oral papillomas: most common in *Ara* spp.
- Oral neoplasias: fibrosarcoma, squamous cell carcinoma
- Trauma
- Ulcerations

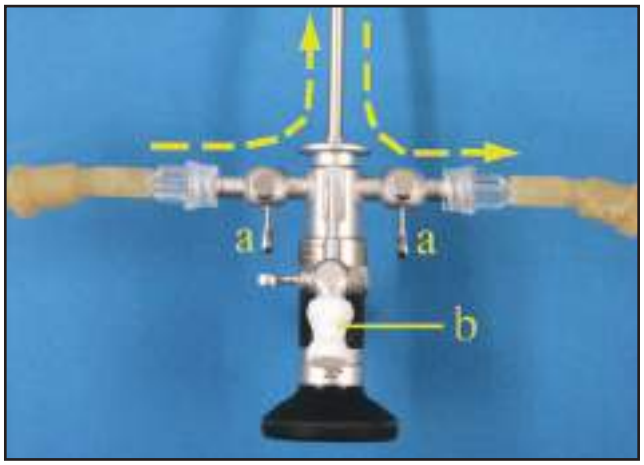


Fig 24.60 | For upper digestive system endoscopy, one uses a sheath with a working channel, here with an endoscope inserted. Hollow organs can be insufflated and flushed using liquids. Tubes for infusion liquids can be attached to the liquid-in and liquid-out taps (a). The arrows demonstrate the direction of the flow of liquids. A port for flexible instruments (grasping biopsy forceps, needle, scissors or various brushes) (b) is shown above the viewing eyepiece.

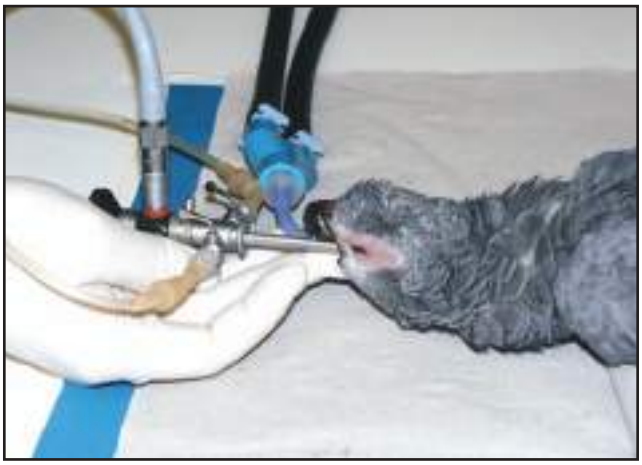


Fig 24.61 | When performing a gastroscopy, the head of the bird must be in a position lower than the body. Aspiration of liquids into the lungs is then easier to avoid. Tracheal intubation of the bird during anesthesia aids in the prevention of aspiration.

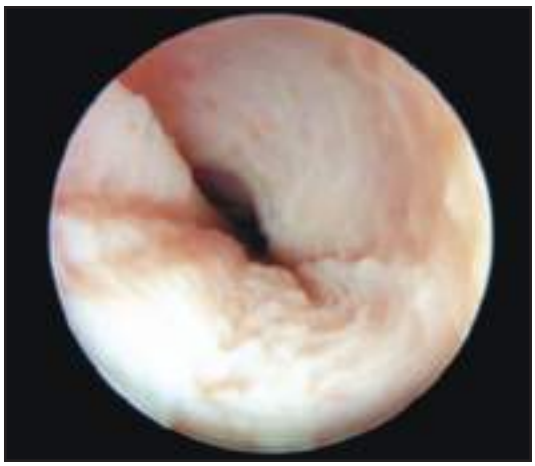


Fig 24.62 | Normal esophagus of a chicken.

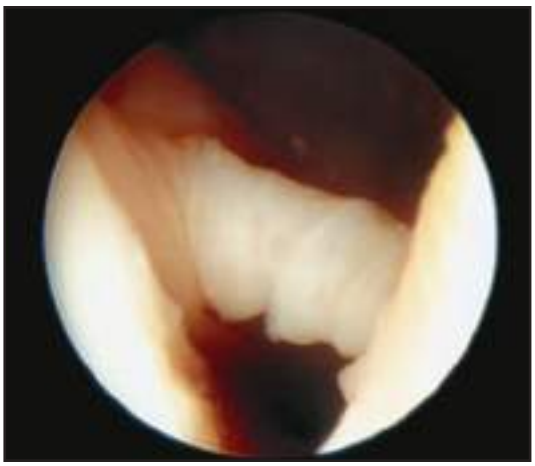


Fig 24.63 | Endoscopic view at the entrance of the crop in a chicken. The oblong structure is the wall between the esophagus and the crop. The other opening is the entrance to the crop.

Prior to gastroscopy, a fasting period is important to allow maximum viewing without the presence of food. Insufflation with air or sterile fluids is commonly used for positioning and advancement of the endoscope. Sterile fluids allow the flushing out of debris and subsequent dilation of the GI tract. This greatly aids in visualization. It is important that the fluid be warm to the touch to avoid decreasing the body temperature of the bird. A working channel is necessary to aim the washing solution. This working channel should have two taps, one for fluids in and one for fluids out (see Fig 24.60). The third port is ideal to allow simultaneous passage of a biopsy or grasping forceps. The fluid inlet is attached to an infusion bag or bottle positioned at a higher elevation. A larger infusion tube is connected to the fluid outlet leading to a collecting container. The two taps allow accurate control of the amount of fluid within the digestive system, expanding the organs as needed for examination. The fluid outlet is closed and the selected portion of the GI tract is dilated until good visualization of the mucosa is achieved. The fluid outlet is then opened in order to flush out mucus and small particles. Opening the fluid inlet again can increase the pressure. To avoid aspiration, fluid should not be allowed to exit the digestive tract through the oral cavity, which could and often does lead to the contents being inhaled. Occlusion of the scope and esophagus to retain the infused air or fluid in the crop can be easily accomplished by placement of digital pressure on the scope and esophagus. Although the tracheal rings are solid in birds, caution

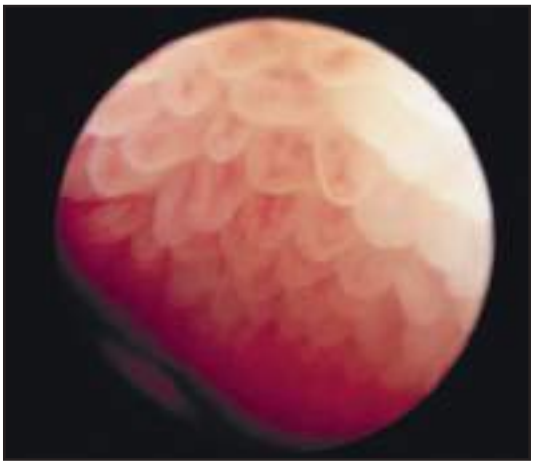


Fig 24.64 | Psittacine cloacal mucosa visualized using liquid insufflation.

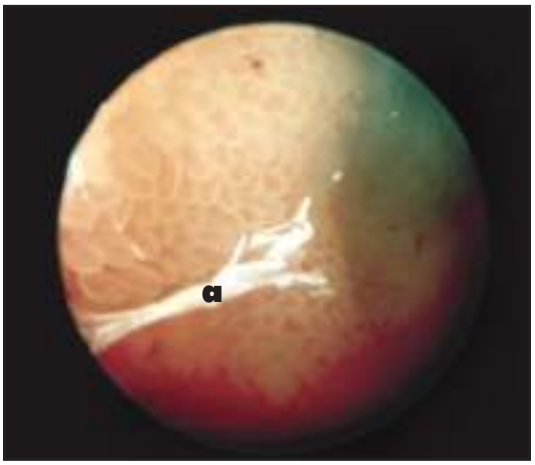


Fig 24.65 | Urates (a) emanating from an ureter on the cloacal surface.

should be used to prevent restriction of normal airflow. In addition, the bird should be positioned in ventral recumbency with the head lower than the body. An endotracheal tube should be in place (Fig 24.61).

The mucosal surfaces of the esophagus and the crop vary between species. The mucous membranes of the esophagus, crop and proventriculus are a homogeneous pink (Fig 24.62). The mucosa of the esophagus is usually smooth; the crop has furrows and the proventriculus papillae (Fig 24.63). Focal dark red or bleeding areas are signs of irritation, which can be due to foreign bodies, infections, ulcerations, or neoplasia. A yellow coating of the mucosa can be seen with trichomoniasis, candidiasis, the diphtheric form of avian pox or vitamin A deficiency or excess. If there is a loss of the proventricular mucous membrane's normal color it might be a sign of a wall suggesting PDD. Biopsy of the proventriculus or the crop and submission for histopathology may confirm a suspected diagnosis of PDD. However the significant risk of dehesience must be considered prior to obtaining a proventricular biopsy. To evaluate the proventriculus in larger psittacines, it may be necessary to introduce the scope through an ingluviotomy incision. A preferred location for this incision is to the left and somewhat dorsally, to avoid postoperative pressure on the crop incision from ingesta. An area of reduced vascularity is ideal. The scope is introduced and advanced carefully into the thoracic esophagus, continuing caudally to the proventricular lumen. This procedure is useful for proventricular biopsies and retrieval of foreign bodies. A flexible scope is mandatory to evaluate a fragile proventriculus (eg, normal lories, large macaws and all cases of PDD) or all ventriculi.

Cloacoscopy Studies

CLOACA

The endoscope simplifies cloacal examination. Insufflation is necessary for viewing to maintain an adequate distance between the scope and tissues under examination. A soft rubber feeding tube may be used, with digital pressure applied around the scope barrel to retain the air or fluid.

When performing a cloacoscopy, the bird is placed in dorsal recumbency and the endoscope is inserted with its working channel. Insufflation is usually done using fluid (see description at Esophagus, Crop, Proventriculus, Ventriculus - Mucosal Examination, above). Feces and urine are almost always present and should be washed out for optimal visualization. The mucosal surface is pink with ureteral papillae (Fig 24.64). Urine can be seen emanating from these ostia of the ureters (Fig 24.65). Hyperemic cloacal membranes are indicative of inflammation or infections. Rough and red raised areas (cauliflower shape) are suggestive of papillomatosis. Inside the cloaca, the openings of the ureters, the rectum and, in female birds, the uterus can be viewed. The oviduct can sometimes be entered and the caudal chambers investigated. The occurrence of fresh blood within the feces is a clinical indication for cloacoscopy, as it may originate from the cloaca, the intestine, the ureters or the uterus.

Otoscopy Studies

EAR

In most species, feathers conceal the opening of the ear canal. The external orifice can vary from <2 mm in small

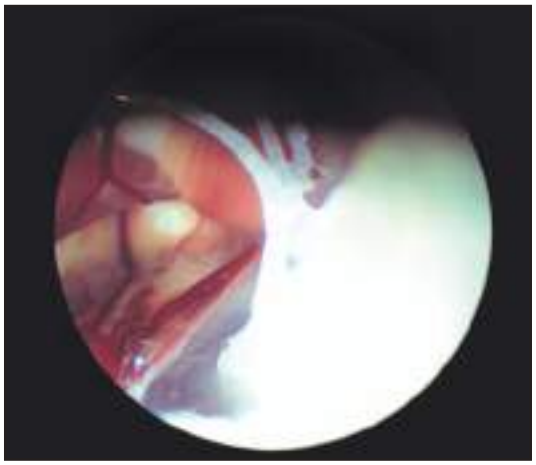


Fig 24.66a | The brachial plexus is visible cranial-lateral to the heart.

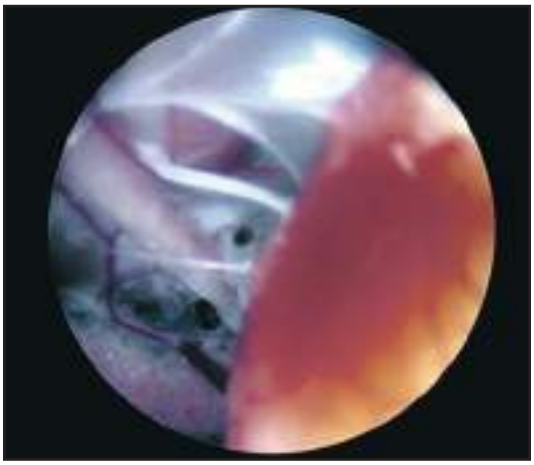


Fig 24.66b | Plexus sacralis dorsal to the kidney.

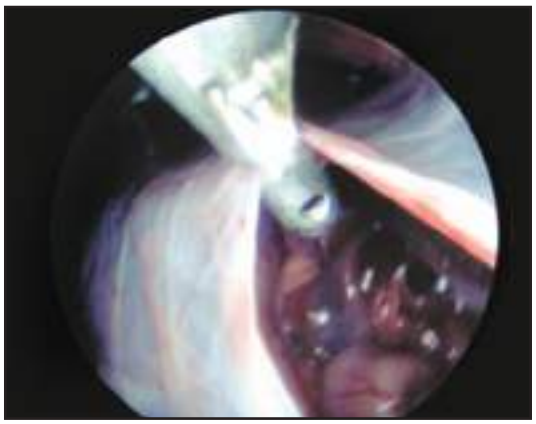


Fig 24.67 | In case of a pansystemic airsacculitis (here milky), the entrance of the scope is the site of choice for a biopsy.

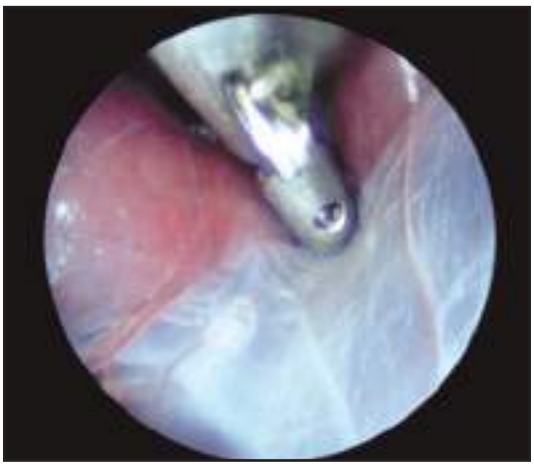


Fig 24.68 | In case of a grossly abnormal liver, a biopsy should be performed at the liver border.

species and up to 6 cm in some large raptors. The normal tympanic membrane is clear and slightly convex. Otitis externa is not a common finding in psittacines, but bacterial, fungal, neoplastic and allergic conditions may occur. In raptors, common findings are bleeding into the ear canal from head trauma.

Nerve Studies

The nerves of the brachial plexus are seen anterior and lateral to the heart (Fig 24.66a) The sacral pluxus sometimes visible dorsal to the kidney (see Fig 24.66b).
Ed Note: Harrison has used endoscopy to evaluate nerve damage. Muscles, nerves, vessels, tendons and ligaments can be examined using the endoscope. In cases of trauma, air or fluids can be injected subcutaneously to provide a path for the scope to follow anatomical structures into the area of trauma. This technique could be useful to investigate brachial plexus evulsion, nerve transection, thrombi, emboli, and traumatic damage to soft tissue.

Endoscopic-guided Biopsies

Coordinating the endoscope and the biopsy instrument can be challenging. The advent of sheaths that are now provided with many endoscopes has simplified this procedure by allowing the biopsy forceps to approach the site without changing the field of vision. The small size of the biopsy forceps used for these procedures usually precludes serious hemorrhage. Endoscopic-guided biopsies allow sampling of organs under direct visualization (Fig 24.67). In general, biopsies of the lung, air sac, liver, kidney, spleen, gonads, proventriculus, ventriculus, thyroid gland and mucosal membranes of esophagus, crop and cloaca are possible using a biopsy forceps within a working channel. Aspiration biopsies are possible using a long, flexible needle with a Teflon cover (see Table 24.6). Puncture of cysts, bone marrow biopsies or lavage sampling are the ideal uses for this needle. In case of a general alteration of an organ, the biopsy should be taken from the organ's border (Fig 24.68). Contraindications of

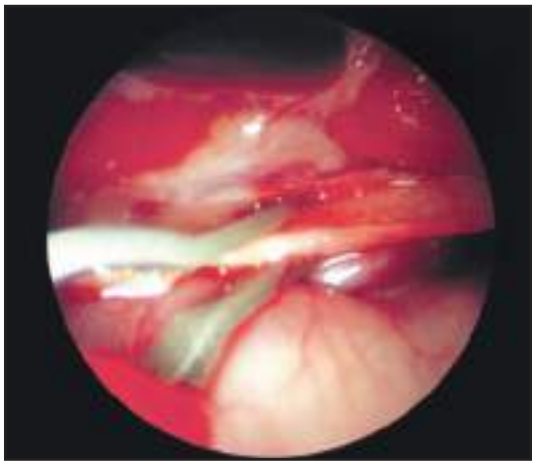


Fig 24.69a | For sterilization of the female bird, the oviduct can be obliterated using a bipolar coagulation forceps. Surrounding tissues must be avoided.

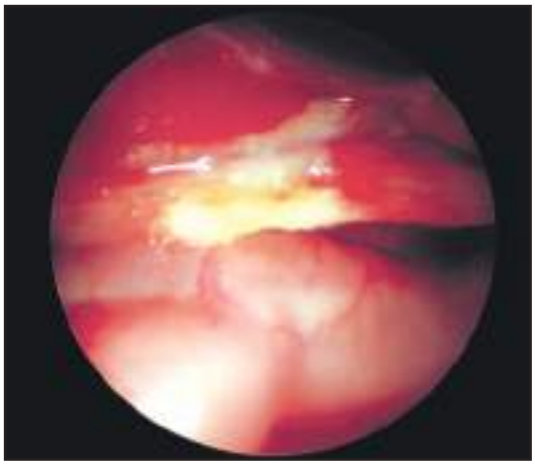


Fig 24.69b | The appearance of the coagulated section of the oviduct that will scar closed.

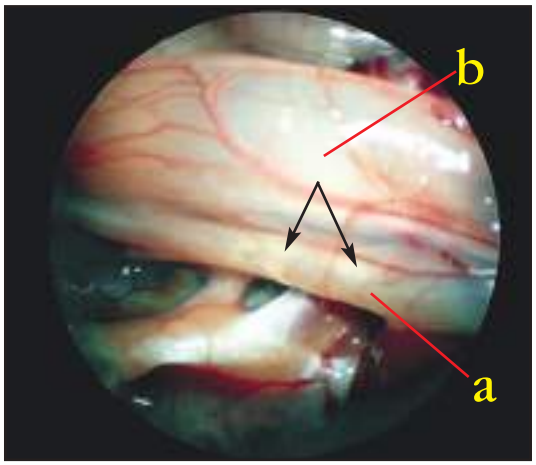


Fig 24.70 | The dichotomy of when to perform female reproductive surgery is amply demonstrated on the immature or involuted side of the question by observing the intimate proximity of the ureter (a) to the uterus (b) in this example. Avoiding collateral damage is imperative. The ureter should be observed for movement of urates (arrows) seen moving during regular contraction cycles. While on the opposite end of the spectrum, the mature or active uterus has up to a centimeter of distance between these structures in a bird as small as a cockatiel, allowing almost no chance for neighboring tissue damage; hemostasis is the challenge. Vessels increase in length but more significantly in diameter and thickness, creating the imminent danger of life-threatening hemorrhage. Some of these vessels are too large for casual coagulation or less than ideal vessel-clamping techniques, resulting in oozing in the first case or loss of the clip and hemorrhage in the latter.

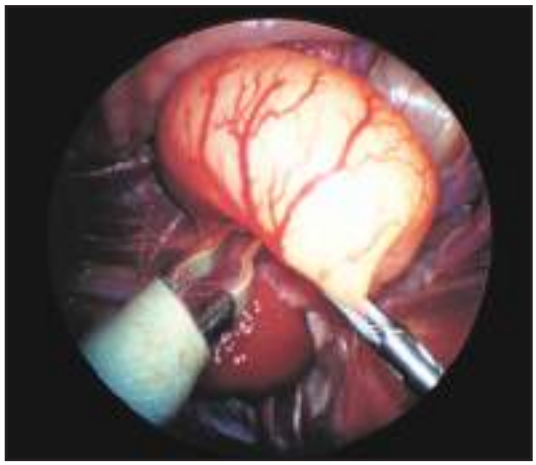


Fig 24.71 | Endoscopic-guided multiple entry for bipolar radiosurgery castration.

biopsies are similar to those mentioned for endoscopy. Endoscopic procedures, in particular tissue biopsies, lead to changes in certain blood values;[1] therefore, planned blood sampling must be performed before endoscopy.

Endoscopic-guided Surgical Procedures

Endoscopic-guided sterilization or castration is possible. This might be indicated in chronic egg laying or birds that are aggressive during breeding season. As castration is quite complex, sterilization represents a quick and easy procedure, as the gonads need not be removed. This can be accomplished using electrosurgery with a bipolar endoscopic forceps (Figs 24.69a,b). Performing this procedure when the bird is sexually inactive reduces the risk of hemorrhage. (Sterilization may hormonally influence behaviors). In juvenile or hormonally inactive females the challenge one is faced with is making the

distinction between the ureter and the quiescent oviduct. The ureter is marked by urates, or its regularly occurring contractions (Fig 24.70). Administering intravenous fluids will increase the likelihood of seeing urates pass down the ureter.

Endoscopic-guided obliteration of air sac granulomas or papillomas in the cloaca is possible. Endoscopic-guided laser diodes have been used to obliterate granulomas within the trachea or air sac. Multiple-entry endoscopic surgery has been developed for resection of tumors or castrations (Fig 24.71). Instruments are guided into the endoscopic field of vision using trocars. Laser or radiosurgery is helpful to maintain hemostasis.

Complication During and After Endoscopy

Hemorrhage is the main complication arising from endoscopy. The kidney can be damaged during penetration of the air sac at the beginning of the laparoscopy. Perforations of the proventriculus may result in fatal peritonitis. If major bleeding occurs, electrocoagulation, oxidized regenerated cellulose or sterile sticks of cotton wool can be used. The bird should be placed at a 45° angle with the head elevated to prevent blood from entering the lungs. This keeps the blood in the caudal air sacs. If a large entry site has been created, the site may need deep sutures to close the muscles and prevent subcutaneous emphysema. Postsurgical closure of air sac defects is usually not necessary. If emphysema occurs, it should be punctured and deflated regularly until these defects close themselves. When performing endoscopy on multiple subjects sufficient sterilization time for the equipment is imperative to avoid transmission of disease.

Product Mentioned in the Text

a. Semi-rigid scopes, infusion/aspiration needle, Storz, www.karlstorzvet.com

b. 22-gauge aspiration needle within a teflon tube, Storz, www.karlstorzvet.com

References and Recommended Reading

1. Lierz M, Ewringmann A, Goebel T: Blood chemistry values in wild raptors and their changes after liver biopsy. Berliner and Muenchener Tieraerztliche Wochenschrift, 1998, 111, pp 295-301.
2. Baileys RE: Surgery for sexing and observing gonad condition in birds. Auk 70:497-500, 1953.
3. Eaton TM: Surgical sexing and diagnostic laparoscopy. *In* Price CJ (ed): Manual of Parrots, Budgerigars and Other Psittacine Birds. West Sussex, UK, Brit Small Anim Vet Assoc, 1989.
4. Hochleithner M: Endoscopy. *In* Altman, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 800-805.
5. Hochleithner M: Endoscopy. Proc Euro Conf Avian Med Surg, 1993, pp 162-166.
6. Kolias GV: Liver biopsy techniques in avian clinical practice. Vet Clin North Amer 14(2):287-298, 1984.
7. Lierz M: Investigations of birds of prey and owls found injured and debilitated in Berlin and Brandenburg. Veterinary Thesis, Free University of Berlin, Germany, 1999.
8. McDonald SE: Endoscopic examination. *In* Burr EW (ed): Companion Bird Medicine. Ames, IA, Iowa State Univ Press, 1987, pp 166-174.
9. Taylor M: Endoscopic examination and biopsy techniques. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 327-354.
10. Taylor M: Endoscopy. Proc Assoc Avian Vet, 1990, pp 319-324.
11. Taylor M: Endoscopic Examination and Therapy of the Avian Gastrointestinal Tract. Sem Avian Exotic Pet Med 8(3):110-114, 1999.
12. Weber MA, et al: Benefits and complications of liver biopsy in birds. Proc Assoc Avian Vet, 2001, pp 211-213.

CHAPTER
25

Advances in Diagnostic Imaging

PETER HELMER, DVM, Dipl. ABVP-Avian

Advances in technology have resulted in many different ways of producing and recording diagnostic images. Conventional black and white radiographic images on film continue to predominate, primarily due to widespread access, ease of use, familiarity and relatively low cost. Digital and computerized radiographic images are not widely accessible in private practice. These latter modalities produce a digital image that can be manipulated with a computer to highlight detail and compensate for less than ideal technique settings. Digital technology will become more affordable and more readily available to the private practitioner in the future.

Radiographic Technique

The diagnostic value of a radiograph is directly proportional to the quality of the radiograph. The small size and fine detail of the avian patient necessitate excellent-quality images. Multiple factors influence the quality of a radiograph.[20] These factors include motion, the speed of the film, focal spot size, focal spot-to-film distance, object-film distance, the use and type of intensifying screen, and the use of a grid. No technique can maximize the benefits of all of these factors, but a reasonable compromise can be obtained that results in radiographs of high quality.

Motion, even in small amounts, results in significant blurring of the image. In order to minimize this artifact, patient restraint is required. Physical restraint of the avian patient for radiography is rarely adequate. Proper positioning of the awake patient is difficult. There is

Fig 25.1 | Mammography cassette[a] opened to reveal the single intensifying screen.

Fig 25.2 | Mammography film[b] has a single-sided emulsion. When loading cassettes, the light side of the film should be in contact with the white side of the cassette.

significant stress and risk of injury to the bird, as well as increased risk of radiation exposure for the handler. Restraint in the form of inhalant anesthesia (isoflurane or sevoflurane) is strongly recommended. The risk of general anesthesia should be assessed for each patient, but usually this risk is far outweighed by the benefits of decreased patient stress, increased quality of the radiographs, less time spent by staff, less exposure to radiation and fewer patient injuries.

Respiratory motion is largely unaffected by appropriate planes of anesthesia. Avian respiratory rates require relatively short exposure times to eliminate the motion artifact. In practice, exposures of 0.01 to 0.05 seconds are routinely used.

Other machine settings also influence radiograph quality. Selection of a smaller focal spot setting, if available, will result in sharper edge definition. Increases in the focal spot-to-film distance will positively affect detail; however, the mAs (milliamperes seconds) or the kVp (kilovolt peak) also must increase proportionately. A compromise focal spot-to-film distance of 40 inches (100 cm) is often used.

Scatter radiation adversely affects the detail of radiographs. This is particularly true when high-detail or mammography screens are used. Close collimation of the primary beam around the areas of interest is important to decrease scatter. Birds are routinely positioned with the film cassette on the tabletop. This decreases the object-to-film distance and maximizes detail. Grids are useful to decrease scatter radiation when the thickness of the object being imaged is greater than 4 inches (10 cm); however, few birds are thicker than this so grids are rarely used.

Film-screen combinations contribute significantly to image quality and exposure settings. A "faster" film will have larger silver halide crystals. The larger size of these crystals makes them more likely to be exposed to x-rays or light, thus they require a lower exposure to produce an image. However, the image will be less sharp than the same image made on a "slower" film with smaller crystals and a higher exposure. Intensifying screens are generally made of rare-earth crystals that fluoresce when struck by x-rays. Mammography cassettes[a] and film[b] offer an excellent combination for high-detail radiographs. The initial cost of the screens is higher, but used screens are often available at discounted prices through human imaging centers. Mammography film is more expensive than standard radiographic film. Mammography cassettes have screens on only one side and mammography film is single-sided emulsion. Proper loading of the cassettes is essential. The dark side of the film faces the dark side of the cassette and the light side faces the light side (Figs 25.1, 25.2). Most automatic processors can routinely develop these films, though their non-standard size may create problems in some models. Mammography film is often still damp after the drying cycle as programmed into most automatic processors. Care should be taken to avoid damage to the developed film and to provide for additional drying time. It also is important to realize that mammography cassettes require a higher exposure setting than typical double-screened cassettes (Table 25.1). Non-screen film such as dental radiography film also provides excellent detail and requires higher exposure settings.

Table 25.1 | Technique Chart Used for Psittacines in the Author's Practice Using Mammography Cassettes[a] and Film[b]

Species	mA	Time (in sec)	kVp (kilovolt peak)
Budgerigar	300	1/10	40
Cockatiel	300	1/10	41-42
Pionus/mini macaw	300	1/10	43
Amazon/African grey	300	1/10	45
Large cockatoo/macaw	300	1/10	48-50

Fig 25.3 | Routine positioning of an anesthetized bird for the ventrodorsal projection.

Fig 25.4 | Routine positioning of an anesthetized bird for the laterolateral projection.

POSITIONING

The principles of positioning the avian patient for radiography are the same as for other species. At least two views, 90° to each other, are suggested. Positioning may be maintained by taping the patient directly to the cassette or to a radiolucent Plexiglas board. Masking tape is usually sufficient in the chemically immobilized patient and has the advantage of not pulling out feathers when it is removed.

The basic views of the body are the laterolateral and the ventrodorsal (Figs 25.3, 25.4). When assessing positioning for the laterolateral view, the two femoral heads should overlie one another, the legs should be pulled caudally, the sternum should be parallel to the film and the wings should be secured dorsally. In the ventrodorsal view, the legs should be pulled caudally. Wings are stretched and secured laterally, and the sternum should directly overlie the vertebral column.

Standard views of the wing are the ventrodorsal (positioned as for the body) and the caudocranial view. The legs are evaluated with routine orthogonal lateral and anterior/posterior views.

Skull radiography is challenging due to overlap of multiple structures, and their complex relationships. In order to optimize interpretation of these films, lateral, ventrodorsal and oblique projections are suggested.[15]

Radiography of the skull is indicated for evaluation of bony structures, including identification of fractures, luxations, hyper- and hypocalcemia, and carcinoma.[8] Sinuses and soft tissue structures may be visualized, but changes in these structures tend to be visible only in advanced stages of disease. Magnetic resonance imaging (MRI) is a more sensitive tool for evaluation of these structures.[16]

The standard oblique views are left 75° ventral-right dorsal and right 75° ventral-left dorsal. The lateral view is obtained with the bird positioned in lateral recumbency with the neck extended. Foam wedges are used to support the skull such that the sagittal plane of the skull is parallel to the film. For the ventrodorsal view, the patient is placed in dorsal recumbency with the neck hyperextended such that the hard palate is parallel to the cassette. Tape is used to secure the beak to the cassette. Additional views may be required on a case-by-case basis. Endotracheal tubes may interfere with interpretation of skull views, especially the ventrodorsal. If pathology is suspected, the endotracheal tube can be removed and an additional radiograph obtained.

GASTROINTESTINAL CONTRAST STUDIES

Contrast studies of the upper and lower gastrointestinal (GI) tract are often indicated based on suspicion of space-occupying masses, ulceration, abnormalities in size or shape of coelomic organs, GI foreign bodies, alterations in GI motility or body wall abnormalities (Figs 25.5a-f).

Thirty percent weight to volume barium sulfate suspension is the most commonly used contrast medium. Approximately 25 ml/kg given by gavage tube into the crop is the suggested guideline. Iohexol[c] (240 mg iodine/ml) diluted 1:1 with tap water also may be used

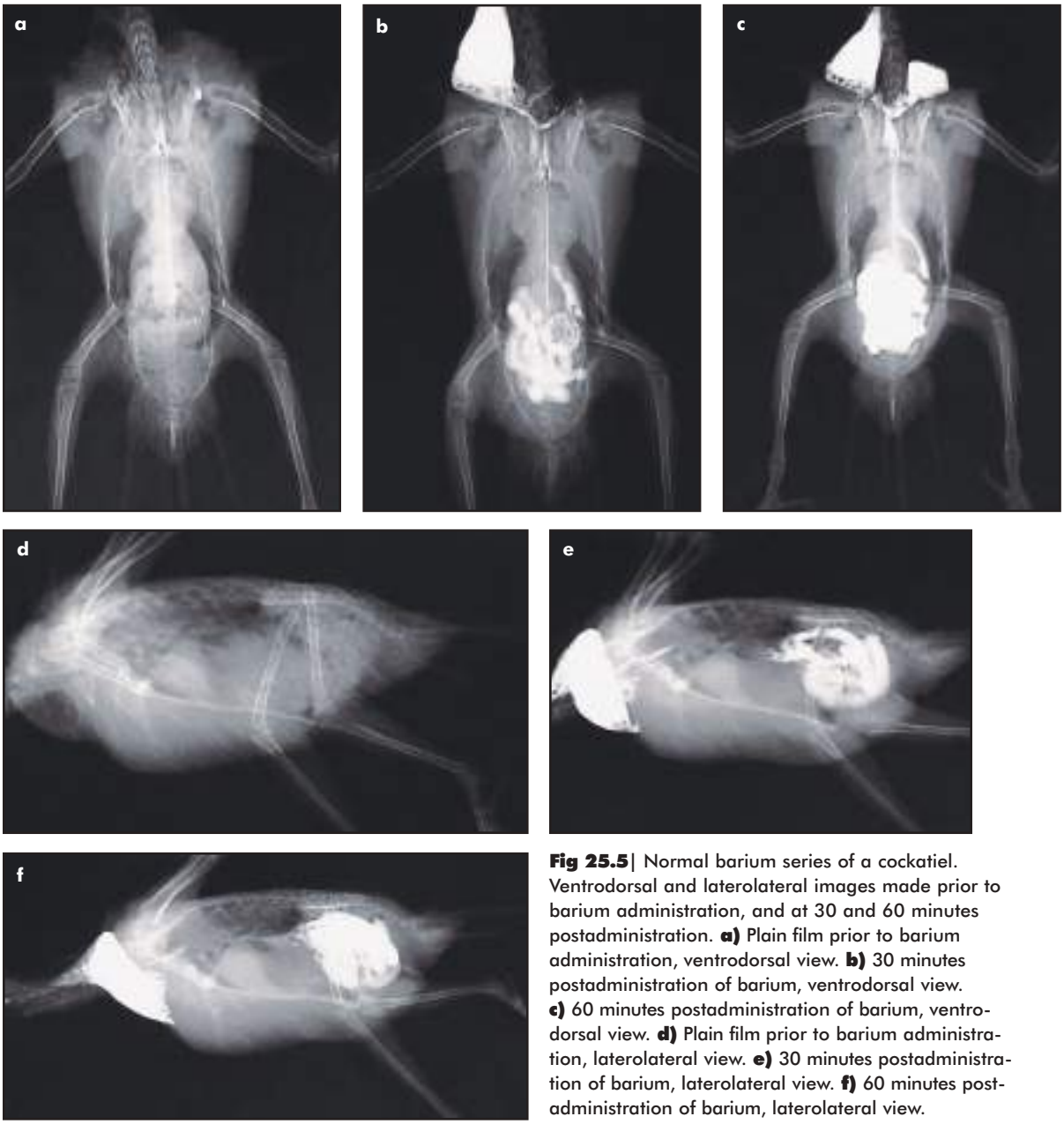


Fig 25.5| Normal barium series of a cockatiel. Ventrodorsal and laterolateral images made prior to barium administration, and at 30 and 60 minutes postadministration. **a)** Plain film prior to barium administration, ventrodorsal view. **b)** 30 minutes postadministration of barium, ventrodorsal view. **c)** 60 minutes postadministration of barium, ventrodorsal view. **d)** Plain film prior to barium administration, laterolateral view. **e)** 30 minutes postadministration of barium, laterolateral view. **f)** 60 minutes postadministration of barium, laterolateral view.

at 25 to 30 ml/kg.[4]

A preliminary study comparing GI transit time of psittacines restrained manually to those chemically restrained with isoflurane revealed no significant differences between the two groups.[11] Regardless of the type of restraint used, great care should be taken to avoid contrast aspiration, especially in the early stages of the study when the crop is distended. Precautions include avoiding external pressure on the crop, maintaining the head in a slightly elevated position and intubation of anesthetized birds.

The use of iohexol[c] results in significantly decreased GI transit time.[4] The crop-to-cloaca transit time of barium is approximately 3 hours, compared to approximately 1 hour with iohexol.[c] If barium is used, films are made at 5, 30, 60, 90, 120 and 180 minutes or possibly longer. A slightly different schedule of 1, 3, 15, 30, 60 and possibly 120 minutes is recommended if iohexol[c] is used. Iohexol[c] also eliminates concerns of contrast material residue if endoscopy or surgery is indicated. If perforation is suspected or possible, iodinated contrast should be used.

Retrograde or cloacally administered barium may be useful in detection of cloacal or colonic abnormalities such as ulcerations or masses. Caution should be used to avoid barium entering the oviduct or ureters.

Ultrasound

Ultrasonography uses the transmission and reflection of sound waves to produce an image. Organ visualization in the avian patient is limited when compared to mammalian species, as the ultrasound waves cannot penetrate the gas-filled air sac system. However, there are several applications for use of ultrasonography in the avian patient.

Due to the small size of most birds and the limited windows of accessibility, the ultrasound transducer must have a small contact area or footprint. Sector transducers, producing a wedge-shaped image, are appropriate as they tend to be smaller than linear transducers and allow good maneuverability. Their disadvantage is a smaller field of view compared to a linear transducer. This is of minimal consequence when dealing with the small avian patient. A transducer frequency of 7.5 MHz or higher is recommended.[9]

There are three acoustic windows to the coelomic viscera of the avian patient: (1) the cranioventral approach, on midline just caudal to the sternum; (2) the caudoventral approach, between the pubic bones; and (3) the lateral approach, in the flank area directly behind the last rib on each side. Based on the size of the bird and the size of the transducer, not all of these windows may be accessible in each patient.

While not always necessary, patients ideally should be fasted for 2 to 4 hours prior to examination. Food and gas within the gastrointestinal tract may impede visualization of other organs. Birds are restrained in dorsal recumbency, with the head and cranial coelom elevated with a 30° foam wedge to assist in visualization. Feathers are either parted or plucked and wetted with a small amount of isopropyl alcohol. Acoustic coupling gel is applied to the skin to provide adequate contact between the transducer and the skin. In the non-pathologic state, liver, heart and active gonads (usually the ovaries) are distinguishable. Spleen, normal kidneys and inactive gonads are difficult to identify.[9]

The indications for ultrasound include investigation of superficial soft tissue masses, hepatomegaly, cardiac disease, renomegaly, disorders of the reproductive tract and identification of ascites.

Hepatomegaly is commonly diagnosed with conventional radiography. The normal liver should not extend caudal to the sternum and has a characteristic coarse, homogeneous echotexture similar to the mammalian liver.[9] Hepatic disease can be focal or diffuse. The abnormal areas may be sampled by fine needle aspiration (FNA) or biopsy for definitive diagnosis. General anesthesia is recommended for both procedures.

Ultrasound-guided hepatic FNA is accomplished using a 22-gauge, 1-inch needle with a 6-inch extension set. A study comparing FNA sampling techniques found that collecting multiple parenchymal samples with the needle under 0.5 ml of suction produced the most high quality hepatic cells with the least hemodilution[14]. In this study of 27 normal Amazon parrots, no morbidity or mortality was associated with hepatic FNA. Percutaneous ultrasound-guided hepatic biopsies may be obtained using a 20-gauge Trucut needle. The depth of the biopsy will vary with the size of the bird.

Echocardiography in the avian patient is in its early stages.[9] The heart is usually visualized cranial to the liver via the cranioventral acoustic window. Standardized imaging planes and chamber sizes have yet to be established. Endocarditis, pericardial effusion, valvular insufficiency and cardiomyopathies may be diagnosed using mammalian criteria as a guide (see Chapter 12, Evaluating and Treating the Cardiovascular System).

The normal kidneys are not visible during ultrasound examination due to their anatomic position within bony depressions of the pelvis and the surrounding abdominal air sacs. Nephromegaly may be detected if the kidneys extend beyond normal landmarks, as described in radiographs discussed above, and can be characterized as focal or diffuse. Ultrasound-guided renal biopsy has not been reported.

Inactive gonads are not generally identified on ultrasonographic examination. Active or enlarged gonadal tissue can be evaluated with ultrasound. In the male, testes enlarged due to seasonal, neoplastic or inflammatory conditions may be identified. A study of the normal sonographic appearance of the reproductive tract of 52 hens of various species identified 17 of 34 (50%) active ovaries, all in hens greater than 70 g body weight.[9] Active ovaries were characterized by the presence of follicles in various states of development. Follicles are initially round with indistinct anechoic or hypoechoic inner structure. As development advances, the more echogenic yolk is visualized. The ovarian parenchyma, identified between follicles, has higher echogenicity and a non-homogeneous appearance due to the presence of rudimentary follicles. As ova progress through the magnum, they exhibit distinct separation of echogenic yolk surrounded by anechoic albumen. The hyperechoic shell is added in the uterus and is easily distinguishable. The shell prevents further examination of the inner structures of the egg. Cysts of the ovary are visualized as clearly defined, rounded, anechoic structures within the parenchyma. Cysts may occur singly or in multiples.

Ultrasonographic examination is particularly valuable in cases of suspected egg binding. Thin-shelled, non-shelled, malformed and fully shelled eggs can be identified. Mineralized eggs are visible as round to oval structures of varying echogenicities resembling onion layers. They are surrounded by anechoic to hypoechoic fluid. Concurrent salpingitis should be suspected when oviduct walls appear thickened. The inflammatory exudate in the lumen of the oviduct may be anechoic to hypoechoic (see Chapter 18, Evaluating and Treating the Reproductive System).

Computed Tomography

Computed tomography (CT) is a cross-sectional imaging modality that uses x-rays to create a digital image or slice (Fig 25.6). that can be reformatted into additional planes of view.[2] Image densities are similar to conventional radiographs. The cross-sectional image eliminates overlying structures so objects of interest are more accurately visualized. CT is best suited for evaluation of bone and air-filled structures. Soft tissue resolution is inferior to MRI (see below). Reports of the use of CT include examination of the skull, sinuses and the lower respiratory tract.[1,6,8,10] Machines vary in acquisition time and width of slice. Typically, the study can be performed in less than 10 minutes. General anesthesia is required for the study.

Magnetic Resonance Imaging

A review of the physics of magnetic resonance imaging (MRI) is beyond the scope of this chapter. Simplistically, a strong external magnetic force is used to align certain atoms within the body about a desired axis. The field is then turned off and the unit senses energy released as atoms return to their resting state.[2] The image produced is cross-sectional.

MRI is particularly useful for imaging soft tissue structures including the brain, spinal cord, coelomic organs and the upper respiratory tract. This MRI modality has been evaluated in the diagnosis and management of chronic sinusitis in psittacines.[16,17] In contrast to conventional skull radiography, where identification of disease was limited to osseous changes or changes in pneumatization, MRI was successful in the identification of caseous plugs, granulomas, a mucocele and a polyp.[16] The accurate localization of these lesions within the sinuses permitted the planning of an optimal surgical approach and drain placement for treatment.

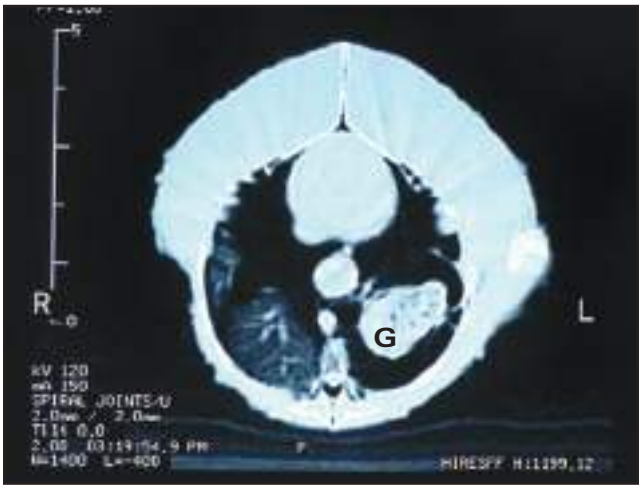


Fig 25.6 | Computed tomography image of a transverse slice through the coelom of an Amazon parrot at the level of the lungs. The right lung is normal. The normal architecture of the left lung is replaced by a cystic mass that was diagnosed as a foreign-body-associated granuloma (G) on histopathologic examination. The heart, keel and normal pectoral musculature are visible toward the top of the image.

Advances in the technology of MRI have decreased the time required for image acquisition, though general anesthesia is routinely required. MRI studies usually take a longer period of time to preform than CT studies. The decreasing cost and increasing availability of this imaging modality will increase its usefulness in avian diagnostics.

Myelography

Techniques for myelography in avian patients have been described.[5] Indications include evaluation of the spinal cord for compressive, traumatic or space-occupying lesions. MRI should be strongly considered as an alternative if feasible. In larger patients (1 kg and greater), a 25-gauge needle is placed at the thoraco-synsacral junction and 0.8 to 1.2 ml/kg of non-ionic iodinated contrast medium is injected into the subarachnoid space (see Chapter 17, Evaluating and Treating the Nervous System).

Excretory Urography

Excretory urography (EU) is indicated for identification of renal mass lesions, ureteral obstruction and abnormalities in renal excretion. The use of sodium diatrizoate (680 mg I/kg), iothalamate sodium (800 mg I/kg), and meglumine diatrizoate (800 mg I/kg) has been reported without adverse effects.[12] Contrast agents are warmed to body temperature prior to intravenous administration, and radiographs are made at 1, 2, 5, 10 and 20 minutes. The technique has been described in

further detail.[12] The absence of a renal pelvis and physiologic variations in avian renal function limit the usefulness of this procedure in birds. See Chapter 16, Evaluating and Treating the Kidneys for further considerations and alternate contrast agent doses.

Products Mentioned in the Text

a. Microvision Detail mammography cassette, Agfa, Greenville, SC, USA
b. Microvision Ci, Agfa, Greenville, SC, USA
c. Iohexol, Omnipaque®, Amersham Health, 1-800-654-0118

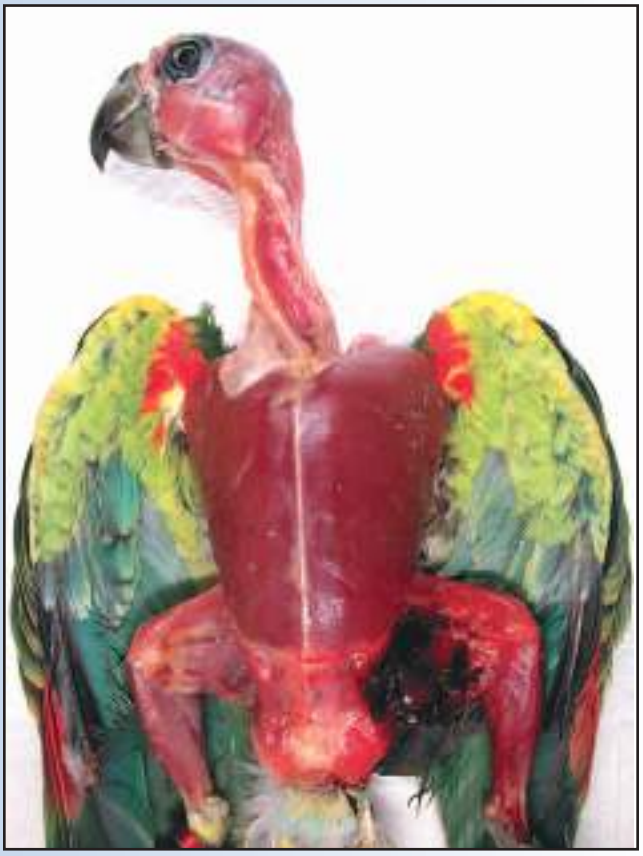
References and Suggested Reading

1. Antinoff N, et al: Correlation between computed tomography and anatomy of the psittacine sinus. Proc Assoc Avian Vet, 1996, pp 367-368.
2. Berry CR: Physical principles of computed tomography and magnetic resonance imaging. *In* Thrall DE (ed): Textbook of Veterinary Diagnostic Radiology 4th ed. Philadelphia, WB Saunders Co, 2002, pp 28-35.
3. Drost WT: Basic ultrasound physics. *In* Thrall DE (ed): Textbook of Veterinary Diagnostic Radiology 4th ed. Philadelphia, WB Saunders Co, 2002, pp 20-28.
4. Ernst SE, et al: Comparison of iohexol and barium sulfate as gastrointestinal contrast media in mid-sized psittacine birds. J Avian Med Surg 12(1):16-20, 1998.
5. Harr KE, et al: A myelographic technique for avian species. Vet Radiol Ultrasound 38(3):187-192, 1997.
6. Jenkins JR: Use of computed tomography (CT) in pet bird practice. Proc Assoc Avian Vet, 1991, pp 276-279.
7. Krautwald-Junghanns ME: Avian imaging. Proc 4th Europ Coll Avian Medi Surg, 2001, pp 343-353.
8. Krautwald-Junghanns ME, Kostka VM, Dorsch B: Comparative studies on the diagnostic value of conventional radiography and computed tomography in evaluating the heads of psittacine and raptorial birds. J Avian Med Surg 12(3):149-157, 1998.
9. Krautwald-Junghanns ME, Riedei U, Neumann W: Diagnostic use of ultrasonography in birds. Proc Assoc Avian Vet, 1991, pp 269-275.
10. Krautwald-Junghanns ME, Schumacher F, Tellhelm B: Evaluation of the lower respiratory tract in psittacines using radiology and computed tomography. Vet Radiol Ultrasound 34(3):382-390, 1993.
11. Lennox AM, Crosta L, Buerkle M: The effects of isoflurane anesthesia on gastrointestinal transit time. Proc Assoc Avian Vet, 2002.
12. McMillan MC: Imaging techniques. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Practice. Delray Beach, FL, HBD International Inc, 1999, pp 246-326.
13. Newell SM: Radiology for the avian patient/Y2K and beyond. Proc Assoc Avian Vet, 2000, pp 421-424.
14. Nordberg C, et al: Ultrasound examination and guided fine-needle aspiration of the liver in Amazon parrots (*Amazona* species). J Avian Med Surg 14(3):180-184, 2000.
15. Paul-Murphy JR, et al: Psittacine skull radiography: Anatomy, Radiographic Technic and Patient Application. Vet Radiol 31(4):218-224, 1990.
16. Pye GW, et al: Magnetic resonance imaging in psittacine birds with chronic sinusitis. J Avian Med Surg 14(4):243-256, 2000.
17. Romagnano A, et al: Magnetic resonance imaging of the avian brain and abdominal cavity. Proc Assoc Avian Vet, 1995, pp 307-309.
18. Rosenthal K, et al: Ultrasonographic findings in 30 cases of avian coelomic disease. Proc Assoc Avian Vet, 1995, p 1.
19. Rosenthal K, et al: Computerized tomography in 10 cases of intracranial disease. Proc Assoc Avian Vet, 1995, p 1.
20. Thrall DE, Widmer WR: Radiation physics, radiation protection, and darkroom theory. *In* Thrall DE (ed): Textbook of Veterinary Diagnostic Radiology 4th ed. Philadelphia, WB Saunders Co, 2002, pp 1-19.

CHAPTER
26

Diagnostic Value of Necropsy

MADELINE A. RAE, BS, DVM, MS, Dipl ABVP-Avian



Gross necropsy and postmortem diagnostic testing are important parts of avian medicine, requiring a systematic approach to the examination of organs and the collection of samples.

Necropsy examination functions as more than a way to satisfy the curiosity of the client, breeder or attending veterinarian — it provides important information that can be used in the diagnosis and treatment of future cases. Clinical signs and clinical pathologic findings are often not definitively explained until necropsy.

Postmortem information can be invaluable in educating clients regarding the seriousness of husbandry, nutritional and infectious disease conditions, thereby preventing them from making the same mistakes with subsequent birds. For grieving owners, necropsy findings can relieve them of some or all of the guilt associated with the death of a beloved pet.

Necropsy findings are an integral part of the flock database from which husbandry, management, treatment, vaccination and quarantine recommendations can be made.

In situations where the client may be dissatisfied with treatment or outcomes, it is wise to have a veterinary pathologist perform the necropsy. Developing a relationship with a veterinary pathologist is also highly recommended so that appropriate samples are submitted, optimizing the chances of arriving at a diagnosis. The veterinary pathologist should have training, experience and interest in companion and free-ranging birds.

Preparing for the Necropsy

The supplies needed for an avian necropsy are listed in Table 26.1. Although bottles or jars of neutral buffered 10% formalin should be available for the collection of specimens for histopathology, some cautions are in order. Ensure that formalin fumes do not contact tissues that are to be cultured for bacteria or viruses, as this can compromise the culture accuracy. Make certain that formalin fumes do not come in contact with blood or tissue cytological smears, as this can severely distort staining and interpretation.

A standardized necropsy form should be used and diagnostic specimen accession forms and instructions should be readily located. All specimen containers should be clearly labeled with the appropriate information. A refrigerator, a freezer, insulated shipping containers and coolant packs should be available for appropriate handling and shipping of specimens. After the necropsy has been performed, the carcass can be frozen and saved for a period of time, as frozen tissues may be useful if initial testing is inconclusive. Have arrangements in place for appropriate disposal of carcasses and infectious wastes.

Table 26.1 | Supplies Needed for Avian Necropsy

- Gram scale
- Apron
- Mask
- Gloves
- Disinfectant
- Necropsy checklist
- Scalpel handle and blades
- Scissors*
- Thumb forceps*
- Small rongeurs or toenail nippers for brain removal and bone cutting
- Ophthalmic scissors and forceps for small passerines, neonate and dead-in-shell
- Sturdy paper plates and/or plastic cutting board
- Microscope slides and coverslips
- Blood and fluid collection tubes
- Sterile saline
- Sterile culturettes (aerobic and anaerobic)
- Sterile cotton-tipped applicators
- Magnifying head loupe
- Good light source
- Sterile sealable plastic bags or sterile plastic vials for sample collection
- Plastic screw-top jars containing 10% neutral buffered formalin
- Sterile syringes and needles
- Stains (Gram's, Wright's, Giemsa or Diff-Quik, acid-fast)
- Butane lighter or other heat source to heat-fix smears for acid-fast staining

*Autoclave one set of scissors and forceps for collecting samples aseptically.

METHOD OF EUTHANASIA

In some instances, it may be appropriate to euthanize a sick bird in order to diagnose a flock problem. The method of euthanasia is important to consider because many injectable agents can cause severe artifacts in tissues. If at all possible, barbiturates should not be injected into the coelomic cavity, thoracic cavity or heart. The barbiturates are very acidic and crystals readily precipitate, causing severe tissue destruction that may obscure gross and histologic lesions. Even intravenous barbiturate solutions can cause intravascular erythrocyte lysis and some tissue damage as the solution pools within blood-filled organs.

Gas anesthetic agents seem to provide the least amount of tissue artifact (less muscle contraction artifact, no cell lysis). Once the gas agents anesthetize the bird, there is the opportunity to collect antemortem blood samples for hematology, serum chemistries, hemoparasite detection, serology and toxicology. The gas anesthetic agents can then be followed with intravenous euthanasia agents, if needed, and the amount of injectable agent necessary is usually quite reduced.

PREPARING THE BODY

The necropsy should be performed as soon after death as is possible. In order to prevent dry feathers from insulating the body and delaying cooling, wet the feathers with a detergent and water solution. The detergent and water also decreases the dispersal of feather and fecal dust into the local environment, thereby decreasing the transmission of infectious agents.

The body should be refrigerated, not frozen. Freezing can create artifacts in the tissues that may seriously obscure histologic lesions. Postmortem autolysis also can obscure histologic lesions, so if necropsy cannot be performed within 3 days, the body should be frozen, realizing that histopathology is likely to be compromised.

When shipping a cooled body, be sure the ice packs (or other frozen coolants) do not directly touch the body as this may freeze the body tissues, especially in very small birds. Wrap the ice packs in bubble wrap or newspaper to prevent freeze damage.

GUIDELINES FOR OBTAINING THE HISTORY

A detailed history should be obtained, just as the clinician would do upon seeing a live bird for the first time in the examination room (Table 26.2) (see also Chapter 6, Maximizing Information from the Physical Examination).

Table 26.2 | Considerations for Obtaining History Information

- Start with the age of the bird and sex, if known. Breeders are often very interested in having the sex of the dead bird confirmed at necropsy. If the dead bird is from a pair that is known to have produced fertile eggs, the knowledge of the mate can be deduced from the confirmation of the dead bird's sex. The breeder then knows which sex of bird to replace in the pair.
- Determine what the bird's purpose is, whether it is a dear pet, a display bird or a breeder bird.
- It is important to obtain a detailed description of the diet fed, and for how long, including any supplements and grit, brands used and their storage. Keeping a stored sample frozen in an airtight container may be useful. Quality control by the manufacturer and storage of formulated diets can be a problem. The resulting products may be over-formulated or rancid, resulting in toxic levels over time.
- Determine if there have been changes in appetite and/or droppings.
- How is water provided and how often is it changed?
- Have feeding practices or the diet changed recently?
- If this is a chick that is being hand-fed, obtain a detailed description of the feeding practices, brand of food with amounts and times per day, as well as any charts documenting weight loss or gain.
- Is the bird allowed to fly freely within the house?
- Ask for a description of the cage and its placement within the house (eg, near the kitchen where exposure to toxic fumes may occur).
- Is bedding material used and how often is the cage cleaned? What products are used for cleaning?
- Are there toys in the cage and does or can the bird chew on them?
- If this bird is from an aviary, a description of the aviary and floor plan can be very helpful.
- How many other birds are in the home or aviary and how many have died in the last year? Are any other birds in the household or aviary ill? Was the deceased bird in quarantine? Are any humans in contact with the ill birds?
- Have there been any recent changes in environmental conditions such as temperature changes?
- Have there been recent additions to the aviary or household?
- Ask about the reproductive history of the bird, such as a recent history of egg laying, dystocia or feeding of chicks.
- Obtain a description of recent clinical signs noticed prior to death and their duration.
- Ask about any other previous illnesses or conditions. Were any medications or treatments given to the bird? Be sure to ask about prescription medications as well as over-the-counter preparations.

PREVENTING CONTAMINATION

Perform the necropsy in a well-lighted, well-ventilated area (preferably under a fume hood), and wear gloves, a mask and, if possible, a disposable apron. Aerosols from feathers, feces and exudates can be infectious. This is particularly important with cases of chlamydophilosis and mycobacteriosis, which can be zoonotic. However, it also is important to contain the feather dander and feces in cases of avian polyomavirus and psittacine circovirus infections, so as not to contaminate the premises, your clothing or other adjacent birds.

Disinfectant solutions should be readily available for clean-up after the necropsy, but neither these solutions nor their fumes should come in contact with tissues being collected, as they may lyse cells and destroy microorganisms needed for culture.

Step-by-Step Necropsy Procedure

The particular routine used for gross necropsy of birds can vary, but what remains the same is that all organs and systems are examined, and the use of a checklist will ensure this. Use the Necropsy Checklist (Table 26.3) to document all findings, both normal and abnormal. Make this checklist a part of the medical record and send a copy to the veterinary pathologist with any fixed tissues.

It is important to collect samples of everything (all organs, the grossly normal and abnormal). Labeling sealable bags and formalin jars prior to the necropsy with the owner's name and the tissues enclosed can save time and prevent interruptions in the flow of the necropsy.

After the necropsy is completed, the decision of which samples to send and what tests to request can be made, and at the very least, the diagnosis will not be cremated with the carcass.

EXTERNAL EXAMINATION

The necropsy begins with an external examination. Record the band number and scan for microchips; these can be removed, labeled and saved as proof of identification. Weigh the bird using a gram scale and record the weight on the checklist. Palpate for obvious fractures; radiographs may be warranted in some instances.

Examine the skin and feathers: often, feather abnormalities may not be visible while the feather remains in the follicle. For example, the concentric pinching of the feather shaft, seen in psittacine circovirus infection, may not be visualized until the feather is plucked from the follicle. Look for stress bars in the wing and tail primaries (Fig 26.1). Collect multiple blood feathers, both plucked and in the follicle, along with any skin lesions (Fig 26.2) and place them in formalin. Check for any signs of

Table 26.3 | Necropsy Checklist

Owner's Name			Date of Necropsy
Animal's Name			Date of Death
Species			Euthanasia Method
Age	Sex		Body Weight at Necropsy
Band/Microchip #	Tattoo?		

Organ(s)	Normal	Abnormal	Description
Skin/Feathers			
Beak/Oral Cavity/Tongue			
Eyes/Ears/Conjunctivae			
Sinuses/Choana/Nasal Cavity			
Skeletal muscle/Bones/Joints			
Liver/Gall Bladder, if present			
Spleen			
Thyroids/Parathyroids			
Trachea/Lungs/Airsacs			
Kidneys/Adrenals			
Testes/Ovary/Oviduct			
Crop/Esophagus			
Proventriculus/Ventriculus			
Duodenum/Pancreas			
Jejunum/Ileum/Ceca, if present			
Colon/Cloaca			
Bursa/Thymus			
Brain/Meninges			
Spinal Cord/Vertebrae/Nerves			
Bone/bone marrow			
Middle & inner ear			
Heart/Great Vessels			
Gut contents wet mount results:			
Gut contents dried smear results:			
Organ impression smear results:			
Tissues in formalin:			
Tissues frozen:			

trauma or bruising. In neonates, closely examine the umbilicus for cleanliness and the adequacy of healing.

Examine the unfeathered portions of the legs and the feet for poxvirus lesions, bumblefoot, herpesvirus pododermatitis and self-mutilation (Figs 26.3, 26.4). Examine the uropygial gland, found at the base of the tail in some species, and collect it for histopathologic evaluation, as this can be a site of chronic inflammation and neoplasia.

Evaluate the beak, both the external and the intraoral surfaces (Fig 26.5). Open the mouth. Look at and under the tongue for abnormalities. Look in the choanal slit for mucus and exudate and for blunting of the choanal papillae (Fig 26.6). Salivary gland enlargement can occur at the base of the tongue and can be due to hypovitaminosis A, bacterial abscesses or, rarely, mycobacterial infections.

The nares and ear canals should be clear and free of debris or exudate. Examine the conjunctivae and the nictitating membranes. In Columbiformes, these tissues can be collected for *Chlamydophila* diagnostics, as they may contain elementary bodies.

The infraorbital sinuses should be opened as aseptically as possible, and swabs or aspirates collected for cytology and culture of bacteria, *Mycoplasma* and fungi (Figs 26.7, 26.8). Bacterial sinusitis is quite common in psittacines, but also occurs in passerine species, and caseous exudate is often seen (Fig 26.9).

In cockatiels (*Nymphicus hollandicus*) with "lockjaw," sinusitis and temporomandibulitis are common, as well as myositis of the mandibular muscles. The mandible and its attached muscles can be placed in formalin for histopathology. In these cases, bacteria such as *Bordetella avium*, *Enterococcus*, *Escherichia coli* and *Enterobacter* may be isolated. It is important to indicate to the

Fig 26.1 | Dystrophic feathers from a sulfur-crested cockatoo (*Cacatua galerita*) infected with psittacine circovirus. Note the concentric pinching of the feather shafts.

Fig 26.3 | Cutaneous pox lesions of the feet in a red-tailed hawk (*Buteo jamaicensis*).

Fig 26.4 | Avascular necrosis of the distal toes of a black-crowned night heron (*Nycticorax nycticorax*).

Fig 26.2 | Dermal lymphosarcoma in a western screech owl (*Otus kennicottii*).

Fig 26.5 | Cutaneous pox involving the commissures of the mouth in a red-tailed hawk.

bacteriology laboratory that *B. avium* is suspected because this organism is somewhat fastidious and colonies may take longer to appear. *Bordetella avium* also may cause tracheitis, bronchitis and pneumonia in cockatiels and rarely in other psittacines.

Several *Mycoplasma* species have been implicated in conjunctivitis and sinusitis in psittacines and passerines, but these require special media for isolation and are recovered uncommonly. In small passerines, cross-sections of the nasal cavity and sinuses can be submitted in formalin, decalcified if needed, and examined histologically. Sometimes the nasal cavity epithelium may be the only site of viral inclusions diagnostic for canarypox. Cryptosporidial rhinitis and conjunctivitis also can be diagnosed with this method.

COELOMIC CAVITY

The coelomic cavity examination is begun by placing the body in dorsal recumbency, incising the skin of the abdomen and peeling it back caudally over the abdomen

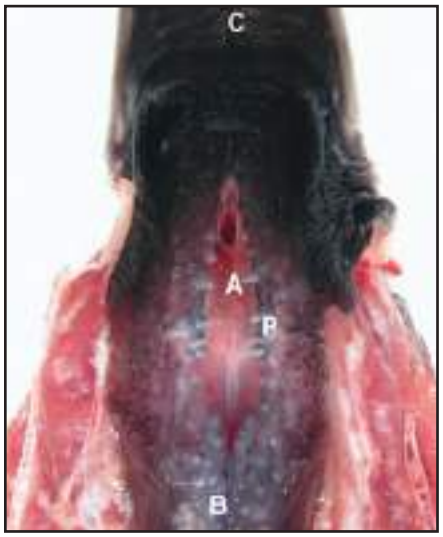


Fig 26.6 | Normal choanal slit of a blue-fronted Amazon parrot (*Amazona aestiva*). Choanal slit (A), caudal palate containing salivary glands (B), choanal papillae (P), palatine beak (C).

Fig 26.7 | View of the avian skull with the lateral wall of the infraorbital sinuses cut away in a blue-fronted Amazon parrot. Exudate may accumulate in the temporomandibular joint (TMJ), especially in cockatiels. Ear (E).

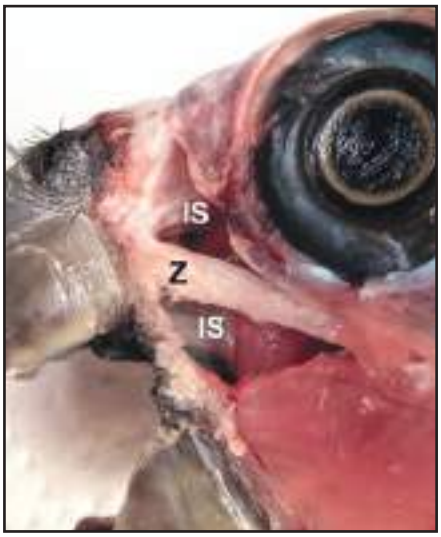


Fig 26.8 | View of the infraorbital sinuses in a blue-fronted Amazon parrot with the lateral wall removed. Infraorbital sinuses (IS), zygomatic or jugal arch (Z).

and cranially over the pectoral muscle mass. Assess the condition of the pectoral muscle, as this is a good measure of weight loss (Fig 26.10). Notice whether subcutaneous fat is present or absent and whether there is any bruising or edema. Grasp the sternum with thumb forceps and slightly elevate, maintaining tension on the abdominal wall. Make a transverse incision with the scalpel blade just caudal to the edge of sternum, being careful not to lacerate the liver. Remove the keel and pectoral muscles in one piece by cutting through the ribs and shoulder girdle with scissors or rongeurs.

Liver and Spleen

Assess the size, color and consistency of the liver. Hepatic margins should be sharp and not extend beyond the caudal edge of the keel in an adult bird (Figs 26.11-26.13). Note whether a gall bladder is present or absent, as not all species have one. Assess the condition of the air sacs and coelomic surfaces that are visible. If coelomic fluid is present, collect it with a sterile syringe for analysis. Aseptically collect samples of air sacs next, since they are delicate structures that readily disappear with further manipulation of the organs. Aseptically collect liver samples: one sample each for bacteriology, virus isolation or DNA probe testing, *Chlamydophila* testing and histopathology, and use any remaining tissue for toxicology and/or impression smears.

Grasp the ventriculus, elevate and incise through the attached membrane/air sac along the left margin and rotate the ventriculus counterclockwise to find the spleen. The spleen is nestled in the curve between the proventriculus and the ventriculus (Fig 26.14). Evaluate the size and shape of the spleen. Determining whether it is of normal size for the bird being necropsied requires some practice, so measuring the diameter can be helpful. The spleen is round in some species, such as Psittaciformes and Galliformes, and elongated or sock-like in Passeriformes and Columbiformes. Note the color and the presence of any pale foci in the spleen. Collect the entire spleen, dividing it into three samples: one for virology, one for *Chlamydophila* diagnostics and one for histopathology. The spleen is the single most important sample for the histopathologic diagnosis of avian polyomavirus infection, since this is where viral inclusions are most abundant.

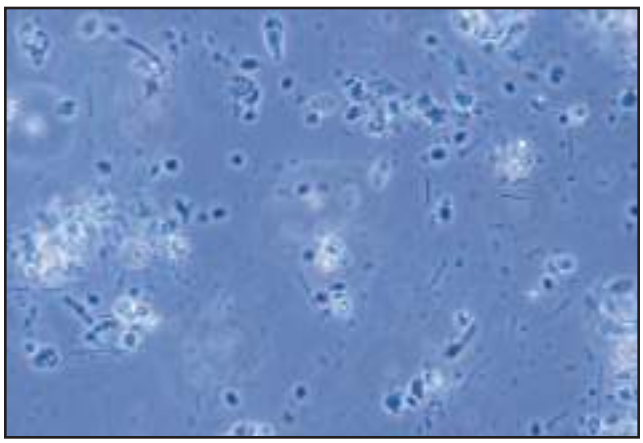


Fig 26.9 | Spirochetes in a wet mount of exudate from the infraorbital sinuses of a cockatiel chick (*Nymphicus hollandicus*), photographed under phase contrast.

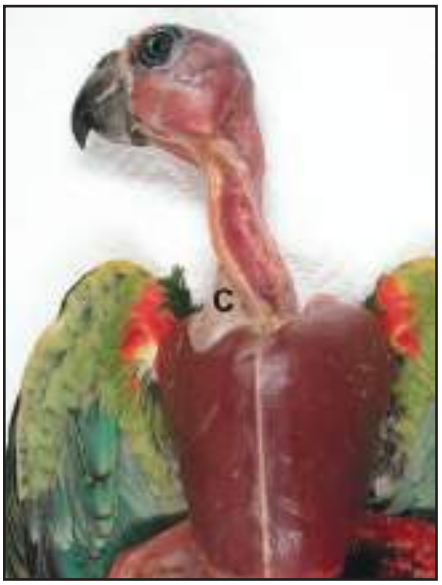


Fig 26.10 | View of the normal pectoral muscles of a blue-fronted Amazon parrot following removal of the skin. Crop (C).

Fig 26.11 | The liver extends beyond the caudal edge of the keel, indicating hepatomegaly in this mature red-tailed hawk with multiple mycobacterial granulomas in the liver.

Fig 26.12 | Marked hepatomegaly in a Lady Gouldian finch (*Chloebia gouldiae*). In this case, the hepatomegaly was due to lymphosarcoma, but a number of diseases can cause marked hepatomegaly in passerines.

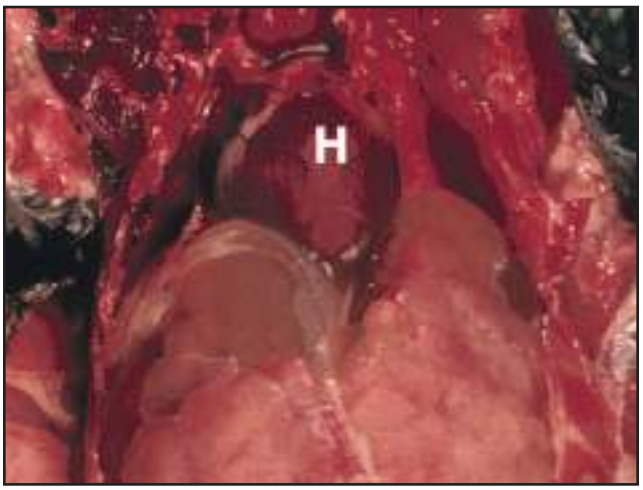


Fig 26.13 | Cholangiocarcinoma in an Amazon parrot (*Amazona* sp.). This tumor can have a very pleomorphic gross appearance. Heart (H).

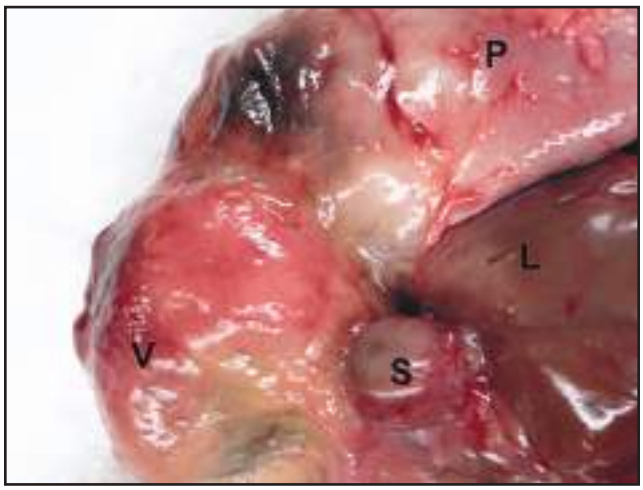


Fig 26.14 | Normal spleen in a blue-fronted Amazon parrot. Spleen (S), liver (L), proventriculus (P), ventriculus (V).

Genitourinary

Reflect the ventriculus and the intestinal tract to the right side of the bird to view the adrenals, gonads and kidneys, leaving the unopened gastrointestinal tract for last to avoid contamination of the other abdominal organs. The adrenals are often obscured by active gonadal tissue, so it is easier to collect the cranial division of the kidney with the adrenal and gonad(s) attached for histopathology. Adrenalitis is sometimes noted in unexplained death and may be the only abnormality in some psittacines with proventricular dilatation disease (PDD).

Sex the bird visually. In most species, only the left ovary and oviduct develop in females, but both testes develop in male birds. The gonads may be pigmented (brown or black) in some species, most notably cockatoos. Note the degree of development of the ovary and oviduct. Is there follicular development? If so, record the general size of the follicles. Is the oviduct hypertrophied?

Open the oviduct to look for exudate and tumors, and collect samples for bacteriology and histopathology as needed. Neoplasms of the oviduct may occur in the mucosa or myometrium. Testicular tumors are common in budgerigars (*Melopsittacus undulatus*) and ducks (Fig 26.15), but seasonal testicular enlargement also occurs. In some species, such as in many passerines, this enlargement can be mistaken for neoplasia; histopathology can usually distinguish between these two changes.

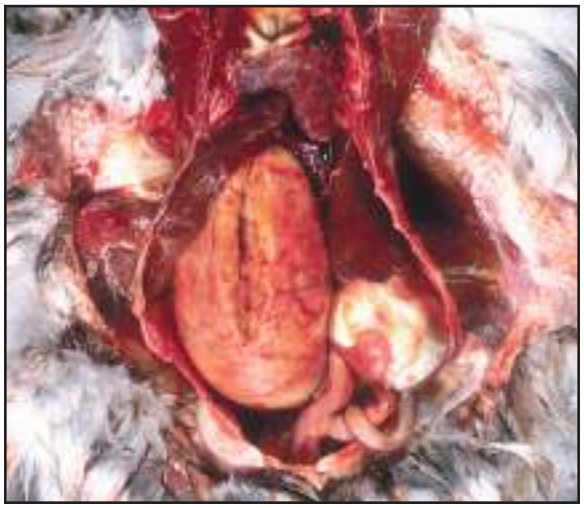


Fig 26.15 | Seminoma of the testes in a mallard duck (*Anas platyrhynchos*).

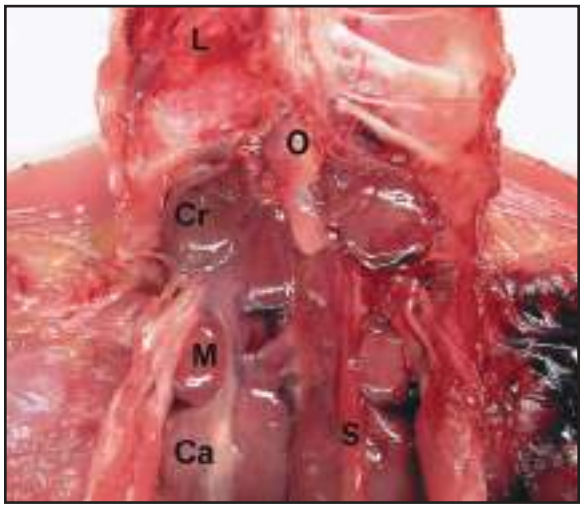


Fig 26.16 | Dissection displaying kidneys, reproductive structures and lung from a normal blue-fronted Amazon parrot. Lung (L), ovary, immature (O), cranial division (Cr), middle division (M) caudal division (Ca) of the kidney, salpinx or oviduct (S).

The kidneys are nestled in the renal fossae of the synsacrum, with the lumbosacral plexus lying deep to the caudal divisions of the kidney bilaterally (Fig 26.16). The ureters run down the ventral surface of the kidneys. In addition to the kidney/adrenal/gonad tissue collected as described above for histopathology, aseptically collect additional renal samples for virology, toxicology and bacteriology (if exudate is present).

In small birds (under 30 g), one can make an en bloc excision of the kidneys still in situ within the synsacrum and place this in formalin. After fixation, renal dissection is easier and/or the synsacrum can be decalcified and cross-sections of kidney together with bone can be cut. After removal of the kidneys, evaluate the lumbosacral plexus, especially in cases of pelvic limb weakness or malfunction. Samples of these nerves can be collected in formalin for histopathologic evaluation.

THORACIC INLET

Move to the thoracic inlet region. Identify the thyroids and parathyroids, located cranial to the heart and adjacent to the carotid arteries bilaterally, and collect them for histopathology (Fig 26.17). Goitrous changes were once quite common in budgerigars, but are less so with the advent of commercial diets. Hyperplastic goiter has been reported in juvenile macaws recently, but the cause is currently unknown. Lymphocytic thyroiditis also may be seen histologically, especially in Amazon parrots (*Amazona* spp.). Thyroid tumors are uncommon, but seem to be observed most often in cockatiels, where they tend to be very vascular.

Normal parathyroids are barely visible. When they are prominent, metabolic bone disease should be considered. Parathyroid hypertrophy is usually the only gross pathologic lesion found in the hypocalcemia syndrome of African grey parrots (*Psittacus erithacus*).

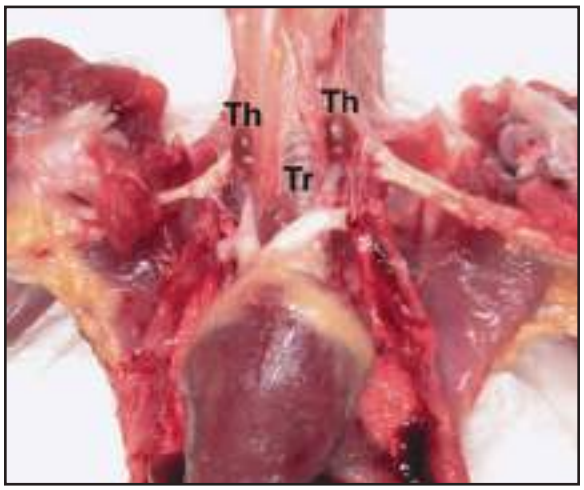


Fig 26.17 | View of the thoracic inlet, thorax and cranial abdomen following removal of the keel and pectoral muscles of a blue-fronted Amazon parrot. Thyroids (Th) are located cranial to the heart on either side of the trachea (Tr), close to the carotid arteries. The parathyroids are located at the caudal margin of the thyroids and are barely visible in normal birds.

In young birds, multiple pale lobules of thymic tissue can be found along the cervical region, from the jaw to the thoracic inlet (Figs 26.18, 26.19). Thymic tissue can be collected for virology and histopathology. Thymomas occasionally are seen as circumscribed, fairly encapsulated masses; they tend to be slow growing, undergoing cystic degeneration with large areas of hemorrhage. Thymic tissue is usually quite difficult to detect in adult birds.

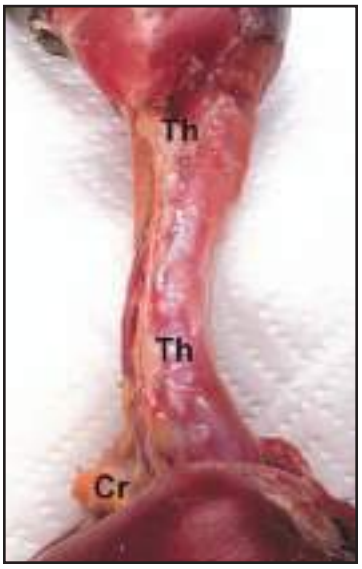


Fig 26.18 | Left lateral view of the neck of a normal young blue-fronted Amazon parrot revealing multiple lobules of thymic tissue along its entire length from the jaw to the thoracic inlet. Thymus (Th), crop (Cr).

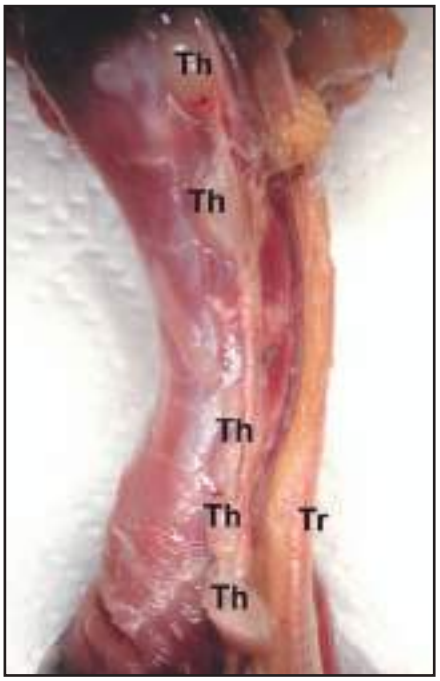


Fig 26.19 | Right lateral view of the neck of a young blue-fronted Amazon parrot revealing multiple lobules of thymic tissue along its entire length from the jaw to the thoracic inlet. Thymus (Th), trachea (Tr).

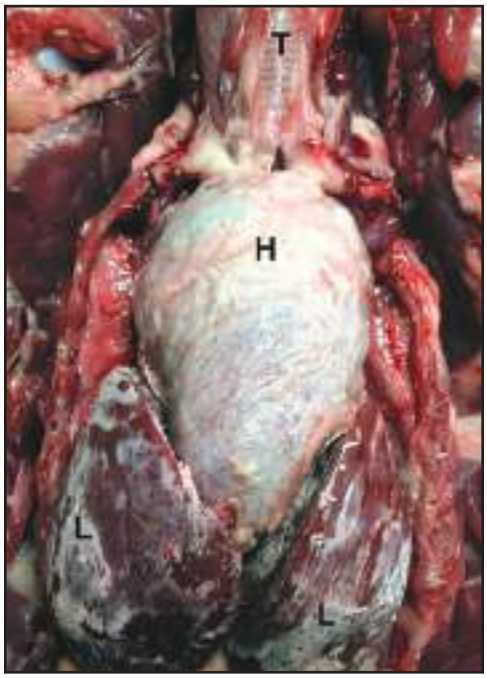


Fig 26.20 | Visceral gout in a red-tailed hawk with severe deposition of urates in the pericardial sac and in the perihepatic membranes. This is a very important gross lesion for the practitioner to recognize, because the urates may dissolve out of the tissue when placed in formalin. A smear of the white material can be examined under polarized light to reveal the typical urate crystal structure. Trachea (T), liver (L), heart (H).

Cardiac

Examine the heart, pericardium and great vessels. Visceral gout can cause the deposition of white, mucoid urate material in the pericardial sac (Fig 26.20). View a smear of this material under polarized light to confirm the presence of uric acid crystals. It is very important to recognize the gross appearance of visceral gout, because formalin fixation may dissolve the uric acid crystals and they may not be visible on histopathology (crystals do not dissolve if fixed in ethanol, but this is usually not a practical fixative for other reasons).

Suppurative pericarditis can be caused by a variety of bacteria such as *Pasteurella* and *Chlamydophila*. Cytologic examination of the pericardial exudate may reveal the causative organisms. Portions of the pericardial sac can be included in the tissues used for *Chlamydophila* diagnostics. Hydropericardium is a common finding in avian polyomavirus infection in juvenile psittacines.

Prior to removing the heart from the thoracic cavity, heart blood can be collected using a sterile syringe and needle for bacteriology. Smears of heart blood can be stained with Wright's stain and examined for hemoprotozoa and microfilaria, or Gram's stained to look for bacteria.

Cardiomyopathy, usually the dilative form, can occur in birds. The cause of the cardiomyopathy is often obscure by the time it becomes a clinical problem, but myocardial degeneration and fibrosis are often seen histologically. After removing the heart and great vessels, open the heart in the direction of blood flow, using water to rinse away blood and clots. (See Chapter 12, Evaluating and Treating the Cardiovascular System for measurements of the heart). Look for thrombi, valvular endocarditis lesions and pale areas in the myocardium. Congenital cardiac anomalies are rarely diagnosed.

Open the great vessels to look for atherosclerosis, which may involve the aorta, pulmonary artery or carotids. Atherosclerosis is characterized grossly by yellowish, raised, intimal plaques, but occasionally may be so severe that the carotids are completely obstructed. Mineralization of the great vessels also may occur in association with atherosclerosis or may be related to renal disease and hypervitaminosis D.

Atherosclerosis is most commonly seen in African grey parrots, where it can be mild to moderate, but also in obese, older Amazon parrots, older macaw species (*Ara* spp.) and captive raptors, where it can be so severe that it results in acute death (Fig 26.21). Atherosclerotic

Fig 26.21 | Atherosclerotic plaques on the luminal surfaces of the great vessels of the heart in an Amazon parrot (*Amazona* sp.). Note the smooth, glistening luminal surfaces of the normal heart on the left.

lesions also may be found in the coronary arteries, but usually these lesions are discovered upon histopathologic examination. Atherosclerosis is a commonly missed diagnosis because the vessels are not opened and examined.

It is best to place most of the heart in formalin so that multiple sections can be cut for histopathologic evaluation. Pale foci or streaks can indicate degenerative myopathy related to vitamin E/selenium deficiency, or myocarditis associated with septicemia or viral diseases such as West Nile virus or PDD. Petechial and ecchymotic epicardial hemorrhages are commonly seen in cases of acute death from avian polyomavirus.

Lungs

Examine the lungs in situ prior to removing them. Avian lungs are fixed in place within the avian thoracic cavity and are not freely moveable. Removal requires gentle teasing of the lung tissue away from the ribs. The avian lung is one tissue in which gross lesions may appear quite significant, but upon histopathologic evaluation turn out to be just passive congestion. Conversely, grossly normal lungs may contain significant histologic lesions. So, it is wise to always include lung for histopathology, even if it appears grossly normal. Collect a portion of the lung for bacteriology and virology and place the rest in formalin for histopathology. Because lesions can be focal or multifocal, it is best to include a large portion of at least one lung for histopathology.

In canaries and mynahs, a Wright's-stained impression smear of lung (along with impressions from liver and spleen) is very important in detecting the monocytic form of atoxoplasmosis, as *Atoxoplasma* organisms are not usually visible on histopathology. *Sarcocystis* organisms, bacteria and fungi also can be seen in impression smears of the lung.

ORAL CAVITY AND GASTROINTESTINAL TRACT

Cutting through the mandible at one of the lateral commissures allows access to the caudal pharynx and visualization of the glottis (Figs 26.22, 26.23). The larynx and trachea can then be opened down to the tracheal bifurcation, looking for hemorrhage, exudate, foreign bodies, granulomas, and parasites such as respiratory mites or *Syngamus* nematodes. Laryngeal papillomatosis can occur in the pharynx and may occlude the glottis (Fig 26.24).

Tissue samples should be collected for histopathology and virus isolation, as well as bacteriology and fungal culture if warranted. Fungal tracheitis, especially at the syrinx, can be diagnosed by cytologic examination of exudate or granulomatous material, fungal culture and/or by histopathology. Rarely, mycobacterial organisms can cause syringeal granulomas.

In canaries, *Enterococcus faecalis* can cause chronic tracheobronchitis; culture of the tracheal lumen is necessary for diagnosis. Viral tracheitis is rare in psittacine birds, but a herpesviral tracheitis, bronchitis and airsacculitis have been reported in *Neophema* parrots. Canarypox can produce a severe tracheobronchitis with intracytoplasmic inclusions. A severe, chronic tracheobronchitis can be seen with *Bordetella avium* in cockatiels; this organism also is often associated with the "lockjaw" syndrome of sinusitis and temporomandibulitis.

Esophagus and Crop

Going back to the pharynx, the cut can extend downward the length of the esophagus and into the crop, looking for lacerations, punctures, peri-esophageal abscesses and other abnormalities. In game birds, the esophagus and crop may exhibit moderate to marked thickening and mucus production due to capillariasis. A wet mount scraping from the crop can reveal the typical bipolar capillarid ova. In juvenile psittacines, thickening and "Turkish towel" appearance of the crop mucosa is often due to candidiasis, and either a wet mount smear, cytology or Gram's stain of a crop mucosal scraping can be diagnostic (Fig 26.25).

Trichomonads can be found in wet mounts from the oral cavity and/or crop of Columbiformes and raptors, but also may occasionally be found in the crops of budgerigars and passerines. The crop contents can be collected in a plastic bag and frozen, if there is any suggestion of toxin ingestion. A large section of crop, to include a large vessel and adjacent nerve, should be collected for histopathology, since PDD lesions are often closely associated with the nerves.

Fig 26.22 | Cutting through the mandible allows opening of the entire oral cavity and better visualization in a blue-fronted Amazon parrot.

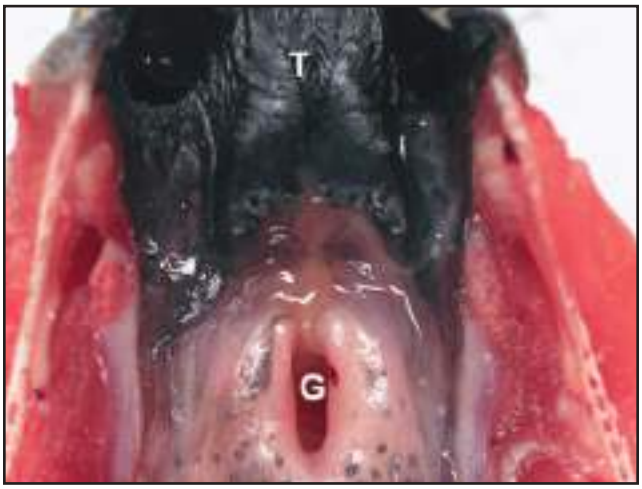


Fig 26.23 | Normal glottis from a blue-fronted Amazon parrot. Tongue (T), glottis (G).

Fig 26.24 | The glottis of this lilac-crowned Amazon parrot (*Amazona finschi*) is occluded by laryngeal papillomatosis.

Fig 26.25 | Severe crop mycosis in a cockatiel chick.

At this point, the esophagus distal to the crop can be transected. Caudal traction of the distal esophagus and sharp dissection of the mesenteric attachments can be utilized to remove the entire gastrointestinal tract.

Bursa of Fabricius

Continue the dissection to make a circular incision around the vent, leaving a margin of intact vent skin and the bursa of Fabricius attached to the tract. The bursa is present in young birds usually less than 6 to 12 months of age and is located dorsal to the cloaca. The bursa should always be collected when it is present and divided in half. Submit one half in formalin for histopathology and save the other half for virology and/or DNA probe testing.

The bursa is important in diagnosing psittacine circovirus, especially in young African grey parrots that die acutely without feather lesions, since the bursa may be the only site where viral inclusions are found. Circoviral inclusions also can be found in the bursa of young pigeon squabs dying from a variety of secondary infections. Lesions in the bursa are often non-specific as to etiology, but can indicate the acuteness or chronicity of stress.

Proventriculus and Ventriculus

Open the distal esophagus with scissors, continuing on into the proventriculus and ventriculus. Evaluate the stomach contents for amount and any foreign material. Washing the contents into a bowl or strainer can allow the food material to be rinsed away, leaving metallic and other foreign bodies behind. Collect and freeze the contents for possible toxicologic analysis. Rinse the mucosa with water and make wet mount and dried smears of mucus and/or mucosal scrapings. Do not separate the proventriculus and ventriculus.

The isthmus (the junction between the proventriculus and ventriculus) is a common site for avian gastric yeast (formerly known as megabacteria); its suggested new name is *Macrorhabdus ornithogaster* (see Chapter 30, Implications of *Macrorhabdus* in Clinical Disorders) (Fig 26.26). Grey-cheeked parakeets (*Brotogeris pyrrhopterus*) seem to be prone to the development of gastric carcinoma at the isthmus and the gross lesions are often unexciting. Collect a large specimen of proventriculus, isthmus and ventriculus (all in one piece if possible), containing at least one large serosal nerve and blood vessel, for histopathology.

In small birds, the entire proventriculus and ventriculus can be placed in formalin. A large specimen

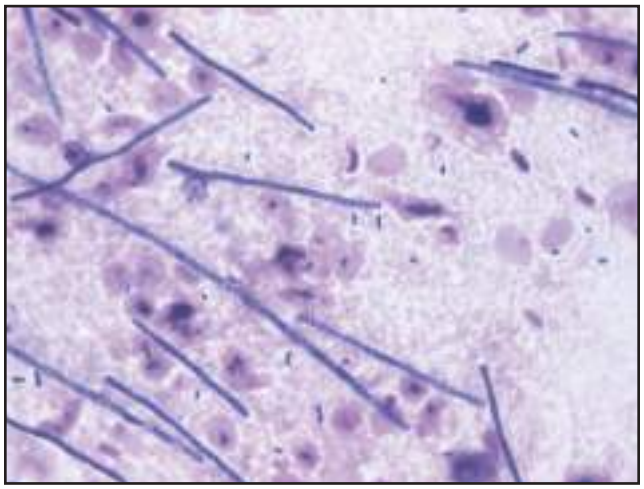


Fig 26.26 | Large gram-positive organisms consistent with the yeast *Macrorhabdus ornithogaster* (formerly known as megabacteria) are seen in a Gram's-stained smear of intestinal contents from a cockatiel. Note the size difference between these fungal organisms and the smaller gram-positive bacilli.

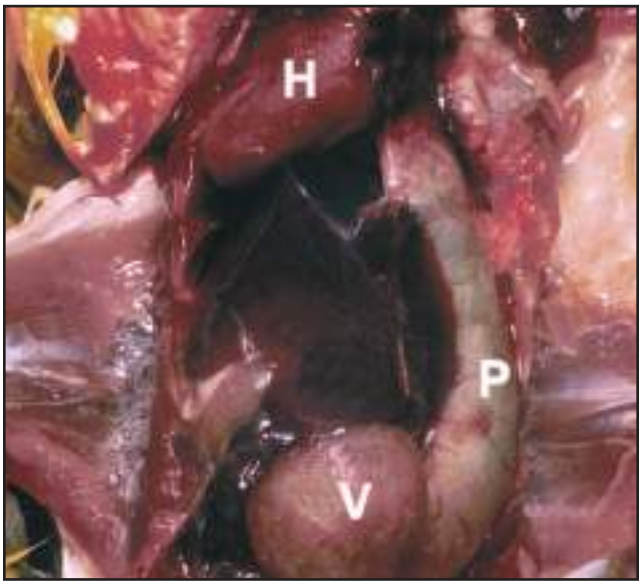


Fig 26.27 | Marked dilatation of the proventriculus (P) in a macaw (*Ara ararauna*) with proventricular dilatation disease (PDD). H= heart, V= ventriculus.

allows multiple sections to be examined by the veterinary pathologist in the search for nerves and plexi. Dilatation of the proventriculus and/or ventriculus is a hallmark gross lesion of PDD, but in juvenile psittacines being hand-fed, these organs also may be dilated as a normal finding. Histopathology is required to differentiate between PDD and normal juvenile underdevelopment of the proventriculus and ventriculus (Fig 26.27).

Foreign body penetration of the ventricular wall can occur in any species, but is most common in waterfowl and ratites. Nutritional muscular dystrophy (degenerative myopathy) can be seen in some species as white streaks in the ventricular muscle as a manifestation of vitamin E/selenium deficiency. Endoventricular mycosis (fungal invasion of the koilin lining of the ventriculus) can be seen histologically and is a common finding in debilitated passerines, despite the usually unremarkable gross appearance.

Duodenum and Pancreas

Open the outflow tract from the ventriculus and proceed into the duodenal loop. The largest limb of the pancreas lies in the duodenal loop mesentery while the small splenic lobe of the pancreas is located adjacent to the spleen. Pancreatic lesions are fairly common histologically, but gross lesions may not be very striking. The pancreas also is one of the first organs to undergo postmortem autolysis.

Quaker parrots (*Myiopsitta monachus*) are prone to the development of acute pancreatic necrosis of unknown etiology. Fat necrosis and serositis may accompany pancreatitis and pancreatic necrosis. Quaker parrots that survive the initial insult may develop severe pancreatic atrophy and fibrosis. Inclusion body pancreatitis can be seen with herpesvirus and adenovirus infections. Lymphoplasmacytic pancreatitis in *Neophema* parrots is associated with paramyxovirus infection. Pancreatic necrosis also is a common lesion in West Nile virus infection.

Vacuolar changes and necrosis of acinar cells may be seen in zinc toxicosis, but these lesions can be readily obscured by even mild postmortem autolysis. The pancreas concentrates zinc in the acinar cells and should be collected for toxicologic analysis, along with liver and kidney, to diagnose zinc toxicity. Collect a sample of pancreas for virology. Also submit in formalin a transverse section through the duodenal loop with pancreas attached, as this helps to identify the duodenum.

Yolk Sac

In neonate, the yolk sac and stalk should be evaluated for the degree of absorption. In psittacine and passerine chicks, the yolk sac is usually quite tiny by 3 days after hatching. Collect a sterile sample of the yolk material for culture and place the rest of the yolk sac (wall and contents) into formalin. Yolk sacculitis and yolk sac retention are common problems in neonatal ratites.

Intestines

Continue opening the intestine through the jejunum and ileum to the ceca (if present in the species) and colon. Collect sections of intestine for histopathology. Opened intestinal sections are usually best, as this gives the mucosa a chance to fix rapidly (Fig 26.28). Do not disturb the mucosa by scraping or handling, as artifacts

Fig 26.28 | Intestinal volvulus in an ostrich (*Struthio camelus*).

can confuse or obliterate the histologic diagnosis.

Wet mounts of intestinal contents (usually two different sites) are helpful in diagnosing parasitic and bacterial problems. Wet mounts should be examined for parasite ova and oocysts, as well as flagellates, yeast and motile bacteria. Sections of bowel can be tied off with string or suture and submitted for culture. In some cases, both aerobic and anaerobic culture may be warranted (Fig 26.29).

If necrotic lesions are encountered in the intestinal mucosa, clostridial disease should be considered. Quail disease caused by *Clostridium colinum* is a common problem in quail, and typical "button ulcers" can be seen in the intestines as well as "crateriform" necrotic lesions in the liver. Clostridial enteritis, usually caused by *Clostridium perfringens*, is becoming more commonly recognized in psittacines, especially nectar eaters such as lories and lorikeets. Clostridial organisms in large numbers can cause acute necrohemorrhagic enteritis. Finding large gram-positive bacilli, with or without spore formation, as the primary organism on a Gram's-stained smear of intestinal contents gives a presumptive diagnosis that should be followed by anaerobic culture (Fig 26.30). Because exposure to oxygen in the air can inhibit clostridia, it is wise to tie off a loop of unopened, affected bowel with string or suture and place it in a sealable bag with the air evacuated prior to sending it for anaerobic culture.

A wide variety of other bacteria can cause enteritis and septicemia. Gram-negative organisms, especially Enterobacteriaceae, are common infectious agents in psittacines and passerines. In addition, *Campylobacter* spp. and *Yersinia pseudotuberculosis* are more common in canaries and exotic finches. *Campylobacter* organisms usually require special media and microaerophilic incubation conditions, so it is wise to alert the bacteriology laboratory when this organism is suspected.

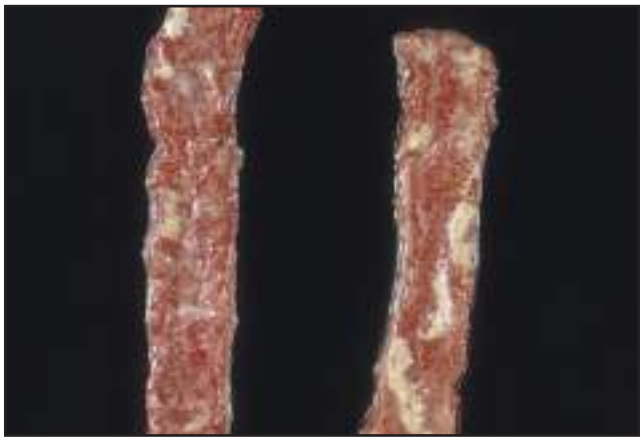


Fig 26.29 | Fibrino-suppurative enteritis in a tufted puffin (*Fratercula cirrhata*).

Multifocal granulomas or thickened areas of bowel can be indicative of mycobacteriosis. These sites should be collected for histopathology, and special acid-fast tissue stains can be applied to paraffin sections to demonstrate the organisms. Alternatively, impressions or scrapings from these sites can be stained with a rapid acid-fast stain for a quick, presumptive diagnosis. Sections of affected bowel can be collected for mycobacterial culture.

Intestinal neoplasia is fortunately uncommon in birds, but needs to be included in the differential diagnosis of thickened or proliferative bowel lesions.

Flagellate protozoa and coccidial organisms also may produce enteritis. Flagellates (including *Giardia* spp. and *Cochlosoma* spp.) are diagnosed by fresh wet mount smears of intestinal contents, but they are nearly impossible to diagnose on histopathology. Coccidiosis can be diagnosed by wet mount smears of intestinal scrapings and by histopathology. Nematode and cestode parasites are uncommon in domestically raised psittacines and passerines, but geographic pockets of these parasites may exist and should always be considered (Fig 26.31). These parasites are still common in ground-feeding and feral or wild birds.

Ceca

Many species of birds do not possess ceca. Psittacines do not. Passerines and Columbiformes have tiny vestigial ceca composed of lymphoid tissue, while Galliformes, Anseriformes and ratites possess large bilateral ceca.

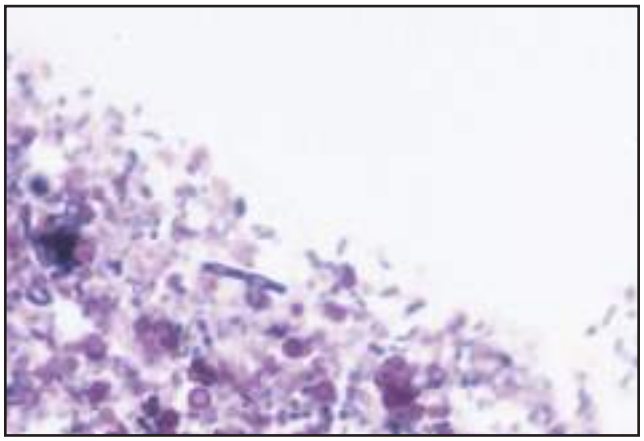


Fig 26.30 | A Gram's-stained smear of intestinal contents from a lory (*Trichoglossus haematodus*) that died of acute necrohemorrhagic enteritis. *Clostridium perfringens* was isolated from the intestinal tract. Note the large gram-positive rods with subterminal spores.

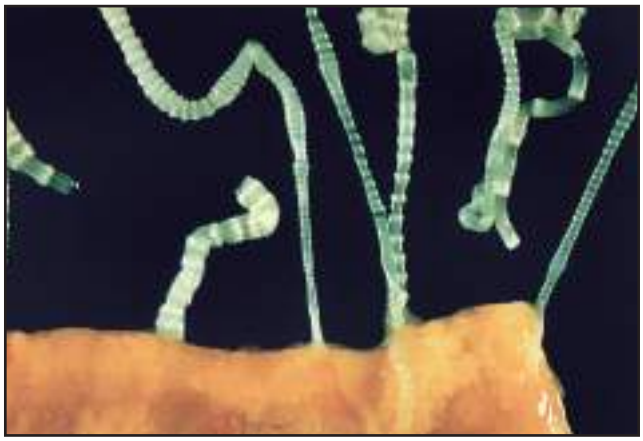


Fig 26.31 | Tapeworms attached to the intestinal mucosa of a great horned owl (*Bubo virginianus*).

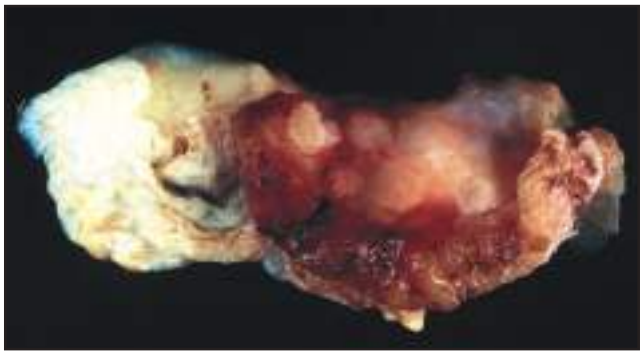


Fig 26.32 | Cloacal papillomatosis is seen in a lilac-crowned Amazon parrot. The cloaca has been opened caudally to cranially.

These should be opened to look for cecal worms and their contents should be included for culture. Cecal contents should be included in culture for *Salmonella*, especially in Galliformes.

Colon

Colon contents should start to look like fecal material as one moves toward the cloaca. Open the cloaca to look for papillomatous lesions, cloacoliths, trauma, inflammatory lesions and neoplasia (Fig 26.32).

In summary, intestinal samples should include the following: wet mounts from at least two different sites, smears for Gram's stain and possibly acid-fast stain, contents for aerobic and possibly anaerobic bacteria or *Campylobacter* culture, tissue for histopathology and ingesta for virology (direct electromicroscopy, virus isolation and/or DNA probes), and toxicology.

NEUROLOGIC

The brain and spinal cord can be very important in the diagnosis of some diseases, especially PDD. The dorsal calvarium should be carefully removed with rongeurs (Fig 26.33). Visualize the brain in situ for any obvious abnormalities such as abscesses, which should be cultured. Remove the brain by inverting the skull and transecting the ventral and cranial attachments (Fig 26.34). Collect a portion of the forebrain for virology and toxicology, and fix the rest of the brain in formalin. In neonates, the brain is so soft that making a cut through the dorsal skull and placing the entire calvarium containing the brain in situ into formalin is recommended. After fixation, the brain will harden somewhat and it can be removed more easily without damaging it.

A similar procedure can be followed for the cervical spinal cord. Cut the vertebral column with cord in situ into 2- to 3-cm pieces and fix in formalin overnight. This process will allow easier removal using rongeurs, with minimal damage to the less fragile, fixed spinal cord. In very small birds, cross-sections of the cervical vertebral column with the spinal cord in situ can be decalcified and examined histologically.

In birds with head tilt or neurologic disease, especially *Neophema* parrots and exotic finches, fix a large portion of the petrous temporal bone containing the middle ear. This bone can later be decalcified by the veterinary pathologist and sectioned to examine the middle ear for inflammation and viral inclusions associated with paramyxovirus infections. Congestion of the vascular sinuses in the bones of the skull is a common finding, but it is significant only if there also is corresponding subdural hemorrhage or bleeding of brain parenchyma.

The eyes can be removed and fixed in formalin if there is any suspicion of blindness, ocular or neurologic disease. Dissection through some orbital bone may be required for removal. Remember that the avian eye has bony scleral ossicles, which may make their sectioning somewhat more difficult. The globes can be transected at the optic nerves or dissection can be carried out

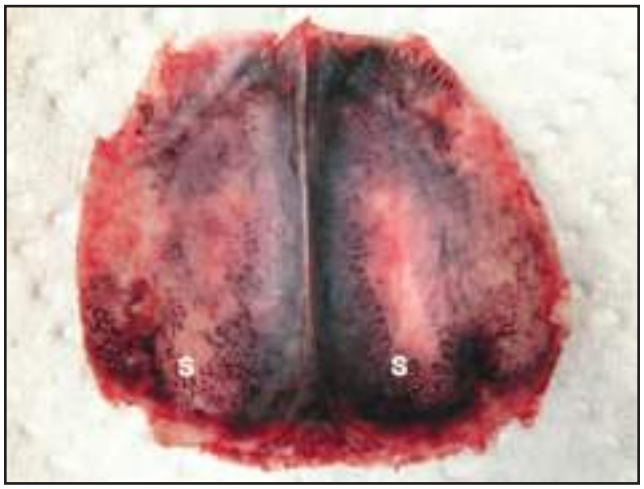


Fig 26.33 | View of the dorsal surface of calvarium of a psittacine bird with congested vascular sinuses (S).

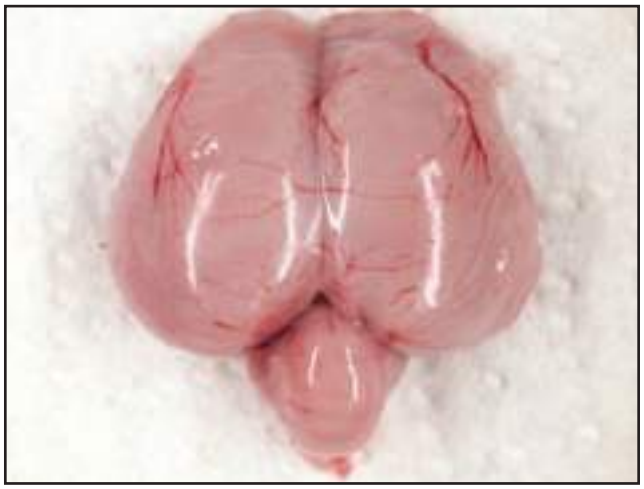


Fig 26.34 | Dorsal surface of a normal brain from a blue-fronted Amazon parrot.

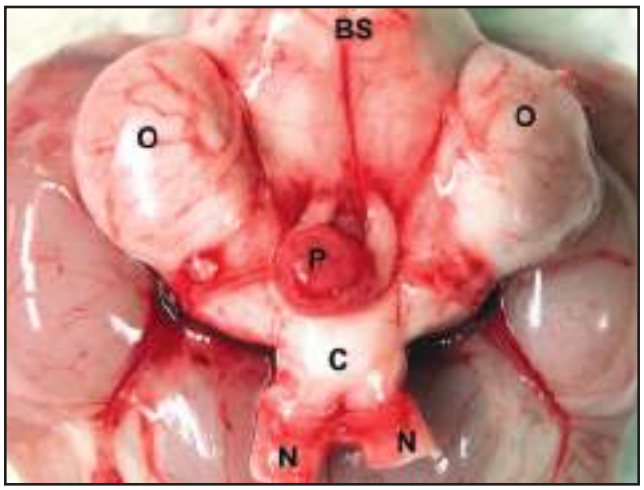


Fig 26.35 | View of the ventral surface of the brain from a blue-fronted Amazon parrot. Pituitary (P), optic lobes (O), brainstem (BS), optic chiasm (C), optic nerves (N).

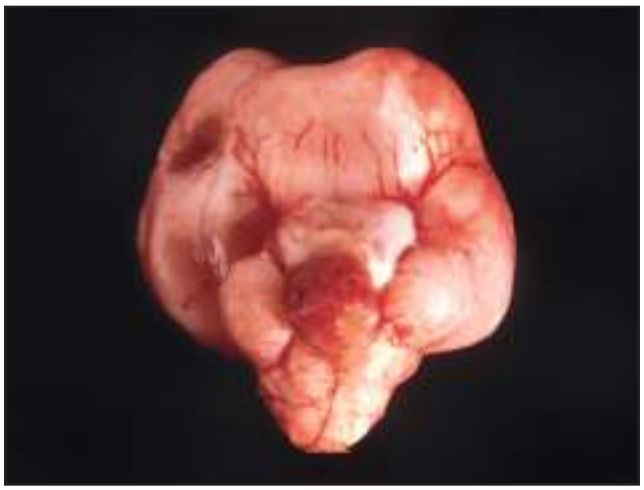


Fig 26.36 | A pituitary tumor in a budgerigar (*Melopsittacus undulatus*).

through the ventral calvarium to keep the optic nerves and chiasm intact and attached to the brain. On the ventral surface of the brain near the optic chiasm is the pituitary (Fig 26.35). Tumors of the pituitary have been reported in budgerigars and cockatiels (Fig 26.36).

MUSCULOSKELETAL

Bone marrow can be collected by aspiration of the femur and smears made and stained for cytologic evaluation. Collect a segment of femur using rongeurs and place it in formalin. Once fixed, the previously fragile bone marrow can be dissected out and examined histologically. Leukemic or aplastic processes can be diagnosed from bone marrow samples, and circovirus inclusions also may occasionally be seen histologically.

Samples of skeletal muscle should be collected for histopathology. Muscular lesions may include trauma, hemorrhage, degeneration, mineralization, and injection or vaccine site reactions. Myositis, degenerative myopathy and *Sarcocystis* infection can be diagnosed histologically (Fig 26.37). Open the joints of the pelvic and thoracic limbs and look for exudate; collect synovial fluid with a sterile syringe for bacterial and mycoplasmal culture, although exudate also can be caseous.

Articular gout can be diagnosed by examining the exudate on cytology or by histopathology. The lesions of degenerative joint disease, periarticular proliferation and proliferative synovitis are fairly common in the joints of the feet, but also may occur in the shoulder, stifle and hock. Any bone or joint lesions demonstrated radiographically should be opened and sampled for culture and histopathology (Fig 26.38).

The flexibility of bones (eg, tibiotarsus, ribs) can be used in the assessment of the adequacy of mineralization. The bones should break with an audible snap if mineralization is normal. The rachitic "rosary" at the costochondral or costovertebral junctions and deformation of the keel or other long bones are obvious lesions of metabolic

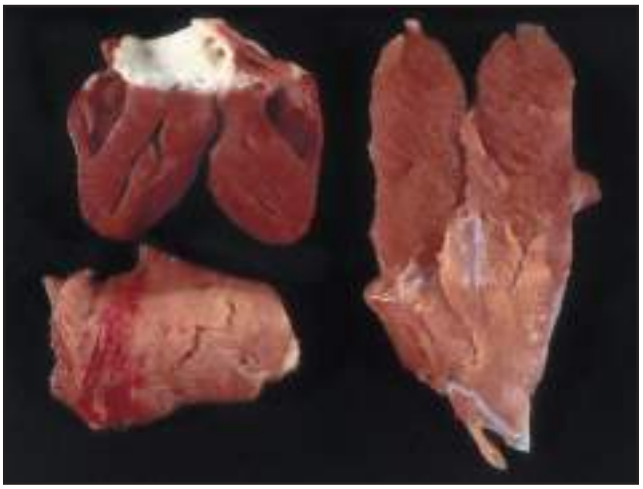


Fig 26.37 | Pale areas in skeletal muscle from an emu (*Dromiceius novaehollandiae*) with degenerative myopathy. The heart muscle appears grossly normal.

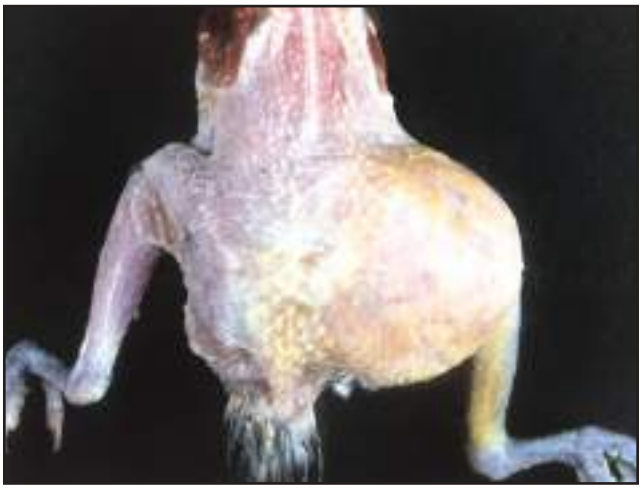


Fig 26.38 | A proliferative osteosarcoma involving the right leg and pelvis of a peach-faced lovebird (*Agapornis roseicollis*).

bone disease. Sections of bone, especially areas of the metaphyses and epiphyses, can be examined histologically for metabolic bone disease.

Ratites are prone to developing angular limb deformities. The "rubber rhea" syndrome is often due to hypophosphatemic rickets. Other ratite limb deformities can be multifactorial, but nutritional imbalances in calcium, phosphorus and vitamin D_3, growth rates and problems with substrates are often implicated. Flock problems with limb deformities in ratites can be investigated through feed/forage analysis and bone ash analysis.

SMALL BIRDS AND DEAD-IN-SHELL

Necropsy of very small birds and neonates (under 15 g) is challenging. One can open the coelomic cavity and thorax and fix the entire body in formalin; opening the ventriculus as well is recommended for best fixation. The veterinary pathologist can then carry out dissection. It is very difficult to get good fixation of tissues in birds weighing more than 20 g, so this technique should not be used for them.

Dead-in-shell and egg necropsies can be performed, but there are limited lesions and testing available. Open the egg as aseptically as possible at the air cell end. Collect samples aseptically for bacterial culture, virology and DNA probes. Assess gestational age and positioning of the embryo or chick. Then place the embryos and membranes in formalin. Histopathologic diagnosis is often limited by autolysis, since the eggs often remain in the incubator for a period of time after embryonic death. Histologic examination of the blastodisk can help determine if the eggs are truly infertile or whether early embryonic death occurred post-fertilization.

Ancillary Testing of Samples Collected at Necropsy

After the necropsy has been concluded, the remaining parts of the carcass can be placed in a sealable plastic bag and frozen until diagnostic testing has been completed. Examine wet mounts of intestinal contents and crop or oral cavity scrapings as quickly as possible in-house for parasite ova, oocysts, motile flagellates, yeast and motile bacteria.

MICROBIOLOGY

Stain impression smears from organs and bone marrow, smears of exudate, or cells from fluid analysis with a Wright's stain and examine for cell types and microorganisms, including bacteria and fungi. If bacteria are seen on the cytologic preparations, a stained smear can be destained and restained with Gram's stain, or another smear can be stained. If macrophages with "ghost bacilli" are seen on the cytologic preparation, an acid-fast stain is in order to attempt to demonstrate mycobacterial organisms.

A Gram's stain of the colonic contents can be performed in-house to provide a quick evaluation of the presence of abnormal bacterial populations, and then followed up with culture. This type of Gram's stain is especially important in detecting possible clostridial organisms, which would then prompt an anaerobic culture. Avian gastric yeast also can be detected in fecal smears or from scrapings of the isthmus stained with cytologic stains.

Composite Samples

A collection of liver, spleen and air sac can be submitted

for *Chlamydophila* diagnostics. A Gimenez or Macchiavello stain can be performed on impression smears of air sac, liver and spleen for the demonstration of elementary bodies. In Columbiformes, conjunctiva and nictitating membrane should be included, as elementary bodies may be confined to this location in these species. Fluorescent antibody and chlamydial culture may be available at certain laboratories, and some also can perform a DNA probe for *Chlamydophila* on a swab from the combined surfaces of liver, air sac and spleen.

Tissues, exudates or swabs can be submitted to diagnostic laboratories for bacterial, mycoplasmal or fungal culture as indicated. With the exception of samples for *Campylobacter*, which does not survive freezing well, these samples can often be frozen if not sent for culture immediately.

Special media is required for the culture of *Mycoplasma* spp. Alert the bacteriology laboratory if fastidious organisms such as *Campylobacter* spp. and *Bordetella avium* are of interest in the particular species or individual bird, as these organisms often require special media and incubation parameters. DNA probe testing for *Salmonella* spp. is available at some laboratories. An antibiotic sensitivity tailored to drugs used in pet avian species also can be requested if other birds on the premises are at risk.

Mycobacterial culture is required for accurate speciation of acid-fast organisms, and special media and handling are necessary. Once *Mycobacterium* isolates are grown on solid media, some laboratories are capable of speciating the organisms by the use of DNA probes and may offer antibiotic sensitivity testing for mycobacterial isolates.

Fungal culture may be requested in cases of suspected mycoses and is often required for accurate identification of the species involved. Antifungal sensitivity testing is available at specialized mycology laboratories.

A pool of parenchymal tissues (liver, spleen, air sac, lung, kidney, brain and bursa if present) and a separate pool of intestinal contents should be refrigerated or frozen for possible virus isolation or DNA probe testing. A combination swab from heart blood and the cut surfaces of liver, spleen, lung, kidney and bursa can be submitted for DNA probe testing for viruses such as psittacine circovirus and avian polyomavirus. Fluorescent antibody techniques on frozen sections of tissue may be available for certain viruses.

Polymerase chain reaction (PCR) tests are available for the detection of certain viruses, such as West Nile virus, on fresh or frozen tissues. It is important to contact the individual laboratory so the most appropriate tissues are submitted, as this can depend on the particular virus or antigen and upon the particular laboratory's technique.

PARASITOLOGY

Direct wet mounts of intestinal contents and crop or oral cavity scrapings prepared and examined at the time of necropsy are invaluable in the diagnosis of trichomoniasis in pigeons, raptors and budgerigars; cochlosomiasis in finches and canaries; and giardiasis in cockatiels and other psittacine and passerine species. Many of these organisms dry up easily, so examination should be performed promptly. Examination of these wet mounts under dark field or phase contrast, if available, may make the detection of flagellates and motile bacteria easier. Inoculating Diamond's media and submitting the media for incubation can attempt culture of some trichomonad parasites.

Microscopy of whole parasites such as nematodes, cestodes, flukes and acanthocephalans may provide morphology that can point to the classification of the parasites, plus characteristic ova may be visible within the helminths. An acid-fast or auramine stain can be performed on smears for the detection of *Cryptosporidium* oocysts, which can be found in the intestine, conjunctiva, nasal cavity or bursa.

A stained smear of the heart blood or lung impression can be examined for microfilaria and hematozoa such as *Plasmodium*, *Hemoproteus* and *Leucocytozoon*. Wright's-stained impression smears of lung, spleen and liver are especially important in canaries and finches for the diagnosis of the monocytic form of atoxoplasmosis, as these organisms may not be visible histologically.

Rarely, flagellates can be demonstrated in impression smears from the lung, trachea, sinus and conjunctiva, which are not readily visible histologically. Other protozoal parasites such as *Sarcocystis*, *Toxoplasma* and *Leucocytozoon* can be found in impression smears of organs.

HISTOPATHOLOGY

Select a group of formalin-fixed tissues with lesions or a group of tissues that commonly contain histologic lesions that could lead to diagnosis and submit them for histopathology. This commonly includes tissues such as liver, spleen, air sac, kidney, lung, trachea, heart, bursa, brain, duodenum/pancreas and proventriculus/ventriculus. Save the remaining formalin-fixed tissues in case the diagnosis is not made with the first set of tissues.

This second set of tissues may include spinal cord, bone marrow, nasal cavity, skin and feathers, bone and joint, middle and inner ear, eyes, tongue, skeletal muscle, thyroid, parathyroid, adrenal, esophagus, crop, jejunum,

ileum, colon, ceca, gall bladder, ovary, oviduct, testes, thymus, nerve (ischiatic, brachial plexus) and beak.

The veterinary pathologist may recommend special diagnostics such as stains for acid-fast organisms, fungi, bacteria, iron or copper, depending on what is seen on the routine hematoxylin- and eosin-stained sections. In special situations, tissues may be embedded in plastic so that electron microscopy can be performed. Direct electromicroscopy also can be performed on intestinal contents or tissue homogenates. In situ DNA hybridization techniques on paraffin-embedded tissues are available for certain viruses such as Pacheco's herpesvirus, adenovirus, avian polyomavirus, psittacine circovirus and paramyxovirus.

Immunohistochemical stains can detect certain antigens from bacteria, fungi, viruses and parasites in paraffin-embedded tissues, and these techniques also can be utilized to detect some cell markers in the diagnosis of tumors. Gene sequencing of certain microorganisms (*Clostridium perfringens*, for example) in formalin-fixed, paraffin-embedded tissues is available at some diagnostic laboratories.

TOXICOLOGY

Toxicologic testing requires some idea of what toxin is being considered. This information often comes from the history and histopathologic findings. Contacting the toxicology laboratory is essential for submission of the most appropriate tissues and amounts.

The most common toxins tested for are heavy metals such as lead and zinc. Usually liver and kidney are required for this analysis, although zinc also accumulates in the pancreas preferentially. Heavy metals also can be detected in foreign bodies, water and feed. Copper accumulation in the livers of swans can be demonstrated qualitatively with special histologic stains for copper, but quantitative levels require toxicologic analysis.

Iron storage disease is most commonly seen in toucans, toucanettes, mynahs and birds of paradise; the condition is rare in psittacines, although there is emerging evidence that lories and lorikeets may be prone to iron accumulation. The special histologic stain, Perl's Prussian blue, can provide qualitative information about the amount of iron in the liver, but, again, quantitative levels are detected by toxicologic analysis.

Poisonous plants can be found in the digestive tract and submitted to a botanist or university botany department for identification. The plants or wood can be frozen until submission to prevent the breakdown of toxic principles. Ingestion of fertilized plants can result in nitrate toxicity, and samples of these plants can be analyzed for the amount of nitrates present.

Polytetrafluoroethylene (PTFE or non-stick coatings) and other toxic inhalation products are rarely detectable in tissues, and the diagnosis is usually made by a history of exposure, the presence of pulmonary edema and hemorrhage, and the exclusion of other causes of death. There is a wide variety of items commonly found in the household that can give off PTFE fumes, including non-stick cookware and appliances such as self-cleaning ovens and electric grills.

Birds also can be sensitive to other inhalants such as carbon dioxide, carbon monoxide and fumes from glues, resins, plastics and paints. Mycotoxins may be implicated in the case of multiple birds suffering liver damage. Aflatoxins can be detected in foodstuffs, but usually by the time chronic liver damage is evident, the offending foodstuff is often no longer available. In the case of acute toxicosis, samples of the feed should be frozen along with liver and kidney, pending further investigation. Contact the toxicology laboratory for specimen requirements and costs.

CHAPTER
27

Update on *Chlamydophila psittaci*

A Short Comment

THOMAS N. TULLY, JR, DVM, MS, Dipl ABVP-Avian, Dipl ECAMS

Fig 27.1 | A bird infected with *C. psittaci* organism may not have clinical signs. This condition can occur in small birds such as this blue budgerigar.

Fig 27.2 | This lutino cockatiel has the upper respiratory signs associated with avian chlamydiosis. The bird tested positive for the disease. Additional therapies may be needed to treat secondary infections that arise as a result of the primary disease.

Chlamydophila psittaci is a zoonotic intracellular bacterial organism that causes the diseases psittacosis in humans and avian chlamydiosis in avian species. Clinical signs often associated with a psittacosis (human) infection include generalized "flu-like" symptoms to more severe pneumonia and complicating health issues. Avian chlamydiosis will present as non-specific clinical signs in a companion bird patient (Fig 27.1). These non-specific signs can include ocular, nasal or conjunctival irritation and discharge, anorexia, depression, dehydration, bright green urates and diarrhea (Fig27.2). Many avian species have been diagnosed with *C. psittaci*, but it is the companion bird species where this disease is the greatest public health concern.

It is often difficult to confirm a diagnosis of avian chlamydiosis because of the intracellular life cycle of the organism, prophylactic treatment of patients with appropriate antibiotics but using inappropriate doses and treatment periods, and the periodic shedding of elementary bodies (the infectious form of the disease). The difficulty to confirm avian chlamydiosis cases encourages the veterinarian to use multiple testing methodologies. Testing methods that can be used either individually or preferably in combination include pathology, antibody testing (direct complement fixation and elementary-body agglutination) and antigen testing (enzyme-linked immunosorbent assay, immunofluorescent antibody tests, polymerase chain reaction amplification technology on choanal/cloacal swabs and blood). Prior to sample submission, the diagnostic laboratory should be contacted for recommendations on proper sample collection, labeling, packaging

and shipment of the sample material. A breakdown in sample handling or shipment delay can adversely affect the reliability of *C. psittaci* test results.

There are several treatment options available to treat suspected or confirmed avian chlamydiosis cases. The treatment options are based on doxycycline as the drug of choice and being administered in the most appropriate way for the patient(s) to receive a therapeutically effective dose for the duration of the 45-day treatment regimen. There have been recent advances in using doxycycline hyclate powder from opened capsules as a seed coating for budgerigars or mixing the powder in water for larger birds.[1] Oral doxycycline (monohydrate or calcium) or intramuscular injections of specific formulations[a] can be effective treating the individual bird or group of birds that will tolerate the stress of capture and drug administration on a regular basis.

Chlamydophila psittaci is a public health concern and can be a deadly, expensive disease within an aviary or to the individual companion bird. Although difficult to diagnose, avian chlamydiosis can be diagnosed and treated if there is an understanding of the available tests, and the proper tests are used to confirm the presence of the disease. Early communication to a client about the ability of this organism to resist improper treatment and the consequences of discontinuing treatment will often lead to owner compliance with antibiotic administration. In hopes of protecting birds and bird owners in the future, research is currently being conducted to improve *C. psittaci* diagnostic testing and to develop a vaccine to protect birds from infection if exposed to the infectious elementary bodies.

See Chapter 1, Clinical Practice for location the compendium of psittacosis control from the CDC. See Chapter 21 Preventative Medicine and Screening for a review of diagnosis and preventive measures.

Product Mentioned in the Text

a. Vibrovenos formulation, Pfizer Laboratories, London

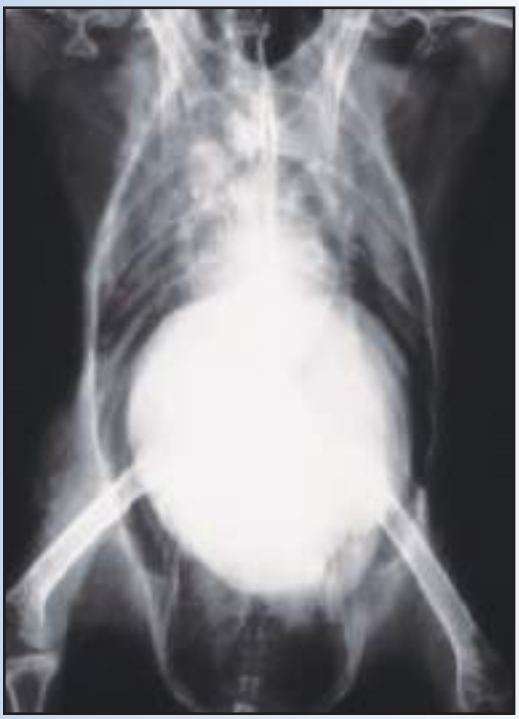
References

1. Smith KA, Eidson M, Johnston WB, et al. Compendium of measures to control *Chlamydophila psittaci* (formerly *Chlamydia psittaci*) infection among humans (psittacosis) and pet birds, 2002. Compend Contin Educ Pract Vet 24:328-335, 374-378, 2002.
2. TullyTN: Update on *Chlamydophila psittaci*, Seminars in Avian and Exotic Pet Medicine, vol 10, No 1 (January) 2001, pp 20-24.

CHAPTER
28

Implications of Mycobacteria in Clinical Disorders

CHRISTAL G. POLLOCK, DVM, Dipl ABVP-Avian



Avian mycobacteriosis is generally a disease of captive populations. In the past, most cases were believed to have been caused by *Mycobacterium avium* and *M. intracellulare*. More recently, however, the atypical organism, *M. genavense*, has emerged as a significant cause of disease. Although the incidence of disease is relatively rare, the potential for avian mycobacteriosis to spread to humans makes this subject pertinent for avian veterinarians. In this chapter, aspects of avian mycobacteriosis, including clinical presentation, therapeutic options, zoonotic potential and diagnostic tests with promising new molecular techniques such as deoxyribonucleic acid (DNA) probes, will be covered.

Mycobacterium spp.

Mycobacterium avium and *M. intracellulare* are frequently grouped together as the *Mycobacterium avium intracellulare* (MAI) complex or *Mycobacterium avium* complex (MAC). Twenty-eight seeknown serotypes of MAI exist.[56] The more pathogenic serotypes 1, 2 and 3 belong to the *M. avium* group.[20,54] Serotypes 4 to 28 make up the *M. intracellulare* group, which is considered relatively avirulent.[31,56,77] Historically, these two species were considered the cause of avian tuberculosis, and they still play an important role in avian mycobacteriosis today.

The atypical organism, *Mycobacterium genavense*, is an important cause of mycobacteriosis, especially in companion birds.[38,40-42] In a recent survey of necropsied pet birds in Europe, *M. genavense* was the predominant mycobacterial species isolated.[49] *Mycobacterium genavense* is a fastidious organism only recently identified. It is most closely related to *M. simiae* and was first isolated from immunosuppressed human patients.[10,19,84] Addi-

tional atypical mycobacteria isolated from companion birds in rare instances include *M. fortuitum*, *M. gordonae* and *M. nonchromogenicum*.[38,41,70,79]

There also are rare reports of disease caused by *Mycobacterium tuberculosis* or *M. bovis*.[80] All avian species studied have been relatively resistant to *M. bovis*.[13] *Mycobacterium tuberculosis* has been reported only in companion parrot species.[29,36,77,80] There are no case reports, as yet, of *M. tuberculosis* in passerines or free-ranging psittacines.[13,48] Infection is probably secondary to close contact with infected humans.[31,48] *Mycobacterium tuberculosis* was cultured from a green-winged macaw (*Ara chloroptera*) 3 to 4 years after active tuberculosis was diagnosed in two human occupants of the household.[85]

Pathogenesis of Disease

SOURCE OF INFECTION

Mycobacterium is a ubiquitous environmental saprophyte most commonly found in soil with heavy fecal contamination or other organic debris.[31] High levels of mycobacteria also might be found in surface water or in marshy, shaded areas.[20,77] Although wild birds are a possible source of infection, they are probably not an important source of disease for captive birds.[6,18,43,77] The prevalence of mycobacteriosis is low (usually <1%) in most free-ranging populations.[24]

TRANSMISSION

Avian mycobacteriosis is usually transmitted by the ingestion or inhalation of soil or water contaminated by feces, or, less commonly, by urine.[29,82] Raptors might become infected by ingesting infected prey. A mechanical arthropod vector is a rare mode of transmission. Vertical transmission also is possible, however, avian mycobacteriosis is generally associated with an immediate halt in reproductive activity.[77]

Pathogenesis

The primary site of entry and initial colonization of mycobacteria is the intestine. Subclinical bacteremia quickly follows and the organism spreads to the liver through the portal circulation. The absence of lymph nodes in the bird then allows mycobacteria to spread hematogenously to distant parenchyma such as the spleen, bone marrow, skin and lungs.[29,36,80]

Disturbance of contaminated surface water might lead to the inhalation of aerosolized mycobacteria and direct colonization of the respiratory tract. Focal skin disease probably occurs secondary to the inoculation of *Mycobacterium* spp. into mucosal or dermal lesions, or by the use of contaminated needles.[36,83]

HOST IMMUNE RESPONSE

The presence of humeral antibodies does not appear to protect against the development of avian mycobacteriosis.[20,82] In the mammal, cell-mediated immunity is much more important, and this also may be true in the bird. Studies evaluating growing layer hens inoculated with *Mycobacterium butyricum* found that dietary linoleic acid may boost cell-mediated immunity.[69]

Clinical Disease

INCIDENCE

The distribution of avian mycobacteriosis is worldwide, although most reports of disease are from northern hemisphere's temperate zones. The incidence of avian mycobacteriosis is reportedly uncommon in some countries, such as Japan.[49,68]

SIGNALMENT

Adult birds, 3 to 10 years of age, are most frequently diagnosed with avian mycobacteriosis.[20,51,80] There is probably no gender predilection, although some reports suggest a slightly higher incidence of disease in the female bird.[42,77,80]

Avian mycobacteriosis has been reported in virtually all avian taxonomic orders, however, susceptibility varies (Table 28.1). The greatest incidence of disease has been in captive collections of waterfowl, parrots, songbirds, and ground-dwelling birds such as farmed ratites and small poultry flocks. Reports also are common in the wood pigeon (*Columba palumbus*) and free-ranging waterfowl. Orders Falconiformes and Gruiformes also are considered susceptible.[6,16,20,24,27,41,43,44,49,59,60,77,80]

Species considered relatively resistant to avian mycobacteriosis include rooks (*Corvus frugilegus*), turtle doves (*Streptopelia risoria*), turkey (*Meleagridis* spp.) and guinea fowl (*Numida meleagris*). Although the flamingo formerly was considered fairly resistant to avian mycobacteriosis, an epidemic was reported in lesser flamingos (*Phoeniconaias minor*) in 1993. More than 18,500 birds died over a 3-month period.[45,77]

CLINICAL PICTURE

There are three forms of avian mycobacteriosis, which have been historically described as classical, paratuberculous or diffuse disease. The most common form of disease in the avian patient involves granulomatous lesions

Table 28.1 | Species Highly Susceptible to Avian Mycobacteriosis[1,6,12,16,17,22,24,27,29,39,41,43,44,46,49,59,60,62,75,77,80,86,87,88]

Order	Species
Anseriformes	• White-winged wood duck (*Aix sponsa*) • Sea ducks (*Somateria fischeri*, *Clangula hyemalis*, *Melanitta* spp.)
Columbiformes	• Wood pigeon (*Columba palumbus*)
Falconiformes	
Galliformes	• Partridge (*Alectoris* spp., *Lerwa* sp., *Ammoperdix* spp., *Tetraogallus* spp.) • Pheasants (*Phasianus colchicus*) • Quail (*Coturnix japonica*)
Gruiformes	• Cranes (*Grus* spp., *Balearica* spp., *Anthropoides* spp.) • Rails (*Rallus* spp., *Laterallus jamaicensis*) • Gallinules (*Porphyrula* spp.) • Coots (*Fulica* spp.)
Passeriformes	• Canaries (*Serinus canarius*) • Sparrows (*Passer domesticus*) • Hooded siskin (*Spinus* sp.) • Lady Gouldian (*Chloebia gouldiae*)
Psittaciformes	• Amazon parrots (*Amazona* spp.) *Amazona ochrocephala auropalliata, Amazona farinosa, Amazona aestiva* • Grey-cheeked parakeet (*Brotogeris pyrrhopterus*) • Budgerigar (*Melopsittacus undulatus*) • Horned parakeets (*Eunymphicus cornutus*) • Pionus (*Pionus* sp.) • Ring-necked parakeets (*Psittacula*)

Table 28.2 | Incidence of Granulomatous Lesions with Avian Mycobacteriosis[29]

	Common	Usually Absent
Charadriiformes (gulls, shorebirds)	✓	
Ciconiiformes (bitterns, herons, egrets, ibises)	✓	
Cuculiformes (cuckoos)	✓	
Falconiformes (hawks, eagles, falcons)	✓	
Galliformes (fowl)	✓	
Gruiformes (coots, cranes, rails)	✓	
Piciformes (toucans)	✓	
Strigiformes (owls)	✓	
Anseriformes (waterfowl)		✓
Columbiformes (pigeons, doves)		✓
Coraciiformes (hornbills, kingfishers)		✓
Passeriformes (songbirds)		✓
Psittaciformes (parrots)		✓

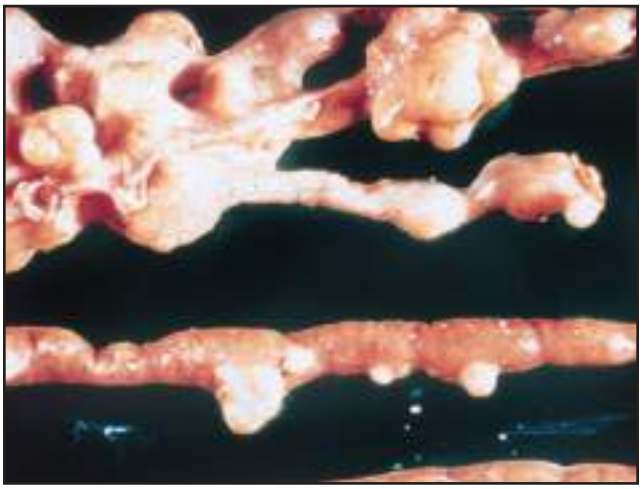


Fig 28.1 | Granulomatous intestinal lesions in a mallard duck (*Anas platyrhynchos*) caused by *Mycobacterium avium intracellulare*.

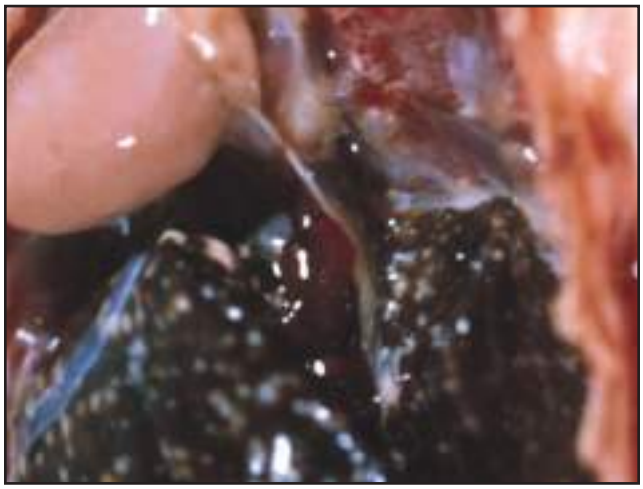


Fig 28.2 | Multifocal granulomatous lesions in the liver of a sandhill crane (*Grus canadensis*) caused by *Mycobacterium* spp.

in the gastrointestinal tract (Fig 28.1) and liver (paratuberculous) (Fig 28.2). Classic avian mycobacteriosis is associated with tubercles in many organs such as the kidney, liver and spleen, while diffuse disease may be associated with diffuse enlargement of affected organs.[24,29,80]

Granulomatous lesions are common in birds of prey but rare in pigeons and doves, waterfowl and most parrots (Table 28.2).[29] Diffuse mycobacteriosis has been most commonly reported in Coraciiformes, such as the kingfisher (*Alcedo* spp.), and Passeriformes.[32,61]

Granulomatous Intestinal Disease

Granulomatous intestinal or paratuberculous disease is associated with muscle wasting and emaciation. Initially the bird will display a good appetite, but anorexia later develops. The presentation of wasting with an increased appetite can lead to the incorrect presumptive diagnosis of proventricular dilatation disease. Additional non-specific signs of illness might include poor feather quality, lethargy, weakness and pallor.[51] Chronic or intermittent diarrhea may also be observed.[12,29,80]

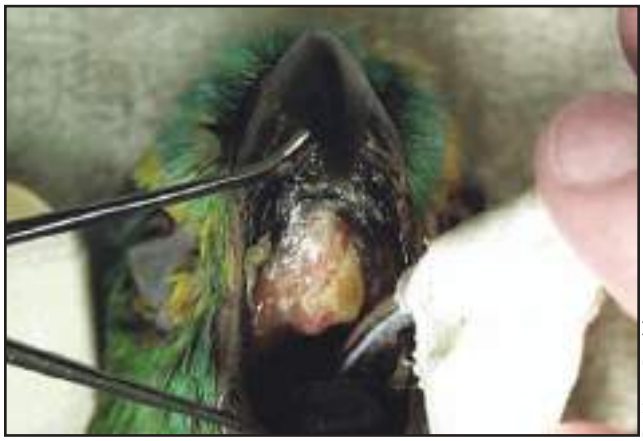
Abdominal distension due to enlargement of the liver or small intestines also might be detected. Ascites is rare, although it has been reported in Canada geese (*Branta canadensis*), mute swans (*Cygnus olor*), tundra swans (*Cygnus columbianus*) and some parrots.[86]

Musculoskeletal Disease

Reports in the literature describe anywhere from 2 to 93% incidence of bony lesions in avian mycobacteriosis.

Dr. Carol Canny

Fig 28.3 | Granulomatous nodules within the skin of a lovebird (*Agapornis roseicollis*) caused by *Mycobacterium* spp.



Dr. Nancy Boedeker

Fig 28.4 | An irregular, firm, fleshy mass in the choana of a blue-fronted Amazon parrot (*Amazona aestiva*) caused by *Mycobacterium* spp.

Acute or chronic lameness, which might be shifting leg in nature, may be observed due to granulomatous lesions in bones or joints. The carpometacarpal and elbow joints are most commonly involved, and skin overlying the affected joints might be thickened and ulcerated.[29,80]

Respiratory Disease

Granulomas within the lungs or compression of air sacs secondary to hepatomegaly can lead to dyspnea or exercise intolerance. Additional signs of respiratory disease are rare with avian mycobacteriosis, although focal nodules have been reported within the infraorbital sinus, nares and syrinx.[14,71,85]

Skin Disease

Tubercle formation within the skin also is rare. Dermatitis, diffuse, non-pruritic thickening associated with xanthomatosis, as well as pale, soft, subcutaneous masses have been reported in association with avian mycobacteriosis (Fig 28.3).[25,29,80]

Ocular Lesions

Granulomatous lesions may be associated with the eyelids, nictitating membranes, retrobulbar tissue and pecten.[1,62,71,77,85] There also is one report of keratitis associated with avian mycobacteriosis.[74]

Miscellaneous Lesions

In rare reports, granulomas have been found within the oropharynx (Fig 28.4), larynx or external auditory canal.[1,29,85] Endocrine abnormalities such as reproductive failure might occur secondary to an infection in the adrenal glands, pancreas or gonads.[80] Avian mycobacteriosis also might be associated with neurologic signs due to involvement of the spinal cord, brain or vertebral column.[52,77,80]

ANTEMORTEM DIAGNOSTICS

Antemortem diagnosis of mycobacteriosis is difficult due to the wide variety of possible clinical signs and physical examination findings. A strong index of suspicion for avian mycobacteriosis can be based on signalment, clinical findings consistent with disease, profound leukocytosis, organomegaly and the cytologic presence of acid-fast bacteria. Serologic techniques such as enzyme-linked immunosorbent assay (ELISA) also may prove helpful in selected cases and are described in the following section on serology. A definitive diagnosis of mycobacteriosis relies on histologic identification or culture of the organism. Molecular techniques, such as DNA probes and nucleic acid amplification, also are promising in the aid of diagnosing avian mycobacteriosis.

Minimum Database

Minimum database results are variable. The complete blood count might reveal a marked heterophilic leukocytosis, monocytosis, thrombocytosis and elevated fibrinogen. Chemistry panel results often are unremarkable, although liver enzymes may be mildly to moderately elevated, and albumin levels may be decreased. Early in the disease process, a polyclonal gammaglobulinopathy also might be detected.[35,77,80]

Imaging

Radiographic findings can include hepatosplenomegaly and dilation of intestinal loops with gas (Fig 28.5).[80] Gastrointestinal contrast radiography may demonstrate a thickened and irregular small intestinal wall. Granulomas may be identified within the bones, lungs or coelom, however, these lesions are more difficult to identify in the bird, since they do not calcify as in mammals.[77]

Radiographic findings involving bone might include lysis and/or sclerosis consistent with osteomyelitis, osteophy-

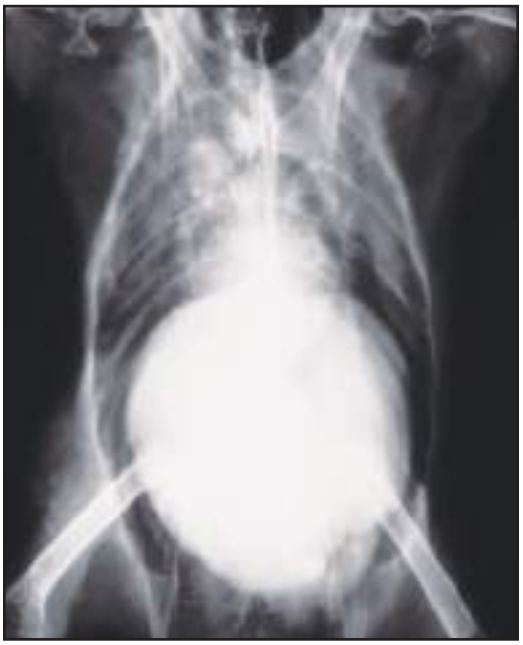


Fig 28.5 | Widening of the cardiohepatic silhouette on a V/D radiograph due to hepatomegaly caused by avian mycobacteriosis in an Amazon parrot (*Amazona* sp.).

tosis around arthritic joints, pathological fractures and endosteal bone densities.[77] Anatomic regions most commonly affected include the midshaft region of long bones, such as the humerus, tibiotarsus and ulna, and the ribs and sternum. There are rare reports of lesions involving the vertebrae and femur.[80] In an unusual case report, chondritis, osteitis and osteomyelitis were described in the nasal bone of a wood duck (*Aix sponsa*) with mycobacteriosis.[27]

Ultrasonography and alternate imaging methods, such as computed tomography, also are potentially useful diagnostic modalities.

Laparoscopy

Laparoscopy is an extremely useful technique for identifying lesions on the serosal surface of the liver, spleen, intestine, lung and air sacs. Granulomas may be visualized as white, yellow or tan round masses, which are soft and easily biopsied.[80]

Cytology

A large number of mycobacteria must be present (5 x 10^4/ml) before the organism can be visualized microscopically; therefore, fecal acid-fast stains serve as a poor screening tool. The Ziehl-Neelsen stain is the standard method for identification of acid-fast organisms. A modified Ziehl-Neelsen stain utilizes peanut oil to reduce damage to the mycobacterial cell wall. Mycobacteria appear pink-red with the Ziehl-Neelsen stain. *Mycobacterium tuberculosis* is rod-shaped, while *M. avium* can appear almost coccoid or as long, beaded rods.[3,77]

A suggestive diagnosis of avian mycobacteriosis may be based on identification of the organism in cytologic samples; however, the acid-fast stain is not a specific test, because non-pathogenic mycobacteria can be transient gastrointestinal inhabitants or environmental contaminants. Therefore, positive acid-fast cytology should be confirmed by culture, histopathology or DNA probe analysis.

Serology

Serologic tests available include hemagglutination (HA), complement fixation (CF) and ELISA. These tests are highly species-specific and they are available for only a limited number of species.

HA is a rapid, easy-to-perform assay run on whole blood or serum. The use of fresh whole blood might produce more sensitive (true positive) results than whole blood in EDTA or serum. Species-specific antigen is required for reliable results. HA has been of some use in waterfowl, domestic fowl, raptors and cranes.[21,35]

ELISA is a sensitive, albeit labor-intensive, test that requires at least 24 hours before results are available. Highly species-specific antigen is required. For instance, a sensitive and specific ELISA exists for mycobacteria in feral barnacle geese (*Branta leucopsis*).[16,19,21,28,77]

CF titers of 1:20 or greater have been used to confirm infection or exposure to *Mycobacterium avium* infection in grey-cheeked parakeets (*Brotogeris pyrrhopterus*).[58]

Culture

Culture has several practical limitations. *Mycobacterium* is only intermittently shed, and careful sample collection is necessary to prevent environmental contamination. Mycobacteria are difficult to culture, and no growth occurs even when proper protocols are followed. If growth does occur, at least 2 to 4 weeks are required for visible colonies to appear and up to 8 weeks of incubation is required. Some strains of *M. avium* require up to 6 months before colonies are identifiable.

Use of a radiometric culture method[a] reduces the length of time needed for culture. This acidic culture medium is especially useful for isolation of the fastidious *Mycobacterium genavense*. In one study, only 3 out of 34 *M. genavense* isolates grew on conventional solid media, while the acidic culture medium supported growth of 23/34 isolates.[41,43]

DNA Probes

The DNA probe assay is a rapid method for identifying various species of *Mycobacterium* grown in culture. Although a very sensitive (95%) and specific (100%) test,

the currently available gene probe[b] is not sensitive enough to directly detect acid-fast bacilli in specimens.[3]

Polymerase Chain Reaction

Molecular techniques may be used to identify organisms grown not only on culture media but also identified within fecal, biopsy or necropsy samples, including formalin-fixed paraffin-embedded sections.[4,34,47] Polymerase chain reaction (PCR) methods[c], based on the amplification and sequencing of the small subunit 16S rRNA gene of mycobacteria, have been used to identify *M. avium*, *M. bovis* and *M. genavense*.[9,47,56,60,76]

One study comparing the sensitivity and specificity of various samples found that fecal specimens had the highest sensitivity by PCR (77.8%). Processing fecal specimens with the zwitterionic detergent, C_{18}-carboxypropylbetaine (CB-18), also might increase the sensitivity of test results.[78]

Intradermal Skin Testing

Intradermal skin testing has been used for decades in poultry in the diagnosis of mycobacteriosis. A small volume (0.1 ml) of avian purified protein derivative tuberculin (PPD) is injected into the wattle or comb and assessed 48 to 72 hours later.[7,55,77]

Unfortunately, intradermal testing has proven unreliable in a number of avian species including pigeons, geese, quail and raptors. In these species, intradermal skin testing is frequently associated with false negatives, especially in the early and late stages of disease. Testing has been attempted on skin over the hock, vent, wing web and eyelid. The skin just behind the ear also has been utilized in ratites.[29,77]

POSTMORTEM DIAGNOSIS

Gross Findings

The gross necropsy findings of avian mycobacteriosis vary widely. Non-specific findings might include muscle wasting, loss of subcutaneous and intracoelomic fat and discolored, poor-quality feathers. There may be no significant gross findings in the diffuse form of mycobacteriosis.[59,80]

Organomegaly

Hepatosplenomegaly is one of the most consistent findings in songbirds and grey-cheeked parakeets (*Brotogeris* spp.) with mycobacteriosis.[11,63] Some birds may also demonstrate an enlarged, pale, firm liver due to amyloid deposition, which may eventually lead to hepatic rupture and hemorrhage.[77] Polycystic livers and cystic granulomas have been reported in waterfowl.[63]

Intestinal Lesions

Distension or thickening of the intestines is an extremely common finding in granulomatous intestinal disease. Intestinal mucosa also may display a "shaggy carpet" appearance caused by prominently thickened or clubbed villi.[77] Proventricular lesions also may be identified.[37]

Granulomas

Tubercles are frequently white, tan or yellow in color, and they might range in size from miliary foci to nodules several centimeters in diameter. Granulomas are most commonly located within the intestinal wall, liver, spleen and bone.[11,24,49,53,77] Granulomas also might be found in subcutaneous tissue as well as a variety of other viscera such as the kidney.[46] Tubercle formation within the skin and lungs is rare.[25,29]

Miscellaneous Lesions

Additional lesions that might be identified with avian mycobacteriosis include the following: proliferative or lytic bony lesions (secondary pathologic fractures are possible),[77] dermatitis or diffuse thickening of the skin associated with xanthomatosis,[80] and pulmonary necrosis or ulceration.[24,53] A single fluid-filled pulmonary cyst has been reported in a cockatoo.[57] An extremely unusual manifestation of mycobacteriosis was reported in fairy bluebirds (*Irena* spp.) with granulomatous cardiopulmonary arteritis.[50]

Histologic Findings

The most common findings reported include granulomatous enteritis, splenitis or hepatitis with variable amounts of acid-fast bacteria. A marked accumulation of macrophages also may be identified within the dermis, mucous membranes and subserosa of the peritoneum and airsacs.[24] Granulomatous intestinal lesions can also be associated with expansion of the intestinal villi by diffuse granulomatous infiltrate and proliferations of epithelial cells within the glands of Lieberkuhn.[29,83]

Large numbers of acid-fast rods are found in lesions caused by *Mycobacterium avium*, while *M. bovis* and *M. tuberculosis* tend to be found in relatively small numbers.[80]

Granulomas generally do not possess a region of central calcification or an extensive necrotic center.[80] The necrotic regions that are identified in avian mycobacterial granulomas are surrounded by epithelioid cells, multinucleated giant cells and lymphocytes.[38,53] Non-caseous granulomas may contain large macrophages with highly vacuolated cytoplasm and numerous acid-fast bacteria.[60]

The diffuse form of avian mycobacteriosis is more difficult to recognize at necropsy, because organomegaly may not be observed. Instead, a diffuse infiltration with

Table 28.3 | Anti-mycobacterial Drugs[5,8,66,81,82]

- Aminoglycosides - Amikacin, Aminosidine, Kanamycin, Streptomycin
- Clofazimine
- Cycloserine
- Dapsone
- Ethambutol
- Ethionamide
- Fluoroquinolones - Ciprofloxacin, Enrofloxacin
- Isoniazid
- Macrolides - Azithromycin, Clarithromycin
- Pyrazinamide
- Rifamycins - Rifabutin, Rifampin
- Tetracyclines - Doxycycline

large, foamy histiocytes may be seen.[23,32,61]

THERAPEUTICS

Humane euthanasia is recommended for birds diagnosed with mycobacteriosis. Birds infected with *Mycobacterium avium* may continuously shed large numbers of organisms into the environment.[77,81] This potential zoonotic risk is especially important in households with immunosuppressed individuals, such as those on chemotherapy, the very young, the elderly and human immunodeficiency virus (HIV)-positive. Any humans in contact with an infected bird should consult a physician for evaluation.

Surgery

Surgical excision may be possible and perhaps even curative for discrete nodules in the skin, subcutaneous tissue or periocular tissue. Medical management is indicated for disseminated avian mycobacteriosis when treatment is deemed appropriate.

Medical Management

There are numerous drugs with anti-mycobacterial activity (Table 28.3). *Mycobacterium avium* isolated from human patients has been reported sensitive to azithromycin, clarithromycin, ciprofloxacin, rifabutin, rifampicin, amikacin, clofazimine and ethambutol. Doxycycline has shown efficacy against atypical mycobacterium like *M. fortuitum*.[81]

Multi-drug therapy should be employed in the treatment of avian mycobacteriosis. Numerous successful combinations have been reported in the literature (Table 28.4).[8,65,66,75,81,82] Due to the intracellular nature of the pathogen, its slow growth, and the bacteriostatic activity of most anti-mycobacterial drugs, an extended course of treatment lasting 4 months or longer is recommended.

Immunotherapy has been a useful adjunct for treatment of human tuberculosis patients.[72,73] Administration of killed *M. vaccae* has some immunomodulatory effects and has been associated with an improvement in survival rates. *Mycobacterium vaccae* was used in a small trial in captive waterfowl; however, results were inconclusive.[11]

Table 28.4 | Multi-Drug Therapy[5,8,66,81,82]

Drug	Therapy
Rifabutin-Ethambutol-Clarithromycin	
Rifabutin	15 mg/kg PO q 24 h
Ethambutol	30 mg/kg PO q 24 h
Clarithromycin	85 mg/kg PO q 24 h (allometrically scaled)
Rifabutin-Ethambutol-Azithromycin*	
Rifabutin	15 mg/kg PO q 24 h
Ethambutol	30 mg/kg PO q 24 h
Azithromycin **or**	43 mg/kg PO q 24 h
Rifabutin **or**	56 mg/kg PO q 24 h
Ethambutol	56-85 mg/kg PO q 24 h
Azithromycin **or** Clarithromycin	43 mg/kg PO q 24 h **or** 85 mg/kg PO q 24 h
Rifampin-Ethambutol-Clofazimine	
Rifampin	45 mg/kg PO q 24 h
Ethambutol	30 mg/kg PO q 24 h
Clofazimine	6 mg/kg PO q 24 h
Rifampin-Ethambutol-Isoniazid**	
Rifampin	45 mg/kg PO q 24 h
Ethambutol	30 mg/kg PO q 24 h
Isoniazid	30 mg/kg PO q 24 h
Rifampin-Ethambutol-Streptomycin	
Rifampin	15 mg/kg PO q 12 h
Ethambutol	10 mg/kg PO q 12 h
Streptomycin	30 mg/kg IM q 12 h
Rifabutin-Ethambutol-Enrofloxacin	
Rifabutin	15 mg/kg PO q 24 h
Ethambutol	30 mg/kg PO q 24 h
Enrofloxacin	30 mg/kg PO q 24 h
Rifabutin-Ethambutol-Ciprofloxacin	
Rifabutin	15 mg/kg PO q 24 h
Ethambutol	30 mg/kg PO q 24 h
Ciprofloxacin	80 mg/kg PO q 24 h
Ethambutol-Cycloserine-Clofazimine-Enrofloxacin (raptors)	
Ethambutol	20 mg/kg PO q 12 h
Cycloserine	5 mg/kg PO q 12 h
Clofazimine	1.5 mg/kg PO q 24 h
Enrofloxacin	10-15 mg/kg PO, IM q 12 h
Lamprene-Seromycin-Myambutol	
Lamprene	1.5 mg/kg PO q 24 h
Seromycin	10 mg/kg PO q 12 h
Myambutol	40 mg/kg PO q 12 h
With advanced cases give	
Ciprofloxacin **or**	20 mg/kg PO q 12 h
Enrofloxacin **or**	15 mg/kg PO q 12 h
Streptomycin	20-40 mg/kg IM q 12 h x 7-10d

*Lower doses have been reported
**Drugs are mixed into a dextrose powder, then mixed into a small amount of food and given PO q 24 h.

CONTROL AND PREVENTION

Mycobacterium is extremely stable in the environment. It is highly resistant to environmental extremes and might

survive for months or years in contaminated soil and surface water or less commonly in feed, feathers or discarded food.[24]

There are no absolute means for control of avian tuberculosis. Quarantine and surveillance programs must strive to identify and eliminate infected animals. Providing complete, balanced nutrition and utilizing good sanitation practices will minimize the impact of disease. Stressors such as overcrowding also must be minimized.[26]

Identification and Elimination of Infected Animals

The poultry industry has relied on the use of intradermal skin testing to identify and then eradicate affected birds. Unfortunately, this screening test has not proven useful in the exotic avian species studied to date.[30,40,51,77] In a zoological or aviary setting, an extended quarantine period of 3 to 6 months should be considered.[40,43] During this time, screening tests should include physical examination, hematology, serum biochemistry, acid-fast fecal smears and serology in those species for which it is available. Laparoscopy, fecal culture and PCR testing also should be considered.

If birds with confirmed mycobacteriosis are not euthanized, they must be kept permanently separated from other birds. Birds that were in contact with mycobacteria-positive individuals also should be quarantined for 1 to 2 years. During this time, periodic retesting every 6 to 12 weeks for mycobacteriosis is recommended.[26,77]

Removal or Prevention of Tuberculosis in the Environment

To reduce the risk of exposure to mycobacteria, carefully consider cage design and sanitation. Prevent contact with wild birds. In aviaries or zoological collections, one should consider solid, non-porous flooring and other easily disinfected surfaces instead of dirt substrate. Footbaths should be utilized to minimize the potential introduction of mycobacteria into the enclosure.[23,43]

Tuberculosis is more resistant to disinfectants than other non-spore-forming bacteria.[77] Compounds with antimycobacterial activity include alcohol, aldehydes, halogens, peroxygens and phenols (Table 28.5).[67] The use of reed biofiltration systems to remove contamination from water also is being investigated.[77]

Table 28.5 | Anti-mycobacterial Compounds[5,8,66,81,82]

- Alcohol
- Aldehydes - Formaldehyde, Glutaraldehyde, Succinaldehyde, Glyoxal
- Halogens - Chlorine-releasing agents, Iodine-releasing agents
- Peroxygens
- Phenolics
- Chemosterilizing gases - Ethylene oxide, Beta-propiolactone

Vaccination

There are only rare reports of vaccination against mycobacteriosis in birds. The bacille Calmette-Guérin (BCG) vaccine, a human product directed against *Mycobacterium tuberculosis*, was tried in poultry but was found to be of little benefit.[33] A vaccine against *Mycobacterium avium* also has been given to poultry and, more recently, captive waterfowl in Britain.[54,64]

ZOONOTIC POTENTIAL OF AVIAN MYCOBACTERIOSIS

Are birds that live in close proximity to people a potential source of tuberculosis? Although the incidence of *M. avium* infection in human acquired immunodeficiency syndrome (AIDS) patients is increasing,[40] these mycobacterial strains are thought to be environmental in origin. Studies using DNA probes have shown that avian strains of *M. avium* rarely infect people.[2] Birds and humans are probably exposed to the same environmental sources of mycobacteria.[31]

CONCLUSION

Avian mycobacteriosis may be caused by MAI or atypical mycobacteria such as *M. genavense*. Birds usually are exposed to mycobacteria through soil or water contaminated by feces. Clinical disease varies with the species and strain of *Mycobacterium* spp., the species of bird affected and the route of transmission. Classically, however, mycobacteriosis is a disease of the gastrointestinal tract and liver in the bird. While identification of disease relies on intradermal skin testing in poultry, this has not proved useful in other avian species. Ancillary testing in nongallinaceous birds should include a complete blood count, imaging, laparoscopy, cytology, serology and PCR testing. A definitive diagnosis is based on culture or histopathology. Euthanasia is recommended for affected birds. Control should focus on identification of affected birds through quarantine and use of appropriate screening tests. Avoiding dirt flooring may reduce exposure to infectious material. Instead, utilize non-porous, easy-to-clean surfaces, appropriate disinfectants and footbaths.

Products Mentioned in the Text

a. BACTEC System, Becton Dickinson, Diagnostic Instrument Systems, Inc, Sparks, MD, USA, www.bd.com/clinical/products/mycob

b. AccuProbe System, GenProbe Inc, San Diego, CA, USA, www.gen-probe.com/prod_serv/mycobac_accuprobe.asp

c. Roche Molecular Systems, Branchburg, NJ, USA, www.roche-diagnostics.com/ba_rmd/products.html

References and Suggested Reading

1. Ackerman LJ, Benbrook SC, Walton BC: *Mycobacterium tuberculosis* infection in a parrot. Am Rev Resp Dis 109(3):388-390, 1974.
2. Angulo FJ, et al: Caring for pets of immunocompromised persons. J Am Vet Med Assoc 205(12): 1711-1718, 1994.
3. Aranaz A, et al: Laboratory diagnosis of avian mycobacteriosis. Sem Avian & Exotic Pet Med 6(1):9-17, 1997.
4. Bascunana CR, Belak K: Detection and identification of mycobacteria in formalin-fixed, paraffin-embedded tissues by nested PCR and restriction enzyme analysis. J Clin Microbiol 34(10):2351-2355, 1996.
5. Bauck L, Hoefer HL: Avian antimicrobial therapy. Sem Avian & Exotic Pet Med 2:17-22, 1993.
6. Behlert O, Gerlach H: Control of avian mycobacterial infection in the pheasantry at Cologne zoo. Berliner und Munchener Tierarztliche Wochenschrift 104(1):12-15, 1991.
7. Bernardelli A, et al: Mycobacterial infections in sea mammals and birds. Revue Scientifique et Technique 9(4):1121-1129, 1990.
8. Beynon PH, Forbes NA, Harcourt-Brown NH (eds): BSAVA Manual of Raptors, Pigeons and Waterfowl. Ames, IA, Iowa State University Press, 1996.
9. Boian M, et al: Identification of *Mycobacterium genavense* in intestinal tissue from a parakeet using two polymerase chain reaction methods: Are pets a reservoir of infection in AIDS patients? AIDS 11(2):255-256, 1997.
10. Böttger EC, et al: Disseminated "*Mycobacterium genavense*" infection in patients with AIDS. Lancet 340(8811):76-80, 1992.
11. Brown MJ, Cromie RL: Weight loss and enteritis. *In* Beynon PH, Forbes NA, Harcourt-Brown NH (eds): BSAVA Manual of Raptors, Pigeons and Waterfowl. Iowa State University Press, Ames, Iowa, 1996, pp 322-329.
12. Butcher GD, et al: Mycobacterium infection in a gray-cheeked parakeet. Avian Dis 34(4):1023-1026, 1990.
13. Butler KL, et al: Experimental inoculation of European starlings and American crows with *Mycobacterium bovis*. Avian Dis 45(3):709-718, 2001.
14. Campbell T: Mycobacterial tracheitis in a double-yellow headed Amazon parrot (*Amazona ochrocephala oratrix*). Exotic Pet Practice 2(1):7, 1997.
15. Clark SL, Collins MT: A single dose oral challenge model of disseminated *Mycobacterium avium* infection. Abstracts General Meeting American Society for Microbiology, 1995, p 139.
16. Clark SL, Collins MT, Price JI: New methods for diagnosis of *Mycobacterium avium* infection in birds. Proc Am Assoc Zoo Vet and Am Assoc Wildl Vet, 1995, pp 151-154.
17. Clippinger TL, Bennet RA, Newell SA: Radiographic diagnosis-Granulomatous pneumonia with intralesional acid-fast bacilli in an Amazon parrot. Vet Radiol Ultrasound 39(3):181-184, 1998.
18. Converse KA, Dein FJ: Tuberculosis in wild birds: Implications for captive birds. Proc Am Assoc Zoo Vet, 1990, p 140.
19. Coyle MB, et al: Laboratory aspects of "*Mycobacterium genavense*," a proposed species isolated from AIDS patients. J Clin Microbiol 30(12):3206-3212, 1992.
20. Cromie RL, et al: Avian immune responses to *Mycobacterium avium*: The wildfowl example. Dev Comp Immunol 24(2-3):169-185, 2000.
21. Cromie RL, et al: A comparison and evaluation of techniques for diagnosis of avian tuberculosis in wildfowl. Avian Pathol 22:617-630, 1993.
22. Cromie RL, et al: Susceptibility of captive wildfowl to avian tuberculosis: The importance of genetic and environmental factors. Tubercle 72(2):105-109, 1991.
23. Derrickson SR, Pickett C: History of avian tuberculosis at the national zoological park: Gaining control through management and husbandry. Proc Am Assoc Zool Parks and Aquariums, 1991, pp 476-484.
24. Dorrestein GM: Bacteriology. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, PA, WB Saunders Co, 1997, pp 275-280.
25. Drew ML, Ramsay E: Dermatitis associated with *Mycobacterium* spp. in a blue-fronted Amazon parrot. Proc Assoc Avian Vet, 1991, pp 252-254.
26. Ensley PK, et al: Avian tuberculosis at the National Zoological Park. Proc Am Assoc Zoo Vet, 1975, pp 203-206.
27. Ferguson SH, et al: *Mycobacterium intracellulare* (*Battey bacillus*) infection in a Florida wood duck (*Aix sponsa*). Am Rev Respir Dis 100:876-879, 1969.
28. Forbes NA, et al: Diagnosis of avian tuberculosis in waterfowl. Proc Assoc Avian Vet, 1993, pp 182-186.
29. Gerlach H: Bacteria. *In* Ritchie B, Harrison G, Harrison L (eds): Avian Medicine: Principles and Applications. Brentwood, TN, HBD Intl, 1999, pp 949-983.
30. Gill IJ, Blandy ML: Control of avian tuberculosis in a commercial poultry flock. Aust Vet J 63(12):422-423, 1986.
31. Grange JM, Yates MD, Broughton E: A review: The avian tubercle bacillus and its relatives. J Appl Bacteriol 68(5):411-431, 1990.
32. Griner LA: Atypical tissue reaction to *M. avium* in Passeriformes and Coraciiformes. *In* Montali R (ed): Mycobacterial Infection of Zoo Animals. Washington DC, Smithsonian Institution Press, 1976, pp 205-207.
33. Guindi SM: Vaccination of fowls against tuberculosis: An experimental study on the immunizing value of BCG vaccine, and a heat-killed tubercle bacilli vaccine (avian type). Proc 1st Annual Veterinary Conference, 1960, pp 93-104.
34. Gyimesi Z, et al: Detection of *Mycobacterium avium* subspecies avium in formalin-fixed, paraffin-embedded tissues of captive exotic birds using polymerase chain reaction. J Zoo Wildl Med 30(3):348-353, 1999.
35. Hawkey C, et al: Hematological changes in domestic fowl (*Gallus gallus*) and cranes (Gruiformes) with *Mycobacterium avium* infection. Avian Pathol 19:223-234, 1990.
36. Hines ME, Kreeger JM, Herron AJ: Mycobacterial infections of animals: Pathology and pathogenesis. Lab Anim Sci 45(4):334-351, 1995.
37. Hoefer HL: Gastric disorders in pet birds. Proc Mid-Atlantic States Assoc Avian Vet, 1997, pp 106-110.
38. Holsboer-Buogo C, et al: Presence of *Mycobacterium genavense* in birds. Schweizer Archiv für Tierheilkunde 139(9):397-402, 1997.
39. Hoop RK: *Mycobacterium tuberculosis* infection in a canary (*Serinus canana* L.) and a blue-fronted Amazon parrot (*Amazona amazona aestiva*). Avian Dis 46(2):502-504, 2002.
40. Hoop RK: Public health implications of exotic pet mycobacteriosis. Sem Avian & Exotic Pet Med 6(1):3-8, 1997.
41. Hoop RK, Böttger EC, Pfyffer GE: Etiologic agents of mycobacteriosis in pet birds between 1986 and 1995. J Clin Microbiol 34(4):991-992, 1996.
42. Hoop RK, et al: Mycobacteriosis due to *Mycobacterium genavense* in six pet birds. J Clin Microbiol 31(4):990-993, 1993.
43. Isaza R: Tuberculosis in all taxa. *In* Fowler ME, Miller RE (eds): Zoo and Wild Animal Medicine, 5th ed. Philadelphia, PA, WB Saunders Co, 2003, pp 89-696.
44. Jahans KL: The isolation of an atypical mycobacterium from three wood pigeons with tuberculosis-like lesions. Res Vet Sci 24(3):378-379, 1978.
45. Kock ND, et al: *Mycobacterium avium*-related epizootic in free-ranging lesser flamingos in Kenya. J Wildl Dis 35(2):297-300, 1999.
46. Marco I, Domingo M, Lavin S: *Mycobacterium* infection in a captive-reared capercaillie. Avian Dis 44(1):227-230, 2000.
47. Miller JM, Jenny AL, Ellingson JL: Polymerase chain reaction identification of *Mycobacterium avium* in formalin-fixed, paraffin-embedded animal tissues. J Vet Diagn Invest 11(5):436-440, 1999.
48. Montali RJ, Mikota SK, Cheng LI: *Mycobacterium tuberculosis* in zoo and wildlife species. Rev Sci Tech Int Epiz 20(1):291-303, 2001.
49. Morita Y, et al: Pathogenicity of *Mycobacterium avium* complex serovar 9 isolated from painted quail. J Vet Med Science 61(12):1309-1312, 1999.
50. Morton LD, et al: Granulomatous aortitis and cardiopulmonary arteritis in fairy-bluebirds (*Irena puella*) with mycobacteriosis. Proc Am Assoc Zoo Vet, 1997, pp 272-273.
51. Mutalib AA, Riddell C: Epizootiology and pathology of avian tuberculosis in chickens in Saskatchewan. Can Vet J 29:840-842, 1988.
52. Odiawo GO, Mukurira JM: Avian cerebral tuberculosis. Vet Rec 122(12):279-280, 1988.
53. Ozcan K, et al: Tuberculosis in geese in Turkey. Avian Dis 45(3):755-759, 2001.
54. Painter KS: Avian tuberculosis caused by *Mycobacterium avium* serotype 3 in captive wildfowl. Vet Rec 140(17):457-458, 1997.
55. Pavlas M, Patlokova V: Occurrence of mycobacteria in sawdust, straw, hay and their epizootiological significance. Acta Veterinaria Brno 54(1):85-90, 1985.
56. Pavlik I, et al: Relationship between IS901 in the *Mycobacterium avium* complex strains isolated from birds, animals, humans, and the environment and virulence for poultry. Clin Diag Lab Immunol 7(2):212-217, 2000.
57. Peavy GM, Silverman S, Howard EB: Pulmonary tuberculosis in a sulfur-crested cockatoo. J Am Vet Med Assoc 169(9):915-919, 1976.
58. Phalen DN, et al: Serologic diagnosis of mycobacterial infections in birds (a preliminary report). Proc Assoc Avian Vet, 1995, pp 67-73.
59. Portaels F, et al: Mycobacteriosis caused by *Mycobacterium genavense* in birds kept in a zoo-11-year survey. J Clin Micro 34(2):319-323, 1996.
60. Ramis A, et al: *Mycobacterium genavense* infection in canaries. Avian Dis 40(1):246-251, 1996.
61. Reed R, et al: Clinical challenge. J Zoo Wildl Med 23(3):383-386, 1992.
62. Rich G: Avian mycobacterial infection. Exotic Pet Prac 5(6):47, 2000.
63. Roffe TJ: Isolation of *Mycobacterium avium* from waterfowl with polycystic livers. Avian Dis 33(1):195-198, 1989.
64. Rossi I: Immunizing potency of inactivated and living *Mycobacterium avium* and *Mycobacterium intracellulare* vaccines against tuberculosis of domestic fowls. Acta Vet 43:133-138, 1974.
65. Rosskopf WJ, Woerpel RW: Avian diagnosis: Laboratory interpretations and case reports, Part 3. Comp Anim Pract 19(1):41-48, 1989.
66. Rupley AE: Respiratory, bacterial, fungal and parasitic diseases. Proc Small Mam and Rept Med Surg, Assoc Avian Vet, 1997, pp 23-44.
67. Russell AD: Activity of biocides against mycobacteria. Soc Appl Bacteriol Symp Ser 25:875-1015, 1996.
68. Sato Y, et al: An occurrence of avian tuberculosis in hooded merganser (*Lophodytes cucullatus*). Avian Dis 40(4):941-944, 1996.
69. Sijben JWC, et al: Dietary linoleic acid divergently affects immune responsiveness of growing layer hens. Poul Sci 79(8):1106-1115, 2000.
70. Silva-Krott I, Brock MK, Junge RE: Determination of the presence of *Mycobacterium avium* on Guam as a precursor to reintroduction of indigenous bird species. Pac Conserv Biol 4:227-231, 1998.
71. Smits B: Non-viral diseases of

psittacines. Surveillance 24(2):17-18, 1997.

72. Stanford JL: Immunotherapy with *Mycobacterium vaccae* in the treatment of mycobacterial disease. J Infect 39(3):179-82, 1999.
73. Stanford JL, Stanford CA: Immunotherapy with *Mycobacterium vaccae* and the treatment of tuberculosis. Soc Appl Bacteriol Symp Ser 25:81S-86S, 1996.
74. Stanz KM, Miller PE, Cooley AJ: Mycobacterial keratitis in a parrot. J Am Vet Med Assoc 206(8):1177-1180, 1995.
75. Stauber EH: Treatment of psittacine pet birds with avian mycobacteriosis-case reports (Behandlung der aviären mykobakteriose bei papageienartigen stubenvögeln -Fall Berichte). VIII. Tagung der Fachgruppe "Geflugelkrankheiten", 1992, pp 231-238.
76. Telenti A, et al: Rapid identification of mycobacteria to the species level by polymerase chain reaction and restriction enzyme analysis. J Clin Microbiol 31(2):175-178, 1993.
77. Tell LA, Woods L, Cromie RL: Mycobacteriosis in birds. Rev Sci Tech Int Epiz 20(1):180-203, 2002.
78. Thornton CG, et al: Processing postmortem specimens with C_{18}-carboxypropylbetaine and analysis by PCR to develop an antemortem test for *Mycobacterium avium* infection in ducks. J Zoo Wildl Med 30(1):11-24, 1999.
79. Tuzova RV: Antigenic activity of bovine and avian type *Mycobacterium* in hens. Trudy-Belorusskii-Nauchno-Issledovatel'skii-Veterinarnyi-Institut 13:30-33, 1975.
80. Van der Heyden N: Clinical manifestations of mycobacteriosis in pet birds. Sem Avian & Exotic Pet Med 6(1):18-24, 1997.
81. Van der Heyden N: New strategies in the treatment of avian mycobacteriosis. Sem Avian & Exotic Pet Med 6(1):25-33, 1997.
82. Van der Heyden N: Update on avian mycobacteriosis, Proc Assoc Avian Vet, 1994, pp 53-61.
83. Van der Schaaf A, Hopmans JLH, Van Beek J: Mycobacterial intestinal disease in wood pigeons (*Columba palumbus*). Tijdschr Diergeneeskd 101(19):1084-1092, 1976.
84. Wald A, et al: Infection with fastidious mycobacterium resembling *Mycobacterium simiae* in seven patients with AIDS. Ann Intern Med 117(7):586-589, 1992.
85. Washko RM, et al: *Mycobacterium tuberculosis* infection in a green-winged macaw (*Ara chloroptera*): report with public health implications. Clin Microbiol 36(4):1101-1102, 1998.
86. Wobeser GA: Other bacteria, mycoplasmas and chlamydiae. *In* Wobeser GA (ed): Diseases of Wild Waterfowl. New York, Plenum Press, 1997, pp 73-74.
87. Woerpel RW, Rosskopf W: Retroorbital *Mycobacterium tuberculosis* infection in a yellow-naped Amazon parrot (*Amazona ochrocephala auropalliata*). Proc Assoc Avian Vet, 1983, pp 71-76.
88. Zorawski C, Skwarek P: Properties of *Mycobacterium avium* and atypical mycobacteria isolated from animals in Poland. Medycyna-Weterynaryjna 32(11): 661-664, 1976.

CHAPTER
29

Implications of Mycoses in Clinical Disorders

ROBERT D. DAHLHAUSEN, DVM, MS

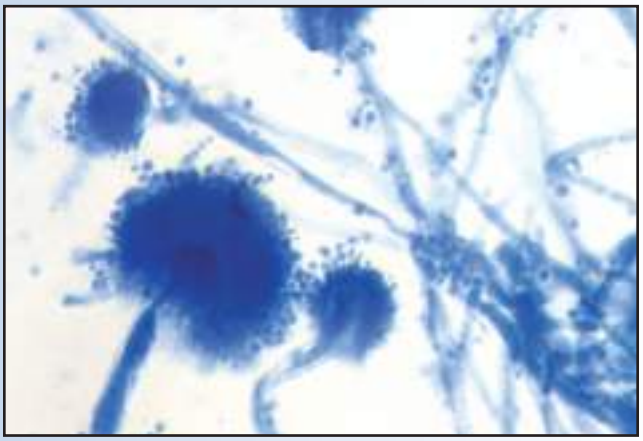


Fig 29.1 | New methylene blue stain of *Aspergillus* conidiophores from an air sac sample obtained at endoscopy.

Mycotic infections are relatively common in avian species. Many fungal agents exist in the environment as soil-borne saprophytes. Most birds are exposed to them in their normal habitat or aviary environment without effect. Nutritional disorders in parrots, stress of captivity in raptors and incubation-related disorders in hatchling galliforms, along with other causes of impaired immune function and environmental factors conducive to fungal proliferation, can cause disease to occur. Respiratory tract aspergillosis and alimentary tract infections due to *Candida* and other yeasts are the most frequent forms of fungal disease observed. The early diagnosis and successful management of systemic fungal disease can present a diagnostic and management challenge to the avian practitioner. Advances in diagnostic methods, improved knowledge of therapeutic agents and better management practices have reduced the morbidity and mortality associated with these agents.

Aspergillosis

Aspergillosis is a non-contagious, opportunistic infection referring to any disease condition caused by members of the fungal genus *Aspergillus*. The organism is an opportunistic, angio-invasive fungus that may act as an allergen, colonizer or invasive pathogen. It can produce both acute and chronic disease varying in spectrum from local involvement to systemic dissemination. It is the most frequent cause of respiratory disease and the most commonly diagnosed fungal disease in pet birds.[70] It also is considered the most common, non-traumatically-induced medical problem in free-ranging birds of prey.[92] The disease is known to occur in a wide variety of captive and free-living birds. Almost all avian species should

be considered as potential hosts susceptible to *Aspergillus* infection.[2]

EPIDEMIOLOGY

Aspergillus spp. are ubiquitous fungi commonly found in nature in the environment, soil and feed grains. They are distributed worldwide and proliferate in environments with high humidity and warm (>25° C) temperatures.[62] Moldy litter, grain and bedding material contaminated with feces are common media for fungal growth. *Aspergillus fumigatus* is the most commonly isolated species from birds with aspergillosis, followed by *A. flavus* and *A. niger*.[22,56] *Aspergillus clavatus*, *A. glaucus*, *A. nidulans*, *A. oryzae*, *A. terreus*, *A. ustus* and *A. versicolor* are among the other species less commonly isolated.

DISEASE PREDISPOSITION

Susceptibility to disease is greatly increased when the immune system is impaired. Immunosuppression is the major factor predisposing birds to the development of opportunistic *Aspergillus* infections. *Aspergillus* spores are widespread in the environment, and many birds may carry them in their lungs and air sacs until immunosuppression or stress triggers clinical disease (Fig 29.1).

Stress alone (a strong immune suppressor) or other factors related to confinement, poor husbandry practice, malnutrition, pre-existing disease and the prolonged use of antibiotics and steroids increase the predilection to disease.[73,95] Overpopulated, poorly ventilated and dusty aviary environments lead to pulmonary and air sac disease. Research has confirmed a causal relationship between high concentrations of *Aspergillus* spp. spores in the environment and aspergillosis. Damp feed or bedding in warm, humid environments and poor ventilation allow for a high concentration of *Aspergillus* spores to develop.[87] Corncob and walnut shell litters can grow *Aspergillus* spp. in aviary environments. Inhalation of large numbers of spores may occur. Birds exposed to the organism in quantities sufficient to establish a primary infection have developed acute disease. There is often a correlation between poor husbandry and a high concentration of spores. Eucalyptus leaves, which have been promoted as a "natural" insect repellent, are often heavily contaminated with this fungus. Acute, severe, untreatable aspergillosis has been associated with their use.[43] Aspergillosis is a common sequela to other respiratory tract disease. A predominantly seed diet, with subsequent malnutrition including vitamin A deficiency, can lead to squamous metaplasia of the oral and respiratory epithelium and the establishment of fungal growth.[73] The incidence of fungal disorders is negligible in an avian practice where the majority of parrots are fed a formulated diet (G.J. Harrison, personal communication, 2003) (see Chapter 4, Nutritional Considerations).

Aspergillosis is the most commonly occurring respiratory disease in captive wild birds.[95] Of the psittacine species, the African grey parrot (*Psittacus erithacus*) and pionus parrots (*Pionus* spp.) are reported to have an increased susceptibility to the development of disease.[9] Localized infection of the nasal passages is observed in Amazon parrots, possibly due to the higher incidence of hypovitaminosis A in this species.[9] Raptorial species at particularly high risk of developing aspergillosis include goshawks (*Accipiter gentilis*), rough-legged hawks (*Buteo lagopus*), immature red-tailed hawks (*Buteo jamaicensis*), golden eagles (*Aquila chrysaetos*) and snowy owls (*Nyctea scandiaca*).[39,92,95] Gyrfalcons (*Falco rusticolus*) are believed to be especially susceptible to *Aspergillus* infection[39,42,92,95]. Among waterfowl, a higher incidence of aspergillosis is seen in swans (*Cygnus* spp.). Captive penguins (*Sphenisciformes*) also are extremely susceptible to developing the disease. In these species, the increased incidence of aspergillosis may reflect environmental and husbandry deficiencies occurring in captivity and/or increased species susceptibility.

ASPERGILLUS PATHOGENESIS

Aspergillus spp. are widespread in nature. Birds are exposed to fungal spores on a regular basis, and many carry them in their lungs and air sacs without ill effect. The nature of disease that occurs is thought to depend on the resistance of the avian host and the number and distribution of fungal spores.[8] Healthy birds exposed to high concentrations of spores can be resistant to infection, whereas immunocompromised patients can become severely affected by minimal exposure.[48,122]

Aspergillosis is usually contracted as the result of a susceptible host inhaling fungal spores. Oral ingestion of spores from moldy feeds may also occur. The fungus is, however, capable of penetrating broken skin and eggshells and can infect developing embryos during the incubation process.

In birds, aspergillosis is predominantly a disease of the lower respiratory tract that usually occurs secondarily to other disease or immunosuppressive processes. Acute and chronic forms of the disease can occur. Infections may be localized or diffuse, depending upon the distribution and growth of fungal organisms. Initial lesions occur mainly in the lungs and air sacs, although the trachea, syrinx and bronchi also may be affected. Infections may spread from the respiratory tract to pneumatized bone or enter the peritoneal cavity by direct extension through the air sac walls. The organism may reach various other tissues by vascular invasion and embolism. The fungus

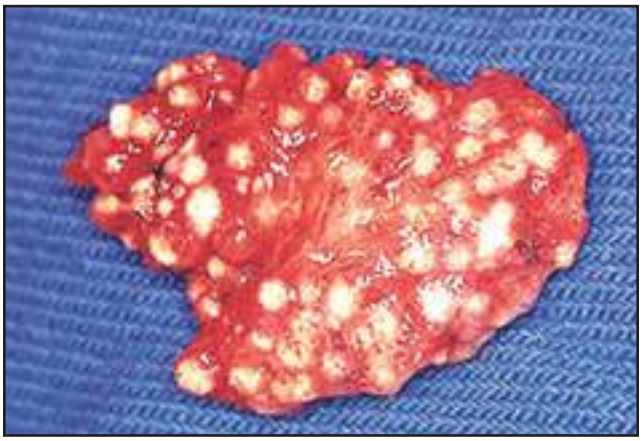


Fig 29.2 | Miliary lung nodules due to inhalation of *Aspergillus* spp. spores.

can infect any organ. Aerogenous and hematogenous dissemination result in diffuse, systemic disease.[49]

In the acute disease, whitish mucoid exudates of fungal growth are present in the respiratory tract. Marked congestion of the lungs and thickening of the air sac membranes occur. Miliary foci of inflammation develop around sites of fungal growth and result in the formation of micronodules (Fig 29.2).[66,120] These predominate in the caudal thoracic and abdominal air sacs and peripheral lung fields. In the chronic form of the disease, multiple nodules may coalesce into plaques and larger granulomatous lesions. Sporulating fungal colonies may develop in the center of these lesions, with extensive adhesions forming between the air sac membranes, lungs and abdominal viscera.

Some *Aspergillus* spp. produce mycotoxins that are immunosuppressive and may be involved in the pathogenesis of the disease. Some mycotoxins also possess carcinogenic activity. Of these, aflatoxin is well known and may induce hepatocellular carcinoma or hepatic fibrosis with subsequent cirrhosis when ingested. Aflatoxins are most commonly associated with *Aspergillus flavus* and may contaminate feeds such as peanuts.[77]

DISEASE

Clinical signs associated with aspergillosis are variable and depend upon such factors as the pathogenesis of initial infection, location of lesions, organ systems involved and host immune defenses. The disease may be either localized or diffuse and often causes a progressive, debilitating illness with high mortality. Aspergillosis predominantly affects the upper and lower respiratory tract, however, any organ system may be involved. Both acute and chronic forms of infection are commonly recognized in birds.

Acute Disease

Acute aspergillosis is a fatal respiratory disease characterized by variable morbidity and high mortality. It is usually seen in young and newly captive birds and occurs when an immunocompromised host inhales an overwhelming number of spores. Onset of clinical disease is rapid and followed by death within several days. Affected birds may show dyspnea, cyanosis, lethargy, anorexia, polyuria, polydipsia and sudden death. A white mucoid exudate and marked congestion of the lungs and air sacs occur. Numerous, diffusely distributed foci of pneumonic nodules also may be variably present.

Acute aspergillosis has been reported to occur in psittacine chicks.[116] Most are septicemic and acutely depressed when illness is observed. In addition to tachypnea, dyspnea and vomiting, abdominal enlargement due to ascites formation frequently develops. The disease is rapidly fatal with few chicks surviving long enough to develop pulmonary granulomas.

Mycotic Tracheitis

Mycotic tracheitis refers to a form of aspergillosis localized in the trachea, syrinx and major bronchi.[59] The pathogenesis is similar to that for acute aspergillosis in an immune-compromised host with exposure to numerous fungal spores. Colonization may occur anywhere along the length of the trachea, but is most often found at the level of the syrinx and tracheal bifurcation (Fig 29.3).[87] Granuloma formation causes a progressive course of life-threatening obstructive airway disease (Fig 29.4). A change in vocalization may be present and is highly suggestive of lesions in the syrinx.[87] Birds also may present with a rapid onset of severe dyspnea, open-mouthed breathing, gurgling respirations or a cough.[45,121] Sudden death also may occur.

Chronic Disease

Chronic aspergillosis is the more commonly observed form of the disease. It generally occurs in older birds that have been in captivity and is the result of long-term malnutrition and stress.[8,59] Previous disease, prolonged antibiotic or corticosteroid therapy and other stress factors are contributory. Some form of immunosuppression is implicated in the chronic disease.[95] This form is often seen in species like the African grey parrot, pionus parrot and Amazon parrot.

Chronic forms of the disease can be quite subtle in onset, and early clinical signs are often non-specific. A change in behavior, reduced level of activity and decreased appetite may be observed. In some instances, exercise intolerance and weight loss, even in light of a good appetite, are the only signs noticed.[73] Birds with

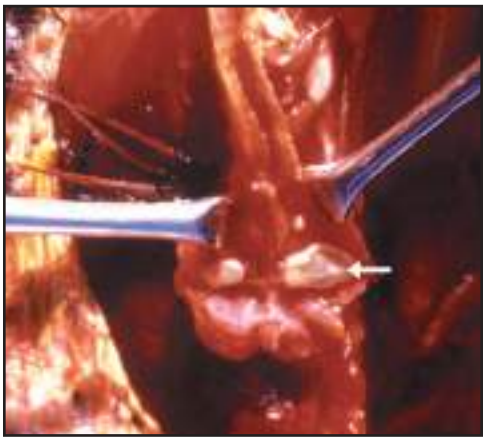


Fig 29.3 | Granuloma (arrow) of the syrinx in a blue-headed pionus parrot (*Pionus menstruus*). The owner noted a characteristic change and weakness in vocalizations several days prior to finding the bird deceased.

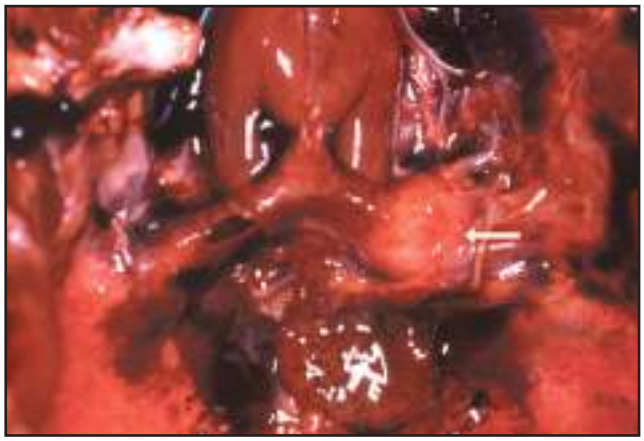


Fig 29.4 | *Aspergillus* granuloma (arrow) occluding the left bronchus in an African grey parrot (*Psittacus erithacus*).

lower respiratory tract involvement may exhibit variable respiratory compromise, depending upon the nature and extent of the lesions present.[53] With the exception of the acute form and mycotic tracheitis, lower respiratory tract aspergillosis is often inapparent. Respiratory signs are usually absent until the disease is extensively developed.[87] Tachypnea and dyspnea are not commonly observed until late in the disease process.[59]

Hepatic signs (biliverdinuria and hepatomegaly) and renal signs (polyuria, polydipsia and renomegaly) may be present. Ascites also may occur. Gastrointestinal involvement (regurgitation, diarrhea and abnormal droppings) is less frequently observed.

Aspergillus air sacculitis is the most frequently encountered form of the disease with extension to the lungs commonly occurring.[87] Aspergillomas may be found throughout the entire respiratory tract but most commonly occur in the posterior thoracic and abdominal air sacs (Figs 29.5, 29.6). In advanced cases, changes occur in the respiratory rate and effort and in vocalization. Dyspnea and tachypnea in an unstressed bird, tail bobbing, open-mouthed breathing and audible respiratory sounds are indications of advanced lower respiratory tract disease.[24,59,87,112] Although auscultation is less valuable in avian patients than in mammals for evaluation of respiratory tract disease, abnormal crackles or clicking noises can indicate air sac involvement.[24]

The time from onset of clinical signs to death ranges from less than 1 week to over 6 weeks.[73] *Aspergillus* hyphae are tissue- and angio-invasive and can cause respiratory hemorrhage and acute death in affected birds at any stage of the disease process (Fig 29.7).

Localized aspergillosis involving the upper respiratory tract often presents as a chronic rhinitis and sinusitis in psittacine birds. Distension of the infraorbital sinus and periorbital soft tissue swellings may be present. Birds may exhibit a unilateral or bilateral nasal discharge that can be serous to purulent in character.[87] Formation of rhinoliths and oronasal granulomas may occur and often obstruct the upper airways, causing wheezing respiratory sounds. Secondary bacterial sinus infections are common. In psittacines, obstruction of the connection between the right and left nares may occur with *Aspergillus* rhinitis. This obstruction may be due to the presence of caseated debris or bony deformation from chronic infection.

Central Nervous System

Encephalitic and meningoencephalitic lesions may occur with disseminated aspergillosis. Necrotizing aspergillosis with dissemination of thrombi to the brain and spinal cord is often associated with ataxia or paralysis, incoordination, tremors and torticollis.[73,95] A trumpeter swan (*Cygnus buccinator*) with ataxia, incoordination and heart murmur had cerebral aspergillomas and *Aspergillus* granulomas in both ventricles of the heart.[5]

Ocular Mycoses

Fungal infections of the eye are rare in birds but have been reported in numerous species.[22,56] *Aspergillus fumigatus* was isolated on conjunctival culture from a blue-fronted Amazon parrot (*Amazona aestiva*) with mycotic keratitis.[56] It was implicated as a cause of severe blepharitis and dermatitis of the eyelids in a falcon/gyrfalcon hybrid (*Falco peregrinus* X *Falco rusticolus*) and keratitis in chickens.[1,10] Most reported occurrences result from the extension of preexisting upper respiratory infections, although ocular trauma and corticosteroid therapy

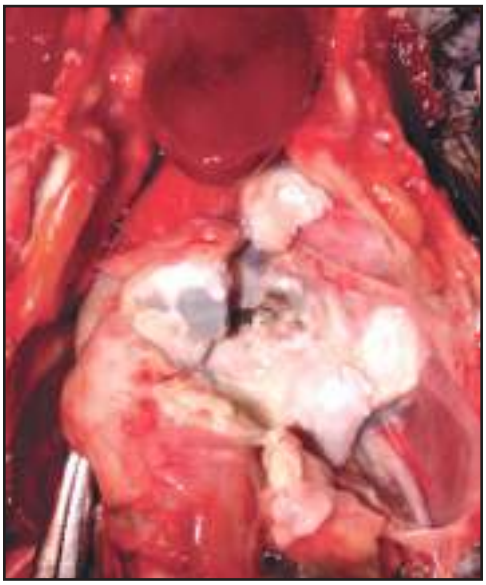


Fig 29.5 | *Aspergillus fumigatus* granuloma exhibiting conidiophore growth in the lung and air sacs of an African grey parrot (*Psittacus erithacus*).

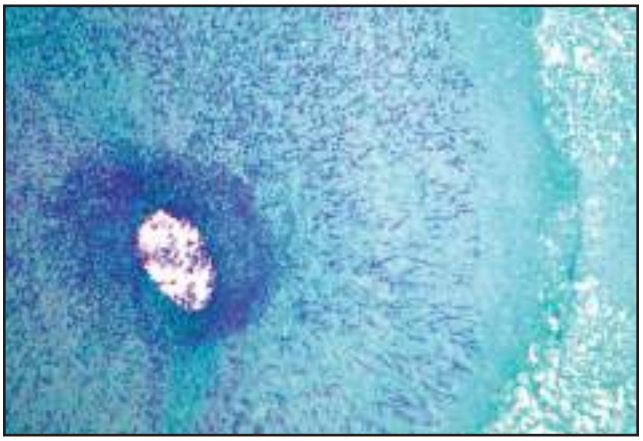


Fig 29.6 | Fungal granuloma in avian lung tissue, central area of necrosis with fungal hyphae. PAS fungal stain.

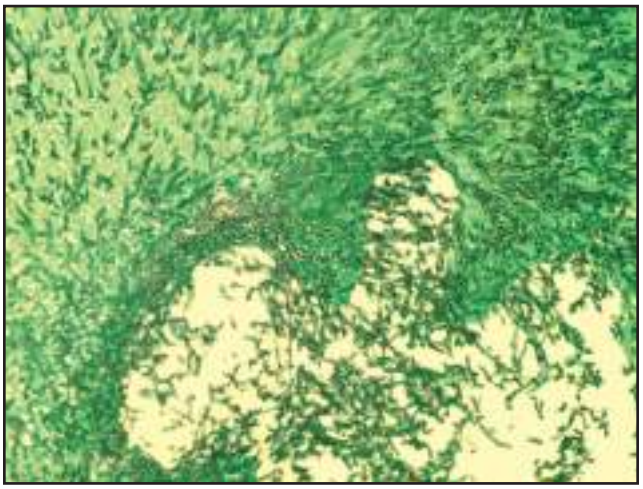


Fig 29.7 | Mycelial growth of *Aspergillus fumigatus* throughout an avian pulmonary granuloma. PAS fungal stain.

are other predisposing factors.[10,56,64] Affected birds may show blepharospasm, photophobia, severe periorbital swelling and conjunctival hyperemia.[22,56] Corneal epithelial erosions and stromal necrosis may result in perforation of the cornea or panophthalmitis, resulting in functional loss of the eye.[56] Characteristic signs with recent stress and/or poor nutrition should make aspergillosis a strong presumptive diagnosis.

ASPERGILLUS DIAGNOSIS

Antemortem diagnosis of aspergillosis is challenging and often requires several diagnostic methods.[15,79] A presumptive diagnosis is often based upon the clinical history, physical exam, clinical impression, complete blood count, radiography and endoscopy. History of a previous stressful event, an immunosuppressive disease or treatment and/or exposure to spore-laden environments is supportive. Clinical signs, especially dyspnea, weight loss, exercise intolerance and chronic debilitation, also suggest aspergillosis; however, a definitive diagnosis requires confirmation of the organism by cytology, culture, histopathology or DNA testing.[8,15,59] Demonstration of the causal agent consistent with the clinical disease and observed lesion(s) is the diagnostic goal.

Hematology and Serum Chemistry

Aspergillosis generally causes a significant heterophilic leukocytosis (25,000 cells/μl to 100,000 cells/μl), which may be associated with both acute and chronic disease.[15,18] White blood cells are often very reactive and a left shift may be present. Heterophils often exhibit degranulation and other toxic signs. Reactive lymphocyte changes also occur and may result in a marked lymphopenia, especially in Amazon parrot species.[44] A monocytosis is often observed in chronic forms of the disease.[19]

A non-regenerative anemia due to the chronic inflammation of the disease is often present. Lack of an appropriate bone marrow response may be evidenced by the absence of polychromasia, reticulocytosis, macrocytosis and/or anisocytosis.[60] Psittacosis and mycobacteriosis can induce similar hemograms.

Serum biochemistry analysis may show an increase in aspartate aminotransferase (AST), lactate dehydrogenase (LDH) and serum bile acids if liver involvement is present. Hypoalbuminemia and hypergammaglobulinemia also are characteristic of the disease.[87]

Plasma protein electrophoresis may help support the diagnosis of aspergillosis in psittacine birds but is not specific for this disease. Observed changes in the plasma protein electrophoretograms are indicative only of a non-specific inflammatory response.

In one study, most (6/7) birds exhibited moderate to marked decrease in the plasma albumin to globulin ratio.[58] Some (3/7) birds showed a moderate to marked increase in β-globulin concentrations. Affected birds may, however, demonstrate normal values for these indices.

Serology

Aspergillus antibody titers have only a moderate predictive value in disease diagnosis. The indirect ELISA assay (The Raptor Center, University of Minnesota) measures *Aspergillus* antibody levels and has shown useful correlation with clinical infections in raptor species, although false-negative results can occur.[95] The test requires species-specific antibody conjugates and is most useful in these species. In a report of falconiform birds with confirmed aspergillosis, 43% (10/23) of the birds had moderate to marked antibody titers, whereas 22% (5/23) had negative titers.[93] In contrast, all 112 owls described in the same report had negative antibody titers despite confirmed aspergillosis in some of these birds. In a study of captive penguins (*Spheniscus humboldti, Spheniscus magellanicus, Pygoscelis adeliae*) with confirmed aspergillosis, many birds had markedly increased titers and only 20% had negative antibody titers.[96]

A negative antibody titer in an infected bird may be explained either by a lack of reactivity between the test conjugate and patient immunoglobulins or by a lack of patient humoral response (immunosuppression). Lack of humoral response also has been attributed to sequestration of the infection site.[26] In raptors, indirect ELISA values in the mid to high range help confirm the diagnosis when aspergillosis diagnosis correlates with the clinical signs shown by the patient.[93] A positive result means active infection, long-term exposure or previous infection antibody.[95] The test should be utilized with hematology, endoscopy, radiology, and tracheal or air sac culture in potential aspergillosis cases. With treatment, the antibody titer generally rises, and with successful treatment then falls to undetectable levels. Failure of the titer to rise or subsequently drop with treatment is a poor prognostic sign.[15]

Aspergillus antibody testing is probably less useful in psittacine species. In a report documenting the sensitivity of serologic testing in detecting aspergillosis in psittacine birds, *Aspergillus* antibody and antigen ELISA titers in infected birds were weakly positive.[58] The incidence of weakly positive antigen titers in clinically normal birds is reported to be high, making these results difficult to interpret clinically.[26,58] Currently, there is no hematologic test that can reliably detect aspergillosis in psittacine birds.[58]

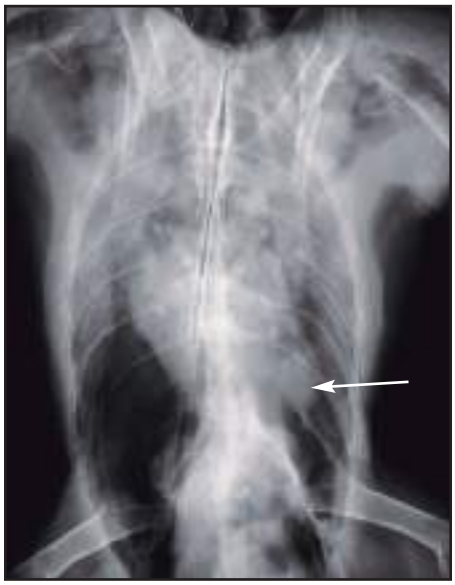


Fig 29.8 | Radiograph of an *Aspergillus* granuloma (arrow) in the air sac of a blue and gold macaw (*Ara ararauna*).

Radiology

Radiographic evaluation can demonstrate the distribution and severity of mycotic lesions in the lungs and air sacs but is usually of limited diagnostic value until late in the disease process. Although mycotic air sacculitis with granuloma formation is well documented in a variety of avian species, lesions are usually advanced before becoming radiographically apparent.[24,87,112] A bronchopneumonia with marked parabronchial patterns is one of the more common radiologic findings.[73] The fibrinous air sacculitis associated with aspergillosis allows for radiographic visualization of the air sac walls.[87] Asymmetry, hyperinflation or consolidation of the air sacs may be evident. A nodular air sacculitis with focal air sac densities is often seen and occurs primarily in the abdominal and, less often, thoracic air sacs. Single or multiple soft tissue densities in the air sacs or lungs are most often granulomas but should be considered non-specific (Fig 29.8).[73] Although intraluminal granulomas of the syrinx, trachea and main stem bronchi are fairly common, they are seldom visualized radiographically. The syrinx is often obscured by soft tissue and bone.[87] Fungal air sacculitis usually causes plaque-like and nodular granulomatous lesions in the air sacs. Even after the successful resolution of clinical *Aspergillus* infection, lungs and air sacs may remain thickened and irregular and appear abnormal both radiographically and via endoscopic examination.

Computed tomography (CT) scans provide a detailed image of all parts of the respiratory tract and are useful for demonstrating small lesions that are not visible on radiographs and for visualizing lesions in the infraorbital sinus, retrobulbar area, trachea and main stem bronchi.[87]

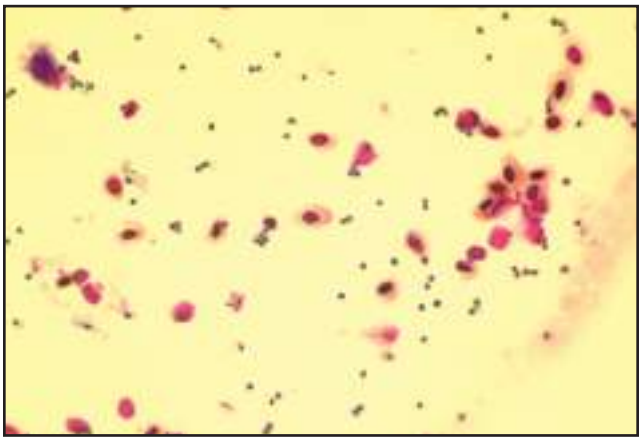


Fig 29.9 | *Aspergillus* spores and avian red blood cells from a clinical sample obtained during diagnostic endoscopy.

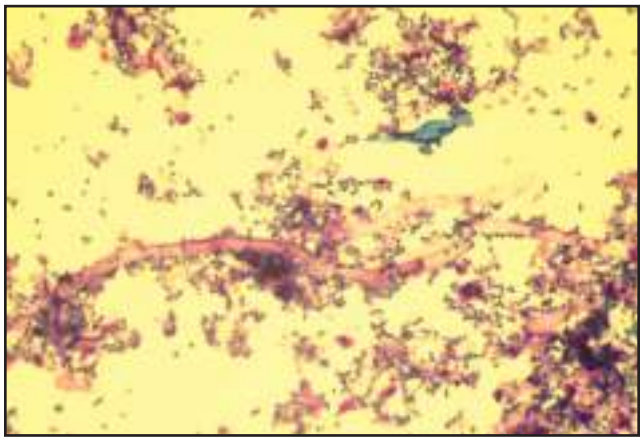


Fig 29.10 | Hyphal elements and spores of *Aspergillus fumigatus* in Gram's-stained clinical specimen collected at endoscopy.

A concurrent hepatomegaly and renomegaly may be visualized on radiography or detected by abdominal palpation.[59]

Endoscopy

Air sac consolidation and granulomas can obscure radiographic detail in the coelomic cavity. Lesions are often best observed by endoscopy, which is an efficient way to detect and sample fungal plaques in the trachea, syrinx or lower respiratory tract.[94,109]

The trachea (depending on species and the size of scope being used) can be visualized down to the level of the syrinx with both rigid and flexible endoscopes. Depending on the extent of the disease and chronicity of the condition, air sacs that are normally thin and transparent may be thickened, cloudy or covered with exudate. Early in the disease process, prominent vascularity of the air sacs may be the only observable abnormality.[95] Fungal air sacculitis usually causes plaque-like, coalescing and nodular granulomatous lesions in the air sacs. Plaques vary from white to yellow in color to green to black on the surface, owing to the development of conidia and fungal spores.[97] The observation of typical lesions by endoscopy with biopsy sample collection for cytology, histopathology or fungal culture can provide a confirmed diagnosis of aspergillosis. Endoscopy is useful in evaluating the extent of infection and monitoring progress during treatment[94] (see Chapter 1, Clinical Practice and Chapter 24, Diagnostic Value of Endoscopy and Biopsy).

Cytological Evaluation

Cytologic evaluation of clinical samples can aid the diagnosis of aspergillosis. Choanal swabs, infraorbital sinus aspirates, tracheal and air sac swabs and washings may reveal *Aspergillus* hyphae and spores (Fig 29.9).[8] Squash preparations of wet mount clinical specimens are prepared and stained with lactophenol cotton blue or methylene blue stain.[95] Microscopic evaluation often reveals long, dichotomously branched (~45°), septate hyphae, 2.5 to 4.5 μm in diameter. Fruiting bodies with spores also may be identified (Fig 29.10).

Confirming the Diagnosis

Histopathology and fungal culture can be used to demonstrate the organism in clinical samples and are important in providing a confirmed diagnosis of the disease. DNA probe testing of clinical specimens also can validate the diagnosis. Tracheal swabs or air sac swabs and granuloma biopsy specimens obtained at endoscopy can be tested specifically for *A. fumigatus*.[a] Genus-specific probes can be used to test for *Aspergillus* spp. in general. Research on the use of DNA probe testing of whole blood samples suggests that this may be helpful in providing a confirmed diagnosis of the disease.

TREATMENT

Amphotericin B

Amphotericin B[b] is an amphoteric polyene macrolide anti-fungal agent.[72] Its mechanism of action is to bind ergosterol, the principal sterol present in the cell membrane of sensitive fungi.[65] It has a wide spectrum of activity, being fungicidal to both *Aspergillus* spp. and *Candida* spp., and to other fungi including *Blastomyces*, *Coccidioides*, *Histoplasma*, *Sporothrix* and *Mucor* spp.

Amphotericin B[b] has been used to treat both systemic and topical fungal infections in birds.[38] It can be administered intratracheally, intravenously, in sinus flushes and through nebulization. Parenteral use quickly establishes fungicidal concentrations, making it a frequent choice for initial therapy. The drug is eliminated by the kidneys and is used for only short duration due to the risk of induced nephrotoxicity. A dose of 1.5 mg/kg IV is administered q8h for a period of 3 to 5 days and in combination with

itraconazole[c], fluconazole[d] or terbinafine[f] orally.[38] Combined nebulization therapy with clotrimazole[e] or terbinafine also is suggested (see Chapter 9, Therapeutic Agents).

Sinus and tracheal aspergillosis are more difficult to treat. Intratracheal use of amphotericin B[b] at 1 mg/kg q8-12 h has been described as supplemental treatment for these infections. Topically, the drug can be very tissue irritating and must be diluted in water (saline inactivates amphotericin B[b]) to reduce the risk of iatrogenic sinusitis or tracheitis.[59] Nebulization with amphotericin B[b] has been reported to cause severe bronchoconstriction in horses.[13] Amphotericin B[b] has been used in nebulization and sinus flushes for 20 years in avian species with good success and no clinical complications.

Itraconazole

Itraconazole[c] is one of the most widely used antifungal agents in birds and is especially effective in combination with nebulized clotrimazole[e] and/or intravenous amphotericin B[b].[40,81,95] It has greater efficacy than ketoconazole[i] or fluconazole[d] against *Aspergillus* spp. and has less potential toxicity than amphotericin B[b].[47,95,115] It has been used effectively to treat aspergillosis in raptors, psittacine birds and waterfowl, and is a recommended treatment of choice in these species.[38,40,81,95]

Itraconazole[c] is eliminated by hepatic metabolism and is generally well tolerated during long-term use.[83] The effective drug dose and potential for adverse effects is variable among avian species. In most psittacine species, effective serum concentrations are attained when therapy is initiated at 5 to 10 mg/kg PO twice daily for 5 days, then once daily for the duration of treatment.[82,87] Temporary anorexia and lethargy were the only side effects observed in an orange-winged Amazon parrot (*Amazona amazonica*) treated with 5 mg/kg itraconazole[c] for 1 year.[83] African grey parrots are reportedly more sensitive to itraconazole[c] and may exhibit adverse drug effects at normal dosage levels. One report suggests that the drug may have contributed to several deaths in this species.[83] A dose of 2.5 to 5.0 mg/kg PO q24h is recommended for use in African grey parrots.[87]

Itraconazole[c] is safe in raptors when dosed at 10 mg/kg per day PO for 3 to 6 months duration.[61] Hawks treated at this level reached steady state plasma levels within 2 weeks, with effective organ tissue levels adequate to treat most fungal infections.[61] Poor distribution in the respiratory tissues after oral dosing in pigeons has been reported, which might limit applicability in this species.[70] A dose of 6 mg/kg q12h in pigeons achieves effective plasma concentrations; however, a comparatively higher dose of 26 mg/kg PO q12h is needed to attain effective respiratory tissue levels.[70]

Itraconazole[c] is marketed as capsules containing coated lactose granules, which are soluble only at low pH. Dissolution of these granules in acid solutions results in improved bioavailability and higher blood itraconazole[c] levels following oral administration in avian patients.[61,83] Absorption also is enhanced when the drug is administered with a fatty meal.[82] It is recommended that itraconazole[c] beads from a 100-mg capsule be divided into five equal parts of 20 mg each. Each 20-mg aliquot is dissolved in 0.4 ml of 0.1 N HCl. The resulting solution is diluted to a final concentration of 5 mg/ml by the addition of orange juice.[61] For avian dosing, the drug is administered by gavage with food. Itraconazole[c] also is available in a 10 mg/ml liquid[c] for oral administration that avoids any concern with sufficient dissolution or suspension.

Other Agents

Fluconazole[d] is effective against aspergillosis although generally less so than itraconazole[c]. It is rapidly absorbed with high bioavailability after oral administration. The drug is widely distributed to the extracellular space, making it useful for treating mycoses of the eye and central nervous system.[71] *Aspergillus* keratomycosis in a blue-fronted Amazon parrot was treated with oral and topical fluconazole[d].[56] A dose of 5 to 15 mg/kg PO q12h is recommended in most psittacine species.[21]

Clotrimazole[e] and enilconazole[k] are relatively insoluble and useful against aspergillosis only at sites that can be treated topically with the drug or reached by inhalation or nebulization. Miconazole[l] is effective for treating fungal keratoconjunctivitis when used in an ophthalmic form with frequent medication intervals (q3-4h).[4,14,41,56]

Terbinafine hydrochloride[f] is a synthetic allylamine antifungal agent that is fungicidal against a wide variety of dermatophytes, molds and fungi. The drug's efficacy is similar to or more effective than amphotericin B[b] and itraconazole[c] against *Aspergillus* spp., and has been used to treat aspergillosis effectively in psittacine species.[28] In addition to its fungicidal property, terbinafine[f] has good ability to penetrate mycotic granulomas. It is readily absorbed after oral administration and has a low potential for adverse side effects. No signs of toxicosis were observed during prolonged drug administration in psittacine birds.[28] The drug has proven very useful as an alternative treatment for avian aspergillosis, especially in cases where more conventional therapies have been ineffective. The combination therapy of terbinafine[f] at 10 to 15 mg/kg PO q12-24h with iatraconazole[c] is a safe and very effective treatment for systemic avian fungal disease.

Nebulization Therapy

Aerosolization therapy is used to augment systemic treatment by delivering drugs directly to the respiratory tract. Target tissues receive high concentrations of the drug while minimizing systemic exposure.[13] In addition, hepatic first-pass metabolism following oral administration is circumvented.[13] Clotrimazole[e] supplied as a 1% solution can be used for nebulization therapy at 30 minutes q24h.[63] However, a 15% aqueous suspension can be compounded, which appears to be more effective than the commercial form (P. Redig, personal communication, 2003). Terbinafine hydrochloride[f] nebulized as a 1 mg/ml aqueous solution (20 minutes q8h) was effective in resolving a non-responsive respiratory aspergillosis in a 6-year-old Congo African grey parrot (*Psittacus erithacus*).[28]

A commercially available disinfectant[g] is useful for treatment of birds within their quarters or en masse when nebulized at a 1:250 aqueous dilution. Nebulization therapy q12h for 3 to 12 weeks has been successful in the treatment of aspergillosis in a 12-week-old Congo African grey parrot and effective in combination with itraconazole[c] for raptorial species.[7,107] It also can be used to fog aviary rooms and pigeon lofts to reduce the level of fungal contamination in these premises[107].

Surgical Treatment

Treatment of respiratory aspergillosis is clinically challenging. Granulomatous lesions may occlude vital respiratory pathways or prove resistant to therapy because of poor drug penetration into the encapsulated lesions.[24,87,112] If nebulization and systemic administration of antifungal agents is not effective, surgical debulking of granulomas may be necessary.[87] For infections of the sinus cavities, trephination of the frontal sinuses permits direct access to granulomas for debridement and topical application of medications in an otherwise inaccessible site.[87]

Rhinitis or sinusitis of *Aspergillus* origin may require enzymatic therapy to dissolve the caseated debris and allow flushing of the affected area with the appropriate antifungal agent. Both hyaluronidase and trypsin-based flushes have been used for this purpose.

Tracheal and syringeal granulomas may precipitate life-threatening respiratory tract obstructions. Granulomas often form just proximal to or at the bifurcation of the trachea. Birds can effectively ventilate through cannulae placed in the clavicular, caudal thoracic or abdominal air sacs.[33,51,98,101] These can provide an alternative airway in emergency cases of tracheal obstruction and can be used for both short- and long-term duration. They can be left in place for periods of up to 1 week, but should be removed when possible to prevent iatrogenically induced air sacculitis.[89,101]

Cannulae also may be used to induce and maintain anesthesia during tracheal, syringeal or oral surgery. For suspected tracheal granulomas, an open-end tom cat catheter can be used to obtain clinical samples and for suction therapy to cannulate the tracheal lumen. Tracheotomy, through a ventral midline incision centered over the thoracic inlet, may be used to access granulomas in the tracheal lumen.[30,89] If the granuloma is at the level of the syrinx, blunt dissection through the interclavicular air sac or an alternative left lateral approach may provide needed access.[89] A clavicular osteotomy may be necessary to improve surgical access to the syrinx in some birds.[89]

Fungal granulomas of the lower respiratory tract are often resistant to medical therapy.[24,87,112] Surgical resection of these masses has been recommended; however, little has been published about techniques and their respective success.[24,89,109] Attempts at surgical removal are often incomplete due to the location of granulomas within the respiratory tract and the need to minimize trauma to adjacent structures.[53] Endoscopy allows access to most of the lower avian respiratory tract with minimal trauma and is an alternate approach for removing these lesions.[109] A detailed knowledge of the avian air sac system and competence with the rigid endoscope is a prerequisite before attempting this procedure[53] (see chapter 24, Diagnostic Value of Endoscopy and Biopsy).

A recent report describes endoscopic debridement and laser ablation as a viable alternative to conventional surgery in the management of lung and air sac granulomas.[53] A gallium-aluminum-arsenide diode laser, employing flexible fibers passed through the operating channel of the rigid endoscope, was used to ablate remaining granulomatous tissues in the air sacs after endosurgical debulking.[53] A small volume of amphotericin B[b] was used to sterilize the remaining lesion. Greater clinical success was achieved by endosurgical debridement and laser ablation, compared with conventional surgical exploration and removal of granulomas or medical therapy alone.[53]

Summary

In treating aspergillosis, proper supportive care including heated environment, fluids and nutritional support are essential. Because affected birds are severely compromised, the risk of secondary bacterial infections is significant. Antibiotic therapy is indicated. Prolonged antifungal therapy for periods up to 4 to 6 months is often necessary for treatment success.

Patients should be closely monitored for clinical improvement and observed for any signs of toxicosis during this period. Treatments are usually continued for 1 month after the complete blood count has returned to normal.[87] Aspergillosis is a preventable disease. Proper

Fig 29.11 | *Candida albicans* in an avian fecal Gram's stain. Yeast in this form proliferates in the lumen of the digestive tract and is responsive to topically acting antifungal agents (nystatin[h]).

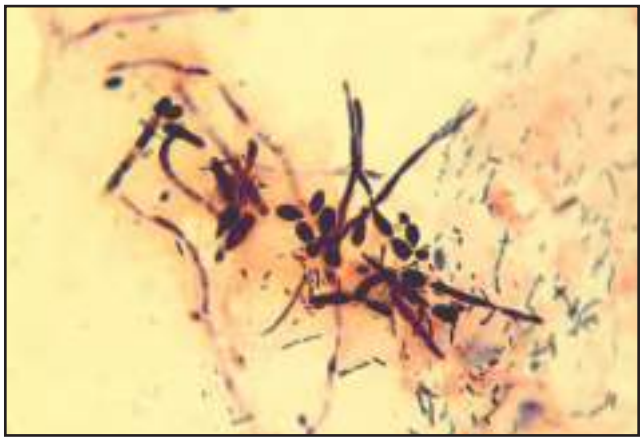


Fig 29.12 | *Candida albicans* exhibiting mycelial growth in a fecal Gram's stain from a cockatiel (*Nymphicus hollandicus*). Yeast in this form is tissue invasive and requires systemic antifungal therapy for proper treatment.

diet and husbandry practices to reduce stress and provide good hygiene will reduce factors that predispose to the development of this disease.

Candidiasis

Candidiasis in birds, also known as thrush, moniliasis or sour crop, refers to infections by yeasts of the genus *Candida*. *C. albicans* is the most commonly implicated species, although *C. parapsilosis*, *C. krusei*, and *C. tropicalis* also may cause disease.[103]

C. albicans is an opportunistic yeast and not regarded as a primary pathogen. Small numbers of the non-budding organism are commonly found in the digestive tract of normal birds and considered normal flora in healthy pigeons.[102] Host defense mechanisms and bacterial flora keep numbers of the organism controlled. *Candida* spp. can proliferate and cause disease when digestive tract flora are severely suppressed. In most cases, the infection is endogenous in origin, occurring secondarily to stress, immunosuppression, inadequate nutrition, poor sanitation, debilitation or in birds that have been extensively treated with antibiotics.

This disease is most often seen in psittacine neonates and cockatiels. Yeasts gain entry into the host by the oral route.[102] The organisms can be transmitted from the parent bird to chicks during regurgitative feeding. The infection also may be spread throughout the nursery population by the use of contaminated fomites and feeding utensils.

Candidiasis affects the mucocutaneous areas of the body and gastrointestinal mucosa, particularly of the oropharynx, crop and esophagus. Affected birds are depressed and may exhibit delayed crop emptying, regurgitation, crop stasis, inappetence and poor digestion of food. Droppings are often abnormal, appearing brownish in color and watery. Affected chicks do not grow or gain weight well and appear stunted.

Lesions vary in severity. They consist of thickening of the digestive tract mucosa with increased mucous and pseudomembranous patches. The choanae may become abscessed with formation of a diphtheritic membrane in the oropharynx as mycelial growth develops.[102] Whitish plaques may be evident under the tongue, in the mouth and most frequently in the crop. In advanced cases, stomatitis and palpable thickening of the ingluvies are present. Endoscopic examination of affected membranes of the oropharynx, crop, esophagus, proventriculus and ventriculus may reveal white-grey to grey-green, thickened and diphtheritic membranes. A characteristic "Turkish towel" thickening of the crop lining is evident in advanced cases.

Diagnosis is made by identifying the organism on wet or Gram's-stained smears from lesions in the oral cavity, crop or cloaca (Fig 29.11). Crop washings and fecal samples also may be used. *Candida albicans* grows as a budding yeast cell, oval in shape, 3.5 to 6.0 x 6.0 to 10.0 μm in size. High numbers of budding yeast confirm the diagnosis. The presence of elongated pseudohyphae suggests more severe infection with deeper tissue involvement (Fig 29.12). The organism can be cultured on mycological agar with cycloheximide and chloramphenicol at 37° C for 24 to 48 hours. Growth appears as shiny and convex round colonies measuring 3 to 5 mm in diameter, pearl white to light cream in color.

Differential diagnoses for inflammation of the upper gastrointestinal tract include bacterial stomatitis, trichomoniasis, capillariasis and nutritional disorders (see Chapter 4, Nutritional Considerations).

TREATMENT

Correction of the diet and husbandry are necessary for successful treatment of candidiasis. Nystatin[h] is the first drug of choice for yeast infections confined to the alimentary tract. It is not absorbed from the digestive tract and is effective for oral or topical use only. Nystatin[h] is fungistatic in action and must come in contact with the organism to be effective. Oral lesions may not respond if the drug is administered by gavage tube beyond this site of infection. The drug also can be applied directly to lesions of the mucous membranes in the oropharynx. The recommended dose of 290,000 units/kg PO q8-12h is safe and effective for use in psittacine neonates.[21] For flock treatment, nystatin[h] can be added to the drinking water at 100,000 IU/L.[102]

Severe yeast infections may be refractory to nystatin[h] therapy. If the organism is resistant to nystatin[h] or is in the hyphal stage, having penetrated the wall of the digestive tract, systemic antifungals are indicated. Fluconazole[d] or ketoconazole[i] are the systemic drugs of choice. Fluconazole[d] is one of the most effective antifungal agents for the treatment of tissue-based yeast infections. A dose of 5 to 15 mg/kg PO q12h is recommended for most avian species.[21] It also is effective against alimentary tract yeast when added to the drinking water at 50 mg/L.[102] Ketoconazole[i] also can be used to treat systemic yeast infections at 10 to 30 mg/kg PO q12h. It can be added to the drinking water at 200 mg/L for flock treatment of pigeons.[102]

Itraconazole[c] has been used in the successful treatment of candidal tracheitis in a blue and gold macaw (*Ara ararauna*) and candidal infection of the uropygial gland in a king penguin (*Aptenodytes patagonicus*).[55] Some *Candida* spp. are, however, extremely resistant to itraconazole[c].[114] The drug is unlikely to achieve therapeutic concentrations at 5 mg/kg and should be used at the higher dose of 10 mg/kg PO q24h.

Oral chlorhexidine[j] at 10 to 20 ml per gallon drinking water for 3 weeks can be used for flock control of *Candida* infections but generally will not eliminate them. Mild cases of candidiasis may respond to acidification (see apple cider vinegar in Chapter 9, Therapeutic Agents).

Macrorhabdosis

Clinical disease caused by the organism historically known as *Megabacterium* has been referred to as megabacteriosis, *Megabacterium*-associated disease and proventricular disease in birds.[6,36] More recent studies have confirmed that the organism is a fungus and represents a new genus of ascomycetous yeast called *Macrorhabdus ornithogaster*.[91,110] The clinical condition in birds is more properly referred to as macrorhabdosis. The reader is referred to Chapter 30, Implications of *Macrorhabdus* in Clinical Disorders in this text for detailed discussion on this topic.

Mycotic Dermatitis

Fungal dermatitis is rarely reported in birds, even though fungi such as *Trichophyton* and *Aspergillus* spp. have been recovered from the feathers, skin and eyes of healthy birds.[32,122] Infections of the integument caused by *Candida albicans*, *Rhodotorula*, *Microsporum gallinae*, *Aspergillus*, *Rhizopus*, *Malassezia* and *Mucor* species have been described.[17,25,85,118] Skin and feather lesions associated with *Aspergillus* have been recognized in pigeons and psittacine birds.[111] *Aspergillus* and *Alternaria* spp. also have been associated with epidermal cysts in the domestic chicken.[108] Other reports of fungal dermatitis include *Trichophyton* in canaries (*Serinus canarius*), *Microsporum gypseum* in budgerigars (*Melopsittacus undulatus*), *Microsporum gallinae* and *Cladosporium berbarum* in chickens (*Gallus gallus*) and *Candida* species in gallinaceous birds.[66,105,111]

Favus, commonly referred to as "avian ringworm," describes a mycotic dermatitis found primarily in gallinaceous birds. It consists of white, crusting lesions of the face, comb and wattles that can extend to the feathered portion of the head. *Microsporum gallinae* is the agent most often involved, although *M. gypseum* and *Trichophyton simii* also have been isolated.

Correction of diet and husbandry issues combined with topical and oral systemic miconazole[l] therapy is usually efficacious in treating mycotic dermatitis in affected birds.

Cryptococcosis

Cryptococcosis is most commonly caused by infection with *Cryptococcus neoformans* var. *neoformans*, an encapsulated saprophytic fungus with worldwide distribution. *Cryptococcus neoformans* var. *gatti* is more geographically restricted because of an ecological association with the river red gum (*Eucalyptus camaldulensis*) and other eucalyptus trees.[34] The organism is commonly found in soils contaminated with bird droppings.[68]

While cryptococcosis is a rare disease of birds, disseminated infection has been reported in the green-winged macaw (*Ara chloroptera*), Moluccan cockatoo (*Cacatua*

moluccensis), thick-billed parrot (*Rhynchopsitta pachyrhyncha*) and North Island brown kiwi (*Apteryx australis mantelli*).[23,27,35,54,100] Infections may involve the respiratory tract, digestive tract and central nervous system, producing necrotic granulomatous lesions and a characteristic thick, pale, gelatinous exudate. The lower temperature of the upper respiratory tract makes it more susceptible than other areas of the body to initial colonization with *Cryptococcus*.[23] Upper respiratory tract involvement can produce facial granulomas that distort the rhamphotheca.[20,27,31] A chronic rhinosinusitis resembling a neoplasm of the rhamphotheca was described in a Major Mitchell's cockatoo (*Cacatua leadbeateri*) and was due to *C. neoformans* var. *gatti*.[90] An encephalitis or meningitis also may occur, causing blindness or paralysis in affected birds.[23,35]

Diagnosis of cryptococcosis should be based on cytology and histopathology in combination with culture rather than culture of nasochoanal swabs or washes alone.[90] *Cryptococcus neoformans* var. *neoformans* and var. *gatti* may be carried asymptomatically in the upper respiratory tract. Wright's-stained smears of gelatinous material often reveal aggregates of encapsulated yeast organisms measuring 6 to 10 μm within 8- to 12-μm non-staining capsules.[90]

Veterinarians must use extreme caution when handling clinical materials that may contain *Cryptococcus* spores. Most human infections occur through contact with contaminated exudates, fecal material and non-clinical infected or diseased birds.[74,78] While human infection with *C. neoformans* var. *neoformans* is well recognized in immunosuppressed patients, infection with *C. neoformans* var. *gatti* is commonly associated with otherwise healthy and immunocompetent individuals.[68,106]

Antifungal agents such as amphotericin B[b], fluconazole[d] or itraconazole[c] have been suggested as treatment for cryptococcosis.[23] Fluconazole[d] administered orally at a dosage of 8 mg/kg q24h for 2 months was successful in resolving bilateral nasal cryptococcosis in an African grey parrot, but the lesions recurred 3 years later.[31]

Although cryptococcosis is a rare disease of birds, the zoonotic potential associated with this infection is significant. Veterinarians must be aware of this disease when diagnosing and treating upper respiratory disease in birds and must remember to discuss the zoonotic potential of this infection with their clients.

Histoplasmosis

Histoplasmosis is an infectious but not contagious mycotic disease that has been reported in poultry and zoo specimens. The soil-borne organism *Histoplasma capsulatum* has worldwide distribution and is endemic in the eastern and central USA. It is commonly associated with fecal material from pigeons and gallinaceous birds, and has the potential to grow within dirt substrates of enclosed aviaries.[8] *Histoplasma* infections in birds produce disease signs similar to those seen with *Cryptococcus* spp. infections. An initial pneumonia can progress to disseminated disease with the formation of necrotic granulomas. Histoplasmosis was identified as the cause of an osteomyelitis and mineralized soft tissue granuloma of the shoulder and antebrachium in a Moluccan cockatoo.[119] The infection should be considered part of the differential diagnosis of granulomatous respiratory disease in avian patients. Diagnosis is based on culture of the organism and histopathologic examination of tissue samples.

Mucormycosis

The order Mucorales includes a number of saprophytic fungi that have been implicated as possible avian pathogens. They have been implicated as an etiologic agent of meningoencephalitis in birds.[11,86] Hyphal invasion of cerebral blood vessels and dissemination of an *Absidia* sp. in the cerebrum was identified as the cause of progressive neurologic defects culminating in seizures in a chattering lory (*Lorius garrulus*).[80] Other clinical syndromes described include air sacculitis in a pigeon (*Columba* sp.), pneumonia in a rock hopper penguin (*Eudyptes crestatus*) and a group of rock ptarmigan (*Lagopus mutus*), and an osteolytic mass involving the ribs and air sacs of a penguin (Spheniscifformes).[12,50,67,84] The feeding of damp, germinated seed has been implicated in disseminated mucormycosis causing alimentary granulomas in a group of canaries (*Serinus canarius*) and nephritis in an African grey parrot; glossitis in an African grey parrot; myocarditis in an Australian parakeet (*Psittacula* sp.); and nasal infection in waterfowl.[16,29,75] *Absidia corymbifera* is the pathogen most often isolated, although *Mucor* and *Rhizopus* spp. also are identified.[66]

Antemortem diagnosis of mucormycosis is difficult because the organisms do not culture well from clinical samples.[69] Histopathology of biopsy specimens is more reliable in confirming the diagnosis.[80]

No effective treatment of mucormycosis in birds has been reported. Amphotericin B[b] is the single most reliable agent used in humans.

Other antifungal medications including nystatin[h], 5-fluorcytosine[m], clotrimazole[e] and miconazole[i] are reported to have no consistent in vivo activity against the Mucorales.[69]

Dactylariosis

Dactyloriosis is a fatal encephalitis of poultry caused by *Dactylaria gallopava*. The organism grows in old sawdust and wood shavings. Infection involves the central nervous system, causing torticollis, incoordination, tremors and sternal recumbency. The respiratory system is a less commonly involved site. The signs and lesions of dactylariosis resemble those caused by aspergillosis. Culture can be used to differentiate the infection.

Resources Mentioned in the Text

a. Veterinary Molecular Diagnostics, Inc, 5989 Meijer Dr, Suite 5, Milford, OH 45150, USA, 513-576-1808
b. Fungizone, Bristol-Meyers Squib Company, Princeton, NJ 08543, USA
c. Sporanox, Janssen Pharmaceutical Inc, Titusville, NJ 08560, USA
d. Diflucan, Pfizer Inc, New York, NY, USA
e. Lotrimin, Schering, Schering Corporation, Kenilworth, NJ 07033, USA
f. Lamisil, Novartis Consumer Health, Inc, Summit, NJ 07901-1312, USA
g. F10, Health and Hygiene Ltd, PO Box 347, Sunning hill 2157, South Africa, www.healthandhygiene.net
h. Nystatin oral suspension, Lederle Standard Products, Pearl River, NY 10965, USA
i. Nizoral, Janssen Pharmaceutica, Titusville, NJ 08560, USA
j. Nolvasan, Fort Dodge Animal Health, Fort Dodge, IA 50501, USA
k. Enilconazole, Janssen, Titusville, N.J. 08560
l. Miconazole, Janssen, Titusville, N.J. 08560
m. 5-fluorcytosine, Rocké Pharmaceuticals, Nutley, N.J. 07110

References and Suggested Reading

1. Abrams GA, et al: *Aspergillus* blepharitis and dermatitis in a peregrine falcon-gyrfalcon hybrid (*Falco peregrinus* X *Falco rusticolus*). J Avian Med Surg 15:114-120, 2001.
2. Ainsworth GC, Austwick PK: Fungal Diseases of Animals. Farnam Royal, Slough, England, 1973.
3. Anderson N: Candida/Megabacteria proventriculitis in a lesser sulphur-crested cockatoo (*Cacatua sulphurea sulphurea*). J Avian Med Surg 7:197-201, 1993.
4. Andrew SE, et al: Equine ulcerative keratomycosis: Visual outcome and ocular survival in 39 cases (1987-1996). Equine Vet J 30:109-116, 1998.
5. Atkinson R: Unusual presentations of aspergillosis in wild birds. Proc Assoc Avian Vet, 1998, pp 177-181.
6. Baker JR: Megabacteriosis in exhibition budgerigars. Vet Rec 131:12-14, 1992.
7. Bailey T, Sullivan T: Aerosol therapy in birds using a novel disinfectant. Exotic DVM 3(4):17, 2001.
8. Bauck L: Mycosis. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 997-1006.
9. Bauck L, et al: Case reports. Proc Assoc Avian Vet, 1992, pp 134-139.
10. Beckman BJ, et al: *Aspergillus fumigatus* keratitis with intraocular invasion in 15-day-old chicks. Avian Dis 38:660-665, 1992.
11. Bennett RA: Neurology. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 723-747.
12. Bigland CH, Graesser FE, Pennifold KS: An osteolytic mucormycosis in a penguin. Avian Dis 5:367-370, 1961.
13. Boothe DM: Drugs affecting the respiratory system. Vet Clin No Am Exotic Pet Med 3:371-394, 2000.
14. Brooks DE, et al: Antimicrobial susceptibility patterns of fungi isolated from horses with ulcerative keratomycosis. Am J Vet Res 59:138-142, 1998.
15. Brown PA, Redig, PT: *Aspergillus* ELISA: A tool for detection and management. Proc Assoc Avian Vet 1994, pp 295-300.
16. Burr EW, Huchzermeyer FW: Mucormycosis in a parrot. Mod Vet Pract 12:961-962, 1982.
17. Burgmann PM: Common psittacine dermatologic diseases. Sem Avian Exotic Pet Med 4:169-183, 1995.
18. Campbell TW: Avian Hematology and Cytology. Ames, IA, Iowa State Univ Press, 1995, pp 3-19.
19. Campbell TW: Hematology. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Applications. Brentwood, TN, HBD Int'l Inc, 1999, pp 177-198.
20. Cannon M: Avian cryptococcosis: A case report. Proc Aust Comm Assoc Avian Vet, 1999, pp 1-5.
21. Carpenter JW, Mashima TY, Rupiper DJ: Exotic Animal Formulary. Philadelphia, WB Saunders Co, 2001.
22. Carrasco L, et al: Application of enzyme-immunochemistry for the diagnosis of aspergillosis, candidiasis, and zygomycosis in three lovebirds. Avian Dis 37:923-927, 1993.
23. Clipsham R, Brit JO: Disseminated cryptococcosis in a green-winged macaw (*Ara*). J Am Vet Med Assoc 183:1303-1304, 1983.
24. Clippinger TL: Diseases of the lower respiratory tract of companion birds. Sem Avian Exotic Pet Med 6:201-208, 1997.
25. Cooper JE, Harrison GJ: Dermatology. *In* Richie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l Inc, 1999, pp 997-1006.
26. Cray C, Zielezienski-Roberts K: Application of *Aspergillus* antigen assay in the diagnosis of aspergillosis. Proc Assoc Avian Vet, 1997, pp 219-221.
27. Curtis-Velasco M: Candidiasis and cryptococcosis in birds. Sem Avian Exotic Pet Med 9:75-81, 2000.
28. Dahlhausen RD, Lindstrom JG, Radabaugh CS: The use of terbinafine hydrochloride in the treatment of avian fungal disease. Proc Assoc Avian Vet, 2000, pp 35-39.
29. Dawson CO, Wheeldon EB, McNeil PE: Air sac and renal mucormycosis in an African grey parrot (*Psittacus eritbacus*). Avian Dis 20:593-600, 1976.
30. Dennis PM, et al: Diagnosis and treatment of tracheal obstruction in a cockatiel (*Nymphicus hollandicus*). J Avian Med Surg 13:275-278, 1999.
31. Doneley B: Cutaneous cryptococcosis in an African grey parrot. Proc Aust Comm Assoc Avian Vet, 1994, p 247.
32. Dupont C, Carrier M, Higgins R: Bacterial and fungal flora in healthy eyes of birds of prey. Can Vet J 34:699-701, 1994.
33. Dustin LR: Surgery of the avian respiratory system. Sem Avian Exotic Pet Med 2:83-90, 1993.
34. Ellis DH, Pfeiffer TJ: Natural habitat of *Cryptococcosis neoformans* var. *gatti*. J Clin Microbiol 228:1642-1644, 1990.
35. Fenwick B, Takeshita K, Wong A: A Moluccan cockatoo with disseminated cryptococcosis. J Am Vet Med Assoc 187:1210-1212, 1985.
36. Filippich LJ, Parker MG: Megabacteriosis and proventricular/ventricular disease in psittacines and passerines. Proc Assoc Avian Vet, 1994, pp 287-293.
37. Filippich LJ, Perry RA: Drug trials against megabacteria in budgerigars (*Melopsittacus undulates*). Aust Vet Pract 23:184-189, 1993.
38. Flammer K: An overview of antifungal therapy in birds. Proc Assoc Avian Vet, 1993, pp 1-4.
39. Forbes NA: Aspergillosis in raptors. Vet Rec 128:263, 1991.
40. Forbes NA, Simpson GN, Goudswaard MF: Diagnosis of avian aspergillosis and treatment with itraconazole. Vet Rec 130:519-520, 1992.
41. Foster CS: Fungal keratitis. *In* Albert DM, Jakobiec FA, Robinson NL (eds): Principles and Practice of Ophthalmology: Clinical Practice. Philadelphia, WB Saunders Co, 1994, pp 171-179.
42. Fransen J, VanCulsem J: Fungal infections in birds in captivity (synopsis). Assoc Avian Vet Today 2:15, 1988.
43. Fudge AM, Reavill DR: Bird litter and aspergillosis warning. J Avian Med Surg 7:15, 1993.
44. Fudge AM: Avian clinical pathology, hematology and chemistry. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 142-147.
45. Fudge AM, Reavill DR, Rosskopf WJ, Jr: Diagnosis and management of avian dyspnea: A review. Proc Assoc Avian Vet, 1993, pp 187-195.
46. Gerlach H: Megabacteriosis. Sem Avian Exotic Pet Med 10:12-19, 2001.
47. Graybill JR: New antifungal agents. Euro J Clin Microbial Infect Dis 8:402-412, 1989.
48. Greenacre CS. Leg paresis in a black palm cockatoo caused by aspergillosis. J Zoo Wildl Med 23:122-126, 1992.
49. Greene R: The pulmonary aspergilloses: Three distinct entities or a spectrum of disease. Radiology 140:527-530, 1981.
50. Hanssen I: Pulmonary phycomycosis in captive rock ptarmigan (*Lagopus mutus*) and willow ptarmigan (*Lagopus lagopus*) chicks. Acta Vet Scand 16:134-136, 1975.
51. Harrison GJ: Selected surgical procedures. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1986, pp 581-582.
52. Henderson GM, Gulland FM, Hawkey CM: Haematological findings in budgerigars with megabacterium and *Trichomonas* infections associated with "going light." Vet Rec.123(19):492-494, 1988.
53. Hernandez-Divers, SJ: Endosurgical debridement and diode laser ablation of lung and air sac granulomas in psittacine birds. J Avian Med Surg 16:138-145, 2002.
54. Hill FI, Woodgyer AJ, Lintott MA: Cryptococcosis in a North Island brown kiwi (*Apteryx australis mantelli*) in New Zealand. J Med Vet Mycol 33:305-309, 1995.
55. Hines RS, Sharkey P, Friday RB: Itraconazole treatment of pulmonary, ocular, and uropygial aspergillosis and candidiasis in

birds-data from five clinical cases and controls. Proc Am Assoc Zoo Vet, 1990, pp 322-327.
56. Hoppes S, et al: Mycotic keratitis in a blue-fronted Amazon parrot (*Amazona aestiva*). J Avian Med Surg 14:185-189, 2000.
57. Huchzermeyer FW, Henton MM, Keffen RH: High mortality associated with megabacteriosis of proventriculus and gizzard in ostrich chicks. Vet Rec 133:143-144, 1993.
58. Ivey ES: Serologic and plasma protein electrophoretic findings in 7 psittacine birds with aspergillosis. J Avian Med Surg 14:103-106, 2000.
59. Jenkins JR. Aspergillosis. Proc Assoc Avian Vet 1991, pp 328-330.
60. Jones MP: Avian clinical pathology. Vet Clin No Am Exotic Anim Pract 1999;2:663-687.
61. Jones MP, et al: Pharmacokinetic disposition of Itraconazole in red-tailed hawks (*Buteo jamaicensis*). J Avian Med Surg 14:15-22, 2000.
62. Jordan FTW: Fungal diseases. *In* Jordan FTW (ed): Poultry Diseases 3rd ed. Philadelphia, Bailliere Tindall, 1990, pp 216-225.
63. Joseph V, Pappagianis D, Reavill DR: Clotrimazole nebulization for the treatment of respiratory aspergillosis. Proc Assoc Avian Vet, 1994, pp 301-306.
64. Kern TJ: Exotic animal ophthalmology. *In* Gelatt KN (ed): Veterinary Ophthalmology 3rd ed. Philadelphia, Lippincot, Williams & Wilkins, 1999, pp 1273-1305.
65. Kerridge D: Mode of action of clinically important antifungal drugs. Adv Microbiol Physiol 27:1-72, 1998.
66. Keymer IF: Mycosis. *In* Petrak ML (ed): Diseases of Cage and Aviary Birds 2nd ed. Philadelphia, Lea & Febiger, 1982, pp 599-605.
67. Kon VM, Linton AH: Mucormycotic infection of birds. Vet Rec 73:1008, 1961.
68. Kwon-Chung KJ: Cryptococcosis. *In* Kwon-Chung KJ, Bennett JE (eds): Medical Mycology. Philadelphia, Lea & Febiger, 1992, pp 397-446.
69. Lehrer RI, et al: Mucormycosis. Ann Intern Med 93:93-108,1980.
70. Lumeij JT, Gorgevska D, Woestenborghs R: Plasma and tissue concentrations of itraconazole in racing pigeons (*Columba livia domestica*). J Avian Med Surg 9:32-35,1995.
71. Luttrull JK, et al: Treatment of ocular fungal infections with oral fluconazole. Amer J Ophthalmol 119:477-481, 1994.
72. Lyman CA, Walsh TJ: Systematically administered antifungal agents. A review of their clinical pharmacology and therapeutic applications. Drugs 44:9-35, 1992.
73. McMillian M, Petrak M: Retrospective study of aspergillosis in pet birds. J Avian Med Surg 3:211-215, 1989.
74. Micalezzi C, Persi A, Parodi A: Primary cutaneous cryptococcosis in an immunocompetent pigeon keeper. Clin Exp Dermatol 22:195-197, 1997.
75. Mitchell G, Esnouf D, Pritchard R: Mucormycosis in canaries (*Serinus canaries*) fed damp germinated seed. Vet Pathol 23:625-627, 1986.
76. Moore RP, Snowden KF, Phalen DN: A method of preventing transmission of so-called "megabacteria" in budgerigars (*Melopsittacus undulatus*). J Avian Med Surg 4:283-287, 2001.
77. Mori T, et al: Systemic aspergillosis caused by an aflatoxin-producing strain of *Aspergillus flavus*. Med Mycol 36:107-112, 1998.
78. Nosanchuck JD, et al: Evidence of zoonotic transmission of *Cryptococcosis neoformans* from a pet cockatoo to an immunocompromised patient. Ann Intern Med 132:205-208, 2000.
79. Oglesbee BL: Mycotic diseases. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 323-331.
80. Orcutt CJ, Bartick TE: Mucormycotic meningoencephalitis and pneumonia in a chattering lory (*Lorius Garrulus*). J Avian Med Surg 8:85-89, 1994.
81. Orosz SE, Frazier DL: Antifungal agents: A review of their pharmacology and therapeutic indications. J Avian Med Surg 9:8-18, 1995.
82. Orosz SE, et al: Pharmacokinetic properties of itraconazole in blue-fronted Amazon parrots (*Amazona aestiva aestiva*). J Avian Med Surg 10:168-173, 1996.
83. Orosz SE, et al: The effects of formulation on the systemic availability of itraconazole in pigeons. J Avian Med Surg 9:255-262, 1995.
84. Panigrahy B, et al: Candidiasis in cockatiel nestlings and mucormycosis in a pigeon. Avian Dis 23:757-760, 1979.
85. Pass DL: The pathology of the avian integument: A review. Avian Pathol 18:17, 1989.
86. Paul-Murphy J: Avian neurology. Proc Assoc Avian Vet, 1992, pp 420-432.
87. Phalen DN: Respiratory medicine of cage and aviary birds. Vet Clin No Am Exotic Anim Pract 3:423-452, 2000.
88. Phalen DN, Tomaszewski E, Davis A: Investigation into the detection, treatment, and pathogenicity of avian gastric yeast. Proc Assoc Avian Vet, 2002, pp 49-51.
89. Pye G: Surgery of the avian respiratory system. Vet Clin No Am Exotic Pet Med 3:693-713, 2000.
90. Raidal SR, Butler R: Chronic rhinosinusitis and rhamphothecal destruction in a Major Mitchell's cockatoo (*Cacatua leadbeateri*) due to *Cryptococcosis neoformans* var. *gatti*. J Avian Med Surg 15:121-122, 2001.
91. Ravelhofer-Rothenender K, et al: Untersuchungen zur taxonomischen Einordung von "Megabakterien. " Proc XII DVG-Tagung über Vogelkrankheiten, 2000, p 15.
92. Redig PT: Mycotic infections of birds of prey. *In* Fowler ME (ed): Zoo and Wild Animal Medicine: Current Therapy 3. Philadelphia, WB Saunders Co, 1993, pp 178-181.
93. Redig PT, Orosz S, Cray C: The ELISA as a management guide for aspergillosis in raptors. Proc Assoc Avian Vet, 1997, pp 99-104.
94. Redig P: Diagnosis of avian aspergillosis. Proc Assoc Avian Vet, 1999, pp 355-357.
95. Redig PT: Aspergillosis. *In* Samour J (ed): Avian Medicine. Philadelphia, Mosby, 2000, pp 275-287.
96. Reidarson TH, McBain J: Serum protein electrophoresis and *Aspergillus* antibody titers as an aid to diagnosis of aspergillosis in penguins. Proc Assoc Avian Vet, 1995, pp 61-64.
97. Richard JL, et al: Recent studies on aspergillosis in turkey poults. Mycopathologia 87:3-11, 1984.
98. Rode J, Bartlow S, Ludders J: Ventilation through an air sac cannula during tracheal obstruction in ducks. J Avian Med Surg 4:98-102, 1990.
99. Rossi G: Histological and immunohistochemical findings in proventricular mucosa of chickens experimentally infected with "megabacterium." Proc Euro Soc Vet Pathol, 2000, p 156.
100. Rosskopf WJ, Woerpel RW: Cryptococcosis in a thick-billed parrot. Proc Assoc Avian Vet, 1984, pp 281-288.
101. Rosskopf WJ: Surgery of the avian respiratory system. Proc Amer Coll Vet Surg, 1988, pp 373-382.
102. Rupiper DJ: Diseases that affect race performance of homing pigeons. Part II: bacterial, fungal, and parasitic diseases. J Avian Med Surg 12:138-148, 1998.
103. Samour JH, Naldo, JL: Diagnosis and therapeutic management of candidiasis in falcons in Saudi Arabia. J Avian Med Surg 16:129-132, 2002.
104. Scanlan CM, Graham DL: Characterization of a gram-positive bacterium from the proventriculus of budgerigars (*Melopsittacus undulatus*). Avian Dis 34:779-786, 1990.
105. Shivaprasad HL: Poultry ophthalmology. *In* Gelatt K (ed): Veterinary Ophthalmology 3rd ed. Philadelphia, Lippincot, Williams & Wilkins, 1999, pp 1177-1207.
106. Speed B, Dunt D: Clinical and host differences between infections with the two varieties of *Cryptococcosis neoformans*. Clin Infect Dis 21:28-34, 1995.
107. Stanford M: Use of F10 in Psittacines. Exotic DVM 3(4)18, 2001.
108. Suedmeyer WK, Bermudez A, Fales W: Treatment of epidermal cysts associated with *Aspergillus fumigatus* and an *Alternaria* sp. in a silky bantam chicken (*Gallus gallus*) Proc Am Assoc Zoo Vet Int Assoc Aquat Anim Med, 2000, p 307.
109. Taylor M: Endoscopic diagnosis of avian respiratory tract diseases. Sem Avian Exotic Pet Med 6:187-194, 2000.
110. Tomaszewski EK, et al: Phylogenetic analysis indicates the "megabacterium" of birds is a novel anamorphic ascomycetous yeast, *Macrorhabdus ornithogaste*r, gen. nov., sp. nov. Int J Sys Evol Micro, 2002.
111. Tudor DC: Mycotic infections of feathers as a cause of feather pulling in pigeons and psittacine birds. Vet Med Small Anim Clin 78:249-253, 1983.
112. Tully TN: Avian respiratory diseases: Clinical overview. J Avian Med Surg 9:162-174, 1995.
113. Ungerechts N: Radiographic signs of proventricular infection with "megabacteria." J Avian Med Surg 4:203, 1990.
114. Van Cutsem J, Van Gerven F, Janssen PAJ: Activity of orally, topically, and parenterally administered itraconazole in the treatment of superficial and deep mycoses: Animal models. Rev Infect Dis 9:15-32, 1987.
115. Van Cutsem J, Janssen PAJ: In vitro and in vivo models to study the activity of antifungals against *Aspergillus*. *In* Vanden Bossche H, MacKenzie DWK, Cauwenbergh G (eds): *Aspergillus* and Aspergillosis. New York, Plenum Press, 1987, pp 215-227.
116. Van Der Hayden N: Aspergillosis in psittacine chicks. Proc Assoc Avian Vet, 1993, pp 1-6.
117. VanHerck H, et al: A bacterial proventriculitis in canaries (*Serinus canaria*). Avian Pathol 13:561-572, 1984.
118. Van Sant F: Impression cytology: New insights into avian skin flora. Proc Assoc Avian Vet, 1999, pp 139-141.
119. Vaughn S: What is your diagnosis? J Avian Med Surg 10:33-35, 1996.
120. Wallach JD, Boever WJ: Diseases of Exotic Animals. Philadelphia, WB Saunders Co, 1983, pp 856-946.
121. Ward FP, Fairchild DG, Vuicich JV: Pulmonary aspergillosis in prairie falcon nest mates. J Wildl Dis 6:80-83, 1970.
122. Wolf ED, Amass K, Olsen J: Survey of conjunctival flora in the eye of clinically captive exotic birds. J Am Vet Med Assoc 11:1232-1233, 1983.
123. Wolff, PL, Petrini KR, Kolmstetter C: An outbreak of aspergillosis in crested wood partridges (*Rollulus roulroul*). J Zoo Wildl Med 23:108-112, 1992.

CHAPTER
30

Implications of *Macrorhabdus* in Clinical Disorders

DAVID N. PHALEN, DVM, PhD, Dipl ABVP-Avian

Fig 30.1 | *Macrorhabdus ornithogaster* stained with calcofluor white M2R and viewed with ultraviolet light (380-420 nm).

History and Identification of the Organism

The information available about macrorhabdosis (aka megabacteriosis, virgamycosis, avian gastric yeast) in birds is confusing for those unfamiliar with the literature and sometimes equally as confusing for those who are. The organism was originally thought to be a yeast because of its staining characteristics.[3] Subsequently, Van Herck, et al, concluded that it was a bacterium, as they were unable to demonstrate cytoplasmic organelles or a nucleus. They did, however, show nuclear-like structures in Giemsa-stained organisms but interpreted them to be "granules."[23] Scanlan and Graham reported isolating a bacterium from the stomach of budgerigars using standard microbiological techniques. The isolated bacterium, however, was smaller than the organism in vivo and was not characterized by periodic acid-Schiff (PAS) or silver stains.[20] Attempts by other investigators to grow this organism with standard microbiological isolation techniques have been unsuccessful. However, Gerlach reported isolation of this organism on MRS medium, a medium used for growing fungi, but was unable to maintain it past a few passages.[8] Huchzemeyer, et al, also reported isolating an organism from the proventriculus of ostriches using MRS agar. This organism had the same biochemical properties as the one isolated by Scanlan and Graham, but was smaller than those seen histologically, and its ability to stain with PAS and silver stains was not reported.[9]

More recent work suggested that the "megabacterium" was, in fact, a yeast. In vivo trials showed that the "megabacterium" was susceptible to amphotericin B but not to antibacterial antibiotics.[4] It also stained strongly with calcofluor white M2R (Fig 30.1) and blancophor BA, stains that bind to chitin and cellulose, products not found in bacteria.[11,16] It grows, albeit slowly, in cell culture media supplemented with dextrose, fetal calf serum and antibacterial antibiotics. A nucleus was demonstrated by electron microscopy, and in situ hybridization with a pan eukaryote rRNA probe was positive.[17] Prior to conclusive determination of the genus and species to which the organism belonged, it was temporarily called avian gastric yeast. Tomaszewski, et al sequenced the ribosomal DNA of this organism and used this information to prove that it was a novel anamorphic ascomycetous yeast that belongs in its own new genus.[22]

Originally, it was proposed that it be named *Virgamycosis avigastricus*, but this name was not accepted. Subsequently, it has been named *Macrorhabdus ornithogaster*.[22]

SIGNS OF INFECTION

There are mixed opinions about whether *M. ornithogaster* can cause disease. Many investigators consider it to be a pathogen, while others have described it as a commensal.[8,20] The truth probably falls in between. It is clear that under some, perhaps most, circumstances, infection with *M. ornithogaster* does not result in clinical signs. It is equally clear that certain individual birds will show signs that can be attributed to infection with this organism, and that the prevalence of disease may be high in some collections and perhaps in some species of birds. Whether this represents variation in the pathogenicity of different strains of *M. ornithogaster* or differing susceptibilities of the affected birds is not known.

Host Range

Macrorhabdus ornithogaster has a wide host range and a worldwide distribution. It was first described in canaries and has subsequently been identified in captive-raised and free-ranging finches.[3,6,13,23] The prevalence of infection in budgerigar aviaries is high, and the percentage of infected birds in aviaries where it is enzootic may range from 27 to 64%, as judged by fecal shedding.[1,4,5]

Macrorhabdus ornithogaster also is commonly found in parrotlets, cockatiels and lovebirds.[5,10,14] Filippich reports that it is seen in several species of captive Australian parrots.[5] *Macrorhabdus ornithogaster* also is reported in ostriches, chickens, turkeys, geese, ducks and two species of ibis.[8,10,12,21] It is suggested that organisms thought to be *M. ornithogaster* also can infect mammals.[19] The organisms used in these experiments, however, appear to be bacteria and not *M. ornithogaster*, and there is no evidence at this time to suggest that it is a pathogen of mammals.

BUDGERIGARS

Filippich describes two clinical presentations in the budgerigar. In the acute presentation, apparently healthy birds suddenly go off feed, regurgitate ingesta (which may be blood stained), and die within 1 to 2 days. In the more common chronic form, affected birds typically appear to be hungry and spend considerable time at the food dish. Instead of eating, however, these birds are grinding their food but not ingesting it. Regurgitation is common, and fresh or dried saliva is often found on the tops of affected birds' heads. Undigested seeds may be present in the droppings. Diarrhea with or without melena also may be present. These birds go through a prolonged period of weight loss (going light) where they appear unthrifty and eventually die. If the affected budgerigar colony is sufficiently large, there will always be a few birds in the collection that will be showing these signs. Birds with clinical signs of infection are reported to have decreased packed cell volumes and low sodium, chloride, phosphate, glucose, cholesterol and aspartate aminotransferase values. When there was gastric ulceration, markedly low total protein concentrations were observed. Contrast radiography revealed, in some birds, a dilated proventriculus and an increased transit time. In one aviary, mature birds with a mean age of 2.7 years were most commonly affected.[5,6] Although clinical signs are more common in middle-aged budgerigars, infection begins very early in these birds and the author has seen large numbers of organisms in the isthmus of nestling budgerigars that were only 12 days old. It is critical to note that other diseases in budgerigars also can cause similar signs; these include candidiasis of the crop or ventriculus, a bacterial ventriculitis, trichomoniasis, enteritis, heavy metal poisoning and neoplasia of the stomach.

PARROTLETS

Parrotlets appear to have an acute onset of disease where they develop regurgitation and may have melena (D. Zantop, personal communication). Infection and disease appear to be most common in the green-rumped parrotlet, especially its color mutations.[14]

LOVEBIRDS

Regurgitation and weight loss were seen in two flocks of lovebirds. Organisms believed to be *M. ornithogaster* were found in significant concentrations in the drop-

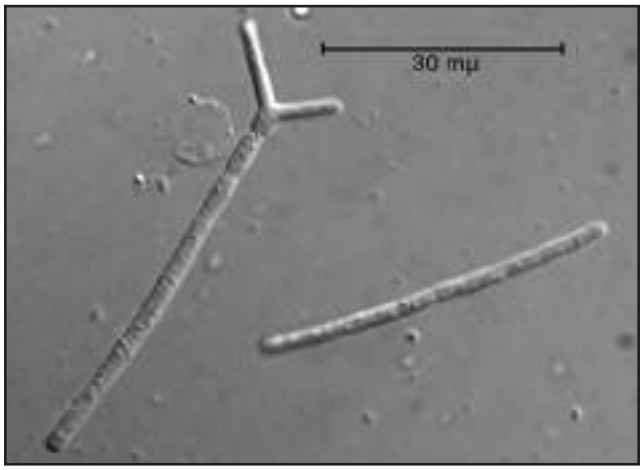


Fig 30.2 | Unstained *M. ornithogaster*. The organism on the right is typical. The Y-form on the left is rarely seen.

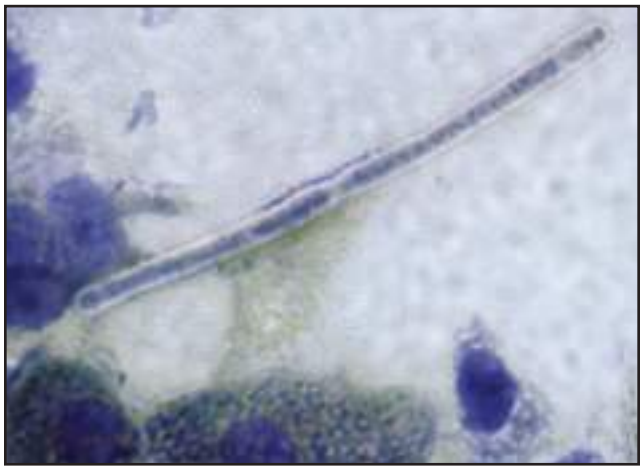


Fig 30.3 | Gram stain of *M. ornithogaster*. Only the cytoplasm stains. Often many of the organisms will stain only faintly or not at all with Gram's stain.

pings of these birds. Psittacine beak and feather disease also was found in affected birds and may have played a role in the ability of *M. ornithogaster* to cause disease in them (T. Lightfoot, personal communication, 2003). It should be noted, however, that psittacine beak and feather disease virus infection is widespread in lovebirds, so this may have been a coincidental finding.

CANARIES AND FINCHES

Signs of disease in canaries and finches are poorly defined. Most bird owners first recognize that there is a problem when a bird is found dead. Typically these birds are thin, suggesting a chronic course of disease that was unrecognized by the owner.[3,23]

OSTRICHES

Disease in ostriches associated with *M. ornithogaster* was described in 10-day-old to 12-week-old chicks. Birds appeared normal but ceased growing and lost weight. Eventually they became weak and died. Birds had soiled vents and were anemic. Diarrhea was observed in some birds while others had dry, pelleted stools. Mortality rates varied from 40 to 80% in affected flocks.[9]

CHICKENS

Two reports describe signs that were seen in flocks of chickens naturally infected with *M. ornithogaster*. In the first report, birds were stunted and prone to eat litter and pick at each other.[10] In the second report, stunting also was seen along with increased mortality and poor laying performance. All but one of the birds in the second study had significant concurrent diseases, making it difficult to know if *M. ornithogaster* acted as a primary or secondary pathogen.[21] Experimental infection with *M. ornithogaster* in white leghorn chickens did not result in clinical signs of disease. However, the feed conversion rate in infected birds was reduced compared to non-infected controls, suggesting that *M. ornithogaster* may have an important economic significance if introduced into poultry flocks.[15]

Detection

Macrorhabdus ornithogaster is a long, straight, narrow rod that is 3 to 4 μm wide and 20 to 80 μm long (Fig 30.2). It will occasionally branch, but this is rare (Fig 30.2). The longer organisms are actually chains of 2 to 4 cells, but the septations between cells are not readily observed. They are gram-positive, but many organisms will not pick up the stain or will only pick up the cytoplasm and not the thick cell wall, and therefore will stain faintly or not at all (Fig 30.3).

Similarly, *M. ornithogaster* stains poorly and variably with quick stains used for cytology.[22] Also, it has been the author's impression that the organism is easily washed off slides during the staining process.

Short of a proventricular scraping or flush, there is no definitive way to detect *M. ornithogaster* infections in the live bird. Many, probably most, sick birds with macrorhabdosis will shed large numbers of organisms in their droppings. These organisms are best observed by making an unstained wet mount of a dropping and examining it under the microscope at 100x and 400x magnifications. Reducing the diameter of the stage diaphragm will make the organisms easier to see (Fig 30.4). Shedding in birds that are not showing signs of illness is highly inconsistent. Examination of five or more droppings may be necessary to find even a few organisms, and in some birds shedding will not be detected at all. The opposite also is true, in that an occasional asymptomatic bird will shed large numbers of organisms. Additionally, fecal screening

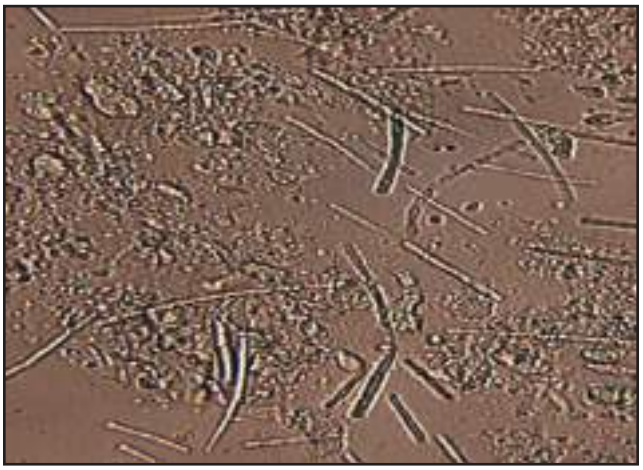


Fig 30.4 | Wet mount of a scraping from the gastric isthmus of a budgerigar with a heavy infection of *Macrorhabdus ornithogaster*. The aperture stage condenser was narrowed to increase contrast.

is complicated because feces contain debris that may be mistaken for *M. ornithogaster*.[14] *Macrorhabdus ornithogaster* stained with calcofluor white M2R is readily visualized with a fluorescent microscope with excitation barrier filters (380-420 nm) (see Fig 30.1). Unfortunately, this technique is not commonly available.[11]

Treatment Options and Interruption of the Infection Cycle

The only antimicrobial tested to date that was effective against *M. ornithogaster* is amphotericin B.[4,14] Treatment was effective only at a dose of 100 mg/kg by gavage twice a day for 30 days. A water-soluble preparation[a] when given for 14 days was not effective. A strain of *M. ornithogaster* resistant to amphotericin B has been identified in Australia.[14] It is not known how widespread resistance to amphotericin B may be. Initial reports in chickens suggested that fluconazole (100 mg/kg q 12 h) showed some promise for treating this organism. Subsequent testing showed that this dose was highly toxic to budgerigars and that mortality was seen even when the dose was reduced to 10 mg/kg q 12 h. A dose of 10 mg/kg q 24 h was less toxic but was not effective.[14] A single report suggested that nystatin was effective for treating *M. ornithogaster* in a small group of goldfinches.[4] Nystatin, however, was not effective in budgerigars in Australia.[7] Iodine preparations, lufenuron, ketoconazole, terbinafine or itraconazole also were not effective against *M. ornithogaster* in other trials.[14] *Macrorhabdus ornithogaster* shedding ceased when a *Lactobacillus* sp. was administered by gavage in treated budgerigars.[7] The birds were not necropsied, so it is not known if they were cured or just temporarily stopped shedding the organism. Previous reports suggesting that *M. ornithogaster* is susceptible to antibacterial antibiotics were erroneous, as the organism they tested was not *M. ornithogaster*.[18,20]

Evidence suggests there are mixed benefits to treating an entire flock of birds for *M. ornithogaster*. This would require that amphotericin B be given by gavage to every bird twice a day for 30 days. Additionally, it would require that the environment be extensively cleaned and disinfected; to date it is not known which disinfectants are effective against *M. ornithogaster*. With these constraints, flock treatment is not likely to result in a flock cure. Filippich, however, suggested that treatment did result in a significant reduction of birds that were shedding the organism.[6] Culling positive birds without treatment did not result in a reduction of shedding. However, it was suggested that culling positive birds after treatment might be of some benefit, as these birds may be infected with amphotericin-resistant strains.

An alternate approach to eliminating the infection from a flock is to incubator-hatch and hand-raise the young. Experimentally, it has been shown that if budgerigar eggs are pulled from the parents and cleaned, and the chicks are not allowed to have contact with the egg or infected birds after hatching, infection does not occur. Hand-feeding nestling day-old parrotlets and budgerigars and keeping them isolated from other birds is not an easy task, but may be one that a breeder is willing to do if this organism is a problem in a flock of valuable breeding birds.

Based on the virtual impossibility of treating large flocks for *M. ornithogaster*, the author recommends treatment of birds showing signs and selectively breeding birds that do not demonstrate signs of disease. Clinical experience suggests that some budgerigars may have a heritable resistance to infection (L. Filippich, personal communication, 1997).

It was thought that *M. ornithogaster* may cause a change in the pH of the stomach. While this seems very unlikely given that *M. ornithogaster* has little or no impact on the acid-secreting cells of the proventriculus, it has led some to speculate that administering agents that would acidify the stomach may be effective in its treatment. A controlled study testing this hypothesis failed to show efficacy of an orally administered acidifying agent.[4]

Findings at Necropsy

Gross necropsy findings are not specific. Birds are typically thin to emaciated and have little or no body fat. There may be ulceration of the ventriculus, proventricu-

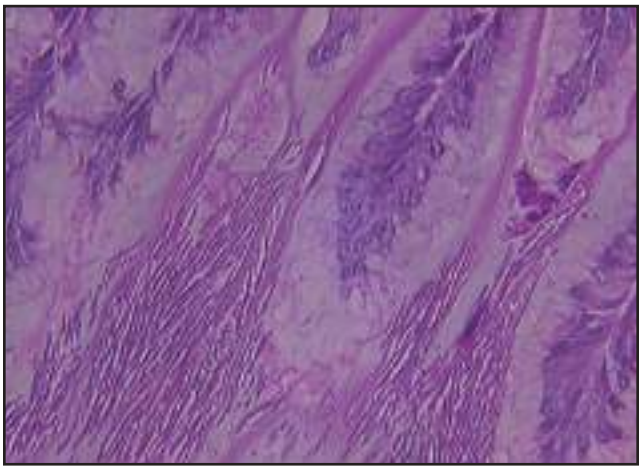


Fig 30.5 | Hematoxylin and eosin-stained section of the gastric isthmus, demonstrating the "log-jam" appearance of *Macrorhabdus ornithogaster* in situ.

lus or both. Most often the distal proventriculus and isthmus, the narrow zone of the stomach between the proventriculus and the ventriculus, is slightly dilated and has a thin wall. Perforation of the proventriculus is reported in some cases. Thick, opaque mucous secretions may cover the mucosa of the proventriculus and isthmus.[1,5,6] Microscopically, the organism is found on the surface of the glands of the isthmus and the transitional koilin. When present in large numbers, these organisms penetrate between the isthmus glands and invade the transitional koilin of the isthmus and the koilin of the ventriculus (Fig 30.5). When the organisms penetrate to the level of the isthmus glands, there is usually a significant accompanying disruption of the koilin. Additionally, isthmus glands appear to atrophy or undergo necrosis. Varying degrees of inflammation were observed in different studies. In the author's experience, inflammation in the budgerigar often is absent or minimal. If there is ulceration there will be a heterophilic response. It is normal for budgerigars, particularly nestling budgerigars, to have mild to moderate lymphoplasmacytic aggregates in the lamina propria of the proventriculus and isthmus. These findings should not be interpreted as a response to *M. ornithogaster* infection. It is possible, however, that these aggregates may become larger and more extensive in some infected birds. Baker found a significant mononuclear cell infiltrate in the mucosa of budgerigars with macrorhabdosis. Birds with chronic disease also showed evidence of goblet cell hypertrophy and fibrosis of the submucosa. Glandular cysts were seen occasionally.[1]

The microscopic lesions in infected chickens resemble those described by Baker in the budgerigar. The normal lymphoplasmacytic aggregates found in the lamina propria and submucosa are markedly expanded, resulting in a prominent widening of the folds of the glands of the isthmus.[12,15,21]

It is important to note that infections with *M. ornithogaster* are common and are often present in birds that die from other causes. Finding *M. ornithogaster* without evidence of koilin disruption and ulceration makes it unlikely that it was the cause of death.

There are reports that *M. ornithogaster* can be found in the intestinal tract and very rarely in other organs.[8] There are bacteria that can have a similar appearance to *M. ornithogaster*; therefore, if it is suspected that organisms outside the stomach are in fact *M. ornithogaster*, sections should be stained with a chitin-specific stain to prove that they are not bacteria.

Products Mentioned in the Text

a. Amphotericin-B, Megabac-S, Vetafarm, Wagga Wagga, Australia, www.vetafarm.com.au

References and Suggested Reading

1. Baker JR: Clinical and pathological aspects of "going light" in exhibition budgerigars (*Melopsittacus undulatus*). Vet Rec 116:406-408, 1985.
2. Davis A, Phalen DN: Sensitivity and specificity of wet-mount examination for the detection of budgerigars infected with *Macrorhabdus ornithogaster*. Forthcoming.
3. Dorrestein GM, Zwart P, Buitellaar MN: Problems arising from disease during the periods of breeding and rearing canaries and other aviary birds. Tijdschr Diergeneeskd 105:535-543, 1980.
4. Filippich LJ, Herdrikz JK: Prevalence of megabacteria in budgerigar colonies. Aust Vet J 76:92-95, 1998.
5. Filippich LJ, O'Boyle DA, Webb R, et al: Megabacteria in birds in Australia. Aust Vet Prac 23:72-76, 1993.
6. Filippich LJ, Parker MG: Megabacteriosis and proventricular/ventricular disease in psittacines and passerines. Proc Assoc Avian Vet, 1994, pp 287-293.
7. Filippich LJ, Perry RA: Drug trials against megabacteria in budgerigars (*Melopsittacus undulatus*). Aust Vet Pract 23:184-189, 1993.
8. Gerlach H: Megabacteriosis. Sem Avian Exotic Pet Med 10:12-19, 2001.
9. Huchzermeyer FW, Henton MM, Keffen RH: High mortality associated with megabacteriosis of proventriculus and gizzard in ostrich chicks. Vet Rec 133:143-144, 1993.
10. Lublin A: A five-year survey of megabacteriosis in birds of Israel and a biological control. Proc Assoc Avian Vet, 1998, pp 241-245.
11. Moore RP, Snowden KF, Phalen DN: A method of preventing transmission of so-called megabacteria in budgerigars (*Melopsittacus undulatus*). J Avian Med Surg 15:283-287, 2001.
12. Mutlu OF, et al: Proventriculitis in fowl caused by megabacteria. Tierärztl Prax 25:460-462, 1997.
13. Pennycott TW, et al: Causes of death of wild birds of the family Fringillidae in Britain. Vet Rec 143:155-158, 1998.
14. Phalen DN, Tomaszewski E, Davis A: Investigation into the detection, treatment, and pathogenicity of avian gastric yeast. Proc Assoc Avian Vet, 2002, pp 49-51.
15. Phalen DN, Moore R: Experimental infection of white-leghorn cockerels with *Macrorhabdus ornithogaster* (megabacterium). Apr-Jan 47(2) 254-260. Avian Dis, 2003.
16. Ravelhofer K, et al: Megabakteriosen bei verscheidenen vogelspezies. DVG Tag Vogelkr 9:95-104, 1998.
17. Ravelhofer-Rotheneder K, et al: Taxonomic classification of "megabacteria" isolates originating from budgerigars (*Melopsittacus undulatus*). Tierärztl Prax 28:415-420, 2000.
18. Rossi G: Histological and immunohistochemical findings in proventricular mucosa of chickens experimentally infected with "megabacterium." Proc Eur Soc Vet Path, 2000, p 156.
19. Rossi G: Possibility of infecting mammals with megabacteria isolated from birds. Vet Rec 147:371-371, 2000.
20. Scanlan CM, Graham DL: Characterization of a gram-positive bacterium from the proventriculus of budgerigars (*Melopsittacus undulatus*). Avian Dis 34:779-786, 1990.
21. Schulze C, Heidrich R: Megabacteria-associated proventriculitis in poultry in the state of Brandenburg, Germany. Dtsch Tierärztl Wschr 108:264-266, 2001.
22. Tomaszewski EK, et al: Phylogenetic analysis identifies the "megabacterium" of birds is a novel anamorphic ascomycetous yeast, *Macrorhabdus ornithogaster*, 534: 1201-1205. Int J Sys Evol Micro, 2003.
23. Van Herck H, et al: A bacterial proventriculitis of canaries. Avian Pathol 13:561-572, 1984.

CHAPTER
31

Implications of Toxic Substances in Clinical Disorders

JILL A. RICHARDSON, DVM

Greg J. Harrison

Fig 31.1 | A lutino cockatiel with excessive yellow feather pigmentation commonly seen in nutritional disorders. Helga Gerlach first proposed that consumption of foreign bodies is mediated by nutrient imbalances. Since that time, the use of formulated diets has become much more prevalent. Practitioners have empirically noted a concomitant and precipitous decline in foreign body ingestion.

Birds are curious in nature and certain dangerous objects may be attractive to them (Fig 31.1). As most pet birds have clipped wings, remain caged or have limited activity outside their cages, toxicosis are not common. However, birds with free household access or free-ranging birds are most at risk of becoming exposed to toxicants. With any potential toxicosis, proper and prompt treatment including stabilization and decontamination is essential. Supportive care is also a critical factor in the full recovery of the bird.

Assessing the condition of the bird is the initial step in managing potential toxicosis. This assessment should be performed quickly and in a manner that will limit stress to the bird. Sedation with isoflurane or sevoflurane gas anesthesia may be required to limit stress or when dealing with fractious birds. Toxic birds are prone to both active and passive regurgitation, and care must be taken to prevent aspiration. To help decrease stress, birds should be examined and maintained in a quiet, warm environment.[1] The assessment should include an evaluation of the respiratory rate and effort, capillary refill time and general attitude (see Chapter 6, Maximizing Information from the Physical Examination). The examination of a bird that is unconscious, in shock, seizuring or in cardiovascular or respiratory distress must be conducted simultaneously with stabilization measures (see Chapter 7, Emergency and Critical Care).

Stabilizing the bird is a life-saving priority.[1] Once the animal is stabilized, a comprehensive history of the bird including exposure history can be obtained. Common presentations of birds that require stabilization include dyspnea, cyanosis, severe depression, emaciation, severe diarrhea, seizures and hemorrhage.[1] Minimal handling is imperative with dyspneic birds in order to decrease


Gwen Flinchum

Fig 31.2 | Fluids being given subcutaneously to a toxic duck.


Greg J. Harrison

Fig 31.3 | Lavaging the digestive system with warmed saline via a crop tube. If lavage is contraindicated, the liquids can be used to dilute toxins and/or aspirate fluids in the crop that may contain toxins

oxygen requirements. Dyspneic birds should be placed in a cage supplemented with oxygen before and during examination.[1] A diuretic may be indicated with the presence of pulmonary edema.[1]

Anti-convulsant therapy such as diazepam at 0.6 mg/kg IM should be given if the bird is seizuring.[7] Intravenous, intraosseous or subcutaneous fluids may be needed if the bird is severely dehydrated (Fig 31.2).

Decontamination

Preventing absorption of the substance is an important step in treating a toxicosis.

DERMAL EXPOSURES

With light dermal exposures, the bird can be gently spritzed with a solution of mild liquid dishwashing detergent and warm water, softly rubbed and then spritzed with plain warm water to remove soap. This process can be repeated as needed, making sure all soap is removed.

A thorough bathing may be indicated with heavy exposures. Following the bath, the bird should be lightly patted dry, kept warm and monitored for signs of hypothermia. Removal of the toxicants from the feathers is contraindicated if the bird is seriously ill; always stabilize the patient first. With corrosive or irritating substances, the bird's skin should be monitored for redness, swelling or pain.

OCULAR EXPOSURES

With ocular exposures, the bird's eyes should be gently flushed with tepid tap water or with physiologic saline. The use of an eyedropper to gently administer the flush is recommended in small birds. Fluorescein staining and follow-up examinations are warranted with exposures to corrosive agents or if clinical signs of redness, pain or ocular discharge occur.

ORAL EXPOSURES

Dilution with milk or water in combination with demulcents is recommended in cases of corrosive ingestion. Close monitoring is recommended following ingestion of corrosive agents, which can lead to tissue necrosis and inflammation of the mouth, esophagus and crop. Severity of injury depends on the concentration and duration of contact.

Never induce emesis in a bird. Emesis is considered unsafe in birds, due to the potential of aspiration and ineffectiveness of emetic medications.[10] Crop lavage may be considered with recent ingestion of toxicants (Fig 31.3). Contraindications to performing a crop lavage include ingestion of corrosive substances or petroleum distillates. With ingestion of corrosive agents, gastric lavage is not recommended.[3,4] Instead, oral dilution with milk or water is preferred.[3,4] Dilution is most effective if it is performed early. Sedation is recommended for frightened or fractious birds. Isoflurane or sevoflurane gases are the optimal anesthetic agents, and an endotracheal tube should be inserted during the process to prevent aspiration. To lavage the crop, body-temperature saline is gently flushed into the crop and aspirated repeatedly (3-4 times).[10]

Activated charcoal is considered a non-specific adsorbent that binds to many substances through weak forces, and prevents their systemic absorption. It is not an effective adsorbent for corrosive substances, petroleum distillates or heavy metals.[3,4,6,15] Activated charcoal can be given to birds with a dosing syringe, an eyedropper or lavage tube, although extreme caution must be used to avoid aspiration. Dosage of activated charcoal in most species

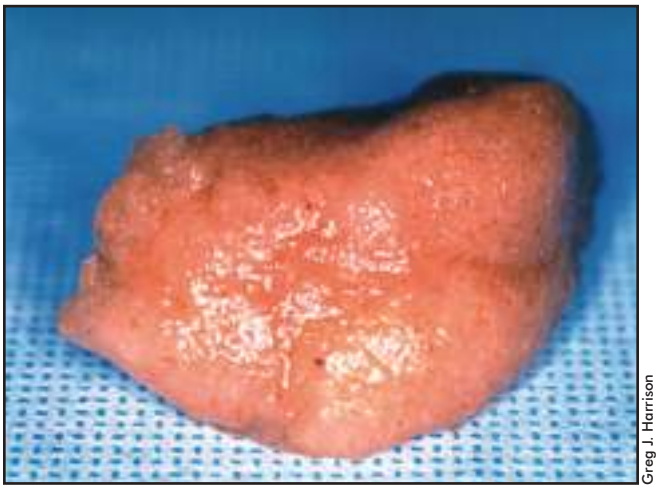

Greg J. Harrison

Fig 31.4 | Psyllium is high in mucopolysaccharides and forms a slick mass that can sweep the digestive tract free of a multitude of toxins. Using more than 2% concentration can lead to gastrointestinal blockage by this hydroscopic mass. Given with balanced electrolytes and dextrose it will simultaneously act to rehydrate the patient.


Greg J. Harrison

Fig 31.5 | Inexpensive pet carriers are often made from hardware cloth. This metal wire is always toxic to chewers as it is high in lead and zinc. This 27-year-old Amazon traveled safely in such a carrier its entire life. The owner was lucky this did not kill the bird.

is 1 g/kg (or 1-3 mg/g body weight).[3,4]

Cathartics are substances that enhance the elimination of activated charcoal, but should be used cautiously in birds. Cathartics can be added to solutions of activated charcoal, or premixed combinations are available. Never use cathartics when the bird is dehydrated. Bulking agents can be useful in removing small solid objects from the bird's gastrointestinal tract, such as lead paint chips. One half teaspoon of psyllium (Fig 31.4) mixed with 60 cc baby food gruel has been suggested as a bulking agent for birds, and can be administered with a dosing syringe or eyedropper.[10] Use extreme caution to avoid aspiration. The mixture may be repeated to ensure complete removal of the objects. Peanut butter also has been recommended as a bulking agent.[10]

The bird should be monitored closely during treatment. Routinely evaluate vital signs and the parameters most likely to be affected. Preventive measures such as gastric protection or antibiotics may be needed. Additional measures such as nutritional and hydration support are key components for full recovery. Daily maintenance fluid requirements in most birds are 50 ml/kg per day.[1] It is extremely important to maintain the bird's nutritional requirements. Hospitalized birds eating voluntarily can be fed their normal diet. Tube-feed ill birds that are not eating, unless vomiting or delayed emptying of the crop is present.[1] Good nursing care should be given until the bird completely recovers. The propensity for hypothermia in a bird that is ill for any reason should be considered and external heat and humidity provided as required.

Common Hazards

HEAVY METALS

Zinc

Sources of zinc include hardware such as wire (Fig 31.5), screws, bolts, nuts, and USA pennies. Pennies minted since 1983 contain 99.2% zinc and 0.8% copper and one penny contains approximately 2440 mg of elemental zinc.[15] The process of galvanization involves the coating of wire or other material with a zinc-based compound to prevent rust. Owners often are not aware of galvanization on the wire used for making cages (Fig 31.6). Food and water dishes also may be galvanized and sufficient zinc may leak into the water or food to create toxicity.

(Ed. Note: In tracking cases of zinc toxicity over 7 years, computer records indicate that in 82 cases of zinc toxicity, approximately half of the clients denied any potential exposure of their birds to zinc or other heavy metal. The idea that a bird out of its cage is truly "supervised" is overrated. Also, powder-coated cages made outside of the USA, especially in China, have been known to use zinc to expedite setting of the powder coating [F. VanSant, personal communication, 2000]).

Although the exact toxicologic mechanisms of zinc in birds or other animals is not known, zinc toxicosis can affect the renal, hepatic and the hematopoietic tissues. Clinical signs of zinc toxicosis in birds may include polyuria, polydipsia, diarrhea, weight loss, weakness, anemia, cyanosis, seizures and death.[2,16] An underreported clinical sign of zinc toxicity is polydipsia with

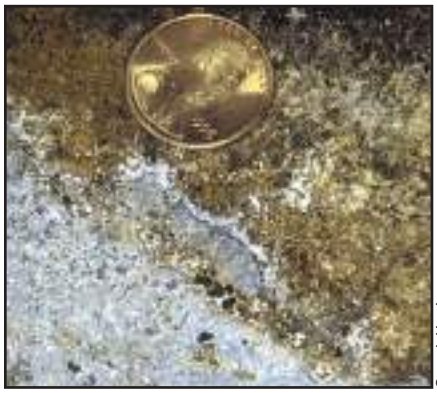

Greg J. Harrison

Fig 31.6 | Two sources of zinc: a USA 1-cent coin and a galvanized metal cage tray. The zinc in the tray has oxidized to its most toxic form — a pure white powder — evident at the margin of the rust and the galvanization.


Greg J. Harrison

Fig 31.7 | Placing a bird unanesthetized in a paper bag is often a safe and accurate way to diagnose metal toxicosis.


Greg J. Harrison

Fig 31.8 | Lead used to balance automobile wheels and fishing weights are modern sources of lead toxicosis.

passive regurgitation of water when the bird is handled. Mild anemia also is frequently encountered. Concurrent marked elevations in the total WBC are noted in the majority of cases of heavy metal toxicosis. Whether this is a true infection or an inflammatory response is not documented, so treatment with antibiotics will depend on the practitioner's evaluation. However, the presence of a high WBC should not convince the practitioner that the discovery of metal in the ventriculus is not the primary etiology of illness.

Diagnosis

Radiography of the abdomen may reveal the presence of metallic objects in the gastrointestinal tract. This need not be a properly positioned radiograph, but rather can be done as a stress-free scan for the presence of metal in the ventriculus (see Chapter 1, Clinical Practice for "bag rad" scan) (Fig 31.7). Serum zinc levels may be obtained using blood collected from plastic syringes (no rubber grommets) and stored in royal blue-top vacutainers or directly into vacutainers with appropriate needle to minimize contamination with exogenous zinc.[15] In general, blood zinc levels of >200 μg/dl (2 ppm) are considered to be diagnostic.[16] The pancreas is considered to be the best tissue for postmortem zinc analysis.[2,16] Pancreatic tissue zinc levels greater than 1000 μg/g are suggestive of a zinc toxicosis.[16]

Treatment

It is imperative to remove the sources of zinc from the gastrointestinal tract. Removal of zinc-containing foreign bodies via endoscopy or proventriculotomy/enterotomy may be required. The success of the removal process can be assessed with radiographs. Since most zinc items swallowed by pet birds are galvanized iron, the use of magnets attached to an enteral tube is an effective means of removing ferrous items that are zinc coated[12] (see also Chapter 14, Evaluating and Treating the Gastrointestinal System). Activated charcoal is not indicated, as it is of little benefit in binding zinc.[15] Bulk cathartics, psyllium (sodium sulfate 125-250 mg/kg), peanut butter, mineral oil and corn oil may aid in the removal of zinc objects from the GI tract. The use of chelators may not be necessary in cases where prompt removal of the zinc source is accomplished. If chelation therapy is instituted, careful monitoring of renal parameters is important for the duration of therapy. Elevated uric acids in heavy metal poisoning and a decrease with therapy have been reported (E. Odberg, personal communication, 2001). The following chelating agents have been suggested for zinc poisoning in birds: Ca EDTA 35 mg/kg BID, IM for 5 days.[7] If needed, the second course of therapy is given after a 5- to 7-day waiting period. If/when the bird is able to tolerate oral medication, D-penicillamine (Cuprimine) can be administered orally at 55 mg/kg BID PO for 1 week.[7] A second course of 1-week therapy can be given, if needed, after a 1-week rest. Succimer, (2, 3 dimercaptosuccinic acid) at 25 to 35 mg/kg for 5 days per week for 3 to 5 weeks also has been used to treat zinc toxicosis in birds.[7] In addition, treatment for symptomatic animals should include blood replacement therapy as needed, parenteral fluids and good nursing care such as force-feeding or hand-feeding.

Lead

Sources of lead include paint, toys, drapery weights, linoleum, batteries, plumbing materials, galvanized wire, solder, stained glass, fishing sinkers (Fig 31.8), lead shot, foil from champagne bottles and improperly glazed bowls.[4,16] Lead is considered to be the most commonly reported of avian toxicosis with acute toxicities more common in captive birds and chronic in free-ranging birds (Fig 31.9).


Greg J. Harrison

Fig 31.9 | Pelicans are presented with fishing lines and hooks swallowed or tangled in their extremities. Radiographs for lead are always indicated to ensure a bird with a lead weight in its digestive system is not released.


Greg J. Harrison

Fig 31.10 | Peregrine falcons are carnivorous and can consume prey that were shot with lead-containing pellets. Radiographs are indicated.


Greg J. Harrison

Fig 31.11 | Blue-winged teal and other water birds can dabble in tidal marshes or ponds that may contain decades-old hunting remnants from lead shot.

Raptors can ingest lead shot from preying on animals that have been shot with or have ingested lead shot (Fig 31.10).[20] Lead toxicosis also has been documented in an Amazon parrot that had been fed portions of game birds that contained lead shot.[18] Between 1983 and 1986, the National Wildlife Health Center examined 1041 moribund or dead waterfowl and diagnosed lead poisoning in approximately 40% (Fig 31.11).[5] Although lead shot has since been banned for hunting waterfowl, spent shot is still present in waterways.[20] Ingestion of 1 to 3 lead shotgun pellets has been reported to be lethal to waterfowl.[4]

Lead affects multiple tissues, especially the gastrointestinal tract, renal and nervous systems. Lead combines with erythrocytes in circulating blood, increasing RBC fragility, anemia and capillary damage. It also can cause segmental demyelination of neurons and necrosis of renal tubular epithelium, GI tract mucosa and liver parenchyma. Clinical signs seen in psittacine birds are often vague and may include lethargy, weakness, anorexia, regurgitation, polyuria, ataxia, circling and convulsions.[4] In some species such as Amazons, hemoglobinuria also may be noted.[11]

Diagnosis

Radiography of the abdomen may reveal evidence of metal in the ventriculus. Blood levels of lead are helpful in confirming lead toxicosis in birds with suspicious radiographic changes.[19] Whole blood levels greater than 0.6 ppm are viewed as diagnostic for lead toxicosis when accompanied by appropriate clinical signs.[19] The basophilic stippling and cytoplasmic vacuolization of red blood cells are not always seen with lead poisoning in avian species.[19]

Treatment

Removal of lead particles via bulk diet therapy, endoscopy or surgery is recommended. Succimer and Ca EDTA are both considered to be effective chelating agents in avian species.[4] Succimer has been reported to decrease blood lead concentration by 87% when given at a dose of 30 mg/kg PO BID for 7 days minimum, with no apparent adverse secondary effects, however, a dose of 80 mg/kg resulted in death.[9] The therapeutic dose of succimer in pet birds is 25 to 35 mg/kg PO BID 5 days a week for 3 to 5 weeks.[7] Calcium EDTA is considered the preferred initial chelator for lead toxicity in birds and is given at a dose of 35 mg/kg BID, IM for 5 days, off 3 to 4 days, and repeated if needed.[7] Fluid therapy is recommended to prevent renal effects from Ca EDTA during treatment.[11] Penicillamine and diethylene triamine pentaacetic acid (DTPA) have also been used to treat avian lead toxicosis.[7]

Since lead can be immunosuppressive, broad-spectrum antibiotics may be indicated.[4] In addition, good supportive care including seizure control is recommended until full recovery.

Nicotine Products

Tobacco products contain varying amounts of nicotine (Table 31.1), with cigarettes containing 3 to 30 mg and cigars containing 15 to 40 mg.[15] Butts contain about 25%

Table 31.1 | Nicotine Content of Common Sources of Nicotine

Nicotine Product	Nicotine Content
Cigarettes	3-30 mg per 1 whole cigarette
Cigarette butts	.75-7.5 mg
Cigars	15-40 mg
Moist snuff	4.6-32 mg/g
Dry snuff	12.4-15.6 mg/g
Chewing tobacco	2.5-8.0 mg/g
Nicotine gum	2-4 mg per piece
Transdermal patches	15-114 mg per patch
Nicotine nasal sprays	10 mg per ml
Nicotine inhaler rods	10 mg per cartridge

of the total nicotine content. Nicotine also is found as a natural form of insecticide. Signs develop quickly in most species, usually within 15 to 45 minutes, and include excitation, tachypnea, salivation and emesis. Muscle weakness, twitching, depression, tachycardia, dyspnea, collapse, coma and cardiac arrest may follow. Death from nicotine toxicosis occurs secondary to respiratory paralysis.[15] A less serious but common response to cigarette smoke deposition on the feathers is feather-destructive behavior.

(Ed. Note: One timneh grey that expired at 21 years of age reportedly had lived its entire life with a heavy smoker. The histopathologic diagnosis of multiple masses in the lungs was carcinoma, but was not definitely labeled as bronchiogenic).

Inhalants

The avian respiratory tract is extremely sensitive to inhalants. Any strong odor or smoke could potentially be toxic (Table 31.2).[17] Polytetrafluoroethylene (PTFE)-coated cookware or cooking utensils can emit toxic fumes when overheated (>280° F).[17] Clinical signs may include acute death, rales, dyspnea, ataxia, depression and restless behavior.[2,10] Hemorrhage and edema in pulmonary tissues leads to respiratory failure and death. Prognosis is usually guarded to poor. Treatment for inhalation toxicosis includes the administration of oxygen, rapidly acting corticosteroids, diuretics, analgesics, parenteral antibiotics and topical ophthalmic antibiotic ointment.[1] A bronchodilator may be needed for bronchospasms[1] (see Chapter 7, Emergency and Critical Care for an updated therapy). In most cases, prognosis is guarded to poor.*

Table 31.2 | Examples of Noxious Inhalants

- Some non-stick surfaces (pots, pans, cookware, irons, ironing boards)
- Heating elements on reverse cycle air conditioners
- Gasoline or other volatile gas fumes
- Any source of smoke
- Automobile exhaust
- Carbon monoxide
- Self-cleaning ovens and drip pans for ranges
- Aerosol sprays
- Cleaning products such as ammonia or bleach
- Paint fumes
- Fumigants
- Candles with lead wicks, scented plug-in items

**Ed. Note: The first-time heating of several new non-stick pans is a frequent finding with PTFE toxicosis. One empirical report (Beckett, personal communication, 2001) had a bird indirectly exposed days later when a wooden perch had been "sterilized" on a PTFE-coated metal cooking sheet and the perch was then later placed in the bird's cage.*

AVOCADO (*Persea americana*)

The toxic principle in avocado is persin, and leaves, fruit, bark and seeds of the avocado have been reported to be toxic to birds and various other species.[10,15,17] Several varieties of avocado are available, but not all varieties appear to be equally toxic. In birds, clinical effects seen with avocado toxicosis include respiratory distress, generalized congestion, hydropericardium, anasarca and death.[10,17] Onset of clinical signs usually occurs after 12 hours of the ingestion, with death occurring within 1 to 2 days of the time of exposure.[10] Small birds such as canaries and budgies are considered to be more susceptible, however, clinical signs have been observed in other species. Treatment for recent avocado ingestion includes decontamination via crop lavage and activated charcoal; bulking diets may help prevent absorption. Close monitoring for cardiovascular and pulmonary signs should follow. With symptomatic animals, treatment with humidified oxygen and minimal handling may be required. Diuretics may be helpful in cases with pulmonary edema.[4]

POISONOUS PLANTS

The following is a partial list of plants that have been shown to cause toxicity in small animals. The severity of signs or toxicity of these plants in birds has not been thoroughly studied.

Potentially Cardiotoxic Plants

- Lily of the valley (*Convallaria majalis*)
- Oleander (*Nerium oleander*)
- Rhododendron species
- Japanese, American, English and Western yew (*Taxus* spp.)
- Foxglove (*Digitalis purpurea*)
- Kalanchoe species
- Kalmia species

Plants That Could Cause Kidney Failure

- Rhubarb (*Rheum* spp.) - leaves only

Plants That Could Cause Liver Failure

- Cycad, Sago, Zamia palms (*Cycad* spp.)
- *Amanita* mushrooms

Plants That Can Cause Multisystem Effects

- Autumn crocus (*Colchicum* sp.)
- Castor bean (*Ricinus* sp.)

Plants Containing Calcium Oxalate Crystals

Peace lilies, Calla lilies, philodendrons, dumb cane,


Greg J. Harrison

Fig 31.12 | Houseplants need to be considered as potential toxicants, especially in birds with livers stressed by nutritional disorders.


Greg J. Harrison

Fig 31.13 | A poinsettia plant growing wild in the rare species breeding aviary in Tenerife, Spain.


Greg J. Harrison

Fig 31.14 | A lantana plant in the same aviary as in Fig 31.13.


Greg J. Harrison

Fig 31.15 | A budgie has been oiled by an ill-informed owner.

mother-in-law, and Pothos plants contain insoluble calcium oxalate crystals. These crystals can cause mechanical irritation of the oral cavity and tongue of birds when plant material is ingested. Clinical signs usually include regurgitation, oral pain, dysphagia and anorexia. The signs are rarely severe and usually respond to supportive care.

- Peace lilies (*Spathiphyllum* spp.)
- Calla lily (*Zantedeschia aethiopica*)
- Philodendron (*Philodendron* sp.)
- Dumb cane (*Dieffenbachia* sp.)
- Mother-in-law plant (*Monstera* sp.)
- Pothos (*Epipremnum* sp.)

(Ed. Note: A common presentation in cockatiels appears to be oral irritation from ingestion of small amounts of Pothos or philodendron species of plants (Fig 31.12)*. In this editor's practice, more than 15 cockatiels have presented over the course of 20 years with documentation of recent chewing on leaves of these plants and almost immediate production of clinical signs. The birds appear acutely depressed and anorectic, but still in good body weight. Examination of the tongue will reveal pronounced erythema, sometimes with obvious ulceration, and hypersalivation. Supportive care for 24 to 48 hours has resulted in 100% recovery. Lack of mortality also has lead to a lack of histopathology, so any additional toxic effects other than oral irritation have not been documented [TTL]).*

Aviculturists need to be sure potentially toxic plants are avoided in the plantings of the aviary (Figs 31.13, 31.14).

OIL-CONTAMINATED BIRDS

Oil spills are not an uncommon problem for aquatic species of birds. According to California's Oiled Wildlife Care Network, bird survival is dependent upon many factors, including the speed of recovery and the species' susceptibility to toxicity and captive stress.[13] The first step when treating oiled birds is to stabilize the animal and provide a warm (approximately 27° C) and stress-free environment.[13] Common presenting clinical signs include respiratory distress and seizures.[13] Following initial stabilization, a thorough exam should be performed. Most affected birds are hypothermic, hypoglycemic, hypoproteinemic and lethargic on presentation.[13] Anemia also has been reported.[13,14] Symptomatic care including nutritional support should be provided as needed.


Greg J. Harrison

Fig 31.16 | When presented to a rehabilitation center, an insectivorous raptor, the burrowing owl, should have pesticide toxicity investigated to document this common but seldom proven condition.


Greg J. Harrison

Fig 31.17 | The insectivorous passerine, such as this painted bunting, is seldom seen when ill. When presented for rehabilitation, pesticide toxicity should be investigated.

With oil contamination, feathers lose the ability to insulate, which can result in hypothermia[1] (Fig 31.15). Oil also can interfere with the animal's buoyancy.[13] Oil can be removed from the feathers once the animal is stable using dishwashing detergent in warm baths. The temperature of water used should be 106° F, and water should be softened to 2 to 3 grains of hardness to help completely remove oil and prevent mineral crystallization in the feathers.[13] Following thorough rinsing, the bird must be placed in a warm environment and allowed to dry. Multiple baths may be needed, however, repeat washings because of incomplete oil or soap removal are associated with increased mortality.[13] Other recommendations for care include the use of lactulose at 0.3 ml/kg PO q 12 h, papaya enzymes, 1 tablet PO q 12 h, aggressive fluid therapy for feather-eating species and warm-water exercise pools.[13]

Editor's Comments

While documentation of environmental toxins is hard to prove as the cause of death in wild birds, one must strongly suspect pesticides when seeing insectivorous birds like burrowing owls (Fig 31.16), the painted bunting (Fig 31.17) and related birds presented to rehabilitation centers with clinical signs consistent with toxicity. When the literature on pesticides is studied (see reference 9 in Chapter 11, Low-Risk Pest Management), it seems rather obvious the veterinary profession is commonly missing pesticide toxicosis.

In 1999, a massive die-off of white pelicans (*Pelecannus erythrorhynchos*), wood storks (*Mycteria americana*), great egrets (*Ardea albus*) and great herons (*Ardea herodias*) occurred on the shore of Lake Apopka in Central Florida. The University of Florida and USA Fish and Wildlife eventually detected the chemicals DDT, toxaphene and dieldrin in lethal levels in these fish-eating birds. These pesticides are believed to be carcinogenic and were banned in the 1970s and 1980s; however, the chemicals can persist for decades in soil and animal tissue.

It was the nation's worst pesticide poisoning in decades. Over 800 documented great white pelicans were killed by this toxic exposure, and the extrapolated number of dead created great concern for the survival of this species in North America. However, political and legal concerns kept the problem from being widely publicized.

Pesticides have tremendous residual potential. Their prolonged half-lives, combined with the general lack of both infrastructure and funding for testing and detection of pollutants, make it likely that a great deal more exposures to toxic pesticides will occur in many species than is suspected or reported.

As veterinarians, we have an opportunity to be cognizant of this dangerous potential, to appropriately diagnose and treat these toxicities in individual birds, and also to report suspected toxicities to responsive authorities, requesting and expecting an appropriate investigative response.

References and Suggested Reading

1. Agnes AE: Critical Care of Pet Birds: Procedures, Therapeutics, and Patient Support. *In* Veterinary Clinics of North America, Exotic Animal Practice. Philadelphia, WB Saunders Co, 1:1 1998, pp 11-42.
2. Bauck L, LaBonde J: Toxic diseases. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 604-613.
3. Beasley VR., Dorman D: Management of toxicosis. Vet Clin North Amer 20:2, 1990, pp 307-338.
4. Beasley VR, et al: A Systems Affected Approach to Veterinary Toxicology. Urbana, IL, University of Illinois Press, 1999, pp 27-69.
5. Beyer WN, et al: Retrospective study of the diagnostic criteria in a lead-poising survey of waterfowl. Arch Environ Contam Toxicol, 35(3):506-512, 1998.
6. Buck WB, Bratich PM: Activated charcoal: Preventing unnecessary death by poisoning. Food Animal Practice. Veterinary Medicine January 1986:73-77.
7. Carpenter JW, Mashima TY, Rupiper DJ: Exotic Animal Formulary 2nd ed. Philadelphia, WB Saunders Co, 2001.
8. Dumonceaux G, Harrison G: Toxins. *In* Avian Medicine: Principles and Application. Brentwood, TN, HBD International Inc, 1999, pp 1030-1052.
9. Hoogesteijn AL, et al: Oral treatment of avian lead intoxication with meso-2,3-dimercaptosuccinic acid. J Zoo Wildl Med 34(1):82-87, 2003.
10. LaBonde J: Toxicity in Pet Avian Patients. Semin Avian Exotic Pet Med 4(1):23-31, 1995.
11. Lightfoot TL: Common Avian Medicine Presentations II. Proceeding Notes. Western Veterinary Conference 2002.
12. Lumeij JT: Gastroenterology. *In* Avian Medicine: Principles and Application. Brentwood, TN, HBD International Inc, 1999, p 503.
13. Mazet JAK, et al: Advances in Oiled Bird Emergency Medicine and Management. J Avian Med Surg 16(2):146-149, 2002.
14. Newman SH, et al. Haematological changes and anaemia associated with captivity and petroleum exposure in seabirds. Compar Haematol Int 9:60-67, 1999.
15. POISINDEX editorial staff: (Toxicologic Management: Nicotine, Zinc, Avocado). *In* Rumack BH, et al (eds): POISINDEX System vol 100. Englewood, CO, Micromedex, 2003.
16. Pushner B, St. Leger J, Galey FD: Normal and toxic zinc concentration in serum/plasma and liver of psittacines with respect to genus differences. J Vet Diagn Invest 11(6):522-527, 1999.
17. Richardson JA, et al: Managing Pet Bird Toxicosis. Exotic DVM 3:1 2001, pp 23-27.
18. Riggs SM, Puschner B, Tell LA: Management of ingested lead foreign body in an Amazon parrot. Vet Human Toxicol 44(6):345-348, 2002.
19. Ritchie BW: Diagnosis, Management, and Prevention of Common Gastrointestinal Disease. Atlantic Coast Veterinary Conference 2002.
20. Shimmel L, Snell K: Case Studies in Poisoning- Two Eagles. Semin Avian Exotic Pet Med 8(1):12-20, 1999.

CHAPTER
32

Implications of Viruses in Clinical Disorders

DAVID N. PHALEN, DVM, PhD, Dipl ABVP

The past 15 years have seen remarkable growth in the understanding of the viral diseases of companion and aviculture birds. Molecular-based and traditional investigative and diagnostic tools allowed scientists to discover and understand the biology of many of the viruses that cause the common diseases seen in these birds. This information resulted in the development of management strategies that, when implemented, mitigate or completely eliminate the risk of several viral diseases. Unfortunately, we still lack critical information on a number of viral diseases and diseases thought to be caused by viruses. Additionally, not all bird owners are aware that they should apply what has been learned, and many are unwilling to do so. Therefore, viral diseases are still a significant threat to captive populations of birds.

Diagnostic Assays Used to Detect Viral Infections

SEROLOGY IN THE LIVE BIRD

Historically, serologic assays were used to screen large groups of animals to determine if a disease agent was present in the flock or herd in question. If a significant number of animals tested positive, then there was sufficient proof that the infectious agent was present and management changes were made accordingly. We ask much more from serology in pet bird and aviary medicine. We ask that each assay applied to a single sample tell us if the bird is or is not currently infected with whatever agent we are interested in; unfortunately, this is often not possible. Birds in the early stages of infection may not have had time to develop antibody. Assays that detect immunoglobulin M (IgM) will not become

positive for 7 to 14 days after infection, while assays that detect immunoglobulin Y (IgY) may take an additional 7 days before they are positive. The other major limitation of serology is that many birds remain seropositive after they are no longer infected with the virus and, without proper knowledge, a practitioner or bird owner could be misled into believing that a serologically positive bird was actively shedding virus.

Sadly, it must be noted that serologic and other diagnostic assays for avian infectious diseases have been and are still being offered commercially that are meaningless or their meaning is not known. For a diagnostic assay for any infectious disease to be valid, it must be tested rigorously in controlled infection trials or by the compilation and careful analysis of clinical data obtained from naturally infected animals. The accuracy of assays that have not been presented and preferably published in a peer-reviewed journal is suspect until proven otherwise.

Enzyme-linked Immunoassay (ELISA)

The ELISA detects antibodies from test plasma that react with viral antigens. To do this, the assay depends on a specific secondary antibody that can recognize the antibody of the bird being tested. If a single species is being tested and a secondary antibody to that species is available, the ELISA is an excellent assay. Cross-reactivity between secondary antibodies made to the antibody of one species of bird and the antibodies of other birds, however, will vary. Therefore, an ELISA using anti-Amazon parrot antibody may work for all species of Amazons and with careful controls may be applied to all species of parrots. It is, however, not likely to work in divergent species, such as passerines or ducks.

Hemagglutination Inhibition Assay (HI)

Several avian viruses, including the psittacine beak and feather disease virus, avian influenza and the paramyxoviruses, when added to red blood cells of the appropriate species will cause them to agglutinate. If the viruses are mixed with diluted serum containing antibodies to that virus, hemagglutination may be inhibited. HI can then be used to detect and quantitate circulating antibody. This assay is highly sensitive; however, non-specific hemagglutins and hemagglutin inhibitors occur in the serum of birds, complicating this assay. At times this assay may prove cumbersome, as some viruses will agglutinate the cells of some species and not others. If the necessary species are rare, this assay becomes impractical. Evidence suggests that the HI may not detect antibody in birds chronically infected with paramyxovirus 3.

Virus Neutralization Assay (VN)

The VN is a very sensitive and specific assay and can detect both IgM and IgY as long as they are neutralizing. In this assay, serum or plasma is diluted and each dilution is incubated with a specific concentration of live virus. The virus-serum mixture is then incubated with cells that are susceptible to infection. The cells are monitored for several days. If antibody is present in the serum and neutralizes the virus, cytopathic effects (CPE) to the cells are prevented. If the serum does not contain antibody, CPE occurs at all dilutions of serum. The antibody titer is defined as the reciprocal of the highest serum dilution that results in a 50% or 100% reduction in CPE.

The biggest disadvantages of the VN are that it requires that live cells be available and the assay itself takes 3 to 5 days. As a result, most laboratories will do this assay only once a week, and the turnaround time may be as long as 2 weeks.

POLYMERASE CHAIN REACTION (PCR) IN THE LIVE BIRD

PCR has been the single most important advancement in the detection of subclinical virus infection of birds. PCR detects viruses by amplifying a portion of the viral DNA, or viral RNA converted to DNA, to detectable levels. Blood, oral and cloacal swabs, tissue swabs and even environmental swabs can be used in this assay. Which samples need to be examined for each virus will depend on the virus and the stage of infection at the time of sampling. Development of a PCR assay requires knowledge of the DNA or RNA sequence of the virus to be detected. It also is necessary to know the variations in the sequences of the specific viruses. If there is considerable genetic variability but little biological variation within a virus, it may be critical to develop an assay that can detect all of the viruses. On the other hand, if significant biological variation is correlated with genetic differences, it may be important to develop multiple PCR assays that can differentiate among the genetic variants.

PCR assays are highly sensitive, but not all PCR assays are equally sensitive. When screening birds for avian polyomavirus, psittacid herpesviruses and the psittacine beak and feather disease virus, it is important to use PCR assays that have the highest level of sensitivity. The most sensitive assays typically use a nested or semi-nested PCR reaction that produces labeled amplification products that can be detected with an automated system. The sensitivity of the PCR assay also can be a disadvantage. Contamination of the sample at the time of collection or at the laboratory can result in false-positive results. It takes contamination with only one infected cell to cause a sample to become positive. Contamination is much more likely to occur when multiple birds are sampled. This technology is rapidly advancing, and it is certain

that the ability to screen for many more diseases soon will be available. Likewise, the cost and convenience of these assays will greatly improve.

POSTMORTEM DIAGNOSTICS FOR VIRAL INFECTIONS

Many viruses leave a characteristic histologic pattern of disease in their victims. Therefore, if the whole bird or a complete set of fixed tissues is submitted to the pathologist, a diagnosis can often be made based on the presence of specific histologic lesions, such as patterns of necrosis, the inflammatory response and the presence of viral inclusion bodies. When inclusions cannot be found or the inclusions are not specific, macerated fresh tissue and even formalin-fixed tissue can be examined for virus particles using electron microscopy. Virus isolation sometimes is necessary to detect specific viruses. Viruses can be isolated in embryonated chicken eggs and in primary cell cultures. Virus isolation often requires multiple blind passages before the virus is detected, and the whole process may take one to several weeks. Not all viruses grow readily in eggs or cell culture, so a negative finding does not conclusively rule out the possibility of a viral infection.

Molecular-based diagnostic assays have greatly improved the pathologist's ability to detect infectious agents in necropsy specimens. Fresh tissues from birds that die with viral infections typically contain high concentrations of virus. This virus is readily detected by PCR, if an assay for that virus is available. Formalin-fixed tissues also can be examined for virus DNA. However, formalin degrades DNA into small pieces; therefore, it is best to screen tissues that have been fixed for only a short time or have been fixed and then imbedded in paraffin within 2 or 3 days. Selecting PCR primers that amplify a short segment of the viral DNA also will increase the chance of detecting the viral DNA in formalin-fixed tissues. In situ hybridization and in situ PCR are techniques where the viral DNA actually can be detected in thin sections of formalin-fixed tissues. These assays have only limited availability and have a reduced sensitivity as compared to PCR of fresh tissue; however, they have important applications under some circumstances.

DNA Viruses

CIRCOVIRUS: PSITTACINE BEAK AND FEATHER DISEASE VIRUS (PBFDV)

Applied Biology

PBFDV is a non-enveloped DNA virus. Its single-stranded genome appears to code for seven proteins. Multiple variants of this virus have been identified, and the DNA sequence of these variants differs up to 11% compared to the originally sequenced variants. Studies in Australia have not shown a host specificity for any one of these variants or an indication that one variant is more pathogenic than others.[4] Work in the USA has identified a variant that is commonly found in lories and lorikeets.[35] The biology of this virus in lories may differ somewhat from other PBFDV variants in other species.[42] Genetic variation in these viruses has significant implications for testing. In order to detect all PBFDV variants, PCR assays must be designed to detect conserved areas of these viruses that are identical in all of them. Alternately, multiple assays that are variant-specific must be used.[35,53]

Infected birds shed virus in feather and skin dander, feces and crop secretions. Transmission has been postulated to result from inhalation of the virus, ingestion of the virus or possibly by movement of the virus across the bursal follicular epithelium. Vertical transmission also has been postulated; however, the overall role that vertical transmission plays in the dissemination of beak and feather infection remains uncertain. Naturally occurring disease predominates in juvenile and young adult birds. Whether birds become persistently infected and develop disease depends on the age and species of the bird exposed and possibly the specific variant of the infecting virus.

Virus replication occurs in a wide range of tissues, including the thymus, bursa of Fabricius, crop, esophagus, intestine, skin and feathers. Virus also has been identified in circulating leukocytes. Feather dysplasia results from virus-induced necrosis and disruption of the epidermal collar, intermediate basal epidermis and feather pulp, and thrombosis and hemorrhage within the feather pulp. Damage to the germinal epithelium of the beak is similar, resulting in the observed gross changes. Necrosis of the bursa, thymus and possibly circulating leukocytes results in varying degrees of immune suppression. Diseases caused by opportunistic pathogens are common in PBFDV-infected birds.[36]

Clinical disease may develop within 2 to 4 weeks in exposed nestling parrots, but prolonged incubation periods of months and possibly even years are more likely when young adult birds are infected. Virus can be detected in the blood before clinical signs are observed. In one report, virus could be detected in an experimentally infected bird 2 days after infection. The onset of viremia may be longer in naturally infected birds.

Clinical Presentation

Species Distribution

Psittacine beak and feather disease (PBFD) occurs in a wide range of wild and captive parrots, particularly the

cockatoos, eclectus parrots (*Eclectus roratus*), budgerigar (*Melopsittacus undulatus*) and lories and lorikeets from Australia, the Pacific Islands and Southeast Asia. African parrots, including the African grey parrot (*Psittacus erithacus*) and lovebirds (*Agapornis* spp.), also are highly susceptible to infection and disease, and infection has been found in the wild African parrots. Infection in Neotropical parrots in captivity occurs at a low to moderate rate, but disease is rare. A small number of macaws and Amazon parrots and a single pionus parrot have been reported with PBFDV.[24] PBFDV infections in wild Neotropical parrots are not documented.

Signs in Cockatoos

PBFDV causes chronic progressive disease in birds older than 8 to 10 months. The large majority of birds with the chronic form of PBFDV first develop lesions between 6 months and 3 years of age. The first signs of PBFDV are subtle. A lack of powder on the beak may be the first indication that powder down feathers are diseased. Some birds will present with a history of a delayed molt. Close inspection of these birds will generally reveal at least a few dysplastic feathers. Both down and contour feathers are affected, but the disease may predominate in one or the other. Initially, diseased feathers are widely scattered and are associated with the pattern of molt. As the disease advances, all feather tracts will become involved, generally in a somewhat symmetrical fashion (Fig 32.1). In advanced cases, only down feathers, a few scattered contour feathers or no feathers at all may remain.

Affected feathers show varying degrees of dysplasia. Hyperkeratosis of the feather sheath is common, resulting in sheath thickening and retention. Growing feathers are short and may be pinched off either at their proximal ends or near their base (clubbing). Thinning of the rachis and recent and previous hemorrhage within the feather shaft is common. In some feathers there is so much disruption of feather growth that the sheath contains only a disorganized mass of keratin. Mildly affected feathers may show bowing, have transverse dystrophic lines and fracture at any location along their length.

Beak lesions are common in the sulphur-crested (*Cacatua galerita*), Major Mitchell's (*C. leadbeateri*), Moluccan (*C. moluccensis*) and umbrella cockatoos (*C. alba*), little corella (*C. sanguinea*) and galahs (*Eolophus roseicapillus*). They are less frequent or entirely absent in other species. These lesions may occur at any stage of the disease but are seen most commonly in birds with advanced disease. Early changes in the beak are the result of hyperkeratosis of its superficial layer. These changes cause beak elongation and overgrowth. Longitudinal fissures develop subsequently. In the terminal stages of the disease, the distal portion of the beak will fracture, leaving underlying necrotic debris and bone. Necrosis of the palatine mucosa causes it to separate from the beak (Fig 32.2). The resulting space fills with caseous material. Beneath the caseous material is bone. These lesions are painful and birds may become partially or completely anorectic. Secondary infections of the beak and oral cavity are common. A pathologic process similar to the one occurring in the beak also may affect the nails of the feet. These lesions, however, generally are not a significant manifestation of PBFD.

If the beak lesions are not severe, birds can live with the PBFD for many years. However, the vast majority of these birds die, either from their primary lesions or from secondary infectious diseases within 6 to 12 months after onset of the signs. Mounting evidence suggests that birds with PBFD have significant alterations in their immune function. As a result, opportunistic pathogens, eg, yeasts and other fungi, both gram-positive and gram-negative bacteria, cryptosporidia and avian polyomavirus, are common complications and often terminal manifestations of PBFD. A survey of cockatoos with PBFD showed that most have high concentrations of avian polyomavirus in their skin and would be expected to be continuously shedding this virus.

Acute PBFD in nestling cockatoos may begin with nonspecific signs such as depression and regurgitation. Feather lesions develop rapidly and are extensive. These lesions may be identical to those seen in the chronic form of PBFD, or more often, annular constricting bands near the base of the feather develop simultaneously in numerous feathers (Fig 32.3). These feathers break off easily and may bleed. They also tend to be loose in the follicles and are easily pulled out. An understated feature of this disease is the discomfort of the nestling. The damaged feathers are painful and these birds do not want to be handled. Like the chronic form of PBFD, an early sign of infection is reduced powder on the beak. This last sign is not specific because young cockatoos do not always groom themselves as intensively as the adults and will routinely have less powder on their beaks. Advanced beak lesions rarely have time to develop, as the acute disease is often rapidly fatal. As with the older birds, the rate of disease progression varies. Infection studies suggest that rapidly fatal disease is likely to occur in umbrella and sulphur-crested cockatoos, whereas a more chronic form of the disease can be expected in galahs.

PBFD was rampant in wild-caught cockatoos imported into the USA and Europe prior to 1992. Importation has ceased or is dramatically diminished; as a result PBFD in cockatoos has become rare. Circumstances are entirely different in Australia, where the disease is common in the wild and infected wild-caught birds sold as pets.


Bob Dahlhausen

Fig 32.1 | A cockatoo with generalized feather dysplasia characteristic of psittacine beak and feather disease.


Bob Dahlhausen

Fig 32.2 | Necrosis of the junction of the rhinotheca and the oral mucosa in a cockatoo with advanced psittacine beak and feather disease.


Bob Dahlhausen

Fig 32.3 | A nestling Moluccan cockatoo with the acute form of psittacine beak and feather disease. All growing feathers are involved. Many are seen to have hemorrhage within their shafts.

Signs in African Grey Parrots

Acute PBFD also occurs in juvenile African grey parrots. In experimentally infected birds, non-specific systemic signs preceded feather lesions. Dystrophic feathers identical to those seen in cockatoos also occur in African grey parrots. Additionally, newly formed contour feathers that would normally be gray will sometimes be red. Red coloration of contour feathers, however, is not specific for PBFD. Not all African grey parrots with PBFD have demonstrable feather lesions. A rapidly fatal form of PBFD was described in 7-week-old to 9-month-old African grey parrots. Birds typically presented with an acute onset of crop status, regurgitation and weakness. Feather loss was present in 3 of 14 birds. Total white blood cell counts fewer than 1000 cells/μl were common. An acute, often massive, liver necrosis also was a common finding, although changes in serum chemistry findings did not consistently correlate with the degree of liver disease. Most of these birds died or were euthanized within 2 weeks of presentation.[36,37]

Signs in Lovebirds

PBFDV infection is extremely common in lovebirds. Up to 40% of the lovebird samples submitted to one laboratory for genetic probing were positive. A survey of commercial lovebird producers in Texas found that 60% of the facilities sampled had PBFDV in their collections. Many, possibly most, PBFDV infections in lovebirds do not result in clinical disease. The dynamics of PBFDV infection in lovebirds have not been studied extensively, but it appears that asymptomatically infected lovebirds are only transiently infected. When disease does occur in lovebirds, it is most common in young adult birds. These birds appear unthrifty, they may shed feathers and not regrow them, or they may have a delayed molt. Dystrophic feathers may predominate, be scattered or absent entirely. Some of these birds survive for many months or years, and some will recover and may eliminate the virus. It has been reported that the lory variant of PBFDV may be common in lovebirds.

Encephalitozoon hellem is a common infection in lovebirds and a potential zoonotic disease. The prevalence of *E. hellem* shedding is significantly higher in lovebirds infected with PBFDV.[3]

Signs in Budgerigars

PBFDV infection is enzootic in some budgerigar breeding facilities, but it is not as widely disseminated as it is in the lovebird. Most affected birds are fledglings. In the author's experience, diffuse feather changes similar to those seen in cockatoos are uncommon. Instead, many of these birds have normal feathering except for the complete absence of primary and secondary wing feathers (Fig 32.4). The owners refer to these birds as runners or creepers. These lesions are not specific for PBFD, and identical feather abnormalities are caused by avian polyomavirus infections. PBFDV and polyomavirus infections in budgerigars also can occur concurrently.

Signs in Eclectus Parrots

PBFD in the eclectus is very similar to that in the lovebird. Dystrophic feathers may or may not be present, but

Fig 32.4 | A budgerigar with "French molt." There is incomplete development of the primary wing feathers and the tail feathers. These lesions are the result of either psittacine beak and feather disease, avian polyomavirus or a concurrent infection with both.

feather quality of clinically affected birds is poor. Many of these parrots are unthrifty and are plagued with other infectious diseases. Fatal polyomavirus infections in adult eclectus parrots have been correlated with concurrent PBFDV infections.

Signs in Lories

PBFD appears to be relatively common in free-ranging Australian rainbow lorikeets (*Trichoglossus haematodus*). Fledgling lorikeets with typical dysplastic feather lesions are found walking on the ground. Histology of these birds reveals characteristic bursal lesions. Approximately one-third of these birds die before their first molt, another third have persistent feather abnormalities, but the remaining birds go on to molt and develop normal feathers (L. Filippich, personal communication, 1997). A similar disease has been described in lory and lorikeet collections in the USA. Some of these birds never develop signs, while others develop transient feather disease and still others develop persistent feather lesions and other manifestations of PBFD.[35]

Signs in Neotropical Parrots

PBFDV infection in New World parrots has been documented in 3 to 5% of samples submitted for PBFDV screening in one laboratory. Disease in these birds, however, is extremely rare. Clinically affected birds have feather lesions essentially identical to those of cockatoos. Also like the cockatoos, disease has been seen in both adult and nestling birds. Resolution of the clinical signs has been documented in some of these birds.

DIAGNOSIS

When typical clinical signs are observed, they strongly support the diagnosis of PBFD. PBFDV infection can be confirmed in birds with clinical disease, those with non-specific signs and inapparently infected birds using a PCR assay of heparinized blood. In capable hands, this is a highly sensitive and specific assay. Birds with clinical signs of PBFD that are positive by PCR have a guarded prognosis. With supportive care, some will live many years with the disease. Uncommonly, clinical signs will resolve in some birds and they will subsequently become virus-negative. Birds infected with the lory variant may be more likely to survive infection. Histopathologic examination of biopsies from PBFD birds also can be used to confirm the clinical diagnosis. Clinically normal birds with a positive test result represent birds in the early stages of infection or birds with a transient subclinical infection or a sample that was contaminated at the time of collection. Clinically normal birds with positive test results should be retested in 90 days. Lories infected with the lory variant have remained positive for over 6 months without showing signs of disease. It is critical to remember that all positive birds are actively shedding virus whether they are showing signs or not. Also, birds that no longer have virus in the blood may continue to shed virus in their feather and skin dander until their next molt.

Before the PCR-based assay was developed, diagnosis of PBFD was made by histopathologic examination of plucked growing feathers or biopsies of feathers and feather follicles. Characteristic changes in the growing feather and its follicle and the presence of virus-induced intranuclear and intra-cytoplasmic inclusion bodies (basophilic globule cells) are considered diagnostic. Similar inclusion bodies are irregularly found in other tissues. In the African grey and eclectus parrots, feather lesions may not be present. In birds without feather lesions, the clinician often does not suspect PBFD. As inclusion bodies may be found only in the bursa, submission of a complete set of tissues is necessary for an accurate diagnosis.[36]

CONTROL

With the advent of a sensitive and specific diagnostic assay for PBFDV, control of this disease has been greatly simplified. All new birds should be tested for the virus at the time of purchase. Alternately, testing can be delayed a month to permit a recently exposed bird time to become viremic. The most conservative method would be to test initially after purchase and repeat the test in 30 days. Testing of new birds is of no value unless all other birds in the aviary also are tested. Although expensive, testing all birds in valuable collections of at-risk species can avert future catastrophic loses. Birds with a positive test result should be immediately removed from the aviary, as they are sources of massive amounts of virus.

The cost of testing individual birds can make it difficult to persuade pet store owners and private individuals to

test all lovebirds and budgerigars. If private owners are unwilling to test these birds but have other birds at risk at home, they should be discouraged from keeping them. Rather than test individual birds coming into a collection, pet stores can require that their sources swab their aviary or holding areas for PBFDV before birds are purchased from them. PBFDV is believed to be highly resistant to commonly used disinfectants; therefore, aggressive cleaning is necessary to eliminate it from a contaminated environment. Following cleaning, PCR of environmental swabs can be used to determine if a facility has been adequately cleaned. Routine environmental testing in veterinary hospitals is recommended. A vaccine for PBFDV is not available at the time of this writing.

CIRCOVIRUS INFECTION IN THE CANARY

A disease with high morbidity and mortality has been reported in nestling canaries (*Serinus canarius*). Affected birds have a distended abdomen and an enlarged gall bladder. Exudate in the air sacs also is reported. Canary fanciers refer to the disease as "black spot," as the enlarged gall bladder can be observed through the nestlings' skin. Lesions characteristic of circovirus infections in other birds are present in these canaries, as are the characteristic intranuclear and cytoplasmic inclusion bodies. Diagnosis is most readily made in birds 10 to 20 days old. The partial DNA sequence of a novel circovirus — named the canary circovirus — was amplified from a flock of canaries experiencing a high degree of mortality. Gross lesions were confined to petechial hemorrhages in two of four birds examined. Microscopic examination of the tissues was not done. This sequence information will permit more extensive studies of this virus in the future.[46]

CIRCOVIRUS INFECTIONS IN COLUMBIFORMES

A circovirus infection also has been documented in pigeons. Unlike the disease seen in psittacine birds, it is not usually associated with abnormal feathering. Signs are rarely specific, and birds generally have other diseases as well. Chlamydophila, mycoplasma, adenovirus and herpesvirus infections and systemic bacterial infections have all been described in pigeons with circovirus infection. It is possible that this virus is immunosuppressive and weakens the pigeon's immune system to a point that other diseases develop[47] (see Chapter 13, Integument).

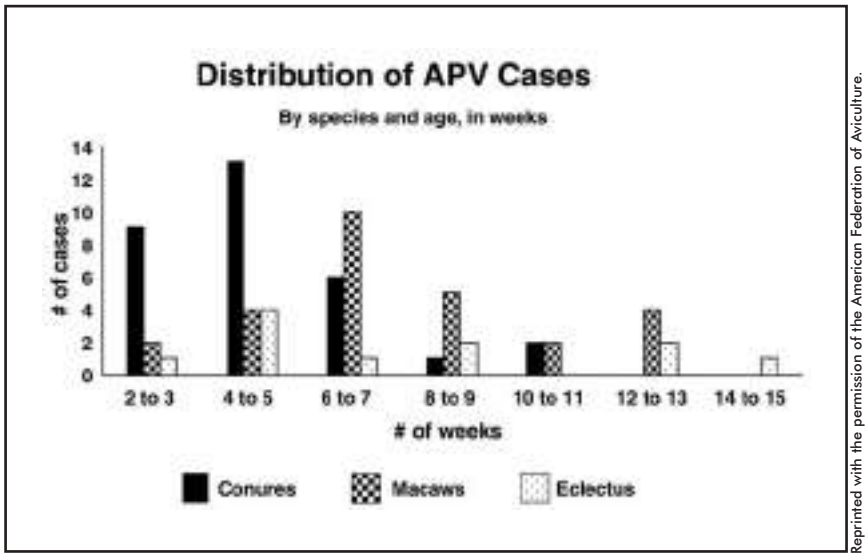
Diagnosis is made by finding basophilic globule cells in tissue sections. A complete set of tissues, including the bursa in the young bird, may be necessary to detect this virus by histopathology. This virus has been sequenced and a PCR assay capable of detecting the virus in tissue has been developed. Using this assay, it was shown that the Columbid circovirus, as it is now called, is widespread in European racing pigeons. It is highly likely that it is present in flocks of feral and domestic pigeons worldwide. Like PBFDV, Columbid circovirus also can be detected in the blood of live birds.[14]

AVIAN POLYOMAVIRUS (APV)

Applied Biology

APV is widespread and can be found in most countries of the world where psittacine birds are raised. It is a nonenveloped DNA virus that codes for six proteins. Some degree of genetic variation has been identified in APV but it is relatively small, and it is assumed that all APV variants have the same host range.[29] The route of natural infection has never been experimentally verified, but infection has been induced through the respiratory route. Given the rapid spread of this virus at bird sales and in the nursery, natural infection through the respiratory system is likely. In the budgerigar, the virus replicates in a wide range of tissues, including growing feathers, skin, liver, spleen, renal tubular epithelium, heart and cerebellum. Clinical signs and necropsy findings are largely associated with tissue distribution of virus replication. Disease in budgerigars is confined to nestlings. Not all budgerigars die with APV infection, and surviving birds shed virus in skin and feather dander and in droppings.[24]

Most non-budgerigar parrots are assumed to be susceptible to APV infection. Disease, however, typically is limited to nestling parrots. Macaws, conures and eclectus parrots are over-represented, although diagnosis of this disease has occurred in most psittacine species (Fig 32.5). Birds become viremic sometime between 1 week and 10 days after infection. Disease, if it is going to occur, develops 10 to 14 days after exposure. It is characterized by generalized hemorrhage, moderate to massive hepatic necrosis, and an immune-complex glomerulopathy. Characteristic karyomegalic changes and intranuclear inclusion bodies are typically found in macrophages and other antigen processing cells, including the mesangial cells of the glomerulus. The vast majority of birds with these lesions die. Adult birds and nestlings that are infected but do not develop disease will remain viremic for a variable period of time and shed virus in their droppings, and possibly in feather dander and skin, before becoming virus-negative. Most infected birds clear the virus after several weeks to several months and, although they maintain a persistent antibody titer, are not thought to be persistently infected.[30]



Reprinted with the permission of the American Federation of Aviculture.

Fig 32.5 | Age distribution of nestling macaws, conures, and eclectus parrots with avian polyomavirus disease, from the literature and birds submitted to the Schubot Exotic Birds Health Center.[2, 5]

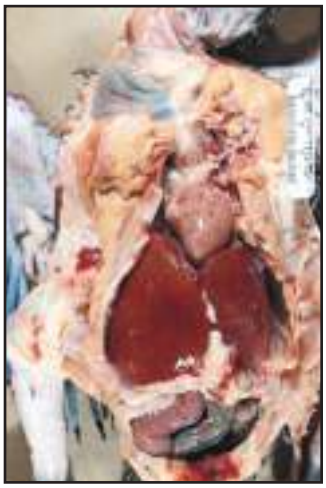


Fig 32.6 | A nestling macaw with avian polyomavirus disease. Note the extensive petechial and ecchymotic hemorrhage and pale musculature.

Clinical Presentations

APV in Budgerigars

Budgerigar breeders first detect this problem in their flocks when there is a sudden increase in the number of dead nestlings in the nest boxes. The nestling mortality rate often is high and may approach 100% when the virus is first introduced to an aviary. If there is no intervention in subsequent breeding seasons, mortality rates will decline but production will always remain depressed. The signs of APV disease in budgerigar nestlings are somewhat variable. Most often, the young birds experience an abbreviated course of disease. At death, birds are found to be stunted and have abnormal feather development, skin discoloration, abdominal distension, ascites, hepatomegaly with localized areas of necrosis and scattered areas of hemorrhage. In some outbreaks, the virus attacks the cerebellum and these birds will have head tremors. Death predominates in birds that are 10 to 20 days old.[25]

Not every budgerigar nestling infected with APV dies. Some never show signs. Other nestlings will fail to develop their primary and secondary wing feathers and tail feathers (see Fig 32.4). These birds have been referred to as runners or creepers, and this form of the disease has been described as French molt. PBFDV or a combination of APV and PBFDV can cause identical lesions. It is possible that one or more additional diseases may cause similar feather disease. Not all budgerigars appear to be equally susceptible to infection and disease. In one study in the United States, English budgerigars were rarely found to be infected with APV, although they were housed with other birds shedding the virus.

APV in Non-budgerigar Parrots

Non-budgerigar parrots are susceptible to APV. Some are highly susceptible to disease, while others rarely, if ever, develop disease. APV disease in these birds occurs at different ages in different birds. In conures, deaths typically occur in birds less than 6 weeks of age. Deaths in macaws and eclectus parrots occur in birds 14 weeks and younger (see Fig 32.5). Most, possibly all, of the nestlings lost are being hand-fed. Infected nestlings appear healthy, show very few premonitory signs and then die suddenly. When signs do occur, they precede death by only a few hours. Observant owners may notice delayed crop emptying, weakness and a generalized pallor or bruising under the skin in the preceding hours before death. Yellow discoloration of the urates is another rare observation. Occasionally, complete blood count and serum chemistry tests can be performed prior to death. Increases in the liver enzyme aspartate aminotransferase are expected. Near death, birds have a marked thrombocytopenia. Birds that die are typically in excellent body condition. Additional findings commonly include generalized pallor with subcutaneous and subserosal hemorrhage and enlargement of the spleen and liver (Fig 32.6). Less commonly, ascites and pericardial effusion may be present.

APV in Lovebirds

APV disease in lovebirds is distinct enough to merit special attention. As in the budgerigar, this disease occurs in nestling birds, and inclusion bodies can be found in multiple organs. Unlike the budgerigar, birds up to 1 year of age also can be affected. This unusual age susceptibility has not been fully explained. However, in at least some of these older birds, concurrent infection with PBFDV also is occurring and may permit APV disease in a bird that would otherwise be resistant to it.

APV in Nestling Cockatoos

A unique presentation of APV disease occurs in nestling cockatoos. These birds present at an age of 4 to 8 weeks with a history of difficulty breathing. Physical examination reveals a severely dyspneic bird that is underweight and may be stunted in its growth. These birds have all the appearances of a bird that has aspirated food. Most of these birds die. Necropsy reveals heavy, wet lungs that may not float in formalin. Histologically, there is a severe generalized interstitial pneumonia with huge numbers of inclusion bodies in what are believed to be type II pneumonocytes. Preliminary sequence data suggests that this form of APV disease is caused by a specific APV variant. This variant is still capable of causing the classic form of APV disease in other susceptible species.[32]

Post-APV Edema and Ascites Syndrome

Some birds that have APV disease survive. An unknown percentage of these birds go on to develop ascites and generalized edema. They still appear bright and alert and may continue to eat and empty their crops, but they are edematous and have a fluid-filled peritoneum. The fluid is a transudate or modified transudate and does not contain inflammatory cells. These birds do not improve and either die or are euthanized. Histologic lesions include a sclerosis of the glomeruli and regenerative lesions in the liver. It is suspected that the edema and ascites syndrome is secondary to hypoproteinemia, either from a failure of albumen production in the liver or a loss of protein from the kidney. Viral inclusion bodies are rare or absent in these birds. This disease very closely resembles the viral serositis lesions described in nestling parrots with eastern equine encephalitis and may be mistaken for it. These birds are still loaded with APV, and PCR of blood or cloacal swabs in the live bird or blood or a liver swab in the dead bird will be strongly positive.[32]

APV in Adult Parrots

APV readily infects adult parrots. Most infections, probably greater than 99.9% of them, are completely asymptomatic. These birds become infected, shed virus for a few weeks or do not shed virus at all, and do not show signs of illness. APV disease, however, has been documented in adult birds. Disease in these birds resembles that seen in the nestling. An atypical form of a progressive virus encephalopathy also has been reported in two cockatoos. Why rare adult birds or groups of birds develop disease is not known in all cases. In many cases, however, adult birds that die with APV disease have concurrent PBFDV infections. PBFDV is believed to be immunosuppressive, allowing APV to cause disease in a bird that would normally be refractory to infection. APV disease does uncommonly occur in adult parrots that do not have concurrent PBFDV infections. In the author's experience, these deaths typically occur in pet stores, suggesting that stress may play a role in the pathogenesis of disease.

Diagnosis

Testing Inapparently Infected Birds

The goal of testing is to detect inapparently infected birds that are shedding virus and to keep them from exposing other birds. Budgerigars, when infected as nestlings, shed virus for 6 or more months. Larger species of parrots, when infected as nestlings, become viremic for 4 to 8 weeks and shed virus from 6 to 16 weeks. Viremia and virus shedding have rarely been detected for as long as 10 months. Birds infected with PBFDV and APV may shed virus continuously. To detect virus-shedding birds, both blood and a combined oral and cloacal swab are examined by PCR. Testing blood alone is not recommended, as viremia ceases before virus shedding ends. Limited studies have been done on the duration of virus shedding in adult birds. However, it appears that after exposure, viremia and virus shedding are absent or greatly abbreviated as compared to nestlings.[25,28]

All birds infected with APV develop a detectable virus-neutralizing (VN) antibody titer within 2 to 3 weeks. The presence of antibody has no bearing on virus shedding, as antibody-positive birds continue to shed virus for many weeks. Also, once a bird develops antibody, most will maintain a high antibody titer many years after they have stopped shedding virus. Even though virus shedding cannot be predicted by serology, serology still has some value in the control of APV. Serology can be used to screen young budgerigars and lovebirds coming out of an aviary or returning from the show circuit. If they are coming from an aviary and are seropositive, they have been recently infected with APV and are most likely shedding virus. If birds that have been on the show circuit are seronegative after a 2-week quarantine, they are not infected with APV.

Postmortem Diagnosis of APV Disease

Gross and microscopic lesions seen in birds that die with APV infection are characteristic. Spleen, liver, lung and kidney are essential tissues to provide to the pathologist. These birds are viremic at the time of death, and swabs of any tissue will be positive by PCR. Immunofluorescent staining of impression smears and in situ hybridization also can be used to confirm infection if other histologic findings are inconclusive.[36]

Prevention and Control of APV[25]

The key to prevention of APV disease is to make sure that birds that are shedding virus are not introduced into an aviary or that materials that might be contaminated with APV are not brought into the aviary. Testing can be an important part of a prevention plan. Excellent

PCR assays have been developed to detect infection in the live bird. Serology can be used to determine if a bird has been infected in the past but does not adequately predict the virus-shedding status of the bird.

Control in Budgerigar Aviaries

If a prevention program for APV infection is to be instituted in a budgerigar aviary, the first step is to make sure that it is not already there. The virus is readily detected by PCR in the environment of aviaries where the infection is enzootic. Alternately, blood and combined oral and cloacal swabs can be used to test nestlings and young adult birds. A virus neutralization assay can be used to detect antibodies to APV. Most birds in an aviary with enzootic APV will be seropositive.

Exhaustive efforts are required to keep APV out of a budgerigar collection. The movement of birds on and off the property must be carefully restricted. All new birds coming onto the property should be seronegative or PCR-negative. Alternatively, environmental swabs of their aviary of origin can be tested by PCR. If the aviary is a commercial aviary, dealers, feed salespeople, delivery trucks and other bird breeders should be banned from the aviary entirely. Young birds taken to the bird dealer and rejected should not be returned to the aviary. Food should be purchased directly from the feed mill so that it is never in contact with other birds. If the aviary is primarily breeding show budgerigars, then all birds going to the show should be quarantined until the end of the show season and tested by serology or PCR before they are returned to the breeding colony.

Budgerigars are not the only birds that can bring APV into a collection. Lovebirds and possibly cockatiels (*Nymphicus hollandicus*) also can be sources. Devastating outbreaks have occurred in budgerigar operations when lovebirds have been introduced into previously closed budgerigar colonies.

Elimination of APV from a budgerigar collection is challenging, but not impossible. The first critical step is to stop breeding. The infection cycle is perpetuated by the constant presence of infected nestlings, fledglings and young adult birds. These birds shed virus for up to 6 months or more after infection, seeding the environment with virus. Chicks are then exposed immediately upon hatch and the cycle continues. Once breeding is stopped, all birds that have not been used for breeding should be removed from the property. Adult birds should be moved to a temporary environment and the aviary totally disinfected. Nest boxes can be cleaned and painted but are better off destroyed and replaced with new boxes. All wood surfaces should either be discarded or cleaned and painted over. After a 6-month hiatus, the adult breeding stock can be returned to the clean aviary and set up for breeding again.

Prevention and Control in Lovebird Collections

The sad state of the matter is that both PBFDV and APV are enzootic in many lovebird aviaries. Oddly, disease in these birds is often rare. Shedding, however, occurs in young lovebirds and may be continuous in birds that are concurrently infected with PBFDV. Breeders who wish to have a lovebird collection that does not have APV should first test their birds for infection. Again, serology using the virus neutralization assay or PCR of swabs from the environment or blood and combined oral and cloacal swabs of individual nestlings and fledglings will readily detect virus. To prevent the introduction of APV to a lovebird aviary, a representative number of each lot of incoming birds is tested by serology or PCR. Alternately, environmental swabs are used to verify that the aviary from which the birds originate is free of APV.

Prevention and Control in Aviaries Breeding Non-budgerigar Parrots

Outbreaks of APV do not occur in adult breeding birds, they occur exclusively in nurseries. Outbreaks occur when birds with inapparent infections, generally nestlings, are introduced to the nursery. If the following rules are followed, APV outbreaks in the nursery are extremely unlikely. Breeders of the larger species of parrots should not breed cockatiels, lovebirds or budgerigars. If they must breed these species, they must test them thoroughly to make sure they are not infected with APV. The breeder should raise only the chicks that they produce. If they must raise chicks from other sources, these birds must be quarantined and tested by PCR before they are brought into the nursery. Extensive and repeated environmental testing of the aviary of origin may be substituted for individual bird testing. Any bird that leaves the nursery and is in direct or even indirect contact with other birds must not be allowed back into the nursery. Adult birds coming into the aviary also should be quarantined and tested for APV. Ideally, people taking care of the nursery would not take care of the adult birds. Often, this is not possible. In these situations, cleaning up and changing clothes before working with the nestlings is recommended.

APV outbreaks in the nursery are devastating. In most cases, once APV is introduced to a nursery it spreads rapidly, so that by the time the first case is recognized most of the nestlings are already infected. This concept is important for two reasons. First, vaccination at this point will do no good. Second, testing during the outbreak will prove only that the virus is widely disseminated. To save money, the aviculturalist should be encouraged to reserve testing to determine when shedding has stopped and the chicks can be sold.

Little can be done to keep exposed chicks from becom-

ing infected with APV in most nurseries. However, efforts should be made to improve hygiene, decrease density of birds and use individual syringes for hand-feeding individual chicks. The most important element in the control of APV outbreaks is to stop bringing babies into the nursery. Chicks can be left in the nest to be raised by the parents or pulled and sent to another facility to be raised. It remains unclear why, but parent-raised chicks (excepting lovebirds and budgerigars) are not reported to develop APV disease. Surviving chicks will shed virus for 8 to 14 weeks, rarely as long as 16 weeks. The older the chick at the time of exposure, the shorter the period of virus shedding. Chicks should be negative by PCR of blood and a combined oral and cloacal swab before they are sold. PCR of the oral and cloacal swab is critical, as viremia ceases before shedding stops.

After the outbreak has stopped, a close inspection of the aviary must be done. Birds that might be shedding virus need to be identified and tested or eliminated from the aviary. Extensive cleaning and disinfection of the nursery also will have to be done. In aviaries where the underlying source of disease has been eliminated, subsequent breeding seasons can be free of the disease.

Preventing APV Disease in the Pet Store

The pet store is one of the most common places where APV outbreaks occur. Most pet stores get their birds from multiple sources. They sell budgerigars, lovebirds and cockatiels, the three species that are most likely to be shedding virus, and many stores will acquire susceptible species when they are still nestlings. To avoid disease, pet stores can use several strategies. The easiest and best method for preventing APV disease in the pet store is to buy only weaned nestlings. These birds will be old enough that, if infected with APV, they will not develop disease. If unweaned nestlings are to be purchased, they should be raised outside of the store until weaned. If nestlings must be in the store, they should be separated from all other birds, and have a person designated to take care of only them and no other birds. The public should not be allowed to handle these birds. Stores that sell high-value nestling parrots should consider limiting their bird sales to these birds only and not selling lovebirds, budgerigars and cockatiels. Establishing long-term relationships with breeders also can reduce the risk of disease transmission. Breeders supplying pet stores should be encouraged to develop a preventive medicine program to develop and maintain APV-free flocks.

Immunization

A commercial APV vaccine[a] is available in the USA. Its value as a tool in the prevention of APV disease is controversial. The author's research has raised several questions about this vaccine and its ability to protect against APV infection. The vaccine is to be given to nestlings that are 4 weeks of age or older and is thought to provide protection to nestlings 2 weeks after the second vaccine, or by the age of 8 weeks. From the dynamics of the disease, however, most birds cannot be immunized early enough in life to be protected. An additional concern is that VN antibody was not detected in nestlings immunized with this vaccine, and immunization of adult birds resulted only in low antibody titers.[28] Clinical trials that claim to show that the vaccine stopped outbreaks of APV disease in nurseries did not study control flocks where the vaccine was not used. If new nestlings were not added to a nursery experiencing an outbreak, deaths would stop on their own within 2 to 4 weeks of the first death.[34] Claims that immunizing already infected birds will result in a shortening of the duration of virus shedding are not documented.

APV is, by and large, a completely preventable disease through appropriate management strategies and selective testing. As a result, the author stresses these avenues of control and does not recommend immunization.

APV Infection and Disease in Non-psittacine Birds

One or more APV strains can infect non-psittacine birds. Several species of passerines have been documented to have classical APV disease. In the author's experience, flocks of Gouldian finches (*Chloebia gouldiae*) are perhaps at greatest risk. Again in the author's experience, mortality is limited to nestling and young adult finches during one breeding season but is not seen again in the following year. Surviving birds have moderate levels of antibody that will neutralize a lovebird-derived APV. APV DNA was detected in the tissues of one finch with PCR primers derived from the psittacine APV sequence, suggesting that this bird was infected with a psittacine variant. However, other studies suggest that another significantly different virus also may infect passerines.

There is a single published report of a rhamphastid dying with an APV disease. The bird was a green aracaris (*Pteroglossus viridis*). The virus sequenced from this bird was found to be nearly identical to those derived from psittacine birds. It was speculated that the original source was a cockatoo. In this study, in-contact birds, including zebra finches (*Poephila guttata*), a kookaburra (*Dacelo novaguineae*) and a Lady Ross turaco (*Musophaga rossae*), became seropositive but did not develop disease.[19]

APV Infection in Free-ranging Birds

There is strong evidence that APV infection occurs in wild birds on multiple continents. A high prevalence of anti-APV antibody was found in free-ranging greater

sulphur-crested cockatoos in Australia.[33] APV disease has not been reported to occur in wild Australian birds, but a disease with characteristic APV lesions was induced in a cockatoo infected with a preparation of PBFD virus derived from the feathers of a wild bird, suggesting that APV was present in these tissues and was copurified with the PBFD virus.[33] Recently, APVs were identified in five buzzards (*Buteo buteo*) and a falcon (*Falco tinnunculus*) in Europe. Genetically, the sequence of the falcon virus was nearly identical to other APV variants of psittacine origin and the virus in the buzzard amplified with PCR primers derived from the sequence of the original APV isolated from a budgerigar. Because of autolysis in the buzzards, the histologic lesions associated with this disease could not be characterized.[17]

Preliminary evidence that APV may occur in wild birds in North America also exists. A house sparrow (*Passer domesticus*) was found to have a glomerulopathy with characteristic APV-like inclusions within mesangial cells and PAS-positive deposits within the mesangium and glomerular capillaries.[24]

Goose Hemorrhagic Polyomavirus

A genetically distinct polyomavirus with limited homology to the avian polyomavirus has been implicated as the cause of the hemorrhagic nephritis and enteritis of geese in Europe. Little is known about the importance of this virus in waterfowl, but it may be widespread, as the histologic lesions that it is reported to cause are not specific.[13]

PAPILLOMAVIRUSES

Diseases caused by papillomaviruses in birds have been described only in wild European finches and imported African grey parrots. The African grey parrots had papilliferous plaques of the commissures of the beak, eyelids and face that became more extensive over the course of the year the birds were monitored. Lesions in European finches predominate on the legs and feet; lesions of the face are rare. These lesions should be differentiated from those caused by poxviruses.[43]

ADENOVIRUSES

Adenoviruses in Companion Birds

Adenovirus infections and disease in companion birds are rare. They have been associated with hepatitis, acute necrotizing pancreatitis, conjunctivitis and a multisystemic disease in lovebirds. However, recent reports of these diseases have been lacking. Adenovirus-associated encephalitis also is a rare disease that has not been recently reported. Characteristic basophilic intranuclear inclusion bodies are infrequently seen in renal tubular epithelial cells in parrots that die with other diseases. These lesions are most common in lovebirds and budgerigars.[21,36]

A fatal adenovirus infection causing hepatitis is described in the nestlings of Senegal parrots (*Poicephalus senegalus*) and related species. The disease occurs sporadically within aviaries. In one collection, the disease occurred in 3 out of 4 years in offspring from a single pair of Senegal parrots. Affected nestlings typically present acutely ill or are found dead. Grossly, the liver is discolored red-black, and scattered yellow-gray areas may be present. Multifocal hepatic necrosis and the presence of large, darkly basophilic intranuclear inclusion bodies within hepatocytes characterize this disease.[36]

The author has seen adenovirus infections in several mixed flocks of finches. Typically, clinical signs are not observed. Concurrent diseases, such as candidiasis and atoxoplasmosis, were common. Poor hygiene and high stocking density may have played roles in these deaths.

Pigeon Adenovirus

Adenoviruses in pigeons cause two distinctly different diseases. The first occurs in pigeons less than 1 year old and may be associated with the onset of the racing season. This virus replicates predominately in the intestinal epithelium, causing villus atrophy. Many birds will develop disease. Signs are those of acute severe enteritis, diarrhea and vomiting. Severely affected birds die, but many uncomplicated infections resolve within 1 week. A common complication of this adenovirus infection is an *Escherichia coli* overgrowth of the intestinal tract. These birds have persistent diarrhea, lose condition and will die if not aggressively treated. *E. coli* overgrowth of the intestine also can result in septicemia and sudden death. Mild to moderate hepatic necrosis may occur in some infections and contribute to the clinical signs and duration of the disease.

A second adenovirus causes massive hepatic necrosis. All ages of birds are susceptible. Disease, however, is sporadic in a flock and spreads slowly. Signs of infection include vomiting and biliverdin-stained urates; however, most birds die before signs are recognized. Birds showing signs die within 24 to 48 hours.

Diagnosis for both adenovirus infections can be made only at necropsy. Treating for dehydration and secondary bacterial infections can mitigate mortality in birds with the enteric adenovirus infection.[8,52]

PSITTACID HERPESVIRUSES (PsHVs)

Applied Biology

PsHVs are alpha herpesviruses that are the causative

agent of Pacheco's disease (PD) and internal papillomatosis of parrots (IP). The PsHV1 virus contains three major serotypes. Two additional serotypes (serotypes 4 and 5) are described, but they are each represented by only a single virus isolate. It is unclear if 5th serotype is a PsHV1 or an entirely different herpesvirus. There are four major genotypes of PsHV1. The viruses in genotypes 1 and 4 comprise serotype 1, the viruses in genotype 2 comprise serotype 2 and the viruses in genotype 3 comprise serotype 3. The single serotype 4 isolate is a genotype 4, but appears to have evolved into a unique serotype.[49] A new herpesvirus, PsHV2, has been discovered. This virus has been identified in mucosal tissues from Congo African grey parrots and a single blue and gold macaw. Most birds were not showing signs of disease, however this virus was found in a mucosal papilloma in one African grey parrot and cutaneous papilloma from another.

The study of the complexity of these viruses and the correlation between genotype and pathotype is still in its infancy, but patterns are beginning to unfold. Current sequence data has allowed the development of PCR primers that can detect all of the viruses discovered to date. This discovery allowed investigators to determine that the PsHVs that cause PD persist in the mucous membranes of the oral cavity and cloaca and can be inconsistently detected in the blood.[31,49,50]

Based on these data, the following epizootiologic picture is proposed. Transmission between birds occurs when a naïve bird is exposed to the oral secretions, droppings or vomitus of a persistently infected bird. The route of infection can be by ingestion or contact with conjunctival or respiratory mucous membranes. The outcome of infection will depend on the genotype of the virus and the species of bird exposed. Genotypes 1, 2 and 3 are highly pathogenic to Amazon parrots. In Europe, genotype 4 PsHV also kills Amazon parrots, but this virus is not found to cause PD in Amazon parrots in the USA.[49] In contrast, genotype 4 is the most common cause of PD in macaws and conures. Cockatiels, cockatoos and other Pacific species of birds are relatively resistant to PD, but when they do develop disease, any of the four genotypes may be responsible. African grey parrots are susceptible to genotypes 2, 3 and 4. Genotype 1 has not been found in African grey parrots with PD, but the number of African greys tested to date is small, so this should be considered only a preliminary finding.

Birds that become infected with PsHVs and either do not develop PD or do develop PD but are treated and survive will become persistently infected and will remain persistently infected for life. There is a possibility that some subclinical infections may result in a cure, but this remains to be proven. There is no evidence that persistently infected birds will subsequently develop PD. However, some persistently infected birds will develop IP. Which birds will develop IP may depend on the species of the bird, the genotype of the virus and as yet undetermined factors.

Most persistently infected birds are readily detected by PCR analysis of blood and combined oral and cloacal swabs. These birds will be consistently positive with repeated samplings.[31] Even though PsHVs are continuously present in the mucosa of persistently infected birds, field data suggest that actual virus shedding or the degree of virus shedding may fluctuate over time. Species persistently infected with PsHVs include the macaws, Amazon parrots, some of the *Aratinga* conures and the Patagonian conure (*Cyanoliseus patagonus*). Increasing evidence also suggests that cockatiels, lovebirds, cockatoos and possibly other species may be persistently infected with one or more PsHVs. Wild-caught birds that have passed through a quarantine station, parent-raised chicks of wild-caught birds and birds that have survived an outbreak of PD are at highest risk for persistent infection.

The incubation period for PD typically ranges from 5 to 14 days. Virus replication occurs in a number of organs, and birds are viremic. Inclusion bodies are most often found in the liver and spleen and to a lesser extent the crop, small intestine and pancreas. Necrosis of the infected cells, particularly hepatocytes, accounts for the clinical signs.[36]

Clinical Presentation

PD occurs almost exclusively in psittacine birds. Disease is most common in avicultural collections, quarantined birds and pet stores. The most common clinical presentation is a dead bird that died with little or no advanced evidence that it was ill. PD occurs most frequently in mixed collections of parrots that contain Amazons, macaws and conures, particularly Patagonian and *Aratinga* conures. The onset of the breeding season or recent changes in the aviary may predispose to virus shedding and PD outbreaks. Clinical signs may precede death in macaws and less frequently in other species. Signs are non-specific and include lethargy, depression and anorexia. Profuse sulfur-colored (biliverdin-stained) urates are another non-specific but consistently reported sign. Regurgitation, bloody diarrhea and terminal central nervous system signs are infrequently reported. Duration of clinical signs ranges from a few minutes to a few days. Only a few birds are known to survive infection once clinical signs develop. Elevation in the serum aspartate amino transferase concentrations and a marked leukopenia are reported in these birds. Radiographically, hepatomegaly, splenomegaly and renal enlargement also are

documented. The number of affected birds can vary from a single isolated case to hundreds.[24]

To the author's knowledge, the only documented naturally occurring case of Pacheco's disease in a non-psittacine bird occurred in a keel-billed toucan (*Ramphastos sulfuratus*). Lesions in this bird and a second keel-billed toucan experimentally infected with Pacheco's virus were characteristic of the psittacine infection. In another toucan (species not reported), a disease resembling Pacheco's disease was described. Herpesvirus virions were identified in the tissues of this bird; however, fluorescent antibody-staining of the tissues with a Pacheco's virus-specific antibody was negative.

Diagnosis

Diagnosis in the live bird is difficult and rarely made. History, signs and laboratory findings are strongly suggestive of PD but are not specific. These birds are strongly positive on PCR of combined oral and cloacal swabs and blood but usually die before the samples can be analyzed. In the author's experience, once a bird is confirmed to have disease and owners know what to look for, they will detect the early stages of the disease in birds, often in time to save them with treatment.[24, 36]

Most birds that die are well muscled and may have recently ingested food. Common gross lesions include hepatomegaly, splenomegaly, renal swelling and serosal and epicardial hemorrhage. The affected liver may be uniformly pale yellow, resembling the appearance of a diffuse lipidosis (Fig 32.7), have a diffuse mottling, or have scattered, irregularly shaped, discolored foci. In many birds, liver lesions are not observed grossly. Less commonly, submucosal hemorrhage of the intestines with or without intraluminal blood also may be present. Because of the acute nature of this disease, gross lesions may be entirely absent in some birds.

Histologically, hepatic necrosis is present in the vast majority of the cases. Varying degrees of splenic lymphoid hyperplasia and necrosis, pancreatitis and enteritis also occur. Eosinophilic and, less frequently, basophilic intranuclear inclusion bodies are found in the liver on the margins of the necrotic areas and in bile duct epithelium. Inclusions will sometimes be present in the spleen, intestinal epithelium, crop and pancreas. Although these lesions are characteristic for PD, the diagnosis can be verified by PCR of tissue swabs, staining impression smears with specific fluorescently labeled anti-Pacheco's virus antibody and in situ hybridization.

Treatment

Mortality in Pacheco's virus outbreaks can be minimized by prophylactic use of acyclovir[b]. In the author's experience, mortality stops within 24 hours after the initiation of flock treatment. Treatment options include administration of acyclovir in the drinking water (1 mg/ml) and food (400 mg/quart of seed) simultaneously or by gavage (80 mg/kg q 8 h). A higher oral dose of 330 mg/kg q 12 h also has been recommended. The necessary length of treatment is not known. The author treats flocks for 7 days and birds with signs of disease for 2 weeks.[23]

Preventing virus spread is another important aspect of bringing a Pacheco's disease outbreak under control. Traffic through the aviary should be minimized and hygiene improved. Additionally, barriers between cages can be erected, or cages can be moved farther apart. Intensive cleaning efforts may result in the increased aerosolization and further dissemination of the virus. Immunization in the face of an outbreak is of questionable benefit, as protective antibody titers would not be expected for 2 weeks after vaccination.

Prevention and Control

Control measures fall into three categories: savvy management practices, testing and immunization. Given that some conure species have repeatedly been implicated in the outbreak of this disease, these birds should not be kept in a mixed collection. General concepts, such as a closed aviary, proper quarantine procedures and acquisition of birds from reputable sources, will help to minimize the likelihood of Pacheco's virus being introduced to an aviary. Adequate spacing between cages and limiting human traffic in the aviary also are important preventive measures. Outbreaks are less likely to occur in outdoor aviaries.

Testing is becoming an increasingly practical means of preventing the introduction of PsHVs into a collection. Persistently infected birds are readily detected by PCR of blood and combined oral and cloacal swabs. Birds that are at highest risk for being persistently infected are those that have survived PD outbreaks and wild-caught parrots and chicks raised by wild-caught parrots. Macaws, Amazon parrots, Patagonian conures and *Aratinga* spp. conures commonly are demonstrated to be infected persistently.

A single Pacheco's disease virus vaccine[c] is currently being marketed in the USA. The serotype of the virus in this vaccine and its ability to protect against all serotypes of PsHVs are not known. Immunizing parrots in mixed collections of high-risk birds may be beneficial.

Internal Papillomatosis of Parrots (IP)

Clinical Manifestations

IP is a disease that primarily affects macaws, Amazon parrots, hawk-headed parrots and, less commonly,

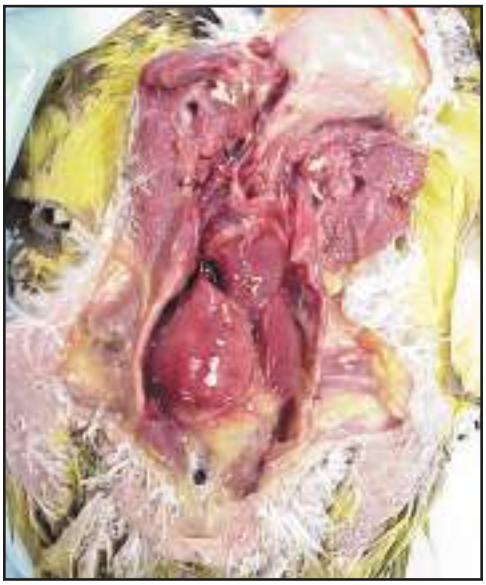


Fig 32.7 | An eclectus parrot with Pacheco's disease. The liver has a diffuse yellow mottling that is caused by extensive hepatic necrosis. Although many cases do not present with this lesion, the presence of this lesion should alert the practitioner or pathologist to the possibility that they are dealing with Pacheco's disease.

Fig 32.8 | Extensive papillomatous changes to the cloacal mucosa of an Amazon parrot.

conures. Most lesions are confined to the oral and cloacal mucosa, although lesions also may be found in the conjunctiva, nasal lacrimal duct, bursa, esophagus, crop, proventriculus and ventriculus. Cloacal lesions are the most common manifestation of IP in Amazon parrots and generally are present in the macaw as well. Oral papillomas are common in macaws. Of the macaws, the green-winged macaw (*Ara chloroptera*) is prone to develop the most widely disseminated form of IP. In these birds, lesions generally are present in both the cloaca and oral cavity and may extend into the esophagus, crop and even the proventriculus and ventriculus. Less frequently, blue and gold (*Ara ararauna*) and scarlet macaws (*Ara macao*) and, uncommonly, an Amazon parrot will develop this diffuse form of IP.[23]

Owners usually first recognize that their bird has IP when they see blood in the bottom of the cage from an ulcerated cloacal papilloma or when the papilloma prolapses through the cloaca (Fig 32.8). Oral lesions may be extensive but rarely result in clinical signs. Papillomatous lesions are rarely static; they wax and wane and may disappear entirely. Often, the only indication that IP is present is a slight roughening of the cloacal mucosa or a thickening of the choanal edges and blunting of the choanal papillae. If the lesions do not spontaneously resolve, each time they recur they generally are more severe. The lesions in some birds will be consistently present. Birds with IP may live for many years and even be reproductively successful. The general life expectancy of these birds, however, is diminished as compared to other birds without this disease. The chronic irritation associated with cloacal lesions and repeated surgeries to remove the lesions may result in cloacal strictures. Birds with diffuse lesions of their upper digestive system often develop a wasting disease that may resemble proventricular dilatation disease.

A small to moderate percent of birds with IP will go on to develop bile duct or pancreatic duct carcinomas. Signs of bile duct carcinomas are not specific. Birds typically lose body condition, appear unthrifty and may have an overgrown beak. Elevated gamma glutamyl transferase levels have been reported in birds with advanced lesions. Bile duct carcinomas are readily demonstrated with ultrasonography and appear as hyperechoic round to irregular masses. Infection with PsHV genotype 3 may predispose to the development of bile duct carcinomas.

Treatment

Birds with cloacal papillomas are often in pain, so it has been common practice to remove all or part of these lesions. Cryotherapy, electrocautery, chemical cautery, laser surgery and sharp dissection have all been used. In the experience of the author, removing part of a lesion will often cause the remainder to regress; these lesions generally return, however. To minimize the risk of stricture formation, surgery is limited to a small portion of the cloaca or not done at all. Carboplatin has been used in a few birds to treat bile duct carcinomas (B. Speer, personal communication, 2001) (see Chapter 35, Surgical Resolution of Soft Tissue Disorders).

Prevention

Mounting evidence strongly suggests the viruses that cause PD are the same viruses that cause IP.[18,27,43,44,48] Depending on the genotype of the viruses and the species involved, however, these viruses can disseminate through a collection without causing Pacheco's disease. The first clue that the virus is there is when birds begin to develop papillomas. Transmission can occur from parent to offspring and between cages of birds. Transmission is much more likely to occur if cages are in close proximity and birds are housed indoors. A meticulous physical examination of birds before entry into a collection coupled with a PCR assay for PsHVs will detect infected birds.

Treating birds with anti-herpesvirus drugs will not cure them of infection and does not appear to impact the course of IP. These viruses presumably persist in a non-replicating form and are not susceptible to these drugs.

Cutaneous Plaques and Papilliferous Lesions of the Foot

Herpesvirus virions are documented in proliferative lesions on the feet of macaws and cockatoos. These lesions show some species-specific variations. Lesions in cockatoos tend to be papilliferous, while those of macaws are raised, depigmented plaques. These lesions regress if treated topically with acyclovir. The sequence of a herpesvirus from a plaque from the foot of a macaw was found to closely resemble that of the PsHVs that cause PD and IP.[48]

MISCELLANEOUS HERPESVIRUS INFECTIONS IN COMPANION BIRDS

An infection resembling a herpesvirus infection is described in Gouldian, melba (*Pytilia melba*) and purple grenadier (*Uraeginthus ianthinogaster*) finches. Clinical signs include weight loss, anorexia, dyspnea, severe conjunctivitis and, less commonly, a head tilt. Mortality ranges from 25 to 100%. Gross and microscopic lesions are found in the conjunctiva, trachea and air sacs.[40]

A herpesvirus has been isolated from English budgerigars in Europe. Its presence has been correlated with reduced hatchability and is believed to be transmitted vertically.

A virus believed to be a mutation of the infectious laryngotracheitis virus of chickens has been observed to cause a severe upper respiratory and tracheal disease in Amazon parrots and Bourke's parakeet (*Neophema bourkii*). The duration of this disease is variable, but clinical signs have been reported to last up to 9 months. This disease is reported in the USA, but if it occurs in the USA, it is rare. Additionally, tracheitis occurs uncommonly in some birds with Pacheco's disease, and this could be mistaken for Amazon tracheitis.

DUCK VIRUS ENTERITIS (DVE)

This herpesvirus has caused massive die-offs of wild ducks on several occasions in the USA. In Texas, the disease is seen on a smaller scale in the early summer on small community or golf course ponds. Generally, these ponds are densely populated with semi-domestic ducks. A common factor in most of the outbreaks is the presence of Muscovy ducks on the pond. There is a significant amount of controversy surrounding this virus and what to do when an outbreak occurs. In the past, attempts were made to euthanize all ducks on the pond in an effort to eliminate the carrier birds. This approach to control is rarely practical and almost certainly will offend some members of the local community. Additionally, genetic-based tests now support the idea that this virus is widespread in wild waterfowl. At this point, the best method of control is to limit the density of ducks maintained on ponds and, if possible, prevent the introduction of Muscovy ducks onto these ponds.[38]

PIGEON HERPESVIRUS (PHV)

PHV has a worldwide distribution and is a particular problem in racing pigeons, as birds are exposed when closely housed with birds from other lofts prior to each race. When the virus is first introduced to the loft, many birds will be affected. Signs include depression, reluctance to move, protrusion of the third eyelid, conjunctivitis, vomiting, rhinitis, dyspnea, anorexia, weight loss and loose green droppings. Characteristic gray to yellow oral and pharyngeal diphtheritic plaques mark this disease. Birds that survive infection appear to remain infected for life. Virus shedding increases during the breeding season, resulting in infection of squabs. Infected squabs become carriers but disease does not occur, possibly because of passive transfer of antibody.[8,23] Egg transmission of PHV does not occur, and fostering eggs from infected flocks under hens that are not infected may break the infection cycle.[23]

POXVIRUSES

Applied Biology

There are many poxviruses. Each poxvirus has its own host range, a range that may include one or several species of birds. Examples of poxviruses with a limited host range include the canary poxvirus, which affects only canaries and canaries hybridized with other species, and the parrot pox that appears to be confined to South American parrots. A mynah poxvirus appears to affect only mynahs, but mynahs may be susceptible to the starling poxvirus.[24,51] In contrast, poxviruses brought into

Hawaii in non-native species have had a devastating impact on native Hawaiian forest birds. Fowl pox can cause disease in a number of gallinaceous birds and appears to be the cause of pox lesions seen in ostrich chicks in the USA.[39]

Poxviruses require an injury to enter the body. Mosquitoes are the most common vectors for poxviruses, allowing the virus to enter the body through a bite wound. When wild-caught nestling blue-fronted Amazon parrots were held in quarantine, incompletely sanitized hand-feeding utensils were believed to spread the virus from one bird to the next. Generally, canary outbreaks occur in birds that are housed outdoors, but conspecific aggression and cannibalism also may result in rapid dissemination if latently infected birds are present in the flock. Rarely, aerosolized virus in feces or feather dander may directly infect respiratory epithelium.

Clinical Presentation

The practitioner is most likely to see this disease in canaries and chickens housed outdoors and in free-ranging birds and young domestic pigeons. Historically, disease was seen in nestling blue-fronted Amazon parrots held in quarantine. This problem is not seen in captive-raised birds and, because the importation of these birds has essentially ceased, this manifestation of disease is no longer seen in the USA.

Three forms of disease are recognized. The so-called dry pox is the most common disease manifestation. In this form of the disease, lesions are most commonly seen around the face (especially the eyelid and commissures of the mouth), on the feet and under the wings (Fig 32.9). Lesions are raised, smooth to nodular, and may ulcerate. Lesions may be small and clinically insignificant to extensive, deforming lids and even the beak. Extreme cases result in lesions that may appear neoplastic. Secondary superficial bacterial and fungal infections occur in ulcerated lesions. Conjunctivitis and keratitis are common when there is extensive lid involvement. Extensive lesions also may impair vision, resulting in the birds not being able to find food. The lesions develop rapidly over the course of several days, but take up to 6 weeks to regress. When they do begin to regress, they regress rapidly.

Wet or mucosal pox is a second manifestation of some poxvirus infections. It is seen in canaries, lovebirds, mynahs and imported blue-fronted Amazon parrots. The disease in canaries is characterized by a unilateral or bilateral blepharitis, chemosis and conjunctivitis. Typically, there will be considerable ocular discharge and the eyelids will swell shut. Diphtheritic lesions of the oral cavity and trachea develop subsequently. These lesions cause the birds to stop eating, and secondary infections of these lesions are common. Extensive oral lesions and tracheal lesions may obstruct airflow and cause asphyxiation. Mortality in canary aviaries is often high. If treated aggressively, some of these birds will survive but may have persistent ocular lesions. Wet pox sometimes may accompany dry pox.

Systemic pox occurs in canaries as an acute onset disease. Chemosis, depression, anorexia and dyspnea characterize this disease. Birds die within a few days. If they survive, they will develop cutaneous lesions. Necropsy findings include air sacculitis and pneumonia.

Diagnosis

The gross appearance of the lesions in the appropriate species is highly suggestive of the disease. Biopsies of mucous membranes and cutaneous masses will reveal the classic large eosinophilic intracytoplasmic inclusion bodies (Bollinger bodies) (Fig 32.10). Impression smears of these lesions also may reveal these inclusion bodies. Because the lesions often ulcerate, inflammatory cells, bacteria and yeasts are likely to be present in scrapings and impression smears.

Treatment

The poxviruses themselves cannot be treated. In the mild form of the disease, treatment is generally not necessary. Severe lesions may cause the birds to stop eating. In these cases, supportive care (tube-feeding and fluids) is indicated. Ulcerated lesions may become infected and antibiotic therapy is indicated in these birds. Vitamin A supplementation also is suggested to be therapeutic. Surgical removal of pox lesions will only cause scarring and should not be attempted.

Prevention and Control

Poxviruses are transmitted by insect bites or by inoculation into abrasions on the skin or mucous membranes. Raising birds indoors or screening the aviary best controls infections in canaries. A canary pox vaccine[d] is available. Immunization of pigeons with the pigeon pox vaccine also is an important means of control. Racing pigeons are immunized[e] no less than 6 weeks before the racing season begins, as the vaccine is live and will cause the birds to show some signs of illness.

RNA Viruses

PARAMYXOVIRUS 1 (PMV-1) EXOTIC NEWCASTLE DISEASE VIRUS

Applied Biology

Nine serogroups of avian paramyxoviruses are recognized.

Fig 32.9 | The dry form of pox in a chicken.

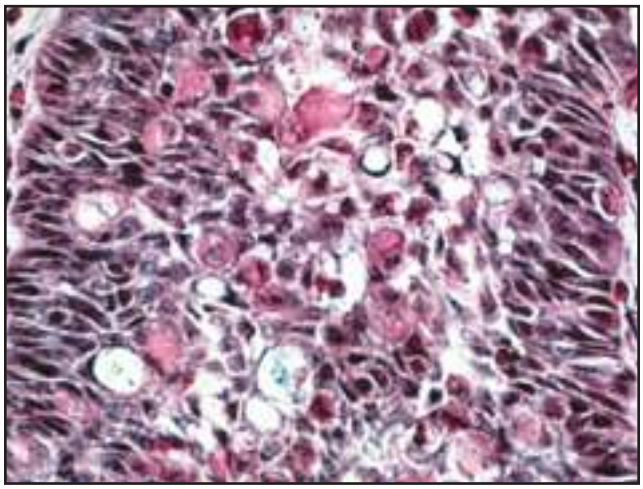


Fig 32.10 | Hematoxylin and eosin-stained section of an avian pox lesion. The round eosinophilic intracytoplasmic inclusions are characteristic of those produced by poxviruses.

Within each paramyxovirus serogroup there may be many strains. PMV-1 or the Newcastle disease virus strains are defined immunologically, genetically and by their pathogenicity to chicken embryos or chicks. Velogenic viruses are highly pathogenic to chickens. These viruses can be divided into those that cause predominately hemorrhagic lesions of the digestive tract — viscerotrophic velogenic Newcastle disease virus (VVND) — and those that cause predominately respiratory and central nervous system lesions — neurotropic velogenic Newcastle disease virus (NVND). Less pathogenic forms that primarily cause disease in young chickens are called mesogenic pathotypes, and those that cause little or no disease in the chicken are called lentogenic pathotypes. The virulence of PMV-1 virus for chickens does not consistently reflect the virulence of the virus in other species, as VVND may cause only mild signs in companion birds, and mesogenic viruses have caused devastating outbreaks of disease in wild birds. The two most important PMV-1 viruses that the practitioner confronts are the highly pathogenic VVND strain known as exotic Newcastle disease virus (END) and pigeon paramyxovirus.[1,24]

EXOTIC NEWCASTLE DISEASE VIRUS

END is a highly virulent virus that has a devastating impact on poultry worldwide. It has been excluded from many nations in the world by strict laws governing the movement of birds. In the USA, all birds entering the country are required to go through a 30-day government-monitored quarantine. Random birds and birds that die in quarantine are tested for END. Illegal movements of parrots across the Mexican border have been responsible for limited outbreaks of END in the USA in the past. The 2003 outbreak occurring in California, Nevada, Arizona and Texas is speculated to have resulted from illegal movements of fighting cocks from Mexico. Infected chickens shed virus through their respiratory system and feces. Inhalation of the virus may be an important means of transmission when birds are in direct contact. Movement of the virus between flocks results from the movement of infected birds and the movement of the virus on contaminated vehicles and other equipment, clothes and feed sacks. END can colonize the conjunctiva of the human eye where it can persist for at least 48 hours, but it is not known if this plays a role in END dissemination. Vertical transmission of virus is not believed to play an important role in END dissemination.

Clinical Presentation and Diagnosis

END in Poultry

END is as likely to appear in small, privately owned collections of chickens as it is in large poultry operations. Therefore, it is entirely possible that private practitioners will be presented with chickens with this reportable disease.

The first indication of END in a flock of chickens may be the sudden onset of mortality with few antemortem signs. Signs are generally non-specific and may involve the gastrointestinal tract, respiratory system, central nervous system or a combination of these systems. Sneezing, coughing, nasal discharge and dyspnea, swelling around the eyes and the head, green diarrhea, depression, weakness, muscle fasciculations, torticollis, paralysis and sudden death are all listed as signs associated with the 2003 outbreak in the southwestern USA. Birds with these signs should be immediately reported to local regulatory veterinarians. Necropsies of these birds are not performed in the clinic but at official diagnostic facilities.

Gross lesions also can be extremely variable. However, lesions that are highly suggestive of END include hemor-

rhagic lesions of the conjunctiva, esophagus, proventriculus, small intestines, ceca and cloaca. Tracheal hemorrhage may be a significant lesion in birds infected with certain strains. Birds with CNS signs may not have gross lesions.

END in Parrots

END has entered the USA on several occasions in smuggled parrots. Although the entry was along the USA-Mexico border, often the disease was not recognized until these parrots had made it to northern states. Outbreaks have typically been associated with nestling yellow-naped (*Amazona ochrocephala auropalliata*) and double yellow-headed Amazon parrots (*A. ochrocephala ochrocephala*). Signs are not specific and include depression, anorexia, weight loss and diarrhea. Respiratory signs may or may not be present. Ataxia, torticollis, opisthotonus, head bobbing, chorea and paralysis may occur in birds surviving the acute form of the disease. The development of neurologic signs in a sick bird should alert the veterinarian that he or she may be dealing with END. Recovery can occur, and recovered birds may shed virus for months to years. If this disease is suspected, a regulatory veterinarian should be immediately contacted.

Prevention and Protection

The first line of defense against END is controlling the movement of birds into a country. When this fails and the disease infects poultry, the situation is catastrophic. Under these circumstances, efforts are made to isolate the virus to a specific geographic area by stopping the movement of birds. Then a door-to-door campaign is undertaken to identify and slaughter all flocks with the disease. An extensive public education effort is necessary to keep this disease from spreading. In affected areas, breeders of companion birds are significantly impacted. Bird shows and sales are banned, and movement of birds out of the quarantined areas is prohibited.

Veterinarians in the quarantined areas and in areas where the disease has the potential of spreading will be asked by aviculturists what they can do to protect their flocks. The most important means of preventing END is to maintain a closed flock. This means that no birds are introduced to the aviary until the outbreak is under control. If owners are outside of the quarantine area and must bring in new birds, these birds are isolated in a separate facility for 30 days before being introduced into a flock. A veterinarian should examine all birds showing any sign of disease. A closed colony also means restricted access to the public. In quarantine areas, visitors who own birds or might have been around birds should be completely barred from the facility. Footbaths should be placed at the entranceway to the aviary, and every effort should be made to make sure that anything brought onto the premises, including food, has not been exposed to other birds.

There is no desire on the part of regulatory veterinarians to unnecessarily kill companion birds. Therefore, in the USA, consideration is given to the specific circumstances of the home or the aviary before action is taken. For maximum protection, birds are housed indoors. If this is not possible, they should be housed in a way that keeps out all wild birds and rodents. A fence preventing loose neighborhood birds from entering the facility is critical. It is highly recommended that all exotic bird owners not keep poultry, or if they do, that the poultry be confined to a cage and isolated the same way the companion birds are isolated. Food and water dishes are covered so the droppings of wild birds cannot contaminate them. An effective biosecurity protocol as described is critical. If END is found in close proximity to an aviary, the aviary is likely to be quarantined. Owners will then be required to follow specific quarantine measures, including having their birds swabbed twice at a 15-day interval. If END is found in a flock, the birds will be euthanized. For more detailed information see the California Animal Health and Food Safety Services Web site at www.cdfa.ca.gov. Immunization of companion birds with Newcastle disease vaccines intended for poultry is not recommended. It also should be noted that immunized poultry still may contract END, and their vaccination status will not impact the outcome of the flock if they are exposed.

PMV-1 IN PIGEONS (PPMV-1)

Applied Biology

Pigeon paramyxovirus-1 (PPMV-1) was first recognized in the early 1980s and has since disseminated throughout the world. It is found in feral pigeon populations and is a significant problem in racing pigeon flocks. Evolution of this virus has resulted in changes in its virulence and clinical manifestations. PPMV-1 is not restricted to pigeons and has been identified in feral Eurasian collared doves (*Streptopelia decaocto*).[45] The strain of virus identified in these doves, however, appears to be adapted to them and varies somewhat from that found in pigeons.[8,24,45]

Clinical Presentation and Diagnosis

PPMV-1 may present in two ways. In the first, neurologic signs predominate. Ataxia and torticollis are the most common signs. The second presentation is polyuria with or without neurologic signs. In both forms of the disease, many birds in a loft will show signs and mortality can be high. Affected birds should be submitted for necropsy to verify the infection.

Prevention

PPMV-1 is disseminated by contact with other pigeons. It is particularly common for it to be introduced to a loft when pigeons are raced, as the birds are in close contact with birds from other flocks prior to the race. Immunization with a PPMV-1 vaccine[f] at least 6 weeks prior to the racing season is recommended on a yearly basis.

OTHER PARAMYXOVIRUSES

Paramyxovirus 2 (PMV-2)

PMV-2 rarely causes disease in companion birds. It has been associated with contact with wild passerine birds, particularly finches, and should be considered a differential for birds that are showing respiratory signs.

Paramyxovirus 3 (PMV-3)

PMV-3 has been recognized in multiple nations around the world, but its basic biology is poorly understood. It appears to cause subclinical infections in some birds and it is these birds that are responsible for the spread of infection. Disease is reported most frequently in *Neophema* species, lovebirds, cockatiels and Amazon parrots, although recently the disease also has been seen in African grey parrots. Signs of infection may be non-specific and precede death by 24 to 48 hours. Birds with a longer duration of signs will develop CNS signs resembling those seen with END. In some cases, respiratory signs also may occur. Chronic infections in *Neophema* species often result in chronic pancreatitis. These birds have voluminous stools that contain undigested starch and fat. PMV-3 infections also are reported in finches with signs of diarrhea, dysphagia, conjunctivitis and dyspnea.[22,24]

Antemortem diagnostic assays have not been consistently effective in identifying birds with disease and asymptomatically infected birds. Traditionally, birds infected with PMVs will produce antibodies that will inhibit virus-induced agglutination of red blood cells. This may be true in some parrots with acute PMV-3, but may not be true in chronic infections. Current efforts to develop an ELISA assay to detect chronically infected birds may help to resolve this issue. Molecular-based technology also may prove to be beneficial in the diagnosis of these infections.

AVIAN INFLUENZA

Avian influenza is a rare disease of companion birds. It is reported to cause non-specific signs as well as signs related to the central nervous system. It is readily recovered from swabs of the cloaca and trachea.

EASTERN EQUINE ENCEPHALITIS (EEE)

The importance of EEE in parrots is unclear. EEE was implicated in a disease of 7- to 12-week-old macaws. The birds showed varied signs from sudden death to decreased appetite with abdominal distention. Grossly, serositis with extensive abdominal effusion was noted. Histologically, hepatic disease, interstitial pneumonia and lymphocytic proventriculitis were consistent findings. This disease has been termed polyserositis. It is the author's experience that this disease is extremely rare. However, edema and ascites occur with some degree of frequency following APV infections, and this disease and its associated lesions may be mistaken for polyserositis.

WEST NILE VIRUS (WNV)

Applied Biology

WNV is a flavivirus that is a member of the Japanese encephalitis virus complex. WNV can be divided into two lineages. Lineage 1 has a wide distribution. It has been isolated from Africa, Europe, the Middle East, India, Australia and the USA. Lineage 2 is confined to Africa. Recently, outbreaks of WNV have occurred in France, Romania, Italy, Russia and Israel. The outbreak in Israel was atypical because the virus had an apparent increased virulence for humans and birds. A strain of WNV essentially identical to the strain found in Israel was first identified in New York City in 1999. By the time of this writing, it has spread widely and has been identified in the majority of states in the USA and in several provinces in Canada. It is expected that it will continue to expand its range into Central and South America in the coming years.[15,20]

Birds are the primary vertebrate host for WNV. Mosquitoes, particularly the members of the genus *Culex*, are the insect vectors. It has been postulated that hippoboscid flies also may be vectors of WNV, but this has not been experimentally tested (M. Taylor, personal communication, 2002). After ingesting blood from a viremic bird, the virus is amplified in the digestive tract and salivary gland of the mosquito. Bird infection occurs when an infected mosquito bites a bird. After a bird is infected, it remains viremic from 4 to 7 days. The magnitude of the viremia depends on the species infected. Crows, magpies, house sparrows (*Passer domesticus*) and other passerines appear to develop the highest concentrations of virus in the blood and have the longest duration of viremia. WNV may persist in the skin after the cessation of viremia, allowing mosquito infection for an as-yet-undetermined period of time after infection.

Preliminary studies suggest that consumption of animals infected with WNV and even ingestion of infected mos-

quitoes will result in infection in some species of birds. Close contact also can result in viral dissemination between birds. Virus can be found in the oral cavity and cloaca of infected birds for 9 or more days after infection, and it is a reasonable hypothesis to assume that it is present in saliva and droppings. WNV persists in the tissues of some species that survive for as long as 14 days after infection. Experiments have not been done to show if it persists longer than this.[16]

Clinical Presentation

WNV can infect and is believed to have caused death in a wide range of species. Birds particularly susceptible to disease caused by WNV include crows, blue jays (*Cyanocitta cristata*), magpies, accipiters, red-tailed hawks (*Buteo jamaicensis*) and several species of northern owls. Ruffed grouse (*Bonasa umbellus*), gulls, house sparrows, robins (*Turdus migratorius*) and mourning doves (*Zenaida macroura*) make a significant number of reported cases, but reported disease in these species is less than 10% of that reported for crows. Naturally and experimentally infected geese appear to be sensitive to disease from WNV. Chickens, turkeys and at least some species of psittacine birds appear to be relatively refractory to disease.

WNV infection has a seasonal distribution in temperate climates. The first cases are seen in the spring and then continue through the summer. A short course of lethargy followed by death may be the only signs seen. Other birds, however, develop signs of central nervous system disease, including ataxia, tremors, weakness, seizures and abnormal head postures prior to death. Anisocoria and impaired vision also were noted in some birds. Observations by practitioners suggest that some birds may show mild signs of illness and then recover.[41]

Diagnosis

Necropsy findings suggestive of WNV disease include intraosseous hemorrhage of the calvaria and hemorrhage of the meninges, mucosa and serosa of the gastrointestinal tract. Splenomegaly is minimal to marked. Focal, linear, or diffuse myocardial pallor also may be present. Microscopic lesions of the brain, heart, pancreas, intestines and spleen are highly suggestive of WNV disease. Infection can be confirmed by isolating the virus from oral and cloacal swabs, brain, heart, kidney, liver, lung and spleen or PCR of these tissues.

Neutralizing virus antibody is detected in the majority of birds within 14 days of infection. Plaque reduction assays and hemagglutination inhibition assays are used to detect antibodies to WNV.[15,16,41]

Prevention

WNV has created panic in the general bird-owning population in the USA and has been of great concern to wildlife and zoo veterinarians. Many zoo veterinarians have elected to use the West Nile virus vaccine[g] for horses to vaccinate birds. The equine dose is 1 ml. Experimental immunization using a 0.5-ml dose given twice 2 weeks apart did not induce an antibody response in cockatiels. A full 1.0-ml dose given three times, 3 weeks apart, did induce an antibody titer in some but not all birds immunized at the Houston Zoo (J. Flanagan, personal communication, 2003). Adverse reactions to the vaccine were not noted. A hemolytic anemia, however, has been reported in lories immunized 1 year after their first set of immunizations the year before. Similar problems have not been noted at the Houston Zoo in birds that have been immunized 2 years in a row (J. Flanagan, personal communication, 2003). Given that it is not known how protective this vaccine is for birds and that at least one institution has seen adverse effects that may be linked to immunization with it, use of this vaccine should be considered a last resort when other mosquito control options are not available. An immunization schedule that has been used has been to give a 1-ml dose of the vaccine every 3 weeks for three immunizations. The vaccine can be divided and given in multiple sites in smaller birds.

Disease in psittacine birds is rare; therefore, immunization of psittacine birds is not recommended at this time. Concerned pet owners should keep their birds indoors in the warmer months of the year.

REOVIRUS

A reovirus was found to be one of several pathogens causing a complex of diseases in recently imported African grey parrots, but also was seen in several other species including cockatoos.[6] Because imported wild birds are rarely seen in practice today in the USA, this disease has essentially disappeared. Wild-caught African grey parrots are, however, still imported into Europe, and this disease continues to be a problem there.

Clinical Presentation

The signs of disease have varied to some degree with the specific outbreak. Signs in outbreaks seen in the USA included depression, weakness, weight loss, edema of the legs and head, and paralysis. Anemia, leukopenia and elevated liver enzymes also are reported. More recent outbreaks in African grey parrots imported into Italy showed respiratory signs, including coughing, nasal discharge and increased lung sounds. The disease had a prolonged course and affected birds died. Australian king parrots also were affected with this disease, but these birds died suddenly.

Natural reovirus infections are often complicated by concurrent infections with multiple other infectious agents, including bacteria, *Aspergillus* spp. and other viruses. Each of these pathogens contributes to the clinical picture of disease.

Diagnosis

Diagnosis in the live bird is probably impossible. However, this disease should be suspected in recently imported wild-caught African grey parrots and birds that are in contact with them. Gross lesions include enlargement of the liver and the kidney, with focal depressed discoloration of the capsular and cut surfaces of the liver. Serosal hemorrhages, enteritis and renal enlargement occur less commonly. Lesions from other pathogens also may be present. Histologic lesions are not specific, and virus isolation is necessary to confirm reovirus infection.

Control

There is no means of control for this disease other than to stop importing wild-caught African grey parrots. Isolation of imported African grey parrots from other imported species may prevent some losses.

RETROVIRUSES

Retroviruses are important causes of disease in waterfowl and gallinaceous birds. Documentation of retrovirus diseases in companion birds is lacking.

Diseases Thought to be Caused by Viruses

PSITTACINE PROVENTRICULAR DILATATION DISEASE (PDD)

Applied Biology

PDD is a disease of unknown etiology, but circumstantial evidence suggests that one or more viruses cause it. Numerous clinical reports document the spread of PDD through a collection after the introduction of birds that subsequently develop disease. Inoculation of tissue homogenates derived from a bird with PDD induced a histologically identical disease in experimentally infected birds. Viruses and virus particles have been identified in a number of birds with PDD. Several viruses have been identified in birds with PDD or in flocks where PDD was a problem. These include eastern equine encephalitis, enterovirus, coronavirus, reovirus, avian paramyxovirus 1 and paramyxovirus 3.[9,10,12] The potential role of paramyxoviruses as the cause of PDD has been strengthened by the development of lesions identical to those seen in PDD in a flock of *Neophema* spp.. They were experiencing an outbreak of PMV-3 infection: these findings were strengthened by the discovery of antibodies to PMV-1 in birds with PDD, and isolation of a low virulent strain of PMV-1 from the spinal cord of 6 of 32 parrots with PDD.[11,12,22] It is hoped that the slow but steady progress made by investigators working with this disease will soon lead to the discovery of its etiologic agent.

Clinical manifestations of the disease are the result of a lymphoplasmacytic inflammation of the nerves of the gastrointestinal tract and brain, spinal cord and peripheral nerves.[5] Lesions also may be found in the heart and adrenal gland. Lesions are rarely diffuse and are variable in their severity. As a result, clinical signs of disease vary from case to case.

Clinical Presentation and Diagnosis

PDD occurs most frequently in African grey parrots, macaws, Amazon parrots, cockatoos and conures, but it is possible that all parrot species are susceptible.[24] It also may occur in non-psittacine birds, as a disease with similar lesions has been observed in Canada geese, a red-tailed hawk and flamingos. There is no sex predilection for PDD. The median age of onset of PDD is 3 to 4 years, but birds as young as 10 weeks and as old as 17 years have been documented with lesions consistent with PDD. Domestically raised and imported birds are equally susceptible to disease. The incubation period for this disease is not known but may be long, as birds isolated from contact with other birds for up to 2 years still have developed this disease.

The number of birds affected by PDD in a collection, the rapidity with which it spreads through a collection and the clinical signs of disease can vary significantly. Birds with the most common form of the disease present with what the owner considers to be an acute onset of disease. There may be a history of regurgitation, anorexia and the presence of undigested seeds in the droppings. Physical examination, however, reveals an emaciated bird. Often the ventriculus can be palpated in the coelomic cavity caudal to the edge of the sternum. Radiographically, the proventriculus is often massively dilated, filling the left side of the coelomic cavity (Figs 32.11, 32.12). Typically, it develops a "J" shape causing the ventriculus to be displaced to the right and ventrally (Fig 32.13). Ultrasound will demonstrate a widely distended proventriculus and ventriculus. Muscle contractions typically are weak, and there is a failure of the junction between the proventriculus and ventriculus to close. Contrast studies show distention of the proventriculus and ventriculus and often the proximal duodenum. Transit time of the contrast material is markedly reduced. Various permutations of this disease may occur, and dilation of the crop, ventriculus, proventriculus or

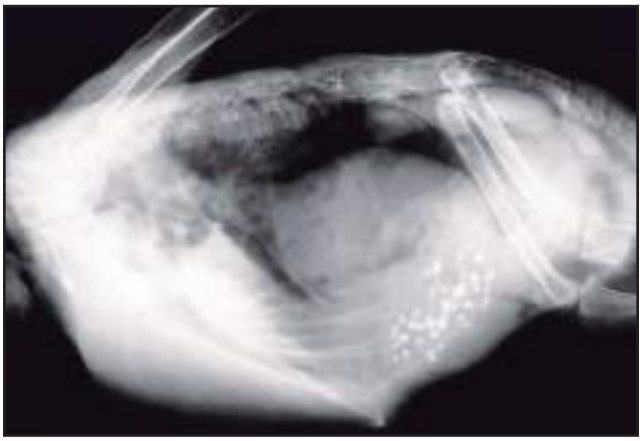


Fig 32.11 | Lateral radiograph of an eclectus parrot reveals proventricular dilatation disease (PDD). Note the massively distended proventriculus.

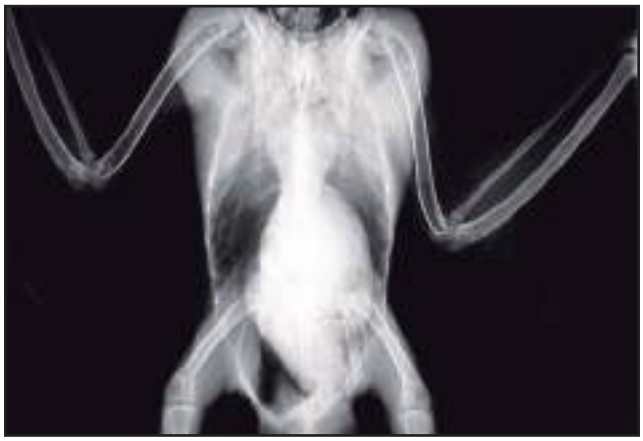


Fig 32.12 | In this ventral dorsal radiograph of the eclectus parrot in Fig 32.11, the hugely distended proventriculus fills the left lateral coelomic cavity.

Fig 32.13 | In the gross necropsy of a eclectus with PDD in Figs 32.11 and 32.12, note the massively distended proventriculus that extends laterally beyond the edge of the left liver lobe. The ventriculus is displaced to the right and ventrally.

duodenum may be seen alone or in combination.

CNS signs may be absent, occur in combination with the gastrointestinal signs, or be the only presenting signs. CNS signs may have an acute onset or be very slowly progressive. They may reflect disease of the brain or the spinal cord, and recent evidence suggests that they also may reflect lesions of the lower motor neurons. Ataxia that may be slowly progressive, proprioceptive deficits, paresis and less commonly paralysis, head tremors and rarely seizures have been reported in birds with PDD.

Clinical pathologic findings are not specific but typically reflect the fact that these birds are starving. High uric acid levels were seen in one of the author's cases, because the bird's neurologic disease prevented it from reaching the water bowl and drinking. Overgrowth of the digestive tract with yeast and gram-negative bacteria occurs as the result of gastrointestinal stasis. Initial attempts at diagnosing this disease by serology and electron microscopy of the feces have subsequently been discontinued.

Demonstrating characteristic lesions in a crop biopsy can make a definitive diagnosis of this disease. Not all birds with PDD have crop lesions, so failure to find lesions in a biopsy does not rule out the disease. It is suggested that biopsying the right cranial ventral aspect of the crop, while making sure that it contains a blood vessel, will increase the probability of a lesion being found.[10] Biopsy of the proventriculus is not indicated, as the proventriculus will already be diseased and is likely to dehisce. A partial thickness biopsy of the ventriculus to get a section of the splanchnic nerves is safe but is rarely done.

The progression of PDD through a flock cannot be predicted. Typically, once it is recognized in the flock, additional birds will develop disease in the following months to years. However, there are times when only an individual bird is affected. Uncommonly, there are outbreaks where multiple birds develop disease within a very short period of time.

As common as this disease is, there are other diseases that can cause nearly identical signs.[2] Gastrointestinal signs identical to those seen in PDD can be caused by neoplasia of the intestines, intestinal foreign bodies and even massive worm burdens that cause intestinal obstruction. Emptying of the proventriculus and ventriculus appears to be inhibited as long as the intestinal tract is distended. As a result, proventricular dilatation occurs in cases of lower bowel obstruction. Inflammatory disease and neoplastic diseases of the ventriculus and proventriculus also can cause gastrointestinal stasis. Another common cause of gastrointestinal stasis is heavy metal poisoning. Heavy metal poisoning is commonly associated with central nervous system signs as well. A chronic wasting disease caused by internal papillomatosis also resembles PDD.

PDD should be considered as a differential in any bird

with CNS disease. Traumatic injuries, heavy metal poisoning, neoplasia, viral, bacterial and fungal infections of the CNS, nutritional deficiencies and hydrocephalus are additional diseases that can cause similar signs.

Treatment

Celecoxib[h], a COX-2 inhibitor, has been advocated as a treatment for PDD.[7] Controlled trials with this drug for the treatment of PDD have not been done, but careful clinical reporting suggests that celecoxib does cause regression of the signs of PDD, and birds that otherwise would have died are still alive up to 2 years after treatment. Successful treatment appears to be more likely if PDD is diagnosed before the bird is extremely debilitated. The recommended treatment protocol is to make a suspension of celecoxib in lactulose and to administer 10 mg/kg orally once a day for a minimum of 6 weeks or until the signs resolve completely. Although lactulose was the first agent used to suspend this drug, others may work just as affectively. Anecdotal information suggests that other COX-2 inhibitors also may be effective.

Additional supportive care will need to be supplied to these birds in addition to celecoxib treatment. Birds that are dehydrated should be given fluids. Liquid diets sometimes will pass through the digestive system while solid diets will not. Secondary yeast and bacterial infections also should be treated.

Prevention and Control

Until the etiologic agent of this disease is identified and an appropriate test for that agent is developed, preventive measures will depend on conservative management practices. A detailed history of the source of all new birds and long quarantine periods (>6 months) will reduce the risk of introducing this disease. Historical findings suggest that this disease is more likely to spread in indoor collections. Keeping breeding birds outdoors when possible and maximizing hygiene, ventilation and cage separation in indoor aviaries may reduce the risk of PDD transmission.

The diagnosis of PDD in a collection can be a devastating blow to the aviculturalist. Remember, however, that sooner or later most large collections of birds that have been assembled from multiple sources will have a case of PDD. Isolating birds in contact with birds that have had PDD may prevent dissemination. Incubator-hatching eggs and hand-raising these chicks in isolation also may break the infection cycle, although this has not been proven scientifically.

SEASONAL MORTALITY IN GREAT-BILLED PARROTS

A disease of unknown etiology is killing great-billed parrots (*Tanygnathus megalorhynchos*) in the USA.[26] A survey of great-billed parrot owners suggests that this disease has killed approximately 50% of the great-billed parrots housed in the southern USA during the period of 1995 to 2000. The disease occurs predominately in Gulf Coast States from July to October. Other species that have had similar signs of this disease include the mealy Amazon parrot (*Amazona farinosa*) and lories. The disease occurs typically in outbreaks, with multiple birds developing signs over a period of 1 to 4 weeks. Both hand-fed nestling parrots and adult parrots are susceptible. Birds housed outside are primarily affected, but disease also has occurred in indoor birds.

Clinical signs of this disease are dramatic. Gastrointestinal motility ceases, so birds stop eating. They may have diarrhea or no droppings at all. Even liquid diets do not pass through the digestive tract. Affected birds rapidly become dehydrated. Birds die within 1 to 3 days, despite aggressive therapy. One case did survive after 2 weeks of treatment, during which time the bird lost nearly 50% of its body weight before its digestive tract began to function again. Intussusception of the intestines occurs in a significant number of cases, resulting in bowel strangulation and necrosis. Blood counts and clinical chemistries show a moderate drop in the total white blood cell count, concentration of the blood from dehydration, an imbalance of electrolytes, elevation in muscle enzymes and an elevation in uric acid, indicating severe dehydration or primary kidney disease or a combination of both.

Consistent specific necropsy findings do not occur. Given the seasonality of this disease, an insect-borne virus has been suspected to cause this disease. However, repeated virus isolation attempts have been unsuccessful. Investigators are currently examining the possibility that a clostridium toxin may be causing this disease.

EPIZOOTIC RESPIRATORY NEOPLASIA OF COCKATIELS

Rapidly growing tumors of the air sacs and lungs occur in cockatiels. Multiple birds in a collection may be affected over a period of several years. The most common clinical sign is the sudden onset of severe dyspnea, although observant owners may see the signs develop over the course of several days. By the time the birds are severely dyspneic, restraint for physical examination or radiographs can be life threatening. If the bird can be radiographed, masses will be seen either in the lung or lungs or in an air sac. These are highly invasive tumors and will penetrate through adjacent vertebrae, compressing the spinal cord. When this occurs, birds present

with a progressive or sudden onset of paresis or paralysis of the legs. Attempts to treat these tumors have not been reported.[24]

These tumors are light tan to yellow. A single pulmonary or air sac mass may be present, but multiple masses are more common. Often they are expanding into the thoracic inlet, resulting in compression of the interclavicular air sac and the trachea. Some of these tumors contain a considerable amount of well-differentiated fat tissue; they may outwardly appear as lipomas. The appearance of the nuclei of the neoplastic cells resembles the nuclear changes seen in cells infected with APV. A history of APV disease has been documented in some of these aviaries, but APV DNA has not been identified in these tumors.

Products Mentioned in the Text

a. Psittimune APV Avian Polyomavirus Vaccine, Biomune, Lenexa, KS, USA 913-894-0230, www.BiomuneCompany.com
b. Zovirax, Burroughs Wellcome Co, Phoenix, AZ, USA 8572-9349
c. Psittimune PDV Pacheco's Disease Vaccine, Biomune, Lenexa, KS, USA 913-894-0230, www.BiomuneCompany.com
d. Poximune Canary Pox Vaccine, Biomune, Lenexa, KS, USA 913-894-0230, www.BiomuneCompany.com
e. Pigeon Pox vaccine, Maine Biological Laboratories, Waterville, ME, USA, 207-873-3989, www.mainebiolab.com
f. Paramyxovirus 1 Vaccine, Maine Biological Laboratory, Waterville ME, USA, 207-873-3989, www.mainebiolab.com
g. West Nile Virus Vaccine, Fort Dodge Laboratories, Fort Dodge, IA, USA, 800-477-1365, www.wyeth.com/divisions/fort_dodge.asp
h. Celebrex, Pharmacia, Pfizer, www.celebrex.com

References and Suggested Reading

1. Alexander DJ: Newcastle disease and other avian Paramyxoviridae infections. *In* Calnek BW (ed): Diseases of Poultry 10th ed. Ames, Iowa State University Press, 1997, pp 541-569.
2. Antinoff N: It isn't always PDD: Three cases with proventricular enlargement. Proc Assoc Avian Vet, 2001, pp 35-37.
3. Barton CE, Phalen DN, Snowden KF: Prevalence of microsporidial shedding in asymptomatic lovebirds: Evidence for a potential emerging zoonosis. J Avian Med Surg, in press, 2003.
4. Bassami MR, et al: Genetic diversity of beak and feather disease virus detected in psittacine species in Australia. Virology 20:392-400, 2001.
5. Berhane Y, et al: Peripheral neuritis in psittacine birds with proventricular dilatation disease. Avian Pathol 30:563-570, 2001.
6. Conzo G, et al: Reovirus infection in two species of Psittaciformes recently imported into Italy. Avian Pathol 30:43-47, 2001.
7. Dahlhausen B, Aldred S, Colaizzi E: Resolution of clinical proventricular dilatation disease by cyclooxygenase 2 inhibitor. Proc Assoc Avian Vet, 2002, pp 9-12.
8. Dorrestein GM: Viral infections in racing pigeons. Proc Assoc Avian Vet, 1992, pp 244-257.
9. Gough RE, Harcourt-Brown N: Psittacine proventricular dilatation disease from a United Kingdom perspective. Midwest Avian Res Expo, 1998, pp 97-103.
10. Gregory CR, et al: Progress in understanding proventricular dilatation disease. Proc Assoc Avian Vet, 2000, pp 269-275.
11. Grund C, Grimm F, Kösters J: Serological studies on persistent PMV-1 infection associated with PDD. Proc Assoc Avian Vet, 1999, pp 19-23.
12. Grund CH, et al: Avian paramyxovirus serotype 1 isolates from the spinal cord of parrots display a very low virulence. J Vet Med B 49:445-451, 2002.
13. Guerin J-L, et al: A novel polyomavirus (goose hemorrhage polyomavirus) is the agent of hemorrhagic nephritis enteritis of geese. J Virol 74:4523-4529, 2000.
14. Hattermann K, et al: A method to diagnose pigeon circovirus infection in vivo. J. Virol Meth 104:55-58, 2002.
15. Johnson DJ, et al: Detection of North American West Nile virus in animal tissue by a reverse transcription-nested polymerase chain reaction assay. Emer Infect Dis 4:739-741,1998.
16. Komar N, et al: Experimental infection of North American birds with the New York 1999 strain of West Nile virus. Emer Infect Dis 9:311-322, 2003.
17. Johne R, Müller H: Avian polyomavirus in wild birds: Genome analysis of isolates from Falconiformes and Psittaciformes. Arch Virol 143:1501-1512, 1998.
18. Johne R, et al: Herpesviral, but no papovaviral sequences, are detected in cloacal papillomas of parrots. Arch Virol 10:1869-1880, 2002.
19. Lafferty SL, et al: Avian polyomavirus infection and disease in a green aracaris (*Pteroglossus viridis*). Avian Dis 43:577-585, 1999.
20. Lanciotti RS, et al: Complete genome sequences and phylogenetic analysis of West Nile virus strains isolated from the United States, Europe, and the Middle East. Virology 298:96-105, 2002.
21. Latimer KS, et al: Diagnosis of avian adenovirus virus infections using DNA *in situ* hybridization. Avian Dis 41:773-782, 1997.
22. Loudis BG: PMV-3 outbreak: Presentation, diagnosis and management. Proc Assoc Avian Vet, 1999, pp 223-227.
23. Messana M, Kösters J, Grund C: Studies on reactivation and transmission of pigeon herpes virus (PHV) for raising PHV-free pigeons (*Columba livia* dom.). Avian Pathol 26:859-864, 1997.
24. Phalen DN: Viruses. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 281-322.
25. Phalen DN: Avian polyomavirus: My thoughts. Amer Fed Avicul Watchbird 25:28-39, 1998.
26. Phalen DN: Mortality in great-billed parrots. Research forum. Assoc Avian Vet, 2002.
27. Phalen DN, Tomaszewski E, Wilson VG: Internal papillomatosis: A herpesvirus connection. Proc Assoc Avian Vet, 1998, pp 45-48.
28. Phalen DN, et al: Avian polyomavirus: More pieces to the puzzle. Proc Assoc Avian Vet, 1998, pp 151-156.
29. Phalen DN, et al: Genetic diversity in twenty variants of the avian polyomavirus. Avian Dis 43:207-218, 1999.
30. Phalen DN, et al: Viremia, virus shedding, and antibody response during natural avian polyomavirus infection in parrots. J Am Vet Med Assoc 217:32-36, 2000.
31. Phalen DN, et al: Diagnosis of parrots infected with Pacheco's disease viruses. Proc Assoc Avian Vet, 2001, pp 87-89.
32. Phalen DN, et al: Genetic diversity of avian polyomaviruses: Clinical implications. Proc Assoc Avian Vet, 2001, pp 171-172.
33. Raidal SR, et al: A serologic survey for avian polyomavirus and Pacheco's disease virus in Australian cockatoos. Avian Pathol 27:263-268, 1995.
34. Ritchie BW, et al: Safety, immunogenicity, and efficacy of an inactivated avian polyomavirus vaccine. Amer J Vet Res 59:143-148, 1998.
35. Ritchie BW, et al: Documentation of a PBFD virus variant in lories. Proc Assoc Avian Vet, 2000, pp 263-268.
36. Schmidt RE, Reavill DR, Phalen DN: Pathology of Pet and Aviary Birds. Ames, Iowa State Press, 2003.
37. Schoemaker NJ, et al: Severe leukopenia and liver necrosis in young African grey parrots (*Psittacus erithacus erithacus*) infected with psittacine circovirus. Avian Dis 44:470-478, 2000.
38. Shawky S, Schat KA: Latency sites and reactivation of duck virus enteritis virus. Avian Dis 46:308-313, 2002.
39. Shivaprasad HL, et al: Genetic and antigenic characterization of a poxvirus isolate from ostriches. Avian Dis 46:429-436, 2002.
40. Shivaprasad HL, et al: Retrospective study of cytomegalovirus-like infection in finches. Proc Assoc Avian Vet, 2002, pp 209-210.
40a. Styles DK, Tomaszewski EK, Phalen DN: A novel psittacid herpesvirus in African grey parrots (*Psittacus erithacus erithacus)*. Avian Path. (Accepted), 2005.
41. Steele KE, et al: Pathology of fatal West Nile virus infections in native and exotic birds during the 1999 outbreak in New York City, New York. Vet Pathol 37:208-224, 2000.
42. Strunk A, et al: Pathobiology and testing recommendations for psittacine circovirus 2 in lories. Proc Assoc Avian Vet, 2002, pp 45-47.
43. Styles D, Phalen DN, Tomaszewski E: Elucidating the etiology of avian mucosal papillomatosis in psittacine birds. Proc Assoc Avian Vet, 2002, pp 175-178.
44. Styles D, Tomaszewski E, Phalen DN: Psittacid herpesviruses in mucosal papillomas of Neotropical parrots. Manuscript in preparation. 2003.
45. Terregino C, et al: Characterization of Newcastle disease virus isolates obtained from Eurasian collared doves (*Streptopelia decaocto*) in Italy. Avian Pathol 32:63-68, 2003.
46. Todd D, et al: Nucleotide sequence-based identification of a novel circovirus of canaries. Avian Pathol 30:321-325, 2001.
47. Todd D, et al: Evaluation of polymerase chain reaction and dot blot hybridization tests in the diagnosis of pigeon circovirus infections. Vet Microbiol 89:1-16, 2002.
48. Tomaszewski E, Wilson VG, Phalen DN: Synchronicity, papillomas, and herpes disease. Proc Assoc Avian Vet, 1999, pp 219-222.
49. Tomaszewski E, Kaleta EF, Phalen DN. Molecular phylogeny of the psittacid herpesviruses causing Pacheco's disease: Correlation of genotype with phenotypic expression. J Virol, submitted, 2003.
50. Tomaszewski E, et al: Detection and heterogeneity of herpesviruses causing Pacheco's disease in parrots. J Clin Micro 39:533-538, 2001.
51. Tripathy DN, Reed WM: Pox. *In* Calnek BW (ed): Diseases of Poultry 10th ed. Ames, Iowa State University Press, 1997, pp 643-660.
52. Vereecken M, de Herdt P, Ducatelle R: Adenovirus infections in pigeons: A review. Avian Pathol 27:13-17, 1998.
53. Ypelaar I, et al: A universal polymerase chain reaction for the detection of psittacine beak and feather disease virus. J Clin Micro16:141-148, 1999.

CHAPTER
33

Updates in Anesthesia and Monitoring

THOMAS M. EDLING, DVM, MSpVM

Although avian anesthesia has made significant advances in the past several years, anesthetizing a bird should never become a procedure to be taken lightly. Unlike common anesthetic procedures in mammals, birds are seldom clinically healthy when anesthesia is utilized. In addition, the anatomy and physiology of birds greatly complicates anesthetic risk. Even a procedure that lasts only 5 minutes can easily put a bird into a hypercapnic state that can be fatal. This being said, birds are now commonly being anesthetized for periods exceeding 2 hours with very little morbidity and mortality. These advances are due to the use of inhalation agents, such as isoflurane[b] and sevoflurane[c], plus better monitoring techniques. The newer monitoring techniques effectively control apnea, hypothermia and hypoventilation, the most commonly experienced problems during anesthesia. As in mammals, there is no formula involving respiratory rate and tidal volume that can be employed to correctly determine the ventilatory status of the patient. The only way to accurately assess ventilation in any animal is through some measure of arterial carbon dioxide. Recent advances in avian anesthesia have shown that capnography (the continuous graphing of the carbon dioxide content of expired air) can be effectively used to monitor anesthesia in birds, and the use of intermittent positive pressure ventilators, electrocardiograms (ECG), pulse oximetry and doppler flow probes have greatly advanced the science of anesthesia in avian species.

Systems Overview

PULMONARY SYSTEM

The avian respiratory system is unique to the animal kingdom. It employs two separate and distinct functional systems for ventilation and gas exchange. The ventilatory components consist of the larynx, trachea, syrinx, intra- and extrapulmonary primary bronchi, secondary bronchi, parabronchi, air sacs, skeletal system and respiratory muscles. The gas exchange system utilizes two types of lungs, the paleopulmonic and neopulmonic. Both of these lung types rely on the parabronchi for gas exchange.

Anatomy

The oronasal cavity is separated from the trachea by the larynx, which opens into the trachea through the slot-like glottis. The larynx extends into the pharynx and attaches directly to the base of the tongue. It consists of four laryngeal cartilages: the cricoids, procricoid, and paired arytenoid cartilages.[19] This is different from the mammalian larynx in that the thyroid and epiglottic cartilages are absent in birds. The functions of the larynx are to open the glottis during inspiration, aid in swallowing, modulate sound and act as a barrier to keep inappropriate matter from entering the trachea.[19] This anatomy makes intubation of most companion avian species easier than mammalian intubation, as the clinician can visualize the glottis and pass the endotracheal tube with relative ease.

The trachea probably exhibits the greatest degree of variability of any of the respiratory system components. The avian trachea consists of four layers: mucous membrane, submucosa, cartilage and adventitia. The cartilaginous layer forms complete rings throughout the length of the trachea.[19] The internal lining of the larynx and trachea is composed of simple and pseudostratified, ciliated columnar epithelium, with simple alveolar mucous glands and goblet cells.[19]

Unusual tracheal variations seen in some avian species include modifications such as an inflatable sac-like diverticulum in emus and ruddy ducks, tracheal bulbous expansions in many male Anseriformes, and the complex tracheal loops and coils located in the caudal neck, keel, thorax or a combination. Another unusual tracheal modification is the trachea is divided by a septum in the cranial portion into two tubes in some penguins, mallards and petrels.[19] Most birds commonly seen in a companion avian practice do not have significant tracheal variations.

The avian tracheal modifications create significant functional consequences relating to anesthesia. The most important is tracheal dead air space. The "typical" bird trachea is longer and wider than that of comparably sized mammals, which increases the tracheal dead space volume. Birds compensate for this increased dead air space volume with a deeper and slower breathing pattern.

The syrinx is the sound-producing organ in birds, located at the bifurcation of the trachea. The shape, size and location of the syrinx in the coelomic cavity vary greatly depending on species. The location and structure of the syrinx explains why an intubated bird can still produce sound. The tracheal tube does not pass through the syrinx, thus the syrinx remains functional if enough force is applied to the air sacs, as in resuscitation efforts, or if the bird vocalizes due to a light plane of anesthesia.

The avian bronchial system is composed of three structural levels.[12] The primary bronchus comprises two components: the extrapulmonary portion that extends from the syrinx to the lung parenchyma and the intrapulmonary portion, which extends throughout the length of the lung ending at the abdominal air sac. In most birds, the secondary bronchus is divided into four groups: medioventral, mediodorsal, lateroventral and laterodorsal, based on their anatomic location.[20]

Most companion birds have nine airs sacs comprised of the paired cervical, single clavicular, paired cranial thoracic, paired caudal thoracic and paired abdominal air sacs. Based on their bronchial connections, air sacs are separated functionally into two groups. The cranial group consists of the cervical, clavicular and cranial thoracic air sacs, and the caudal group comprises the caudal thoracic and abdominal air sacs. Functionally, the air sacs serve as bellows to the lung. They provide airflow to the relatively rigid avian lung during both inspiration and expiration; but, the air sacs are poorly vascularized and contribute less than 5% of the total respiratory system gas exchange.[17]

VENTILATION

In birds, unlike in mammals, both inspiration and expiration require muscular activity. When the inspiratory muscles contract, the internal volume of the coelomic cavity increases. Pressures within the air sacs become negative, relative to ambient atmospheric pressure, and air flows from the atmosphere into the respiratory system, across the gas exchange surfaces of the lungs and into the air sacs.[12] The reverse process occurs with contraction of the expiratory muscles: air flows from the air sacs across the gas exchange surface of the lungs and out to the atmosphere. During the resting phase, the system stops halfway between inspiration and expiration.

Gas Exchange

In the avian patient, gas exchange occurs in the parabronchus. The parabronchi are long, narrow tubes that have numerous openings into chambers termed atria. The atria have funnel-shaped ducts (infundibula) that lead to air capillaries.[25] The air capillaries form an anastomosing, three-dimensional network interlaced with a similarly structured network of blood capillaries where gas exchange takes place.[12]

There are two types of parabronchial tissues in the avian lung. The paleopulmonic parabronchial tissue, consisting of parallel, minimally anastomosing parabronchi, is found in all birds. The second type, neopulmonic parabronchi tissue, is a network of anastomosing parabronchi located in the caudolateral portion of the lung.[25] Penguins and emus have only paleopulmonic parabronchi. Pigeons, ducks and cranes have both types of tissues, with the neopulmonic parabronchi accounting for 10 to 12% of the total lung volume in these species.[12] In songbirds and parrots, the neopulmonic parabronchi are more developed and may account for 20 to 25% of the total lung volume.

During inspiration and expiration, the direction of gas flow in the paleopulmonic parabronchi is unidirectional, whereas the direction of gas flow in the neopulmonic parabronchi is bi-directional. A process involving aerodynamic valving controls the direction of gas flow through the intrapulmonary primary bronchus, secondary bronchi and the paleopulmonic parabronchi.[26]

The gas exchange system in the avian parabronchi is best described with a crosscurrent model. In this system, parabronchial gas is constantly changing in composition as it flows along the length of the lung. The degree to which capillary blood is oxygenated and carbon dioxide is eliminated depends on where the blood contacts the blood-gas interface. Another important note in this crosscurrent design is that it is not dependent on the direction of gas flow. Gas exchange occurs equally well with gas flow originating from either end of the parabronchus.

The respiratory gas volume per unit body mass of the avian respiratory system is 2 to 4 times that of a dog. Although the volume of gas in the parabronchi and air capillaries is only 10% of the total specific volume in birds, in the mammalian lung it is 96%. This results in a small functional residual capacity (FRC) in birds and makes periods of apnea critical. Without airflow through the lungs, gas exchange does not occur and the physiological acid-base balance maintained by the respiratory system is made ineffective. Apneic episodes will cause significant problems during anesthesia and must be carefully managed.

Control

The control of ventilation in birds is complex and poorly understood. In both birds and mammals, respiration originates as a rhythmic motor output from the central nervous system. Reflexes, in response to changes in activity and the environment, modulate the basic rhythm. Birds also possess central and arterial chemoreceptors involved in ventilatory control. Central chemoreceptors respond to changes in the partial pressure of arterial carbon dioxide ($PaCO_2$) and pH, while arterial chemoreceptors are sensitive to changes in the partial pressure of arterial oxygen (PaO_2), $PaCO_2$ and pH. These arterial receptors account for the ventilatory response to hypoxia in birds and mammals. In addition, birds have a unique group of receptors, termed intrapulmonary chemoreceptors (IPCs), located in the parabronchial mantle. IPCs are acutely sensitive to CO_2 and insensitive to hypoxia, and affect the rate and depth of breathing on a breath-to-breath basis.

CARDIOVASCULAR SYSTEM

The avian heart is a four-chambered muscular pump very similar in function and physiology to that of mammals. In contrast, birds have a proportionally larger heart, larger stroke volume, lower heart rate, higher blood pressure and a higher cardiac output than comparably sized mammals.[30] Birds also possess both right and left cranial vena cavae, their aorta arches to the right and they have a tricuspid (right AV) valve with a single leaf. Sympathetic and parasympathetic nerves innervate the atria and ventricles.[30]

The main cardiac sympathetic neurotransmitters are norepinephrine and epinephrine.[30] Excitement increases the concentration of these neurotransmitters, especially epinephrine, which has significant implications because inhalant anesthetics, especially halothane, sensitize the myocardium to catecholamine-induced cardiac arrhythmias. Hypoxia, hypercapnia and anesthetics each depress cardiovascular function.

The conduction system of the avian heart consists of the sinoatrial node, the atrioventricular node and its branches, and Purkinje fibers.[30] Birds have type 2 Purkinje fibers that completely penetrate the ventricular myocardium. Ventricular activation appears to originate from both the endocardium and epicardium and vice versa, an adaptation thought to facilitate synchronous beating at high heart rates.

Special Considerations

AIR SACS AND POSITIONING

Air sacs in birds, as previously discussed, do not contribute significantly to gas exchange, and therefore do not play a major role in the uptake of inhalation anesthetics, nor do they accumulate or concentrate anesthetic gases.

The position of the patient during anesthesia can alter ventilation. While a bird is in dorsal recumbency, normal ventilation is reduced. This is primarily due to the weight of the abdominal viscera compressing the abdominal and caudal thoracic air sacs, and reducing their effective volume. While in dorsal recumbency, adequate ventilation can be achieved through the use of intermittent positive pressure ventilation (IPPV).

Because of the flow-through design and the crosscurrent gas exchange in the avian respiratory system, it is possible to provide inhalation anesthetics from either the trachea via the glottis or a cannulated air sac. Cannulation also offers an effective means to ventilate an apneic bird or patient with an obstructed trachea.

DIVE RESPONSE

In some birds, especially waterfowl, episodes of apnea and bradycardia can occur during induction of anesthesia due to a physiologic response termed a dive response. It is thought to be a stress response mediated by stimulation of trigeminal receptors in the beak and nares. It can be elicited simply by placing a mask snugly over a bird's beak without anesthetic gas involvement. The dive response usually happens during the initial phase of induction of gas anesthesia with a mask. If the dive response occurs, turn off the anesthetic gas, remove the mask from the bird's head and provide oxygen to the bird's beak via open anesthetic mask until the bird has recovered.

INTERMITTENT POSITIVE PRESSURE VENTILATION

Providing manual IPPV during inhalation anesthesia is a tried-and-true method for maintaining an animal in a normal physiologic state. The next step in the progression of avian anesthesia is the use of mechanical ventilators to provide IPPV. Mechanical devices can provide a much greater consistency in this effort and free the clinician or technician for other critical duties during surgical procedures. There are two types of assisted ventilation machines — volume-limited and pressure-limited. The volume-limited delivers a set tidal volume, regardless of airway pressure, and the pressure-limited delivers a tidal volume until a predetermined airway pressure is reached. In pressure-limited ventilation, if the airway becomes occluded, the machine will deliver a lower tidal volume for the same airway pressure. Similarly, changes in lower respiratory compliance over time may alter tidal volume at a given pressure. Thus, gradual hypoventilation may result without the operator becoming aware. In contrast, if the endotracheal tube becomes occluded during pressure regulated volume-limited ventilation, the resulting high airway pressure triggers an alarm that will alert the operator. If the system leaks, gradual hypoventilation may develop due to a loss of a portion of each tidal volume.

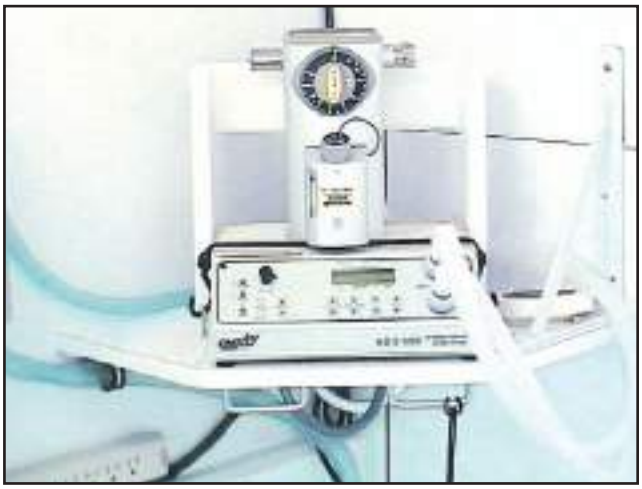


Fig 33.1 | Sevoflurane vaporizer[a] mounted to the ADS (anesthesia delivery system) 1000 mechanical ventilator. The ventilator allows mask induction, and then changes to an endotracheal tube with the flip of a switch.

Use caution when using all ventilators during surgical procedures in the coelomic cavity, where there is a large opening in an air sac. Because volume-limited ventilators deliver only a preset volume of anesthetic gas, it is almost impossible to control ventilation and anesthesia because most of the anesthetic gas leaks from the opening in the air sac. Although difficult, it is possible to control ventilation under the same circumstances with a pressure-limited ventilator because this type of system will continue to supply anesthetic gas until the preset pressure is achieved. There will be continuous flow from the ventilator through the respiratory tract if there is a large rent.

Pressure- and volume-limited ventilation machines are commonly used in veterinary practice, and manufacturers offer mechanical ventilators designed specifically for small or laboratory animals. These machines are excellent choices for avian anesthesia (Fig 33.1).

It has been hypothesized that it is possible to reverse the direction of gas flow within the avian lung during PPV. Because the crosscurrent gas exchange system is not

dependent on the direction of flow, a reversal of gas flow will not adversely affect gas exchange.[9]

General Considerations

HISTORY

A complete and thorough history, obtained from the bird's owner, is perhaps the most important information one can acquire prior to anesthesia. The history will provide invaluable information concerning husbandry issues and events that will lead you toward specific concerns. Observant owners will recognize changes in their bird's behavior prior to the clinical manifestation of disease. The history should include all parameters concerning husbandry. These include cage size, cage construction, cage grate, substrate, cage location, perches, toys, food/water bowls, other animals in the household, exposure to other birds (boarding, visits, bird club meetings, etc), exposure to toxins, supervision, changes in household, cleaning (agents used, frequency, etc), and vaccination history, to name a few pertinent factors.

PHYSICAL EXAMINATION

After obtaining the history, quietly observe the bird as it perches in its cage. Watch for signs of awareness and attention to its surrounding environment, body position, feather condition, and respiratory rate and depth. After the initial observations, a thorough physical examination should be performed. Remove the bird from its cage and examine it for any abnormalities. Special attention should be given to the nares, oral cavity, choanal slit, glottis, abdomen, cloaca and muscle mass covering the keel. The heart, lungs and air sacs should be auscultated for signs of disease. Observation of the bird's respiratory rate and depth after handling, when the bird is back on its perch, also provides valuable information on the respiratory condition of the patient. If the physical exam, history or other parameters warrant, blood should be collected for evaluation. Refer to Chapter 6, Maximizing Information from the Physical Examination.

ACCLIMATION

Placing a bird into a new or different environment is usually a stressful situation. When a bird is stressed, it generally will attempt to hide any signs of illness. This can make observing the bird for signs of infirmity difficult. If time and physical parameters allow, acclimating the bird prior to anesthesia can help. If the bird does become acclimated to the new environment, signs of disease that were masked while the bird was stressed may become evident, but they can rarely hide respiratory distress. It also is possible that the patient will not become acclimated to the new surroundings during the period of time allowed. In these cases, the bird's stress level will elevate and its disease state may worsen. The bird may refuse to eat and drink, and will need to be given supportive care. When managing the highly stressed bird, it is best to bring it into the clinic as closely as possible to the time of anesthesia, while still allowing time for a complete physical evaluation. Preliminary testing such as blood work can be done prior to the day anesthesia will be performed.

FASTING

Ensuring that the crop is empty prior to anesthesia is very important due to the hazards associated with regurgitation. There is controversy as to the length of time a bird should be fasted prior to induction. Because of a bird's high metabolic rate and poor hepatic glycogen storage, it has been recommended that fasting be limited to no more than 2 to 3 hours. However, when working with cockatiel-sized and larger birds in good physical condition, removing their food the night before and their water 2 to 3 hours prior to anesthesia does not appear to be harmful.[7] Always palpate the crop before anesthesia. If there are residues, especially liquid, they can be aspirated prior to anesthesia.

RESTRAINT

The use of proper physical restraint is important in the management of avian patients. Improper capture or restraint can result in serious physical trauma such as fractures, lacerations and dislocations. Owners will judge the veterinarian's clinical abilities on how their bird looks after its visit. Birds must be restrained so that the legs and wings are not allowed to flail. In psittacine restraint, the head must be controlled at all times. In species such as macaws with bare cheek patches, restraining the bird by placing your fingers on these cheek patches can cause bruising, which inevitably will cause the owners to lose confidence in your abilities. Each avian species has its own unique defense mechanism that needs to be addressed to ensure proper restraint. The beak of a parrot can cause significant soft tissue injury and its feet can inflict painful scratches. Birds of prey use their talons as their defense mechanism. Their feet must be carefully restrained or serious injury will result. Although most raptors do not bite as a general rule, great horned owls can use their beaks very effectively. Cranes and herons typically use their long, pointed beaks to attack the eyes of their handlers.

The most common method of restraint is a soft, tightly-woven towel appropriately sized to be able to encircle the patient. The bird is carefully grasped through the

Fig 33.2 | Non-rebreathing circuit. The non-rebreathing anesthesia circuits allow for the removal of CO_2 with high oxygen flow (100-200 ml/kg/minute). This type of circuit also is capable of almost instantaneous changes in the percentage of anesthetic gas delivered to the patient when the vaporizer settings are adjusted.

Fig 33.3 | Standard small-mammal face masks with latex gloves fitted across the openings, producing tight-fitting induction masks. The hole should be slightly smaller than the neck size of the animal being induced, and the animal's head should fit completely inside the mask. When fitted correctly, it is possible to provide intermittent positive pressure ventilation in some circumstances.

towel, being careful to control the animal while allowing the sternum freedom of movement for respiration. The animal can either be left in the towel or removed from the towel, depending on the skill and comfort level of the clinician or technician restraining the bird. When using a towel for restraint during an examination it is easy for a bird to become hyperthermic, so be careful to monitor the patient for signs of overheating. During a physical exam, a bird of normal weight will start to pant if wrapped in a towel for more than 5 minutes; therefore, obese birds should be examined within 1 to 2 minutes.

Inhalant Anesthetics

BREATHING CIRCUITS AND GAS FLOW

Non-rebreathing circuits such as Magill, Ayre's T-piece, Mapleson systems a-f, Jackson-Rees, Norman mask elbow and Bain circuit are typically used during companion bird anesthesia. These systems rely on a relatively high fresh gas flow rate to remove carbon dioxide. They offer advantages over a rebreathing circuit such as an almost immediate response to vaporizer setting changes and a lower resistance to breathing. Oxygen flow in a non-rebreathing circuit should be 2 to 3 times the minute ventilation or 150 to 200 ml/kg per minute[23] (Fig 33.2).

INDUCTION METHODS

Mask induction techniques are used with companion birds in most circumstances. The masks can range from commercially available small animal masks to plastic bottles and syringe cases (Fig 33.3). The size and shape of the mask is dependent upon the size and shape of the bird's head and beak. The mask should be stable and as small as possible. Eye lubrication should be administered at this time. During induction, the entire head of the bird should be placed inside the mask, being careful not to cause eye and beak damage.

A disposable latex glove can be placed over the mask opening with a central hole cut for insertion of the head. The hole should be roughly the same size as the bird's neck (Fig 33.4). When fashioned properly, the glove will provide a seal around the patient's neck tight enough to allow for positive pressure ventilation. The tight seal also helps reduce the amount of waste gas released into the environment (Fig 33.5). Too tight of a seal may occlude major vessels.

Other methods of induction have been successfully used. Clear plastic bags can be used to completely enclose a cage and induce anesthesia for patients that are difficult to control. Anesthetic chambers also have been used for induction. Both of these techniques can be effective, but have their disadvantages. The anesthetist cannot physically feel how the bird is responding during induction or have the ability to auscultate the animal using these methods. In addition, the bird can injure itself when not being restrained during the excitement phase of anesthesia.

Several induction techniques have been described in the literature,[1] including the use of preoxygenation techniques and slowly increasing the concentration of the gas anesthetic agent until the desired effect has been attained. This method does induce anesthesia, but has the disadvantage of taking longer to achieve a loss of

Fig 33.4 | Induction masks can be made from a variety of existing articles such as plastic bottles, syringe cases, pill containers and plastic containers. Only the shape of the animal's head and your imagination limit the variety of induction masks.

Fig 33.5 | Mask induction of a rose-breasted cockatoo. Note that the head is completely enclosed in the mask and the opening of the mask is covered with a latex glove.

consciousness and may increase the excitement level of the patient. The most common method is to simply place the induction mask over the head of the bird with a high oxygen flow rate (1-2 L/minute), adjust the anesthetic vaporizer concentration to a high concentration (depending on the anesthetic agent, 4 to 5% for isoflurane[b], 7-8% for sevoflurane[c], individual setting is necessary) and securely restrain the bird for the few seconds it takes to achieve induction. The vaporizer setting is then reduced to a setting near the minimum anesthetic concentration (MAC). When performed correctly, this method reduces the induction time and stress level on the bird.

INTUBATION

When short (10 minutes or less), non-invasive procedures such as radiography, blood collection, and physical examinations are to be performed, intubation is usually not necessary. If the procedure is to be invasive or longer than 10 minutes, intubation of the patient can be crucial.

Most birds 100 g in body weight and larger can be intubated with minimal difficulty. It is possible to intubate birds as small as 30 g in body weight, but they present a much greater challenge. In these smaller birds, an endotracheal tube can be fashioned using a red rubber catheter of an appropriate diameter (Fig 33.6). Some birds have unique anatomical features, such as the ventral crest in some hornbills and median tracheal septum in some penguins, which can interfere with intubation. In psittacine birds, intubation can be difficult because the glottis is located at the base of the fleshy tongue.

Care must be taken during intubation to ensure that the trachea is not damaged. The endotracheal tube should provide a good seal with the glottis, but should not fit tightly. If the tube is cuffed, the cuff should not be inflated or should be inflated with tremendous care. An overinflated cuff can cause damage to the tracheal mucosa because of the complete cartilaginous rings. Tracheal damage may not become apparent for several days following intubation, when the bird presents with dyspnea due to a stricture in the lumen of the trachea.

The most common problem associated with intubation of companion birds is airway obstruction. Small endotracheal tube diameters and cold, dry gases increase the probability of a complete or partial airway obstruction. As the airway becomes occluded, the expiratory phase of ventilation is prolonged.[12] The obstruction can be corrected by extubating the patient and cleaning the endotracheal tube. Positive pressure ventilation, even during spontaneous breathing, can help prevent the formation of endotracheal mucus plugs, even in the smallest patients.

Once the patient is intubated, the endotracheal tube should be securely attached to the lower beak. This will help prevent the endotracheal tube from becoming dislodged while preparing the bird for the procedure and also will help reduce the likelihood of tracheal damage (Fig 33.7). Another important aspect to address at this juncture of the procedure is caring for the patient's eyes. The eyes of most companion birds are prone to physical damage, since they protrude from their heads. A method to help reduce eye damage is to provide a ring of soft material to encircle the eye that is going to be closest to the table. An eye lubricant also should be administered at this time.

ANESTHETIC POTENCY

Anesthetic potency can be expressed in many ways. One method is to assess the MAC of the inhalation anesthetic during surgical anesthesia. The MAC is generally defined as the minimum alveolar concentration of an anesthetic that produces no response in 50% of patients exposed to

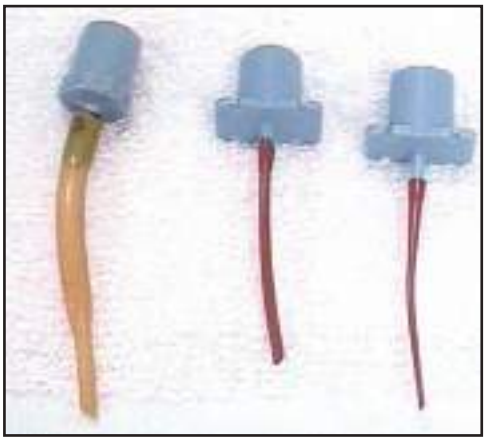


Fig 33.6 | Examples of endotracheal tubes. Cole and red rubber catheter endotracheal tubes can be fashioned for smaller patients.

Fig 33.7 | Intubation of the bird in Fig 33.5 with a modified Cole endotracheal tube. Notice that the endotracheal tube is taped to the lower beak to help avoid damaging the trachea and to allow for a better seal with the glottis.

painful stimulus. MAC values are measured as the end-tidal concentration of anesthetic and are not vaporizer settings. In birds, the term MAC is not appropriate because birds do not have an alveolar lung. It has been suggested that in avian species, MAC be defined as the minimum anesthetic concentration required to keep a bird from purposeful movement to a painful stimulus.[13] The MAC for isoflurane[b] in cockatoos, ducks, and Sandhill cranes is 1.44%,[12] 1.32%[15] and 1.35%,[9] respectively.

Another method of comparing anesthetic agents is by their ability to cause respiratory depression and apnea in an animal. The Anesthesia Index (AI) can predict this effect: the lower the AI, the greater the chance of apnea. The AI for isoflurane[b] in dogs, cats, horses and ducks is 2.51,[29] 2.40,[29] 2.33[28] and 1.0[15] respectively. These values indicate that isoflurane[b] depresses ventilation more in birds than in mammals.

INHALATION AGENTS

Inhalation anesthetic agents are used to produce general anesthesia. Their safe use requires knowledge of their pharmacologic effects and physical and chemical properties. Anesthetic doses required for surgery produce unconsciousness (hypnosis) and hyporeflexia. With unconsciousness (no response) all pain physiologically still occurs. Inhalation anesthetics provide optimal control of anesthesia, rapid induction and recovery from anesthesia, and relatively few adverse side effects.[21]

There are two agents currently used for inhalation anesthesia by most avian practitioners, isoflurane[b] and sevoflurane[c]. A third agent, desflurane, recently has become available, although due to its specialized vaporizer and pungent odor it is doubtful whether it will ever become a commonly used anesthetic agent in avian practice. However, even with the relative high cost of sevoflurane, it quickly is becoming the inhalation anesthetic agent of choice for companion avian practitioners. All three of these inhalation anesthetic agents produce dose-dependent central nervous system, respiratory and cardiovascular effects.

Isoflurane[b] currently is the preferred choice for avian anesthesia due to its low relative cost, comparatively rapid induction and recovery, low blood solubility and minimal metabolism. In addition, it does not sensitize the heart to catecholamine-induced arrhythmias.[22]

Sevoflurane also has been shown to be an excellent anesthetic agent, as it has a lower blood gas partition coefficient than isoflurane[b] (sevoflurane[c] = 0.69, isoflurane[b] = 1.41) and is therefore less soluble in blood, although it is less potent (MAC 2-3%). This decreased solubility accounts for the shorter recovery time and time to standing with sevoflurane compared with isoflurane[b].[8] In critical or prolonged surgical procedures, the use of sevoflurane[c] can help increase the chance of a successful outcome due to the faster recovery time when compared to other inhalation anesthetics. In addition, sevoflurane does not cause respiratory tract irritation, as do isoflurane[b] and desflurane[d], and therefore reduces the stress involved with mask induction.

Desflurane[d] is a less potent (MAC 6-8%) anesthetic agent that requires a specialized temperature-controlled and pressurized vaporizer to accurately deliver the anesthetic agent to the patient. It has the lowest blood gas partition coefficient (0.42) of the three agents and tissue solubility (desflurane[d] and sevoflurane[c] blood-brain tissue coefficient = 1.3 and 1.7, respectively).[7] These physical attributes provide for a faster recovery time than those of isoflurane[b] and sevoflurane[c], especially after prolonged anesthetic procedures. In humans, the pungency of desflurane[d] caused respiratory tract irritation, cough-

ing, breath holding and laryngospasms, and thus is not used for mask inductions.[7]

Even when using these reliable anesthetic agents, anesthesia remains risky because it depresses ventilation at concentrations required for surgery.[13] Specifically, as the concentration of isoflurane[b] increases, the $PaCO_2$ increases, which manifests clinically as a respiratory acidosis.[13]

Halothane is no longer considered to be a safe and reliable anesthetic agent in avian species. As in mammals, halothane sensitizes the heart to catecholamine-induced cardiac dysrhythmias. Fatalities due to pre-existing high levels of circulating catecholamines in stressed birds have been associated with halothane.

Injectable Anesthetics

There are many inherent disadvantages associated with the use of injectable anesthetic agents, the most notable being significant species variation, cardiopulmonary depression, prolonged and violent recoveries, and the difficulty involved in delivering a safe and effective volume.[12] The advantages of injectable anesthesia are few and are mostly related to cost and ease of administration. The disadvantages significantly outweigh the advantages in all but the most severe situations, such as field conditions where inhalant anesthesia is not feasible. See Chapter 1, Clinical Practice for a field anesthesia setup. If the decision is made to use injectable anesthesia in companion avian species, the clinician should realize that most of the positive attributes associated with inhalation anesthesia cannot be exploited. When using any of the injectable anesthetic agents, it is advisable to intubate the patients and monitor their physiologic state as if they were undergoing general inhalation anesthesia. Also, an I.V. catheter should be in place for rapid vascular access for fluid therapy and pharmaceuticals

PREANESTHETICS

Parasympatholytic Agents

The use of routine parasympatholytic agents in avian species, as in mammals, is no longer thought to be necessary and is, in fact, counterproductive in most circumstances. Parasympatholytic agents (atropine and glycopyrrolate) in avian species exacerbate thickening of salivary, tracheal and bronchial secretions, and increase the risk for airway obstruction.

Tranquilizers

Tranquilizers such as diazepam and midazolam are benzodiazepines that have excellent muscle relaxant properties. They lack analgesic properties whether used alone or in combination with primary anesthetics such as ketamine. I.V. diazepam can be used to tranquilize a bird prior to mask induction with an inhalant anesthetic, thus reducing the stress involved with the procedure.[12] Uptake of diazepam is slow and unpredictable with I.M. injection. An important feature of diazepam, in contrast to midazolam, is its shorter duration of action leading to a shorter recovery time. Midazolam is more potent and longer lasting than diazepam, and does not adversely affect mean arterial blood pressure and blood gases in select avian species. I.M. uptake is rapid and almost complete. When midazolam is given to geese, raptors and pigeons, the effects last for several hours after the termination of anesthesia, which can be undesirable.[31]

Alpha-adrenergic Agents

Xylazine, medetomidine and other related $alpha_2$-adrenergic agonists have sedative and analgesic properties. They can have profound cardiopulmonary effects including second-degree heart block, bradyarrhythmias and increased sensitivity to catecholamine-induced cardiac arrhythmias. When used alone in high doses, xylazine is associated with respiratory depression, excitement and convulsions in some species.[12] Hypoxemia and hypercapnia were observed in Pekin ducks (*Anas domesticus*) given xylazine, and a combination of xylazine and ketamine.[14] When used in combination with ketamine, the sedative and analgesic effects of xylazine are enhanced. One positive aspect of this class of drug is that an overdose or slow recovery can be treated with an alpha-adrenergic antagonist reversal agent such as yohimbine or atipamezole.

Opioids

Opioids are commonly used as a premedication in small mammal medicine, both for presurgical analgesia and to reduce the amount of inhalation anesthesia necessary to achieve a surgical plane. In pigeons, it appears that the kappa opioid receptors account for the majority of the opioid receptor sites.[18] Thus butorphanol, a kappa agonist, may be a better analgesic than mu opioid agonists. In addition, it has been demonstrated that butorphanol reduces the concentration of isoflurane[b] necessary to maintain anesthesia in cockatoos.[3] Please refer to Chapter 8, Pain Management for a comprehensive assessment of analgesia in avian species.

In a recent study it was found that the administration of hydromorphone to healthy dogs undergoing elective ovariohysterectomy or castration may result in transient increases in $PaCO_2$ postoperatively, and that the administration of hydromorphone or butorphanol may result in transient decreases in PaO_2; however, the increases and decreases were mild and within reference limits.[2] Due to

the unique physiology of avian species, it is possible that the postoperative increases in $PaCO_2$ and decreases in PaO_2 due to the administration of opioids may be more pronounced. More study in this area is necessary to reach a conclusion.

Local Anesthetics

Local anesthetics are an excellent tool for preemptive analgesia in avian species. However, local anesthetics do not provide any relief from the stress involved with the restraint and handling of the awake bird.

It has been demonstrated in humans and animals that pain is easier to prevent than to treat. In fact, it has been shown that the repeated stimulation of the neurons that mediate nociception in the dorsal horn of the spinal cord can cause them to become hypersensitized. The morphology of these neurons actually changes and becomes "wound up," and as a result, the response to subsequent incoming signals is changed. The neuronal hypersensitivity continues even after the noxious stimulus stops and can last 20 to 100 times longer than the original stimulus.[32] The technique of administering a preoperative local anesthetic to block the transmission of noxious stimuli can prevent or attenuate the "windup." This procedure is especially effective for painful procedures such as amputations, fracture repairs and coelomic surgeries. However, when using local anesthetic agents, care must be taken not to induce seizures or cardiac arrest with an overdose. One common method to ensure the correct dosage is to calculate the maximum safe dose and, if necessary, dilute it to a more convenient volume for administration. See Chapter 8, Pain Management for an indepth review of local anesthetic.

In summary, local anesthetic agents should be used primarily as an adjunct to general anesthesia in helping to prevent "windup." In general, they should not be used for local anesthesia in an awake bird due to the high levels of stress involved with handling and restraint, except in rare circumstances where a companion bird is not overly stressed and the procedure is simple such as in the case of a broken toenail.

General Anesthetics

Ketamine hydrochloride, a cyclohexamine, produces a state of catalepsy and can be given by any parenteral route. When used alone, ketamine is suitable for chemical restraint and moderate analgesia for minor surgical and diagnostic procedures, but is not suitable for major surgical procedures.[14]

Ketamine is generally used in conjunction with other drugs such as diazepam or xylazine to improve the quality of the anesthesia by providing more muscle relaxation or increased analgesia. There appears to be significant species variation when using ketamine in birds. For example, in several raptor species and waterfowl, the commercially available form of ketamine induced poor-quality chemical restraint and anesthesia.[14]

Propofol is a substituted phenol derivative developed for intravenous induction and maintenance of general anesthesia. Its major advantage is that it has a rapid onset and recovery, and thus has very little residual or cumulative effect. Its major disadvantage is its dose-dependent cardiovascular and respiratory depression[16] (see Chapter 8, Pain Management).

Patient Monitoring

Monitoring the avian patient during anesthesia is the most critical aspect of the process. The bird must be maintained and monitored correctly, and appropriate responses to the animal's physiologic state performed in a timely fashion.

Apnea, hypoventilation, hypothermia and regurgitation are the most common problems experienced during anesthesia and, because of the bird's small FRC, periods of apnea are critical. Without adequate airflow through the lungs, gas exchange does not occur and the physiologic acid base balance maintained by the respiratory system is made ineffective.

RESPIRATORY SYSTEM

Both the respiratory rate and tidal volume should be monitored during anesthesia to help assess the adequacy of ventilation. These parameters can be supervised by observing the respiratory rate and pattern and ausculting the coelomic cavity. But, there is no formula involving respiratory rate and tidal volume that can be employed to correctly determine the ventilatory status of the patient. The only way to accurately assess ventilation in an animal is through some measure of arterial CO_2.

One study in African grey parrots (*Psittacus erithacus*) suggests that capnography can be effectively used to monitor arterial CO_2. The study indicates that the End Tidal Carbon Dioxide ($ETCO_2$) consistently overestimates arterial CO_2 by approximately 5 mmHg.[4] When using capnography in avian patients, a side stream capnograph should be utilized and the dead air space associated with the endotracheal tube must be minimized. The capnograph can be connected to the breathing circuit through an 18-gauge needle inserted into the lumen of the endotracheal tube adapter (Figs 33.8, 33.9). It is imperative that the needle does not obstruct the luman of the endotracheal tube.

Fig 33.8 | Cole endotracheal tube shortened to reduce dead air space. The endotracheal tube adapter has been modified to allow the attachment of a side stream capnograph. This has been accomplished by mounting an 18-ga needle into the lumen of the adapter with the needle bevel facing the patient.

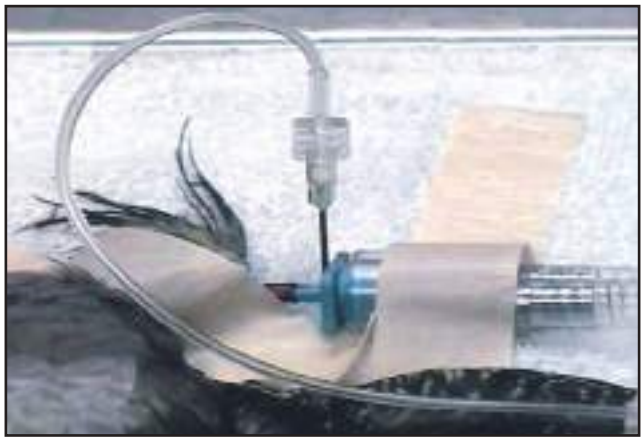


Fig 33.9 | Example of a red rubber catheter with the endotracheal tube adapter modified to allow side stream capnography on a cockatiel.

Positive pressure ventilation through the use of a mechanical ventilator or by manual compression of the reservoir bag can be used to effectively maintain the avian patient during inhalation anesthesia. During positive pressure ventilation, airway pressures should not exceed 15 to 20 cm H_2O to prevent volutrauma to the air sacs. When using positive pressure ventilation, adjust the ventilations per minute mechanically or manually according to the $ETCO_2$, striving to maintain the patient within normal physiologic ranges. A guideline that can be used with some caution was found in studies with African grey parrots. An $ETCO_2$ of 30 to 45 mmHg indicated adequate ventilation during inhalation anesthesia in African grey parrots.[4]

CIRCULATORY SYSTEM

The heart can be monitored through a number of noninvasive methods such as pulse, auscultation and an electrocardiogram (ECG). Monitoring the pulsations of blood through a peripheral artery can assess heart function. The Doppler flow probe[e] is an effective means to monitor pulse rate and rhythm. Standard bipolar and augmented limb leads can be used to monitor and record the ECG, which reports the electrical activity of the heart. The ECG must be able to detect and accurately record the high heart rates (up to 500 bpm) associated with avian species. A new technique to obtain ECG readings is through the use of an esophageal probe[f] (Figs 33.10, 33.11). The probe is inserted into the esophagus of the patient and attached to the standard ECG via an adapter (Figs 33.12, 33.13). This technique achieves accurate readings and eliminates the need for attaching leads to the limbs. It is difficult to directly measure arterial blood pressure in psittacine birds due to their small size, the invasiveness of the procedure and the cost of the equipment.

CENTRAL NERVOUS SYSTEM

Eye reflexes, jaw tone, cloacal reflex, pedal reflex and muscle relaxation can be used to assess avian patients during anesthesia. One study described the ideal anesthetic level as when the patient's eyelids were completely closed, pupils mydriatic, the pupillary light reflex was delayed, the nictitating membrane moved slowly over the entire cornea, the muscles were all relaxed and all pain reflexes were absent.[10] Breathing depth and frequency are the best manual assessment tools.

OXYGENATION

Ensuring that an adequate PaO_2 is present in a patient's arterial blood is very important. An intubated bird on 100% oxygen during inhalation anesthesia is usually well oxygenated. Problems such as apnea, ventilation perfusion mismatch and tracheal obstructions can significantly alter the partial pressure of arterial oxygen. The only accurate method for determining a patient's arterial oxygen status is through arterial blood gas analysis. Mucous membrane color can be used to monitor change but is not effective in a critical patient. Studies using pulse oximetry indicate that while it is a valuable tool for assessing mammalian oxygen saturation, it is not consistently accurate on avian patients[27] (Fig 33.14). It is very important that the anesthetist be careful not to interpret sufficient oxygenation as being adequately ventilated. Birds can be well oxygenated and at the same time be extremely hypercapnic.[6] PaO_2 is not a reliable indicator of the ventilatory status of a bird or any other animal.

TEMPERATURE

Studies show that without thermal support anesthetized birds rapidly lose heat. The flow of dry anesthetic gases through the respiratory system, removing feathers for sterile skin preparation, surgical skin preparation liquids,

Fig 33.10 | Patient with a companion ECG probe[f] (green line).

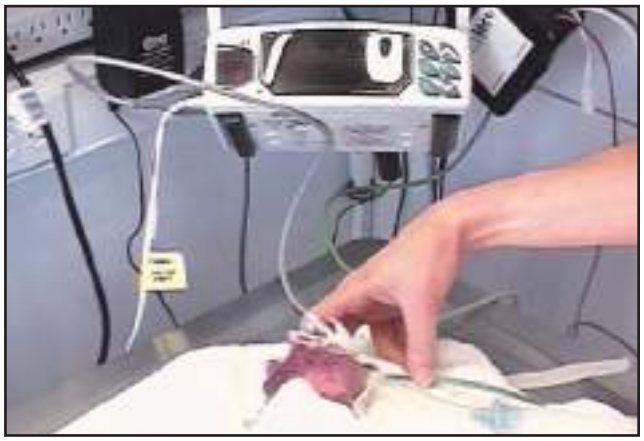


Fig 33.11 | Overview of the patient attached to the companion ECG probe connected to the combination ECG and pulse oximetery monitor[f]. Notice the adapter box to the right of the monitor (black box). The adapter allows standard ECG leads to connect to the esophageal ECG probe.

Fig 33.12 | ECG probe[f] with adapter. This adapter box connects standard ECG leads to the esophageal probe.

Fig 33.13 | ECG probe[f]. This probe is inserted into the esophagus of the patient and eliminates the need of attaching separate ECG leads.

blunted physiologic responses to the reduction in body temperature, the small body mass in relation to the surface area and many other factors all serve to quickly reduce a bird's body temperature during anesthetic procedures. Hypothermia is the most common problem associated with prolonged anesthesia, as it decreases the requirement of anesthetic, and it also causes cardiac instability and prolongs recovery. Hypothermia also is significant after anesthesia because hypothermic patients must then use critical energy reserves to generate heat by shivering. There are many methods for ensuring that a bird's body temperature does not drop significantly during anesthesia, including a circulating water blanket, heated surgery tables, warm towels, warm IV fluids and warm air blankets. When using warm air blankets, be certain to keep the patients' eyes well lubricated, as these devices tend to dry out the animals' eyes. The most effective method for providing heat during anesthesia appears to be from providing an external heat source.[24] Korbel showed that medical oxygen has no moisture content and this caused evaporation from the mucous membranes, dropping body temperature. If he warmed that oxygen and added moisture, he got negligible body temperature loss during anesthesia.[9,10]

Recent research has demonstrated that forced-air warming systems[k] more effectively minimize hypothermia in avian patients while undergoing inhalant anesthesia than the traditional thermal devices (eg, circulating water blankets and infrared heat emitters) (Tully, T. unpublished data, 2001).

Temperature can be reliably monitored with a long, flexible thermistor probe inserted into the esophagus to the level of the heart. Cloacal temperature monitoring can be accurate, but is dependent on body position and cloacal activity over time.

Anesthetic Emergencies

Emergency situations arising from anesthesia no longer

Fig 33.14 | Several manufacturers offer combination anesthesia monitoring equipment such as this pulse oximeter and ECG instrument[f].

occur as frequently as in the past. It is now common to perform anesthesia on a psittacine bird for 2 or more hours and have a low incidence of morbidity and mortality. This is primarily due to the advances in the practitioner's ability to monitor and maintain the patient within its normal physiologic parameters. Although emergencies are not common, emergency drugs should be prepared and available prior to anesthesia.

COMMON EMERGENCY TREATMENTS

- **Doxapram**[g] - Positive inotrope, direct action on respiratory centers in the medulla, stimulates ventilation.
- **Isotonic Crystalloid** - Hypotension: Expand blood volume and increase tissue perfusion.
- **Epinephrine HCl**[h] - Positive inotrope, initiates heartbeats, increases heart rate and cardiac output.
- **Atropine**[i] - Parasympatholytic effects, may correct supraventricular bradycardia or a slow ventricular rhythm by stimulating supraventricular pacemakers.

Recovery

Recovering the patient following inhalation anesthesia is usually a rapid process once the anesthetic gas is turned off. Disconnect the bird from the anesthetic circuit and flush the circuit with fresh oxygen. Reconnect the bird to the circuit and continue the recovery with the patient on 100% oxygen.

Most birds will initially experience muscle fasciculations as they become lighter. If the bird is being auscultated at this time, the heart sounds will become less audible due to the muscle movement.[4] Care should be taken not to incorrectly interpret this as a deep plane of anesthesia. This is especially important during surgical procedures. As the bird becomes lighter, more apparent movements such as wing flutter and leg withdrawal will become evident. When the patient starts exhibiting jaw movement, it should be extubated to keep it from severing the endotracheal tube. The bird should be held lightly in a towel in an upright position once extubated. Wrapping the towel too tightly will not only inhibit breathing, but can lead to excessive retention of body heat leading to hyperthermia. The mask used for induction, without the latex glove, should be placed over its head or beak to provide oxygen. The patient should be held in this fashion, being careful not to inhibit the movement of the sternum, until it can hold itself upright. At that point it should be placed in a dark, padded box and the box placed in a heated, oxygenated cage. This will allow the animal to fully recover in a less stressful environment. If a bird has not been fasted correctly prior to surgery, regurgitation can occur during recovery. Most patients will appear fully recovered from anesthesia within 30 minutes to 2 hours.

Products Mentioned in the Text

a. ADS 1000 Penlon Sigma Delta — Sevoflurane Vaporizer, Penlon Limited, Radley Road, Abingdon, OX14 3PH, UK; Positive Pressure Ventilator, Engler Engineering Corp, 1099 E. 47th St, Hialeah, FL, USA
b. Isoflo, Abbot Laboratories, North Chicago, IL, USA
c. Ultane, Abbot Laboratories, North Chicago, IL, USA
d. Suprane, Anaquest, Madison, WI, USA
e. Parks Doppler Pediatric Probe, Parks Electronics, Aloha, OR, USA
f. Cardio Companion ECG Probe; Cardio Companion Esophageal Lead; ECG-Pulse oximeter V3404, SurgiVet Inc, SurgiVet Anesco, Veterinary Surgical Products, Waukesha, WI, USA
g. Dopram-V, Fort Dodge Laboratories, Fort Dodge, IA, USA
h. Epinephrine 1:1000, Vedco, Inc, St. Joseph, MO, USA
i. Atropine, Elkins-Sinn, Inc, Cherry Hill, NJ, USA
j. Respirot, Novartis, Agro Benelux BV, Animal Health Sector, Stepvelden 10, Rousendiial, The Netherlands
k. Bair Hugger, www.bairhugger.com

Parts of this chapter are reprinted or paraphrased from Exotic DVM 5(3):15-20, 2003 and from BSAVA Manual of Psittacine Birds 2nd Ed 2005, pp 87-96. Permission granted by Zoological Education Network, Inc and BSAVA 2005.

References and Suggested Reading

1. Abou-Madi N: Avian Anesthesia. *In* Heard DJ (ed): Vet Clin No Am. Exot An Prac 4:147-167, 2001.
2. Campbell VL, Drobatz KJ, Perkowski SZ: Postoperative hypoxemia and hypercarbia in healthy dogs undergoing routine ovariohysterectomy or castration and receiving butorphanol or hydromorphone for analgesia. J Am Vet Med Assoc 222:330-336, 2003.
3. Curro TG, Brunson DB, Paul-Murphy J: Determination of the ED50 of isoflurane and evaluation of the isoflurane-sparing effect of butorphanol in cockatoos (*Cacatua* spp.). Vet Surg 23:429-433, 1994.
4. Edling TM: Gas anesthesia: How to successfully monitor and keep them alive. Proc Assoc Avian Vet, 2001, pp 289-301.
5. Edling TM, et al: Capnographic monitoring of African grey parrots during positive pressure ventilation. J Am Vet Med Assoc 219:1714-1717, 2001.
6. Eger El II: New inhalational agents: Desflurane and sevoflurane. Can J Anaesth 40(5):R3-R5, 1993.
7. Franchetti DR, Kilde AM: Restraint and anesthesia. *In* Fowler ME, (ed): Zoo and Wild Animal Medicine. Philadelphia, WB Saunders Co, 1978, pp 359-364.
8. Greenacre CB, Quandt JE: Comparison of sevoflurane to isoflurane in Psittaciformes. Proc Assoc Avian Vet, 1997, pp 123-124.8.
9. Korbel R: Vergleichende Untersuchungen zur Inhalationsanaesthesie mit Isofluran (Forene) und Sevofluran (SEVOrane) bei Haustauben (*Columba livia* Gmel., 1789, var. *domestica*) und Vorstellung eines Narkose-Referenzprotokolls fuer Voegel. Tierarztliche Praxis 26 (K):211-223, 1998.
10. Korbel R, et al: Aerosacular perfusion with isoflurane: An anesthetic procedure for head surgery in birds. Proc Assoc Avian Vet, 1993, pp 9-37.
11. Kramer MH: Managing endotracheal tube mucus plugs in small birds. Exotic DVM 4(5):9, 2002.
12. Ludders JW, Mathews N: Birds. *In* Thurmon JC, Tranquilli WJ, Benson JG (eds): Lumb and Jones Veterinary Anesthesia 3rd ed. Baltimore, MD, Williams & Wilkins, 1996, pp 645-669.
13. Ludders JW, Rode J, Mitchell GS: Isoflurane anesthesia in sandhill cranes (*Grus canadensis*): Minimal anesthetic concentration and cardiopulmonary dose-response during spontaneous and controlled breathing. Anesth Analg (Cleve) 68:511-516, 1989.
14. Ludders JW, Rode JA, Mitchell GS: Effects of ketamine, xylazine and a combination of ketamine and xylazine in Pekin ducks. Am J Vet Res 50(2):245-249, 1989.
15. Ludders JW, Mitchell GS, Rode J: Minimal anesthetic concentration and cardiopulmonary dose response of isoflurane in ducks. Vet Surg 19:304-307, 1990.
16. Machin KL, Caulkert NA: The cardiopulmonary effects of propofol in mallard ducks. Proc Am Assoc Zoo Vets, 1996, pp 149-154.
17. Magnussen H, Willmer H, Scheid P: Gas exchange in air sacs: Contribution to respiratory gas exchange in ducks. Respir Physiol 26:129-146, 1976.
18. Mansour A, et al: Anatomy of CNS opioid receptors. Trend Neurosci 11:308-314, 1988.
19. McLelland J: Larynx and trachea. *In* King AS, McLelland J (eds): Form and Function in Birds Vol 4. London, Academic Press, 1989, pp 69-103.
20. McLelland J: Anatomy of the lungs and air sacs. *In* King AS, McLelland J (eds): Form and Function in Birds Vol 4. London, Academic Press, 1989, pp 221-279.
21. Muir WW, Hubbell LA: Inhalation anesthesia. *In* Muir WW, Hubbell LA (eds): Handbook of Veterinary Anesthesia. St. Louis, MO, Mosby, 2000, pp 154-163.
22. Muir WW, Hubbell LA: Pharmacology of inhalation anesthetic drugs. *In* Muir WW, Hubbell LA (eds): Handbook of Veterinary Anesthesia. St. Louis, MO, Mosby, 2000, pp 164-181.
23. Muir WW, Hubbell LA: Anesthetic machines and breathing systems. *In* Muir WW, Hubbell LA (eds): Handbook of Veterinary Anesthesia. St. Louis, MO, Mosby, 2000, pp 210-231.
24. Phalen DN, Lau MT, Filippich LJ: Considerations for safely maintaining the avian patient under prolonged anesthesia. Proc Assoc Avian Vet, 1997, pp 111-116.
25. Powell FL, Scheid P: Physiology of gas exchange in the avian respiratory system. *In* King AS, McLelland J (eds): Form and Function in Birds Vol 4. London, Academic Press, 1989, pp 393-437.
26. Scheid P, Piiper J: Aerodynamic valving in the avian lung. Acta Anaesth Scand 33:28-31, 1989.
27. Schmitt PM, Gobel T, Trautvetter E: Evaluation of pulse oximetry as a monitoring method in avian anesthesia. J Avian Med Surg 12(2):91-99, 1998.
28. Steffey EP, et al: Enflurane, halothane, and isoflurane potency in horses. Am J Vet Res 38:1833-1836, 1977.
29. Steffey EP, et al: Isoflurane potency in the dog and cat. Am J Vet Res 39:573-577, 1978.
30. Sturkie PD: Heart and circulation: Anatomy, hemodynamics, blood pressure, blood flow. *In* Sturkie PD (ed): Avian Physiology 4th ed. New York, Springer-Verlag, 1986, pp 130-166.
31. Valverde A, et al: Determination of a sedative dose and influence of midazolam on cardiopulmonary function in Canada geese. Am J Vet Res. 51(7):1071-1074, 1990.
32. Woolf CJ, Chong MS: Preemptive analgesia: Treating postoperative pain by preventing the establishment of central sensitization. Anesth Analg 77(2):362-379, 1993.

CHAPTER
34

Surgical Resolution of Orthopedic Disorders

PETER HELMER, DVM, Dipl ABVP-Avian;
PATRICK T. REDIG, DVM, PhD

Orthopedic injuries are common in pet bird practice. Some of the more common causes are falling, an impact with a window or ceiling fan, a crushing incident such as being stepped on, or an encounter with a dog or cat.

Definitive fixation of a fracture is rarely an emergency. Due to the traumatic nature of most of these injuries, first priority must be given to stabilizing the patient. Emergency treatment of shock, hemorrhage and sepsis are covered elsewhere. It is important to assess the patient holistically, including diet, husbandry and concurrent medical conditions, without focusing solely on the obvious injuries.

Prior to examination, obtain a thorough history from the owner. Upon initial examination of the patient, orthopedic problems may manifest as lameness, a wing droop, paresis, a swelling or an open wound. Following initial stabilization of the patient, further investigation of these abnormalities is warranted.

The initial orthopedic exam is generally performed with the bird awake. Prior to handling the bird, visually assess it in its cage:

- Does the bird bear weight equally on each leg?
- Are the wings held symmetrically and in the proper position?
- Does the bird grip a perch?
- Is the overall body posture correct?

Greg J. Harrison

Fig 34.1 | Wetting with alcohol or removing feathers over a fracture site often reveals the contusion.

Following the initial visual examination, restrain the animal and systematically assess the skeletal system. The skull is palpated and the feathers covering the head parted to examine the head for hemorrhage or other injuries. In small birds, transillumination of the skull may identify intracranial bleeding. The keel is palpated for evidence of a fracture. Palpation and visualization of the entire length of the vertebral column may reveal deviations or swellings. Wings and legs are examined beginning at the proximal aspect and progressing distally. Long bones are assessed for fractures, deviations and swellings, and joints are assessed for appropriate range of motion. The contralateral appendage may be used as a normal for comparison. Be careful examining extended wings, as iatrogenic fractures may result from the bird trying to flap while improperly restrained.

Suspicion or identification of orthopedic injury requires further examination under anesthesia. Isoflurane or sevoflurane are the agents of choice. While under anesthesia, a more complete evaluation of suspicious areas may be performed, including investigation of masses, minor wound debridement and investigation, wetting or plucking of feathers for closer inspection (Fig 34.1), and radiographs may be made.

Radiographic technique and positioning are covered elsewhere. At a minimum, two orthogonal views are required for evaluation of a particular area. Radiographs of the non-affected contralateral limb are often helpful for comparison, especially as anatomy is quite variable between genera of birds.

Following identification of an orthopedic problem, a treatment plan must be formulated. Fractures should be temporarily immobilized with splints or bandages until the patient is otherwise stable enough to undergo surgery for repair.

One must keep in mind that splints and bandages are usually meant for temporary fixation only.

The selection of an appropriate technique for definitive repair will depend on several variables, including the size of the patient, the degree of postoperative return to function required, cost, the skill of the surgeon and concurrent medical conditions.

The importance of the first of these conditions, the size of the patient, is often underestimated. Many avian orthopedic techniques were developed for use in raptors weighing approximately 1 kg. While it is true that "tie-in" fixators (TIF) was developed on birds around 1 kg in weight, available hardware is such that it can be readily applied to birds 65-100 grams. These techniques may not be feasible in the 20-g canary. The reduction of load associated with smaller size and higher surgical morbidity and mortality of small patients often contribute to a decision to manage fractures more conservatively in small patients.

The goal of every surgeon should be to return each patient to the pre-injury level of activity; however, this is not always possible. Consideration must be given to the quality of life of a non-releasable wild bird postsurgically. Given the same circumstance, a pet parrot may have an excellent quality of life.

The ideal method of fixation must always be offered and recommended, but lower cost alternatives may have to be considered. A lower cost option may sacrifice postoperative return to function, but in a pet this may be an acceptable compromise. Preoperative communication with owners may be as important as the surgical fixation technique.

Finally, all surgeons do not possess the same skill and experience with avian fracture repair. Referral of orthopedic cases to a more experienced surgeon should be considered.

Principles of Orthopedic Repair

The basic fundamental principles of orthopedic repair are similar to other species. The repair technique should promote a functional union of the fragments, share the load on the bone during healing, allow early return to function and have a low morbidity. The ideal hardware for use in this repair would be versatile, effective, adjustable, lightweight, inexpensive and associated with minimal complications.

Although the principles of repair are the same as in other veterinary patients, some important differences exist:

- Bone cortices are thinner and more brittle, resulting in

less holding power for hardware.
- There is less soft tissue covering the bones. As a result, blood and nerve supplies are commonly injured. Fracture segments tend to be unstable and commonly penetrate the skin, with bacterial contamination a common sequela. These fragments of exposed bone are non-viable and commonly form sequestra if incorporated into the repair.
- Bone grafting is not common, as there is little cancellous bone to harvest.
- Load bearing must be rapidly restored to the legs, as locomotion is bipedal.

The healing patterns of avian bone have been examined.[8,19] In adult birds, the amount of time needed for radiographic and histologic union of unilateral radius and ulna fractures in an experimental setting was 5 weeks with internal fixation and 8 weeks with external coaptation.[8] The majority of the callus tissue during healing is derived from the periosteal surface, and the blood supply to the periosteum from surrounding soft tissues is very important. The intramedullary circulation appears to be of less significance in avian bone healing than in mammals.[19]

General Methods of Fracture Fixation

CAGE REST

Very few avian fractures are satisfactorily treated with this method of repair. Fractures of the digits as well as greenstick fractures of young birds may be managed in this way. Cage rest also may be appropriate in the management of fractures of non-weight-bearing bones in very small birds such as canaries and finches. Consider decreasing light levels to decrease activity; however, adequate light must be provided at least twice daily for feeding. Also consider the use of smooth-sided cages without perches (eg, aquarium or plastic carrier) to prevent climbing.

EXTERNAL COAPTATION

The use of splints and bandages for fracture repair in the avian patient is limited. Bandages tend to be bulky and cumbersome. They require prolonged immobilization of joints and usually result in poor alignment of fracture fragments. Though this type of repair may be initially less expensive, return to function is typically prolonged and may not be as complete as with internal fixation. Coaptation may be considered if:
- Full return to function is not required.
- Fractures are pathologic as a result of metabolic bone diseases.
- Bones are too soft to hold hardware.
- The patient is too small for internal fixation alternatives.
- The surgical or anesthetic risk is judged to be too great.

Commonly used splints include the "figure-8" wrap, which may or may not include a body wrap, the Altman splint, and Robert-Jones bandages with the incorporation of splinting material. The application of the figure-8 wrap and the Robert-Jones bandage are covered in other texts.[7] A description of the application of the Altman splint is found below in Managing Fractures of the Specific Bones, Tibiotarsus.

The use of titanium IM pins may have advantages in comparison to the traditional stainless steel pin. Titanium has a "memory" and can be bent past 180° and spring back to its normal position (Fig 34.2). The titanium pin is measured against the radiograph of the fractured bone. The pin is inserted into the proximal or distal fragment (Fig 34.3). The pin is bent and inserted into the opposite fragment, thus avoiding the need to transgress a joint (Fig 34.4) (G. Harrison, personal communication, 2003). If a larger pin than shown in Fig 34.4 is needed for larger birds, a single titanium IM pin is not used, as the force to bend the larger pin will shatter the bone. Multiple smaller diameter pins are placed in stacking fashion to facilitate bending.

Orthodonture rubber-band impactions also have been used successfully on the tibiotarsus (Fig 34.5) (G. Harrison, personal communication, 2003) (see Chapter 14, Evaluating and Treating the Gastrointestinal System).

HYBRID FIXATORS

The use of both intramedullary (IM) pins and external skeletal fixators (ESF) has been well described for the management of avian fractures.[7] More recently, the use of hybrid fixators, or "tie-in" fixators (TIF), has become more popular. This technique combines an IM pin linked to an ESF (Fig 34.6). Advantages include the relative ease of application, use of a smaller diameter IM pin than would otherwise be used, which causes less damage to the intramedullary blood supply, an increase in resistance to bending forces compared to either ESF or IM pin alone,[1] a decrease in migration of the IM pin or the cross-pins, and the ability to gradually remove hardware over time, a process called dynamization, which gradually increases the load bearing of the bone.

The diameter of the IM pin should fill 50 to 60% of the medullary cavity. Following placement, the pin is bent at 90° where it exits the bone. Two or more threaded cross-pins are placed. Threaded pins have been demonstrated to have superior bone-holding strength in avian cortices when compared to non-threaded pins.[4] In one recent

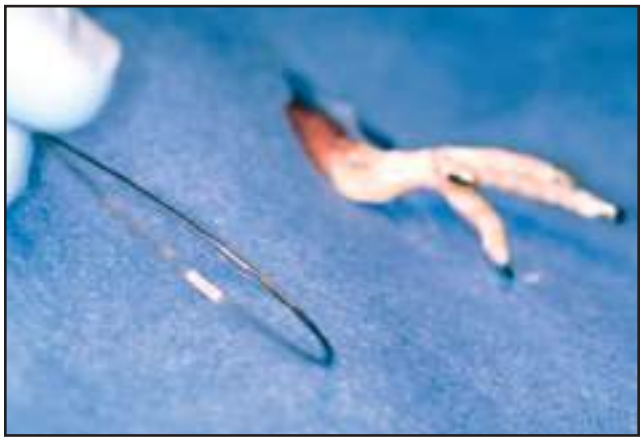

Greg J. Harrison

Fig 34.2 | Titanium pin bent past 180°.

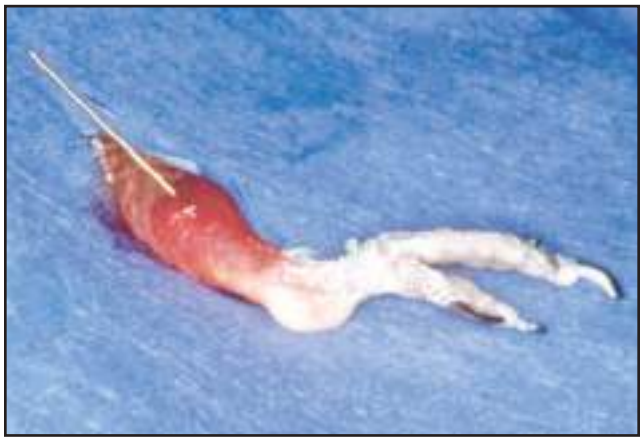

Greg J. Harrison

Fig 34.3 | Titanium pin, precut and placed in the distal fragment via the fracture site.

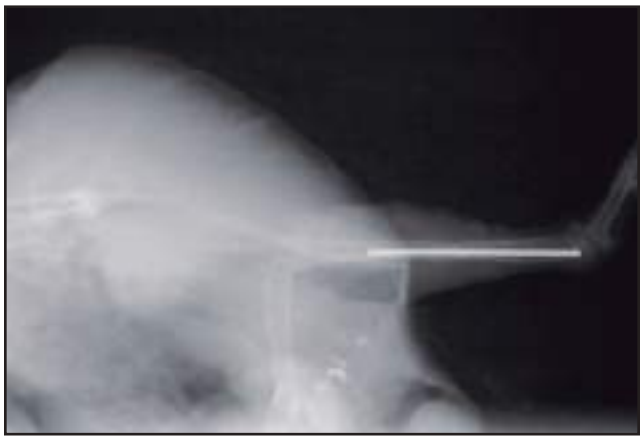

Greg J. Harrison

Fig 34.4 | Radiograph of a titanium pin in the marrow cavity, having never transversed a joint surface.

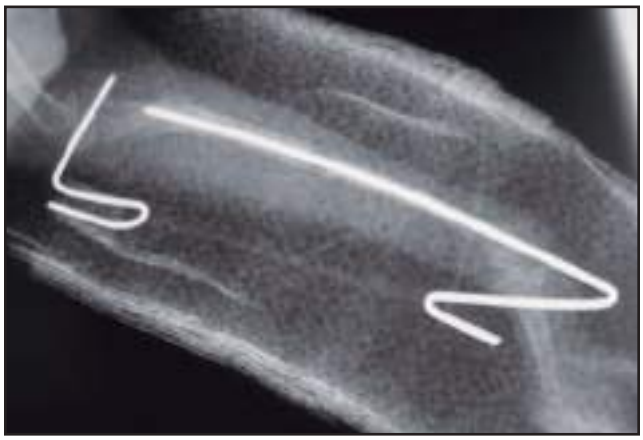

Greg J. Harrison

Fig 34.5 | Bending a hook shape into the distal end of a stainless steel transverse pin and the distal end of the distal fragment pin can allow a periodontal rubber band to be used to apply traction to a fracture. A topical bandage covering helps prevent the rubber band from being removed inadvertently.

study, a significant difference in pull-out strength was not demonstrated when comparing positive profile threaded pins vs. negative profile threaded pins.[4] However, positive profile pins have a higher locking strength at the pin-bone interface and may be considered advantageous in larger bones. The cross-pins are linked to the IM pin with either a metal or acrylic bar. Several ways have been described for linking the pins including rubber tubing (ie, Penrose drain, IV tubing, PVC tubing) that is filled with methylmethacrylate (ie, hoof repair material[a]) or car body filler. Also available is a methylmethacrylate putty[b] that is semi-solid. This material is kneaded and applied without the need for any tubing. The fumes emitted from this product are significantly less than the hoof repair material.

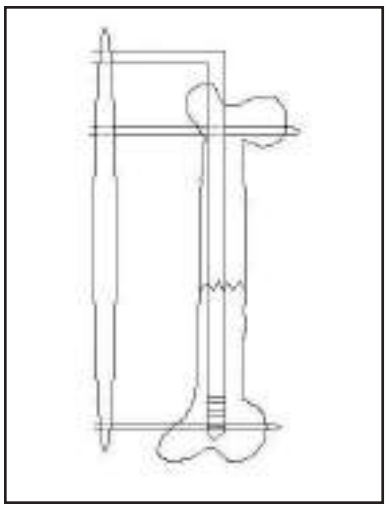


Fig 34.6 | Schematic of a tie-in fixator (TIF) comprising an IM pin linked to two cross-pins.

Managing Fractures of Specific Bones

THORACIC LIMB

Fractures of the scapula, coracoid and clavicle are managed conservatively in all sizes of birds. Figure-8 bandages with body wraps are left in place for approximately 3 weeks, then radiographs are made for reevaluation. Previous recommendations for internal fixation of coracoid fractures, compared to bandaging, resulted in lower success rates in birds of prey, even when severe displacement of fragments existed.[16]

Luxations of the elements of the shoulder girdle also are managed conservatively, with the exception of subluxation of the proximal end of the coracoid from the cranial aspect of the sternum. Open reduction of these subluxations is performed via a standard approach to the coracoid. The musculature of the keel is detached and reflected laterally to expose the proximal coracoid. An elevator or osteotome is used for reduction. Transarticular cerclage wire or pins may be placed if instability persists. The wing is bandaged for 3 weeks postoperatively with physical therapy beginning about 10 days postoperatively.

Patagium

The patagium is a soft tissue structure comprising muscular, elastic and tendinous tissues that extends from the shoulder to the metacarpus. This structure forms the leading edge of the wing during flight. Injury to the patagium may result in perforation or tearing. Healing of these injuries often results in contraction of the web, altering the conformation and restricting extension of the wing.

Sutures do not hold well in patagial tissue. This can be overcome by suturing a piece of cardboard slightly larger than the defect over the area. The splint should be replaced every 7 to 10 days until the defect is healed. The support of the cardboard allows extension of the wing during healing.

Humerus

Fractures of the humerus are classified anatomically into one of three zones: (1) The proximal zone, which extends from the tubercles to the pectoral crest, (2) the diaphyseal zone extending from just distal to the pectoral crest to the apex of the distal curvature of the bone, and (3) the distal zone, which is the curved portion of the bone adjacent to the elbow.

Proximal humeral fractures that are minimally displaced often heal well with a figure-8 wrap combined with a wrap to immobilize the wing against the body wall. However, in order to maximize postoperative flight chances, or in cases of displacement, internal fixation is required. Generally the proximal fragment is too small to drive two cross-pins for external fixation, and there is insufficient purchase for an IM pin. A tension band method of fixation has been described as the most effective technique in the management of these fractures.[16]

The proximal humerus is approached from the dorsal aspect. The major pectoral muscle and the deltoid muscle are elevated from their attachments to the pectoral crest. Two small-diameter pins are driven to exit at the dorsal and ventral aspects of the pectoral crest. Following fracture reduction, the pins are driven into the distal fragment, which results in tension exerted against the medullary cavity. This advancement can be difficult, as the pins bend against the bone and each other. The pins should be driven in an alternating fashion, advancing each only a small amount at a time. The wires are left projecting from the head of the humerus for future removal. This fixation, in addition to wrapping the wing to the body for approximately 1 week, is sufficient in birds under 300 g.

In larger birds, following placement of the cross-pins, a hole is drilled approximately 1 cm distal to the fracture site and another just caudal to the exit point of the wires. A wire is passed through these holes and tightened in a figure-8 pattern to complete the tension band.

Fractures of the humeral diaphysis tend to be oblique and are best managed with a TIF device. The radial nerve, which must be identified and preserved, crosses the dorsal aspect of the humerus at approximately one half of its length.

There are two methods of placing the IM pin in the humerus: 1) the retrograde (ie, away from the fracture site) method that is generally used for fixation of open fractures, and 2) the normograde (ie, toward the fracture site) method for closed fractures.

For retrograde IM pin insertion, the patient is placed in ventral recumbency and the humerus is approached from the dorsal aspect. The diameter of the IM pin should be slightly larger than 50% of the diameter of the marrow cavity. The pin is introduced at the fracture site and driven retrograde, exiting the proximal humerus just distal to the shoulder. The bone chuck is then attached to the free end of the pin and the pin withdrawn until the interval end is flush with the fracture site. The fracture is reduced and the pin driven into the distal fragment. Care must be taken not to penetrate the distal end of the humerus, as damage to the triceps tendon and joint damage are common sequelae.

Fig 34.7 | Dorsal view of the correct introduction site for normograde placement of an IM pin in the humerus. Skin has been removed for illustration purposes.

Fig 34.8 | Dorsal view of the correct sites for placement of cross-pins in the humerus. Skin has been removed for illustration purposes.

Normograde pin insertion is often possible in closed diaphyseal fractures (Fig 34.7). A small skin incision is made on the dorsal aspect of the distal humerus just proximal to the lateral (or dorsal) humeral condyle. Following caudal retraction of the triceps tendon, a non-threaded pin is introduced. The fracture is reduced and the pin is driven proximally to engage the cortex of the proximal humerus in the midsection of the pectoral crest.

Regardless of the manner of IM pin insertion, positive profile interface K-wires are placed at the proximal and distal aspects of the humerus to link externally to the IM pin (Fig 34.8). The distal pin is placed first. A small skin incision is made just proximal to the highest point of the lateral (or dorsal) condyle and aimed toward the ventral condyle. The pin is driven until a full thread extends through the opposite cortex. Prior to placing the proximal pin, the wing is folded against the body to properly align the rotation of the fragments. The midsection of the free edge of the pectoral crest is palpated and fingers walked along it until the high point is reached. The pin is driven, parallel to the distal one, until both cortices are engaged. The free end of the IM pin is bent at 90° approximately 2 cm from the skin. The three pins are attached as previously described. It is not recommended to place the distal ESF pin directly through the condyles in fractures of either the distal humerus or the distal tibiotarsus. In many species the intercondylar sulcus is sufficiently deep that the pin will skewer the tendon (triceps or gastrocneumius) that rides in that sulcus. The pin is placed in a slightly proximal position: at the humeral epicondyle to which the tendon of the common digital extensor and supinator muscle arise and in the tibiotarsus, proximal to the supratendinal ridge.

Fractures of the distal humerus (ie, those that occur within 2 to 3 bone diameters of the distal humeral condyles) are problematic, as there is insufficient space for an IM pin to gain purchase in the distal fragment. A cross-pinning technique for these supracondylar fractures has been described.[16]

A skin incision is made dorsally to approach the fracture. The distal fragment is isolated and elevated while protecting the soft tissues and avoiding the separation of comminuted fragments from their soft tissue attachments. Two K-wires are placed in retrograde manner, at an angle such that they exit the fragment on the opposite side of the marrow cavity. When pin ends are flush with the fracture line, the fracture is reduced and the pins are driven into the proximal fragment. Movement is alternated between the pins, advancing about 0.5 cm at a time until properly seated. Pins are placed in the proximal and distal humerus as previously described and the elements attached to form a hybrid fixator device.

Radius and Ulna

The method of repair of fractures of the radius and ulna will depend upon the integrity of the other bone of the pair. External coaptation is a viable option in small companion birds when either the radius or the ulna is fractured and the displacement of fragments is minimal. Potential complications include patagial contraction as a result of prolonged immobilization and the formation of synostosis between any displaced fragments and the other bone. This significantly affects the bird's ability to fly, as both lift and descent require the radius to rotate about the ulna.

Internal fixation of the radius and/or ulna may be accomplished with ESF, IM pins, or a combination forming a TIF. The prognosis for diaphyseal fractures is good; however, some very proximal radius and ulna fractures may be managed only by transarticular ESF, with a very poor prognosis for return to flight. Repair of avulsion fractures of the olecranon has not been reported.

In cases of fracture of both the radius and ulna, fixation

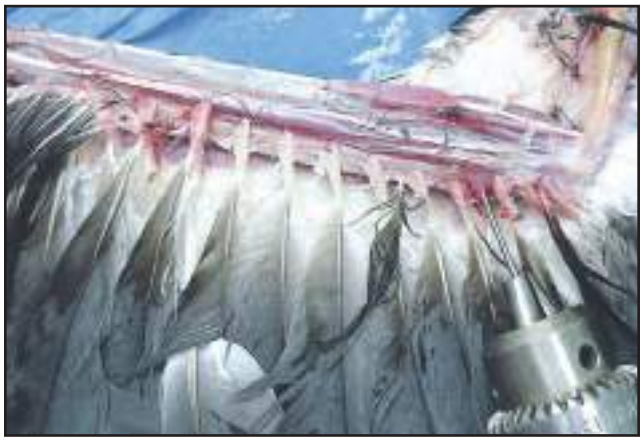


Fig 34.9 | Dorsal view of the correct introduction site for normograde placement of an IM pin in the ulna. Skin removed for illustration purposes.

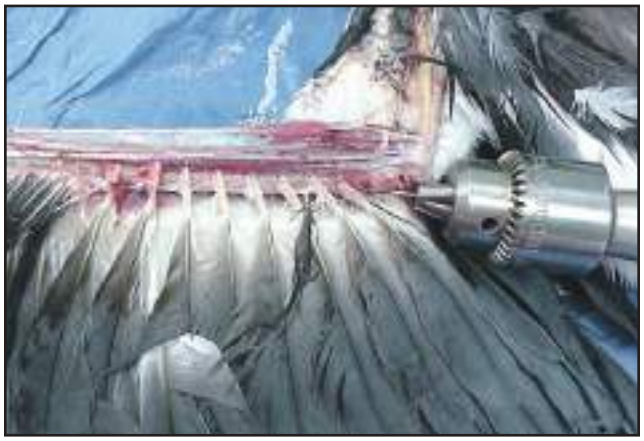


Fig 34.11 | Dorsal view of the normograde placement of an IM pin in the ulna: final alignment. Skin removed for illustration purposes.

of the radius is mandatory, while repair of the ulna is somewhat optional. Given the greater rigidity obtained and the shorter healing time, ulnar fixation is generally recommended. A combination of IM pin fixation of the radius and TIF fixation of the ulna is effective.

IM pins may be placed in the radius and the ulna, however, the method and location of placement is very different. In the radius, the pin is introduced at the site of the fracture and driven retrograde to exit at the distal end of the bone as the carpus is held in flexion. The pin will exit cranial to the carpal joint as the distal radius curves caudally. The fracture is reduced and the pin driven into the proximal fragment. Blunting the proximal tip of the pin may aid in avoiding penetration into the elbow joint.

The ulna is pinned in normograde fashion. Retrograde placement is contraindicated, as it risks exiting the pin at the olecranon and damaging the joint. The covert and down feathers are plucked from the dorsal and caudal aspects of the wing distal to the olecranon. Secondary flight feathers are left untouched. The pin insertion

Fig 34.10 | Dorsal view of the normograde placement of an IM pin in the ulna: gradually reducing the angle of introduction. Skin removed for illustration purposes.

point is on the caudal aspect of the ulna between the shafts of the second- and third-to-last secondary feathers. A small skin incision is made and the pin introduced at nearly a right angle to the caudal bone cortex. As the pin is slowly driven through the cortex, the angle is gradually reduced to become aligned with the long axis of the ulna (Figs 34.9-34.11). The fragments are manipulated into reduction and the pin is seated in the distal aspect of the bone. The proximal ulnar cross-pin is placed between the proximal end of the ulna and the IM pin. It should not impinge upon the elbow joint. The carpal joint must be avoided when placing the distal cross-pin.

Elbow Luxations

Moderate success has been reported in the surgical repair of caudodorsal luxations of the elbow with the following technique.[16] A skin incision is made over the dorsal surface of the wing, and the distal end of the humerus and the proximal antebrachium are exposed. Exposure of the joint is improved by transecting the tendon of origin of the supinator muscle if it remains intact. The proximal end of the ulna is levered into place by inserting a flat periosteal elevator between the proximal ulna and the dorsal (or lateral) humeral condyle and levering the ulna distally until it aligns with the humeral condyle. Application of traction to the distal ulna may be beneficial. The cut ends of the supinator muscle are resutured and a pseudocollateral ligament is made by suturing the edge of the triceps tendon to the common digital extensor tendon. Following closure, a transarticular ESF is placed for 7 to 10 days.

Carpal and Metacarpal Bones

Factors that complicate the management of these fractures include the paucity of soft tissue structures around these bones to protect them and provide blood supply as well as the high incidence of open comminuted fractures

Fig 34.12 | Preoperative view of a fracture of the major and minor metacarpal bones.

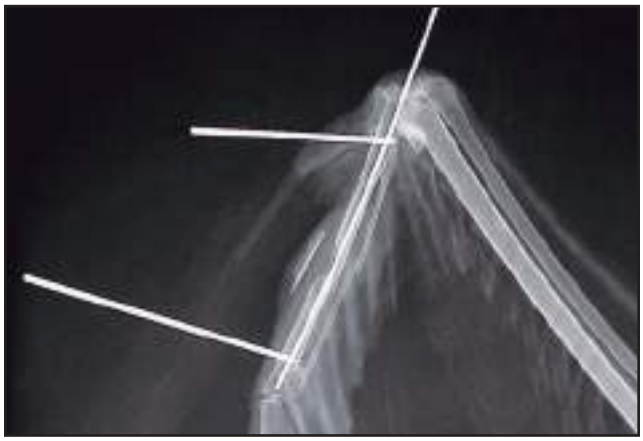


Fig 34.13 | Postoperative view of a reduced fracture of the major and minor metacarpal bones. The IM pin has not yet been bent nor attached to the cross-pins.

as a result of high-energy collisions with wires or projectile impacts. Fractures of the metacarpal bones have a lower rate of successful fixation than other avian fractures.

There are two main techniques used in the management of metacarpal and carpal bone fractures: the curved-edge sandwich splint, and ESF (Type I). The later has yielded the best overall results.

Curved-edge sandwich splints are suitable for stabilizing closed, easily reduced metacarpal fractures. These splints are formed with a right-angle bend in the edge of the splint that runs along the leading edge of the wing. The splint material is cut to a length equal to the distance from the cranial edge of the metacarpus to the distal phalanx and wide enough to cover five to six primary feather bases. The material is laid on the ventral surface of the wing, and the cranial edge is bent at 180° and trimmed flush with the dorsal wing surface. Overlapping adhesive tape strips are applied lengthwise to the dorsal and ventral surfaces of the feather shafts to keep the splint in place. Tape edges are pressed together to provide strength.

ESF are ideal for highly comminuted fractures of the metacarpal bones with extensive soft tissue damage. The fragments can be reduced with minimal soft tissue manipulation.

TIF are not ideal due to the great care that must be taken when placing the IM pin, as there is significant risk of injury to the distal radius. For placement, the pin is introduced at the fracture site and driven proximally while the carpal joint is held in flexion. Following reduction of the fragments, the pin is seated in the distal fragment and bent at 90° to attach to the fixator (Figs 34.12, 34.13). IM pins alone are unsuitable options for metacarpal fracture fixation due to the risk of injury to the radius, coupled with the need for supplemental external coaptation to control rotation forces.

PELVIC LIMB

The femoral head is connected to the acetabulum by the femoral capital ligament and supported by thickenings of the joint capsule. Traumatic rupture of the femoral capital ligament results in dislocation of the joint.

In small birds, closed reduction and strict cage rest may be effective in the management of coxofemoral luxations; however, in larger species an open reduction is preferred. A cranio-dorsal approach to the joint avoiding the antitrochanter is favored, as exposure is increased. The femoral head is replaced in the acetabulum and soft tissues repaired. Strict cage rest for 3 weeks postoperatively is necessary.

Pins should not be used to fix the femoral head into the acetabulum, as there is a high risk of damaging the kidney and its blood supply on the medial aspect of the pelvis. Additionally, only the rim of the acetabulum is bony in birds; the rest of the structure is fibrous.

Femur

The femur is surrounded by the *iliotibialis* and *femorotibialis medius* muscles cranially, the body wall medially, and the *flexor cruris* and *puboischiofemoralis pars medialis* muscles caudally. The ischiatic vein, artery and nerve are identified caudolaterally proximally and pass more laterally toward the distal femur. The surgical approach to the femur is made from the lateral aspect.

Fractures of the proximal femur are managed with a tension band apparatus similar to that described for the proximal humerus. The cross-pin technique described for fixation of supracondylar fractures of the humerus is well suited for distal femoral fractures.

Fig 34.14 | Lateral view of the placement of an IM pin and cross-pins (hypodermia needles) in the femur. Note the neurovascular bundle coursing caudal to the bone. Skin removed for illustration purposes.

Diaphyseal fractures are best managed with a TIF. The IM pin is introduced at the fracture site and retrograded to exit at the hip. Following fracture reduction, the pin is driven into the distal fragment. The cross-pins are placed from slightly craniolateral to caudomedial to best avoid the neurovascular bundle (Fig 34.14). The proximal pin is placed through the femur just distal to the dorsal acetabular rim. The distal pin is placed through the condyles. IM pins alone are often sufficient in birds under 100 g.

Luxations of the Stifle

Luxations of the stifle or femorotibial joint may be managed using one of several methods. One recently reported method involves the placement of IM pins into the femur and the tibiotarsus at the stifle.[3] The free ends of these pins, projecting at the stifle, are bent parallel to each other, and with the leg in normal perching position, the pins are held together with a small amount of acrylic. Transarticular ESF placement, with threaded pins placed in the femur and tibiotarsus and connected with acrylic, has also been described.[6] Ruptured cruciate and collateral ligaments may be repaired using PTFE (Teflon) suture material as a replacement. Hypodermic needles are used to drill holes in the bone and the suture material is then threaded through the needles.

Tibiotarsus

Tibiotarsal fractures are very common in pet birds and in falconry birds. Psittacines tend to have mid- to distal diaphyseal fractures, whereas proximal-third fractures are more common in the raptors used in falconry. Tibiotarsal fractures are usually closed and the prognosis for repair is good. Damage to the tibial and or fibular nerves is not uncommon and must be evaluated. Also, particularly in raptors, bumblefoot of the opposing limb is a concern.

Patients under 300 g will heal very well with a combination of an IM pin or K-wire and external coaptation in the form of an Altman splint. Hypodermic needles make excellent IM pins, and a 22-gauge needle works very well in budgerigars, cockatiels and similar-sized birds. The pin is introduced into the cranial aspect of the proximal tibiotarsus, avoiding the patellar ligament, and following fracture reduction, advanced into the distal fragment. This is easily accomplished without a chuck, using the plastic hub of the needle to grasp. The hub is then trimmed off the needle. This IM pin technique is combined with an Altman splint to control rotation forces.

To place an Altman splint (Figs 34.15a-d), the bird is placed in lateral recumbency. Overlapping strips of adhesive tape are placed laterally and medially in a horizontal fashion, with the sticky side toward the limb, beginning just proximal to the stifle and continuing distally to immobilize the hock. The sticky sides of the tape are pressed together with hemostats on the cranial and caudal aspects of the limb. The limb should be splinted in a normal perching position. Several layers may be necessary to give strength to the splint, or the tape may be coated with a small amount of cyanoacrylic glue to strengthen it. As an alternative to white adhesive tape that can be difficult to remove, self-adhering bandage material[c] can be used in much the same way. Healing takes approximately 3 weeks.

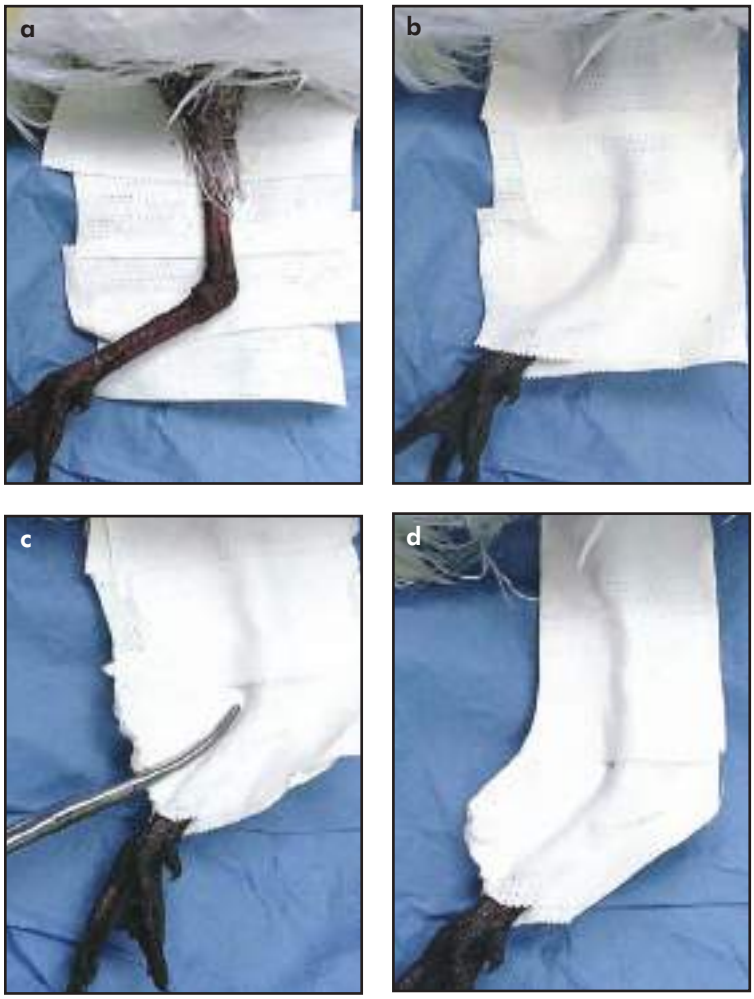
In larger birds, a TIF is the fixation method of choice (Figs 34.16, 34.17). The IM pin may be placed in a normograde fashion as previously described or in retrograde fashion from the fracture site. Pins should not exit into the intertarsal joint, as the tendons of the digital flexor muscles pass through the tibial cartilage in this area and may be damaged.

In planning a surgical approach or placement of the cross-pins, the fibula and neurovascular bundle laterally, and the gastrocnemius muscle caudally, dictate a craniomedial approach. The distal cross-pin is placed from lateral to medial through the condyles. The proximal cross-pin should be introduced on the craniolateral aspect just distal to the tibial plateau and cranial to the fibula. The pin is directed caudomedially. These pins are attached to the IM pin to form the fixator. Type II ESF fixators also have been used with great success in the management of tibiotarsal fractures.

Rotational or angular deformities may be corrected with an osteotomy and either ESF or a TIF.[6]

Tarsometatarsus

The shape of the tarsometatarsus varies among families of birds. The tarsometatarsus of hawks has a flat, C-shaped cross-section with little medullary cavity, whereas in parrots, the bone is rounder with a larger cavity. IM



Figs 34.15a-d | Step-by-step application of an Altman splint.

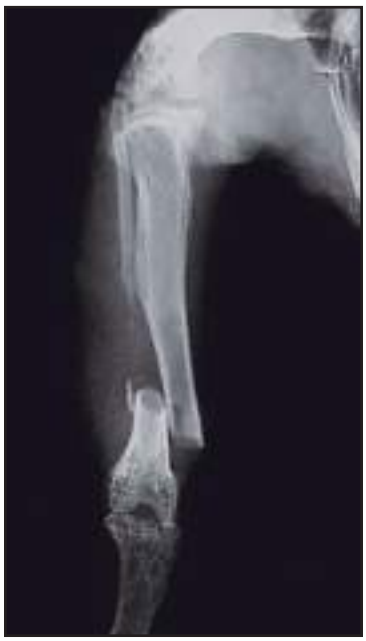


Fig 34.16 | Preoperative view of the fracture of the tibiotarsus and fibula.

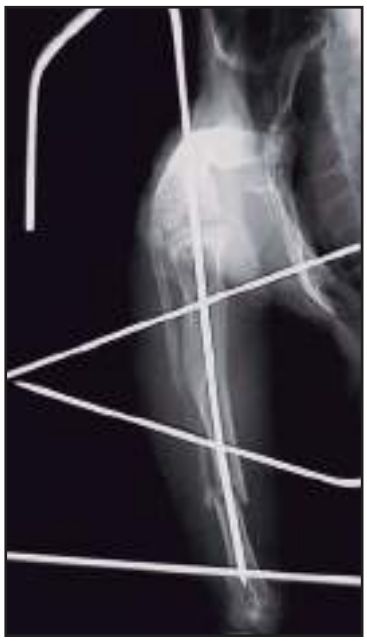


Fig 34.17 | Postoperative view of a reduced fracture of the tibiotarsus and fibula. The IM pin has not yet been attached to the cross-pins.

pins are not recommended, as the flexor tendons that run on the caudal aspect of the bone are affected. ESF cross-pins must be placed with caution to avoid the metatarsal artery and extensor tendons running dorsally, and the flexor tendons ventrally. Splinting of tarsometatarsal fractures with an L-shaped metal splint works well. The toes should be allowed to function freely during recovery to avoid immobilization of the flexor tendons during callus formation.

Phalanges

Closed phalangeal fractures are left unsupported and treated with cage rest. The flexor tendons and their sheaths provide good support. Splinting results in the formation of adhesions and a stiff toe. Compound fractures commonly result in osteomyelitis, and amputation should be considered. Dislocations of phalangeal joints may be reduced under anesthesia without the need for external support. Damaged collateral ligaments may be repaired with 3-0 or 4-0 polyglactin suture.

POSTOPERATIVE CARE

Gauze sponges are applied between the connecting bar and the skin to provide mild compression and to absorb fluids exuding around the pins. These should be changed daily. Pain is managed with opioids or NSAIDs. Analgesia is discussed in Chapter 8, Pain Management and elsewhere in this book. Perioperative antibiotic therapy with bactericidal antibiotics such as cefotaxime[d], enrofloxacin[e], or clavulanated amoxicillin[f] is warranted. In patients where osteomyelitis is a concern, clindamycin[h] is indicated. The use of antibiotic-impregnated polymethylmethacrylate (AIP-MMA) beads (also called MMP beads) should be considered in patients with open, contaminated fractures with infected bone.

Postoperative radiographs are always indicated to assess the alignment and apposition of the reduction, regardless of the fixation technique used. Radiographs should be repeated in 10 to 14 days to assess healing. Fractures will often heal with significant fibrous callus that will not be initially evident radiographically. Palpation of the fracture site yields additional information regarding fracture stability.

Complications

AMPUTATION

The ability of an individual bird to deal with either thoracic or pelvic limb amputation depends on the bird's size, demeanor and required return to function. Amputation through bone is preferred to disarticulation. The bone end will atrophy and maintain adequate soft tissue coverage.[18]

Wing amputations are quite feasible in most parrots, though balance is significantly affected. Most birds will learn to adapt. The wing is generally amputated at the junction of the proximal and middle thirds of the humerus, leaving sufficient soft tissue for closure.

Pelvic limb amputation always carries with it the concern of development of bumblefoot on the opposing limb. Parrots, especially the smaller varieties, fed formulated diets and offered appropriate perches, tolerate pelvic limb amputation well. There are two common sites for amputation: the proximal tarsometatarsus and midfemoral. The advantage of a tarsometatarsal amputation is the creation of a weight-bearing stump covered by the thick, scaly skin in the area. Some birds will traumatize this stump, and a midfemoral amputation site is cosmetic as well as leaving adequate soft tissue for closure. Postoperatively, birds often benefit from wider, padded perches until they regain their balance.

MALUNION/NON-UNION

Malunions and non-unions are the results of instability at the fracture site. Management includes ensuring adequate immobilization of the fracture, which may include additional apparatus, additional coaptation or a decrease in the activity level of the bird. If a callus is present, this material may be removed and packed into the defect as a graft. In more long-standing cases, bone grafting may be required. A piece of bone from the carina may be harvested, chopped into small pieces and packed into the defect. This is cortical bone and may become a sequestrum.

OSTEOMYELITIS

As previously mentioned, many avian fractures are open and contaminated. Routine antibiotic therapy is instituted as discussed. Cases of postoperative osteomyelitis are managed with surgical debridement and antibiotic therapy based on culture and sensitivity. Lincomycin[i] and clindamycin[h] are generally the drugs of choice.

Implantation of AIPMMA beads may be beneficial, as high concentrations of antibiotic are reached in their surrounding area. Consider *Aspergillus* sp. and *Mycobacterium* spp. as etiologic agents in unresponsive cases.

SEPTIC ARTHRITIS

Joints may become infected through a direct penetrating wound or via the hematogenous route. Clinical signs include lameness and swelling of the joint. Diagnosis is obtained through radiographs, cytology and culture of the joint fluid. Radiographic signs of septic arthritis include increased radiodensity of the subchondral bone and osteolysis as the disease progresses. Treatment combines daily irrigation of the joint with saline and an antibiotic, in conjunction with oral antibiotics based on culture and sensitivity. Radiographic bone changes are a poor prognostic indicator. Although the infection may be controlled, a decrease in the range of motion of the joint should be anticipated.

BUMBLEFOOT

Bumblefoot, a combination of pressure sores and infection of the plantar aspect of the foot, is a common condition of captive birds of prey and waterfowl and also is seen in psittacines, primarily cockatiels and Amazon parrots. Predisposing factors include obesity, poor diet, inactivity, inappropriate perches and uneven weight bearing, as is often the case in pelvic limb injuries.

Initial lesions are recognized as hyperemia and flattening of the skin of the digital and metatarsal pads, the sites of maximum weight bearing (Type I) (Figs 34.18, 34.19). These lesions progress if untreated and bacterial invasion of the subcutis occurs, resulting in a scab and mild swelling (Type II) (Figs 34.20, 34.21). Some may further progress to form a caseous abscess with marked swelling and pain (Type III) (Fig 34.22). Infection of the tendon sheaths results in an infection and corresponding cellulitis tracking toward the intertarsal joint and the digits, flexor tendon rupture (Type IV) (Fig 34.23), osteoarthritis of the sesamoid bone ventral to digit II, and septic arthritis of the tarsometatarsal-phalangeal joints (Type V) (Fig 34.24).

Treatment and prognosis depend on the degree of disease, but all birds with lesions should have the following changes made:

- Correct dietary deficiencies. In parrots, it is crucial to convert to a formulated diet.
- Alter perching surfaces to allow more even weight bearing. Wrapping wood perches with bandaging material[c] or covering perches with artificial turf or other carpeting material will result in a different weight distribution each time the bird perches. Cement and sandpaper-covered surfaces must be eliminated.
- Reduce the bird's weight. This will often accompany conversion to a formulated diet from a high-fat seed diet.


Greg J. Harrison

Fig 34.18 | Early Type I bumblefoot in a parrotlet (*Forpus* sp.) that selected only oats from a seed/pellet diet.


Greg J. Harrison

Fig 34.19 | Advanced Type I bumblefoot in a budgerigar (*Melopsittacus undulatus*) on an all-seed diet.


Greg J. Harrison

Fig 34.20 | Type II bumblefoot in a flamingo fed an all-grain diet and housed on a poor floor surface.

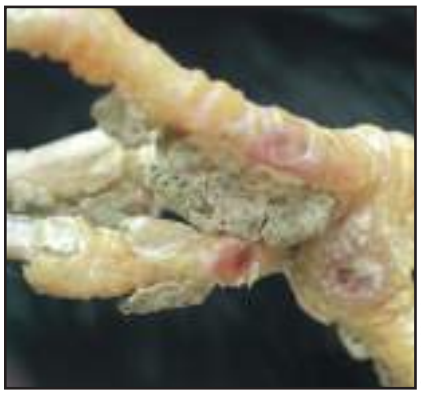

Greg J. Harrison

Fig 34.21 | Type II bumblefoot in a mynah bird (*Gracula religiosa*) with iron storage disease.


Greg J. Harrison

Fig 34.22 | Advanced Type III bumblefoot in a cockatiel (*Nymphicus hollandicus*) fed an all-seed diet.


Greg J. Harrison

Fig 34.23 | Type IV bumblefoot in a 20-year-old cockatiel (*Nymphicus hollandicus*) with deformed bones, tendons and ligaments fed an all-seed diet.

- Increase exercise. Pets should be encouraged to walk around on various surfaces and must be let out of their cage. Allow flight.

The above changes are usually sufficient for treatment of Type I disease. Refractory cases should be evaluated for other systemic disease that may be decreasing perfusion to the foot or otherwise delaying healing.

All birds with Type II-V bumblefoot should be managed with antibiotics. The choice of drugs is based on culture and sensitivity testing. Good initial choices include the amoxicillin/clavulanic acid[f] combinations, enrofloxacin[e], or Lincocin[i].

Type II bumblefoot may be treated with thorough cleaning of the feet and application of medication to soften the scab, such as hydrocolloidal wound dressing material or sodium fusidate[j] ointment. Once the scab is removed, the underlying wound is sutured and bandaged.

More advanced cases (Types III, IV, V) require surgical intervention. Treatment is aimed at wound debridement and re-epithelialization. AIPMMA beads may be left in the wound to increase antibiotic levels locally.[5] Large wounds are managed with a purse-string suture and allowed to granulate in. A rigid foot cast is made to support the foot and raise the plantar surface to avoid contact with the perch.


Greg J. Harrison

Fig 34.24 | Type V bumblefoot in an American bald eagle (*Haliaeetus leucocephalus*) with improper diet and housing.

Prognosis for Types IV and V bumblefoot is guarded to poor. The disease often results in deformity and dysfunction of the toes.

Products Mentioned in the Text

a. Technovit, Jorgensen Laboratories Inc, Loveland, CO, USA www.jorvet.com, www.3m.com/us/healthcare/professionals/animalcare
b. ESF putty, Jorgensen Laboratories Inc, Loveland, CO, USA www.jorvet.com
c. Vetrap Bandaging Tape, 3M Animal Care Products, St. Paul, MN, USA
d. Claforan, cefotaxime, Hoechst-Roussel Pharmaceuticals, Kansas City, MO, USA www.aventis- us.com/PIs/claforan_TXT.html
e. Baytril, Bayer, Shawnee Mission, KS, USA www.bayeranimalhealth.com
f. Clavamox, Pfizer Animal Health, Exxon, PA, USA www.pfizer.com/ah
g. Suitably sized IM pins are available in the US from IMEX Veterinary Inc, Longview, TX, USA , www.imexvet.com
h. Clindamycin- Antirobe - Upjohn, Kalamazoo, MI. 616-329-8244
i. Lincomycin- Lincocin Upjohn, Kalamazoo, MI. 616-329-8244
j. Sodium fusidate ointment, Fusidic acid www.drugs.com/xq/cfm/pageID_0/search_fusidic%20Acid/start_31

References and Suggested Reading

1. Aron DN, et al: Experimental and clinical experience with an IM pin external skeletal fixator tie-in configuration. Vet Comp Ortho Traumatol 4:86-94, 1991.
2. Bennett RA, Harrison GJ: Soft tissue surgery. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l Inc, 1999, pp 1096-1137.
3. Bowles HL, Zantop DW: A novel technique for luxation repair of the femorotibial joint in a monk parakeet (*Myiopsitta monachus*). J Avian Med Surg 16(1):34-38, 2002.
4. Degernes L, Roe SC, Abrams CF: Holding power of different pin designs and pin insertion methods in avian cortical bone. Vet Surg 27:301-306, 1998.
5. Forbes NA: Antibiotic-impregnated polymethylmethacrylate beads in the treatment of bumblefoot, osteomyelitis and other localized infections in birds. Proc Assoc Avian Vet Aust Com, 2000, pp 82-87.
6. Harcourt-Brown NH: Orthopedic conditions that affect the avian pelvic limb. Vet Clin Nor Am Exot An Pract 5(1):49-81, 2002.
7. Martin H, Ritchie BW: Orthopedic surgical techniques. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l Inc, 1999, pp 1137-1169.
8. Newton CD, Zeitlin A: Avian fracture healing. J Am Vet Med Assoc 170(6):620-625, 1977.
9. Orosz SE: Clinical considerations of the thoracic limb.Vet Clin Nor Am Exot An Pract 5(1):31-48, 2002.
10. Orosz SE: Surgical anatomy of the pelvic limb. Proc Assoc Avian Vet, 1999, pp 389-398.
11. Orosz SE: Avian surgical anatomy of the proximal pelvic limb. Proc Assoc Avian Vet, 2002, pp 317-321.
12. Peirone B, et al: Femoral and humeral fracture treatment with an intramedullary pin/external fixator tie-in configuration in growing dogs and cats. Vet Comp Ortho Traumatol 15:85-91, 2002.
13. Redig PT: Decision making in avian orthopedics. Proc Assoc Avian Vet, 1998, pp 253-263.
14. Redig PT: General aspects of avian orthopedic surgery: The pelvic limb. Proc Assoc Avian Vet, 1999, pp 399-411.
15. Redig PT, Korbel RT, Grimm F: Laboratory procedures for avian orthopedics. Proc European Assoc Avian Vet, 2001, pp 323-330.
16. Redig PT: Master class: Anatomical and surgical considerations of the avian thoracic limb. Proc Assoc Avian Vet, 2000, pp 429-438.
17. Redig PT: Orthopedic fixation for long bone fractures of raptors and other large birds: Pelvic limb. Proc Assoc Avian Vet, 2002, pp 323-335.
18. Weigel, JP: Amputations. *In* Slatter DS (ed): Textbook of Small Animal Surgery 2nd ed. Philadelphia, WB Saunders Co, 1993, pp 1901-1910.
19. West PG, et al: Histomorphometric and angiographic analysis of bone healing in the humerus of pigeons. Am J Vet Res 57(7):1010-1015, 1996.

CHAPTER
35

Surgical Resolution of Soft Tissue Disorders

HEATHER L. BOWLES, DVM, Dipl ABVP-Avian, Certified in Veterinary Acupuncture (Chi Institute); ESPEN ODBERG, DVM; GREG J. HARRISON, DVM, Dipl ABVP-Avian, Dipl ECAMS; JACK J. KOTTWITZ, DVM

Espen Odberg

Preparation and Considerations for Avian Surgery

PATIENT EVALUATION

A full signalment and history should be obtained and a physical examination performed prior to anesthesia and surgery (see Chapter 6, Maximizing Information from the Physical Examination and Chapter 34, Surgical Resolution of Orthopedic Disorders).

Preoperative diagnostics may include a complete blood count, serum chemistry and electrolyte profile, and other serologic or hematologic tests. The surgeon should evaluate the risk/benefit ratio for each diagnostic test, particularly in smaller avian patients (<100 g). Presurgical blood collection may cause hypovolemia and/or anemia that may dispose these birds to life-threatening intraoperative hemorrhage. Hematologic findings that may preclude or delay elective surgery are similar to those in domestic animals. These include the following:

1. An elevated hematocrit, which may indicate dehydration or cardiovascular compromise.
2. A decreased hematocrit. Anemic patients have increased surgical risks, particularly if there is perioperative hemorrhage.[5] Severe anemia (ie, PCV <20%) requires correction or treatment prior to surgery.
3. A decreased total protein, which may delay healing as well as indicate underlying metabolic disease.
4. Leukocytosis, which may indicate an infectious or inflammatory process, requiring pre-, peri- and postoperative antimicrobial therapy. Perioperative antibiotic or antifungal agents should be administered when indicated.

5. Hypocalcemia that may pose an anesthetic risk, compromise cardiac and neurologic function, and impair bone healing subsequent to fracture repair.[1] Evaluation of both total calcium and ionized calcium is strongly recommended prior to surgery[1] (M. Stanford, personal communication, 2000), particularly in those patients with associated malnutrition or history of egg production.
6. Organ system disease (eg, renal, pancreatic), which may require medical therapy prior to surgery and may influence anesthetic choices.
7. Hepatic insufficiency which may debilitate the patient due to decreased hepatic production of clotting factors as well as reduced liver function, that may affect various critical aspects of patient homeostasis.
8. Reduced thrombocyte counts, which may predispose the patient to coagulopathy.
9. Electrolyte imbalances.[1,5,8]

Radiographs and ultrasonography may be utilized in establishing a diagnosis, treatment plan and prognosis, as well as determining the patient's degree of anesthetic and surgical risk.

Cardiovascular function should be thoroughly evaluated prior to anesthesia and surgery. Arrhythmias, murmurs, tachycardia, bradycardia and pulse abnormalities (pulse deficits or jugular pulses) should be investigated with radiographs, electrocardiology, echocardiography and evaluation of blood pressure.[5,87]

Anorexia, disease and stress may all contribute to nitrogen imbalance. Starvation and disease may result in a hypermetabolic state, and stress may cause an initial hypometabolic state followed by a hypermetabolic state. Hypermetabolism increases the body's protein requirement. Birds have a higher protein requirement than mammals. Protein demands are further increased because there is increased need for tissue repair, blood cell production and antibody production with surgery.[5,87]

Carbohydrates are a nitrogen-sparing energy source and are recommended for correction of a stress-related negative nitrogen balance. A successful postsurgical patient requires a positive nitrogen balance to facilitate tissue repair, and a source of non-protein energy to meet their increased caloric requirements.[5] Patients with a decreased blood glucose level should be supported intravenously with dextrose as part of their fluid therapy pre-and intraoperatively.[1]

Resting basal metabolic rate (BMR) may be determined by using the formula BMR kcal/kg per day = $(K)BW^{.75}$. Additional energy is required for growth, reproduction, disease and tissue repair. Severe trauma and sepsis may increase the patient's energy requirements 1.5 to 3 times their resting requirement. There are several commercially available supplemental diets that can be utilized to meet these nutritional requirements, ranging from juvenile hand-feeding formulas to avian critical care diets[5] (see Chapter 7, Emergency and Critical Care).

Malnutrition may lead to obesity, vitamin A deficiency and other nutritionally related diseases. These conditions may pose anesthetic and surgical risks, and predispose the patient to infection, delayed healing and/or coagulopathies.[5]

PATIENT PREPARATION

Fasting

Birds normally maintain relatively low hepatic glycogen stores. Liver glycogen stores may decrease as much as 90% during a 24 to 36 hour fast and possibly more in small birds. However, it is important for the crop to be completely empty prior to anesthetizing the patient to prevent regurgitation and aspiration. Therefore, general guidelines include a short fast of 2 to 4 hours for birds <300 g body weight, 5 to 8 hours for birds >300 g body weight, 2 to 4 hours for frugivores and 24 to 36 hours for larger raptor species weighing 2 to 4 kg. It is generally recommended to leave water available until 1 hour prior to anesthesia.[5]

Decreased gastrointestinal motility may alter these recommendations. The crop should be thoroughly palpated immediately prior to anesthesia. If surgery must be performed and the crop is not completely empty, fluid or liquefied food may be aspirated through a feeding tube and/or the head should be elevated during the surgery to prevent regurgitation and aspiration of crop contents. It is advisable to intubate these patients to protect the airway.[5] Following intubation, cotton balls or gauze can be placed in the caudal pharynx to prevent reflux of crop contents.

Anesthesia and analgesia are indicated to promote patient comfort and reduce pre-, peri- and postoperative stress (see Chapter 8, Pain Management and Chapter 33, Updates in Anesthesia and Monitoring).

Positioning

Patient positioning varies with the surgical approach. Lateral and dorsal recumbent positions are most common (Fig 35.1). Respiration may be impaired when the avian patient is placed in ventral recumbency. Procedures that may require ventral recumbency include excision of the uropygial gland, surgery of the pygostyle and excision of dorsal feather follicle cysts.[1]

When the patient has been properly positioned, atraumatic adhesive tape such as masking or certain medical tapes may be used to secure the patient. Restraint

Espen Odberg

Fig 35.1 | The lateral and ventrodorsal patient positions are most commonly used for avian surgery. Care should be taken to avoid placing pressure on the thorax with surgical drapes, instruments or the surgeon's hands, as birds require movement of the keel and intercostal muscles to expand the thorax for respiration. Tape that is not traumatic to skin and feathers, such as masking or certain medical tape, may be used to position the patient.

Espen Odberg

Fig 35.2 | Prior to surgery, all feathers 2 to 3 cm from the surgical site should be removed. Standard aseptic technique applies to avian surgery. Chlorhexidine diacetate (0.05%), povidone iodine (1.0%) or chlorhexidine gluconate (4%) rinsed with alcohol and saline are all effective for presurgical skin disinfection.

boards are commercially available and are preferred by some surgeons. It is helpful to secure the patient to a platform that then may be repositioned as needed to facilitate intraoperative adjustments in surgical access.[1]

Birds require movement of the keel and intercostal muscles for respiration, therefore, care should be taken to avoid placing any pressure on the thorax with surgical drapes, instruments or the surgeon's hands.[1]

THERMOREGULATION

Hypothermia is a significant concern, particularly in small avian species. Several heating systems exist including circulating water heating pads, electric heat pads, heated forced air systems and overhead heat sources. Some of these may not provide adequate supplemental heat to the patient and others may mechanically interfere with the surgeon and/or anesthetist[5,67] (see Chapter 33, Updates in Anesthesia and Monitoring).

Creating a Sterile Surgical Field

In preparation for surgery the feathers should be removed in a 2 to 3 cm radius beyond the edges of the surgical site (Fig 35.2). It may not be possible to remove all the feathers from the surgical field due to skin trauma, species-specific skin fragility, location of the surgical site, need to retain flight feathers or patient body size. Removal of the rectrices or remiges, which insert into the periosteum of the associated bones, may increase postoperative pain. These larger feathers may instead be wrapped with sterile non-adherent bandage material. The remaining feathers may be retracted from the surgical field with light adhesive tape such as masking tape. A small amount of alcohol may be used to lightly wet feathers and smooth them away from the surgical site;[5] however, alcohol should not be used when radiosurgery or electrocautery will be employed. Alternately, sterile, water-soluble lubricant jelly may be used to smooth the feathers out of the surgical field.[5]

Standard aseptic surgical techniques apply to avian surgical procedures. Skin disinfectant scrub should be used to minimize the risk of bacterial contamination of the surgical site without damaging the skin. Chlorhexidine diacetate (0.05%) and povidone iodine (1%) have been found equally effective for skin preparation, and have no significant effect on wound healing. Chlorhexidine gluconate (4%) is equally effective when rinsed with alcohol or saline. Saline provides the benefit of not predisposing patients to hypothermia and does leave sufficient residual chlorhexidine solution bound to the skin. One study demonstrated that 50% of dogs developed erythema, edema, papules, wheals and weeping of serum from the skin when the skin was prepared with povidone iodine. This may suggest that chlorhexidine solutions are a less irritating class of disinfectants.[5]

Aseptic preparation of the cloaca is difficult. Maximum disinfection can be achieved by irrigating the cloaca with chlorhexidine gluconate (4%), then infusing antibiotic ointment.[5]

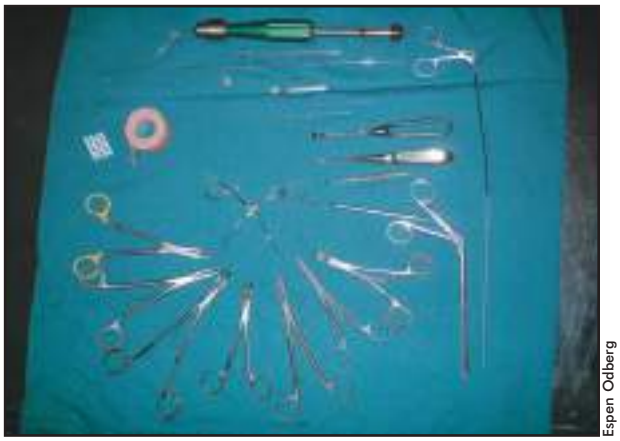
There are several lightweight surgical drapes available. Clear adhesive drapes allow the surgeon and anesthetist to monitor the rate and depth of respiration, and may be more effective in maintaining body temperature.

Greg J. Harrison

Fig 35.3a | A traditional small-animal thumb forceps with teeth is shown. This instrument has a curved tip and unique jaws for handling delicate tissues.

Greg J. Harrison

Fig 35.3b | The forceps in Fig 35.3a have unique jaws (40x). The central row of teeth is elevated to fit into a depression on the opposite jaw. The apposing jaw has a recession for the ridge to fit in. On either side of the ridge and recession are alternating teeth and an offset space to receive a tooth. This setup maximizes tissue contact. Dr. Harrison has found this to be the most versatile instrument in avian surgery.



Espen Odberg

Fig 35.3c | The basic minimal surgical pack should include top-quality instruments with carbide steel inserts. Minimal requirements for a basic surgical pack include Brown-Adson tissue forceps, Adson tissue forceps, smooth-tipped and toothed Bishop-Harmon tissue forceps (used for smaller patients), a larger pair of DeBakey tissue forceps, two or more curved-tip Halstead mosquito hemostats, tenotomy or small Metzenbaum scissors, and two needle holders. The recommended needle holders include a small Mayo-Hegar or Olsen-Hegar needle holder for 3-0 to 5-0 suture, and a smaller Castroviejo or Mathieu-type needle holder for 5-0 to 8-0 suture. The surgical pack also should include small, sterilized gauze pads and cotton swabs.

INSTRUMENTATION

Surgical instruments for avian surgery must be appropriately sized and designed to atraumatically manipulate delicate tissue (Figs 35.3a-c). Microsurgical tools such as those utilized in human vascular surgery, ophthalmic instruments and instruments specifically designed for small veterinary patients may be used. Microsurgical instruments should be of a standard length, counterbalanced and have miniature tips (Fig 35.4a). The length allows for balancing in the hand (Figs 35.4b,c). When utilized correctly, the arm provides stability while the fingers carefully move the tip. This balance, stability, and rounded shape allow for smooth, precise movement, thereby preventing any trauma to delicate tissues, blood vessels or nerves (Fig 35.4a-f). Jeweler's instruments are often used for their small size; however, these are usually not ergonomically designed for microsurgery. Several courses are available for microsurgical training. Microvascular and human plastic surgery techniques are particularly helpful with avian patients.[1,4,5]

Small, angled (60-90°) mosquito hemostats and hemostatic clip applicators are useful for avian celiotomies (Figs 35.4g-j). These allow increased accessibility to viscera and blood vessels located deep within the coelomic cavity. Angled DeBakey neonatal vascular clamps[a] are useful for ovariectomy.[2,4]

Sterile gavage and feeding tubes may be used for irrigation, to moisten tissue or to flush hollow viscera. Tuberculin or other small syringes may be used for suction, as some traditional suction units may traumatize delicate tissues. Mini-Frazier suction tips and Poole-type suction tips may be used in avian patients. The Poole-type suction tip may be fashioned from a rubber feeding tube by creating multiple fenestrations.[1,4,5]

Abdominal retractors must adequately retract tissue without causing tissue trauma (Fig 35.5). Mini-Balfour and Alm retractors may be used in larger avian species such as macaws and Amazon parrots. Heiss retractors and ophthalmic eyelid retractors may be used in small avian species such as cockatiels and budgerigars. Lone Star retractors are lightweight and allow the surgeon to achieve retraction at several areas surrounding the surgical site.[1,4,5]

HEMOSTASIS

Hemostasis is of the utmost importance in birds. Minor hemorrhage can result in severe compromise to these small patients. Large blood vessels are located just below the dermis and care should be taken to either avoid severing or to preempt bleeding prior to transection of these vessels. Several tools exist to assist the surgeon. These include chemical cautery agents, metal clips, radiosurgery, electrocautery and lasers.[1,5] Hemoclips[b] are small, atraumatic, stainless steel clips applied with hemostat-type applicators (Fig 35.4i). These applicators facilitate access into small, deep, difficult to reach areas.[1,5]

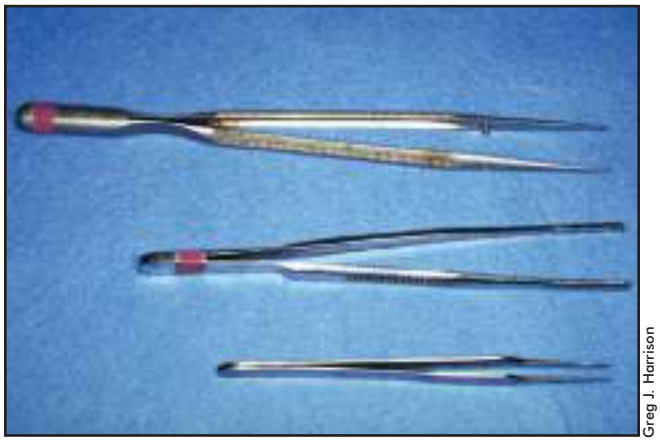

Greg J. Harrison

Fig 35.4a | Bottom to top: Standard delicate-tissue, small-animal thumb forceps. Regular thumb forceps. Microsurgical thumb forceps.


Greg J. Harrison

Fig 35.4b | Microsurgical forceps in Fig 35.4a showing the counterbalance and scooped-out area that fits over the web of the index-thumb finger area of the operator's hand. Grasping the round handles approximates using a writing instrument.

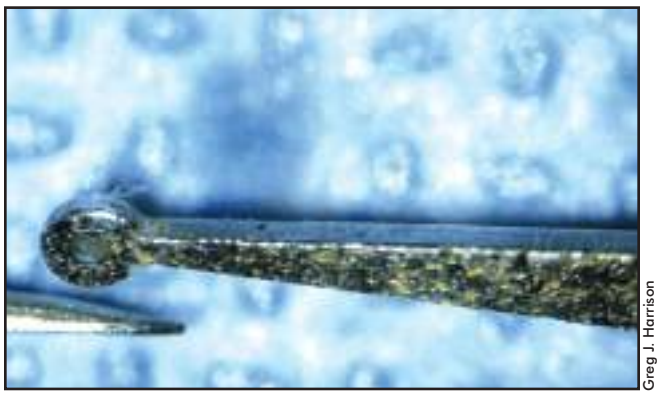

Greg J. Harrison

Fig 35.4c | The two tips available for microsurgical forceps (40x). On the left bottom is a fine needle tip that tends to slip if any tissue volume is grasped. On top is a circular or ring tip with titanium dust on the contact surface to aid in grasping. This greatly improves delicate tissue handling and reduces bleeding caused by slipping with the other forceps.

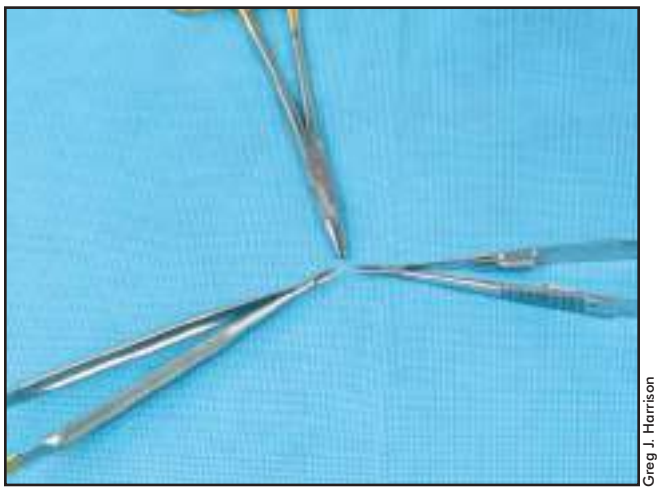

Greg J. Harrison

Fig 35.4d | Needle holders. Top: Standard small-animal surgical needle holder. Right: Delicate-tissue needle holder. Left bottom: Microsurgical needle holder. The extra-fine tip and the round handles allow increased speed and accuracy of suturing when handling 6-0 to 10-0 swaged-on suture material.

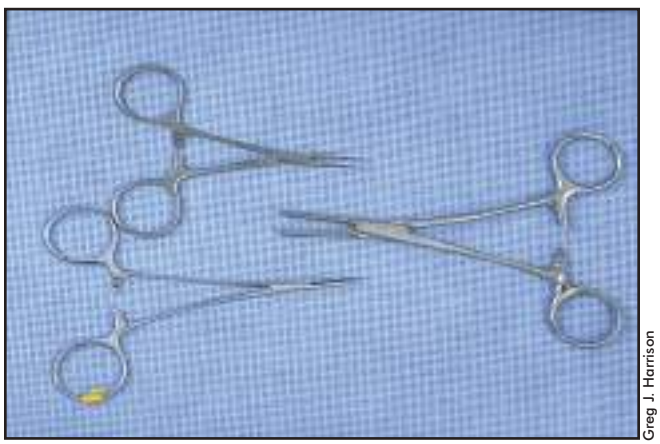

Greg J. Harrison

Fig 35.4e | Top to bottom: Delicate-surgery hemostat. Standard hemostat. Microsurgical hemostat.

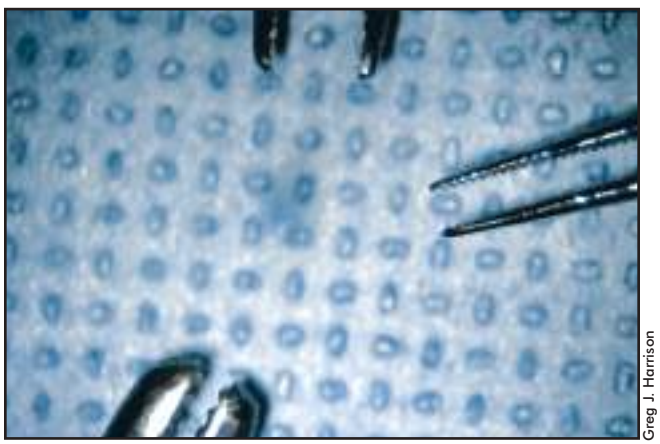

Greg J. Harrison

Fig 35.4f | Hemostat tips. Top: Delicate-surgery hemostat. Right: Microsurgical hemostat. Bottom: Standard hemostat. The microsurgical hemostat allows pinpoint grasping. Only the tiny area of concern is grasped. If needed these hemostats can be touched with the radiosurgery tips to coagulate the vessel contained within. Cauterization of surrounding soft tissue should be minimized to prevent unnecessary necrosis.

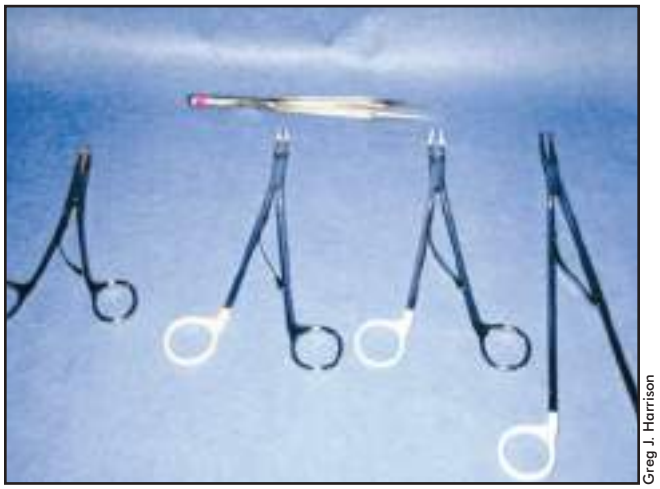


Fig 35.4g | Various hemoclip applicators. Left to right: Small hemoclip with 20° tip. Small hemoclip with 90° tip. Medium hemoclip with 20° tip. Medium hemoclip with 90° tip. Top: Microsurgical forceps for size comparison.

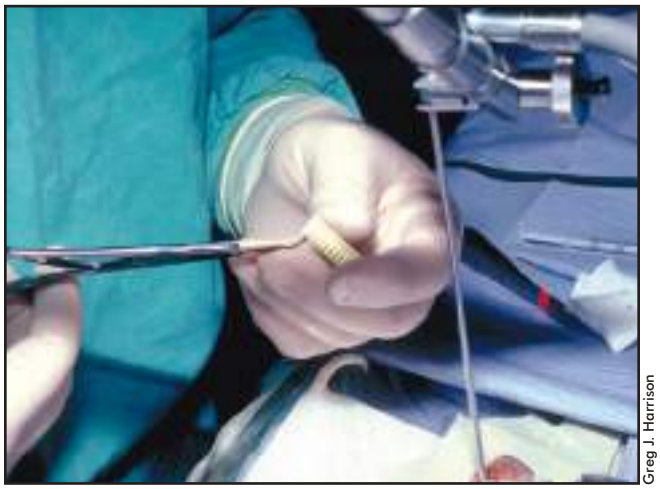


Fig 35.4h | Loading the hemoclip applicators from a clip cartridge.

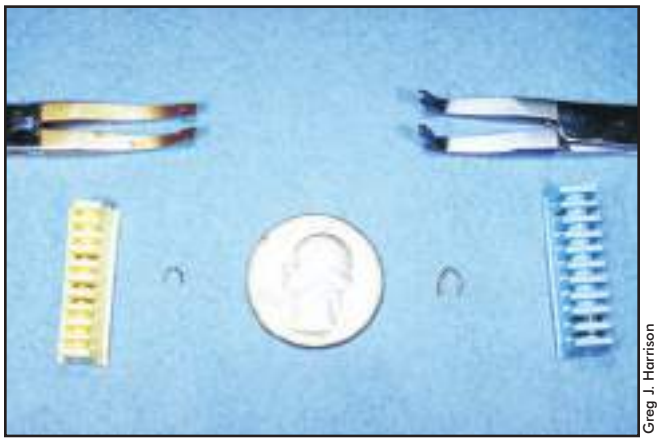


Fig 35.4i | Comparison of a small hemoclip applicator (left), a medium hemoclip applicator (right) and a US 25-cent coin.

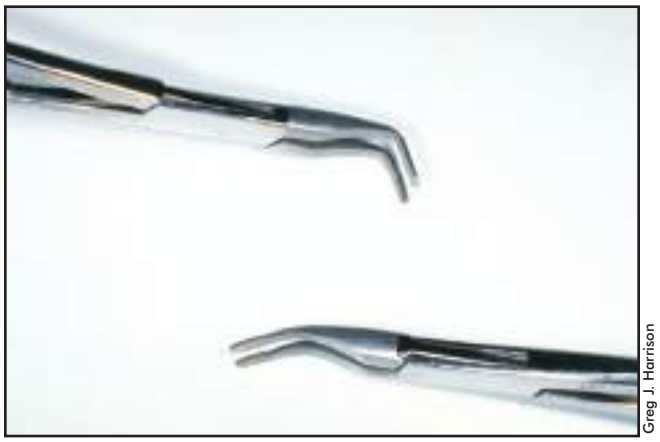


Fig 35.4j | Side view of the hemostat applicator tips. Top: 90° tip. Bottom: 20° tip.

Gel foam, surgical spears, Monsel's solution[c], chemical cautery, collagen sheets, beaded polysaccharide powder[d] and direct manual pressure can assist in the control of minor hemorrhage.[1,5]

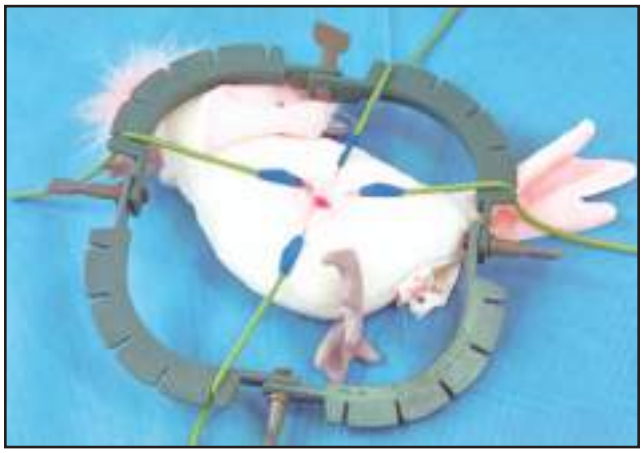
WOUND HEALING

Wound healing has been thoroughly evaluated in mammals and determined to occur in a sequential series of events. These include the inflammatory stage, fibroblastic stage, epithelialization phase, contraction phase and the remodeling phase. Cellular and vascular processes of the inflammatory stage have been evaluated in chickens and are similar to that described in mammals. Birds lack significant subcuticular tissue, therefore primary skin closure is often necessary. Excessive scar tissue does not typically form in birds (Fig 35.6).[5]

RADIOSURGERY

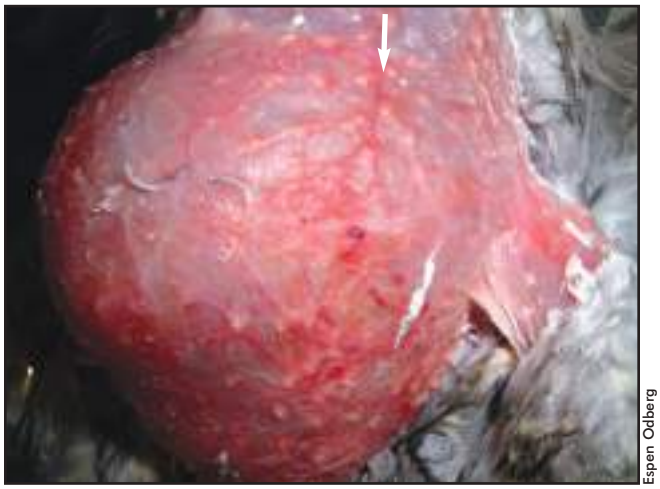
Radiosurgery utilizes high-frequency (2-4 MHz) alternating current to generate energy waves that create vibration and molecular intercellular heat (Fig 35.7a). This results in vaporization of water and rupture of affected cells while the electrode remains cool. The frequency may be manually set to cut tissues or coagulate blood vessels. Coagulation occurs when the current density dehydrates cells and coagulates the cellular contents (Figs 35.7b-e).[4,5]

When the radiosurgery unit is set for monopolar operation, it utilizes two electrodes: an active electrode and an indifferent electrode or ground plate. This concentrates the current density at the tip of the active (smaller) electrode. Monopolar radiosurgical technique is indicated for gross tissue manipulation in larger avian patients (>2 kg). The ground or indifferent electrode should be placed in contact with the patient as close to the surgical field as possible. Patient contact is improved with the application of a contact gel to the patient and ground. Alternatively, the ground plate may be permanently mounted under the surgery table and the patient placed



Greg J. Harrison

Fig 35.5 | Adjustable wound retractor frame and two tension bands hooked into one another. The location and tension on the band can be adjusted after inserting the hook into the surgical tissue. Then the band is slipped into a V-shaped slot in the frame at the ideal location and the desired pressure is applied.



Espen Odberg

Fig 35.6 | The scar from a previous surgery is visible (arrow). There may be minor adhesions between skin and the abdominal wall; however, scar formation is generally minimal in the avian patient.

on a non-metal material such as a towel. Such material will prevent thermal burns to the dependent aspect of the patient. This is particularly important in thin patients with prominent bony sites that provide small conduction points that can generate high localized temperatures. The active electrode must be kept clean and free of char and debris. An excessive amount of char or debris will interfere with conduction, thereby creating drag through the tissue, inhibiting cutting action and increasing coagulation. This may delay wound healing and predispose the patient to wound dehiscence. Several types of electrode tips are commercially available. Ball-tipped electrodes create significant tissue destruction for fulguration and coagulation of large amounts of tissue; loop electrodes are useful to obtain tissue biopsies and surgically excise tissues; and fine wire electrodes are utilized for incisions. It is useful to have some tips (such as those used in dentistry) that function in a wet field for effective coagulation during hemorrhage.[4,5]

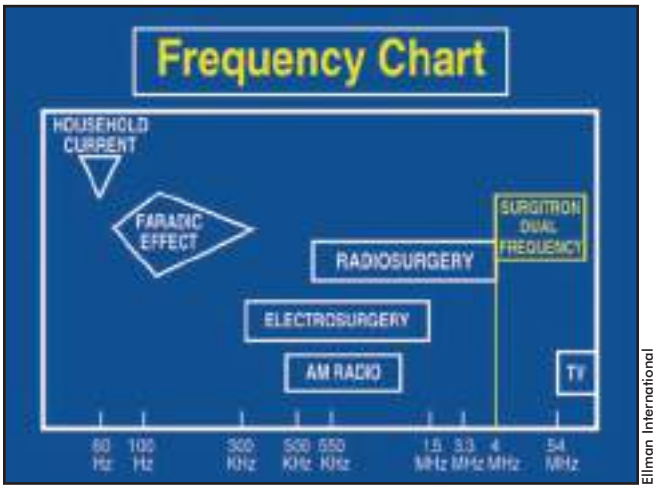
If cryosurgery is performed in conjunction with radiosurgery, it is important to note that radiosurgical tools will not work on frozen tissues.[4,5]

Bipolar radiosurgical forceps are useful, particularly in small avian patients (<2 kg) (Fig 35.7f). These allow for hemostatic control at the tip without the use of a ground plate, as one of the tips serves as the active electrode and the other as the indifferent electrode. The current passes from one tip, the active electrode, to the other, the indifferent electrode, without passing through the entire patient. Closer proximity of the two electrodes alters the transmitted wave currents from those transmitted by monopolar electrodes, resulting in more precise control and less reflux hemorrhage. The cut settings are used for tissue incisions. The cut/coagulation settings are indicated for vessels that are difficult to coagulate and for controlled cutting with coagulation properties (ie, as for organ biopsy). The coagulation settings are used for tissue fulguration. The material and design of the bipolar forceps, as well as proper calibration of the machine, determine the efficiency and performance of bipolar radiosurgery (Fig 35.7e).[4,5]

Bipolar forceps may be used to make primary skin incisions, to incise through muscle with minimal hemorrhage, and to coagulate cutaneous blood vessels prior to incision with a scalpel blade or scissors. The skin may be grasped and elevated with thumb forceps, then incised with bipolar radiosurgical forceps. This incision is then extended by inserting the indifferent electrode of the bipolar forceps subcutaneously to the full extent of the desired incision. The forceps are then apposed with the skin between them and the incision performed is extended by dragging the tips over the full thickness of the skin. This will incise the skin and coagulate the blood vessels. Correct settings and proper use of radiosurgery does not cause discoloration of the skin lateral to the incision (ie, if the skin is discolored, tissue damage has occurred and primary intention healing is unlikely).[5]

Tissue incisions may be performed with monopolar tips as well. Wire-type tips may be used as an "electrosurgical scalpel". The current should be initiated prior to touching the tissue. Often, higher settings are necessary in birds as compared to mammals due to the lower water content of the skin, which may result in less coagulation of associated vessels and hemorrhage. Cutting ability may be improved in very dry skin by moistening the skin with saline.[4,5] Feather follicles and their associated blood supply should be preserved whenever possible.[5]

When using the bipolar forceps for hemostasis, the forceps tips are relaxed as current is applied, providing a

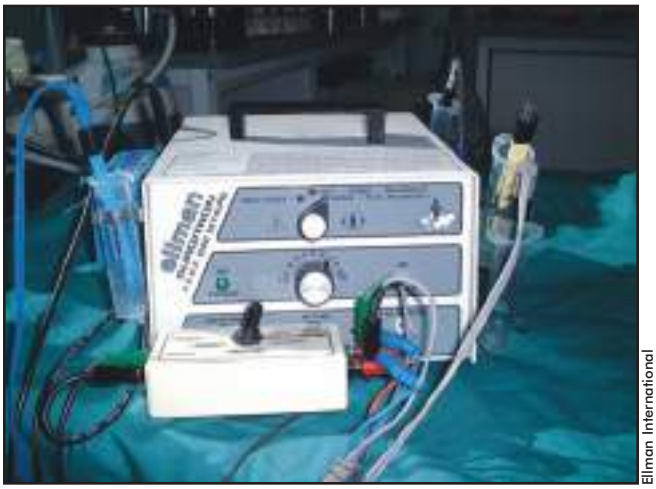


Ellman International

Fig 35.7a | Frequency chart.

Ellman International

Fig 35.7b | The multifrequency radiosurgery generator for performing avian surgery with minimal hemorrhage.

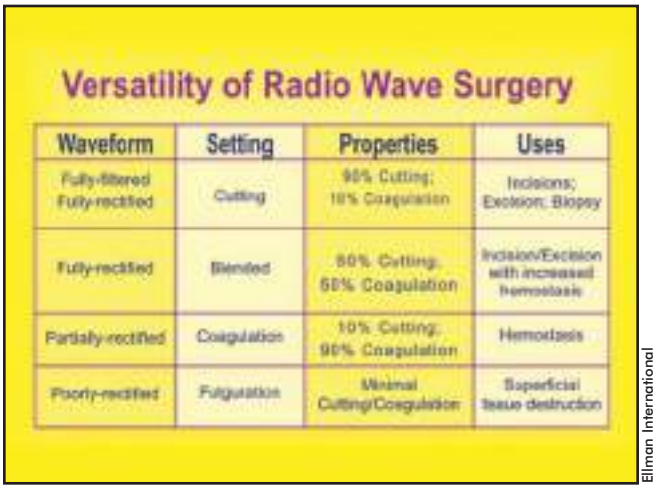


Ellman International

Fig 35.7c | This unit has been modified to allow multiple bipolar or unipolar hand pieces with a flick of a switch.

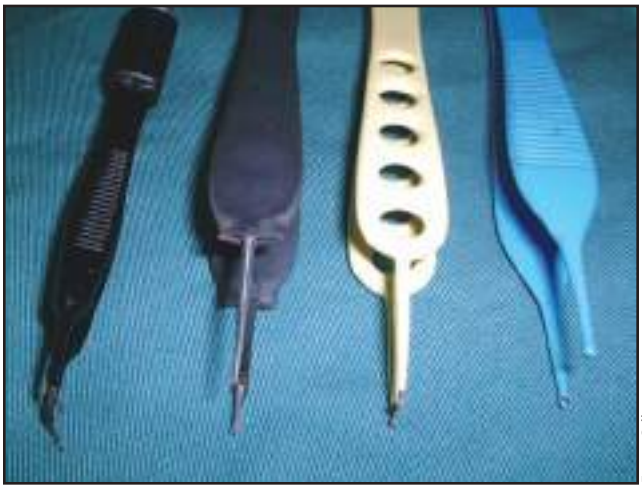
Ellman International

Fig 35.7d | Advantages of radio wave surgery.



Ellman International

Fig 35.7e | Versatility of radio wave surgery.



Espen Odberg

Fig 35.7f | Various bipolar forceps. Left to right: The Harrison modification and three custom ring-tip models.

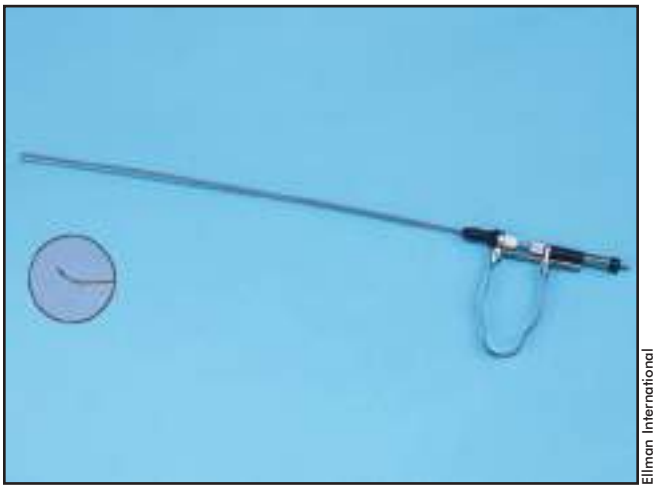


Fig 35.7g | The trigger handle allows endoscopic bipolar applications of a radio frequency instrument.

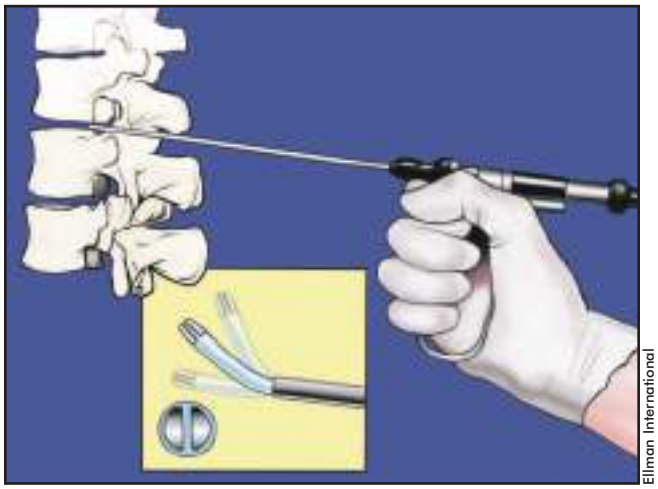


Fig 35.7h | The trigger handle has directional control. This illustration is from use in human disc disease therapy.

small gap between the two sides through which the radio current flows, thereby sealing the vessel. It is important to clean the forceps tips frequently. Accumulated blood and tissue can adhere to the clot and subsequently destroy it when the forceps are removed. Forceps tips are currently being developed with new materials that do not accumulate blood and tissue. When using the coagulation settings, the vessel may retract within the tissue due to vasospasm. This results in temporary hemostasis until the vessel relaxes, but hemorrhage may recur. A new modification that can be used with an endoscope has been developed (Figs 35.7g,h).

LASER SURGERY

Light Amplification by the Stimulated Emission of Radiation (LASER) relies on the production of electromagnetic radiation in response to photon emission by a lasing medium. Electrical energy excites a lasing medium (carbon dioxide, diode or argon) contained within an optical laser chamber, which, upon returning to a steadier electrical state, loses energy and generates photons in the form of electromagnetic radiation or light. These photons are directed from the optical laser chamber as monochromatic electromagnetic radiation transmitted via a series of lenses and delivery fibers in a focused and controlled laser beam. The type of lasing medium will alter the wavelength and frequency of the radiation.[38,69]

Carbon dioxide and diode lasers are most commonly used in veterinary medicine. Both produce an immediate region of vaporization, surrounded by a zone of irreversible photothermal necrosis and a zone of reversible edema. Laser surgical incisions seal blood vessels, nerves and lymphatics for controlled hemostasis, analgesia and postoperative edema. Carbon dioxide lasers operate at a wavelength of 10,600 nm. They may be operated with a focused beam, ideal for cutting, or a defocused beam, used for vaporizing tissue. They provide accurate, non-contact surgery with minimal tissue penetration and minimal collateral thermal injury (0.05-0.2 mm from the incision), as compared to the diode laser, which offers a zone of thermal injury of 0.3 to 0.6 mm from the incision. Thermal penetration is relatively superficial, penetrating only 50 to 100 μm in depth.[38,54,69]

Diode lasers operate at a wavelength of 635 to 980 nm. They may be operated in direct contact with tissue (contact mode) or at a distance from tissue (non-contact mode). They have the ability to operate in a fluid environment (intestinal tract, fluid-filled coelom) and provide improved hemostasis, with the ability to coagulate blood vessels up to 2 mm in diameter, as compared to the CO_2 laser, which coagulates blood vessels up to 0.6 mm in diameter. Operation results in deeper tissue penetration and is relatively less precise. Diode lasers have been used for photocoagulation of retinal and other ocular tissues, chromophores enhanced tissue ablation and coagulation, laser welding and photodynamic therapy. In addition, the diode laser has the fiberoptic ability to operate with several endoscopes.[38,54,69]

Laser is particularly useful for avian surgery, where hemostasis is of great concern. Surgical indications include salpingohysterectomy, orchidectomy, and limb amputation. It is also beneficial in the excision of granulomas, abscesses, and neoplasms. Endoscopic diode laser techniques provide minimally invasive access to several anatomic sites as well as endoscopic hemostatic control. Superficial lung and air sac granulomas within the cranial thoracic, caudal thoracic and abdominal air sacs have been successfully ablated with the 810-nm diode laser.[21,37,61]

Bob Doneley

Fig 35.8a | Avascular necrosis of the phalanges can occur as a result of circumferential constriction due to fibers from cage bedding or as a condition of undetermined etiology. In the case of this bird, cloth fibers became entwined around the foot, leading to the visible digit swelling and potential for necrosis of the digits. Removal of the constricting fibers, appropriate topical antibiotics and bandage application are necessary to treat this condition.

Greg J. Harrison

Fig 35.8b | Manmade fibers tend to entrap digits. The threads are removed using 40x magnification and a bent-tip hypodermic needle as a cutting tool. When the constricting band is not accompanied by foreign material, treatment is similar to that described in Fig 35.8a. The circumferential bands may require debridement and suturing.

ILLUMINATION AND MAGNIFICATION

Avian surgery often requires delicate handling of small structures. Coelomic surgery is performed within a deep cavity, which obstructs visualization. Therefore, magnification and illumination equipment are essential (see Chapter 1, Clinical Practice). The ideal avian surgical light source provides optimum illumination with minimal heat transfer to the patient that may result in tissue dehydration and surgeon discomfort. *(Ed Note: Although overhead lights that produce significant heat have been shown to be effective in helping to prevent hypothermia during avian anesthesia, forced warm air blankets also are effective and do not have the disadvantage of tissue desiccation and over heating of the surgeon).* The small size of the avian patient necessitates intense light, precisely focused. This often requires changing the angle of the light to provide illumination and avoid shadows. A three-headed, flexible fiberoptic light source provides ideal illumination and the ability to change the focal area of the light. Often, 250-watt bulbs with no less than 20,000 lux are required.[1,4,5]

Ocular head loupes and operating binoculars with a halogen light source also may be used for magnification and illumination. These allow the surgeon to set the light source on the surgical site, and magnification and illumination move with the surgeon. Operating microscopes are useful for avian patients, particularly those weighing less than 1 kg, and are advantageous for handling blood vessels in larger birds as well. An operating microscope with a lens objective approximately 150 power and a 12.5 power binocular objective is most useful.[1,4,5]

A less expensive but also less effective option for increased magnification and illumination includes adjustable magnifiers with attached lights available for sewing and other home uses.

SUTURE MATERIALS AND ADHESIVES

Significant information is available regarding appropriate selection of suture material in veterinary and human medicine. Suture material utilized in birds must be minimally reactive and of an appropriate size. Tissue reaction to five suture materials has been evaluated in pigeons at 3, 7, 15, 30, 60, 90 and 120 days following implantation in the body wall. These include polyglactin 910, polydioxanone, monofilament nylon, medium chromic catgut and stainless steel. Pigeons developed a marked granulocytic inflammatory response to medium chromic catgut that diminished during the evaluation period. The suture was still present at the end of the study, indicating prolonged absorption of the material. Polyglactin 910 caused the most inflammatory reaction and was the most quickly absorbed, being completely gone by day 60. Polydioxanone, like polyglactin 910, is absorbed by hydrolysis. Unlike polyglactin 910, however, it caused minimal tissue reaction and absorption was occurring by day 120. Nylon and stainless steel are non-absorbable materials that caused minimal tissue reaction. However, the stiffness may make them mechanically irritating to surrounding tissues, and these were more often associated with hematoma, seroma and caseogranuloma formation.

This study concluded that chromic catgut should be avoided; slowly absorbed monofilament and synthetic materials absorbed by hydrolysis rather than proteolysis are recommended when prolonged wound healing is expected. Rapidly absorbed, braided, synthetic suture materials absorbed by hydrolysis are recommended when the benefit of rapid absorption outweighs the disadvantage of possible pronounced inflammatory reaction. Monofilament suture material has the advantage of minimizing trauma and cutting of tissue when compared to multifilament material. Taper-point needles are usually indicated in avian surgery, as compared to cutting needles to prevent tearing of tissues being sutured. Cutting needles may be useful for suturing thicker, tougher tissues such as the feet of larger species.[4,5,10]

Cyanoacrylate tissue adhesives hold tissues in apposition to allow healing. The cyanoacrylate monomer is a liquid that polymerizes in a small amount of water present in tissues. However, it is important not to allow the acrylic to run between the apposed tissues, as this physical barrier will delay wound healing. One should be cautious when using these adhesives in the presence of anesthetic gases with which they are reactive. They may also cause ocular irritation and vomiting in avian patients. The fumes of some cyanoacrylate tissue adhesives may cause respiratory irritation as well.[5]

POSTOPERATIVE CARE

Postoperatively, the patient should be placed into a temperature-controlled incubator once it is able to stand without ataxia. Temperature should be set according to each individual's optimal requirements (27-30° C or 81-86° F for most psittacines) and supplemental humidified oxygen is beneficial to those patients at risk for hypoxia/hypercapnea during recovery.[1,5,9,41]

Perches should be avoided until the bird has recovered sufficiently to balance and grip well. Once the patient can balance and grip, perches of low height are recommended. Food and water are not introduced until the patient has fully recovered to prevent regurgitation and aspiration.[1,5,9,41]

The likelihood of postoperative self-trauma varies with the species, the individual patient and the surgical procedure performed. Avian patients generally do not traumatize their surgical incisions. Some clinicians advocate leaving longer-than-average suture ends to allow the bird to groom these as they would a feather.[1,5,9,41]

Occasionally an Elizabethan or other collar is necessary to provide a mechanical barrier to self-induced trauma to the surgical site. In all cases where a collar is first applied, the bird should be monitored following the collar application. Agitation, depression, or inability to access food or water must be noted and corrected. If the bird can still traumatize the surgical site, collar adjustment also would be needed. Applying the collar prior to surgery may enhance acceptance of an Elizabethan or foam collar. This allows the bird to adjust to the collar, thus minimizing postoperative stress. Antianxiety or sedative medications such as diazepam may be given prior to collar application to decrease stress. Initially applying a small collar and increasing the size as necessary also may improve patient acceptance. Some birds will not tolerate restraint collars and customized body suits may be used to prevent self-trauma.[1,5,9,41]

Soft Tissue Surgery

SKIN

Birds have a relatively thin, dry epidermis. In feathered regions, the skin may be only 10 cell layers thick. The dermis is loosely attached to the underlying muscle fascia with very little subcutaneous tissue except in the distal extremities where it is firmly adhered to the underlying bone.[9]

Constriction and Avascular Necrosis of the Digits

Avascular necrosis of the phalanges of the pelvic limbs may occur secondary to circumferential constriction caused by fibers, scabs or necrotic tissue. These constrictions compromise vascular flow to and from the distal phalanx, leading to edema and necrosis (Figs 35.8a,b). If detected early, the toe may be salvaged and amputation avoided. This condition is particularly common in pediatric patients. Proposed etiologies include low relative humidity, egg fiber-related strictures from hatching, septicemia and ergot-like intoxication. Increasing the environmental humidity and applying topical creams to promote hydration of the affected tissue may be effective in resolving early lesions. Eschars should be debrided and the digit cleaned and bandaged with a hydroactive dressing and allowed to heal by secondary intention. If tissue fibers are the inciting cause, a full-thickness linear skin incision should be made over the dorsal aspect of the constricted region. This area is then bandaged with a hydroactive dressing to prevent scab formation. Complete healing may require weeks to months. A circumferential excision of the constricted region followed by anastomosis of the skin may be performed. The constricted tissue is surgically removed with a scalpel blade. One to two subcutaneous sutures are placed to prevent excessive tension on the skin repair. The skin edges are apposed with several simple interrupted sutures placed superficially to prevent disruption of the vascular supply

by resulting eversion of the skin edges, which will delay wound healing. After the skin edges are apposed, 2- to 3-mm superficial release incisions should be made on the lateral and medial aspects of the digit at the site of the anastomosis. This will allow postoperative swelling to occur without constriction. A hydroactive dressing is applied and the digit bandaged. Magnification and illumination such as a head loupe or operating microscope are useful to visualize the fibers.[1,9]

If the distal phalanx has developed complete avascular necrosis, amputation is necessary. Amputation should be performed just proximal to the proximal end of the necrotic tissue, where a good vascular supply still is present. An incision is made in the skin and subcutaneous tissue circumferentially. One or two sutures are placed in the subcutaneous tissue to relieve tension on the skin incision. Several simple interrupted sutures are placed in the superficial epidermis to prevent eversion of the skin edges, which will delay healing. The digit is bandaged and sutures removed in 10 to 14 days[1,9] (see Amputation of Digit later in this chapter).

It is best to avoid the use of electrocautery or radiocoagulation to prevent damage to the minimal vascular supply of the digits. If a tourniquet is utilized to control hemorrhage, it should be used for only a limited time to avoid vascular compromise.[1,9]

Passerines are prone to developing avascular necrosis of the pelvic limbs or digits due to entanglement with synthetic fibers such as nesting string. Microsurgical tools or a bent 25-gauge needle may be used to remove the constricting fibers. The tip of the needle can be used to elevate the fibers and the edge of the needle used to severe the material. A hydroactive dressing is applied and the area bandaged.[1,9]

Passerines also may develop constrictions secondary to hyperkeratosis or to the development of excessively large scales over the pelvic limbs and digits. Malnutrition and *Knemidokoptes* sp. infection have both been implicated as etiologies. These conditions may predispose the patient to *Staphylococcus* spp. infections. In most cases, skin lesions will resolve with correction of nutritional problems or treatment with ivermectin. In severe cases, it may be necessary to surgically debride these hyperkeratotic lesions or enlarged scales. Microsurgical instruments and magnification are useful for manipulating these small structures. Antibiotic emollient creams will soften and hydrate the skin while treating bacterial infection.[1,9]

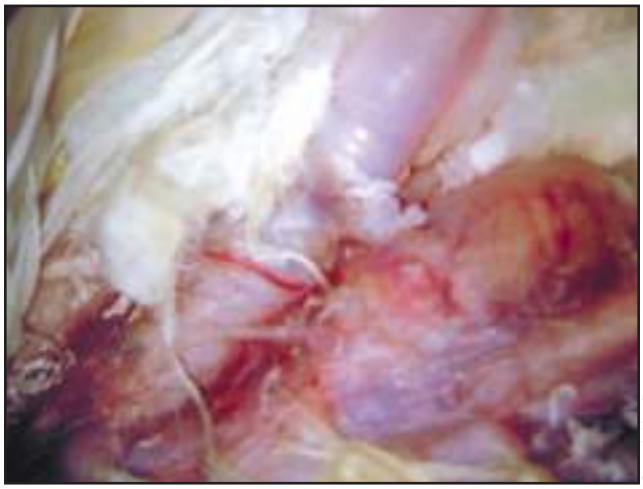
Feather Cysts

Feather cysts are usually formed as a result of injury to or deformation of the follicle.[36b] Direct trauma, mechanical and chemical cautery also are inciting causes.

Damage to one side of the follicle may result in asymmetrical feather growth. The developing feather may then grow in an arch back toward the body, forming a feather cyst (Figs 35.9a-i). Certain species of canaries, particularly the Norwich, Gloucester and their crossbreeds, may develop cysts as a result of abnormally formed feathers (Fig 35.10). These birds have been genetically selected to produce a downy, soft feather type that predisposes them to feather cysts. Malnutrition, viral, bacterial and parasitic infections may result in formation of feather cysts as well.[1,9,36b]

The surgeon should examine the cyst to determine whether it contains a viable or devitalized feather. Perform an initial evaluation of the feather by making a small incision at the distal aspect of the cyst and examining the contents. Every attempt should be made to salvage viable feathers and tissue, particularly those involving tail rectrices and flight feathers.[1,9]

If excision is required, use of a scalpel blade offers the benefit of complete excision of the affected follicle without damage to adjacent follicles. Damage to adjacent feather follicles and/or their blood supply may disrupt feather formation and result in the formation of additional feather cysts. Radiosurgical fulguration has been performed successfully; however, the adjacent feather follicles may be damaged due to difficulty in controlling the extent of tissue destruction. Feather cysts typically have good vascular supply, so hemostasis is required.



Greg J. Harrison

Fig 35.9a | Feather cysts in parrots usually form as a result of injury to the follicle. Direct trauma to the feather follicle can result in abnormal feather growth, thus promoting feather cyst formation. Improper wing trim, malnutrition, viral, bacterial and parasitic infections may result in the formation of poor-quality feathers that are easily damaged. This cyst has formed in the follicle of the dorsal major covert overlying primary remex VIII. While the cyst is dorsal, this follicle inserts on the ventral aspect of the major metacarpal bone. The underlying primary feather structures form the strongest boundary, so the cyst herniates dorsally. Approaching the cyst from this dorsal aspect fails to address the germinal tissue found at the point of insertion.

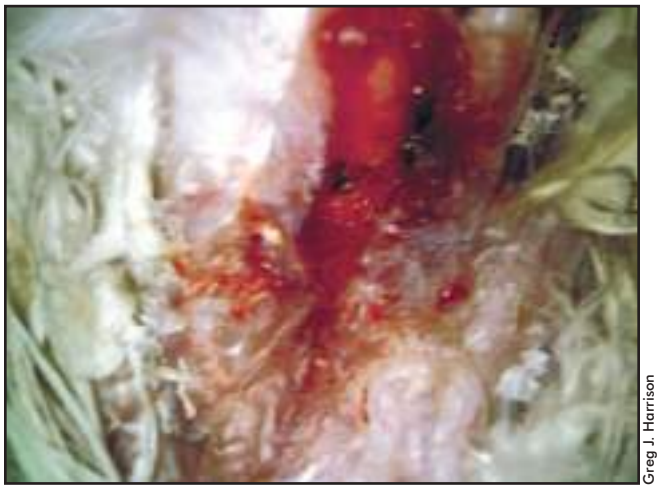


Fig 35.9b | Incising alongside the cyst using radiosurgery bipolar forceps.

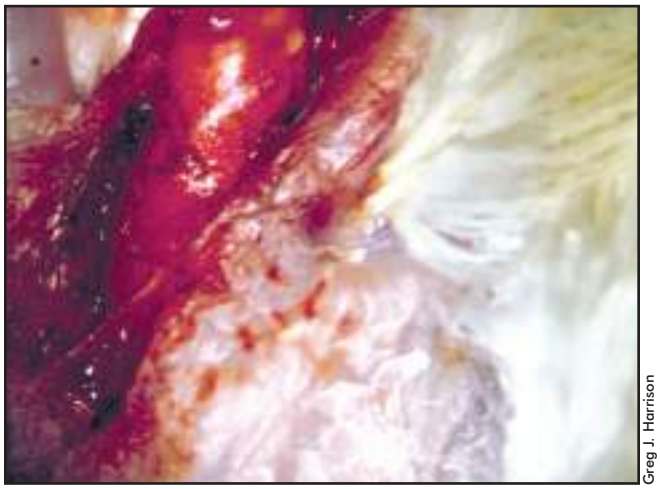


Fig 35.9c | Freeing the cyst dorsally from surrounding feather structures.

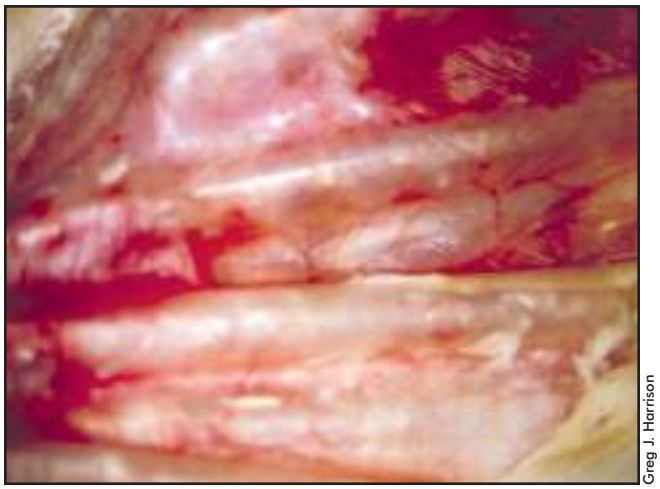


Fig 35.9d | Next the ventral surface of the wing is incised between primary VII and VIII, allowing access to the insertion of the cystic follicle on the ventral aspect of the metacarpal bone of the manus.

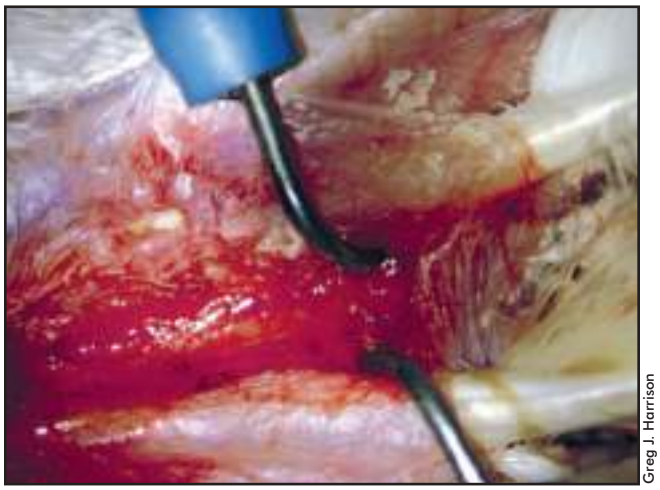


Fig 35.9e | Retractor bands used to improve the view and hold the unaffected follicular tissues out of the way of the radio wave's energy field to avoid damage. In Harrison's opinion, feather damage and failure to resect the point of insertion are the major causes of cyst surgery failure and cyst recurrence.

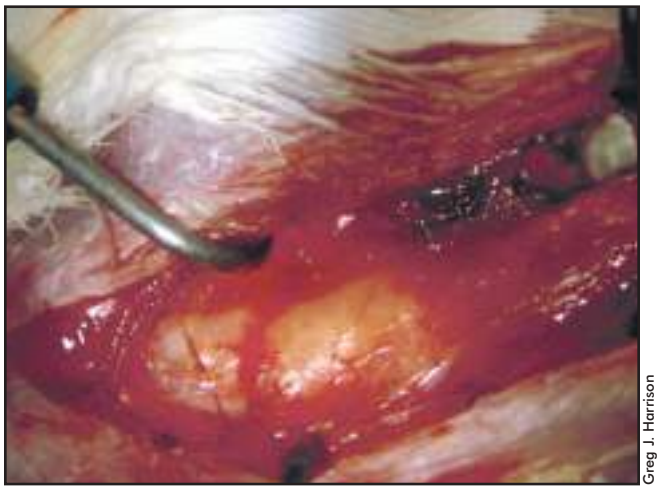


Fig 35.9f | Dissection of the follicular tissue. Retractor bands are repeatedly relocated to minimize trauma during cyst removal.

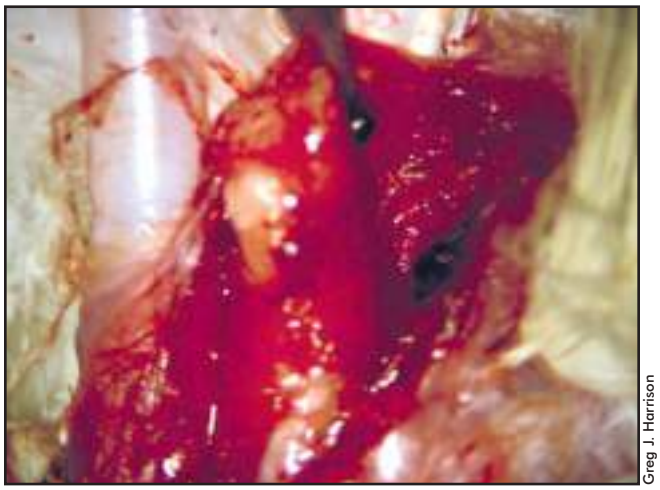
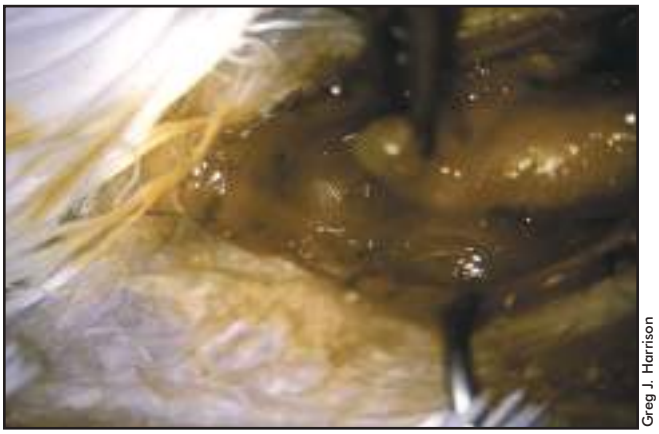
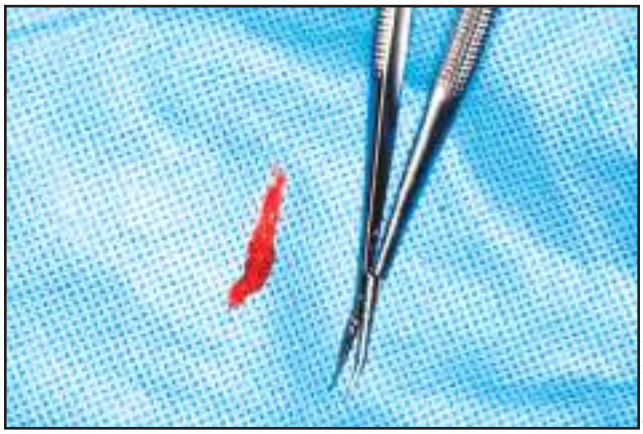


Fig 35.9g | The freed follicle is traced to its insertion and the nutrient vessel is coagulated.



Greg J. Harrison

Fig 35.9h | The full cystic follicle is removed intact.



Greg J. Harrison

Fig 35.9i | Cyst removed intact and microsurgical needle holder.

Espen Odberg

Fig 35.10 | Multiple feather cysts can occur on the body, either over a regional area or in a particular feather tract. Multiple cysts may be removed surgically using an elliptical or fusiform excision followed by primary closure with a simple continuous pattern using a monofilament suture.

A tourniquet, direct manual pressure applied to the surgical site or polysaccharide beads[d] may aid in hemostasis. The site may be sutured or left to heal by second intention and bandaged with a hydroactive dressing. As adjacent feathers begin to regrow, debris should be removed carefully and the bandage changed frequently to prevent interference with developing feathers. Laser excision may improve hemostasis. Occasionally the cyst contains necrotic material but the follicular epithelium is not damaged, making new feather growth possible. In these cases, the follicle is incised and necrotic material removed. The follicle is then irrigated with sterile saline and the edges of the incision apposed. The site may be bandaged and local and/or systemic antibiotic therapy initiated. New feather growth must be carefully monitored for formation of cysts and disfigurement of new feathers. Feathers may fail to regrow if the epithelium has been severely damaged.[1,9]

Occasionally a cystic follicle may be salvaged by marsupialization, particularly if there is a single cyst or a large follicle. An incision is made over the center of the cyst with a scalpel blade parallel to the direction of feather growth. Hemorrhage is controlled with a tourniquet, manual pressure or by ligation. Radiocautery or chemical hemostatic agents may damage the epithelium and further damage the follicle. The contents of the cyst are removed and the lining sampled for cytology and bacterial culture. Redundant tissue is excised and the follicle irrigated with sterile saline solution. The margin of the cyst is then sutured to the adjacent skin using a simple continuous pattern with a fine monofilament suture. New feather growth must be carefully monitored for formation of further cysts or disfigured feathers. Feathers may fail to form if the epithelium is severely damaged.[1,9]

Occasionally multiple feather cysts are present on the body, either over a regional area or in a particular tract. Multiple cysts may be removed using an elliptical or fusiform excision followed by primary closure with a simple continuous pattern using a monofilament suture material. Radical excision of an entire pteryla of affected feathers has been described. This is typically necessary in canaries. A fusiform incision is made around the affected pteryla. The primary vascular supply to the tract, and any large blood vessels are ligated or coagulated. The affected follicles are removed along with the surrounding skin. The remaining skin edges are apposed using a simple continuous pattern with a monofilament suture.[1,9]

Feather cysts of the tail may be severe and disfiguring. Birds may traumatize these and develop secondary infections. Cysts can be excised as described previously. Amputation of the pygostyle is an option in those patients suffering from multiple cysts or those that have severe trauma to the surrounding soft tissues and underlying bone. This is performed by blunt dissection to the coccygeal vertebrae and disarticulation at the sacrococcygeal junction. Soft tissues are closed routinely and care should be taken not to enter the cloaca. Intraoperative hemorrhage and postoperative pain may be severe, and hemostasis and analgesia are crucial to a successful outcome.[1,9]

Skin and Follicular Biopsy

Skin biopsy is indicated as part of a diagnostic process for dermatoses including feather dysplasia, feather-destructive behavior, auto-mutilatory conditions, ulcerative dermatitis, hyperkeratinization and feather loss (see Chapter 13, Integument). Selection of biopsy sites is crucial to accurate diagnostic results. A sample with an actively growing feather, an entire follicle and surrounding skin should be obtained for conditions affecting the feathers. The surgical site should be gently prepared, avoiding vigorous scrubbing that may irritate the skin and alter the histopathology results. The follicle is grasped with atraumatic forceps and the feather, follicle and skin excised with scissors or a blade. Disposable skin punch biopsy instruments also work well. Minimal pressure should be exerted with these biopsy punches due to the thin avian skin and absence of significant subcutaneous tissue. It is best to avoid cautery prior to excising the sample to prevent cellular and tissue changes to the edges, which may complicate histopathologic analysis. Radiosurgery may be used to control hemorrhage after the sample is obtained. Closure is routine.[57]

Cranial Skin Defect Repair with an Advancement Pedicle Flap

Soft and hard tissue injury to the cranium may occur due to trauma from various sources including predator attack or attack from other birds, ceiling fan injuries and cage trauma. Species that commonly "flush" such as quail may be susceptible to cranial trauma if the caging does not have significant height to allow for flight. Soft tissue injuries that expose the cranium may result in chronic, non-healing wounds and devitalized cranial bone, which impedes formation of granulation tissue and epithelial migration. The skin covering the cranium is firmly adhered to the cranium. Primary and secondary closure of larger wounds may be difficult once skin edges have begun to fibrose. In addition, closure may create tension when apposing the skin edges, creating deformation of the eyelids and even exposure keratitis. Some wounds may be allowed to heal by second intention under a hydrophilic bandage material.[27,73]

A pedicle advancement flap is preferable to simple closure to surgically resolve larger defects to the soft tissue covering the cranium. Advancement flaps remain continuous with the donor site thereby maintaining blood supply to the subdermal plexus within the flap. Pedicle advancement flaps have a greater chance of remaining viable, as compared to free grafts, particularly when the recipient site is poorly vascularized. In addition, full thickness skin and subcutaneous grafts will provide normal feather coverage if they contain pterylae.[33,55,62-65,73]

Initially, the wound is assessed to determine the degree of necrosis of the skin and cranial bone, and to examine the size of the defect to be repaired. The cranial skin is undermined and the skin edges are debrided, removing just enough skin to obtain a good vascular supply. If necessary, small rongeurs may be used to carefully remove devitalized bone; obviously, the cranium should not be completely penetrated. Initiating two skin incisions at the lateral aspects of the caudal end of the defect creates the pedicle flap. These incisions are continued caudolaterally over the cervical region. Divergence of the lateral incisions creates a wider base that increases the blood supply to the subdermal plexus, and compensates for the tendency for inadvertent convergence of the incisions when applying lateral skin tension where the skin is more mobile.[33,55,62-64] These incisions should be made with either a scalpel blade or sharp surgical scissors to maintain integrity of the ends of the blood vessels that will be advanced cranially. The pedicle is undermined bluntly to prepare the flap for advancement. The skin flap is advanced rostrally and the skin edges apposed with small monofilament suture in a simple continuous or interrupted pattern using a taper-point needle.[27,73]

If the defect is too large to correct with a single surgery, the initial procedure should close as much of the defect as possible and the remaining defect can be covered with a hydroactive dressing. A second surgical flap may be created or a staged closure of the defect using horizontal mattress sutures may be performed to completely repair the defect.

Repair of Ulcerative Lesions of the Sternum (Carina of the Keel)

Ulcerative lesions to the carina of the keel are most often traumatically induced (see Chapter 1, Clinical Practice). Thermal burns, foreign bodies, poxvirus, mycobacteriosis or other stressful situations such as heat and overpopulation have been implicated as well. Improper wing trimming, particularly in heavier bodied birds (African grey parrots and Amazon parrots) may cause the bird to impact the floor when it attempts to fly. This trauma may create a bruise or a laceration of the skin over the carina. Scars from previous traumatic episodes are often evident upon routine physical examination. Auto-mutilation of the tissue overlying the carina may occur as well. Secondary bacterial infection, often involving anaerobes, is common in this location.[39]

Traumatic lesions of the carina often heal readily if the patient is prevented from falling and re-injuring the site. Conversely, repeated trauma and/or auto-mutilation may cause more severe and chronic lesions. These lesions may require extensive surgical debridement. Extensive lesions may prevent primary closure and may heal by second intention.

Surgical correction is accomplished with the patient in dorsal recumbency. The feathers are removed in a 2-cm diameter around the circumference of the wound and the area surgically prepped. The skin and subcutaneous tissues are debrided until healthy tissue is encountered. It may be necessary to debride devitalized pectoral muscle and affected portions of the carina of the keel. Radiosurgery may be used for hemostasis. Tissue and bone samples should be submitted for bacterial culture and histopathologic examination. The elevated origins of the pectoral muscles are sutured together over the keel or anchored to the cartilaginous portion of the keel in an interrupted horizontal mattress pattern with absorbable monofilament suture. The skin is closed in a simple interrupted or continuous pattern with monofilament suture. There is often considerable tension on these incision sites, therefore, it may be necessary to place tension-relieving sutures lateral to the incision. One method described involves placing interrupted horizontal mattress sutures through the skin and pectoral muscle tied over gauze sponges just lateral to the medial incision. The wings may be bandaged to the body to prevent extension and movement that would place additional tension on the suture site, and a restraint collar or body suit is usually necessary if auto-mutilation has occurred. Defects that are too large to close surgically may heal by second intention. Gentle irrigation and frequent bandage changes with a sterile hydrophilic dressing will assist in healing.[39]

Xanthoma

Xanthomatosis results from the accumulation of lipid-laden macrophages, giant cells, free cholesterol and variable degrees of fibrosis. Xanthomas often occur at the distal wing, but have been found in other locations as well. These masses may be locally invasive and wide margins may be necessary to completely excise and prevent recurrence. Some birds may mutilate these lesions, causing ulceration and secondary infection. Elevated serum cholesterol, trauma and genetic predisposition in some species have been implicated in the formation of xanthomas. Dietary correction may be curative in some species and in some individuals. However, very large, painful, hemorrhagic or infected xanthomas often require surgical resection.

Masses may be removed with bipolar or monopolar radiosurgery, taking care to avoid damage to remaining feather follicles and their blood supply. The site may be closed if there is enough remaining tissue or allowed to heal by second intention and bandaged with a hydroactive dressing (Figs 35.11a-g). If extensive subcutaneous tissues and bone are involved, amputation of the affected area may be necessary.[9]

Uropygial Gland

The uropygial gland is located dorsal to the tail. It is absent in Amazon parrots (*Amazona spp.*) and the hyacinth macaw (*Anodorhynchus hyacinthinus*) and may be reduced in size in some cockatiels. Disease of this gland and/or its papillae is not uncommon and surgical correction may be necessary. Absence of papilla feathers may indicate a problem with glandular function. Left untreated, a gland may rupture, causing inflammation and significant scar tissue formation. Simple impaction of the gland may respond to medical therapy and gentle expression of the contents. If the impaction cannot be alleviated by conservative therapy, small incisions may be made over the affected lobe(s) of the gland, the contents expressed, and the gland irrigated with saline. Antibiotics and analgesics may be indicated during recovery.[9] Neoplastic conditions of the uropygial gland with secondary infection occur with some frequency (see Chapter 13, Integument).

Chronic impaction and/or infection unresponsive to medical therapy and neoplasia of the uropygial gland may require surgical removal of the affected gland. The patient is placed in lateral or semi-ventral recumbency. Intermittent positive pressure ventilation and close monitoring of respiration is necessary when positioned ventrally to ensure movement of the sternum is not reduced and respiration not impaired. The head may be elevated and a pad may be placed under the tail, with the tail rectrices taped in place to elevate the sacrum and improve exposure and visualization of the uropygial gland. The surgical site is aseptically prepared. The gland is bilobed and each lobe receives its vascular supply from a vessel that branches at the cranial, middle and caudal portions of the gland. These vessels and other surrounding vessels require ligation or bipolar radiocoagulation. The gland may extend deep to the synsacrum and caudally to the insertion of the tail feathers.[9]

A fusiform incision is made via unipolar or bipolar radiosurgery around the circumference of the gland. This is initiated caudal to the papilla and continued craniolaterally along both sides of the gland. Dissection of the gland is initiated at the caudal aspect of the gland and extended circumferentially and cranially until the gland is removed. Mosquito hemostats or thumb forceps may be used to apply gentle traction on the gland, facilitating removal. The strongest attachments are associated with the muscle fibers at the cranial border of the gland. Hemorrhage must be strictly controlled by radiocoagulation, manual pressure and/or hemostatic products.[9] The deeper fascia is closed with absorbable monofilament suture in a simple continuous or interrupted pattern, depending on the amount of tension present. Subcutaneous and skin closure is routine. Extensive tissue trauma, neoplasia or

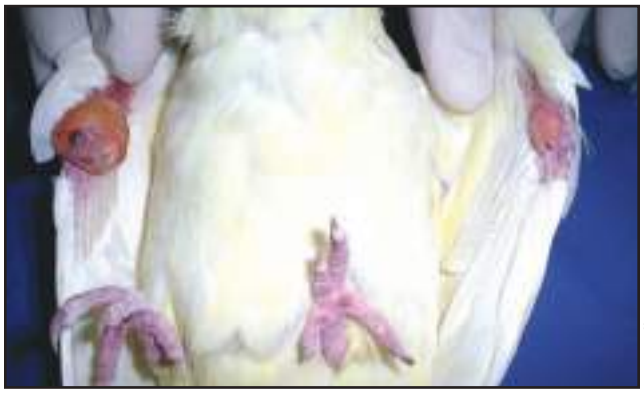

Espen Odberg

Fig 35.11a | Xanthomas often occur at the distal wing, but have been found in other locations. Note the balding plantar foot patterns and the discoloration of the feathers. These are indicative of malnutrition and related disorders.

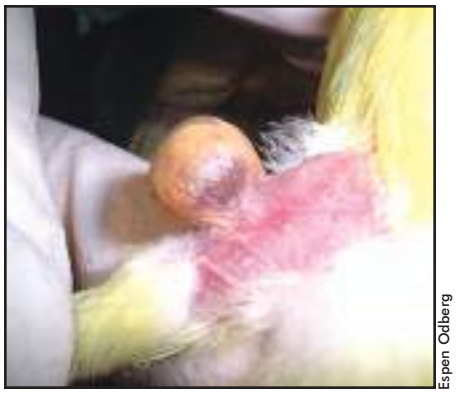

Espen Odberg

Fig 35.11b | Xanthomas that are well demarcated and/or pedunculated may need to be excised.

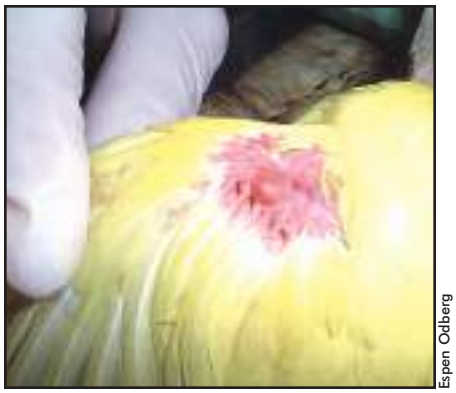

Espen Odberg

Fig 35.11c | Xonthomas that are closely associated with feather follicles may be excised, being cautious not to cause follicle damage.


Espen Odberg

Fig 35.11d | Removal of such well-defined distal wing xanthomas can be performed by making a small skin incision and gently teasing the contents out with a sterile cotton swab.

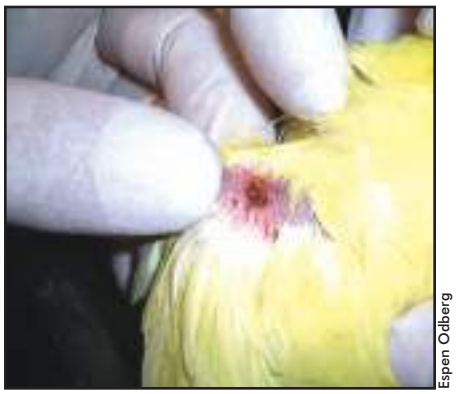

Espen Odberg

Fig 35.11e | Hemorrhage can be controlled with a bipolar or monopolar radiosurgical unit.

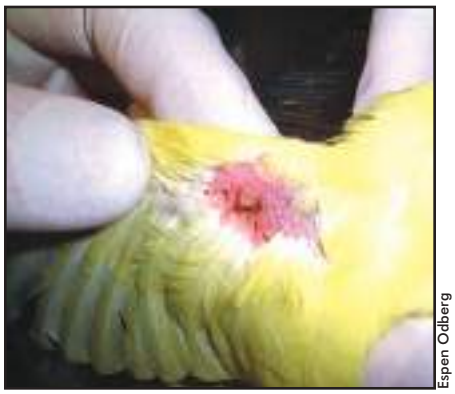

Espen Odberg

Fig 35.11f | After using radiosurgical hemostasis allow a few moments to make sure no oozing occurs.

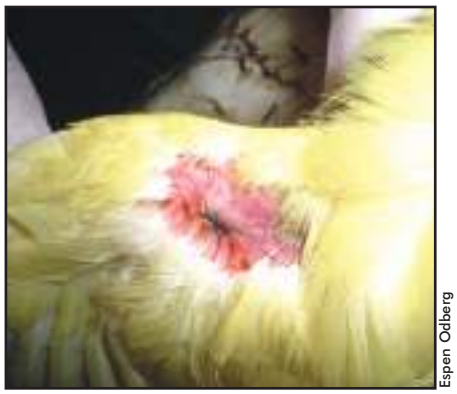

Espen Odberg

Fig 35.11g | The skin is closed in a simple interrupted suture pattern with a monofilament suture.

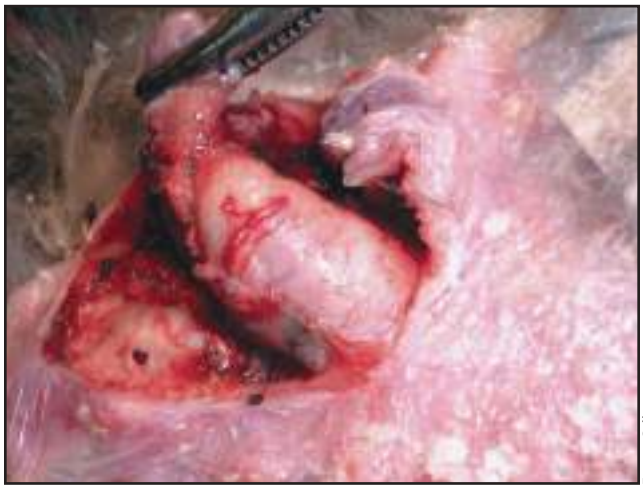
rupture of the uropygial gland may require additional dissection and debridement. An additional caudal incision perpendicular to the dorsal midline incision may be necessary. If the remaining defect is too large to allow full closure, staged closure or healing by second intention may be necessary. Any open defects should be bandaged under a hydroactive dressing to promote granulation and prevent exposure. Antibiotics and analgesics should be administered as appropriate to each patient. Dehiscence, damage to the follicles of the rectrices and infection are potential complications (Figs 35.12a-e).[9]

Pododermatitis

Treatment and surgical intervention in severe presentations of pododermatitis are outlined in Chapter 13, Integument.

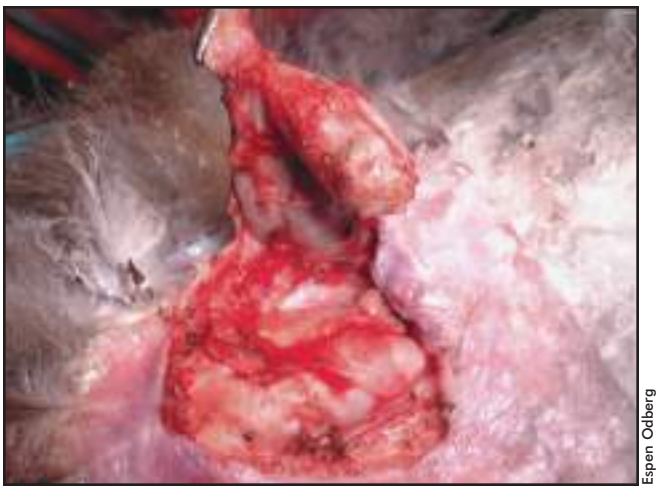
Espen Odberg

Fig 35.12a | The feathers of the uropygial gland and the feathers of the skin dorsally should be removed prior to surgical removal of the gland. Care must be taken when removing these feathers to prevent gland rupture or hemorrhage. Once the feathers of the papilla are removed, material may drain from the gland. This material will need to be cleaned and a gentle routine surgical scrub performed prior to surgery. A fusiform incision is made around the uropygial gland papilla, remaining dorsal to the tail feathers.



Espen Odberg

Fig 35.12b | The tissues underlying the skin are gently dissected and the skin flap is gently reflected dorsally and cranially. Hemorrhage is controlled by coagulation with a bipolar radiosurgical unit. The difference in the appearance between the left (impacted) and the right (non-impacted) side of the gland are apparent in this picture.



Espen Odberg

Fig 35.12c | The largest vessels of the uropygial gland are located on the cranial aspect of the gland along the muscular attachments. By utilizing the duct as a handle and working caudal to cranial, the underlying vessels can be visualized and coagulated with the radiosurgical unit. Removing the gland requires careful dissection and thorough examination for bleeding vessels.

Espen Odberg

Fig 35.12d | The skin is apposed using 5-0 monofilament nonabsorbable suture. Beginning the sutures in the middle of the incision will allow for easier alignment of the skin flap.

Surgery of the Upper Respiratory System

RHINOLITH REMOVAL

Birds may develop rhinoliths secondary to chronic rhinitis and malnutrition. These masses are often formed from desiccated secretions and debris and cause a physical obstruction to respiration. The nares and opercula may become severely eroded and disfigured and damage is often permanent. Clinical signs include sneezing, upper respiratory sounds, inflation of the associated infraorbital sinus, nasal discharge and picking at the affected nares with a toenail. The nares will appear impacted with material, but it is important to recognize the normal anatomy and not mistake the operculum for abnormal material (Figs 35.13a,b). A strong light source, magnification and gentle probing may be required to identify a small rhinolith (Figs 35.14a,b). Surgical head loupes with halogen light sources are particularly useful.[1,9,71]

Nasal tissues are friable and vulnerable to traumatic

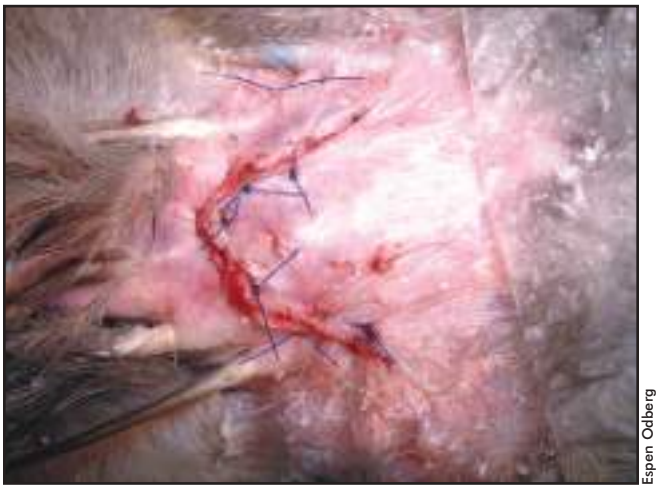


Fig 35.12e | The appearance of the incision following resection of the uropygial gland and completion of the skin closure (see Fig 35.12d).

probing, which will also predispose the mucosa to infection. Prior to attempting removal, warm saline drops should be applied to the affected naris and associated rhinolith. This will ease removal and decrease trauma to the associated tissues. A stainless steel aural curette may be used to gently elevate and remove the mass from the naris. A lacrimal cannula may be used in smaller avian patients such as budgerigars and passerines. Samples should be obtained for cytology, and bacterial and fungal culture. The nares should be flushed with a disinfectant (eg, dilute chlorhexidine or F10 solution) after removal of the bulk of the mass to remove any small pieces or material and to assist in resolution of any pathogens.[79] Appropriate antibiotic or antifungal therapy should be initiated and subsequent flushing performed. Malnutrition should be treated through diet correction. Proper air filtration, humidification and frequent bathing are necessary to prevent recurrence. The rest of the respiratory system should be thoroughly evaluated to identify other concurrent disease.[1,9,71]

INFRAORBITAL EXPLORATION AND TREPHINATION

Infraorbital sinusitis is a common disease in pet birds. This may lead to rhinitis, conjunctivitis, lacrimal infections, and if left untreated, may result in abscess and necrosis of the infraorbital sinus and osteomyelitis of the surrounding bone. Clinical signs include sneezing, nasal discharge, picking at the nares and choana with the toes, inflation of the infraorbital sinus, periorbital swelling and conjunctivitis. Hypovitaminosis A, low environmental humidity and environmental inhalant irritants may predispose birds to secondary bacterial and fungal infections. A sinus flush with sterile, non-bacteriostatic saline may be performed to obtain samples for cytology, and bacterial and fungal cultures.[1,9,71] If flushing of the sinus via the nares and other medical therapy (eg, nebulization) is not effective in establishing drainage, a surgical approach may be used.

To access the infraorbital sinus, the patient is placed in dorsal recumbency and ophthalmic lubricating ointment applied to both eyes. An incision is initiated at the rostral aspect of the infraorbital diverticulum of the infraorbital sinus midway between the eye and the external nares. It is continued caudally, staying parallel with the lateral aspect of the head. Caution must be taken not to penetrate the ocular orbit located caudally. This area is extremely vascular and hemorrhage may be controlled by the use of a laser, radiosurgery, direct pressure or commercial hemostatic products. The sinus must be thoroughly explored, as mucoid, purulent or caseous material may be located within the nasal cavity, within the beak, and between the sinus and the nasal cavity caudal to the turbinates. The sinus cavity should be well irrigated with sterile saline prior to closure and it may be necessary to remove affected periorbital bone. Closure is achieved in a simple continuous pattern with monofilament suture.[1,9,71]

Supraorbital trephination may be necessary to gain access to the dorsal and caudal areas of the infraorbital sinus, which cannot be accessed with nasal flushing and sinus aspiration. This will allow direct irrigation of these affected areas. The skin is incised to expose the frontal bone. Holes are made in the bone with a sterile rotary tool just above the eye. These holes are angled toward the midline. Cortical bone is removed until cancellous bone above the sinus is visible. Drilling is then advanced and widened to an appropriate diameter. Samples are obtained for cytology and culture, and the sinus irrigated with an appropriate solution such as saline, chlorhexidine, F10, water-soluble antibiotics and/or antifungals. The solution should pass through the choana and into the oral cavity to confirm that the trephination is accurately located. With this irrigation, fluid will enter the oral cavity; therefore, the patient should be intubated and the head positioned to allow the fluid to exit the mouth. To prevent aspiration, the oral cavity may be packed with an absorbent material to collect any excess fluid. The trephination site should be irrigated often and the site may need to be reopened, as healing occurs quickly. This procedure may be performed bilaterally, particularly in species such as passerines, in which the right and left infraorbital sinuses do not communicate. Once therapy is no longer required, the sites will heal quickly with minimal scarring.[1,9,71]

CHOANAL ATRESIA

Choanal atresia has been reported in African grey parrots

Fig 35.13a | Normal naris in a lovebird.

Fig 35.13b | Avian patients with malnutrition and subsequent squamous metaplasia and chronic respiratory infections can develop rhinoliths. Erosions of the operculum and nares may result in permanent disfigurement of the nostrils, as shown in this lovebird.

Fig 35.14a | An African grey demonstrates a mild or early stage of accumulation of debris on the operculum. Left untreated, this condition would likely progress to a rhinolith.

Fig 35.14b | A normal naris in an African grey on a formulated diet. Normal powder down is naturally coating the operculum, illustrating the need for showering.

(*Psittacus erithacus erithacus*) and one white cockatoo (*Cacatua alba*). The choana may either be entirely absent or there may be a membrane present that prevents communication between the nasal cavity and the pharynx. Rhinography and an endoscopic examination reveal this lack of communication.[14,36a,71]

A choanal communication may be created with the nasal cavity by hand-drilling a ⅛- or 7⁄64-inch Steinmann pin into each naris, through the nasal choanae, to enter the choana. An 8 French red rubber catheter is then passed from one naris, through the choana, and exited through the other naris. Previously cut slits in the tubing allow mucus to drain. This creates a loop of rubber tubing across the cere with each end passing through the nares to the choana. The ends are tied and secured behind the head. The tube is left in place for 4 to 6 weeks to allow formation of a permanent communication. Nasal flushes with saline are performed twice daily for 7 to 10 days to prevent mucus from occluding the holes (Figs 35.15a-n).[14,36a,71]

RUPTURE OF THE CERVICOCEPHALIC AIR SAC

Hyperinflation of the cervicocephalic air sac has been attributed to chronic infection and/or inflammation, while rupture may occur with trauma, with the former condition being more common. Location of the site of occlusion of normal air flow or rupture of the air sac in traumatic cases may not be identifiable. Smaller avian species typically suffer from generalized overinflation or rupture, while hyperinflation or subcutaneous emphysema is generally confined to the dorsum of the neck. A cutaneous Teflon stent may be surgically implanted at the highest point of the head to allow air to escape. The stent must be carefully monitored for occlusion with debris.[1,9]

A skin incision is made just large enough to insert a 5-mm Teflon stent. Sutures are pre-placed in the four pairs of holes in the flange of the stent. The suture enters the one hole from the external side, doubles back, and passes through the other hole from the lateral side. Once all four sutures are placed, the stent is implanted. A 22-gauge needle is inserted through the skin at the proper location for one end of the suture material to be inserted through the needle to be exteriorized through the skin. This procedure is repeated for the remaining three sutures and the sutures tied in place. The stent may become occluded and may require cleaning with a swab or needle. Occasionally the cervicocephalic air sac may be so excessively hyperinflated that it may interfere with prehension of food or even traumatize the cornea. Excess redundant skin may require resection after releasing the excess air and deflating the air sac.[1,9]


Don Harris*

Fig 35.15a | An African grey with epiphora from choanal atresia.

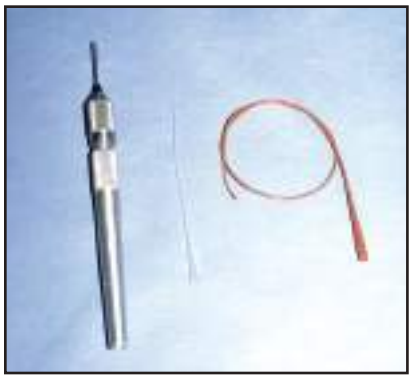

Don Harris*

Fig 35.15b | (From left to right) 0.065 K-wire, with or without the chuck, 3.5 French closed-end tomcat catheter, No. 5 French rubber feeding tube.

Don Harris*

Don Harris*

Fig 35.15c1,2 | To access the nasal passageway, the K-wire pin must be introduced in a direction perpendicular to the long axis of the head. The initial approach is the most important step, as it determines the site of choanal perforation.


Don Harris*

Fig 35.15d | The tip of the pin (with or without the chuck) is introduced through the nostril beneath the turbinate. While maintaining contact with the ventral surface of the nasal cavity, the pin is angled medially until the ventral midline of the nasal cavity is encountered. The exact ventral midline must be located blindly - based on "feel." In birds that do not have an osseous blockage, the membrane can be determined by some "give" in the distal end of the pin as the midline is slowly approached. Or, instead of a soft membrane, you may encounter a "slot" in the bony tissue into which the pin tends to slip. At the midline of the ventral aspect of the nasal cavity, the pin is directed with minimal pressure and rotation to puncture through into the oral cavity.*

*Figs 35.15a-n used with the permission from Zoological Education Network.[36a]


Don Harris*

Fig 35.15e | When the perforation has been made, the K-wire is removed, and a tomcat catheter is inserted into the naris and passed in the same direction into the oral cavity.


Don Harris*

Fig 35.15f | The tomcat catheter is introduced into the oral cavity with a hemostat which is used to pull it through the newly created opening.

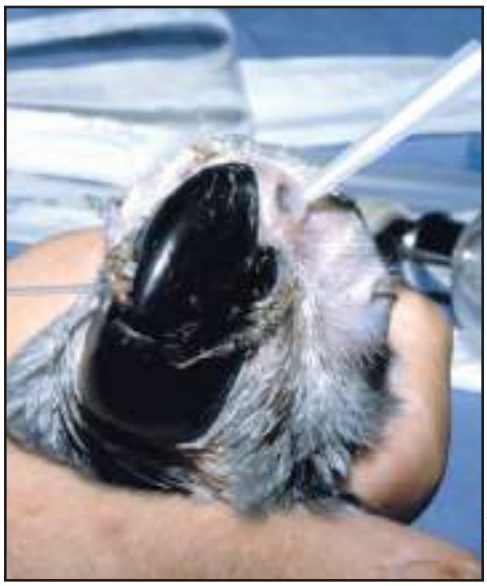

Don Harris*

Fig 35.15g | When the catheter has been pulled most of the way through, the proximal end must be trimmed to fit through the nasal passage.


Don Harris*

Fig 35.15h | The distal end of the feeding tube is introduced into the proximal end of the catheter.


Don Harris*

Fig 35.15i | A mark is made at the point where the two join together snugly. The feeding tube is removed and the catheter is trimmed at that point.

Don Harris*

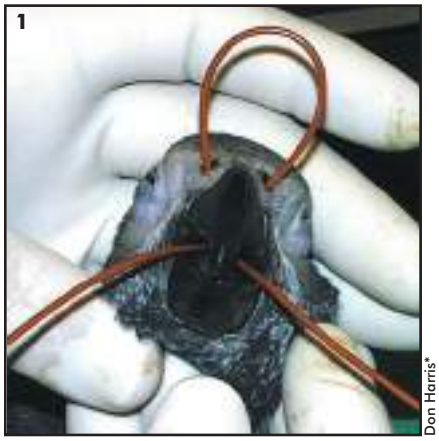
Don Harris*

Fig 35.15j1,2 | After the connection has been made, the catheter is used to pull the feeding tube through the nasal perforation and into the oral cavity.

*Figs 35.15a-n used with the permission from Zoological Education Network.[36a]

Fig 35.15k | After one end of the feeding tube is introduced into one nostril, the procedure is repeated by introducing the other end of the feeding tube into the other nostril. The free ends of the tube are pulled through so that the middle of the feeding tube is retracted onto the dorsal surface of the cere.

Fig 35.15l | At the point where the tube exits one nostril and enters the other, the tube itself must be trimmed to allow the passage of mucus from the nasal cavity externally. If this is not done, the sinuses fill with nasal mucus and the tube would need to be removed, drained and reinserted.



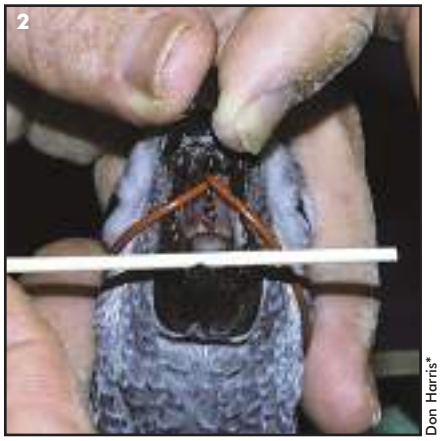


Fig 35.15m1,2 | The feeding tube actually creates a figure 8 configuration where one end enters the left nostril and comes out the right side of the mouth; the other end of the feeding tube enters the right nostril and exits the left side of the mouth. The ends are tied behind the bird's head.

Fig 35.15n | A chin strap can be devised to help hold the tube in place.

*Figs 35.15a-n used with the permission from Zoological Education Network.[36a]

Placing a one-way valve connecting the cervicocephalic air sac to the clavicular air sac may also be used to treat rupture of the cervicocephalic air sac. An approach is made through the left lateral thoracic inlet. The tube is inserted into the hyperinflated air sac, directed caudally along the esophagus, through the thoracic inlet, and into the cranial aspect of the clavicular air sac. The tube is sutured to the *longus coli* muscle to prevent migration, but no attempt is made to suture the air sac around the tube. Skin closure is routine. This method does pose a risk associated with leaving a foreign object in the body. The risk/benefit ratio should be considered prior to surgery, as many birds function well with a persistently hyperinflated cervicocephalic air sac.[1,9]

Air sac hyperinflation may be treated by an alternative procedure that may be used alone or in combination with those previously described. This procedure is particularly useful if the hyperinflated air sac poses a mechanical obstruction to respiration, food intake and physical movement. The air is removed by making a small incision in the overlying skin and air sac, thereby deflating and collapsing the air sac. Redundant skin and air sac are excised, and the skin and air sac sutured to the underlying tissue in several places to prevent extensive re-inflation. The surgical site is closed with a simple continuous pattern using monofilament suture. Traumatically induced subcutaneous emphysema may be alleviated by surgically incising the site to remove the excess air from under the skin.

Although postsurgical subcutaneous emphysema is possible, it is uncommon even in birds that have undergone extensive surgery to the air sacs and associated bone.[1,9]

Thoracic Surgery

TRACHEAL OBSTRUCTION

Foreign material such as seeds, granulomas, inflammation, scarring post-trauma, or concretions of epithelial cells and mucous may occlude the trachea or syrinx, resulting in respiratory distress. Clinical signs include respiratory distress, dyspnea and vocal change. A history of recent anesthesia and intubation should raise concern regarding iatrogenic tracheal trauma, particularly if the endotracheal tube cuff was inflated. Foreign material may be visible in the trachea by wetting the overlying feathers with alcohol and transilluminating the trachea. Often foreign material is located at the syrinx or main bronchi, and therefore may not be visible during an examination. The trachea also may be assessed and obstruction diagnosed with both radiographs and tracheoscopy.[9,14,18,71]

Treatment varies with the severity of the disease, size of the patient and anatomy of the trachea. Certain species such as swans and cranes possess elongated, tortuous tracheas with portions being located within the thorax, making access to the distal trachea difficult. Emergency treatment includes oxygen supplementation and possible placement of an air sac cannula to create a patent airway and stabilize the patient prior to further care. Please refer to Chapter 7, Emergency and Critical Care for a complete description regarding placement of air sac cannulas in birds. An appropriately sized needle may be temporarily placed through the trachea just distal to the foreign body or granuloma to prevent distal migration of the obstructing material, particularly during endoscopic retrieval or debridement. In certain avian species, the pessulum, a midline syringeal cartilage, may be present, which may impede access to a syringeal or bronchial foreign body or granuloma.[1,9,14,18,71]

Many foreign bodies may be retrieved and infectious or inflammatory granulomas may be debrided via tracheoscopy. This is an effective and minimally invasive procedure that should be pursued prior to tracheotomy. Establishment of a patent airway is necessary for respiration and anesthesia. An air sac cannula should be placed until the obstruction is removed. If the obstruction is due to a granuloma or inspissated material and mucus, a small tube such as a urinary catheter or an endotracheal tube may be advanced to the point of obstruction and used to attempt aspiration of the foreign material. Samples should be submitted for cytology, bacterial and fungal cultures. Appropriate antibiotics, antifungals, and nebulization should be continued post-operatively until resolution is achieved.[1,9,18,71,84]

If unsuccessful, or if the patient's trachea is too small to allow passage of an endoscope, a tracheotomy may be necessary. The patient is placed in dorsal recumbency and the area from the mandible to 1 to 2 cm distal to the thoracic inlet is surgically prepared. A transverse tracheotomy of approximately 50% of the tracheal circumference is performed on the ventral tracheal surface. The entire tracheal diameter should not be transected in order to maintain its anatomic alignment, reduce tension on the surgical closure and prevent disruption of the vascular supply. Stay sutures are placed around the tracheal rings adjacent to the tracheotomy site to atraumatically manipulate the trachea. Foreign material may be grasped and removed, gently debrided, suctioned, or material cranial to the incision may be pushed cranially to exit through the glottis. Simple interrupted sutures are preplaced to incorporate one to two tracheal rings on each end of the incision using small, absorbable monofilament suture. Knots are tied external to the tracheal lumen to prevent granuloma formation intratracheally. If the trachea completely separates during the procedure, an anastomosis may be performed in the same fashion, closing the entire circumference of the trachea. Soft tissue, subcutaneous and skin closures are routine (Figs 35.16a-f).[1,6,7,9,14,18,71,84]

Due to the predilection of masses and foreign bodies to be located at the level of the syrinx, surgery is often focused on the thoracic inlet. An operating microscope or halogen-illuminated magnification head loupe is necessary for optimal visualization. The patient is positioned in dorsal recumbency and gas anesthesia is delivered via an air sac cannula. A sterile swab or feeding tube should be placed in the esophagus to facilitate identification and avoid iatrogenic trauma. The skin is incised from the right clavicular-sternal junction to the clavicular-coracoid junction just lateral and ventral to the crop. The overlying skin is gently elevated from the crop and the surrounding tissues bluntly dissected from the crop to avoid tearing the crop or transecting surrounding blood vessels. Once the crop is freed from its clavicular attachments it should be reflected to the left. The trachea is identified by its complete cartilaginous rings. The sternotracheal muscles traverse obliquely and are transected near their caudolateral tracheal attachments. A large blood vessel lies between the muscle bellies and should be coagulated prior to transection of the muscles.[1,6,7,9,14,18,71,84]

Once access to the thoracic inlet has been achieved, it is helpful to elevate the cranial end of the restraint board to improve visualization deep into the thoracic inlet. The interclavicular air sac is bluntly dissected. A blunt hook is looped under the syrinx at the tracheal bifurcation and gently pulled cranially for better visualization. Tracheotomy, foreign body retrieval, granuloma debridement and closure are as described previously.[9]

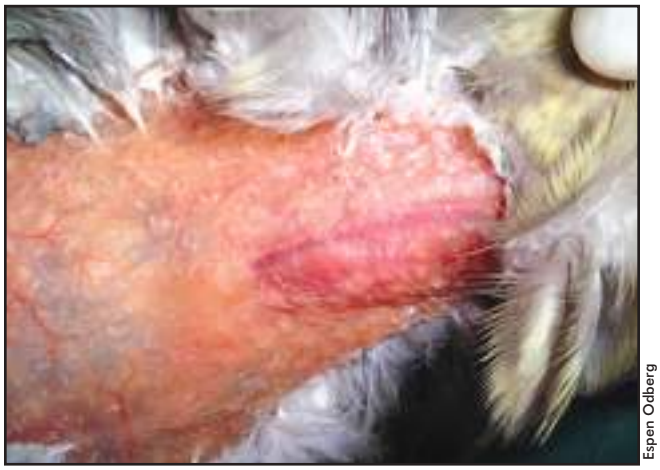


Fig 35.16a | Surgical preparation for tracheal surgery involves placing the patient in dorsal recumbency. The area from the mandible to 1-2 cm distal to the thoracic inlet is surgically prepared by removing the feathers and scrubbing the skin with an appropriate presurgical scrub.

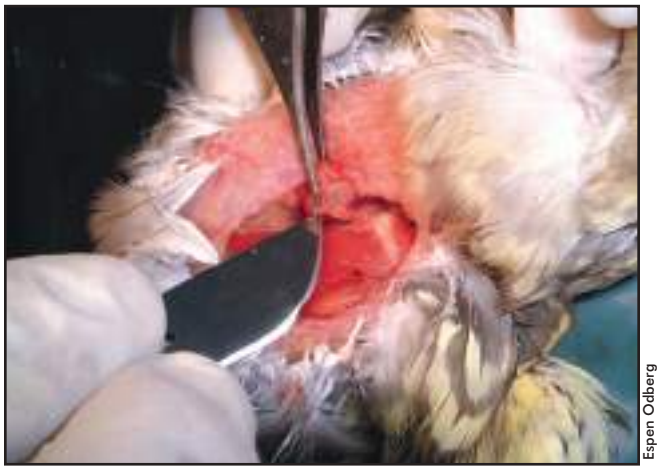


Fig 35.16b | A transverse tracheotomy incision is made on the ventral trachea, approximating 50% of the tracheal circumference.

Fig 35.16c | Care should be taken not to exceed 50% of the tracheal circumference with the tracheotomy incision. This is critical in order to maintain anatomic alignment, reduce tension on the surgical closure and prevent disruption of the vascular supply.

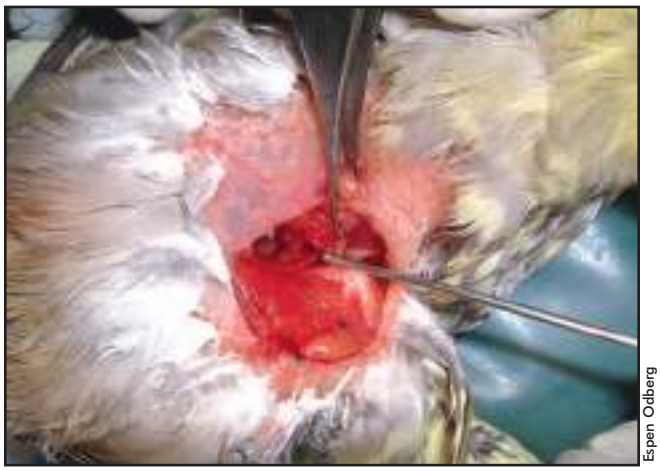


Fig 35.16d | A small-diameter endoscope can be inserted into the trachea to identify foreign material.

Fig 35.16e | A small-diameter suction tip, feeding tube or endoscope can be inserted into the trachea and gentle suction applied to remove any aspirated material.

Fig 35.16f | Simple interrupted sutures are pre-placed to incorporate one or two tracheal rings on each end of the incision using small, absorbable monofilament material. Knots are tied external to the tracheal lumen to prevent granuloma formation intratracheally. If the trachea completely separates during the procedure, an anastomosis may be performed in the same fashion, closing the entire circumference of the trachea.

Certain species such as Amazon parrots, small macaws and smaller birds have shorter primary bronchi, and cranial retraction of the syrinx may result in avulsion of the bronchi from the lung. Therefore, a left lateral approach to the syrinx may be preferable. The patient is positioned in right lateral recumbency. An incision is made over the second and third ribs. These ribs are exposed by blunt dissection and transected at both ends to allow complete removal. This will expose the cranial portion of the lung. The cranial portion of the lung is gently dissected and reflected from its attachments with a moistened cotton-tipped applicator. The jugular vein, pulmonary artery and branches of the subclavian artery are then identified and should be avoided. Dissection between these vessels is performed to access the syrinx. A 2- to 3-mm incision is made in the syrinx using bipolar radiosurgical forceps at the junction with the left primary bronchus. A foreign body may be removed or granuloma debrided via tracheoscopy, suction or gentle manual removal. The syringeal incision heals by second intention. The lung is repositioned into its normal anatomic position and the ribs are not replaced. Soft tissue, subcutaneous and skin closure are routine.[1,9,71]

PNEUMONECTOMY

Surgical removal of lung tissue is indicated for biopsy and for removal of abscesses, granulomas and primary lung neoplasia. Biopsy of lung tissue may be performed by endoscopy. This is an effective and less invasive procedure if the desired site for biopsy is accessible via laparoscopy. There is no discrete pleural space in birds and the visceral and parietal pleura are in close approximation. The dorsal pulmonary parenchyma is contoured tightly to the ribs and intercostal spaces, which facilitates the surgical approach. The avian lung is more vascular and the intrinsic clotting mechanism appears to be less efficient, as compared to mammals, therefore, hemorrhage is a concern.[1,9]

The patient is placed in right or left lateral recumbency, depending on the site desired for biopsy, and the surgical area prepared routinely. A lateral celiotomy is performed. The lungs may be approached through the caudal thoracic air sac or through the intercostal space by removing one or two ribs. The affected lung tissue is carefully elevated using a sterile, moistened cotton-tipped applicator and isolated using vascular or hemostatic clips. The tissue is excised between the clips, leaving them with the viable portion of the lung for hemostasis. No studies exist to determine the amount of lung tissue that may be safely removed or the physiologic effects of pneumonectomy. However, clinically, patients appear to recover well after partial pneumonectomy. Closure includes apposing the intact ribs with stainless steel suture, cerclage wire or non-absorbable monofilament suture. If a caudolateral thoracotomy has been preformed and a portion of the last rib had to be removed for maximum exposure, resulting in unsuitable tissue to close the musculature, a stitch surrounding the remaining rib, passing caudally to and around the ipsilateral pubic bone can produce the tension needed to bring the tissues into apposition. Skin closure is routine.[1,6,7,9]

AIR SAC GRANULOMA RESECTION

Air sac granulomas are usually identified via radiographs, endoscopy or occasionally by ultrasound. If resection is indicated, a celiotomy is performed based on the relative location of the granuloma (see Celiotomy under Surgery of the Gastrointestinal Tract below).

Surgery of the Gastrointestinal Tract

ORAL CAVITY

Keratinized cysts, abscesses, oral papillomas, neoplastic masses and traumatically induced wounds may be found on the tongue, choana, glottis, submandibular cleft and salivary glands. Chronic vitamin A deficiency may result in the accumulation of keratin within cyst-like structures and the formation of caseous abscesses in the oral cavity, submandibular skin and salivary glands.[73] These may interfere with respiration and swallowing. If respiration and food intake are not compromised, it may be beneficial to perform a fine needle aspirate to obtain samples for cytology, bacterial and fungal cultures. Appropriate antibiotic, antifungal and parenteral vitamin A therapy may reduce the size of the abscess and promote encapsulation, thereby reducing the size and vascularity of the mass to be removed. Medical treatment listed previously, including supplementation with beta-carotene, has occasionally been reported to resolve these abscesses.[1,9,73]

Submandibular abscesses may be resected by incising the skin overlying the masses on the ventral neck. Abscesses within the oral cavity may be less accessible and extremely vascular. It is important to intubate these patients to prevent blood and debris from entering the airway, and pre- and postoperative endoscopic examination is helpful to fully assess the oral cavity and choana. Radiosurgery or laser may be used to incise the abscess and to control hemorrhage. The contents of the abscess are removed and the site irrigated with an appropriate disinfectant. If present, the capsule should also be resected if this can be accomplished without clinically significant hemorrhage. Samples are collected for cytology, histopathologic examination, bacterial and fungal cultures. The remaining defect is left to heal by second intention. Abscesses or cysts located on the palatine area

and choanal slit may be removed in the same manner. This area is extremely vascular and hemostasis is crucial to prevent severe hemorrhage. Some surgeons recommend temporary ligation of the palatine arteries during the procedure.[1,9,73]

Papillomatous masses may be removed from the choanal slit, glottis or pharynx with radiosurgery, laser or cryosurgery. Removal with chemical cauterization must be carefully controlled to prevent severe damage to adjacent tissues. Excision is usually not curative and recurrence is common. Papillomatous masses are often located in other regions of the gastrointestinal tract and cloaca. Hepatic and pancreatic carcinoma are associated with papillomatosis. The reader is referred to other sections of this text for a thorough description of papillomatosis and associated disease conditions (Figs 35.17a-c, 35.18a-c).[1,9,73]

Traumatic injuries to the tongue may result in significant hemorrhage, pain and failure to eat. If topical anticoagulants and chemical cautery fail to control hemorrhage, a mattress suture may be placed with an absorbable monofilament suture. The knot of the ligature is placed on the ventral surface of the tongue.[73]

Neoplasia of the tongue has been reported. These masses may be removed by radiosurgery or laser.[73] Complete excision with adequate margins may be difficult and it may be beneficial to ablate the surrounding tissue. A feeding tube or frequent tube-feeding may be necessary for alimentation.

PHARYNGOSTOMY

A pharyngostomy is most often performed in order to place a feeding tube. This is indicated if the patient is anorectic, or if it is necessary to bypass the oral cavity, the esophagus and/or the crop. The patient is placed in left lateral recumbency and the right side of the neck is surgically prepped from the caudal aspect of the mandible to the midcervical region. A small incision is made through the skin and the underlying esophagus is identified. A moistened cotton-tipped applicator or mosquito hemostat is inserted through the mouth and pharyngeal region and visualized through the skin. A small 1- to 2-mm incision is made over this swab in an avascular area. The tube is grasped with the ends of the mosquito hemostat to facilitate entry into the crop and advanced through the lower esophageal sphincter to the proventriculus, depending on the location of the pathology or disease condition that necessitated placement of the feeding tube. The external end of the tube is then sutured in place with two simple interrupted sutures, incorporating the skin and esophageal crop wall on both sides of the incision. The area is bandaged to protect the site, directing the external portion of the tube dorsally to prevent it being chewed or manipulated by the patient. When no longer needed, the sutures are cut and the tube removed. The esophagus/crop and skin can be left to heal by second intention.[1,9] A step-by-step pharyngostomy is shown in (Fig 35.19a-g); the procedural details are similar to those used in other species.

ESOPHAGEAL PERFORATION

Esophageal perforation may be caused by the use of a firm feeding tube, struggling of the patient during tube-feeding, enthusiastic feeding response while a feeding tube is inserted into the crop, or thermal burns followed by necrosis with or without fistulation. Food may enter the subcutaneous space through the lacerated or necrotic esophagus. Severe edema, infection, sepsis, toxemia and necrosis may result. Rapid emergency and supportive care must be instituted. Surgical repair will vary according to the extent of tissue damage, necrosis and infection (see Chapter 14, Evaluating and Treating the Gastrointestinal System, Figs 14.12a-f). The patient is placed in dorsal or lateral recumbency, depending on the location of the tissue damage, and repositioning may be necessary to gain access to all affected areas. A skin incision is made through the overlying skin with a blade, monopolar or bipolar radiosurgery, or with a laser. The subcutaneous and underlying esophagus is then examined to determine the extent of disease. If affected tissues appear healthy, immediate debridement, irrigation and closure may be possible. However, often these patients are diagnosed days to weeks after the initial perforation occurred and severe necrosis and infection are present. These patients may require multiple debridements and irrigation procedures. The external affected area should be bandaged with hydrophilic dressing to promote tissue granulation. Final surgical closure must be delayed until the necrotic tissue has been delineated and resected. Once this is achieved, the esophagus may be closed in a simple continuous inverting pattern, and the subcutaneous and skin closure is routine. A pharyngostomy tube may be placed extending through the lower esophageal sphincter to bypass the esophagus during feeding until the esophagus is healed.[1,9]

CROP BURN REPAIR

Thermal crop damage with or without fistula formation may occur when overheated juvenile feeding formula is stored within the crop immediately after feeding. These thermal burns may range from minor and inapparent to severe and life threatening. The extent of tissue injury and necrosis may not be evident for several days to weeks. An attempt at immediate surgical repair may fail due to progressive tissue necrosis. With severe or extensive crop burns, the patient is often both septic and toxic.


Espen Odberg

Fig 35.17a | A macaw with severe oral papillomatosis. The choanal slit is occluded with hypertrophic tissue, as is the majority of the oropharynx.

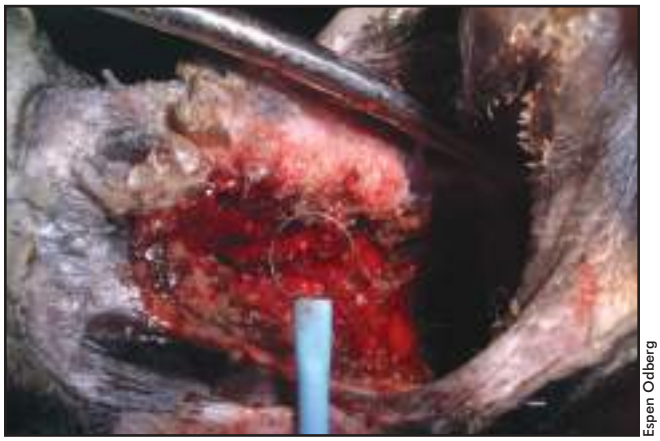

Espen Odberg

Fig 35.17b | A loupe-monopolar radiosurgical tip can be used to debulk oral masses. Care must be taken not to damage adjacent tissues.


Espen Odberg

Fig 35.17c | Silver nitrate can be used to debulk oral papillomas, but care must be taken not to cause chemical damage to adjacent structures within the oral cavity.

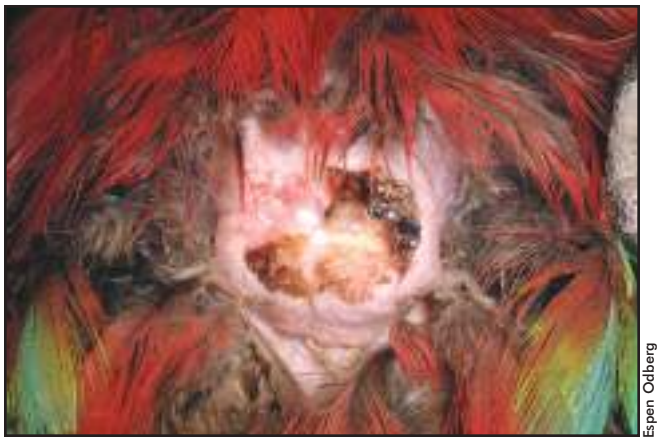

Espen Odberg

Fig 35.18a | Papillomas may be found on the mucosal surface of the cloaca, oropharynx, esophagus/crop, proventriculus, ventriculus, bile ducts and pancreatic ducts. Cystic regression and recurrence is extremely common, and *E. coli* and *Clostridium* spp. are often isolated from the cloaca of affected birds. Surgical resection is recommended, particularly if the mass is causing straining to defecate, secondary cloacal infection, fecoliths, hematochezia, and cloacal prolapse.


Espen Odberg

Fig 35.18b | Cloacal papillomas may be visualized by applying gentle pressure to either side of the vent. Insertion of lubricated cotton-tipped applications may aid in eversion of the cloacal mucosa and allow visualization of the prominent papillomatous tissue. Papillomas are identified by a characteristic "cobblestone" appearance of the mucosa.

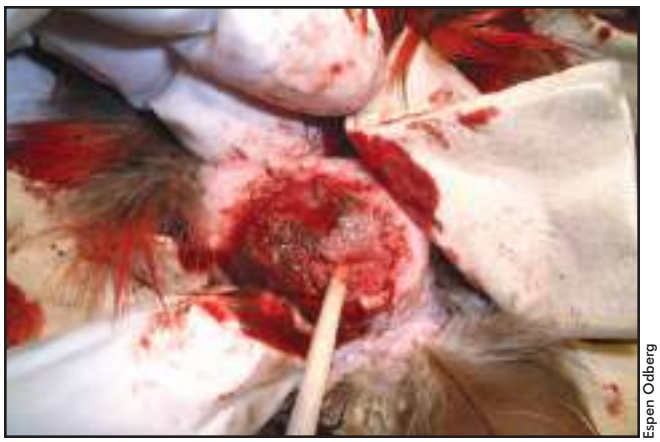

Espen Odberg

Fig 35.18c | Methods for removal of cloacal papillomas include silver nitrate cauterization, cryosurgery, radiocautery, laser surgery, and blade excision. The mass and affected cloacal wall may be everted manually and the mass debulked with any of these methods. If silver nitrate is used, as in this photo, the area must be thoroughly flushed with saline to prevent cauterization of normal mucosa as soon as sufficient tissue has been cauterized to debulk the mass.

Pharyngostomy – step by step Figs 35.19a-g


Scott Echols

Fig 35.19a | The length of the tube to be used is determined by measuring from the crop to the level of the proventriculus.

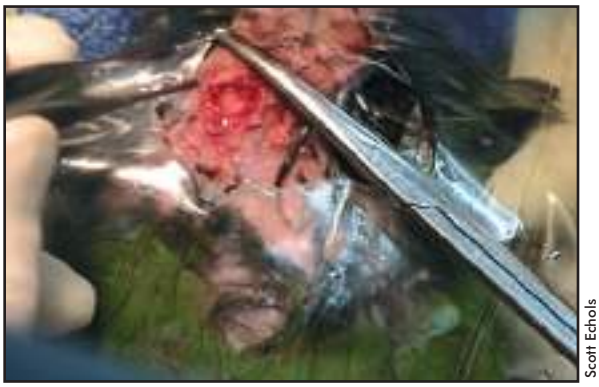

Scott Echols

Fig 35.19b | The skin over the crop is incised.


Scott Echols

Fig 35.19c | The crop tissue is exteriorized and incised just enough to allow tube insertion.

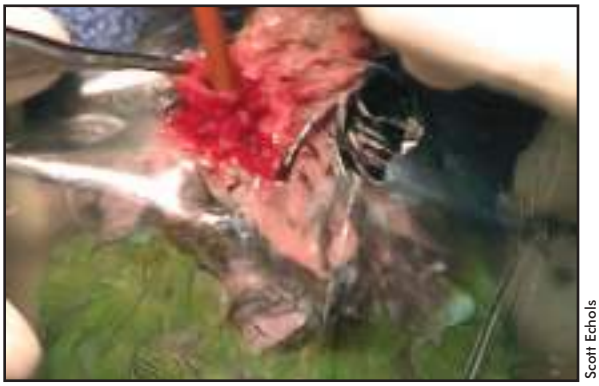

Scott Echols

Fig 35.19d | The tube is placed in the crop and slowly advanced toward the thoracic inlet just ventral to the trachea. The surgeon's index finger guides the tube within the esophagus, using the trachea as a guide, advancing into the thoracic esophagus and proventriculus.

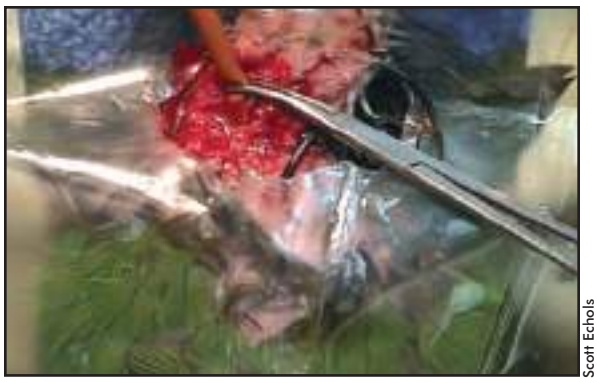

Scott Echols

Fig 35.19e | The tube is positioned for suturing.


Scott Echols

Fig 35.19f | A purse-string suture is placed around the tube to prevent crop contents from leaking.


Scott Echols

Fig 35.19g | The tube is coiled alongside the head. A bandage is applied that allows syringe access, but prevents the bird from pulling or scratching out the tube.

Despite aggressive medical therapy, some of the patients will succumb within the first hours to days following presentation. Patients in this condition are not surgical candidates. Therefore, topical treatment, hydrophilic bandaging and supportive care are indicated. If a fistula occurs prior to delineation of the affected area and the establishment of a granulation bed, it is important to initiate supportive care. A pharyngostomy tube must be placed if the fistula is large enough as not to allow any appreciable food storage or if weight loss is documented. Otherwise the bandage covering the region may assist in containing the formula.[1,9] Medical treatment alone may resolve less severe burns, with shrinkage of the scar tissue closing the potential deficit. When the fistulated area has begun to granulate, then surgical repair should be performed.[1,9]

The patient is anesthetized and placed in dorsal or lateral recumbency, depending on the location of the fistula. The area and surrounding skin are prepared aseptically. Do not use alcohol, as it may gain access to the esophagus and damage the serosa. A circumferential incision is made around the edges of the fistula and the adhered skin is separated from the ingluvies by blunt dissection. Care should be taken not to extend the fistula more than is necessary for removal of necrotic tissue. Placement of a tube or swab into the esophagus from the oral cavity will aid in the delineation of the crop. It is important to note that the skin is normally attached to the crop by two layers of striated muscle that form a sling-like support for the diverticulum of the crop. Once the crop is separated from the skin, the crop is closed in a simple continuous inverting pattern and the overlying skin closed in a simple continuous pattern. The skin and crop should be closed in two separate layers, as there is an increased risk of dehiscence if the two layers are closed together (Figs 35.20a-e).[1,9]

Occasionally thermal burns are so severe that very little viable tissue remains. The length of the crop should be maintained if possible. Esophageal strictures are more likely to develop if resection and anastomosis are performed than if only a thin strip of esophageal tissue is preserved and allowed to granulate over a stent. If enough viable tissue is present, it may be sutured over a pharyngostomy tube. A longitudinal incision with a transverse closure will increase the diameter of the esophagus and may reduce the risk of esophageal stenosis. The patient must receive frequent small feedings of a soft or liquid diet until the crop stretches and the holding capacity increases. If there is not enough viable esophageal tissue present to close the defect, it may be allowed to heal by second intention while a pharyngostomy tube is in place. Alternatively, a dermoplasty may be performed once healthy granulation tissue is present. A rotating skin flap will usually cover the defect.[1,9]

INGLUVIOTOMY AND CROP BIOPSY

Pet birds, particularly neonates, are susceptible to ingestion of foreign materials. Feeding tubes, substrate and small toys are commonly ingested. The foreign materials will obstruct food passage and irritate the crop. Small objects may be retrieved from the crop by esophagoscopy or by manual retropulsion and withdrawal with a hemostat or tissue forceps. Manual retropulsion is noninvasive, but may result in inadvertent concurrent retropulsion and subsequent aspiration of liquid from the crop. Care must be taken not to damage the crop, esophagus, pharynx, oral cavity and choana, and a thorough examination of all structures should be performed after retrieving the object to note any trauma or remaining pieces of material. An endoscopic exam of the oral cavity, choana, pharynx, esophagus and crop after removal of foreign substance is useful to determine if there is any damage to these structures.[1,9]

Indications for an ingluviotomy include foreign body retrieval, endoscopic access to the proventriculus and ventriculus, or biopsy of the crop for histopathologic evaluation. The patient is placed in dorsal recumbency and skin prepped routinely. It is important that the patient be intubated and, if there are contents within the crop, it is recommended to occlude the upper esophagus with a moistened gauze sponge to prevent any refluxed ingesta from entering the airway. An incision is made through the skin over the left lateral portion of the crop. This may be performed by scalpel, monopolar or bipolar radiosurgery, or with a laser unit. The skin is bluntly dissected to identify the crop. Stay sutures are placed in the crop wall to assist in manipulation of the crop, facilitate incising the crop and to prevent uncontrolled exit of material within the crop. The crop is then incised at the cranial aspect of the left lateral side of the sac. This area of the crop is less prone to stress as the crop fills and is not within the path of a feeding tube should the patient require tube-feeding postoperatively. This crop incision should be made with a scalpel blade or sharp scissors to preserve the integrity of small blood vessels. Radiosurgery should be used only to coagulate vessels. If the ingluviotomy is being performed for foreign body retrieval or for access to the proventriculus and ventriculus for an endoscopic exam, the incision into the crop should be performed in a relatively avascular region to control hemorrhage. If the purpose is to collect a biopsy of the crop for histopathologic examination —such as those performed as part of a diagnostic workup in patients demonstrating clinical signs consistent with proventricular dilatation disease —this biopsy should be collected from a vascular region, as it is crucial

Crop Burn Repair – Step by Step Figs 35.20a-e

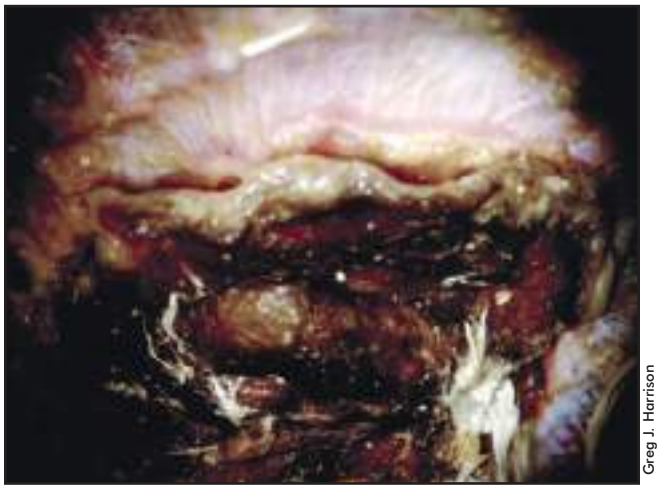


Fig 35.20a | A severe crop burn that has been allowed several days to granulate. With the initial edema gone, the tissue layers can be more easily identified and separated for repair (6x).

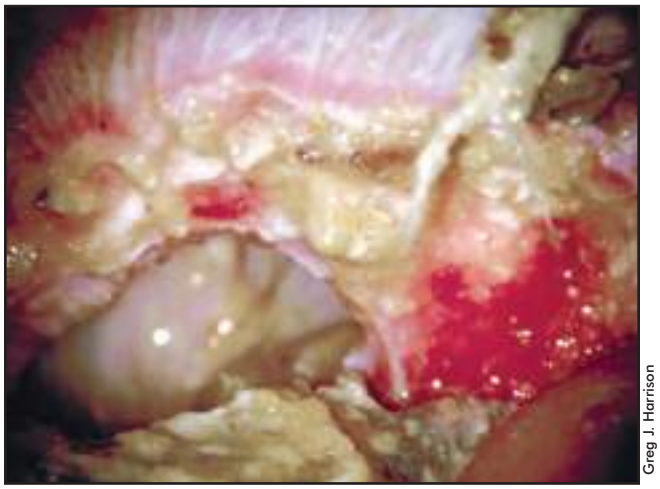


Fig 35.20b | The necrotic skin and the anterior wall of the crop have been removed in the scab (6x).

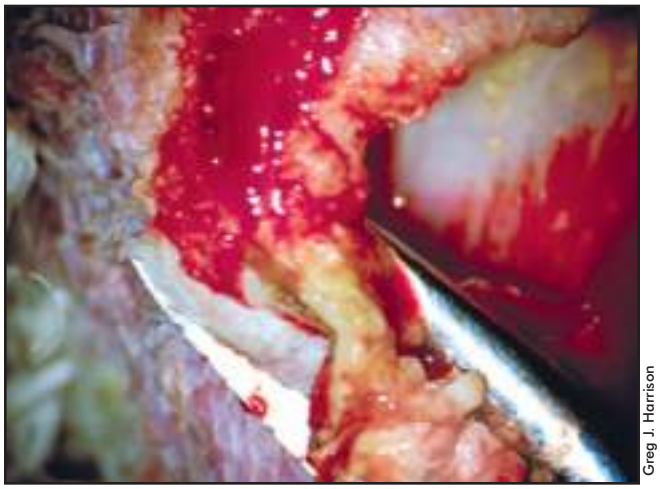


Fig 35.20c | Scissors are used to cut the granulated union of the skin and the crop into separate layers (6x).

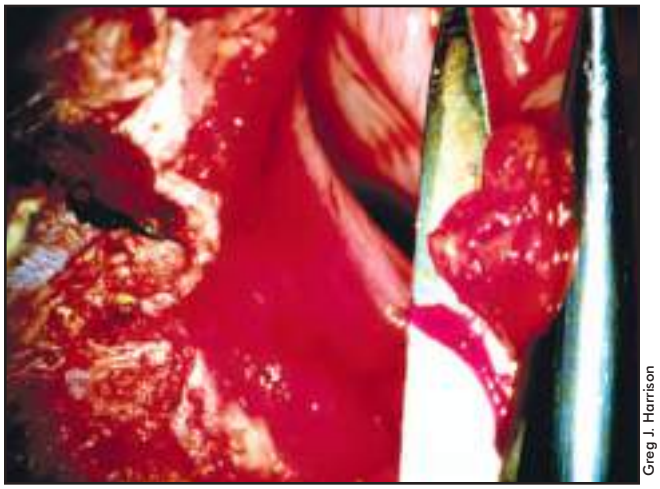


Fig 35.20d | Trimming of devitalized or granulated tissue (6x).

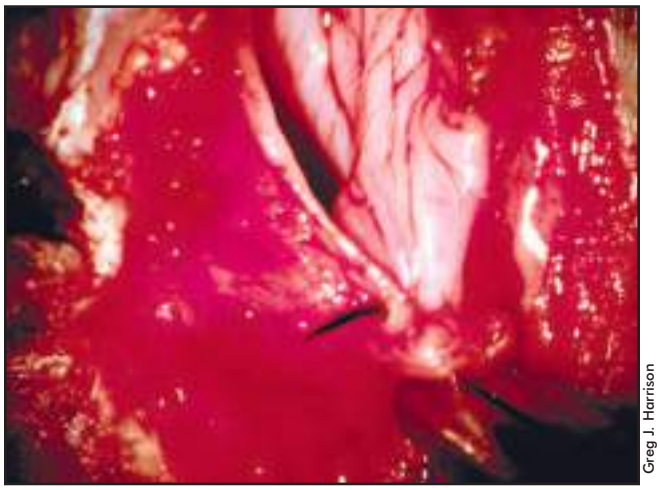


Fig 35.20e | A simple interrupted inverting suture pattern is used to close the crop (6x).

to collect nerve tissue that typically can be found in close proximity to blood vessels. Closure of the crop is accomplished with a simple continuous pattern oversewn with an inverting pattern with an absorbable monofilament suture on an atraumatic needle. Skin closure is routine (Figs 35.21a-h).[22,25,57]

ESOPHAGEAL STRICTURE CORRECTION

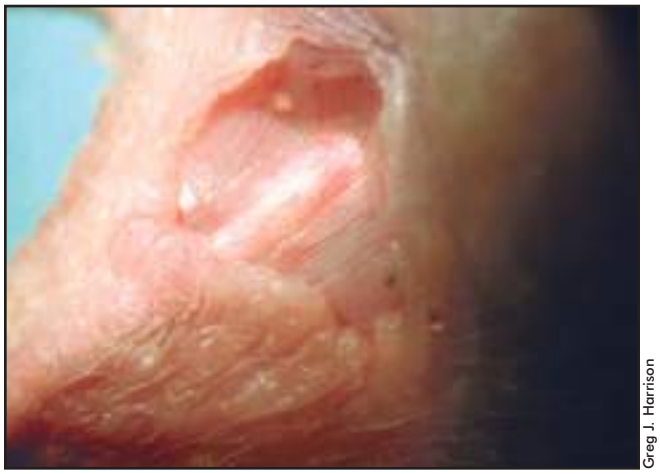
Esophageal strictures may develop as a result of previous infection (trichomoniasis, capillariasis, candidiasis), trauma from tube-feeding, thermal or caustic burns, ingestion of a foreign body or secondary to iatrogenic surgical trauma. Therefore, once an esophageal stricture is diagnosed, the underlying etiology must be determined and addressed. It may be necessary to place a pharyngostomy tube for alimentation. The stricture may be resolved by passing a series of tubes of increasing diameter through the oral cavity and esophagus past the stricture over a period of several weeks.[25]

CELIOTOMY

There are several surgical approaches to the avian coelom. These include the left lateral, right lateral, ventral midline, and cranial, mid and caudal transverse approaches. Skin incisions vary with the surgical procedure and amount of coelomic exposure required. The surgeon should evaluate the skin and subcutaneous tissues overlying the surgical site for any sign of trauma, fatty infiltration, infection and necrosis. The surgical approach should maximize exposure of the coelomic organs, but minimize the involvement of diseased skin and subcutaneous tissues. For any celiotomy, the cranial part of the patient should be elevated between 30 to 40° to prevent fluids used for irrigation, or coelomic fluid, from flowing craniad and entering the respiratory tract following penetration of the air sacs. If coelomic fluid is present, as much should be aspirated as possible or surgery delayed until the fluid is resorbed. If coelomic fluid is aspirated while the patient is anesthetized, it may be necessary to adjust the vaporizer setting as the air sacs are allowed to completely expand and anesthetic gas concentration increases.[1,9]

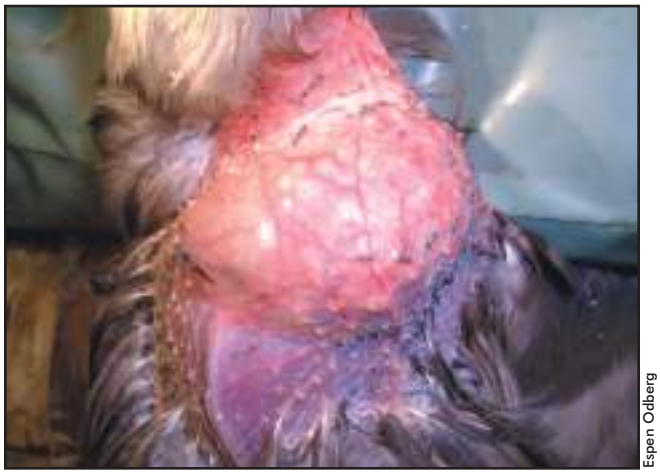
The left lateral celiotomy provides the best exposure to the proventriculus, ventriculus, female reproductive tract, left testicle, spleen, left kidney and the left ureter. The patient is placed in right lateral recumbency and the site surgically prepped. The caudodorsal border of the sternum is palpated, and the pelvic bones including the cranial pubis are identified. The left leg may be retracted caudally, creating a fold from the knee to the lateral margin of the sternum (knee web) to increase exposure to the cranial coelom (Figs 35.22a-h). Alternatively, the leg may be retracted cranially to increase surgical exposure to the caudal coelom (Figs 35.22i-p). Lung tissue can be visualized percutaneously between the fifth through seventh ribs in smaller birds. The *latissimus dorsi* and *iliotibialis cranialis* muscles obscure visualization of lung tissue in larger species. The skin is incised using monopolar or bipolar radiosurgery or with a laser from the cranial to caudal left paralumbar region. At the cranial edge of the knee web, just caudal to the last rib, the incision is continued caudoventrally to pass through the groove of the groin web to the region of the pubic bone. Once the skin incision is complete, the left leg may be further retracted caudodorsally to expose the abdominal wall. A branch of the superficial medial femoral artery and vein, visible passing over the lumbar fossa toward the pubis, should be cauterized. The abdominal wall incision is initiated in the external abdominal oblique

Ingluviotomy – Step by Step Fig 35.21a-h



Greg J. Harrison

Fig 35.21a | Cadaver showing a cotton-tipped applicator coming up the esophagus into the crop and the delicate nature and transparency of the crop's tissues.



Espen Odberg

Fig 35.21b | Distended crops may occur with primary disorders or lower gastrointestinal dysfunction.

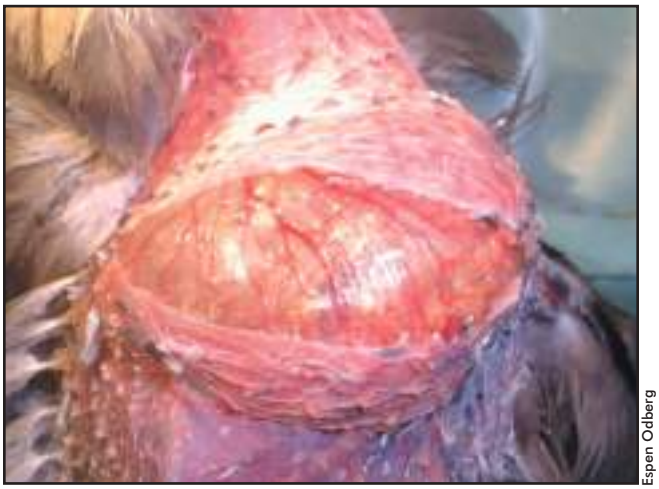

Espen Odberg

Fig 35.21c | An incision is made through the skin over the crop using a monopolar or bipolar radiosurgery unit or a laser.

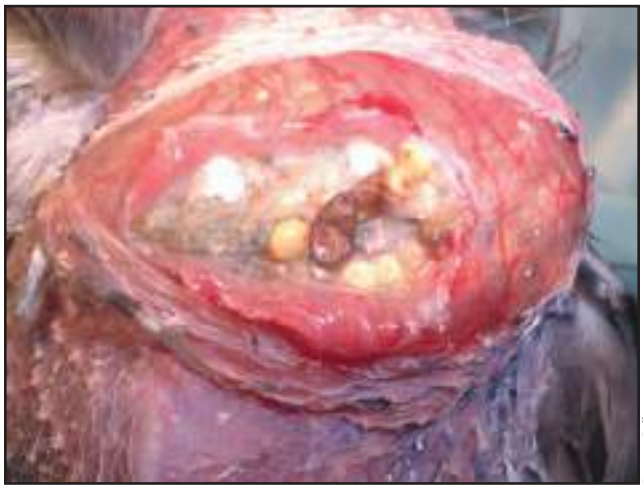

Espen Odberg

Fig 35.21d | The crop has been incised in this picture revealing a large amount of seed and other material within the crop lumen.

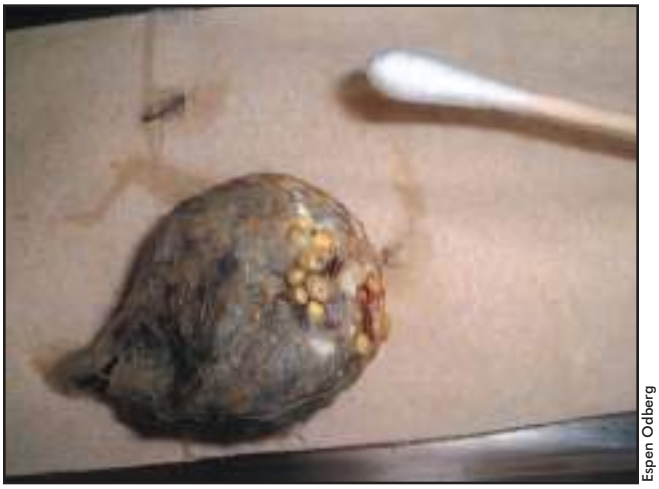

Espen Odberg

Fig 35.21e | The seed and fibrous foreign material present within the crop have been removed. Fibers from a rug had formed a matrix for a bezoar in this bird.

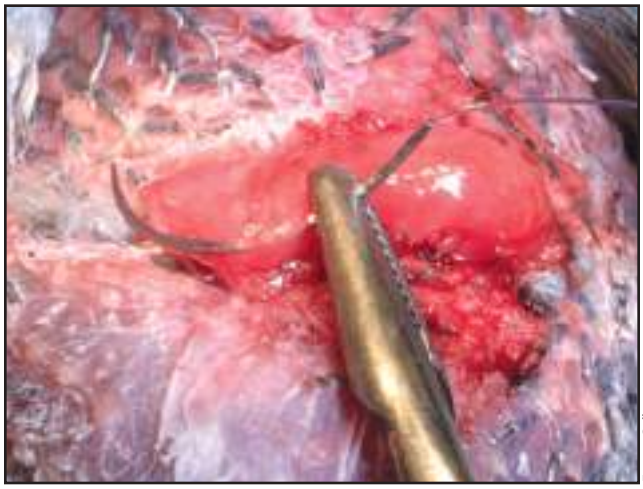

Espen Odberg

Fig 35.21f | Closure of the crop is performed with an interrupted suture pattern using an absorbable monofilament suture and an atraumatic needle. The needle pictured here is a cutting needle (vs an atraumatic needle) and is very large for use in the crop, thus increasing the risk of trauma and tearing of the tissues.


Espen Odberg

Fig 35.21g | Once the incision is closed, saline should be injected into the crop, as pictured, or infused via gavage tube in order to identify any areas that are not completely sealed. If leaks are noted, additional sutures should be placed and the patency checked again. It is important to aspirate the fluid from the crop and keep the head elevated to prevent aspiration during anesthesia recovery.

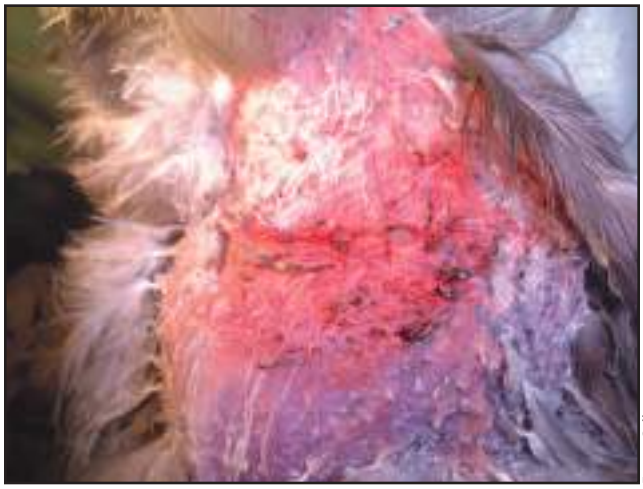

Espen Odberg

Fig 35.21h | Skin closure after an ingluviotomy.

Left Lateral Celiotomy – Step by Step Figs 35.22a-p


Scott Echols

Fig 35.22a | Left lateral celiotomy with the left leg retracted caudally is the best approach for hysterectomy, ovary, adrenal, anterior kidney or testicular investigations.


Scott Echols

Fig 35.22b | Site is plucked, surgically prepared and draped.


Scott Echols

Fig 35.22c | Close-up view of draped site.

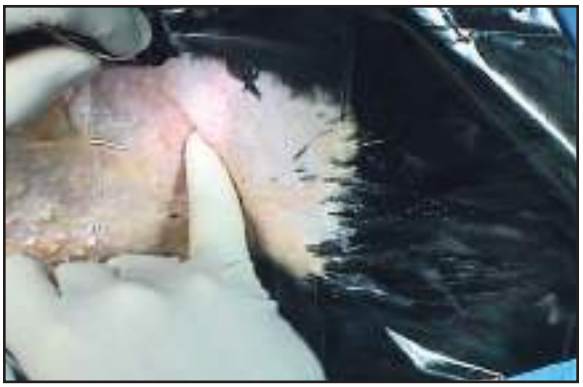

Scott Echols

Fig 35.22d | Finger identifying the anterior border of the iliotibialis cranialis muscle, just caudal to the last rib. This is the paralumbar fossa.

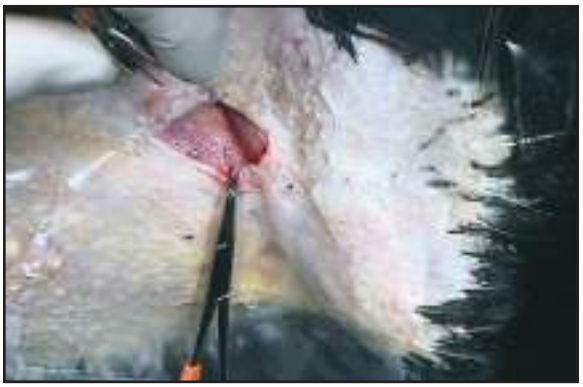

Scott Echols

Fig 35.22e | The incision is made in the middle of the paralumbar fossa.


Scott Echols

Fig 35.22f | The last rib is identified and elevated.


Scott Echols

Fig 35.22g | The last two ribs are transected.

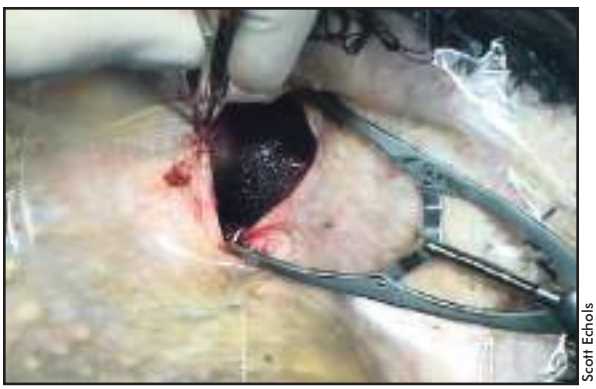

Scott Echols

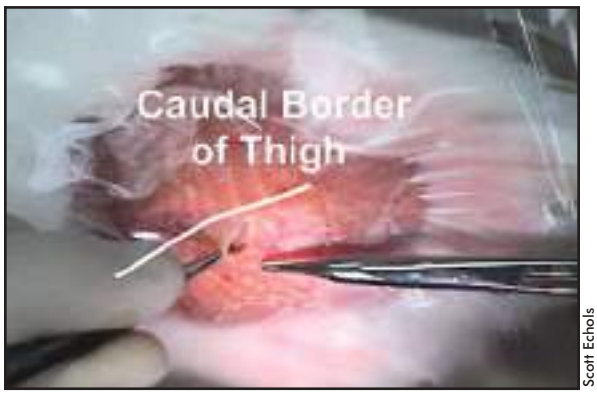
Fig 35.22h | A retractor is in place to maximize visualization.


Scott Echols

Fig 35.22i | Left lateral celiotomy with the left leg pulled anterior. This allows the ideal approach for the proventriculus, ventriculus, spleen, liver and caudal intestine tissues for surgical exploration.

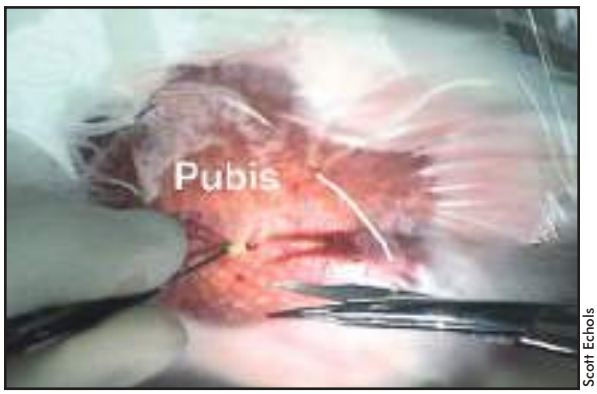

Scott Echols

Fig 35.22j | Area plucked free of feathers.



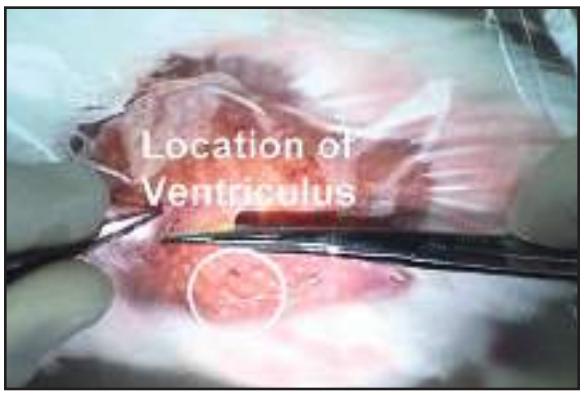
Scott Echols

Fig 35.22k | Site of entry and associated structure locations.



Scott Echols

Fig 35.22l | Site of entry and associated structure locations.



Scott Echols

Fig 35.22m | Site of entry and associated structure locations.

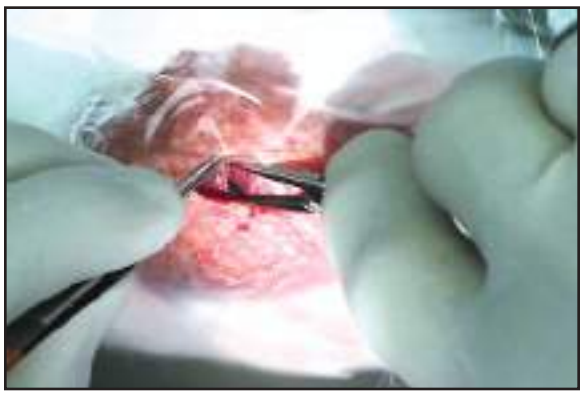

Scott Echols

Fig 35.22n | Skin incision and identification of caudal thigh muscle allows safe entry into the coelom without invading coelomic organs or tissues.

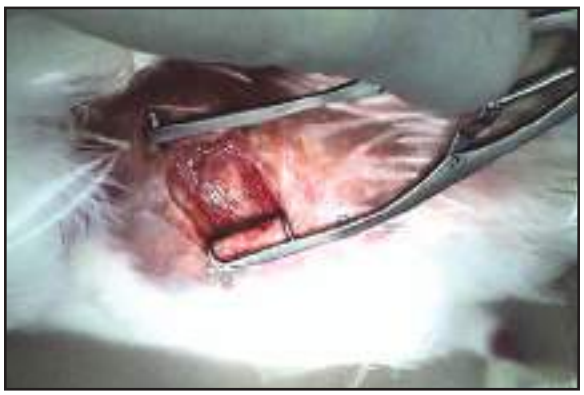

Scott Echols

Fig 35.22o | Retractor in place.

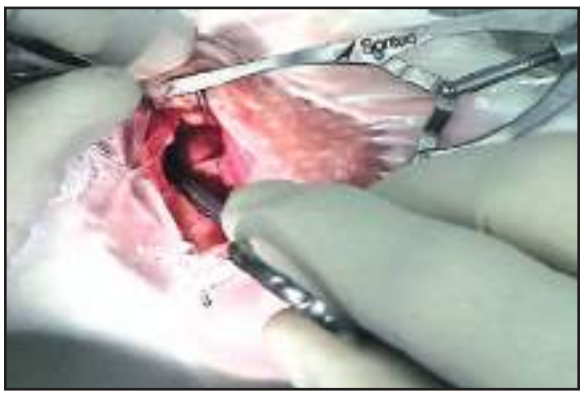

Scott Echols

Fig 35.22p | Exploration of the coelom anterior to the ventriculus.

muscle, just caudal to the last rib. This incision is extended caudally through the internal abdominal oblique and *transverses abdominis* muscles to the cranial aspect of the pubis.[1,9,25,78]

In order to achieve adequate exposure to the cranial coelom, the last one to two ribs may be transected. The intercostal blood vessels course along the cranial edge of each rib and require ligation or coagulation. In small birds, these vessels may be coagulated by inserting the indifferent electrode of the bipolar radiosurgical forceps inside the thoracic wall, lightly apposing the electrodes at the cranial edge of the rib and then activating the electrodes. In larger species, it is recommended to clamp these vessel cranial to the ribs, transect the ribs and then apply a hemostatic clip to the respective vessel. Occasionally it is necessary, particularly in larger birds, to transect the last two ribs dorsally and ventrally and remove this section of the rib entirely.[1,9,25,42]

Retraction of the abdominal wall may be maintained by a Heiss, Alm, mini-Balfour or Lone Star retractor. Retraction in smaller patients such as budgerigars may be achieved by gently applying a Halstead or Hartman mosquito forceps to the skin flap. This will avoid hemorrhage that may occur with retractors that penetrate tissue. Alternatively, ophthalmic lid retractors may be used. As the caudal thoracic air sac is entered, the caudal aspect of the lung is visible, including the ostium of the bronchi entering the abdominal air sac. The liver is noted ventrally and the proventriculus dorsally. If the abdominal air sac is entered, the lung will lie dorsolaterally. The intestines are apparent and may be gently manipulated with a moistened cotton-tipped applicator, ring-tipped ophthalmic forceps or microvascular surgical forceps. Toothed forceps traumatize intestinal tissue and may cause perforation. The proventriculus is located medially and is suspended by air sacs and suspensory ligaments. The intestines may be retracted caudally and ventrally to reveal the left kidney located dorsomedially in the coelom. The ovary or left testicle is visible at the cranial edge of the kidney and the adrenal gland noted between the gonad and the cranial division of the kidney. Obesity and organomegaly may alter anatomic location and obscure visualization of certain organs.[1,9,42]

Transected ribs are not surgically reattached during closure, but left in the correct anatomic location surrounded by soft tissues. If the seventh and eighth ribs have been removed, tension-relieving sutures must be placed from the abdominal musculature to the sixth rib. The abdominal musculature is closed with absorbable monofilament suture in a simple interrupted or continuous pattern. Skin closure is routine.[1,9]

A ventral midline, transverse or combination approach to celiotomy provides surgical access to the middle and both sides of the coelomic cavity. These approaches provide access to the small intestine, pancreas, liver, testes, oviduct (when enlarged) and cloaca.[1,9,24]

The patient is positioned in dorsal recumbency and the site surgically prepared. Most avian species have a relatively large superficial vein located subcutaneously on the ventral abdomen. This vein may be coagulated prior to incising the abdominal wall to prevent hemorrhage. The skin is incised on the ventral midline from the caudal sternum to the interpubic space. The linea alba is identified and tented upward midway between the caudal sternum and interpubic space. It is then carefully incised with bipolar radiosurgical forceps, and the surgeon may inspect the underlying tissue for adhesions or other attachments to the peritoneum. If adhesions to the abdominal wall are noted or strongly suspected, the coelomic cavity may be evaluated with the use of an endoscope prior to extending the incision. These adhesions may be broken down with the use of a cotton-tipped applicator or other blunt instrument. If adhesions cannot be broken down, an alternative surgical approach may be necessary. The incision is extended cranially and caudally by inserting the indifferent electrode under the linea alba, apposing the tips with the linea in between them and initiating the current while dragging the forceps cranially and caudally. This will incise the abdominal wall while preventing any hemorrhage. *Extreme caution must be practiced not to extend this incision too deeply to prevent iatrogenic trauma and laceration to the duodenum and pancreas, which lie from left to right just inside the abdominal wall.*[1,9,24]

A transverse and ventral combination celiotomy may be performed to increase exposure to the coelomic cavity. An incision may be performed in the cranial region (5-7 mm from the caudal border of the sternum), mid-abdomen or caudal region (5-7 mm from the cranial border of the pubic region). Care must be taken when making a caudal transverse incision, as intestinal loops often lie just under and may be attached to the abdominal musculature. A transverse incision is performed on one or both lateral sides caudal to the sternum, leaving sufficient tissue caudal to the sternum to allow subsequent closure. The size of the incision should be large enough to provide adequate exposure, but small enough to minimize escape of anesthetic gas and minimize hypothermia. Please refer to Chapter 33, Updates in Anesthesia and Monitoring for a discussion regarding anesthetic considerations for the avian patient during a celiotomy. The abdominal wall is closed using a simple continuous or interrupted pattern with an absorbable monofilament suture. Skin closure is routine.[1,25,57]

Proventriculotomy and Ventriculotomy

Proventriculotomy is indicated for the removal of foreign or toxic material from the proventriculus or ventriculus if endoscopic retrieval is unsuccessful or impossible.[1,9,42,78] The patient is positioned in right lateral recumbency. A left lateral celiotomy is performed to provide the best exposure to the proventriculus and ventriculus. The ventral suspensory structures must be dissected bluntly to retract the proventriculus caudally. Stay sutures are placed in the wall of the ventriculus to exteriorize and manipulate both structures. The proventriculus is fragile and may tear or bruise if manipulated with toothed forceps or if stay sutures are placed in this organ. Certain atraumatic microsurgical instruments may be used to manipulate the proventriculus. The coelomic cavity should be packed with moist gauze sponges to prevent gastric contents from leaking into the coelom and assist in minimizing escape of anesthetic gas.[1,9,42]

The proventriculotomy is initiated at the isthmus (junction of the proventriculus and ventriculus) with scissors. This incision is extended into the body of the proventriculus. Hemorrhage from the cut surface of the proventriculus is controlled by radiocautery. Proventricular contents and foreign material may be removed by suction or with a small curette. The lumen should be well irrigated after foreign body removal, and an endoscope may be used to perform an examination of the proventriculus prior to closure to ensure that all material has been successfully removed. The proventriculus is closed using a simple continuous pattern oversewn with a continuous inverting pattern using a fine, absorbable monofilament suture with an atraumatic needle. The inverting pattern should extend beyond the incision on both ends and the integrity of the closure evaluated with the injection of sterile saline. Abdominal wall and skin closure are routine.[1,9,42]

Postoperative fasting is not necessary and the patient should be offered food and water once completely recovered. The strength of the incision is strongest immediately postoperatively and during the fibroblastic stage. Leakage of gastric contents is not an infrequent occurrence due to the lack of omentum in birds. If the proventricular wall appears thin or friable, it may be necessary to place a duodenal feeding tube for temporary alimentation. This will allow enteral alimentation of the patient while bypassing the proventricular incision during healing (Figs 35.23a-k).[1,9,42]

The ventriculus may be accessed either through a proventriculotomy incision or through the ventriculus itself. The ventriculus is extremely vascular with heavy musculature in psittacine species, and healing postoperatively can be prolonged. Therefore, access to the ventriculus through the previously described proventricular incision is preferred when possible. The incision is initiated at the isthmus and extended into the ventriculus. The entrance into the ventriculus may be gently dilated to insert instruments to remove any foreign material, apply suction and irrigate the lumen. The ventriculus may be assessed with an endoscope to ensure complete removal of any foreign material.

An alternate approach to the ventriculus is through a cranial transverse celiotomy just caudal to the sternum. The ventriculus is identified and gently rotated clockwise to expose the thinnest portion of the ventricular wall. A longitudinal incision is performed with monopolar radiosurgery through the ventricular wall to access the luminal contents. The incision is typically 1 cm in length in a 400-g bird. Material may be removed with an appropriately sized spatula or curette. Foreign material may be removed from the proventriculus as well. The incision is closed using a horizontal mattress pattern with a slowly absorbable or non-absorbable monofilament suture. Care should be taken that the ventricular lumen is not penetrated during closure.[1,9,42]

The serosal surface of the proventriculus or ventriculus also may be biopsied when evaluating a patient for proventricular dilatation disease. It is important to biopsy a vascular region to ensure that the surgeon has obtained nervous tissue. Abdominal wall and skin closure are routine.[1,9,42]

Intestinal Resection/Repair/Anastomosis Surgery

Intestinal surgery may be indicated in the following presentations:

1. Obstructions, either from foreign bodies or secondary to adhesions or scarring.
2. Intestinal neoplasia.
3. Undiagnosed intestinal disease requiring biopsy.
4. Traumatic incidents, often involving bite wounds from predator species.
5. Congenital herniation and strangulation.
6. Repair of iatrogenic damage to the intestine occurring during celiotomy.

The patient is placed in dorsal recumbency and a midline or transverse celiotomy is performed. The vascular supply to the small intestine is via the celiac artery to the duodenum and the cranial mesenteric artery to the jejunum and ileum. Any necrotic bowel is resected gently using microsurgical instruments to prevent damage to healthy intestinal tissue. An anastomosis is performed using a 6-0 to 10-0 absorbable monofilament suture on a one-fourth-circle atraumatic needle. Typically six to eight simple interrupted sutures are necessary for end-to-end anastomosis. Abdominal wall and skin closure are routine.[1,9,25,42]

Proventriculotomy via Left Lateral Celiotomy – Step by Step Figs 35.23a-k

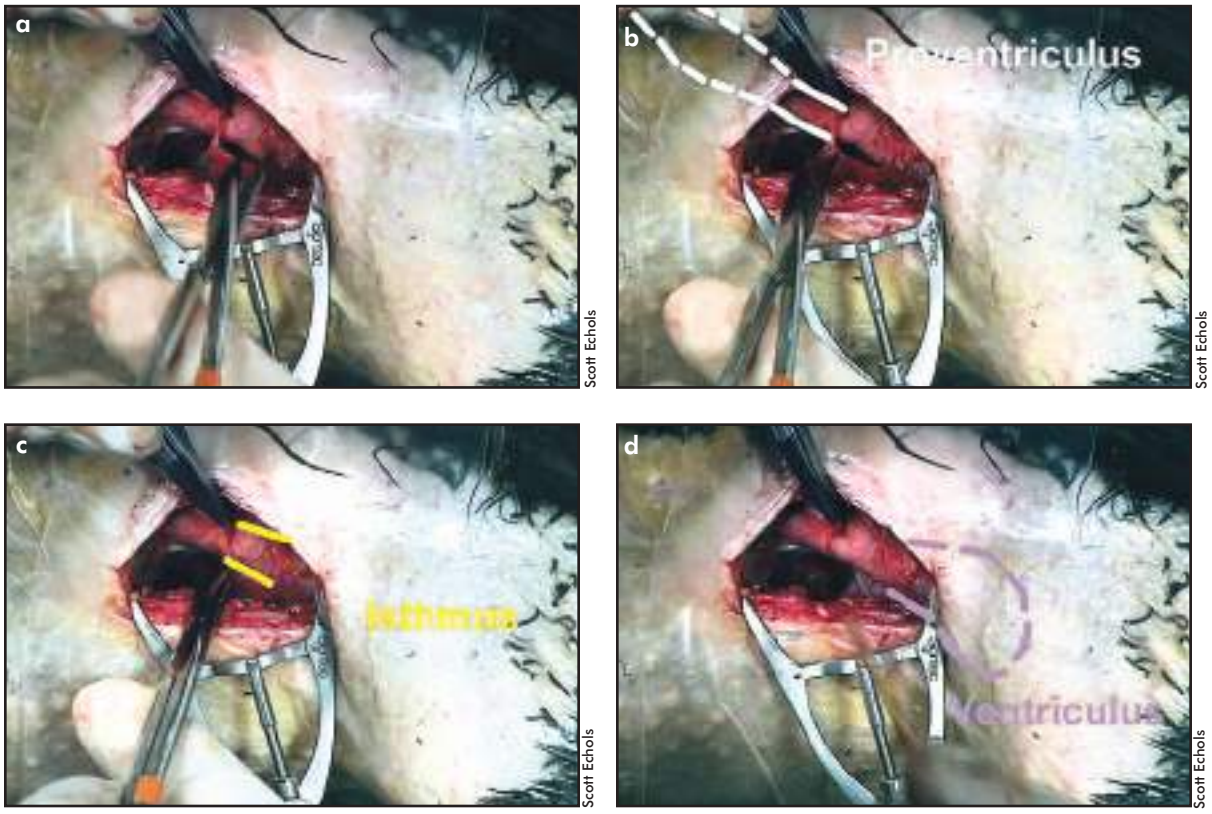


Fig 35.23a-d | Approaching the proventriculus to free it for a proventriculotomy.

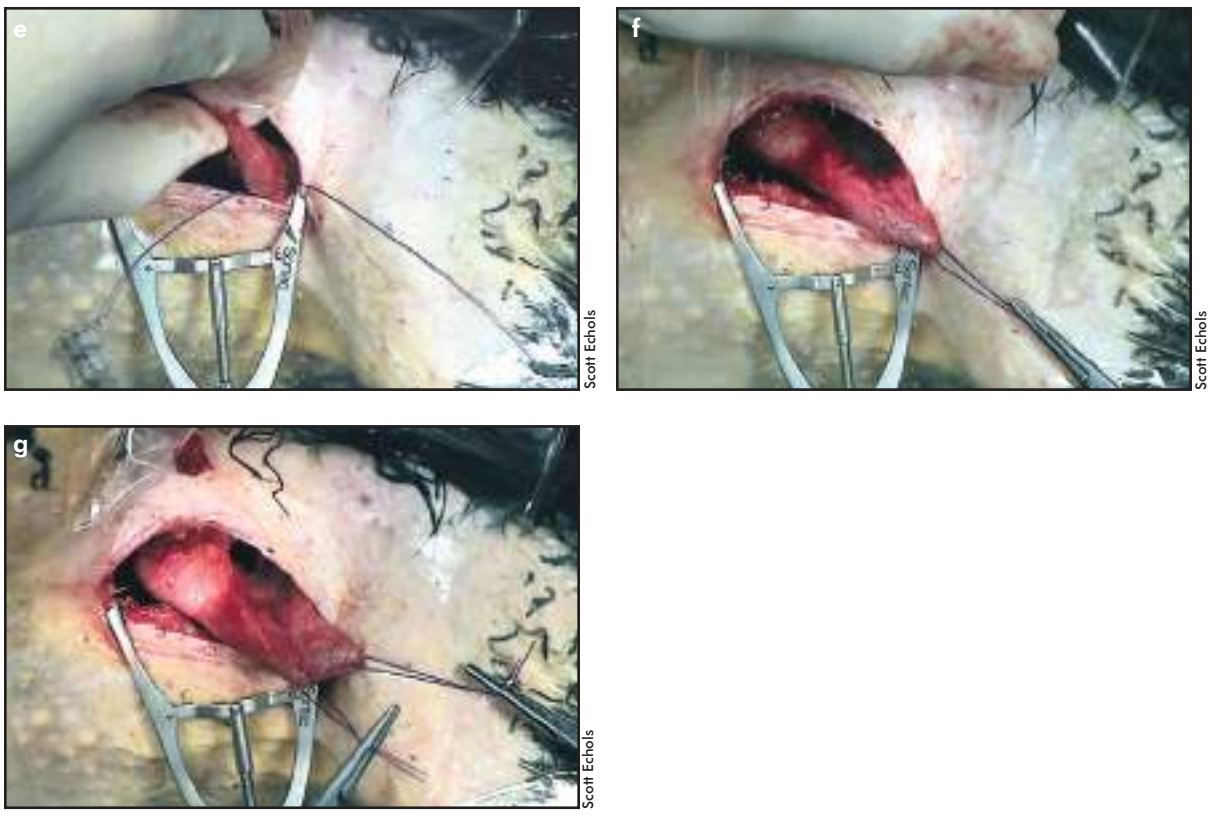
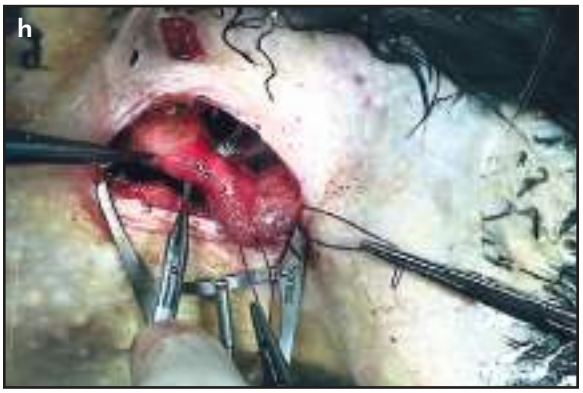


Fig 35.23e-g | Attaching suture to the muscular tissues of the proventricular-ventricular junction and freeing the proventriculus from surrounding attachments to allow exteriorization for the organ entry.



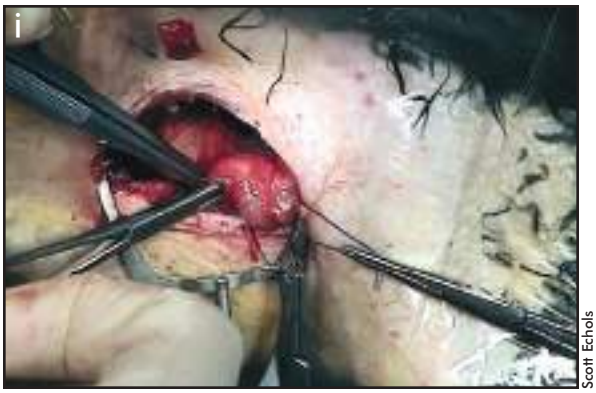


Fig 35.23h-j | Access to the lumen of the proventriculus and removal of contents and metal object noted on radiograph.

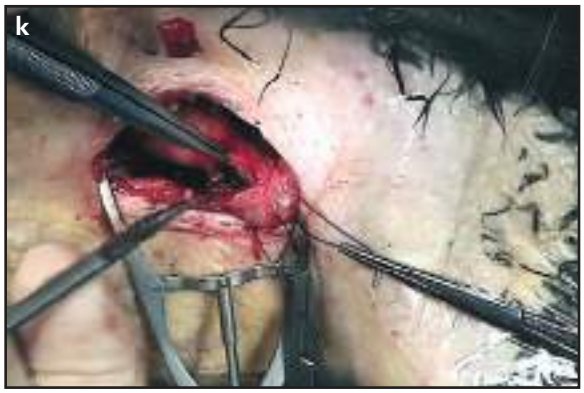


Fig 35.23k | Proventricular closure.

Enteral feeding tubes may be placed in the duodenum. This is indicated if it is necessary to bypass a diseased portion of the alimentary system. The patient is placed in dorsal recumbency and a midline or transverse celiotomy is performed. An indwelling jugular catheter, no less than one-third the diameter of the small intestine, is placed through the left abdominal wall and into the descending duodenal loop. The catheter is then advanced gently through the descending and ascending duodenal loops and the needle withdrawn from the intestine and abdominal wall. One to two sutures are placed with 5-0 monofilament in the intestine and abdominal wall to secure the intestine to the body wall and provide a tight seal. The patency and seal of the catheter is tested by injecting sterile saline solution and the celiotomy is closed routinely. The external portion of the catheter is secured using monofilament suture. The excess catheter is coiled and secured to the patient anterior to the leg and under the wing. An appropriate type and amount of a liquid diet is used to aliment the patient.[1,9,25,42] Hyperosmotic diets may cause an osmotic diarrhea. The amount to be fed should be divided into equal volumes and injected 4 to 6 times daily at a rate of approximately 1 ml/15 seconds to allow the intestine to accommodate for the volume. The catheter should be flushed with warm water or lactated Ringer's solution before and after injecting the food to prevent obstruction of the catheter.[1,9]

The patient and surgical site must be monitored closely for leakage and coelomitis, and for damage to the catheter and/or the incision site. Once the catheter is no longer needed, the suture is cut, the catheter removed and the incision is left to heal by second intention.[1,9,25]

Surgery of the Reproductive Tract

ANATOMY OF THE FEMALE REPRODUCTIVE TRACT

The avian oviduct is divided into five anatomic regions, which are microscopically distinguishable (see Chapter 18, Evaluating and Treating the Reproductive System).

The avian oviduct is suspended via the dorsal and ventral ligaments within the coelomic cavity. The cranial, middle and caudal oviductal arteries run through the dorsal mesentery vascular supply to the oviduct. Species variations exist, but in general the cranial oviductal artery arises from the left cranial renal artery, aorta or external iliac artery. The middle oviductal artery arises from the left internal iliac artery or the pudendal artery. Venous blood from the cranial oviduct enters into the caudal vena cava via the common iliac vein and venous blood from the caudal oviduct enters the renal portal or

hepatic system[24,43,45,59] (see Chapter 7, Emergency and Critical Care Fig 7.16-7.19).

OVOCENTESIS AND MANUAL EGG DELIVERY

Egg binding and dystocia occur in many pet bird species. If medical therapy fails to deliver the egg, ovocentesis or manual delivery may be attempted prior to a celiotomy. The clinician should be prepared to perform a celiotomy when attempting ovocentesis or manual delivery, as retropulsion of the egg and oviductal rupture, resulting in an ectopic egg, shell fragments or yolk coelomitis, are potential complications. A celiotomy with or without salpingohysterectomy may, in some cases, be a more rapid and effective method of resolving dystocia (see Chapter 7, Emergency and Critical Care Fig 7.16-7.19).

SALPINGOHYSTERECTOMY

Salpingohysterectomy involves removal of the oviduct. Salpingohysterectomy is indicated to prevent egg production, resolve disease conditions associated with egg production, infectious/inflammatory disease of the oviduct, oviductal neoplasia, oviductal torsion, oviductal prolapse, and weakening or herniation of the abdominal wall secondary to chronic reproductive activity.[3,9,24,34,51,76] It is not typically performed as a preventive procedure due to possible risks, namely hemorrhage, to the patient during the procedure. However, techniques such as endoscopic salpingohysterectomy in juvenile cockatiels have been described as preventive procedures for removal of the oviduct in juvenile birds.[72] During sexual and egg-laying activities, the oviduct is hypertrophied and blood supply to the ovary and oviduct is significant. It is recommended to delay surgery if possible until the reproductive tract is in an inactive state, thereby reducing the risk of hemorrhage to the patient. Egg production may be stopped, and ovarian and oviductal size and vascularity may be reduced by medical therapy prior to surgery. Please refer to Chapter 18, Evaluating and Treating the Reproductive System for a complete description of medical therapy to reduce egg production.[1,9,24]

The size and condition of the oviduct varies with the reproductive and physiologic state of the patient. Birds suffering from previous reproductive disease, particularly coelomitis, may have significant adhesions, which complicate surgical removal of the oviduct. These may include adhesions between the oviduct and the kidney, the cloaca and other coelomic structures. Caution must be taken when separating these adhesions, as hemorrhage and tearing of the affected tissue may occur. A hormonal feedback loop presumably exists between the uterus and the ovary to control follicular development and ovulation. In many birds following salpingohysterectomy, follicles will develop but will not progress to ovulation. However, some birds, namely Anseriformes, will develop large follicles and ovulate freely into the coelom. These ova may be resorbed without incident, but some birds will develop coelomitis. Clients should be informed of this potential and medical therapy to control ovulation, ovariectomy or cryosurgery of the ovary may be considered in these patients.[9,24,43]

For salpingohysterectomy, the patient may be placed in right lateral recumbency and a left lateral celiotomy is performed. Alternatively, the patient may be placed in dorsal recumbency and a ventral midline celiotomy with or without a midabdominal transverse incision may be performed.[9,24,49] The ovary is visible at the cranial pole of the left kidney, adjacent to the adrenal gland. It may be necessary to retract the proventriculus and ventriculus ventrolaterally to improve exposure of the oviduct. The convoluted oviduct lies along the dorsal body wall in proximity to the caudal vena cava. The ventral ligament, responsible for these oviductal convolutions, courses caudally and becomes a muscular cord at the vagina. This ligament is dissected with bipolar radiosurgery to allow the oviduct to be released and positioned in a linear fashion.[1,9,76]

The fimbria of the funnel portion of the infundibulum lies caudal to the ovary and is elevated to expose the dorsal attachments. The dorsal ligament that suspends the uterus and a branch of the ovarian artery course(s) caudally along the uterus from the base of the infundibulum. A small blood vessel is identified from the ovary through the infundibulum and is coagulated and transected with bipolar radiosurgical forceps or ligated with a hemostatic clip. If it is accidentally transected without coagulation, it retracts dorsal to the ovary and is irretrievable. Manual pressure and application of a small piece of absorbent gelatin sponge or beaded polysaccharide powder may be used to achieve hemostasis. The remaining suspensory tissue may be dissected with bipolar radiosurgical forceps.[1,9,24]

The oviduct is retracted ventrocaudally once the infundibulum is free. This exposes the dorsal suspensory ligament, several small blood vessels and branches of the ovarian artery, which should be coagulated or ligated with hemostatic clips. As the dissection is continued caudally toward the cloaca, the ureter is identified as a white, tubular structure extending from the kidney to the cloaca. The ureter courses along the terminal colon and enters the cloaca, and should be identified and avoided. The uterus is ligated at its junction with the cloaca with hemostatic clips, using caution not to trap the left ureter (Figs 35.24a-j).[1,9,24]

Alternatively, if the oviduct does not contain any infectious material, two hemostatic clips may be placed at the

mid-magnum region and the tissue between them transected. This results in the removal of two shorter sections. The cranial portion is retracted ventrally and bipolar radiosurgical forceps are used to coagulate vessels and transect the oviductal ligament, thereby freeing the cranial oviductal section from the dorsal body wall and facilitating its removal. Once the cranial portion is removed, the caudal portion is retracted ventrally and bipolar radiosurgical forceps are used to coagulate vessels and transect the oviductal attachments caudally toward the cloaca. The ureter is identified and avoided. A hemostatic clip or ligature is applied at the junction of the uterus and cloaca, avoiding the ureter, and the uterus is transected with radiosurgical forceps or scissors and removed. It is important to ligate the entire circumference of the uterus to prevent any postoperative reflux and leakage of feces and urates from the cloaca into the coelom. Closure is routine and previously described. Samples are collected for cytology, bacterial culture and histopathologic examination, when indicated.[1,9,24]

CAESAREAN SECTION/OVIDUCTAL-SPARING CELIOTOMY

It is often recommended that a salpingohysterectomy be performed when a celiotomy is necessary for reproductive problems such as removal of an egg that is bound. However, it may be necessary to salvage the reproductive capabilities of some avian patients. In addition, it may be prudent in some patients to initially remove the problematic egg without performing a hysterectomy, employ medical therapy to reduce the size and vascularity of the oviduct, and perform a salpingohysterectomy at a later time. The surgical approach varies with the location of the egg. If located cranially, a left lateral celiotomy is recommended, and if caudally located, a ventral midline approach with or without a transverse incision provides optimal exposure. The oviduct is incised directly over the egg, avoiding obvious blood vessels, and the egg removed. The oviduct is examined for gross abnormalities and samples are collected for cytology, bacterial culture and histopathologic examination. The oviduct is closed with a simple interrupted or continuous pattern using an absorbable monofilament suture. An inverting pattern is not recommended since this may reduce the oviductal luminal diameter. Abdominal and skin closure are routine. It is recommended to rest the hen from reproductive stimuli for a minimum of 2 to 4 weeks and if possible for the remainder of the reproductive season or longer, based on culture and histopathologic results. It is crucial to identify and correct the etiology that initiated dystocia prior to resuming breeding.[24]

REMOVAL OF THE OVARY AND OVARIAN BIOPSY

Partial or complete ovariectomy may be indicated in patients that suffer from ovarian neoplasia, ovarian granulomas, persistent follicular activity, oophoritis and ovarian cysts that do not resolve with medical therapy. It is a challenging procedure and often poses great risk to the patient. Due to this risk, medical alternatives including hormonal manipulation and intralesional chemotherapeutic administration should be explored. It also is important to note that none of these procedures have been satisfactorily studied in pet birds. Laparoscopic ovarian biopsy is generally preferred. Hemorrhage is a significant potential complication and the clinician should be prepared to perform an emergency celiotomy during the laparoscopic procedure.[1,24,57]

The avian ovary is attached to the cranial kidney and the dorsal body wall by the mesovarian ligament and receives its blood supply from the ovarian artery, which originates from the left cranial renal artery or directly from the aorta. Accessory ovarian arteries also may arise from adjacent arteries. The ovarian artery further divides into many branches, with the greatest blood flow directed to any large preovulatory follicles that are present. Ovarian veins join to form the main anterior and posterior veins that drain into the overlying vena cava. Multiple left ovarian veins may be present and drain into the cranial oviductal vein. Venous blood then enters the common iliac vein and finally the vena cava. The cranial oviductal vein may be too short or too poorly developed to identify. Multiple short veins appear to enter the common iliac vein over the length the dorsal base of the ovary.[24,34,43,45,59]

The ovary is tightly adhered to its dorsal attachments. This makes complete excision of the ovary extremely difficult and poses significant risk of hemorrhage to the patient. The avian ovary is attached to the cranial renal artery by a short stalk and the attachment to the common iliac vein is intimate and extensive. Life-threatening hemorrhage often occurs from a lacerated common iliac vein during ovariectomy.[24]

Ovariectomy and salpingohysterectomy may be attempted in the juvenile bird as an effort to prevent future reproductive disease. Ovariectomy has been described in many poultry studies. Unfortunately, most articles poorly elucidate the exact technique or associated complications.[52,66,70,81,86] Any of these procedures still pose significant risk to the hen, and clients should be well counseled regarding the potential for hemorrhage and complications. A ball-tipped electrocautery probe may be used to coagulate ovarian follicles of immature hens. However, this procedure results in ovarian regeneration

Salpingohysterectomy via Left Lateral Celiotomy – Step by Step Figs 35.24a-j

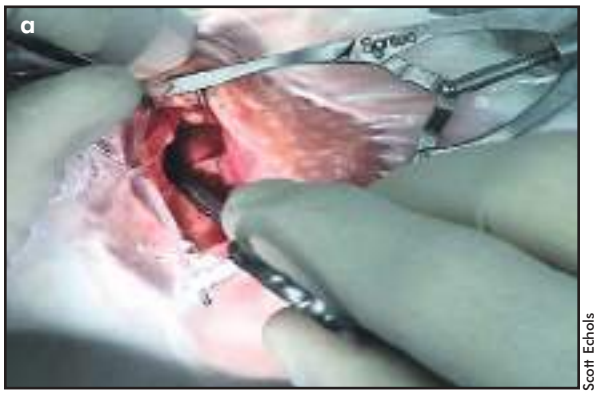


Fig 35.24a | From the left lateral approach with the leg caudal, the bird will undergo a salpingohysterectomy.

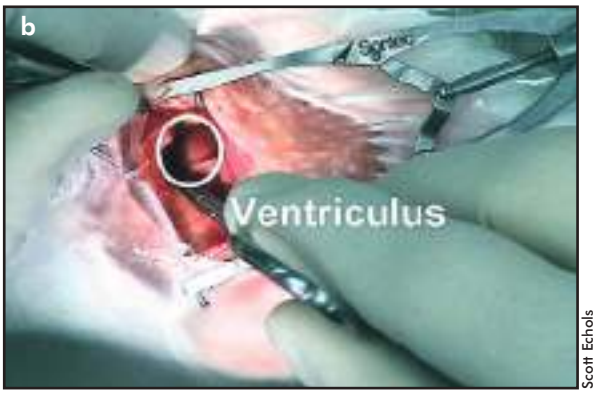
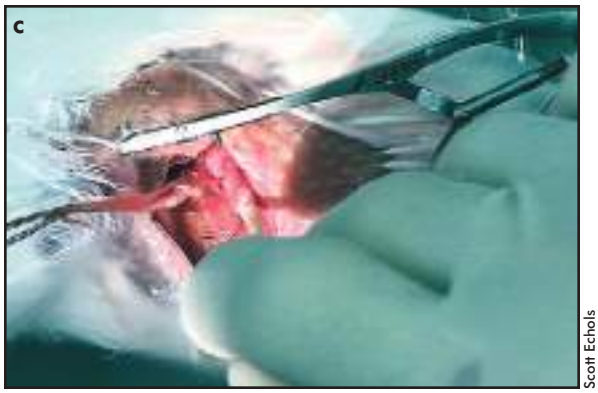
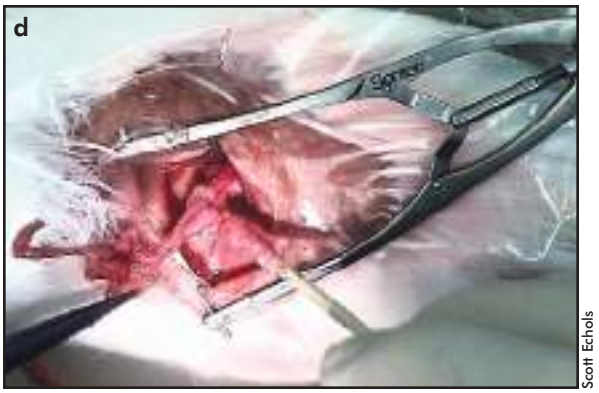
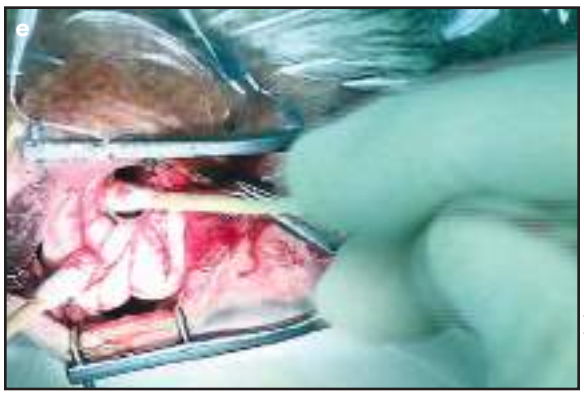


Fig 35.24b | Opening the confluent wall of the air sacs and the suspensory tissues of the proventriculus to allow entry into the hepatoperitoneal cavity.







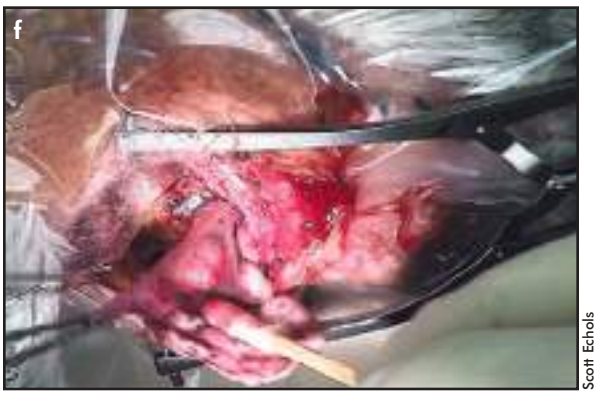


Fig 35.24c-f | Identifying and exteriorizing uterine tissues for a salpingohysterectomy and/or ovary removal. The use of cotton-tipped applicators allows the very fragile uterus to be manipulated. The blood supply comes from the dorsal aspect of the salpinx and uterus, or from vessels via the ovarian or cloacal areas. Exteriorizing the body of the uterus and transecting it, allows the maximum visualization of the vessels and surrounding tissue that must not be traumatized. Bisecting a large uterus as shown in the lower example can simplify exteriorization.

in mature hens. A procedure to remove the ovary of juvenile hens includes manually removing the ovary in toto. The caudal end of the ovary is grasped with angled hemostats and pulled gently in a cranial direction with clear separation from the dorsally located common iliac vein. When performing this procedure it is important to stop immediately if any resistance occurs to prevent tearing of the overlying vein.[9,24]

Ovariectomy in the adult hen must include removing ovarian follicles or cysts, debulking the mass of the ovary and then removing the ovary just ventral to its blood supply. The patient is placed in right lateral recumbency and a left lateral celiotomy is performed. Any large preovulatory follicles are either manually debulked or aspirated. Blood-filled follicles may represent previously ruptured blood vessels from an invasive mass and caution must be taken when removing these to prevent hemorrhage. Ovarian cysts should be aspirated. When aspirating

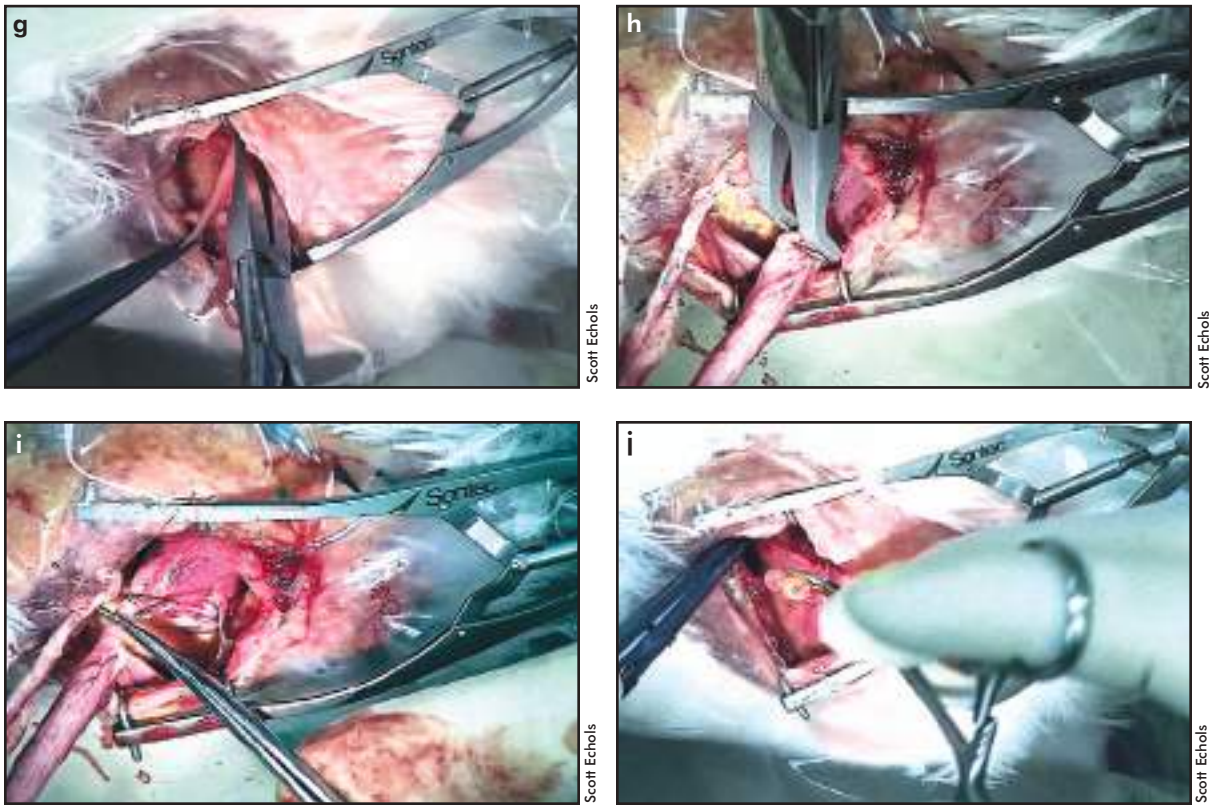


Fig 35.24g-j | Hemostatic clips are applied to the portion of the uterus that attaches to the cloaca. The uterus can then be transected and removed. The anterior vessels from the uterine ligament are ligated with hemoclips in a similar manner (Sealing these vessels with radio current alone is not sufficient). If hemostasis is not achieved in a rapid and thorough manner, the accurate placement of oxidized regenerated cellulose mesh may be required. Closure is routine. Some birds (especially budgies) have very thin paralumbar musculature and sutures tend to tear easily. Including the medial tissues of the thigh and encircling a rib with suture and then a layer of the cellulose mesh to fill the deficit has worked in such cases. Topical and systemic analgesics are needed.

follicles or cysts, a small (23-25 gauge) butterfly catheter may be inserted into the most avascular region to prevent hemorrhage and the contents aspirated. Care must be taken not to spill aspirated contents, particularly in conditions such as oophoritis, which could result in development of infectious coelomitis. Once follicles and/or cysts have been removed or aspirated and collapsed, the ovarian surface is visible and prepared for debulking. Leakage of cystic contents does not typically result in coelomitis in non-infectious conditions.[24]

An angled DeBakey neonatal vascular clamp is applied dorsal to the ovary to occlude the vascular supply. This clamp is atraumatic, remains in the surgical site without obstructing the surgeon's view and provides hemostasis. This clamp must be applied parallel to the spine to avoid entrapping the aorta and peripheral nerves. The ovarian tissue is excised with the use of a monopolar radiosurgical wire loop until very little tissue protrudes through the hemostatic clamp. The vascular hemostat is carefully opened, but left in place while the area is monitored for hemorrhage. If hemorrhage occurs the hemostat may be replaced. If possible, the vascular clamp is then opened, moved dorsally and reapplied to the ovarian base. This process is continued until the overlying vasculature is clearly visible and the course of the common iliac vein may be seen.[24]

Once the bulk of the ovary has been removed, the vascular supply must be securely ligated and the remaining ovarian tissue excised. One to two hemostatic clips are applied dorsal to the vascular clamp with 90° clip applicators. It is recommended to apply the first clip in a caudal-cranial direction and the second clip in a cranial-caudal direction to incorporate the entire vascular supply. The remaining ovarian tissue is excised with the use of a monopolar radiosurgical wire loop. Another procedure may be pursued if the ovarian attachment to the common iliac vein is too extensive to apply hemoclips, or if there is erosion into the overlying vessel, as with some invasive ovarian neoplasia. The common iliac vein is ligated with a hemoclip just caudal to the ovary and cranial to its junction with the caudal renal vein. When performed properly, the ovarian artery and common iliac veins are effectively clamped. This allows the surgeon to carefully dissect the ovarian tissue from the overlying vessels. If necessary, the ventral wall of the common iliac vein may be safely removed. It is important to note that

there is a significant risk of damaging the left adrenal gland, significantly altering blood flow through the renal portal system and the cranial renal division, and damaging the overlying kidney and lumbar and/or sacral nerve plexus. Closure is routine and the patient must be monitored for postoperative hemorrhage.[9,24]

There are reports of carbon dioxide laser ablation and cryosurgical destruction of the ovary and neoplastic tissue (R. Wagner, personal communication, 2003). These techniques may allow for more complete removal of ovarian tissue and less risk of hemorrhage, however, ablation must be strictly controlled to prevent damage to the adrenal gland, vascular supply to the left kidney and local nerves. Removal of avian gonads with laser often resulted in severe hemorrhage intra- and postoperatively.[2]

ANATOMY OF THE MALE REPRODUCTIVE SYSTEM

The male avian reproductive system includes the testes, the epididymis and the ductus deferens. The testes are located just ventral to the cranial division of the kidneys and are connected to the dorsal body wall by the mesorchium. The epididymis is located at the testicular hilus at the dorsomedial aspect of the testes, and continues caudally lateral to the ureter and terminates at the urodeum as papillae ventral to the ureteral ostium. The testicular artery arises from the cranial renal artery and provides most of the arterial blood supply to the testes. Budgerigars and passerines have a seminal glomus at the distal ductus deferens that forms a prominent projection and serves for sperm storage. There may be an accessory artery that arises directly from the aorta. Venous blood either returns directly to the vena cava or forms a common vessel with the adrenal veins. Testicular vasculature may vary among avian species or individuals.[24,46,48,59]

ORCHIDECTOMY AND TESTICULAR BIOPSY

Indications for orchidectomy include testicular neoplasia, testicular cysts, and infectious and inflammatory conditions of the testicle(s) that are unresponsive to medical therapy.[1,9,24,30,83] Laparoscopy is the preferred method for testicular biopsy.[15-17]

Castration techniques have included simple extraction in chickens (caponization), laser ablation, intravascular suction and complete surgical excision. Testicular regrowth is extremely common unless the entire testicle is completely removed. Removal of testicles carries significant risk of hemorrhage and should not be used in place of behavioral therapy, environmental manipulation, or exogenous hormone therapy for testosterone-related behavioral disease. In addition, castrated Gamble's and scaled quail maintained ornate breeding plumage and exhibited overt aggression, demonstrating that these behaviors were either learned or resulted from the influence of hormones other than testicular-produced testosterone.[24,60]

Medical therapy is instituted prior to surgery to reduce the size of active testicles. Several surgical approaches have been described. The patient may be placed in dorsal recumbency and a ventral midline celiotomy with or without a transverse flap is performed. This provides access to both the left and right testicles if both are to be removed. Alternatively, a lateral celiotomy may be performed to gain access to either the left or right testicle. It is possible to access the opposite testicle from a lateral incision by incising the midline junction of the corresponding air sacs.[9,24]

The testicle is gently retracted ventrally and a 90° vascular hemostat or hemostatic clip is applied to the base of one testicle with 90° clip applicators incorporating its vascular supply. The hemostat or clip must be applied parallel to the spine to avoid entrapping the aorta and peripheral nerves. If possible, a second clip is applied just ventral to the first. One clip is applied in a cranial-caudal direction and the second clip in a caudal-cranial direction to incorporate the entire vascular supply. The base of the testicle is incised between the hemostat or clip and the ventrally applied clips with a scalpel blade, scissors or radiosurgery. Alternatively, the testicular tissue may be debrided with the use of a monopolar radiosurgical wire loop or excised with scissors until very little tissue protrudes through the hemostatic clips. The vascular hemostat is carefully opened, but left in place while the area is monitored for hemorrhage. If hemorrhage occurs the hemostat may be replaced and another hemostatic clip applied dorsal to the previous clips, or if hemorrhage is minor a small piece of hemostatic gelatin sponge or beaded polysaccharide powder may be placed over the area. Any remaining testicular tissue that protrudes through the hemostatic clip may be ablated with electrocautery or a laser. Residual testicular tissue may result in tissue hyperplasia and produce reproductive hormones. Closure is routine and the patient must be monitored for postoperative hemorrhage.[9,24]

VASECTOMY

Indications for vasectomy in birds include providing "teaser males" and to control reproduction. This procedure is not typically performed in pet birds. In the budgerigar, the patient is placed in dorsal recumbency and a 3- to 7-mm incision is made lateral to the cloacal sphincter. The fat and abdominal musculature is carefully dissected to enter the coelomic cavity. An operating microscope is used to locate the ductus deferens and a 5-mm section of the ductus deferens is excised. The skin

is closed routinely. It is recommended to repeat the procedure on the other side 2 weeks later.[24,77]

In the finch, a 3-mm incision is made 5 mm lateral to the cloaca with the use of an operating microscope. The fat and abdominal musculature is incised to access the seminal glomera. The ductus deferens is carefully separated from the ureter and one or more sections excised without ligation. The skin is closed routinely.[11,24]

Vasectomy in larger avian patients is performed via transection of the ductus deferens via lateral or transverse celiotomy or laparoscopy. This procedure has been described in Japanese quail (*Coturnix japonica*). During a laparoscopic approach, the patient is placed in right or left lateral recumbency and the leg pulled cranially. A laparoscope is inserted at the apex of an inverted V created by the semitendinosus muscle as it passes over the last rib. The testis and ductus deferens are identified and distinguished from the kidney, adrenal gland, ureter and common iliac vein. The proximal ductus deferens is isolated and grasped with biopsy forceps. This section of the ductus deferens is transected and removed through the biopsy sheath. Care must be taken not to damage the ureter and common iliac vein. Closure is routine. The patient is repositioned on the contralateral side and the procedure repeated.[24,44]

Cloacal Surgery

CLOACAL PROLAPSE REPAIR

Cloacal surgery is indicated in those patients suffering from prolapse of the cloaca for removal of cloacoliths, and for cloacal papilloma debridement. Old World psittacines, particularly cockatoos, may develop intermittent or permanent prolapse of the cloaca. Reduced sphincter tone, chronic masturbation and straining, and chronic bacterial cloacitis have been implicated as causes for this disease. A thorough history and medical evaluation often elicits the cause. Medical therapy may include appropriate antibiotics based on cytology and bacterial culture and sensitivity and counseling clients to create a non-reproductive environment.[1,9,24]

If unresponsive to medical therapy, surgical intervention may be necessary to prevent the cloaca from prolapsing. Minor prolapse may be resolved by placing temporary mattress sutures on both sides of the vent or by placing two transverse sutures across the vent. Sutures should not be placed in the vent itself due to potential damage to the innervation. Purse-string suture of the vent is contraindicated due to frequent postoperative cloacal atony. Any procedure involving the surgical fixation of the cloacal wall to the abdominal musculature or ribs will interfere with the normal physiologic movement during voiding and egg laying, and may result in significant discomfort to the patient.[1,9,24]

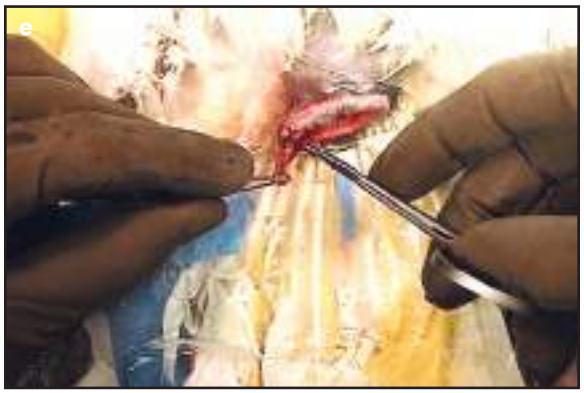
Occasionally, cloacal prolapse is due to or results in atony of the vent sphincter. Narrowing the diameter of the vent or performing a ventplasty may treat this condition. This may be accomplished by one of two procedures. Two triangular-shaped wedges are excised from the superficial surfaces of each side of the vent, without traumatizing the muscular or nervous tissue. This creates a reduction in the vent opening by one-third to one-fourth. Simple interrupted sutures are placed with monofilament nylon through the skin and subcuticular tissues. Another procedure to accomplish reduction of the vent opening includes incising one-half to three-fourths of the margin of the circumference of the vent to provide a cut surface for healing. Simple interrupted sutures are placed from one side of the vent to the other to partially close the vent opening, thereby preventing prolapse of the cloaca[1,9,24] **(Figs 35.25a-l)**.

Current theory attributes cloacal prolapse, in many cases, to behavioral and/or nutritional causes. In the interim, however, surgical reduction of cloacal prolapse may be required. Cloacopexy, following various techniques (see text) tends to be a temporary fix at best, unless underlying causes are addressed.

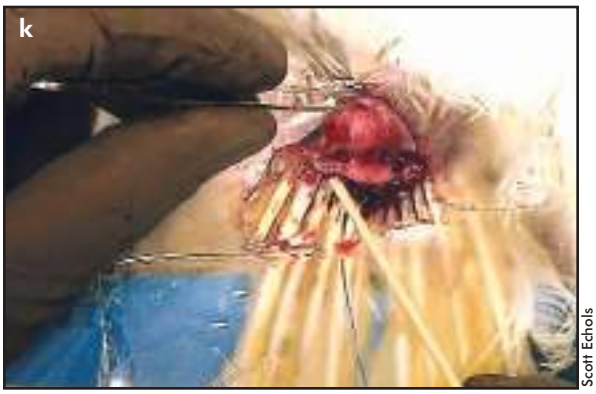
A percutaneous cloacopexy may be performed. This may offer only a temporary resolution, but it is much less invasive than the more extensive procedures later described. *(Ed. Note: With behavioral modification and hormonal manipulation, many practitioners are finding the need for more invasive cloacopexy unnecessary. The percutaneous technique has, in these editors' experience, supplied sufficient support to allow for the institution of medical and environmental therapy).*

The patient is placed in dorsal recumbency and the abdomen surgically prepared from the caudal sternum to the pubis. The prolapsed tissue is replaced manually or with a lubricated cotton-tipped applicator or gloved lubricated index finger. The applicator may be left in the cloaca to delineate the location of the ventral cloacal wall, or finger or appropriately sized syringe case may be inserted into the cloaca. Two to three percutaneous sutures are placed using monofilament nylon while the intracloacal object gently holds the ventral cloacal wall against the abdominal wall. This aids in displacing intracoelomic organs so as not to entrap them in between the abdominal and cloacal walls. Potential complications include entrapment or perforation of the ureters, rectum, duodenum and pancreas. This is avoided if the suture placement is restricted to the ventral aspect of

Prolapsed Cloacal Repair - Vent Resection and Reduction – Step by Step Figs 35.25a-l



Figs 35.25a-h



Figs 35.25i-l

Figs 35.25a-l | Prolapsed cloaca is currently considered a malnutritional and/or behavioral disorder. Until corrections for such problems become effective, the prolapses need to be replaced and maintained with some surgical method. Cloacopexy has been used for years, but often proves insufficient for long term treatment. Ventplasty is used to reduce the diameter of the orifice. A thin, superficial dermal layer is removed (c-i) and a routine closure is made. Appropriate space must be maintained to allow passing of droppings but retention of tissues. Over-closing results in retention of droppings. Adjusting the opening in such a case should occur within a matter of hours. Such surgery is often done in cockatoos (notorious self-mutilators) and devices applied to the neck to avoid mutilation are advised, as are pain medicines both systemically and topically.

the body wall, directly lateral to the linea alba. It is important to note that any surgery that places the cloaca in a fixed position will interfere with the dynamic action of the cloaca during defecation and micturition.[1,9,24]

Birds with chronic cloacal prolapse often have elongated the distal colon from constant straining and subsequent protrusion from the vent. Therefore, although care is taken to remain ventral in the placement of the cloacopexy sutures, this distended colon may be forced to make a "U" turn when the ventral cloaca is sutured too extensively in a cranial direction. This can result in folding of the colon on itself and a functional colonic obstruction. If fecal material is not produced within a reasonable period of time post-cloacopexy, the veterinarian should consider removal of the most cranial abnormal cloacopexy suture.

A circumcostal or rib cloacopexy may be performed in patients suffering from severe cloacal prolapse. The patient is placed in dorsal recumbency and a ventral midline celiotomy is performed. Placing a lubricated cotton-tipped applicator, gloved finger, or syringe case will facilitate identification and manipulation of the cloaca. The cloacal wall is identified and bluntly dissected from the surrounding fat and subcutaneous tissues. Fat tissue on the ventral surface of the cloaca must be excised for a successful outcome. The ribs are pushed caudally while the abdominal incision is elevated manually. This will bring the ribs into view to facilitate suture placement. An absorbable monofilament suture material is passed around the last rib on the right and left sides. These sutures are then passed through the full thickness of the ventral aspect of the craniolateral extent of the urodeum. Large tissue sections must be used for suture placement and it appears to be necessary to penetrate the cloacal lumen. The sutures are tied with enough tension to slightly invert the vent. Several other sutures are then placed between the body and cloacal walls. The cloaca also may be sutured to the caudal border of the sternum instead of the ribs if excessive inverting tension is placed on the cloaca when sutured to the ribs. There may be increased discomfort associated with this surgical

procedure when compared to others.[9,24]

A third cloacopexy technique includes fixing the coprodeum to the abdominal wall. A 2-to 5-mm incision is made in the serosal surface of the coprodeum approximately 5 to 10 mm lateral to the ventral midline of both the right and left sides. Corresponding paramedian incisions are made in the peritoneal surface of the body wall cranial enough so as to result in slight inversion of the vent. Three to four absorbable monofilament sutures are placed in the serosal surfaces of the coprodeum and body wall so as to appose the subserosal surfaces.[9]

An additional cloacopexy technique involves closure of a celiotomy incision to include the cloacal and body wall. The patient is placed in dorsal recumbency and a ventral midline celiotomy is performed. The cloaca is reduced and the lubricated cotton-tipped applicator left in the cloaca to aid in identification and manipulation of the cloaca. Fat is excised from the ventral cloacal surface. The abdomen is closed incorporating the cloacal wall. An absorbable monofilament suture is passed through one side of the body wall, through the full thickness of the cloaca and through the other side of the body wall in a simple interrupted pattern. The overlying skin is closed routinely. Performing the same procedure via a caudal transverse celiotomy approach offers similar benefits, but additionally it avoids incising the cloaca should a future ventral midline celiotomy be necessary.[9]

CLOACAL MASS EXCISION

Internal papillomatosis is reported in New World psittacine species including macaws, Amazon parrots, hawk-headed parrots and conures. Papillomas may be found on the mucosal surface of the cloaca, oropharynx, esophagus/crop, proventriculus, ventriculus, bile ducts and pancreatic ducts. Cystic regression and recurrence is extremely common and *E. coli* and *Clostridium* spp., are often isolated from the cloacas of affected birds. Surgical removal is recommended, particularly if the mass is causing secondary cloacal infection, fecaliths, hematochezia, cloacal prolapse or if the bird is straining to defecate, indicating a mechanical obstruction. In addition, cloacal leiomyosarcoma has been reported in a blue-fronted Amazon parrot (*Amazona aestiva*).[9,28,68]

Methods for removal of cloacal papillomas include silver nitrate cauterization, cryosurgery, radiocautery, laser surgery and excision with a scalpel. The mass and affected cloacal wall may be everted manually and the mass debulked with any of these methods. If silver nitrate is used, the area must be profusely flushed with saline to prevent cauterization of normal mucosa as soon as sufficient tissue has been cauterized to debulk the mass (Figs 35.18a-c).[1,9]

A cloacotomy may be performed to increase exposure to the affected mucosa and allow complete removal of a large occlusive mass. The patient is placed in dorsal recumbency and the abdomen surgically prepped from the caudal edge of the sternum to the vent. A ventral midline incision with or without a caudal transverse incision is made with a scalpel blade, scissors, radiosurgery or laser through the skin from the mid-abdomen to the ventral vent opening. The underlying vent sphincter muscle and the cloacal mucosa are incised with scissors to expose the entire cloaca. The mass may be removed with chemical cauterization, cryosurgery, radiocautery, laser surgery or excision with a scalpel. When chemical cauterization is used during a cloacotomy, extreme caution must be taken due to increased potential of damage to normal adjacent tissue. Hemorrhage may be controlled with radiocautery. The cloacal mucosa is apposed where the mass is excised with a small, absorbable monofilament suture in a simple continuous pattern. The cloacal mucosa is closed in a simple continuous pattern and the vent sphincter is apposed using a horizontal mattress pattern, both utilizing absorbable monofilament suture. The skin is closed in a simple continuous or interrupted pattern with a monofilament suture. An appropriate antibiotic should be used peri- and postoperatively. Surgical complications may include hemorrhage, scarring, stricture formation, fecal and urate retention, and incontinence.[1,9,23]

Mucosal stripping has been reported in a lilac-crowned Amazon parrot (*Amazona finschi*) for removal of cloacal papillomas. Recurrence of the papilloma occurred at the mucosal border adjacent to the sphincter 4 weeks postoperatively and cloacal anatomy was severely disrupted. Significant pain, lethargy, prolonged recovery, weight loss, leukocytosis and stricture of the cloaca also occurred. Therefore, mucosal stripping is reserved for cases where extensive debulking, is necessary. This may need to be done in a step wise fashion; resecting only a portion of the mucosa is less invasive.[2]

COELOMITIS

Coelomitis may occur secondary to ectopic ovulation, inflammatory and infectious conditions of the gastrointestinal, respiratory and reproductive systems. Many patients will recover with medical therapy and those patients that do require surgical intervention may benefit from medical treatment and supportive care prior to surgery.[12,13,31]

If appreciable coelomic fluid is present, it is recommended to either delay surgery or perform an abdominocentesis prior to surgery. Abdominocentesis, if performed, must be accomplished precisely on the ventral midline to prevent coelomic fluid from escaping from

peritoneal cavities and gaining access to the respiratory system. This escape of peritoneal fluid into the air sacs also can occur during celiotomy. This may result in life-threatening respiratory disease.[9,24]

The patient is placed in dorsal recumbency with the head and cranial body slightly elevated. Coelomic fluid is aspirated as described above, if indicted a ventral midline celiotomy is performed. A ventral midline approach may avoid transection of the air sacs and any fluid may be suctioned or drained. It may be necessary to adjust the vaporizer setting, as anesthetic depth may be altered when depth of respiration changes. The intestines are retracted atraumatically to gain exposure to the reproductive tract and other coelomic organs. Yolk material and tissue debris are gently removed and the coelomic cavity examined for abnormalities including tissue adhesions.[24] A salpingohysterectomy with or without ovariectomy should be performed to prevent future disease. There is risk of respiratory compromise to the patient if air sacs are transected and additional intracoelomic fluid accumulates postsurgically. There often are significant adhesions present between coelomic structures, particularly between the oviduct, and the kidney, ureter and the cloaca. Salpingohysterectomy requires breaking down these adhesions to allow excision of the oviduct. Tearing of these structures and hemorrhage are potential complications. Occasionally oviductal adhesions are too extensive to allow removal of the oviduct. Patients frequently have abdominal distension and muscular dysfunction postoperatively.[9,24] Some clinicians alleviate this by removing excess muscular tissue or by rolling the muscular tissue to create a muscular stent prior to closure of the coelomic cavity.

Miscellaneous Surgical Procedures

AMPUTATION

Limb amputation appears to be well tolerated by psittacines, however, emotional concerns of the owners often arise and may present a need for careful counseling by the surgeon prior to committing the bird to the surgery. When amputation is recommended not as a result of severe trauma to the bird, but rather due to neoplasia, nonunion fractures or chronic infection, the surgery may be postponed for 1 to 2 days to allow the owner time to reach a decision.

In cases of amputation due to neoplasia or infection, pre-operative radiographs are necessary to assure that affected bone, which may extend proximal to the visibly affected skin and soft tissue, is completely excised.

Prior to performing an amputation, the surgeon should make sure that the surgical team and equipment are prepared for the potentially life-threatening hemorrhage that can occur. At a minimum, a preoperative hematocrit and total protein should be performed to provide an indication of the overall health status of the patient. Extreme care must be taken in small avian patients and patients with a low hematocrit, as death can occur in these patients from the loss of a relatively small amount of blood. The use of a tourniquet is recommended as a means of controlling intraoperative hemorrhage during most amputations. Commercially available rubber small-animal tourniquets have the potential for causing severe skin trauma. These types of tourniquets can be used if they are well padded. A soft nylon rope, 1/2" rolled gauze, rubber band held by a hemostat, or piece of thick (>1-0) catgut suture provide alternatives. An assistant is necessary to hold the ends of the rope or suture and control the amount of pressure exerted. Ideal pressure will occlude the blood flow to the distal portion of the limb without causing trauma to the skin and underlying tissue. If skin trauma is noted, the placement of the tourniquet must be altered. If it is determined preoperatively that a tourniquet is not to be used, then careful dissection and ligation of the blood vessels is necessary. This may significantly increase the length of the surgery, but is absolutely necessary to prevent hemorrhage. If a tourniquet is not initially applied, it should be kept readily available in the event of severe hemorrhage. Being able to rapidly apply a tourniquet intraoperatively may mean the difference between life and death for an avian patient.[5]

Appropriate perioperative analgesia will significantly decrease the stress to the patient. If possible, it also is recommended that the avian patient be preconditioned to an Elizabethan collar or "sweater," which will prevent picking at the sutures of the amputation site until the skin is fully healed. Use of these devices and this period of adjustment are especially important for reducing the stress to the patient and decreasing postoperative complications The potential for hemorrhage makes prevention of postoperative picking at the skin and sutures critical.

Amputation of the Wing

When a wing amputation is performed, it is desirable to perform the surgery as distal as possible. This will allow retention of a portion of the normal function of the wing for balance. Some avian patients may traumatize the amputation site and a more proximal amputation site may be necessary. Wing amputation can be divided into three different categories: distal, mid-wing and proximal. A distal wing amputation can be defined as an amputation performed distal to the carpal joint. Indications for this surgery include inoperable neoplasia of the

distal wing, severe trauma or chronic infection. These typically occur in small birds such as budgies, lovebirds or cockatiels, making the need for amputation not uncommon in these species.[26,40]

Birds weighing less than 150 g should be anesthetized and placed in dorsal recumbency. The feathers distal to the carpal joint should be removed and the area aseptically prepared. Depending on the size of the bird, one or two hemostatic clips can be placed across the distal portion of the carpal joint firmly enough to crush the bone. The portion of the wing distal to the hemoclips can then be removed with a scissors leaving 5 mm of tissue. This small amount of tissue allows for the placement of skin sutures and provides tissue in the event of the hemoclip slipping. The incision is then closed with non-absorbable 5-0 monofilament suture with an atraumatic needle of equal size or smaller than the suture. The hemoclips can typically be removed in 2 to 6 weeks. Appropriate postoperative pain management is necessary. The bird should be observed carefully postoperatively to ensure that it is not picking at the hemoclips or sutures. If necessary, an Elizabethan or tube collar can be applied.[5,9]

Mid-wing or elbow joint disarticulation is indicated for small to medium-sized birds with trauma, nonunion radial or ulnar fractures, neoplasia or infection of the distal third of the wing. By performing the amputation at this point, the bird will lose its ability to fly but will maintain the use of the wing for balance. The bird should be anesthetized and placed in lateral recumbency. If possible, the feathers 1 to 2 cm proximal and the feathers distal to the elbow joint should be removed and the skin aseptically prepared. A tourniquet can be applied at the level of the mid-humerus to decrease the risk of severe hemorrhage.[9,20,40]

A circumferential skin incision is made distal to the elbow joint with a radiosurgical unit, laser or scalpel. Care must be taken to make the incision such that sufficient skin remains to allow closure of the incision. The insertion of the antibrachial muscles and related soft tissues are transected with radiosurgical forceps at the level of the elbow joint. The ligaments of the elbow are transected and the articular cartilage of the humeral condyles is carefully removed with rongeurs or scissors. Horizontal mattress sutures with an appropriately sized, absorbable monofilament suture are used to suture the muscle over the distal humerus. If large vessels are identified, they should be ligated with absorbable monofilament suture. The tourniquet is carefully loosened and the sutured tissue examined for any signs of hemorrhage. Hemostasis can be provided by coagulation with a bipolar radiosurgical forceps, identifying and ligating bleeding vessels, or applying very small hemoclips. The skin is closed in a horizontal mattress pattern with an appropriately sized, non-absorbable monofilament suture. Placing the suture deep into the muscle is desirable to prevent the possible formation of a hematoma postoperatively. The skin of the wing is very fragile and avoiding the formation of a hematoma may be difficult. It is important to provide postoperative pain management and an Elizabethan collar may be necessary to prevent picking at the incision.[9]

A proximal or proximal-humeral amputation is indicated for chronic trauma to the distal wing, neoplasia, nonunion or open, severely contaminated fractures and severe infection. The bird will lose the ability to utilize its wing for balance, but most psittacines appear to be able to adjust to this without complications. The patient should be anesthetized and placed in dorsal recumbency. The feathers should be removed and the skin aseptically prepared. The distal portion of the wing can be wrapped or covered with appropriate surgical draping material. Application of a soft tourniquet significantly decreases the possibility of severe hemorrhage. The tourniquet should be placed on the proximal humerus. If a mid-humeral amputation is to be performed, the tourniquet should be placed around the shoulder joint, incorporating the insertion of the pectoral muscles. If a proximal humeral disarticulation is to be performed the tourniquet should be placed around the shoulder joint including the brachial plexus.[9]

A circumferential incision at the mid-humerus is made using a radiosurgical unit, laser or scalpel. It is important to leave sufficient tissues to allow for closure of the skin. Also, it should be noted that despite the presence of a tourniquet, some hemorrhage may occur. Ligatures are placed around the distal portion of the muscles using appropriately sized, absorbable monofilament suture. The suture size should be based on the size of the bird and large enough that when tightened, there is compression without cutting of the tissue. The muscles are then transected at their musculotendinous junctions near the elbow joint using a radiosurgical unit. Blood vessels are identified and coagulated or ligated with 3-0 or 4-0 absorbable monofilament suture prior to being transected. The tourniquet should be loosened and any bleeding vessels ligated or coagulated with the radiosurgical unit. The muscles of the humerus are bluntly dissected from their tendinous attachments to the bone. The humerus is transected in the proximal third of the bone or, alternatively, the proximal humeral joint is disarticulated and the synovial tissues of the glenoid fossa are removed with appropriately sized rongeurs. The pneumatic nature of the proximal humerus makes good tissue coverage of the end of the bone very important. Inserting a piece of gelatin or collagen foam into the

distal portion of the remaining bone will ensure that blood does not travel through the humerus into the rest of the respiratory system. Impregnating this material with an appropriate antibiotic may be useful in preventing or aiding in the treatment of soft tissue or bone infection. The muscles are then sutured over the stump using a horizontal mattress or simple interrupted pattern with 3-0 or 4-0 absorbable monofilament suture. The skin and subcutis are apposed using 3-0 or 4-0 nonabsorbable monofilament suture in a simple interrupted or horizontal mattress pattern. The incision site should be monitored carefully for postoperative hemorrhage. Due to the potential for hemorrhage through the pneumatic portion of the humerus, the patient should be observed carefully for signs of respiratory distress. This surgery can be very stressful to the avian patient and may necessitate several days of hospitalization postoperatively. As with all amputations, postoperative analgesics are imperative (Figs 35.26a-g). Injection of a lidocaine derivative intraoperatively in the area of the radial nerve will also decrease post-operative pain.[9,40]

Amputation of the Leg

Most companion avian species function well with only one leg, especially psittacines. Care must be taken when considering leg amputation in avian patients. If the leg is amputated distally, the avian patient will attempt to use the remaining stump for ambulation. Contraindications for leg amputation include obesity, osteoarthritis of the contralateral leg or if the bird is unable to utilize its wings to assist in balancing. It is important to provide appropriate perching material to allow ease of grip and prevent perch-associated pododermatitis postoperatively. Amputation is indicated in avian patients with severe non-union fractures, severely contaminated open fractures, neoplasia, severe trauma or infection of the distal leg. Because a large portion of the femur is located within the skin of the body wall, a midfemoral amputation provides for adequate tissue to cover the end of the bone, prevents self-mutilation and is cosmetically acceptable. In captive raptors, postoperative pododermatitis of the contralateral foot is a common sequela, however, it is rarely observed in psittacines fed an appropriate diet and supplied with appropriate perches.[9,20,29,58,74]

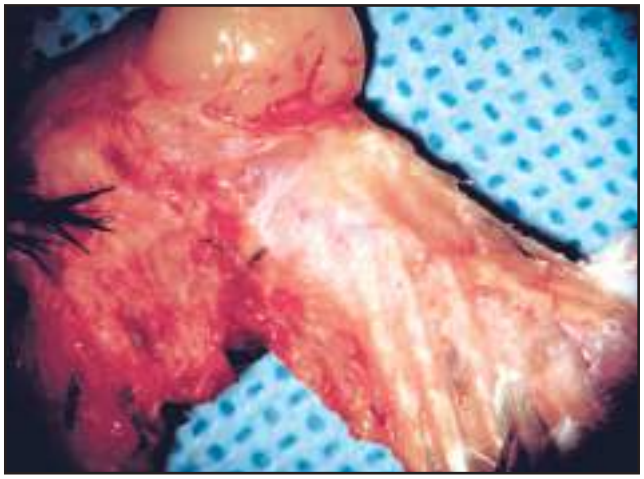
The patient should be anesthetized, placed in dorsal or lateral recumbency, and the feathers removed from the ventral abdomen distally to the stifle joint. The skin is aseptically prepared utilizing a standard orthopedic technique. The skin incision is made with a radiosurgical bipolar forceps along the web of the knee in the contour of the abdomen and semicircular incisions are made at the level of the stifle. Sufficient skin must remain to provide for closure without undue tension. Ligatures are placed around the distal portion of the muscles using 1-0 or 2-0 absorbable monofilament suture. Blood vessels are identified and ligated with 3-0 or 4-0 absorbable monofilament suture prior to being transected. A periosteal elevator is utilized to elevate the muscles from the proximal femur to the mid diaphyseal region. A bone cutter, osteotome, Gigli wire or other instrument appropriate for the patient's size is used to transect the femur. The muscles are then sutured over the stump with 3-0 or 4-0 absorbable monofilament suture in a simple interrupted or horizontal mattress pattern. The skin and subcutaneous tissues are apposed using 3-0 or 4-0 nonabsorbable monofilament suture in a horizontal mattress pattern. As with proximal humeral amputations, this surgery is very stressful for the avian patient and may necessitate several days in the hospital postoperatively. Appropriate pain management is imperative.[9,58,74]

Amputation of the Digit

Toe amputation is indicated in the event of severe trauma, neoplasia, avascular necrosis or infection of any of the digits. The tissue damage may be so severe or multiple toes may be involved such that amputation of the distal portion of the foot is also required. The same surgical techniques are utilized for both the toes and the feet. The patient is anesthetized, placed in dorsal recumbency and the distal portion of the leg aseptically prepped. It should be noted that the avian patient may present to the hospital with a large amount of fecal material on their feet. This requires careful cleaning prior to surgery because of the added risk for contamination and infection of the surgical site. Appropriate postoperative antibiotic therapy is necessary.[9,35]

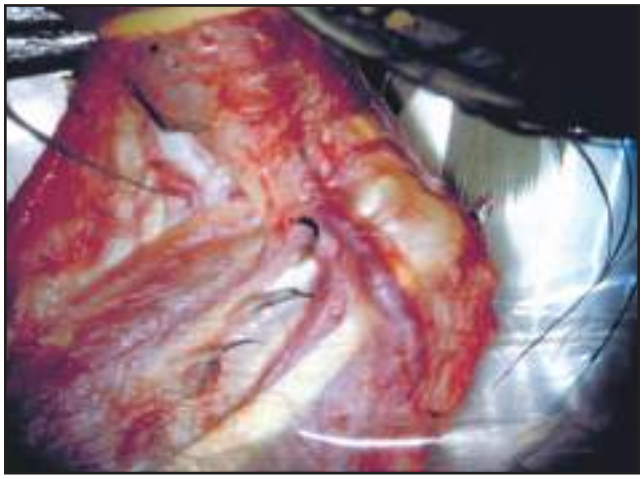
It is possible to perform two types of digit amputations, a proximal joint disarticulation and a phalangeal mid-diaphyseal amputation. A tourniquet composed of a thick suture material or a soft nylon rope is applied to the mid-tibiotarsal bone to control hemorrhage for both procedures. The site of a joint disarticulation of a digit should be at the joint proximal to the affected area. The skin should be incised distal to the stump to allow for sufficient skin for closure. A bipolar radiosurgical unit or scalpel blade can be used to make parallel horizontal incisions on either side of the toe. Alternatively, an incision is made around the dorsal two-thirds of the toe. It is important not to incorporate the plantar aspect of the digit with this approach. The phalanx is amputated at the proximal end of the bone. The joint may be disarticulated utilizing rongeurs, a scalpel blade, laser or electrosurgical unit. Any exposed joint surface can be removed prior to skin closure. Close in a horizontal mattress pattern with appropriately sized (3-0 to 5-0), nonabsorbable monofilament suture. If a horizontal incision was made, the edges of the skin are apposed with the knot tied on the dorsal surface of the skin. If a plantar

Wing Amputation – Step by Step Figs 35.26a-g



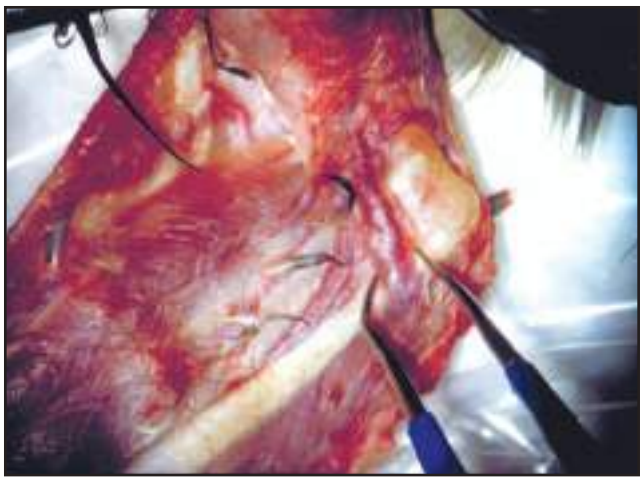
Greg J. Harrison

Fig 35.26a | An aggressive tumor involving the carpus was an indication for wing amputation. Harrison prefers to amputate at a joint, thus the elbow was the chosen site.



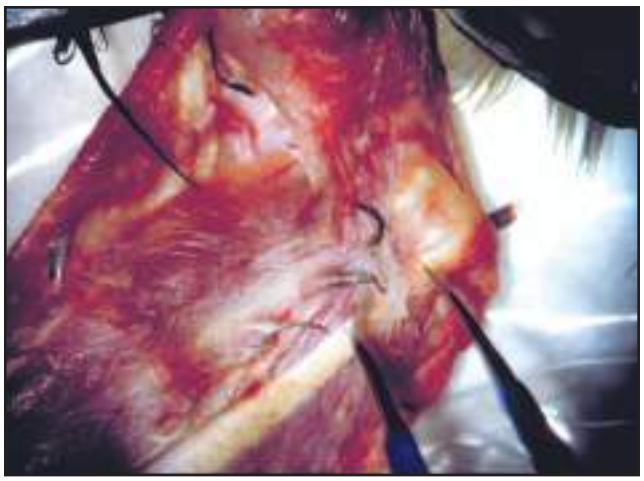
Greg J. Harrison

Fig 35.26b | The brachial vein is the first vessel encountered when amputating a wing at the elbow joint. Note the rubber band tourniquet that is secured by a hemostat.



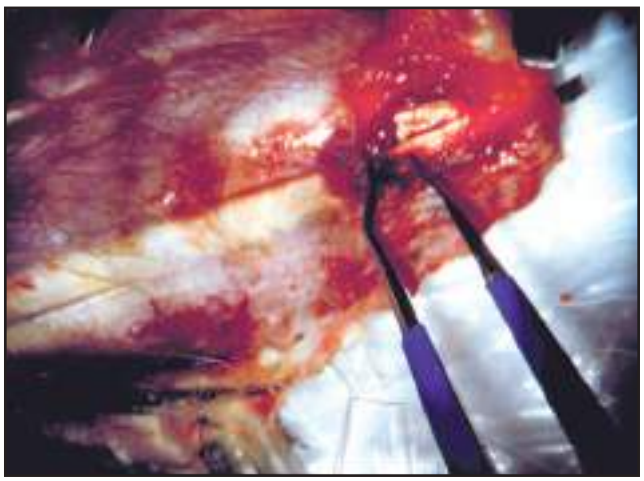
Greg J. Harrison

Fig 35.26c | The radiosurgery forceps are placed around the vessel and the coagulation setting is used to seal the vessel.



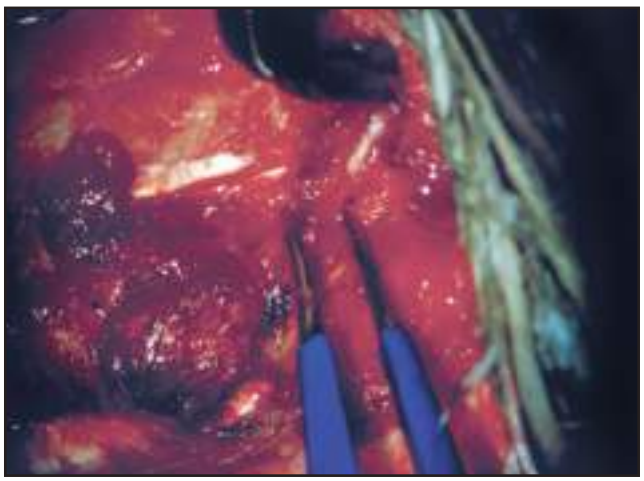
Greg J. Harrison

Fig 35.26d | The cutting current is used to incise the vessel and surrounding tissue.



Greg J. Harrison

Fig 35.26e | Vessels, ligaments and tendons are transected in a similar manner. Transection of muscle is avoided whenever possible.



Greg J. Harrison

Fig 35.26f | If the upper wing is involved, the wing can be amputated at the proximal ⅓ of the humerus or at the shoulder joint. Larger diameter vessels in this area will require more extensive and careful ligation. Muscles will require transection, however, sufficient musculature should be retained to cover the remaining portion of humenal bone in the case of amputation at this level. If amputation is performed by disarticulation of the shoulder joint, muscle should be retained and sutured to fill the dead space and prevent seroma formation.


Espen Odberg

Fig 35.26g | Skin closure after proximal wing amputation.

skin flap was created, the skin is sutured to cover the end of the bone. Placing the most dorsal suture initially will allow for symmetrical apposition of the sides. The thicker skin of the plantar surface of the toe will provide additional protection for the end of the bone.[9,35]

A mid-diaphyseal amputation is generally performed on small avian species. The skin is cut proximal to the affected tissue in a circumferential manner utilizing a radiosurgical unit or scalpel blade. The skin is then carefully retracted, exposing the underlying bone. The diaphysis of the bone is transected using rongeurs or scissors. The skin is then pulled distally to cover the exposed bone. Sufficient tissue is removed from the dorsal portion of the remaining skin of the toe to create a flap of plantar skin that will cover the end of the bone. The skin is then sutured using 4-0 or 5-0 non-absorbable monofilament suture in a horizontal mattress pattern. The affected toe and incision should be monitored closely for postoperative signs of infection. An Elizabethan collar or bandaging of the foot, can be utilized to prevent picking at the incision postoperatively.[9]

ABDOMINAL WALL HERNIA REPAIR

Abdominal wall hernias may be congenital or acquired. The etiology is undetermined, but several disease conditions have been implicated. Hyperestrogenism leading to weakening of the abdominal musculature has been suggested as a predisposing cause in budgerigars (*Melopsittacus undulatus*) and cockatiels (*Nymphicus hollandicus*). Chronically egg-laying hens and changes in calcium metabolism may contribute to muscular atony. Lack of exercise, malnutrition, obesity, space-occupying masses, organomegaly, trauma, or chronic masturbation and straining may result in weakening of the abdominal musculature and abdominal distension.[9,50,53,56]

Most "abdominal hernias" in birds do not in fact have an opening in the aponeurosis of the abdominal muscles. A true hernia is defined as a protrusion of an organ through connective tissue or through the abdominal wall in which it is usually enclosed. Therefore, a thorough examination is crucial to accurately diagnose an abdominal hernia. Herniation may include separation of the aponeurosis of the abdominal musculature at the ventral midline, allowing coelomic viscera to displace outside the muscular body wall. Clinical signs may include disease associated with entrapment of intestinal loops. "False hernias" or abdominal distension requires investigation of primary etiologies, therefore, a thorough examination and diagnostic protocol is important to accurately assess the patient's condition. In many patients, false hernias are of little clinical consequence and do not require surgery. In addition, surgical repair may carry significant risk. Respiratory compromise may result when reducing the coelomic contents due to increased pressure on the abdominal and thoracic air sacs. Herniorrhaphy is indicated if the abdomen is being traumatized due to distension causing abrasion or ulceration by contact with the perch or floor, herniation of coelomic viscera poses risk to the patient, secondary clinical disease such as egg binding, intestinal obstruction, cloacal urolithiasis or difficulty expressing urates develop. Abdominal hernia and distension have been associated with hepatic lipidosis, reproductive tract disease, intracoelomic lipomas and peritoneal cysts.[9,50]

Pre-operative radiographs, with gastrointestinal contrast media if needed, will identify the location of structures within the distended or herniated abdomen.

The patient is placed in dorsal recumbency and a ventral midline or an elliptical incisional celiotomy is performed. Caution must be taken when making the midline incision to avoid iatrogenic trauma to underlying viscera. Herniated viscera are gently replaced and the hernia repaired while patient respiration is closely monitored. The distended abdominal wall is trimmed on either side of the linea alba to create a normal anatomic abdominal wall. The abdominal wall is then sutured in a simple interrupted or continuous pattern with absorbable monofilament suture and the overlying skin closed routinely. If the body wall defect is extremely large, a mesh implant may be considered for repair.[1,9]

ABDOMINAL MASS EXCISION OR BIOPSY

Surgical excision or biopsy of neoplastic masses is indicated for several different types of disease conditions. Removal often requires prolonged anesthesia, strict

hemostasis and careful anatomic dissection. This predisposes the patient to hypothermia, hemorrhage and metabolic compromise. Surgical procedure varies with the organ affected. Laser surgery may show some promise for removal of neoplastic and granulomatous masses.[3,9,51]

When a biopsy and histopathologic diagnosis are obtained, or when complete surgical resection carries an unacceptable risk, alternative treatments may be preferred. Recent advances in oncology, including intralesional cisplatin and carboplatin, have shown promise for various abdominal neoplasias (see Chapter 20, Overview of Tumors).

LIPOMA EXCISION

Lipomas are frequently the result of obesity. Some species demonstrate a predisposition to development of lipomas (see Chapter 13, Integument). Correction of malnutrition, obesity and increased activity level often will reduce the size of lipomas.[9] Medical treatment including supplementation with L-carnitine, or levothyroxine if a hypothyroid condition has been accurately diagnosed, has not met with consistent results.[19,75] It is important to note that liposarcomas, leiomyosarcomas and other masses that mimic lipomas have been reported in pet psittacines.[56]

Lipomas that are well encapsulated are generally simple to excise. However, large, diffuse or broader-based lipomas can pose a significant patient risk when excision is attempted. Occasionally, loss of adequate vascular supply will result in central necrosis and ulceration. Large abdominal lipomas may be prone to inadvertent trauma and damage to the overlying skin.[9] The Cavitron ultrasonic surgical aspirator (CUSA) has been used to safely resect lipomas in budgerigars. This ultrasonically powered aspirator selectively fragments and aspirates parenchymal tissue while sparing vascular and ductal structures. Preliminary evaluation suggests that the CUSA may provide reduced tissue necrosis and hemorrhage, increased visibility, shortened operating and anesthetic duration, and reduced recovery time when compared to blade resection, bipolar cautery and CO_2 laser excision.[85]

RENAL BIOPSY

Biopsy of the kidney may be performed through a lateral, ventral midline or combination ventral midline-transverse celiotomy.[9] In addition, a dorsal pelvic approach has been described.[80] Renal biopsy may be performed via laparoscopy. The reader is referred to Chapter 24, Diagnostic Value of Endoscopy and Biopsy for a complete description of this procedure.

Products Mentioned in the Text

a. Vascular clamps, Sontec Instruments, www.sontecinstruments.com
b. Weck Hemoclips, Solvay Animal Health, Inc., Mendota Heights, MN, USA
c. Gordon Laboratories, Upper Darby, PA, USA
d. HemoBlock, Abbot Laboratories, North Chicago, IL, USA
e. Silverglide Nonstick, Select-Sutter, Germany

References and Suggested Reading

1. Altman RB: Soft tissue surgical procedures. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, PA, WB Saunders Co, 1997, pp 704-732.
2. Antinoff N: Treatment of a cloacal papilloma by mucosal stripping in an Amazon parrot. Proc Assoc Avian Vet, 2000, pp 97-100.
3. Bauck L: Neoplasms. *In* Rosskopf WJ, Woerpel RW (eds): Diseases of Cage and Aviary Birds. Baltimore, MD, Williams & Wilkins, 1996, pp 480-489.
4. Bennett RA: Instrumentation, preparation, and suture materials for avian surgery. Semin Avian Exotic Pet Med 2(2):62-68, 1993.
5. Bennett RA: Surgical Considerations. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 1081-1095.
6. Bennett RA: Techniques in avian thoracoabdominal surgery. Proc Assoc Avian Vet, 1994, pp 45-57.
7. Bennett RA: Thoracic surgery and biopsy. Proc Assoc Avian Vet, 1995, pp 297-299.
8. Bennett RA: Preparation and equipment useful for surgery in small exotic pets. Vet Clin North Amer Exotic Anim Pract 3:563-586, 2000.
9. Bennett RA, Harrison GJ: Soft Tissue Surgery. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 1096-1136.
10. Bennett RA, et al: Histologic evaluation of the tissue reaction to five suture materials in the body wall of rock doves (*Columbia livia*). J Avian Med Surg 11(3):175-182, 1997.
11. Birkhead TR, Pellatt JE: Vasectomy in small passerine birds. Vet Rec, 1989, pp 125, 646.
12. Bowles HL: Diagnosis and management of female avian reproductive diseases. Proc Assoc Avian Vet, 2001, pp 349-357.
13. Bowles HL: Reproductive diseases of pet bird species. Vet Clin Exotic Anim 5:489-506, 2002.
14. Clippinger TL, Bennett RA: Successful treatment of a traumatic tracheal stenosis in a goose by surgical resection and anastomosis. J Avian Med Surg 12(4):243-247, 1998.
15. Clubb S, et al. Endoscopic testicular biopsies for evaluation of fertility in psittacine birds. Proc Assoc Avian Vet, 2002, pp 133-137.
16. Crosta L, Burkle M: New perspectives in avicultural medicine including testicular biopsy. Exotic DVM 3(3):75-76, 2001.
17. Crosta L, Burkle M, Timossi L: Testicular biopsy in psittacine birds: technique and histological findings. Proc European AAV Conf, 2003, pp 113-116.
18. Dennis PM, et al: Diagnosis and treatment of tracheal obstruction in a cockatiel (*Nymphicus hollandicus*). J Avian Med Surg 13(4):275-278, 1999.
19. De Voe RS, Trogdon M, Flammer K: Diet modification and L-carnitine supplementation in lipomatous budgerigars. Proc Assoc Avian Vet, 2003, pp 161-163.
20. Diagle JC, et al: What Is Your Diagnosis? J Avian Med Surg 13(1):50-54, 1999.
21. Divers SJ, Stahl S: Diode laser surgery on birds. Proc Am Assoc Zoo Vet, 2001.
22. Doolen M: Crop biopsy-a low risk diagnosis for neuropathic gastric dilatation. Proc Assoc Avian Vet, 1994, pp 193-196.
23. Dvorak L, Bennett RA, Cranor K: Cloacotomy for excision of cloacal papillomas in a Catalina macaw. J Avian Med Surg 12(1):11-15, 1998.
24. Echols MS: Surgery of the avian reproductive tract. Semin Avian Exotic Pet Med 11(4):77-195, 2002.
25. Forbes NA: Avian gastrointestinal surgery. Semin Avian Exotic Pet Med 11(4):196-207, 2002.
26. Freeman KP, et al: Radiation therapy for hemangiosarcoma in a Budgerigar. J Avian Med Surg 13(1):40-44, 1999.
27. Gentz EJ, Linn KA: Use of a dorsal cervical single pedicle advancement flap in 3 birds with cranial skin defects. J Avian Med Surg 14(1):31-36, 2000.
28. Gerlach H: Viruses. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 887-888.
29. Gilsleider E: Ratite Orthopedics. Semin Avian Exotic Pet Med 3(2):81-91, 1994.
30. Gorham SL, Ottinger MA: Sertoli cell tumors in Japanese quail. Avian Dis 30:337-339, 1986.
31. Gorham SL, Akins M, Carter B: Ectopic egg yolk in the abdominal cavity of a cockatiel. Avian Dis 36:816-817, 1992.
32. Greenacre CB, Watson E, Ritchie BW: Choanal atresia in an African grey parrot (*Psittacus erithacus erithacus*) and an umbrella cockatoo (*Cacatua alba*). J Assoc Avian Vet 7:19-22, 1993.
33. Hannon DE, Weber TD: Use of a single pedicle advancement flap for wound repair in a great horned owl (*Bubo virginianus*).

Proc Assoc Avian Vet, 1995, pp 285-290.
34. Harcourt-Brown NH: Torsion and displacement of the oviduct as a cause of egg-binding in four psittacine birds. J Avian Med Surg 10(4):262-267, 1996.
35. Harcourt-Brown NH: Orthopedic conditions that affect the avian pelvic limb. Vet Clin N Amer Exotic Anim Pract 5(1):49-81, 2000.
36a. Harris D: Resolution of choanal atresia in African grey parrots. Exotic DVM 1:13-17, 1999.
36b. Harrison GJ: Microsurgical procedure for feather cyst removal in a citron-crested cockatoo (*Cacatura sulprurea citrinocristatala*) J. Assoc. Avain Vet 17(2): 86-90, 2003.
37. Hernandez-Divers SJ: Endoscopic diode laser surgery in birds. Exotic DVM, 3(3):73-74. 2001.
38. Hernandez-Divers SJ: Diode laser surgery: principles and application in exotic animals. Semin Avian Exotic Pet Med 11(4):208-220, 2002.
39. Hochleithner M, Hochleithner C: Surgical treatment of ulcerative lesions caused by automutilation of the sternum in psittacine birds. J Avian Med Surg 10(2):84-88, 1996.
40. IJzer J, Dorrestein GM, Van Der Hage MH: Metastatic subcutaneous sarcoma and abdominal carcinoma in a peach-faced lovebird (*Agapornis roseicollis*). Avian Pathol 31(1):101-104, 2002.
41. Jenkins JR: Postoperative care of the avian patient. Semin Avian Exotic Pet Med 2(2):97-102, 1993.
42. Jenkins JR: Surgery of the avian reproductive and gastrointestinal systems. Vet Clin North Amer Exotic Anim Pract 3:673-692, 2000.
43. Johnson AL: Reproduction in the female. *In* Whittow GC (ed): Sturkie's Avian Physiology 5th ed. San Diego, CA, Academic Press, 2000, 569-596.
44. Jones RG, Redig PT: Endoscopy guided vasectomy in the immature Japanese quail (*Coturnix coturnix japonica*). Proc European AAV Conf, 2003, pp 117-123.
45. King AS, McLelland J: Female reproductive system. *In* King AS, McLelland J (eds): Birds: Their Structure and Function 2nd ed. Philadelphia, PA, Bailliere Tindall, 1984, pp 145-165.
46. King AS, McLelland J: Male reproductive system. *In* King AS, McLelland J (eds): Birds: Their Structure and Function 2nd ed. Philadelphia, PA, Bailliere Tindall, 1984, pp 166-174.
47. King AS, McLelland J: Choana and vent. Birds: Their Structure and Function, Bailliere Tindall, London, 1984, pp 187-198.
48. Kirby JD, Froman DP: Reproduction in males. *In* Whittow GC (ed): Sturkie's Avian Physiology 5th ed. San Diego, CA, Academic Press, 2000, pp 597-615.
49. Kramer MH, Harris DJ: Ventral midline approach to avian salpingohysterectomy. Exotic DVM, 4(4):23-27, 2002.
50. Langlois I, Jones MP: Ventral abdominal hernia associated with hepatic lipidosis in a red lory (*Eos bornea*). J Avian Med Surg, 15:3, 216-222.
51. Latimer KS: Oncology. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Publishing, 1994, pp 640-672.
52. Lea RW, Richard-Yris MA, Sharp PJ: The effect of ovariectomy on concentration of plasma prolactin and LH and parental behavior in the domestic fowl. Gen Comp Endocrinol 101:115-121, 1996.
53. MacWhirter P: A review of 60 cases of abdominal hernias in birds. Proc Annu Conf Assoc Avian Vet 1994, pp 27-37.
54. Mader DR: The use of lasers in exotic animal surgery. Exotic DVM, 3(3):70-72, 2001.
55. Mallay AD, Whitbread TJ: The integument. *In* Benyon PH, Forbes NA, Harcourt-Brown, NH (eds): Manual of Raptors, Pigeons, and Waterfowl. Cheltenham, British Small Animal Veterinary Association, 1996:129-139.
56. Martin HD: Abdominal hernia with formation of a urate concretion in a cockatiel. J Amer Vet Med Assoc 18(9):1332-1333, 1986.
57. McCluggage DM: Surgery of the integument-selected topics. Semin Avian Exotic Pet Med 2(2):76-82, 1993.
58. Olsen GH: Orthopedics in Cranes: pediatrics and adults. Semin Avian Exotic Pet Med 1994, 3(2):73-80, 1994.
59. Orosz S: Anatomy of the urogenital system. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, PA, WB Saunders Co, 1997, pp 614-622.
60. Orr MG: Avian castration technique without testicular artery/vein ligation. Proc Assoc Avian Vet, 1994, pp 23-26.
61. Parrott-Nenezian T: Using the diode laser in avian endoscopic surgery. Proc Assoc Avian Vet, 2000, pp 249-251.
62. Pavletic MM: Skin flaps: classification and techniques. Proc Annu Conf CO Vet Med Assoc. 1989, 50:137-138.
63. Pavletic MM: Atlas of Small Animal Reconstructive Surgery. Philadelphia, PA, JB Lippincott, 1993.
64. Pavletic MM: Pedicle Grafts. *In* Slatter D (ed): Textbook of Small Animal Surgery. Philadelphia, PA, WB Saunders Co, 1993, pp 295-325.
65. Peacock EE: Wound Repair 3rd ed. Philadelphia, PA, WB Saunders Co, 1984.
66. Petrowski MI, Wong EA, Ishii S: Influence of ovariectomy and photostimulation on luteinizing hormone in the domestic turkey: evidence for differential regulation of gene expression and hormone secretion. Biol Reprod 1993, 49:295-299.
67. Phalen DN, Mitchell ME, Cavazos-Martinez ML: Evaluation of three heat sources for their ability to maintain core body temperature in the anesthetized avian patient. J Avian Med Surg 1996, 10(3):174-178.
68. Phalen DN: Viruses. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, PA, WB Saunders Co, 1997, pp 309-312.
69. Polanyi TG: Physics of the surgical laser. Adv Surg Oncol 1:205-215, 1978.
70. Proudman JA, Opel H: Daily changes in plasma prolactin, corticosterone, and luteinizing hormone in the unrestrained, ovariectomized hen. Poult Sci 68:177-184, 1989.
71. Pye GW: Surgery of the avian respiratory system. Vet Clin North Amer Exotic Anim Pract 3:693-714, 2000.
72. Pye GW, et al: Endoscopic salpingohysterectomy of juvenile cockatiels (*Nymphicus hollandicus*). J Avian Med Surg 15(2):90-94, 2001.
73. Rich GA: Surgery of the head. Semin Avian Exotic Pet Med, 2(2):69-75, 1993.
74. Robins PK, Pokras M: Congenital anomalies in a mourning dove (*Zenaida macroura*). J Avian Med Surg 9(3):182-184, 1995.
75. Rosenthal KL, Johnston M: Hypothyroidism in a red-lored Amazon (*Amazona autumnalis*). Proc Assoc Avian Vet, 2003, pp 33-36.
76. Rosskopf WJ, Woerpel RW: Soft tissue surgery. *In* Rosskopf WJ, Woerpel RW (eds): Diseases of Cage and Aviary Birds. Baltimore, MD, Williams & Wilkins, 1996, pp 675-693.
77. Samour JH, Markham JA: Vasectomy in budgerigars (*Melopsittacus undulatus*). Vet Rec 120:115, 1987.
78. Speer BL: Chronic partial proventricular obstruction caused by multiple gastrointestinal foreign bodies in a juvenile umbrella cockatoo (*Cacatua alba*). J Avian Med Surg 12(4):271-275, 1998.
79. Stanford M: Use of F10 in psittacines. Exotic DVM 3(4):18, 2001.
80. Suedmeyer WK, Bermudez A: A new approach to renal biopsy in birds. J Avian Med Surg 10(3):179-186, 1996.
81. Terada O, Shimada K, Saito N: Effect of oestradiol replacement in ovariectomized chickens on pituitary LH concentrations of mRNA encoding LH beta and alpha subunits. J Reprod Fertil 111:59-64, 1997.
82. Tully TN, et al: Liposarcomas in a monk parakeet (*Myiopsitta monachus*). J Assoc Avian Vet 8(3):120-124, 1994.
83. Turk JR, Kim J, Gallina AM: Seminoma in a pigeon. Avian Dis 25:752-755, 1981.
84. Westerhof I: Treatment of tracheal obstruction in psittacine birds using a suction technique: a retrospective study of 19 birds. J Avian Med Surg 9(1):45-49, 1995.
85. Wilson H, et al: Surgical applications of Cavitron ultrasonic surgical aspirator (CUSA) in birds. Proc Assoc Avian Vet, 2002, pp 29-31.
86. Zadworny D, Etches RJ: Effects of ovariectomy or force feeding on the plasma concentrations of prolactin and luteinizing hormone in incubating turkey hens. Biol Reprod 36:81-88, 1987.
87. Zantop DW, Bowles HL: Evaluating avian patients with the parks Doppler flow monitor. Exotic DVM 2(2):44-45, 2000.

CHAPTER
36

Management of Waterfowl

GWEN B. FLINCHUM, BS, MS, DVM

The order Anseriformes includes swans, ducks, geese and screamers. Numerous excellent references are available for specific medical and biological information on these birds.[1,9,11,12,13] Some basic biological facts of which veterinarians should be aware are discussed in the following sections.

Adult male ducks, geese and swans are referred to as drakes, ganders and cobs, respectively. Adult female ducks, geese and swans are called ducks, geese and pens. Young ducks, geese and swans (older than 3 weeks) are referred to as ducklings, goslings and cygnets.

FEATHER CHARACTERISTICS

Molting

Most waterfowl go through a complete molt following breeding season. This molt may last 3 to 6 weeks. During this time, flight feathers are lost, thus the birds are flightless. Most members of the subfamily Anatinae (eg, Shelducks, dabbling ducks, perching ducks, diving ducks) molt twice yearly. Birds of the family Anhimidae (screamers) and the magpie goose (*Anseranas semipalmata*) molt gradually and do not pass through a flightless period.

Feather Color and Sexual Dimorphism

Birds of subfamily Anserinae, ie, whistling ducks, swans, geese, Cape Barren geese (*Cereopsis novaehollandiae*) and freckled ducks (*Stictonetta naevosa*), have plumage that is monomorphic in all species. Magellan geese (*Chloephaga picta*) and kelp geese (*Cloephaga hybrida*) are the exceptions. Greater Magellan ganders have a white head, neck and breast, while females are reddish brown. Kelp geese males are pure white, while females are striped with different hues of brown. Birds of the subfamily Anatinae frequently have dimorphic plumage. Males typically have iridescent coloration with outstanding patterns. Ducks are usually sexually dimorphic

Fig 36.1 | A fence separates two sides of the aviary so birds are able to fly over the fence into the back of the aviary to escape other more aggressive birds.

Fig 36.2 | Back side of a two-sided aviary into which threatened birds may fly.

except for the Pekin, American black duck (*Anas rubripes*) and Mexican breeds. It is a hobbyist's theory that male ducks have a curl to their tail feathers at maturity that is not present in females.

ANATOMIC VARIATIONS

Trachea

Swans have an elongated trachea. The trachea of trumpeter swans extends into the sternum, turns on itself and then re-enters the syrinx. The ruddy duck (*Oxyura jamaicensis*) has an inflatable tracheal sac.

Syringeal Bulla

Most male ducks have a left-sided enlargement that can be visualized on radiographs.[10] This is called the syringeal bulla and should not be misinterpreted as pathologic. This structure is absent in swans and geese.

Phallus

Male Anseriformes have an erect phallus that is covered with papillae. By placing gentle pressure on the sides of the cloaca, the phallus can be exteriorized, thus determining the bird's sex as male. Females lack a phallus and instead have two small labia-like structures. Because this type of sexing necessitates turning the bird upside down, geese and swans are most easily done at a young age.

Husbandry

ENVIRONMENTAL/ENCLOSURE CONSIDERATIONS

Enclosed Versus Open Ponds

There are numerous advantages to keeping waterfowl in enclosed areas. The greatest advantage is protection from outside predators. In addition, it is more difficult for wild birds to enter enclosed areas, and this decreases the chances of introduction of disease into the collection. Another advantage is that birds in enclosed areas do not have to be pinioned. A disadvantage of enclosed areas is that the overall area is smaller than open enclosures, and this may lead to territorial aggression, harassment and injury to smaller birds by more dominant birds. This can be avoided by providing a tiered enclosure in which smaller, less dominant birds can fly up to the second level to escape harassment. It is important to avoid overcrowding in aviaries (Figs 36.1, 36.2).

Nylon netting[a,b,c] works well to enclose pens. In colder areas, the netting will be weighted down with snow or ice, which will cause damage to the netting. This can be corrected by having the netting attached to several wires (Fig 36.3). The wires are then connected to a hand crank. Loosening the netting with the crank alleviates tension, so that damage to the netting is less likely (Lubbock, personal communication[d]).

Open ponds provide more space and greater opportunity for grazing. However, birds in open areas are susceptible to predation, especially if they are unable to fly. In some cases, predation can be avoided by enclosing the area with a tall fence lined with electric wire. Furthermore, larger species such as mute swans can be very aggressive and are thus less likely to be attacked.

Protection from vermin also is an important consideration. Poisonous bait for rats, mice or roaches can be placed inside long plastic or metal tubes and put outside the reach of birds. However, there have been reports of warfarin toxicity resulting from the birds' ingestion of mouse droppings (Lubbock, personal communication).

Fig 36.3 | Nets with cable support.

Fig 36.4 | Skirting can be placed around the bottom perimeter of the cage to minimize rodent invasion.

Figs 36.5-36.7 | Various types of houses can be used for waterfowl.

In general, extreme care must be taken when using rodenticides or other poisons near an aviary. Other deterrents for vermin include a 2 to 3 foot (1 m) tall sheet of metal lined with electric wire at the bottom, placed around the bottom perimeter of the cage (Fig 36.4).

Smaller waterfowl can be provided with various types of houses for nesting (Figs 36.5, 36.6). These can be made from large, hollowed-out logs (Fig 36.7) or flat pieces of wood. One breeder has had success using slatted boxes (Fig 36.8) because small waterfowl normally build nests in reeds or grasses, and the staggered pieces of wood in slatted boxes simulate stalks of grasses or reeds. Apparently, this imparts a feeling of privacy and security for birds, thus facilitating nesting (Lubbock, personal communication[d]).

Ground cover is an important aspect of enclosure design. Hard surfaces such as concrete should be avoided, as these can contribute to bumblefoot. Grass or dirt surfaces are less irritating to feet, although they are harder to keep clean. A small (20 x 20 feet or 7.5 x 7.5 m) swan enclosure requires twice monthly re-sodding to keep the environment ideal (Montgomery, personal communication[e]).

Fig 36.8 | Slatted houses emulate reeds or grasses and impart a sense of privacy for nesting waterfowl.

Water Quality

In small enclosures, artificial ponds can be built. These are made of concrete, then painted with waterproof

Fig 36.9 | Small concrete pools are easy to clean and maintain.

Fig 36.10 | Swans and diving ducks require ponds 3–4 feet deep.

Fig 36.11 | Water aerator for a large pond.

Fig 36.12 | Reeds and water plants can be useful in water filtration.

epoxy pool paint (Fig 36.9). These types of pools can be drained and cleaned on a regular basis. A water depth of 2 feet is adequate for most Anseriformes. Swans and diving ducks require 3 to 4 feet of water (Fig 36.10). Water quality in larger ponds is affected by the amount of ammonia present. Ammonia forms near the bottom surface of the pond subsequent to the degradation of fecal matter, food and other organic substances. It then rises to the surface of the pond where it facilitates algae growth. Above-water aerator systems can be utilized to remove ammonia before it reaches the pond surface. A network of underwater pipes that is connected to a central fountain achieves this (Fig 36.11). The pipes carry water (and thus ammonia) from the bottom surface area to the fountain where it is discharged into the air. This eliminates ammonia and oxygenates the water, which results in cleaner, fresher water with less algae.

Filtration systems also can be utilized to maintain water quality. There are hundreds of systems available and many of these can be researched on the Internet.[f] The majority of these are for smaller ponds, with 25,000-gallon capacities being maximum size. Submersible filters are very effective in maintaining water quality. However, care must be taken when using these, as there have been reports of birds being pulled under water and trapped in the filtration system. Reeds and water plants can act as natural water filters when placed at exit and entry points of water flow (Fig 36.12). If water quality becomes out of balance—usually too much nitrogen and phosphorous combined with warm weather and bright sunlight—algae will bloom. A dark blue coloring agent[g] that blocks the light so algae cannot grow is available.

Species Compatibility

In general, the smaller the enclosure area, the more potential there is for territorial aggression. As previously mentioned, tiered cages will allow smaller species to fly away from more aggressive birds. Inbreeding can occur and should be avoided. Likewise, interspecies breeding is undesirable and can be avoided by not having many different species of geese or ducks in one pen. Table 36.1 lists waterfowl species that should not be mixed.

Table 36.1 | Waterfowl Species That Should Not Be Housed Together

Keep Isolated as a Single Pair	Exceptions	Specific Non-mix Combinations
Swans, especially: • Coscoroba	• Two pairs of black swans may be kept together if the area is large enough, as long as they are released together. • Never release a young pair into the territory of an established pair.	• Even on large lakes, never mix two pairs of trumpeter swans. • Never mix a pair of Bewick's swans with a pair of whistling swans.
Ducks, especially: • Screamer • Bronze-winged • Pink-eared • Hartlaub's • Comb • Shelducks • White-winged wood • Musk • Crested • New Zealand teals (brown teals or brown ducks)	• More than one pair of comb ducks and white-winged wood ducks can be released together if the area is large, but do not release new birds into an existing group. • Never release a young pair into the territory of an established pair.	• Avoid keeping any subspecies together.
Geese, especially: • Sheldgeese (Andean) • Egyptian • Cereopsis geese • Spur-winged	• Never release a young pair into the territory of an established pair.	• Hawaiian geese (Néné) and cackling Canada geese. • Avoid keeping any subspecies together.

Adapted from BSAVA Manual of Raptors, Pigeons, and Waterfowl, edited by Peter H. Beynon, Neil A. Forbes and Nigel H. Harcourt-Brown (1996) with permission of BSAVA.

Fig 36.13 | Royal mute swans (*Cygnus olor*).

Fig 36.14 | Black swans (*Cygnus atratus*).

Species Considerations for a Collection

Choosing which waterfowl to have in a collection depends on a number of considerations. These include price and availability of birds, aggression quotient (ie, larger birds such as swans can be quite aggressive during mating season), susceptibility to predation (smaller, pinioned birds are more likely to be attacked) and size of enclosure and water quality. Figs 36.13-36.18 show some commonly kept swans.

Banding Birds

Once birds are acquired, placing leg bands is recommended, especially if there are two or more birds. This helps identify birds for breeding and health monitoring. Bands that are placed on the upper part of the leg (on the tibiotarsus) have a lesser chance of getting caught and causing injury (Fig 36.19). In addition, bands can be placed on the left leg of females and the right leg of males for quicker sex identification. Plastic leg bands have an advantage over aluminum because they are expandable (Fig 36.20). Aluminum bands can become bent, thus placing pressure on the leg or causing constrictions. Metal leg bands increase the incidence of frostbite injury in cold climates.

Diet

There are several commercially formulated diets available for waterfowl.[h,i,j] These simplify meeting growing, maintenance and breeding nutritional requirements. Waterfowl kept on corn and lettuce diets frequently have dietary deficiencies that manifest as joint pain/lameness and bumblefoot. Grass and plants should be available for foraging.

Fig 36.15 | Whooper swan (*Cygnus cygnus*).

Fig 36.16 | Black-necked swans (*Cygnus melanocoryphus*).

Fig 36.17 | Coscoroba swans (*Coscoroba coscoroba*).

Fig 36.18 | Trumpeter swan (*Cygnus buccinator*).

Fig 36.19 | Bands placed on the upper part of the leg have less chance of becoming entrapped.

Fig 36.20 | Plastic leg bands are suitable for identification of waterfowl.

Fig 36.21 | A 50-foot ski rope can be wound on a reel to be used for swan capture.

Fig 36.22 | Rope-across-pond method of capturing swans: one person stands on each side of the pond holding an end of the rope.

Fig 36.23 | The rope is stretched across the pond and moved toward the swans.

Fig 36.24 | As the rope is moved toward the swans, they are herded in the desired direction for capture.

Management of Patients

CAPTURE AND RESTRAINT

Capturing pinioned waterfowl for examination in an open environment can be challenging. Most are excellent swimmers but are not proficient runners; therefore, a primary goal is to manipulate the birds out of the water so they can be more easily captured on land. This can be accomplished in a number of ways.

Capture Methods

Rope-across-pond Method

This method (Figs 36.21-36.23) involves pulling a long rope across the diameter of the pond. The birds attempt to swim away from the rope and can be herded up onto shore (Fig 36.24). A 50-foot (yellow) nylon ski rope can be purchased from a hardware or boating store. This can be wound upon a reel (such as that used to wind an electrical extension cord) for easy access (see Fig 36.21). If a longer length is needed, two cords can be spliced together. A disadvantage of this method is that birds will quickly overcome their fear of the rope and learn to swim under or over the rope to avoid capture.

Net Restraint

Pole nets[k] can be used to capture waterfowl (Fig 36.25). This method actually works well for capturing swans and geese from a small boat.

Throw Nets

Circular nets with a weighted outer perimeter[l] can be thrown over birds (Fig 36.26). These are best used on land or in very shallow water. Throw nets should be inspected prior to use and damaged weights removed to avoid lead weights falling off in the pond.

Manual Capture

Swans and geese can be grasped gently but firmly by the base of the neck, then covered with a towel to prevent injury from the wings to the handler. Both large and

Fig 36.25 | A pole net can be used for capturing waterfowl.

Fig 36.26 | Circular throw nets can be used for capture on land.

Fig 36.27 | Waterfowl can be restrained by grasping the humerus on each wing at the shoulder. Such restraint applied on the lower wing can result in injury.

Fig 36.28 | Large waterfowl should be carried under the arm with the head facing backward. Additionally, the head can be covered to avoid bites.

small waterfowl can be restrained by holding the base of the wings (Fig 36.27). Smaller birds can be carried in this manner; however, larger birds should be carried under one arm with the head facing backward and the body and feet supported with the other hand (Fig 36.28).

Restraint for Travel

Birds can be wrapped in a towel or pillowcase (Fig 36.29), then encircled with cohesive flexible bandage material.[m] Caution should be taken so that the bird is not wrapped too tightly. In addition, birds should be monitored for overheating in hot weather.

Drug Immobilization

Chemical immobilization has been reported for use in waterfowl; however, this has not proven to be an effective adjunct to capture for many reasons.[9] Drugged birds may go into the water and subsequently drown. When oral agents are mixed with food, over-consumption may cause an overdose with subsequent death. In addition, recovery from such drugs may be prolonged and may take up to 8 hours.

Fig 36.29 | Large waterfowl can be wrapped in a pillowcase to restrain for travel.

Fig 36.30 | Feathers of a healthy swan.

Fig 36.31 | Frayed, dirty feathers of an unhealthy bird.

Fig 36.32 | Bumblefoot in a mute swan.

Fig 36.33 | The feet of healthy swans have no cracked areas or lesions.

Dangers to Handlers During Capture and Restraint of Swans and Geese

Wings

Swans and geese use wings defensively. The distal humerus and olecranon can cause contusions. It is important to avoid having the handler's head anywhere near the wings, as serious injury can occur. This is true with any large bird, but especially dangerous when handling the spur-winged goose.

Toenails

Waterfowl have short but very sharp toenails. These can cause painful scratches.

Beak

Most waterfowl have serrations on the edges of their beaks. They are capable of causing bruises when they bite or pinch. Handlers also must beware of the potential for an eye injury from biting birds.

PHYSICAL EXAMINATION

Feather Quality

Feathers should be smooth and regular in appearance (Fig 36.30). Frayed, dirty, bent or broken feathers may indicate lack of preening or abnormal molting (Fig 36.31). Water should roll off normal feathers, and feathers should not have a wet appearance. Feather color should be consistent, ie, white feathers should be white, not brown or dirty.

Skin and Foot Quality

The skin is best examined over the chest muscle and on the feet and legs. Skin should not be cracked or flaky. The bottoms of the feet should be examined for bumblefoot lesions (Fig 36.32). Ideally, the bottoms of the feet should have small patterns of scale with no balding areas or scabs (Fig 36.33). Limping is generally a sign of joint or foot problems.

Body Weight

General body weights for waterfowl are available in table form.[9] Body weight should be recorded at every physical exam. Deviations from previously recorded weights may indicate disease. Many free-ranging waterfowl have palpable keel bones. A prominent keel bone indicates

excessive weight loss with probable disease. Large birds can be easily weighed using a human scale; obtain the holder's weight and then hold the bird, subtracting the difference of the two weights. This is an ideal way to weigh larger species.

Eyes, Nose, Mouth

Eyes should be clear with no redness or discharge. In collections where inbreeding is allowed, ocular abnormalities may be seen (corneal or ocular opacities). The nares should have no discharge and be bilaterally symmetrical. The oral cavity should be examined and should be free of excessive redness, white plaques or brown mucous. Breathing should not be open-mouthed or labored. Auscultation of lungs and air sacs should reveal little or no sound. Wheezing or crackling may indicate respiratory illness.

Feces

It is not unusual for waterfowl to have some loose feces, however, feces should not have a foul odor. Fecal color should be brown but will depend on diet. Excessively green feces, urine and urates may indicate liver disease.

Overall Behavior

Sick birds will sequester themselves away from other birds. Often, sick birds will be at the bottom of the pecking order, so their feathers will be more dirty, plucked and ragged than those of other birds. Another indication of illness is reluctance get up and move around. Sick birds spend inordinate amounts of time lying down or sleeping. Male swans will harass female and juvenile birds in an attempt to mate. If not interrupted, some infirm birds have been drowned by aggressive males.

TESTING RECOMMENDATIONS

Blood Testing

The recommendations for blood tests in waterfowl are similar to those commonly performed in other birds and include the following: complete blood count, advanced serum chemistry panel with bile acids, serum protein electrophoresis, chlamydial PCR testing and aspergillosis serology. Chlamydiosis serology has not been rewarding in waterfowl.

Blood Collection

The safest place for blood collection is the medial metatarsal vein (Fig 36.34). The cutaneous ulnar vein also can be used but there are more problems associated with using this vein. For example, birds do not like having the wing restrained and tend to struggle more during wing venipuncture. This can lead to head injury to the handler. Furthermore, hematoma formation is more

Fig 36.34 | Blood is easily collected from the medial metatarsal vein.

Table 36.2 | Hematology and Serum Chemistry Values for Waterfowl

Species	White-winged Wood Duck	Hawaiian Goose	Canada Goose	Swan
	n = 30	n = 10	n = 15	n = 50
RBC ($x10^{12}$/l)	2.6-3.48	2.35-2.89	2.25-3.35	1.96-2.9
PCV (l/l)	0.46-0.57	0.38-0.45	0.35-0.49	0.32-0.5
Hb (g/l)	122-181	129-170	122-172	110-165
MCV (fl)	163-177	156-161	162-178	164-200
MCH (pg)	46.6-51.9	54.9-59.3	47-58.7	52.9-65.5
MCHC (g/l)	270-321	340-380	342-363	290-365
WBC ($x10^9$/l)	4.7-9.4	6.2-13.4	3.0-5.15	6.3-22.0
Heterophils ($x10^9$/l)	2.7-5.6	0-5.57	0.5-2.7	3.33-14.67
Lymphocytes ($x10^9$/l)	0.65-4	0-7.74	0.8-3.8	0.9-9.77
Monocytes ($x10^9$/l)	0.15-0.76	0-0.28	0.15-0.8	0.05-1.39
Eosinophils ($x10^9$/l)	0-0.3	0-0.6	0-0.5	0.11-3.5
Basophils ($x10^9$/l)	0.1-0.09	0-0.6	0-0.25	0-0.82
Thrombocytes ($x10^9$/l)	—	—	—	—
Fibrinogen (g/l)	—	<3.5	—	—
	n = 18		n = 10	n = 50
Total protein (g/l)	34-54	—	37.3-56.3	35.5-54.5
Albumin (g/l)	10-25	—	17.5-23.6	12.0-21.5
Globulin (g/l)	26.4-29.41	—	20.3-42.6	23.0-35.5
A:G ratio	—	—	—	0.43-0.65
Urea (mmol/l)	0.76-1.05	—	0.8-3.56	0.1-2.4
Creatinine (umol/l)	6-14	—	4-11	18-89
Uric acid (umol/l)	165-691	—	—	126-700
Bile acids (umol/l)	—	—	—	—
ALT (SGPT) (u/l)	0-67.5	—	—	10.59
ALP (u/l)	0-198	—	0-149	—
GGT (u/l)	0-14	—	1-10.5	4.26
AST (SGOT) (u/l)	9.8-43.2	—	—	17-112
CK (u/l)	—	—	—	124-894
LDH (u/l)	—	—	145-435	165-724
Glucose (mmol/l)	8.0-13.4	—	—	6.2-12.6
Cholesterol (mmol/l)	—	—	—	3.0-7.8
Inorg phosphate (mmol/l)	0.55-1.66	—	—	0.7-2.36
Calcium (mmol/l)	2.01-2.52	—	—	2.19-2.89
Sodium (mmol/l)	—	—	—	132-150
Potassium (mmol/l)	—	—	3.9-4.7	3-5
Chloride (mmol/l)	—	—	101-133	—

Reproduced from BSAVA Manual of Raptors, Pigeons, and Waterfowl, edited by Peter H. Beynon, Neil A. Forbes and Nigel H. Harcourt-Brown (1996) with permission of BSAVA.

likely when using the cutaneous ulnar vein. The jugular vein is not commonly used for blood collection due to difficulty in visualization of the vein, especially in long-necked birds.

Blood Parameters

Hematology and serum chemistry values for waterfowl are listed in Table 36.2.

Fecal Tests

Direct fecal smears are a simple way to check for internal parasites. This is easily done by smearing a small amount of fresh feces on a microscope slide, adding a few drops of lactated Ringer's solution, adding a cover slip and examining under a microscope. *Giardia* spp. or other protozoan organisms as well as parasite eggs can be visualized on direct fecal smears. Fecal Gram's stains can be used to identify *Cryptosporidia* spp. and budding yeast. Gram-negative bacteria and *Clostridia* spp. are not unusual in Gram's stains of asymptomatic waterfowl and should not necessarily be treated.

Endoscopy

Endoscopy is routinely performed via the left or right lateral approach as with parrots. Because swans and geese have elongated tracheas, tracheal endoscopy is difficult, since few endoscopes will reach far enough to visualize the trachea in its entirety. A flexible endoscope can be used, as the trachea of most birds over 1000 g will pass a 3-mm scope.

Other recommended diagnostic tests include radiology, histopathology, necropsy and culture and sensitivity of feces, throat or skin lesions.

TREATMENT RECOMMENDATIONS

Fluid Therapy

Intravenous catheterization can be most easily done in the medial metatarsal vein. This vein is easy to access and not highly prone to hematomas, and birds tolerate catheter placement well there. The biggest disadvantage of using the medial metatarsal vein is that some agents injected into the legs will go through the renal portal system and be excreted by kidney tubules before entering the general circulation.[8] However, the author has experienced successful treatment results following fluid and antibiotic therapy administered via this method. Catheter placement is implemented in the same manner as with dogs and cats. A 20- to 24-gauge Teflon catheter can be used. It is recommended that catheters not be left in for more than 48 hours. If long-term catheterization is necessary, a vascular access device may be necessary.[6] Catheterization also may be done in the cutaneous ulnar vein; however, as previously mentioned, there is a greater possibility of hematoma, and this is more dangerous to the handler in larger birds. Jugular catheterization is difficult due to visualization difficulties discussed earlier.

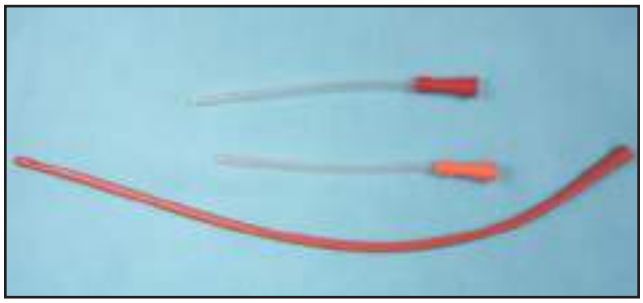


Fig 36.35 | Red rubber catheters or silicone feeding tubes can be used for gavage-feeding waterfowl.

Intraosseous catheterization can be performed in the tibiotarsus or ulna but is more painful than intravenous catheterization. Such catheter placement often necessitates anesthesia, and some patients may not be candidates for anesthesia. Bone infection may occur if the process is not done aseptically.

If a patient is not hypoproteinemic, subcutaneous fluids with hyaluronidase[n] have been preliminarily purposed to be as effective as intravenous fluids.[5]

Oral fluids should also be given in tube-feedings in mildly dehydrated birds.

Gavage-Feeding

Soft tubes work well for gavage feeding waterfowl. Swans and geese can be gavage fed with 14-Fr (16") red rubber catheters.[o,p] Smaller birds can be fed using silicone feeding tubes[o,p] (Fig 36.35). There are a variety of formulas available for nutritional support in debilitated patients.[q]

Medications

Table 36.3 lists medications commonly used for waterfowl.

SURGICAL PROCEDURES

Pinioning

Pinioning, the surgical removal of the tip of the wing from the alula distally to render a bird flightless, is a common procedure in waterfowl. When done early (at 2-3 days old), this procedure is virtually bloodless and stress free (Figs 36.36-36.38). Pinioning older birds is more difficult because stress and excessive bleeding can occur. The proper technique for pinioning is to remove metacarpals III and IV and leave the alula intact to cover the amputated area. If the alula is removed, repeated trauma to the stump can occur on a regular basis.

Table 36.3 | Medications Commonly Used for Waterfowl

Generic	Trade Name(s) and Manufacturer	Dosage(s) and Route(s)	Main Indications
ANTIBACTERIAL AGENTS			
Amoxicillin	Amoxinsol 50 soluble powder (Univet)	1 g/3 L drinking water* Medicated drinking water should be provided on alternate days for 3 days, ie, 2 days of medication.	Sensitive bacterial infections
Chlortetracycline	Aureomycin soluble powder (Cyanamid)	1000 ppm (18.2 g/kg feed) in feed for 45 days	Chlamydiosis
Co-trimazine (trimethoprim + sulfadiazine)	Cosumix Plus soluble powder (Ciba); Duphatrim Poultry Suspension (Solvay Animal Health) (*Bactrin, Roche*)	1 ml/5 L drinking water* for 5-7 days	Sensitive bacterial infections
Doxycycline	Ronaxan tablets (Rhône Mérieux) (Henry Schein, Roerig)	50 mg/kg PO BID for 3-5 days (45 days for chlamydiosis) or 240 ppm in feed for 45 days	Sensitive bacterial infections, especially chlamydiosis
	Steriject (Pfizer)	75 mg/kg IM once weekly for 6 weeks	Chlamydiosis
Enrofloxacin	Baytril 2.5% or 5% injection 2.5% or 10% oral solution or tablets (Bayer) (*Baytril 2.7%, Haver/Diamond*)	10-15 mg/kg IM or PO BID for 5-7 days	Sensitive bacterial infections. Useful for bacterial hepatitis or septicemia in neonates. Used widely in growing chickens and poultry of all ages without any incidence of articular cartilage problems: at normal therapeutic levels (10-15 mg/kg BID) it is unlikely to produce joint deformity in neonatal waterfowl (or in raptors or pigeons).
		4 mg in 20 ml saline for a 1-kg bird – daily nasal flushing for 10 days	Treatment of sinusitis
Lincomycin	Lincocin soluble powder (Upjohn)	10 g/5 L drinking water* for 5-7 days	Pasteurellosis, mycoplasmal tenosynovitis
Lincomycin/spectinomycin	Linco-Spectin 100 soluble powder (Upjohn)	3 g/4 L drinking water* for 3-7 days	Mycoplasmal tenosynovitis, sinusitis
Oxytetracycline	Various long-acting injections.	200 mg/kg IM daily for 5-7 days	Pasteurellosis and other sensitive bacterial infections
	Terramycin soluble powder (Pfizer)	37 g/15 L drinking water* for 5-7 days	
Tylosin	Tylan 50 or 200 injection (Elanco) (Butler)	20-30 mg/kg IM TID for 3-7 days; or 100 mg in 10 ml saline, daily nasal flush for 10 days.	Mycoplasmosis
	Tylan tablets (Elanco)	20 mg/kg PO TID for 3 days	
	Tylan soluble powder (Elanco)	2.5 g/5 L drinking water* for 3 days	
ANTIFUNGAL AGENTS			
Itraconazole	Sporanox capsules (Janssen)	10 mg/kg PO SID for 7-10 days for prophylaxis, or BID for 4-6 weeks for therapy	Aspergillosis
Nystatin	Nystan oral suspension (Lagap) (*Myco 20, Squibb*)	300,000 units (3 ml)/kg PO BID for 7 days	Candidiasis
ANTIPROTOZOAL AGENTS			
Clazuril	Appertex (Harkers)	5-10 mg/kg PO every 3rd day on 3 occasions	Coccidiosis
Co-trimazine (trimethoprim + sulfadiazine)	Cosumix Plus soluble powder (Ciba); Duphatrim poultry suspension (Solvay Animal Health) (Bactrin, Roche)	60 mg/kg (combined constituents) PO BID, 3 days on, 2 days off, 3 days on	Coccidiosis Do not use in dehydrated birds
	Duphatrim 24% injection (Solvay Duphar)	30 mg/kg SC, 3 days on, 2 days off, 3 days on	
Pyrimethamine	Daraprim (Glaxo-Wellcome)	0.25-0.5 mg/kg PO BID for 30 days	*Sarcocystis* spp., toxoplasmosis
Pyrimethamine/ sulfaquinoxaline	Microquinox (C-Vet Livestock Products)	60 mg/L drinking water*, 3 days on, 2 days off, 3 days on	Coccidiosis
Toltrazuril	Baycox (Bayer) (Bayvet)	1 ml of 2.5% solution/2 L drinking water* for 48 hours	Coccidiosis
ENDOPARASITICIDES			
Chlorsulon	Curatrem (MSD Agvet)	20 mg/kg PO 3 times at 2-week intervals	Control cestodes and trematodes
Fenbendazole	Panacur 2.5% or 10% liquid, 8-mg capsules (Hoechst)	20 mg/kg PO once	Control nematodes
Flubendazole	Flubenvet (Janssen)	240 ppm (2.4 kg/ton) in feed for 7 days	Control nematodes
Ivermectin	Ivomec 1% cattle injection (MSD Agvet)	200 μg/kg SC or PO once	Control nematodes and nasal or duck leeches
Levamisole	Various, eg, Levacide (Norbrook) (*Ripercol-L, American Cyanamid*)	25-50 mg/kg SC once	Control nematodes

Table 36.3 | Medications Commonly Used for Waterfowl (Continued)

Generic	Trade Name(s) and Manufacturer	Dosage(s) and Route(s)	Main Indications
ENDOPARASITICIDES (continued)			
Mebendazole	Mebenvet (Janssen) (*Telmin, Pitman-Moore*)	5-15 mg/kg PO daily for 2 days	Control *Syngamus* trachea
		120 ppm (1.2 g/ton) in feed for 14 days	Control nematodes
Praziquantel	Droncit (Bayer) (*Bayvet*)	10-20 mg/kg SC or PO once. Repeat after 10 days	Control cestodes
		10 mg/kg SC or PO daily for 14 days	Control trematodes
MISCELLANEOUS			
Atropine	Atropine injection (C-Vet)	0.1 mg/kg IV or IM every 3-4 hours	Anticholinesterase poisoning, eg, carbamate
D-penicillamine	Distamine (Dista) (*Cupramine, Merck; Depen, Wallace; Titratabs, Wallace*)	55 mg/kg PO BID for 7-14 days	Heavy metal poisoning
Dexamethasone	Dexafort (Upjohn)	2 mg/kg SID for 2 days only	Treatment of shock, or anti-inflammatory
Diazepam	Valium (Roche)	0.5-1.0 mg/kg IV or IM, BID or TID, as required	Control of fits
Dinoprost tromethamine (PGF 2, alpha), $PGE_{1 or 2}$	Lutalyse (Upjohn)	0.02-0.1 mg/kg IM or topically onto cloacal mucosa, once	Egg binding
Doxapram	Dopram injection (Willows Francis) (*Robins*)	10 mg/kg IV once	Respiratory stimulant
Iron dextran	Vet Iron injection (Animalcare) (*Butler, Lextron, Vedco*)	10 mg/kg IM. Repeat in 1 week	Anemia
Ketoprofen	Ketofen (Rhône Mérieux) (*Fort Dodge/Aveco*)	1 mg/kg IM SID for 1-10 days	Pain relief, arthritis
Magnesium sulfate crystals	Magnesium sulfate (various)	0.5-1.0 g/kg PO SID for 1-3 days	Increase gut motility. Aid passage of lead if present in intestines
Metoclopramide	Emequell (Pfizer) (Reglan, Robins)	2 mg/kg IV or IM TID as required	Anti-emetic. Control of gut stasis, eg, sour crop
Oxytocin	Oxytocin S (Intervet); Oxytocin (Leo) (*Butler, Lextron, Vedco*)	3-5 IU/kg IM	Egg binding
Pralidoxime mesylate	Contact National Poisons Bureau regarding availability (*Protopam, Wyeth-Ayerst*)	100 mg/kg IM. Repeat once after 6 hours	Organophosphate and acetyl-cholinesterase poisoning, eg, carbamate
Sodium calcium edetate	Sodium Calcium edetate (Strong) (Animalcare) (*Calcium Disodium Versonate, 3M Pharmaceuticals*)	10-40 mg/kg IV or IM BID for 5-10 days with concurrent fluid therapy	Lead poisoning. No need to dilute

Reproduced from BSAVA Manual of Raptors, Pigeons, and Waterfowl, edited by Peter H. Beynon, Neil A. Forbes and Nigel H. Harcourt-Brown (1996) with permission of BSAVA.

**Ed. Note - To be effective, drink water medication needs to be administered to waterfowl with no water to swim in and the source elevated to avoid bathing.*

Another advantage to pinioning birds at a young age is that birds can be easily sexed before pinioning. Subsequently, the wing pinioned can indicate the sex of the bird, ie, females will be pinioned on the left wing and males will be pinioned on the right.

Tendonectomies of the *extensor carpi radialis* tendon or the insertion point of the superficial *pectoralis* muscle are surgeries that have been described for rendering birds flightless. These techniques are not always effective.[9] Furthermore, most waterfowl owners feel that it is not cost effective to do these surgeries.

Displaced Tendon Repair (Luxation of the Achilles Tendon)

Surgeries have been described for the repair of Achilles tendon luxation.[2,9] Depending on the cause (which is often due to malnutrition), prognosis for recovery is poor, especially in heavier birds. Placement of the affected bird in a sling apparatus to take weight off the legs may improve prognosis.

Orthopedic Repairs

Leg fractures in swans and geese may carry a more guarded prognosis for healing due to large body size and short legs. Immobilization is imperative and may cause stress to the patient. Otherwise, fracture treatment and repair is identical to that done in other birds.

Reproduction

Average Biological Data

Table 36.4 lists sexual maturity, clutch size and incubation period of various waterfowl.

Generally incubation periods are: ducks, 28 to 30 days;

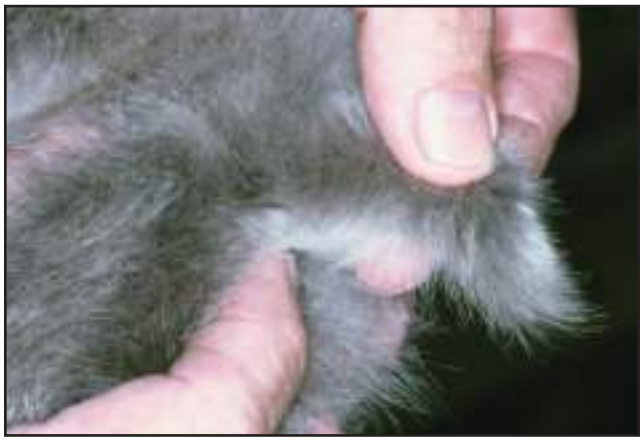


Fig 36.36 | Cygnets if they are to be pinioned it should be done at 2 to 4 days of age.

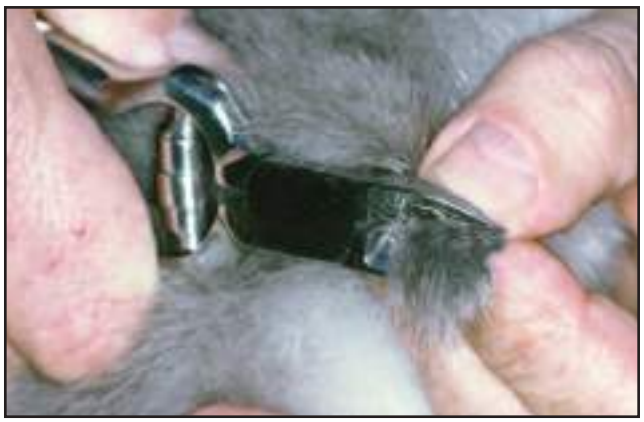
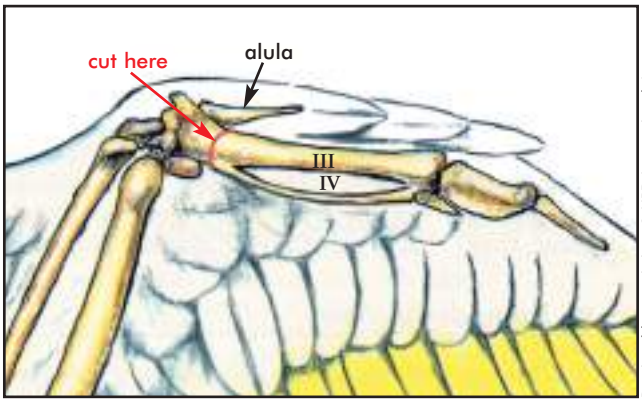


Fig 36.38 | Pinioning metacarpals III and IV are cut as close to the alula as possible with sterile clippers.



Reprinted by permission. © HBD International, Inc.

Fig 36.37 | Anatomy for pinioning. The alula is identified as a landmark and spread away from the carpus. Metacarpals III and IV are shown.

Table 36.4 | Waterfowl Reproductive Data

Species	Sexual Maturity (years)	Clutch Size	Incubation Period (days)
Mute swan	5	4-8	35-40
Pink-footed goose	2	3-5	26-27
Bar-headed goose	2	4-6	27
Hawaiian goose	2	3-5	29
Red-breasted goose	2	3-7	23-25
European wigeon	1	7-11	23-25
Mallard	1	8-12	23-29
Common eider	1	3-6	25-30
Tufted duck	1	6-14	23-25
Mandarin duck	1	9-12	28-30
Muscovy duck	1	8-15	35
European goldeneye	1	9-11	27-32

geese, 20 to 28 days; swans, 30 to 40 days; and Shelducks, 27 to 30 days.

Age of Sexual Maturity

Most ducks are sexually mature at 1 year of age. Geese usually mature in about 2 years. Swans reach sexual maturity at about 5 years.

Artificial Incubation

There are a variety of incubators (Fig 36.39, 36.40), brooders and hatchers (Fig 36.41) available that are suitable for waterfowl.[r,s,t,u] For artificial incubation in waterfowl, incubator temperatures of 99.3° F (37.3° C) and 85% humidity are desirable.

Brooder rooms should be designed so they are easy to clean with adequate ventilation, heating and cooling capacities (Fig 36.42). A heat source (such as a 150-watt heat lamp) should be provided in one area such that the young birds can get close to or back away from the heat as needed. The temperature should be about 95 to 99° F (35-37.2° C) initially, and then gradually decreased over a 3-week period.[9] Never allow chicks to become chilled.

Food and water should be placed at the end opposite from the heat source. Large colored marbles may be placed in water dishes to encourage the birds to learn how to drink. It is not advisable to use hay, shavings, straw or newspaper in the enclosure, as these may be consumed.

Young birds may be ready for outside pens at 2 to 4 weeks of age. Concrete pens with epoxy-painted pools can be constructed. Shallow pools three-fourths inch deep are recommended to acclimate the young birds to water (Fig 36.43). A heat source can be provided in the pen. The pens pictured have doors that can open to outside pens (Fig 36.44). These help acclimate birds as they are going from the controlled indoor environment to the less-controlled environment of outside pens. Once acclimatized to the outdoor pens, the birds can then be moved to larger growing pens.

Waterfowl Diseases

An extensive table of bacterial, fungal, viral and parasitic diseases of Anseriformes is available elsewhere.[9] Listed

Figs 36.39, 36.40 | Different types of incubators are available for waterfowl.

Fig 36.41 | Hatcher for waterfowl eggs.

Fig 36.42 | Brooder room.

Fig 36.43 | Shallow ponds help acclimate young birds to water.

Fig 36.44 | Indoor pens that open to outdoor pens make excellent transition housing.

Fig 36.45 | Heart of a mute swan with gout secondary to bacterial septicemia.

Fig 36.46 | Angel wing in an adult Abyssinian blue-winged goose (*Cyanochen cyanopterus*).

below are some of the most commonly observed disease syndromes.

Malnutrition

Dirty, broken, frayed feathers that do not repel water effectively evidence malnutrition. Frequently, affected birds have secondary bumblefoot due to dietary insufficiencies. Leg and joint lameness with a reluctance to move are other clinical signs, especially in younger birds. Hepatic lipidosis is common in malnourished birds. These birds also are frequently immunosuppressed and thus have secondary bacterial infections. The use of balanced, formulated diets appears to mitigate, if not alleviate, many of these clinical signs.

Bumblefoot

Many waterfowl with bumblefoot (see Fig 36.32) show no signs of lameness. In fact, many cases are not noticeable until the bird is restrained and examined. The underlying cause is usually malnutrition, although rough surfaces and excessive egg laying also can be contributing causes. Because bumblefoot is usually a chronic inflammatory condition, amyloidosis is a common sequela.

Trauma

Most of the larger waterfowl cannot survive if injury, such as that incurred from a dog, alligator, raccoon or turtle attack, results in the disuse of one leg. Heavy body size is not supported well by the one remaining leg. *Pasteurella multocida* is a frequent concern from scratch or bite wounds of predators.

Amyloidosis

Amyloidosis is a condition in which normal organ cells are replaced with a proteinaceous amorphous, eosinophilic, acellular material. Although the exact pathogenesis is unknown, amyloidosis is thought to be associated with stress of close confinement, chronic primary diseases or inflammatory conditions. Acute death frequently occurs, and liver and kidney biopsies are currently the only antemortem diagnostic tests. Where clinical signs occur, affected birds appear lethargic, have a lack of appetite, and are reluctant to stand. Gout is seen frequently subsequent to kidney failure associated with amyloidosis.

Gout

Gout (Fig 36.45) occurs secondary to renal failure. Causes of renal failure include toxicoses, chronic infection and amyloidosis.

Angel Wing

Angel wing (Fig 36.46) is a condition in which the distal portion of the wing appears flipped outward. Young swans and geese are most susceptible to this condition. Angel wing is caused by excessively rapid growth of feathers in relation to muscle development. As a result, growing flight feathers cause excess stress (weight) on carpal muscles, making the carpal portion of the wing hang and twist outward. Possible causes for angel wing include manganese or vitamin E deficiency, hypovitaminosis D_3, genetic factors, over-feeding and excessive dietary protein. If angel wing is noticed soon after the condition develops, it may be corrected by taping the wing in a normal position for 3 to 5 days. However, if the condition is left uncorrected until adulthood, the carpus can become traumatized, with amputation the best solution.

Non-specific Joint Inflammation/Lameness

Clinical signs of non-specific joint disorders include lameness or reluctance to move. This is seen frequently in young birds that are fed primarily "scratch grains"

(cracked corn, wheat, barley or oats) or large amounts of lettuce. In these cases, improvement is seen when the birds are switched to a pelleted diet. This condition also is seen in older birds (especially swans) as arthritis or septic joint infections. Depending on the cause, some decrease of pain and inflammation can be seen with flunixin-meglumine (1-10 mg/kg IM). Carprofen (5-10 mg/kg PO q 24 h) also may be effective. Frequently, bumblefoot develops or worsens as a result of excessive weight placed on the unaffected leg.

Fire Ant Stings

Fire ants are common in Florida. Stings manifest as necrosed areas on the foot web. When healed, these areas show up as defects in the foot web. In the aviary, 5% carbonate dust will help control fire ants. Improperly applied this can be toxic to birds.

Maggot Infestation

Maggot infestation occurs when old wounds (cuts or bites) go undetected or neglected. Infestation can occur in as few as 24 hours. Hydrogen peroxide helps flush out maggots, or they can be removed manually with forceps. Prognosis for tissue recovery depends on the amount of necrosis and length of time the wound has been left unattended. Carcasses that harbor maggots may be a source of botulism toxins if maggots are consumed.[9]

Products and Personal Communications Mentioned in the Text

a. Toprite Netting, Lakewood, NJ, USA; jacissel@Compuserve.com; 1-800-631-2234
b. Mike Gamebird Netting and Sight Barriers, Blue Mountain, AL, USA; 1-256-237-9461
c. BF Products Inc., Harrisburg, PA, USA; 1-800-255-839
d. Sylvan Heights Waterfowl II, M Lubbock and A Lubbock, Scotland Neck, NC, USA; 252-826-5038
e. Montgomery, R, Palm Beach, FL
f. Pinnacle filter, MacArthur Water Gardens, Bethesda, MD, USA
g. Pond Algae Blocker - Destroyers. Algae Fix. Aquarium Pharmaceuticals, www.aquariumpharm.com/pcalgae.htm, 1-800-847-0659, Chalfont, PA. www.macarthurwatergardens.com; 1-800-695-4913
h. Mazuri, PMI Foods, St. Louis, MO, USA; www.mazuri.com; 1-314-768-4592
i. Reliable Protein Products, Palm Desert, CA, USA; www.zoofood.com; 1-760-321-7533
j. High Potency Fine Pellets, Harrison's Bird Foods, Brentwood, TN, USA;1-800-346-0269; www.harrisonsbirdfoods.com
k. Tomahawk Mighty Net, Tomahawk Live Traps and Equipment, Tomahawk, WI, USA; 1-800-272-8727, www.livetrap.com
l. Tomahawk Throw Net, Tomahawk Live Traps and Equipment, Tomahawk, WI, USA; 1-800-272-8727, www.livetrap.com
m. 3M Vetwrap, 3M Worldwide, www.mmm.com; 1-888-364-3577
n. Wydase, www.tricarepharamacy.com
o. Feeding Tube and Urethral Catheter, Sovereign, Sherwood Medical, St. Louis, MO, USA
p. 17 Fr, 5.5 mm disposable silicone tubes, Veterinary Specialty Products, Boca Raton, FL, USA; www.vet-products.com; 1-800-362-8138
q. Emeraid Products, Lafeber Company, Cornell, IL, USA; www.Lafeber.com; 1-800-842-6445
r. Petersime Incubator Company, Gettysburg, OH, USA; 1-888-255-0067
s. Lyon Electric Company, Chula Vista, CA, USA;1-619-216-3400
t. Brinsea, Titusville, FL, USA; www.brinsea.co.uk; 1-888-667-7009
u. Humidaire Incubator Company, Madison, OH, USA; hatch@bright.net; 1-800-410-6925

References and Suggested Reading

1. Beynon PH, Forbes N, Harcourt-Brown NH (eds): Manual of Raptors, Pigeons and Waterfowl. Shurdington, Cheltenham, BSAVA, 1996.
2. Brown CS: An innovative repair method for slipped tendon. Proc Assoc Avian Vet, 2000, pp 127-131.
3. Carpenter JW, Mashima TY, Rupiper DJ: Exotic Animal Formulary. Philadelphia, WB Saunders Co, 2001, pp 109-194.
4. Game Bird and Conservationists' Gazette magazine, www.gamebird.com.
5. Griffin C, Snelling LR: Use of hyaluronidase in avian subcutaneous fluids. Proc Assoc Avian Vet, 1998, pp 239-240.
6. Jenkins J: Hospital techniques and supportive care. *In* Altman RB, Clubb SL, Dorrestein GM, Quesenberry K (eds): Avian Medicine and Surgery. Philadelphia, PA, WB Saunders Co, 1997, p 240.
7. Johnson-Delaney CA. Poultry and waterfowl. *In* Exotic Companion Medicine Handbook. Lake Worth, FL, Zoological Education Network, 2000.
8. Lumeij JT: Nephrology. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Intl, Inc 1999, p 539.
9. Olsen JH: Anseriformes. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Intl, Inc 1999, pp 1236-1275.
10. McMillan MC: Imaging techniques. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Intl, Inc 1999, p 277.
11. Todd F. The Natural History of Waterfowl. Vista, CA, Ibis Publishing Co, 1997.
12. Flinchum G: Exotic pet care: Swans. Exotic DVM 2(1):36-38, 2000.
13. Gardner G, et al: Selected Topics in captive swan medicine and surgery. Exotic DVM 5(4): 33-38, 2003.

CHAPTER

37

Management of Racing Pigeons

JAN HOOIMEIJER, DVM

Open flock management, which is used in racing pigeon medicine, assumes the individual pigeon is less important than the flock as a whole, even if that individual is monetarily very valuable. The goal when dealing with racing pigeons is to create an overall healthy flock composed of viable individuals. This maximizes performance and profit. Under ideal circumstances, problems are prevented and infectious diseases are controlled. In contrast, poultry and (parrot) aviculture medicine is based on the principles of the closed flock concept. With this concept, prevention of disease relies on testing, vaccinating and a strict quarantine protocol — measures that are not integral to racing pigeon management.

This difference is due to the very nature of the sport of pigeon racing; contact among different pigeon lofts (pigeon houses) constantly occurs. Every week during the racing season, pigeons travel — confined with thousands of other pigeons in special trucks — to the release site. Pigeons from different lofts are put together in baskets. Confused pigeons frequently enter a strange loft. In addition, training birds may come into contact with wild birds during daily flight sessions. Thus, there is no way to prevent exposure to contagious diseases within the population or to maintain a closed flock. The pigeon fancier also must be aware that once a disease is symptomatic, the contagious peak has often already occurred, so preventive treatment is too late. Treatment at this point may be limited to minimizing morbidity and mortality.

Fig 37.1a | The appearance of the head of a "modern" male racing pigeon, is a result of selective breeding. The differentiation of the sexes of the modern racing pigeon by head structure has become much less dependable than in the wild type.

Fig 37.1b | The head of an "old fashioned" or "show racer" male pigeon makes visual sexing easy.

What Makes a Loft Successful?

QUALITY OF THE PIGEONS

The health and racing abilities of racing pigeons are limited by the genetics of the parents. This selection process has even altered the appearance of the birds (Figs 37.1a,b). Using strong selection criteria when pigeons are paired ensures that the quality increases with each successive generation. Most pigeon fanciers select on the basis of performance and forget that other major factors to consider might include features such as resistance to diseases. For example, respiratory problems are common during the racing season, and it appears that heredity partially determines resistance. Thus, the veterinarian should play a role in establishing selection criteria.

It is critically important to understand that there is a difference in genetic susceptibility among different pigeons. Selecting pigeons that are not clinically affected by a disease outbreak helps increase disease resistance in the future. This is the principle of "survival of the fittest". During the first 10 weeks, 25 to 30% of the young birds should be culled. An average of an additional 60% will be culled or lost over the next 3 years.

CRITERIA FOR SELECTION OF BREEDERS

A winning racing pigeon is not necessarily the best choice for breeding. Similarly, the best breeding bird may not always have a good racing record.

Theoretically, the best performing racing pigeons are the result of cross-breedings. One chooses a bird from a certain line based on race performance and breeds it to a bird that is genetically predisposed to disease resistance. Offspring from such crossings are generally stronger and more viable but may not themselves make the best breeding pigeons (Figs 37.2a,b).

The quality of the breeding pigeon is determined by the achievements of its youngsters. There are several important qualities to select for in young birds. The birds should be visually healthy and strong and prove resistant to contagious diseases. Vigor, personality and intelligence are equally important. Birds should be creative in interacting with the rest of the flock, as successful cohabitation is required. Well-developed orientation skills are vitally important in gauging a pigeon's winning potential. Pigeons should be able to discern the most efficient route to return home without delay.

These criteria are applied at the earliest age, starting with eggs. Eggs that develop slowly and squabs that hatch late should be culled. The growth, development and feather quality are early characteristics that can be used to evaluate young pigeons. Physical abnormalities or delay in physical development are reasons to cull a bird.

Squabs should be able to fly by the age of 5 weeks. When youngsters start to fly around the loft, it is important to observe flight differences. The fancier should keep notes and compare observations among individual pigeons. Differences also can be noticed among youngsters during weaning. Ideally, a pigeon will find the water supply and start eating and drinking without any hesitation. It is not a good sign when a fledgling is slow to learn. Similarly, when it comes to finding a place to perch in the loft, which bird gets the most preferred spots? The ones that are chased away from the food source or ideal perches should be culled.

There will frequently be young pigeons that have a wet eye, a brown cere or abnormal feces. Even if these

Fig 37.2a | A typical breeding facility.

Fig 37.3 | Typical wavy or "milled " edges of primary wing feathers show a lack of condition.

Fig 37.2b | A female with youngsters.

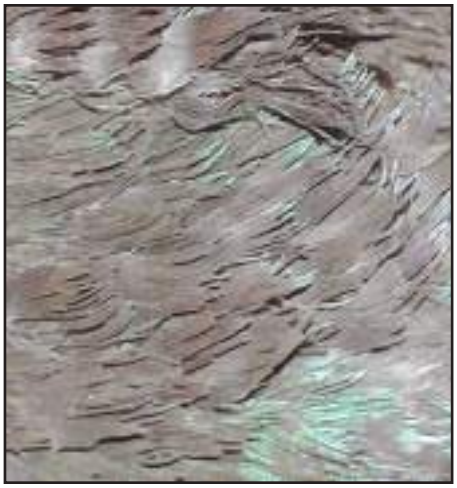


Fig 37.4 | Contour feathers are missing the proper structure to keep them smooth, and the normal shiny color has been lost. (Further examples showing lack of condition).

pigeons recover quickly, they should be culled. Culled birds should be recorded in the parents' records so improved matings can be undertaken in the future.

Criteria For Selection Of Birds To Race

The fancier's selection criteria for choosing birds to race often determines the success of his program. The author's current advice is to race a maximum of 50% of the available pigeons per race. When there is illness or other problems, racing a smaller percentage is advised. The decision to race an individual pigeon should be based on the condition of the bird, not on its previous performances. A decision so based can prevent the loss of good pigeons from over or misuse (Figs 37.3, 37.4). There are only a few very good pigeons. To race those pigeons in their prime is the goal, and this is more important than winning a particular race.

To judge the success of a selection program, the fancier should answer these questions: Is the percentage of the pigeons winning points in races increasing? Are there fewer disease problems, especially during the racing season? Are there fewer birds lost at the end of races? Has the profit increased?

If the answers are negative, then the pigeons may not be in the best physical condition (Fig 37.5). Birds that are under-conditioned not only perform poorly, they do not have enough resistance to infection during transport. Such birds commonly have subclinical problems, especially respiratory disease (see Respiratory Problems, below). An opportune time to evaluate pigeons is immediately after their return from a race. A bird's recovery speed and any problems seen immediately after a race should be documented.

FANCIER'S COMMITMENT TO QUALITY

Year after year, champions come from lofts where the owners are committed to selecting quality birds. Good fanciers plan the purchase of new pigeons based on characteristics that will improve the flock rather than

Fig 37.5 | A section of this feather's vane is defective following a molt, supporting the evaluation of lack of proper condition.

Fig 37.6 | A pigeon loft, displays adequate but not excessive amounts of glass.

purchasing a new bird that has won a certain race. Old breeding stock should be kept pure to conserve good qualities in case cross-breedings do not work out as planned. While infectious diseases are still present and do spread among most of the lofts during the racing season, this commitment to quality ensures that infectious diseases generally don't play a major role in ideal lofts. On the other hand, an outbreak of herpesvirus infection can be devastating to young pigeons under any situation.

Fanciers should be educated on establishing high quality standards in their loft as well as their birds. They then can base husbandry decisions on logic, not on what other fanciers tell them. For example, in the past, a fancier often made the decision to put more glass in the loft roof, because everyone was putting glass in their roofs. The increased level of light was perceived by many to be advantageous, especially in the early months of the year. However, many fanciers did not think about the disadvantages. More glass resulted in greater temperature fluctuations. The excess glass promoted excess humidity in cool months, while it led to the loft becoming too hot and dry in the summer.

THE LOFT

Climate in the Loft

The loft's climate should be warm, dry and without draft (Fig 37.6). The environment of the pigeon house must be constantly monitored because temperature and humidity fluctuate with changes in the weather, as well as between day and night. A common error during cold, wet weather is to allow too much ventilation, which leads to a house colder on the inside than out. Instead, ventilation should be reduced and supplemental heat added. At the beginning of the season, the loft temperature should be at least 12° to 13° C (53-55° F) during the night. The humidity should not be over 70%.

During warm, dry periods, many pigeon houses are insufficiently ventilated, becoming hot and dusty and creating respiratory signs that fail to respond to any medication. The temperature should not be over 28° C (82° F). Otherwise, pigeons will start drinking more and eating less, which has a negative influence on their general condition. For example, this causes wetter droppings, which can incubate bacteria, fungus and yeast. The short-term instillation of smoke into the compartment during construction allows airflow to be visualized. The air inflow should enter above the level of the pigeons and exit out the roof.

The quality of pigeon houses is highly variable. During inspection, a check list will aid in identifying problems and making suggestions to correct these areas in the facility (eg, insulation materials used, ventilation errors).

The Number of Pigeons in the Loft

Overcrowding is an important cause of a poor loft environment (Fig 37.7). Crowding promotes feather dust, wet feces and excess moisture in expired air. Space is another consideration, as each pigeon needs its own space and territory. When birds are constantly competing for space, the number of birds is too high. Males are more competitive, so there should be fewer cocks than hens or chicks per cubic meter. When young pigeons reach 7 to 8 weeks of age, they begin to compete in the pecking order.

Over the past 10 years, the tendency of many fanciers has been to develop larger flocks each year, despite the fact that their facilities are not designed for expansion (Fig 37.8). Pigeon fanciers do not improve performance of the racing flock by simply producing more birds. Too many pigeons in improper facilities creates stress, increases dust, and, therefore, increases susceptibility to respiratory and other disease.

Fig 37.7 | A pigeon loft with a crowded outdoor flight cage creates an undesirable stressful situation.

Fig 37.8 | A youngster was mutilated as a result of overpopulation and stress.

Loft Space Guidelines

To avoid the problems of overcrowding, each loft should contain a maximum of two young pigeons, two adult females and one adult male per cubic meter. Therefore, in a loft of 2 x 2 x 2 meters (8 cubic meters), there can be 16 young pigeons, 16 adult females and 8 adult males. Other recommendations suggest a maximum population density of half this number per unit space. During fledging, some overpopulation is acceptable because of the better climatic conditions in the early months of the year, including low temperature and high humidity. Overpopulation is very unfavorable during the racing season (ie, high temperature, low humidity), so a major cull must occur just prior to the racing season.

Separating Groups by Age

The adverse effects of housing birds of different ages together are well-known in the poultry industry. The same effects are seen in mixed-age groups of racing pigeons. By advancing the breeding season and breeding the same stock several times each year, many fanciers are creating and maintaining several different age groups within a flock. This is acceptable if the facility has been designed to accommodate multiple age groups.

The goal is to breed enough young pigeons so the fancier has the opportunity during the first 2 to 3 months to select the pigeons that exhibit the desired qualities, culling the balance to prevent overcrowding and avoiding the pitfalls such as stress and disease that come with keeping too great a number of individuals.

Veterinary Support

The aims of veterinary support are to ensure a healthy flock, to minimize the losses and to maximize success during the racing seasons.

PREVENTIVE MEASURES

Proper husbandry practices should be followed at all times. This includes areas both in and around the loft.

Monitoring Procedures

Particular attention must be paid to general body condition, molting or feather disturbances, signs of *Salmonella* spp., ectoparasites and endoparasites that may be present in the feces. A swab from the crop and/or cloaca should be obtained for cytology and Gram's stains and cultures if disease is suspected in a bird. Necropsies of poor performers or birds showing signs of disease can be important for monitoring the disease status of the flock.

Vaccinations

Routine vaccinations are imperative. Regulations regarding these may vary among countries. Paramyxovirus vaccine (Fig 37.9) should be given after 3 weeks of age and thereafter once a year about 3 weeks before breeding. Poxvirus vaccine should be given after 5 weeks of age and thereafter once a year at least 3 weeks before racing season. Salmonella vaccine is given after 5 weeks of age, then twice at 2-week intervals and 3 weeks before breeding.

Preventive Medications

Trichomoniasis (Fig 37.10) is one the most common infectious diseases among racing pigeons in the Netherlands. Trichomoniasis causes an erosion of the pharyngeal mucosa and increases susceptibility to other infectious agents (Fig 37.11). Hexamitiasis is also common.

Examination of young pigeons at the time of vaccination against paramyxovirus has shown that 35 to 40% of these birds, which show no clinical signs, are infected with

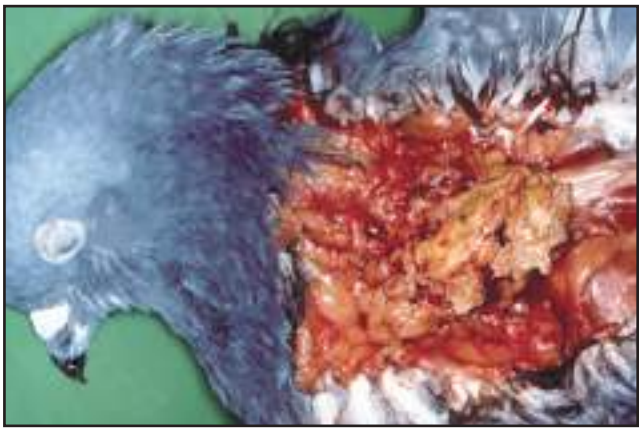


Fig 37.9 | Granuloma formation as a result of a vaccine reaction after using an oil-based paramyxovirus vaccine.

Fig 37.11 | Clinical signs of a combined infection with herpesvirus and trichomoniasis.

trichomonads. When clinical signs of respiratory disease are present, this percentage is much higher.

To control trichomoniasis, breeding pigeons should be treated when they are incubating their eggs. The offspring should then be treated at 5 weeks of age. Treatment consists of ronidazole once daily for 6 days and repeated for 3 to 4 days every 3 weeks. During the racing season, the pigeons should be treated every weekend upon their return from a race.

Medications should play only a minor role in the management of diseases in a loft. Use of medication should be based on a confirmed diagnoses. Refer to the literature for a more detailed discussion of medications and specifics on diseases.[12] Unfortunately, European fanciers are encouraged to overuse medications. Companies that sell medicaments frequently advertise their products in publications designed for pigeon fanciers. Treatment for coccidiosis or other endoparasites and Salmonellosis should only be performed when these conditions have been diagnosed. A common misconception among fanciers is that these treatments will improve racing performance.

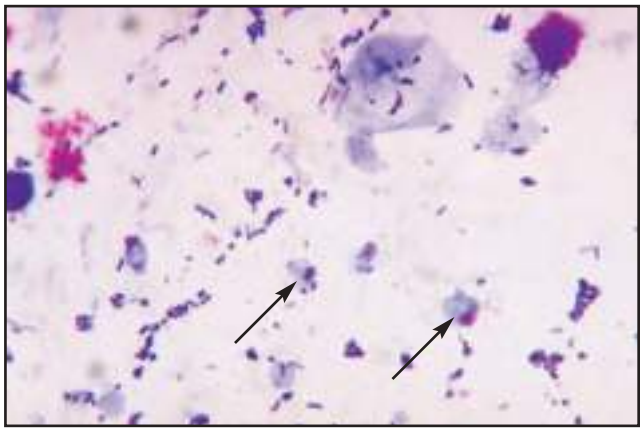


Fig 37.10 | Stained slide preparation of trichomoniasis (arrows).

The dosage of any medication is based on body weight. In general, 20 pigeons drink about 1 L of water per day, but intake can vary. Medications may also be administered in the food. A small amount of yogurt may be used as a vehicle in which to suspend medication that will adhere to the food.

The Presence of *Escherichia coli (E. coli)*

The presence of *E. coli* is considered a normal inhabitant of pigeons' intestinal flora. When *E. coli* is cultured at necropsy, it is important to look for concurrent viral diseases, especially circovirus, herpesvirus and adenovirus. Many fanciers will request bacterial cultures of sick or dead birds, and *E. coli* is frequently isolated. The fancier may mistakenly assume that the *E. coli* is the primary pathogen. Antibiotics sold over the counter are then administered to these birds. In the face of a viral infection, these antibiotics may not only be ineffective, but may also destroy the natural intestinal flora.

Veterinary Inspection of the Loft

The quality of veterinary support depends on the experience and knowledge of the veterinarian. The flock is considered the patient. Prevention is the creed. The veterinarian and the fancier should base their relationship on mutual respect and trust. The veterinarian's physically visiting the pigeon loft is an important part of the veterinary program.

When evaluating the loft, the veterinarian should note the physical facilities, including the direction the loft faces (preferably southeast); what materials were used for construction, insulation and roofing; the size of each loft; the amount of glass in front and on the roof; and what type of ventilation system is in use. He or she should note any trees, bushes, fences and buildings around the facility and evaluate them for safety and appropriateness (eg, ensure that there are no toxic plant materials or fencing that could prove harmful).

Fig 37.12 | Feather/quill mites.

Figs 37.13a | The slender pigeon louse, which can be considered harmless in small numbers, is a sign of poor health in large numbers.

Figs 37.13b | The small pigeon louse, which can cause much distress, is often found around the neck feathers and uropygial gland areas.

Fig 37.14 | Feces in the morning without distress at night.

Fig 37.15 | Feces in the morning as the result of distress at night because of mites and lice.

In evaluating the fancier's parasite control, the veterinarian should perform a thorough inspection of the birds for ectoparasites (Figs 37.12, 37.13a,b). Fecal examinations should be performed for endoparasites. Examining the cleanliness of the loft in general (Figs 37.14, 37.15), and the food and water sources in particular, is important for control of both endoparasites and potentially infectious microorganisms.

During this inspection, the veterinarian can verify that the population density and age separation criteria are appropriate, as well as noting and discussing nutrition, hygiene and other husbandry concerns that are significant.

Contagious Diseases

Contagious diseases are common, especially during the racing season. As discussed previously, preventing contagious diseases in racing pigeons is impossible (Fig 37.16). As a result, one must consider an individual flock as part of the whole pigeon population in the racing area and even as part of the whole racing pigeon sport, because birds travel between countries.

PREDISPOSING FACTORS THAT DETERMINE THE PATHOGENICITY OF A DISEASE

While it may not be possible to avoid all contagious infections in racing pigeons, there are many factors that influence the severity and scope of disease. Healthy, well-maintained birds will suffer lower morbidity and mortality than immune compromised birds. Some factors that increase the pathogenicity of opportunistic diseases include: malnutrition, overpopulation (birds lower on the pecking order are more susceptible), suboptimal climate conditions within the loft, combining different age groups in the same loft, adding new pigeons to the flock, stressful situations within the flock, and racing birds that are not in proper condition. Diseases and vectors such as salmonellosis, trichomoniasis, heximitasis, mites, lice and endoparasites, can be immunosuppressive and increase morbidity and mortality of contagious diseases.

INFECTIOUS DISEASES AND PARASITES

While the fancier and veterinarian must be vigilant for a wide range of possible debilitating afflictions, some are more prevalent than others. The main infectious illnesses of racing pigeons include: viral diseases, chlamydophilosis

Fig 37.16 | Release of pigeons from the transport trucks to start a race.

Fig 37.17 | Green-yellow urine is often a first sign of adenovirus and/or herpesvirus infection.

Fig 37.18 | The death of youngsters within 1 day can be associated with circovirus or salmonellosis.

and parasitic diseases such as lice, mites, roundworms (*Ascaridia*, *Capillaria*), tapeworms, coccidia, *Hexamita* spp., trichomonads and trematodes. Additionally, birds may contract combined infections, known as "ornithosis complex", of the upper respiratory tract.[12]

VIRAL DISEASES

In most viral outbreaks, an otherwise healthy flock that is properly managed will have only a few birds sick and only a small percentage will die.[12]

Paramyxovirus and Poxvirus

Vaccines are available for annual protection against paramyxovirus and poxvirus in racing pigeons. By law, in Europe, pigeon fanciers must vaccinate their pigeons against paramyxovirus. There is evidence that pigeon paramyxovirus and the paramyxovirus that causes Newcastle disease in poultry are distinct. Poxvirus infection is transmitted by mosquitoes. During an outbreak, do not allow the pigeons to bathe. The ideal environment would include higher-than-normal temperatures, good ventilation and controlled humidity. Disinfection of the pigeon loft is important when a viral infection is suspected. It also is essential that all aspects of management and husbandry be evaluated and corrected if necessary.

Adenovirus

Adenovirus type 1 is especially common in young pigeons. Adenovirus type 2 is seen mainly in older pigeons, causing acute hepatitis and acute death. Pigeons that appeared to be healthy at the time of putting them in the baskets for a race can be dead on arrival at the race venue. Clinical signs of an adenovirus outbreak include reduced appetite, excessive drinking, regurgitation, loose voluminous feces, abnormal urine (yellow to green instead of white) (Fig 37.17), weight loss and acute death.

Herpesvirus

Clinical signs may include epiphora, rhinorrhea and diphtheric plaques in the mouth, throat and/or trachea similar to those seen with trichomoniasis or poxvirus. Anorexia, polydipsia and yellow-green urates may be present due to hepatic involvement. Herpesvirus can also cause central nervous system signs, such as paresis and paralysis. Differential diagnoses for the encephalitic form include paramyxovirus, salmonella, intoxications and trauma. A definitive diagnosis is made post-mortem via histopathology and/or viral culture.

Circovirus

First reported in 1990 in California, circovirus also has been found in Europe since 1995. The main characteristics are juvenile mortality and multiple secondary infections (Fig 37.18). Outbreaks of other infectious diseases are common because of the immunosuppression caused by circovirus. Anemia and paralysis may also be seen, as well as feather abnormalities similar to those seen in affected psittacines.

Pigeons infected with circovirus may not respond to a vaccination against paramyxovirus, poxvirus or salmonella, resulting in serious consequences. When pigeons vaccinated against paramyxovirus develop the disease, the legislation in Europe requires that all pigeons in close contact with affected birds be euthanized.

The most important tool for diagnosing circovirus is to submit tissues of infected organs for histopathologic examination. The bursa of Fabricius is a target organ in birds less than 6 weeks of age. In Belgium, there is a concerted effort underway to create a special pigeon circovirus PCR test.

Management of a Viral Disease Outbreak

Diseased pigeons must be culled from the loft as soon as clinical signs are noted. It is important to realize that

asymptomatic pigeons may still be infected. Not every pigeon gets sick or dies when infected with a contagious viral disease. When faced with an outbreak of paramyxovirus or poxvirus, immediate vaccination, using precautions to prevent further spread of disease, is imperative. Sick pigeons must be examined for the presence of other infectious diseases. Endoparasites and/or salmonella often need to be addressed. Antibiotics should not be used unless there is a confirmed bacterial infection. Reduce the number of young pigeons to a maximum of two per cubic meter.

To support recovery of the pigeons a combination of amino acids, lactobacillus, minerals, electrolytes and vitamins should be provided in the food. Add apple cider vinegar or citric acid (1 g/L) to the drinking water to decrease gastrointestinal pH. Alternating the administration of echinacea and green tea may decrease the incidence of secondary bacterial and yeast infections. Increased fresh air ventilation will decrease the concentration of viruses and aerosolized irritants.

RESPIRATORY PROBLEMS

Respiratory problems are among the more common reasons for poor racing performance and are generally caused by a combination of environmental factors and microbial agents. Pigeons may be subclinical but demonstrate diminished performance. A thorough examination may reveal conjunctivitis, dacrocystitis and/or pharyngitis. The breathing sounds of a normal pigeon, like those of other birds, should be inaudible even when the bird is held to the ear or auscultated.

Clinical Signs Associated with Mild Upper Respiratory Problems

The fancier should always be alert to clinical signs that can signal respiratory problems in the flock. Some of the more easily noticed signs may include "swollen head" due to the feathers being more erect, especially around the ears; similarly, the feathers around the head and neck may no longer be smooth, but become rough and dull. The ceres may no longer be dry with the normal white collection of powder down, but instead are a bit darker or even grayish. Additional clinical signs include epiphora, head shaking and scratching, sneezing and dyspnea. The nictitating membrane is often inflamed and swollen and remains extended; fanciers call it "the film". The bird may exhibit frequent swallowing and tongue movement. The mucous membranes in the pharynx and esophagus become swollen, and often the choanal slit is closed. Mucus is often visible around the glottis and may become viscous and whitish. The choanal papillae may change in shape and, in more chronic cases, even disappear.

Fig 37.19 | Mild respiratory problems may show only a very subtle dampness around the naris.

Fig 37.20 | Severe respiratory problems including conjunctivitis.

Clinical signs can be very subtle (Fig 37.19), which is why most pigeon fanciers do not suspect an infectious disease even when performance is disappointing. Poor race finishers should be examined by a veterinarian in order to detect the more subtle clinical signs.

Clinical Signs Associated with Severe Upper Respiratory Problems

Severe upper respiratory clinical signs are more readily noted and can include a dirty, wet, discolored cere; conjunctivitis with discoloration of the eyelids; wet areas on the shoulder or back feathers from eye wiping; and respiratory congestion (Fig 37.20). The sounds may be dry or moist. In severe cases where a tracheitis or other deeper respiratory problem is involved, the pigeon may be gasping for air with an open beak and have exaggerated abdominal and tail movements. Yellowish flakes may be observed on the mucous membranes of the oropharynx and tongue and in the trachea. These may be combined with diphtheritic membranes.

Infectious agents that may be involved in respiratory diseases of pigeons include pigeon herpesvirus, pigeon poxvirus, circovirus, *Chlamydophila psittaci*, *Trichomonas* sp., *E. coli*, *Hemophilus* spp. and *Mycoplasma* spp.

Differential Diagnoses for Respiratory Diseases

Poorly performing birds should be thoroughly examined. In making a list of differential diagnoses, as in all birds, many other disease conditions can present as respiratory distress, including egg binding, serositis, tumors, cysts, severe infestation with worms or coccida, cardiovascular disease and anemia. The infectious agents previously mentioned must be considered, in addition to less commonly occurring mycotic infections. Husbandry, including air quality and ventilation, may also be contributory. Inhalation of toxic fumes may also cause upper and or lower respiratory signs (Table 37.1).

In practice, one is often contending with a combination of these different agents and the complicating factors of dust, crowding and malnutrition.

Treatment of Respiratory Diseases

Treatment will depend upon the etiologic agents involved and may include vaccination, improved ventilation, dietary supplementation as previously outlined and antimicrobials when indicated.

Several affected individuals should be culled while implementation of prevention and treatment protocols are instituted for the remainder of the flock.

Table 37.1 | Differential Diagnoses Based on Clinical Signs

Clinical Signs	Possible Causes
Loose, unformed feces (Fig 37.21)	• Endoparasites such as capillaria (Figs 37.22, 37.23), ascaridiasis (Fig 37.24), coccidiosis, hexamitiasis, trematodiasis, cestodiasis • Bacterial infections including *Salmonella typhimurium* var. *Copenhagen, Streptococcus bovis* • Viral infections including adenovirus, herpesvirus • Intoxications, excessive ingestion of moss, sand • Stress
Polyuria/polydipsia	• Paramyxovirus • Excessive dietary salt • Intoxications • Severe enteritis • "Spraying" is a lay term that refers to polyuria and loose feces. It occurs in youngsters of about 10 days of age when the parents stop feeding crop milk. It also is seen during the stress of mating. Causes for such symptoms should be investigated
Respiratory problems	• Improper loft conditions, eg, dust • Unseasonal weather: too hot, too cold or wet • Ornithose-complex • Trichomoniasis (Fig 37.25), tumors (Fig 37.26) • Herpesvirus • Tracheitis/air sacculitis • Obstructions within the trachea, syrinx (foreign body, mycoses, diphtheria) • Pneumonia • Ascites, egg binding, tumors, serositis, foreign body • Circulation problems • Obesity • Excessively high temperature • Bad general physical condition • Overpopulation, stress • Mixing different age groups
Diphtheric mucous membranes	• Trichomoniasis • Herpesvirus • Poxvirus (Fig 37.27) • There are many causes for these small yellow dots: *E. coli* (Fig 37.28) • Candidiasis
Disturbance of equilibrium	• Paramyxovirus • Herpesvirus • Salmonella • Intoxication with substances such as dimetridazole, (Fig 37.32) or aminopyridine salt • Encephalitis - trichomoniasis (Figs 37.29-37.30) • Trauma • Debilitation and emaciation-seen in birds returning from a race in inclement weather • Anemia
Drooping wing, leg lameness	• Salmonella • Arthritis • Myositis caused by *Streptococcus bovis* • Muscle damage subsequent to trauma • Fractures • Joint luxations (Fig 37.33) • Paralysis subsequent to egg laying (Fig 37.34) • Paramyxovirus • Leg swells under band (Fig 37.35)
Abnormal feathers	• Malnutrition-for dietary considerations[12] • Can be caused by medication such as fenbendazole administered during the molting period • Corticosteroids are reason for a delay in molting and typical growth lines (Fig 37.36) • Paramyxovirus • Circovirus • Chronic diseases such as salmonellosis

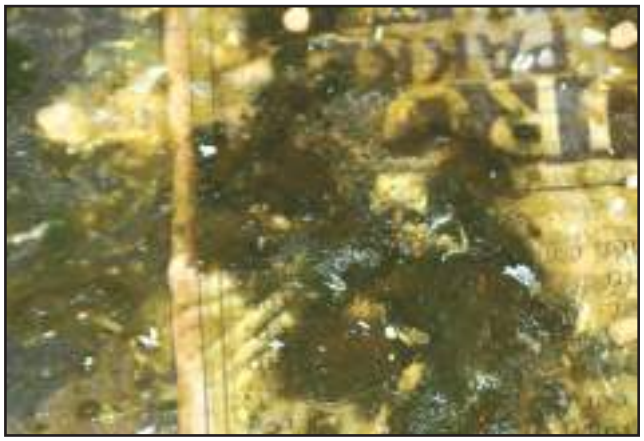


Fig 37.21 | Abnormal feces caused by hexamitiasis in young pigeons.

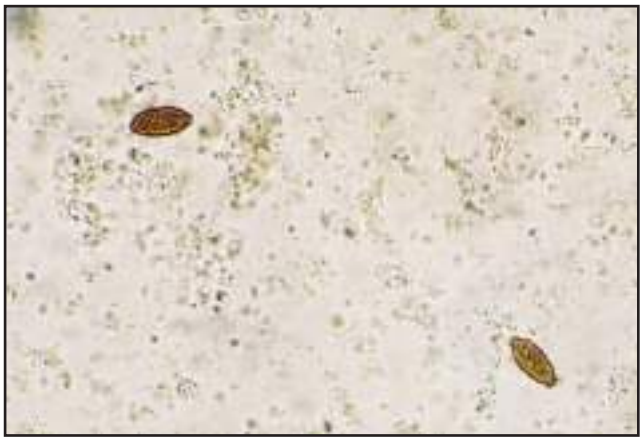


Fig 37.22 | Eggs of capillaria.

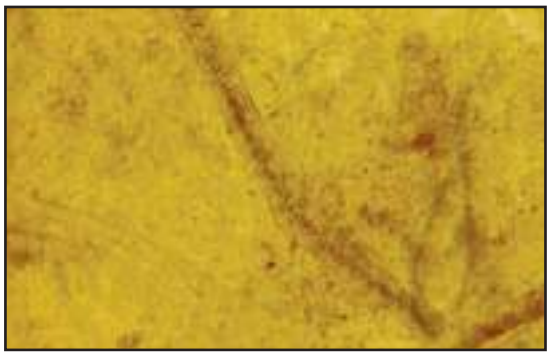


Fig 37.23 | Capillaria under the microscope, note the eggs of the capillaria in the parasite's body.

Fig 37.24 | Intestines with ascarid obstruction.

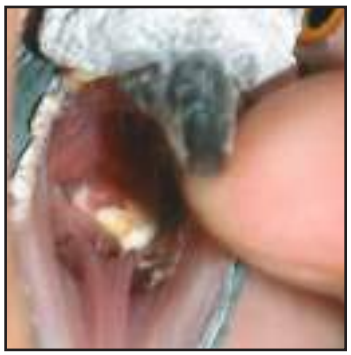


Fig 37.25 | Obstruction of the glottis by a diphtheric mass often seen with trichomoniasis.

Fig 37.26 | Swelling of the mucous membranes caused by a carcinoma.

Fig 37.27 | Poxvirus lesions of the eyelids, beak and tongue.

Fig 37.28 | White spots are the accumulation of cellular debris in the mucus glands of the palate. Histopathologically these are often described as "lymphoid aggregates" and suggest a cellular immunologic reaction in response to a locally infectious agent.

Fig 37.29 | Torticollis in a young pigeon caused by an unusual infection with trichomoniasis.

Fig 37.30 | The youngster in Fig 37.29 at necropsy showing yellowish fluid surrounding the brain.

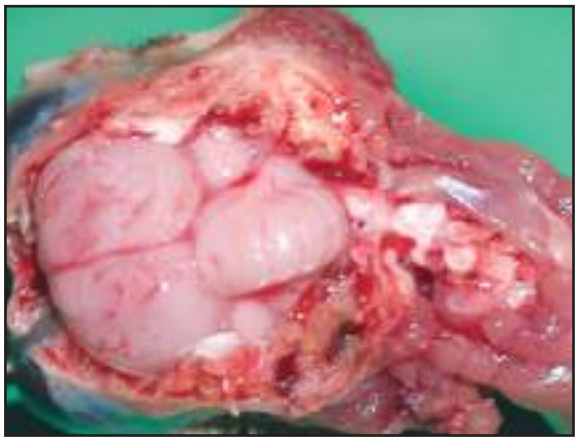


Fig 37.31 | Necrotic brain tissue in the bird in Figs 37.29 and 37.30.

Fig 37.32 | Hemorrhage as a result of dimetridazole intoxication causing central nervous signs.

Fig 37.33 | Luxation of the shoulder causing the wing tip to point upward.

Fig 37.34 | Reversible paralysis after egg laying.

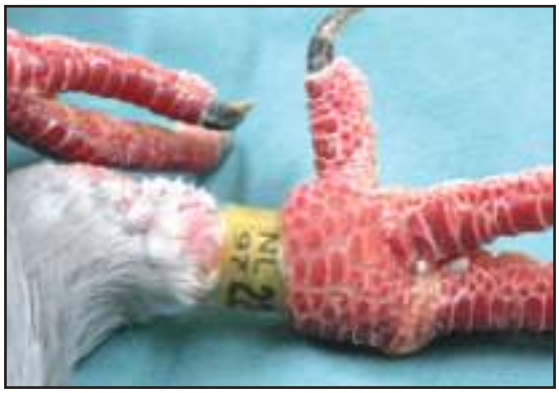


Fig 37.35 | A leg band has been accumulating hyperkeratotic debris from malnourished skin and the resultant constriction acts like a tourniquet, requiring band removal to save the leg.

Fig 37.36 | Feather growth abnormalities are seen after administration of corticosteroids or fenbendazole overdose.

References and Suggested Reading

1. Abadie J, Nguyen F, et al: Pigeon circovirus infection: Pathological observations and suggested pathogenesis. Avian Path 30:149-158, 2001.
2. Dorrestein GM: Viral infections in racing pigeons. Proc Assoc Avian Vet, 1992, pp 244-257.
3. Duchatel JP, Jauniaux T, et al: Premiere mise en evidence en Belgiqiue de particules ressemblant a des circovirus chez le pigeon voyageur. Annales de Medicine Veterinaire 142:425-428, 1998.
4. Hooimeijer J, Dorrestein GM: Pigeons and doves. *In* Altman RB, Clubb SL, Dorrestein GM, Quesenberry K (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 886-909.
5. Hooimeijer J: Principles of veterinary support in racing pigeons. Proc Assoc Avian Vet 1993, pp 151-155.
6. Marshall R: Management of pigeon diseases. Proc Assoc Avian Vet 1990, pp122-135.
7. Pennycott TW: Pigeon diseases: Results from a Scottish diagnostic laboratory. Proc Assoc Avian Vet 1994, pp 231-239.
8. Rupiper DJ, Ehrenberg M: Introduction to pigeon practice. Proc Assoc Avian Vet 1994, pp 203-211.
9. Rupiper DJ, Ehrenberg M: Practical pigeon medicine. Proc Assoc Avian Vet 1997, pp 479-497.
10. Rupiper DJ: Diseases that affect race performance of homing pigeons. Part 1: husbandry, diagnostic strategies, and viral diseases. J Avian Med Surg, 12:70-77, 1998.
11. Rupiper DJ: Diseases that affect race performance of homing pigeons. Part 2: bacterial, fungal and parasitic diseases. J Avian Med Surg, 12:138-148, 1998.
12. Vogel C, Gerlach H, Loffler M: Columbiformes. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 1200-1217.
13. Vogel K: Taubenkrankheiten. VEB Deutscher Landwirtschaftsverlag, Berlin, 1983.

CHAPTER
38

Management of Galliformes

GARY D. BUTCHER, BS, MS, DVM, PhD, Dipl ACPV

Greg J. Harrison

Fig 38.1 | In warm climates, game cocks are housed on "string walks". A leg leash, just long enough for the bird to reach a shelter but not to fight with other birds, allows group confinement. Fighting of cocks is considered inhumane and illegal in some countries; however, it is a part of the culture in others.

Members of the order Galliformes are found on every continent except Antarctica. The red junglefowl, common turkey and helmeted guinea fowl have been domesticated for centuries. Their descendants, through selective breeding, are of considerable economic importance today. Some varieties are very plentiful in the wild, while others like the Japanese quail (*Coturnix japonica*) and various pheasants are approaching a level of complete domestication.

Many Galliformes are commonly maintained as game and food (meat and/or eggs) birds. Some are stable in captivity under variable ambient conditions, easy to breed and inexpensive. Other species are from niches with specific environmental requirements and need specialized diets, humidity and temperature ranges to survive. Currently, commercial production of chickens and turkeys in the USA for food has surpassed that of the beef, pork and fish industries. In 1900, per capita consumption of chicken was 1 pound and had risen to 80 pounds by the year 2000!

In this chapter, "domestic fowl" means *Gallus gallus, forma domestica* (domestic form of the red junglefowl); "domestic turkey" is *Meleagris gallopavo, forma domestica* (domestic form of the common turkey) and "domestic guinea fowl" is *Numida meleagris, forma domestica* (domestic form of the helmeted guinea fowl) (Table 38.1).

Maintaining, breeding, treating or commercially dealing with gallinaceous birds may be regulated by laws that govern the protection of animals, property rights, exchange of goods, liability, epornitics, food for human consumption, hunting and (international) transport of animals. In the USA, voluntary federal and state programs such as the National Poultry Improvement Plan (NPIP) provide testing for specific diseases to facilitate interstate and international transport of fowl. Laws are

Table 38.1 | Families and Subfamilies of Gallinaceous Birds

Family (Subfamilies)	No. of Genera	No. of Species
Cracidae (cracids)	10	43
Megapodiidae (megapodes)	7	12
Phasianidae (phasianids)	70	203
Numidinae (guinea fowl)	4	6
Pavoninae (peafowl)	2	3
Meleagridinae (turkeys)	1	2
Argusianinae (peacock pheasants and argus pheasants)	3	8
Phasianinae (pheasants)	8	21
Lophophorinae (monals)	1	3
Pucrasiinae (koklass)	1	1
Ithagininae (blood pheasant)	1	1
Gallinae (junglefowl)	1	4
Tragopaninae (tragopans)	1	5
Galloperdicinae (spurfowl)	1	3
Ptilopachinae (stone partridge)	1	1
Perdicinae (partridges, snowcocks, francolins, Old World quail)	27	98
Odontophorinae (New World quail)	9	31
Tetraoninae (grouse)	9	16

presently in place and/or being considered in some states that would prevent the interstate or international transport of fowl for purposes of fighting (Fig 38.1).

Anatomy and Physiology

Considering the large number of birds in the order Galliformes, there are surprisingly minimal anatomic and physiologic differences as compared to other animal orders. Likely, the strict physical requirements for flight have limited variability. Several peculiarities should be discussed. In the circulatory system, for example, most gallinaceous birds have right and left internal carotid arteries; however, the megapodes have only the right internal carotid artery. The respiratory rates, heart rates and rectal temperatures of some gallinaceous birds are listed in Table 38.2 and are highly variable, depending on the metabolic rate and physiology of the specific bird.

INTEGUMENT

Many gallinaceous species develop a durable, vascularized thickening of the corium in the ventral thoracic region called a brooding spot. The feathers in this region are temporarily lost and body heat is transferred directly from the brooding bird to the eggs.

The preen gland in the domestic fowl consists of two bilaterally symmetric lobes, each with one secretory duct opening into the uropygial papillae. Some breeds of domestic fowl have two uropygial papillae. Tail-less breeds of the domestic fowl and the argus pheasants

Table 38.2 | Respiratory Rate, Heart Rate and Rectal Temperature of Selected Gallinaceous Birds

Bird Group	Respiratory Rate (per min)	Heart Rate (per min)	Temperature (°C)
Domestic fowl	12-37	220-360	41.2
Domestic turkey	28-49	93-163	40.7
Pheasant	12-37	—	—
Bobwhite quail	—	—	44.0
Common quail	40-85	249-494	42.2

(*Argusianus argus*) have no preen gland. A brush-like feather tuft that absorbs secretions from the gland is present on the uropygial papillae. This feather tuft is absent in the megapodes.

Some gallinaceous birds have unique skin appendages. Junglefowl possess marked unpaired carneous combs consisting of a wide intermediate layer, which is formed of a fibrillar network filled with mucus-like substances that impact elastic stability of the comb. The strongly vascularized corium and the epidermis cover the intermediate layer. Feathers are present on the comb bonnet in some domestic fowl breeds. The paired wattles of the throat are similar in structure to the comb (Fig 38.2). Like the comb, the size of the wattles is influenced by hormones, and both are better developed in cocks than in hens. Paired cheek or earlobes are located ventral to the auditory canal and are of varying colors. It has been suggested that the color of the earlobe is related to the color of the eggshell in *Gallus gallus*.

The structure of the skin appendages on the head and neck of turkeys varies from those described in junglefowl. These appendages have no elastic intermediate layer but do have superficial, muscular and vascular layers. The dewlaps of turkeys are smooth, increase and decrease in size and can change color. Turkeys have a single snood on the forehead that can readily increase or decrease in length. Numerous red caruncles are located on the poorly feathered blue skin of the head. A beard consisting of tough, dark bristles is present at the border between the neck and chest. Turkey hens have more poorly developed skin appendages than cocks, and a beard is found occasionally in older hens, probably as a result of hormonal changes.

In New World quail (Odontophoridae), the edge of the lower bill is serrated or slightly jagged. An osseous process, which can be large in some species or subspecies, exists near the junction of the upper bill and cranium of helmeted guinea fowl and some cracids (Cracidae). This helmet consists of a cone of spongy bone covered by the corium and a keratinized epidermis. The wattles of the helmeted guinea fowl (*Numida meleagris*) are white to light blue and, like the helmet, are larger in cocks than in hens. Some other phasianids,

Fig 38.2 | Developed comb and wattles in the rooster (foreground) and hen are shown. The comb of the male is typically more prominent and brightly colored; in this case, it is cyanotic due to feed impaction of the crop.

some megapodes (Megapodiidae) and some cracids also possess ornamental appendages of the head and neck. In some species, these appendages are visible only during mating displays. Some breeds of the domestic fowl, some megapodes, some francolins, (Peliperdixidae), some tragopans (Tragopanidae) and some pheasants (Phasianidae) have completely featherless heads and necks, or featherless areas of the head or neck. Unfeathered areas of skin frequently are colored. Many grouse species (*Bonasa* spp.) have red-colored supraocular combs. These unfeathered regions become swollen during mating season.

The cocks of many gallinaceous birds have spurs, which are osseous eminences originating from the tarsometatarsus and are covered by keratinized epidermis. If spurs occur in hens, they are poorly developed and often have no osseous component. The cocks' spurs are frequently sharp and can easily injure rivals, hens, clients or veterinarians. Cracids and grouse do not have spurs. In the common pheasant, annual rings are formed in the epidermis at the base of the spurs and can be used to determine the minimum age of the bird.

Adaptations to Low Temperatures

The feet and toes of grouse are feathered. In ptarmigans (*Lagopus* spp.), even the plantar surface of the foot is covered with fur-like feathers. Long nails and keratinous pins on both sides of the digits facilitate locomotion on snow. Dense plumage and a thick layer of subcutaneous fatty tissue help protect against the cold. Hair-like feathers cover the nostrils. In ptarmigans, shivering for the active production of body heat starts only below -12° C.

Plumage

The chicks of all gallinaceous birds are nidifugous (can ambulate and self feed) and hatch with a downy plumage. The deck feathers (tectrices), flight feathers (remiges) and tail feathers (rectrices) form the contour feathers of the plumage. The number of rectrices varies among different species: the domestic fowl has 7 pairs; the Bulwer's wattled pheasant has 12 to 16 pairs. Ornamental feathers can originate from different portions of the plumage, including tail coverts (peafowl), rectrices (many pheasants) and chin feathers (capercaillies). Birds that are indigenous to open terrain often have a patterned plumage that serves as camouflage. Some species like the golden pheasant show polychromatism of the plumage.

Dark periorbital feathers hide the eyes of many gallinaceous birds. Attempting to escape from predators by running or flying in open terrain is a poor defense; thus, most ground-dwelling gallinaceous birds remain immobile when predators approach, and flee only as a last-ditch effort to escape.

Gallinaceous birds generally have well-developed afterfeathers (hypopennae). In some cracids, the vanes of the first primaries are curved and narrow, which, when a bird flies, produce a unique sound that is used to mark its territory.

Most gallinaceous birds molt naturally once a year, generally after the breeding season. Gallinaceous birds retain their ability to fly during a molt. The secondaries are molted in a divergent pattern from an inner starting point. The rectrices are molted randomly. The willow ptarmigan lives in a subarctic-type habitat and molts three times a year in order to adapt to color changes in the environment, with the winter plumage being mainly white. Some grouse (capercaillies and ptarmigans) even molt the horny sheath (rhamphotheca) of the bill (in small pieces) after the breeding season. Ptarmigans also replace their nails. Molting of commercial chickens is often done on a scheduled basis to improve the level of egg production and quality of the eggshell.

Some birds (notably grouse [Tetraoninae], pigeons [Columbidae]) undergo a stress-induced physiologic response when attacked by predators, which results in release of the feathers (the shock or fright molt). The predator or handler is left with a collection of feathers and the bird escapes.

Gallinaceous birds normally fly at a low level, have a high-frequency wing flap and tire quite rapidly. Their flight is often limited to gliding for short distances. Some species lead a nomadic life. Birds that dwell in high mountainous regions in the summer usually move to lower altitudes in the winter. The only true migratory gallinaceous birds are the common quail (*Coturnix*

coturnix) and the Japanese quail. Some gallinaceous birds move by running, which is assisted by quick flapping of the wings. A normal cruising speed for the common pheasant would be 33 km/h (20.5 mph), while the common turkey cruises through the forest at 24 km/h (15 mph). The nidifugous chicks of the gallinaceous birds are able to fly shortly after hatching. The chicks of the phasianids first attempt to fly at the age of 10 to 16 days, and the cracid chicks start to fly 3 to 4 days after hatching. Megapode chicks, which are not tended by their parents, are able to fly short distances just after hatching.

LOCOMOTOR SYSTEM

The furcula (wishbone) of the domestic fowl is V-shaped and has a ventral process. In the crested and plumed guinea fowl (Numidinae), an indentation exists at the junction of the two clavicles. This indentation holds the U-shaped loop of the elongated trachea. The medial notch of the sternum extends far cranially, and fibrous membranes connect the lateral and medial notches. In this region, the sternum does not protect the liver, and injections, abdominocentesis or handling procedures must be carefully performed.

The ground-dwelling phasianids generally have a long femur, tibiotarsus and tarsometatarsus to facilitate ambulation, while the tree-dwelling cracids have shorter tarsometatarsi. The legs of all gallinaceous birds are well muscled. Cracids are active climbers, and other gallinaceous birds need strong feet and legs to scratch the ground in search of food. The toes of cracids and megapodes are on the same plane, whereas the first toe of the phasianids originates more proximally than the other digits. The first digit of the gallinaceous birds is oriented mediocaudally and the three other digits are directed cranially. Some breeds of the domestic fowl have five digits, with the additional digit being located medial to the first.

RESPIRATORY SYSTEM

Desert-dwelling gallinaceous birds such as sand partridges (*Ammoperdix heyi*) possess well-developed salt glands situated in an osseous indentation above the eyes. This extrarenal excretory organ for salt empties through a duct into the nasal cavity.

The cocks or both genders of some gallinaceous birds have elongated tracheas. The additional length produces a U-shaped or circular loop in the trachea that lies between the skin and the muscle layer in the ventral thoracic or cranial abdominal region. In helmeted curassows, the loop extends to the cloaca, and in some other cracids, it extends to the caudal end of the sternum. Crested and plumed guinea fowl and the common capercaillie also have elongated tracheas. Although the function of the loop is not fully understood, it may be involved in generating deep sounds.

The tracheobronchial syrinx of gallinaceous birds is a simple structure. The neopulmo, which is the phylogenetically younger portion of the lung, is well developed in Galliformes. A phylogenetic increase in the size of the neopulmo is accompanied by a decrease in the size of the caudal thoracic air sacs. The common turkey has a well-developed neopulmo and has no caudal thoracic air sacs. Four clavicular air sacs are recognized in gallinaceous embryos. In the common turkey, only two of the four clavicular air sacs merge with the unpaired cervical air sac, and two clavicular air sacs remain distinct. In other birds, all the embryonic clavicular air sacs merge into one. With these adaptations, the common turkey has only seven air sacs, while most gallinaceous birds have nine air sacs: the unpaired clavicular air sac, and the paired cervical, cranial thoracic, caudal thoracic and abdominal air sacs.

ALIMENTARY TRACT

Most gallinaceous birds have a pointed bill (rostrum) that is used to pick up food. In grouse, the bill is stronger and is used for cutting tough vegetable matter. In gallinaceous birds, the cere is usually limited to the base of the upper bill; however, in cracids, two-thirds of the bill is covered by the cere.

The tongue of gallinaceous birds is shaped like an acute triangle, is stabilized by a bone and has no intrinsic musculature. Most gallinaceous birds have a crop. This esophageal diverticulum is missing in small cracids and snowcocks, and in its place is a slight bulge in the diameter of the esophagus or only an increased stretchability of the esophagus. The sage grouse (*Centocercus minimus*) and some other North American grouse (*Artemisia* spp.) use a diverticulum in the middle part of the esophagus for territorial display and not for the storage of food. During display, the "inflatable esophageal air sacs" are inflated to expose featherless, brightly colored skin. The organ also may play a part in amplifying the voice.

The ventriculus and its associated musculature are well developed in most gallinaceous birds. Grouse and snowcocks (*Tetraogallus himalayensis*), which eat extremely rough food, possess the most heavily muscled ventriculi. The sage grouse, which feeds on soft food, has a thin-walled ventriculus.

The secretory ducts of the liver and the pancreas open into the duodenum. Gallinaceous birds have a gall bladder and two bile ducts. In the domestic fowl, the pancreas extends to the apex of the duodenal loop and

generally has three secretory ducts. The largest pancreas is found in gallinaceous birds that feed on grain.

All gallinaceous birds have well-developed ceca. Peristaltic movements of the small intestine and antiperistaltic movements of the rectum transport fluid and small food particles into the cecal lumen. The contents of the ceca are dark-colored and have a sticky consistency. The size of the ceca will increase or decrease, depending on the amount of crude fiber in the diet. In some species, bacterial digestion of cellulose occurs in the ceca. Species like grouse and snow cocks, which feed on foods with high amounts of crude fiber, have particularly well-developed ceca.

The cecal flora probably plays an important role in the synthesis of vitamins and the metabolism of nitrogen. Uric acid that enters the cloaca is transported into the ceca by antiperistaltic movements of the rectum and is used for the synthesis of amino acids produced by bacteria and available to the bird. The ceca are usually emptied once a day, typically in the morning.

URINARY AND REPRODUCTIVE SYSTEMS

The testicles are generally yellowish or white but can be pigmented in some species like the common capercaillie or in some breeds of the domestic fowl. The testicles enlarge during the breeding season. Fertile semen is not produced between breeding seasons. The ductus deferens and, in some species, an enlarged area of the caudal ductus deferens serve as reservoirs for the storage of semen. Gallinaceous cocks have a non-erectile phallus.

Husbandry

Most gallinaceous birds are best maintained in combination indoor and outdoor aviaries and can live to 6 to 20 years, depending on the species (Table 38.3). In general, the available space should be as large as possible. In some countries, law stipulates the minimum areas.

A pair of pheasants can be maintained and bred in an aviary with a floor space 4 x 6 m with an additional 4-m^2 shelter. A common pheasant cock with five to six hens needs 30 to 38 m^2. An aviary for peafowl should be at least 3 m wide x 3 m deep x 3 m high. These species are best maintained in open-air enclosures or big gardens. One pair of bobwhite (*Colinus virginianus ridgwagi*) or California quail (*Callipepla californica*) needs a minimum of 1.5 m x 1.5 m floor space. For grouse, small aviaries 4 m deep x 8 m wide are recommended, because these birds may injure themselves if they fly into netting at the high speeds attained in larger flights.

Table 38.3 | Longevity of Selected Gallinaceous Birds

Bird	Years
Peafowl	Approx. 20
Bobwhite quail	Approx. 6
Grouse	8-10
Common pheasant	10-18
Cracids	20+

Greg J. Harrison

Fig 38.3 | A pheasant is provided with protection from the elements in an enclosure with a simple A-frame shelter at a popular zoological garden. Note the bent toes, a form of metabolic bone disease from malnutrition.

Many Galliformes prefer to roost in elevated positions, making the height of an aviary important. Shelters should be provided to protect birds from sun, wind and rain (Fig 38.3). Tropical or subtropical species maintained in cold climates require an indoor aviary or, if kept outdoors in winter, a heated shelter. The mesh size of netting should be small enough to prevent a bird from placing its head through the mesh, and preventing a predator from injuring or killing the bird. It also should prevent the smallest predators from entering the aviary. Some gallinaceous birds, especially the common pheasant and many quail,[23] fly straight up when panicked. For this species, the top netting in an enclosure should be loose to provide some give and reduce the chances of head[24] (scalping) and neck injuries. An opaque barrier can be placed at the back of the aviary, extending up to one-half of the height, to provide extra visual security for the birds.

Ground dwellers like some quail, partridges and francolins do not need elevated perches. Perches should be placed far enough from walls or wire netting to prevent the tail or wing feathers from contacting these surfaces. Peafowl, Reeve's pheasant (*Syrmaticus reevesii*), argus pheasants and Phoenix fowl (a strain of red junglefowl) require especially high perches placed 3 to 4 m above the ground to accommodate their long tail feathers.

Sharp corners should be avoided in designing the aviary. Curved corners or dense bushes planted in the corners reduce the possibility of trauma.

Shrubs also help to landscape an aviary and provide shelter for the birds; however, the aviary should not be over-planted. Too many plants will make an aviary difficult to clean. Natural turfs are attractive but are not recommended when keeping birds that are highly susceptible to infectious diseases. An aviary with a concrete floor that is covered with an exchangeable layer of sand meets the needs of sensitive species (like grouse or the cheer pheasant) and is better than natural soil. Plants may be grown in containers that are removed when the aviary needs cleaning.

Snowcocks need large rocks for perching and shaping their bills. Some species like monals, eared pheasants (*Crossoptilon crossoptilon*) and the cheer pheasant (*Catreus wallichi*) use their upper bill to search the soil for roots and insects. If these birds are maintained on artificial substrate, natural abrasion of the bill will not occur and manual trimming will be necessary. Gallinaceous birds do not bathe in water. Most gallinaceous birds like to take dust or sand baths. The placement of abrasive materials on the plumage may function to lightly abrade and polish the edges of the feathers and may help reduce the number of external parasites as long as the sand itself is not contaminated. Insect powders should be used only if they are known to be nontoxic for the species concerned and only if the birds do in fact have parasites. In the winter, willow ptarmigan bathe in the snow.

Various bird species should generally not be mixed in one aviary because of possible interspecific aggression and the potential transmission of infectious agents. If species are combined, it is best to mix birds that do not compete for the same food or biotope and have originated from the same geographic region.[25] Ground-dwelling gallinaceous birds can be combined with bush- or tree-living species like thrushes, babblers, starlings, bulbuls and doves (with the exception of the ground pigeon); however, mixing of species is not recommended. Predatory species, including birds that feed on eggs, should not be combined with gallinaceous birds.

Silver pheasant (*Lophura nycthemera*), eared pheasant (*Crossoptilon auritum*), golden pheasant (*Chrysolophus pictus*), Lady Amherst's pheasant (*Chrysolophus amherstiae*), Elliot's pheasant (*Syrmaticus ellioti*), and Indian peafowl can be maintained in open-air enclosures that are fenced but not covered. Birds in open-air enclosures must have sufficient hedges, bushes or trees for protection. Higher trees should be available for roosting. Fruit trees or oaks (some are poisonous) provide a food source as well as cover. Clipping the wings before introducing it to new surroundings should reduce the flight capacity of a bird.

Losses to predators can occur in open-topped facilities, particularly with respect to chicks. Rare species should not be maintained in an open-topped enclosure. A breeder who uses open-topped enclosures should expect that the loss of a bird to a predator is the responsibility of the breeder and not the fault of the predator. Some gallinaceous birds are noisy, especially the Indian peafowl and guinea fowl during the breeding season, and should be maintained in secluded areas to avoid complaints from neighbors.

Nutrition

Many diseases and problems in captive Galliformes are directly or indirectly related to malnutrition. Breeders of gallinaceous birds should be aware of the natural foods consumed by any species maintained in captivity. Conclusive data on the nutritional demands (with respect to maximal egg or meat production and not for longevity and appearance) is available only for the domestic fowl, domestic turkey and the Japanese quail. Some information is available for the domestic guinea fowl and less has been determined for the common pheasant. All nutritional guidelines for other gallinaceous birds are based on experience.[26] Special care must be exercised when feeding commercial turkey and/or chicken feeds. Levels of calcium, protein and energy vary considerably among the starter, grower, layer and finisher rations. As well, many commercial poultry feeds contain antibiotics and other drugs (anticoccidials) that may be harmful to some birds or other animals on the premises.

Generally, the protein requirement increases at the beginning of the mating season because of egg and semen production. After the breeding season, the amount of protein in the feed should be gradually reduced. With any change in the diet, the new food should be mixed slowly into the daily diet until the conversion is complete.

"EASY" BIRDS

Many gallinaceous birds are omnivorous. The nutritional requirements of common pheasant, golden pheasant, peafowl, guinea fowl, turkeys, partridges and New World quail are relatively easy to provide. Commercial diets for domestic fowl, domestic turkey, common pheasant and Japanese quail are available in many countries. Pellets designed for turkeys can be used in species without special requirements. Adding fresh green plants to the diet

provides the birds with nutritional diversity. Grass or corn silage also can be offered in small quantities. During the breeding season, the diet should contain 20 to 25% crude protein. Outside the breeding season, a maintenance diet containing less than 20% crude protein is best. Commercial diets for the domestic turkey are usually better suited for pheasants than diets developed for domestic fowl. Feeding is best accomplished by providing small portions of the diet several times a day in the non-breeding season and offering food ad libitum during the breeding season.

Most New World quail are primarily seed eaters and are easy to feed. Forest-adapted species may be largely insectivorous and have higher and more specific protein requirements in comparison to other gallinaceous birds. Cracids are mainly, but not exclusively, vegetarians. They can be sustained on pellets containing 21% crude protein supplemented with fruits but no grains. During the breeding season, they are fed soybean paste, chopped hard-cooked eggs, chopped meat or mealworms (larvae of the meal beetle) with calcium as a supplement. The primarily meat diet of these birds results in odiferous feces. Megapodes can be fed a commercial poultry diet.

Birds with a High Protein Requirement

Some gallinaceous birds like peacock pheasants (*Polyplectron bialcaratum*), argus pheasants and the roulroul (crested wood partridge) do best with high-protein diets. In addition to high-protein turkey or pheasant diets, adult peacock pheasants should be fed mealworms, chopped meat, fruits and a small quantity of grain. Green plants are rarely consumed by these species. The roulroul is fed a commercial soft feed for insectivorous birds, mixed with live insects, chopped hard-cooked eggs and chopped meat with calcium as a supplement.

"DIFFICULT" BIRDS

Some gallinaceous birds consume almost exclusively vegetable material. The koklass (*Pucrasia marolapha*), the blood pheasant (*Ithaginis cruentus*), snowcocks, tragopans and grouse are examples. Feeding these species with game bird pellets or, even worse, with commercial diets for domestic fowl and turkeys results in obesity, reduced fertility and imbalances in the intestinal microflora. These species should be maintained only where natural-type foods are available year-round. These gallinaceous birds should be fed large amounts of fresh vegetables. Pellets should be provided only in small quantities, if at all. Koklass naturally feed on ferns, grasses, leaves, mosses, buds and berries. In captivity they should be provided soft green plants, fruits and berries, and no grains. In the summer, grasses and lucerne (alfalfa) can be provided. Spinach, romaine lettuce and fresh, frozen vegetables can be substituted in the winter months. Free-ranging blood pheasants feed on mosses, lichen, ferns, grass tips and conifer needle-buds. They browse constantly in planted aviaries. Their chicks feed on these plants immediately after hatching.

Tragopans consume oak trees, bamboo sprouts, grasses, mosses, oak nuts, berries and a few insects. In captivity, tragopans can be fed lucerne, grasses, cucumbers, apples and different kinds of berries. In the spring, summer and autumn, grouse feed on a variety of plants. In the winter, most grouse species are restricted to consuming one or a few plant species. During the winter season, the spruce grouse, capercaillies and other grouse species feed almost exclusively on conifer needles, the black grouse on birch buds, and ptarmigans on buds from different deciduous trees (birch, alder, willow).

Captive grouse should receive natural foods or at least large amounts of leaves, grass and berries supplemented with a limited quantity of pellets and grain. Capercaillies and ptarmigans require a diet high in crude fiber. Even with strict attention to the diet, the bacterial fecal flora in capercaillies in captivity is similar to the fecal flora of the domestic fowl and differs substantially from the fecal flora of free-ranging capercaillies. The tannin and essential oil content of natural food plants may support the growth of autochthonous intestinal flora in free-ranging grouse. In the sage grouse, leaves and sprouts of the North American big sagebrush are the sole winter food and the main portion of food in the summer.

Some commercial poultry diets contain coccidiostatic agents. Halofuginone is toxic for the common pheasant, guinea fowl and the common partridge. Monensin is toxic for guineafowl. The presence of antimicrobial agents can be life-threatening in species that depend on a functional cecal flora and fauna (eg, grouse) for proper digestion. In general, the effects of coccidiostats and other medical feed supplements on gallinaceous birds have not been sufficiently studied. It is safer to provide food without these potentially toxic supplements.

All gallinaceous birds should have access to grit, when not fed strictly an artificial diet. The grit container should be emptied and refilled regularly because birds select only stones that are suitable for their body mass. Pellets or complete rations have an adequate supply of calcium and should not be supplemented with lime or crushed shell. Fresh, clean water must be available at all times for all species.

CHICKS

During their first few weeks of life, free-ranging gallinaceous chicks feed mainly on live invertebrates like insects,

larvae of insects, worms and snails in order to obtain the protein levels needed to sustain rapid growth. Starting at 5 to 6 weeks of age, the protein requirements begin to decrease and the intake of carbohydrates increases to meet energy requirements. By 6 months of age, most young gallinaceous birds have reached a mass equivalent to that of adults. The quantity of carbohydrates in the diet must then be reduced to prevent obesity.

Feed should be provided to newly hatched chicks on a large, flat plate on which they can move around and practice pecking. By 5 to 7 days of age, food can be offered in larger containers. The change from the plate to larger containers should occur by offering feed in both containers at the same time. Small chicks may drown in large water containers.[28] Reducing the drinker depth by placing stones or glass marbles in the container will reduce losses.

Chicks of unpretentious species (common pheasant, peafowl, guinea fowl) are initially fed a starter diet like turkey starter (28% crude protein) and are transferred to a lower protein diet like turkey grower (18-20% crude protein) from the eighth to eighteenth week of age.[29]

Chicks of the vegetarian species are difficult to feed. It is best to provide these birds with foods that are similar to those eaten by their free-ranging counterparts. A diet composed of turkey starter mixed with mealworms, ant cocoons, chopped hard-cooked eggs, diced romaine lettuce, spinach, dandelion and other green plants is a viable substitute. In several species (some grouse), chicks obtain food by pecking at the ground and by cutting off parts of plants with the bill. In these species, it is important that chicks be provided intact plants that are placed in the ground or tied in bundles to facilitate natural food-gathering behavior. Chicks that are to be released into the wild must be introduced to their natural foods to prevent starvation. Perhaps chicks are imprinted with food shapes and colors, or at the least they learn what foods to consume from the hen.

The chicks of some gallinaceous birds will not pick downward in the first days of life. This is because peacock pheasants, crested argus, great argus and some other gallinaceous hens feed their chicks for several days after hatching. Argus pheasant chicks can be enticed to peck by offering live food (mealworms). Monal chicks fed mealworms will pick at their siblings' toes.

Reproduction

Some gallinaceous birds breed easily in captivity, while others rarely reproduce. Breeding failures are an indication that the birds are not provided a suitable environment or there may be medical problems with the individual birds.[30] Some pheasant and quail species are approaching a level of domestication that is advantageous for both the captive animal and the breeder. Comparatively, "semi-domesticated" animals are of no value if offspring are to be released to the wild with the intent of reintroducing genetic diversity into dwindling populations. Genetic selection and breeding to achieve color variants increase the expression of genetic abnormalities, semi-lethal factors and susceptibility to disease. The clutch size and incubation times for commonly maintained gallinaceous birds are listed in Table 38.4. Parameters for artificial incubation are listed in Table 38.5.

GENERAL CONSIDERATIONS

Gallinaceous birds to be used for breeding purposes should be introduced to each other before the breeding season in surroundings that are novel to both the males and females. The female should be introduced to the enclosure a few hours prior to the male. In some species, it is possible to keep several males together if there are no females present. If females are present, only one male should be housed in an aviary or in one compartment. In monogamous species, only a single pair should be housed together.[28]

Males of some species are very aggressive. During the breeding season, they may attack other males, other bird species or even the keeper. Pursuit by the male and mock escape by the female is normal behavior in some species like eared pheasants and francolins. If there is insufficient space for the hen to escape, she may be injured or killed by the cock. Beak trimming or restricting the flight capabilities of the male can prevent injuries to the hen, but are inferior procedures to providing adequate space for a pair of birds to behave normally. Densely planted aviaries that provide a hen with areas to hide still may have inherent problems. Fiberglass panels leaned against the wall or concrete tubes provide similar protection and are easy to clean.

For species in which there are substantial differences in body size between the genders, aviaries can be designed to allow the hen to visit the cock when she wishes. Small holes just big enough for the hen are used to connect adjacent enclosures. This allows the hen to enter the cock's enclosure while preventing the cock from entering the hen's area. This is an effective method for breeding birds like the common capercaillie. In some species, the hen chooses the most attractive of several cocks and if only one cock is available, breeding may not occur if the hen does not like the cock. In some species, the visual or acoustic presence of other males is necessary to stimulate display and mating behavior.

Table 38.4 | Clutch Sizes and Incubation Times of Gallinaceous Birds

	Species	Clutch Sizes	Incubation Time (days)
Megapodiidae			
	Alectura lathami	25-30	46-54
Cracidae			
	Ortalis spp.	3	26-28
	Penelope spp.	2-3	27-29
	Aburria spp.	2-3	unknown
	Penelopina spp.	2	unknown
	Oreophasis spp.	2	unknown
	Nothocrax spp.	2	28
	Mitu spp.	2	29-30
	Pauxi spp.	2	30
	Crax spp.	2	29
Phasianidae			
Perdicinae	*Lerwa* spp.	5-7	unknown
	Tetraogallus spp.	5-8	26
	Tetraophasis spp.	4	unknown
	Arborophila spp.	3-5	20-21
	Perdix spp.	8-20	24-25
	Alectoris spp.	8-14	24-26
	Bambusicola spp.	4-6	18-20
	Francolinus spp.	4-8	19-21
	Pternistis spp.	3-9	18-20
	Scleroptila spp.	3-6	22
	Dendroperdix spp.	4-9	19
Numidinae	*Guttera* spp.	8-10	unknown
	Numida spp.	8-12	27
	Acryllium spp.	10-14	23-24
	Agelastes spp.	12	unknown
Pavoninae	*Afropavo* sp.	3-4	26-27
	Pavo spp.	3-5	28-30
Meleagridinae	*Meleagris* spp.	8-15	28
Argusianinae	*Polyplectron* spp.	2	18-23
	Rheinardia spp.	2	25
	Argus spp.	2	24-25
Phasianinae	*Chrysolophus* spp.	5-12	22-23
	Phasianus spp.	8-12	22-24
	Graphephasianus spp.	6-12	24
	Syrmaticus spp.	7-15	24-25
	Colophasis spp.	6-8	25-28
	Lophura spp.	5-15	22-25
	Crossoptilan spp.	4-14	24-28
	Catreus spp.	9-14	26

	Species	Clutch Sizes	Incubation Time (days)
Phasianidae (Continued)			
Lophophorinae	*Lophophorus* spp.	4-5	27
Pucrasiinae	*Pucrasia* spp.	5-7	20-21
Ithagininae	*Ithaginis* spp.	5-12	27
Gallinae	*Gallus* spp.	5-8	19-21
Tragopaninae	*Tragopan* spp.	4-10	28-31
Ptilopachinae	*Philopachus* spp.	4-6	unknown
	Peliperdix spp.	2-6	unknown
	Ortygornis spp.	4-8	18-19
	Perdicula spp.	4-8	22
	Cryptoplectron spp.	4-7	16-18
	Ammoperdix spp.	8-14	22-24
	Synoicus spp.	4-12	20-22
	Coturnix spp.	7-14	16-20
	Margaroperdix spp.	5	unknown
	Caloperdix spp.	8-10	18-20
	Melanoperdix spp.	5	unknown
	Rollulus spp.	4	18-20
	Haematortyx spp.	8-9	unknown
	Rhizothera spp.	5	unknown
Odontophorinae	*Colinus* spp.	7-28	22-23
	Callipepla spp.	9-17	22-23
	Oreotyx spp.	6-15	24-25
	Philortyx spp.	8-12	22-23
	Dendrortyx spp.	4-7	28-30
	Odontophorus spp.	4-5	26-27
	Dactylortyx spp.	5	unknown
	Cyrtonyx spp.	6-16	24-25
Tetraoninae	*Tympanuchus* spp.	5-17	24-25
	Bonasa spp.	11	24
	Tetrastes spp.	7-11	23-25
	Centrocercus spp.	7-13	25-27
	Dendragapus spp.	7-10	24-25
	Falcipennis spp.	4-10	21-22
	Lagopus spp.	6-9	20-23
	Lyrurus spp.	7-10	26-27
	Tetrao spp.	5-12	26

Most gallinaceous birds incubate eggs on the ground and should be provided with flat trays containing moss, foliage or hay for nesting material. Tragopans, the Congo peafowl (*Afropavo congosis*), the bronze-tailed peacock pheasant (*Pavo cristatus*), the crested argus pheasant, the mikado pheasant (*Syrmaticus mikado*), the Salvadori's pheasant (*Lophura inornata*) and the cracids nest in trees. A box placed approximately 150 cm from the ground and filled with hay and foliage can be used as an artificial nest. A slanted limb should be provided for easy access to the nest. Nests of ground- and tree-nesting birds should be inconspicuous to provide the pair with visual security, but should be placed such that the birds can easily look out.

Most gallinaceous birds are non-determinant layers and if the first clutch of eggs is removed, the hen will lay a second and sometimes a third clutch. Hatching is genetically determined and should not normally be assisted. Because gallinaceous chicks are nidifugous, the family can stay together only if all the chicks hatch at the same time. Synchronization of the hatch dates can occur by two mechanisms: 1) The hen does not incubate the

Table 38.5 | Parameters for Artificial Incubation of Some Gallinaceous Birds

Species	Incubation Chamber		Hatching Chamber	
	Temp. (°C)	Humidity (%)	Temp. (°C)	Humidity (%)
Common pheasant (*Phasianus colchicus*)	37.5	60	37	85
California quail (*Callipepla californica*)	38.5-39.0	50-60	—	80
Common capercaillie (*Tetrao urogallus*)	37.5	60-70	36.5-37.0	80-90
Black grouse (*Tetrao tetrix*)	37.4	55-60	—	85-90
Ruffed grouse (*Bonasa umbellus*)	37.5	60-65	—	70-75
Chukar partridge (*Alectoris chukar*)	37.5	65	37	85

Greg J. Harrison

Fig 38.4 | The red junglefowl hen is commonly used by aviculturists to incubate the eggs of species that commonly abandon their eggs.

clutch until the last egg has been laid, allowing the eggs to cool (which slows the process of embryogenesis); or 2) The chicks in a clutch synchronize hatching through audible signals. This latter process occurs in species like the Japanese quail. When sounds are heard from other eggs, the chicks increase the speed of hatching. When no sounds are heard from other eggs, the most developed chicks reduce their speed of hatching. Most gallinaceous chicks are independent by 3 months of age. The exception is the megapode chick, which is independent immediately after hatching.

Foster Breeding

The hens of some gallinaceous birds are unreliable brooders in captivity. Cracid, common pheasant and nearly all species of New World quail hens are not reliable brooders in captivity. These hens can be encouraged to produce two or three clutches per year instead of one by using foster parents or an incubator for hatching eggs. Chinese silk fowl (*Bambusicola thoracica*) and bantams make excellent foster parents (Fig 38.4). Domestic turkey hens can be used to incubate the eggs of larger gallinaceous birds. Small and fragile eggs should be placed under golden pheasant hens, which are cautious brooders and excellent care providers. During the last week of incubation, the eggs of tropical birds being raised in dry climates should be moistened with a clean mister once a day. After hatching, the hens and chicks can be placed in a small enclosure that is moveable and can be placed on fresh grassy areas on a daily basis. Chicks are prone to chilling the first few days posthatch and must have supplemental body heat from the attending hen or a heat lamp where appropriate.

The disadvantages of foster parenting are as follows:

- Crushing of small, fragile eggs by heavy or clumsy adults
- Premature cessation of brooding if the natural incubation period of the foster hen is shorter than the fostered eggs
- Trauma or death of the chicks if the hen recognizes them to be strange (this is a particular problem when behavioral incompatibilities exist between the hen and chicks)
- Transmission of infectious agents between hen and chicks
- Inappropriate imprinting

Placing the eggs in an incubator for the last third of the incubation period can reduce infanticide and disease transmission (this method is often used for grouse). A hen of the same species should rear chicks that are to be released into the wild.

For many pheasants, the percentage of carbon dioxide in the incubator must be increased up to approximately 1% (verified with a gas detector) during the last 2 days of incubation. This is achieved by reducing the intake of fresh air and serves to stimulate the hatching process. Chicks should be taken out of the incubator immediately after hatching.

SPECIFIC REPRODUCTIVE CHARACTERISTICS

Megapodes

Megapode eggs differ from those of other gallinaceous birds, owing to their uncommon brooding biology. The eggs are not incubated by the parents but by solar heat, fermentation heat or geothermal energy. One egg can reach a size of up to 17% of the hen's body mass. The eggs are thin shelled and contain a large yolk that is rich in lipids. Cocks or both sexes begin constructing an induction mound out of foliage and earth when the air temperature and atmospheric humidity reach a certain level. The hens lay their eggs every 2 to 3 days in previously prepared holes, which are quickly covered after ovi-

position. Eggs are deposited in a mound, with the pointed pole downward, and they are not turned during incubation. They do not have a fixed air chamber or chalaza.

The birds may determine the temperature of the mound, and perhaps other parameters, with the bill or tongue. The mean temperature in the incubation mound is around 34° C. The incubation mound is cooled when needed by scratching holes. This allows carbon dioxide to escape and oxygen to enter. The incubation period varies from 45 to 90 days, depending on the temperature in the mound. Brush turkey chicks leave the mound 24 to 30 hours after hatching. Normally, megapode chicks do not come into contact with their parents, which function only to care for the incubation mound. The chicks join their siblings that have hatched at around the same time. Megapodes are sexually mature by 1 year of age.

The Australian brush turkey (*Alectura lathami*) is easy to maintain and breed in captivity, and is the most common captive representative of the megapodes. This species is monogamous. In one breeding season, an Australian brush turkey hen lays about 25 to 30 eggs.

Cracids

Cracids are Central and South American species that are considered monogamous. The breeding season lasts from March until July. Most nests are well hidden in a fork or branch of a tree, but some species are ground-nesters. Only the hen incubates the eggs. A clutch consists of two to three eggs, which are rough shelled with wide pores and a uniform white color. Newly hatched chicks are immediately able to climb trees. The family stays together until the next breeding season. Sexual maturity occurs by 2 years of age.

Turkeys

The common turkey is polygamous. Behavior of free-ranging birds is dramatically different from that of domesticated breeds. The brain volume of domesticated turkeys is 3% smaller (brain:body weight ratio) than that of their wild-type conspecifics. The nest is formed of a flat depression in the soil and may be padded with leaves, grass or twigs. The chicks are able to fly at 2 weeks of age. Several hens, together with their offspring, typically associate in a flock in the winter. The young birds leave their mother before the next breeding season. Young turkeys are sexually mature at 2 years of age.

New World Quail

New World quail are monogamous. Both parents participate in building the nest and brooding the chicks. Young birds are sexually mature by 1 year of age, in some species even earlier. Outside the breeding season, the gregarious New World quail live together in large family groups (coveys). At the beginning of the breeding season, the older cocks become very aggressive toward young cocks. Captive bobwhite quail have become polygamous and it is possible to keep one cock with two hens, indicating the effects of domestication.

Grouse

Some grouse species like ptarmigan, ruffed grouse, hazel hen (*Tetrastes bonasia*), spruce grouse (*Falcipennis canadensis*) and blue grouse (*Dendragapus obscurus*) are monogamous. In these species, cocks should not be allowed to see or hear other cocks. Hazel hen males may attack the female if a rival can be heard but not seen. Other grouse species are polygamous. In these species, the hen chooses one cock from a group of displaying males. One cock may be chosen to mate with several hens. Hens in captivity breed best when allowed to choose between two or more cocks. The cocks, which are housed in different compartments of an aviary, may see and hear each other if there are enough hiding places for the hens. In most grouse only the hen provides chick care. The chicks of different species can be distinguished by the varying color patterns on the head and back plumage. Most grouse are sexually mature at 1 year of age. Crossbreeding between different genera and species occurs in free-ranging birds. Similarities in the appearance and display behavior of hens seem to induce cocks to crossbreed. Hens will choose cocks of another species if a representative of their own species is not available.

Peafowl

The Congo peafowl is monogamous. The nest is always built in a tree. Both parents care for the chicks. The Indian (*Pavo cristatus*) and the green peafowl (*Pavo muticus*) are polygamous. In captivity, it is possible to keep one cock with four to five hens. The hens care for the clutch and the chicks, which mature slowly. Hens reach sexual maturity in the second year and cocks in the third year of life. The green peafowl is more aggressive than the Indian peafowl, but has a more pleasant call.

Pheasant

Most pheasant species are polygamous. One common pheasant cock can be kept with five to six hens (Fig 38.5). The hens make poor care providers in captivity. They tend to be indiscriminate in the placement of eggs and will not incubate the eggs. Young common pheasants are sexually mature at 1 year of age. Free-ranging golden pheasants are monogamous, but in captivity one cock can be kept with three to four hens. The hens are exceptional care providers and defend their chicks. Young golden pheasant hens are sexually mature within 1 year, cocks within 2 years. Lady Amherst's pheasant

Greg J. Harrison

Fig 38.5 | A male Reeves pheasant has worn a path in its aviary in an attempt to find mates or compete with other males.

cocks and hens can be aggressive during the breeding season. Only a few of the birds found in captivity are purebred. Both male and female argus pheasants, peacock pheasants and the copper pheasant establish and defend their own territories. Males should be introduced to females for only a short time during the breeding season to prevent aggressive behavior and traumatic injuries to both genders.

Junglefowl and Domestic Fowl

Junglefowl can either be monogamous or polygamous. The hens can breed year-round, but the main breeding season is from February to May in the northern hemisphere. A red junglefowl cock can be maintained with three to four hens. The young birds are independent at an age of 4 months and sexually mature after the first year. Many domestic fowl breeds have lost much of their brooding behavior, and eggs must be artificially incubated.[32]

GENDER DETERMINATION

Many gallinaceous birds show a marked sexual dimorphism. The size (height and width), the body mass (weight), the color of the plumage, the shape of certain feathers, the presence of spurs, and the length and color of the tail feathers assist in gender determination between adults of some species (Table 38.6). In some breeds of domestic fowl, fertile cocks may have plumage that resembles that of hens.

Highly skilled individuals can determine gender by examining the cloaca in 1-day-old chicks or adults. The cloacal examination in newly hatched chicks of small bird species must be carefully done. Holding a chick too tightly can cause asphyxiation. Restraint of a chick for gender determination should start by gently pressing on the abdomen from both sides distal to the keel bone to stimulate defecation. The procedure is then similar to that described for Anseriformes (see Chapter 36, Management of Waterfowl).

Behavioral clues like dominance and certain mating rituals may suggest a gender but are not always indicative. Under certain conditions, the hens of some gallinaceous birds behave like and can have plumage like the males. DNA analysis or endoscopic examination of the gonads provides definitive determination of gender in species with similar morphologic characteristics.

Table 38.6 | Gender Determination of Selected Species of Gallinaceous Birds Without Marked Sexual Dimorphism[28]

Genus	Plumage Identical	Plumage Similar	Differences
Megapodiidae			
Alectura spp.	X		Cocks have neck appendages
Cracidae			
Ortalis spp.	X		Voice of cock is deeper
Penelope spp.	X		In some species, iris colors differ
Nothocrax spp.	X		In cocks, the tracheal loop is palpable
Pauxi spp.	X		In hens, plumage is sometimes a red phase
Phasianidae			
Numidinae			
All genera	X		Cock's call has 3 syllables; hen's call has 2 syllables
Argusianinae			
Polyplectron spp.		X	Hen's plumage is dull; cocks have spurs
Phasianinae			
Crossoptilon spp.	X		In general, cocks have spurs
Catreus spp.		X	Cocks have long, sharp spurs
Ptilopachinae			
Ptilopachus spp.		X	
Perdicinae			
Tetraogallus spp.		X*	In some species, cocks have short spurs
Arborophila spp.		X*	In some species, cocks have short spurs
Bambusicola spp.		X*	
Francolinus spp.	X		
Pternistis spp.	X		In some species, cocks have spurs
Scleroptila spp.	X		Cocks have spurs
Ortygornis spp.	X		Cocks have spurs
Coturnix spp.		X	
Odontophorinae			
Odontophorus spp.		X*	
Tetraoninae			
Tympanuchus spp.		X	
Bonasa spp.		X	
Tetrastes spp.		X	
Lagopus spp.	X		Only in winter

*Some species of the genus are very similar, but not identical

ARTIFICIAL INSEMINATION

Artificial insemination is of economic importance in the domestic turkey and domestic guinea fowl. Domestic turkey cocks, like domestic fowl cocks, are fertile year-round, except during periods of extreme heat or during the molt period. Domestic guinea fowl cocks are not fertile all year and artificial insemination is used to induce year-round production.

The semen is collected by massaging the caudal region of the back or the abdomen, followed by stimulation of the cloaca. Fecal contamination of the semen may occur. It is best to collect the semen directly from the spermatic duct with a syringe and a blunted hypodermic needle. The semen may be diluted with Ringer's or Tyrode's solution by up to a factor of three.

Avian semen has a short half-life and must be used as quickly as possible. The semen is introduced with a syringe and a blunted hypodermic needle into the hen's oviduct. It is best to inseminate the hen just after she has laid an egg. This ensures that the oviduct is open, providing the semen with unrestricted access to the infundibulum.

Restraint

Cocks with spurs can injure handlers, especially when they become increasingly aggressive during the mating season. The beak also can serve as a weapon. Although serious injuries are rare, the face and the eyes of handlers should always be protected from a bird's beak, even in small species. The legs of a gallinaceous bird should be the initial focus for restraint.

Catching gallinaceous birds in an aviary can be done gently with a long, hooked stick. The birds should never be restrained by the feathers alone. The whole body must be secured to prevent a shock molt. Shock molt is most common in tail feathers but other feathers can be involved. Birds can be nearly "bald" after several failed restraint attempts. In larger species, the base of the wing is fixed along side the body with one hand and the legs are controlled with the other hand. The abdomen should be supported from below. If assistance is not available, a large bird can be restrained against one's body. Birds can usually be calmed by placing a loose-fitting, lightweight cotton sock over the head to reduce vision.

Disease Considerations

Gallinaceous birds are susceptible to a wide variety of viral, bacterial, mycoplasmal, parasitic, chlamydial, rickettsial and fungal agents (Table 38.7). Information on these diseases may be found in other literature.

NUTRITIONAL DISEASES

Vitamin C deficiency does not occur in most birds; however, it has been reported in willow ptarmigan chicks and may occur in other grouse chicks. Though the chicks are able to produce endogenous vitamin C (as all gallinaceous birds probably can), the internal production is not sufficient in the first weeks of life, and has to be augmented by the intake of vitamin C from natural food plants (eg, blueberries). Clinical signs of vitamin C deficiency are abnormal behavior, enteritis, ruffled plumage, weakness of the wings and legs, bone fractures, retarded growth and death before the age of 4 weeks. Characteristic necropsy findings include weight loss, pale and edematous skeletal muscles, petechial hemorrhage in the muscles and mild subcutaneous edema. Fractures in the diaphysis of the humerus, radius, ulna, femur and tibiotarsus with massive callus formation and lateral twisting of the tibia also may occur. Feeding chicks natural foodstuffs high in vitamin C will prevent deficiency. Birds with poor quality diets often develop pododermatitis (Fig 38.6).

INTEGUMENT CONCERNS

Amputation of the comb or the wattles may be indicated following extensive injury, infection or frostbite. Adequate hemostasis is necessary to prevent fatal hemorrhage. Occasional trimming of the keratinous tip of the bill is necessary if the horny layer grows too fast, or if insufficient abrasive materials are available to facilitate

Fig 38.6 | Chickens on poor-quality diets often develop bumblefoot. The pain from the bumblefoot causes chickens to lay down frequently, which may lead to sternal ulcers.

Table 38.7 | Checklist of Infectious Diseases in Gallinaceous Birds

Viruses

Poxviridae
- Avian pox

Herpesviridae
- Infectious laryngotracheitis
- Marek's disease
- Quail Herpesvirus
- Turkey Herpesvirus

Adenoviridae
- Quail bronchitis
- Quail necrotizing hepatitis
- Inclusion body hepatitis
- Egg drop syndrome (infectious salpingitis)
- Marble spleen disease
- Hemorrhagic enteritis of turkeys
- Chicken splenomegaly
- Adenovirus infection of the blue grouse, Guinea Fowl

Parvoviridae
- Parvovirus infection of chickens
- Parvovirus-like infection of turkeys

Circoviridae
- Infectious anemia

Reoviridae
- Viral arthritis
- Other reovirus infections
- Rotavirus infections

Birnaviridae
- Infectious bursal disease

Togaviridae
- Eastern and St. Louis encephalitis
- Avian serositis
- Louping-ill
- Israel turkey meningoencephalitis

Coronaviridae
- Coronaviral enteritis of turkeys (bluecomb disease)
- Infectious bronchitis

Rhabdoviridae
- Rabies

Paramyxoviridae
- Newcastle disease
- PMV-2 infection
- PMV-3 infection
- Turkey rhinotracheitis
- Swollen head syndrome

Orthomyxoviridae
- Avian influenza, fowl plague

Retroviridae
- Leukosis
- Reticuloendotheliosis
- Lymphoproliferative disease of turkeys

Picornaviridae
- Avian encephalomyelitis
- Turkey viral hepatitis
- Infectious nephritis

Bacteria

Staphylococcus spp.
- Staphylococcosis

Streptococcus spp.
- Streptococcosis

Mycobacterium avium
- Tuberculosis

Eryspelothrix rhusiopathiae
- Erysipelas

Listeria monocytogenes
- Listeriosis

Clostridium spp.
- Ulcerative and necrotic enteritis (*Cl. colinum* and *Cl. perfringens*)
- Botulism (toxin of *Cl. botulinum*)

Escherichia coli
- Colibacillosis
- Coligranulomatosis

Salmonella spp.
- Salmonellosis

Klebsiella spp.
- *Klebsiella* infection

Yersinia pseudotuberculosis
- Pseudotuberculosis

Pseudomonas spp.
- *Pseudomonas* infection

Aeromonas hydrophila
- *Aeromonas* infection

Bordetella avium
- Bordetellosis (turkey coryza)

Campylobacter spp.
- Avian hepatitis

Borrelia anserina
- Spirochetosis

Treponema spp.
- Infectious typhlitis in chickens

Pasteurella spp.
- Fowl cholera

Actinobacillus salpingitidis
- Actinobacillosis

Haemophilus spp.
- *Haemophilus* infection

Francisella tularensis
Tularemia

Mycoplasma

Mycoplasma spp.
- *Ureaplasma* sp.

Chlamydia

Chlamydophila psittaci
Chlamydiosis

Rickettsia

Coxiella burnetii
- Query (Q) fever

Aegyptianella pullorum
- Aegyptianellosis

Mycoses

Aspergillus spp.
- Aspergillosis

Candida albicans
- Candidiasis

Dactylaria gallopavo
- Dactyloriosis

Trichophyton spp.
- Favus

Mycotoxicoses

Toxins of *Aspergillus* spp., *Penicillium* spp., *Fusarium* spp. and others

Parasites

Protozoal Parasites
- *Trypanosoma avium*
- *Spironucleus meleagridis*
- *Histomonas meleagridis* (Blackhead disease)
- *Trichomonas* spp.
- *Chilomastix gallinarum*
- *Entamoeba* spp.
- *Endolimas* spp.
- *Eimeria* spp.
- *Toxoplasma gondii*
- *Sarcocystis* spp.
- *Cryptosporidium* spp.
- *Haemoproteus* spp.
- *Leucocytozoon* spp.
- *Plasmodium* spp.

Metazoal Parasites

Trematodes
- *Prosthogonimus* spp.

Cestodes
- *Davainea proglottina*
- *Raillietina* spp.
- *Amoebotaenia cuneata*
- *Choanotaenia infundibulum*
- *Hymenolepis* spp.
- *Metroliasthes lucida*
- *Fimbriaria fasciolaris*

Nematodes (in digestive tract)
- *Capillaria* spp.
- *Trichostrongylus tenuis*
- *Heterakis* spp.
- *Ascaridia* spp.
- *Gongylonema ingluvicola*
- *Cheilospiruro* spp.
- *Dispharynx nasuta*
- *Tetrameres* spp.
- *Subulura* spp.

Nematodes (in respiratory tract)
- *Syngamus trachea*

Nematodes (in the eye)
- *Oxyspirura* spp.

Nematodes (in other locations)
- *Aproctella stoddardi*
- *Singhfilaria hayesi*

Acanthocephalans
- *Mediorhynchus papillosus*

Arthropods
- External parasites like lice, fleas, flies, mosquitoes, midges and ticks occur in most gallinaceous birds. Mites occur above all in intensively reared gallinaceous birds, predacious bugs in some gallinaceous birds.

normal wear. The excessive horn is pared off prudently with a sharp knife without cutting into the viable parts of the bill.

Cannibalism may occur in some Galliformes and is characterized by vent picking, feather pulling, toe picking, head picking and egg eating. Overcrowding, incorrect feeding, an inappropriate daylight cycle, high light intensity, poor housing conditions (eg, high proportion of toxic gases in the air), genetic predisposition and other factors may all promote cannibalism.

Beak trimming has been successful in commercial poultry production facilities, when performed by experienced personnel, to reduce the incidence of cannibalism and resulting injury. Removal of the comb and wattles is sometimes performed by commercial poultry breeders to reduce losses associated with aggressive behavior, especially associated with males. These procedures may not be suggested in gallinaceous birds raised for hobby. When performed improperly, these procedures may interfere with the bird's ability to eat, may result in infection and even affect the bird's social ranking in the flock. The bill is not only important for the uptake of food but also has sensory functions and is necessary for preening. Damage to the beak should be considered a substantial handicap. In most cases, cannibalism can successfully be prevented by correcting the deficiencies in the birds' environment; however, once feather picking is started, beak trimming or other management changes such as separating the birds or use of an anti-picking ointment[a] may be necessary to break the cycle (Fig 38.7).

Trimming of the flight feathers in one wing can be used to prevent birds from escaping from open aviaries or to reduce the mobility of an aggressive cock during the breeding period. Usually all but the outermost two primaries and the innermost three secondaries are transected, creating an effective and cosmetic wing trim. With one wing trimmed, the bird is unbalanced and cannot gain speed during flight. Because the feathers will be replaced during the next molt, trimming must be repeated annually in adults. Under certain circumstances, it may be necessary to trim both wings. Other methods like pinioning or cutting the short tendon of the *extensor carpi radialis* make birds permanently unable to fly. The client should be made aware of the consequences of these procedures. Birds unable to fly or ambulate normally would be more susceptible to attack by pets and wild animals such as raccoons. In many countries, such practices are outlawed for humane reasons.

VACCINATION CONSIDERATIONS

Vaccination programs used in the commercial broiler, turkey and layer industries may be very comprehensive and complex. These enterprises often have large numbers of birds, 1 million or more in many cases, on a single premises. Thus, disease prevention rather than treatment is the goal. Disease prevention is achieved through strict programs whereby disease organisms are prevented entry onto the premises by using biosecurity and by vaccination. Vaccination programs for each farm unit are designed specifically to provide maximum protection to the birds, while being economic and causing minimum stress to the flocks. Disease challenge risk in an area also must be considered in the program design.

Fig 38.7 | Anti-picking lotion may help reduce cannibalism.

In collections where gallinaceous birds are maintained, the goal likewise should be disease prevention by preventing entry of disease organisms onto the premises. Vaccination, as the second line of defense, is important in reducing losses in high-risk areas where disease challenge occurs. The primary obstacle in vaccinating collections of gallinaceous birds is availability of quality commercial vaccines. Vaccines for fowl are readily available for commercial use, but not for smaller collections. Due to market considerations, poultry vaccines are produced in vials containing 3000 or more doses per vial. Once vaccines are reconstituted, in the case of lyophilized products such as pox, infectious bronchitis, Newcastle, infectious bursal disease and infectious laryngotracheitis, they must be administered promptly (within 2 hours). As only small numbers of birds may be vaccinated at a time, vaccination may not be feasible. Other vaccines such as Marek's require storage in liquid nitrogen and must be administered to chicks promptly after hatching to be effective. These vaccines also must be used within 1 hour after careful thawing and mixing of the vaccine, as they are very fragile, cell-associated products. In the case of Marek's disease, birds will be exposed to this ubiquitous field virus soon after hatch in most instances, thus prompt vaccination is essential if it is to be efficacious (Fig 38.8).

Vaccination programs for gallinaceous birds in smaller collections are therefore limited to diseases that are more

Fig 38.8 | Vaccination of recently hatched chicks against Marek's disease.

virulent in nature and are high risk in a particular region. A disease such as infectious bronchitis, which causes only a mild and transitory respiratory condition in some gallinaceous birds and in which multiple serotypes are circulating, often is not considered for vaccination.

Diseases commonly considered for vaccination if endemic or if virulent strains are of concern in the area include Marek's disease, infectious laryngotracheitis, pox, Newcastle and infectious bursal disease (Figs 38.9a,b).

Although live-type vaccines are labile and must be stored under refrigerated conditions in the dark, have expiration dates, are sold in doses of 5000 or more per vial, and the contents of the vaccine must be used promptly following reconstitution, vaccination would often be indicated for valuable collections. Veterinarians, often working with clients from a number of collections, may divide the lyophilized vaccine pellet prior to reconstitution, using aseptic technique, to reduce vaccine waste and cost.

Vaccinations are often used in response to post mortem diagnosis. This is not always possible (Fig 38.10).

Product Mentioned in the Text

a. Anti-Pick Lotion, Vineland Laboratories, 1-800-846-3547, www.vinelandlabs.com

Figs 38.9a,b | A chicken is presented with respiratory disease. Note exudates around the eye and sinuses. Infectious bronchitis is mild and transitory and is not usually considered for vaccination.

Greg J. Harrison

Fig 38.10 | Two coincidental findings in a dead rooster: an eye worm and a fly "strike". Fly eggs will hatch into maggots. The cause of death was not determined.

References and Suggested Reading

1. Amand WB: Husbandry of Galliformes. *In* Fowler ME (ed): Zoo and Wild Animal Medicine. Philadelphia, WB Saunders Co, 1978, pp 295-296.
2. Amand WB: Surgical problems. *In* Fowler ME (ed): Zoo and Wild Animal Medicine. Philadelphia, WB Saunders Co, 1978, pp 304-306.
3. Amand WB: Disease description. *In* Fowler ME (ed): Zoo and Wild Animal Medicine. Philadelphia, WB Saunders Co, 1978, pp 309-319.
4. Amand WB: Clinical pathology. *In* Fowler ME (ed): Zoo and Wild Animal Medicine. Philadelphia, WB Saunders Co, 1978, pp 320-322.
5. Aschenbrenner H: Rauhfusshühner. Hannover, Verlag M & H Schaper, 1985.
6. Barnes EM, Impey CS: The occurrence and properties of uric acid decomposing anaerobic bacteria in the avian caecum. J Appl Bact 37:393-389, 1974.
7. Baumel JJ, et al: Nomina Anatomica Avium. London, Academic Press, 1979.
8. Calnek BW, et al (eds): Diseases of Poultry. Wolfe Publishing Ltd, 1991.
9. Cootes ME, et al: Intestinal synthesis of vitamins of the B complex in chicks. Brit J Nutr 22:493-500, 1968.
10. Fenna L, Boag DA: Filling and emptying of the Galliforme caecum. Can J Zool 52:537-538, 1974.
11. Franchetti DR, Klide AM: Restraint and anesthesia. *In* Fowler ME (ed): Zoo and Wild Animal Medicine. Philadelphia, WB Saunders Co, 1978, pp 303-304.
12. Gasaway WC: Cellulose digestion and metabolism by captive rock ptarmigan. Comp Biochem Physiol 54A:179-182, 1976.
13. Gylstorff I: Blut, Blutbildung und Blutkreislauf. *In* Mebner A, Harfiel W (eds): Handbuch der Geflügelphysiologie. Gustav Fischer Verlag Jena, 1983, pp 280-393.
14. Gylstorff I, Grimm F: Vogelkrankheiten. Stuttgart, Verlag Eugen Ulmer, 1987.
15. Hanssen I: Micromorphological studies on the small intestine and caeca in wild and captive willow grouse (*Lagopus lagopus lagopus*). Acta Vet Scand 20:351-364, 1979.
16. Hanssen I, et al: Vitamin C deficiency in growing willow ptarmigan (*Lagopus l. lagopus*). J Nutr 109:2260-2278, 1979.
17. Johnsgard PA: The Grouse of the World. Croom Helm, University of Nebraska Press, 1983.
18. Johnsgard PA: The Quails, Partridges, and Francolins of the World. Oxford, Oxford University Press, 1988.
19. Kaal TF: Geschlechtsmerkmale bei Vögeln. Hannover, Verlag M & H Schaper, 1982.
20. Keller H: Hornringe am Sporn der Hühnervögel zur Altersbestimmung. Lebensmitteltierarz 5:11, 1954.
21. Kösters J, Korbel R: Zur Frage des Schnabelkürzens beim Geflügel. Tierärztl Umschau 43(11):689-694, 1988.
22. Korbel R: Praxis der Injektions- und Blutentnahmetechniken am Vogelpatienten. Tierärztl Prax 18:601-611, 1990.
23. Korbel R: Zwangsmassnahmen beim Vogelpatienten. Teil 1, Teil 2, Tierärztl Prax 20:59-64. 164-170, 1992.
24. Korbel R, Kösters J: Einige von Tierhaltern geforderte oder durchgeführte Operationen an gesunden Vögeln unter tierschutzrechtlichen Aspekten. Tierärztl Prax 17:380-381, 1989.
25. Lopez JE: The Cracidae. Avic Mag 85:210-215, 1979.
26. Michigan State University: Managing gamebirds. Extension Bulletin E 692. Resource Development Series 44. Michigan State University, 1974.
27. Pendergast BA, Boag DA: Seasonal changes in the internal anatomy of spruce grouse in Alberta. The Auk 90:307-317, 1973.
28. Raethel HS: Hühnervögel der Welt. Melsungen, Neumann-Neudamm, 1988.
29. Schales C: Untersuchungen über die antibakterielle Wirkung ätherischer Öle and hydrophiler Inhaltsstoffe aus Koniferennadeln auf Bakterien aus dem Kot von in Gefangenschaft gehaltenen Auerhühnern (Tetrao urogallus L, 1758) *in vitro*. Diss Med Vet, München, 1992.
30. Schales K: Untersuchungen über die aerobe Flora und C*lostridium perfringens* im Kot von freilebenden und in Gefangenschaft gehaltenen Auerhühnern (*Tetrao urogallus L,* 1758). Diss Med Vet, München, 1992.
31. Snyder RL: Feeding and nutrition. *In* Fowler ME (ed): Zoo and Wild Animal Medicine. Philadelphia, WB Saunders Co, 1978, pp 296-302.
32. Sturkie PD: Body fluids: Blood. *In* Sturkie PD (ed): Avian Physiology. New York, Springer Verlag, 1986, pp 102-121.
33. Spearman RIC, Hardy JA: Integument. *In* King AS, McLelland J (eds): Form and Function in Birds. Vol. 3, London, Academic Press, 1985, pp 1-56.
34. Vollmerhaus B, Kinowatz F: Haut und Hautgebilde. *In* Nickel R, et al: Lehrbuch der Anatomie der Haustiere. Band V. Anatomie der Vögel. Berlin, Verlag Paul Parey, 1992, pp 16-49.
35. Vallmerhaus B: Spezielle Anatomie des Bewegungsapparats. *In* Nickel R, et al: Lehrbuch der Anatomie der Haustier, Band V. Anatomie der Vögel. Berlin, Verlag Paul Parey, 1992, pp 54-154.
36. Vollmerhaus B, Sinowatz F: Atmungsapparat. *In* Nickel R, et al: Lehrbuch der Anatomie der Haustier, Band V. Anatomie der Vögel. Berlin, Verlag Paul Parey, 1992, pp 159-175.
37. Vollmerhaus B, Sinowatz F: Verdauungsapparat. *In* Nickel R, et al: Lehrbuch der Anatomie der Haustier, Band V. Anatomie der Vögel. Berlin, Verlag Paul Parey, 1992, pp 176-221.
38. Waibl H, Sinowatz F: Harn- und Geschlechtsapparat. *In* Nickel R, et al: Lehrbuch der Anatomie der Haustier, Band V. Anatomie der Vögel. Berlin, Verlag Paul Parey, 1992, pp 224-264.
39. Wallach JD, Boever WJ: Game birds, waterfowl and ratites. *In* Diseases of Exotic Animals II. Medical and Surgical Management. Philadelphia, WB Saunders Co, 1983, pp 830-889.
38. Wiese DR. Fuller MK: Artificial insemination in the brown-eared pheasant. World Pheasant Assoc J III, 1977-1978, pp 90-95.
41. Wolters HE: Ordnung Phasianiformes. *In* Die Vogelarten der Erde. Hamburg, Paul Parey, 1975-1982, pp 100-109.
42. World Pheasant Association (WPA): Census 1982.

CHAPTER
39

Management of Canaries, Finches and Mynahs

PETER SANDMEIER, Dr med vet, Dipl ECAMS; PETER COUTTEEL, DVM

Johan Van der Maelen

Fig 39.1 | A white recessive color canary is shown.

Johan Van der Maelen

Fig 39.2 | A red color canary is shown.

The order Passeriformes (Table 39.1) is the largest of all avian orders and consists of 63 families comprising 5206 different species. The order is extremely heterogeneous, containing granivorous, insectivorous, frugivorous, omnivorous and carnivorous species ranging in size from a few grams to over 1 kilogram.

The species commonly seen in private veterinary practice include canaries, New World finches, Old World finches, waxbills, cardinals and mynahs (see Table 39.1).

The canary (*Serinus canarius*) can be considered a domesticated species. Spanish monks in monasteries as far back as 1402 achieved first breeding. Following a French expedition to the Canary Islands, this bird was introduced to France and Italy and later into the rest of Europe.[17] Nowadays, canary fanciers have a wide range of activities, including preservation of old and rare breeds as well as breeding new color mutations.[32] An important aspect of their hobby is showing and judging their birds. There are three groups of canaries: song canaries, color canaries and form canaries.

Song canaries include Harzer (Germany), Malinois (Belgium), Timbrado (Spain) or American singers (USA). Color canaries are divided into two groups. The melanin group includes black, brown, agate and isabel birds. The lipochrome group includes white (Fig 39.1), red (Fig 39.2) and yellow (Fig 39.3) canaries.

Form canaries are a diverse group, distributed among "frill," "type," "shape," "crested" (and "crest-bred") and "feather pattern" birds. Frills include Parisian frill, Frisé du nord, Frisé du sud, Frisé Suisse, Gibber Italicus, Giboso Espagnol, Padovano and Fiorino (Fig 39.4). Type


Peter Coutteel

Fig 39.3 | A yellow color canary is shown.

canaries include Belgian bult, Scotch fancy, Münchener, Japan hoso, Lancashire, Yorkshire, Berner and Rheinländer. Shape canaries include border, Fife fancy, Norwich, Raza Espagnol and Irish fancy. Crested and crest-bred birds include Gloster consort, Gloster corona and German crest. The Lady Gouldian Finch is a popular finch amongst fanciers (Fig 39.5).

Anatomy and Physiology

Some anatomic and physiologic characteristics observed in canaries and finches include an extremely high basal metabolic rate and high body temperature (42° C, 108° F). The foot of the passerine bird is anisodactylous: 3 digits forward and 1 digit backward.[105] All passerine species have a crop, but in canaries and finches, the crop is much smaller than in psittacines, pigeons or chickens. There is no production of crop milk, but crop contents are regurgitated to feed the young. A proventriculus and ventriculus are present; the cecae are rudimentary. The spleen is oblong rather than spherical. Nestling Estrildidae finches have species-specific luminous mouth patterns to attract their feeding parents.[71] The right and left nasal sinuses do not communicate as they do in Psittaciformes. Song canaries and other species are able to produce two sounds simultaneously by using both bronchial ends of the syrinx. The neopulmo as well as the paleopulmo divisions of the lungs are well developed in Passeriformes. Most Passeriformes have seven air sacs as opposed to the nine air sacs in Psittaciformes. The cranial thoracic air sacs are fused to a single air sac, as are the clavicular air sacs.[71]


Peter Coutteel

Fig 39.4 | A form canary, Parisian frill, is shown.


Greg J. Harrison

Fig 39.5 | Lady Gouldian finches are dimorphic with the male in front showing the most and brightest color. The female in the background has less color especially on the head.

Table 39.1 | Selected Passeriformes Families

Family	Scientific name	English name	German name
Corvidae		Crow, jay, magpie	Krähen
Sturnidae		Starling	Stare
	Gracula religiosa	Hill mynah	Beo
Passeridae		Sparrow	Sperlinge
Ploceidae		Weaver	Webervögel
Estrildidae		Waxbill	Prachtfinken
	Poephila guttata	Zebra finch	Zebrafink
	Chloebia gouldiae	Lady Gouldian finch	Gouldsamadine
	Erythrura spp.	Parrot finch	Papageienamadine
	Lonchura striata var. domestica	Society or Bengalese finch	Japanisches Mövchen
Fringillidae		Finch	
	Serinus canarius	Canary	Kanarienvogel
	Carduelis spinus	Siskin	Zeisig
	Carduelis carduelis	Goldfinch	Stieglitz
	Carduelis chloris	Greenfinch	Grünfink
	Fringilla coelebs	Chaffinch	Buchfink
	Pyrrhula pyrrhula	Bullfinch	Gimpel
Emberizidae	*Melopyrrha nigra*	Cuban finch	Kubafink
	Cardinalis	American Cardinal	Kardinale

Reproduction[19]

BREEDING FACILITIES

Nowadays, canaries are mostly bred indoors, often within a building in the garden or close to the house (Fig 39.6), sometimes inside the house in a cellar or an attic. The birds are kept in pairs in breeding cages (50 x 40 x 40 cm) and require artificial lighting. In a traditional European facility, each cage would have drinking water, seed mixture, soft food, cuttlefish bone, grit, nesting material, conditioning seeds and bath water that is changed on a regular basis. The most suitable substrates for the cage bottom are newspaper, plain brown paper or small pieces of wood. Some breeders use cages with wire bottoms. Wire is easy to clean but can be dangerous for the youngsters sitting on the bottom of the cage as entanglement of the feet may occur.

Some breeding facilities have cages with nest boxes inside the breeding cage; others have breeding cages with the nest boxes outside (Figs 39.7-39.9). In order to collect or candle the eggs and leg band the youngsters, it is easier to have outside hanging nest boxes. Additionally, breeders have found that leaving a clutch with the parents until weaning leads to fewer babies produced each year. If they try to let the parents breed with the babies present, the subsequent eggs may be infertile because the babies interfere with the breeding process in some way. The parents may be disturbed and not incubate the eggs properly or the parents may abuse and pluck the youngsters, trying to drive them away. To overcome this, the babies are maintained in accessible but separate enclosures. Where outside hanging "baby cages" are used, the youngsters are fed through the wire. The youngsters of the first clutch are put in a cage next to the parents, separated by a wall containing 2-cm-diameter openings (Fig 39.10). The parents can prepare for the next round of eggs and babies more successfully, while continuing to feed the first clutch through the holes until they are completely weaned.

After weaning, the youngsters are put together in large cages so they can exercise their flight (Fig 39.11). Individual perches are very important to prevent picking. Often an older male is placed together with the youngsters to feed those that still beg for food.

BREEDING PERIOD

In Europe most breeders artificially extend the daily photoperiod. The reasons for using this technique of light manipulation are: 1) to control the breeding period and start reproduction before the natural breeding season (so most intensive work is done before July summer holidays); 2) to prepare the youngsters for the exhibitions at

Peter Coutteel

Fig 39.6 | A canary breeding facility with netting on the windows helps prevent mosquito problems.

Peter Coutteel

Fig 39.7 | Breeding cage separates youngsters of the first clutch in a baby cage (left) from the breeding pair preparing the second round (right).

Peter Coutteel

Fig 39.8 | Between every two breeding cages (with inside hanging nest pans), there is a common baby cage for the youngsters in a canary breeding facility.

Peter Coutteel

Fig 39.9 | A nest box, outside the main cage, makes access easier.


Peter Coutteel

Fig 39.10 | Youngsters are fed by their parents on the other side through little holes in the separation wall of the cages.

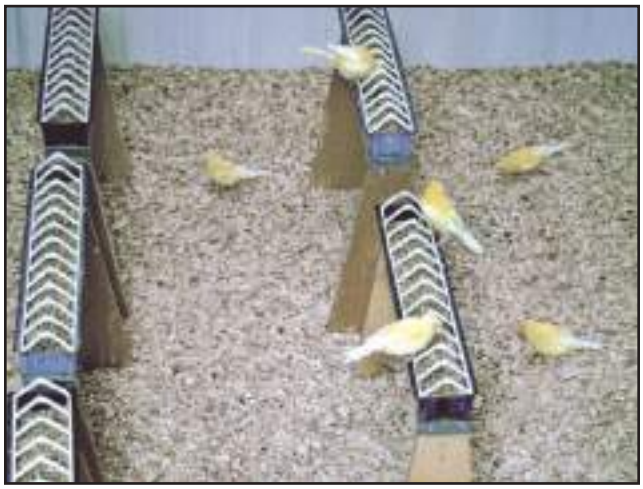

Peter Coutteel

Fig 39.11 | Young canaries are housed in a large cage for flight exercise. Often an older male is placed together with the youngsters to feed them. The substrate is small pieces of wood.

the end of the year (molt period has to be completed prior to exhibition); and 3) to produce more clutches and youngsters. (*Ed. Note: These techniques are used to improve profit and may not be ideal for the parent birds' over-all health).*

TECHNICAL ASPECTS OF LIGHT

The spectrum of optical radiation lies between 10^2 nm in the ultraviolet (UV) and 10^6 nm in the infrared (IR) spectrums. This wavelength range is subdivided into seven bands: UV-C, UV-B, UV-A, visible light, IR-A, IR-B and IR-C. The UV-B band in natural sunlight allows for the multistep conversion of 7-dehydrocholesterol into 1,25 dihydrocholecalciferol (vitamin D_3), which is then reabsorbed by the skin or ingested during preening.[98] Vitamin D_3 is very important for calcium metabolism. If there is a lack of incoming sunlight, full spectrum lamps can be used to induce the internal vitamin D production in the skin. Also, environmental contamination can be reduced by UV light. The action spectrum for inactivation of microorganisms reaches its maximum at about 265 nm (DNA absorption). If artificial light is provided for breeding birds, the kind of radiation produced by the lamps is an important factor.

Luminance

A minimum luminance (500 to 1000 lux) is needed (lux = lumen/m^2). Lumen gives an indication of the total amount of light produced by a light source per second. Adding the lumen of all lamps present in the breeding room and dividing it by the square meters of the room will give an idea of the quantity of lux. Sunlight produces approximately 100,000 lux.

Frequency

Normal fluorescent lamps do not give continuous light, but flicker like a stroboscope at approximately 50 times per second (50 Hz). The human eye does not discern this frequency and the effect on the behavior of birds is not completely known. However, recent studies reveal that birds may have a spatial difference of 160 frames per second.[63]

The stroboscopic effect of fluorescent light may lead to stress and may negatively influence the general condition of the bird. If many lamps are used at the same time or a combination of bulb lamps and fluorescent light is used, the stroboscopic effects will be less marked. The latest development is the HF (high frequency) lamp. These lamps have a frequency of 28,000 Hz, have a longer life and make dimming possible. When using artificial light, a dimmer should be used to simulate dawn and twilight. In Belgium, 92% of the breeders are using fluorescent lamps and 8% are using bulb lamps.[18]

Color Temperature

Color temperature is the measure for descriptions of "warm" or "cold" light and is expressed in degrees Kelvin. Examples of color temperature include incandescent light (2700° K), warm white (3000° K), cool white (4000° K), daylight (5000° K), and cool daylight (6500° K). The higher the color temperature of the light (higher degrees Kelvin), the more blue the color of the light and the more UV light produced. Cool daylight[b] is ideal.

Ambient Light Temperature

Fluorescent lamps give the highest light output at a temperature of 20° C (68° F). These lamps are very sensitive to temperature. At a low ambient temperature, the buffer gases inside the lamp will disintegrate and there will be

less light output. Therefore, the use of multiple lamps simultaneously is recommended, in case of defects.

Nightlights

A small amount of light (7 W) during the night has a calming effect. If a bird is startled or otherwise disturbed, this lighting will allow it to regain its perch.

Photoperiodic Stimulation

The photoperiod is the ratio of light to dark. A 15:9 ratio means 15 hours of light and 9 hours of darkness. The photoperiod is very important in those species of birds that originate from zones with seasonal changes. In temperate-zone birds such as canaries, ovarian development and testicular growth are synchronized by a combination of increasing day length, the presence of a partner and an available nesting site.[93]

The normal physiologic pathway for photoperiod stimulation has been described.[93] Retinal photoreceptors give stimulation through the optic nerve to the hypothalamus. Releasing hormones from the hypothalamus stimulates the adenohypophysis to produce gonadotropins, which, in turn, stimulate the gonads to release sex hormones. Another factor that can stimulate the hypothalamus is the presence of light rays that penetrate the spongy bones of the head.[93]

These stimulating factors encourage testicular growth in the male bird (up to five times in size) and production of fertile sperm. In the female bird, strong development of the ovaries and follicular growth occur. Experience shows that male canaries need a slightly longer exposure to extended daylight than females. This can be obtained by placing the males in isolation 2 weeks in advance, so that light stimulation may begin earlier. The reason that males need a longer preparation period is probably due to the fact that, in nature, males receive extra incentives, such as the song of another male while marking out their territories and trying to attract a partner. These incentives are often not present in an artificial breeding situation.

The Role of Daylight Length

Most problems in canary breeding are due to errors in the manipulation of the light cycle. Canaries need a minimum of 14 to 15 hours of daylight to begin breeding (nest building and production of eggs). With this amount of light, they are also able to feed their youngsters adequately and raise them properly. The cycle of light is also a major factor in determining whether breeding is sustained. If the length of daylight is submitted to fluctuations, the birds may receive conflicting hormonal incentives and negative endocrine feedback. The result can be an early molt and the birds may cease breeding. There are two methods of manipulating the length of daylight.[18]

Gradually Increasing Daylight Length:

Using this technique, the amount of daylight is gradually increased on a weekly basis. Depending on how quickly this is done, it may take a period of 2 months to extend the 8 to 10 hours of natural daylight to 15 hours. If a weekly addition of 30 minutes (5 additional minutes per day) is used, it will take approximately 10 weeks to obtain this result. This means that the fancier needs 2 to 3 months preparation before breeding can begin. Gradually increasing the length of the day is closest to natural stimuli and is used by more than 80% of the fanciers. Fifteen hours of daylight length appears to be ideal. Poor annual breeding results with higher chick mortality occur when the daylight length exceeds 17 hours.

Immediate Increase to Full Daylight Length:

The daylight length can also be increased suddenly from 10 to 15 hours. In this case, the birds reach breeding condition after 3 to 4 weeks, but most are unable to maintain good results throughout the full breeding season. However, some fanciers do have good results with this method. This method of sudden increase, used by approximately 10% of the breeders, often leads to poor fertilization of the first clutch, which normalizes subsequently, and higher mortality of females.

BREEDING CONDITIONS

Normally, canaries will begin breeding when the following conditions are fulfilled: photoperiod stimulation and appropriate minimum daylight length (as discussed), maturity, good health, an acceptable partner, presence of nest and nesting material and a minimum temperature of 15° C (59° F), during daytime.

Maturity

Most canaries will reach sexual maturity in a few months but good fertility will only start at approximately 10 months of age. Sometimes there are fertility problems when birds born in the last clutch of the former year are bred too early in the following year.

General Health

Many diseases and breeding failures in captive Passeriformes are the result of husbandry problems. Medication is often incorrectly used to attempt correction of husbandry-based problems. Primary infectious disease is less commonly encountered. The ideal avian veterinarian/aviculturist relationship starts before the breeding season when the birds are routinely examined. A physical examination is performed to determine whether each bird is in good condition. Examination includes the head (eyes, ears and nares), abdomen (liver, gastrointestinal tract),


Peter Coutteel

Fig 39.12 | A nesting material ("sharpie") of small white fibers of cotton is well accepted by canaries.


Peter Coutteel

Fig 39.13 | Small white fibers of cactus (*Agave rigidus*) are another source used for nesting material.

feathers (state of molt, external parasites and discolorations), wings (lacerations, feather cysts), feet and digits (hyperkeratosis, pox lesions and pododermatitis). Crop swabs are collected to determine if trichomoniasis or candidiasis is present. Fecal samples are taken for bacteriology, and blood samples are collected by venipuncture for serologic diagnosis of paramyxovirus and avian influenza. Finally, a fecal examination is performed to assess for coccidiosis, macrorhabdosis, helminth infections, yeast, protozoal cysts (*Giardia* sp.) and cochlosomiasis (diagnosis of cochlosomiasis requires fresh, warm stool). Sometimes a fecal Gram's stain is performed to interpret the levels of gram-positive and gram-negative bacteria and for the detection of *Candida* and *Macrorhabdus* spp.

Acceptable Partner

In canary breeding, there are generally few problems with the acceptance of the partner. Even during the breeding season, the female bird allows copulation with a different male for each round. If necessary, male birds can be used for several females simultaneously, with a maximum of three females for each male.

Nest and Nesting Material

Breeders use several different types of nest pans. Plastic nest pans have a very smooth surface that is easy to clean and makes movement of the nest possible. Often, holes are drilled in the bottom of the nest in order to attach the nest with wire. Stone nest pans can be well impregnated with a water-soluble insecticide. Sometimes self-made wooden or bamboo nest pans are used. They have the disadvantages of being difficult to clean and disinfect and must be changed after every clutch. Several types of nesting material are available. "Sharpie" consists of small white fibers of cotton, washed and cut in small pieces of approximately 3 to 4 cm (Fig 39.12). This nesting material is well accepted by canaries, and most Belgian breeders utilize it. Sisal fiber, of the plant *Agave rigidus*, is a very fine organic nesting material (Fig 39.13). It is important to note that constriction of a digit can occur due to entanglement in the fine fibers. If not treated, vascular necrosis and digit loss can result. Horsehair and moss are no longer used because they cannot be disinfected.

Temperature and Humidity

Most fanciers maintain the temperature in the breeding room at approximately 15° C (59° F) at the start of the breeding season. If the temperature is higher, the females start laying eggs even before pairing. Temperature is regulated with various heating devices (electrical, central heating, gas and fuel oil). During the breeding season and summer, the temperature may fluctuate. Therefore, good ventilation should be provided to remove exhaust gas and to eliminate temperature extremes. The temperatures should range from 15° to 25° C (59-77° F). In the authors' experience the temperature should not exceed 35° C (95° F).

The humidity in the breeding room should be kept within the range of 60 to 80%. Maintaining the humidity at the lower end of this range minimizes the development of pathogens. It is important to have sufficient humidity at the time of hatching. Therefore, breeders often moisturize the eggs just prior to hatching with a spray of warm tap water in the nest or by plunging the eggs for a second into a cup of warm water (40° C, 104° F).

PREPARATION FOR THE BREEDING PERIOD

Trimming

Before pairing birds, each bird should be physically

Peter Coufteel

Fig 39.14 | The pericloacal region of a canary (Gloster corona) is trimmed 1 cm ventral to the cloaca to prevent the feathers from interfering in semen transfer.

examined and eventually "trimmed". Because of the heavy plumage of some canary varieties (especially type canaries), it is preferable to trim the plumage surrounding the pericloacal region approximately 1 cm above the cloaca and horizontally prior to breeding (Fig 39.14). This reduces the potential for the caking of excrement around the vent and may increase the possibility of copulation. The nails should be checked and cut shorter if necessary so that good positioning is possible during copulation. Loose perches should be repaired prior to nail trimming. Trimming of the eye feathers can also be important so that the male can see the female. Sometimes there is an overgrowth of the upper beak, which can be trimmed with an electric grinding tool.[a] Traditionally, breeders used cuttlebone for that purpose, but recent nutritional experience shows the problem is more complex than just calcium deficiency and a lack of beak exercise. Nutritional assessment is important in birds with overgrown beaks.

Disinfecting

The breeding cages should be disinfected before introducing birds. No single disinfectant is active against all microorganisms or useful in all situations, so the chemical must be carefully chosen. Commonly used and readily available disinfectants include sodium hypochlorite (household bleach), quaternary ammonium products, chlorhexidine, glutaraldehyde and formalin.

Cleaning and disinfecting the breeding cages, perches and nest pans should be performed prior to introducing the birds. Also, prevention of red mites, northern mites and lice with an appropriate insecticide is advised. A common product utilized in Belgium is permethrin. Alternative pest control agents include insecticidal soaps used for plants, fresh garlic and diatomaceous earth. When using diatomaceous earth, the unpolished form (used for swimming pool filters) should be selected. When this form of diatomaceous earth is dusted onto the birds, the needle-like points penetrate the insect's exoskeleton causing desiccation. To reduce mosquitoes, mosquito netting or screened windows may be necessary. The carbon dioxide generator has advanced outdoor control. Female mosquitoes feed on blood, to which they are attracted by exhaled carbon dioxide. A new machine uses bottled gas to generate the CO_2 bait. The mosquitoes are then trapped in an inescapable collection bottle. If used as directed and combined with traditional preventive measures, mosquito population control is achieved.

Some canaries are housed outside in open aviaries, in which case, disease control and medicating individuals can be impractical. It is difficult to completely eliminate infectious agents once they are introduced into a planted aviary. Free-living birds and rodents may be transmitters of protozoal diseases, helminth infestations and bacterial infections.

PAIRING BIRDS

It is important to predetermine the sex of the individual birds in order to ensure they are paired appropriately. In males, the caudal end of the ductus deferens forms a mass called the seminal glomerulus. During the breeding season, the seminal glomerula push the cloaca walls into a "cloacal promontory" (Fig 39.15). Females do not develop this projection and have a flatter vent (Fig 39.16). The male is placed into the female's cage when the birds are ready for pairing. In the classic situation, one male and one female are together for the whole breeding season and rear the youngsters together. Alternatively, one high-quality cock may be used for pairing with several females. After copulation, the male is separated, and each female will rear her youngsters alone. Fertile sperm may be stored in the female's sperm glands (tubular glands within the oviductal wall located at the uterovaginal junction) and may fertilize eggs for approximately 8 to 10 days.[93]

The regulation of sexual behavior is altered by the effects of social interactions (eg, song of other male birds), which can exert powerful effects on reproductive success and overall well-being of the individual birds.[80]

Genetic Considerations

There should also be appropriate genetic selection of breeding birds regarding origin, color, type and feathers. Feather cysts are usually seen in the Frill, Norwich and other soft feather breeds and are believed to be a genetic disorder. Likewise, cataracts in canaries may have a genetic origin.[33]


Peter Coufteel

Fig 39.15 | A male canary is trimmed in the pericloacal region. The cloaca is perpendicular to the tail and points down, a "cloacal promontory".


Peter Coufteel

Fig 39.16 | A female canary is also trimmed in the pericloacal region, but the cloaca points in the same direction as the tail.

Other genetic abnormalities may cause mortality. One example of this is seen when pairing crested canaries such as the gloster (with crest = corona, without crest = consort).[71] The corona gloster is heterozygous for the autosomal crest gene. Birds that are homozygous for the crest gene die. Pairing a corona with a consort yields 50% corona and 50% consort. Pairing two corona phenotypes will yield 25% consort, 50% corona and 25% dead chicks. Another example of genetic mortality occurs with the dominant white color gene. Canaries that are homozygous for the white gene die,[71] while heterozygous birds are white. There is 100% mortality in chicks if two heterozygous dominant white birds are paired. If white birds are paired with other colors, 50% of the chicks will be heterozygous for the dominant white gene and 50% will be the other color.

EGG COLLECTION

During the egg-laying period, eggs are collected and replaced with artificial eggs while the female continues to lay her clutch. The eggs are kept in a cool place (15 to 20° C, 59 to 68° F, humidity 60 to 80%), in numbered boxes with seed at the bottom and put back into the nest when the female starts sitting. This is done to achieve uniform growth in the youngsters. The average clutch size of type canaries is 4 eggs. Color canaries often have a clutch size of 4 to 7 eggs. One should avoid writing on the eggs, especially with an alcohol pencil. Also, soft cloth or rubber gloves should be used while handling eggs in order to avoid contamination (eg, hand creams and cosmetics), which can result in the death of the chick.

Nutrition

Most passerines are primarily seed-eating birds. They may consume up to 30% of their body weight in food daily (compared with 10% for larger parrots). The basal metabolic rate is approximately 65% higher than in non-passerines. Granivorous birds are believed to need grit for digestion.[32]

The majority of commercially available seed mixtures are multideficient.[32] Seeds are deficient in vitamins A, D_3, E, K, lysine and methionine and have a poor Ca/P ratio. In Belgium, seed mixtures for canaries contain canary seed (62%), Niger seed (2%), rapeseed (22%), hemp seed (3%), peeled oats (8%) and linseed (3%). During the winter, 50% canary seed is mixed with this seed mixture to prevent obesity. Research has shown the white canary has a much higher vitamin A requirement level than other canaries.[37]

For good health and breeding results, canaries are traditionally fed soft food (egg food). The formulas of available commercial egg foods vary greatly. The current recommendation is to follow the advice of an experienced passerine veterinarian, as several formulas are used with good breeding results. They contain essential vitamins, amino acids and minerals. During the breeding period, soft food should be given on a daily basis and offered fresh three times a day. Most of the available soft food contains 16 to 18% protein. Soft food is often moisturized just before administration to make it more acceptable. Sometimes soaked seed or couscous is mixed with the soft food to improve the taste. Soaked, germinated seeds contain many additional vitamins.

Soluble grit sources, such as cuttlefish bone (*Sepia* spp.),

oyster shell, limestone (calcium carbonate), marble (crystalline limestone) and gypsum (calcium sulfate), are used as calcium supplements and are usually completely digested by birds. Insoluble grit consists of items such as sand, quartz and granite and can lead to health problems (eg, impaction of the crop, proventriculus and gizzard) if it is overconsumed.[109] Beach sand is shell (and soluble), but contamination with salt excludes its use.

Fruits, vegetables and many free-growing plants such as dandelion, chickweed and parsley are good sources of fiber, vitamins and minerals.

Sometimes color additives are used to manipulate the color of the plumage. For example, red-colored canaries are fed canthaxantines or ß-carotene 2 weeks before the breeding season until the end of the molt period. Spirulina algae is known as a natural source of coloration, however, it may not be effective to acquire the level of coloration desired. Yellow-colored canaries are supplemented with lutein for enhancing the desired "yellow color" for exhibition.

Because of the poor rate of breeder performance and a high rate of nutritional disorders on the seed-supplemented diet, trials offering pellets or mash containing a balanced proportion of the required nutrients are proving a good alternative to a traditional diet mentioned previously. One should not indiscriminately supplement a formulated diet with calcium, vitamins or minerals.

Most small passerines drink from 250 to 300 ml/kg body weight of water each day (desert birds such as zebra finches are an exception).[71]

Diagnostic Procedures

HISTORY AND PHYSICAL EXAMINATION

A careful consideration of the history is essential because the physical examination and other diagnostic procedures are often not rewarding in Passeriformes due to the size and nature of the birds (Table 39.2).

Examination of Cage or Aviary

Pet birds will often be presented individually or in a small group in their own cage. Points to observe include size, hygiene status, perches, substrate, food and water containers, toys and cuttlefish bone or other supplements. Although this might not give much information about a certain disease, it will help assess the owners and their standard of knowledge regarding hygiene, nutrition and general requirements of the birds.

Table 39.2 | Checklist of Questions to Obtain Patient History

- Number of birds owned, number of sick birds
- Clinical Signs
- Age of affected birds
- Species, breeds involved
- Diet: seed mixture, soft food or formulated diet
- Food supplements (eggs, soaked seeds, vitamins, trace elements, amino acids, live food)
- Drinking water supplements (vitamins, trace elements, amino acids; frequency)
- Breeding method (artificial light, foster parents, baby cages)
- Vaccinations (poxvirus)
- Contact with birds (newly acquired, shows)
- Medications already given

When dealing with a breeding facility, the aviary should be visited. This is the only way to assess important aspects such as general hygiene, nutrition and management procedures.

Observation of the Bird

Before handling and restraining a sick bird for a physical exam, it should be observed from a distance, either in its own cage or, if it is presented in a cardboard box, in a small cage that can be placed directly on a scale.

Restraint and Physical Examination

A systematic approach with good restraint of the bird is important. This allows the physical exam to become routine, which shortens the procedure and reduces the stress factor to the often already compromised patient. The systematic approach assures that nothing is overlooked.

The necessary equipment and basic procedure for performing a physical examination are listed in Table 39.3.

DIAGNOSTIC SAMPLING

Fecal Examination

A direct wet mount of fresh, warm stool should be examined for *Cochlosoma, Giardia, Candida, Macrorhabdus,* bacteria, plant material, chitin skeletons, urates and powderdown feathers. Gram's stains should not contain *Macrorhabdus*, yeast (*Candida*) or bacteria (or only low numbers of gram-positive rods or cocci). Flotation can reveal coccidian oocysts (common), or helminth eggs (seldom seen in canaries and finches).

Crop Swabs

A crop swab can be obtained by using a cotton-tipped applicator moistened in saline (Fig 39.17) or by administration of approximately 0.2 ml saline into the crop with a syringe and small feeding needle and then re-aspirating


Peter Coufteel

Fig 39.17 | A cotton-tipped applicator in saline is used for a crop swab.


Peter Coufteel

Fig 39.18 | The right jugular vein in a canary is used for blood sampling.

Table 39.3 | Physical Examination Checklist

Equipment
- Paper towel for capture and restraint
- Jeweler's loupe (for magnification)
- Bright light source (otoscope or endoscope light source)
- Alcohol
- Aural thermometer
- Digital gram scale
- Small observation cage

Procedure
- Prepare all necessary equipment.
- Capture and restrain bird with a paper towel.
- Listen for respiratory sounds.
- Extreme respiratory distress warrants releasing the bird and considering delay of further procedures. Exceeding the oxygen reserve of a small passerine with respiratory impairment can be rapidly fatal.
- Examine the eyes, ears and nares. Look for exudates, crusts, pox lesions, cataracts, sinusitis.
- Examine the oral mucosa and tongue. Look for white plaques (candidiasis, bacterial infections, trichomoniasis).
- Measure body temperature using aural thermometer, which is a fast, low-stress method. Use three readings for an average temperature. In sick small passerines the body temperature is below 40° C (104° F).
- Assess the pectoral muscle mass and color (anemia) and the presence of fat. Look at the skin for signs of dehydration (red, dry skin).
- Examine the abdomen. Assess the internal organs, which can be observed easily through the abdominal wall for signs of hepatomegaly, dilatation of the intestines, ascites or presence of urine in the cloaca. (If the feathers cannot be parted correctly consider applying a drop of alcohol over the abdomen. Be aware that this can cause hypothermia).
- If necessary wet feathers over neck and observe trachea for mites.
- Examine the feathers for the state of molt, ectoparasites, broken feathers, discoloration, alopecia or dirty feathers around the vent and along abdomen.
- Examine the wings and legs for skeletal deformities, fractures, wounds and feather cysts.
- Examine the feet and toes for signs of hyperkeratosis, pox lesions and pododermatitis.
- Place bird in a small cage standing on a digital scale. Register weight. If necessary, cover with a towel.
- Only at this stage (after the bird is no longer in the hand), discuss findings and further testing with the owner.

it. A direct warm wet mount can reveal the presence of *Trichomonas, Candida, Macrorhabdus* or bacteria.

Samples for Bacteriology

Samples for bacteriology may include feces, a cloacal swab, nasal discharge or a skin swab.

Blood Samples

A blood sample may be obtained by venipuncture of the jugular vein (Fig 39.18). No more than 1% of a bird's body weight should be collected, unless this is a terminal procedure for serology prior to euthanasia and necropsy. Hematology includes hematocrit, total protein and blood smear for blood parasites. Serology may indicate paramyxoviruses (PMV), *Chlamydophila* spp. or *Toxoplasma* spp. Table 39.4 lists selected normal hematologic and serum biochemical values.[33]

NECROPSY

Necropsy is a good procedure to find the cause of death or to confirm a diagnosis, especially when dealing with flock problems. The carcass must be refrigerated imme-

Table 39.4 | Hematologic and Chemistries Reference Ranges

Parameter	Canary	Finch	Mynah
PCV (%)	45-60	45-62	44-55
RBC ($10^6/\mu l$)	2.5-4.5	2.5-4.6	2.4-4.0
WBC ($10^3/\mu l$)	4-9	3-8	6-11
Heterophils (%)	20-50	20-65	25-65
Lymphocytes (%)	40-75	20-65	20-60
Monocytes (%)	0-1	0-1	0-3
Eosinophils (%)	0-1	0-1	0-3
Basophils (%)	0-5	0-5	0-5
AP (IU/L)	146-397	—	—
AST=SGOT (IU/L)	145-345	150-350	130-350
LDH (IU/L)	120-350	—	600-1000
Ca (mmol/L)	1.28-3.35	—	2.25-3.25
P (mmol/L)	0.52-1.81	—	—
Glucose (mmol/L)	11-22	11-25	10.5-19.4
TP (g/L)	20-45	30-50	23-45
Creatinine (mmol/L)	8.8-188	—	8.8-53.0
Uric acid (μmol/L)	—	—	237-595
K (mEql/L)	2.7-4.8	—	3.0-5.1
Na (mEql/L)	125-154	—	136-152

Data reprinted with permission from Dorrestein and Elsevier Science.[32]

Table 39.5 | Necropsy Procedure

History:

- Number of birds owned, number of sick birds.
- Identification of carcass: species, leg band.
- Inspect packing and feathers for ectoparasites.
- External examination: feathers, eyes, ears, nostrils, vent; condition of pectoral muscles.
- Wet carcass with alcohol and pluck feathers. Open carcass from sternum to cloaca as well as from sternum up to thoracic inlet and on along neck up to the mandible.
- Assess air sacs and serosal surfaces.
- Remove heart, then the liver and intestinal tract, by cutting through the esophagus and bending the viscera away from the body cavity at the cloaca, leaving lungs, kidneys and gonads in the body.
- Assess all organs macroscopically.
- Swab obvious lesions as well as liver and heart blood for bacteriological cultures.
- Slice all parenchymatous organs (liver, spleen, kidney, pancreas, lung, heart, gonads).
- Open all tubular organs (gastrointestinal tract, trachea).
- Direct wet preparation and flotation of gastrointestinal content.
- Cytology of impression smears of freshly cut surface of liver, spleen, lung, etc, and scrapings of the mucosa of crop, proventriculus and intestine.
- Collect tissues for histopathology in buffered formalin.
- Freeze tissue for virology.

diately after death, and necropsy must be performed within 72 hours. A systematic approach is essential (Table 39.5).

Treatment Techniques

DRUG ADMINISTRATION

Parenteral Administration

As in all avian species, drugs can be administered by intravenous, intramuscular or subcutaneous injection. Drug dosing should be based on the exact weight determined by an electronic gram scale and administered using a precise 0.3-ml insulin syringe, which permits dosing as little as 0.005 ml. Due to the size and nervous character of small passerines, most injectable drugs are administered by intramuscular injection into the cranial third of the pectoral muscle. Applying pressure on the injection site with a dry cotton-tipped applicator can control bleeding. In dehydrated patients, fluids can be administered subcutaneously in the inguinal skin fold.

Oral Administration

Direct oral administration allows accurate and regular dosing but is feasible only in individually diseased birds housed in a small hospital-sized cage. Expecting an owner to individually treat a flock of small passerines housed in a larger cage or aviary will result only in poor owner compliance and a counterproductive stressful situation for the birds being chased through the aviary twice a day.

Medication of the drinking water and soft food is often the only feasible option for flock treatment. Drawbacks of which to be aware include: 1) reduced and irregular water intake, resulting in inadequate or irregular blood levels with therapeutic failure and development of drug resistance; 2) no measurable blood levels during the night due to the high metabolic rate and rapid drug elimination; and 3) the potential for drug toxicoses in dry, hot weather (eg, dimetridazole or furazolidones). Oral drug administration should be via drinking water and soft food simultaneously. Dosages are listed in Table 39.6.[34]

ANESTHESIA

The anesthetic of choice is isoflurane (or sevoflurane) gas, as for other avian species. The bird may be captured and restrained using a paper towel and masked down with 4 to 5% isoflurane. Anesthesia can be maintained at approximately 1.5 to 2.0% isoflurane; the anesthetic depth can be adjusted by monitoring the respiration rate and reflex status. A suitable mask may include a syringe case, syringe or a dropper bottle with the base cut off.

Table 39.6 | Therapeutics Administered via Soft Food and Water in Flock Treatment of Canaries and Small Passerines

Drug	Concentration in Drinking Water (mg/L)	Concentration in Soft Food (mg/kg)
Amoxicillin	200-400	300-500
Amphotericin B	100-200	100-200
Ampicillin	1000-2000	2000-3000
Chlortetracycline	1000-1500	1500
Dimetridazole	125	—
Doxycycline	250	1000
Enrofloxacin	200	200
Fenbendazole	25	25
Furazolidone	200-300	300
Ivermectin	9	—
Metronidazole	100	100
Neomycin	200	200
Nystatin	200,000 IU	200,000 IU
Polymyxin	100,000 IU	100,000 IU
Ronidazole	350	350
Spectinomycin	200-400	400
Spiramycin	200-400	400
Sulfachloropyridazine	150-300	—
Sulfadimidine	150	—
Toltrazuril	180	—
Trimethoprim/sulfonamide	200	200
Tylosin	250-400	400

Note: The birds should be treated at the same time through the drinking water and through the food.
Data reprinted with permission from Dorrestein and Elsevier Science.[32]

Use of a heating pad as well as humidified anesthesia gas will minimize drying out of the mucous membranes and loss of body heat.

SURGERY

Surgery techniques are the same as in other avian species. Special considerations due to the small blood volume and the high metabolic rate include: the use of minimally invasive techniques (eg, removing egg-bound eggs from the cloaca and not by abdominal surgery), bipolar radiosurgical forceps (which allows for hemorrhage control), binocular magnification loupes and a good light source. Postoperative recovery should occur in a dark, warm incubator at 30° to 32° C (86-98° F).

FRACTURE SPLINTING

Fractures of the wing are relatively uncommon and may be treated by external splinting and bandaging with a minimal amount of padding. Fractures of the tarsometatarsus and tibiotarsus are more common. A sandwich adhesive or masking tape splint gives excellent results. The knee and tarsal joint are positioned in a moderately flexed position, and tape is applied to the medial and lateral sides of the fractured leg. The tape is molded to the contour of the leg using hemostat forceps. Instant glue is applied to the outer surface of the tape and then a second layer of tape is applied over the glue-covered surface. The tape is trimmed to make it as small and light as possible. The patient is maintained under anesthesia for another few minutes to allow the glue to dry and form a hard, stable cast. This glue-hardened masking tape splint can be removed with scissors, with the patient under isoflurane anesthesia to avoid refracturing the leg.

Viral Diseases

POXVIRUS

Roughly 232 avian species in 23 orders have been reported to acquire a natural poxvirus infection. It is likely that many more species are susceptible to avipoxviruses. Many of these reported avian species belong to the order of Passeriformes.[9] Poxvirus infections are particularly common in canaries (*Serinus canarius*) and other Fringillidae.[32] Of clinical importance to the avian practitioner is canary pox.[51] The disease is mainly transmitted from latently infected birds by bloodsucking insects, including mosquitoes and red mites.[59]

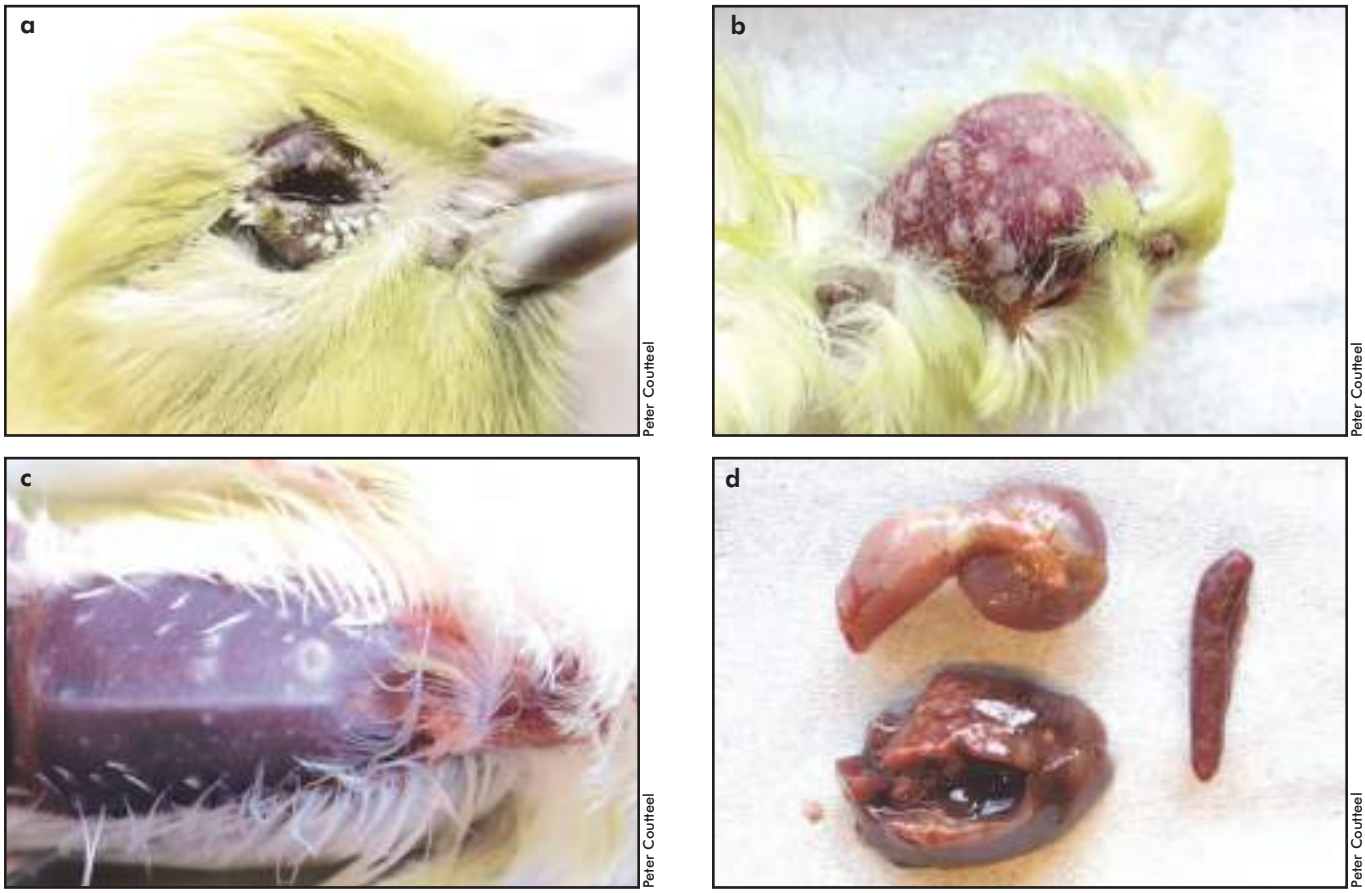
Transmission can also occur through direct contact with an infected bird or indirect contact with contaminated objects such as gloves or hands. Because poxviruses are not capable of penetrating intact epithelium, they must enter the skin through abrasions (eg, caused by cannibalism, territorial behavior, feather picking, aggressive preening or handling and consumption of scabs).[97] Theoretically, pox infections can occur during the whole year. However, most outbreaks are diagnosed in the late summer and autumn, because of the prevalence of mosquitoes. Birds of all ages can develop disease, but young birds (older than 3 weeks) are especially susceptible.

Clinical Disease

The three different clinically recognized symptoms are the cutaneous or external form, the internal diphtheroid form and the septicemic form.[59] Whereas finches often develop the cutaneous form, canaries will often develop the internal diphtheroid or septicemic form, making pox infections in canaries a disease with a high mortality.[17]

Cutaneous Form

This form is localized on the toes and legs and around the eyes, nares and beak (Figs 39.19a-c). These unfeathered body regions are easily accessible to blood-sucking insects. As the lesions progress they develop from papules of roughly 2 to 4 mm diameter to vesicles that open spontaneously, dry out and then become crusts. Tumor-like pox lesions that do not develop vesicles and



Figs 39.19 | **a,b,c)** Shown are pox lesions on the eyelids, back of head and sternum. **d)** A hemorrhage in the liver and enlarged spleen is shown in a bird with pox.

crusts have also been described.[38,112] The contaminated birds rub their eyes and beaks against the perches. They also may pick the lesions on their legs until they start bleeding. The lesions can be treated locally with iodine, but often the bird will lose a nail or even a digit. The mortality rate, however, is low in this form and the lesions will heal spontaneously after 3 to 4 weeks.[17]

Diphtheroid Form

Lesions occur on the mucosa of the tongue, pharynx and larynx. The fibrinous lesions are gray to brown and caseous.[51] In severe cases birds have difficulty swallowing or exhibit dyspnea.

Septicemic Form

This is the predominant form diagnosed in canaries. Birds show severe dyspnea and become apathetic and cyanotic. They are unable to eat or drink, and mortality can be as high as 90 to 100%. In contrast to other disorders, such as tracheal mites or bacterial infections of the upper respiratory system, the breathing is silent without clicking or growling sounds.

Diagnosis

Antemortem in-house cytology and postmortem histopathology often reveal intracytoplasmic inclusions (Bollinger bodies) in epithelial cells.[59] Macroscopic postmortem findings are often unspectacular (Fig 39.19d).

Prophylaxis

Attenuated live vaccines[c] that are applied using the wing-web method are commercially available for canaries and crossbreeds. A whitish swelling or scab at the injection site observed 8 to 10 days post-vaccination indicates a successful vaccination. Protection lasts approximately 6 months. Vaccination should be performed early in the year, prior to mosquito season. Solid immunity develops 2 to 4 weeks after vaccination. Fledglings should be at least 4 weeks old. Vaccination can be repeated without any risk if vaccination status is unknown. A new needle should be used for every bird so that blood-borne diseases, including poxvirus, are not transmitted. Vaccinated birds should be separate from newly acquired non-vaccinated birds.[17]

Treatment

There is no therapy for the septicemic poxvirus infection. Only a preventive vaccination offers solid protection. In case of an outbreak, the following measures

should be taken: separate diseased birds (gasping birds and birds with cutaneous lesions), consider emergency vaccination and begin antimicrobial therapy against secondary bacterial and fungal infections. Multivitamin (especially vitamins A and C) supplementation may help with epithelial turnover and immune system support. Iodine should be applied locally to cutaneous and mucosal pox lesions. Non-steroidal antibiotic eye ointment may be applied. Access to blood-sucking insects must be prevented, and cages and perches much be thoroughly disinfected. An emergency vaccination in the face of an outbreak is controversial. This may result in the recombination between field and vaccine virus strains, inducing severe disease in the entire flock.[51] Handling of the birds can also induce skin abrasions, creating a port of entry for the poxviruses.

Differential Diagnoses

The following should be considered as differentials for the cutaneous form of poxvirus infection: bacterial and mycotic dermatitis, *Knemidokoptes* mites, fiber constriction and conjunctivitis. *Trichomonas* and *Candida* should be considered as differentials for the diphtheroid form, and for the septicemic form, consider bacterial upper respiratory disease, *Atoxoplasma, Trichomonas, Sternostoma, Chlamydophila, Aspergillus, Syngamus* and *Paramyxovirus* (PMV) infections.

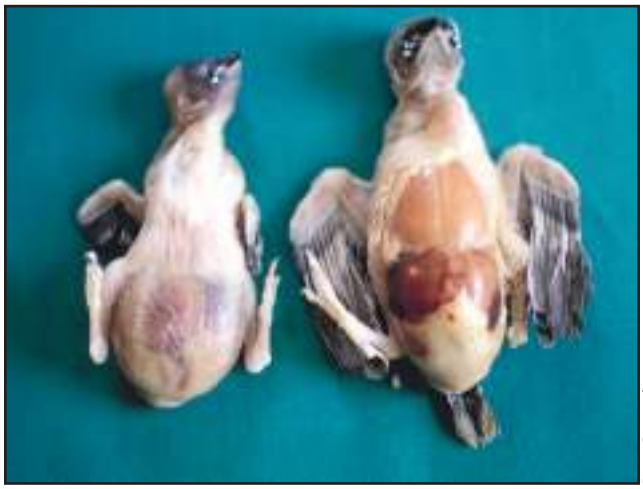
POLYOMAVIRUS

Avian polyomavirus (APV) has been associated with mortality in Passeriformes of all ages. Reports include disease in canaries, greenfinches (*Carduelis chloris*), goldfinches (*Carduelis carduelis*), Gouldian finches (*Erythrura gouldiae*), painted finches, golden-breasted starlings (*Cosmopsarus regius*), seedcrackers (*Pyrenestes* sp.) and bluebills (*Spermophaga haematina*).[22,48,71,99]

Recently, 20 different avian polyomavirus variants have been determined.[85] A parsimony tree was constructed containing three major branches. All European viruses were confined to Branch I, but APVs from the USA represented all three branches. Polyomaviruses from different avian species were also on each of the three branches, suggesting that species-specific pathotypes have not developed.

Clinical Disease

No specific clinical entity is recognized in birds with APV, but reported clinical signs include acute mortality, nonspecific signs of disease (fluffed bird), delayed fledging, poor feather development, abdominal hemorrhages (Fig 39.20) and long, tubular, misshapen beaks in surviving fledglings.[17,95]



Peter Coufteel

Fig 39.20 | Abdominal hemorrhages in these Lady Gouldian finch (*Erythrura gouldiae*) chicks was due to polyomavirus.

Diagnosis

Serodiagnosis of APV has traditionally been performed using a serum neutralization test. Recently a blocked enzyme-linked immunosorbent assay for the detection of avian polyomavirus-specific antibodies was developed in Germany.[61]

DNA probes used to detect avian polyomavirus in psittacines are commercially available. This probe, however, will not detect the virus in seedcrackers.[48] It does not appear clear in which passerine cases these probes are capable of detecting avian polyomavirus. A DNA in situ hybridization analysis using a probe designed to recognize nucleotide sequences coding for a major viral structural protein was highly sensitive in the detection of APV infection in seedcrackers and bluebills.[48]

Necropsy

Avian polyomavirus lesions that can be expected on gross postmortem examination include a pale swollen liver, splenomegaly, and perirenal, serosal, intestinal and hepatic hemorrhage.[51] Histologic changes include inflammation and necrosis of the liver, spleen, myocardium, intestinal tract and bone marrow. These organs also will demonstrate intranuclear inclusion bodies.[95]

Prophylaxis and Treatment

A commercially available inactivated avian polyomavirus vaccine for parrots has not been shown to induce protection in Passeriformes. In one case, over 80 Passeriformes of various species were vaccinated. The nestling mortality caused by polyomavirus did not decrease in the breeding season following the vaccination trial.[99] Other than supportive measures, there is no treatment for diseased Passeriformes. In aviaries experiencing high nestling mortality, discontinuing all breeding during one season can often control the outbreak of clinical disease.


Peter Coutteel

Fig 39.21 | This melba finch (*Melba astrilde*) is exhibiting torticollis as a result of paramyxovirus (PMV-3).


Peter Coutteel

Fig 39.22 | Papillomavirus with wart-like skin masses is shown in a siskin (*Carduelis spinus*).

Differential Diagnoses

Differential diagnoses include all pathogens causing sudden death in Passeriformes (Table 39.7).

PARAMYXOVIRUS

Of the nine known serotypes of paramyxoviruses (PMV-1 through PMV-9), only PMV-1, -2 and -3 are known to cause disease in Passeriformes.[96,104]

PMV-1

As in all avian species, PMV-1 can cause a variety of disease signs in Passeriformes. These include watery diarrhea, respiratory signs, sudden death and, less commonly, neurologic signs.[32,71] Most species of free ranging birds have been found to be susceptible to some strain on PMV-1. PMV-1 is a reportable disease in most countries.

PMV-2

PMV-2 infections in Passeriformes appear to be mild and self-limited. They are endemic in finches originating from northern Africa. Experimentally, they cause mild upper respiratory tract disease.[51]

PMV-3

The most common serotype observed in Passeriformes is PMV-3. Commonly affected species are African and Australian finches.[17,32] Clinical signs include conjunctivitis, anorexia, diarrhea, voluminous starchy stools, dyspnea and torticollis (Fig 39.21).[70,102,104] Diagnosis is based on serology or virus isolation. Serologically, there are cross-reactions with PMV-1. An exact differentiation is possible with monoclonal antibodies.[51] Virus isolation is performed by hemagglutination inhibition using antisera specific for PMV-3 on inoculated cell cultures.[59,104] Gross postmortem lesions can be minimal and are nonspecific. Histologic lesions consist of encephalitis, myocarditis and pancreatitis associated with intranuclear and intracytoplasmic inclusion bodies.[104] Although no commercial PMV-3 vaccination is available, an inactivated vaccine was shown to produce sufficient immunity to withstand challenge in canaries.[8] Differential diagnoses include toxins (organophosphates, dimetridazole), trauma, atoxoplasma and bacterial meningoencephalitis (Table 39.8).

HERPESVIRUS

Herpesviruses are much less a clinical problem in Passeriformes than in many other avian orders. Uncharacterized herpesviruses causing necrosis of the liver, spleen and bone marrow have been reported in finches (Estrildidae), canaries and weavers (Ploceidae).[60]

CYTOMEGALOVIRUSES

Cytomegalovirus can cause severe conjunctivitis, pseudosymblepharon (crust sealed eyelids) and acute illness, often with a high mortality rate, in Australian and African finches.[17,24,103] Diagnosis is based on histopathology with cytomegaly and karyomegaly of epithelial cells, hemorrhage in the lung and bronchi and diphtheroid lesions in the esophagus and choana. Basophilic intranuclear inclusion bodies within epithelial cells of the conjunctiva and respiratory tract can be observed.[17,94] Treatment consists of supportive measures including tube-feeding and keeping the eyelids free of forming crusts. Differential diagnoses of conjunctival lesions include infections with poxvirus, *Chlamydophila, Mycoplasma* or other bacteria.[24] For differential diagnoses of sudden death, see Table 39.7.

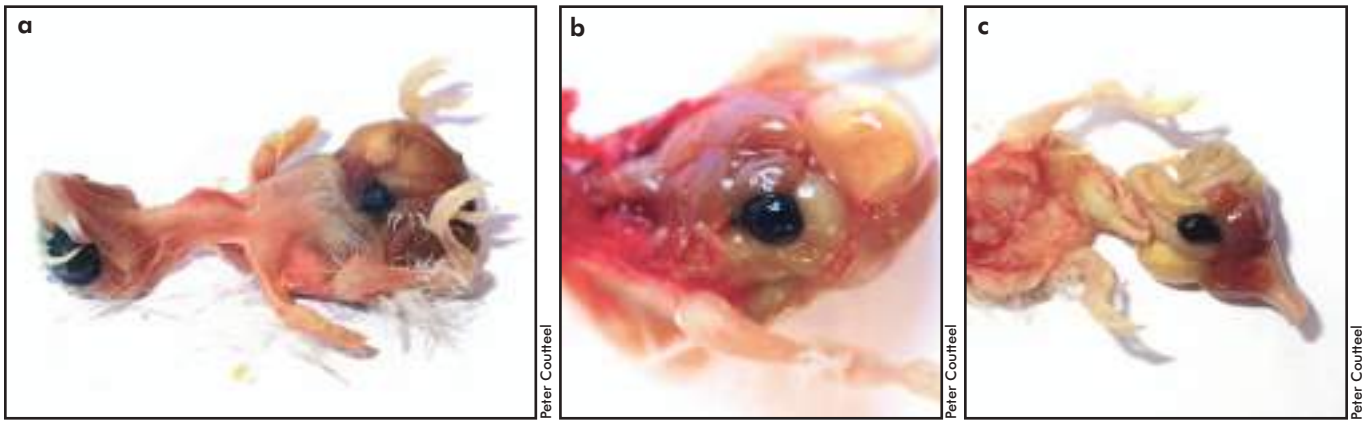
PAPILLOMAVIRUS

Proliferative wart-like skin masses observed on the legs and feet of Fringillidae (especially siskins [*Carduelis spinus*] and European chaffinches [*Fringilla coelebs*]) are caused by papillomaviruses (Fig 39.22). In other passerine species, however, lesions appear to be rare.[95]

Table 39.7 | Causes of Sudden Death and Increased Mortality in Passiformes

	Infectious	Parasitic	Other
Canaries and Finches (Fringillidae)	★ *E. coli* (nestlings) ★ Mycobacteria (adults) ★ *Macrorhabdus* (all ages) ★ *Yersinia pseudotuberculosis* (adults) ★ Poxvirus (fledglings) ● Herpesvirus (all ages) ● Polyomavirus (all ages) ● Circovirus (nestlings) ● *Chlamydophila* (all ages) ● *Salmonella* (all ages) ● Other bacteria (nestlings) ● *Candida* (nestlings) ● *Aspergillus* (adults) ❍ PMV-1 (all ages) ❍ *Campylobacter* (nestlings)	★ *Atoxoplasma* (fledglings) ★ *Dermanyssus* (adults) ● *Isospora* (all ages) ● *Sternostoma* (adults) ❍ *Cryptosporidium* ❍ *Sarcocystis* ❍ *Toxoplasma* ❍ *Cochlosoma* ❍ Blood parasites ❍ Microsporidia ❍ *Syngamus* ❍ Spiruroids	● Inhalant toxins (PTFE, CO) ● Ingested toxins ● Cranial trauma ● Starvation ● Visceral gout ● Blood loss ● Predators (eg, cats) ● Neoplasia ● Diseases of the urogenital tract ❍ Ingested foreign bodies ❍ Iron storage disease ❍ Hepatic lipidosis ❍ Cardiac disease
Waxbills (Estrildidae)	★ *E. coli* (nestlings) ★ *Campylobacter* (nestlings) ● Polyomavirus (all ages) ● Herpesvirus-cytomegalovirus (all ages) ● *Chlamydophila* (all ages) ● *Salmonella* (all ages) ● Mycobacteria (adults) ● *Yersinia pseudotuberculosis* (adults) ● Other bacteria (nestlings) ● *Macrorhabdus* (all ages) ● *Candida* (all ages) ● *Aspergillus* (adults) ❍ PMV-1 (all ages) ❍ Poxvirus (fledgling)	● *Cochlosoma* (nestlings, fledglings) ● *Isospora* (all ages) ● *Sternostoma* (adults) ● *Dermanyssus* (nestlings) ❍ *Cryptosporidium* ❍ *Sarcocystis* ❍ *Toxoplasma* ❍ Blood parasites ❍ Microsporidia ❍ *Syngamus* ❍ Spiruroids	● Inhalant toxins (PTFE, CO) ● Ingested toxins ● Cranial trauma ● Starvation ● Visceral gout ● Blood loss ● Predators (eg, cats) ● Neoplasia ● Diseases of the urogenital tract ❍ Ingested foreign bodies ❍ Iron storage disease ❍ Hepatic lipidosis ❍ Cardiac disease
Mynah (Sturnidae)	★ *Aspergillus* ● *Chlamydophila* ● *E. coli* ● Mycobacteria ● *Salmonella* ● *Yersinia pseudotuberculosis* ● Other bacteria ● *Macrorhabdus* ● *Candida* ❍ PMV-1 ❍ Poxvirus	● *Coccidia* ● Spiruroids ❍ *Dermanyssus* ❍ *Cryptosporidium* ❍ *Sarcocystis* ❍ *Toxoplasma* ❍ Blood parasites ❍ Microsporidia	★ Iron storage disease ★ Ingested foreign bodies ● See canaries ❍ Hepatic lipidosis ❍ Cardiac disease

★ Common ● Occasional ❍ Seldom



Figs 39.23a-c | Circovirus, congestion of the gall bladder, is expressed as "black spot disease," which is shown in a 2-day-old canary prior to and after necropsy.

Table 39.8 | Causes of Central Nervous System Signs

	Infectious	Parasitic	Other
Canaries and Finches (Fringillidae)	● PMV-3 ❍ PMV-1 ❍ Mycobacteria ❍ *Listeria* ❍ *Aspergillus* ❍ *Sarcocystis*	● *Atoxoplasma* (fledglings) ❍ *Toxoplasma*	● Trauma (cranium, spinal cord) ● Toxins (organophosphates) ● Pharmacologic agents (dimetridazole) ❍ Hepatoencephalopathy ❍ CNS neoplasia ❍ CNS abscess ❍ Hypovitaminosis B_1 ❍ Vitamin E/selenium deficiency ❍ Hypocalcemia ❍ Epilepsy ❍ Age - improper diet (arteriosclerosis, infarct)
Waxbills (Estrildidae)	★ PMV-3 ❍ PMV-1 ❍ Mycobacteria ❍ *Listeria* ❍ *Aspergillus* ❍ *Sarcocystis*	❍ *Toxoplasma*	● Trauma (cranium, spinal cord) ● Toxins (organophosphates) ● Pharmacologic agents (dimetridazole) ❍ Hepatoencephalopathy ❍ CNS neoplasia ❍ CNS abscess ❍ Hypovitaminosis B_1 ❍ Vitamin E/selenium deficiency ❍ Hypocalcemia ❍ Epilepsy
Mynah (Sturnidae)	❍ PMV-1, -3 ❍ Mycobacteria ❍ *Listeria* ❍ *Aspergillus* ❍ *Sarcocystis*	❍ *Toxoplasma*	● Trauma (cranium, spinal cord) ● Toxins (organophosphates) ● Pharmacologic agents (dimetridazole) ❍ Hepatoencephalopathy ❍ CNS neoplasia ❍ CNS abscess ❍ Hypovitaminosis B_1 ❍ Vitamin E/selenium deficiency ❍ Hypocalcemia ❍ Epilepsy

★ Common ● Occasional ❍ Seldom

Papillomavirus lesions can bleed profusely and can often cause leg band constriction.[17] Papillomavirus-like infections causing epithelial proliferations at the commissure of the beak and on the head in canaries have been described in Belgium.[28] Treatment consists of local disinfection. An autogenous vaccine can be considered, although no data exist to prove the efficacy in Passeriformes.[17] Differential diagnoses include hyperkeratosis, poxvirus, *Knemidokoptes* mites and trauma.

CIRCOVIRUS

A disease called "black spot" by European canary breeders has been proven to be caused by a circovirus. The disease is observed in hatchlings and nestlings and has a high mortality. Signs include abdominal enlargement and congestion of the gall bladder (visible as a black spot through the skin) (Figs 39.23 a-c).[17] Feather loss and lethargy in finches also have been associated with circovirus.[78] Diagnosis is based on recognizing inclusion bodies on histopathology of the bursa of Fabricius or the presence of 18-nm viral particles on electron microscopy. PCR techniques for psittacine circovirus (psittacine beak and feather disease—PBFD virus) fail to demonstrate viral presence, indicating that the canary circovirus differs genetically from the PBFD virus.[52] Nucleotide sequencing showed the virus to be more closely related to the Columbid circovirus than to the PBFD virus.[111] Differential diagnoses include *Atoxoplasma, Isospora, E. coli* and other causes of mortality in nestlings (see Table 39.7).

Bacterial Diseases

The general principles for diagnosing, treating and controlling bacterial disease in Passeriformes are similar to those in other avian orders.[71] There is normally no bacterial growth on routine aerobic microbiologic cultures taken from passerines. Stained fecal smears collected from normal canaries and finches reveal either no bacteria or low levels of gram-positive rods or cocci.[17,71]

CHLAMYDOPHILA PSITTACI

The causative agent of chlamydiosis is *Chlamydophila psittaci*.[113] Passeriformes appear to be less susceptible than Psittaciformes to chlamydiosis.[71] Clinical signs are nonspecific and include general apathy, diarrhea and nasal and ocular discharge. Mortality is usually low.[32]

Diagnosis is normally made at necropsy. Identification of the organism within macrophages in impression smears


Peter Coutteel

Fig 39.24 | This canary has *Mycoplasma* conjunctivitis.


Peter Coutteel

Fig 39.25 | This breeding female looks dirty and wet and has sweating disease.

of internal organs or air sac walls can be made by use of a Macchiavello, Gimenez or Stamp stain.[113] Antigen may be identified from a swab of the cut surface of internal organs or air sac walls by use of an ELISA or PCR.[30,77]

For treatment, water-soluble doxycycline hyclate, at a dose of 280 mg/L, has been proven to achieve plasma doxycycline concentrations >1 μg/ml in cockatiels.[44,88] Doxycycline hyclate at 280 mg/L in the drinking water and simultaneously 280 mg/kg in soft food are clinically effective for the treatment of chlamydiosis in canaries and finches and cause no side effects. Often citric acid is added to the drinking water. In Europe a pharmaceutical company produces water soluble doxycyclinum hyclatum[d] in combination with citric acid. This combination makes the product more water soluble and less sensitive to light.

MYCOPLASMA spp.

Mycoplasma spp. are often associated with conjunctivitis and other signs of upper respiratory disease in canaries and finches (Fig 39.24). Epizootic conjunctivitis caused by *Mycoplasma gallisepticum* has been reported in house finches (*Carpodacus mexicanus*) in the eastern US.[27] Diagnosis is based on PCR or culture of conjunctival swab samples, but isolation of this organism is often difficult.

Treatment with ciprofloxacin ophthalmic solution and tylosin at 1 mg/ml in the drinking water resolved mycoplasma infections in house finches.[73] Suspected cases of mycoplasma infections should be treated with tylosin, tetracycline or enrofloxacin.[71]

Differential diagnoses include *Enterococcus faecalis* and other bacterial causes of upper respiratory disease (see Table 39.9), *Mycoplasma, Atoxoplasma, Isospora,* toxins, liver disease, *Sternostoma* and *Macrorhabdus*.

GRAM-NEGATIVE BACTERIA

Escherichia coli

E. coli is probably the most important bacterial cause of diarrhea and nestling mortality in canaries and finches. Various other Enterobacteriaceae also can be involved. Apparently healthy adult birds can be carriers of *E. coli,* resulting in clinical problems during the breeding season.[17] *E. coli* is a secondary pathogen and should be considered the sequel to a primary problem, such as poor hygiene, unsuitable housing, unbalanced diet or other management-related problems. Primary pathogens such as *Atoxoplasma*, circovirus or polyomavirus also may be present.[32]

The typical clinical presentation in a breeding flock includes diarrhea, dehydration and cachexia. The nests as well as the breeding females are dirty, wet and yellowish (sweating disease) (Fig 39.25). The youngsters die before they can be leg-banded on day 6 or 7.[17] Diagnosis is based on aerobic cultures of fecal samples or internal organs in septicemic disease.

Treatment consists of antibiotics, chosen on the basis of culture and sensitivity results, and should be administered in drinking water and egg food from one day before hatching until 6 days after hatching. At the same time, other management-related problems must be addressed.[17]

Differential diagnoses include other bacterial infections that cause diarrhea and mortality, *Atoxoplasma, Isospora,* polyomavirus, circovirus, *Chlamydophila,* toxic enteritis and PMV-1.

Salmonella

Salmonella typhimurium is the most commonly isolated *Salmonella* species in Passeriformes.[47] Salmonellosis is clinically (and at necropsy) very similar to *Yersinia*

pseudotuberculosis infections.[59] Many birds will die without preliminary signs, but chronic disease can also occur. The existence of clinically healthy carriers in canaries and finches has not been researched.[32] On necropsy, small yellow miliary bacterial granulomas can be observed on a dark and swollen liver and spleen. Sometimes focal necrosis in heart, lung and pectoral muscle can be seen.[17] Diagnosis is confirmed on culture.

Treatment consists of antibiotics chosen on the basis of culture and sensitivity. Antibiotics commonly used include sulfonamid-trimethoprim, enrofloxacin and marbofloxacin. Elimination of salmonellosis is often difficult, and an antibiotic treatment should be combined with counseling on good hygiene and disinfection. The success of a flock treatment should be monitored in serial bacterial cultures at 3 to 6 weeks after therapy by examining pooled fecal samples in enrichment medium.[32]

Differential diagnoses of necropsy lesions include *Yersinia pseudotuberculosis* and mycobacterial infections. A large number of viral, bacterial and parasitic pathogens must be considered in the differential diagnoses of the clinical presentation of unspecific disease and flock mortality (see Table 39.7).

Yersinia pseudotuberculosis

Yersinia pseudotuberculosis infects a wide range of avian and mammalian species, including humans. It is particularly common in a variety of passerine species and also in rodents.[49] The pathogen is thought to be indigenous to central and northern Europe, but is now diagnosed throughout the world. The disease is most common in autumn, winter and spring months and is believed to be transmitted by rodents and migratory birds.[17,47]

The clinical signs are nonspecific but normally include peracute mortality in adult birds.[17] Diagnosis is based on the typical small, yellow, miliary granulomas on a dark and swollen liver and spleen (Fig 39.26) and impression smears and bacteriological cultures revealing gram-negative coccoid rods.[49] Clinically diseased birds usually die before treatment can be initiated, but the rest of the flock should be treated based on culture and sensitivity. Enrofloxacin has been shown to completely protect canaries from experimental infection with *Yersinia pseudotuberculosis,* whereas groups of canaries treated with doxycycline, chloramphenicol, ampicillin and sulphamerazine-trimethoprim suffered mortality rates between 30 and 44%.[53]

Differential diagnoses of the typical necropsy lesions include *Salmonella typhimurium* and mycobacterial infections.

Peter Coutteel

Fig 39.26 | Miliary granulomas as seen on this canary's dark and swollen spleen are indicative of *Yersinia pseudotuberculosis*.

Campylobacter spp.

Campylobacter fetus subsp. *jejuni* is common in tropical finches and canaries. Society finches can be asymptomatic carriers.[17,59] Clinical signs include apathy in adult birds and high mortality in nestlings. Diseased birds develop yellow diarrhea and pale, voluminous droppings.[17] A fecal Gram's stain reveals comma- to S-shaped gram-negative rods. Culture requires special media and a microaerophilic environment.[47] Campylobacter are often resistant to multiple classes of antibiotics, and the disease frequently recurs despite antibiotic treatment.[47,49] Doxycycline, erythromycin or enrofloxacin could be good choices for flock treatment.[17,47] Thorough cleaning and disinfecting of the aviary may help prevent reinfection.[49]

Pseudomonas spp.

Pseudomonas aeruginosa is the most important pathogen in this genus. Infections often originate from contaminated drinking water, misting bottles or inappropriately prepared sprouted seeds.[71] Clinical signs of disease commonly include foul-smelling diarrhea, but air sacculitis and infections of the oropharynx may also be present. Diagnosis is based on culture, using routine aerobic media.

Treatment is often difficult because *Pseudomonas aeruginosa* is resistant to many antibiotics. Quinolones often offer an adequate treatment.[47] Effective treatment is based on antibiotics (selected by sensitivity testing) combined with elimination of the source of contamination.[32] Differential diagnoses to consider are listed in Tables 39.9 and 39.12.

Other Gram-negative Bacteria

Other gram-negative bacteria such as *Citrobacter, Klebsiella, Serratia, Pasteurella, Bordetella, Borrelia, Actinobacillus* and *Haemophilus* spp. also have been isolated from diseased Passeriformes.[49,71]

Table 39.9 | Causes of Respiratory Clinical Signs

	Infectious	Parasitic	Other
Canaries and Finches (Fringillidae)	★ Poxvirus ★ *Enterococcus faecalis* ★ *Streptococcus* spp. ● *Chlamydophila* ● *Mycoplasma* ● *Pseudomonas* ● Other bacteria ● *Aspergillus* ❍ PMV-1, -2	★ *Atoxoplasma* ★ *Trichomonas* ● *Sternostoma* ❍ *Syngamus* ❍ *Cryptosporidium* ❍ *Sarcocystis* ❍ *Toxoplasma* ❍ *Dermanyssus*	★ Abdominal distension (various causes) (see Table 39.10) ● Inhalant toxins ❍ Foreign body inhalation ❍ Neoplasia
Waxbills (Estrildidae)	★ *Chlamydophila* ● *Mycoplasma* ● *Pseudomonas* ● Other bacteria ● *Aspergillus* ● Cytomegalovirus ● Poxvirus ❍ PMV-1, -2	★ *Sternostoma* ● *Syngamus* ● *Trichomonas* ❍ *Cryptosporidium* ❍ *Sarcocystis* ❍ *Toxoplasma* ❍ *Dermanyssus* (anemia)	★ Abdominal distension (various causes) (see Table 39.10) ● Inhalant toxins ❍ Foreign body inhalation ❍ Neoplasia
Mynah (Sturnidae)	★ *Aspergillus* ● *Chlamydophila* ● *Mycoplasma* ● *Pseudomonas* ● Other bacteria ❍ Poxvirus ❍ PMV-1	● *Syngamus* ● *Trichomonas* ❍ *Cryptosporidium* ❍ *Toxoplasma*	★ Iron storage disease ● Abdominal distension - various causes (see Table 39.10) ● Inhalant toxins ❍ Foreign body inhalation ❍ Neoplasia

★ Common ● Occasional ❍ Seldom

GRAM-POSITIVE BACTERIA

Enterococcus faecalis

Enterococcus faecalis (formerly *Streptococcus bovis*) is a frequent inhabitant of the passerine alimentary tract.[47] It also is associated with chronic tracheitis, pneumonia and air sacculitis.[25] Affected birds have increased respiratory sounds, voice changes and dyspnea. Form canaries are especially sensitive. Treatment with antibiotics will improve the clinical signs, but individual birds can seldom be completely healed. Differential diagnoses include *Sternostoma tracheacolum*, other bacterial infections of the upper respiratory tract, poxvirus, PMV-1, *Chlamydophila, Aspergillus, Atoxoplasma,* and *Trichomonas* and causes of abdomen distension (Table 39.10). Concurrent infections with *Enterococcus faecalis* and *Sternostoma tracheacolum* (tracheal mites) can occur.

Mycobacterium spp.

Mycobacterial infections were the most commonly diagnosed bacterial diseases in a review of 546 necropsies performed between 1991 and 1997 in Passeriformes in Switzerland. Over 8% of all necropsied Passeriformes suffered from a mycobacterial infection, and 40% of all diagnosed bacterial infections in this avian order were caused by *Mycobacterium* spp.[1]

Clinical signs are nonspecific but include chronic "sick bird", diarrhea, weight loss and death.[36] The classic disease with tubercles in the organs is seldom seen in Passeriformes. These results suggest that mycobacterial disease is often overlooked in passerine necropsies. Acid-fast-stained impression smears of the intestinal wall and liver should be performed on every necropsied passerine.[1] Although classically described as being caused by *Mycobacterium avium,* the advent of PCR techniques for species identification has (at least in Europe) shown *Mycobacterium genavense* to be the most commonly isolated species.[56,57,83,86,90,100,110] *Mycobacterium tuberculosis* has recently been diagnosed in a canary.[55]

In Passeriformes, mycobacterial infections are almost exclusively diagnosed postmortem. Gross necropsy is often unrewarding, with nonspecific hepato- and splenomegaly and thickened intestinal walls. Acid-fast bacilli can, however, be found in the eyelids and many internal organs including liver, jejunum, ceca and/or colon, spleen, lung, air sac, iris, brain, bone marrow, muscle and kidney.[57,83,100] No treatment of mycobacterial diseases in Passeriformes has been described.

Other Gram-positive Bacteria

In addition to the gram-positive species described above, infections with *Erysipelothrix rhusiopathiae, Listeria monocytogenes, Clostridium perfringens,* as well as various *Staphylococcus* spp. and *Streptococcus* spp. have been described in Passeriformes.[71]

Table 39.10 | Causes of Abdominal Enlargement

	Hepatomegaly	Reproductive Tract	Other
Canaries and Finches (Fringillidae)	★ *Atoxoplasma* ★ *Isospora* ★ *Yersinia pseudotuberculosis* ★ Mycobacteria ● Circovirus ● *Chlamydophila* ● Other bacteria ❍ Metabolic liver disease	★ Egg binding ● Salpingitis/Impacted oviduct ❍ Egg-related peritonitis ❍ Ovarian cysts	★ Coccidiosis ● Neoplasia ● Adipositas ● Ascites, hepatic ❍ Ascites, heart ❍ Ascites, hypoproteinemia ❍ Peritonitis
Waxbills (Estrildidae)	● *Yersinia pseudotuberculosis* ● Mycobacteria ● *Chlamydophila* ● Other bacteria ❍ Metabolic liver diseases	● Egg binding ❍ Salpingitis/Impacted oviduct ❍ Egg-related peritonitis ❍ Ovarian cysts	● Coccidiosis ● Neoplasia ● Adipositas ● Ascites, hepatic ❍ Ascites, heart ❍ Ascites, hypoproteinemia ❍ Peritonitis
Mynah (Sturnidae)	★ Iron storage disease ● *Chlamydophila* ● Other bacteria ❍ Other metabolic liver diseases	● Egg binding ❍ Salpingitis/Impacted oviduct ❍ Egg-related peritonitis ❍ Ovarian cysts	★ Ascites, iron storage disease Other causes of ascites ❍ Coccidiosis ❍ Neoplasia ❍ Peritonitis

★ Common ● Occasional ❍ Seldom

Fungal Diseases

MACRORHABDOSIS

Classically known as a common disease in budgerigars ("going light"), this organism was previously termed "megabacteria" or "avian gastric yeast". Observation has been made of this organism in a wide range of passerine species.[23,50,43,84] Historically described as "megabacteriosis" during the last 20 years, there has been frequent debate on the description of this as a large gram-positive bacterium. Recent investigations in Germany proved that the so-called megabacteria are indeed fungi,[91,92] and Phalen has now renamed the pathogen, *Macrorhabdus ornithogaster*.

Clinical Disease

Chronic depression and weight loss are typical of macrorhabdosis. Birds are always hungry and stay close to the food bowl, eating large quantities of soft food. Regurgitation is not a clinical sign in passerines. Droppings often contain undigested seeds. The patient may be anemic with pale muscles. The liver becomes visible due to the proventricular dilatation. Other diseases that either may have triggered macrorhabdosis or developed as secondary diseases following macrorhabdosis must be considered.

Diagnosis

Diagnosis is based on microscopic examination of a fecal sample. The organism is easily recognized on a wet mount or following a Gram's stain using a 1000 magnification[17,50] (Fig 39.27). Failure to find Macrorhabdus organisms does not prove that the bird is not infected, as shedding begins only after a certain stage of disease and then may occur irregularly. Microscopic examination of sequential fecal samples will increase the sensitivity. Pooled fecal samples from an aviary will give good information on the status of infection within a group of birds. The organism appears in proventricular scrapings after necropsy. Note that routine fungal culture will not yield growth of this fastidious organism.

Necropsy

A distended, thick-walled proventriculus, often with white mucus on the mucosal surface, is revealed upon necropsy (Fig 39.28). Sometimes, ulcerations and small petechial bleedings of the mucosa can be detected.

Prophylaxis and Treatment

Traditional therapy consists of supportive care, including a warm, controlled environment and administration of amphotericin B. Antibiotics for secondary infections can be given. The patient should have access to soft food ad libitum. Concurrent diseases should be treated accordingly.

Differential Diagnoses

Chronic bacterial infections such as mycobacteriosis and *Chlamydophila* as well as parasitic infections such as *Atoxoplasma, Isospora,* and *Cochlosoma* may be suspected. Toxins and nutritional changes should also be considered.

CANDIDIASIS

Candida is a common environmental contaminant and in low numbers is considered a normal inhabitant of the

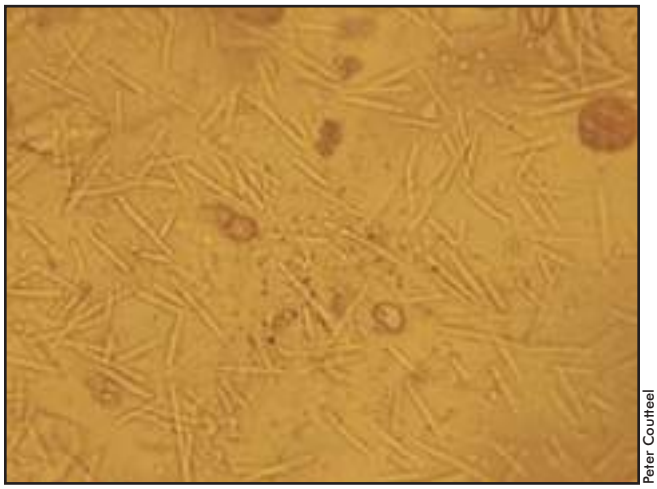

Peter Coufteel

Fig 39.27 | Macrorhabdus (400x, unstained) is easily recognized on a wet mount or following a Gram's stain.


Peter Coufteel

Fig 39.28 | Macrorhabdosis, distended proventriculus, is shown in this canary.

gastrointestinal tract.[59,115] It is considered an opportunistic pathogen. Predisposing factors include prolonged antibiotic therapy, malnutrition, spoiled food, hand feeding, stress (crowding) and the juvenile immune system.[17] Disease is not overly common in Passeriformes but is observed more often in tropical finches than in canaries.[32] Clinical signs include regurgitation, anorexia, diarrhea, whole seeds in the feces, crop stasis, gas formation and tilted appearance with elevation of the abdomen and tail.[108] Diagnosis is based on demonstrating large numbers of budding yeast, which can be visualized on a crop swab or in a fecal sample. At necropsy, a thickened crop wall and white coating of the mucosa, as well as possible white plaques in the oropharynx, can be observed.

Prophylaxis and Treatment

Prophylaxis is based on prevention of predisposing factors (listed above). Treatment may include antimycotic drugs, such as nystatin (not absorbed from the gastrointestinal tract), fluconazole or ketoconazole (systemic drugs). Medicated drinking water and soft food may be offered.[17] Differential diagnoses include *Trichomonas*, poxvirus, bacterial stomatitis, ingluvitis or enteritis, macrorhabdosis and hypovitaminosis A.

ASPERGILLOSIS

Aspergillosis is one of the most commonly diagnosed diseases in mynahs.[58,62] It is occasionally diagnosed in canaries and finches. Predisposing factors include a warm and humid environment, overpopulation and contaminated food and environment. Recently imported birds are especially susceptible.

Clinical Disease

Mynahs may experience loss or change of voice, peracute dyspnea caused by a syrinx granuloma, chronic dyspnea and depression. In finches, aspergillosis is an acute disease with respiratory distress.[17]

Diagnosis

Definitive diagnosis of aspergillosis in a live bird is difficult. Diagnostic procedures in mynahs may include tracheoscopy, endoscopy and culture of suspicious lesions.

Necropsy

Typical aspergillosis lesions within the lung and air sac walls and syrinx granulomas may be seen. Culture, Gram's stain, wet mount or histopathology can provide a definitive diagnosis.

Prophylaxis and Treatment

Concentration of Aspergillus spores in the environment (moldy fruits on bottom of cage, general poor hygiene) should be reduced as well as susceptibility of the bird by providing a balanced diet (ensuring adequate vitamin A, in particular), improving the room climate, increasing ventilation and reducing stressors. Treatment consists of giving antimycotics orally and via inhalation, as in psittacines.

Differential Diagnoses

Iron storage disease and other causes of respiratory disease in mynahs (see Table 39.9) and dyspnea due to abdominal enlargement (see Table 39.10) must be considered.

DERMATOMYCOSES

The most common etiologic agents are *Microsporum gallinae* and *Trichophyton* spp.[71] Clinical signs include feather loss and hyperkeratosis predominantly on the head and neck (Figs 39.29, 39.30).[17] Diagnosis is based on culture or biopsy and histopathology. Treatment with topical miconazole or several sprays with enilconazole


Peter Coufteel

Fig 39.29 | Dermatomycosis is shown on this canary's head.


Peter Coufteel

Fig 39.30 | Dermatomycosis is shown on the dorsal surface of this canary's wing.

are effective. Systemic antimycotics will provide improvement but probably not eliminate the infection. Differential diagnoses include *Knemidokoptes* mites, bacterial dermatitis, feather picking, cannibalism and hyperkeratosis (Table 39.11).

Parasitic Diseases

COCCIDIAL DISEASES

Atoxoplasma

The taxonomy of *Atoxoplasma* spp. is controversial. Some authors suggest that this genus should be placed in the *Isospora* genus.[68,114] This disease is also called "Lankesterella" or "big liver disease".[17] *Atoxoplasma* spp. appear to be host specific.[68] The species affecting canaries has been named *Atoxoplasma* or *Isospora serini*.[17,32]

Unlike other Eimeriidae species, the asexual life cycle of Atoxoplasma takes place in internal organs and not in the intestinal mucosa.[59] The life cycle of the organism begins with the host's oral ingestion of oocysts.[81] Oocysts excyst the sporozoites within the intestinal tract. Sporozoites penetrate the intestinal wall and spread in lymphocytes and macrophages to parenchymal organs. Affected organs include lung, liver, spleen, pancreas, pericardium and intestinal epithelium. Several generations of asexual schizogony in these organs produce merozoites. Merozoites migrate back to the intestinal mucosa. Gametogony (sexual cycle) of the merozoites produce oocysts. Oocysts are excreted with the feces.

This is a common flock disease in canaries but only occasionally diagnosed in exotic finches. It has been a devastating disease for the captive population of the endangered Bali mynah (*Leucospar rothschildi*).[79,81,114]

Clinical Disease

Typically, this is a disease of young canaries aged 2 to 9 months.[32] The affected bird will appear fluffed up and will be debilitated and anorectic. It will have diarrhea and a red, swollen vent. Hepatomegaly is visible through the abdominal wall caudal to the sternum. Mortality is variable, but up to 80%.[32] Occasionally, a patient will exhibit neurologic signs, such as epileptiform seizures and intermittent weakness. It may exhibit respiratory distress.[5,121]

Diagnosis

Definitive antemortem diagnosis is difficult because after the acute phase, only a few *Atoxoplasma* oocysts are excreted.[32,59] Fecal flotation shows oocysts with 2 sporocysts, each containing 4 sporozoites. Microscopic differentiation from Isospora is not easy: *Atoxoplasma serini* oocysts = 20.1 x 19.2 μm, *Isospora canaria* oocysts = 24.6 x 21.8 μm. A PCR assay[c] has been developed that will detect an 18S rDNA fragment of *Atoxoplasma* species in feces, blood and tissues of infected birds.[68]

Necropsy

Necropsy reveals severe splenomegaly, hepatomegaly and dilated bowel loops.[17] Intracytoplasmic inclusion bodies will appear in mononuclear cells in impression smears or on histopathology of the lung, liver and spleen.[5,17,72]

Prophylaxis

Sound husbandry practices must be observed: avoid overcrowding, practice good hygiene and provide proper nutrition.[81] Newly acquired birds must be quarantined and screened with multiple fecal flotations for the presence of *Atoxoplasma*. Adult canaries can be asymptomatic carriers and will shed oocysts sporadically. In collections with recurrent disease, consider annual coccidial treatment prior to the breeding season.

Treatment

Clinically diseased individuals usually die before they respond to treatment. Anticoccidial drugs such as toltrazuril, sulfachloropyridazine (Esb3 30%) or other sulfonamides may be given. Atoxoplasmosis is considered resistant to treatment; however, Esb3 30% at 150

Table 39.11 | Possible Causes of Diseases of the Skin, Feathers and Extremities

	Extremities	Feathers	Skin/Subcutis/Eye/Ear
Canaries and Finches (Fringillidae)	★ Fiber constriction ★ Hyperkeratosis ★ Pododermatitis ★ Fractures ★ Leg band constriction ● Cutaneous pox ● Papillomavirus ● *Knemidokoptes* mites ● Burns ● Frost bite ● Luxations ● Bite wounds ● Arthritis ❍ Leg deformities (malnutrition) ❍ Degenerative joint disease	★ Feather cysts ★ *Dermanyssus* (red mites) ★ Molt (light, temperature) ★ Spoiled feathers (diarrhea, sitting on floor) ★ Poor husbandry ● Polyomavirus ● Ectoparasites ● Feather picking, cannibalism ● Loss of color (nutritional) ● Baldness (hormonal, malnutrition?) ❍ Circovirus	● Cutaneous pox ● *Knemidokoptes* mites ● Bacterial dermatitis ● Dermatomycoses ● Neoplasia ● Abscesses ● Adiposity ● Otitis ● Conjunctivitis/Blepharitis ● Uropygial gland impaction or neoplasia ● Wounds
Waxbills (Estrildidae)	★ Fiber constriction ★ Hyperkeratosis ★ Pododermatitis ★ Fractures ★ Leg band constriction ● Cutaneous pox ● Papillomavirus ● *Knemidokoptes* mites ● Burns ● Frost bite ● Luxations ● Bite wounds ● Arthritis ❍ Leg deformities (malnutrition) ❍ Degenerative joint disease	★ *Dermanyssus* (red mites) ★ Molt ★ Spoiled feathers (diarrhea, sitting on floor) ★ Poor husbandry ● Polyomavirus ● Ectoparasites ● Feather picking, cannibalism ● Feather cysts ● Loss of color (nutritional) ● Baldness (hormonal?)	● Cutaneous pox ● *Knemidokoptes* mites ● Bacterial dermatitis ● Dermatomycoses ● Neoplasia ● Abscesses ● Adiposity ● Otitis ● Conjunctivitis/Blepharitis ● Uropygial gland impaction or neoplasia ● Wounds
Mynah (Sturnidae)	★ Fiber constriction ★ Hyperkeratosis ★ Pododermatitis ★ Fractures ★ Leg band constriction ● Cutaneous pox ● Papillomavirus ● *Knemidokoptes* mites ● Burns ● Frostbite ● Luxations ● Bites from psittacines ● Arthritis ❍ Leg deformities (malnutrition) ❍ Degenerative joint disease	★ *Dermanyssus* (red mites) ★ Molt ★ Spoiled feathers (diarrhea, sitting on floor) ★ Poor husbandry ● Polyomavirus ● Ectoparasites ● Feather picking, cannibalism ● Feather cysts ● Loss of color (nutritional) ● Baldness (hormonal?)	★ Chronic ulcerative dermatitis ● Cutaneous pox ● *Knemidokoptes* mites ● Bacterial dermatitis ● Dermatomycoses ● Neoplasia ● Abscesses ● Adiposis ● Otitis ● Conjunctivitis/Blepharitis ● Uropygial gland impaction or neoplasia ● Wounds

★ Common ● Occasional ❍ Seldom

mg/L of drinking water 5 days a week every week from the moment of diagnosis until after molting has proven to stop production of oocysts, although it will not influence the intracellular stages.[32]

Differential Diagnoses

E. coli, mycobacteria and other bacterial pathogens, *Isospora, Macrorhabdus, Chlamydophila,* polyomavirus, circovirus and toxins must be considered as differentials.

Isospora

The life cycle is completed in the intestinal tract (unlike atoxoplasmosis but like all other Eimeriidae species). Species-specific *Isospora canaria* is common in canaries. Isolates from over 50 species of passerines other than canaries represent different *Isospora* species.[17] Clinical signs are diarrhea and emaciation (Fig 39.31)[32] as well as dilated bowel loops causing abdominal distension. *Isospora* does not always cause clinical disease.

Diagnosis is based on large amounts of oocysts observed on fecal flotation, which, unlike in atoxoplasmosis, are secreted on a continuous basis. Treatment with anticoccidial drugs will be more successful than against *Atoxoplasma* spp. Differential diagnoses include *Atoxoplasma* spp., *E. coli,* mycobacteria, *Macrorhabdus,* other enteric bacteria, toxic enteritis and sudden nutritional change (Table 39.12).

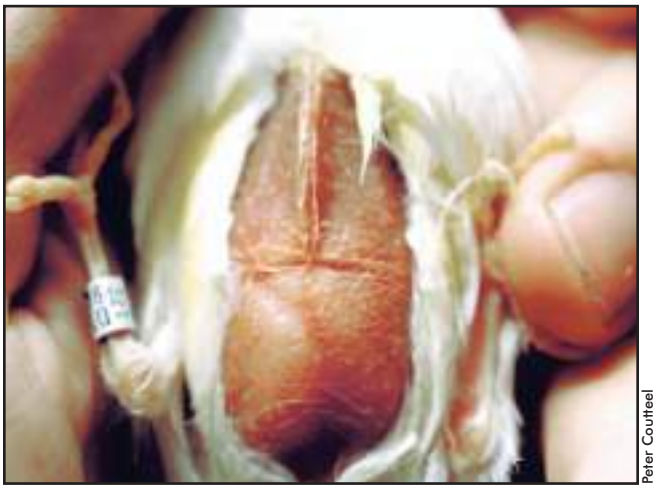

Peter Coufteel

Fig 39.31 | A canary presented with coccidiosis had emaciation and abdominal swelling.

Cryptosporidium

The clinical significance is not fully understood, but cryptosporidiosis appears to be emerging as a serious disease threat in many avian species, including canaries and finches.[17,81] Autoinfection can occur because of the endogenous sporulation.[13] It may infect and cause disease in the mucosal epithelial cells of the gastrointestinal, respiratory and urinary tracts.[17] The very small oocysts (4 x 8 μm) with 4 sporozoites can be found in fecal flotations. The small size and low numbers of excreted oocysts make diagnosis difficult. Additional diagnostic methods include acid-fast staining, direct immunofluorescence staining or ELISA of fecal samples.[20] Differential diagnoses include other causes of gastrointestinal (see Table 39.12) and respiratory (see Table 39.9) disease.

Sarcocystis

Sarcocystis spp. have an obligatory two-host life cycle. The definitive host is the opossum, whereas a wide variety of avian species can be intermediate hosts.[81] In addition, cockroaches, rats and flies can serve as transport hosts. In the avian intermediate host, merozoites enter striated muscle, where they can be observed macroscopically. Clinical signs can range from inapparent infection to anorexia, diarrhea, weakness, dyspnea, ataxia and death. Diagnosis is based on demonstrating sarcocysts on histopathology of muscle biopsies or after necropsy.

Toxoplasma

Cats and other felids are the only definitive hosts for *Toxoplasma gondii*. Essentially, any warm-blooded animal can be infected as an intermediate host by ingestion of oocysts excreted by cats. Most infections in birds are subclinical and asymptomatic.[81] In the acute phase of infection, birds may show severe respiratory signs.[59] In chronic infections, typical signs include blindness, ataxia and torticollis.[32,67,116] Antemortem diagnosis is difficult and based on an increasing antibody titer. Prevention is based on eliminating direct and indirect contact with cats. Treatment described includes pyrimethamine at 0.5 mg/kg orally every 12 hours for 14 to 28 days or diclazuril at 10 mg/kg orally every 24 hours on days 0, 1, 2, 4, 6, 8 and 10.[59] Differential diagnoses include other causes of central nervous system signs (see Table 39.8).

FLAGELLATE DISEASES

An unidentified flagellate found in the crop has been described as a possible cause of a dermatologic disease in canaries. Clinical signs include feather loss on the crown and neck, often accompanied by signs of dermatitis.[16]

Cochlosoma

Motile flagellates found in the feces of finches have been identified as *Cochlosoma* sp. or *Cochlosoma anatis*-like protozoa, which are classically transmitted by asymptomatically infected society finches, often used as foster parents.[17,42,87] Most commonly affected species include red-headed parrot finches, Bengalese and Lady Gouldian finches.[42]

Clinical Disease

Adult birds are usually asymptomatic carriers. Disease most commonly affects young birds from 10 days to 6 weeks of age.[32] Clinical signs include dehydration, diarrhea, whole seeds in the droppings, yellow staining of nestlings due to contact with their droppings, or death.

Diagnosis

Diagnosis requires demonstration of motile flagellates in saline smears from fresh warm feces.[12] Cochlosoma are smaller than giardia and hard to find in a fecal exam. They can be detected histologically in the intestinal wall (JMM Cornelissen, personal communication).

Prophylaxis and Treatment

All newly introduced adult finches should be screened via fresh fecal smears and, if necessary, treated during quarantine. All breeding finches should be screened and treated, especially foster parents, prior to breeding. Ronidazole can be administered at 50 mg/L drinking water and at 50 mg/kg in soft food for 7 days.[17] Dimetridazole (100 mg/L drinking water for 5 days) may also be helpful. Overdosage of dimetridazole or metronidazole results in reversible CNS signs. Treatment often involves an antibiotic for secondary intestinal infections.

Differential Diagnoses

Differential diagnoses include *Isospora* and other enteric parasites (see Table 39.12), *E. coli,* and other bacterial infections that cause diarrhea and juvenile mortality, *Macrorhabdus* and toxic enteritis.

Table 39.12 | Causes of Diarrhea

	Infectious	Parasitic	Other
Canaries and Finches (Fringillidae)	★ *E. coli* ★ *Macrorhabdus* ★ Mycobacteria ● *Chlamydophila* ● *Yersinia pseudotuberculosis* ● *Campylobacter* ● *Pseudomonas* ● Other bacteria ● *Candida* ❍ PMV-1, -3	★ *Atoxoplasma* ★ *Isospora* ● *Trichomonas* ❍ *Cochlosoma* ❍ *Cryptosporidium* ❍ *Giardia* ❍ *Ascaridia* ❍ *Capillaria* ❍ Cestodes	★ Sudden nutritional change (fruit, vegetables) ● Starvation ● Liver disease ● Toxins ● Kidney disease (polyuria, increased urate excretion)* ❍ Pancreatitis ❍ Neoplasia ❍ Grit over-consumption ❍ Cloacal disease ❍ Diseases of the salpinx
Waxbills (Estrildidae)	★ *E. coli* ★ *Macrorhabdus* ★ Mycobacteria ● PMV-3 ● *Chlamydophila* ● *Yersinia pseudotuberculosis* ● *Campylobacter* (more common than in canaries) ● *Pseudomonas* ● Other bacteria ● *Candida* ❍ PMV-1	★ *Isospora* ★ *Cochlosoma* ● *Trichomonas* ● Cestodes ❍ *Cryptosporidium* ❍ *Giardia* ❍ *Ascaridia* ❍ *Capillaria*	● Starvation ● Liver disease ● Toxins ● Kidney disease (polyuria, increased urate excretion)* ❍ Pancreatitis ❍ Neoplasia ❍ Grit over-consumption ❍ Cloacal disease ❍ Diseases of the salpinx
Mynah (Sturnidae)	★ *E. coli* ● *Chlamydophila* ● *Yersinia pseudotuberculosis* ● *Campylobacter* ● *Pseudomonas* ● Other bacteria ● *Candida* ❍ PMV-1	● *Coccidia* ● *Trichomonas* ● *Ascaridia* ● *Capillaria* ● Cestodes ❍ *Cryptosporidium* ❍ *Giardia*	★ Iron storage disease ● Intestinal foreign body ● Starvation ● Liver disease ● Toxins ● Kidney disease (polyuria, increased urate excretion)* ❍ Pancreatitis ❍ Neoplasia ❍ Grit over-consumption ❍ Cloacal disease ❍ Diseases of the salpinx

★ Common ● Occasional ❍ Seldom *Polyuria and polyurates can create a loose stool but that is not a true diarrhea

Trichomonas

Infections with *T. gallinae* are seen sporadically in canaries and finches.[12,32] Birds of all ages can be affected, but the infection is most predominant in younger birds. Clinical signs include respiratory changes, regurgitation and emaciation. Trichomoniasis can cause thick, yellow caseous lesions of the infraorbital sinus.[66] Diagnosis is based on a fresh wet mount of a crop swab. At necropsy, a thickened opaque crop wall with plaque development is noted (Fig 39.32). Imidazoles are used for treatment as for *Cochlosoma* spp. Differential diagnoses include poxvirus, *Candida* and other causes of respiratory signs (see Table 39.9).

Giardia

Giardia has also been reported in association with gastrointestinal disease in Passeriformes.[71]

BLOOD PROTOZOA

Protozoa are regularly observed in blood smears of clinically healthy captive and free-ranging Passeriformes species and are occasionally associated with primary disease. The most commonly encountered blood protozoa include *Plasmodium* spp., *Hemoproteus* spp., *Leucocytozoon* spp., *Trypanosoma* spp. and *Piroplasma* spp.

MICROSPORIDIA

Encephalitozoon hellem has been diagnosed in a variety of avian species from different orders. Although it has not yet been described in Passeriformes, there is no reason why it should not infect birds of this order.[11]

NEMATODES

Syngamus trachea (Gapeworm or Red Worm)

Syngamus trachea is common in Sturnidae (starlings) and Corvidae (crows) and must be considered in all Passeriformes housed in outdoor aviaries. The life cycle is direct, but earthworms can act as transport and accumulation hosts. Adult worms live in permanent copulation within the lumen of the trachea.[17]


Peter Coutteel

Fig 39.32 | An oral probe is passed down the opened esophagus in this canary with trichomoniasis. The canary has a thickened crop wall with plaque development.


Peter Coutteel

Fig 39.33 | *Syngamus trachea* (gapeworm or red worm) is shown in a canary's opened trachea.

Clinical Disease

Respiratory distress, gasping, coughing or sneezing is commonly observed in affected birds.[17] Head shaking and dried blood at the beak commissure may also be observed.

Diagnosis

Tracheoscopy or tracheal transillumination reveals adult worms (Fig 39.33). Fecal flotation will reveal ovoid eggs (70 to 100 μm x 43 to 46 μm) with a thick operculum at each pole similar to eggs of *Capillaria* spp. The number of excreted eggs does not correlate with the presence or severity of clinical signs.

Prophylaxis and Treatment

All newly introduced birds must be quarantined and screened by fecal flotation and, if necessary, treated. Contact with natural soil and earthworms must be prevented. A regular annual or biannual worming program should be instituted. Oxygen supplementation should be considered, and dexamethasone administered intratracheally or intramuscularly may be needed to reduce inflammation and mucosal swelling. If size allows, adult worms can be removed with an endoscope and biopsy forceps. Flubendazole, fenbendazole or ivermectin can be used to kill the adult worms that will then be coughed up or swallowed.

Differential Diagnoses

Differential diagnoses include *Sternostoma tracheacolum, Chlamydophila psittaci, Mycoplasma* spp., bacterial rhinitis, tracheitis, pneumonia, aspergillosis, *Trichomonas* spp., *Cryptosporidium* spp. and causes of abdominal distension.

Spiruroidae

Acuaria spp. and *Geopetitia* spp. are most commonly described.[46,64,68] Development proceeds via arthropodal intermediate hosts, such as cockroaches, crickets and mealworm beetles.[46] In the bird, Spiruroidae inhabit the proventricular and ventricular mucosa under the koilin lining.[17] There are no typical clinical signs beyond general malaise and increased mortality. Diagnosis is based on fecal flotation. On necropsy, inflammation of the proventricular and ventricular mucosa is noted. Adult spirurids can be observed under the koilin lining of the ventriculus. Spiruroidae are difficult to treat, with ivermectin most likely to be effective. Differential diagnoses include chronic bacterial enteritis, macrorhabdosis, proventricular candidiasis, coccidiosis, *Cochlosoma* spp. and other helminths.

Other Nematodes

Ascaridia spp. and *Capillaria* spp., commonly observed in Psittaciformes, are of less significance in canaries and finches. *Capillaria* spp. are not only found in the intestines but can also inhabit the oropharynx and crop, causing white plaques. Filarial nematodes, such as *Serratospiculum* spp., *Diplotriaena* spp. and *Splendidofilaria* spp., have been reported in Passeriformes. Most infections are not associated with clinical disease.[71]

CESTODES

All tapeworms require arthropods as intermediate hosts and are most common in finches (eg, parrot finches) that are fed live food.[17,71] Many different species have been described, but in most cases they cause no clinical disease.[71] Clinical signs of disease include emaciation, diarrhea and debilitation. Proglottids or eggs can be demonstrated in fecal flotation samples, but excretion is irregular. Prophylaxis includes limiting access to intermediate hosts and using insect-proof screening. Treatment with praziquantel can be effective.

Fig 39.34 | Tracheal mites (*Sternostoma* spp.) are seen as small black dots in this opened trachea.

Peter Coufteel

Fig 39.35 | Red mites (*Dermanyssus gallinae*) are shown in the dampened feathers of this canary.

TRACHEAL MITES

Sternostoma tracheacolum should be called tracheal mites and not air sac mites because the mites are normally present in the trachea and syrinx.[71] Common in Lady Gouldian finches and other exotic finches, they are less common in canaries.[17]

Clinical Disease

Clinical signs include wheezing, gasping, open-mouthed breathing, head shaking, loss of voice, cessation of singing and respiratory distress. Mortality is low.

Diagnosis

Tracheal illumination after wetting the feathers may show the mites, recognized as small black spots, in the lumen of the trachea. Lack of visualization does not rule out their presence.[17] The use of the 1.2 mm endoscope is possible, even in the unanesthetized patient. The authors warn that the inexperienced practitioner may kill the bird with this procedure, either by puncturing the trachea or suffocating the bird.

On necropsy, mites can readily be recognized macroscopically within the opened trachea (Fig 39.34).

Prophylaxis and Treatment

Prophylaxis is difficult, as recognition of carriers with a low burden is not possible. One should consider treating all newly acquired birds during quarantine. Individual birds can be treated with ivermectin or doramectin. Ivermectin or doramectin can be diluted with 1:10 with propylene glycol or sesame oil, respectively. The drug is applied as a "spot on" to the bare skin dorsolateral to the thoracic inlet (at the site of jugular blood collection), at the rate of 1 drop per bird up to 50 g and repeated after 7 to 10 days. The potential of propylene glycol toxicity can be avoided by using doramectin that contains sesame oil. It may be helpful to hang a dichlorvos strip near (but out of reach of) the birds.[17]

Differential Diagnoses

For differential diagnoses, consider *Enterococcus faecalis* and other bacterial causes of upper respiratory disease, poxvirus, *Chlamydophila, Atoxoplasma, Trichomonas, Aspergillus* and *Syngamus* (see Table 39.9).

SCALY MITES

Knemidokoptes pilae cause hyperkeratotic lesions on the feet and the base of the beak.[17] Pruritus is noted only by attentive owners. Diagnosis is based on recognizing the small bore holes within the hyperkeratotic lesions or demonstrating the scaly mites in scrapings of the lesions. One may treat locally with plant oil to suffocate the mites in conjunction with ivermectin "spot on". Differential diagnoses include hyperkeratosis, dermatomycoses, papillomavirus, fiber constriction or poxvirus (see Table 39.11).

BLOOD-SUCKING MITES

Dermanyssus gallinae (red mites) hide in nests and other dark areas in the aviary and attack the birds only at night[17] (Fig 39.35). *Ornithonyssus sylviarum* (northern or fowl mite) spends its entire life cycle on the host.[32] This mite can cause high mortality, especially in nestlings. Clinical signs include general depression, anemia, respiratory distress and pruritus. Diagnosis is based on recognizing the mites on the bird or in nests or under perches. The dark excrement of the mites can be found under the nests. During hot weather, the mite population can explode within days. Preventive measures should be initiated before the breeding season. New

Peter Coutteel

Fig 39.36 | Feather mite (*Analges* spp., *Megninia* spp.) eggs (nits) are shown in the tail feathers of a canary.

Peter Coutteel

Fig 39.37 | The effects of quill mites (*Syringophilus* spp., *Dermoglyphus* spp.) are fault lines and hemorrhages in the feather's shaft.

nests and perches should be installed and the breeding cages thoroughly cleaned. In aviaries with a known history of red mites, prophylactic use of an insecticide prior to the breeding season may be considered.

The birds may be dusted with a permethrin or carbaryl powder using a salt shaker or sprayed (along with the environment, including the nesting material) with a dilution of carbaryl (5 g 85% carbaryl powder/L water). Fipronil is also effective and well tolerated. Following application of any insecticide, the cage or room should be vacated and thoroughly cleaned with nests and perches replaced with new ones.

OTHER ECTOPARASITES

Quill mites, epidermotic mites (Fig 39.36), and lice of a variety of species have been described in Passeriformes.[32,71] These mites cause general nervousness and feather damage. Quill mites are a major problem for exhibition birds.[17] Many flight and tail feathers develop horizontal lines and hemorrhages in the shaft (Fig 39.37). Diagnosis can be made by opening the base of the shaft of a growing feather and detecting the parasite microscopically. Discovery should cause the owner to rethink general hygiene and management practices. Treatment is similar to that used for blood sucking mites.

Metabolic Diseases

IRON STORAGE DISEASE

Together with aspergillosis, iron storage disease is the most common disorder diagnosed in mynah birds.[1,62] Passerine families with higher incidence of iron storage disease include starlings (Sturnidae), tanagers (Emberizidae), bulbuls (Pycnonotidae) and birds of paradise (Paradisaedae).[36] (Toucans [Rhamphastidae] are not Passeriformes but belong to the order Piciformes and are also commonly diagnosed with iron storage disease).

Various theories exist concerning the pathogenesis of iron storage disease. High iron absorption from the intestines, regardless of body iron stores (similar to hereditary hemochromatosis in humans) is one possibility.[75] High or toxic levels of iron in the diet may be a cause or contributing factor.[21] The incidence of iron storage disease in mynahs has, however, not decreased despite the development of controlled-iron diets. Ascorbic acid in citrus fruits and green leafy plants may enhance the bioavailability of iron; thus, high levels of ascorbic acid (vitamin C) in the diet of captive birds may be a factor.[4] Iron storage disease can, however, also develop in birds specifically fed a diet lacking citrus fruits. Lack of substances such as tannin in the diet of captive birds compared to the high tannin content of water in the rain forests is another possibility. Tannin acts as a natural mineral chelating agent and therefore decreases the bioavailability of iron in the intestinal tract.[119] Concurrent disease conditions could be responsible for the degree and nature of the pathologic changes described in cases of iron storage disease. In avian species not classically considered predisposed to iron storage disease, such as psittacines, the disease is observed histologically in many individuals that succumb to a concurrent disease.[14]

Clinical Disease

Clinical signs include abdominal swelling due to ascites and hepatomegaly, dyspnea due to abdominal swelling, apathy and cachexia, loss of intense coloring of beak and periocular skin in mynahs and sudden death.

Diagnosis

Definitive diagnosis is possible only by histopathology of a liver biopsy sample or quantitative determination of the hepatic iron concentration.[15] It is important to know

the species of a diseased patient. Radiography will show hepatomegaly with or without ascites. Values of liver enzymes, such as AST, LDH and AP as well as bile acids, can be increased. Many mynahs will develop a hypoproteinemia.[62] Total serum iron and total iron binding capacity do not correlate with the presence or degree of iron storage disease in rhamphastids.[118]

Necropsy

A swollen liver with an orange-brown or marbled appearance and transudate within the abdomen may be observed on necropsy. On histopathology, deposition of iron pigment may be seen in hepatocytes and hepatic Kupffer cells, as well as in the spleen, gut wall, myocardium, kidney and pancreas.

Prophylaxis and Treatment

A low-iron diet with less than 60 to 100 ppm (mg/kg) of iron should be fed.[119] Food items containing ascorbic acid, such as citrus fruits, should not be offered, while tannin-rich tea such as oak bark tea may be used to replace drinking water several days a week.

Acute respiratory distress and apathy due to ascites must be relieved by abdominocentesis. Consider diuretics to help resolve ascites. In stable patients, phlebotomies at the rate of 1% of the bird's body weight may be performed once a week. This should induce mobilization of hepatic iron for erythrocyte production. Deferoxamine at 100 mg/kg SC q24h over a 4-month period has been proven to normalize the liver iron concentration in toucans.[15] General supportive liver therapy should be considered, using phytotherapeutics such as dandelion, milk thistle or artichoke, possibly in combination with lactulose (see Chapter 4, Nutritional Considerations).

Differential Diagnoses

Differential diagnoses include *Aspergillus* and other causes of respiratory distress in mynahs (see Table 39.9) and primary hepatic or cardiac disease. For other causes of abdominal distension (see Table 39.10).

OTHER METABOLIC DISORDERS

Amyloidosis of the liver can be observed on necropsy and histopathology of finches such as Lady Gouldian finches.[71] Hepatic lipidosis can be observed in canaries and finches fed a high-fat diet and lacking exercise.

Visceral gout due to hyperuricemia secondary to renal disease is a regular necropsy finding. Diseased birds may show signs of polyuria/polydipsia and apathy, but the most common clinical sign is sudden death. Articular gout is less commonly diagnosed in Passeriformes than in psittacines.

Skeletal malformation caused by hypovitaminosis D, in conjunction with calcium and phosphorus deficiency or imbalances, can be observed in Passeriformes as in all other avian species.

Toxic Diseases

INHALANT TOXINS

Canaries and finches are particularly susceptible to inhalant toxins because they exchange more air per gram of body weight than do larger birds. Dangers include carbon monoxide exposure (cages in car garages, leaks from gas heaters), overheated polytetrafluoroethylene (non-stick cookware), carpet freshener,[29] hair spray, glues, paints and smoke. Disinfectants used in the breeding cages, such as formaldehyde, also pose a hazard. Necropsy reveals nonspecific pulmonary congestion, pulmonary edema and/or hemorrhage (Fig 39.38). Diagnosis is based on a careful history of the bird and its environment.

PLANTS

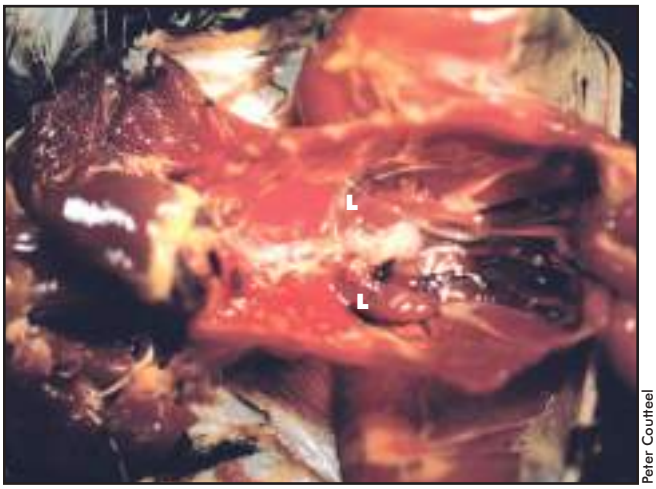
Plant toxicoses are rare. The majority of ingested plants will merely cause mild gastrointestinal signs. Plants that have proven to result in toxic reactions in canaries include avocado (*Persea americana*), dieffenbachia (*Dieffenbachia* spp.), foxglove (*Digitalis pupurea*), lupine (*Lupinus* spp.), oleander (*Nerium oleander*) and yew (*Taxus media*).[2]

PESTICIDES

Organophosphates inhibit acetylcholinesterase and cause clinical signs such as anorexia, diarrhea, ataxia, tremors and seizures.[3,7] The main source of organophosphates is the inappropriate use of insecticides that are used to combat ectoparasites, such as insecticide spray with an alcohol base administered directly on the bird, insecticide spray used in the nest pans and the addition of insecticide strips to the cage.

PHARMACOLOGIC AGENTS

Many drugs used at doses higher than recommended will cause toxicity in passerines as in other avian species. Drugs with a low therapeutic range that can easily induce signs of toxicity (neurologic signs) in canaries and finches include dimetridazole and furans.[40]



Peter Coufteel

Fig 39.38 | A canary presented with PTFE (polytetrafluoroethylene) intoxication had acute erythema and edema of lungs (L).


Peter Coufteel

Fig 39.39 | A feather cyst (arrow) is shown in a canary.

Selected Miscellaneous Diseases

NEOPLASTIC DISEASES

Passeriformes have a low incidence of tumors compared to other avian orders.[71] Reported neoplasms include tumor-like pox lesions,[38] leukosis in canaries[71] and thymoma in a finch.[65]

FEATHER CYSTS

Feather cysts are common in heavily feathered canaries (Norwich, crest and crestbred) and new color canaries.[17] Due to the histologic features, it has recently been suggested that feather cysts in canaries should be considered benign tumors, and the name "plumafolliculoma" has been proposed.[122] The condition appears to be hereditary but other factors may be involved. The most common localizations include the wings (Fig 39.39), on the back, in the shoulder region and on the chest. The cysts can occur individually or in multiple locations. Depending on the stage of the molt, the cysts will contain blood and gelatinous material or dry keratinous material.[17] Individual cysts can be lanced and curetted but will likely recur. A better option is the surgical removal of the entire cyst.[71] Typically, a hemostat is placed at the base of the cyst. The entire cyst is then removed using a radiosurgical unit. Minimal bleeding is controlled using a ferric subsulfate stick or tissue glue. If necessary, a few stitches can be placed. This procedure works well for cutaneous cysts but fails in wing cysts because the origin of the cyst is the germinal epithelium at the base of the feather follicle. On the wing, this occurs at the insertion on a bone.

CHRONIC ULCERATIVE DERMATITIS (CUD)

Chronic ulcerative dermatitis (CUD) is regularly observed in mynahs.[62] The etiology is unknown, but possibilities include metabolic diseases of the liver, malnutrition, polyomavirus, bacterial dermatitis, fungal dermatitis, lipoma, squamous cell carcinoma and giardiasis. Clinical disease is similar to CUD observed in *Agapornis* spp. and budgerigars. It is most common in the axillary body region, but the entire trunk and upper legs can be affected. It is normally pruritic. Treatment is frustrating and includes controlling secondary bacterial and fungal infections, using collars, improving diet and controlling pruritus with local anesthetics or local phytotherapeutic agents such as calendula gel. Adding an antiseptic, such as chlorhexidine in glycerol, to the bath water can help control secondary infections.

FIBER CONSTRICTION OF THE EXTREMITIES

The fibers derived from nesting material are a common cause of digital swelling, inflammation and necrosis of extremities in canaries and finches. Swelling of the feet and legs should be examined using magnification and a good light source to determine if fibers are involved. Fibers should be cut using a pointed scalpel blade or a 26- to 30-gauge needle, and the incisions should be made parallel to the long axis of the legs. Necrotic or mummified digits must be amputated. Hemorrhage is a complication after removal. Local antibiotics and bandaging may be applied.

HYPERKERATOSIS OF THE EXTREMITIES

Hyperkeratosis, which is common in canaries (Fig 39.40), may be caused by genetics (soft feather breeds),

old age, malnutrition and hormonal imbalances.[36] Treatment consists of softening and removing the hyperkeratotic lesions with water soluble creams and addressing predisposing factors, such as malnutrition, and removing leg bands. The differential diagnoses include *Knemidokoptes* mites and papillomavirus-induced warts.

CONSTRICTIONS OF THE EXTREMITIES

Possible causes of constriction of the extremities include leg bands that are too small, hyperkeratosis, swelling due to trauma or inflammation, *Knemidokoptes* mites or pox lesions.[17] Bands may be removed by application of oil and then gentle removal using a special leg band scissors or the drill of a dental unit. The primary disease is treated as necessary.

OTHER DISORDERS OF THE EXTREMITIES

Other disorders of the extremities may include frostbite, injury from wire netting or burns (eg, from hot stove plates, candles or heaters). Missing toenails or toes (bitten off by small psittacines kept in mixed aviaries or overgrown toenails that become entrapped) are common.

EGG BINDING

Pathogenesis and predisposing factors to egg binding are the same as in other avian species. Clinical signs include fluffed-up appearance of a female bird during the breeding period or a female bird sitting continuously in the nest or on the bottom of the cage. The egg can normally be palpated but definitive diagnosis is based on radiology. Conservative treatment, consisting of stabilization of the patient in a warm, humid environment, subcutaneous infusions, parenteral calcium, and PGE_{1or2} applied to the cervix may lead to spontaneous laying of the egg. If this does not occur, manual removal of the egg should be considered as soon as the bird is stable.

Procedure

The bird is induced with isoflurane anesthesia and placed in dorsal recumbency (being cognizant of the increased respiratory effort often present in the egg-bound patient). Warmed lubrication jelly is infused into the cloaca using a 2-ml syringe and a cow teat cannula. Careful digital pressure is applied until the egg can be visualized at the vagino/cloacal junction through the vent. More lubrication jelly is applied between the mucosa of the vagina and the eggshell. The ovocentesis is performed using a 5-ml syringe and an 18-gauge needle. Negative pressure is applied via the syringe at the same time the egg is collapsed by digital pressure through the abdominal wall. Constant, caudally directed digital pressure is applied so the collapsed eggshell can be visualized through the cloaca. Lubrication jelly is continually applied as necessary. Using hemostats, the collapsed eggshell is grasped, and the entire collapsed egg is gently removed. This is normally possible because the broken eggshell is still attached to the shell membrane. Any additional eggshell fragments are removed as necessary. Although this procedure can cause small lacerations, it is still considerably less traumatic than a surgical cesarean. Recovery in a warm humid environment is usually rapid and uneventful.

Peter Coufteel

Fig 39.40 | Hyperkeratosis is shown in the feet of a canary.

OTHER DISEASES OF THE FEMALE REPRODUCTIVE TRACT

Other disorders of the female passerine may include prolapse of the oviduct, excessive egg laying, salpingitis or egg-related peritonitis.

CONCUSSIVE HEAD TRAUMA

A panicked bird flying into a window or wall (often due to the detection of a predator) can induce concussive head trauma (Fig 39.41). Clinical signs include: a depressed bird sitting on the floor; neurological signs such as torticollis, opisthotonos, seizuring, paresis of a wing or leg; blood in the oral cavity, nares or ear canal; ophthalmologic lesions such as blindness, hemorrhage into the anterior or posterior chamber along with uveitis and possibly anisocoria; and sudden death. On necropsy, a hematoma on the skull can be observed. Treatment consists of: controlling swelling and inflammation with manitol; oxygen; controlling seizures (eg, with diazepam); providing supportive care, including adequate fluids (5% dextrose); and maintaining the bird in a cold, dark and quiet environment. Prognosis is guarded to poor, depending on the extent of cranial trauma.


Peter Coutteel

Fig 39.41 | Trauma on the head of a bullfinch (*Pyrrhula pyrrhula*), due to its head impacting against the wire, is commonly seen with young birds in new cages.


Peter Coutteel

Fig 39.42 | A rupture of the air sac is shown in this canary.

AIR SAC RUPTURE

Air sac rupture may be caused by trauma (bird flying into a window or wall) or chronic obstructive pulmonary disease. Clinical signs include swelling in the dorsolateral neck area due to rupture of the clavicular air sac (Fig 39.42). Treatment consists of puncturing the air sac swelling with a needle and addressing any primary disease. The likelihood of recurrence after puncturing is high.

Resources Mentioned in the Text

a. Dremel, Racine, WI, USA, 1-800-437-3635, www.dremel.com
b. True-light, Philips Lighting TLD 96 or TL950 series, Osram Biolux, www.lighting.philips.com
c. Poximune, Canary Pox Vaccine, 1-913-894-0230, www.Biomunecompany.com
d. Soludox® 15% water soluble doxycycline in citric acid. Eurovet, www.eurovetanimalhealth.com
e. PCR test for atoxoplasmosis, Department of Medical Microbiology and Parasitology, College of Veterinary Medicine, University of Georgia

References and Suggested Reading

1. Albicker-Rippinger P, Hoop RK: Common diseases in psittacines and passerines: Postmortem findings. Tierärztl Prax 27(K):245 254, 1999.
2. Arai M, Stauber E, Shropshire CM: Evaluation of selected plants for their toxic effects in canaries. J Am Vet Med Assoc 200(9):1329-1331, 1992.
3. Augspurger T, et al: Mortality of passerines adjacent to a North Carolina cornfield treated with granular carbofuran. J Wildl Dis 32(1):113-116, 1996.
4. Bains BS: Role of vitamin C in mineral metabolism. Poult Digest (Australia) April-May, 1992.
5. Baker DG, et al: An unusual coccidian parasite causing pneumonia in a northern cardinal (*Cardinalis cardinalis*). J Wildl Dis 32(1):130-132, 1996.
6. Bauck L, Brash M: Survey of diseases of the lady gouldian finch. Proc Assoc Avian Vet, 1999, pp 204- 212.
7. Bauck L, LaBonde J: Toxic diseases. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 604-613.
8. Bennewitz D: Zur Immunprophylaxe der PMV-3 Infektion bei Sittichen und Passeriformes. DVG-Tagung, Vogelkrankheiten, 1988, pp 86-103.
9. Bolte AL, Meurer J, Kaleta EF: Avian host spectrum of avipoxviruses. Avian Pathol 28:415-432, 1999.
10. Brue RN: Nutrition. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 63-95.
11. Christen C: Enzephalitozoonosis in birds: An overview. Proc Assoc Avian Vet, 2002, pp 183-185.
12. Clipsham R: Avian pathogenic flagellated enteric protozoa. Sem Avian Exotic Pet Med 4(3):112-125, 1995.
13. Clubb S: What is your diagnosis? Cryptosporidiosis in the gouldian finch. J Avian Med Surg 11(1):41-42, 1997.
14. Cork SC: Iron storage diseases in birds. Avian Pathol 29(1):7-12, 2000.
15. Cornelissen JMM, Ducatelle R, Roels S: Successful treatment of a channel-billed toucan (*Ramphastos vitellinus*) with iron storage disease by chelation therapy: Sequential monitoring of the iron content of the liver during the treatment period by quantitative chemical and image analyses. J Avian Med Surg 9(2):131-137, 1995.
16. Cornelissen JMM, Dorrestein GM: A new dermatologic disease possibly caused by flagellates in the gastrointestinal tract. Proc Assoc Avian Vet Europ Comm, 2003, pp 17-18.
17. Coutteel P: Canaries and finches in avian practice. Proc Assoc Avian Vet Europ Comm, 1997, pp 371-386.
18. Coutteel P: The importance of manipulating the daily photoperiod in canary breeding. Proc Assoc Avian Vet Europ Comm, 1995, pp 166-170.
19. Coutteel P: Veterinary aspects of breeding management in captive passerines. Sem Avian Exotic Pet Med 12(1):3-10, 2003.
20. Cray C, et al: Comparison of diagnostic assays for the detection of *Cryptosporidium*. Proc Assoc Avian Vet, 2002, pp 127-129.
21. Crissey SD, et al: Hepatic iron accumulation over time in European starlings (*Sturnus vulgaris*) fed two levels of iron. J Zoo Wildl Med 41(4):491-496, 2000.
22. Crosta L, Sironi G, Rampin T: Polyomavirus infection in Fringillidae. Proc Assoc Avian Vet Europ Comm, 1997, pp 128-131.
23. De Herdt P, et al: Megabacterium infections of the proventriculus in passerine and psittacine birds: Practice experiences in Belgium. Proc Assoc Avian Vet Europ Comm, 1997, pp 123-127.
24. Desmidt M, et al: Cytomegalo virus-like conjunctivitis in Australian finches. J Assoc Avian Vet, 1991, pp 132-136.
25. Devriese L A, et al: Tracheitis due to *Enterococcus faecalis* infection in canaries. J Assoc Avian Vet 4(2):113-116, 1990.
26. Devriese LA, et al: Streptococcal and enterococcal infections in birds. Vlaams Diergeneeskundig Tijdschrift 63(4):112-111, 1994.
27. Dhondt AA, Tessaglia DL, Slothower RL: Epidemic mycoplasma conjunctivitis in house finches from eastern North America. J Wildl Dis 34(2):265-280, 1997.
28. Dom P, et al: Papilloma-like virus infections in canaries (*Serinus canaria*). Proc Assoc Avian Vet Europ Comm, 1993, pp 224-231.
29. Doolen M: A case of thirteen deaths associated with carpet freshener toxicity. Proc Assoc Avian Vet, 1994, pp 93-94.
30. Dorrestein GM: Diagnostic necropsy and pathology. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 158-169.
31. Dorrestein GM: Passerine and softbill medicine and surgery. Proc Assoc Avian Vet, 1997, pp 437-452.
32. Dorrestein GM: Passerines. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 867-885.

33. Dorrestein GM: Passerines and exotic softbill medicine. Proc Assoc Avian Vet, 2000, pp 363-378.
34. Dorrestein GM: Diagnostic approaches and management of diseases in captive passerines. Sem Avian Exotic Pet Med 12(1):11-20, 2003.
35. Dorrestein GM: Passerine and softbill therapeutics. Vet Clin No Am Exot Anim Pract 3(1):35-57, 2000.
36. Dorrestein GM, Kummerfeld N: Singvögel. *In* Gabrisch K, Zwart P (eds): Krankheiten der Heimtiere. Schlütersche Verlag, Hannover, 2001, pp 327-396.
37. Dorrestein GM, Schrijver J: A genetic disorder of vitamin A metabolism is recessively white canaries. Tijdschr Diergeneeskd 107(21):795-9, 1982.
38. Dorrestein GM, van der Hage MH, Grinwis G: A tumour-like pox-lesion in masked bull finches (*Pyrrhula erythaca*). Proc Assoc Avian Vet Europ Comm, 1993, pp 232-240.
39. Dorrestein GM, et al: Quill mite (*Dermoglyphus passerinus*) infestation of canaries (*Serinus canaria*): Diagnosis and treatment. Avian Pathol 26:195-199, 1997.
40. Dumonceaux G, Harrison GJ: Toxins. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 1030-1052.
41. Dustin LR: Clinical conditions in a pet canary practice. J Avian Med Surg 4(2):80-82, 1990.
42. Filippich LJ, O'Donoghue PJ: Cochlosoma infections in finches. Aust Vet J 75(8):561-563, 1997.
43. Filippich LJ, Parker MG: Megabacteria and proventricular/ventricular disease in psittacines and passerines. Proc Assoc Avian Vet, 1994, pp 287-293.
44. Flammer K, Whitt-Smith D, Papich M: Plasma concentrations of doxycycline in selected psittacine birds when administered in water for potential treatment of *Chlamydophila psittaci* infections. J Avian Med Surg 15(4):276-282, 2001.
45. Frasca S, Hinckley L, Forsyth MH: Mycoplasma conjunctivitis in a European starling. J Wildl Dis 33(2):336-339, 1997.
46. French RA, et al: Parasitology and pathogenesis of *Geopetita aspiculata* (Nematoda: Spiruridae) in zebra finches (*Taeniopygia guttata*): Experimental infection and new host records. J Zoo Wildl Med 25(3):403-422, 1994.
47. Fudge AM: Diagnosis and treatment of avian bacterial diseases. Sem Avian Exotic Pet Med 10(1):3-11, 2001.
48. Garcia A, et al: Diagnosis of polyomavirus infection in seedcrackers (*Pyrenestes* sp.) and bluebills (*Spermophaga haematina*) using DNA in situ hybridization. Avian Pathol 23(3):525-537, 1994.
49. Gerlach H: Bacteria. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 949-983.
50. Gerlach H: Megabacteriosis. Sem Avian Exotic Pet Med 10(1):12-19, 2001.
51. Gerlach H: Viruses. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 863-948.
52. Goldsmith TL: Documentation of passerine circoviral infection. Proc Assoc Avian Vet, 1995, pp 349-350.
53. Haesebrouck F, et al: Effect of antimicrobial treatment on the course of an experimental *Yersinia pseudotuberculosis* infection in canaries. Avian Pathol 24:273-283, 1995.
54. Handbook of the Birds of the World: The internet bird collection. Linx editions. www.hbw.com/ib/. Date accessed: February 21, 2003.
55. Hoop, RK: *Mycobacterium tuberculosis* infection in a canary (*Serinus canaria*) and a blue-fronted Amazon parrot (*Amazona amazona festiva*). Avian Dis 46:502-504, 2002.
56. Hoop RK, Böttger EC, Pfyffer GE: Etiological agents of mycobacterioses in pet birds between 1986 and 1995. J Clin Micro 34(4):991-992, 1996.
57. Hoop RK, Ossent P, Pfyffer GE: *Mycobacterium genavense*: A new cause of mycobacteriosis in pet birds? Proc Assoc Avian Vet Europ Comm, 1995, pp 1-3.
58. Jones MP, Orosz SE: The diagnosis of aspergillosis in birds. Sem Avian Exotic Pet Med 9(2):52-58, 2000.
59. Joseph V: Infectious and parasitic diseases of captive passerines. Sem Avian Exotic Pet Med 12(1):21-28, 2003.
60. Kaleta EF: Herpesviruses of birds: A review. Avian Pathol 19:193-211, 1990.
61. Khan MSR, et al: Development of a blocking enzyme-linked immunosorbent assay for the detection of avian polyomavirus-specific antibodies. J Virol Meth 89(1,2):39-48, 2000.
62. Korbel R, Kösters J: Beos. *In* Gabrisch K, Zwart P (eds): Krankheiten der Heimtiere. Schlütersche Verlag, Hannover, 2001, pp 397-428.
63. Korbel RT: Ultraviolet perception in birds. Proc Assoc Avian Vet, 1999, pp 77-81.
64. Kuebber-Heiss A, Juncker M: Spiruroidosis in birds at an Austrian zoo. Proc Assoc Avian Vet Europ Comm, 1998, pp 157-160.
65. Latimer KS, Rakich PM, Weiss R: Thymoma in a finch. J Avian Med Surg 15(1):37-39, 2001.
66. Leger JS, Read DH, Shivaprasad HL: Passerine protozoal sinusitis: An infection you should know about. Proc Assoc Avian Vet, 1998, pp 157-160.
67. Lindsay DS, et al: Central nervous system toxoplasmosis in roller canaries. Avian Dis 39:204-207, 1995.
68. Little SE, et al: Developing diagnostic tools to further our understanding of *Atoxoplasma* species. Proc Assoc Avian Vet, 2001, pp 157-159.
69. Loomis MR, Wright JF: Treatment of iron storage disease in a Bali mynah. Proc Am Assoc Zoo Vet, 1993.
70. Loudis BG: PMV-3 outbreak: Presentation, diagnosis and management. Proc Assoc Avian Vet, 1999, pp 223-227.
71. Macwhirter P: Passeriformes. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Brentwood, TN, HBD Int'l, Inc, 1999, pp 1173-1199.
72. Martinez F, Munoz E: *Atoxoplasma* spp. in a hybrid passerine (*Serinus canarius x Carduelis cannabina*). Avian Pathol 27:420-422, 1998.
73. Mashima TY, et al: Evaluation of treatment of conjunctivitis associated with *Mycoplasma gallisepticum* in house finches (*Carpodacus mexicanus*). J Avian Med Surg 11(1):20-24, 1997.
74. Massey JG: Clinical medicine in small passerines. Proc Assoc Avian Vet, 1996, pp 49-53.
75. Mete A, et al: A comparative study of iron retention in mynahs, doves and rats. Avian Pathol 30:479-486, 2001.
76. Moore RP, Snowden KF, Phalen DN: A method of preventing transmission of so-called "megabacteria" in budgerigars (*Melopsittacus undulatus*). J Avian Med Surg 15(4):283-287, 2001.
77. Murray JM, et al: Use of PCR testing in diagnosing chlamydiosis. Roundtable discussion. J Avian Med Surg 14(2):122-128, 2000.
78. Mysore J, et al: Feather loss associated with circovirus-like particles in finches. Proc Am Assoc Vet Lab Diagn, Histopathology section, 1995.
79. Norton TM, et al: Bali mynah captive medical management and reintroduction program. Proc Assoc Avian Vet, 1995, pp 125-136.
80. Ottinger MA, Bakst MR: Endocrinology of the avian reproductive system. J Avian Med Surg 9(4):242-250, 1995.
81. Page CD, Haddad K: Coccidial infections in birds. Sem Avian Exotic Pet Med, 4(3):138-144, 1995.
82. Patton S: Diagnosis of *Toxoplasma gondii* in birds. Proc Assoc Avian Vet, 1995, pp 75-78.
83. Peccati C, Croci C, Böttger EC: Mycobacteriosis by *Mycobacterium genavense* in captive bred finches. Proc Assoc Avian Vet Europ Comm, 1999, pp 27-29.
84. Pennycott TW, et al: Causes of death of wild birds of the family Fringillidae in Britain. Vet Rec 143(6):155-158, 1998.
85. Phalen DN, et al: Genetic diversity in twenty variants of the avian polyomavirus. Avian Dis 3(2):207-218, 1999.
86. Phalen DN: Avian mycobacteriosis. *In* Bonagura JD (ed): Kirk's Current Veterinary Therapy XIII: Small Animal Practice, 2000, pp 1116-1118.
87. Poelma FG, et al: Cochlosomosis: A problem in raising waxbills kept in aviaries. Tijdschr Diergeneeskd 103(11):589-593, 1978.
88. Powers LV, Flammer K, Papich M: Preliminary investigation of doxycycline plasma concentrations in cockatiels (*Nymphicus hollandicus*) after administration by injection or in water or feed. J Avian Med Surg 14(1):23-30, 2000.
89. Quiroga MI, et al: Diagnosis of atoxoplasmosis in a canary (*Serinus canaria*) by histopathologic and ultrastructural examination. Avian Dis 44:465-469, 2000.
90. Ramis A, et al: *Mycobacterium genavense* infection in canaries. Avian Dis, 40(1):246-251, 1996.
91. Ravelhofer-Rotheneder K, et al: "Megabacteria": Taxonomic classification and significance as pathogen in various bird species. Proc Assoc Avian Vet Europ Comm, 2001, pp 189-190.
92. Ravelhofer-Rotheneder K, et al: Taxonomic classification of "megabacteria"-isolates originating from budgerigars (*Melopsittacus undulatus*). Tierärztliche Praxis 28(6):415-420, 2000.
93. Ringer RK: Selected physiology for the avian practitioner (reproduction). *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1986, pp 78-80.
94. Ritchie BW: Herpesviridae. *In* Avian Viruses: Function and Control. Lake Worth, FL, Wingers Publishing, 1995, pp 171-222.
95. Ritchie BW: Papovaviridae. *In* Avian Viruses: Function and Control. Lake Worth, FL, Wingers Publishing, 1995, pp 127-172.
96. Ritchie BW. Paramyxoviridae. *In* Avian Viruses: Function and Control. Lake Worth, FL, Wingers Publishing, 1995, pp 253-283.
97. Ritchie BW: Poxviridae. *In* Avian Viruses: Function and Control. Lake Worth, FL, Wingers Publishing, 1995, pp 285-311.
98. Ryan T: Use of light in aviculture and avian medicine. Proc Assoc Avian Vet, 1999, pp 179-186.
99. Sandmeier P, et al: Polyomavirus infections in exotic birds in Switzerland. Schweizer Archiv für Tierheilkunde 141(5):223-229, 1999.
100. Sandmeier P, Hoop RK, Bosshart G: Cerebral mycobacteriosis in zebra finches (*Taeniopygia guttata*) caused by *Mycobacterium genavense*. Proc Assoc Avian Vet Europ Comm, 1997, pp 119-122.
101. Scott JR: Passerine aviary diseases: Diagnosis and treatment. Proc Assoc Avian Vet, 1996, pp 39-48.
102. Shihmanter E, et al: Isolation of avian serotype 3 paramyxoviruses from imported caged birds in Israel. Avian Dis 42:829-831, 1998.
103. Shivaprasad HL, et al: Retrospective study of cytomegalovirus-like infection in finches. Proc Assoc Avian Vet, 2002, pp 209.
104. Shivaprasad HL: An overview of paramyxovirus 3 (PMV-3) infection in psittacines and passerines. Proc Assoc Avian Vet, 1998, pp 147-149.
105. Speer BL: Taxonomy for the avian veterinarian: The orders and families of birds. Proc Assoc Avian Vet, 2000, pp 379-399.

106. Sreter T, Varga I: Cryptosporidium in birds: A review. Vet Parasitol 87:261-279, 2000.
107. Stoll R, et al: Molecular and biological characteristics of avian polyomaviruses: Isolates from different species of birds indicate that avian polyomaviruses form a distinct subgenus within the polyomavirus genus. J Gen Virol 74:229-237, 1993.
108. Suedmeyer WK, Haynes N, Roberts D: Clinical management of endoventricular mycoses in a group of African finches. Proc Assoc Avian Vet, 1997, pp 225-227.
109. Taylor EJ: An evaluation of the importance of insoluble versus soluble grit in the diet of canaries. J Avian Med Surg 10(4):248-251, 1996.
110. Tell LA, Woods L, Cromie RL: Mycobacteriosis in birds. Revue Scientifique et Technique - Office International des Dpizooties 20(1):180-203, 2001.
111. Todd D, et al: Nucleotide sequence-based identification of a novel circovirus of canaries. Avian Pathol 30:321-325, 2001.
112. Tully TN, et al: What is your diagnosis? (Avipoxvirus in a canary *Serinus canaria*). J Avian Med Surg 10(3):199-202, 1996.
113. Tully TN: Update on *Chlamydophila psittaci*. Sem Avian Exotic Pet Med 10(1):20-24, 2001.
114. Upton SJ, et al.: A new species of *Isospora* (Apicomplexa: Eimeriidae) in canaries (*Serinus canaria*) from Rothschild's mynah, *Leucopsar rothschildi* (Passeriformes: Sturnidae), and comments concerning the genera *Atoxoplasma*. Syst Parasit 48:47-53, 2001.
115. Velasco MC: Candidiasis and cryptococcosis in birds. Sem Avian Exotic Pet Med 9(2):75-81, 2000.
116. Williams SM, et al: Ocular and encephalic toxoplasmosis in canaries. Avian Dis 45:262-267, 2001.
117. Woods LW, Latimer KS: Circovirus infection of nonpsittacine birds. J Avian Med Surg 14(3):154-163, 2000.
118. Worell A: Further investigations in Ramphastids concerning hemochromatosis. Proc Assoc Avian Vet, 1993, pp 98-107.
119. Worell AB: Toucans and mynahs: *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1997, pp 910-917.
120. Young P: Selected herpesviral diseases of birds. Sem Avian Exotic Pet Med 4(2):62-71, 1995.
121. Zantop DW: Respiratory distress in two canary aviaries. Proc Mid-Atlantic States Assoc Avian Vet, 1995, pp 195-196.
122. Zwart P, Grimm F: Plumafolliculoma (feather cyst) in canaries (*Serinus canaria*): A benign tumor. Proc Assoc Avian Vet Europ Comm, 2003, pp 241-247.

CHAPTER
40

Management of Raptors

JAIME SAMOUR, MVZ, PhD, Dipl ECAMS

Fig 40.1 | Birds of prey have been used for hunting for thousands of years. This practice is believed to have started in Central Asia and its popularity soon spread across the Mogul and Ottoman empires.

The first manuscript in the Arab literature related to raptor medicine, "Benefits of Birds and the Comprehensive Treatment of their Diseases," was written by Adham bin Mahres Al Bahili during the reign of the 4th Caliph Haroon Al Rashid Al Abbasi (765 to 808 A.D.). This was a manuscript comprising 153 chapters in which issues such as diagnoses and diseases were first described. This manuscript was enriched by the participation of Al Hajaj bin Khaytamah during the reign of the 5th Caliph Mohammed Al Amin bin Haroon Al Rashid Al Abbasi (786 to 813 A.D.).

Sadly, there was a crucial hiatus in the continuing contribution of Arab literature to raptor medicine with the Mogul occupation of Baghdad in 1258 A.D. (Fig 40.1). Throughout the succeeding decades, falconry continued to decline in the domains of the Ottomans and Moguls. However, deep into the Arabian Peninsula, Bedouins continued the seasonal traditions of trapping, training and hunting with falcons as a means of supplementing their basic diet.

Falconry is still widely practiced in the Middle East, but is now considered a sport. The large population of falcons maintained annually in captivity prompted the creation of modern medical and research facilities in several countries in the region. These centers have contributed significantly in enhancing our knowledge of raptors during the past 25 years, particularly in falcon medicine (Fig 40.2). Similarly in North America and Europe, dwindling populations of peregrine falcons and bald eagles prompted the creation of rescue and rehabilitation centers and captive breeding programs.

Owing to the need to respond to medical and conservation trends, raptor medicine has become one of the fastest growing disciplines in avian medicine. This has

Fig 40.2 | The saker falcon (*Falco cherrug*) is the most popular species used in the sport of falconry in the Middle East. This species is the largest of the desert falcons. Arab falconers recognize several colors and color patterns for this species. Shown is a much-prized white saker falcon.

Fig 40.3 | The correct way to handle a large raptor is demonstrated on an immature white-tailed sea eagle (*Haliaeetus albicilla*). The towel placed around the body prevents damage to the feathers.

Fig 40.4 | Vultures have strong curved beaks capable of inflicting serious injuries during handling. Only trained personnel provided with suitable equipment (eg, leather gloves and aprons) should handle such birds.

Fig 40.5 | A typical free-flying molting room in the Middle East measures 15 x 8 x 5 m and is provided with red desert sand as a substrate. A single platform-type perch is placed around the room. Some birds have already been released, while others wait on stands until they are settled into the new environment.

been due to the dedication of individuals on both sides of the Atlantic and in the Middle East. This chapter is dedicated to these pioneers who, by their example, have inspired the author over the years.

Capture, Restraint and Transportation

Most raptors kept in captivity have strongly curved upper beaks and long, sharp, curved talons (the exception being vultures). Therefore, only well-trained personnel wearing adequate protective equipment (eg, leather gloves and aprons) should undertake handling and restraint of any of those species (Fig 40.3).

The techniques for capture and restraint vary with the circumstances (Fig 40.4). Birds kept free-flying in aviaries can be captured using nets fitted on long poles and later restrained by hand (Fig 40.5). Many birds are presented to veterinarians in transport crates. The position of the bird should be assessed before introducing the hands and arms into the crate. Make sure beforehand that the bird can easily fit through the door. Never introduce unprotected hands inside a crate holding a raptor. Trained raptors are easy to handle, since such birds are commonly hooded and can be captured on a gloved fist or a perch. The author favors capturing medium to small raptors by hand, then wrapping them with a soft towel to avoid damage to the feathers. Masking tape can be used to secure the towel around the body (Fig 40.6). Two operators should always be involved in the handling of large raptors (eg, vultures, eagles). Commonly, one oper-

Fig 40.6 | Two operators are involved when handling a saker falcon. One technician holds the leash with the gloved hand in case the procedure is aborted, and a second grasps the bird and casts it down on a soft cushion. The falcon is then wrapped with a thin towel to prevent feather damage.

Fig 40.7 | Weighing is an integral part of the medical management of raptors. A technician weighs a saker falcon using an electronic scale accurate to 1 g.

ator places one arm around the wings and holds the bird firmly against his/her body; the second hand holds the legs. While holding the legs, one finger should be placed between the tarsi to prevent lacerations to the skin around the joints. A second operator holds the bird's head and provides further assistance, if required.

Commercially available transport cages used for dogs and cats are suitable for transporting raptors. However, slight modifications such as placing burlap or canvas over grills and attaching pads to the roof of the cage, perches and/or carpet on the floor are all highly recommended measures. Trained hooded raptors usually travel well on perches when the cage is placed in the back of the vehicle or within the cabin of an airplane. During transport, it is highly recommended to protect the tail feathers with a tail guard made of lightweight cardboard or radiology film, fixed to the feathers with masking tape.

CLINICAL EXAMINATION

Clinical examination is similar to that performed on psittacines. The questions related to the clinical history of a bird or a flock can be grouped under three main headings: general clinical details, housing and feeding/watering (Tables 40.1-40.3).

Weighing

Raptors should be weighed upon presentation and regularly thereafter while under medical care. The body weight is a useful measurement in the assessment of health and disease and in monitoring the response to treatment. In addition, the weight of the bird is important for other purposes, such as sex determination, taxonomy or testimony in legal cases. Very often, a trained hooded raptor can be enticed to stand on a scale fitted with a perch. Commercially available electronic scales provided with perching surfaces are recommended (Fig 40.7).

Table 40.1 | General Clinical Details*

- Body weight
- Age
- Sex
- Origin (wild-caught/captive-bred)
- Chief clinical signs
- Duration of disease
- Attitude
- Flight performance
- Frequency and consistency of mutes
- Molting status
- Reproductive status
- Previous medication/treatments

Table 40.2 | Housing*

- Layout of cage/enclosure/aviary
- Size (height, length, width)
- Structural materials (posting, mesh, shade, wind breakers)
- Flooring (substrate)
- Furniture (perches, nesting ledges)
- Feeding and watering utensils
- Location in relation to other aviaries, buildings, roads
- Vegetation in and around aviary
- Proximity of livestock, hay stores, gardening materials
- Companions (number, sex)
- Contact with feral species

Table 40.3 | Feeding/Watering*

- Diet (food items)
- Dietary management (feedings, seasonal changes)
- Food item source, storage and handling
- Appetite
- Water consumption
- Crop emptying time
- Vomition/regurgitation
- Casting

*Modified[76]

Conversely, birds can be placed on a scale while anesthetized or wrapped in a soft towel. Large birds such as eagles and vultures can be weighed together with the handler stepping onto a scale and the weight of the handler subsequently subtracted. The scales' accuracy should be +/- 1% of the body weight of the bird. With raptors used for falconry, the weight of falcon accessories (hood, jesses, bells) has to be taken into consideration. It is paramount importance that the bird be weighed with an empty crop.

PHYSICAL EXAMINATION

In common with other avian species, the physical examination of raptors involves handling and restraint. However, trained hooded raptors can be partially examined on the fist of the handler or while standing on a perch. It is important to carry out a significant part of the physical examination without handling and restraining the bird (Table 40.4). If the bird is a free-living or untrained individual, it will require restraint. Wrapping the bird with a soft towel is always very helpful in avoiding injuries to the main flight feathers. Extreme care should be exercised when examining a raptor. If the bird catches a handler with one or both feet during the handling process (commonly known as being "footed" or "taloned"), it is dangerous to attempt unlocking the grip. In most cases, the best solution is to release the bird. Subsequently, the bird can be recaptured within the room or aviary or recovered back to the glove if the bird is fitted with a leash and jesses.

Endurance Test

The endurance or stress test is very useful in assessing both the health and diseases of the respiratory system of captive raptors. This test can be carried out only on trained, small to medium-sized raptors, eg, hawks or falcons used in falconry. The respiratory rate is first obtained with the bird completely at rest and away from any noise or disturbance. As a general rule, the respiratory rate of a healthy raptor is 20 to 25 respirations per minute. Then, while on the glove, the bird is stressed by letting it vigorously flap its wings for 30 seconds. After this time, the bird is placed back on its perch and is allowed to rest for 2 minutes. The respiration is then assessed based on frequency and nature. After 2 minutes of complete rest, the respiratory rate is again obtained, which, in a healthy hawk or falcon, should be similar to the original prestress rate. Birds with lower respiratory system diseases would show deep, mostly abdominal respiration, with bobbing of the tail and body. The rate might be elevated to 80 to 120 respirations per minute. Radiology, endoscopy and hematology analyses are all highly recommended if a bird fails the endurance test. Examples of other causes of increased respiration following the endurance test include anemia, hyperthermia, septicemia, cardiac disease and ascites.

Table 40.4 | Systematic Examination of Raptors

Anatomical Site	Examination
Eyes and eyelids	Symmetry, appearance, shape, discharge, wounds, swellings
Beak (upper/lower)	Appearance, grooves, cracks, splits, shape
Nares (nostrils)	Symmetry, shape, foreign material (eg, sand), discharge
Oropharynx	Color, swellings, caseous masses, blunted papillae, foreign bodies (eg, bones, tendons, string)
Ears	Symmetry, foreign bodies (eg, sand, ectoparasites), discharge
Neck/crop	Swellings, wounds, impaction
Body (chest and back)	Wounds, swellings, pectoral muscle mass, ectoparasites
Cloaca	Urate/feces seepage, swelling, masses
Wings	Symmetry, wounds, swellings, fractures, feather integrity
Tail	Swellings, feather integrity
Uropygial gland	Size, shape, quantity and consistency of oil
Legs	Symmetry, wounds, swellings, fractures
Feet	Symmetry, grip strength, skin condition, temperature, swellings, wounds, talon condition

Clinical Laboratory Diagnosis

Clinical laboratory diagnosis is an essential component of raptor medicine. In this respect, veterinarians should be aware of the various assays available and be familiar with the protocols for the collection, submission and analyses of samples for the various assays (Table 40.5).

ADMINISTRATION OF MEDICATION

There is a wide choice of routes for the administration of medications to raptors. Each has its own advantages and disadvantages that should be taken into consideration before a particular route is selected (Table 40.6).

Bandages, Dressings and Casts

The placement of bandages, dressings and casts is part of the routine care of injured raptors or part of post-surgical protocols (Table 40.7).

The bandage most widely used among veterinarians working with raptors is a flexible self-adherent bandage[a] that has revolutionized bandaging in veterinary medicine. It should be noted that this material and similar bandages will tighten when wet. Therefore, birds should be kept indoors, and access to open containers of water provided in the cages should be limited or monitored (Table 40.8). For further information on dressing and bandages of raptors, the reader is referred to a recent comprehensive publication.[8]

Foot castings are conforming devices commonly used in raptor medicine to prevent pressure-related trauma to a

Table 40.5 | Diagnostics

Discipline	Specimens	Assays
Hematology	Blood samples for partial or complete hematology analyses	RBC, Hb, PCV, MCV, MCH, MCHC, WBC, differential white cell count, thrombocyte count and fibrinogen estimation
Blood chemistry	Blood samples for partial or complete blood chemistry analyses	Glucose, total protein, albumin, globulin, A:G ratio, urea, uric acid, creatinine, bile acids, ALT (SGPT), ALP, GGT, AST (SGOT), CK, LDH, cholesterol, triglycerides, calcium, sodium, potassium, chloride, ionized Ca, Phos
Parasitology	Biopsies, intestinal content, feathers, fecal samples, skin scrapping, swabs, tissue samples, worms (whole/section)	Direct wet smear, flotation, histology, staining with methylene blue stain, Lugol's iodine, rapid stains
Bacteriology	Aspirates, air sac/tracheal/crop washing, biopsies, swabs, tissue samples	Aerobic and anaerobic cultures, antibiotic sensitivity testing, staining with Gram's stain and Ziehl-Neelsen stain
Mycology	Aspirates, air sac/tracheal/crop washing, biopsies, impression smears, swabs, tissue samples	Fungal culture, clarifying with potassium hydroxide, rapid stains, lactophenol cotton blue, India ink
Serology	Blood samples	Antibody and antigen (eg, ELISA, IFA, PCR)
Virology	Blood samples, tissues, swabs	Embryonated egg culture, cell culture, electron microscopy, PCR
Cytology	Aspirates, impression smears, swabs	Staining with Gimenez stain, Papanicolaou stain, Macchiavello stain, rapid stains
Histopathology	Biopsies, tissue samples	Staining with hematoxylin and eosin stain, periodic acid-Schiff stain, PCR

Table 40.6 | Routes Commonly Used for the Administration of Medications in Raptors

Route of Administration	Site of Administration	Remarks
Oral in water or food	Oropharynx, crop	Raptors do not always drink. It might be possible to hide medication in food.
Oral gavage		Danger of aspiration of medication. Tubes or cannulae with sharp ends should be avoided.
Topical	Eyes, eyelids, ear, oropharynx, crop, skin	Excessive use of creams or ointments might cause matting of feathers; antiseptic solutions and sprays might stain feathers.
Intramuscular	Pectoral muscles, quadriceps muscles	Main choice is pectoral muscles, avoid using long needles; irritant injections might cause muscle damage.
Subcutaneous	Dorsal neck, crural area	When administering a large volume, distribute in different sites.
Intravenous	Jugular vein (right), basilic vein, saphenous vein	Avoid lacerating vein; ensure hemostasis post procedure.
Intraosseous	Proximal tibiotarsus, distal ulna	Placement of intraosseous cannula; efficient in debilitated birds.
Intranasal	Nasal cavities	Avoid irritating substances.
Intrasinal	Infraorbital sinuses	Avoid irritating substances; effective at treating sinusitis through flushing and direct injection.
Intratracheal	Trachea, tracheobronchial syrinx, bronchi	Avoid irritating substances; avoid large volumes.
Inhalation therapy (nebulization)	Upper respiratory system	Effective in the treatment of upper and lower respiratory disease; might lead to environmental contamination.

Table 40.7 | Use and Functions of Bandages

1. Avoid further damage (eg, self-inflicted lesions)
2. Prevent desiccation of tissue
3. Avoid contamination following surgical intervention
4. Holding dressings in place
5. Providing localized pressure to prevent hemorrhage following trauma or surgery
6. Prevent further trauma (eg, fractures)
7. Minimize pain post-surgery
8. Maintain intravenous or intraosseous fluid lines

Table 40.8 | Types of Dressings and Application

Type	Application
Adhesive Dressings	
Transparent dressings[b]	Thin film with a non-latex hypoallergenic adhesive. Allows vapor and oxygen exchange, protects from outside contamination.
Non-Adhesive Dressings	
Hydrocolloid dressing[c]	Adheres to skin but not to wound, creating a gelatinous mass over wound that provides a suitable environment for healing.
Moisture/vapor-permeable dressing[d]	Adequate maintenance of an aerobic environment under the dressing preventing scab formation and promoting epithelialization.
Low-adherent absorbent dressing[e]	Dry dressing with maximum absorbance ideal for infected suppurating wounds.
Petrolatum-impregnated fine mesh gauze[f]	Ideal for large areas of skin damage (eg, abrasion); prevents skin desiccation.

newly created wound in the postoperative treatment of pododermatitis or bumblefoot. Conversely, these can be used to prevent pressure sores to the opposite foot after fracture repair of a leg or to provide comfort during the non-surgical treatment of early bumblefoot lesions. Several materials and designs have been proposed over the years, ranging from a semi-rigid bridge made of thermoplastic tape[g] under the foot, rigid shoes made of styrene plastic polymer,[66] and form-fitting bandages hardened with epoxy glue[70] to a doughnut made of a ring heavily covered with soft bandaging (N.A. Forbes, personal communication). The author prefers to use a soft shoe made from 15- or 20-mm-thick rubber sheet, a material commonly used to make beach sandals. The form of the shoe is cut and shaped with a sharp blade. Round shapes to accommodate the toes and the ball of the foot are made using a red-hot rod. The shoe is fitted to the foot with a light conforming bandage.

Surgery

Many of the surgical procedures and ancillary techniques used in general avian medicine are applicable to raptors (Table 40.9). A short review of the different procedures and the different applications follows. There are other more specific surgical procedures pertinent to raptors and brief descriptions are provided.

KEEL INJURIES

Raptors in general, particularly those used in the sport of falconry, are prone to injuries of the carina, or prominence of the ventral median section of the keel, sustained when they crash on the ground during training exercises with lures or during fights with quarry. The most common type of injury is a longitudinal wound involving the skin and underlying tissue, but sometimes the lateral muscular mass is involved. If the injury is recent, closure should be attempted using conventional surgical techniques; if chronic, dry scabs and fibrous tissue should be debrided. In more severe cases with trauma or osteomyelitis of the carina, debridement of affected bone might be necessary to allow adequate closure.

CLAW DETACHMENT

Most raptors have long, curved talons integrated with a hard, highly keratinized casing covering the dorsal and lateral aspects, and a ventral, much softer plate. Very often, due to excessive length, deformities or trauma, claws can become detached, leading to extensive hemorrhage and exposure of the last phalanx. Intervention should be accomplished as soon as possible to maintain viability of the germinal tissue of the claw. The use of a hydrocolloid dressing[c] and a conforming bandage is indicated. These should be changed every 5 to 7 days. Regrowth of the claw is possible, but this is a slow process sometimes requiring up to 2 to 3 months. Supplementation with 25 μg/kg biotin PO[h] is recommended during the regrowth period.

Distal Necrosis

This term is commonly used to describe necrosis of the terminal ends of the digits. The condition is often associated with extensive scabbing related to pox infection or frostbite. In the Middle East, there is a condition characterized by avascular necrosis of the distal end of the third digit (Fig 40.8). This is more often seen at the end of the hunting season and is very likely associated with cardiovascular changes as a result of a sudden cessation of exercise. A similar theory has been postulated for the development of bumblefoot in raptors.[26,29] Distal necrosis invariably leads to amputation of the digit (Fig 40.9).

Table 40.9 | General Surgical Procedures and Indications in Raptors

Surgery	Indication
Sinusotomy	Removal of trichomoniasis granulomas, draining and flushing in severe obstructive bacterial or fungal sinusitis
Ingluviotomy	Removal of foreign bodies and trichomoniasis granulomas, removal of ingested food in sour crop
Tracheotomy	Removal of aspergillomas and trichomoniasis granulomas from tracheal lumen and syrinx
Celiotomy	Removal of aspergillomas pre- or post-medical treatment
Proventriculotomy Ventriculotomy	Removal of foreign bodies, impaction
Salpingohysterectomy	Removal of uterus and oviduct, sterilization of females
Orchidectomy	Removal of testes, sterilization of males
Vasectomy	Sterilization of males (libido intact)
Cloacopexy	Correction of cloacal prolapse

Table 40.10 | Common Endoscopy Applications in Raptor Medicine*

Application	Anatomical Site
Otoscopy	External auditory canal
Rhinoscopy	Nasal cavities
Pharyngoscopy	Oropharynx
Tracheoscopy	Trachea/tracheobronchial syrinx/bronchi
Ingluvioscopy	Crop
Esophagoscopy	Esophagus
Gastroscopy	Proventriculus, ventriculus
Celoscopy	Coelomic cavity
Cloacoscopy	Cloaca

*Modified[77]

Ring Constriction

This is a condition characterized by an annular or circumferential constriction at a particular area of the toes. The etiology in many cases remains unclear. In the Middle East, ring constriction is very often observed around the hallux or first toe of hunting falcons caused by entanglement with the thin, rope-like jesses used in Middle Eastern falconry (Fig 40.10). Treatment of ring constriction entails the removal of the annular scab and attempted closure with conventional suturing techniques. If suturing is not possible due to a large gap between the wound edges, hydrocolloid dressings[c], together with a conforming bandage changed at regular intervals, is used to achieve healing by granulation and epithelialization (Fig 40.11) (see Chapter 35, Surgical Resolution of Soft Tissue Disorders).

ENDOSCOPY

Endoscopy (*Greek: endon = within, skopein = to examine*) is an essential medical procedure used routinely in raptor medicine as an ancillary technique in the diagnosis of certain medical conditions (eg, aspergillosis, candidiasis) (Fig 40.12), assisting in the collection of

Fig 40.8 | Distal necrosis is a poorly understood condition in raptors. In falcons, the middle digit is most often affected. The etiology might involve vascular disorders originating from a sudden cessation of activity.

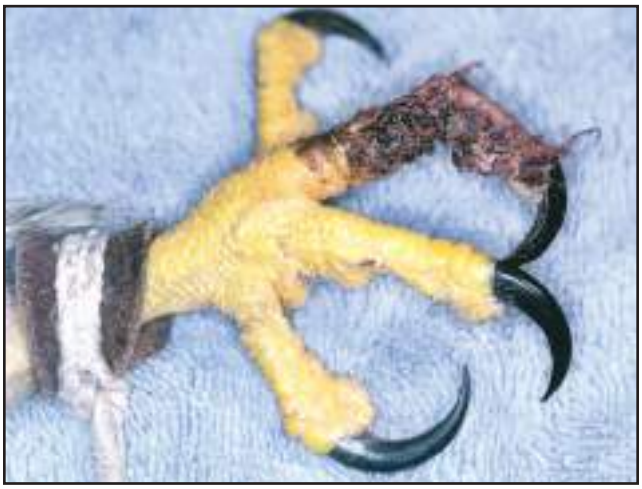


Fig 40.9 | Peregrine falcon (*Falco peregrinus*) with a severe self-inflicted injury in the middle digit. This is a common occurrence in newly wild-caught individuals of this species. The damage was extensive, necessitating complete amputation of the digit.

Fig 40.10 | Arab falconers use jesses fitted with a thin cotton or nylon string. This string very often becomes entangled around the hallux or first digit, leading to ring constriction.

Fig 40.11 | Severe ring constriction around the hallux. Satisfactory resolution can be achieved by periodic application of hydrocolloid dressings to the affected area. More severe cases may require surgical reconstruction.

Fig 40.12 | Rigid endoscopes are normally used to examine the upper digestive tract of raptors under anesthesia. Candidiasis and esophageal trichomoniasis are two of the diseases diagnosed with the aid of endoscopy.

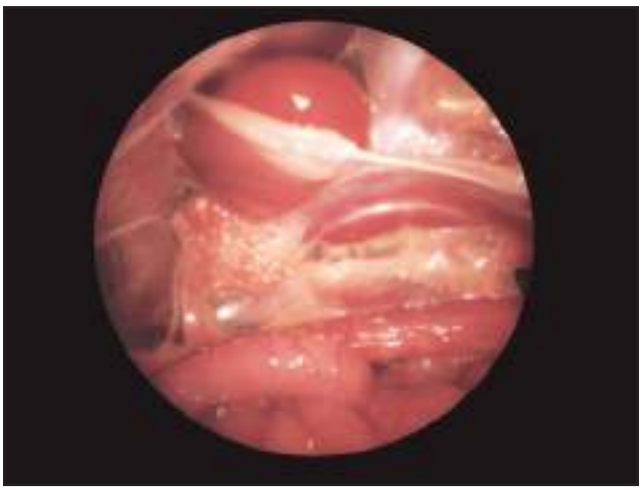


Fig 40.13 | Endoscopic view of the reproductive tract of an immature bird. Endoscopy offers the opportunity to examine the organs directly for signs of health and disease and to collect biopsies.

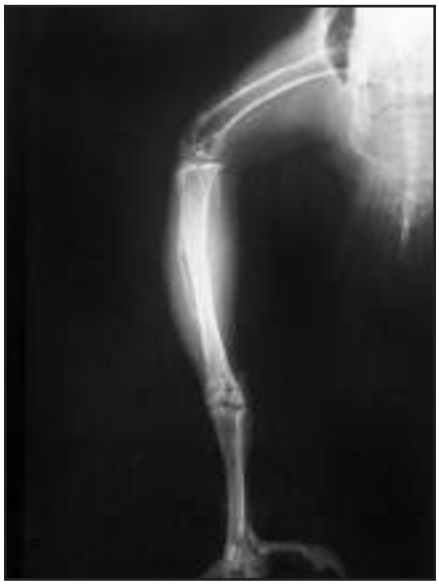


Fig 40.14| Fracture of the tibiotarsal bone in a saker falcon. This is the most common type of fracture encountered in clinical practice with captive raptors. Fractures of this bone tend to occur in newly tethered birds and during training exercises.

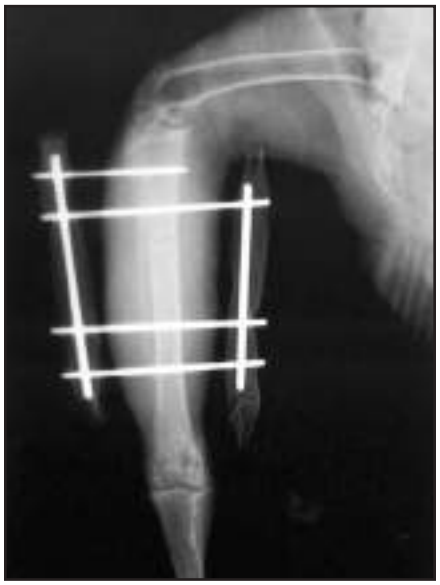


Fig 40.15 | The bird in Fig 40.14. The fracture was repaired using a shuttle pin and a type II external skeletal fixator. Two acrylic bars and additional metals pins within the tubing were used to maintain the fixator in position and provide extra strength.

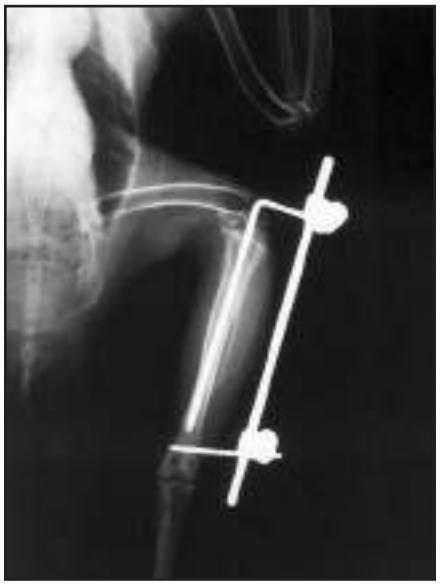


Fig 40.16 | A similar fracture of the tibiotarsus in a gyr-peregrine hybrid falcon. This fracture was repaired using an intramedullary pin inserted in a normograde fashion from the tibial crest, a positive profile threaded pin placed distally and tie-in using a bar and clamps.

Fig 40.17 | The falcon in Fig 40.16. Note the bandage covering the external skeletal fixator and the shoe placed on the opposite foot to prevent bumblefoot. In uncomplicated cases, full healing should be expected in 4 to 6 weeks.

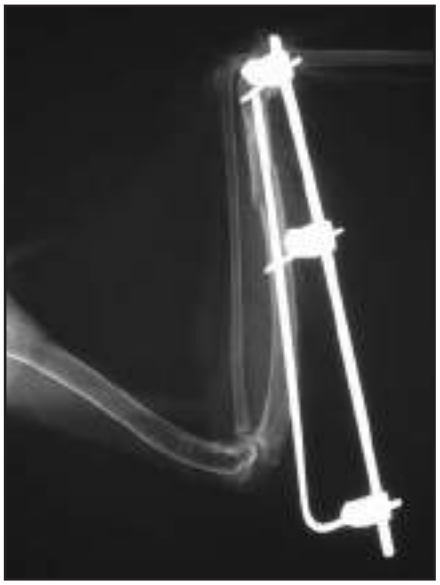


Fig 40.18 | Oblique fracture of the distal ulna repaired using an intramedullary pin inserted in normograde fashion from the proximal ulna, two positive profile threaded pins placed in the proximal and distal fragments, and tie-in using a bar and clamps.

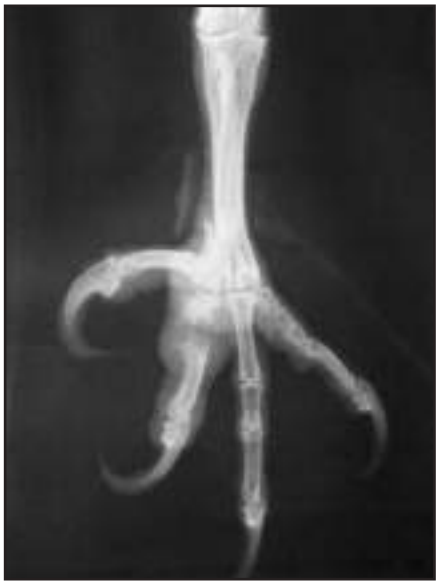


Fig 40.19 | Fracture of the first phalanx of digit 2 in a saker falcon. There was extensive soft tissue swelling and lysis of the bone fragments requiring amputation of the digit.

biopsies (eg, liver biopsy) and assisting in the performance of some intracoelomic surgical interventions (eg, vasectomy) (Fig 40.13, Table 40.10) (see Chapter 24, Diagnostic Value of Endoscopy and Biopsy).

ORTHOPEDIC SURGERY

Raptors are very often presented to rescue and rehabilitation centers with luxations and fractures[62] caused by inadequate management, trapping, injuries by larger migratory raptors and collision with moving vehicles or stationary objects (eg, fences, towers, utility poles) (Figs 40.14-40.17). The goal of return to flight and the associated need for retention of soft tissue and joint integrity make orthopedics in raptors destined for either

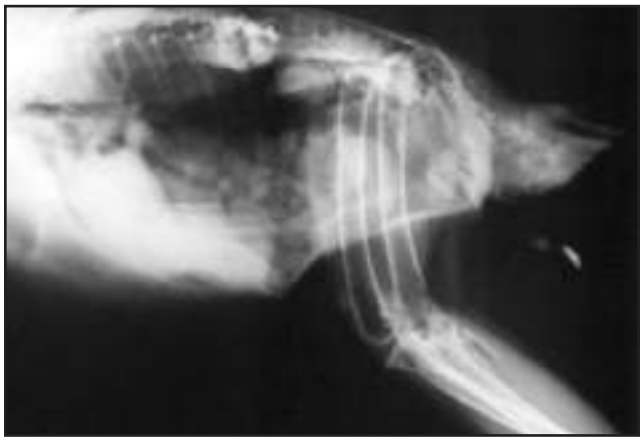


Fig 40.20 | Lateral radiograph of a falcon showing extensive osteoarthritic changes of the vertebral synsacral joint. The only clinical sign was the inability to maintain a straight position while standing. Damage tends to occur due to collision-type injuries.

falconry or release a different proposition than it is for pet psittacines. Table 40.11 summarizes orthopedic techniques used in raptor medicine (Table 40.11, Figs 40.15-40.20). For further information, the reader is referred to published information.[25,62,64,72] (See Chapter 34, Surgical Resolution of Orthopedic Disorders).

Pododermatitis

Pododermatitis or bumblefoot is a common medical condition of captive raptors and is characterized by inflammation and often abscessation of the sole of the foot or the plantar aspect of the digits. This condition appears to be caused by a combination of factors including poor nutrition, obesity, inadequate perches, lack of exercise, poor blood circulation to the foot and cardiovascular changes at the end of the hunting season[26,29] (Figs 40.21, 40.22). Some raptor species appear to be more susceptible to this condition than others. For instance, the incidence of bumblefoot appears to be higher in falcons but

Table 40.11 | Techniques for Fracture Repair in Raptors[62]

Anatomical Site	Technique
Coracoid	Conservative treatment (eg, immobilization of the wing).
Humerus Proximal	Tension band technique. Two Kirschner wires driven normograde cross-pin fashion, tension band formed by cerclage wire.
Mid-shaft	Intramedullary pin type I external skeletal fixator tie-in technique. Steinmann intramedullary pin normograde fashion driven from the distal end of the humerus. Placement of two positive profile threaded pins, one in the dorsal condyle, and the second placed close to the curvature of the pectoral crest. Tie-in with a fixator bar and clamps[i], thermoplastic tape[g] or acrylic bar[k].
Distal	Intramedullary pin type I external skeletal fixator tie-in technique. Two Kirschner wires driven normograde cross-pin fashion, placement of a positive profile threaded pin[i], on the curvature of the pectoral crest. Tie-in with a fixator bar and clamps[i], thermoplastic tape[g] or acrylic bar[k].
Radius and Ulna	"Figure eight" coaptation bandage. Intramedullary pin type I external skeletal fixator tie-in technique. Normograde placement of Steinmann intramedullary pin, normograde fashion driven from the proximal end of the ulna and Steinmann intramedullary pin normograde or retrograde fashion in the radius, placement of two or more positive profile threaded pins tie-in with a fixator bar and clamps, thermoplastic tape[g] or acrylic bar[k].
Major Metacarpal	Coaptation with splint[f] or thermoplastic tape[g] splints. Intramedullary pin type I external skeletal fixator tie-in technique. Steinmann intramedullary pin or Kirschner wire in normograde or retrograde fashion, placement of two or more positive profile threaded pins tie-in with thermoplastic tape[g] or acrylic bar[k].
Femur Proximal	Tension band technique. Two Kirschner wires driven normograde cross-pin fashion, tension band formed by cerclage wire.
Mid-shaft	Intramedullary pin type I external skeletal fixator tie-in technique. Steinmann intramedullary pin retrograde fashion, placement of two or more positive profile threaded pins tie-in with a fixator bar and clamps, thermoplastic tape[g] or acrylic bar[k].
Distal	Two Kirschner wires driven normograde cross-pin fashion, placement of a positive profile threaded pin on proximal femur. Tie-in with a fixator bar and clamps, thermoplastic tape[g] or acrylic bar[k].
Tibiotarsus	Intramedullary pin type I external skeleton fixator tie-in technique. Steinmann intramedullary pin normograde fashion driven from the tibial crest, placement of a positive profile threaded pin distally tie-in with a fixator bar and clamps, thermoplastic tape[g] or acrylic bar[k].
Tarsometatarsus	Type II external skeletal fixator. Placement of four pins, tie-in with fixator bar and clamps, thermoplastic tape[g] or acrylic bar[k].

Fig 40.21 | Plantar view of the foot of a saker falcon showing a large area of dry skin in the center of the sole. The adjacent papillae are flattened, having lost the form and texture due to the use of inadequate perches and lack of exercise.

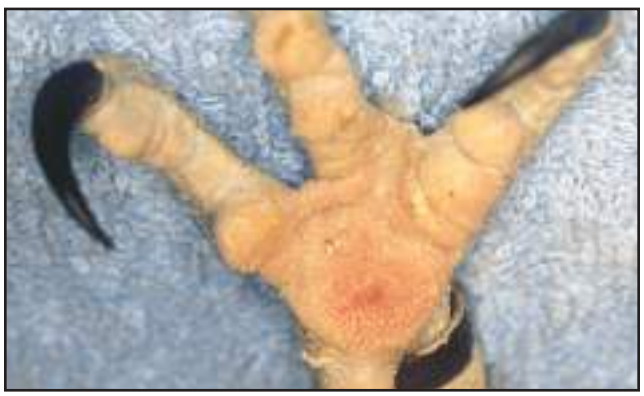


Fig 40.22 | The foot in Fig 40.21. The dry skin was removed by brushing gently, exposing underneath an early ulcerative lesion in the skin. This is, in the author's opinion, the most common presentation of bumblefoot in captive raptors.

Fig 40.23 | Severe bilateral bumblefoot in a saker falcon, with soft tissue swelling and underlying infection. Such cases are not amenable to treatment due to large skin deficits that remain after scab removal. The use of skin flaps has been attempted with little success.

seldom occurs in hawks.[26] In most cases, the preferred therapy involves the surgical removal of scabs and adjacent necrotic and purulent tissue, followed by suturing to achieve healing by first intention (Fig 40.23). The placement of antibiotic-impregnated polymethylmethacrylate beads within the newly created cavity improves the rate of healing.[69] If the skin deficit is relatively large and complete closure is not possible, partial closure might be attempted with a purse-string suture together with hydrocolloid dressings[c] to promote healing by second intention. The use of foot casting together with conforming bandages as part of the postoperative treatment is highly recommended (see previous section on foot casting) (Fig 40.24).

IMPING

Imping, a medieval falconry term, is the art of repairing fractured or bent feathers. The integrity of the primary and tail feathers is of the utmost importance for flight and performance in raptors destined for release back into the wild or for those used in the sport of falconry. Feathers tend to suffer severe bends or fractures during captivity in rescue and rehabilitation centers (poor aviary or holding cage design), training or hunting (crash landings or fighting with quarry) or because of inadequate handling and transport (no tail guard). Imping is usually carried out by total or partial feather replacement or by external splinting[45,78,89] (Fig 40.25). See Table 40.12 for a list of materials used for imping.

For a total and partial feather replacement, it is necessary to procure a feather that is of the same species, side (eg, wing feather), size, sex, age and color. Rescue and rehabilitation centers and medical facilities concerned with raptors usually maintain a collection of molted feathers and feathers obtained from carcasses. It is recommended to carry out feather examination and feather repair procedures under general inhalant anesthesia.

Fig 40.24 | A custom-made protective shoe made from a soft rubberized material. These are used to protect surgical sites in the sole of the foot and to prevent pressure sores on the opposite foot after orthopedic surgery.

Fig 40.25 | Materials and equipment required for feather repair and total or partial feather replacement.

Table 40.12 | Materials and Instruments for Imping

- Scissors, small, sharp, fine-pointed
- Nail cutter, medium and large (eg, cat and dog size)
- Imping needles, made from steel hairpins, long 50 mm x 1.5 mm, medium 40 mm x 1.5 mm, short 30 mm x 1.5 mm, fine 25 mm x 1 mm
- Hair clips (aluminum) 100 mm long
- Nail file, fine
- Methacrylate glue
- Epoxy glue, fast setting
- Baking soda, finely powdered
- Pliers, curved, fine-tipped
- Bamboo pegs, different diameters (barbecue skewers are ideal)
- Knife, interchangeable blades
- Cardboard cards, thin, 5 cm x 5 cm square

Total Feather Replacement

Total feather replacement is indicated when the feather is fractured at the proximal section of the feather shaft. After examination and determining the number of feathers for

Fig 40.26 | This falcon sustained a fracture of a tail feather with complete loss of the fragment. The fracture occurred in the first third of the shaft, requiring full feather replacement.

Fig 40.27 | A similar feather was procured and cut to the same size as compared with the feather on the opposite side of the tail. A pre-made short bamboo peg was placed between the two fragments and glued into position using rapid-setting epoxy glue.

Fig 40.28 | Tail of the falcon in Figs 40.26 and 40.27, showing the new feather in place. In addition, the two deck or central feathers were repaired using the partial replacement technique.

Fig 40.29 | This saker falcon has suffered fractures of primaries 1st, 2nd and 3rd (8th, 9th and 10th according to the Western ornithology) with loss of feather fragments. Partial feather replacement is indicated in this particular case.

repair, the area should be prepared. First, the covert feathers are deflected backward and held in place with masking tape to expose the base of the shaft. The fractured feather is cut approximately 15 to 25 mm from the skin with a nail cutter (Fig 40.26). The new feather is placed in position to assess the length, making sure to maintain bilateral symmetry with the opposite wing when replacing a wing feather, or the opposite side if replacing a tail feather. If the feather from the opposite side is missing, the veterinarian or technician should follow the feathering pattern of the wing or tail characteristic of the species (eg, in a peregrine falcon, primary 10 [No 1 in Arab falconry] is approximately 5 to 8 mm shorter than primary 9 [No 2 in Arab falconry]). This general ornithological knowledge is essential to carrying out feather replacement adequately. The feather is cut, and a bamboo peg about 80 to 100 mm long is prepared by sharpening both ends to approximate the diameter of the shaft of the new feather and the empty shaft of the wing. The wooden peg is first glued into the shaft of the new feather with fast-setting epoxy. Additional glue is then placed (eg, injected with a 1-ml tuberculin syringe) into the shaft, making sure the feather is properly aligned. A small piece of cardboard should be placed under the imping site to prevent the glue from smearing onto adjacent feathers (Fig 40.27). The wing or the tail should then be closed in the natural anatomical position and all the feathers held in place with hair clips until the glue is set (Fig 40.28).

Partial Feather Replacement

Partial feather replacement is indicated if the fracture has occurred at the mid-shaft or at the distal end of the feather. If a fracture is complete and the feather fragment

Fig 40.30 | Feather fragments from the same species, size, side (eg, left wing), sex, age and color have to be procured. Imping needles of a suitable size are used to join the fragments. The needle is first fixed into the distal fragment using methacrylate glue. The fragment is then pre-placed into the fractured fragment to check alignment before gluing into position.

Fig 40.31 | The falcon in Fig 40.30 with the three feathers repaired. It is highly recommended to place an external splint as that described in Figs 40.32 and 40.33 to reinforce the ventral aspect of the imping surface. The dorsal aspect can be filed to produce a smooth surface and then colored using a brown marker.

Fig 40.32 | A severe bend in the mid shaft of a main flight feather. Note that the integrity of the feather shaft has been maintained.

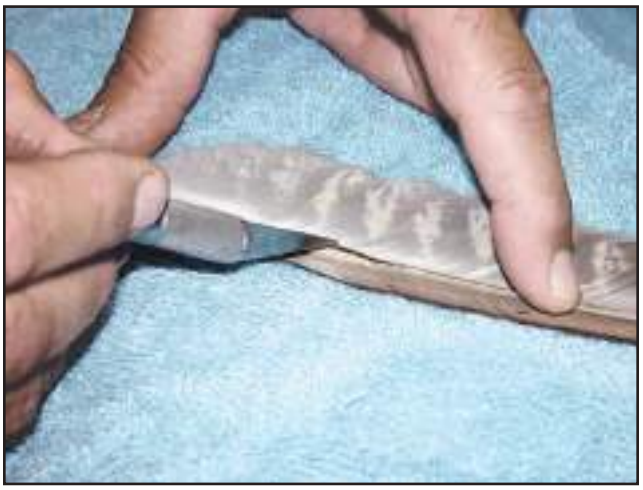


Fig 40.33 | The preliminary stage of the technique used by the author for feather repair involves splitting the ventral aspect of the shaft, 12 to 15 mm in either direction from the bend, using a sharp knife.

is missing, a similar fragment must be procured from a donor feather (Fig 40.29). Conversely, the fragment might then be reattached. In both cases, the ends of the fragments are smoothed out with a fine-pointed scissor and a fine nail file. A previously prepared imping needle of suitable length and diameter is carefully inserted in both fragments to make a narrow channel. The needle is then fixed onto the fragment with a small amount of methacrylate glue. The fragment is attached onto the rest of the feather and checked for correct alignment. Additional glue is then applied onto the free end of the needle of the fragment, which is then attached to the rest of the feather (Fig 40.30). Pressure should be applied over the imping site with fine-tipped, curved hemostats or pliers for approximately 30 seconds to allow the glue to set. The dorsal and ventral aspects of the fracture line are then filed with a fine nail file. In partial replacement, it is strongly recommended to apply a ventral external splint in addition to the method described above to produce a more satisfactory and efficient result. The dorsal aspect of the imping site can be colored, if necessary, with a marker pen (Fig 40.31).

Bent Feather Repair

Moderate or severe bending might occur at different levels of the shaft. Bends are repaired using the external splinting technique. The bend is straightened on its dorsoventral axis with a pair of fine-tipped, curved hemostats or pliers (Fig 40.32). The ventral aspect of the feather shaft is then split 12 to 15 mm in either direction from the bend (Fig 40.33). A suitable imping needle is placed in the newly created groove and secured firmly

Fig 40.34 | The second stage of this technique involves the placement of an imping needle within the newly created groove. Methacrylate glue is then used to secure the needle into position.

Fig 40.36 | After completing the splint, fine-grain nail files are used to smooth the newly created surface. The final product blends very well with the shaft of the feather, resulting in a satisfactory cosmetic repair.

with methacrylate glue (Fig 40.34). The ventral surface of the feather shaft around the bend is roughened with a fine nail file. A thin layer of methacrylate glue is smeared onto the site approximately 10 mm to either side of the bend. A small amount of baking soda is sprinkled directly onto the freshly glued surface (Fig 40.35). The sodium bicarbonate binds with the glue creating a strong cement-like layer over the bend. The procedure can be repeated two or three times to create a thicker layer if this proves necessary. The upper surface and the edges of the newly created layer are filed with a fine nail file (Fig 40.36). The external splint is translucent, making the need for coloring unnecessary.

COPING

Coping is a medieval term meaning the trimming and reshaping of talons and beak. Overgrown talons and

Fig 40.35 | The final stage of feather repair involves the creation of an external splint using a combination of methacrylate glue and sodium bicarbonate to produce a cement-like layer over the original bend.

Table 40.13 | Materials and Instrumentation for Coping

- Nail cutter, guillotine-type
- Nail cutter, large, human-type
- Nail file, fine
- Methacrylate glue
- Baking soda, finely powdered
- Knife, interchangeable blades
- Silver nitrate pencil

beaks are commonly found in captive raptors due to the insufficient wear associated with the use of soft, inadequate perching surfaces and lack of a natural diet. More severe cases indicate malnutrition. See Table 40.13 for a list of materials used for coping.

Talons

Knowledge of the size and morphology of the talons of different raptor species is essential. Overgrown talons are best trimmed with a guillotine-type nail cutter. The talon is then reshaped with a sharp blade and nail files. Excessive trimming would invariably lead to hemorrhage. The use of a thermocautery or a silver nitrate pencil is usually sufficient to arrest any hemorrhage. The use of a lanolin or paraffin-based hand cream is recommended after trimming and reshaping of the talons. Raptors used in the sport of falconry should have the talons trimmed before placing them into molting chambers (Fig 40.37).

Beak

In raptors, the shape of the beak is species-specific. For instance, *Buteo* spp. and *Accipiter* spp. possess a hooked beak design for tearing at prey, while the beak of *Falco* spp. has a tomial tooth used presumably to sever the head of prey. Knowledge of the normal

Fig 40.37 | Overgrown and deformed talons are the result of the use of inadequate perching surfaces. The talons of the hallux are more susceptible to deformities if birds are confined to small round perches such as the Arab-style stand.

Fig 40.38 | Saker falcon with a long deformed beak after the molting season. Coping, an old medieval term, is the art of trimming and reshaping of the beak and talons of raptors. Coping of the beak is usually carried out using nail cutters and fine-grain nail files.

Fig 40.39 | Severe bilateral splitting of the upper beak with lateral deviation of the lower beak in a peregrine falcon due to neglect. The coping of such a condition requires several sessions over several months to fully correct the different defects.

Fig 40.40 | Deep unilateral groove in the beak of a saker falcon. This condition commonly originates from scratch injuries sustained at the anterior aspect of the nares, damaging the germinative tissue of the beak and leading to a growth disorder.

morphology of beaks in raptors is essential before undertaking any reshaping.

An overgrown beak is simply trimmed back with human-type nail cutters, and the tip reshaped with nail files or a rotary grinding tool (Fig 40.38). The lateral aspect of the beak of captive raptors is prone to cracks and fissures, which can be prevented by providing hard surfaces (stones) where the bird can both clean its beak and file it regularly to maintain its shape. Cracks and fissures should be trimmed as soon as possible to prevent more serious conditions such as fractures (Fig 40.39). Lateral grooves on one or both sides of the beak are found commonly in raptors, but particularly in falcons (Fig 40.40). It has been suggested that these grooves in falcon beaks are due simply to old age. An alternate explanation, adhered to by this author, involves the grooming practices of falcons. During routine grooming, falcons tend to scratch the head with their talons. Puncture wounds to the eyes or scratches to the eyelids and the nares are often observed. During scratching, a falcon can easily hook a talon on the anterior aspect of the nares causing a laceration. This wound is created on the edge of the germinative tissue of the beak, creating a groove as the beak grows. Very little can be done to correct this condition. Temporary filling of the defect with methacrylate glue and baking soda or commercially available beak repair kits has been successful. However, as the beak grows, continuous repairs will be required every 8 to 12 weeks.

HOSPITAL CARE

In order to be treated for selected medical conditions, raptors must be hospitalized (Fig 40.41). Free-living raptors usually tolerate small cages, provided these are fitted with curtains and maintained in quiet, semi-dark rooms. Falconry birds can be kept unhooded on wall or floor perches. Hoods are commonly used only during

Fig 40.41 | Large raptors can be kept in small, secluded aviaries during hospitalization. This griffon vulture (*Gyps fulvus*) was admitted for the treatment of a compound fracture involving the humerus.

routine cleaning and for handling purposes. Hospitalized raptors should be fitted with a tail guard as described in the earlier section on capture and restraint.

Handling of raptors for medication administration and cleaning should be kept to a minimum; non-intrusive cages with a tray fitted below a slatted floor are ideal. Furthermore, a significant amount of medications can be administered hidden in food (eg, tablets or powdered medication inside the heart of a quail). There are cases, however, in which medication has to be administered by injection, nebulization or topically, and handling is therefore necessary. Hooded raptors are easier to maintain under hospital conditions. Changeable substrate (eg, carpeting, sand, newspaper) under floor or wall perches allows easy maintenance of such birds. The administration of medicaments to such birds also is facilitated, since hooded raptors can, for instance, be given intramuscular injections in the chest musculature by a single operator without restraint.

DIET AND FEEDING STRATEGY

Hospitalized raptors can be fed a variety of food items such as feathered and unfeathered quail, mice, rats, ducklings, rabbits, day-old chicks and fish for species such as ospreys (*Pandion haliaetus*) and bald eagles (*Haliaeetus leucocephalus*). Thiamine supplementation at a rate of 2 mg/kg PO weekly is indicated when using frozen-thawed fish. The daily provision of casting material in the form of fur and feathers is of the utmost importance. Casting material should be offered only to raptors in which the gastrointestinal tract is working normally. However, raptors very often refuse to eat, and it might prove necessary to force-feed them in order to meet their daily nutritional requirements. For a list of materials used to force-feed raptors, see Table 40.14. For a complete description of dietary management of captive raptors, the reader is referred to comprehensive previously published accounts.[3,61]

Table 40.14 | Force-feeding Materials

- Stomach tube, blunt end, plastic (5 mm, 8 mm or 12 mm in diameter)
- Feeding syringe, 60 ml
- Lubricant[l]
- Formula container

Table 40.15 | Force-feeding Formula[e]

Formulae	Ingredients	Instructions
Formula 1 - Induction	Add 15 ml water to 20 g ground whole unfeathered quail +10 g ground beef or chicken liver + ¼ teaspoon glucose-electrolyte preparation in powder form[m].	Administer 20 to 30 ml of mix per kg body weight, 3 or 4 times daily during first day of admission.
Formula 2 - Intensive	Add 20 g of ground whole unfeathered quail +10 g ground beef or chicken liver +1 whole egg + ¼ teaspoon glucose-electrolyte preparation in powder form[m].	Administer 30 to 40 ml of mix per kg body weight, 3 or 4 times daily; weigh daily, until the desired body weight is reached.
Formula 3 - Maintenance	Add 5 ml water to 20 g of unfeathered ground quail + 10 g ground beef liver + ¼ teaspoon glucose-electrolyte preparation in powder form[m].	Administer 30 to 40 ml of mix per kg body weight, 3 or 4 times daily; weigh daily.

Force-feeding is the introduction of nutritional formula into the alimentary tract by means of a stomach tube, and injected using a large syringe. There are several commercially available diets that can be used. However, the author favors the force-feeding diets and feeding strategy for raptors shown in Table 40.15. In fish-eating species, fish can be incorporated into the diet by substituting it for the quail, beef or chicken.

To force-feed a raptor, the thoroughly lubricated feeding tube is passed gently into the back of the oropharynx, through the crop and then into the esophagus toward the right side of the neck. Great caution should be exercised while manipulating the tube within the crop to avoid folding the tube or causing a penetrating wound through the crop wall. During force-feeding, the neck of the bird should remain extended to discourage regurgitation. Feeding tubes and syringes must be thoroughly washed, disinfected and rinsed (1 ml quaternary ammonium and biguanadine compounds disinfectant[n] in 500 ml water for 2 to 5 minutes) prior to using them on another patient.

BIOSECURITY

Biosecurity is defined as a series of measures undertaken within a building or building complex to prevent the propagation and spread of diseases. Housing a large

Fig 40.42 | Disinfection is the backbone of any biosecurity program. The technician is using a fogging unit loaded with a solution of commercially available disinfectant[n] within a falcon room. Note that the procedure is being carried out in the presence of the birds.

number of raptors (eg, captive breeding program) within the same facility represents a potential risk if basic biosecurity rules are not followed. The risk becomes more significant when there is a constant flow of raptors from different origins such as hospitals or rescue and rehabilitation centers. For instance, an epornitic outbreak of pox within a facility housing 15 different species of raptors was reported in the USA. The author has observed similar outbreaks of pox and Newcastle disease in falcon molting and breeding facilities in the Middle East. In some cases, outbreaks of Newcastle disease were traced to the consumption of infected quail and pigeons. In other instances, pox and Newcastle disease were traced to affected falcons placed in close contact with healthy birds without having gone through a quarantine period.

One of the main pillars of any biosecurity program is disinfection. Disinfection is defined as a procedure intended to eliminate from a particular defined area any pathogenic organism or to render them inert with one or a combination of chemicals. There are many products available on the market that could be used within a biosecurity program. Such products should be non-toxic, non-irritating, non-corrosive and, ideally, biodegradable. Once a suitable product has been obtained, the next step is to design and implement a disinfection program for the facility (Fig 40.42).

The disinfection program followed at the Fahad bin Sultan Falcon Center facilities in Riyadh, Kingdom of Saudi Arabia (Table 40.16) is based on the use of a recently introduced quaternary ammonium and biguanadine compounds disinfectant[n].

Table 40.16 | Disinfection Protocol (using F10 disinfectant)[n]

Area	Applications	Frequency	Dilution
Reception			
Walls	Spray and wipe clean	Weekly	1:500
Counter	Spray and wipe clean	Twice a day	1:250
Floor perch	Spray and brush	Twice a day	1:250
Waste bins	Spray inside of new bin liner	Daily	1:250
Floors	Sweep clear, apply with mop	Daily, twice a day	1:500
Air space	Fog	Weekly	1:125
Air conditioning filters	Spray and leave to dry	Weekly	1:250
Examination rooms/Operating theatre			
Walls and cabinets	Spray and wipe clean	Twice a week	1:250
Door handles	Spray and wipe clean	Twice a day	1:250
Worktables and sinks	Spray and wipe clean	Twice a day	1:250
Examination table	Spray and wipe clean	After each use	1:250
Towels	Soak overnight, wash	Daily	1:250
Equipment	Handles and switches, spray and wipe clean	Daily	1:250
Trolleys	Spray and wipe clean	Daily	1:250
Telephones, light switches	Spray and wipe clean	Daily	1:250
Waste bins	Spray inside of new bin liner	Daily, twice a day	1:250
Floors	Sweep clear, apply with mop	Daily, twice a day	1:500
Floor drains, sink wastes	Pour solution down drain	Weekly	1:500
Air space	Fog	Weekly	1:125
Air conditioner filters	Spray and leave to dry	Weekly	1:250
Post-mortem room			
Carcass fridge	Wash out with water, spray and leave to dry	After defrosting	1:250
Safety cabinet, table	Wash out with water, spray and leave to dry	After each use	1:250
Hospital/Quarantine/Molting wards			
Rooms	Fog	Weekly	1:125
Carpeting, artificial turf tops	Spray, brush down surfaces and perches	Daily, twice a day	1:250
Water bowls	Wash, soak	Daily	1:250
Walls and cabinets	Spray and wipe clean	Weekly	1:250
Door handles	Spray and wipe clean	Twice a day	1:250
Worktables and sinks	Spray and wipe clean	Weekly	1:250
Examination table	Spray and wipe clean	After each use	1:250
Equipment	Handles and switches, spray and wipe clean	Daily	1:250
Trolleys	Spray and wipe clean	Daily	1:250
Telephones, light switches	Spray and wipe clean	Daily	1:250
Waste bins	Spray inside of new bin liner	Daily, twice a day	1:250
Floors	Sweep clear, apply with mop	Daily, twice a day	1:250
Floor drains, sink wastes	Pour solution down drain and wastes	Weekly	1:500
Air space	Fog	Weekly	1:125
Footwear decontamination	Spray or use footbath	Before and after using	1:250
Air conditioner filters	Spray and leave to dry	Weekly	1:250

Infectious Diseases

Infectious diseases are those disorders produced by the invasion and propagation of microorganisms in body tissues. Infectious diseases may produce no clinical signs, or they may cause a myriad of reactions, including localized cellular injury due to competitive metabolism, toxins, intracellular replication or antigen-antibody response. Infectious diseases can be divided into four main groups: Parasitic, bacterial, viral and fungal diseases.

PARASITIC DISEASES

Parasites (*Greek*: *Parasitos*) are organisms that live upon or within other living organisms, at the hosts' expense. Parasites are often found associated with free-living and captive raptors. The relationship between the parasite and its host does not necessarily translate into overt disease. However, both intrinsic (eg, severe weight loss, immunosuppression, toxicosis, collateral infectious disease) and extrinsic (eg, heat stress) factors can exacerbate the effect of parasites, resulting in adverse clinical signs.

In general, parasites can be classified as ectoparasites (*Greek: ecto = outside*) and endoparasites (*Greek: endon = inside*). However, in the context of this chapter, the main parasites of raptors will be described under the grouping macroparasites (arthropods, helminths) and microparasites (protozoa, hematozoa).

Macroparasites

Arthropods

Arthropods or ectoparasites are invertebrates commonly found associated with the integument of free-living and captive raptors.[29,42,92] All ectoparasites of raptors are classified under the phylum Arthropoda, classes Insecta and Arachnida, and include mainly ticks and mites (Acarina), fleas (Siphonaptera), larvae of flies and louse flies or hippoboscids (Diptera) and chewing lice (Phthiraptera, formerly Mallophaga).[42] Some species of ectoparasites (eg, lice, mites) live permanently on the host while others (eg, ticks, flies) only temporarily (Table 40.17).

For more information on geographical distribution, pathogenesis and suggested control of ectoparasites in raptors, the reader is referred to excellent reviews recently published.[42,92,109]

The treatment used by the author for the control of ectoparasites in raptors is an insecticide containing 1.25 g/L permethrin, 6.25 g/L piperonyl butoxide and 20 mg/L methoprene[o]. This is sprayed lightly under the wings, over the neck and back, and under the tail. Meticulous cleaning and spraying of the rooms, cages or aviary might be required.

Table 40.17 | Common Ectoparasites Found in Raptors

Hard ticks	*Ixodes ricinus, I. arboricola, Hyalomma marginatum, H. rufipes, Rhipicephalus turanicum, Amblyomma lepidum*
Soft ticks	*Argas persicus, A. reflexus, Ornithodoros* spp.
Mites	*Dermanyssus gallinae, Ornithonyssus sylviarum, Knemidokoptes mutans, Kurodaia haliaeeti, Bonnetella fusca, Boydaia falconis*
Fleas	*Echidnophaga gallinacea, Ceratophyllus gallinae*
Larvae of flies	*Lucilia* spp., *Calliphora* spp., *Prosimulium* spp., *Carnus hemapterus, Passeromyia heterochaeta*
Louse flies	*Ornithomya avicularia, Pseudolynchia canariensis, Icosta Americana, Ornithomya anchineuria*
Lice	*Craspedorrhynchus* spp., *Aegypoecus* spp., *Laemobothrion tinnunculi, Degeeriella rufa, D. discocephala stelleri, Falcolipeurus* spp. *Kelerinirmus rufus camtschaticus, Caracaricola chimangophilus, Pterophilus sudanensis, Colpocephalum zerafae*

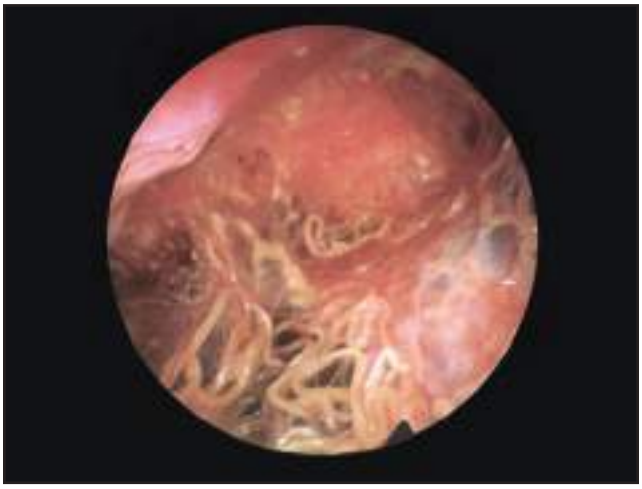


Fig 40.43 | Large number of *Serratospiculum seurati* filarial worms within the coelomic cavity of a saker falcon. Falcons often carry a heavy parasitic burden without displaying any clinical signs. However, the falcon depicted in this photograph had severe air sacculitis and associated dyspnea.

Helminths

Helminths (*Greek: Helmins = worm*) are a group of parasitic worms with various structural and behavioral characteristics that inhabit different organ systems of raptors. These can be classified under the following groups: trematodes (flukes), cestodes (tapeworms), nematodes (roundworms) and acanthocephalans (spiny-headed worms).[44]

It is generally believed that large numbers of certain parasites can coexist within the host without posing any serious health threat. For instance, exceedingly large numbers of *Serratospiculum seurati* filarial worms have been observed within the coelomic cavities of recently caught free-living saker falcons upon routine endoscopy in the Middle East[85] (Fig 40.43). The endoscopic examinations were carried out as an integral part of health assessments. In most cases, finding such heavy parasitic burdens was incidental and did not correlate with any obvious clinical signs. However, in severe infections, *S. seurati* has been found associated with pneumonia, air sacculitis and early lesions of aspergillosis.[85]

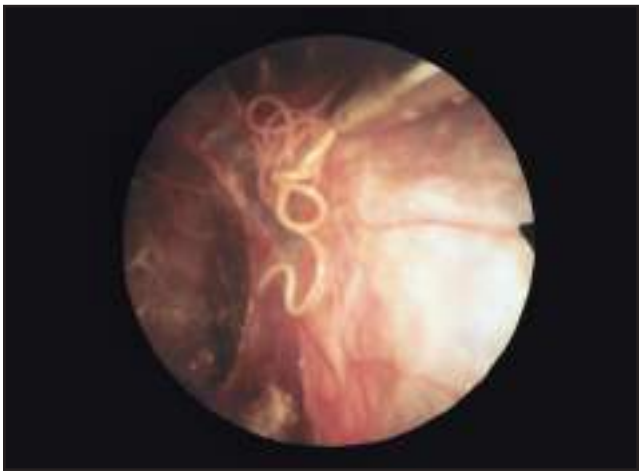


Fig 40.44 | It is a common and widespread practice to manually remove *Serratospiculum seurati* filarial worms from the coelomic cavity following ivermectin treatment. This is unnecessary in most cases as the worms are commonly absorbed. The current treatment of choice for serratospiculiasis is 1 mg/kg PO q7d x 2 weeks of moxidectin.

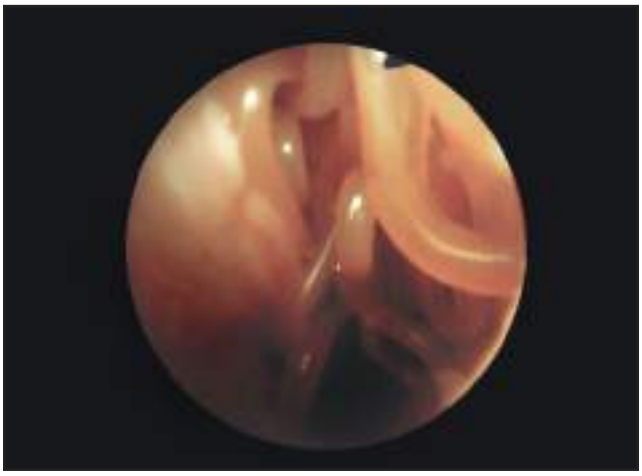


Fig 40.45 | Adult *Physaloptera alata* worms attached to the esophagus of a lanner falcon (*Falco biarmicus*). This was an incidental finding during a routine endoscopy examination of the upper digestive tract. The ova of this species are very similar to those of *Serratospiculum seurati*, but differ slightly in size and shape.

It is a good practice to screen captive birds for parasites a minimum of two times a year. This should be part of a much wider preventive medicine program. When possible in captive breeding projects, up to three fecal samples should be collected at intervals of 24 to 48 hours to provide a better qualitative and quantitative assessment of the presence of parasites. It is imperative to educate raptor owners and keepers to the fact that the treatment of parasites involves more than administering pharmacological compounds. Other important aspects of parasitic control include strict hygiene measures and modifications to aviaries and enclosures to prevent access to intermediate hosts (eg, placement of pebbles as substrate in open aviaries to prevent raptor's access to earthworms).

The following table (Table 40.18) provides a list of the most common endoparasites of raptors. For further information on the taxonomy and distribution of endoparasites in raptors, the reader is referred to a comprehensive and excellent review recently published.[44]

Table 40.18 | Common Endoparasites Found in Raptors

Trematodes	*Clinistomum complanatum, Nematostrigea serpens, Neodiplostomum attenuatum, Strigea falconis, S. falconispalumbi*
Cestodes	*Anomotaenia mollis, Cladotaenia globifera, C. armigera, C. cylindracea, Hymenolepis exilis, Idiogenes flagellum, Matabelea fuhrmani, Mesocestoides perlatus*
Nematodes	*Baruscapillaria falconis, Capillaria tenuissima, C. falconis, C. contorta, C. strigis, Cyathostoma americana, C. brodskii, C. lari, Diplotrianea falconis, Eucoleus dispar, Porrocaecum angusticolle, P. depressum, Serratospiculum seurati, S. tendo, Serratospiculoides amaculata, Syngamus trachea, Synhimantus laticeps, S. hamata, Physaloptera alata, Procyrnea leptoptera, P. mansioni, Tetrameres accipiter*
Acanthocephalan	*Centrorhynchus aluconis, C. buteonis, C. globocaudatus, C. kuntzi, C. olssoni, C. robustus, C. spinosus, C. tenuicaudatus, Mediorhynchus armenicus, M. papillosus*

Treatment

Praziquantel is widely used for the treatment of trematodes and cestodes at the dose rate of 50 mg/kg PO or SC as a single dose.[97] Alternatively, niclosamide at the dose rate of 125 mg/kg PO also as a single dose has been used for the treatment of cestodes. The author, however, currently is successfully using a compound mixture[p], providing a total of 10 mg/kg PO praziquantel, for the control of trematodes and cestodes in falcons, administered in two doses 1 week apart. In addition, this compound provides 10 mg/kg oxfendazole. The pharmacological action of oxfendazole on nematodes in raptors has not been ascertained. Fenbendazole, at the dose rate of 25 mg/kg PO for 3 to 5 consecutive days, is used very commonly for the treatment of nematodes in raptors.[29,47,96] Levamisole has been recommended for the treatment of nematodes in raptors at the dose rate of 10 to 20 mg/kg PO for 2 consecutive days.[47] Alternatively, fenbendazole at the dose rate of 25 mg/kg PO for 5 consecutive days has been suggested.[29] Ivermectin also has been widely used for the treatment of nematode infections, in particular those involving *Serratospiculum seurati*[46,85] in the Middle East. Ivermectin at the dose rate of 200 μg/kg IM or SC has been used to stunt the parasites and allow subsequent surgical removal of adult worms.[46,85] However, doses of up to 3 mg/kg IM have been suggested for the control of *S. seurati* infections in hybrid gyrfalcons.[46] The author, in common with other clinicians, has found that ivermectin at the dose rate of 2 and 3 mg/kg IM or SC can lead to temporary blindness lasting up to 48 hours in saker, peregrine, Barbary (*Falco pelegrinoides*) and lanner falcons.[85] The author favors the use of moxidectin[p] at the dose rate of 1 mg/kg

PO q7d x 2 weeks, for the treatment of *Serratospiculum seurati*, *Capillaria falconis*, *Physaloptera alata* and acanthocephalan infections in falcons (Figs 40.44-40.45).

Microparasites

Protozoa

Trichomonas gallinae is perhaps the single most important protozoan affecting raptors worldwide. The disease, known in falconry terminology as frounce, is typically characterized by the appearance of caseous lesions in the upper digestive system, including the tongue, oropharynx, crop and esophagus[93] (Fig 40.46). Trichomoniasis also can affect the upper respiratory tract of raptors, affecting the nasal cavities, the infra-orbital sinuses, the trachea and the tracheobronchial syrinx.[79,93] Carnidazole[q], at the dose rate of 20 mg/kg PO[10] to 120 mg/kg PO[100] as a single dose, has been used in the treatment of clinical trichomoniasis in raptors. Conversely, metronidazole can be used at the dose rate of 50 mg/kg PO SID for 5 consecutive days.[93] Currently, the author uses metronidazole[r] at a higher dose, 100 mg/kg PO for 3 consecutive days, in the treatment of trichomoniasis in falcons, as this appears to be more effective (Figs 40.47-40.52).

Several different species of coccidia of the genera *Caryospora*, *Eimeria*, *Sarcocystis* and *Frenkelia* (Table 40.19) are relevant in raptor medicine.[42,44] Species of the genus *Caryospora* appear to be the more pathogenic to raptors, particularly to young birds under captive conditions. Weight loss, reduced appetite, regurgitation and vomiting, blood in feces, diarrhea and acute death characterize clinical coccidiosis.[19] Similar clinical signs, together with poor performance during training exercise, have been frequently observed by the author in captive falcons in the Middle East, particularly in juvenile peregrine, Barbary and lanner falcons. The treatment of choice for coccidiosis is toltrazuril[s] 25 mg/kg PO.[40] This product is known to have a bitter taste and tends to induce regurgitation in a significant number of raptors immediately after administration. The author administers toltrazuril with a crop cannula to falcons at this dose rate, mixed 1:1 with a cola-based soft drink to mask its bitter taste, for 2 consecutive days. The incidence of regurgitation is significantly reduced with this method.

Table 40.19 | Common Microparasites of Raptors

Coccidia	*Caryospora kutzeri, C. boeri, C. henryae, C. falconis, C. megafalconis, C. neofalconis, C. uptoni, C. buboni, Frenkelia microtis, F. glareoli Sarcocystis cernae, Eimeria accipitris, E. asturi*
Hematozoa	*Haemoproteus tinnunculus, H. brachiatus, H. elani, H. nisi, H. janovyi, H. syrnii, Plasmodium elongatum, P. fallax, P. circumflexum, P. lophurae, P. relictum, P. vaughani, Leucocytozoon toddi, L. ziemanni, Trypanosoma bramae, T. noctuae, T. santodiasi, T. syrnii, T. avium, T. fiadeiroi, T. guyanense, T. langeroni, T. everetti, T. fiadeiroi, T.corvi*

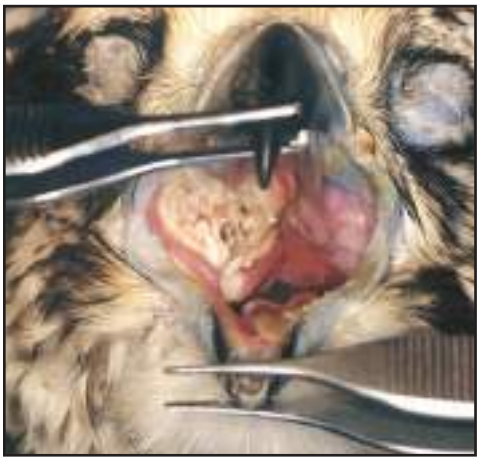


Fig 40.46 | A large caseous lesion produced by infection with the protozoan *Trichomonas gallinae* in the lateral oropharynx of a saker falcon. Infection usually occurs when captive raptors are fed pigeons or doves. Raptors living in urban or suburban areas are prone to contract trichomoniasis because they have access to feral populations of pigeons.

Hematozoa

Hematozoa (*Greek: haima = blood, zoa = animals*) are protozoan parasites living in the blood. The most relevant species in raptor medicine (Table 40.19) are classified under the genus *Haemoproteus*, *Leucocytozoon*, *Plasmodium* and *Trypanosoma*.[42,44,59] There appears to be a relatively high incidence of some hematozoa in free-living birds. In a recent survey, up to 11% of 976 Falconiformes and up to 13% of 173 Strigiformes were found positive for hematozoa in Germany.[43] The impact of these hemoparasites on their host is not well understood. In general terms, it is believed that the pathogenicity of most hemoparasites for raptors is relatively low. However, hematozoa infections have been directly implicated in severe clinical disease and even the deaths of individuals. It has been shown that infections with *P. relictum* can cause severe clinical disease in gyrfalcons, while several deaths in nestling owls have been attributed to *Parahaemoproteus*[101] spp. and *Leucocytozoon* spp. infections.[32] In addition, the deaths of a sub-adult saker falcon[91] and a common kestrel (*Falco tinnunculus*)[56] were attributed to severe hypochromic anemia produced by heavy parasitic infections of *Babesia shortti*.

Suggested treatment for hematozoa includes the use of chloroquine[t] at the initial dose rate of 25 mg/kg PO, followed by 15 mg/kg at 12, 24, 48 hours together with primaquine[t] at the dose rate of 0.75 mg/kg PO.[29,31]

BACTERIAL DISEASES

There are numerous bacterial diseases that have been described in different species of raptors worldwide. A brief description is therefore included here of the most

Fig 40.47 | Unilateral trichomoniasis infection in the nasal cavity of a saker falcon. The caseous mass is bulging through the palate. This type of infection very often leads to moderate to severe obstruction of the nasal cavity and difficulty eating. Note the food debris accumulated at the cranial aspect of the palate. In extreme cases, this condition could lead to a fistula formation between the oral and nasal cavities.

Fig 40.48 | Severe unilateral trichomoniasis infection affecting the supraorbital region of a saker falcon. The infection usually enters through the supraorbital diverticulum of the infraorbital sinus. Note the large, multiple caseous masses bulging through the skin.

Fig 40.49 | The same falcon as in Fig 40.48 after treatment and 2 weeks after surgery. The falcon was administered metronidazole for 3 consecutive days and an antibiotic for 7 days, after which time the caseous masses were surgically removed.

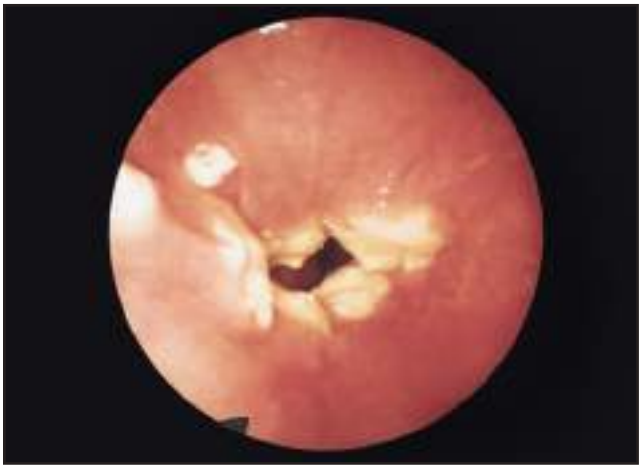


Fig 40.50 | Multiple nodular trichomoniasis lesions at the cranial aspect of the thoracic esophagus as seen from the crop. The masses had created a virtual ring, producing stenosis. The growths were not palpable from the crop, therefore illustrating the need to examine the upper digestive tract by endoscopy in selected cases based on the clinical history.

Fig 40.51 | An extreme case of trichomoniasis infection in a saker falcon. The appearance in this falcon is misleading, as it gives the impression that it had just eaten and has a full crop.

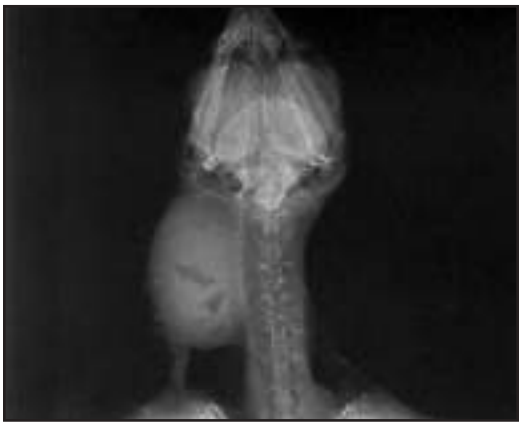


Fig 40.52 | A dorsoventral radiograph of the same falcon as in Fig 40.51, clearly showing a single large caseous mass within the crop. In most cases, masses such as this can be retrieved through the oropharynx after a course of antiprotozoal therapy.

frequently reported conditions. For a comprehensive review of bacterial diseases in raptors, the reader is referred to a recent publication on this subject.[9]

Tuberculosis is a bacterial disease produced by *Mycobacterium avium avium*. This condition is considered rare in North American raptors[65] but is frequently found in the United Kingdom and continental Europe.[9,29] The disease is characterized by the presence of tubercles primarily in the liver, spleen, gastrointestinal tract and bones. Subcutaneous tubercles also have been described in raptors.[9,29] Diagnosis with the tuberculin test is unrewarding.[29] The use of radiology in bone-related infections has proved useful.[65] For a more accurate diagnosis, suspected birds should be subjected to endoscopic examination and subsequent liver biopsy and histopathology analysis. Treatment of tuberculosis is not recommended in raptors, particularly when the birds are in close contact with humans; but if treatment is deemed appropriate, a number of therapeutic agents have been proposed.[7] The treatment protocol should run parallel to an adequate biosecurity program targeted at containing the disease (see Chapter 28, Implications of Mycobacteria in Clinical Disorders).

The gram-negative microorganism *Chlamydophila psittaci*, produces chlamydiosis. This microorganism has been detected serologically in both captive[15,22] and free-living raptors,[21] but very few cases of actual clinical chlamydiosis have been recorded in the literature.[15,54] The disease is characterized by unilateral or bilateral periocular swelling and conjunctivitis, rhinitis and lime green-colored urates.[15] Suggested treatments include the administration of doxycycline at the dose rate of 50 mg/kg BID PO for 45 days[15] or 100 mg/kg IM every 5 days for a total of six doses,[29] or azithromycin at 40 mg/kg once a week PO for up to 4 weeks[102] (see Chapter 27, Update on *Chlamydophila psittaci*).

There are very few reports in the literature on the incidence of clostridial enterotoxemia in raptors.[108] This disease is produced mainly by *Clostridium perfringens* type A/B, although *C. histolyticum* also has been implicated on a single occasion.[108] *Clostridium perfringens* is characterized by the production of potent toxins that are readily absorbed from the gastrointestinal tract, resulting in severe illness and death. Mismanagement of food items before and after freezing has been blamed as a possible cause for the proliferation of the microorganism.[29,108] In addition, the management system used widely by falconers — providing meat soaked overnight in water to falconry birds — has been proposed as a potential risk for the proliferation of *C. perfringens*.[29] In most cases of infection, the course of disease is peracute or acute. In peracute cases, death occurs after a relatively short period of general depression. In acute cases, clinical signs include general depression, reduced appetite, regurgitation, soft brown feces progressing rapidly to reddish and hemorrhagic, pastel green-colored urates and recumbency.[29] Suggested treatments for the acute form include the use of oxytetracycline at the dose rate of 100 mg/kg SID for 5 days or a single dose of a doxycycline long-acting preparation at the dose rate of 100 mg/kg.[29] Three falcons that displayed typical clinical signs of clostridial enterotoxemia survived after the administration of a bovine polyvalent gangrene antiserum IV.[108]

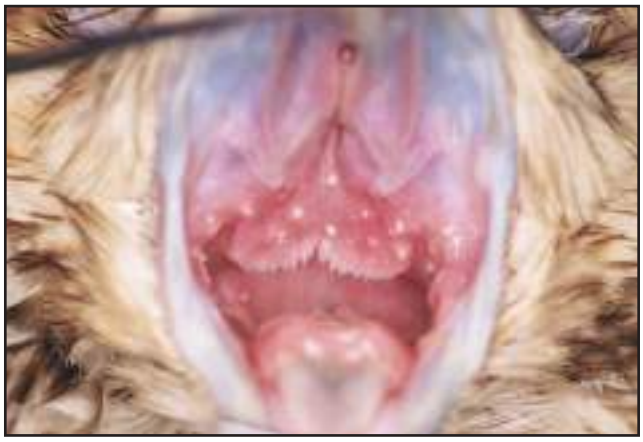


Fig 40.53 | *Pseudomonas aeruginosa* stomatitis as a sequel to a recent trichomoniasis infection. Note the characteristic multiple caseous masses present in the caudal and lateral aspects of the oropharynx and on the tongue. In most cases, the tongue is grossly enlarged, preventing birds from eating normally.

Pseudomoniasis is an infectious disease produced by the rod-shaped, gram-negative bacterium *Pseudomonas aeruginosa*. In avian species, *P. aeruginosa* is considered to be an opportunistic pathogen and seldom is considered the primary source of infection.[12,23] *Pseudomonas aeruginosa* is commonly associated with upper respiratory tract infections in psittacine birds[60] and is often isolated from respiratory tracts of juvenile ostriches.[12] It is believed that predisposing factors such as injuries to mucosal membranes, general weakness produced by systematic diseases, immunosuppression and reduced normal flora might lead to infections with *P. aeruginosa*.[60] Recently, stomatitis produced by *P. aeruginosa* as a sequel to trichomoniasis was described in falcons in the Middle East.[80] The disease was characterized by the presence of small, nodular, white-yellow caseous masses, ranging in size from 2 to 5 mm distributed across the oropharynx (Fig 40.53). In addition, the tongue displayed numerous nodular masses distributed across the dorsal and lateral aspects, varying in size from 0.5 to 5.0 mm, resulting in a gross enlargement of the tongue to the extent that the falcon could not close its beak.[80] Treatment includes administering antibiotics such as piperacillin and amikacin or tobramycin in combination, daily

curetting of caseous masses and the application of oral antiseptics.

There have been reports in the literature of bacterial findings during microbiology surveys in raptors. Some of the bacteria isolated probably can be considered normal or transient flora, and therefore their presence will not necessarily result in disease. For instance, the culture of some bacteria, such as *Pseudomonas* spp., from infected wounds is relatively common, but is not necessarily involved in the infectious process.[9] One such finding includes *Mycoplasma* in different species of free-living raptors[49,50] and captive falcons in the Middle East.[48] However, as previously stated, "Today's commensal is tomorrow's pathogen".[9] Under some circumstances, some bacteria might be pathogenic due to immunosuppression and stress, resulting in clinical disease. For instance, mycoplasmosis was recently diagnosed in a free-living saker falcon nestling affected with skeletal deformity due to perosis.[14] Other bacteria that have been implicated in clinical disease include *Pasteurella multocida*,[55] *Salmonella enteriditis*, *S. typhimurium*,[16] *Campylobacter jejuni*,[16] *Yersinia pseudotuberculosis*,[16] *Escherichia coli*[9] and *Staphylococcus aureus*.[9]

VIRAL DISEASES

The most significant viral diseases in raptors include Newcastle disease, avian pox and raptor herpesvirus infection.

Newcastle disease is an infectious viral disease produced by avian paramyxovirus serotype 1.[53,71,104,107] In falcons, the disease is characterized by gastrointestinal signs in the earliest stage of the disease, followed by central nervous system signs including ataxia, head tics and tremors, and wing and leg paralysis.[107] Annual vaccination with inactivated vaccines is highly recommended.[9,29,106] A vaccine derived from local strains in the Middle East has been used successfully in different species of birds including falcons.[107]

Avian pox is a viral disease known to affect captive[37,82] and free-living raptors.[95] The interrelationship of the different strains is not clearly understood. Pox infections in raptors are not considered fatal. However, the loss of an eye due to scratching, the loss of digital function due to avascular necrosis of digital tendons and the sloughing of talons due to distal necrosis are very often observed. In addition, pox infections on the cere can lead to permanent damage to the nostrils, the nasal septum, conchal structures and, in extreme cases, to avascular necrosis of the nasal bridge (Figs 40.54, 40.55). Treatment of early-stage lesions consists of cauterizing, debriding the upper layer and application of alcohol-based antiseptic solutions (eg, merthiolate, mercurochrome or iodine). This treatment tends to arrest the development of first-stage lesions into scabs. Scabs are best treated only with antiseptic solutions. The addition of glycerol or liquid paraffin wax to the antiseptic solution is beneficial. Scabs on the cere are treated by surgical removal and the periodic application of hydrocolloidal dressings[86] (Figs 40.56-40.59). Diagnosis of the disease is possible by identifying the virus at electron microscopy or by detecting the presence of Bollinger's intracytoplasmatic bodies in cytologic or histologic preparations of the skin of affected individuals.[104] Annual vaccination with commercially available live vaccines has been suggested.[104] Recently, a new strain of the virus was isolated from falcons in the Middle East, and studies are under way to develop a more species-specific live vaccine.[5]

Raptor herpesvirus infection is a disease that affects captive and free-living Falconiformes and Strigiformes.[24] Three distinct species of the virus have been identified to date including Falconid HV1, Strigid HV1 and Accipitrid HV1.[24,104] Affected individuals invariably die within 2 or 3 days after manifesting signs, including anorexia, pastel green-colored urates and general depression. The infection produces widespread characteristic white-yellow necrotic foci in the parenchyma of the liver and spleen[29] (Fig 40.60). Considerations for the production of a vaccine have been extensively discussed.[67,68] Recently, an attenuated vaccine has been produced and used successfully in common kestrels.[105]

Other viral infections affecting raptors worldwide include the West Nile virus,[98] avian influenza[51] and avian polyomavirus.[36] A comprehensive review of the viral diseases affecting raptors has been published elsewhere[9] (see Chapter 32, Implications of Viruses in Clinical Disorders).

FUNGAL DISEASES

Aspergillosis is the most common infectious disease affecting captive raptors.[63] The disease is caused mainly by *Aspergillus fumigatus*, although other fungi of the same genus, such as *A. flavus* and *A. niger*, also have been implicated.[9,29] Some species appear to be more susceptible than others to contracting the disease. For instance, raptor species that have been identified as prone to contract aspergillosis in captivity include the golden eagle (*Aquila chrysaetos*), goshawk (*Accipiter gentilis*), gyrfalcon (*Falco rusticolus*), immature red-tailed hawk (*Buteo jamaicencis*) and snowy owl (*Nyctea scandiaca*).[63] Aspergillosis is usually contracted through heavy or mild spore inhalation over a period of time, together with factors that compromise immune function. Examples of these factors include recent capture, poorly ventilated quarters, neonatal and geriatric conditions, corticosteroids, exposure to respiratory irritants and lead toxicosis.[63] After exposure, the disease might follow

Fig 40.54 | A large scab on the cere of a saker falcon produced by an infection with avian pox. Note that the naris is completely obstructed. The removal of the scab and application of hydrocolloid dressings is indicated. Complete healing has been achieved in 2 to 3 weeks following this technique.

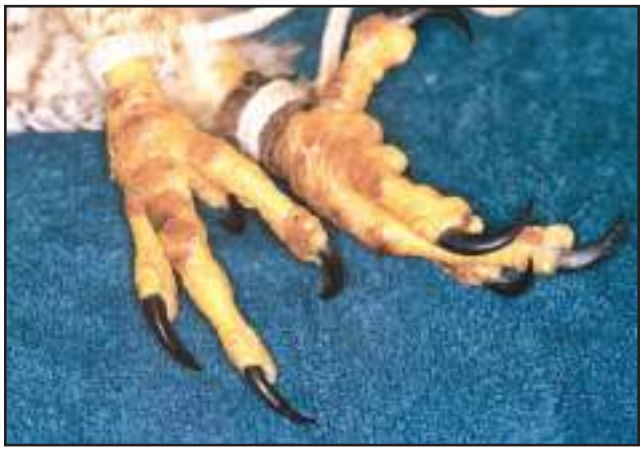


Fig 40.55 | Avian pox infection on both feet of a peregrine falcon. Note that the lesions are still in the pustular stage. The electrocauterization of such lesions and the application of an alcohol-based antiseptic are indicated. The primary objective of such therapy is to prevent the development of scabbing.

Fig 40.56 | Early stage of the development of pox infection in a male saker falcon. Note that the eyelids and the cere are equally affected. Pox infections in the Middle East follow a seasonal pattern and are relatively common during the spring.

Fig 40.57 | A pustular-type pox lesion on the commissure of the mouth of a saker falcon. This type of lesion is relatively rare in raptors.

Fig 40.58 | A large fistula in the commissure of the mouth of a saker falcon created by the presence of a large pox scab. Prompt and effective treatment is required in order to arrest the development of early lesions into scabs in this anatomical site. Surgical reconstruction of the mouth is indicated in this type of presentation.

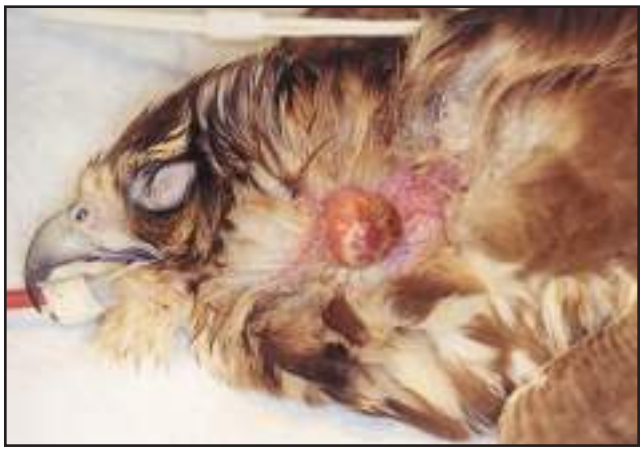


Fig 40.59 | An unusual nodular growth produced by avian pox on an apterium of the neck of a saker falcon. The dry type of avian pox infection tends to develop in unfeathered areas of the body, mainly on the cere, eyelids, tarsus and digits. These dry lesions are infrequently observed on other anatomical sites.

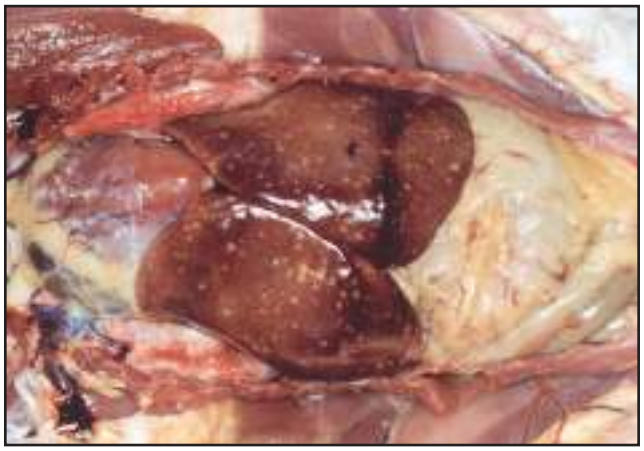


Fig 40.60 | Herpesvirus infection in a saker falcon. The liver is slightly enlarged. Note the presence of characteristic multiple necrotic foci disseminated across the liver. The bird was presented with a history of anorexia and passing pastel green-colored urates.

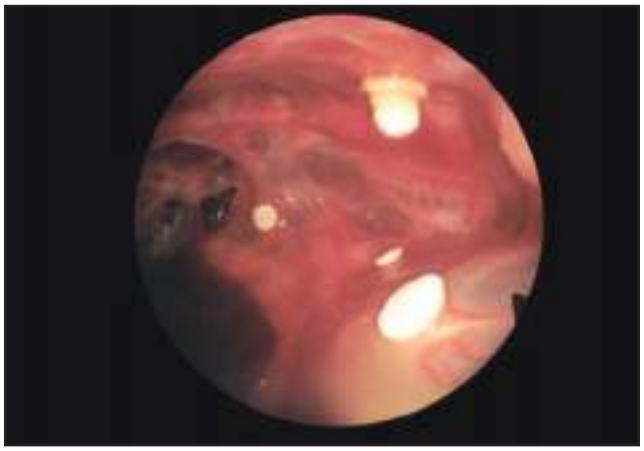


Fig 40.61 | Early lesions of aspergillosis in the coelomic cavity of a saker falcon. Stress associated with overexertion and recent capture, exposure to decaying vegetable matter and immunosuppression have all been implicated in the development of this fungal disease. Some raptor species appear to be more susceptible to developing the disease.

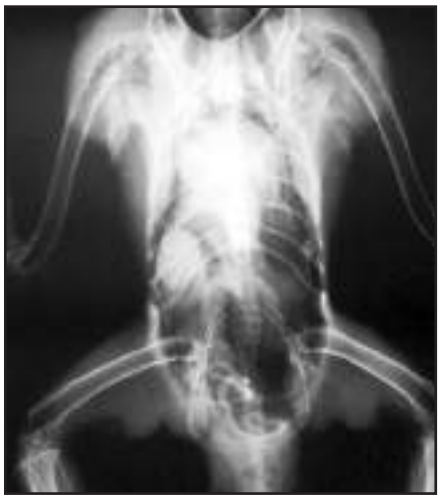


Fig 40.62 | Large aspergilloma in the coelomic cavity of a saker falcon as observed on a ventrodorsal radiograph. It is very common for raptors to develop a single, large aspergillosis lesion.

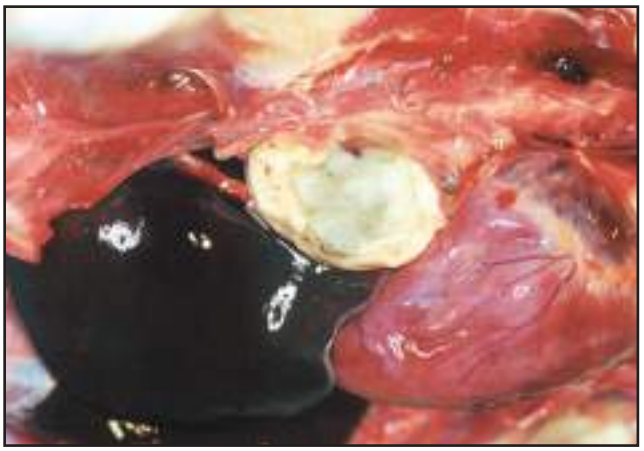


Fig 40.63 | A postmortem examination of the bird in Fig 40.62. Note the size and location of the already sectioned aspergilloma showing sporulating fungal colonies of *Aspergillus fumigatus*.

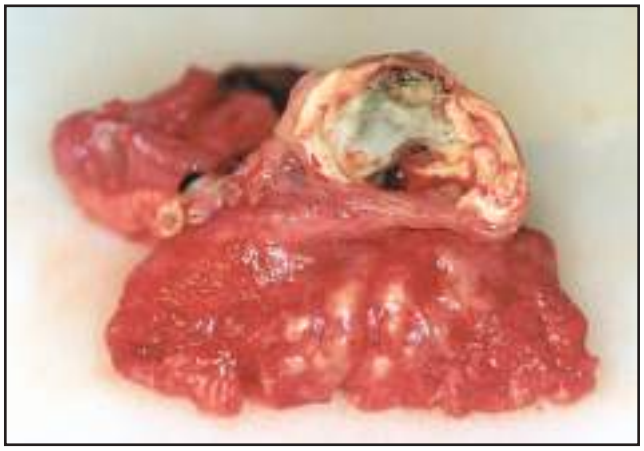
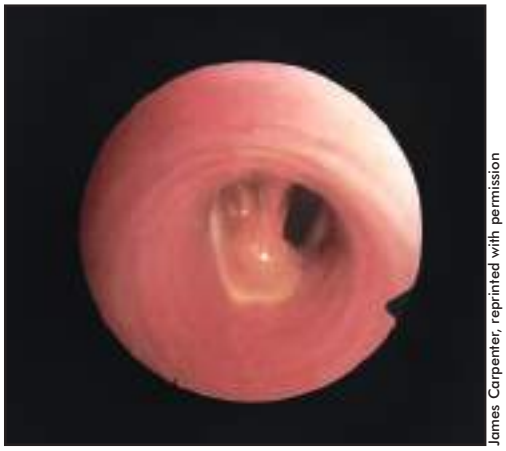


Fig 40.64 | A dissection of the aspergilloma and the lungs of the bird in Figs 40.62 and 40.63. Note the numerous smaller aspergillomas developing in the lungs.



James Carpenter, reprinted with permission

Fig 40.65 | Unilateral obstruction of one primary bronchus in a saker falcon produced by infection with *Aspergillus fumigatus*. Severe bilateral obstruction requires the placement of an air sac cannula to alleviate dyspnea.

an acute or a more chronic course depending on exposure and immunosuppression. The acute form is characterized by the development of extensive miliary granulomas throughout the lungs. The more chronic forms might include the development of small to large granulomas mainly in the tracheobronchial syrinx, pericardium, lungs and air sac system[63] (Figs 40.61-40.65). At the onset of the disease, clinical signs typically include subtle changes in behavior, reduced appetite, shredding and flicking of food, lack of stamina and decreased flight performance. Respiratory signs including dyspnea, wet rales and change of voice, and usually follow behavioral changes. Diagnosis is made through a detailed clinical history, tracheal and air sac swabs submitted for cytologic examination and culture, endoscopy examination, hematology, enzyme-linked immunosorbent assay (ELISA) and antigen capture test.[63] Treatment typically consists of itraconazole[u] PO at the dose rate of 5 to 10 mg/kg BID for 5 days then decreased to q24h for 2 to 3 months, amphotericin B[v] intratracheally (IT), 1 mg/kg diluted to 1 ml volume BID for 5 days, and nebulization with clotrimazole for 1 hour BID for 2 to 3 months.[63] Surgery is usually recommended to remove aspergillomas located at the syrinx and within the coelomic cavity. A 1:10 solution of enilconazole[w] 0.5 ml/kg BID intratracheally (IT) and nebulization with a 1:10 dilution for 20 minutes QID also have been recommended.[17] Recently, nebulization with a quaternary ammonium and biguanadine compound disinfectant[n] has been proposed.[4,103]

Candidiasis, or thrush, is a fungal disease produced primarily by the yeast *Candida albicans*. The disease is usually endogenous in origin and can be triggered by predisposing factors including immunosuppression, stress, prolonged use of antibiotics and nutritional deficiencies.[57] In raptors, candidiasis is characterized by the presence of amorphous diphtheritic membranes that are whitish gray to gray-green in color and affect mainly the crop[88] (Fig 40.66). There are several other diseases that affect the upper digestive tract in raptors. These include vitamin A deficiency, *Pseudomonas aeruginosa* stomatitis, trichomoniasis and capillariasis. The importance of establishing a complete list of differential diagnoses prior to instituting therapy is stressed.[88] Raptors affected with candidiasis demonstrate clinical signs including reduced appetite or anorexia, shredding and flicking of food, regurgitation and progressive weight loss.[88] The diagnosis is suggested through the clinical history and observation via endoscopic examination of the characteristic "Turkish towel" appearance of the crop. The blastospores of *C. albicans* can be demonstrated in stained cytological preparations obtained from affected areas. Traditionally, the treatment of candidiasis in raptors has included nystatin, ketoconazole, itraconazole or fluconazole. Topical use of a miconazole preparation[x] applied BID for 3 to 5 days has recently been described[88] (see Chapter 29, Implications of Mycoses in Clinical Disorders).

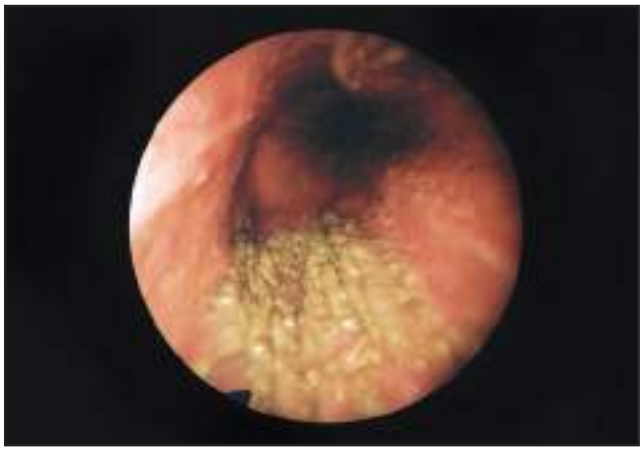


Fig 40.66 | Infection with *Candida albicans* in the crop of a saker falcon. Candidiasis is often associated with immune depression, delay in emptying of the crop and nutritional deficiencies. Note the typical "Turkish towel" appearance of the mucosal membrane of the crop.

Non-infectious Diseases

Non-infectious diseases include a diverse group of medical conditions caused by a single or multifactorial etiology. While for some of these conditions the etiology is clearly understood, such as in the case of metabolic bone disease, the causes of others such as amyloidosis are not known.

Palpation of the pectoral muscles, weighing the bird and assessing its behavior are commonly performed to determine body condition in captive raptors. The assessment of pectoral muscle mass on its own is not a reliable indicator of body condition, as most of the body fat is deposited within the coelomic cavity, under the wings around the scapular areas and in the lower abdomen. The best way of assessing body condition in captive raptors is a combination of the three parameters mentioned above.

METABOLIC BONE DISEASE

Metabolic bone disease is a term that encompasses a series of medical conditions that affect mainly the mineralization of bones, including osteoporosis, osteomalacia, rickets, fibrous osteodystrophy and nutritional secondary hyperparathyroidism. These conditions are usually related to mineralization disorders of the osteoid due to Ca and/or vitamin D_3 deficiencies. Other diseases affecting the growth, mineralization, maturation and maintenance of bone include the twisting and bending of long bones and slipped tendon, which are commonly seen in fast-growing birds.[39] Correct diet, adequate feeding management and vitamin/mineral supplementation are

essential in breeding raptors in captivity.[29] Excellent analysis and recommendations for raptor nutrition have been published elsewhere.[18] (*Ed. Note: While the Author does not do so, this reference advocates day-old chicks which may have an acceptable Ca:P but there is insufficient calcium. At the University of Guelph they fed chicks to growing Northern Harriers and they all developed metabolic bone disease).* Treatment is usually indicated and includes changes in the diet and vitamin/mineral supplementation.[29] However, individuals already exhibiting folding fractures and severe bending of long bones should be humanely destroyed, as treatment is seldom successful.

Disorders of the Digestive System

SOUR CROP

Sour crop in raptors is a medical emergency condition caused by the retention of food in the crop, resulting in content decomposition leading to rapid bacterial proliferation, septicemia and death. The condition might result simply from over-feeding. This is a common occurrence in falconry birds when enthusiastic falconers allow a particular bird to gorge itself to rapidly increase the body weight. Factors that might influence crop stasis include low body condition, gizzard impaction with casting or foreign material, or general disease. Usually birds with sour crop are presented in a state of endotoxic shock with a swollen crop that is filled with macerated and foul-smelling, decomposing food. Affected birds tolerate general anesthesia with isoflurane very well. An endotracheal tube should be placed carefully to avoid aspiration pneumonia. Gently massaging the crop contents toward the mouth and retrieving this material with blunt-tipped forceps should remove the food. Extreme care should be exercised during the massaging process to avoid lacerations or penetrating wounds to the crop if there are bones present in the crop. In extreme cases, an ingluviotomy can be performed to remove impacted contents. After removal of the decomposing food, a tube should be passed down to the proventriculus, and approximately 50 ml/kg of 0.1% chlorhexidine or 1:1000 of quaternary ammonium and biguanadine compounds disinfectant[n] solution can be flushed to decrease the bacterial load present in the upper GIT. The author favors the use of marbofloxacin[y] 15 to 20 mg/kg PO or IM for 3 to 5 days SID in the postoperative care of sour crop. Depending on the bird's condition, force-feeding might be necessary for 2 to 3 days. An excellent account of the medical management of sour crop has been previously described.[61]

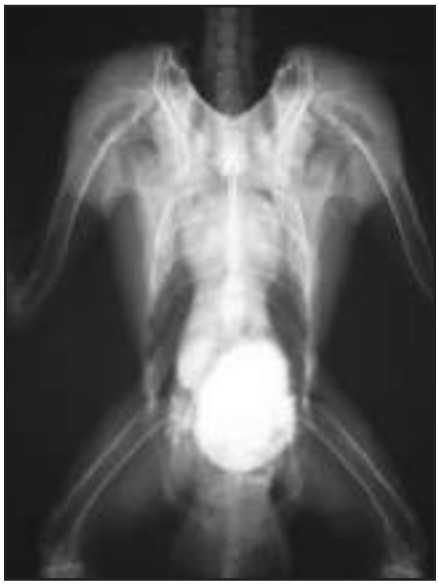


Fig 40.67 | Ventrodorsal radiograph of a saker falcon showing the presence of foreign material in the gizzard. The ventriculus was repeatedly flushed retrieving a large amount of red desert sand. The bird was presented with a history of delay in emptying the crop, reduced appetite and progressive weight loss.

GASTROINTESTINAL FOREIGN BODIES

Gastrointestinal tract (GIT) foreign bodies are a relatively common occurrence in raptors. One of the most significant findings includes lead pellets or lead fragments in the GIT, usually the ventriculus, of raptors fed on shot prey. A full account of the diagnosis and management of lead toxicosis will be addressed later in this chapter. Other foreign materials that have been found include grass, synthetic strands from artificial turf, sand and grit (Fig 40.67). In general, these are materials that tend to adhere to the offered food when it falls on the ground. Small quantities of these materials are usually cast out (grass, artificial turf strands) or passed out with fecal material (sand and grit). However, large quantities of foreign material can significantly reduce the capacity of the proventriculus and ventriculus or cause impaction in these sites.

Falconry birds appear to be the most commonly affected. In the Middle East, it is very common to be presented with a falcon with a history of reduced appetite or complete anorexia, delayed crop emptying and regurgitation. Radiographically, large amounts of sand and medium-sized grit have been observed in the ventriculus. In some cases, it has been estimated that approximately 50 to 75% of the total capacity of the ventriculus has been occupied by foreign material. In one case involving a wild-caught gyrfalcon, the ventriculus and proventriculus were completely impacted with grass. This bird died before any treatment could be instituted. The wet weight of the grass retrieved from the ventriculus was 40 g. It also is

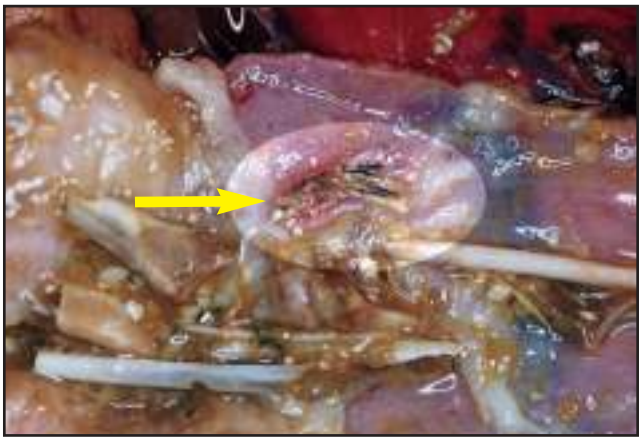


Fig 40.68 | A unusual case of a penetrating wound on the wall of the gizzard of a saker falcon caused by a sharp, bony splinter (arrow). The bird died of acute peritonitis. Other bone fragments with sharp edges also are present. Under normal circumstances, bones are cast out the following day after a meal. However, entire long bones (eg, femur) should be not be offered to captive raptors.

Fig 40.69 | Severe sinusitis and associated conjunctivitis in a saker falcon. Note the large amount of clear discharge originating from the nares and the nasolacrimal duct. Sinusitis may be the result of systemic (eg, chlamydiosis), localized infections (eg, trichomoniasis) or secondary bacteria.

common to find large amounts of impacted casting material (fur, feathers, bones). Retrieval of foreign material from the gizzard can be attempted by ventricular lavage. For large falcons (1.0 to 1.5 kg), the author favors stomach tubes commonly used to feed neonatal lambs. This procedure should be undertaken with the bird under isoflurane anesthesia. The placement of an endotracheal tube is essential to avoid aspiration. The stomach tube is lubricated with water-soluble gel[l] and passed gently into the gizzard. A 60-ml syringe is then attached to the tube and the stomach is flushed repeatedly with warm (32-35° C) water. The quantity of water used depends on the amount of foreign material present. Usually, 180 to 240 ml is sufficient to dislodge all material. In severe cases of ventricular impaction, surgical removal of foreign bodies is indicated. The author has used a rigid endoscope and 33 cm long modified grasping forceps, to remove casting material, lead pellets and other foreign bodies from the gizzards of falcons (Fig 40.68).

Disorders of the Respiratory System

Rhinitis is the inflammation of the mucosal membrane of the nasal cavities, which may be due to the presence of foreign bodies, rhinoliths, trichomoniasis or bacterial invasion (Fig 40.69). Captive falcons have the tendency to face the wind while tethered out in the open. Sand storms are relatively common in the Middle East during the training and hunting season. Very often, fine dusty sand finds its way into the nares, producing mechanical occlusion within the nasal cavities.[74] Trichomoniasis and associated invading bacteria affecting the nasal cavities of captive falcons have been previously described.[93] The only clinical sign usually observed is fluttering of the skin immediately above the affected nares. In severe and chronic cases, the presence of foreign material and rhinoliths, and complete obstruction of the nares is obvious on physical examination. Treatment consists of the manual removal of obstructing debris with a fine blunt probe or a pair of fine-tipped round forceps, together with the instillation of saline drops. Sometimes, several sessions are required to remove all foreign material. Flushing the nasal cavities of affected individuals is highly recommended, as this tends to dislodge foreign material more efficiently (Figs 40.70-40.72). In severe or chronic cases, partial retrieval of foreign material before flushing is often necessary. The flushing solution is usually prepared with an antiseptic (chlorhexidine 50 ml/kg of 0.1%) or an antibiotic (enrofloxacin or marbofloxacin) in normal saline. The author favors a 1:250 solution with quaternary ammonium and biguanadine compounds[n]. Construction of a device for flushing the nares of affected falcons has recently been described in the literature.[81]

Sinusitis is the inflammation of the infraorbital sinuses, produced mainly by invading bacteria. In most cases, sinusitis is produced as a sequel to rhinitis caused primarily by obstruction and subsequent proliferation of bacteria. Supraorbital trichomoniasis infection was recently described in saker falcons.[79] Intranasal flushing with antibiotics or antiseptics has some effect in mild cases. However, intrasinal injection of solutions such as those used for rhinitis is highly recommended. Strict aseptic and sterile measures should be observed when injecting into the sinus. The use of antibiotics such as marbofloxacin 15 to 20 mg/kg PO, IM SID for 5 to 7 days is indicated.

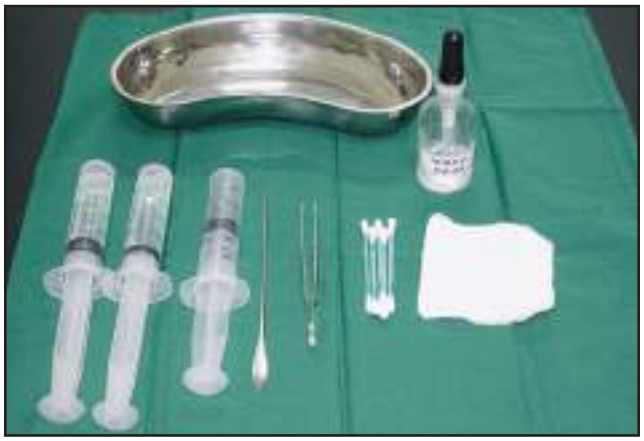


Fig 40.70 | One of the most effective collateral treatments in the therapeutic management of rhinitis and sinusitis is the systematic flushing of antiseptic, disinfectant or antibiotic solutions through the nares of affected individuals. The equipment commonly used to clean the nares and to flush the nasal cavities is shown here.

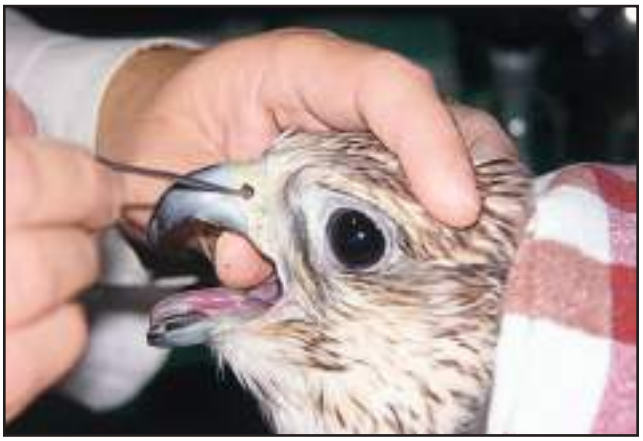


Fig 40.71 | Small round-tipped probes are very useful to remove rhinoliths from the nares and relieve obstructions. It is important to soak rhinoliths with saline solution drops during the removal process. Extreme care must be exercised to avoid injuries to the concha.

Fig 40.72 | A nasal flushing of a saker falcon in progress. Large syringes (eg, 20 ml) fitted with a small rubber plunger at the end are useful for this procedure. Note that the falcon has been wrapped with a towel in order to avoid damage to the feathers.

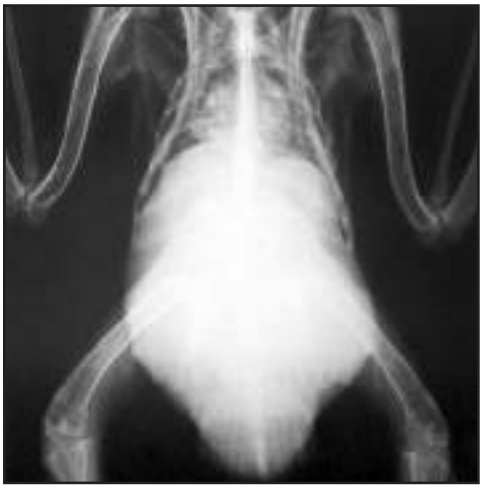


Fig 40.73 | Amyloidosis and related ascites in a saker falcon. The etiology of amyloidosis in raptors is poorly understood. Therapeutic management of amyloidosis is still in its infancy. Some success has been obtained by removal of the ascitic fluid and the administration of glucose and ascorbic acid, and general support therapy.

Non-infectious Diseases

Amyloidosis (*Greek: Amylon = starch, eidos = form*) is a disorder affecting primarily the liver, although the spleen, kidneys and even the brain[99] can be involved. The condition is characterized by the deposition of amyloid within the parenchyma of the liver, engulfing and obliterating the hepatocytes.[30] Amyloid deposition appears to be stimulated by chronic disease involving antibody-antigen production, such as aspergillosis, bumblefoot or tuberculosis. However, the author has encountered several mild to severe cases of amyloidosis in falcons with no previous history or evidence of disease. It is possible, therefore, that subclinical disease might also stimulate deposition of amyloid in the liver. Nutrition or nutritional management also may play an important role, as the majority of cases seen by the author have been in falcons that have undergone dietary changes such as the removal of one of the main food items (eg, day-old chicks) several weeks before the onset of disease (Fig 40.73).

Disorders of the Nervous System

There is a series of disorders involving the nervous system in captive raptors, produced by nutritional deficiencies, infectious diseases, toxicosis and lesions of the central and peripheral nervous systems.[9] For a comprehensive review of the different neurological disorders affecting raptors, the reader is referred to a recent publication.[9] However, an account of some of the most

recently described disorders and those commonly observed by the author in falcons in the Middle East is presented here.

Progressive paralysis of the hind limbs might be the result of lead toxicosis, botulism (G. Harrison, personal communication, 2003), aspergillosis, kidney disease, trauma, arthritic changes in the vertebral synsacral joint due to trauma,[25] Newcastles disease[107] and atypical parasitic migration.[28] The latter finding might be a more common occurrence that has gone largely undiagnosed in geographical areas, including the Middle East, where parasitic infections with different species of the genus *Serratospiculum* are very common. Fish-eating raptors such as the osprey, fed frozen/thawed fish, require supplementation with thiamine (vitamin B_1) as part of dietary management to avoid paralysis of the hind limbs.[65] In addition, lumbosacral plexus avulsion has been suspected in tethered falcons presented with unilateral paresis or paralysis. Some of these cases have responded well to non-steroidal anti-inflammatory agents such as meloxicam[z] (0.1 mg/kg SID PO or IM for 3 to 5 days) and administration of 25 mg/kg vitamin B_1[aa] IM for 3 to 5 days or 25 mg/kg vitamin B_1[bb] PO for the same period or longer. Stargazing, convulsions and seizures have been observed in captive raptors, primarily in falcons[29] **(Figs 40.74, 40.75)**.

In the Middle East, falcons are commonly presented with CNS signs including moderate to severe tilting of the head or opisthotonos. These signs are exacerbated when the hood is in place. In severe cases, these falcons might even fall backward from the perch when disturbed. In addition, falcons are often presented with a history of having experienced a "fit during a training session or while simply standing on the perch". The etiology might be multifactorial, but deficiencies of B-vitamin complex[29] and vitamin E[9] have been implicated. In such cases, the author favors administering 1 ml/kg IM of a multivitamin preparation (providing 15,000 IU vitamin A, 25 μg vitamin D_3, 20 mg vitamin E, 10 mg vitamin B_1, 5 mg vitamin B_2, 25 μg vitamin B_{12}, nicotinamide 35 mg, 25 mg d-panthenol[cc]). This is usually followed by administering PO tablets of a multivitamin preparation[dd] (providing 2000 IU vitamin A, 250 IU vitamin D_3, 5 IU vitamin E, 2 mg vitamin K, 10 mg niacin, 2.5 mg pantothenic acid, 0.4 mg iodine, 1 mg vitamin B_1, 8 μg pyridoxine, 0.1 μg vitamin B_{12}, 0.5 mg folic acid, 80 μg choline bitartrate, 0.2 μg biotin, 0.4 mg riboflavin) administered directly or within the food for 1 or 2 weeks, in powder form (containing per 1 g 1000 IU vitamin A, 125 IU vitamin D_3, 2.5 IU vitamin E, 1 mg vitamin K, 5 mg niacin, 1.2 mg pantothenic acid, 1.2 mg riboflavin, 0.4 mg vitamin B_1, 0.4 mg pyridoxine, 0.5 μg vitamin B_{12}, 0.2 mg folic acid, 0.4 μg choline bitartrate, 0.1 μg biotin, 2 μg iodine) administered daily in drinking

Fig 40.74 | Saker falcon presented with severe torticollis. The problem was present only when the bird had its hood in place. Severe torticollis may lead to falcons falling from the perch due to loss of balance. The etiology of this condition is believed to be the result of vitamin B complex deficiency.

Fig 40.75 | Saker falcon displaying opisthotonos, a common central nervous sign associated with vitamin B complex deficiency in raptors.

Fig 40.76 | Unilateral flaccid paralysis of the foot in a saker falcon. The etiology of this condition is not clearly understood, but avulsion of the nervous plexus or injury to individual nerves has been suggested as the possible cause.

water for 1 or 2 weeks. Success in treating such cases appears to be related to how promptly the treatment is instituted. Recovery and full cessation of clinical signs have been observed when treatment was administered within 24 to 48 hours after the initial signs were noticed. However, the etiology of this and other diseases remains unclear and there is, therefore, a need to conduct further research into the etiology, diagnosis and pathogenesis of neurological disorders affecting raptors (Fig 40.76) (see Chapter 4, Nutritional Considerations).

Disorders of the Eye and Ear

Conjunctivitis is the inflammation of the membrane that lines the eyelid and the exposed surface of the sclera, characterized by hyperemia and, in severe cases, discharge. The condition in raptors is usually associated with the presence of foreign bodies such as feather particles, sand or dust. Foreign material also can be lodged under the nictitating membrane or lower eyelid. If the presence of foreign material is suspected, the bird should be examined under general anesthesia and the eye repeatedly flushed with normal saline solution or a proprietary eye solution[ee] and any visible foreign material removed with the aid of sterile cotton swabs. The treatment of conjunctivitis might include ophthalmic drops[ff] BID for 3 to 5 days. In more severe cases and in the presence of inflammation, the use of corticosteroid (betamethasone) and antibiotic (neomycin) drops[gg] BID for 2 to 3 days might be considered.

Corneal lacerations and keratitis are probably the most common types of injuries seen in raptors. Most of the injuries are caused by trauma. Lacerating wounds to the cornea are often observed in falcons in the Middle East. Falcon trappers use a procedure called "sealing" to tame newly caught free-living falcons. The procedure consists of placing a stitch on the proximal edge of the lower eyelids with a standard stitching needle and a fine cotton thread. The two threads are then secured with a knot on the top of the head to close shut or "seal" the eyes. During the process it is very common to produce penetrating wounds and lacerations to the cornea while stitching. Mild to severe injuries to the cornea usually benefit from temporary tarsorrhaphy and the use of antibiotic eye drops.[58] Corneal injuries also can result when one of the laces used to close and open the hood is inadvertently left inside and in direct contact with the eye. The use of fluorescein stain in the assessment of the integrity of the cornea is recommended, as eye drops or ointments containing corticosteroids should not be used in the presence of corneal lacerations since these might delay healing.

Uveitis is the inflammation of the vascular middle area of the eye including the iris, ciliary body and choroid. Uveitis and tears in the iris are commonly seen in raptors involved in collision accidents and trauma to the head.[38] Mild to severe cases of uveitis can be resolved successfully with the use of a non-steroidal anti-inflammatory agent such as meloxicam[z] at the dose rate of 0.1 mg/kg SID PO or IM for 5 to 7 days, and the instillation of corticosteroid and antibiotic drops[gg] BID for 2 to 3 days.

Otitis is the inflammation of the ear and is relatively rare in raptors. In falcons, otitis is usually produced by the presence of foreign material, ectoparasites or trichomoniasis. Affected individuals usually present with a mild to severe head tilt, feather matting and a thick purulent discharge around the ear opening. The treatment of otitis consists of removing caseous and purulent material with normal saline and cotton swabs and the instillation of corticosteroid and antibiotic drops[gg] BID for 3 to 5 days. Trichomoniasis should be treated with metronidazole 100 mg/kg SID PO for 3 days. The author has observed severe cases of trichomoniasis causing marked injury and destruction of vital structures of the middle and inner ear. These cases carry a poor prognosis.

Miscellaneous Medical Disorders

TOXICOSIS

Ammonium chloride toxicosis is a common medical disorder in falcons in the Middle East. At the end of the molting season, many falconers routinely administer ammonium chloride to their falcons. The theory behind this practice is belief that ammonium chloride dissolves the fatty layer accumulated in the stomach throughout the long molting season, resulting in a hungrier bird. Other falconers prefer to administer this substance only if a falcon failed to make a kill or showed little interest in a chase.

This toxic agent is usually administered to falcons in the form of a large crystal. Three to five minutes after administration, falcons vomit violently, bringing up large quantities of thick white to green-yellow mucus. However, often the large crystal breaks down into smaller fragments within the crop, resulting in only partial vomition of the crystal originally swallowed, an event that usually goes unnoticed by the falconer. Acute ammonium chloride toxicity can cause death within 10 to 15 minutes after administration. Rapid loss of appetite, progressive weight loss and the passing of characteristic dark metallic green mutes characterize chronic toxicosis. Terminal signs include incoordination, inability to stand, seizures and

Fig 40.77 | Lead toxicosis in a peregrine falcon. Common clinical signs of lead toxicosis include ataxia, hock sitting, hyperesthesia and convulsions. Falcons in the Middle East are commonly affected by ingesting lead pellets or lead fragments found in the carcasses of shot prey offered by falconers.

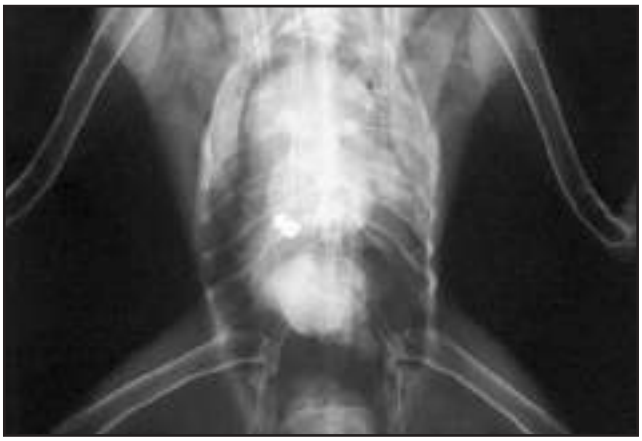


Fig 40.78 | A large lead pellet found in the gizzard of a saker falcon. The bird was presented in a state of collapse and displaying severe convulsions. The pellet was retrieved using a flexible endoscope.

opisthotonos, followed by death. The severity of clinical signs depends on the total amount of ammonium chloride ingested.[94] Mild toxicosis cases respond well to fluid therapy and force-feeding for 3 to 5 days.

Falconers in North America and Europe have used "rangle" since medieval times to produce the same effect as that obtained using ammonium chloride by falconers in the Middle East. "Rangle" are small stones commonly found along river banks. Field biologists have observed many species of falcons and other birds of prey, in many different parts of the world, ingesting these stones as part of their normal behavior. The stones are commonly cast out the following day. This phenomenon also has been observed in captivity, and the provision of such stones within aviaries should be promoted.

Clinical lead toxicosis in raptors is characterized by ataxia, paresis or paralysis of the wings and legs, amaurosis and convulsions (Figs 40.77, 40.78). In addition, the author has consistently observed the presence of hyperesthesia in falcons affected by clinical lead toxicosis. Diagnosis of lead toxicosis is carried out by detecting the presence of lead pellets or lead fragments within the gastrointestinal tract through radiography and/or the measurement of lead levels in whole blood. The use of a novel diagnostic system[hh] was recently published.[87] Lead pellets are normally removed either by gastric lavage or via endoscopy-assisted retrieval with rigid or flexible endoscopes and long grasping forceps. Lead fragments are normally removed through gastric lavage only (Figs 40.79-40.81). In falcons, the treatment recommended by the author is disodium calcium edetate[ii] at the dose rate of 50 mg/kg administered undiluted BID IM. Retesting is carried out every 5 days until the lead level is lower than 20 μg/dl. Falcons have been treated for up to 23 days without observing any deleterious effect.[87]

Pesticide toxicosis is a general term used to encompass groups of chemicals used to eradicate unwanted and destructive animals and plants. These different chemicals can be classified under three main groups: herbicides, insecticides and rodenticides. In captivity, most cases of toxicosis are due to negligence or accident. Often the instructions provided by the manufacturer are not adequately followed or are ignored, and products are placed in direct or indirect contact with the birds or their food, water or living quarters. In the Middle East, the author has observed several cases of toxicosis in which falconers have sprayed falcons with products commonly used against mosquitoes, flies and cockroaches. In a recent case a saker falcon was presented with hyperesthesia, ataxia, paresis of wings and legs and anorexia. The bird in question was sprayed with a product[jj] containing prallethrin 0.055%, tetramethrin 0.216% and d-phenothrin 0.095%. The bird survived and made a successful recovery with supportive therapy including the administration of 20 ml/kg lactated Ringer's SC SID for 5 days, vitamin B_1 IM SID for 5 days, vitamin B complex IM and force-feeding via a stomach tube with a mixture of whole ground quail, chicken liver and a carbohydrate/electrolyte solution (see Table 40.15).

Pesticides have produced a significant negative impact on free-living raptor populations in different parts of the world. In some areas, whole populations of raptors have been decimated due to the indiscriminate use of pesticides via direct toxicosis, the production of thin-shelled eggs or breeding disorders. Most incidents are related to the use of pesticides to control unwanted insects such as grasshoppers, or rodents such as voles and rats, or birds such as the red-billed quelea (*Quelea quelea*). In general, raptors are the species most commonly affected in most geographical areas of the world by the widespread use of pesticides, as they occupy a prominent place at

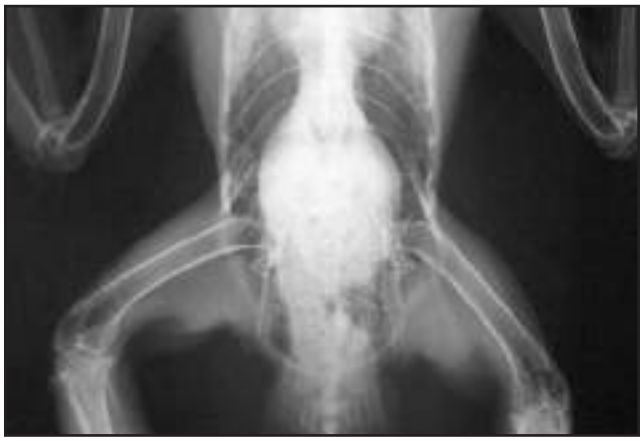


Fig 40.79 | A radiograph showing lead dust and lead fragments in the gizzard of a saker falcon. Often, lead pellets strike a bone, producing small lead fragments. Shot prey always contains this potential health hazard and should not be offered to raptors.

Fig 40.80 | The recommended way to remove lead dust and lead fragments is by repeatedly flushing the gizzard. It is important to place an endotracheal tube in order to avoid inhalation pneumonia during the process.

Fig 40.81 | Small blunt-end tubes commonly used to feed neonate lambs are useful for flushing the ventriculus of small to medium-sized raptors. Note the large syringe (60 ml) attached to the tube. Warm water (32-35° C) is commonly used as the flushing solution. A total volume of 180 to 240 ml is often necessary to dislodge all lead particles.

Fig 40.82 | A large lipoma near the shoulder joint of a saker falcon. This type of neoplasia is rare in raptors. The growth was successfully removed using standard surgical procedures.

the pinnacle of the food chain[9] (see Chapter 31, Implications of Toxic Substances in Clinical Disorders).

NEOPLASIAS

A recent publication lists that 122 neoplasms of 39 types recorded from 120 birds were identified in 44 species of raptors.[20] The most common types found in the survey include fibrosarcomas (17 cases), adenocarcinomas (16 cases), squamous cell carcinomas (13 cases) and papillomas (6 cases). A thyroid adenocarcinoma in a bald eagle (*Haliaeetus leucocephalus*)[6] and a thyroid cystadenocarcinoma in a saker falcon[90] have recently been described. These findings suggest that the incidence of neoplasms in captive raptors is not as rare as previously believed (Fig 40.82). More research is needed to determine the incidence of neoplasms in free-living raptors (see Chapter 20, Overview of Tumors).

UROPYGIAL GLAND IMPACTION

The uropygial gland, also known as preen or oil gland, is a bilobed gland located near the base of the tail. The gland secretes a lipoid sebaceous liquid, thick and transparent in appearance and with a characteristic light musky smell. The oil is spread on the plumage by the birds during preening to provide waterproofing. The secretion is carried by a number of ducts to an external papilla covered by a tuft of down feathers forming what is commonly known as the wick. One of the most common disorders affecting the uropygial gland is impaction. This is usually characterized by moderate to severe bilateral enlargement of the gland and a dry wick. Often the ducts of the wick are obstructed with hardened secretion forming a plug (Fig 40.83). The application of hot water compresses over the area and a gentle milking massage usually resolves the obstruction. More severe cases might require surgical intervention. In these cases,

two small incisions are made parallel to the wick, the contents are expressed and the area is irrigated with an antibiotic solution. Usually no suturing is required.

Molting Disorders

Molting is a natural process involving the periodic shedding and replacement of feathers within an annual cycle. In Falconiformes, the molt usually begins with the loss of primary feather No 4 progressing sequentially inward and outward from this point. Accipiters, harriers, Harris hawks, New World vultures and kites, however, follow a descending pattern similar to that of Passeriformes, in which the shortest innermost primary is shed first, proceeding sequentially toward the longer outermost feather. Large vultures and eagles tend to have a more erratic molting pattern, in which feathers from different locations are shed without following a particular order.[29]

Raptors in captivity often suffer from molting disorders including delayed molt, sudden shedding of a large number of feathers from one particular area, twisted feathers, stress or fret bars, "pin" feather and "pinched" feathers (Fig 40.84). Molting disorders might be the result of ecto- and endoparasitism, trauma, follicular infection or use of certain pharmacological agents during molting, but are most frequently the result of nutritional deficiencies.[52] The need to provide a suitable environment, an appropriately diverse diet and adequate vitamin/mineral supplementation during the molting period cannot be overstated. (*Ed. Note: If the proper diet is fed, supplementation can lead to toxicities).*

Molting can be induced by confining raptors to secluded environmental chambers and gradually modifying the photoperiod, temperature and humidity. The molting process also can be induced and accelerated with 1 mg/kg levothyroxine sodium (thyroxine) SID PO until molt is initiated.[5] Other authors have suggested doses of 25 μg SID PO for 1 week, followed by 50 μg SID for 1 week, then 75 μg SID for 1 week, then 50 μg SID for 1 week and finally 25 μg SID for 1 week for a red-tailed hawk (*Buteo jamaicencis*) weighing 1.4 kg.[52] It has long been recognized in the ancient Arab literature that feeding sheep thyroid gland can induce molting in raptors. Caution has been expressed about how to satisfy the voracious appetite of raptors for the first few days after they have consumed approximately 25 g of beef thyroid gland. The use of thyroxine to induce and accelerate molting in raptors is prescribed only in selected cases of delayed molt (Fig 40.85). This therapy should be accompanied by an adequate and balanced diet and suitable housing conditions throughout the entire molting period.

Fig 40.83 | Falconers in the Middle East often use needles in an attempt to open obstructed ducting of the uropygial gland. Poor asepsis and inadequate procedure often leads to infection and necrosis.

Fig 40.84 | A saker falcon without the main flight feathers. The falcon was administered one of the numerous commercial products widely available in the Middle East to induce molting. The powder is believed to be desiccated thyroid gland.

Fig 40.85 | Feathers molted prematurely during growth and development. The presence of such feathers, commonly called "pinched feathers," is the result of follicular infection, nutritional deficiencies or high stress during the molting period.

Physiological Reference Values

Table 40.20 | Hematological Reference Values of Selected Species of Raptors[2]

Assay	Red–tailed hawk (*Buteo jamaicensis*)	Great horned owl (*Bubo virginianus*)	Bald eagle (*Haliaeetus leucocephalus*)	Peregrine falcon (*Falco peregrinus*)	Gyrfalcon (*Falco rusticolus*)
	n=10	n=10	n=8	n=14	n=12
PCV (%)	44.6 (2.6)†	43.3 (2.9)	44 (4)	44 (4)	49 (2)
TP (g/dl)	4.3 (0.5)	5.1 (0.6)	4.0 (1)	2.65 (1.18)	2.94 (0.38)
WBC (x10^3/μl)	6-8	6-8	12.8 (4.8)	8.7 (2.2)	4.6 (1.7)
Heterophils (%)	35 (11.1)	47 (10.7)	75 (13)	65 (12)	51 (5)
Lymphocytes (%)	44 (8.9)	27 (7.0)	18 (10)	35 (13)	47 (5)
Monocytes (%)	6 (3.2)	9 (3.6)	3 (3)	0 (0)	1 (1)
Basophils (%)	2 (1.3)	Rare	Rare	0 (0)	Rare
Eosinophils (%)	13 (3.8)	1 (1.2)	4 (3)	0 (1)	1 (1)

†Standard deviation in parentheses.

Table 40.21 | Hematological Reference Values of Selected Species of Raptors[7,34]

Assay	Lanner falcon (*Falco biarmicus*)	Lagger falcon (*Falco jugger*)	Saker falcon (*Falco cherrug*)	Peregrine falcon (*Falco peregrinus*)	Merlin (*Falco columbarius*)
	n=42	n=13	n=50	n=70	n=33
Hb (g/L)	122-171	128-163	115-165	118-188	132-179
RBC (x10^{12}/L)	2.63-3.98	2.65-3.63	2.54-3.96	2.95-3.94	2.85-4.1
PCV (L/L)	0.37-0.53	0.39-0.51	0.38-0.49	0.37-0.53	0.39-0.51
MCV (fl)	127-150	123-145	124-147	118-146	105-130
MCH (pg)	42.3-48.8	38.0-47.7	41.4-45.4	40.0-48.4	36.0-45.9
MCHC (g/L)	317-353	312-350	304-349	319-352	340-360
WBC (x10^9/L)	3.5-11.0	5-9	3.8-11.5	3.3-11.0	4.0-9.5
Heterophils (x10^9/L)	1.65-8.8	3.5-6.57	2.6-5.85	1.4-8.55	3.2-4.03
Lymphocytes (x10^9/L)	1.1-5.13	1.7-4.0	0.8-4.25	1.1-3.3	1.2-1.56
Monocytes (x10^9/L)	0.0-0.9	0.0-0.85	0.0-0.8	0.1-0.86	0.0-0.5
Eosinophils (x10^9/L)	0.0-0.2	0.0-0.2	0.0-0.2	0.0-0.3	0.0-0.15
Basophils (x10^9/L)	0-0.45	0.17-0.83	0.0-0.45	0.0-0.6	0.0-0.15
Thrombocytes (x10^9/L)	5-40	12-35	12-25	6-46	—
Fibrinogen (g/L)	<4	<4	<3.5	<4.2	<4

Table 40.22 | Hematological Reference Values of Selected Species of Raptors

Assay	Saker falcon[84] (*Falco cherrug*)	Peregrine falcon[13] (*Falco peregrinus*)
	n=25	n=48
Hb (g/dl)	15.93±0.38 (13.3-21.2)	14.82±1.32 (11.6-19.1)
RBC (x10^{12}/L)	2.65±0.08 (2.05-3.90)	3.49±0.21 (2.76-4.05)
PCV (L/L)	0.47±0.59 (0.42-0.53)	0.40±0.38 (0.26-0.58)
MCV (fl)	183.16±3.84 (135.8-219.5)	117.51±7.70 (100.8-176.0)
MCH (pg)	60.74±1.42 (50.62-78.94)	—
MCHC (g/dl)	33.28±0.63 (28.33-40.0)	—
WBC (x10^9/L)	5.7± 0.31 (2.8-8.4)	12.56±3.06 (7.6-21.2)
Heterophils (x10^9/L)	4.14±0.24 (2.18-5.96)	4.52±1.2 (1.38-7.53)
Lymphocytes (x10^9/L)	1.33±0.09 (0.52-2.29)	5.52±1.36 (1.75-7.53)
Monocytes (x10^9/L)	0.21±0.03 (0.04-0.64)	0.25±0.03 (0.12-0.62)
Eosinophils (x10^9/L)	0	2.3±0.9 (1.0-4.77)
Basophils (x10^9/L)	0.08±0.01 (0-0.32)	—
Thrombocytes (x10^9/L)	0.41±0.03 (0.17-0.76)	2.97±1.2 (1.25-7.15)
Fibrinogen (g/L)	2.82±0.14 (1.78-4.7)	—

Saker falcon: Results expressed as mean ± standard error of mean (minimum and maximum in parentheses).
Peregrine falcon: Results expressed as mean ± standard deviation (minimum and maximum in parentheses).

Table 40.23 | Hematological Reference Values of Selected Species of Raptors[27]

Assay	Turkey vulture (*Cathartes aura*)	Egyptian vulture (*Neophron percnopterus*)	Buzzard (*Buteo buteo*)	Golden eagle (*Aquila chrysaetos*)	Caracara (*Polyborus plancus*)	Secretary bird (*Sagittarius serpentarius*)
	n=10	n=4	n=6	n=4	n=9	n=4
Hb (g/dl)	16.3 (15.7-17.3)	14.8 (13.3-16.5)	12.9 (11.6-14.6)	13.8 (12.1-15.2)	16.2 (13.1-20.6)	16.7 (15.2-18.6)
RBC ($x10^{12}$/L)	2.7 (2.4-2.9)	2.3 (1.9-2.6)	2.4 (2.2-2.7)	2.4 (1.9-2.7)	2.8 (2.5-3.3)	2.18 (2.0-2.3)
PCV (L/L)	0.54 (0.51-0.58)	0.43 (0.5-0.46)	0.38 (0.34-0.42)	0.41 (0.35-0.47)	0.46 (0.38-0.59)	0.46 (0.42-0.50)
MCV (fl)	204 (194-224)	190 (183-206)	159 (151-171)	174 (160-184)	165 (149-173)	208 (201-216)
MCH (pg)	61.7 (58.6-65.0)	67.7 (65.2-72.9)	53.8 (48.8-57.5)	58.9 (56.3-62.7)	57.8 (51.6-62.4)	76.7 (73.4-80.8)
MCHC (g/dl)	30.2 (28.6-32.0)	35.2 (35.0-35.5)	33.9 (31.4-36.0)	34 (32.3-35.9)	35.2 (34-36)	36.9 (35.3-5.6)
WBC ($x10^9$/L)	20.1 (10.5-31.9)	7.6 (4.7-10.6)	9.1 (4.6-13.9)	13.1 (11.7-14.7)	6.8 (3.3-11.6)	8.1 (6.8-10.0)
Heterophils ($x10^9$/L)	11.8 (6.7-19.8)	4 (1.2-5.5)	5.5 (2.3-8.8)	10.4 (9.5-12.7)	4.2 (0.6-5.9)	5.3 (3-9)
Lymphocytes ($x10^9$/L)	3.3 (0.8-5.6)	2.5 (1.5-3.4)	1.7 (1.1-2.4)	2.2 (1.6-3.2)	2.4 (0.9-5.6)	2.4 (0.8-4.2)
Monocytes ($x10^9$/L)	(0.0-0.4)	(0.0-0.4)	0	0	(0.0-0.6)	(0.0-0.4)
Eosinophils ($x10^9$/L)	(1.5-7.5)	(0.3-1.4)	(0.1-3.1)	(0.2-0.6)	(0.0-0.3)	(0.0-0.2)
Basophils ($x10^9$/L)	(0.0-2.3)	0	(0.0-0.6)	(0.0-0.2)	(0.0-0.3)	(0.0-0.4)
Thrombocytes ($x10^9$/L)	14 (7-22)	13 (6-15)	27 (18-36)	14 (4-21)	27 (18-35)	9 (7-10)
Fibrinogen (g/L)	—	1.6 (1.0-1.9)	2.3 (1.3-3.3)	2.9 (2.0-4.1)	2.4 (1.2-3.8)	2.7 (2.0-3.3)

Table 40.24 | Hematological Reference Values of Selected Species of Raptors[27]

Assay	Barn owl (*Tyto alba*)	European eagle owl (*Bubo bubo*)	African eagle owl (*Bubo africanus*)	Spectacled owl (*Pulsatrix perspicillata*)	Tawny owl (*Strix aluco*)	Boobook owl (*Ninox boobook*)
	n=10	n=14	n=6	n=4	n=14	n=5
Hb (g/dl)	14.2±1.5 (12.7-16.4)	14.2±1.5 (11.7-16.8)	15.8 (13.9-19.9)	14.2 (12.4-16.3)	14.6±1.1 (12.9-16.4)	15.1 (14.4-15.9)
RBC ($x10^{12}$/L)	2.7±0.3 (2.2-3.0)	1.9±0.2 (1.4-2.3)	2.4 (2.1-2.8)	1.6 (1.4-1.8)	2.5±0.2 (2.0-2.9)	2.5 (2.4-2.9)
PCV (L/L)	0.46±0.03 (0.42-0.51)	0.39±0.04 (0.31-0.45)	0.45 (0.41-0.53)	0.42 (0.5-0.45)	0.40±0.03 (0.36-0.47)	0.42 (0.40-0.45)
MCV (fl)	176±22 (145-216)	207±17 (178-239)	189 (171-214)	261 (245-267)	158±9 (147-177)	172 (165-175)
MCH (pg)	51.1±5.7 (44.9-60.7)	75.1±8.1 (67.1-87.1)	66.4 (58.9-76.2)	87.8 (86.1-89.1)	56.8±4.8 (49.8-66.6)	61.5 (60.8-61.6)
MCHC (g/dl)	31.8±2.2 (28.9-34.9)	36.3±2.0 (33.8-38.4)	35.1 (33.9-36.6)	33.7 (32.3-36.2)	36.3±0.09 (34.9-38.0)	36 (34.8-5.3)
WBC ($x10^9$/L)	16.6±4.2 (11.5-22.3)	10.8±4.0 (5.3-18.6)	6.2 (4.7-8.0)	9.6 (6.9-11.1)	6.7±3.3 (2.4-11.8)	6.4 (3.7-11.2)
Heterophils ($x10^9$/L)	8.9±3.0 (5.2-12.5)	6.9±3.2 (2.6-11.8)	3 (1.3-5.2)	4.9 (2.8-7.6)	3.4±2.0 (1.1-7.2)	4.6 (2.3-9.1)
Lymphocytes ($x10^9$/L)	5.0±1.7 (2.5-7.5)	3.8±1.9 (1.9-6.7)	2.3 (1.9-3.2)	4.3 (2.7-7.3)	3.3±1.4 (0.9-5.1)	1.4 (0.9-1.7)
Monocytes ($x10^9$/L)	(0.0-1.0)	0	0	0	(0.0-0.3)	(0.0-0.5)
Eosinophils ($x10^9$/L)	(0.0-2.5)	(0.0-1.6)	(0.0-1.0)	(0.0-0.6)	(0.0-1.9)	(0.0-0.5)
Basophils ($x10^9$/L)	(0.0-0.9)	(0.0-0.6)	(0.0-0.6)	(0.0-0.4)	(0.0-0.9)	(0.0-0.2)
Thrombocytes ($x10^9$/L)	33±15 (14-58)	15±3 (9-17)	22 (14-29)	18	17±5 (10-24)	—
Fibrinogen (g/L)	2.7±0.5 (1.9-3.3)	3.3±0.9 (1.4-5.0)	5.2 (3.6-7.7)	7 (6.4-8.8)	3.6±0.7 (2.6-5.3)	2.8 (1.6-3.8)

Table 40.25 | Blood Chemistry Reference Values of Selected Species of Raptors[73]

Assay	Gyrfalcon (*Falco rusticolus*)	Great horned owl (*Bubo virginianus*)	Red shouldered hawk (*Buteo lineatus*)	Peregrine falcon (*Falco peregrinus*)	Bald eagle (*Haliaeetus leucocephalus*)
RBC (x10^{12}/L)	3.21	1.7	2.4	3.96	2.46
HCT (%)	50.5	32.0	40.9	60.0	37.9
Hb (g/dl)	18.6	12.2	15.0	23.5	14.6
MCV (fl)	157.0	188.0	171.0	152.0	154.0
MCH (pg)	58.1	71.5	62.6	59.2	59.2
MCHC (g/dl)	36.9	38.0	36.7	39.1	38.4

Automated Hemogram-typical Erythrocytic Data

Table 40.26 | Blood Chemistry Reference Values of Selected Species of Raptors[73]

Assay	Lanner falcon[35] (*Falco biarmicus*)	Gyrfalcon[1] (*Falco rusticolus*)	Peregrine falcon[1,35] (*Falco peregrinus*)	Saker falcon[35,83] (*Falco cherrug*)	Merlin[35] (*Falco columbarius*)
	n=26	n=12	n=55, 14	n=38	n=39
Total protein (g/L)	33-42	28.9	25-40	27-36	27.5-39
Albumin (g/L)	9.6-16	7.3	8.3-12.5	9-12.3	8.6-16.1
Globulin (g/L)	21.2-28.8	—	16-28	18-28	17.2-25
A:G ratio	0.44-0.57	—	0.4-0.55	0.45-0.57	0.47-0.58
Urea (mmol/L)	1.3-2.7	—	0.9-2.8	0.5-2.6	—
Creatinine (µmol/L)	5-75	—	41-91	23-75	16-50
Uric acid (µmol/L)	318-709	828.56	326-675	320-785	174-800
Bile acids (µmol/L)	—	—	20-118	20-90	—
ALT (SGPT) (u/L)	—	—	15-51	36-55	—
ALP (u/L)	180-510	257	97-350	285-450	54-310
GGT (u/L)	—	—	0-7	0.8-5.9	—
AST (SGOT) (u/L)	30-118	97	50-105	45-95	50-125
CK (u/L)	350-650	402	357-850	355-651	521-807
LDH (u/L)	434-897	—	625-1210	551-765	320-630
Glucose (mmol/L)	11-15	17.65	11-16	12-14	9-12
Cholesterol (mmol/L)	3-8.8	—	3.9-10.5	4.5-8.6	3-7.8
Inorg phosphate (mmol/L)	0.68-2	—	0.77-2.1	0.72-2.16	0.95-1.79
Calcium (mmol/L)	2.07-2.45	2.4	2.1-2.56	2.15-2.61	2-2.45
Sodium (mmol/L)	152-164	160	153-164	154-161	155-170
Potassium (mmol/L)	1.0-2.1	1.99	0.9-1.7	0.8-2.3	1.0-1.8
Chloride (mmol/L)	—	125	117-127	114-125	—
Amylase (u/L)	—	—	—	—	—
Bilirubin (µmol/L)	—	—	78.15	8.55-27.53	—
Blood urea nitrogen (mmol/L)	—	3.33	2.32	—	—
Phosphorus (mg/dl)	—	3.57	3.35	—	—
Triglycerides (mmol/L)	—	—	—	0.79-1.25	—

Table 40.27 | Blood Chemistry Reference Values of Selected Species of Raptors[27]

Blood Chemistry Assay	Red-tailed hawk[1,33,41] *(Buteo jamaicensis)*	Harris' hawk[33,35] *(Parabuteo unicinctus)*	Northern goshawk[35] *(Accipiter gentilis)*	Golden eagle[33] *(Aquila chrysaetos)*
	n=10	n=17	n=24	n=7
Total protein (g/L)	33-45	31-45.7	26.3-42.0	25-39
Albumin (g/L)	9-12	13.9-17	8.8-12.4	10-14
Globulin (g/L)	—	21-29.4	18.0-29.2	—
A:G ratio	—	0.46-0.55	0.4-0.57	—
Urea (mmol/L)	—	0.7-1.9	—	—
Creatinine (μmol/L)	44.2-106.08	20-59	41-94	53.04-106.08
Uric acid (μmol/L)	446.1-1058.74 mmol/L	535-785	511-854	261.71-713.76 mmol/L
Bile acids (μmol/L)	—	—	—	—
ALT (SGPT) (u/L)	31	—	—	—
ALP (u/L)	45-90	20-96	15.6-87.5	15-36
GGT (u/L)	—	2.0-6.9	3.0-7.6	—
AST (SGOT) (u/L)	113-180	160-348	176-409	95-210
CK (u/L)	1124	224-650	218-775	—
LDH (u/L)	470-775	160-563	120-906	320-690
Glucose (mmol/L)	17.32-22.87	12.2-15.7	11.5-15.9	13.88-22.65
Cholesterol (mmol/L)	2.59-3.88	6.6-13.1	4.0-11.5	2.59-4.91
Inorg phosphate (mmol/L)	—	0.8-2.14	0.8-1.97	—
Calcium (mmol/L)	2.1-2.5	2.1-2.66	2.15-2.69	1.85-2.38
Sodium (mmol/L)	157	155-171	—	—
Potassium (mmol/L)	2.6-4.3	0.8-2.3	—	—
Chloride (mmol/L)	125	113-119	—	—
Amylase (u/L)	—	—	—	—
Bilirubin (μmol/L)	8.55-10.26	8.55-20.52	—	5.13-8.55
Blood urea nitrogen (BUN) (mmol/L)	3.33	—	—	—
Phosphorus (mg/dl)	1.8-4.1	3.0-4.4	—	1.9-3.6
Triglycerides (mmol/L)	—	—	—	—

Table 40.28 | Blood Chemistry Reference Values of Selected Species of Raptors

Blood Chemistry Assay	Bald eagle[1,33] *(Haliaeetus leucocephalus)*	Tawny eagle[35] *(Aquila rapax)*	Northern eagle owl[35] *(Bubo bubo)*	Great-horned owl[1] *(Bubo virginianus)*
	n=8	n=13	n=20	n=10
Total protein (g/L)	30-41	29-41.4	30.1-34.5	43.3
Albumin (g/L)	8-16	11.5-18.0	11.1-13.5	12.7
Globulin (g/L)	—	25.3-28.4	18.7-22.4	—
A:G ratio	—	0.44-0.55	—	—
Urea (mmol/L)	—	0.8-2.7	0.9-2.9	—
Creatinine (μmol/L)	35.36-88.4	31-59	31-49	—
Uric acid (μmol/L)	327.14-880.30 mmol/L	413-576	475-832	814.88 mmol/L
Bile acids (μmol/L)	—	—	—	—
ALT (SGPT) (u/L)	25	—	—	39
ALP (u/L)	23-30	17.1-69.7	—	31
GGT (u/L)	—	1.0-2.7	—	—
AST (SGOT) (u/L)	153-370	124-226	—	287
CK (u/L)	383	—	—	977
LDH (u/L)	250-580	211-369	—	—
Glucose (mmol/L)	15.82-22.20	10.2-14.5	13.5-21.7	19.76
Cholesterol (mmol/L)	3.88-6.26	7.9-10.7	3.9-7.1	—
Inorg phosphate (mmol/L)	—	1.2-1.78	1.15-1.94	—
Calcium (mmol/L)	2.05-2.65	2.21-2.66	2.16-2.61	2.55
Sodium (mmol/L)	156	153-157	—	156
Potassium (mmol/L)	3	1.5-3.1	—	2.8
Chloride (mmol/L)	120	114-123	—	122
Amylase (u/L)	1158	—	—	—
Bilirubin (μmol/L)	3.42-8.55	—	—	1.2
Blood urea nitrogen (BUN) (mmol/L)	2.21	—	—	3.57
Phosphorus (mg/dl)	3.03	—	—	4.34
Triglycerides (mmol/L)	2.4-3.2	—	—	—

Table 40.29 | Body Weight of Selected Species of Raptors

Name	Scientific Name	Male (g)	Female (g)	General (g)
Turkey vulture	*Cathartes aura*	—	—	850-2000
American black vulture	*Coragyps atratus*	—	—	1100-1900
King vulture	*Sarcoramphus papa*	—	—	3000-3750
California condor	*Gymnogyps californianus*	—	—	8000-14000
Andean condor	*Vultur gryphus*	11000-15000	8000-11000	—
Osprey	*Pandion haliaetus*	1200-1600	1600-2000	—
Long-tailed buzzard	*Henicopernis longicauda*	447-630	570-730	—
American swallow-tailed kite	*Elanoides forficatus*	—	—	375
Mississippi kite	*Ictinia mississippiensis*	—	—	220-390
Plumbeous kite	*Ictinia plumbea*	190-267	232-280	—
Red kite	*Milvus milvus*	—	—	757-1221
Black kite	*Milvus migrans*	—	—	567-941
Brahminy kite	*Haliastur indus*	—	—	320-673
White-bellied sea-eagle	*Haliaeetus leucogaster*	2120-2900	2900-3400	—
African fish-eagle	*Haliaeetus vocifer*	1986-2497	3170-3630	—
Pallas's fish-eagle	*Haliaeetus leucoryphus*	—	—	2040-3700
White-tailed sea-eagle	*Haliaeetus albicilla*	4100	5500	—
Bald eagle	*Haliaeetus leucocephalus*	—	—	3000-6300
Steller's sea-eagle	*Haliaeetus pelagicus*	—	—	4900-9000
Palm-nut vulture	*Gypohierax angolensis*	—	—	1361-1712
Bearded vulture	*Gypaetus barbatus*	—	—	4500-7100
Egyptian vulture	*Neophron percnopterus*	—	—	1600-2200
Hooded vulture	*Necrosyrtes monachus*	—	—	1530-2600
African white-backed vulture	*Gyps africanus*	—	—	4150-7200
Indian white-backed vulture	*Gyps bengalensis*	—	—	3500-6000
Long-billed vulture	*Gyps indicus*	—	—	5500-6300
Rüppell's griffon	*Gyps rueppellii*	—	—	6800-9000
Rufous-breasted sparrow hawk	*Accipiter rufiventris*	—	—	180-210
Sharp-shinned hawk	*Accipiter striatus*	82-125	144-208	—
Cooper's hawk	*Accipiter cooperii*	235-300	413-598	—
Black sparrow hawk	*Accipiter melanoleucus*	430-490	650-980	—
Northern goshawk	*Accipiter gentilis*	517-1170	820-1509	—
Red goshawk	*Erythrotriorchis radiatus*	630-640	1110-1370	—
Common black hawk	*Buteogallus anthracinus*	793	1199	—
Swainson's hawk	*Buteo swainsoni*	683-936	937-1367	—
White-tailed hawk	*Buteo albicaudatus*	—	—	850-884
Red-tailed hawk	*Buteo jamaicensis*	690-1300	900-1460	—
Eurasian buzzard	*Buteo buteo*	525-1183	625-1364	—
Long-legged buzzard	*Buteo rufinus*	590-1281	945-1760	—
Ferruginous hawk	*Buteo regalis*	1050	1230	980-2030
Rough-legged buzzard	*Buteo lagopus*	600-1377	783-1660	—
Harpy eagle	*Harpia harpyja*	4000-4800	7600-9000	—
Great Philippine eagle	*Pithecophaga jefferyi*	—	—	4700-8000
Lesser spotted eagle	*Aquila pomarina*	—	—	1100-2000
Greater spotted eagle	*Aquila clanga*	—	—	1500-2500
Tawny eagle	*Aquila rapax*	—	—	1696-3100
Steppe eagle	*Aquila nipalensis*	—	—	2400-3900
Spanish imperial eagle	*Aquila adalberti*	—	—	2500-3500
Eastern imperial eagle	*Aquila heliaca*	—	—	2450-4530
Wahlberg's eagle	*Aquila wahlbergi*	—	—	437-1400
Golden eagle	*Aquila chrysaetos*	2840-4550	3630-6665	—
Wedge-tailed eagle	*Aquila audax*	2025-4000	3180-5300	—
Verreaux's eagle	*Aquila verreauxii*	3000-4150	3100-5800	—
Bonelli's eagle	*Hieraaetus fasciatus*	—	—	1600-2400
African hawk-eagle	*Hieraaetus spilogaster*	1150-1300	1444-1750	—
Booted eagle	*Hieraaetus pennatus*	709	975	—
Little eagle	*Hieraaetus morphnoides*	530-810	745-1250	—
Martial eagle	*Polemaetus bellicosus*	—	—	3012-6200

Table 40.30 | Body Weight of Selected Species of Raptors (continued)

Name	Scientific Name	Male (g)	Female (g)	General (g)
Long-crested eagle	*Lophaetus occipitalis*	912-1363	1367-1523	—
Ornate hawk-eagle	*Spizaetus ornatus*	1000	1450	—
Secretary bird	*Sagittarius serpentarius*	—	—	2300-4270
Crested caracara	*Polyborus plancus*	834	953	1150-1600 (Chile & Peru)
Laughing falcon	*Herpetotheres cachinnans*	567-686	626-800	—
African pygmy-falcon	*Polihierax semitorquatus*	—	—	54-67
Lesser kestrel	*Falco naumanni*	90-172	138-208	—
Common kestrel	*Falco tinnunculus*	136-252	154-314	—
Mauritius kestrel	*Falco punctatus*	178	231	—
Seychelles kestrel	*Falco araea*	73	87	—
American kestrel	*Falco sparverius*	80-143	84-165	—
Red-necked falcon	*Falco chicquera*	139-178	190-305	—
Red-footed falcon	*Falco vespertinus*	—	—	130-197
Amur falcon	*Falco amurensis*	97-155	111-188	—
Eleonora's falcon	*Falco eleonorae*	350	388	—
Aplomado falcon	*Falco femoralis*	—	—	261
Merlin	*Falco columbarius*	150-210	189-255	—
Eurasian hobby	*Falco subbuteo*	131-232	141-340	—
African hobby	*Falco cuvierii*	125-178	186-224	—
New Zealand falcon	*Falco novaeseelandiae*	252-500	420-594	—
Black falcon	*Falco subniger*	510-710	610-1000	—
Lanner falcon	*Falco biarmicus*	500-600	700-900	—
Lagger falcon	*Falco jugger*	—	—	525-850
Saker falcon	*Falco cherrug*	730-990	970-1300	—
Gyrfalcon	*Falco rusticolus*	961-1321	1262-2100	—
Prairie falcon	*Falco mexicanus*	500-650	700-975	—
Peregrine falcon	*Falco peregrinus*	—	—	550-1500
Taita falcon	*Falco fasciinucha*	212	306	—
Greater sooty-owl	*Tyto tenebricosa*	500-700	875-1150	—
Lesser sooty-owl	*Tyto multipunctata*	430-450	540	—
Common barn owl	*Tyto alba*	555 (Malaysia) 227-418 (Australia)	612 (Malaysia) 220-475 (Australia)	187-455 (Europe, N. Africa), 266-470 (S. Africa), 400-700 (N. America), 387-558 (Surinam), 264 (Galapagos Is.)
White-fronted scops-owl	*Otus sagittatus*	—	—	120
Reddish scops-owl	*Otus rufescens*	—	—	77
Sandy scops-owl	*Otus icterorhynchus*	69-80	61-80	—
African scops-owl	*Otus senegalensis*	—	—	45-123
Eurasian scops-owl	*Otus scops*	—	—	60-135
Western screech-owl	*Otus kennicottii*	131-210	157-250	—
Eastern screech-owl	*Otus asio*	166	194	—
Vermiculated screech-owl	*Otus vermiculatus*	—	—	100-110
Great horned owl	*Bubo virginianus*	680-1450	1000-2500	—
Eurasian eagle-owl	*Bubo bubo*	1500-2800	1750-4200	—
Spotted eagle-owl	*Bubo africanus*	490-620	640-850	—
Brown fish-owl	*Ketupa zeylonensis*	—	—	1105
Snowy owl	*Nyctea scandiaca*	700-2500	780-2950	—
Vermiculated fishing-owl	*Scotopelia bouvieri*	975	975	—
Spotted wood-owl	*Strix seloputo*	1011	—	—
Brown wood-owl	*Strix leptogrammica*	—	—	500-700
Tawny owl	*Strix aluco*	440	553	—
Hume's owl	*Strix butleri*	—	—	214-220
Spotted owl	*Strix occidentalis*	520-700	550-760	—
Barred owl	*Strix varia*	630	800	—
Rusty-barred owl	*Strix hylophila*	285-340	345-395	—
Chaco owl	*Strix chacoensis*	360-435	420-500	—

Table 40.31 | Body Weight of Selected Species of Raptors (continued)

Name	Scientific Name	Male (g)	Female (g)	General (g)
Ural owl	*Strix uralensis*	500-950	570-1300	—
Great grey owl	*Strix nebulosa*	800-1175	925-1700	—
African wood-owl	*Strix woodfordii*	240-270	285-350	—
Spectacled owl	*Pulsatrix perspicillata*	—	—	590-980
				1050-1250 (Pulsatrix)
Northern hawk-owl	*Surnia ulula*	270-314	320-345	—
Eurasian pygmy-owl	*Glaucidium passerinum*	50-65	67-77	—
Collared owlet	*Glaucidium brodiei*	53	63	—
Pearl-spotted owlet	*Glaucidium perlatum*	36-86	61-147	—
Northern pygmy-owl	*Glaucidium californicum*	62	73	—
Amazonian pygmy-owl	*Glaucidium hardyi*	—	—	55-65
Red-chested owlet	*Glaucidium tephronotum*	80-95	75-103	—
Elf owl	*Micrathene whitneyi*	—	—	41
Little owl	*Athene noctua*	162-177	166-206	—
Spotted owlet	*Athene brama*	—	—	115
Burrowing owl	*Athene cunicularia*	130-185	120-250	—
Boreal owl	*Aegolius funereus*	90-115	120-195	—
Rufous owl	*Ninox rufa*	1200	980	—
Southern boobook	*Ninox boobook*	250	315	—
Morepork	*Ninox novaeseelandiae*	156	170	—
Short-eared owl	*Asio flammeus*	200-450	280-500	—
Marsh owl	*Asio capensis*	—	—	225-375

Note: See literature on body weights presented above[10,11] and on body weight of the vermiculated fishing owl (*Scotopelia bouvieri*).[75]

Acknowledgments

The author extends thanks to HRH Prince Fahad bin Sultan bin Abdulaziz Al Saud for his support to the advancement of falcon medicine and to Mr. Basil Al Abbasi, Director General, for his continuing interest in the clinical and research program of the Falcon Specialist Hospital and Research Institute; to Shinto K. John for compiling the reference tables; to Generoso Quiambao for preparing the photographic material; and to Dr. Jesus Naldo for proofreading the manuscript and for providing useful comments.

Products Mentioned in the Text

a. Vetrap, 3M Health Care, St. Paul, USA
b. Tegaderm, 3M Health Care, St. Paul, USA
c. Granuflex (DuoDERM), ConvaTec Ltd, Deeside, UK
d. OpSite, Smith & Nephew Medical Ltd, Hull, UK
e. Melolin, Smith & Nephew Medical Ltd, Hull, UK
f. Nu Gauze Sponges, Johnson and Johnson, New Brunswick, USA
g. Hexcelite, Hexcel Corp, Dublin, CA, USA
h. Biotin, Arnolds Veterinary Products Ltd, Shrewsbury, UK
i. Steinmann intramedullary pins, positive profile threaded pins, Kirschner wires, fixator bar and clamps, Veterinary Thermoplastic, Imex Veterinary Inc, Longview TX, USA
j. Sam Splint, Moore Medical Corp, New Britain, CT, USA
k. Technovit, Jorgensen Laboratories Inc, Loveland, CO, USA
l. KY Jelly, Johnson & Johnson Medical Ltd, Gargrave, Skipton, UK
m. Spark, Vetafarm, Wagga Wagga, Australia
n. F10, Health and Hygiene (Pty), Ltd, Sunninghill, South Africa, www.healthandhygine.co.za/products_select.asp
o. Falcon Insect Liquidator, Vetafarm, Wagga, Wagga, Australia
p. Wormout, Imox, Vetafarm, Wagga Wagga, Australia, www.vetafarm.com.au
q. Spartrix, Harkers Ltd, Bury St. Edmunds, Suffolk, UK
r. Metronidazole Tablets, Regent-GM Laboratories, Ltd, London, UK
s. Baycox, Bayer Plc, Bury St. Edmunds, Suffolk, UK, www.baycox.com
t. Arlen, Chloroquine phosphate, Primaquine phosphate, Winthrop Pharmaceuticals, New York, New York, USA
u. Sporanox, Itraconazole 10 mg/ml, Janssen-Cilag Ltd, High Wycombe, Buckinghamshire, UK
v. Fungizone, Amphotericin BP 50 mg, ER Suinn & Sons Ltd, Hounslow, UK
w. Imaverol, Enilconazole 100mg/ml, Janssen Animal Health, High Wycombe, Buckinghamshire, UK
x. Daktarin Oral Gel, Janssen-Cilag Ltd, High Wycombe, Buckinghamshire, UK
y. Marbocyl, Vétoquinol UK Ltd, Bicester, Oxon, UK
z. Metacam, Boehringer Ingelheim, Ingelheim/Rhein, Germany
aa. Vitamin B1 Injection, Bimeda UK, Knowsley Industrial Park North, Liverpool, UK
bb. Benerva, Roche Products Ltd, Welwyn Garden City, UK
cc. Duphafral Multivitamin 9, Fort Dodge Animal Health Ltd, Southhampton, UK
dd. Multivet, Soluvet, Vetafarm, Wagga Wagga, Australia
ee. Optrex, Crookes Healthcare Ltd, Nottingham, UK
ff. Genticin eye drops, Roche Products Ltd. Welwyn Garden City, UK
gg. Betsolan eye and eardrops, Pitman-Moore Ltd, Crewe, Cheshire, UK
hh. LeadCare Blood Lead Testing System, ESA Inc, Chelmsford, MA, USA
ii. Sodium Calcium Edetate, Animalcare, Dunnington, UK
jj. Pif Paf, Reckitt Benckiser Arabia FZE, Dubai, UAE

References and Suggested Reading

1. Altman RB, et al (eds): Appendix I, Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997.
2. Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, p 1020.
3. Arent LR, Martell M: Care and Management of Captive Raptors. St. Paul, The Raptor Center, University of Minnesota, 1996.
4. Bailey T, Sullivan T: Aerosol therapy in birds using a novel disinfectant. Exotic DVM 3(4):17, 2001.
5. Baronetzky-Mercier A, Seidel B: *In* Göltenboth R, Klös HG: Krankheiten der Zoo und Wildtiere. Berlin, Blackwell Wissenschafts-Verlag, 1995, pp 443-465.
6. Bates G, Tucker RL, Ford S, Mattix ME: Thyroid adenocarcinoma in a bald eagle (*Haliaeetus leucocephalus*). J Zoo Wild Med 30(3):439-442, 1999.
7. Beynon PH, Forbes NA, Harcourt-Brown NH: Appendix - Formularies. *In* Beynon PH, Forbes NA, Harcourt-Brown NH: Manual of Raptors, Pigeons and Waterfowl. Cheltenham, UK, British Small Animal Vet Assoc Ltd, 1996, p 339.
8. Burke HF, Swaim SF, Amalsadvala T: Review of wound management in raptors. J Avian Med Surg 16(3):180-191, 2002.
9. Cooper JE (ed): Birds of Prey, Health and Diseases. Oxford, Blackwell Science Ltd, 2002.
10. del Hoyo J, Elliot A, Sargatal J (eds): Handbook of the Birds of the World. Lynx Edicions Vol 2, 1994.
11. del Hoyo J, Elliot A, Sargatal J (eds): Handbook of the Birds of the World. Lynx Edicions Vol 5, 1999.
12. Dorrestein GM: Bacteriology. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 255-280.
13. Dötlinger HS, Bird DM: Hematological parameters in captive peregrine falcons (*Falco peregrinus*). Falco Newsletter No 4. The Middle East Falcon Research Group, National Avian Research Center, United Arab Emirates, 1995.
14. Erdélyi K, Tenk M, Dén Á: Mycoplosmosis associated perosis type skeletal deformity in a saker falcon nestling in Hungary. J Wild Dis 35(3):586-590, 1999.
15. Forbes NA: Epidemic and endemic chlamydiosis in a collection of raptors. *In* Samour JH (ed): Proceedings of the Specialist Workshop. Middle East Falcon Research Group, Abu Dhabi, United Arab Emirates, 1995, pp 3-8.
16. Forbes NA: Chronic weight loss, vomiting and dysphagia. *In* Beynon PH, Forbes NA, Harcourt-Brown NH: Manual of Raptors, Pigeons and Waterfowl. Cheltenham, UK, British Small Animal Vet Assoc Ltd, 1996, pp 189-196.
17. Forbes NA: Respiratory problems. *In* Beynon PH, Forbes NA, Harcourt-Brown NH: Manual of Raptors, Pigeons and Waterfowl. Cheltenham, UK, British Small Animal Vet Assoc Ltd, 1996, pp 180-188.
18. Forbes NA, Rees Davies R: Practical raptor nutrition. Proc Assoc Avian Vet, 2000, pp 165-167.
19. Forbes NA, Simpson GN: *Caryospora neofalconis*: An emerging threat to captive-bred raptors in the United Kingdom. J Avian Med Surg 11(2):110-114, 1997.
20. Forbes NA, Cooper JE, Higgins RJ: Neoplasms of birds of prey. *In* Lumeij JT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network Inc, 2000, pp 127-145.
21. Gerbermann H, Korbel R: Findings in free-living raptors infected with *Chlamydia*. VIII DVG-Tagung über Vogelkrankheiten. München, 1992, pp 105-124.
22. Gerbermann H, Jakoby JR, Kösters J: Incidence of *Chlamydia* in an aviary station of birds of prey. J Vet Med 5:739-748, 1990.
23. Gerlach H: Bacteria. *In* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine: Principles and Application. Lake Worth, FL, Wingers Pub Inc,1994, pp 949-983.
24. Gough RE, Capua I, Wernery U: Herpesvirus infection in raptors. *In* Lumeij JT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network Inc, 2000, pp 9-11.
25. Harcourt-Brown NH: Foot and leg problems. *In* Beynon PH, Forbes NA, Harcourt-Brown NH: Manual of Raptors, Pigeons and Waterfowl. Cheltenham, UK, British Small Animal Vet Assoc Ltd, 1996, pp 147-168.
26. Harcourt-Brown NH: Bumblefoot. *In* Samour JH (ed): Avian Medicine. London, Harcourt Publishers Ltd, 2000, pp 126-131.
27. Hawkey C, Samour JH: The value of clinical hematology in exotic birds. *In* Jacobson ER, Kollias GV Jr (eds): Contemporary Issues in Small Animal Practice. London, Churchill Livingstone, 1988.
28. Hawkins MG, et al: Atypical parasitic migration and necrotizing sacral myelitis due to *Serratospiculoides amaculata* in a prairie falcon (*Falco mexicanus*). Avian Dis 45:276-283, 2001.
29. Heindenreich M: Birds of Prey, Medicine and Management, Oxford, Blackwell Science Ltd, 1997.
30. Hoefer HL: Diseases of the gastrointestinal tract. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 419-453.
31. Huckabee JR: Raptor therapeutics. Vet Clin N Am Exot An Pract 3(1):91-116, 2000.
32. Hunter DB, Rohner C, Currie DC: Mortality in fledgling great horned owls from black fly *Hematophaga* and *Leucocytozoon*. J Wild Dis 33(3):486-491, 1997.
33. Ivins GK, Weddle GD, Halliwell WH: Hematology and serum chemistries in birds of prey: *In* Fowler ME (ed): Zoo and Wildlife Medicine 2nd edition. Philadelphia, WB Saunders, 1986, pp 286-290.
34. Jennings IB: Haematology. *In* Beynon PH, Forbes NA, Harcourt-Brown NH (eds): Manual of Raptors, Pigeons and Waterfowl. Cheltenham, UK, British Small Animal Vet Assoc Ltd, 1996, p 75.
35. Jennings IV: Appendix 8.1 Haematology and clinical chemistry values. *In* Beynon PH, Forbes NA, Harcourt-Brown NH (eds): Manual of Raptors, Pigeons and Waterfowl. Cheltenham, UK, British Small Animal Vet Assoc Ltd, 1996, pp 75-78.
36. Johne R, Müeller H: Avian polyomavirus in wild birds: Genome analysis of isolates from Falconiformes and Psittaciformes. Arch Virol 143:1501-1512, 1998.
37. Kaaden OR, Riddle KE, Wernery U: *In* Samour JH (ed): Falcon pox in the Middle East. Proc Specialist Workshop, Middle East Falcon Research Group, Abu Dhabi, United Arab Emirates, 1995, pp 21-23.
38. Kellner SJ: Eye and eyelid injuries: *In* Samour JH (ed): Avian Medicine. London, Harcourt Publishers Ltd, 2000, pp 120-123.
39. Kirkwood J: Metabolic bone disease, twisting and bending deformities of the long bones in growing birds, slipped tendon. *In* Samour JH (ed): Avian Medicine. London, Harcourt Publishers Ltd, 2000, pp 170-176.
40. Klueh PN: Untersuchungen zur therapie und prophylaxe der *Caryospora* infektionen der falken mit toltrazuril sowie die beschreibung von zwei neuen Caryospora-Arten (*C. megafalconis* n.sp. und *C. boeri* n.sp.). Vet Med Dis, Hannover, 1994.
41. Kollias GV, McLeish J: Effects of ketamine hydrochloride in red-tailed hawks (*Buteo jamaicensis*), biochemical and hematology. Comp Biochem Physiol (c), 1978, p 60, p 211.
42. Krone O, Cooper JE: Parasitic diseases. *In* Cooper JE (ed): Birds of Prey, Health and Diseases. Oxford, Blackwell Science Ltd, 2002, pp 105-120.
43. Krone O, et al: Hemosporidia of birds of prey and owls from Germany. Acta Protozool 40:281-289, 2001.
44. Lacina D, Bird DM: Endoparasites of raptors: A review and an update. *In* Lumeij JT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network Inc, 2000, pp 65-100.
45. Lierz M: Imping feathers in birds of prey. Exotic DVM 1.6:13-15, 2000.
46. Lierz M: Evaluation of the dosage of ivermectin in falcons. Vet Rec 148:596-600, 2001.
47. Lierz M, Remple D: Endoparasitenbefall bei Großfalken in Dubai. Kleintierpraxis 42:757-768, 1997.
48. Lierz M, et al: Mycoplasmas in captive falcons in the United Arab Emirates. Proc Assoc Avian Vet, 2000, pp 485-486.
49. Lierz M, et al: Isolation of *Mycoplasma meleagridis* from the free-ranging birds of prey in Germany. J Vet Med Series B: Infectious Diseases and Veterinary Public Health 47:63-67, 2000.
50. Lierz M, et al: Detection of *Mycoplasm* spp in raptorial birds in Germany. *In* Lumeij JT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network Inc, 2000, pp 25-34.
51. Magnino S, Fabbi M, Moreno A: Avian influenza virus (H7 serotype) in a saker falcon in Italy. Vet Rec 146:740, 2000.
52. Malley AD, Whitbread TJ: The integument. *In* Beynon PH, Forbes NA, Harcourt-Brown NH: Manual of Raptors, Pigeons and Waterfowl. Cheltenham, UK. British Small Animal Vet Assoc Ltd, 1996, pp 129-139.
53. Manwell RJ, et al: Newcastle disease (Avian PMV 1) viruses in raptors. *In* Lumeij JT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network Inc, 2000, pp 3-8.
54. Mirande LA, Howerth EW, Poston RP: Chlamydiosis in a red-tailed hawk (*Buteo jamaicensis*) J Wildl Dis 28(2):284-287, 1992.
55. Morishita TY, et al. Lesions associated with *Pasteurella multocida* infection in raptors. Avian Dis 41:203-213.
56. Muñoz E, Molina R, Ferrer D: *Babesia shortti* infection in a common kestrel (*Falco tinnunculus*) in Catalonia (northeastern Spain). Avian Pathol 28:207-209, 1999.
57. Oglesbee BL: Mycotic diseases. *In* Altman RB, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 323-331.
58. Park F, Gill J: Use of 360 degree conjunctival flaps in a peregrine falcon. Proc Assoc Avian Vet, Australia, 2000, pp 163-170.
59. Peirce MA: Hematozoa: *In* Samour JH (ed): Avian Medicine. London, Harcourt Publishers Ltd, 2000, pp 245-252.
60. Reavill D: Bacterial diseases. *In* Rosskopf W, Woerpel R (eds): Diseases of Cage and Aviary Birds 3rd ed. Baltimore, MD, Williams & Wilkins, 1996, pp 596-612.
61. Redig PT: Nursing avian patients. *In* Beynon PH, Forbes NA, Harcourt-Brown NH: Manual of Raptors, Pigeons and Waterfowl. Cheltenham, UK. British Small Animal Vet Assoc Ltd, 1996, pp 42-46.
62. Redig PT: Fractures. *In* Samour JH (ed): Avian Medicine. London, Harcourt Publishers Ltd, 2000, pp 131-165.
63. Redig PT: Fungal diseases: Aspergillosis: *In* Samour JH (ed): Avian Medicine. London, Harcourt Publishers Ltd, 2000, pp 275-287.
64. Redig PT: Effective method for management of avian fractures and other orthopedic problems. Proc Eur Assoc Avian Vet, 2001, pp 26-42.
65. Redig P, Ackermann J: Raptors. *In* Tully TN, Lawton M, Dorrestein GM (eds): Avian Medicine. Oxford, Butterworth-Heinemann, 2000.
66. Remple JD: Raptor bumblefoot: A new treatment technique. *In* Redig PT, et al (eds): Raptor Biomedicine. Minneapolis, University of Minnesota Press, 1993, pp 154-160.

67. Remple JD: Falcon herpesvirus and immune considerations for the production of a vaccine. *In* Samour JH (ed): Proc Specialist Workshop. Middle East Falcon Research Group, Abu Dhabi, United Arab Emirates, 1995, pp 9-20.
68. Remple JD: Considerations on the production of a safe and efficacious falcon herpesvirus vaccine. *In* Lumeij JT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network Inc, 2000, pp 13-23.
69. Remple JD, Forbes NA: Antibiotic-impregnated polymethylmethacrylate beads in the treatment of bumblefoot in raptors. *In* Lumeij JT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network Inc, 2000, pp 255-263.
70. Riddle KE, Hoolihan J: Form-fitting, composite-casting method for avian appendages. *In* Redig PT, et al (eds): Raptor Biomedicine. Minneapolis, University of Minnesota Press, 1993, pp 161-164.
71. Ritchie BW: Avian Viruses: Function and Control. Lake Worth, FL, Wingers Publishing Inc, 1995, pp 253-311.
72. Rodriguez-Quiroz J: Review of an intramedullary pin/external skeletal fixator tie-in technique for repair of bone fractures in birds. Exotic DVM 3(4):14-16, 2001.
73. Rosskopf W, Woerpel R (eds): Diseases of Cage and Aviary Birds 3rd ed. Maryland, Williams & Wilkins, 1996.
74. Samour JH: Falcons, falconry and veterinary medicine in the Middle East. Proc Avian Vet Assoc, 1996, pp 233-239.
75. Samour JH (ed): Avian Medicine. London, Harcourt Publishers Ltd, 2000.
76. Samour JH: Clinical examination. *In* Samour JH (ed): Avian Medicine. London, Harcourt Publishers Ltd, 2000, pp 15-27.
77. Samour JH: Endoscopy. *In* Samour JH (ed): Avian Medicine. London, Harcourt Publishers Ltd, 2000, pp 60-72.
78. Samour JH: Imping. *In* Samour JH (ed): Avian Medicine. London, Harcourt Publishers Ltd, 2000, pp 108-111.
79. Samour JH: Supraorbital trichomoniasis infection in two saker falcons (*Falco cherrug*). Vet Rec 146:139-140, 2000.
80. Samour JH: *Pseudomonas aeruginosa* stomatitis as a sequel to trichomoniasis in captive saker falcons (*Falco cherrug*). J Avian Med Surg 14(2):113-117, 2000.
81. Samour JH: Making a safe intranasal flushing device. Exotic DVM 2(4):16, 2000.
82. Samour JH, Cooper JE: Avian pox in birds of prey (Order Falconiformes) in Bahrain. Vet Rec 132:343-345, 1993.
83. Samour JH, D'Aloia M-A: Normal blood chemistry of saker falcon (*Falco cherrug*). Avian Pathol 25:175-178, 1996.
84. Samour JH, D'Aloia M-A, Howlett JC: Normal haematology of captive saker falcons (*Falco cherrug*). Comp Hematol Intl 6:50-52, 1996.
85. Samour JH, Naldo J: Serratospiculiasis in captive falcons in the Middle East: A review. J Avian Med Surg 15(1):2-9, 2001.
86. Samour JH, Naldo J: Therapeutic management of pox lesions on the cere of captive falcons. Proc Assoc Avian Vet, 2001, pp 233-236.
87. Samour JH, Naldo J: Diagnosis and therapeutic management of lead toxicosis in falcons in Saudi Arabia, J Avian Med Surg 16(1):16-2o, 2002.
88. Samour JH, Naldo JL: Diagnosis and therapeutic management of candidiasis in falcons in Saudi Arabia. J Avian Med Surg 16(2):129-132, 2002.
89. Samour JH, Naldo JL, Kutty IM: Exotic DVM. Forthcoming.
90. Samour JH, et al: A thyroid cystadenocarcinoma in a saker falcon (*Falco cherrug*). Vet Rec 149:277-278, 2001.
91. Samour JH, Peirce M: *Babesia shortti* infection in a saker falcon (*Falco cherrug altaicus*). Vet Rec 139:167-168, 1996.
92. Samour JH, Silvanose C: Parasitological findings in captive falcons in the United Arab Emirates. *In* Lumeij JT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network Inc, 2000, pp 117-124.
93. Samour JH, Bailey TA, Cooper JE: Trichomoniasis in birds of prey (Falconiformes) in Bahrain. Vet Rec 136:358-362, 1995.
94. Samour JH, Bailey TA, Keymer IF: The use of ammonium chloride in falconry in the Middle East. Vet Rec 137:269-270, 1995.
95. Scope A, Frey H: Diseases and mortality causes in captive and free-ranging bearded vultures (*Gypaetus barbatus*). *In* Lumeij JT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network Inc, 2000, pp 157-162.
96. Smith SA: Diagnosis and treatment of helminths in birds of prey. *In* Redig PT, et al (eds): Raptor Biomedicine. University of Minnesota Press. Minnesota, 1993, pp 21-27.
97. Smith SA: Parasites of birds of prey: Their diagnosis and treatment. Semi Avian Exotic Pet Med 5(2):97-105, 1996.
98. Steele KE, et al: Pathology of fatal West Nile virus infections in native and exotic birds during the 1999 outbreak in New York City, New York. Vet Pathol 5:208-224, 1999.
99. Swanson JR, Veatch J, Trieb DK: CNS symptoms in a tucuman Amazon (*Amazona tucumana*) due to amyloidosis in the brain. Proc Assoc Avian Vet, 1994, pp 275-277.
100. Ueblacker SN: Trichomoniasis in American kestrels (*Falco sparverius*) and eastern screech owls (*Otus asio*): *In* Lumeij JT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network Inc, 2000, pp 59-64.
101. Valentin A, Haberkorn A, Jakob W: Massive malaria-infektion mit *Parahaemoproteus* sp. in schenee-eulen (*Nyctea scandiaca*) und deren behandlung mit primaquin. Verhanlungshericht des 36 internationalen symposiums über die erkrankungen der zoo und wiltiere 36:401-404, 1994.
102. Verwoerd D: Observations of the use of azithromycin in falcons. Falco 16:19-20, 2000.
103. Verwoerd D: Aerosol use of a novel disinfectant as part of an integrated approach to aspergillosis in falcons in the UAE. Falco 17:15-18, 2001.
104. Wernery U: Viral diseases. *In* Samour JH (ed): Avian Medicine. London, Harcourt Publishers Ltd, 2000, pp 264-275.
105. Wernery U, et al: Production of a falcon herpesvirus vaccine. Berliner und Münchener Tierärztliche Wochenschrift 112(9):339-344, 1999.
106. Wernery U, et al: Avian paramyxovirus serotype 1 (Newcastle disease virus) infections in falcons. J Vet Med 39:153-158, 1992.
107. Wernery U. et al: Newcastle disease in captive falcons. *In* Samour JH (ed): Proc Specialist Workshop. Middle East Falcon Research Group, Abu Dhabi, United Arab Emirates, 1995, pp 24-5.
108. Wernery U, et al: Clostridium enterotoxemia in Falconiformes in the United Arab Emirates. *In* Lumeij JT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network Inc, 2000, pp 35-41.
109. Zucca P: Arthropods, protozoa, helminths. *In* Samour JH (ed): Avian Medicine. London, Harcourt Publishers Ltd, 2000, pp 219-225.

CHAPTER

41

Management of Captive Ratites

BOB DONELEY, BVSc, FACVSc (Avian Health)

Auckland Zoo

Fig 41.1 | North Island brown kiwi.

Although the demand for veterinary input into the ostrich and emu industries is not as great today as it was in the 1990s, veterinarians are still called on to give advice and to treat ratites of all descriptions. This chapter seeks to present new and updated information on these flightless birds as well as refer the reader to references where more comprehensive information may be found.

Classification

Ratites are not an order or family of birds. Rather, they are a group of birds sharing similar physical characteristics. They are all flightless birds, with elongated necks and relatively long legs adapted for walking and running (Fig 41.1, Table 41.1). The name ratite comes from the Latin *ratis*, or raft. This refers to the flattened, raft-like sternum that lacks a keel and ventral musculature — an adaptation to their loss of flight. Ratites are naturally found on three continents (Africa, Australia and South America) and two islands (New Guinea and New Zealand), all in the Southern Hemisphere. Nevertheless, commercial development of the ostrich and emu industry is seen on every continent except Antarctica.

Table 41.1 | Classification of Ratites.
There are 11 species of birds grouped as ratites. They come from four orders and five families.

Common Name	Order	Family	Species	Subspecies
Ostrich	Struthioniformes	Struthionidae	*Struthio camelus*	*S.c. camelus* (North African) *S.c. molybdophanes* (Somali) *S.c. massaicus* (Massai) *S.c. australis* (South African)
Emu	Casuariiformes	Dromaiidae	*Dromiceius novaehollandiae*	—
Southern/double-wattled cassowary	Casuariiformes	Casuariidae	*Casuarius casuarius*	—
Bennett's/little cassowary	Casuariiformes	Casuariidae	*C. bennetti*	—
Northern/single-wattled cassowary	Casuariiformes	Casuariidae	*C. unappendiculatus*	—
Greater rhea	Rheiformes	Rheidae	*Rhea americana*	—
Darwin or lesser rhea	Rheiformes	Rheidae	*R. pennata* (formerly *Pterocnemia pennata*)	—
Tokoeka kiwi	Apterygiformes	Apterygidae	*Apteryx australis*	—
Brown kiwi	Apterygiformes	Apterygidae	*A. mantelli*	—
Little spotted kiwi	Apterygiformes	Apterygidae	*A. oweni*	—
Great spotted kiwi	Apterygiformes	Apterygidae	*A. haasti*	—
Rowi kiwi	Apterygiformes	Apterygidae	*A. rowi*	—

Some classifications place the tinamous (Order Tinamiformes; Family Tinamidae), a South American partridge-like bird, in the ratite group. They share some physical characteristics with the ratite groups described above, especially the rhea.

Clinical Anatomy and Physiology

Ratite anatomy and physiology are similar to other avian species, particularly poultry and psittacines, although there are some clinically significant differences.[3,31,34,63]

INTEGUMENT

The skin of ratites is thicker than that of many other avian species; consequently, ostrich and emu hides are in demand by the leather industry. Rough handling easily damages the skin, and scarring or bruising detracts from the hide's value. Care must be taken when handling these birds to avoid such damage. Kiwi skin is particularly thick and includes a prominent hypodermis for fat storage (Fig 41.2). Sternal callosities (dermal thickening for weight bearing when in sternal recumbency) often are present over the sternum of ostriches, emus and rheas. A similar condition also frequently is present over the ventral-cranial portion of the pubic bone in the ostrich. The plantar surface of the foot has a dermal pad of packed vertical rods of cornified tissue, with underlying paired, tubular fat bodies for additional padding.

The toenails are blunted in ostriches, but are sharper in other species, especially cassowaries, which can have 12-cm spikes on the medial toes. Prior to the breeding season, male North Island brown kiwis can store as much as 50% of their body mass as subcutaneous and intracoelomic fat. The feathers of ratites lack the barbules seen in other species, and so the feather vane does not interlock tightly. This gives ratite feathers a unique hair-like appearance, but also makes them less water-resistant. Contrary to earlier work, the presence of filoplumes has been reported in ostriches.[6] These slender feathers

Auckland Zoo

Fig 41.2 | Dissection showing massive deposits of subcutaneous and intracoelomic fat in a male North Island brown kiwi early in the breeding season (September). Ironically, the bird died after being caught in a leg-hold trap set for its mammalian predators.

arise from small follicles alongside the larger follicles of contour feathers. The presence of a large number of filoplumes (and therefore their follicles) can detract from the value of a hide. There may be a genetic factor for these filoplumes, and their presence should be considered a selection criterion for breeding birds.[6]

Thermoregulation in ostriches is achieved by evaporative cooling from the respiratory tract and convection heat loss through the skin. The thighs of the ostrich, normally covered by the wings, are bare of feathers. Contour feathers on the body can be erected or flattened voluntarily. A heat-stressed ostrich will hold its wings away from the body and erect its contour feathers, thus maximizing heat loss from the body (Fig 41.3). Conversely, in

Fig 41.3 | Heat stress in an ostrich chick. Note the raised feathers and the wings held away from the body, thus maximizing heat loss. In these conditions, appetite can be reduced by as much as 40%, with resultant stunting and mortality.

Auckland Zoo

Fig 41.4 | North Island brown kiwi chick showing vestigial wing and axillary apteria (featherless skin), which is the site used to implant subcutaneous identity microchips.

cold weather, the ostrich will flatten its feathers and wings against the body, conserving body heat. It may also lie down on its sternum, further reducing the area of skin available for convective heat loss.

MUSCULOSKELETAL SYSTEM

With the loss of flight, ratites have lost the need for pneumatized bones. Consequently, with the exception of a pneumatized femur in the ostrich and emu, ratite bones are heavier and denser than those of other avian species. In the ostrich, the thoracic girdle (coracoid, clavicle and scapula) has fused, a further adaptation to the loss of flight. Ostriches and rheas have relatively large wings, while the wings of other ratites are comparatively much smaller (Fig 41.4). Ostriches have two toes (digits 3 and 4), the rest have three (digits 2, 3 and 4) all forward, and all toes have four phalanges on each digit.

In the ostrich, emu and rhea, the muscles of economic importance are found on the pelvic limb and along the lumbar vertebrae. This makes these muscles unsuitable for intramuscular injections, as abscesses or scar formation could lead to downgrading or condemnation of the carcass. In all ratites, the reliance on the legs for locomotion means that particular care must be taken that intramuscular injections do not result in pain and lameness. The lack of pectoral muscles on the ventral sternum leaves the muscles alongside the thoracic vertebrae as the area most suitable for injections. Note that care must be taken to minimize hide damage through repeated injections.

DIGESTIVE SYSTEM

Ratites are primarily herbivorous (although cassowaries are known to eat small mammals), and their digestive tracts are basically similar to those of other herbivorous birds.[31] The exception is the kiwi, which is primarily insectivorous.[18] Its anatomic and physiologic differences reflect the evolutionary influences of the forages available in their natural habitat.

The esophagus lies on the right side of the neck. It contains numerous longitudinal rugae that allow expansion when a bolus of food is swallowed. The ostrich cock, when displaying territorial behavior or seeking to attract a mate, inflates the esophagus with air and then expels it with a loud "boom". There is no crop in any of the ratites, and the esophagus opens directly into the proventriculus. There is no esophageal/proventricular sphincter; therefore, regurgitation of proventricular contents can be of concern during anesthesia.

In the ostrich, the large, thin-walled proventriculus lies caudal to the ventriculus. In other ratites, the proventriculus is cranial to the ventriculus. The proventriculus of the rhea is small, while those of the kiwi, emu and cassowary are intermediate to those of the ostrich and the rhea. The ventriculus in the ostrich is separated from the proventriculus by a large opening that facilitates the surgical removal of ventricular foreign bodies via a proventriculotomy.

Ratites lack cellulase needed to digest plant fiber and therefore rely on fermentation of the fiber in the intestinal tract. This fermentation requires a slow rate of passage through the tract and an area where microbes can colonize without being swept away.[16] This is where major anatomic differences exist among ratites.

Ostriches and rheas have a comparatively longer rectum and larger ceca than the emu and cassowary, perhaps indicating their development as hindgut fermenters of relatively poor-quality fodder. The ventriculus of the cassowary lacks a koilin lining. The emu and cassowary,

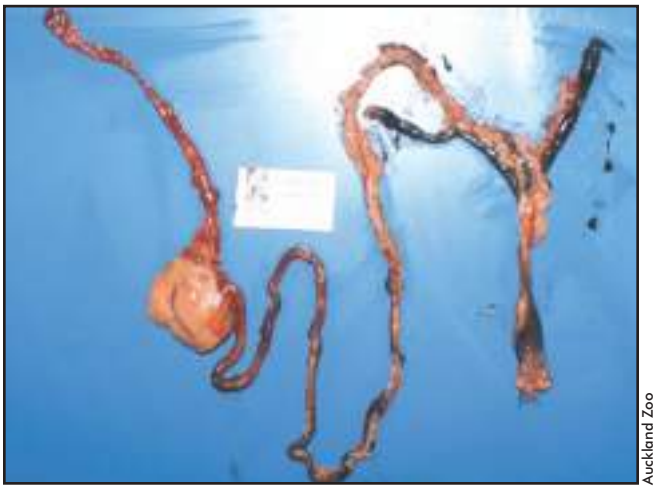

Auckland Zoo

Fig 41.5 | Dissected normal gastrointestinal tract from a kiwi showing paired ceca.

Table 41.2 | Gastrointestinal Transit Times in Ratites

Species	Transit time
Ostrich (immature)	36 hours
Ostrich (mature)	48 hours
Emu	5-6 hours
Cassowary	—
Rhea	—
Kiwi	5-20 hours

evolving on a comparatively higher quality diet, have much longer small intestines, but small, non-functional ceca and a relatively shorter rectum. Overall, the intestinal tracts of the emu and cassowary are relatively shorter than those of the rhea and ostrich. The kiwi has long, paired ceca and a very short rectum (Fig 41.5). There is no gall bladder in the ostrich or emu, while the cassowary has one. Gut transit times vary with age and species (Table 41.2). For example, the emu has the capability of retaining larger fibrous particles in the ileum, allowing for greater fermentation.

RESPIRATORY SYSTEM

Despite their adaptations to a terrestrial lifestyle, ratites have retained a respiratory tract similar in structure and function to that of other avian species. Unlike other birds, however, the sternum of the ratite does not move during respiration. It is fixed to enable it to support the weight of the bird when resting. Respiration, therefore, relies on the lateral movement of the ribs, a fact that should be considered during anesthesia and recovery.[63]

Like other avian species, the tracheal rings of most ratites are complete cartilaginous rings. The exception is the emu, where a longitudinal cleft 6 to 8 cm long is found on the ventral surface of the trachea 10 to 15 cm cranial to the thoracic inlet. A membrane covers this cleft. As the emu matures, the cleft develops into an expandable pouch, which can be inflated to produce a booming noise in hens and a growling noise in cocks.[31] This can be a complication during anesthesia if positive pressure ventilation is used, as gases may be directed into this pouch and away from the respiratory system. This can be overcome by wrapping the base of the neck with a non-adhesive bandage during anesthesia.[31,63]

The syrinx in ratites is poorly developed compared to other birds, a reflection on their lack of vocalization. Although the anatomy of the air sacs is similar to that of other birds, their capacity is much reduced.[63]

The respiratory rate varies according to age and ambient temperature. Ostrich chicks have respiratory rates of 12 to 60 breaths per minute, compared to the adult resting rate of 6 to 12 per minute. In hot conditions, this adult rate can increase to 40 to 60 breaths per minute.[31,34]

REPRODUCTIVE SYSTEM

Like other birds, most ratite hens develop only the left ovary and oviduct. The exception is the kiwi, which has two functional ovaries and oviducts.[14] The oviduct(s) open into the urodeum. In the ostrich, it is possible to pass a guarded culture swab into the first few centimeters of the caudal oviduct through this opening from the urodeum. This opening can be found within a small mound on the left side of the urodeum, in the 10 o'clock position. On the ventral floor of the proctodeum is a small genital mound. In the ostrich, a small (1 to 3 cm) phallic-like structure arises from this mound. An even smaller structure is present in the emu, but only the mound itself is present in the cassowary and rhea.

Ratite cocks have a phallus. This differs from the mammalian penis in that there is no urethra within it, and it plays no role in urination. Ostrich and kiwi phalluses are solid structures, unlike those of the emu, cassowary and rhea, whose phalluses are spiral-shaped with a cavity and a partially inverted sleeve that everts during erection. This gives the appearance of a urethra, but it is a blind-ended structure.[31] In all ratites, the non-erect phallus is found on the floor of the proctodeum. During erection, the phallus becomes engorged and is everted from the cloaca. Semen is directed along a dorsal phallic sulcus into the hen's cloaca.

The sex of ratites can usually be determined by palpation or examination of the cloaca (Table 41.3). In small chicks, the cloaca can be everted by digital pressure and visually examined. In older chicks and adults, one or more fingers can be inserted into the proctodeum and the ventral floor palpated to detect the presence or absence of a phallus (Fig 41.6). It must be noted that in

Table 41.3 | Sex Differentiation of Ratites[31]
In the much smaller kiwi, cloacal palpation is difficult, and the length of the bill and tarsus (both being longer in the larger female) generally determines sex. This difference becomes evident at approximately 6 months of age.

Species	Juvenile, Male	Juvenile, Female	Adult, Male	Adult, Female
Ostrich	Phallus round in cross section, 1-4 cm long, dorsal sulcus	Phallic-like structure flattened in cross section, 0.5-1.0 cm long, no sulcus	Phallus 20-40 cm, curved	Phallic-like structure small, 1-4 cm long, straight
Emu	Hollow tube 0.5-1.0 cm, spirals as bird gets older	Genital mound, slight prominence on genital mound	Spiralled phallus, hollow tube, 3-12 cm long	Genital mound, slight prominence on genital mound
Rhea	Similar to emu, but more elongated	Genital mound with no prominence	Similar to emu	Genital mound with no prominence
Cassowary	Phallus 0.5-1.0 cm, triangular in cross-section	Small genital mound	Triangular-shaped phallus pointed caudally	Genital mound
Kiwi	—	—	Triangular-shaped phallus pointed caudally	—

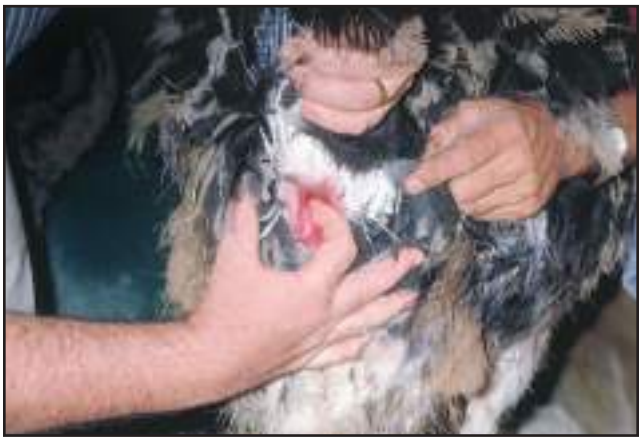


Fig 41.6 | Cloacal sexing in an ostrich to detect the presence of a phallus.

some individual juveniles, this method is inaccurate because of variations in phallic size, and the clinician must be prepared to repeat the procedure when the bird is older. Visually sexing a ratite by its size or plumage color, while often accurate, can be misleading. In the ostrich, for example, a lack of estrogen gives the cock bird its striking black plumage. However, some infertile hens have a similar lack of estrogen and have the same black plumage as cocks. These so-called "black hens" have been the subject of several lawsuits when sold as cocks without cloacal palpation to confirm sex.

The ratite cock has paired intra-abdominal testes that increase dramatically in size with the onset of the breeding season (Table 41.4). Outside of this breeding season, the testes return to their quiescent size and semen production ceases. Semen collection for assessment of fertility is unsuccessful at this time and should not be attempted.[26]

Ratites are seasonal breeders, although some individuals will breed year round. Ostriches, kiwis and rheas start to breed as day length increases, while emus breed as the day length starts to shorten. Cassowaries breed in late winter-early spring, coinciding with the peak availability of fruit in their natural habitat. Ostrich hens often will start

Table 41.4 | Sexual Maturity in Ratites
The age of onset of sexual maturity varies among species, sex, and differing nutritional planes and management systems.

Species	Age of Sexual Maturity
Ostrich	24–36 months
Emu	20–24 months
Rhea	18–24 months
Cassowary	42–48 months
Kiwi	42–54 months

to lay before the cock has come into peak sexual activity, so the first few eggs in each season often are infertile.

URINARY SYSTEM

Ratites have a urinary system similar to that of other birds and, like other birds, produce copious amounts of dilute urine that is resorbed and concentrated in the cloaca and caudal rectum. Ostriches are capable of expelling urine without defecating, unlike most other birds. They are unable to urinate except when standing, and an ostrich that has been recumbent for some time will have a cloaca distended with urine.[34]

Nutrition

In the last few years, the interest in farming some of the ratites has dramatically increased our knowledge of their nutritional requirements. However, research into this area is far from complete, and new information and concepts are becoming known each year.[1,8,15,16,18,34,47] Key areas to consider in ratite nutrition are as follows:

- The adaptation to a terrestrial life-style has resulted in nutritional requirements very different from those of flighted birds.
- There are significant differences among ratites in anatomy and physiology and consequently their nutritional requirements.


Greg J. Harrison

Fig 41.7 | Free-ranging emus in Australia.

Fig 41.8 | The cassowary is primarily frugivorous.

- There are significant differences between juvenile and adult ratites (of the same species) in anatomy and physiology and consequently their nutritional requirements.
- There are economic aspects of feeding farmed ratites that must be considered when formulating diets.

The terrestrial lifestyle of ratites does not require the easily digested and rapidly utilized high-energy diets needed by flighted birds. Many ratites in their natural habitat live in semi-arid environments, grazing on bulky, high-fiber, low-quality roughage. Lacking cellulase to digest this fiber, ratites have to rely on fermentation of the fiber in their relatively long intestinal tracts to obtain the full benefit of their diet. Emus (Fig 41.7), with comparatively shorter intestinal tracts and vestigial cecum, are able to selectively retain larger fiber particles in the distal ileum allowing fermentation.[1]

A notable exception is the cassowary (Fig 41.8), living in rain forests and sclerophyll forests of New Guinea and North Queensland, which is primarily frugivorous (fruit eating) although other plants and even small animals are readily eaten. The kiwi feeds in leaf litter, eating 40 to 45% small invertebrates, 40 to 45% earthworms and 10 to 15% plant material.[18]

Juvenile ratites differ markedly from the adults. In the first week of life, the abdomen is dominated by the presence of the large yolk sac. Juvenile ratites, in their first few weeks of life, are nutritionally dependent on the quality of the parental diet and its effect on the yolk composition. The yolk provides fat, fat-soluble vitamins and some protein, and the yolk sac gradually reduces in size as the yolk is utilized. At the same time, the gastrointestinal tract increases in size. By 2 to 3 weeks of age, the yolk sac is usually completely resorbed, and the abdominal viscera are dominated by the proventriculus, cecum and large intestine. Fiber digestibility parallels this same trend. In the ostrich, for example, fiber digestibility at 3 weeks of age is only 6%, increasing to 58% by 17 weeks. Fat digestibility also increases in a similar fashion, except that the 3-week-old chick has a fat digestibility of approximately 44%.[1,28]

With the development of the ratite industry as a commercial enterprise (with meat, oil and leather as its products), economic factors become pivotal in diet selection with the desire to reach a good slaughter weight at an early age. In much of South Africa, for example, ostriches are traditionally slaughtered at 14 months of age. This situation is rapidly changing toward younger age processing. There are some types of ostriches that can attain body weights in excess of 95 kg as early as 7 to 8 months of age (Black, personal communication). However, the economic benefits of being able to slaughter birds months earlier than previously attained must be offset against the disadvantages of this rapid weight gain, namely angular limb deformities and other growth abnormalities.

Ratite nutrition, therefore, requires the development of diets for each species, as well as each life stage.[1]

PRACTICAL FEEDING

Diets for the two main commercial species, the ostrich and emu, have been researched in some detail, but specific information on the other species is lacking. Prestarter and starter diets should be fed ad libitum (Tables 41.5, 41.6). Older birds should be fed 1.0 to 1.5 kg of a formulated diet per day. As the bird matures, the amount of good-quality fiber can be increased and the amount of formulated diet reduced. At 4 to 6 months, fiber should be no more than 33% of the diet; this can be increased to up to 60% in birds weighing more than 95 kg. The weight gains of birds on these diets, while slightly less than that of birds on 100% formulated diet,

Table 41.5 | Diet Recommendations for Emus[1,8,34,47]

Diet	Age (months)	Est. Body Weight (kg)	Crude Protein (%)	Calcium (%)	Fiber (%)	Comments
Starter	0-1	0.5-1.3	17-25	1.1	>4	Feed ad lib.
Grower	1-16	2-40	16.5-23.0	0.9	>4	Can be used as a maintenance diet (see below).
Market	—	—	14	0.7	>4	Higher energy diet fed in the 2-3 months prior to slaughter in order to maximize fat deposition.
Breeder	20-24	40-45	15-25	2.1 (Females only)	>4	Feed 1.0-1.5 kg/bird per day. Intake drops by 30-50% with onset of the breeding season and increases again at the end of the season. This diet is nutrient dense and is fed ad lib from 1 month prior to breeding till 1 month after egg laying ceases.
Maintenance	16+	40-45	12-20	0.9	*	Intake varies with day length, peaking 1-2 months before maximum day length and then dropping as day length decreases.

*6% at 3 weeks then up to ≤60% at 17 weeks on

Table 41.6 | Diet Recommendations for Ostriches[1]

Diet	Age (months)	Est. Body Weight (kg)	Crude Protein (%)	Calcium (%)	Fiber (%)
Pre-starter	0-2	0.8-10.5	25	1.2-1.5	—
Starter	2-4	11-28	21.5	1.2-1.5	>4
Grower	4-6	29-52	17	1.2-1.5	>4
Finisher	6-10	53-90	13.5	0.9-1.0	6
Post-finisher	10-20	91-110	8.5	0.9-1.0	—
Maintenance	Mature	—	8	0.9-1.0	6-60
Breeder ♀	Laying	—	14	2.0-2.5	8-60

are not significantly less. This feeding regime can result in a cost savings of up to 80%.[16]

Emus and Ostriches

See Tables 41.5, 41.6 for practical feeding recommendations for ostriches and emus.

Rheas

Given the anatomic similarities of the digestive tracts of ostriches and rheas, it is reasonable to assume that their nutritional requirements are similar.[1] A survey of free-ranging greater rheas in Argentina showed that cultivated pasture, especially alfalfa, made up 39 to 63% of the diet. Grasses and rye made up the bulk of the balance, with seed and small invertebrates making up only a small portion.[46]

Cassowaries

The short length of the cassowary's intestinal tract and its subsequent rapid gastrointestinal transit time means that cassowaries require a large volume of food—up to 10% of their body weight daily. Most zoos and wildlife parks feed each bird 4 to 5 kg of fruit daily, including tomatoes, bananas, apples, papayas, pears, watermelon, grapes, mangoes, plums, nectarines, cherries, kiwi fruit, figs, cantaloupe, eggplant, sweet potatoes and carrots. This often is supplemented with animal protein (day-old chicks, mice, rats), vitamins and minerals. Chicks are fed a similar diet in smaller proportions. A commercial diet is available in the USA for carnivores.[c]

Kiwis

The Auckland Zoo in New Zealand feeds its captive kiwis a diet consisting of lean ox heart, diced fruit and vegetables, yeast, wheat germ, sunflower oil, calcium carbonate and a vitamin-mineral premix. This is supplemented with earthworms and invertebrates contained in provided leaf litter.[39] Captive kiwi chicks are introduced to worms and other invertebrates from 10 days of age and then rapidly convert to the artificial diet.

Behavior

An understanding of behavior in free-ranging and captive ratites is necessary to decrease stress and maximize production in both farmed birds and zoological specimens. Research has primarily focused on the ostrich.

There are four major behavioral areas of clinical interest: social behavior, feeding behavior, courtship and breeding behavior, and behavior of chicks.

SOCIAL BEHAVIOR

In their natural state and out of the breeding season, ostriches are gregarious animals, coming together in large flocks of varying ages and sexes, particularly around water holes. Within these flocks, however, there does appear to be a family social structure, with each group headed by a dominant male. During the breeding season these large flocks dissipate, with pairs and single birds being commonly seen. Free-ranging ostriches rarely associate or interact with other species, preferring to ignore them.[23]

In captivity, ostriches are generally run together as pairs and trios. Interaction between other ostriches seems to be mainly confined to males pacing the fence alongside each other in a territorial display. Occasionally this behavior can be detrimental to reproductive success, as males spend more time confronting each other than they do mating. However, colony breeding of ostriches is not uncommon, and confrontation does not appear to

be a hindrance to production.

Rheas are not nomadic birds, as they are rarely subjected to the effects of drought. One cock and several hens will reside in a family territory during the breeding season, with other cocks vigorously expelled. However, these territorial disputes disappear outside the breeding season, and flocks of up to 50 birds may form.[40]

Ostriches, emus and rheas in general show little aggression toward each other outside their breeding season.[40]

Cassowaries, on the other hand, tend to be loners, living a solitary existence within an apparently defined territory. When meeting another cassowary or another animal, cassowaries react aggressively, stretching their bodies and rumbling lowly. If this fails to dissuade the intruder, an aggressive attack may follow. For this reason, they should not be housed with other species, and humans bushwalking in their natural territory need to be cautious, particularly during the breeding season.[55]

With the exception of the Stewart Island brown kiwi, where juveniles stay with their parents in a social group for at least their first year, kiwis also are highly territorial, solitary birds.[49]

FEEDING BEHAVIOR

Time-activity budget studies of wild ostriches indicate that they spend approximately 33% of the daylight hours feeding. The amount of time spent feeding was affected by flock size and sex. In smaller flocks more time was spent in vigilant behaviors, watching for predators and rivals. Males spent more time being vigilant than females. As flock size increased, females spent more time feeding while males decreased their feeding time. It is thought that this may be due to increased rivalry among males in larger flocks. This behavior is replicated in captive birds.[23] Captive females appear to have longer feeding bouts due to their higher energy demands associated with egg production. Males, on the other hand, feed for shorter periods due to competitiveness with other males.[56]

Ostriches prefer to eat natural vegetation, apparently preferring it to a formulated diet. Green annual grasses and forbs are preferred, although leaves, flowers and fruit also are consumed. Ostriches also eat stones, presumably to aid in the grinding and digestion of food. Abnormal feeding behaviors, such as the consumption of dirt and branches, are thought to be due to stress.[23,34]

Emus prefer high-quality foodstuffs, grazing on green plant materials, seed heads, ripening fruit, berries and insects.[47] This higher quality diet may be a reflection of their shorter digestive tract and decreased gut transit times, 5 to 6 hours as opposed to 48 hours in ostrich.

Cassowaries feed predominantly on fallen fruit, but will eat almost anything, including dead rats, birds, small reptiles and fungi. As frugivores, they play an important role in the rain forest ecology, spreading undigested seeds throughout their territory. It is recorded that 21 species of rain forest plants require passage through the cassowary's digestive tract to germinate. A cock bird rearing chicks will pick up a food item and drop it in front of the chicks, clapping his beak to draw their attention to the object.[55]

Kiwis are unique among ratites in being strictly nocturnal and having a highly developed sense of smell. The slit-like paired nostrils are situated at the distal tip of the bill and are driven into the soil and leaf litter in search of a wide range of invertebrate prey. These include caterpillars and pupae of moths, larvae and adult beetles (especially chafer beetles), wood lice (slaters), centipedes, millipedes, slugs, snails, cicada nymphs, crickets, wetas, ants and spiders.[49] Berries and seeds of native plants such as hinau (*Elaeocarpus dentatus*) and miro (*Prumnopitys ferruginea*) also form a small but significant part of the diet.[49]

COURTSHIP AND BREEDING BEHAVIOR

Ostriches

Ostriches breed in a defined breeding season, usually during the spring and summer months. Hens will usually display courtship behavior before the cocks and may even lay the first few eggs before being mated. The hen displays in front of an appropriate mate, lowering her head and spreading and fluttering her wings (Fig 41.9). She opens and closes her beak rapidly, making a soft noise as she does so.

Male courtship behavior begins more slowly. He develops a deep red coloration of the beak and featherless skin over the tarsometatarsal area. In some species, the skin of the neck and thighs also develops a red blush. Aggression toward other males increases, and the erect phallus is prolapsed from the cloaca and displayed. Territorial and display behavior such as booming and kantelling increase. The cock drops to his hocks, spreads his wings and swings his head from side to side, striking his back at the end of each swing.

When both birds are ready to mate, the hen will sit down and extend her head and neck along the ground. The cock approaches and sits astride her, just off to the right. The phallus enters the hen's cloaca and mating proceeds with some kantelling behavior displayed. Mating takes approximately 1 minute.

The free-ranging ostrich has a communal nesting system,

Fig 41.9 | A mature ostrich hen shows reproductive behavior by opening her wings and making a soft "cluck".

usually utilized by one cock, one "major" hen, and two "minor" hens.[40] The cock digs a number of shallow scrapes, one of which is chosen by the major hen. Egg laying proceeds in this nest, but the other minor hens may lay in the same nest. The major hen lays approximately 11 eggs; the minor hens may contribute another 15 to 25 eggs. Egg laying occurs in the late afternoon and early evening, during which time both cock and hen attend the nest. The clutch size builds up over a period of up to 30 days, but incubation does not begin until the clutch is complete. Once the major hen starts to incubate, she will selectively remove many of the eggs of the minor hens from the center of the nest, pushing them to the periphery of the nest where they do not develop.[23]

Emus

Emus also are seasonal breeders, laying their eggs in late autumn and winter.[71] Emu hens are dominant to the cock and make a drumming noise during the breeding season. The male responds with a growling noise. Emu males are primarily monogamous, with one cock pairing with a hen for the breeding season.[44] When ready to mate, the hen drops to the ground, extending her cloaca. The cock drops to his hocks behind her and intromission occurs. The cock picks the back of the hen's neck during mating.[33]

Emu hens start their laying by depositing eggs randomly around a pen. When a nest site is chosen, the remaining eggs are laid there and the dispersed eggs are collected by the cock and relocated to the nest site. After 6 to 10 eggs have been laid, the cock will start to incubate the eggs. Although further eggs laid near him are rolled under to join the others, in many cases the hen will stop laying or move on to find another non-incubating mate.[59] At this stage the cock will hardly move, standing only to turn the eggs. His body weight will significantly decrease — a limiting effect on production.[71]

Rheas

Rhea cocks also incubate the eggs, but a hen may seek out more than one mate. Cocks will defend their territory and nest from other males through physical aggression such as shoving and beak grasping.[40] Once the cock has established his territory, he attracts hens with his courtship displays and behavior. He prepares a shallow depression in the ground as a nest, into which multiple hens will lay. Females lay every 2 to 3 days until the nest contains approximately 13 to 30 eggs, at which point the cock will not allow the hens' access to the nest. The eggs are incubated for approximately 35 to 40 days, with the chicks hatching synchronously over 24 to 28 hours.

Cassowaries

Cassowaries breed in late winter/early spring coinciding with the peak availability of fruit. As the breeding season approaches, the hen becomes more tolerant of cocks and eventually breeding pairs form. A pair remains together for several weeks until the hen is ready to lay in a shallow scrape in the rain forest floor. The male displays to the hen by "dancing" around her, inflating his esophagus and emitting a series of low booming noises. The hen reciprocates by standing with her neck upright and her head tucked down, emitting a low rumble while vibrating her neck. When she is ready to mate, the hen sits down; the cock approaches and, if the hen does not move away, he pecks at her neck, squats and mates from behind. The whole process takes 30 minutes.[55] Once the eggs are laid, the hen leaves and takes no further interest in them. She may seek out another cock and repeat the whole ritual. Up to 8 eggs may be laid in a single clutch, although it is rare that this many hatch and survive.[55]

Kiwis

Kiwis live in stable pairs within a territory that may be aggressively protected. They lay only two eggs, one from each ovary, usually 24 days apart.[14] They are usually laid in a burrow and are incubated by the male. (The exception is the Stewart Island brown kiwi, where the female does assist incubating the eggs, possibly due to the colder weather). Eggs are laid between July and February, and lost clutches are not immediately replaced. The male emerges from the burrow each night for a few hours to feed until within a few days of the eggs' hatching. During the incubation period (70-90 days), the male can lose up to 17% of his body weight.[52]

BEHAVIOR OF CHICKS

In the natural state, hatching of ostrich eggs takes place over 2 to 3 days, during which time the chicks remain "brooded" by an adult. When all have hatched, the chicks form crèches of up to 30 individuals, overseen by

Fig 41.10 | Ostrich chicks feeding. Note the colored mat, which attracts the chicks' attention.

a single pair of adults. These crèches often will merge when they meet, although older chicks are not usually accepted into younger groups. When the chicks are approximately 1 year old, they are left by their guardians in compact peer groups to fend for themselves.[23]

Ostrich chicks are naturally gregarious and do not do well if reared in solitude.[23] Lacking parental security and the comfort of their crèche, isolated chicks (both free-ranging and captive-bred) will suffer severe stress and anxiety.[34] Captive-bred chicks imprint on their human caretakers and look to them for parental security. If the caretaker leaves, the chicks become stressed, leading to decreased food intake, gastric stasis and eventual death. This can be overcome by setting a routine for the chicks in their first 6 weeks (better enabling them to deal with stressful situations), and by having a caretaker with the chicks as much as possible.[42]

A time/activity budget study of captive ostrich chick behavior showed that chicks aged 7 to 14 days spent equivalent amounts of time feeding from the floor and walking around their enclosure (27.7% and 23.1%, respectively).[23] Lacking an adequate thermoregulatory capacity, chicks must spend some time under a brooder lamp warming themselves (11.2%). Presumably, free-ranging chicks would fulfill this requirement by brooding under an adult guardian. The balance of their day was spent pecking at objects (10.2%), standing (6.7%), drinking (5.8%) and feeding from a tray (3.5%).

This study would indicate that feeding chicks from a tray or dish is less successful than placing the food directly on the ground (Fig 41.10). As 10% of their time was spent pecking at other objects, it would appear that pecking is a means for ostriches to locate food. As they learn by mimicry, this behavior spreads rapidly through a group of chicks. Another study indicated that chicks prefer the colors green and white, pecking at these colors in preference to other colors.[24]

Aberrant behaviors, such as pecking at non-food items and other chicks, can become highly repetitive and habitual. Pecking at other chicks may be an aggressive behavior but is more likely to be misdirected feeding behavior or a response to stress. Once this behavior becomes habitual, it is difficult to stop. Chicks that peck the most have the slowest growth rates and may even die.[23]

Emus

Emu chicks spend their first week primarily huddled under a heat source. Any activity is done as a group and is usually initiated by older chicks. Feeding as a group is of short duration; as an individual, it is even shorter. As the chicks get older, they become more active, spending more time running, chasing, pecking at objects and feeding. Sudden disturbances will induce clustering behavior, all the chicks coming together in a huddle. Emu chicks, like ostriches, learn by mimicry, usually by watching the behavior of older chicks.[30]

Rheas

Rheas also rear their chicks in crèches, overseen by a single male. Newly hatched chicks are readily adopted into these crèches with good survivability of the new chicks. Smaller chicks adopted into groups of larger chicks have a higher mortality.[41] The precocial chicks, gray with darker stripes, will stay with the male for approximately 6 months, often taking shelter under the wings of the male when he lies down. Juvenile birds may stay together for several years until reaching sexual maturity (Smith, personal communication).

Cassowaries

Cassowary cocks look after their chicks until they are about 9 months old, although chicks of 16 months have been seen accompanying the cock. When the chicks are approaching maturity, the cock chases them away and they establish their own territories.[55]

Kiwis

The kiwi hen, while not sharing the incubation with the cock, remains nearby and spends time with the male and chicks once they are hatched. Chicks are precocial and independent within approximately 14 to 20 days, but remain with or near their parents for up to several years. With the exception of the Stewart Island brown kiwi, kiwi chicks are fully independent after their first 3 weeks of life.[49]

Production Management

With the advent of commercial farming of some ratites species and improved captive breeding of others in

zoological parks, the management of these birds has received much attention in recent years. Decreased production and most disease problems in ratites often can be traced directly to management faults and errors. An understanding of the management principles of ratite production is, therefore, essential when assessing problems with these birds.

With the modernization of the ostrich industry, more research on management of this species has been done than for any other ratite. Unless otherwise stated, the following information refers to ostriches. Care must be taken in extrapolating this to other ratites.

EGGS

Incubation

Artificial incubation of eggs is widely practiced in all ratite species in an endeavor to increase production. Egg characteristics and incubation requirements for ratite species are contained in Table 41.7.

Given healthy, fertile eggs, there are nine parameters for successful artificial incubation: selection of eggs for incubation, egg collection and sanitation, egg storage, incubator temperature, relative humidity, ventilation, turning and positioning of eggs, hygiene and monitoring and record keeping.

Selection of Eggs for Incubation

In the early days of the recent ostrich "boom," every egg was worth a lot of money, and eggs that should not have been incubated were set. The lesson learned was that these eggs rarely hatched, or, if they did, they produced weak chicks. Not only was the labor intensity in maintaining these eggs and chicks not comparable to the results obtained, but they also acted as "disease multipliers", adding greatly to farm problems.

Undersized eggs or those with poor shell quality have a tendency to lose excessive moisture content during incubation, producing weak, dehydrated chicks. Conversely, oversized eggs or those with excessively thick shells do not lose enough moisture, and the resultant chicks are edematous and weak. Neither type of chick survives well.

It is therefore not advisable to set eggs that are heavily contaminated, cracked, undersized (eg, ostrich eggs less than 900 g), oversized (eg, ostrich eggs greater than 1500 g) or that have poor shell quality (eg, too thick or too thin).

Egg Collection and Sanitation

Microbial contamination of eggs can be responsible for significant losses of fertile eggs, with infection rates ranging from 13.4 to 67% of dead-in-shell ostrich eggs. Most isolates are of fecal or soil origin.[22] Clearly, success rates can be improved if this contamination is minimized or avoided.

Table 41.7 | Egg Characteristics and Incubation Requirements of Ratite Species[62,66]

	Ostrich	Emu	Rhea	Cassowary	Kiwi
Eggs per year	40-60	20-40	40-60	3-10	1-6
Egg weight (g)	900-1700	500-700	400-700	500-700	400-450
Incubation period (days)	41-43	48-50	36-41	47-54	70-90
Temperature (° C)	36.0-36.4	35.2-35.5	36.0-37.2	36.1-36.7	35.5-36.5
Relative humidity (%)	22-36	35-50	55-70	55-70	60-65

Kiwi data from Auckland Zoo (unpublished)

Ratites should be provided with clean, dry, well-drained nest sites. Many birds will accept an artificial nest site, so care should be taken to locate it in an easily accessible area that meets the previous requirements. Wherever possible, eggs should be collected several times daily, with the last collection just before sunset. This regime will minimize the contact time the egg has with possible contaminants, which may be drawn through the shell as the egg cools.

Sanitation of eggs remains controversial, but obvious gross contamination should be removed with a soft, dry brush. The decision on whether to then wash the eggs, fumigate them or irradiate them with ultraviolet (UV) light will depend on local experience and available equipment. One study indicated that UV irradiation gave better hatchability results than washing but was only marginally better than doing nothing at all.[65] In this study, washing the eggs in a disinfectant bath resulted in decreased hatchability. If washing is required for badly contaminated eggs, the wash solution should be maintained at 40° C to minimize the solution being drawn through the eggshell pores.

Egg Storage

The realities of modern farming practices mean that ostrich eggs should be set only once or twice weekly. This allows a better allocation of labor and other resources. As embryonic development does not appear to proceed at temperatures under 25° C,[25] it is feasible to store eggs below this temperature for periods not exceeding 7 days without significant effects on hatchability. A common practice is to store the eggs at 15 to 20° C, turning them twice daily for 3 to 7 days. As the storage time increases, early embryonic mortality rises and can reach 100% by 17 days.[22]

Prewarming the eggs prior to storage may increase

hatchability. It has been demonstrated that heating to 36° C for 4 hours prior to less than 6 days' storage at 17° C increased hatchability by 8%.[11] The same study showed that warming stored eggs for 16 hours prior to incubation had no significant effect on hatchability.

Incubator Temperature

Incubation temperatures below those recommended in Table 41.7 may result in slow-developing chicks that hatch late; conversely, temperatures that are too high may lead to premature hatching.

The recommended temperatures may represent a compromise between the higher temperatures required for an early embryo and the lower temperature needed for a late embryo generating its own metabolic heat.[22] Most ratite producers compensate for this metabolic heat by moving late-term embryos to a hatcher operating at 1° to 2° C lower than the incubator. These hatchers also operate at a higher relative humidity, and turning is stopped.

Relative Humidity

As the embryo develops, the egg loses moisture (and, therefore, weight) through the shell. Numerous studies have shown that hatchability is maximized when this weight loss is between 12 and 18%. The amount of moisture loss is dependent on the porosity of the shell and the relative humidity of the air surrounding the egg. As most incubators can only increase the relative humidity of room air (they lack the ability to dehumidify it), it is recommended that they be placed in an air-conditioned room fitted with dehumidifiers.

Most ratite eggs incubated at the recommended relative humidity shown in Table 41.7 will lose 12 to 18% of their weight during incubation. However, variables such as shell porosity, altitude, room relative humidity and incubator characteristics can all affect the degree of weight loss. Regular weighing of eggs allows for adjustment of the relative humidity in order to achieve the desired weight loss.

Ventilation

The embryonic cells utilize oxygen, and carbon dioxide is produced. The embryo exchanges these gases initially across the yolk sac membrane and later across the chorioallantoic membrane. Without an efficient exchange, the embryo will die. The rate of exchange is determined by the porosity of the shell and the gas concentration gradient across the shell. It is, therefore, imperative that the incubator maintain a constant flow of fresh air over the eggs, usually achieved by a fan.[25] Kiwis are an exception and are best incubated in a still-air incubator. At Auckland Zoo, kiwi eggs are cooled outside the incubator for 1 hour per day during the first 55 days of incubation.[66] This simulates egg temperatures recorded in the nest corresponding to the departure of the male for foraging during natural incubation.[19]

Turning and Positioning of Eggs

Failure to turn eggs results in poor development of extra-embryonic membranes, a reduction in the amount of extra-embryonic fluid produced and poor utilization of albumen by the embryo. Turning is, therefore, essential during incubation up to transfer to a hatcher.[22,25] Various degrees and rates of turning have been described, but hatchability increases as frequency increases up to 8 times daily. Beyond this, there is little difference in hatchability. The degree of turning is dependent on the frequency of turning, with low-frequency turning requiring large-degree turns.[22]

Incubating eggs horizontally for 2 weeks and then vertically for the remainder of incubation appears to maximize ostrich egg hatchability.[22]

Kiwis turn their large eggs up to 180 degrees in the early stages of incubation but, due to the imbalance caused by the large air cell, can only rock them from side to side in the later stages.[19] Artificially incubated eggs are turned on a 2-day cycle, 45 degrees at a time. On the first day, the egg is turned clockwise 90 degrees in two stages and then 90 degrees counterclockwise in two stages. On the second day this is reversed. No turning is done after 55 days' incubation.[66]

Hygiene

The buildup of microbial contamination can lead to embryonic and chick deaths through bacterial infection. It is, therefore, essential that strict hygiene measures be in place in the incubator room and that the incubator is regularly cleaned and fumigated. Bacterial colony counts can be used as a means of monitoring the level of hygiene in an incubator.[25]

Monitoring and Record Keeping

Regular assessment of incubating eggs by weighing and candling is essential to detect problems before they become overwhelming. Several advantages of efficient candling practices have been described, and include the early identification and removal of contaminated/infected eggs, early identification of embryonic death at different stages, and the identification of malpositioned eggs.[2] The ability to make these judgments early allows the early removal of non-viable eggs (thus freeing up valuable incubator space), minimizes contamination and assists in the identification of incubator problems.

Accurate records of setting dates, weight loss, expected hatch dates, outcomes, stages of embryonic death and

Figs 41.11a,b | Brooder shed for ostrich chicks allows sufficient room for exercise. Note the heat lamp.

subsequent chick viability are essential for the clinician trying to assess an incubator problem. Without these records, the task is almost impossible, and much time will be lost monitoring production and generating new records. Producers must have the necessity for good record-keeping strongly impressed on them.

CHICKS

Incubation techniques for ratite eggs have improved to a point where it is reasonable to expect 70 to 90% of fertile eggs to hatch. Adult ratites are generally robust animals with few serious health problems. Chick survivability is, therefore, the greatest limitation to successful production. Reported mortality rates of chicks less than 3 months of age range from 10 to 50%, decreasing to 10% between 3 and 6 months, and 5% up to 12 months.[68]

The ratite chick is precocial, ie, independent of its parents, but its early viability is dependent on the input of the hen into the egg. Calcium for bone development and growth initially comes from the eggshell; minerals, protein and vitamins come from the albumen; fat and immunoglobulins come from the yolk. Therefore, parental nutrition is a key factor in chick survivability. Other factors, such as egg size, weight loss during incubation, microbial contamination and genetics also will have an effect.

Ostrich chicks in particular are susceptible to the effects of stress. Stressors such as temperature extremes, inadequate ventilation, overcrowding, inadequate exercise, poor hygiene, social stresses and incorrect nutrition are all detrimental to growth and survivability of chicks.[28] Management of chicks requires that these stressors and others are identified and minimized (Figs 41.11a,b).

While the yolk sac and its contents are important to the chick, the theory that chicks should not be fed for the first few days to encourage absorption of the yolk appears to be incorrect. With the notable exception of kiwis, ratite chicks need to start eating as early as possible to allow the functional development of the gastrointestinal tract.[68] They may need an older chick or a chicken to teach them to eat, and the feeding behavior of chicks should be utilized to encourage them to eat as soon as possible. Kiwi chicks begin to probe for food after 5 to 7 days, but rarely begin feeding until their yolks have been fully absorbed at approximately 10 days of age (Jakob-Hoff, personal communication). Because the nutrition of ratite chicks is significantly different from that of adults, dietary management is essential to achieve optimal growth rates. Growth rates of chicks as well as the incidence of problems related to nutrition (such as angular limb deformities) should be monitored.

Many different chick-rearing systems have been used around the world. Concrete runs, sand, bare ground, mobile sheds, permanent structures, lucerne (alfalfa) pasture have all been utilized under different conditions. The one constant seems to be that what works well in one geographical area does not work nearly as well in other areas. Variables such as genetics, climate, available nutrition and labor cost all have an effect on the suitability of chick-rearing systems.

A unifying concept in farmed ratite chick rearing is that of "all in-all out" — the batching of chicks according to their age and body weight and the maintenance of these groups as they progress through the farm system. The advantages of this system have been described and include tailoring the rearing system to the age of the group, moving the birds less so there is less stress, and minimizing the spread of disease by preventing chick movement in or out of the group.[68] A review showed that "mixed weight" groups grew more slowly than "same weight" groups, indicating that social pressures affected the growth of chicks.[24] Such social pressures may be less pronounced in groups of chicks of similar ages and weights.

Management of chicks must aim to minimize stress and maximize growth rates. Biosecurity, vermin control, farm

traffic flow, quarantine and isolation of sick chicks also are necessary to reduce losses due to disease. The integration of these two concepts is paramount to the success of chick rearing.

BREEDERS

Ostriches appear to be induced breeders, ie, the hen requires the stimulation of the presence of a male in order to lay eggs. This makes tools such as artificial insemination, commonly used in the poultry industry, less useful and more complicated.[17] The use of vasectomized "teaser" birds may be one option, but practicality and economics would require fertile cocks to run with egg-laying hens. This can be done as pairs, trios or colonies.

Colonies of ostriches can range in size from 9 to 150 birds, with males making up 20 to 30% of the flock. Although colony breeding is recommended by some authors[33] on the grounds of economics, fertility and ease of management, others believe that pairs and trios are more productive.[23,34] Additionally, selection of the best breeding stock requires accurate records of breeding success and chick performance/data that cannot be obtained from colony breeding.

The separation of sexes out of the breeding season and their reintroduction at the start of the season can be a powerful stimulus for breeding.[33] This management tool often is overlooked or neglected, usually due to space constraints.

Emus

Emus are primarily monogamous and will pair with only one hen per season. After mating, the cock broods the eggs and takes no further interest in breeding. In this species, artificial insemination may be advantageous.[44] Many Australian farms allow breeding birds to run as a flock and then separate pairs as they form. This allows compatibility to develop and retains some degree of genetic selection.

Cassowaries

Cassowaries, by nature solitary and potentially aggressive, may not tolerate the presence of a mate outside the breeding season. This needs to be assessed on a case-by-case basis. Young birds paired early appear to make the best breeders, as adults newly introduced to each other may take several years to develop a pair bond. As the breeding season approaches, cassowaries should be placed within visual proximity to each other, separated by a fence. When they are seen to be interacting, they can share the same enclosure.[55]

Kiwis

Captive kiwis must be paired carefully. These territorial birds are likely to fight when first introduced and can inflict fatal wounds with their strong feet and claws. Generally, new pairs are established by placing them in adjoining pens so they can hear and smell each other for some weeks to months before an introduction is attempted. Introducing the birds outside the breeding season in a neutral territory and with widely separated feeding stations and roosting boxes can minimize fighting. However, even with these precautions it is important that the introduction and first few nights together be closely supervised, either by video or direct observation. Once the birds have settled down and are eating regularly, a successful pairing can be assumed. Kiwis tend to pair for life, but have been known to form a new pair bond should one of the birds die (Jakob-Hoff, personal communication).

FLOCK HEALTH SCHEMES

With the advent of the commercial phase in ostrich and emu farming, ie, the production of meat and leather as distinct from breeder (investment) birds, the emphasis of veterinary services has moved away from the individual bird to the overall production of the farm. Veterinary input now focuses on areas such as farm design, general management, selection of stock, nutrition, and disease control and prevention.[27] An indication of the effect of various parameters on farm profitability is shown in Table 41.8.

These figures are based on production of a farm in South Africa. The magnitude and ranking of these parameters will vary slightly from farm to farm and from country to country. Obviously, some of these, especially the income per slaughter bird, chick mortality rates, hen productivity and feed cost, can have a major impact on profitability. These parameters are well within the capacity of veterinarians to have an effect through a farm flock health scheme. A well-designed and carefully implemented ostrich flock health scheme is a feasible way of achieving improved performance and profitability.[7]

Before implementing a scheme, the veterinarian first needs to assess the following:

- The farm layout and facility design
- The management practices employed in all aspects of the farm from incubation to breeder management
- The nutritional basis of all rations currently in use
- Complete performance records over the last 2 to 3 years
- Any major problems encountered — past or present
- The health status and quality of the ostriches of all ages

Table 41.8 | Effects on Farm Profitability[7]

Parameter	Increase by	Effect on profitability
Income per slaughter bird	10%	55% increase
Chick mortality	10%	35% decrease
Eggs/hen and hatchability	10%	31% increase
Feed cost	10%	27% decrease
Cost of contract work	10%	10% decrease
Labor cost	10%	3% decrease
Cost of drugs	10%	1% decrease

From this, a written plan can be prepared along the following lines:

- Evaluation of the present layout and performance of the farm
- Issues requiring improvement and their relative priority
- Realistic targets for future performance
- Aspects of a flock health scheme required to attain such targets
- Requirements for ongoing monitoring[7]

This plan must take into consideration the farmer's willingness and ability to participate in such a scheme, his/her aims and goals, and the economic realities of implementing the scheme. The scheme must be cost-effective for the farmer or it will not succeed.[27]

Effective implementation of flock health schemes involving facility design, record keeping, genetic selection programs, preventive health measures, quarantine principles, biosecurity practices, nutritional analysis and monitoring of management practices can improve the efficiency of ostrich farms and increase financial returns to the ostrich producer.[7]

Clinical Examination

DISTANT EXAMINATION AND HISTORY

As with any animal, the examination of a ratite begins with a distant examination and history taking. As most ratites being examined will be farmed birds or zoological specimens, a history of their management, diet, previous medical history and behavior is vital in understanding the problem presented to the clinician.

While taking a history, the clinician is well advised to stand back and watch the bird before capture and restraint takes place. Species' differences in behavior must be understood. For example, it is normal for a cassowary to be isolated and aloof. However, an ostrich that is doing the same may well be ill. Chicks behave differently than adults. Abnormalities in gait, respiration, appetite, posture or feathering often can be best assessed before the bird is stressed by capture. A fecal examination should be conducted if possible. When as much information as possible has been collected, the bird should be restrained in order to be more closely examined.

HANDLING AND RESTRAINT

Not only are many of the larger ratites capable of inflicting serious injury on people, but also their economic value often lies with the condition of their hide and meat. As such, handling and restraint techniques must ensure that no harm befalls either the bird or the people doing the handling. Time spent planning and preparing to work with a ratite is never wasted!

Wherever possible, adult ostriches should be removed from their own enclosure (which they have come to regard as their territory) before an attempt is made to handle them. If time permits, allowing the birds to become accustomed to being fed in a smaller enclosure (eg, a shed or mobile cattle yard) for a few days can make capture simple and safe. Once the birds are comfortable entering the enclosure, merely closing a gate behind them can capture them. If time does not allow this approach, several people working together can herd the birds into a corner of the pen or into a small enclosure. As ostriches are quite adept at running past individual handlers, creating a "mobile fence" by suspending a length of shade cloth between handlers can effectively herd the birds to where they can be captured. This whole procedure must be done calmly and smoothly, with a minimum of running, shouting and arm-waving.

Once confined, individual ostriches can be caught and examined. An ostrich is generally more amenable when it is "hooded", ie, a tubular cloth hood is placed over its head restricting its eyesight. To do this, the neck is grasped just below the head, either by hand or with a shepherd's crook, and the head is pulled down to a horizontal plane (Fig 41.12). Care must be taken not to damage the neck and jugular vein, as fatal hematomas have resulted from trauma to the vein by careless handling. The handler has the hood bunched up over the free hand, which then grasps the beak (Fig 41.13). The hood is then pulled over the head and the head is released. The ostrich is then immediately restrained with a hand on the cranial sternum and another on the pubic bones. Large birds may require two handlers, one on either side of the bird. Under no circumstances should the bird be given the opportunity to run while hooded, as injury invariably results.

Emus

Emus do not tolerate a hood, although Jakob-Hoff (personal communication) has found them useful as long as

Fig 41.12 | Capture and restraint of an ostrich using a "shepherd's crook" and a hood.

Fig 41.13 | Preparing to place a hood on an ostrich.

the hooded bird, can detect no light. Once placed in a small enclosure, approaching an emu from behind and hooking an arm around the base of the neck near the thoracic inlet can restrain it. The bird is pulled back against the handler's body and the other hand is used to grasp the upper neck. The bird is then tilted backward into a more human-related upright position relative to the normal emu body position. Once it has settled, the upper neck can be released.[45]

Cassowaries

Cassowaries are dangerous to handle. Physical restraint can be achieved by herding the bird into a corner. The handlers use padded sheets of plywood to block the bird. They must be braced against the impact of a running or jumping bird, and the handlers must be aware of the cassowary's ability to jump. Once the bird is cornered, it can be approached from behind and pushed firmly to the ground by applying body weight squarely onto its back. Once it is sitting, a second person can restrain the tarsometatarsus to immobilize the bird. Care must be taken not to injure the leg. A hood can be tried, but not all birds will tolerate it.[55] Chemical sedation may be required for further examination. This can be given after physical restraint or by darting before examination. Ketamine, diazepam, xylazine and zolazepam-tiletamine have all been used with unpredictable results. Medetomidine was trialed at 0.26 to 0.31 mg/kg (captive birds) and 0.38 to 0.54 mg/kg (free-ranging birds) with good results.[70] External stimuli must be avoided once the birds are sedated to avoid auditory stimulation.

Kiwis

Kiwis are easily restrained by grasping both legs above the hock joint between the thumb and middle finger of the right hand with the index finger between the two legs (Fig 41.14). The left hand supports the ventral and lateral body into a sitting posture in the crook of the right elbow. Directing the bird's bill and eyes under the cover of the left arm while in this position will help calm it. An alternative method, used when handling kiwi in the field, is to grasp the two legs as described and hold the bird in the upside-down position. The dorsal body is then placed on the lap of the holder with the head similarly placed under the left arm. The birds tolerate this remarkably well for the considerable period of time it takes to apply a radio transmitter and take measurements and diagnostic samples (Jakob-Hoff, personal communication).

PHYSICAL EXAMINATION

Once the bird has been safely restrained, the physical examination can proceed (Table 41.9). A systematic and thorough evaluation is essential, and the use of a comprehensive examination form can be invaluable. A detailed guide to the examination of the ostrich is available.[4] A summary of this technique can be applied to all ratites. After the bird is caught and identified (leg band, microchip), the following are performed:

- Externally examine the eyes, beak, nares, ears and oral cavity.
- Check the body condition by palpating the spine.
- Auscultate the thorax, determining the heart and respiratory rates and checking for abnormal respiratory and cardiac sounds at several sites.
- Auscultate the proventriculus and ventriculus.
- Palpate the abdomen, including the proventriculus and ventriculus.
- Observe the feet and legs for abnormalities.
- Palpate the limbs and determine the alignment of the tibia.
- Examine the skin and feathers for abnormalities and parasites.
- Examine the vent and digitally sex the bird.

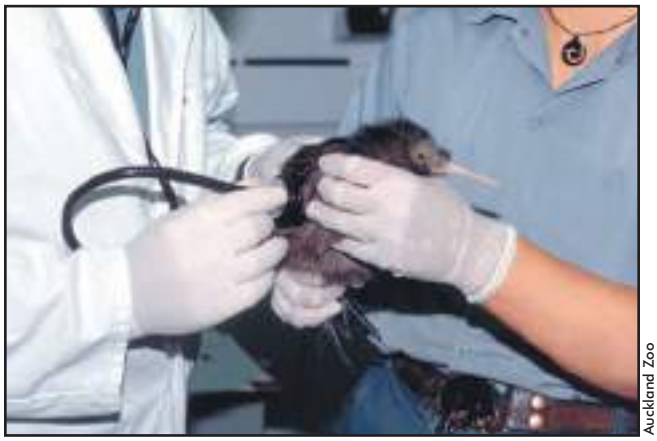

Auckland Zoo

Fig 41.14 | Examining a kiwi chick at Auckland Zoo.

Fig 41.15 | The basilic vein is suitable for blood collection only in the ostrich.

Table 41.9 | Some Physical Characteristics of Adult Ratites

	Weight (kg)	Heart Rate	Respiratory Rate
Ostrich	80-150	30-60	6-12
Emu	35-55	42-76	13-21
Cassowary	85	35-90*	20-44
Rhea	25	—	—
Kiwi	1.5-4.0	70-240	12-60

*Recorded under sedation

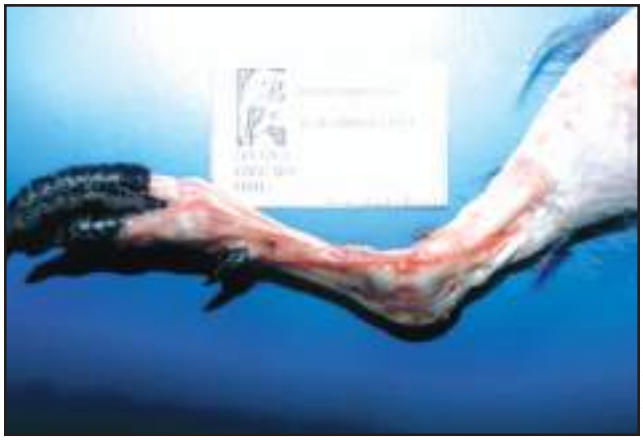

Auckland Zoo

Fig 41.16 | Kiwi dissection shows the medial metatarsal vein, the only accessible vein for blood collection in this species.

Clinical findings should be recorded as the examination proceeds, especially if there are several birds to be examined at the same time. A standardized examination form is invaluable in this respect; not only does it allow accurate recording, it also ensures the clinician does not overlook part of the examination (see Chapter 6 Maximizing Information from the Physical Examination).

DIAGNOSTIC TESTING

If appropriate, samples for laboratory analysis can be collected following the examination.

Several veins are readily accessible for venipuncture in ratites other than the kiwi. The right jugular vein is suitable for the smaller ratites and juveniles. Care should be taken in the larger ratites, because the thin-walled jugular can tear easily, leading to a potentially fatal hematoma. The basilic vein can be used in ostriches (Fig 41.15), but the small vestigial wings in other ratites make this vein unsuitable in other species. The medial metatarsal vein is accessible in all species, but care must be taken that adequate restraint is in place before this vein is utilized. In kiwis, this is the only usable vein (Fig 41.16), the jugular being inaccessible due to the overlying layer of thick, fatty skin (Jakob-Hoff, personal communication).

A blood smear should be made immediately and the rest of the sample placed in plain tubes or, preferably, lithium heparin. If the samples cannot be analyzed rapidly, it is advisable to centrifuge the sample and refrigerate the plasma or serum.

In ostrich hens with a history of reproductive disease (embryonic deaths, poor shell quality, irregular laying, misshapen eggs), a swab can be taken of the distal oviduct for cytology and culture. With the hen hooded and perhaps restrained, a gloved and lubricated hand is placed into the urodeum, and the oviduct opening is located. This opening is found in the 10 o'clock position in a small papilla. A finger can be passed through this opening into the distal oviduct and a guarded mare swab introduced alongside the finger. A limitation of this technique is that only the caudal 10 to 15 cm of the oviduct can be accessed; a disease process occurring more proximally may be missed. Common pathogens isolated include *E. coli*, *Pseudomonas* spp. and *Klebsiella* spp. Fecal contamination is not uncommon.[4]

Several authors have described semen collection in ostriches.[4,26,36] It should be attempted only in mature, reproductively active birds; young or sexually inactive birds will produce little or no semen. Although artificial insemination techniques are being developed in ostriches, semen collection and assessment are important parts of an infertility assessment. Minimal restraint should be

Table 41.10 | Ratite Hematology and Biochemistry Reference Ranges

	Ostrich	Emu	Cassowary	Rhea	Kiwi
PCV (%)	45 (41-57)	47.4 (39-57)	48.1 (33.5-58)	45.5 (29-59)	46 (38-54)
Hb (g/L)	140-172	136-170	174 (135-200)	126 (64-170)	—
MCHC (g/L)	347-412	352-433	352-433	451 (444-457)	250 (110-333)
WBC (x10⁹/L)	18.7 (10-24)	14.9 (8-21)	17.55 (8.6-31.6)	11.8 (4.1-25.7)	11.6 (8.7-14.5)
Heterophils (x10⁹/L)	10.8-16.6	8.0-13.1	11.1 (6.4-20.9)	7.4 (0.5-20.0)	6 (4.0-8.2)
Lymphocytes (x10⁹/L)	2.2-7.7	1.5-6.6	5 (2.0-9.5)	3.6 (0.5-7.0)	4.2 (2.5-5.9)
Eosinophils (x10⁹/L)	0-0.37	0-0.9	0.3 (0.2-0.4)	0.4 (0.05-0.7)	0.18 (0.7-0.29)
Monocytes (x10⁹/L)	0-0.75	0-0.15	1.1 (0.1-2.8)	0.5 (0.04-1.6)	0.3 (0.1-0.5)
Basophils (x10⁹/L)	0-0.37	0.15	0.4 (0.19-0.8)	0.4 (0.07-1.6)	0.56 (0.09-1.3)
CK (U/L)	800-6508	70-818	365-1335	0-2640	521-971
AST (U/L)	226-547	80-380	269-1399	20-192	64-138
Bile Acids (μmol/L)	2-34	2-30	—	—	—
Serum Protein (g/L)	24-53	34-44	45-75	34-62	54-62
Uric Acid (mmol/L)	0.59-8.9	0.59-8.3	0.24-4.5	0.17-1.4	0.3-0.38
Ca (mmol/L)	2.0-3.4	2.2-3.2	2.3-3.0	2.6-8.2	1.85-3.1
Glucose (mmol/L)	9.1-18.3	5.6-13.5	5.5-12.8	2.1-8.8	3.0-3.9
LDH (U/L)	408-1236	318-1243	—	269-1640	2380

used, as excessive stress may inhibit ejaculation. The administration of 5 to 10 IU of oxytocin intravenously may produce a better sample.[60] The phallus is manually extruded from the cloaca, and the dorsal base (where the ductus deferens papillae are located under the uroproctodeal fold) is gently but firmly massaged. Ejaculation occurs after a variable time period, and the semen can be collected as it runs down the dorsal phallic groove.

The use of an artificial cloaca to collect semen in emus has been described.[44] Using a "teaser" hen, the artificial cloaca is placed over the phallus as the cock attempts to mate with the hen. This technique has been quite successful in collecting from these birds.

Interpretation of Clinical Pathology

The interpretation of hematology and biochemistry in ratites is very similar to that of other avian species. It should be noted that ratites normally have much higher creatine kinase (CK) values than other avian species. Table 41.10 shows reference ranges for ratite species. An excellent guide to the interpretation of ratite clinical pathology also is available.[32]

NECROPSY TECHNIQUES

As the ratite industries become more commercial, the value of the necropsy to quickly and accurately determine a diagnosis and etiology continues to grow (Fig 41.17). Necropsy offers both the most accurate diagnosis and the most cost-effective option. These advantages should be impressed on both farmers and zookeepers, and the value of having both fail-to-hatch eggs and dead birds submitted for veterinary examination emphasized.

Egg Necropsy

All eggs that fail to hatch should be necropsied. Not only does an egg necropsy allow a determination of the stage and possible cause of embryonic death, it also provides the clinician (and farmer) with a true indication of fertility. On several occasions, this author has been asked to investigate infertility problems, only to discover (via egg necropsy) that the problem was actually one involving embryonic mortality.

The technique is simple. The egg is first weighed and, if possible, the weight loss during incubation is calculated. The shell quality is examined (surface texture, porosity and cleanliness). The egg is then candled. Candling allows identification of the air cell, an indication of its size and mobility, and an idea as to whether an embryo or infection is present. Hairline cracks in the shell also can be identified. This information is recorded and then the next step is taken.

The egg is placed vertically onto a support (a ring of PVC pipe is sufficient) with the air cell uppermost. The shell over the air cell is broken with a drill or a sharp tap of a hammer, revealing the air cell. The shell is then broken away to the level of the inner shell membrane forming the base of the air cell. This membrane is peeled away with fine forceps, revealing the egg contents (Fig 41.18a).

At this stage, a swab for culture of bacteria and fungi can be taken from the albumen or inside the shell. If an embryo is not obviously present, the blastodisk on the yolk should be examined. The yolk around this disk is less dense than the rest of the yolk, so it will float uppermost. In an infertile egg, the disc will appear as a small white point. A very early embryo will appear as a small white "doughnut" with a patch of yolk in the middle. Any blood vessel development at all is confirmation of fertility (Fig 41.18b).

Fig 41.17 | This intra-abdominal fat in a mature ostrich at slaughter represents a significant financial loss to the farmer in wasted food conversion.

If an embryo is present, two determinations must be made: the positioning of the embryo (if it is late term) and the stage of development.

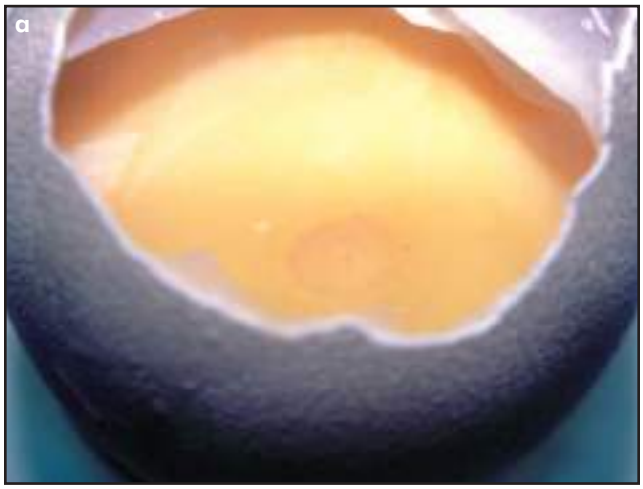
Embryonic position is observed before removing the embryo from the egg. The normal late-term embryo is positioned with the head next to the air cell, right side uppermost and turned to the right, with the beak adjacent to the right foot and shoulder; legs flexed on either side of the body and ventral to the shoulders; and the spine following the long axis of the egg. Any other position is classified as a malposition, and may account for embryonic death. Descriptions and possible causes of classical malpositions are described in Table 41.11.

A measurement of the embryo's crown-to-rump length and an assessment of its growth and development can give an indication as to whether this is an early, mid-term or late-term embryo.

A procedure for egg necropsies has been described.[12] In summary, once the embryo is removed from the egg, the following procedure is recommended:

1. Record position of yolk sac and percentage that is external.
2. Weigh embryo and record crown-to-rump length.
3. Measure thickness of edema (if present) over proximal neck and thighs.
4. Remove and weigh yolk sac (if necessary, incise abdomen).
5. Record gender.
6. Collect samples from the chorioallantois, yolk and proventriculus for culture.
7. Collect tissues from multiple organs, including the membranes, in formalin.

By determining the stage and cause of embryonic death, the clinician can then start to narrow down the search for the underlying cause of embryonic mortality (Table 41.12).



Figs 41.18a,b | An egg necropsy can identify early embryonic deaths in fertile eggs: **a)** degenerated germinal disc; **b)** degenerating vessels.

Necropsy of Chicks and Older Birds

As with any necropsy, a thorough and methodical procedure is essential to obtain the best results. If the bird is still alive, it should be weighed (if possible) and blood collected for hematology and biochemistry. A physical examination should be conducted and then the bird humanely euthanized. Care must be taken to ensure that the euthanasia method does not affect likely necropsy findings.

The body can be opened through a midline incision with the bird in dorsal recumbency, but an alternative is a lateral approach. The body is placed in lateral recumbency, the upper leg is abducted, and an incision is made along the ventrolateral border of the body. The skin is reflected, the coxofemoral joint is disarticulated and the leg is further abducted. The abdominal muscles and ribs can then be transected and reflected dorsally. This gives a good exposure of the internal organs.

Table 41.11 | Embryonic Malpositions

Malposition	Description	Possible Causes (if known)
I	Head is down between the legs	High incubator temperature
II	Chick is rotated within the egg, with the head at the end opposite to the air cell	Egg position and low temperature during incubation
III	Head is rotated to the left, with the head under the left wing	Egg position, temperature and parental malnutrition
IV	Beak is away from the air cell, rest of the body is normally positioned	Egg position
V	Feet over the head	—
VI	Head over the right wing	Parental nutrition

The relative size and position of the organs is noted, and culture swabs are taken from the liver and proventriculus. Individual organs are removed and examined, and tissue samples are placed in formalin for histological evaluation. Proventricular contents should be examined and the contents assessed for possible impaction. Intestinal contents should be assessed as to consistency and volume. A written report should be prepared and placed in the farm's records.

Diseases of Ratite Chicks

Studies of ostrich chick mortality indicate that most deaths occur before 2 months of age and that chicks of this age are most likely to die of infectious diseases. After 2 months of age, death is usually the result of leg problems. Mortality rates from all causes drop significantly after 4 months (More, personal communication). In South Africa, chick mortality is reported to be as high as 40 to 50% up to 3 months and 10 to 30% from 3 to 6 months. Mortality from 3 to 12 months dropped to 5%.[68] Problems with chicks are a major limiting factor in successful ratite production.

DISEASES OF THE INTEGUMENT

Congenital deformities of the beak are occasionally seen in ostrich chicks. The more common of these deformities include scissor beak and a downward deviation of both rhinotheca and gnathotheca. These rarely cause any significant problems to the chick, and treatment is rarely warranted. Severe deformities can interfere with the prehension of food and, unless the chick is valuable, euthanasia may be the preferred option. Manganese deficiency in parental diets is reported to cause a shortening of the lower beak.[34]

Mycotic dermatoses involving *Aspergillus* spp., *Trichophyton* spp. and *Microsporum* spp. have been reported.[34] Treatment with 10% enilconazole wash[a] is the recommended treatment.

Table 41.12 | Troubleshooting Embryonic Mortality[40,64]

Problem	Possible Causes
Floating air cell	• Rough handling of egg • Parental nutritional deficiencies • Genetics
Early embryonic mortality	• Parental nutritional deficiencies • Delayed egg collection • Incorrect storage – temperature incorrect, stored too long • Shaking/jarring of eggs • Incubator problem – temperature, turning, ventilation • Formaldehyde fumigation of eggs in first 3 days
Mid-term embryonic mortality	• Parental nutritional deficiencies • Infection • Inadequate turning • Incubator problem – temperature, turning, ventilation • Lethal genes
Late-term embryonic mortality	• Malpositions • Incubator problem – temperature, humidity, turning, ventilation • Infection • Parental nutritional deficiencies
Air cell pip, fail to hatch	• Hatcher problem – temperature, humidity, ventilation
Malformed chicks	• Incubator temperature too high • Parental nutritional deficiencies • Genetics • Teratogens
Edematous chicks	• Humidity too high • Shell too thick • Inadequate ventilation
Sticky chicks	• Humidity too high • Shell too thin
External yolk sac	• Temperature too high • Edematous chick • Inappropriate intervention to assist hatch

Avipoxvirus has been reported in ostrich chicks in South Africa, Israel, the United States and Australia.[53,58] The proximity of poultry and the presence of suitable vectors contribute to outbreaks. Chicks from the age of 2 weeks are affected, with both cutaneous and diphtheritic forms reported. Vaccination from 10 days of age with fowl pox vaccine combined with biting insect control will usually limit or prevent outbreaks.

Hyperkeratosis of the skin around the head, eyelids, beak commissures, neck and feet has been observed in ostrich chicks and is thought to be nutritional in origin. Supplementation with zinc and biotin often is curative. It must be distinguished from mycotic infections.

A biotin-responsive plumage abnormality has been recorded in wild-hatched North Island brown kiwi chicks simultaneously parasitized by a hemoparasite of the genus *Babesia*. The feather abnormalities included complete feather loss from the ventral-lateral trunk and thighs and retention of keratinized sheaths on dorsal contour feathers, giving the plumage a dry, spiky texture. The hemoparasite infection was associated with pronounced pyrexia, and it was speculated this might have interfered with biotin absorption as has been recorded in poultry.[38]

DISEASES OF THE GASTROINTESTINAL TRACT

Candidiasis in ratite chicks can result from immunosuppression, malnutrition or prolonged antibiotic therapy. Plaque-like lesions can be observed in the oropharynx, although the infection can extend through to the ventriculus. Cytology and/or culture are diagnostic. Treatment with nystatin or ketoconazole is usually successful.

Other fungal infections, including zygomycosis and macrorhabdosis (megabacteriosis), have been reported in ostrich chicks.[34,58] Treatment is the same as in other avian species.

Bacterial enteritis is a common problem in ostrich chicks (Fig 41.19). *Salmonella* spp., *E. coli* and *Pseudomonas* spp. are the most common isolates. Clinical signs are not limited to just the intestinal tract, as septicemia frequently develops. Diarrhea, swollen joints, keratitis, corneal ulceration, depression and anorexia are common clinical signs. Hepatic granulomas are frequently seen on necropsy of chicks dying from *Pseudomonas* septicemia or colibacillosis. Although antibiotic therapy, based on culture and sensitivity testing, can save some chicks, severely affected birds usually die. It is essential that investigating clinicians do all that is possible to identify the source of the infection, as management issues significantly contribute to such outbreaks.

Clostridial enteritis may be an over-diagnosed problem. *Clostridium perfringens* is a normal inhabitant of the ostrich intestinal tract and its identification on fecal culture does not necessarily constitute a diagnosis of clostridial enteritis.[34] Abrupt dietary changes, starvation, stress, anthelmintics and vaccinations have been associated with outbreaks. Ingestion of soil and substrate contaminated with clostridial spores also may be a contributing factor. Acute mortality, occasionally with antemortem anorexia and depression, is the hallmark of this infection. Necropsy reveals varying degrees of enteritis associated with clostridial spores, and *Clostridium* spp. can be cultured from affected chicks. Treatment with tetracycline, penicillin and zinc bacitracin has been described.[34,58] A vaccination protocol using *Cl. perfringens* Type B and D vaccines at either 1 week or 1 month, or at 3 and 6 weeks, also has been described.[34]

Adenoviral enteritis has been described in ostrich chicks in the USA.[54] Affected chicks, usually older than 2 months, show marked depression, anorexia and diarrhea. Mortality is usually greater than 90%. Vertical and horizontal transmission can occur. Infectious bursal disease has been reported in the United States. Affected chicks show depression, anorexia and diarrhea over a 3- to 4-day period before death.[58]

Fig 41.19 | Intestinal volvulus in a 6-week ostrich chick. This may be secondary to enteritis and is usually diagnosed postmortem.

Endoparasitism can be a major problem in young ratites, particularly if they are reared with adults or have access to adult fecal material. An excellent review of ratite parasites is available.[21] *Libyostrongylus douglassii* (wire worm) is a major problem on some ostrich farms on most continents, with mortality rates of affected chicks approaching 50%. It causes a diphtheritic proventriculitis (vrotmaag: rotten stomach), with clinical signs including weight loss, depression, anorexia and death. Strongyle eggs identified on fecal flotation should be cultured, as larval identification is needed to diagnose *L. douglassii*. Levamisole, fenbendazole and ivermectin have been reported as effective treatments.

Many other helminths have been identified in ostriches and other ratites, including trichostrongyles, ascarids, acanthocephalans and cestodes. Protozoal parasites, including *Cryptosporidia*, *Balantidium* spp., *Histomonas meleagridis*, *Spironucleus* and coccidia, have been reported in many ratite species. Coccidiosis is by far the most important gastrointestinal parasite of captive kiwis, and regular monitoring is important. Cumulative fecal samples collected over 4 days are examined to reduce the possibility of missing this intermittently shed parasite. The infestation is readily treated with a single dose of toltrazuril[b] (25 mg/kg PO).[37]

Prolapses of the cloaca and rectum in young chicks can occur secondarily to enteritis. Treatment of these prolapses involves replacement with a gloved, lubricated finger and correction of the initiating cause. If the prolapse recurs, the use of a purse-string suture may be required. This suture can be placed under local anesthesia and should be left in place for no more than 3 days (Black, personal communication).

Treatment of enteritis in ostrich chicks revolves around

several key principles: early recognition that a problem exists; identification and isolation of affected chicks; identification of the causative pathogen; and appropriate treatment.

Batching of chicks and good biosecurity measures offer the best chance of success in stopping an outbreak.

DISEASES OF THE RESPIRATORY SYSTEM

The artificial environment in which young ratites are usually reared — hatchers, brooders and chick pens — often are poorly ventilated, and respiratory disease results.

Aspergillosis is a common disease in ratites (Fig 41.20). *Aspergillus fumigatus* and *A. flavus* are commonly isolated from affected birds; *A. niger* and *A. terreus* may not be clinically significant (Black, personal communication). In chicks, the disease is associated with poorly ventilated hatchers and brooders. Eggs contaminated with *Aspergillus* spp. from nest substrate placed in a hatcher cause a buildup of spores, which are then inhaled by newly hatched chicks. Alternatively, chicks placed in poorly ventilated, unhygienic brooder pens may be exposed to spores. If an immunocompromised chick inhales sufficient spores, an overwhelming fulminating "brooder pneumonia" may result, often followed by relatively acute death. In other cases, an air sacculitis may result, which may not become clinically apparent for weeks, months or even years. Treatment is usually unrewarding because of the often chronic, granulomatous nature of the disease. Ketoconazole, amphotericin B and itraconazole have been used to treat systemic aspergillosis, while inhalation of smoke generated by enilconazole bombs[c] have been used to treat pneumonia and air sacculitis.

Pseudomonas aeruginosa, Pasteurella haemolytica, P. multocida, Haemophilus spp. and *Bordatella* spp. have been isolated from chicks with respiratory infections.[34] *Mycoplasma* spp. also has been isolated.

An important factor in the pathogenesis of respiratory disease in ratite chicks is ammonia toxicosis. Ammonia arising from the feces and urine builds up in chick sheds overnight when the chicks are closely confined. Overcrowding and poor ventilation in the shed will lead to ammonia toxicosis. This should be suspected when, upon opening the shed in the morning, the chicks are lethargic and yawn frequently. In more severe cases, keratitis may develop. The smell of ammonia in the shed is strong. The irritation to the airways can lead to secondary infections as described above. Prevention revolves around management: overcrowding should be avoided

Fig 41.20 | An ostrich hen with advanced aspergillosis exhibits severe respiratory distress.

(allow 0.5 to 1.5 m^2 floor space per chick). Chick sheds should be cleaned daily to reduce the buildup of feces and urine, and ventilation should be improved. As ammonia is heavier than air and will therefore settle closer to the floor, ventilation must be provided at chick level. Ventilation grills 15 to 30 cm above the floor combined with exhaust vents in the roof can dramatically improve the situation.

DISEASES OF THE YOLK SAC

The newly hatched chick receives some nutrients and possibly immunoglobulins from the yolk sac, which is attached to the intestine via the vitello-intestinal duct. Yolk sac contents are drained by this duct and via absorption by mesenteric blood vessels around the yolk sac. A few days before hatching, the yolk sac is drawn into the abdomen, and the abdominal muscles close over it at the navel. The yolk is resorbed over 10 to 14 days, and only a vestigial remnant remains after 3 weeks.

Occasionally the umbilicus fails to close fully, exposing a portion of the yolk sac. If the exposure is only small, dressing it with iodine and bandaging until closure is complete may be all that is required. Larger exposures may require surgical correction if economically practical. Affected chicks should be monitored carefully for omphalitis or yolk sac retention.

Omphalitis, or yolk sac infection, can be a multifactorial problem seen in chicks less than 1 week of age. Chicks that have been stressed by incorrect incubator/hatcher temperature and humidity or chicks that are weak or undersized at hatch are predisposed to bacterial infection. This infection usually arises from poor hygiene in the hatcher or brooder, although transovarial and transoviductal infections and shell contamination also can be involved. Many chicks that develop yolk sac retention may subsequently develop omphalitis. The incidence on

Table 41.13 | Prevention of Omphalitis and Yolk Sac Retention*

- Optimal incubation conditions
- Avoidance of early or inappropriate intervention during hatching (an exposed yolk sac has a much higher chance of becoming infected)
- Good hatcher hygiene
- Minimal handling after hatching
- Adequate umbilical care
- Adequate nutrition and exercise

*Black (personal communication)

many farms can be reduced by correcting incubator and hatcher problems and hygiene and applying an iodine ointment and bandage to the umbilicus of newly hatched chicks (Table 41.13). If chicks from a particular hen show a high incidence of omphalitis (without other chicks being affected), that hen needs to be investigated for the possibility of salpingitis.

Yolk sac retention, on the other hand, is a failure of the yolk sac to be resorbed in the absence of infection. This is primarily a management problem, with possible faults lying in incubator or hatcher problems, chick nutrition and exercise. Unless secondary omphalitis develops, clinical signs do not become evident until the chick is 2 to 6 weeks old and the autolyzing yolk begins to release possible toxic substances that are absorbed by the mesenteric blood vessels. These chicks fail to thrive and usually start to lose weight. The yolk sac often is palpable in the abdomen or is detectable via ultrasound.

As antibiotics do not penetrate the yolk sac, treatment for both omphalitis and yolk sac retention is the surgical excision of the yolk sac. Although a relatively simple procedure, the success rate for omphalectomy is not high due to pre-existing toxemia and immunosuppression.

DISEASES OF THE MUSCULOSKELETAL SYSTEM

"Wry neck" or torticollis is seen occasionally in all ratite species, with emus been the most frequently affected. The muscles and tendons of the neck cause abnormal positioning of the neck and head. Suggested etiologies include vitamin E and selenium deficiencies, teratogens, parental malnutrition, excessive handling and turning of the egg during incubation, and skeletal malformation. Splinting and dietary supplementation may improve many chicks.[61]

Tibiotarsal rotation (or angular limb deformity) is one of the most common and serious limb deformities seen in ratite chicks (Fig 41.21). The tibiotarsus rotates along its long axis, leading to an outward rotation of the limb (Fig 41.22). Mild cases may show only a "windmilling" gait when running, ie, the affected leg swings out rather than going straight ahead. Severe cases may be so badly rotated that the chick has great difficulty in walking and standing. Numerous causes have been suggested, indicating this problem's multifactorial nature. Possible etiologies may include diets excessively high in protein and/or energy, calcium/phosphorous imbalances, leg injuries, lack of exercise and heating of chick pen floors. Although no hereditary component has been conclusively identified, it appears that certain breeding pairs may produce offspring that have a high growth rate. These offspring, if fed a diet that can maintain or encourage this high growth rate, will develop a higher incidence of tibiotarsal rotation if they are exercised inadequately. Various surgical corrections have been described, but none has shown consistent long-term success. In kiwi chicks, "splayed legs", which occur when the brooder box substrate is too slippery to afford adequate traction, can readily be corrected with hobbles if identified in the early stages (Fig 41.23) (Jakob-Hoff, personal communication).

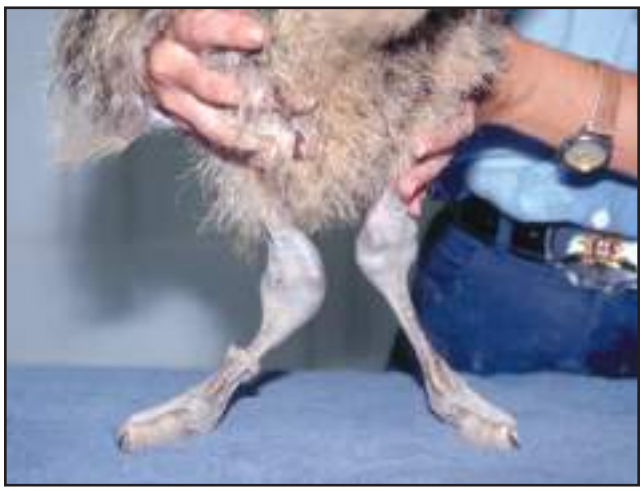


Fig 41.21 | Angular limb deformity in an ostrich chick raised on an oxalate-rich pasture, causing calcium deficiency.

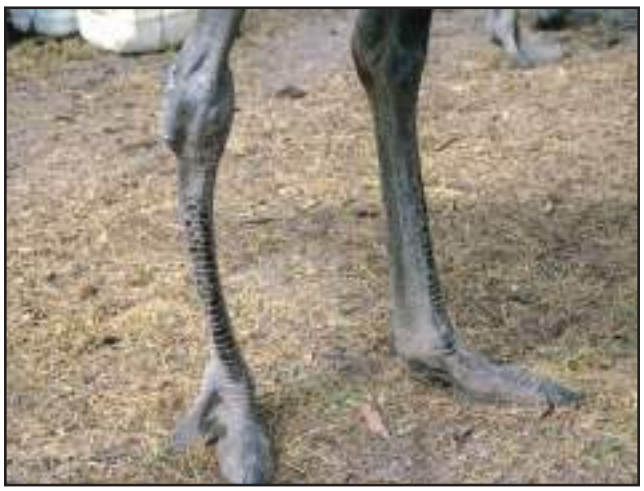


Fig 41.22 | Bowed tarsometatarsus, mature ostrich. This is usually the result of juvenile nutritional imbalance.


Auckland Zoo

Fig 41.23 | Splay leg in a young kiwi chick. At this age, this condition is reversible through the application of hobbles and use of a non-slip substrate.

Rickets is the result of a deficiency of vitamin D_3. This can be due to dietary imbalances, lack of sunlight or intestinal malabsorption. It results in enlargement of the joints and bowing of the femur, tibiotarsus and tarsometatarsus.[61] Identifying and rectifying the inciting cause(s) may correct early cases. Other causes of bowed legs include nutritional calcium and phosphorous deficiencies or imbalances.

Rolled toes appear to occur in two distinct groups. The first group is chicks aged less than a week. Within a day or two of hatching, the toe begins to roll medially. Parental malnutrition and incubator errors have been implicated as causative factors. Corrective splinting or taping of the affected toe usually rectifies the roll within a few days. Chicks older than 4 to 5 weeks make up the second group. In these chicks, the rolled toe appears to be a variation of the angular limb deformity problem. Splinting may help with these chicks, but the prognosis is not as good unless dietary and exercise factors are corrected.

"Slipped tendon" refers to the lateral luxation of the flexor tendon of the gastrocnemius muscle out of its position on the caudal aspect of the hock joint. This can be mild, with immediate relocation of the tendon when the joint is straightened, or so severe that the tibiotarsus tears through the skin. Acute, mild cases may be successfully treated with bandaging and splinting. More severe cases, or cases where the luxation has been present for several hours, carry a poor prognosis. Surgical repair of the tendon retinaculum is required in these cases but is usually unsuccessful because of the weight-bearing stresses placed upon it.

OSTRICH FADING SYNDROME (CHICK FADING SYNDROME)

In the early 1990s, a syndrome in ostrich chicks less than 6 months old, characterized by weight loss, anemia and death, started to appear on ostrich farms throughout the world. Mortality rates vary from 20 to 80%. Affected chicks progressively lose weight with wasting of the paralumbar muscles. Ascites often develops. Urate pasting around the vent is common as the chick weakens. A nonregenerative anemia is a frequent clinical finding.

Necropsy reveals a multitude of pathologies including rhinitis, pharyngitis, proventriculitis, enteritis, pneumonia, air sacculitis, hepatic abscessation and splenic atrophy. The most consistent histological finding is non-suppurative enteritis.[13]

The syndrome appears to have an infectious etiology, but numerous pathogens have been recovered from affected chicks: *Cryptosporidia*, gram-negative and gram-positive bacteria, fungi and yeast, and a range of viruses. No consistent pathogen has been identified.[13,61]

Given the wide range of findings and secondary pathogens isolated, it is generally presumed that a primary immunosuppressive agent is responsible for this syndrome. Work in Australia suggested that a retrovirus may be the causative agent, but this has yet to be confirmed.[48]

A major problem with this disease is the tendency of many farmers to categorize all chick mortalities as fading syndrome and decline further work-up. Clinicians must educate their clients against this tendency. The good news is that in recent years the incidence of this disease appears to have declined. Until the causative agent is identified, good biosecurity measures and treatment of secondary pathogens is the best that can be offered.

Diseases Of Adult Ratites

Mortality rates in ostriches decline rapidly with age. This appears to apply to all ratites, with few of the disease processes described for chicks applying to adult birds. Discussed below are some of the problems specific to adult birds, as well as some that can affect all age groups.

DISEASES OF THE INTEGUMENT

Skin lacerations are not uncommon in captive ratites, often occurring as injuries inflicted by collisions with wire fences. Although these respond well to basic treatment principles, it must be noted that scarring will significantly detract from the hide value in birds such as ostriches and emus. Fence construction and handling procedures must be done with the aim of minimizing such injuries.

Bacterial and mycotic dermatitis and folliculitis do not appear to be common but can occur in excessively humid climates. Early recognition and treatment is essential to prevent or minimize damage to the hide.

An exudative dermatitis beginning around the external auditory meatus and progressing to involve the ventral and dorsal neck has been reported in captive kiwis whose normal vitamin/mineral supplement had been accidentally omitted from the diet. The condition responded dramatically to B-complex vitamin injections after 5 to 6 days.[10]

Ectoparasites are common in ratites, with ticks, lice and mites being found on most species. While the primary clinical focus is on the damage caused to plumage and hide by lice such as the ostrich louse, *Struthioliperirus struthionis*, and mites such as the quill mite, *Gabucinia bicaudatus*, other problems have been recorded. Tick paralysis, caused by an ixodid tick, *Hyalomma truncatum*, has been reported in South Africa, while an argasid tick, *Argus persicus*, can cause anemia in chicks and transmit aegyptianellosis from chickens to ostriches.[21] The kiwi tick, *Ixodes anatis*, is suspected to be the vector of the *Babesia* spp. found in these birds.[50] Regular spraying with 2% malathion or pyrethrins needs to be part of the husbandry of ratites.

DISEASES OF THE DIGESTIVE SYSTEM

Mycobacterial infections have been reported in ostriches, emus, cassowaries, rheas and kiwis.[29,34,58] Affected birds typically show chronic weight loss accompanied by a marked leukocytosis. Necropsy shows multiple granulomas throughout the liver parenchyma and intestinal serosa, extending into the mucosa. *Mycobacterium avium* is usually identified either by culture or PCR. Although some reports[58] contend that intradermal tuberculin testing is diagnostic, this is not confirmed by other reports.[29] At this time, intradermal testing should be used to confirm the presence of the disease in a flock, rather than in individuals. Although the zoonotic potential of *M. avium* strains isolated from ratites has not been fully ascertained, treatment is not recommended and affected birds should be euthanized.

Sick or stressed ostriches of all ages may start to ingest foreign or indigestible material, which blocks (impacts) the proventriculus. As more material (high-fiber grasses, stones, dirt, sticks) is added, the proventriculus becomes distended, but no ingesta can move through. Affected birds become anorectic, weak and lose weight. They may regurgitate water after drinking. Fecal output diminishes or ceases altogether. The impacted proventriculus is usually palpable. Young birds can be treated by gastric lavage while suspended upside down. Older birds may require a proventriculotomy.[34] Although some authors[34] advocate against the use of mineral oil, it has been used successfully in sand and gravel impaction when combined with psyllium (Doneley, personal observation). Treatment is of value only if the original stressor has been removed, behavioral stress minimized and access to substrate and/or foreign material prevented.[35]

Eastern equine encephalitis (EEE) has been reported in emus in North America. Affected birds show depression, recumbency and hemorrhagic diarrhea, followed by death. Prevention revolves around minimizing exposure to biting insects and a vaccination protocol using a commercial equine inactivated EEE vaccine.[58]

An acute central nervous system disease manifesting as extreme depression and convulsions has been observed in a number of captive North Island brown kiwi. Some fatalities have occurred while others have made a full recovery over a number of days to weeks with supportive care. Although response to antibiotics has suggested a bacterial etiology, not all affected birds responded similarly (Jakob-Hoff, personal communication).

DISEASES OF THE RESPIRATORY SYSTEM

The most common respiratory disease seen in ratites is air sacculitis associated with infection by *Aspergillus* spp. Unlike the "brooder pneumonia" seen in young chicks, this is a chronic disease with clinical signs often appearing within a few weeks of a stressful event such as transport. Affected birds lose weight and show an increased respiratory effort, seen as an increased respiratory rate (greater than 20 breaths per minute in a non-stressed bird), increased sternal lift and open-mouth breathing. Care must be taken to observe suspect birds before they are handled or heat stressed, as this can replicate the signs of respiratory distress. Some birds may cough. Auscultation over the paralumbar fossa may detect rasps, squeaks and friction rubs at peak inspiration and expiration. Leukocytosis is frequent, but serology is unreliable, as many birds appear to fail to seroconvert and will test falsely negative.

Aspergillosis is not contagious among birds, but it is not uncommon to find several birds infected from a common source. Treatment with antibiotics (for secondary infections), itraconazole orally and fumigation with enilconazole smoke bombs[c] appears to be effective in causing remission of clinical signs in many birds, although recurrence months or years later is common (Doneley, personal observations).[43]

Avian influenza, caused by a number of strains of the

influenza virus, has been reported in ostriches in South Africa and Denmark. Chicks are apparently more susceptible than are adults, but all age groups can be affected. Clinically, infected birds show severe depression, respiratory signs, ocular discharge and green urates. Treatment is usually unsuccessful, and vaccination relies on the identification of the viral strain present. Prevention is best achieved by preventing contact between ostriches and free-ranging birds.[35]

DISEASES OF THE NERVOUS SYSTEM

Newcastle disease, caused by paramyxovirus serotype 1 (PMV1), has been reported in ostriches in Africa and Israel and in rheas in Brazil. The virus is documented in 236 avian species, confirming widespread susceptibility of birds to this disease.[58] Young birds appear to be more susceptible, with up to 50% mortality. Affected birds initially show a slight head tilt, frequent scratching of the head and a tic of the neck muscles. This progresses on to torticollis, uncontrolled head movements and finally an inability to lift the head off the ground. Death occurs in 2 to 3 days.[35] There is no treatment, although some birds recover spontaneously. Vaccination is carried out on some farms in South Africa with apparently good results.[9,57,67]

Western equine encephalitis (WEE) has been reported in emus and rheas in North America.[69] Affected birds are initially depressed, progressing on to paresis, recumbency and finally paralysis. Mortality rates are less than 20%, with recumbent birds dying within 48 hours. Vector control and vaccination are usually effective in preventing this disease.[58]

Borna Disease is seen in 2- to 8-week-old ostrich chicks in Israel. Affected chicks show paresis and reluctance to move, progressing on to paralysis and finally death from dehydration.[58]

Cerebral nematodiasis has been reported in ostriches and emus in North America. *Chandlerella quiscali* and *Baylisascaris* spp. have been isolated from birds showing signs including ataxia, abnormal gait, muscle weakness, recumbency and death.[21]

DISEASES OF THE REPRODUCTIVE SYSTEM

Disorders of the Female Reproductive Tract

The quality of the egg and its shell can be a reflection of the health of the hen and reproductive tract that produced it; consequently, veterinarians are occasionally called on to comment on abnormal eggs. A number of abnormalities have been observed (Table 41.14).

Table 41.14 | Assessment of Egg Quality

Observed Abnormality	Possible Causes
Shell too thin	• Immaturity of hen • Malnutrition • Salpingitis causing rapid transit of egg through tract • Genetics
Shell too thick	• Excessive calcium supplementation
"Stress lines" – ridging and grooving of the egg shell	• Salpingitis due to infection or excessive egg laying • Copper deficiency
Shell texture chalky	• Immaturity of hen • First eggs of the season
Blood on the shell	• Maiden hen • Salpingitis
Excessive porosity	• Immaturity of hen • Start or end of laying season • Nutrition • Genetics
Floating air cell	• Rough handling by bird or people • Genetics
Cracked or damaged shells	• Eggs laid while standing up • Rough handling by bird or people
Abnormal shape	• Salpingitis • External pressure on oviduct compressing the egg as it forms eg, fluid in abdomen due to yolk-related peritonitis

Yolk-related peritonitis occurs when yolk is deposited into the abdomen rather than passing down the oviduct. The infundibulum in ostriches is a large, delicate membrane that moves up and surrounds a developing ovum. Any inflammation of the oviduct or infundibulum can result in this process not proceeding normally, and the ovum fails to enter the oviduct. Alternatively, the ovum does enter the oviduct, but retropulsion pushes it cranially and out through the infundibulum. The yolk constituting the ovum provokes a peritoneal inflammatory response, producing large volumes of fluid as a result. Affected hens usually have a history of having been good layers that suddenly stop laying eggs or may produce abnormally shaped eggs. Examination shows distension of the abdomen and a flaccid, enlarged vent. A fluid wave can be balloted on cloacal examination, and ultrasound can detect the fluid accumulation. Hematology reflects a marked leukocytosis. Abdominocentesis, performed with an appropriate catheter (eg, 14 ga in an ostrich) on the flank behind the last rib, reveals a yellowish pink fluid. Early or mild cases may respond to antibiotic and anti-inflammatory therapy, but severe cases may require a flank laparotomy to allow drainage of the fluid and lavage to remove yolk material.

Salpingitis can be due to infection (ascending or descending) or bruising following excessive or difficult egg laying. The bird may show any or all of the following signs: cessation of egg production, cloacal discharge, stress lines on eggs (Fig 41.24), increased embryonic mortality, or signs of ill health. Physical examination and laboratory work-up other than a leukocytosis may be unrewarding. Ultrasonography may reveal caseous mate-

Fig 41.24 | Stress lines in an ostrich egg from a hen with salpingitis.

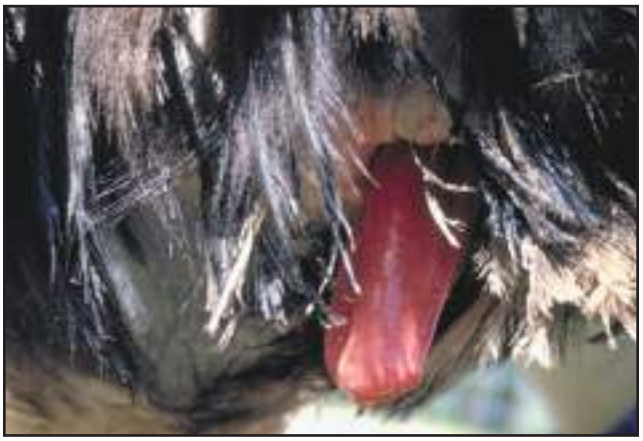


Fig 41.25 | Prolapsed phallus, mature ostrich cock.

rial and fluid within the oviduct, and an oviduct swab may detect the presence of infection and/or inflammation. Antibiotic therapy should be based on culture and sensitivity, and cases with caseous exudate in the oviduct may require the surgical insertion of a Foley catheter into the magnum or isthmus, followed by normograde flushing to expel debris from the oviduct.[33]

Two ostriches with caseous salpingitis were treated with dinoprost tromethamine, a prostaglandin $PGF_{2\alpha}$ salt.[d] In both cases, the hens were treated with one 5-mg injection. This was followed within 15 minutes by signs of abdominal discomfort (grunting, straining to urinate and defecate, and restlessness), which lasted for approximately 1 hour. Systemic antibiotic therapy was started and given for 1 week after the prostaglandin. Follow-up ultrasonography 48 hours after the prostaglandin showed that the caseous material within the oviduct had gone, and both hens went on to breed successfully within 6 months (Doneley, personal observation). Although this treatment caused considerable discomfort to the patients, with further investigation it may prove to be a viable alternative to surgery. *Ed Note: Try PGE_{1or2} either applied to cervix or injected into the oviduct to reduce pain and dilate the cervix. This probably is a more expensive therapy then owners would prefer, but it is a more humane therapy.*

Egg binding is uncommon in ratites, or at least uncommonly diagnosed. With the exception of the kiwi, the size of ratite eggs relative to body size is quite small, so the clinical signs associated with egg binding in other species are not seen in ratites. The only clinical sign may be cessation of egg laying. In smaller ratites, the egg may be palpable in the abdomen, but in larger ratites (or cases where the diagnosis is uncertain), ultrasound is usually diagnostic. Conservative treatment with oxytocin and calcium may resolve some cases, but surgery may be required.

Although uncommon, prolapse of a large portion of the oviduct of mature egg-producing hens can occur. The prognosis for these hens is generally very poor. Treatment must be instigated as soon as possible after the prolapse and requires general anesthesia and careful replacement of the oviduct, followed by a purse-string suture. If the oviduct has been prolapsed for a significant time, tissue viability often is compromised. Antibiotics and anti-inflammatory drugs are given postoperatively. An ultrasound examination is strongly recommended to detect the possibility of the presence of egg retention or other oviduct pathology (Black, personal communication).

In contrast to oviductal prolapses, an apparent cloacal prolapse is seen in immature ostrich hens aged 12 to 20 months before egg production begins. The immature oviduct secretes an albumen-like fluid as it matures. This normally drains freely into the cloaca and is occasionally noticed by observant farmers. In some birds, a persistent membrane covers the opening of the oviduct into the urodeum. This prevents the fluid from draining. As pressure builds up behind this membrane, a balloon-like structure arising from the urodeum forms and eventually bulges out through the vent, appearing as a prolapse 5 to 10 cm in diameter. Once the diagnosis is confirmed by cloacal examination, the membrane can be incised with a scalpel and the newly created opening widened manually. This allows the fluid in the oviduct to drain and the problem resolves. Prophylactic antibiotic coverage is at the clinician's discretion.

Disorders of the Male Reproductive Tract

Phallic prolapse can occur in both immature and mature birds (Fig 41.25). Immature birds attempting to mate inappropriately can damage the phallus, leading to a prolapse. As well as the treatment described below, these birds need to be isolated from other birds to allow them time to sexually mature. Mature birds can develop

a phallic prolapse following trauma or infection of the phallus and its surrounding structures or because of excessive sexual activity. Occasionally, an unhealthy bird will develop a prolapse due to generalized weakness.

Purse-string suture techniques should be a treatment of last resort in these birds. If the phallus is not traumatized or becoming desiccated, conservative treatment is usually sufficient. Isolation and sexual rest, combined with lubrication and frequent replacement of the phallus into the cloaca, may be sufficient. Anti-inflammatory and antibiotic therapy may be useful adjuncts. If the phallus is suffering significant trauma or drying out, a temporary purse-string suture combined with anti-inflammatory and antibiotic therapy may be required.

Infertility

The consistent production of infertile ("clear") eggs requires a thorough veterinary investigation. Before beginning such an investigation, it is vital to review records and procedures. All eggs that fail to develop or hatch should be necropsied as described earlier. Numerous cases of assumed infertility have subsequently been shown to be early, or even mid-term, embryonic deaths.

Possible causes for true infertility include the following:

- **Incompatibility:** Some birds simply are not compatible and will not mate. Occasionally, if birds are run as trios or in colonies, a dominant hen may prevent mating with other hens by driving them away from the cock.
- **Immaturity:** Many farmers have unrealistic expectations of when they can expect their birds to perform. As hens generally mature earlier than cocks, egg production may occur before the cocks are physically capable of mating.
- **Environmental disturbance:** If birds are feeling threatened by disturbances in their immediate environment (such as construction work, traffic, dogs), they may show little interest in mating. The presence of another cock bird in the adjoining enclosure may trigger territorial behavior and inhibit mating.
- **Nutrition:** Excessively fat or excessively thin birds often show little interest in mating. Imbalances in vitamin and mineral content of the diet may cause decreased spermatogenesis.
- **Poor health:** Sick or injured birds may not mate.
- **Time of season:** As most ratites are seasonal breeders and the hens come into breeding condition before the cocks, the first few eggs of a breeding season may be infertile.
- **True male infertility:** This is uncommon and can be assessed by semen collection and evaluation.

MISCELLANEOUS DISEASES

Erysipelas is occasionally diagnosed in emus in Australia and the United States. *E. rhusiopathiae* is transmitted by ingestion of contaminated soil and through skin lacerations. Typically, affected birds die with few clinical signs other than a short period of depression. Necropsy shows hepatomegaly, splenomegaly and petechial hemorrhage on serosal surfaces of the viscera. Culture of *E. rhusiopathiae* from the liver, spleen or heart blood is diagnostic. Affected birds can be treated with penicillin or quinolone antibiotics. The turkey vaccine can be used in susceptible birds at 6 weeks, 20 weeks and 40 weeks.[58]

Salmonellosis has been reported in ostriches, emus, kiwis and rheas. *Salmonella pullorum, S. typhimurium* and *S. arizonae* have been isolated, with both vertical and horizontal transmission possible. Carrier birds, rodents, free-living birds and mammals can act as reservoirs for the infection. Clinical signs can include embryonic and neonatal mortality, and depression, diarrhea and sudden death in juvenile and adult birds. Antibiotic therapy (eg, quinolones) can suppress clinical signs but may create chronic carriers. Good biosecurity measures are necessary to both prevent infection and to limit its spread.[58]

Anthrax, caused by *Bacillus anthracis*, has been diagnosed in ostriches in South Africa. Spores lying dormant in the soil for many years act as the reservoir for infection. Death occurs acutely with few clinical signs. Demonstration of the characteristic organism in blood smears is diagnostic. There is no recommended treatment. Carcasses should be disposed of by incineration (*Editors Note: Burned Spores are liberated and this may not be an ideal practice)* or deep burial. An inactivated vaccine is available.[58]

A necrotizing typhlitis, associated with a mixed spirochete and trichomonad-like protozoan, has been reported in rheas. Juveniles older than 1 month are susceptible, with affected birds showing depression and anorexia. The mortality rate may exceed 50%. Necropsy shows distension and hyperemia of the ceca and colon, with fibronecrotic and pseudomembranous changes. Oral metronidazole and parenteral lincomycin reduce mortality in affected flocks.

Plasmodium spp. have been reported in ostriches in Africa, in rheas in Brazil and in emus in North America. *Leukocytozoon* spp. are considered common in juvenile ostriches in South Africa.[34] An as-yet-unidentified *Babesia* sp. has been isolated from North Island brown kiwis in New Zealand and is considered common in wild populations. A *Hepatozoon* sp. also has been recovered from the same birds.[38,50] Affected birds of all species show weakness and weight loss, with a regenerative anemia. Treatment of

avian malaria is discussed in other textbooks.

Aegyptianella pullorum, a rickettsia, is transmitted by argasid ticks in Africa and causes anemia, pyrexia and death in ostrich chicks. Diagnosis is by detection of the organism in erythrocytes. Treatment with tetracyclines is usually curative.[34]

Anesthesia and Surgery of Ratites

The size and potential danger of many ratites present unique challenges to the avian anesthetist. An anesthetic regime must offer a quick, safe and minimally stressful induction, consistent maintenance and a rapid recovery, while at the same time minimizing the exposure of the operator to risk of injury from the patient. To these ends, a number of anesthetic regimes have been devised.

Masking with isoflurane at 5% at oxygen flow rates of 2 to 4 liters per minute can usually safely and easily induce ratites weighing less than 15 to 20 kg. Once induced the bird can be intubated and maintained on 2 to 3% isoflurane at 1 to 2 liters per minute. Assisted ventilation may be advantageous in longer procedures. Recovery is usually rapid, and the bird should be manually restrained or wrapped in a towel until fully conscious.

Larger ratites will require injectable induction, followed by intubation and gaseous maintenance. The following are some suggested protocols:

- Sedation with intramuscular azaperone (0.5-2.0 mg/kg), followed 10 to 15 minutes later with an intravenous combination of ketamine (8-10 mg/kg) and diazepam (0.2-0.4 mg/kg), mixed in the same syringe and given as a bolus[5]
- Tiletamine-zolazepam (2-8 mg/kg) intravenously[51]
- Alphaxalone/alphadolone (2.2-4.8 mg/kg) intravenously[51]
- Ketamine (5 mg/kg IM or 2.2 mg/kg IV) plus xylazine (1 mg/kg IM or 0.25 mg/kg IV)[20]
- Medetomidine (0.26-0.54 mg/kg IM) has been used in cassowaries for sedation and restraint,[70] and at 0.1 mg/kg for sedation of ostriches[20]

It is important when anesthetizing large ratites to ensure that adequate padding is placed under the pelvis and femur to avoid peroneal paralysis. If possible, the bird should be positioned in sternal recumbency to assist ventilation. If lateral or dorsal recumbency is necessary, assisted ventilation will be required at a rate of 6 to 12 breaths per minute. The expandable pouch in the trachea of emus must be bandaged during this procedure.

Analgesia and intravenous fluid administration will contribute toward a smooth course of anesthesia. Opioids such as butorphanol or NSAIDs, such as flunixin, carprofen and meloxicam, have been used in ratites. Care should be taken to avoid both hypothermia and hyperthermia, and regular assessment of cloacal temperature is useful in long procedures.

For anesthetic recovery, the bird should be placed into sternal recumbency. This is a potentially dangerous time for the patient. Birds startled by loud noises or other stimuli may rear up and fall over backward, causing themselves injury and even killing themselves. For this reason they should be placed in a quiet, dark area and movement kept to a minimum. Placing them in a crate or horse trailer or surrounding them with hay bales can prevent them from rolling into lateral recumbency. Ostriches should be hooded, and remain hooded, until they can hold their heads upright unaided.

ABDOMINAL SURGERIES[5]

Chicks

Most laparotomies in small ostrich chicks are performed with the bird in dorsal recumbency.

The approach for yolk sac surgery or exploration of the abdominal cavity is via a ventral midline incision. In yolk sac excisional surgery, an elliptical incision incorporating the umbilicus is utilized. Immediately below the skin is an extremely thin abdominal lining. This lining consists of a connective tissue layer with a black-pigmented peritoneum immediately below. Care should be taken in incising these layers, as the dilated yolk sac can be very close or even adhered to the peritoneum. Once the abdomen is exposed, the incision may have to be lengthened to enable the yolk sac to be exteriorized. Again, care must be taken as the yolk sac may rupture if excessive pressure is applied to it. The vitelline duct and blood vessels are ligated between the yolk sac and duodenum, and the yolk sac is removed. The abdomen can be irrigated using a warmed saline solution, and a small amount of aqueous antibiotic can be infused (eg, amoxicillin or ampicillin). This is particularly important if the yolk sac has been ruptured either before or during the surgery. Occasionally the yolk sac may be very inflamed and adhered to the abdominal wall or intestine. Careful dissection is necessary to remove all yolk sac tissue without significant damage to the intestinal serosa. The abdominal incision is closed in two layers: muscle and skin.

A proventriculotomy is performed via an incision over the proventriculus on the left ventral abdomen. This incision normally is sited within the left abdominal pterylae. If the incision is properly sited, the surgical

area is isolated from the rest of the abdomen by fascia and air sac lining, and suturing of the proventriculus to the abdominal wall is unnecessary. If the incision is positioned too far back toward the posterior abdomen, the intestines can be directly visualized and accessed. This area should then be closed by suturing before the proventriculus is entered to avoid contamination of the abdomen by spillage of proventricular contents. The relatively thin proventricular wall is located just below the thin ventral abdominal wall. The proventriculus and the ventriculus can both be accessed via this approach.

The ventriculus should be approached via a proventriculotomy rather than directly incising the ventriculus. Any free fluid in the proventriculus can be aspirated and then the impacting material or foreign bodies can be removed. The closure is in three layers: proventriculus, abdominal wall and skin. The proventricular closure is achieved using a continuous Lembert-type suture pattern using absorbable synthetic suture material. The abdominal wall is closed with similar suture material. The skin closure can be done with absorbable or non-absorbable material.

Older Chicks and Adults

Abdominal surgery on older ostrich chicks and adults is usually performed with the birds in lateral recumbency, lying on their right side.

Entry into the abdominal cavity is via an incision through the left abdominal wall in the flank apterylae. The position and orientation of this incision will be determined by the nature of the condition leading to the laparotomy. For example, most egg yolk peritonitis surgeries are performed via a vertical incision about halfway between the back of the left thigh and the posterior margin of the abdomen. In some instances, a horizontal incision may be used (eg, to gain greater oviduct exposure), or even a T-type incision combining both can be used. The horizontal incision usually results in greater hemorrhage and is subjected to much greater tension on closure. The skin is incised to reveal two muscle/fascial layers directly below (there is very little subcutaneous tissue). Between the second muscle/fascial layer and the peritoneum is a variable and sometimes excessive fat layer. The peritoneum is opened by puncture using blunt-pointed scissors. The peritoneum is pigmented (gray color of varying darkness) and very tough.

With yolk-related peritonitis, significant yolk-stained fluid can be present in the abdomen. This fluid will immediately begin draining through the surgical opening, often under pressure because of the bird's positioning. This fluid can be aspirated or allowed to passively drain. Tilting the bird's abdomen downward facilitates drainage. The abdomen can be manually explored to detect the presence of inspissated yolks, retained eggs or neoplasia. The aim of the surgery is to extensively flush the abdomen until all yolk debris has been removed. This can be a very difficult exercise in view of the size of the abdomen, the length of intestine present and the relatively difficult access. The abdomen is flushed with 0.25% Betadine in saline until no further debris is found and the fluid escaping the abdomen becomes clear. This can take up to 12 to 14 liters of fluid. A final liter of saline and antibiotic is instilled into the abdomen, and 250 ml of this solution is usually infused into the oviduct lumen via a needle through the anterior oviduct.

The oviduct is visualized to detect any pathology such as edema, inflammation or neoplasia. The entire length is palpated to detect any blockages or retained eggs. The infundibulum also is visually inspected. It is imperative to avoid much handling of the infundibulum. This is a highly motile, thin and delicate structure, which is easily torn, bruised or inflamed. It is also important to ensure that the infundibulum is kept well away from the incision site during suturing of the peritoneum.

The closure of the abdomen is normally done in four or five layers. The peritoneum is closed with synthetic absorbable suture in a simple continuous pattern. The two muscle/fascial layers are then closed either separately or as one layer using similar material. A subcutaneous suture may then be done using a lighter absorbable suture. The skin is usually closed with non-absorbable suture. This skin closure is an interrupted pattern of simple or mattress sutures.

The prognosis associated with this procedure is influenced by the length of time the condition has been present and the degree of damage found in the abdomen. Prognosis for survival is excellent, but prognosis for future productivity is guarded, with expected success rates, with experience, of about 66%.

Severe salpingitis cases may best be treated surgically. An intrauterine infusion of an appropriate antibiotic saline solution is employed via a needle or Foley catheter introduced into the proximal oviduct (magnum). A volume of 250 to 1000 ml is used, depending on the extent and nature of the infection. Care must be taken to avoid retrograde passage of fluid back into the abdomen via the infundibulum.

Some retained eggs may be accessed via the cloaca and removed manually while the bird is anesthetized. Eggs higher in the oviduct can be removed only surgically. The site of the retention can be previously ascertained by an ultrasound examination. Access to the lower oviduct is difficult. The oviduct is very friable and has

poor suture-holding properties. After egg removal from the incised oviduct, the wall is sutured carefully using synthetic absorbable suture material in a continuous Lembert-type pattern.

Proventriculotomy surgery in older birds is done in a fashion similar to that of chicks with the amount of abdominal fat being the major difference. It is not uncommon to encounter up to 15 cm of abdominal fat during the surgical approach. This makes access limited in some cases and creates some difficulty for easy suturing.

Occasionally, a decision may be made to perform the laparotomy with the bird in dorsal recumbency and the incision made on the ventral abdomen. This is rarely done, but may be required to access some intestinal problems (especially those involving the small intestine). In these cases, it is necessary to slightly extend the legs to avoid potential nerve and muscle injury at the level of the upper thigh during the procedure.

Intestinal volvulus is not uncommon in ostriches and is especially associated with diet changes. Some cases will occur without any obvious predisposing trigger. Gut-penetrating injuries also can occur after ingestion of sharp objects such as sticks, nails and wire. Diagnosis is based on clinical signs (anorexia, depression, abdominal pain, lack of fecal passage), abdominal paracentesis, hematology and possible radiology. Ostriches appear to have very little tolerance to musculoskeletal pain, but show remarkable tolerance to visceral and abdominal pain. It is possible that birds with significant peritonitis and avascular necrosis of a section of gut may show only mild symptoms of abdominal pain.

The laparotomy site is usually the left lateral abdomen, as described previously. A decision to access the abdomen via a midline approach may be made if the problem is suspected in the upper small intestine or if there is insufficient access via the lateral route. If non-viable intestine is found requiring resection and anastomosis, a poor prognosis should be given. The large intestinal wall is extremely thin and very difficult to suture successfully. Peritoneal lavage using saline and a saline/antibiotic solution is necessary in all cases, as invariably a septic peritonitis is present. If surgical intervention is early enough, it is possible to correct a volvulus or intussusception without further complications, provided gut viability is present.

Postoperative Care

Apart from cases of yolk sac excision or egg yolk peritonitis, most laparotomy cases in ostriches do not require intensive postoperative care.

Chicks recovering from yolk sac excisional surgery require close monitoring, heat support and frequent tube feeding. The tube feeding may be necessary for only 24 hours or may be required for several days until the chick becomes self-sufficient and is regularly gaining weight.

Most laparotomy cases require immediate postoperative antibiotics. The final choice of antibiotics will be determined by any culture/sensitivity testing previously carried out. Tube feeding in some chicks also may be required during the postoperative period. Suture removal, if necessary, is done at a minimum of 2 weeks after surgery.

SKIN LACERATIONS

Generally, lacerations of the skin of the neck, body and upper thighs heal very well once sutured. Some neck lacerations may heal well with minimal intervention. Lower limb wounds are less rewarding due to tension and secondary infection. If possible, administration of general anesthesia to recently traumatized birds is avoided. Local anesthesia, if possible, is safer. If the trauma is too severe for the use of local anesthesia, it is preferable to determine the status of the bird by blood sampling and evaluating the full blood examination and biochemistry profile. This should be combined with immediate stabilization of the patient using intravenous fluids, analgesics, and antibiotics.

Traumatic penetrating wounds to joints and tendon sheaths should be treated vigorously using local and systemic antibiotics. If infection is suspected, culture and sensitivity testing is essential. Joint lavage will be necessary if fistulae and significant joint infection are already present.

ORTHOPEDICS

The likelihood of the veterinary surgeon being requested to perform orthopedic procedures on ostriches is now quite low. Severe leg fractures or luxations usually result in a decision to euthanize the bird. Most cases of beak trauma can be handled medically. It is quite surprising to see ostriches suffer very little setback, even with major beak trauma resulting in significant defects. Wing fractures will commonly repair after simple taping of the wing back into correct position. This is best achieved by taping wing feathers to body feathers at several sites. This avoids the stress of complete body wrap taping of wings. Severe wing fractures can be handled by amputation if necessary.

EYES

Surgical repair of eyelid lacerations is common and usually rewarding.

Eyelid closure to treat corneal ulcers and keratitis can be

performed as described in many textbooks, and may even be done using local anesthesia. The strength of the eyelid muscles is surprising and requires the placement of quite heavy-gauge suture material.

Enucleation can be performed in chicks. In adults this is a difficult procedure with significant risk of hemorrhage. Adult hens have been reported to not breed following unilateral enucleation. This is not always the case, as some birds, apparently in severe pain prior to enucleation, have bred successfully within weeks of the surgery.

Cataract removal using phacoemulsification techniques has been carried out in Australia and the United States (Fig 41.26). This is becoming less common now that bird values have reduced.

Periorbital abscessation resulting from bacterial sinusitis is relatively common in ostrich chicks. These tend to result in discrete swellings on the skull in a dorsomedial position relative to the eye. After local anesthesia, an incision can be made into this swelling to enable debridement and excision of firm gelatinous-like pus. Local irrigation with appropriate antibiotics combined with systemic antibiotic therapy after culture and sensitivity testing are needed to effect a normally uneventful recovery.

Fig 41.26 | Luxated cataract lens in the ostrich.

Acknowledgements

I thank the following people for their invaluable contributions to this work. Doug Black (Ostrich Health Consultant, Moama, New South Wales) gave me valuable advice about writing this chapter, adding to the advice on ostriches he has given me over the last 10 years. Richard Jakob-Hoff (Auckland Zoo, New Zealand) and Hugh Robertson (Science and Research Unit, Department of Conservation, New Zealand) provided much of the information on kiwis, making unique information widely available for the first time. Dale Smith (Ontario Veterinary College, University of Guelph, Canada) discussed with me many aspects of this chapter.

Products Mentioned in the Text

a. Enilconazole wash, Imaverol®, Coopers Australia, North Ryde, NSW www.coopersanimalhealth.com.au
b. Toltrazuril, Baycox®, Bayer Australia, Pymble, NSW, www.baycox.com
c. Enilconazole smoke bombs, Clinafarm, Janssen, Sterwin, Belgium
d. Dinoprost tromathamine (prostaglandin PGF2a) Lutalyse, Pharmacia Upjohn, Rydalmere, NSW, www.lutalyse.com
e. Mazuri, www.mazuri.com

References and Suggested Reading

1. Angel CR, Scheider SE, Sell JL: Ratite nutrition. *In* Tully TN, Shane SM: Ratite Management, Medicine and Surgery. Malabar, FL, Krieger Publishing, 1996, pp 11-30.
2. Badley AR: Improved assessment of the fertility and development of ostrich eggs by more efficient candling. *In* Deeming, DC (ed): Improving Our Understanding of Ratites in a Farming Environment. Manchester, UK, 1996, pp 146-148.
3. Bezuidenhout AJ: Anatomy. *In* Deeming DC (ed): The Ostrich: Biology, Production and Health. Oxon, UK, CABI Publishing, 1999, pp 13-49.
4. Black DG: Ostrich examination. Post-Grad Comm Vet Sci, University of Sydney. Sydney, Australia, 1995.
5. Black DG: Common medical and surgical conditions of ostriches. *In* The Basics of Avian Medicine. Proc 2789, Post Grad Foundation Vet Sci, University of Sydney, 1996, pp 243-258.
6. Black DG: Genetic, nutritional and management effects on ostrich hide quality. Proc Assoc Avian Vet Aust Comm, 1999, pp 43-51.
7. Black DG: Ostrich flock health. Sem Avian Exotic Pet Med 10(3): 117-130, 2001.
8. Blake JP: Nutritional recommendations for emus. *In* Deeming, DC (ed): Improving Our Understanding of Ratites in a Farming Environment. Manchester, UK, 1996, pp 109-110.
9. Blignaut A, et al: Antibody production in ostriches in response to vaccination with La Sota Newcastle disease virus vaccines. Proc 2nd Intl Ratite Cong, South Africa, 1998, pp 199-200.
10. Boardman WSJ: Suspected biotin deficiency in North Island brown kiwi. Kokako Bull Wildlife Soc New Zeal Vet Assoc 1(1):5, 1994.
11. Brand Z, et al: The effect of preheating of ostrich eggs prior to storage and setting in commercial hatcheries. Proc 2nd Intl Ratite Cong, South Africa, 1998, pp 152-154.
12. Button K: Ostrich incubation practices and necropsy technique. Proc Aust Ostrich Assoc, Post Grad Comm Vet Sci, University of Sydney, 1993, pp 69-72.
13. Button C, Kabay M, Rawlin G: Ostrich fading syndrome in Australia. In Deeming, DC (ed): Improving Our Understanding of Ratites in a Farming Environment. Manchester, UK, 1996, pp 35-38.
14. Calder WA: The kiwi and its egg. *In* Fuller E (ed): Kiwis: A Monograph of the Family Apterygidae. Auckland, NZ, SeTo Publishing, 1990, pp 155-171.
15. Cilliers SC: Feedstuff evaluation, metabolisable energy and amino acid requirements for maintenance and growth in ostriches. Proc 2nd Intl Ratite Cong, South Africa, 1998, pp 12-23.
16. Cilliers SC, Angel CR: Basic concepts and recent advances in digestion and nutrition. *In* Deeming DC (ed): The Ostrich: Biology, Production and Health. Oxon, UK, CABI Publishing, 1999, pp 105-128.
17. Cloete SWP, van Schalkwyk SJ, Brand Z: Ostrich breeding: Progress towards a scientifically based strategy. Proc 2nd Intl Ratite Cong, South Africa, 1998, pp 55-62.
18. Cockrem J: Diet and nutrition of captive kiwi. *In* Billing T (ed): Proc New Zealand Conservation Management Group Kiwi Workshop, Auckland Zoo, 1998, pp 69-73.
19. Colbourne R: Operation nest egg. *In* Billing T (ed): Proc New Zealand Conservation Management Group Kiwi Workshop, Auckland Zoo, 1998, pp 41-46.
20. Cornick-Seahorn JL: Anesthesiology of ratites. *In* Tully TN, Shane SM: Ratite Management, Medicine and Surgery. Malabar, FL, Krieger Publishing, 1996, pp 79-94.
21. Craig TM, Diamond PL: Parasites of ratites. *In* Tully TN, Shane SM: Ratite Management, Medicine and Surgery. Malabar, FL, Krieger Publishing, 1996, pp 115-126.
22. Deeming DC, Ar A: Factors affecting the success of commercial incubation. *In* Deeming DC (ed): The Ostrich: Biology, Production and Health. Oxon, UK, CABI Publishing, 1999, pp 159-190.
23. Deeming DC, Bubier NE: Behavior in natural and captive environments. *In* Deeming DC (ed): The Ostrich: Biology, Production and Health. Oxon, UK, CABI Publishing, 1999, pp 83-104.
24. Deeming DC, et al: A review of recent work on the Behavior of

young ostrich chicks with respect to feeding. *In* Deeming, DC (ed): Improving our Understanding of Ratites in a Farming Environment. Manchester, UK, 1996, pp 20-21.
25. Doneley RJT: Principles of incubation. Proc Assoc Avian Vet Aust Comm, 1996, pp 155-167.
26. Doneley RJT: Semen collection in the ostrich. Proc Aust Ostrich Assoc, 1996.
27. Doneley RJT: Flock health schemes and preventative medicine: The veterinarian's view. Proc Aust Ostrich Assoc, 1996.
28. Doneley RJT: Ostrich paediatrics. Proc Assoc Avian Vet Aust Comm, 1997 pp 1-20.
29. Doneley RJT, et al: Mycobacterial infection in an ostrich. Aust Vet J 77(6):368-370, 1999.
30. Elston JJ, Beck MM, Scheideler SE: Behavioral analysis of emu chicks and breeding adults. Proc 2nd Intl Ratite Cong, South Africa, 1998, pp 177-180.
31. Fowler ME: Anatomy. *In* Tully TN, Shane SM: Ratite Management, Medicine and Surgery. Malabar, FL, Krieger Publishing, 1996, pp 1-10.
32. Fudge A: Clinical hematology and chemistry of ratites. *In* Tully TN, Shane SM: Ratite Management, Medicine and Surgery. Malabar, FL, Krieger Publishing, 1996, pp 105-114.
33. Hicks-Alldredge KD: Reproduction. *In* Tully TN, Shane SM: Ratite Management, Medicine and Surgery. Malabar, FL, Krieger Publishing, 1996, pp 47-57.
34. Huchzermeyer FW: Diseases of ostriches and other ratites. Agriculture Research Council, Onderstepoort, Republic of South Africa, 1998.
35. Huchzermeyer FW: Veterinary problems. *In* Deeming DC (ed): The Ostrich: Biology, Production and Health. Oxon, UK, CABI Publishing, 1999, pp 293-320.
36. Irons PC, et al: Semen collection and evaluation in the ostrich. *In* Deeming DC (ed): Improving Our Understanding of Ratites in a Farming Environment. Manchester, UK, 1996, pp 157-159.
37. Jakob-Hoff R, Buchan B, Boyland M: Kiwi coccidia: North Island survey results. Kokako Bull Wildlife Soc New Zealand Vet Assoc 6(1):3-5, 1999.
38. Jakob-Hoff R, Twentyman C, Buchan B: Clinical features associated with a haemoparasite of the North Island Kiwi. Proc Joint Conf Wildlife Disease Assoc and Wildlife Soc, NZ Vet Assoc, 2000, pp 23-24.
39. Jakob-Hoff R: Establishing a health profile for the North Island brown kiwi (*Apteryx australis mantelli*). Proc Intl Joint Conf World Assoc Wildlife Vet, Wildlife Dis Assoc, Aust Assoc Vet Conservation Biologists and Wildlife Soc, NZ Vet Assoc, 2001, pp 135-140.
40. Jensen J, Johnson H: Husbandry and medical management of ostriches, emus and rheas. Wildlife and Exotic Animal Teleconsultants, Texas, USA, 1992.
41. Lábaque C, Navarro JL, Martella MB: Chick adoption and subsequent survival in the greater rhea. Proc 2nd Intl Ratite Cong, South Africa, 1998, pp 175-176.
42. Lambrechts H, Huchzermeyer FW, Swart, D: Integrating ostrich Behavior in commercial farming systems. Proc 2nd Intl Ratite Cong, South Africa, 1998, pp 167-169.
43. Love SCJ, Gill HS: Aspergillosis in ostriches: Epidemiology, treatment and diagnosis. Proc 5th Aust Ostrich Assoc Conf, 1995, pp 53-59.
44. Malecki IA, Martin GB, Lindsay DR: Semen collection and production of spermatozoa in the male emu. *In* Deeming, DC (ed): Improving Our Understanding of Ratites in a Farming Environment. Manchester, UK, 1996, pp 159-160.
45. Mouser D: Restraint and handling of the emu. *In* Deeming, DC (ed): Improving Our Understanding of Ratites in a Farming Environment. Manchester, UK, 1996, pp 41-45.
46. Navarro JL, et al: Diet of wild greater rheas in an agroecosystem of southern Córdoba (Argentina). *In* Deeming, DC (ed): Improving Our Understanding of Ratites in a Farming Environment. Manchester, UK, 1996, pp 110-111.
47. O'Malley PJ: An estimate of the nutritional requirements of emus. *In* Deeming, DC (ed): Improving Our Understanding of Ratites in a Farming Environment. Manchester, UK, 1996, pp 92-108.
48. Peach P: Detection of retroviral infections and their application to the detection of a novel retrovirus isolated in ostriches diagnosed with ostrich fading syndrome. Proc Assoc Avian Vet Aust Comm, 1997, pp 143-149.
49. Peat N: The incredible kiwi. Random Century in association with Television New Zealand, 1990.
50. Peirce MA, Jakob-Hoff RM, Twentyman C: New species of Apterygidae in New Zealand. J Nat Hist, in press.
51. Perelman B: Health management and veterinary procedures. *In* Deeming DC (ed): The Ostrich: Biology, Production and Health. Oxon, UK, CABI Publishing, 1999, pp 321-346.
52. Potter MA, Fordham RA, McLennan JA: Reproductive biology of kiwi. *In* Deeming, DC (ed): Improving Our Understanding of Ratites in a Farming Environment. Manchester, UK, 1996, pp 161-162.
53. Raidal SR, Gill JH, Cross GM: Pox in ostrich chicks. Aust Vet J (73):32-33, 1996.
54. Raines AM: Adenovirus infection in the ostrich (*Struthio camelus*). Proc Assoc Avian Vet, 1993, pp 304-312.
55. Romer L: Cassowary Husbandry Manual. Workshop materials, Currumbin Sanctuary Conservation Unit, Currumbin, Queensland, 1997.
56. Ross EJ, Deeming DC, Dawkins MS: Feeding and vigilance Behavior of breeding ostriches in a farming environment in Britain. *In* Deeming, DC (ed): Improving Our Understanding of Ratites in a Farming Environment. Manchester, UK, 1996, pp 24-26.
57. Schaetz L, et al: Investigation into the immune response of ostrich chicks (*Struthio camelus*) after vaccination against Newcastle disease. Proc 2nd Intl Ratite Cong, South Africa, 1998, pp 195-198.
58. Shane SM, Tully TN: Infectious diseases. *In* Tully TN, Shane SM: Ratite Management, Medicine and Surgery. Malabar, FL, Krieger Publishing, 1996, pp 127-146.
59. Sharp PJ, et al: Neuroendocrine control of incubation Behavior in the emu. *In* Deeming, DC (ed): Improving Our Understanding of Ratites in a Farming Environment. Manchester, UK, 1996, pp 162-1163.
60. Soley JT, Groenewald HB: Reproduction. *In* Deeming DC (ed): The Ostrich: Biology, Production and Health. Oxon, UK, CABI Publishing, 1999, pp 129-158.
61. Speer BL: Developmental problems in young ratites. *In* Tully TN, Shane SM: Ratite Management, Medicine and Surgery. Malabar, FL, Krieger Publishing, 1996, pp 147-154.
62. Stewart J: Ratite incubation. Proc Assoc Avian Vet, 1992, pp 358-359.
63. Stewart J: Ratites. *In* Ritchie BW, Harrison GJ, Harrison LR: Avian Medicine: Principles and Application. Brentwood, TN, HBD Intl Inc, 1999.
64. Stewart J: Hatchery management in ostrich production. *In* Tully TN, Shane SM: Ratite Management, Medicine and Surgery. Malabar, FL, Krieger Publishing, 1996, pp 59-67.
65. Van Schalkwyk SJ, et al: The influence of different disinfection protocols on the hatching performance of ostrich eggs. Proc 2nd Intl Ratite Cong, South Africa, 1998, pp 157-159.
66. Vercoe D: Auckland Zoo native fauna conservation centre breeding program: Achievements for the 2001/2002 season. Unpublished internal report, Auckland Zoo, 2002.
67. Verwoerd DJ: Newcastle Disease in ostriches: A review of experimental data. Proc 2nd Intl Ratite Cong, South Africa, 1998, pp 192-194.
68. Verwoerd DJ, et al: Rearing environments around the world. *In* Deeming DC (ed): The Ostrich: Biology, Production and Health. Oxon, UK, CABI Publishing, 1999, pp 191-216.
69. Vorster JH, Olivier AJ: Diseases affecting the central nervous system of ostriches in southern Africa. Proc 2nd Intl Ratite Cong, South Africa, 1998, pp 201-204.
70. Westcott DA, Reid KE: Use of medetomidine for capture and restraint of cassowaries (*Casuarius casuarius*). Aust Vet J 80(3):150-153, 2002.
71. Williams KM, et al: Growth, sexual development and carcass composition in intact and surgically or hormonally gonadectomised male and female emus. Proc 2nd Intl Ratite Cong, South Africa, 1998, pp 75-80.

CHAPTER
42

Management of Zoo and Park Birds

LORENZO CROSTA, DVM, LINDA TIMOSSI, DVM;
MARCELLUS BÜRKLE, DVM

The role of the veterinary staff in a zoological setting involves the active promotion of animal health and well being in addition to the treatment of overt diseases. It is uncommon for a zoo veterinarian to be involved strictly in avian medicine, although this may occur when a veterinarian is employed in a zoological garden that is a bird park. More commonly, an avian veterinarian will be consulted when problems with avian species are encountered in a mixed species zoologic collection.

The Purposes of a Zoo or Bird Park

A zoological garden or bird park, whether private or open to the public, is a place where animals (birds) are exhibited. One of the most important concerns of the zoo is the appearance of the displayed animals. Zoos are constantly under pressure from animal rights activists. In many countries, including the USA, zoological gardens have legal requirements, including animal welfare rules and legislation, to which they must adhere.

Additional purposes or goals of zoologic parks include:

- Maintenance of the genetic pool of selected species.
- Geographically orienting management and exhibition of animals according to their natural distribution (eg, South American birds located contiguously).
- Gaining biologic and ecologic information regarding the applicable species.
- Performing specific research projects in areas of nutrition, behavior, genetics and disease.

Routine Zoo Veterinary Work

The veterinarian's work in a zoo involves adherence to strict protocols including meticulous record keeping. A thorough understanding of the concepts and challenges of aviculture is required. Management, husbandry and medical and surgical protocols must be developed and performed (Table 42.1).

Medical Equipment

Zoo veterinary equipment for the avian collection does not differ significantly from that encountered in a normal exotic animal clinic (Table 42.2). Its availability will depend on budget and ease of outside sourcing. Housing for hospitalized birds is outlined in Table 42.3.

Depending on local veterinary support, the zoo veterinary clinic may need a full range of surgical and diagnostic tools including microbiology, hematology, blood chemistry and cytology, histopathology, toxicology, virology and special tests, some which may be submitted to outside laboratories.

The Importance of Biology and Taxonomy

Recent discoveries in zoology, field biology, morphology, ethology and DNA-based genetics need to be monitored and, where appropriate, implemented into the medical protocols. Understanding these issues allows the zoo veterinarian to form an idea about the correct husbandry conditions (eg, environment, temperature, diet) to provide. Taxonomy of a closely related species for which good medical information is available will be invaluable in the approach to any novel species.

Table 42.1 | Recommended Veterinary Routines for Zoo and Park Birds

Daily
- Evaluate hospitalized patients
- Review laboratory results received
- Develop or alter treatment based on the above
- Examine nursery birds, park exhibits and breeding birds, making appropriate notes, examining individuals, obtaining samples and retrieving birds for hospitalization as needed
- Perform any required surgeries

Weekly
- Interdepartmental meetings to review schedules and plans for routine procedures

Monthly
- Routine deworming and fecal examinations
- Vaccinations as appropriate

Yearly
- Annual physical examination of every animal in the park; plan further testing, if warranted
- Evaluate unsuccessful breeding pairs

Table 42.2 | Recommended Medical Equipment for Zoo and Park Birds

Endoscopy
- Both rigid (standard avian) and flexible (larger bird) endoscopes of various diameters

Surgery
- Standard surgical equipment and facilities for exotics

Anesthesia
- A portable anesthesia machine for larger patients such as ratites

Radiology
- A portable x-ray machine is often needed in a zoo

Ultrasound
- A portable ultrasound machine equipped with various sized probes

Cages, hospital cages and aviaries
- A variety of enclosures for various avian patients is desirable (see Table 42.3)

Table 42.3 | Recommended Cages for Different Avian Patients

Avian Patient	Recommended Cage
Critically ill birds	They need a warmed cage, ideally an Intensive Care Unit (ICU). This applies to any avian patient in critical condition. The hospital cage should be connected to an O_2 delivery system, with the temperature and humidity controlled.
Potential infectious patients	A zoo must have a special area for animals that may carry an infectious disease. This area should be separate from the quarantine for incoming birds.
Psittacines	See Chapter 7, Emergency and Critical Care.
Birds of prey (diurnal and nocturnal)	See Chapter 40, Management of Raptors.
Cranes, storks and flamingos	Although these animals have totally different feeding habits, they have similar hospital needs. Flooring must provide adequate traction. Hard rubber mats are ideal since they can be disinfected. Flamingos must be housed with access to the water and food blend that they consume via filtration through their beaks.
Small passerines	Some species may be very shy and easily stressed, so English-style cages (with 3 solid walls) are preferred.
Ratites	See Chapter 41, Management of Captive Ratites.
Penguins and auks	Penguins belong to the Order Sphenisciformes. There are 17 living species of penguins, divided into 6 genera. All penguin species live in the southern hemisphere. The term "auk" includes species of the Alcidae family, 21 species in the world, which include the guillemot, or murre (*Uria aalge*), razorbill (*Alca torda*) and puffin (*Fratercula artica*). Penguin and auk species require species-specific environments, but they all need to swim, preferably in a relatively cold saltwater pool. If a zoo includes penguins and/or auks among its species, an appropriate area should be built for quarantine and hospitalization room(s). An ideal room for nursing these aquatic birds meets the following requirements: 1. A saltwater pool within easy access of the birds. 2. Environmental air and water temperatures cooled to 0° C (32° F). 3. Water and air filtration systems (to decrease the potential for aspergillosis).
Anseriformes	See Chapter 36, Management of Waterfowl.
Galliformes	See Chapter 38, Management of Galliformes.
Columbiformes	Pigeons and doves, like the two preceding groups, vary in size, husbandry and dietary requirements. For short-term nursing, they can be kept in cages similar to ones for psittacines or birds of prey. Some of the shyest of the species (ie, the large crowned pigeons [*Goura* spp.]) should be kept in a very quiet environment (see Chapter 37, Management of Racing Pigeons).

Fig 42.1 | Cooperation with the gardeners helps achieve pleasant looking cages, while avoiding toxic plants.

Fig 42.2 | Some bird species, such as these rockhopper penguins (*Eudyptes crestatus*), have extreme temperature and humidity requirements.

The Importance of Teamwork

A common problem encountered during setup of a new exhibit or refurbishing an existing one is the lack of team work. Often the architects, biologists, curators, keepers and veterinarians have different perspectives on what the ideal exhibit should be for a given species, but all these points of view must be collated before the building or refurbishing work begins. The basic issues of an exhibit design should be addressed in the preplanning phase. Once the building work has started, it is often impossible or too expensive to make changes.

Experienced, committed keepers understand the behavior and personalities of the animals. They know the daily cleaning and feeding routines and can offer important suggestions about materials best suited for furniture, fencing, drainage and access to aviaries. Keepers who work with aggressive birds (eg, ratites and large cranes) can be helpful in developing solutions for their safe transport from the exhibit to holding areas.

The veterinarians and gardeners, or horticultural department if there is one, are involved in the selection and control of the vegetation to be planted, thereby eliminating the use of toxic plant species inside the exhibit (Fig 42.1).

Additionally, keepers' suggestions are invaluable when planning service areas and pest control.

HOUSING

Housing is one of the most important aspects of keeping birds in a zoo. In an up-to-date setting, animals must not only be healthy but also be exhibited in the most "natural" environment possible.

Temperature and Humidity

Most commonly kept zoo and park birds do well in the average temperate climate, but exceptions do exist. Most tropical birds, especially smaller ones, need temperature-controlled housing for winter, in addition to warm spots, even during summer nights. There also are birds that need some accessible areas away from extreme heat to thrive during the summer months (Fig 42.2). Some of these species are easily identified (eg, penguins, snowy owls, gyrfalcons). Bird species that are represented in different climates by multiple subspecies, such as the peregrine falcon (*Falco peregrinus*) or the osprey (*Pandion haliaetus*), offer special challenges. It is better to obtain a local subspecies in order to avoid stress due to inappropriate climate.

Light Cycles

Most bird species are highly dependent on photoperiod for their reproductive cycle, but other physiological issues (eg, molting) also are light-cycle dependent. This is important for birds originating from the extreme northern or southern latitudes and must be taken into consideration when designing an exhibit for these species.

Flooring

Flooring is of primary importance in a birdhouse (Figs 42.3-42.5). When approaching bird management, the avian veterinarian must keep in mind two important things:

- Avian species have evolved with multiple anatomic and functional variations of the feet (eg, webbing, talons, feet for wading), which require the appropriate caging materials in captivity.
- Birds are bipedal; therefore, damage to one foot places 100% of the weight-bearing load on the remaining contralateral foot. For all but the smaller species, this increased load and decreased mobility can quickly lead to affliction of the plantar surface of the remaining foot.

Fig 42.3 | Natural flooring and a proper diet promote healthy feet, as in these demoiselle cranes (*Grus* [*Anthropoides*] *virgo*).

Fig 42.5 | Aquatic species, such as these little blue penguins (*Eudyptula minor*), need a proper pool to allow swimming and prevent foot problems.

Roofing

Some non-flying species (eg, ratites) do not need a roof. Also, some birds have limited ability to fly because they have been pinioned or their wings are regularly trimmed. This often applies to ducks, geese, swans, flamingos and sometimes cranes and storks. When a roof is designed, several factors are taken into consideration.

It is important to know if the birds will or will not be on exhibit. If so, the roof will have to be both effective and have a natural look. "Invisible" nets are available and work well in these cases. Because some species may bite and chew on the roof, this should be considered when selecting roofing material.

Another factor is the ability of a species to climb and become trapped in a mesh roof. Roofing for birds of prey and other birds that might become trapped in mesh can be made with wooden lathing strips, 2 cm thick and 10 cm wide. The strips are arranged horizontally, 5 cm apart, allowing resting birds to see the sky and receive fresh air, rain and snow, if advisable; when the birds are flying, this roof will appear as a solid wall

E. Albertini, Monticello Breeding Center, Italy

Fig 42.4 | Natural displays should include the different plants that occur in a bird's natural habitat. Marsh owl (*Asio capensis*) shown.

and the birds will not fly into it (Figs 42.6a,b).

Vegetation

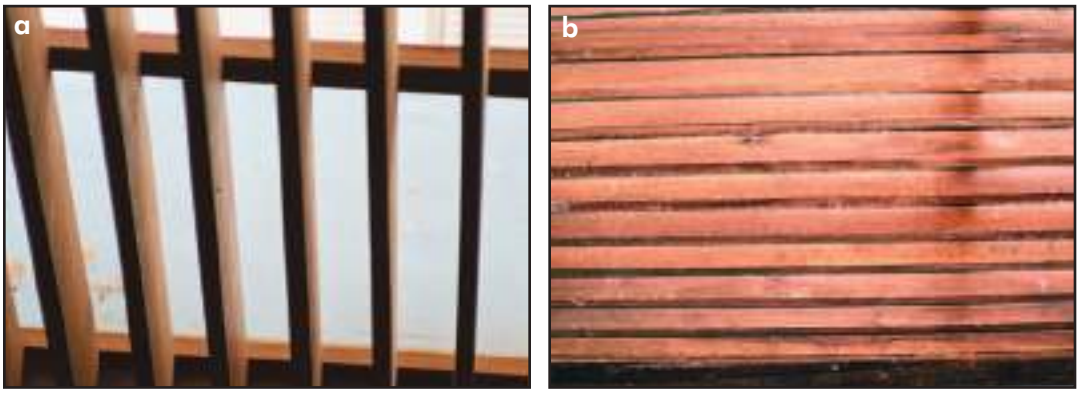
Most bird species like plants and branches in their aviaries for hiding, perching and nesting, but not all trees are appropriate for planting. Normally, large zoos employ botanists or experienced gardeners who can help veterinarians and curators select local plants suitable for installation in large aviaries or exhibits.

Perches

Perches, whether provided alone or in conjunction with planted trees, must offer a good base for both rest and exercise. Ideally, perches should provide a variety of textures, diameters and elasticities. When perches are made of a variety of different materials, sizes and shapes, they will provide the birds' feet with exercise, equal weight distribution and natural nail trimming (Figs 42.7a-c).

Food and Water Delivery Systems

In most bird collections, food and water are provided twice daily. During the summer and year long in warm climates, food and water easily become polluted by bacteria, fungi and protozoa and can be the source of serious health problems. Feeding schedules and equipment must be adjusted according to the type of food (eg, seeds, dry, wet, fish, live food) used for the different bird species. In general, hygiene is the most important issue. Food and water bowls must be changed daily, and good cleaning and disinfection procedures are necessary to avoid spreading pathogens from one enclosure to another.



Figs 42.6 a,b | A properly designed roof for flying birds (ie, birds of prey) will appear to the birds as open air or solid, depending on perspective.

Fig 42.7 | A selection of branches and other natural perches to exercise the feet is a must in exhibits for wild species. **a)** Great grey owl (*Strix nebulosa*). **b)** Red-tailed black cockatoo (*Calyptorhynchus banksii*). **c)** A variety of branches in an aviary.

In some facilities, water is delivered through an automatic watering system. The system's pipes must be checked on a regular basis for hydrophilic bacteria, especially *Pseudomonas* sp. Cleaning and disinfecting the pipes should take place on a regular basis. Natural exhibits with a lake or waterfall as the main source of the birds' water intake should have a high-quality filtration system. Weekly bacteriologic and chemical tests (eg, nitrogen and chlorine) should be performed using one of the available commercial kits. Using a dipping media[a] for urine bacteriology allows identification of type and number of bacteria in the water.

Nests

For many species, nests are necessary for reproduction and may be a stimulus for breeding activity. Although some exceptions do occur (eg, macaw and cockatiel), most psittacines will not breed without a nest box.

Searching for a nest site and building a nest can play important roles in mating behavior and breeding success. Less commonly, species like king penguins (*Aptenodytes patagonica*) do not build nests but incubate their single-egg clutches on their feet, which are covered by a skin fold. Even these birds need to be provided with an adequate breeding area where they are protected against weather and feel safe from actual or perceived threats, such as predators.

Choosing the best nest sites and adequate nest shapes for different types of birds falls to the biologists and curators, but the veterinarian's medical input should be included in the decision-making process (Figs 42.8, 42.9).

Nest Hygiene

Depending on the species, nests can easily become dirty with excrement from the offspring. Parasites and bacteria proliferate in this organic medium. Consequently, selection of bedding material is important. In general, bedding material should be clean and free of pathogens. Inadequate bedding material has been reported as a cause of aspergillosis in neonatal parrots and other avian

Fig 42.8 | Nest sites should meet species-specific needs. A diamond dove (*Geopelia cuneata*) is shown.

Fig 42.10 | A nest box for lories, built using wire mesh for the floor, allows the liquid feces to drop and ensures better ventilation.

species. Ectoparasites can be avoided by adding 5% carbaryl powder to the bedding material at the beginning of the breeding season. Bedding material should be routinely tested for bacterial and fungal contamination. Toxic substances also can cause problems in this respect. Wood shavings, widely used as nest bedding, must exclude the presence of paints, resins and wood preservatives.

The condition of the bedding material must be monitored during the breeding season. This should be undertaken in cooperation with the curator or animal keeper in order to not disturb the breeding pair. In some species, like lories and lorikeets that feed on a liquid-based diet resulting in voluminous stools, it may be necessary to change or add new bedding material during egg incubation or while the young are still in the nest. For these birds, a specially designed nest box has proven to be effective against pollution. Instead of a wooden floor, the nest box is built using wire mesh, allowing the liquid feces to drop through and ensuring better ventilation (Fig 42.10).

Cleaning and disinfecting the nest after the breeding season should be carried out carefully. Wooden nest boxes may require replacement. Natural trunks should

Fig 42.9 | Empty trunks can be used as nests for selected species. Shown is a palm trunk nest provided to Pesquet's parrots (*Psittrichas fulgidus*).

be cleaned as carefully as possible, and appropriate disinfecting solutions, which allow the birds to use the nesting site again, should be used. In species like the gentoo penguin (*Pygoscelis papua*), which uses stones for nesting, the stones should be removed and carefully cleaned after the breeding season. Permanent nesting sites, eg, caves for puffins (*Fratercula arctica*) or other cave-breeding species, must be designed for ease of cleaning and disinfecting. The design of a nest should be a compromise between the natural breeding behavior of the species, its specific hygiene requirements and the captive situation (Figs 42.11a,b).

Controlling the Nest Site

Nests should be designed in a way that allows evaluation of parents, eggs and chicks (Fig 42.12). In case of irregularities in the behavior of the parents, evaluating and/or removing the offspring may be necessary.

Risks to the Offspring

Leaving the nest for the first time after weaning is a risky situation for young birds, and fractures or other traumatic injuries can occur. Therefore, nests should be designed to minimize the risk of fledglings flying into wire mesh or other dangerous structures of the cage. To help prevent such accidents, tree branches can be placed in front of the nest a few days before fledging.

DISEASES

Identification of Sick Animals

Routine controls include daily rounds during which all the aviaries and exhibits are inspected, and, if war-

Figs 42.11 a,b | In an exhibit housing cave nesters and bird species that build nests using stones, special attention to the hygiene involved in such an exhibit is required. Shown are gentoo penguins (*Pygoscelis papua*) on a nest and a pair of Humboldt penguins (*Spheniscus humboldti*) in the cave nest.

E. Albertini, Monticello Breeding Center, Italy

Fig 42.12 | Nests should be designed in a way that allows control of parents, eggs and chicks. A great grey owl (*Strix nebulosa*) with a 1-day-old chick is shown.

ranted, samples are collected for laboratory evaluation (see Table 42.4).

Species-specific Diseases

Viral diseases tend to be more species-specific than bacterial or fungal diseases. In parrots, one might see psittacine circovirus, avian polyomavirus or Pacheco's disease. Other viral diseases that are specific to selected bird groups and encountered in zoo settings are avian poxvirus infections and herpesvirus infections (HV).

Some HV may be extremely dangerous for flocks of sensitive species. HV are generally pathogenic for a single group of birds, but other bird families, even those not very taxonomically close, may sometimes be susceptible. For example, pigeon herpesvirus causes disease in pigeons, Arctic falcons and sometimes in owls. Typically, herpesvirus infections cause neoplastic, hemorrhagic or necrotic lesions. A group of these diseases causes necrotic hepatosplenitis in falcons, eagles, owls, cranes, storks and pigeons.

When an outbreak of disease occurs in a zoo, bird movement should be limited.

Multi-species Diseases

Most bacterial and viral infections potentially are transmittable through many avian species and orders. In this respect, an extremely strict routine for disinfection of selected areas in the zoo (eg, food preparation, chick hand-rearing station, quarantine and, of course, the hospital) is important. A good strategy for avoiding bacterial resistance to disinfectants is to rotate the use of three to four different disinfectants; at selected times, a fogger for disinfecting the most unreachable spots also can be used.

It is extremely important to know the most common interspecific diseases in order to rate the risk of cross-species transmission in case of an outbreak. Bacterial examples are *Mycobacterium avium, Yersinia pseudotuberculosis, Salmonella* spp., *Pasteurella multocida, Chlamydophila psittaci, Erysipelothrix rhusiopathiae* and *Listeria monocytogenes*.

Paramyxovirus-1(PMV-1) is distributed worldwide, and all bird species are considered susceptible to PMV-1. Influenza A viruses have been recovered from several different bird orders, including species very common in zoos and bird parks, such as Anseriformes, Galliformes, Falconiformes, Psittaciformes, Sphenisciformes and Gruiformes. It also has been recovered from some mammals.

Introduced Diseases

The introduction of new animals may lead to the introduction of unexpected diseases. The chance of introducing a disease through ectoparasites or other pests traveling with the crates, cages or boxes of newly arrived animals also must be considered; hence, it is important to institute strict quarantine measures and thorough disinfection (if

Table 42.4 | Routine "On-site" Check for Park and Zoo Birds

1. Behavior
   Look for any behavioral changes (the bird is quieter, sleeps, is nervous, turns away from the partner).
2. Physical posture
   The bird sits hunched over.
   The bird sleeps standing on both feet (healthy birds usually sleep on one foot).
   The bird wags tail (indicates respiratory diseases or egg retention).
   The bird favors its extremities (one or both wings are hanging down slightly; the bird obviously tries not to strain one of its feet).
3. Plumage
   At the first stage of a disease, birds quite often ruffle a few neck or head feathers (which also may be just a threatening posture).
4. Eyes
   At the very beginning of a disease, the animal's eyes are slightly—very slightly—closed. You will have to take a very close look. At the same time, the eyes are no longer shining. Seriously ill birds may have the eyes completely closed.
5. Stool
   Pay attention to the quantity, color and consistency. Increased or reduced stool quantity may indicate a disorder.
   Also feces' color and consistency should be monitored.
6. Ingestion of food and water
   Is the ingestion of food and water normal? Does the bird eat or drink more or less than usual?

not destruction) of crates, cages or boxes used for the importation of animals.

Zoonosis

The incidence of diseases acquired by humans from birds is low (Table 42.5). The most frequent diseases of concern are usually chlamydiosis and salmonellosis. Quarantine and strict hygiene measures are the only effective methods for avoiding bird-to-human transmission of diseases. Most zoonoses are transmitted via the fecal/oral route. Birds in contact with visitors should be tested on a regular basis for potential zoonoses, and results must be archived to prove negative test results in case of a new occurrence. Positive birds should be removed from visitor contact for security reasons and treated or euthanized as appropriate.

QUARANTINE

Quarantine guidelines have been established by the American Zoo and Aquarium Association (www.aza.org) and should be adapted to each situation. Quarantine measures include all birds that are newcomers to the collection (and those that return after being on loan to another facility or from a different exhibit). All newcomers are subjected to a minimum 6 weeks' quarantine period. Multiple quarantine rooms of varied sizes should be available for this purpose. All quarantine rooms should have separate, low-pressure ventilation systems. After a short, 2- to 4-day period of adaptation, each bird is subjected to a thorough medical examination, which includes the procedures listed in Table 42.6.

Birds may be integrated into the collection after the quarantine period in the absence of any detectable disease or infection. Special attention should be paid to behavior and feeding of new animals in the first weeks of quarantine. Food and water should be supplied in a manner equivalent to the animals' former feeding conditions at the original facility. Changes to the food and feeding schedule should be made gradually to avoid stress to the birds. A separate keeper for the quarantined birds is preferred to avoid disease transmission. Separate clothes, boots and hand and shoe disinfection baths are critical.

COMMON SENSE NUTRITION

Working in a zoo presents the veterinarian with a variety of different species, often with very unusual or specialized diets. In most circumstances, specific commercial diets are not readily available. Hence, it is important to work jointly with other professionals such as zoologists, biologists and nutritionists in an effort to customize the most appropriate diet for a given species.

In addition to the foods manufactured specifically for psittacines, diets are also commercially available for mynah birds, canaries, small finches, pigeons, thrushes, cranes, emus, flamingos, game birds, ostriches, waterfowl, aquatic ducks and toucans. Other diets are often used according to "local traditions," "avicultural myths" and keepers' experiences, which may or may not be ideal. For best results, a veterinarian should work with a nutritionist in developing a feeding plan.

Imitation of natural nutrition can be very successful, but this may not always be possible or desirable for the following reasons:

- Some or all diet components are not available.
- Nutritional requirements in captivity are different from those in the wild.
- Some foods cannot be digested in the absence of other unknown or unavailable items.
- Birds do not recognize the offered diet as food.

If a diet for birds has to be revised, especially in cases of birds for which little accurate data are available or when birds have been fed for a long time on an alternate diet, discretion is needed. Generally speaking, if a bird flock has been fed a locally developed diet for a prolonged period, it can be assumed that the diet and general management were reasonable if the birds maintain good

Table 42.5 | Zoonotic Diseases Potentially Present in a Bird Park

Disease	Etiology
Bacterial	
Campylobacteriosis	*Campylobacter jejuni*
Colibacillosis	*Escherichia coli*
Erysipeloid	*Erysipelothrix rhusiopathiae*
Listeriosis	*Listeria monocytogenes*
Miscellaneous bacterial infections	*Klebsiella* spp., *Pseudomonas* spp., *Vibrio* spp.
Pasteurellosis	*Pasteurella* spp.
Psittacosis	*Chlamydophila psittaci*
Salmonellosis	*Salmonella typhimurium*
Tuberculosis	*Mycobacterium avium, M. genavense*
Yersiniosis	*Yersinia pseudotuberculosis*
Mycotic	
Aspergillosis	*Aspergillus fumigatus*
Cryptococcosis	*Cryptococcus neoformans*
Dermatophytosis	*Trichophyton gallinae, Microsporum gypseum*
Histoplasmosis	*Histoplasma capsulatum*
Viral	
Influenza A	Orthomyxovirus type A
Newcastle disease	Avian Paramyxovirus type 1
West Nile virus	Flavivirus

physical condition. Normal laboratory examination results, successful reproduction, good quality feathering and a normal physical examination would warrant a gradual and cautious approach to dietary alterations (see Chapter 4, Nutritional Considerations).

BEHAVIORAL CONSIDERATIONS

In zoo and bird park collections, particular attention should be given to the choice of animals occupying neighboring cages due to potential incompatibility among species or individuals and aggressive or challenging behavior during the breeding season. This is particularly true for some parrot species, such as the hawk-headed parrot (*Deroptyus accipitrinus*), which seems to be affected by the presence of other pairs of the same species. Male macaws (*Ara* spp.) are protective of their partners during the breeding season, and a neighboring male of the same species may affect the mating behavior of the pair and create a stressful situation that can compromise breeding success; males are more occupied with challenging each other than with breeding.

On the other hand, parrot families such as white cockatoos (*Cacatua* spp.) appear to benefit from the presence of conspecific birds. If they are housed to allow visual and vocal contact, they will display and call to each other, and this situation might contribute to breeding success.

Sound knowledge of the natural behavior of the species is helpful when choosing the most suitable species to be neighbors. Sometimes it is only a question of common sense when choosing neighboring birds so that one of the species is not stressed, especially in a zoo collection where birds and their natural predators are maintained. An incorrect choice can create a stressful situation for the animals and will lead, at best, to the abnormal appearance and behavior of the animals. In most circumstances, a stressed bird will soon become a sick bird as well.

Table 42.6 | Recommended Medical Check-up for Birds in Quarantine

- Bacteriological analysis (cloacal, choanal, tracheal)
- Mycological analysis (cloacal, choanal, tracheal)
- Parasitological analyses (endo- and ectoparasites)
- Checking for viral infections, (eg, circovirus and polyomavirus in the case of parrots and Newcastle disease for all birds)
- Checking for *Chlamydophila* (test for antigens and antibodies)
- Hematological and blood chemistry analysis
- Other tests, depending on the species

MIXED SPECIES EXHIBITS

The idea of keeping different species in a mixed exhibit is common in zoos. The major challenge is to weigh the benefits and risks that a mixed exhibit can offer. It is easy, practical and economically more convenient to use one enclosure to display different species. Further, a mixed exhibit can be more attractive and educational for visitors, and a combined species exhibit can have a positive effect on the physical and mental behavior of the animals. From an educational point of view, most zoos prefer that animals belonging to the same geographical area be shown in the same mixed exhibit.

Despite these advantages, the negative points of a mixed-species exhibit are the risk of aggression among the animals and competition for position, space and food in the exhibit itself. It is important to be knowledgeable regarding the natural environment and behavior of the species to be combined and even more important to know the temperament of the individual animal in a mixed-species exhibit.

To successfully develop a mixed-species exhibit, specific guidelines should be followed:

- Avoid combining animals that occupy the same ecological niche because they will compete for space and food. Try to use all the space of the enclosure, combining terrestrial species with arboreal and/or aquatic species.
- Primates are always problematic to combine in mixed-species exhibits due to their inquisitive nature and the fact that they are often aggressively territorial in captivity. Nevertheless, there are examples of birds successfully exhibited with primates. Golden lion tamarins

(*Leontopithecus rosalia*), screech owls (*Otus* spp.) and red-capped cardinals (*Paroaria* spp.) have successfully been housed together.
- Avoid housing closely related species together in order to prevent the risk of hybridization.
- Provide an abundance of items the animals may compete for: perches and branches of different sizes, feeding points, nesting areas and shadows. For example, when keeping multiple hummingbirds together, a bird can be harassed by its cage mates when there are insufficient feeders (although occasional mock fights are normal behavior in hummingbirds).
- Provide the animals with the option of hiding from others, when necessary, by offering visual barriers such as plants and rocks.
- Avoid combining species that can be antagonistic in nature.
- Combine animals of different sizes when working with animals occupying the same niche. Large species have a tendency to accept the presence of small animals. Nevertheless, the smaller species may challenge the larger if the species is of a particularly aggressive or protective nature. Aggressive species can be combined with others only in a very large exhibit.

Combining birds of different sizes requires additional consideration. For example, very small nocturnal birds of prey (eg, *Athene* spp., *Glaucidium* spp.) can be housed with some waterfowl, but particular care should be taken when combining toucans (*Ramphastidae*) with passerines or other small birds like doves, to avoid predation. Cranes, waterfowl, ibises and storks are often displayed together. Some parrots also may be kept together in walk-through aviaries. Several lory (*Loriidae*) species are commonly housed together in an aviary exhibit.

For birds that have the same or similar diets, or the diet of one species represents part of the diet of another, adequate distribution of food can be accomplished by providing feeding sites accessible to only one of the species (eg, flying vs non-flying species) or providing the birds with additional feeding sites.

Some examples of successful mixed-species exhibits include:
- Bali mynah (*Leucopsar rothshildi*) with Asian glossy starling (*Aplonis panayensis*), Prevost's squirrel (*Callosciurus prevostii*) and mouse deer (*Tragulus* spp.)
- Bali mynah with chevrotain (*Tragulus napu*)
- Goffin's cockatoo (*Cacatua goffini*) and Eleonora's cockatoo (*Cacatua g. eleonora*) with bettongs (*Bettongia penicillata*)
- Emu (*Dromaius novaehollandiae*) and cassowary (*Casuarius* spp.) with red kangaroo (*Macropus rufus*) and dama wallaby (*Macropus eugenii*)
- Crowned crane (*Balearica* spp.), marabou stork (*Leptoptilos crumeniferus*), storks (*Ciconia* spp.) and ostrich (*Struthio camelus*) with giraffe (*Giraffa camelopardis*) and antelope (*Tragelaphus* spp. and *Gazella* spp.)
- Yellow weaver (*Ploceus subaureus*), black-winged bishop (*Euplectes hordaceus*), golden-breasted starling (*Cosmopsarus regius*) and speckled pigeon (*Columba guinea*) with dik-dik (*Madoqua dirkii*).

WORKING (SHOW) BIRDS

Most zoo parks and some zoos have bird shows and/or walk-through aviaries, putting both visitors and birds into direct contact. The potential risk of bird-to-human and human-to-bird disease transmission has been addressed.

The training and working schedules of show birds are readily accepted by some animals that benefit both mentally and physically from this activity, but other birds may be stressed by this routine. This work-derived stress will influence the birds' immune systems and make them more susceptible to both infectious and non-infectious diseases. In this respect, good teamwork with trainers is important in identifying subtle changes before they become problems.

Generally speaking, show birds are individually examined two to four times per year. This examination includes a discussion with the trainers and careful inspection of the resting area where the birds live. Tests for major viral diseases are performed when a new bird is introduced to the group and then repeated as needed (see "Quarantine"). Birds are regularly screened for parasites, especially if they are free-flying animals. Blood tests, including CBC and basic biochemistries, are performed annually. Tests for chamydiosis and other bacterial diseases are performed at least twice a year, especially when direct contact with visitors occurs. Finally, a fecal screening for the presence of acid-fast bacteria is performed annually on free-flying animals.

COMMON ACCIDENTS AND ESCAPES

Zoo birds are exposed to some of the same risks as pet and aviary birds, such as poisoning, foreign body ingestion and aggression by cage mates. Situations may be unique to zoo settings.

Aggression can be inflicted by a conspecific bird or by a cage mate belonging to a different species (Fig 42.13) (see "Mixed-species Exhibits"). In the case of trauma, therapy follows emergency protocols.

When a bird escapes and flies into a dangerous animal's exhibit, it will probably be killed. If the predator is not

Fig 42.13 | A conspecific bird can inflict injury due to aggression. A bite wound on the wing of a Humboldt penguin (*Spheniscus humboldti*) is shown.

dangerous to humans (eg, small felids or canids), the bird can be netted and removed. In any other case, measures should be taken to anesthetize the predator and avoid further escape of the bird by putting a net in front of the hiding place before attempting to rescue it or being ready with a water hose to soak the bird's feathers to prevent further escape.

VETERINARIANS AND BIRD CONSERVATION

It is believed that a natural rate for extinction is about one species every 100 years. But since 1800, more than 100 bird species have become extinct, which is 50 times higher than the natural rate. Zoo veterinarians must become more involved in bird conservation programs, to prevent disease and other factors from contributing to species extinction (Fig 42.14).

Veterinarians' contributions to conservation programs can also include clinical care for individuals of highly endangered species (the highest standard veterinary care must be provided) and the design and implementation of breeding and release programs.

Fig 42.14 | Spix macaws (*Cyanopsitta spixii*) are the most emblematic endangered parrot species.

Domestically bred birds must be fully evaluated for diseases prior to becoming part of a reintroduction program. A health check for psittacines might include the following: physical examination, complete blood count analysis, fecal parasitology, cloacal and choanal cultures, radiographs and endoscopy (biopsy) as well as testing for avian influenza, circovirus, paramyxovirus, herpesvirus, *Chlamydophila,* adenovirus and avian tuberculosis.

The veterinarian may lend his or her expertise to the release program and subsequent monitoring and handling of the subject birds.

Product Mentioned in the Text

a. Uriline, bioMérieux SA, 69280 Marcy-l'Etoile, France, Phone +33-4-78872000, http://www.biomerieux.com

References and Suggested Reading

1. Abrey A: The management of a multi-species bird collection in a zoological park. *In* Tully TN, Lawton MPC, Dorrestein GM (eds): Avian Medicine. Oxford, UK, Butterworth-Heinemann, 2000, pp 364-385.
2. Aguilar RF: Order Falconiformes (hawks, eagles, falcons, vultures): Raptor medicine and surgery. *In* Fowler ME, Cubas ZS (eds): Biology, Medicine, and Surgery of South American Wild Animals. Ames, Iowa State University Press, 2001, pp 118-124.
3. Angel R, Plassé RD: Developing a zoological avian nutrition program. Proc Am Assoc Zoo Vet, 1997, pp 39-43.
4. Asterino R: Diseases and care of wild passerines. *In* Rosskopf WJ Jr, Woerpl RW (eds): Diseases of Cage and Aviary Birds. Baltimore, Williams & Wilkins, 1996, pp 965-980.
5. Baer JF: A veterinary perspective of potential risk factors in environmental enrichment. *In* Shepherdson DJ, Mellen JD, Hutchins M (eds): Second Nature, Environmental Enrichment for Captive Animals. Washington, Smithsonian Institution Press, 1998, pp 277-301.
6. Bailey TA, et al: The medical dilemmas associated with rehabilitating confiscated wildlife: Experience at the environmental research and wildlife development agency, Abu Dhabi, monitoring the recovery of Houbara bustards (*Clamydotis u. macqueenii*) from avian pox and Newcastle disease. Proc European Assoc Zoo Wildlife Vet, 2000, pp 237-241.
7. Bauck L: Psittacine diets and behavioral enrichment. Sem Avian Exotic Pet Med 7(3): 135-140, 1998.
8. Beklova M, Pikula J: Hazards of rodenticidal baits to game birds. Proc European Assoc Zoo Wildlife Vet, 2000, pp 249-252.
9. Breitweiser Mullins BA: Fluid therapy in penguins. Proc Assoc Avian Vet, 1993, pp 158-160.
10. Clubb SL: Aviculture medicine and flock health management. *In* Altman RB, Clubb SL, Dorrestein GM, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 101-121.
11. Coles BH: Galliformes. *In* Tully TN, Lawton MPC, Dorrestein GM (eds): Avian Medicine. Oxford, UK, Butterworth-Heinemann, 2000, pp 266-295.
12. Cubas ZS: Order Piciformes (toucans, woodpeckers) medicine: Family Ramphastidae (toucans). In Fowler ME, Cubas ZS (eds): Biology, Medicine, and Surgery of South American Wild Animals. Ames, Iowa State University Press, 2001, pp 188-199.
13. Curro TG, Langenberg J, Paul-Murphy J: A review of lameness in long-legged birds. Proc Avian Vet, 1992, pp 265-270.
14. Degernes LA: Introduction to wild bird medicine. Proc Basic Avian Med Symposium, Assoc Avian Vet, 1994, pp 97-41.
15. Degernes LA: Toxicity in waterfowls. Sem Avian Exotic Pet Med 4(1):15-22, 1995.
16. De Herdt P, Devriese L: Pigeons. *In* Tully TN, Lawton MPC, Dorrestein GM (eds): Avian Medicine. Oxford, UK, Butterworth-Heinemann, 2000, pp 312-338.
17. Doest OEA: Macaw reintroduction to prevent extinction: Fiction or reality? Proc Assoc Avian Vet, 2000, pp 145-148.
18. Dorrestein GM: Medicine and surgery of canaries and finches. In Rosskopf WJ, Jr, Woerpl RW (eds): Diseases of Cage and Aviary Birds. Baltimore, Williams & Wilkins, 1996, pp 915-927.
19. Dorrestein GM: Passerines. In Altman RB, Clubb SL, Dorrestein GM, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 867-885.
20. Dorrestein GM: Passerines and exotic softbills. In Tully TN, Lawton MPC, Dorrestein GM (eds): Avian Medicine. Oxford, UK, Butterworth-Heinemann, 2000, pp144-179.
21. Dorrestein GM, et al: Iron in zoo-animals, frequency and interpretation of the findings. Proc European Assoc Zoo Wildlife Vet, 2000, pp 243-248.
22. Echols SM, Speer BL: Management of avian flock emergencies. Vet Clin No Am Exot Anim 1(1):59-75, 1998.
23. Fowler ME: Order Phenicopteriformes (flamingos): Biology, management in captivity, and medicine. *In* Fowler ME, Cubas ZS (eds): Biology, Medicine, and Surgery of South American Wild Animals. Ames, Iowa State University Press, 2001, pp 95-102.
24. Fowler ME: Order Anseriformes (ducks, geese, swans): Captive management and medicine. *In* Fowler ME, Cubas ZS (eds): Biology, Medicine, and Surgery of South American Wild Animals. Ames, Iowa State University Press, 2001, pp 105-114.
25. Fowler ME: Order Strigiformes (owls): Biology, medicine, and surgery. *In* Fowler ME, Cubas ZS (eds): Biology, Medicine, and Surgery of South American Wild Animals. Ames, Iowa State University Press, 2001, pp 125-132.
26. Fowler GS, Fowler ME: Order Sphenisciformes (penguins): Biology, captive management, and medicine. *In* Fowler ME, Cubas ZS (eds): Biology, Medicine, and Surgery of South American Wild Animals. Ames, Iowa State University Press, 2001, pp 53-64.
27. Fox N: Managing a breeding program. *In* Fox N: Understanding the Bird of Prey. Surrey, BC, Hancock House Publishers Ltd, 1995, pp 58-110.
28. Fox N: Equipment and facilities. *In* Fox N: Understanding the Bird of Prey. Surrey, BC, Hancock House Publishers Ltd, 1995, pp 112-174.
29. Frey H, Zinc R: Aspects of management within the European bearded vulture (*Gypaetus barbatus*) reintroduction. *In* Lumeij JT, Remple JD, Redig PT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network. 2000, pp 281-288.
30. Gerlach H: Galliformes. *In* Altman RB, Clubb SL, Dorrestein GM, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 944-959.
31. Gerlach H: Anseriformes. *In* Altman RB, Clubb SL, Dorrestein GM, Quesenberry K (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 960-972.
32. Greenwood AG: Veterinary support for in situ avian conservation programs. Proc Assoc Avian Vet, 1995, pp 163-169.
33. Harlin RW: Husbandry and diseases of domestic pigeons. *In* Rosskopf WJ, Jr, Woerpl RW (eds): Diseases of Cage and Aviary Birds. Baltimore, Williams & Wilkins, 1996, pp 944-950.
34. Harper JE, Skinner ND: Clinical nutrition of small psittacines and passerines. Sem Avian Exotic Pet Med 7(3):116-127, 1998.
35. Harris DJ: Care of the critically ill patient. Sem Avian Exotic Pet Med 3(4):175-179, 1994.
36. Hatt JM: Nutrition research in zoo animals. In Nijboer J, Hatt JM, Kaumanns W, et al (eds): Zoo Animal Nutrition. Fürth, Germany, Filander Verlag, 2000, pp 11-19.
37. Heatley JJ, Mikota SK, Olsen G, et al: Antipredator conditioning of Mississippi sandhill cranes (Grus canadensis pulla). Proc Assoc Avian Vet, 2000, pp 143-144.
38. Heidenreich M: Management of raptors in captivity. *In* Heidenreich M: Birds of Prey, Medicine and Management. Oxford, UK, Blackwell Science Ltd, 1997, pp 5-23.
39. Heitzmann-Fontenelle J: Order Ciconiiformes (herons, storks, ibises): Ciconiiformes medicine. In Fowler ME, Cubas ZS (eds): Biology, Medicine, and Surgery of South American Wild Animals. Ames, Iowa State University Press, 2001, pp 84-87.
40. Hernandez M, Aguilar RF: Steroid and fluid therapy for treatment of shock in the critical avian patient. Sem Avian Exotic Pet Med 3(4):190-199, 1994.
41. Holz P, Naisbitt R: Fitness level as a determining factor in the survival of rehabilitated raptors released back into the wild: Preliminary results. *In* Lumeij JT, Remple JD, Redig PT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network, 2000, pp 321-325.
42. Hooimeijer J, Dorrestein GM: Pigeons and doves. *In* Altman RB, Clubb SL, Dorrestein GM, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 886-909.
43. Houston DC, Fidgett A: What are the nutritional requirements for making a good bird egg? *In* Nijboer J, Hatt JM, Kaumanns W, et al (eds): Fürth, Germany, Zoo Animal Nutrition. Filander Verlag, 2000, pp 51-56.
44. Houston DC: Digestion strategies in meat and fish eating birds. *In* Nijboer J, Hatt JM, Kaumanns W, et al (eds): Zoo Animal Nutrition. Fürth, Germany, Filander Verlag, 2000, pp 57-62.
45. Jenkins J: Ratite medicine and surgery. *In* Rosskopf WJ, Jr, Woerpl RW (eds): Diseases of Cage and Aviary Birds. Baltimore, Williams & Wilkins, 1996, pp 1002-1006.
46. Jennings J: Order Piciformes (toucans, woodpeckers): Captive management family Ramphastidae (toucans). *In* Fowler ME, Cubas ZS (eds): Biology, Medicine, and Surgery of South American Wild Animals. Ames, Iowa State University Press, 2001, pp 186-188.
47. Jones MP: Developing a veterinary care program for captive birds of prey. Proc Assoc Avian Vet, 1998, pp 183-187.
48. Joseph V: Preventive health programs for falconry birds. Proc Assoc Avian Vet, 1995, pp 171-178.
49. Joseph V: Selected medical topics for birds of prey. Proc Assoc Avian Vet, 1996, pp 261-266.
50. Joseph V: Emergency care of raptors. Vet Clin No Am Exot Anim 1(1):77-98, 1998.
51. Kirkwood J, Bailey T, Samour J, et al: Management-related diseases. *In* Samour J (ed): Avian Medicine. London, UK, Mosby, 2000, pp 170-218.
52. LaBonde J: The medical and surgical management of domestic waterfowl collections. Proc Assoc Avian Vet, 1992, pp 223-233.
53. LaBonde J: Toxicity in pet avian patients. Sem Avian Exotic Pet Med 4(1):23-31, 1995.
54. LaBonde J: Private collections of waterfowls. Proc Assoc Avian Vet, 1996, pp 215-223.
55. LaBonde J: Medicine and surgery of mynahs. *In* Rosskopf WJ, Jr, Woerpl RW (eds): Diseases of Cage and Aviary Birds. Baltimore, Williams & Wilkins, 1996, pp 928-932.
56. LaBonde J: Medicine and surgery of gallinaceous birds. *In* Rosskopf WJ, Jr, Woerpl RW (eds): Diseases of Cage and Aviary Birds. Baltimore, Williams & Wilkins, 1996, pp 951-956.
57. LaBonde J: Medicine and surgery of Anseriformes. *In* Rosskopf WJ, Jr, Woerpl RW (eds): Diseases of Cage and Aviary Birds. Baltimore, Williams & Wilkins, 1996, pp 956-964.
58. Langenberg J: The role of a veterinarian in an avian conservation program. Proc Assoc Avian Vet, 1995, pp 157-161.
59. Loudis BG, Sutherland-Smith M: Methods used in the critical care of avian patients. Sem Avian Exotic Pet Med 3(4):180-189, 1994.
60. Luebbert JAR: Canada goose (Branta canadensis) rehabilitation: A natural history guide for veterinarians. Proc Assoc Avian Vet, 1996, pp 245-253.
61. Luebbert JR: Wild trumpeter swan (*Cygnus buccinator*) rehabilitation: Natural history for veterinarians. Proc Assoc Avian Vet, 2000, pp 149-156.
62. Morgan KN, Line SW, Markowitz H: Zoos, enrichment, and the skeptical observer: The practical value of assessment. *In* Shepherdson DJ, Mellen JD, Hutchins M (eds): Second Nature, Environmental Enrichment for

Captive Animals. Washington, Smithsonian Institution Press, 1998, pp 153-171.
63. Morishita TY: Clinical assessment of gallinaceous birds and waterfowls in backyard flocks. Vet Clin No Am Exot Anim 2(2):383-404, 1999.
64. Norton TM, et al: Bali mynah captive medical management and reintroduction program. Proc Assoc Avian Vet, 1995, pp 125-136.
65. Olsen GH: Common infectious and parasitic diseases of quail and pheasants. Proc Assoc Avian Vet, 1993, pp 146-150.
66. Olsen GH, Carpenter JW: Andean condor medicine, reproduction and husbandry. Proc Assoc Avian Vet, 1995, pp 147-152.
67. Olsen GH, Spalding M, Langenberg J: Veterinary aspects of releasing endangered whooping cranes (*Grus americana*) in Florida. Proc Assoc Avian Vet, 1996, pp 255-259.
68. Olsen GH, Langernberg JA, Carpenter JW: Medicine and surgery. *In* Ellis DH, Gee GF, Mirande CM (eds): Cranes: Their Biology, Husbandry, and Conservation. Blaine, WA, Hancock House Publishers, 1996, pp 137-174.
69. Olsen GH, Carpenter JW: Cranes. *In* Altman RB, Clubb SL, Dorrestein GM, et a; (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 973-992.
70. Olsen GH: Cranes. *In* Tully TN, Lawton MPC, Dorrestein GM (eds): Avian Medicine. Oxford, UK, Butterworth-Heinemann, 2000, pp 215-227.
71. Pokras MA: Clinical management and biomedicine of sea birds. *In* Rosskopf WJ, Jr, Woerpl RW (eds): Diseases of Cage and Aviary Birds. Baltimore, Williams & Wilkins, 1996, pp 981-1001.
72. Pollock C: Special avian species. Proc Assoc Avian Vet, 2001, pp. 411-425.
73. Prudente do Amaral P, Sanfilippo LF: Order Ciconiiformes (herons, storks, ibises): Management in captivity. *In* Fowler ME, Cubas ZS (eds): Biology, Medicine, and Surgery of South American Wild Animals. Ames, Iowa State University Press, 2001, pp 83-84.
74. Redig PT: Health management of raptors trained for falconry. Proc Assoc Avian Vet, 1992, pp 258-264.
75. Redig PT: Raptors. *In* Altman RB, Clubb SL, Dorrestein GM, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 918-928.
76. Reding PT, Ackermann J: Raptors. *In* Tully TN, Lawton MPC, Dorrestein GM (eds): Avian Medicine. Oxford, UK, Butterworth-Heinemann, 2000, pp 180-214.
77. Remple JD: Considerations on the production of a safe and efficacious falcon herpesvirus vaccine. *In* Lumeij JT, Remple JD, Redig PT, et al (eds): Raptor Biomedicine III. Lake Worth, FL, Zoological Education Network. 2000, pp 13-23.
78. Robinson I: Seabirds. *In* Tully TN, Lawton MPC, Dorrestein GM (eds): Avian Medicine. Oxford, UK, Butterworth-Heinemann, 2000, pp 339-363.
79. Roudybush TE: Psittacine nutrition. Vet Clin No Am Exot Anim 2(1):111-125, 1999.
80. Routh A, Snaderson S: Waterfowl. *In* Tully TN, Lawton MPC, Dorrestein GM (eds): Avian Medicine. Oxford, UK, Butterworth-Heinemann, 2000, pp 234-265.
81. Samour J: Veterinary medicine, falcons, and falconry in the Middle East. Proc Assoc Avian Vet, 1996, pp 233-239.
82. Seidensticker J, Forthman DL: Evolution, and enrichment: Basic considerations for wild animals in zoos. *In* Shepherdson DJ, Mellen JD, Hutchins M (eds): Second Nature, Environmental Enrichment for Captive Animals. Washington, Smithsonian Institution Press, 1998, pp 15-29.
83. Speer BL: Taxonomy for the avian veterinarian. Proc Assoc Avian Vet, 1999, pp 305-322.
84. Stahl S, Kronfeld D: Veterinary nutrition of large psittacines. Sem Avian Exot Pet Med 7(3):128-134, 1998.
85. Staples G: Appendix 7. *In* Harrison GJ, Harrison LR (eds): Clinical Avian Medicine and Surgery. Philadelphia, WB Saunders Co, 1986, pp 667-668.
86. Stonebreaker R: Ratites. *In* Altman RB, Clubb SL, Dorrestein GM, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 929-943.
87. Stringfield CE: Medical management of the California condor (*Gymnogyps californianus*). Proc Assoc Avian Vet, 1998, pp 173-174.
88. Suedmeyer WK: An introduction to falconry. Proc Assoc Avian Vet, 1993, pp 164-172.
89. Swengel SR, Carpenter JW: General husbandry. *In* Ellis DH, Gee GF, Mirande CM (eds): Cranes: Their Biology, Husbandry, and Conservation. Blaine, WA, Hancock House Publishers, 1996, pp 31-43.
90. Timossi L, Crosta L, Bürkle M: Management and general medical issues of a mixed species penguin collection in closed environment. Proc European Assoc Zoo Wildlife Vet and European Wildlife Disease Assoc, 2002, pp 283-285.
91. Tully TN: Ratites. *In* Tully TN, Lawton MPC, Dorrestein GM (eds): Avian Medicine. Oxford, UK, Butterworth-Heinemann, 2000, pp 228-233.
92. Van De Water D: Raptor rehabilitation. *In* Rosskopf WJ, Jr, Woerpl RW (eds): Diseases of Cage and Aviary Birds. Baltimore, Williams & Wilkins, 1996, pp 1007-1028.
93. Worrell AB: Medicine and surgery of toucans. *In* Rosskopf WJ, Jr, Woerpl RW (eds): Diseases of Cage and Aviary Birds. Baltimore, Williams & Wilkins, 1996, pp 933-943.
94. Worrell AB: Toucans and mynahs. *In* Altman RB, Clubb SL, Dorrestein GM, et al (eds): Avian Medicine and Surgery. Philadelphia, WB Saunders, 1997, pp 910-917.
95. Worrell AB: Ramphastids. *In* Tully TN, Lawton MPC, Dorrestein GM (eds): Avian Medicine. Oxford, UK, Butterworth-Heinemann, 2000, pp 296-311.
96. Zdziarski JM, Abou-Madi N, Zuba JR: Veterinary consideration in the shipping and receiving of avian species. Proc Am Assoc Zoo Vet, 1993, pp 358-371.
97. Bürkle M, Gerlach H: Medical monitoring of a parrot collection. Proc Intl Parrot Conv, 2002.

Appendices

Appendix I. Avian Hematologic Reference Ranges*

Determination	African grey	Amazon	Budgerigar	Canary	Cockatiel	Cockatoo	Conure	Eclectus
WBC x $10^3/\mu l$	5-11	6-11	3-8.5	4-9	5-10	5-11	4-11	4-10
RBC x $10^6/\mu l$	2.4-3.9	2.4-4	2.4-4	2.5-3.8	2.2-3.9	2.4-4.2	2.5-4.1	2.4-3.9
HCT (%)	40-48	40-50	40-50	40-49	40-49	40-48	40-49	40-47
Hets (%)	55-75	55-80	50-75	50-80	55-80	55-80	55-75	55-70
Eos (%)	0-2	0-1	0-2	0-2	0-2	0-2	0-2	0-1
Baso (%)	0-1	0-1	0-1	0-1	0-2	0-1	0-1	0-2
Monos (%)	0-3	0-3	0-2	0-1	0-2	0-1	0-2	0-2
Lymphs (%)	25-45	20-45	25-45	20-45	20-45	20-45	25-45	30-45

Determination	Jardine's	Lory	Lovebird	Macaw	Parakeet	Pionus	Quaker	Senegal
WBC x $10^3/\mu l$	4-10	4.5-8.5	3-8.5	6-12	4.5-9.5	4-11.5	4-10	4-11
RBC x $10^6/\mu l$	2.4-4.2	2.5-4.1	2.3-3.9	2.4-4.2	2.2-3.9	2.4-4	2.3-4.1	2.4-4.1
HCT (%)	38-48	39-50	38-50	39-48	39-48	40-47	40-49	39-48
Hets (%)	55-75	50-70	55-80	58-78	50-75	50-75	55-80	55-75
Eos (%)	0-1	0-2	0-1	0-1	0-2	0-2	0-1	0-1
Baso (%)	0-1	0-1	0-1	0-1	0-1	0-1	0-2	0-1
Monos (%)	0-2	0-2	0-3	0-3	0-2	0-2	0-3	0-2
Lymphs (%)	25-45	23-45	20-45	20-45	25-45	25-45	20-45	25-45

*Provided by University of Miami Avian and Wildlife Laboratory: RBC and WBC count by Unopette method from blood collected in EDTA; Spun HCT; Diff based on 100-cell count using smear made at the time of sample acquisition.

Appendix II. Avian Hematologic Reference Ranges for Clinically Normal Psittacines**

In an attempt to establish ranges for clinically normal birds of various psittacine species, the following criteria were used to determine the acceptability of individual values for incorporation into the following statistics:

1) The bird was presented for a wellness examination, at which time the sample was obtained.

2) At least one previous CBC was performed and documented at the same hospital, utilizing the same techniques, and was also performed during a wellness examination.

3) No other concurrent disease was detected or treated, with the exception of behavioral concerns.

4) The total WBC did not vary more than 25% between the two values.

Determination	African greys	Amazon sp.	Budgerigars	Cockatiels	Umbrella cockatoos	Aratinga conures	Eclectus parrots
	(n = 150)	(n = 150)	(n = 50)	(n = 200)	(n = 100)	(n = 50)	(n = 25)
WBC x $10^3/\mu l$	8-11	7.5-12.5	2.5-6.5	4.0-8.8	7-11.5	7.0-11.7	7.0-11.8
PCV (%)	42-50	44-49	42-53	45-57	38-48	42-49	41-49

Determination	Lovebirds	Macaws	Pionus sp.	Quaker (monk) parakeets	Senegal parrots
	(n = 25)	(n = 100)	(n = 25)	(n = 50)	(n = 25)
WBC x $10^3/\mu l$	4.5-9.0	8.5-15.5	7.0-11.5	5.5-12.5	6.5-12
PCV (%)	39-51	39-52	44-51	38-48	37-48

** Compiled 1989-2001 by Teresa L. Lightfoot, DVM, Dipl ABVP-Avian: All calculations were done using the Eosinophil Unopette method; an average of the total WBC counts for the two or more CBCs that were performed was used to develop the above ranges.

Appendix III. Avian Biochemistry Reference Ranges*

Determination	African grey	Amazon	Budgerigar	Canary	Cockatiel	Cockatoo	Conure	Eclectus
Alk phos (U/L)	20-160	15-150	10-80	20-135	20-250	15-255	80-250	150-350
ALT (U/L)	5-12	5-11	5-10	5-11	5-11	6-12	5-13	5-11
AST (U/L)	100-365	130-350	145-350	145-345	95-345	145-355	125-345	120-370
Amylase (U/L)	210-530	205-510	200-500	190-485	205-490	200-510	100-450	200-645
BUN (mg/dL)	3-5.4	3.1-5.3	3-5.2	3-5	2.9-5	3-5.1	2.8-5.4	3-5.5
Ca (mg/dL)	8.5-13	8.5-14	6.5-11	5.5-13.5	8-13	8-13	7-15	7-13
Chol (mg/dL)	160-425	180-305	145-275	150-400	140-360	145-355	120-400	130-350
Creat (mg/dL)	0.1-0.4	0.1-0.4	0.1-0.4	0.1-0.4	0.1-0.4	0.1-0.4	0.1-0.4	0.1-0.4
CO_2 (mmol/L)	13-25	13-26	14-25	14-26	13-25	14-25	14-25	14-24
CPK (U/L)	165-412	55-345	90-300	55-350	30-245	95-305	35-355	220-345
GGT (U/L)	1-10	1-12	1-10	1-14	1-30	1-45	1-15	1-20
Glu (mg/dL)	190-350	190-345	190-390	205-435	200-445	185-355	200-345	145-245
LDH (U/L)	145-465	155-425	145-435	120-450	120-455	220-550	120-390	200-425
Lipase (U/L)	35-350	35-225	30-300	29-255	30-280	25-275	30-290	35-275
Phos (mg/dL)	3.2-5.4	3.1-5.5	3.0-5.2	2.9-4.9	3.2-4.8	2.5-5.5	2-10	2.9-6.5
Potassium (mmol/L)	2.9-4.6	3-4.5	2.2-3.9	2.2-4.5	2.4-4.6	2.5-4.5	3-4.5	3.5-4.3
Sodium (mmol/L)	157-165	125-155	139-165	135-165	130-153	130-155	135-149	130-145
Total bili (mg/dL)	0-0.1	0-0.1	0-0.1	0-0.1	0-0.1	0-0.1	0-0.1	0-0.1
Total protein (g/dL)	3-4.6	3-5	2.5-4.5	2.8-4.5	2.4-4.1	3-5	3-4.2	2.8-3.8
Trig (mg/dL)	45-145	49-190	105-265	60-265	45-200	45-205	50-300	70-410
Uric acid (mg/dL)	4.5-9.5	2.3-10	4.5-14	4-12	3.5-10.5	3.5-10.5	2.5-11	2.5-11

Determination	African grey	Amazon	Budgerigar	Canary	Cockatiel	Cockatoo	Conure	Eclectus
Bile acids (μmol/L)	13-90	18-60	15-70	23-90	20-85	25-87	15-55	10-61
T_4 μg/dL)	0.2-1.5	0.1-1.1	0.3-1.5	0.3-1.8	0.4-1.9	0.3-1.8	0.3-1.9	0.3-2.0
Pre-albumin (g/dL)	0.03-1.35	0.35-1.05	0.48-1.21	0.35-0.98	0.8-1.6	0.24-1.18	0.18-0.98	0.05-0.74
Albumin (g/dL)	1.57-3.23	1.9-3.52	0.79-1.35	0.81-1.23	0.7-1.8	1.8-3.1	1.9-2.6	2.3-2.6
Alpha-1 (g/dL)	0.02-0.27	0.05-0.32	0.08-0.21	0.08-0.16	0.05-0.30	0.05-0.18	0.04-0.23	0.09-0.19
Alpha-2 (g/dL)	0.05-0.25	0.07-0.32	0.05-0.16	0.05-0.22	0.05-0.30	0.04-0.36	0.08-0.26	0.11-0.21
Beta (g/dL)	0.35-0.66	0.38-0.76	0.35-0.75	0.3-0.71	0.3-0.78	0.35-0.82	0.38-0.77	0.35-0.62
Gamma (g/dL)	0.11-0.71	0.17-0.76	0.15-0.55	0.16-0.63	0.11-0.53	0.21-0.65	0.32-0.61	0.22-0.51
A/G ratio	1.6-4.3	1.9-5.0	1.5-4.1	1.3-4.5	1.5-4.3	2.0-4.5	2.2-4.3	2.6-4.1

* University of Miami Avian and Wildlife Laboratory: All reference ranges obtained from heparinized plasma samples; regular chemistry performed on Ortho (Kodak Ektahem) 700XR; TP by non-temperature compensated refractometer; bile acids and T_4 by RIA; EPH by Beckman Paragon SPEP II gels; represent adult ranges.

Determination	Jardine's	Lory	Lovebird	Macaw	Parakeet	Pionus	Quaker	Senegal
Alk phos (U/L)	80-156	75-155	10-90	20-230	20-120	80-290	70-300	70-300
ALT (U/L)	5-12	5-13	5-13	5-12	5-12	5-12	5-11	5-11
AST (U/L)	150-278	150-350	110-345	100-300	145-395	150-365	150-285	100-350
Amylase (U/L)	100-425	90-422	90-400	150-550	150-525	200-500	100-400	190-550
BUN (mg/dL)	2.8-5.6	2.7-5.7	2.8-5.5	3-5.6	3.1-5.3	3-5.4	2.9-5.4	2.9-5.4
Ca (mg/dL)	7-13	6.5-13	8-14	8.5-13	5.5-13.5	7-13.5	7-12	6.5-13
Chol (mg/dL)	100-300	95-295	95-335	100-390	150-400	130-295	100-295	130-340
Creat (mg/dL)	0.1-0.4	0.1-0.4	0.1-0.4	0.1-0.5	0.1-0.4	0.1-0.4	0.1-0.4	0.1-0.4
CO_2 (mmol/L)	14-25	14-26	14-25	14-25	14-25	14-24	14-26	14-25
CPK (U/L)	110-310	110-330	52-245	100-300	50-400	100-300	110-320	100-330
GGT (U/L)	1-15	1-16	2.5-18	1-30	1-12	1-18	1-15	1-15
Glu (mg/dL)	199-348	200-300	195-405	145-345	205-345	125-300	200-350	140-250
LDH (U/L)	119-335	115-330	105-355	70-350	145-445	125-380	120-300	150-350
Lipase (U/L)	30-255	25-250	30-320	30-250	30-220	30-250	25-225	35-250
Phos (mg/dL)	2-6.8	2-6.5	2.8-4.9	2-12	2.9-4.9	2.9-6.6	2.9-6.5	2.5-9.5
Potassium (mmol/L)	3-4.5	3-4.4	2.1-4.8	2-5	2.3-4.2	3.5-4.6	2.8-4.6	3-5
Sodium (mmol/L)	133-153	130-155	125-155	140-165	138-166	145-155	140-155	130-155
Total bili (mg/dL)	0-0.1	0-0.1	0-0.1	0-0.1	0-0.1	0-0.1	0-0.1	0-0.1
Total protein (g/dL)	2.8-4	2-3.5	2.8-4.4	2.1-4.5	3-4.4	2.2-4	2.8-3.6	3.5-4.4
Trig (mg/dL)	60-130	65-140	45-200	60-135	55-250	60-225	50-200	45-145
Uric acid (mg/dL)	2.5-12	2.8-11.5	3.5-11	2.5-11	4.5-12	3.5-10	3.5-11.5	2.3-10

Determination	Jardine's	Lory	Lovebird	Macaw	Parakeet	Pionus	Quaker	Senegal
Bile acids (μmol/L)	25-65	20-65	13-65	6-35	18-79	14-60	25-65	20-85
T_4 (μg/dL)	0.2-1.5	0.3-1.2	0.2-2.3	0.2-1.9	0.4-1.8	0.5-1.9	0.4-2.1	0.5-2.3
Pre-albumin (g/dL)	0.18-0.32	0.48-0.76	0.6-1.2	0.05-0.7	0.6-1	0.19-0.93	0.48-1.13	0.19-0.64
Albumin (g/dL)	1.85-2.23	1.26-1.96	2-2.8	1.24-3.11	1.9-3	2.19-3.29	1.26-2.52	1.45-2.28
Alpha-1 (g/dL)	0.07-0.15	0.04-0.14	0.08-0.21	0.04-0.25	0.06-0.16	0.1-0.19	0.04-0.25	0.02-0.20
Alpha-2 (g/dL)	0.08-0.15	0.04-0.23	0.08-0.25	0.04-0.31	0.07-0.20	0.08-0.15	0.05-0.28	0.08-0.16
Beta (g/dL)	0.38-0.66	0.35-0.58	0.34-0.68	0.48-0.68	0.22-0.45	0.30-0.60	0.41-0.63	0.36-0.58
Gamma (g/dL)	0.26-0.51	0.13-0.29	0.2-0.48	0.2-0.5	0.19-0.30	0.18-0.42	0.13-0.48	0.14-0.23
A/G ratio	2.9-3.5	2.3-4.0	2.5-4.6	1.6-4.3	4-5.3	3.4-5.0	2.2-3.2	2.2-3.9

* University of Miami Avian and Wildlife Laboratory: All reference ranges obtained from heparinized plasma samples; regular chemistry performed on Ortho (Kodak Ektachem) 700XR; TP by non-temperature compensated refractometer; bile acids and T_4 by RIA; EPH by Beckman Paragon SPEP II gels; represent adult ranges.

For assistance on biochemistry conversions: www.vin.com/scripts/labquest/converthtml.pl

Index

A

B

C

D

E

F

G

H

I

J

K

L

M

N

O

P

Q

R

S

T

U

V

W

X

Y

Z