AVIAN MEDICINE

THIRD EDITION

EDITED BY

Jaime Samour, MVZ (Hons), PhD, Dipl ECZM (Avian)
Director, Wildlife Division, Wrsan,
Abu Dhabi, United Arab Emirates

ELSEVIER

ELSEVIER

3251 Riverport Lane
St. Louis, Missouri 63043

AVIAN MEDICINE, THIRD EDITION ISBN: 978-0-7234-3832-8

Library of Congress Cataloging-in-Publication Data

Avian medicine (Samour)
Avian medicine / edited by Jaime Samour.—Third edition.
p. ; cm.
Includes bibliographical references and index.
ISBN 978-0-7234-3832-8 (hardback : alk. paper)
I. Samour, Jaime, editor. II. Title.
[DNLM: 1. Bird Diseases. 2. Birds. SF 994]
SF994
636.5′0896–dc23

2015032018

Content Strategy Director: Penny Rudolph
Content Strategist: Brandi Graham
Associate Content Development Specialist: Laura Klein
Publishing Services Manager: Hemamalini Rajendrababu
Project Manager: Manchu Mohan
Senior Book Designer: Margaret Reid

Printed in China

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

I would like to dedicate the 3rd edition of Avian Medicine to my wife Merle with my most heartfelt gratitude for her support and understanding to the professional and man in me; your unreserved and unconditional love over the past 20 years is a true reflection of who you are. To my sons Omar and Adam and my daughters Miriam and Yasmeen with my deepest love and expecting that one day they will also follow the teachings of my boyhood hero, Jim Corbett, and in his own words, "try and make this world a better place for others to live in." (Jim Corbett, 1875-1955, Naini Tal, India).

Jaime Samour
Wildlife Division, Wrsan
Abu Dhabi
United Arab Emirates, 2015

CONTRIBUTORS

M.M. Apo, BCs, ACS
Administrative Assistant, Wildlife Division, Wrsan,
Abu Dhabi, United Arab Emirates

T.A. Bailey, BSc, BVSc, MRCVS, Cert Zoo Med, Dipl ECZM (Avian), MSc (Wild Animal Health), PhD
Wildlife Consultant, Goetre Farm,
Pembrokeshire, UK

B. Barca Ruibal, LicVet, CertZooMed, MSc (Wild Animal Health), MPhil, MRCVS
Al Aseefa Falcon Hospital,
Dubai, United Arab Emirates

J.V. Baskar, BVSc
Senior Veterinarian,
Central Veterinary Research Laboratory,
Dubai, United Arab Emirates

H. Beaufrère, Dr med vet, PhD, Diplomate ACZM, ABVP (Avian), ECZM (Avian)
Service Chief, Avian and Exotic Service,
Health Sciences Centre, Ontario Veterinary College,
University of Guelph, Ontario, Canada

Professor J.E. Cooper, DTVM, FRCPath, FSB, CBiol, FRCVS
RCVS Specialist in Veterinary Pathology, Diplomate European College of Veterinary Pathologists, European Veterinary Specialist Zoological Medicine; Visiting Professor, Faculty of Veterinary Medicine, University of Nairobi, Kenya,
Department of Veterinary Medicine, University of Cambridge, Cambridge, UK

M.E. Cooper, LLB, FLS
Solicitor (not in private practice); Visiting Lecturer, Faculty of Veterinary Medicine, University of Nairobi, Kenya; Honorary Research Fellow, Durrell Institute of Conservation and Ecology (DICE), The University of Kent, Canterbury, Kent, UK

P. Coutteel, DVM
Clinic TRIGENIO,
Nijlen, Belgium

M. Cowan, BVSc (Hons I), MANZCVS (Avian Health)
Brisbane Bird and Exotics Veterinary Service,
Brisbane, Queensland, Australia

L. Crosta, DVM, PhD
Veterinari Montevecchia,
Montevecchia (LC), Italy

F.J. Dein, VMD, MS
School of Veterinary Medicine,
University of Wisconsin, Madison, WI,
United States of America

A.B. del Rio, DVM, PhD, Dipl CLOVE
Department of Medicine and Animal Surgery,
Ophthalmology Services Chief,
Veterinary Teaching Hospital, Murcia University,
Murcia, Spain

R. Doneley, BVSc, FANZCVS (Avian Health) CMAVA
Head Avian and Exotic Pet Services,
University of Queensland Veterinary Medical Centre,
The University of Queensland,
Gatton, Queensland, Australia

N.A. Forbes, BVetMed, CBiol, MIBiol, Dipl ECZM (Avian)
Vets Now Referrals,
Swindon, UK

B. Gartrell, BVSc, PhD, MANZCVSc (Avian Health)
Institute of Veterinary, Animal & Biomedical Sciences,
Massey University, New Zealand

S. Hammer, Dr med vet
Certified Specialist in Zoo and Wild Animals,
Director, Zoo Goerlitz,
Goerlitz, Germany

Professor J. M. Hatt, Dr med vet, MSc, Dipl ACZM, Dipl ECZM (Avian)
Clinic Director and Senior Veterinarian of Zurich Zoo, Clinic for Zoo Animals,
Exotic Pets and Wildlife, University of Zürich,
Zürich, Switzerland

M.G. Hawkins, VMD Dip ABVP (Avian)
Department of Medicine and Epidemiology,
School of Veterinary Medicine,
University of California,
Davis, California, USA

C. Hebel, Dr med vet
Garmisch-Partenkirchen, Bavaria, Germany

J.C. Howlett, RVN, BSc (Hons), Dip Nat Sci
Al Ain Zoo,
Al Ain, United Arab Emirates

J. Kinne, Dr med vet, Cert Vet Path, Cert Trop Vet
Head Department of Pathology,
Central Veterinary Research Laboratory,
Dubai, United Arab Emirates

Professor M.E. Krautwald Junghanns, Dr med vet, Dr med vet habil
Dipl ECZM (Avian), National Approved Specialist for Birds and Reptiles,
Selected Member of the National Academy of Sciences, Leopoldina,
Director of Clinic for Birds and Reptiles, University of Leipzig,
Leipzig, Germany

O. Krone, Dr med vet
Leibniz Institute for Zoo and Wildlife Research, Department of Wildlife Diseases,
Berlin, Germany

M.P.C. Lawton, BVetMed; CertVOphthal; CertLAS; CBiol; MSB; DZooMed; FRCVS
Lawton & Stoakes Veterinary Surgeons, Harold Wood,
Essex, UK

C. Liu, PhD
Cell Biologist, Central Veterinary Research Laboratory,
Dubai, United Arab Emirates

M.N.F. Magno, DVM
Senior Veterinary Officer,
Wildlife Division, Wrsan,
Abu Dhabi, United Arab Emirates

A. Melillo, Dr med vet
Omniavet Veterinary Clinic,
Rome, Italy

D. Monks, BVSc (Hons), CertZooMed, FANZCVSc (Avian Health), Dip ECZM (Avian)
Brisbane Bird and Exotics Veterinary Service, Brisbane,
Queensland, Australia

A. Montesinos, LV, MSc
Centro Veterinario Los Sauces,
Madrid, Spain

K. Morgan, BVSc, MANZCVS (Avian Health), PGDipVCS, PhD
Institute of Veterinary, Animal & Biomedical Sciences,
Massey University, New Zealand

J.L. Naldo, DVM
Nad Al Shiba Veterinary Hospital,
Dubai, United Arab Emirates

J. Paul-Murphy, DVM Dip ACZM, Dip ACAW
Department of Medicine and Epidemiology,
School of Veterinary Medicine,
University of California,
Davis, California, USA

M. Pees, Dr med vet, Dipl ECZM (Avian, Herpetology)
Clinic for Birds and Reptiles, University of Leipzig,
Leipzig, Germany

M.A. Peirce, PhD, CBiol, FSB, FZS
MP International Consultancy,
Bexhill-on-Sea,
East Sussex, UK

H. Pendl, Dr med vet
PendlLab, Diagnostic Microscopy, Hematology, Cytology, Histopathology in Birds and Reptiles,
Steinhausen, Switzerland

J. Perlman, PhD
Wildlife Nutrition Consultants,
Alexander, Arkansas, USA

D. Phalen, DVM, PhD, Dipl ABVP (Avian)
Wildlife Health and Conservation Centre,
Faculty of Veterinary Science, University of Sydney,
Sydney, Australia

J. Ponder, DVM
The Raptor Center, College of Veterinary Medicine,
University of Minnesota,
St. Paul, Minnesota, USA

Professor P.T. Redig, DVM, PhD
Director Emeritus, The Raptor Center,
College of Veterinary Medicine,
University of Minnesota,
St. Paul, Minnesota, USA

M.D. Saggese, DVM, MS, PhD
College of Veterinary Medicine,
Western University of Health Sciences,
Pomona, California, USA

J. Samour, MVZ (Hons), PhD, Dipl ECZM (Avian)
Director, Wildlife Division, Wrsan,
Abu Dhabi, United Arab Emirate

D. Sanchez-Migallon Guzman, LV, MS, Dipl ECZM (Avian, Small Mammal), Dipl ACZM
The Companion Exotic Animal Medicine Service,
University of California Davis School of Veterinary Medicine,
Davis, California, USA

P. Sandmeier, Dr med vet, Dipl ECZM (Avian)
Small Animal and Avian Practice,
Baden-Daettwil, Switzerland

P. Schnitzer, Dr med vet
Veterinari Montevecchia,
Montevecchia (LC), Italy

N.J. Schoemaker, DVM, PhD, Dipl ECZM (Small Mammal, Avian), Dipl ABVP (Avian)
European Veterinary Specialist in Zoological Medicine (Small Mammal),
Division of Zoological Medicine, Department of Clinical Sciences of Companion Animals,
Faculty of Veterinary Medicine, Utrecht University,
Utrecht, The Netherlands

Professor Dr. R.K. Schuster, Dip EVPC, FTA Parasitology, FTA Tropical Veterinary Medicine
Head of Parasitology Department, Central Veterinary Research Laboratory,
Dubai, United Arab Emirates

C. Silvanose, BSc, MLT, DPath, PhD
Laboratory Manager, Dubai Falcon Hospital,
Dubai, United Arab Emirates

L. Timossi, Dr med vet
Veterinari Montevecchia,
Montevecchia (LC), Italy

Y.R.A. van Zeeland, DVM, MVR, PhD, Dip. ECZM (Avian, Small Mammal), European Specialist in Zoological Medicine (Avian)
Division of Zoological Medicine,
Department of Clinical Sciences of Companion Animals,
Faculty of Veterinary Medicine, Utrecht University,
Utrecht, The Netherlands

P.M. Wencel, DVM
Avi Expert Veterinary Clinic,
Lublin, Poland

Professor U. Wernery, Priv Doz Dr Dr habil
Scientific Director, Central Veterinary Research Laboratory,
Dubai, United Arab Emirates

M.B. Wernick, Dr med vet, Dipl ECZM (Avian)
ExoticVet GmbH,
Jona, Switzerland

M. Ziccardi, DVM, MPVM, PhD
Oiled Wildlife Care Network, Karen C. Drayer Wildlife Health Center,
One Health Institute, School of Veterinary Medicine, University of California,
Davis, California, USA

P. Zsivanovits, Dr med vet, Dipl ECZM (Avian)
Tierärztliche Praxis für Vogelmedizin,
Wahlstedt, Germany

P. Zucca, DVM, PhD, BSc Psych
Zooanthropology Unit, Healthcare Services Agency,
Trieste, Italy

Professor P. Zwart, DVM, PhD, Dipl ECZM (Herpetology)
Diplomate European College of Veterinary Pathologists (ECVP), Diplomate European College of Zoological Medicine (ECZM; Herpetology),
Professor Emeritus in Diseases of Exotic Animals,
Department of Veterinary Pathology, Utrecht University,
Utrecht, The Netherlands

FOREWORD

I am writing this as my wife, Margaret, and I sit at Ngutuni Lodge, in Kenya, overlooking the water hole. The country is in the middle of a prolonged drought. The air is alive with the sound of thirsty African elephants (*Loxodonta africana)*, zebra (*Equus quagga burchellii*), and buffalo (*Syncerus caffer*) jostling for access to a drink from this much depleted water resource. Also at the pool, at a respectful distance from the mammals, are European white storks (*Ciconia ciconia*), Egyptian geese (*Alopochen aegyptiacus*), and crowned plovers (*Vanellus coronatus*), Namaqua doves (*Oena capensis*) have flown past and a pallid harrier (*Circus macrourus*) is circling overhead. Red-billed oxpeckers (*Buphagus erythrorhynchus*) periodically fly from the backs of the buffalo, land in the mud, circle in the air, and return to their hosts. Occasional flocks of red-billed quelea (*Quelea quelea*) descend in a cloud; they touch the water and, in a moment, are away again.

The scene reminds me of the words and thinking of one of my greatest heroes. This was John Hunter (1728-1793), the anatomist and surgeon, who was instrumental in the establishment of Britain's first veterinary college in London in 1791. Hunter was a countryman, with a keen knowledge of animals, plants, and the environment. He famously stated in his teaching that "*Nothing in nature stands alone.*" He thereby recognized at an early stage of his life, before the concept was fashionable, that there is an interconnectedness between individual organisms, other species, and the habitats that they share.

So what is the significance of all this to the third edition of Dr Jaime Samour's internationally acclaimed *Avian Medicine*? It is relevant because the book is not only about diseases of captive birds, how to diagnose ailments, and how to carry out treatment. This is also a scholarly treatise concerned with the health and welfare of birds. Its contributors, who hail from diverse countries, include veterinarians and biologists. Many of these are active in conservation. As such, although the primary aim of the book *is* to assist those working with birds in captivity, the information that it contains can readily be translated into practical assistance to the Class Aves on a much wider scale.

Wild (free-living) birds are threatened by habitat destruction, malicious persecution, poisoning, and infectious diseases. Attempts to halt the decline or disappearance of any one taxon require the input of skilled personnel from many disciplines, both professional and "amateur," including ecologists and field naturalists. To these, however, need to be added others, especially avian biologists and aviculturists. As populations of wild birds decline, the need to manage them becomes crucial. Such management may be carried out *in situ*, by (for instance) the provision of nest boxes or supplementary feeding; or *ex situ*, implying captive breeding. These and other techniques necessitate both the practical skills and "green finger" touch of those who keep birds and the increasingly sophisticated specialist abilities and techniques of veterinarians who seek to keep them healthy.

It will come as no surprise that I welcome this latest edition of Jaime Samour's book. My reasons for this are twofold. For a start, it will do much for birds in captivity. Aviculture has been practised for thousands of years and has been a feature of every civilization for which a recorded history exists. Nearly 40 years ago, in a now often forgotten manuscript, "The earliest records of aviculture" (*Avicultural Magazine* [1978] 84[4]), the Canadian R. M. Allison argued that the keeping of birds is deeply rooted human behavior—and went on to lament that this is too often ignored by "wildlife officialdom," to the detriment of many otherwise well-intentioned conservation policies.

The practice of Avian Medicine, in contrast—even allowing for early seminal studies on diseases of poultry, pigeons, and falcons in Mesopotamia (now Iraq) and a few other locations in the Middle East—has really only come of age as a specialist, evidence-based discipline during the past three decades. The many contributors to the different editions of Jaime Samour's *Avian Medicine* are amongst those who can take pride in this achievement.

The second reason I applaud the publication of the third edition of this book is because, as intimated above, its beneficial influence will not be restricted to birds in captivity. The advances in veterinary and biological knowledge crystallised in its text and often depicted graphically in its fine illustrations will, without doubt, contribute to the protection and conservation of the Class Aves on a global scale. The importance of understanding and practicing "ecosystem health" has never been more relevant. We need to look in a holistic way at the many current threats to biodiversity, to species survival, and to human well-being. The answer to diagnosing and treating most of these afflictions lies in identifying and correcting the multifarious insults that we are wreaking on our planet. This will not be an easy task, but the sooner the complex mosaic of manifestations ("clinical signs") that constitute global decline can be fully understood, the sooner the healing process can start. Scholarly texts on individual taxa have an important part to play, especially where they address questions of health and disease. Jaime Samour's contribution, through skillfully editing and executing this book and focusing attention on the needs of birds, is an indispensable component.

John E. Cooper, DTVM, FRCPath, FSB, CBiol, FRCVS,
Diplomate of the European College of Veterinary Pathologists,
Diplomate of the European College of Zoological Medicine
Wildlife Health Services (UK),
The Durrell Institute of Conservation and Ecology (DICE),
The University of Kent, Canterbury, England

PREFACE

My interest in the wild and its inhabitants began when I was a young child growing up in my native country, El Salvador. There was a cinema across the street from my home where I used to spend many Saturday afternoons watching movies of African jungle adventures. I remember vividly every detail of each movie, the sights of which still flash across my eyes and the sounds of which still resound in my ears after all this time. Later, my interest in wildlife medicine was inspired from watching a television series of a wildlife veterinarian running a wildlife rescue and rehabilitation center in Africa. He always wore safari-style khaki-colored clothing and a matching hat. I was always amazed to see him using his remote darting rifle to capture a full-grown African leopard (*Panthera pardus*) and send it into a deep sleep in a blink of an eye.

Little did I know then that the eerie sound in the background of those African jungle adventure movies came from a kookaburra (*Dacelo* sp.), a kingfisher native to Australia and New Guinea, or that the snake sliding across a branch was a boa constrictor (*Boa constrictor*) originally from Central and South America, or the elephants used in the movies were Indian elephants (*Elephas maximus indicus*) with large plastic ears. Yet again, little did I know that there has never been an anesthetic agent, as used by the wildlife veterinarian in the television series that could send a full-grown leopard into a deep sleep in a blink of an eye. Over time I realized that this was all to do with Hollywood and the strategies and paraphernalia used for movie making. Then the time came to move on.

I was still in high school when I started looking for opportunities to go abroad to study veterinary medicine in wildlife. It was shortly after qualifying from veterinary college that the long awaited opportunity came with an acceptance letter to undertake a residency for six months at the world famous London Zoo at Regent's Park. I arrived in London one day in late October 1981 with a copy of the Oxford English dictionary in one hand and a suitcase full of thermal clothing in the other, intending to begin a new phase in my career and to fulfill my long awaited dream. At London Zoo every day was different and full of exciting opportunities to learn. For instance, in the morning I could be assisting with the examination of a large silverback gorilla (*Gorilla gorilla*), at noontime helping with the collection of blood samples from a giant panda (*Ailuropoda melanoleuca*), and in the early afternoon attending to a skin condition in a white rhino (*Ceratotherium simum*). I had never been so fascinated in my entire life.

I am not sure when it came to me, but gradually I became aware that somewhere in the darker corners of all the excitement and the attention were the birds, the reptiles, the amphibians, the fishes, and the invertebrates. Soon I began to understand that there was much more to zoological collections than mammalian species. I was privileged to visit a great number of zoological collections across Great Britain and continental Europe, and I encountered the same situation in almost every collection. Therefore while mammalian medicine and husbandry was developing at gigantic strides and receiving all the attention, this was not the case for the lower classes. This appeared to follow the same trend everywhere in the zoological world. It was then that I decided to center my attention on Avian Medicine and husbandry and to try to make a difference.

Very early in my stay at the zoo I was asked to learn how to use the existing endoscopy equipment at the Animal Hospital, since we were going to use this relatively new technique to carry out a sex-determination project at the two collections of the Zoological Society of London. This extended over time to basically all major zoological and avicultural collections in mainland Great Britain and lead to specimen exchanges, a kind of marriage bureau if you like, between collections to maximize captive breeding. This was all possible by the support of my mentor, Mr. David Jones, to whom I will always be grateful for believing in me and giving me this magnificent opportunity.

My interest in sex determination lead to my involvement with cage design, nutrition and diets, artificial incubation and rearing methods, and then to artificial insemination. My other mentor, Dr. Christine Hawkey, then suggested that I should pursue a higher academic degree. I had never thought about this before, but it was a very exciting development. To make a long story short, instead of spending six months at the London Zoo as originally planned, I ended up staying for the better part of 7 years. My PhD centered on avian reproductive physiology covering areas as diverse as the anatomy of the cloaca of the budgerigars (*Melopsittacus undulatus*), the gonadal cycle, semen collection techniques, semen cryopreservation, and artificial insemination. The first part of the study consisted of using the budgerigar as a biological model to develop techniques that could be applied to endangered species. The second part consisted of the applied aspect of the study using the peregrine falcon (*Falco peregrinus*) as a model.

I would have loved to stay in England after completing my PhD. My dream was to establish an avian breeding center outside London to breed species such as the highly endangered Rothschild's mynah (*Leucopsar rothschildi*), the Mauritius kestrel (*Falco punctatus*), and other species in need of integrating into a captive breeding programs. Unfortunately at the time, the London Zoo was undergoing severe financial difficulties and the general mood was somber and uncertain. So with great sadness I left London at the end of 1987 to take a position in the Middle East. I still have the drawings I made of the proposed endangered avian breeding center. Those drawings are all that is left of my dream.

My association with falcons and falconry in the Middle East started in 1983 when I was asked to fly to the State of Bahrain to attend a bilateral bumblefoot case in a saker (*Falco cherrug*) falcon that belonged to the King of Saudi Arabia. If I say that from that day onward I became fascinated with falcons it would be an understatement and I have been devoted to falcon medicine ever since. Consequently, during my entire professional career spanning the past 30 years or so, I have strived to promote falcon medicine as a true specialty.

This book is a testament to the advances, not only in falcon medicine, but also in Avian Medicine in general worldwide. In the previous editions there has been a great emphasis on using photographic material from species seldom included in other books such as falcons and bustards. I have tried, together with the group of the most generous contributors I have ever worked with, to balance this with other species. I sincerely hope the result is satisfactory to the reader. You will notice some welcome additions to the table of contents as well as new contributors from every corner of the world.

I would like most wholeheartedly to wish the best to all students, veterinary surgeons, and those who acquire this book. May Avian Medicine continue inspiring many to embrace this unique specialty.

Jaime Samour
Wildlife Division
Wrsan, Abu Dhabi
United Arab Emirates, 2015

ACKNOWLEDGMENTS

I would like to thank Penny Rudolph, Content Strategy Director; Jolynn Gower, Content Development Manager; Brandi Graham, Content Development Specialist; Laura Klein, Associate Content Development Specialist; Deepthi Unni, Team Lead Production; Manchu Mohan, Project Manager; Katie Gutierrez, Content Coordinator; and Anna Schook, Processing and Development from Elsevier for their support, patience, and understanding throughout the production of this book.

I also would like to thank HH Sheikh Sultan bin Zayed Al Nahyan, the son of a true conservationist, for his outstanding interest and dedication to preserving the flora and fauna of the United Arab Emirates and for his continuing support to the activities of the Veterinary Science Department, Wildlife Division, Wrsan.

Jaime Samour
Wildlife Division
Wrsan, Abu Dhabi
United Arab Emirates
January 2015

1

Housing, Environment, and Public Awareness

Melodiya Nyela Magno

The fundamental needs of housing and the right environment for birds are not too different from that of humans. Housing for captive birds must provide protection from natural elements, changing weather, and natural predators; provide a sense of security; and must reflect a reasonable degree of sanitation. Most important, although commonly overlooked or ignored, is that the environment within the aviary or enclosure must encourage natural behavior including grooming, foraging, and breeding. Satisfying these fundamental needs promotes the health and well-being of birds, thus maximizing their quality of life in captivity (Fig. 1-1).

CAGE AND AVIARY DESIGN

When designing an aviary for any species of bird, aviculturists commonly encounter conflicting views ranging from promoting the esthetic aspect of the aviary, promoting the health and well-being of the bird, and providing a functional design that allows adequate cleaning and maintenance (Fig. 1-2). However, birds can also adapt to the aviary setup provided by the keeper. Arboreal or tree-dwelling species that descend occasionally to the ground to drink or bathe, such as the green turaco (*Turaco persa*), can be seen consistently roosting on a 1-meter high perch in the absence of a tall tree or a very high perch (Fig. 1-3). Birds unable to adapt to a new and unsecured environment eventually develop signs of stress and chronic diseases and may even die. Indications of stress range from feather plucking (e.g., psittacine species), stereotypical behavior, nervousness, and immune suppression. Excessive aggression related to overcrowding and space constraints can also be an issue of concern. An example of this was observed in a small flock of chukar partridges (*Alectoris chukar*) reported to have individuals displaying torticollis and head tilting. Newcastle disease was initially suspected; however, this was a case of space constraint as aggressive individuals pecked on the lateral aspect of the head of companions leading to ear injury and impaired balancing ability.

The design and appearance of cages is important in zoological collections and bird parks. Ideally, the cage should be covered with black PVC-coated mesh or painted black to achieve a "see-through effect," making the interior of the cage stand out and more obvious to viewers (Fig. 1-4).

Providing a great degree of freedom is one of the common goals in designing an aviary. This can be achieved by constructing large landscaped aviaries. However, capturing birds housed in such large enclosures for regular health checks and prophylaxis can be a challenge and heightens the risk for injury. In the process of pursuit and capture, birds could suffer injuries such as neck dislocations and wing or leg fractures. This is particularly important in species with long necks and legs (e.g., flamingos, storks, cranes). Birds may even escape from an open aviary by jumping or flying over fenced enclosures, making capture difficult.

The type of materials used in constructing aviary and aviary furniture is important. Newly installed galvanized wire may predispose psittacine birds to zinc toxicosis because they instinctively cling to cage mesh using their beaks. Washing and brushing down newly installed mesh using vinegar before transferring the birds helps minimize such cases (Fig. 1-5). Small cages can also be constructed with temporary sliding partitions that can be removed gradually to join two adjacent cages and increase the space. Aviaries should be constructed taking into consideration adequate exposure to the sun and minimizing direct strong wind.

Aviary design must incorporate the needs of the species housed. Cold moist conditions can be detrimental to desert species and hot conditions may cause problems for species from temperate and cold climates. Excessively cold conditions are involved in wing-tip edema syndrome of raptors and in toe frostbite for flamingoes. Translucent panels should be used to allow natural light exposure if enclosures are totally covered (e.g., seclusion). Alternatively, suitable artificial lighting should be provided.

In hot climates, birds must be kept cool. In the Middle East, outdoor falcon aviaries often have water-cooling systems at both ends and fans placed outside to directly cool air to the main perches. Most of the birds cool off by bathing and then sitting near the fan. Many bird species require indoor air-conditioning accommodation during the hotter months of the year (June to September).

Clean air is essential for the welfare of captive birds and keepers alike. Poor ventilation predisposes birds to respiratory infections and is considered a major contributory factor in the development of aspergillosis in falcons.

One of the major problems with raptors in captivity is the development of bumblefoot (pododermatitis). The cause of this is multifactorial, but the type of perch is critical. Perches covered with AstroTurf or coconut matting are ideal. A choice of perches with different diameters should be provided for all species of birds that perch, especially passerines.

In bird species such as Psittaciformes, digit and pedal injuries can be minimized by constructing double-walled partitions between aviaries distanced by a small gap to prevent bites by neighboring birds. For birds frequently staying on the ground, such as gallinaceous and anseriform species, foot injuries can be avoided by providing soft substrate such as sand and soil instead of concrete floors. The use of mats as flooring can be dangerous for species that habitually peck on the ground, such as ratites, and can lead to foreign-body obstruction of the gastrointestinal tract.

FIGURE 1-1 Places with consistently good weather such as in the tropics, do not require indoor facilities to keep the birds from harsh cold temperature, unlike in temperate regions.

FIGURE 1-2 A creative way of presenting owls' aviaries to the visiting public.

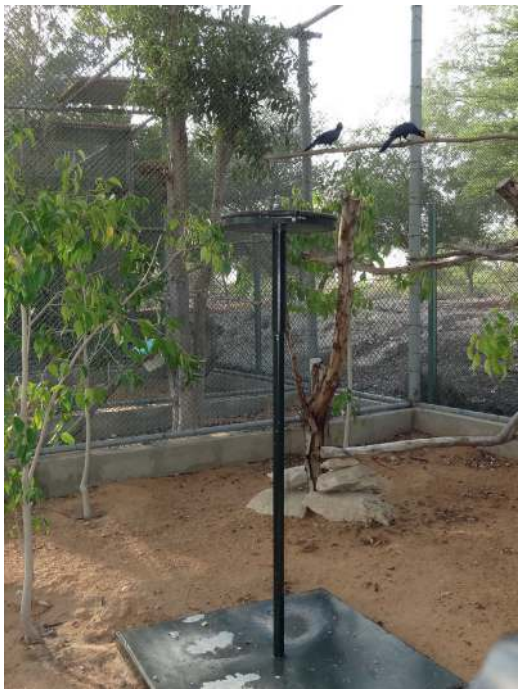


FIGURE 1-3 Arboreal (tree dwelling) species benefit from raised feeders and should not be fed on the ground.

FIGURE 1-4 Painting the cage mesh black creates a "see-through" effect, making the interior of the cage stand out and more visible from a distance compared with an aviary with unpainted mesh. Note the difference between the aviary in the back with its mesh painted black with the one in the front.

CAGE AND AVIARY MANAGEMENT

Eager aviculturists must realize that the moment they decide to house a bird, a creature with a great urge for freedom, in an aviary or cage, they must take full stewardship over its environment (Fig. 1-6). Essentially, the aviculturist must realize that a cage or an aviary is a controlled environment. The degree of control over this closed environment dictates the extent and gravity of health conditions that would arise throughout the captive life of a bird.

At the very start of aviary planning and construction, a proactive approach must be made by creating a setup that reinforces efficient cleaning and maintenance of the aviary. This begins with the selection of appropriate construction material because this can dictate the degree of sanitation that is required and feasible. Wood and other porous materials retain contamination and are difficult to sanitize. Gravel and sand floor substrate can be more challenging to sanitize compared with a concrete floor. The design of the ground of the aviary should ensure that the flooring is leveled and prevents stagnation of water after raining or cleaning because this could predispose birds to drinking contaminated water. However, the aviculturist must not construct a sterile aviary that is easy to clean and disinfect, but that is uninteresting and boring for the birds.

Feeding birds in captivity entails the provision of a variety of food items, optimal food quality, adequate food hygiene, and an efficient feeding strategy. It is important to provide the critical nutrients specific for the species in the collection. Pesquet parrots (*Psittrichas fulgidus*), for instance, require a diet that is high in vitamin A and fiber; otherwise, they succumb more easily to candidiasis. Food hygiene can be

FIGURE 1-5 Breeding aviary for a captive breeding program. Well-designed and strongly built aviaries are required to house medium to large psittacines in captivity. It is recommended to brush the mesh of newly built aviaries with a solution of vinegar to avoid zinc toxicosis.

FIGURE 1-7 Food presentation is very important. Offering fruit slices makes the large surface of food available for passerine species.

FIGURE 1-6 Close proximity of the perch to the nest is important. It allows more activity and interaction near the nest as the male tries to attract the female for nesting.

achieved by observing a good hand-washing routine and implementing adequate sanitation practices in the food preparation area. Applying the appropriate feeding strategies is very important to fulfill the nutritional requirements of individuals (Fig. 1-7). This could involve effective food presentation and setting up feeding stations at strategic locations, which can be achieved by first observing the behavior of individuals within a flock establishing a particular territory or a favorite roosting site within the enclosure. The latter should be the basis of both the location for the feeding stations and the number of feeding stations to be provided. When dealing with a flock, not all individuals come regularly to a central feeding station to feed. Keeping staff should expect that some individuals fear the more aggressive individuals and wait for them to finish feeding, ending up consuming the contaminated leftovers. In carnivorous species housed in groups, this entails a simple feeding strategy of providing an adequate food level; otherwise, cannibalism would occur.

Managing contamination involves an efficient waste-management system and a thorough sanitation program. Sound waste-management starts with providing the right amount of food for the bird population, resulting in less leftover food. This would significantly reduce the length of time for cleaning. Often excess contaminated food can be consumed by birds in the early morning the following day when they are most hungry and begin to seek food. The location of perches must be strategic and should not be placed over feeding stations to prevent fecal matter from falling onto the food. In a large aviary setting and with minimal manpower available to implement cleaning, *spot cleaning* practices (cleaning selectively areas where contamination concentrates) can save time and effectively reduce contamination. Personnel can get the most out of this practice if they prioritize cleaning critical areas such as feeding stations and areas beneath perches and favored roosting sites. Daily spot cleaning is more advantageous than conducting a major general cleaning after waste material has built up, forming fecal cakes. Another practice in preventing buildup of contamination is regular replacement of perches and other aviary furniture and changing the floor substrate (e.g., sand, soil) seasonally or as needed before saturation of fecal matter occurs. Changing the floor substrate seasonally prevents reinfection with parasitic forms such as nematode ova and protozoa cysts. Persistent infection due to *Capillaria* spp. and *Coccidia* spp. has been observed in birds of paradise housed in aviaries with soil substrate.

Cleaning and sanitation is a crucial aspect of good husbandry. It involves dry and wet cleaning. Dry cleaning refers to removal of organic matter. Wet cleaning refers to the use of detergent or soap and water to remove tough dirt buildup and removal of oily layers and biofilms. Disinfection of cleaned surfaces completes the sanitation process. Cleaning is a time-consuming process and it is likely that personnel will take shortcuts.

The use of outdoor footbaths can be controversial, since exposure to sunlight can deactivate chemicals and the presence of organic matter from footwear can reduce the effectiveness of disinfectant. Spraying the wheel of the vehicle with disinfectant, after spraying with water to remove organic matter, is more effective for controlling contamination.

Management of pools and ponds is critical because contamination by hydrophilic microorganisms can be a major issue. Water pump and filtration failure can lead to catastrophic situations and mortalities. In some institutions, ozone has been used to control contamination in pools and algal buildup.

Indoor environments for birds require a larger investment and a greater deal of husbandry. In the tropics, indoor environments could be difficult to manage because of buildup of humidity and the risk of hyperthermia when cooling systems breakdown. Sanitation using disinfectants can result in accumulation of fumes or odor, such as with bleach (sodium hypochlorite), resulting in respiratory irritation. Ammonia buildup should be prevented, especially in carnivorous birds (e.g., piscivores and raptors), because they consume more dietary protein so more ammonia is released from their fecal matter compared with other species of birds. Aside from harmful fumes from chemicals, cigarette smoke is unhealthy for birds. Birds kept in an indoor environment may have limited access to sunlight, resulting in hypovitaminosis D and metabolic bone disease. The provision of UV lamps can improve the situation.

Aviary management also entails several ways of controlling aggression to minimize morbidity and mortality. A limited number of nesting sites and nesting material can result in aggression. Sex ratio is a critical factor. Pheasants in captivity, for example, can be kept successfully if the ratio of one male to five females is maintained. Physical barriers such as thick vegetation can provide hiding places from aggressive individuals, but the downside of this practice is that such barriers would reduce visibility of birds from the viewing public. For bird species that could destroy plants, such as parrots, it is advisable to provide cut branches with leaves to minimize the destruction of aviary vegetation. In addition, to territorial species such as leafbirds (*Chloropsis* spp.), provision of large leaves for hiding is enough to modulate their aggression. Some personnel practice wing clipping on aggressive individuals to reduce the incidence of bullying. If this procedure is not done properly it can result in birds falling and acquiring keel injuries or more severe trauma (see Chapter 8).

Excess materials or refuse from maintenance work and construction in or around aviaries should be cleared after completion. Leftover plastic or nails and pieces of wire could be ingested by birds out of curiosity or hunger resulting in foreign-body obstruction and may require surgical intervention if not excreted normally in the feces.

EXERCISE

Every captive bird must have access to an exercise area. Young ratites require exercise to develop strong legs and falcons require a flying area to develop flight muscles and to reduce the incidence of pressure sores and bumblefoot (Fig. 1-8). Excessively long cages can be a problem for flying birds because the bird may build up speed and accidentally collide with the wall of the aviary, resulting in serious injury. Some of the larger breeding establishments now house young falcons in circular cages so that the birds can fly without risk of injury in a corner. Although more expensive, this may be the cage design of the future.

Unfortunate situations occur when birds are treated like ornaments or sources of entertainment and amusement (e.g., passerines and psittacines) and are kept in small cages, which is the most unnatural way of keeping birds. In general, small cages should accommodate twice the wingspan of the bird and where the head and the tail do not touch the roof and the flooring, respectively, when the bird is perching. The perch size should be appropriate such that the tips of the digits do not touch each other and its grip should cover three fourths of the total circumference of the perch. Adequate perch size and type of perch surface should promote even distribution of body weight to the digits and metacarpals, preventing pressure sores or pedal inflammation. In addition to the latter, obesity can develop from a sedentary lifestyle due to minimal opportunity for exercise compounded by the provision of high-energy commercial diets (e.g., all seed diets in parrots).

FIGURE 1-8 Providing bouncy perches strengthens the leg muscles of arboreal species of birds in captivity. As an alternative to monofilament wire, simple materials such as a green clothesline (plastic-coated braided wire) and an eye bolt provide more flexibility and can be useful in hanging bouncy perches. Note that the clothesline is camouflaged.

SECURITY

A double-door system is essential for most aviaries, but hanging a cloth or plastic strips at the entrance also works. Before entering an aviary it is best to alert the birds to your presence because they may panic, resulting in injuries. Handling some species of birds in the dark and with the aid of a small flashlight is an excellent practice. Raptors kept in seclusion tend to be especially nervous, requiring special care during approach. Some species (e.g., goshawks, cockatoos) display serious intersex aggression and require a hiding area in the breeding aviaries. Other species are gregarious and can be maintained with minimal aggression provided an adequate number of nest sites are installed. Aside from vegetation, shading cloth can be used as physical barriers to neighboring birds. Never keep a predator species and a prey species adjacent to other birds unless there is a physical barrier in between creating a completely opaque partition.

Pests can lead to significant economic losses in a valuable collection of birds. For example, rats can contaminate food and cages with urine and feces, transmitting disease such as salmonellosis. They can also kill small birds, or worse, destroy electrical cables, increasing the risk of fire. Packs of foxes and dogs have been reported killing birds in open exhibits and even large ratites such as an adult ostrich. The presence of pests reduces the sense of security of birds in captivity. In one instance, crowding and nervousness was observed in an aviary of pheasants in the presence of a cobra. Aside from pests, aviaries should be safe from human and animal disturbance. Rats, mice, and snakes not only kill young birds but may disturb the adults, resulting in poor breeding outcomes. The nesting area is especially vulnerable. Placing a sheet of Perspex behind a wall-mounted nest box is an excellent way to prevent predators from climbing into the nest box. Dogs and foxes can be a problem, requiring a solid wall around the perimeter of the aviary. Cats can also be a problem, but most birds become accustomed

to them provided they do not disturb nesting areas. Many species of captive birds are valuable, so steps must be taken to protect the aviary from thieves and vandals. In certain situations closed-circuit television and burglar alarm systems are both ideal security measures.

An efficient pest-control program is highly recommended and can be implemented as early as when aviary design is decided and construction is started. Good aviary designs effective in pest proofing the cage and environment, such as installing barriers at the top or at the base of the fencing, can minimize incidents of intrusion by canines (e.g., foxes, feral dogs), felines, and rodent species and eventually prevent mortalities and morbidities (Fig. 1-9, *A* and *B*). For smaller pests, entry of free-flying passerines (e.g., sparrows, starlings, etc.) into aviaries can be prevented by installing mesh with 1/2- to 1-inch fenestration. For snakes, drains can be entry points and should have fitted covers to keep them closed when not in use. For ant infestation, hanging cages are regarded as safe for birds less than 100 grams, which can die of anaphylactic shock and pain after acquiring excessive ant bites. In countries with a tropical climate, the aviary should be fitted with mosquito netting to prevent the transmission of disease carried by mosquitoes such as avian pox and malaria. The netting can also be extended to cover unroofed areas of aviaries, thus allowing contaminated fecal matter of free-flying birds to be sieved by the netting and desiccated under the sun, killing infective forms of parasites before they fall into the aviary. This method was even implemented in zoological gardens in Hong Kong at the height of the bird flu outbreaks to reduce fecal contamination by free-ranging birds. Mosquito netting can also be used to prevent bee colonies from establishing inside aviaries.

Preventive approaches to pest control can also be achieved with sound husbandry practices. Simple housekeeping can reduce clutter and potential breeding areas for rats. Removal of uneaten food in aviaries can prevent attracting wild birds and rodents that feed on food leftovers. Another option is to provide enough food for the bird to finish, resulting in minimal discarded food. In tropical climates during the rainy season, the practice of eliminating stagnant water is important to control mosquito breeding.

Pest control in some areas and under some circumstances can be achieved by using glue traps, spring traps, and poisoned bait. However, rats can be suspicious and adapt to trapping methods. Different options and techniques should be used periodically to avoid such adaptability. It is important to use poisons cautiously, making sure that the poisoned bait is situated outside the enclosures, and that it is absolutely impossible for the poisoned rat to enter the cage of a carnivorous species.

Another aspect of security is implementing safety features that distance aggressive and dangerous birds from the viewing public. This prevents visitors from offering unwanted items to the bird, such as inedible objects and unnatural food items, or worse, their own fingers and hands when they attempt to touch the birds on display (Fig. 1-10).

FIGURE 1-9 Aviary designs as practical ways for pest control. **(A),** Paddock appropriate for large flightless birds with fox-proofed fencing 3 meters high. **(B),** Aviary with rat-proofing 2 feet high at its base.

ENVIRONMENTAL ENRICHMENT

In the wild, birds are preoccupied with a diverse range of activities including finding shelter, guarding its territory, grooming, foraging, finding a mate, nest building, incubation, and raising chicks. Similarly, the environment of birds in captivity must cater to these activities. It is a constant challenge for a keeper to make the life of birds in confinement more interesting. It would be advantageous for keepers to set up a barren, sterile, and low-maintenance aviary that requires less work; however, this would be at the expense of enriching the life of birds under their care.

To know the appropriate enrichment to provide, the keeper must first be aware of the biology of the species and complement this with first-hand observation by patiently checking, at different times of the day, the birds' tendencies and behavior. From the information gathered, the type of enrichment strategy can be determined and implemented. Species that inhabit countries with wet and dry seasons would benefit from aviaries fitted with sprinkler systems that can simulate rain. Many larger collections operate a misting system for tropical birds, although care must be taken to avoid chilling. Birds are

FIGURE 1-10 Health and safety features can be installed. Fencing keeps visitors, especially children, away from an aggressive raptor.

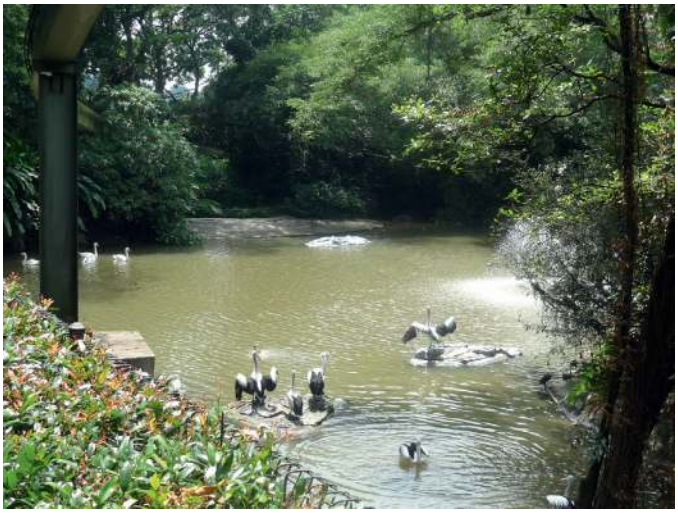


FIGURE 1-11 Though far from their natural coastal setting, a man-made pond with fish provides a good quality of life in captivity for a flock of pelicans.

photoperiodic organisms and light can dictate the onset of their breeding and molt cycles. The provision of insects can trigger breeding of insectivorous birds and other passerines, since insect populations rise on the onset of the rains in the tropics. It is advisable, before offering the insects to the birds, to gut-load them with minerals to increase the dietary calcium content and eventually support egg production. Providing and maintaining an adequate photoperiod within the aviary or cage is important. Extended lighting periods may result in disturbance of the reproductive cycle and molting, and may lead to feather plucking. Most species can be housed under a 12-hour lighting regimen. However, species in a breeding program should be exposed to 14 to 16 hours of lighting.

Birds exhibit varying degrees of socialization. Certain bird species are solitary (e.g., cassowaries) and some are communal (e.g., lories, cockatoos, flamingos; Fig. 1-11). Keeping communal species in solitary confinement can be detrimental to their mental and psychological health. However, in certain species having just two individuals with well-established pair bonding can be considered enrichment in itself since a partner can provide endless allopreening, sound enrichment, and companionship.

Examples of Enrichment for Birds

- A dish of sand can encourage sand bathing and be a natural way to control ectoparasites in the feathers.
- Provision of fresh branches or a wooden perch with bark encourages foraging and beak hygiene in parrots and raptors, respectively. By wiping and rubbing their beaks on rough surfaces, birds are able to prevent beak overgrowth and maintain the normal shape of their beak.
- Provision of projecting branches for hornbill species enables them to clean the interior of their beaks and prevent fungal infections.
- Incorporating food items with a container of dried leaves such as nuts and seeds for parrots and live prey such as mice for raptors can encourage foraging behavior.
- Provision of live fish in man-made ponds.
- Plants that produce seeds (e.g., legumes, palm trees) can be attractive to Columbiformes such as the pied imperial pigeons (*Ducula bicolor*) and psittacines such as the palm cockatoo (*Probosciger aterrimus*).
- Plants producing nectar that attracts insects.
- Rotten logs with inhabiting arthropods (termites), for insectivorous species.
- A tray with soil and arthropod/larvae such as crickets without limbs and mealworms to promote "soil probing" by insectivorous species (e.g., purple gallinulle [*Porphyrio martinica*]).
- Hanging a plastic container with holes and containing overripe fruit such as banana or papaya, to encourage breeding of fruit flies (*Drosophila* spp.) and provide for small insectivorous species such as the Oriental white eye (*Zosterops palpebrosus*) and sunbirds (Nectariniidae).
- Food must not be completely peeled or cut to provide a challenge during feeding.

MENTAL STIMULATION

In the past, the mental health of captive birds was given low priority. However, this has changed dramatically in the past 10 years and pet owners and keeping staff in bird parks and zoological collections are providing ways to stimulate natural behavior of birds under their charge. For instance, toys for pet birds are now very popular with parrot owners. Many birds also seem to benefit from listening to the radio and television, especially when the owner is not in the house. Captive gyrfalcons play with tennis balls in molting aviaries for hours on end. In bird parks or zoological collections, food-seeking behavior may be stimulated by placing food on different sites of the aviary and at irregular times of the day. This also encourages exercise. Parrots like to chew wood, and this activity can keep them active for hours and may also stimulate nesting behavior. Providing natural food can also stimulate birds in a captive environment.

Most captive birds appear to enjoy bathing. This provides a natural activity and improves plumage quality. Shallow bathing basins provided with slopes are advised, as a wet bird may have difficulty getting out of a deep bath.

INFORMATIVE AND EDUCATIONAL LABELING

Aviaries located in zoological collections and birds parks should have informative labels and graphic displays identifying the species. These

FIGURE 1-12 Educating the public about imprinting sends a message about the disadvantages of hand rearing rescued juvenile raptors. This signage states that "human imprinted" birds are unable to survive in the wild.

FIGURE 1-14 Pedagogic displays can help promote public awareness of the advantages of sustainable development strategies benefiting bird life in the wild.

FIGURE 1-13 An anthropomorphic sign reinforces the public to relate birds to humans and creates a better awareness of bird anatomy and structure.

should also contain detailed biological data of the different species to increase public awareness. This is a key responsibility of modern institutions because a variety of messages on the species can be conveyed, ranging from its geographical distribution and feeding habits, issues on imprinting, government regulations, and efforts from institutions to preserve the species in captivity (Fig. 1-12).

Anthropomorphizing birds with the aid of graphic displays can reinforce the association of anatomy and physiology between birds and humans (Fig. 1-13). The sign in Figure 1-14 is a simple and easy way to promote better understanding of the biology of birds in general.

2

Avian Intelligence, Clinical Behavior, and Welfare

Paolo Zucca

"It is essential to understand our brains in some detail if we are to assess correctly our place in this vast and complicated universe we see all around us."

Francis Crick

The recent discoveries of avian brain functions and organizations, their implications in the development of the new avian brain nomenclature, and the recent findings in the field of avian cognition are keystones for the understanding of avian clinical behavior and welfare. Historically, ethologists and ornithologists focused their attention, respectively, on the mechanisms of avian associative learning and avian behavioral ecology/ethology (Emery, 2006), and only during the last decade have scientists' attention shifted toward birds as models for the comparative studies of intelligence and vertebrate brain evolution. This chapter discusses recent findings in the field of avian brain neuroarchitecture and gives a brief overview of the most important topics in the field of avian cognition, clinical behavior, and welfare. These subjects cover a wide and complex scientific area and it is not possible to describe them comprehensively in a short book's chapter. For more information, please refer to the references and suggested further reading listed at the end of this chapter. Although this chapter refers to the whole Class *Aves*, some avian species/groups like corvids and parrots will be treated more extensively because of their special cognitive abilities, which are comparable to those of primates.

AVIAN BRAIN

Birds are able to perform problem-solving tasks and other complex cognitive tasks to the same extent as primates despite their lower brain weight to body weight ratio (Emery, 2004; Emery and Clayton, 2004; Rogers, 1997; Zucca, 2002). However, for many decades people and also scientists did not consider birds as intelligent animals and expressions such as "bird brain," or "dodo," which are found in numerous European languages, are proof of this (Emery, 2004; Zucca, 2007). The bad reputation of the avian brain could stem from the studies of Ludwig Edinger (1855-1918), who is known as one of the founders of modern neuroanatomy. He made many important neuroanatomical discoveries, but he also thought, combining Darwin's theory of evolution and a nineteenth-century version of Aristotle's "*scala naturae*," that brain evolution was progressive and not linear, from fish to amphibians, to reptiles, to birds and mammals, to primates and finally, to humans, ascending from lower to higher intelligence in a chronological series (Jarvis *et al.*, 2005). In a "folk" evolutionary scale, birds are usually positioned somewhere between reptiles and mammals (Fig. 2-1). The common notion that birds' brains are simple and that without a six-layered cortex birds could not be intelligent persisted for many years throughout the twentieth century. Starting from the 1950s there has been increasing evidence of the cognitive abilities of birds, and these early behavioral studies set the stage for a re-evaluation of the avian brain that appeared to be far more complex than was originally presumed (Jarvis *et al.*, 2005).

Even though the brain of a bird is made up of neurons and glial cells as in mammals, the neuroarchitecture is different (Rogers, 1997). In mammals, the mental operations generating cognitive abilities are associated with the prefrontal cortex (PFC), which has a "laminated" (layered) structure, whereas the corresponding structure in birds, the *nidopallium caudolaterale*, has a mostly nonlaminated structure (Güntürkün, 2005; Jarvis *et al.*, 2005; Vallortigara, 2000). Anatomical, neurochemical, electrophysiological, and behavioral studies show these structures are highly similar and extremely comparable (Güntürkün, 2005). According to a new understanding of avian brain organization, it has been estimated that, as in mammals, the adult avian *pallium* comprises about 75% of the telencephalic volume (Fig. 2-2) and processes information similar to mammalian sensory and motor cortices (Jarvis *et al.*, 2005). This knowledge is the basis for the re-evaluation of avian intelligence (Zucca, 2002).

An appropriate computer science metaphor when comparing mammalian brain to avian brain has been made by Pepperberg (1999). Mammalian brains are like the IBM PC while avian brains are like the Apple Macintosh. These different computers use the same wires and similar input gives similar results, but the wires are organized differently and these computers need different programs to achieve the same results.

Avian Brain Lateralization

Cerebral lateralization is an evolutionary ancient adaptation of the brain that contributes to biological fitness (Vallortigara *et al.*, 1999). It has been described in several species of vertebrates and also invertebrates. Each cerebral hemisphere is specialized in processing specific cognitive functions, and it primarily processes the input from the contralateral eye in fish, reptiles, and birds and in many species of mammals with laterally placed eyes (Fig. 2-3). The left hemisphere, which primarily processes input from the right eye, controls responses that require considered discrimination between stimuli and for control of motor responses involving object manipulation. The right hemisphere controls rapid species-typical responses, expresses intense emotions, and controls visuospatial processes centered on relational properties of the spatial layout (Rogers and Andrew, 2002; Vallortigara and Rogers, 2005).

The behavioral consequences of this neuroanatomical architecture are quite visible. Birds preferentially use their left or right eye for

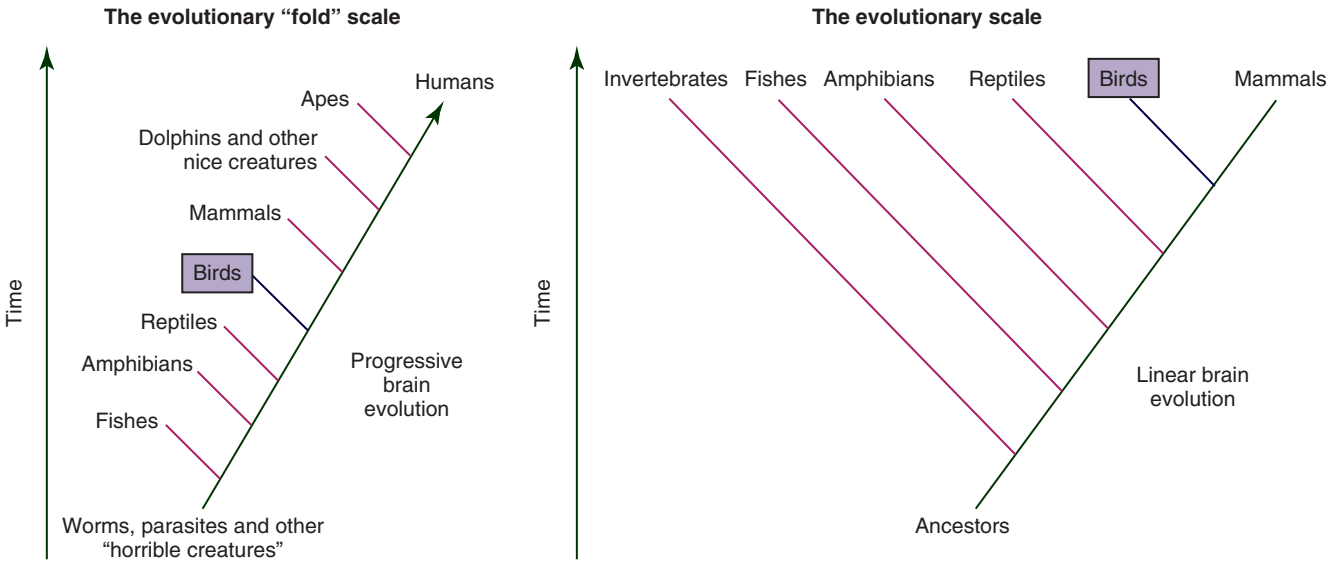


FIGURE 2-1 The progressive "folk" brain evolution scale (*left*) and the linear brain evolution scale (*right*), which underline how all living creatures have had the same time to evolve their nervous systems.

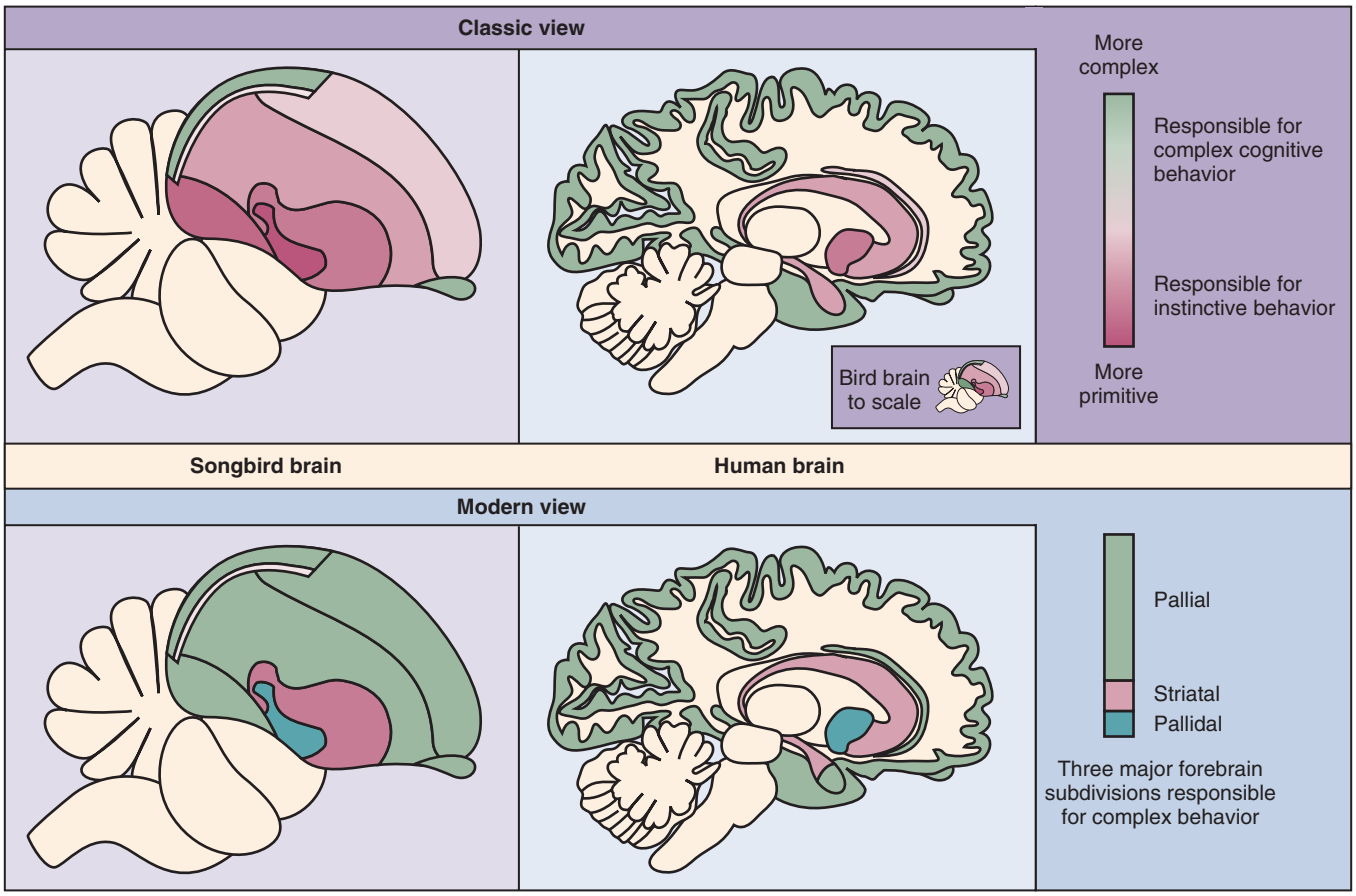


FIGURE 2-2 Avian and mammalian brain relationships. Side view of a songbird (zebra finch) and a human brain. Upper images report the classic view and the lower images represent the modern view of avian and mammalian brain relationships according to the Avian Brain Nomenclature Consortium. (From Jarvis ED, Güntürkün O, Bruce L, et al: *Nat Rev Neurosci* 6:151–159, 2005.) (Courtesy Professor Erich Jarvis, Duke University, North Carolina; Zina Deretsky, National Science Foundation; and Avian Brain Nomenclature Consortium.)

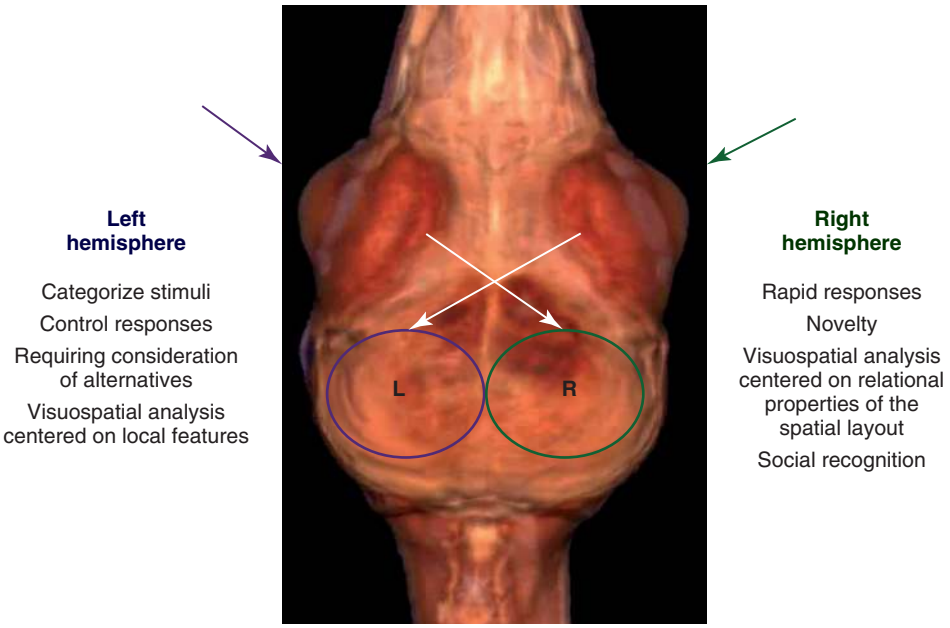


FIGURE 2-3 Each cerebral hemisphere primarily processes the input from the contralateral eye and specializes in processing specific cognitive functions. (From Zucca P: *Mind of the avian patient: cognition and welfare*, Proceedings of the 9th European Conference of the Association of Avian Veterinarians, Zurich, Switzerland, 2007.)

viewing, respectively, novel or familiar stimuli (Dharmaretnam and Andrew, 1994; Zucca and Sovrano, 2008). Recent studies demonstrated that neuroanatomical asymmetries in starlings (*Sturnus vulgaris*) are not only a prerogative of the central nervous system but also a peripheral sensory organ like the *retina* shows a morphological asymmetry in terms of photoreceptor distribution. According to these results it is possible to hypothesize that, at least in this avian species, color discrimination would be best accomplished with the left eye while movement detection is best performed by the right eye (Hart *et al.*, 2000).

The distribution of lateralization may occur at the individual or population level. If most of the individuals of a population exhibit a side preference, but there is approximately an equal number of right and left preferences, lateralization is manifested at the individual level. When the majority of individuals show the same directional bias, then we can say that lateralization is present at the population level (see Vallortigara and Rogers, 2005 for a detailed review). As reported by Ghirlanda and Vallortigara (2004), behavioral and brain lateralization at the population level is the rule rather than the exception among vertebrates.

There is evidence supporting the idea that lateralized brains are more efficient in terms of cognition and fitness (Rogers *et al.*, 2004; Vallortigara *et al.*, 2005). For instance, it has been proved, in the domestic chicken, that cerebral lateralization enhances brain efficiency in cognitive tasks that demand simultaneous but different use of both hemispheres (Rogers *et al.*, 2004). The existence of a fitness advantage in terms of enhanced cognitive functions due to laterality also has been found in Australian parrots (Magat and Brown, 2009). Brain asymmetry is a universal phenomenon characterizing not only cerebral control of cognitive or emotion-related functions but also cerebral regulation of somatic processes. The manifestation of brain laterality in the control of bodily processes is as pronounced as that of cognitive or emotional functions, and stress responsiveness to acute or chronic diseases seems to be mediated, respectively, by the right and the left hemisphere (Wittling and Schweiger, 1993; Wittling, 1997, 2001).

FIGURE 2-4 A group of hooded crows (*Corvus cornix*) during foraging behavior. The two individuals on the right show clearly a standing position on the right leg. During data collection, especially when working in the field, it is better to score the bird's motor behavior from the side to avoid parallax errors.

It is useful for the clinician to know the degree and strength of laterality bias of the avian patient because a patient with a laterality pattern different from the population trend could require much more attention because of the evolutionary health cost of being left-handed. One of the easiest procedures to assess the laterality bias in a bird is to record the position of the bird leg while grasping food (parrots) or standing (many other avian species) in relation to one another. Sampling is made when the bird maintains the bipedal standing position for at least 5 seconds yielding 30 observations for each subject (Fig. 2-4). The standing behavior can be subdivided into standing with the left forelimb advanced (SL), standing with the right forelimb advanced (SR), or standing square (SS), i.e., with legs level with one another (Zucca *et al.*, 2011). The laterality index (LI) of motor

preferences for each subject can be calculated as (SR/SR + SL) × 100, where SR is the number of times observed standing with the right foreleg advanced and SL is the number of times observed standing with the left foreleg advanced (LI > 0.5 = right forelimb bias; LI < 0.5 = left forelimb bias; LI ≅ 0.5 is not significant.

The understanding of the laterality bias of an avian species can be used as a tool to predict the behavior and emotional state of the avian patient. When a hemisphere is selectively involved for solving a specific task, our attention is directed to the visual hemifield of that hemisphere by orienting the contralateral eye and the head to the target (turning on the opposite side). From a neurobiological point of view, several avian species like chicks and quails have a dominant right hemisphere for social recognition. The left eye is used preferentially to scrutinize familiar stimuli while the right eye scrutinizes novel ones. Quails turn leftward when viewing a stranger (right eye), but they turn rightward (left eye) when viewing a companion (Zucca and Sovrano (2008). Therefore we can infer that a quail shows cooperative or uncooperative behavior by simply observing the preferential eye used during the first steps of interaction with a conspecific or a veterinarian. The understanding of the neural mechanisms responsible for these signals allows us to infer the emotional state of an "avian patient" and to understand in which category (*familiar/known* or *foreign/unknown*) the animal places the observer. This could be useful during handling procedures, pairs formation, assessment of social interactions, and during pets versus human interactions (Zucca *et al.*, 2009).

Avian Senses and Perception

According to Jakob von Uexküll (1934), the perceptual world in which an organism exists and acts as a subject *Umwelt* (the world that is around the animal) is not the same thing as the *Innenwelt* (the world perceived and internalized). One of the most deceptive illusions is the belief that there is only one world in which all living creatures are contained and that space, time, and sensorial perception are the same for all species. Actually, the subjective worlds are countless and each animal species has its own combination of sensory windows related to its evolutionary history and environmental pressures (Sovrano *et al.*, 2013). Birds are primarily "sight animals" because the visual system is the most important sense for acquiring information from the surrounding environment (Fig. 2-5). Also their sense of hearing is very well developed while smell, taste, and touch show greater variability in terms of importance among the class *Aves*. Furthermore, many avian species are able to sense the world in a way that humans cannot. Echolocation (biosonar), solar compass, magnetic compass, and sensitivity to air pressure are frequently defined as "exotic senses" because they are neglected in the human sensorial experience (see Table 2-2).

FIGURE 2-5 The trichromatic vision of man cannot penetrate fully the colors perceived and internalized by the birds that have pentachromatic vision. Therefore the dorsal feathers of a male golden pheasant (*Chrysolophus pictus*), which seem to us extremely colorful and bright, are actually carriers of chromatic messages far more complex than those visually perceived by humans: they hide an invisible world under the visible one.

The key to understanding avian senses is variability; there are more than 10,000 different bird species colonizing virtually all the continents. The range of sensory specialization is vast among the Class *Aves* and it varies from one Family to another according to the different habitat and the related environmental pressures (Zucca, 2002). Table 2-1 gives a brief overview of the avian senses, whereas Table 2-2 lists the known avian "exotic senses".

The internalized world of an animal is not simply a matter of sensory perception: other factors such as attention, motivation, emotional profile, personality, and individual attitudes are all pieces of a larger puzzle representing the cognitive phenotype of a single individual. The input process of the same sensorial fragments generates a peculiar behavioral output that is different for each individual bird.

Attention and Selective Attention

Attention can be defined as the choice process that a bird makes when selecting relevant *stimuli* from the external environment and when inhibiting irrelevant information, switching among these, as the situation requires. It has been demonstrated that a genetic base for selective attention exists in some species of birds; for instance, one-day-old chicks are strongly attracted to bright objects and peck them much more than other objects in the surrounding environment. This genetic-based selective attention helps them discover water because they do not have any specific sense for detecting it. Chicks discover water only when they touch it for the first time with their beak.

Motivation

Motivation can be defined as the internal reversible processes that are responsible for behavioral changes. A modification of the internal state of the bird modifies its response to a fixed external *stimulus*. For instance, if a raptor is not hungry, it will ignore any prey that passes close to it. Motivation is very important to understanding avian behavior because a lack of response to a current *stimulus* might reflect the inability of the bird to cope with this task, or a deficit in selective attention, but it might also reflect lack of motivation.

Emotions

Fundamental emotional systems are a common phylogenetic inheritance in vertebrates (Kotrschal, 1995), and the study of behavior is one of the preferred methods for the study of emotions. Emotions arouse observable behavior that reveals the state of the animal and represents a major motivational basis for this behavior. It is clear that animals exhibit emotional behavior that suggests an emotional experience similar to ours; they share a common evolutionary history with humans and emotions simply evolved and are widespread among vertebrates because they are useful, adaptive, and increase animal fitness. Aside from the countless anecdotal examples, there is a great deal of evidence that suggests the existence of an emotional life in animals (Sovrano *et al.*, 2013).

Anatomical evidence: The limbic system includes several subcortical brain structures involved in motivational processes, emotions, and in the enrichment of emotional memories. This system is present in all species of mammals and, because of recent progress in the revision of avian neuroanatomy (Jarvis *et al.*, 2005), it is possible to identify analogue areas in the brains of birds.

Functional evidence: Brain functional scanning technology demonstrated that crows use the same part of their brains as do humans when confronting a known danger (Marzluff *et al.*, 2012).

TABLE 2-1 Avian Senses

Avian Senses	Short Description
Vision	Most birds have excellent vision because of an evolutive pressure for controlling their body in a three dimensional world Flight requires a fast and sensitive input related to the location of objects in the surrounding area Able to process visual information rapidly Large visual fields and great visual acuity Compared with humans they have superiority in the field of color vision Pentachromatic vision that extends the frequency range of colors to which the bird is sensitive Some species also can extend their vision in the ultraviolet spectrum and polarized light Able to focus close up for feeding and at the same time see far away on both sides scanning for predators Central, elongated, or two *fovea*
Hearing	Birds, especially songbirds, have the most complex auditory signals among vertebrates This sense is very important for communicating with each other Some avian species hear and emit sounds above the range of the human perception
Smell	The sense of smell seems to be the least developed sense for many species of birds, although several avian species use smell to locate food from great distances (vultures, albatrosses, petrels, and other seabirds), find their way home (pigeons), or recognize kin from their odors (some petrel species identify kin from the wax smell they use to preen their feathers; Bonadonna and Sanz-Aguilar, 2012) Researchers compared the diameter of the olfactory bulb with the diameter of the encephalon of various species of bird and obtained interesting results that confirm the abovementioned behavioral observations: house sparrows (5%), diurnal raptors (14%-17%), nocturnal raptors (18%), pigeons (22%), pelicans (37%), and kiwi (33%) (Bang and Cobb, 1968) There is genetic evidence that many bird species have a well-developed sense of smell; it has been provided by analyzing the number of olfactory receptor genes in different avian species (Steiger *et al.*, 2008).
Taste	Birds do have a sense of taste and they have taste receptors like many other vertebrates Many species, including parrots, fruit-eating birds, and other species, can taste sweet, but it seems that their responses to sour, bitter, and salty vary species by species This variability in terms of sensitivity among avian species seems to be related to the importance of the sense of taste for finding food sources
Touch	Sense of touch is vitally important to birds, particularly for flight because with their feathers, birds obtain physical information from the surrounding environment; during flight they adjust their motor responses according to the different environmental changes Sense of touch is also used by several species of birds like oilbirds, corvids, and nightjars that have special feathers called rictal bristles around the base of the beak: the sense of touch might be useful when drumming into mood or catching insects The most widely distributed mechanical tactile receptors in the body of birds are the Herbst corpuscles found in the beak, in the legs, and in the feathered skin (Gottschaldt, 1985)

TABLE 2-2 Avian Exotic Senses

Avian Exotic Senses	Short Description
Echolocation	Birds' echolocation is restricted to lower frequencies audible to humans; their system has a poorer resolution than the ultrasonic (>20 kHz) biosonar of most bats and toothed whales, and for these reasons it is labeled frequently as rudimentary Echolocation has been found in at least 16 existent bird species (oilbirds and swiftlets) and has evolved several times in avian lineages (Brinkløv *et al.*, 2013)
Magnetic compass (magneto reception)	Many avian species use the earth's magnetic field to navigate
Sun compass	Orientation from the position of the sun and the time of the day
Sensitivity to air pressure	Sensitivity to changes in air pressure—differences in altitudes of 10 meters has been found in pigeons (Kreithen and Keeton, 1974)

Neuro-endocrine evidence: Manifestation of an emotion is connected to precise biochemical changes in the brain. For instance, happiness and stress activate the secretion of different neurotransmitters and neurohormones, and this phenomenon occurs in a similar manner both in man and in animals. The treatment of emotional and behavioral disorders of domestic animals, including birds, is normally performed with the same drugs used in human medicine for the treatment of analog emotional disturbances.

Behavioral evidence: Humans are strongly attracted to the emotional characteristics of the animals with which they share their life. All parrot owners are absolutely convinced that their birds feel emotions similar to those of humans, and any manual about parrots mentions specifically for each species at least a couple of emotional characteristics analog to humans. This emotional approach is often labeled as anthropomorphism in science. However, things are changing because of extensive research performed both in the

laboratory and in the field. Many animals exhibit a profound prostration following the loss of a friend or a partner, and Jane Goodall observed this phenomenon by studying chimpanzees (Goodall, 1991). The "depression by abandonment" is a well-known phenomenon in the avian field, with particular reference to social species such as parrots.

Positive Emotions

Pleasure plays an important role in an animal's life and has great significance to humankind's relationship with other animals (Balcombe, 2009). It is generally accepted that animal welfare is not simply the absence of negative experiences, but requires that the animal should also have positive experiences. There are still a few papers that deal with these aspects (Boissy *et al.*, 2007). Also from the regulatory point of view, pain and suffering of animals are present in the regulatory repertoire of many countries and lacks a formal recognition of the fact that animals can experience positive emotions (Balcombe, 2006; Fig. 2-6).

We must dispel the common thought that the days of animals are a continuous alternation between obtaining food and escaping from predators. Many species of animals belonging to different classes, from birds to mammals, show behavior of play and social interactions suggesting the existence of a positive emotional life. The emotional categories most frequently used to describe the emotional state of parrots are love, fear, joy, loneliness, boredom, depression, anger, and distrust. It is important to underline that an animal frequently experiences more than one emotional category at the same time.

Positive emotional needs increase the importance of proper social interaction, mental stimulation, and environment enrichment. These needs should not be considered as a priority only for some avian social species like psittacines; avian veterinary surgeons should avoid an emotional approach to avian intelligence and should not underestimate the cognitive abilities and the related mental/welfare needs of their non-psittacine patients. The morphological and behavioral characteristics of some avian groups such as raptors, which are proud, noble, and strong, have led avian veterinary surgeons to underestimate these birds' capacity to feel pain and to be stressed by a stimulus-poor environment. Pain therapy, environmental enrichment, and "behavioral therapy" should enter the daily work of every avian practice and wildlife rescue center, and owners should be informed of the "mental lives" of their avian pets (Zucca, 2002, 2007).

FIGURE 2-6 Pecking the tails of cats is a risky play behavior of hooded crows (*C. cornix*), and it is not strictly related to food access. The same behavioral pattern has been observed with hooded crows that peck the tail feathers of griffon vultures (*Gyps fulvus*).

INTELLIGENCE AND COGNITION

The great development of cognitive studies on birds in the past decades suggests that certain cognitive abilities that, until not long ago, were attributed only to a few species of primates, seem to be widespread not only in mammals but also in the Class *Aves* (Emery, 2004; Vallortigara and Rogers, 2005; Zucca, 2007). Historically, early studies of avian behavior were focused on ecological aspects rather than on intelligence (Marler, 1996). Furthermore, avian intelligence tests were initially based on long checklists of different problems that a species may or may not be able to solve. However, cognitive abilities of birds did not evolve by themselves randomly in a sterile laboratory, but their expression is the result of the bird's abilities to cope with different environmental pressures. Therefore the avian mental skill looks very different from species to species according to their evolutionary context. As mentioned previously, the ability of solving a cognitive task is related not only to the evolutive environment but also to motivation, individual mental abilities, and anatomical features of each avian species; for instance, corvids and parrots have different beaks, and this anatomical feature strongly influences their problem-solving strategy when compared with the same cognitive task (Auersperg *et al.*, 2011). Although there are more than 9000 species of birds, it is clear that some avian groups show cognitive abilities that can be compared with those of great apes. For instance, corvids and parrots comply with the same social and ecological constraints as apes, and their ape-like cognitive abilities are an example of convergent evolution of the development of intelligence among vertebrates (Emery and Clayton, 2004; Emery, 2006).

Avian cognition can be defined as the study of the mental abilities of birds. Historically it was developed out of developmental and comparative psychology, but many other scientific disciplines like ethology, ecology, and anthropology contributed to the comparative study of minds. These different theoretical approaches to the study of the avian mind strongly influenced the choice of experimental paradigms and investigation methods used by researchers. The following is a brief overview of the most important topics in the study of avian cognition and intelligence: Table 2-3 gives a short list of attributes and abilities that have been used to study avian intelligence, whereas Table 2-4 contains a list of cognitive attributes/abilities investigated in some avian species. The sources used in this overview are listed in the bibliography and suggested readings to allow more in-depth research.

Theory of Mind

The study of the theory of mind in animals is still an open and controversial subject because this high cognitive skill is strictly related to the existence of animal thinking and self-awareness. However, it has been proved that a great variety of animal and bird species, such as corvids or parrots, have high cognitive abilities and show complex behaviors like imitation, teaching, and tactical deception that probably require the ability to attribute mental states to oneself and to other animals.

Consciousness

This is probably the most controversial topic in the field of animal cognition, and there is a great scientific debate about the definition, the existence, and the evolution of this mental ability. One of the simplest definitions of animal consciousness identifies this ability as the quality or state of self-awareness or being aware of any external object. According to the Cambridge Declaration on Consciousness (2012):

Text continued on p. 18

TABLE 2-3 A Short List of Attributes and Abilities Used to Study Avian Intelligence

Attributes and Abilities	Short Description
Amodal completion and illusory perception	Amodal completion is the perception of partly occluded pictures while illusory perceptions are cases in which perceptual systems misrepresent a "real" sensory stimulus (Fig. 2-7)
Art discrimination (good and bad)	Humans have the ability to create art but pigeons seem to be able to discriminate between good and bad children's drawings in strong concordance with the judgment of adult humans (Watanabe, 2010)
Biological movements patterns	Some avian species seem to show a spontaneous preference to approach/avoid certain biological motion patterns, suggesting the existence of evolutive avoiding mechanisms of predators
Communication	Vocal communication, semantic (meanings of the calls have been demonstrated in several avian species); innate ability for grammatical structures like nouns, adjectives, and verbs (parrots); starling vocalizations may have recursive structures The existence of gestural communication in birds (ravens) has been proved
Concept learning	Concepts are the mental categories that help classify objects, events, or ideas; birds, for instance, seem to understand the concepts of same–different, equivalence, and generalization
Cooperative behavior and teamwork	Several avian species, such as corvids or falcons, cooperate while searching for food
Imitation and teaching	Not just the simple reproduction of behavior but also the understanding of the relationship between that behavior and the model (i.e., the existence of a substrate of intentionality) Target behavior should not be part of the observing bird's behavioral repertoire
Learning	Easily defined as a modification of behavior as the result of individual experience: song learning, social learning (jackdaws do not automatically recognize predators and need to learn it from their relatives), etc.
Means–end relationship ("naive" or "folk physics")	How the animal understands the functional properties of tools These tests were originally used by developmental psychologists to study infants because understanding means–end relationships is a key step in human cognitive development This paradigm has been used several times in ravens, Eurasian jays, and several other avian species
Memory	Capacity of encoding, storing, and retaining past experiences and information that birds pick up during their life Memory can be classified according to temporal criteria and coding criteria Several kinds of memories have been investigated in birds, such as short-term/long-term, implicit/explicit, episodic-like (what, when, and where; Fig. 2-8), etc. Planning for the future; recalling incidents from the past; mentally modeling the thinking of their peers; hiding and caching food; and remembering food resources, events, and enemies are behaviors with great survival value for several avian species
Mental rotation of images	Pigeons seem to be relatively better than humans at discriminating mirror-image shapes
Mirror recognition	This test requires that the bird understands that one's own mirror reflection does not represent another individual but oneself; it has been demonstrated only in European magpies (Fig. 2-9).
Navigation	Orientation ability toward a defined final destination that the animal cannot see or feel from the place of departure Many mechanisms have been proposed for bird navigation: landmarks, sun compass, star compass, magnetic map or magnetoception, polarized light, and olfactory maps All of these mechanisms might integrate into each other in a navigation path-integration process
Numbers and counting	Relative numerosity judgment (subitizing): rapid accurate and confident judgments of numbers performed for small numbers of items more or less of something Counting is the action of finding the number of elements of a finite group of items (Fig. 2-10; Pepperberg, 1994).
Object permanence	Objects are separate entities that continue to exist even when out of the observer's sight This cognitive ability is very important for all vertebrates, and it has been investigated in several species of birds
Play behavior	Not only training for learning novel behavior in young subjects (who mimic adult survival behavior), but it has a main role in the cognitive and motor development of birds: according to affective neuroscience, there are many links between play behavior and brain neurogenesis
Painting styles discriminations	Pigeons can discriminate novel color slides of paintings of Monet and Picasso by style artist categorization, and they use different cues for different discriminations (Watanabe *et al.*, 1995).
Tactical deception (bluff)	Action of propagating or transmitting false/misinformation by an animal to another of the same or different species It does not imply necessarily consciousness, although it requires higher brain functionality and complex cognition background

TABLE 2-3 A Short List of Attributes and Abilities Used to Study Avian Intelligence—cont'd

Attributes and Abilities	Short Description
Time perception	Time of occurrence (circadian phase) and temporal intervals Very important for sun navigation: it seems birds have a time compensation ability to make allowances for changes in the sun's position over the course of the day Smaller animals tend to perceive time as if it is passing in slow motion; the ability to perceive time on very small scales may be the difference between life and death for fast-moving organisms (Healy *et al.*, 2013; Fig. 2-11).
Social behavior	According to several researchers, the more social birds are, the smarter they tend to be A complex social behavior is frequently used as an indirect assessment instrument of intelligence in highly social avian groups such as parrots and corvids
Tool use	Requires a complex cognitive level A tool can be defined as a physical object other than the animal's body that has been modified to fit a purpose or for a future use Described in several avian groups like parrots, corvids, finches, vultures, gulls, owls, etc. (Fig. 2-12; see also Table 2-2).

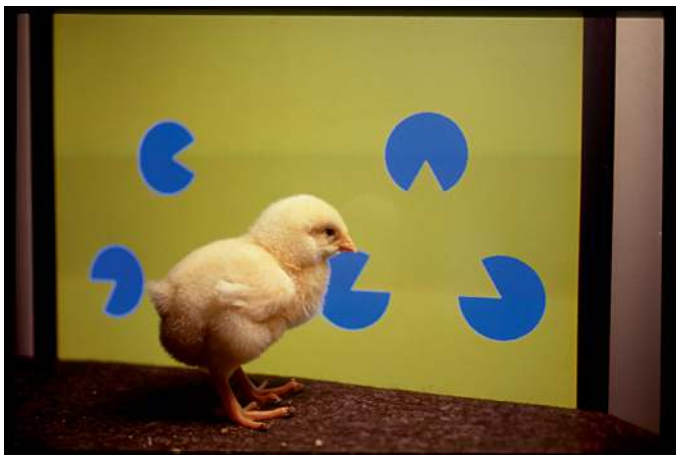


FIGURE 2-7 Chicks, as many other avian species, "see" the illusory triangle of Kanizsa. (Courtesy Professor Giorgio Vallortigara, University of Trento, Italy.)

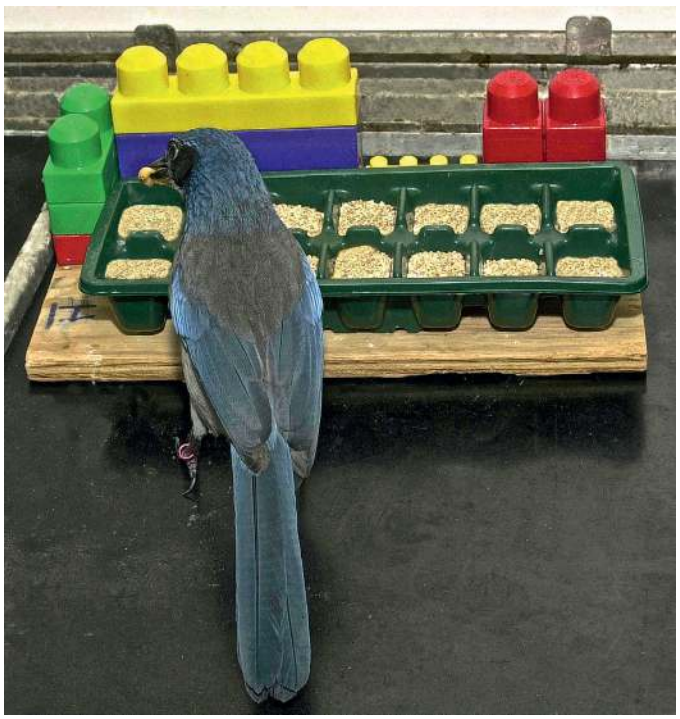


FIGURE 2-8 The recollection of past experiences allows us to recall what a particular event was and where and when it occurred—a form of memory that is thought to be unique to humans. Clayton and Dickinson (1998) demonstrated that scrub jays (*Aphelocoma coerulescens*) remembered what food items they had cached where and when they had cached them, fulfilling the behavioral criteria for episodic-like memory in nonhuman animals. (Courtesy Professor N. S. Clayton, University of Cambridge, UK.)

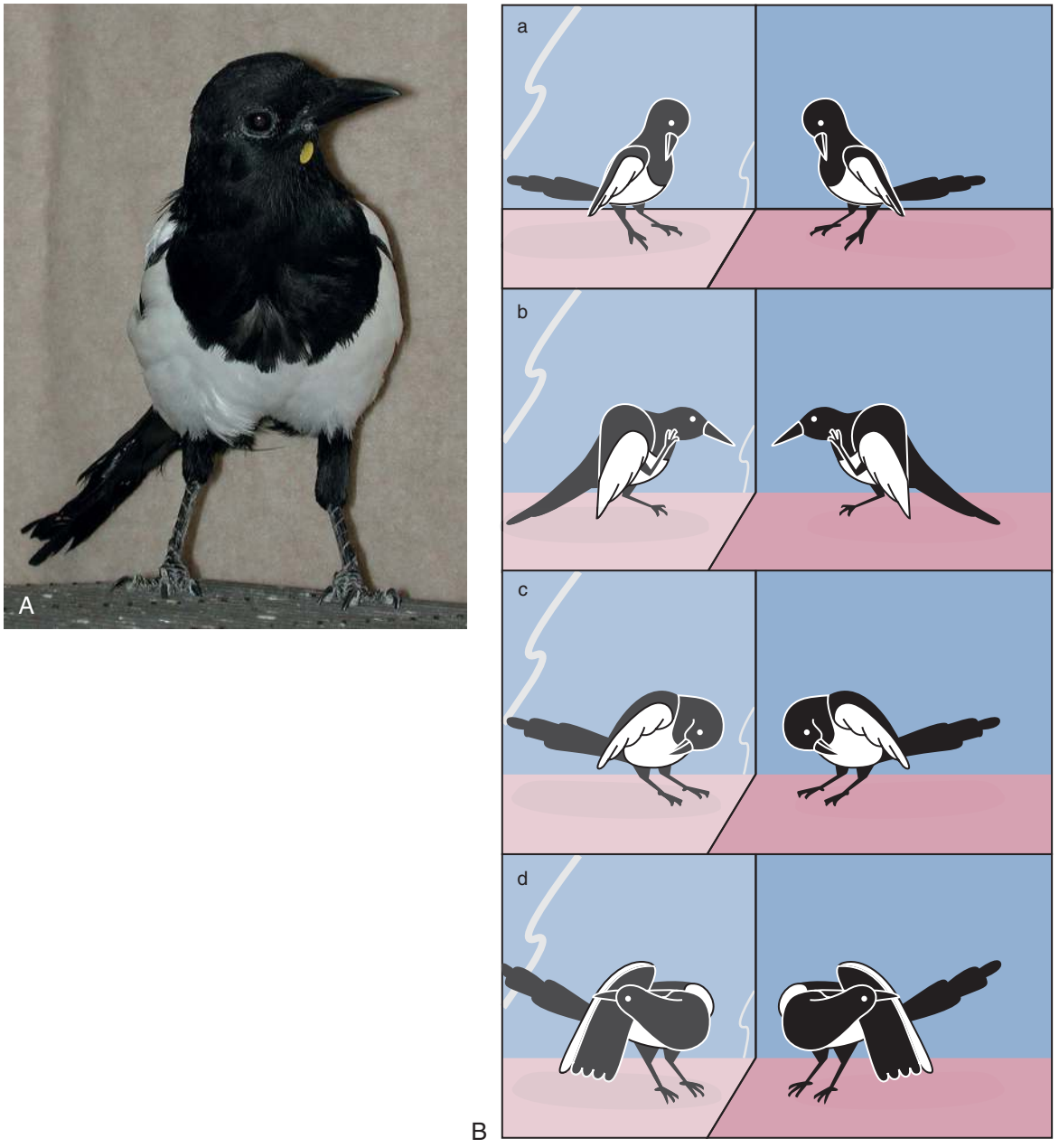


FIGURE 2-9 **(A),** Self-directed behavior classified by responses to a mirror has been investigated in several mammals like apes, dolphins, and elephants. Prior *et al.* (2008) gave the first evidence of mirror self-recognition in a non-mammalian species, the magpie (*Pica pica*). They suggested that essential components of human self-recognition have evolved independently in different vertebrate classes with a separate evolutionary history. **(B),** Examples of the behavior that were used for quantitative analysis. (**A** and **B** were mark-directed behavior.) (From Prior H, Schwarz A, Güntürkün O: *PLoS Biol* 6(8):e202, 2008.)

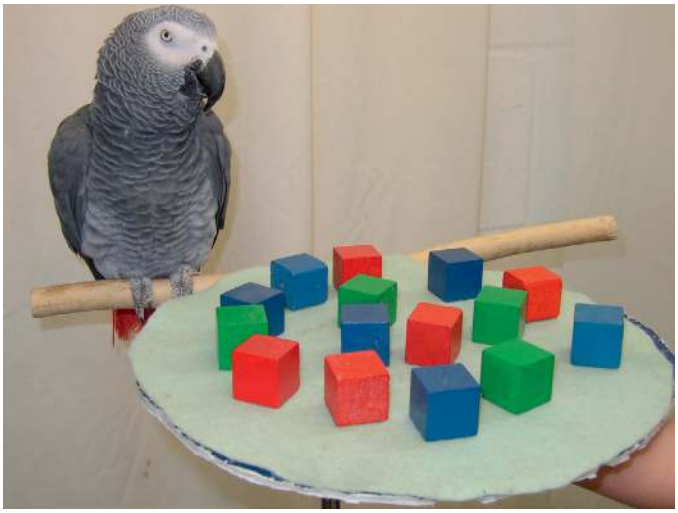


FIGURE 2-10 Alex, an African grey parrot, while participating in a numerical competence task (Pepperberg IM: *J Comp Psychol* 108:36–44). (Courtesy Alex Foundation, Professor Irene Pepperberg, Harvard University, Boston Mass.).

FIGURE 2-12 A New Caledonian crow (*C. moneduloides*) with a tool in the beak. Hunt (1996) gave the first evidence of tool manufacture and use in an avian species. (Courtesy Professor Gavin Hunt, The University of Auckland, New Zealand.)

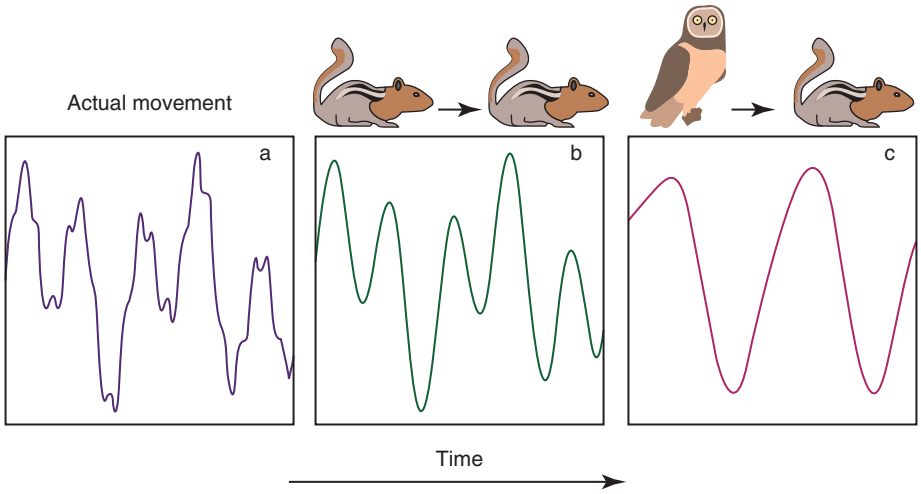


FIGURE 2-11 The ability of an organism to track a moving object depends on the time integral over which the individual can obtain its information. This is determined by its ability to resolve temporal information. Where an animal, such as a ground squirrel, displays complex movement **(a),** conspecifics may perceive the individual as moving according to a first-order integral of its actual movement owing to its high temporal resolution abilities **(b)**. However, a species with lower temporal resolution abilities, such as a short-eared owl, may perceive the motion as an even higher order derivative of the actual motion, meaning information of prey motion at finer temporal scales is not available to it **(c)**. (From Healy K, McNally L, Ruxton GD, et al: *Anim Behav* 86(4):685–696, 2013.)

TABLE 2-4 List of Cognitive Attributes/Abilities Investigated in Some Avian Species

Avian Species	Cognitive Attributes/Abilities Investigated
Pigeons	Discrimination and categorization (painting style, trees, water, cars, humans, paintings styles, same–different), geometry and landmark representation, memories, homing, future planning, navigation, inferential reasoning, brain lateralization, physical cognition, execution of sequential planning, echoic memory, picture object recognition, abstract–concept learning (same–different)
Parrots (African grey parrots, keas, and others species)	Abstract concepts of discrimination and categorization, numerical competence, reciprocity, cooperative problem solving, vocal learning, object permanence, exclusion performance, different types of memories, tool use, brain lateralization, physical cognition and naive physics, conspecific discrimination, experimenter cue, flexibility of problem solving, lifelong vocal learning, exclusion tasks, synchronization to a musical beat, discrimination of discrete and continuous amount, experimenter-given cues, referential signaling, zero-like concept
Corvids (ravens, rooks, jays, nutcrackers, New Caledonian crows, etc.)	Means–end comprehension, naive physics, memories (spatial, working, episodic-like, etc.), future planning, flexibility in problem solving, transitive inference, symbolic distance, rapid problem solving, numerical competence, theory of mind, tactical deception, exclusion tasks, cooperative problem solving, trap tube test
Chickens	Visual illusion
Greater hills mynah	Object-discrimination learning
Vultures	Problem solving (turkey vultures), tool use
Humming birds	Memory, spatial navigation, spatial learning
Zebra finches	Physical cognition
Cowbirds	Spatial cognition
Songbirds	Auditory perception, pattern generalization, working memory
Starlings	Affective state, video playback, behavioral plasticity
Darwin finches	Physical cognition (trap tube test), tool use
North Island robin	Large quantity discrimination
Canary	Song modulation and attractiveness, social transmission of information, memory and discrimination tasks, detour behavior
Many avian species	Time biological clock, associative learning, insight, social learning, phonetic and syntactic processing abilities
Crossbills	"Larger than" concept
Magpie	Self-recognition, object permanence
Ring dove	Stage 4 object permanence
Quails	Imitative learning, memories, detour behavior, social recognition
Java sparrows	Auditory discrimination
Herring gulls	Detour behavior

Birds appear to offer, in their behaviour, neurophysiology, and neuroanatomy a striking case of parallel evolution of consciousness. Evidence of near human-like levels of consciousness has been dramatically observed in African grey parrots. Mammalian and avian emotional networks and cognitive microcircuitries appear to be far more homologous than previously thought. Moreover, certain species of birds have been found to exhibit neuronal sleep patterns similar to those of mammals, including REM sleep and, as was demonstrated in zebra finches, neurophysiological patterns, previously thought to require a mammalian neocortex. Magpies in particular have been shown to exhibit striking similarities to humans, great apes, dolphins and elephant studies of mirror self-recognition.

CLINICAL BEHAVIOR

There are more than 9000 bird species living on the earth showing an incredible biodiversity in terms of behavioral repertoire (ethogram). The following will give a noncomprehensive overview of the neurological and behavioral disorders of captive birds, discussing the diagnostic and therapeutic approach to these frequent disorders.

The Concept of "Abnormal Behavior"

Abnormal behavior in birds can be defined in many different ways according to the classification criteria used. A behavioral pattern can be considered to be abnormal when (1) its prevalence, frequency, intensity, or latency is statistically significantly different from the average population values for that class, sex, and age; (2) it does not have any adaptive value for the survival and fitness of the individual; (3) it causes physical damages to itself or to other birds; and (4) it does not belong to the ethogram of that avian species

The Diagnostic Approach

The Gestalt psychology (from the German *Gestalt* [i.e., shape, form]) is a particular approach to psychology that assumes we always perceive a unity of form and that the whole is greater than the sum of the parts. According to Konrad Lorenz, the psychology of Gestalt is the main source of knowledge (Wuketits and Lorenz, 1990), and this theoretical approach applied to behavioral studies explains that animal behavior cannot be encoded with an atomistic approach, analyzing its individual components. Instead, this clinical process needs a global comprehensive understanding of the living being and the behavior that animals exhibit.

A Gestalt approach to the diagnosis of an avian behavioral problem is very useful from the methodological point of view because it helps avian veterinarians investigate the whole world of the avian pet instead of focusing their attention only on the specific behavioral disorder and treating the symptom without reducing or eliminating the etiology of the disorder. The investigation process requires the establishment of a good relationship between the veterinarian, the owner, and the pet as suggested by Seibert and Sung (2013). The first behavioral examination of a pet bird should take longer than a normal clinical investigation because the practitioner needs to collect a wide range of historical information. Table 2-5 lists the main information fields that the avian veterinarian needs to achieve a correct behavioral diagnosis. Although the primary goal of the avian veterinarian is to treat the pet bird, this process cannot be completely accomplished without basic behavioral and personality profiling of the owner. The most important information that avian veterinarians should infer from owner behavior is to understand if he is telling the truth about pet management. As mentioned by several authors (Welle and Wilson, 2006; Orosz, 2008), without an open and honest rapport with the bird owner, it is very difficult to assess the behavioral situation and improve it.

Medical Etiologies of Behavioral Disorders

Many neurological diseases and medical problems can lead to behavioral changes in avian patients. The dichotomy of mind–body interaction and the question if the behavioral disorder has a medical (body) or psychological (mind) etiology makes it hard to achieve a clear diagnosis for a behavioral disorder, since frequently both systems are involved at different degrees and levels. The differential diagnosis for an avian patient usually considers a psychological basis for a behavioral problem only when medical/physical causes have been excluded (Fig. 2-14, *A* and *B*). The following is a noncomprehensive list of neurological diseases and medical problems that can cause behavioral changes in the avian patient. Additional information may be obtained from the references and suggested reading.

- Metabolic and nutritional problems include hypocalcemia, hypovitaminosis, hepatic encephalopathy, and hypoglycemia
- Infectious (viral, bacterial, fungal, or parasitic)
- Circulatory diseases (brain ischemia, ischemic strokes, and cerebral atherosclerosis)
- Epilepsy and Lafora body disease
- Toxic (heavy metals, botulism, salt, nicotine, alcohol, insecticides, other)
- Traumatic
- Neoplastic (brain tumors)
- Otitis
- Ophthalmic diseases and deficiencies

Behavioral Disorders and the Therapeutic Approach

The choice of a correct therapeutic approach requires categorization in terms of the adaptive value of the behavioral problem: if the abnormal/undesired behavior has a strong adaptive value in terms of fitness and survival for a certain avian species, the extinction of this behavior will probably be impossible and wrong from the therapeutic approach. On the other hand, other undesirable behavior with a lower fitness value could be easily eliminated by proper modification techniques such as negative or positive reinforcements, substituting other less undesirable behavior, or teaching new desired behavior (Lightfoot and Nacewicz, 2006).

Table 2-6 lists the most frequent behavioral disorders (abnormal or undesired) of companion pet birds. Many disorders belong to different categories, and frequently a bird exhibits more than one undesired behavior simultaneously. Like Table 2-5, this table is quite "parrot oriented" although many of these disorders also are widespread among non-psittacine captive birds. Therefore the list should be considered

TABLE 2-5 Main Fields of Behavioral Investigation Form "Parrot-Oriented" That Can Be Used with Minor Changes also for Other Avian Species

Main Fields	Details
Signalment	Species, age, gender
Bird source and development	Pet store, breeder, other, wild-caught, hand raised, weaning, previous owner(s)
Physical environment	Cage: size, furnishing and perching, contents, location in the house/outdoor, environmental enrichment (toys etc.), play areas, bathing, etc., falconry block, bow and screen perch, etc.
Time	Day schedule: time in cage, out of cage, daily photoperiod and sleeping arrangements Social schedule: alone or with others (birds or humans)
Social environment: owner and other human interactions	Daily time spent with the bird; type and nature of interactions, play, work, etc.; family member interactions, other humans (hosts, strangers, etc.) interactions, other animal species; sociogram* of the family including the pet bird and all the other humans and animals (Fig. 2-13)
Feeding	Type of food, feeding schedule
Bird behavior	With the owner, with other humans, with other family animals; time allocation and related behavior; restrain responses; novelty reaction; qualitative sociogram† of behavioral responses related with the previous social categories: play, aggressive, fair, anxiety, happiness
Sexual behavior	Describe the situation, assess intensity
Abnormal/undesirable behavior	Describe the situation, assess gravity
Changes	Describe any recent changes of the environment or social group
Follow-up	Tracking over time frequency, intensity, latency, and timing of the abnormal undesirable behavior

*A sociogram is a graphical representation of the structure and the social links and interindividual relations that a person or an animal has within a group, such as a family.

†A qualitative sociogram illustrates the assessment of the interactions among the social group (negative, positive, and neutral) and the related behavioral responses (play, aggressive, fear, anxiety, happiness, etc.).

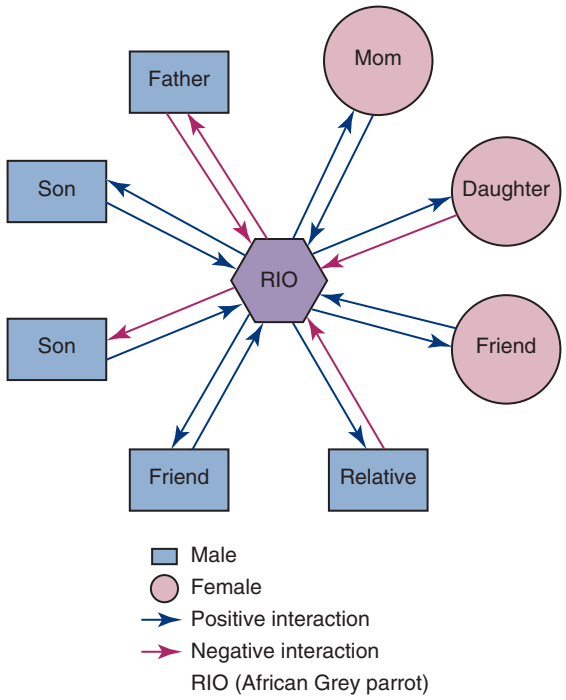


FIGURE 2-13 A clinical qualitative sociogram: positive and negative interactions among the family group, relatives, friends, and a pet African grey parrot (RIO).

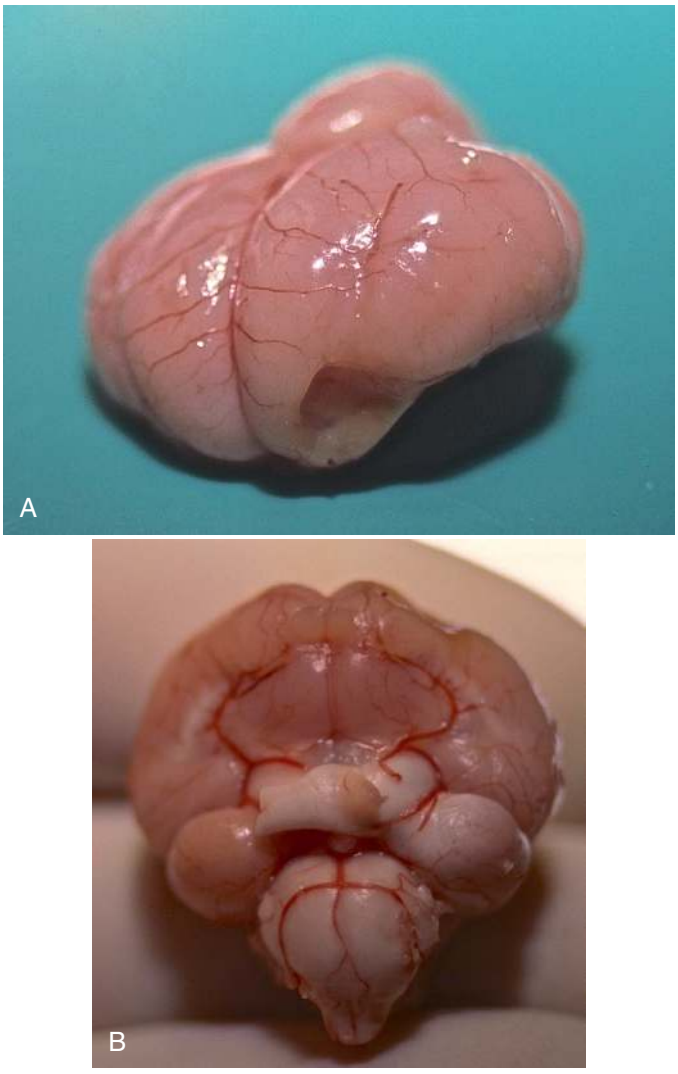


FIGURE 2-14 Postmortem findings of chronic and degenerative brain lesions of traumatic origin in a European kestrel (*Falco tinnunculus*) where the left optical nerve is broken **(A)** and the brain is severely damaged **(B)**.

nonexhaustive. For more information about avian behavioral problems and behavioral therapy/training, please refer to the suggested reading.

Behavioral Pharmacology

There are several drugs available for behavioral pharmacological treatment of avian pet birds as summarized in Table 2-7. However, before starting any pharmacological therapy please remember that you should:

- Exclude all medical causes;
- Exclude all environmental causes;
- Exclude all social factors;
- Consider carefully the behavioral differences between a solitary and a social species;
- Focus your therapeutic goal (i.e., eliminate a behavior or simply reduce its intensity and/or frequency);
- Keep in mind that pharmacological therapy is most efficient if combined with behavioral therapy/training;
- Remember that physical restraint and drug treatment are the last chance to treat behavioral problems, and they play a minor role in terms of importance in the behavioral medicine of pet birds compared with environmental and social changes.

For more information about psychotropic drugs and their use in avian medicine please refer to Carpenter (2013), Tully (1997), and the formulary section of the avian medicine books listed in the suggested readings.

AVIAN WELFARE

Animal welfare is a complex subject that can be analyzed from different perspectives with a strong emotional impact. There is no single solution to this problem, because by questioning the ways in which people benefit from animals, we also investigate the way in which people interact with the natural world. The welfare level of nonpoultry avian species varies according to the welfare level of the human society where they live. The same species can be kept as a pet bird in one country and be perceived as food in another (Sovrano *et al.*, 2013).

In the past, the "five freedom platform" (freedom from thirst and hunger; discomfort; pain, injury, and disease; freedom to express normal behavior; and freedom from fear and distress) has been implemented by a cognitive and emotional approach to animal welfare that can be summarized by the "three positive conditions" necessary to achieve a good welfare level as suggested by Webster (2008): living a natural life, being fit and healthy, and being happy. Many species of birds exhibit complex cognitive and learning abilities, and the recent advancements in the field of avian neuroanatomy and cognitive ethology are the basis for the achievement of better levels of welfare.

Avian veterinary surgeons should avoid an emotional approach to avian welfare and should not underestimate the cognitive abilities and the related mental/welfare needs of their non-psittacine patients. According to the recent advancements in the field of avian neuroanatomy

TABLE 2-6 Most Frequent Avian Behavioral Problems, Their Etiologies, and Therapeutic Approaches

Behavioral Problem	Medical and Behavioral/Psychological Etiology	Therapeutic Approach: Preventing and Correcting
Feather plucking, feather damaging behavior, pterotillomania (own feathers)	Medical: Malnutrition, wrong or poor diet, allergies, metabolic and systemic disease, environment, wrong photoperiod (no more than 12 hours of daylight), infections (psittacosis, polyomavirus–budgerigar fledging disease, circovirus– psittacine beak and feather disease), endoparasites, ectoparasites, skin- and feather-related diseases, lead and zinc toxicosis, neoplasia; a genetic base was found in orange-winged Amazon parrots (Garner *et al.*, 2006). Behavioral/psychological: stressful or boring environment, sleep deprivation, sexual deprivation or frustration, other social or stressful factors, attention seeking, separation anxiety, overcrowding, environmental changes, obsessive-compulsive disorders ([OCDs]; see below), stereotypes, feather clipping	Therapeutic intervention must treat both medical and behavioral etiologies at the same time Medical diagnosis, environmental and social improvement and enrichment, behavioral modification training, pharmacological therapy and psychotropic drugs The owner should be very careful not to reward this behavior, especially by means of an increased attention; at the opposite end he should ignore the bird and leave the room (Low, 2001).
Feather pecking (pecking and pulling the feather of other individuals)	Especially in poultry and ostriches Nutrient deprivation, redirect behavior from ground pecking or dust bathing	*Ad libitum* feeding, balanced diet, good housing and husbandry, reduced flock density
Biting/aggressive behavior/excessive territoriality	Especially parrots but also other species and/or imprinted subjects Dominance (especially hand-reared birds), territorial aggression, fear (especially wild-caught or parent-bred birds), sexual behavior or pain (Low, 2001; Davis, 1991) The meaning of the aggression can be different according to the target (owner, other birds, etc.) According to some authors, in parrots it is a learned behavior, and in fearful birds it is a result of a fear of human (Wilson, 1999)	Identify the specific etiology and work on the elimination of every reinforcement that supports this behavior Improve the owner's ability to read the parrot/bird body language, reduce dominance by means of environmental changes (perches not higher than the owner's chest, new location of the cage, etc. (Davis, 1991; Wilson, 1999; Low, 2001)
Screaming and excessive vocalization	Screaming behavior is an essential component of the ethogram of several avian species, such as parrots. Etiology: attention-seeking behavior, fear vocalization, happiness vocalization, distress, injuries, environmental or social stress, wrong emotional interaction with the owner, living in a turbulent environment, communication attempts with other birds that are far from the cage, outside or inside the house, boredom, location of the cage far from the owner's room (Davis, 1991; Lawton, 1996; Low, 2001; Wilson, 1999)	Sometimes it is very difficult to assess the limit between a normal and abnormal vocalization. Classic example of an undesirable behavior that cannot be eliminated, but only reduced in intensity and frequency or substituted with a less undesirable behavior only when inappropriate
Stereotypies	Repetitive, unvarying, and functionless behavioral patterns that are often performed by captive and domesticated animals housed in barren environments that try to cope with an inadequate environment or mental stimulation that does not satisfy normal behavioral needs: pacing, perch circles, corner flips, route trace; wire chewing, food manipulation, feather pecking, begging, destructive behavior, self-mutilation, cage overuse, feathers clipping, perseveration in appropriate behavioral responses Caged parrot stereotypes share the same mechanism stereotypies as human schizophrenia and autism and reflect a general disinhibition of the behavioral control mechanisms of the dorsal basal ganglia (Garner *et al.*, 2003; Meehan *et al.*, 2004).	Stereotypies are mutually exclusive of OCDs and should not be pharmacologically treated as such Usually it is very difficult to get rid of or reduce after they have been adopted by birds Environmental and psychological enrichment associated with proper pharmacological therapy might decrease the intensity of stereotypies
Obsessive-compulsive disorders (OCDs)	OCD is an anxiety disorder characterized by unreasonable thoughts and fears (obsessions) that lead to repetitive behavior (compulsions) Symptoms are frequently time-consuming and might cause a reduction of social interaction Several medical and psychological causes might be involved in OCDs.	Although they have different neuronal substrates from stereotypies (Garner *et al.*, 2003), it is very difficult to achieve a differential diagnosis in an avian patient because from a clinical point of view executive motor impairments might be similar

Continued

TABLE 2-6 Most Frequent Avian Behavioral Problems, Their Etiologies, and Therapeutic Approaches—cont'd

Behavioral Problem	Medical and Behavioral/Psychological Etiology	Therapeutic Approach: Preventing and Correcting
Sexual and reproductive behavior	Overbonding with humans (usually by imprinted birds), parental deprivation, sexual frustration, regurgitation on objects, chronic egg laying, infantile behavior (begging in imprinted birds)	Chicks always should be hand raised with other birds and after weaning should be kept in social groups for normal social and sexual development Chronic egg laying: allow one laying cycle and after 3 to 4 weeks remove the nest and the eggs Add pharmacological treatment when necessary (medroxyprogesterone)
Anxiety, phobias	Anxiety is an emotional response to stimuli that are potentially dangerous While a fear response usually has clear, potentially dangerous stimuli, frequently anxious birds exhibit this behavioral pattern without any well-defined threat Anxious psittacines keep their body feathers tight, their necks extended, and their eyes wide open in the absence of any threat Phobia can be defined as a strong, persistent fear of certain situations, objects, activities, or persons. As reported for depression, this disorder is strongly related to the individual coping strategy and personality	The affected bird would like to escape as far as possible from the anxiety–phobia's origin; therefore, the diagnostic key for this disorder is the identification and elimination/reduction of the causal phenomena
Depression, apathy	Apathy can be defined as an absence or suppression of emotion, feeling, concern, and attention to things generally found to be exciting or moving (indifference and impassiveness). It is one of the main symptoms of several psychological disorders, like depression, that is a persistent feeling of sadness or loss of interest in things that were once pleasurable. Very frequently sleep and alimentary abnormalities commonly accompany the ailment	It is quite difficult to make a differential diagnosis between apathy and depression because the tendency of an animal to become apathetic or depressed could be related to environmental conditions and to his personality, motivation, and individual coping strategy

TABLE 2-7 Psychotropic Drugs Used in Birds

Class	Drug	Dosage	Comments and Species
Antihistamines			
Inhibit histamine receptors	Diphenhydramine	2-4 mg/kg by mouth every 12 hours 2 mg/L drinking water	Most species, may cause sedation and mild hypnotic effects
	Hydroxyzine	2.0 mg/kg by mouth every 8 hours 30-40 mg/L drinking water	Most species, mild sedative effects
Barbiturates anticonvulsant	Phenobarbital sodium	1-7 mg/kg by mouth every 8-12 hours 50-80 mg/L drinking water	Most species, mild sedative effects but also may cause deep sedation Idiopathic epilepsy, self-mutilation, feather picking Start with a low dosage range and increase for refractory seizures
Benzodiazepines			
Sedative, anxiolytic	Diazepam	0.25-0.5 mg/kg intramuscularly Intravenously every 24 hours × 2-3 days 10-20 mg/L drinking water	Used for stress-associated feather-picking, facilitates acceptance of Elizabethan collar, control of seizures
	Lorazepam	0.1 mg/kg by mouth every 12 hours	Macaw aggressive behavior; feather picking

TABLE 2-7 Psychotropic Drugs Used in Birds—cont'd

Class	Drug	Dosage	Comments and Species
Butyrophenones			
Dopamine antagonist tranquilizer	Haloperidol	0.1-0.4 mg/kg by mouth every 24 hours 6.4 mg/L drinking water × 7 months	Self-mutilation, aggression, feather picking May cause anorexia or depression; death reported in macaws
Opioid antagonist	Naltrexone	1.5 mg/kg by mouth Every 8-12 hours × 1-18 months	Most species, self-injured behavior, feather picking Contraindicated in birds with liver disease Doses may need to increase up to 6× to obtain effects
	Naloxone	2 mg/kg intravenously	May be used to determine the response of stereotypic behavior to antagonist therapy, reduction of the behavior within 20 minutes
Progestins	Megestrol acetate	2.5 mg/kg by mouth every 24 hours × 7 days then 1-2× per week 10-20 mg/L drinking water × 7-10 days, then 1-2× per week	Most species, feather picking behavior, calming effects but many severe side effects (diabetic-like) Sexual behavior problem, feather picking
Selective serotonin reuptake inhibitor	Fluoxetine	0.4 mg/kg by mouth every 24 hours 2-3 mg/kg by mouth every 12-24 hours	Antidepressant, feather picking, may cause sedation
	Paroxetine	1-2 mg kg by mouth every 24 hours	Macaws, ibis, feather picking, self-mutilation, requires long-term therapies
Tricyclic antidepressant inhibits serotonin reuptake, antihistamine Alleviates anxiety and depression	Amitriptyline	1-5 mg/kg by mouth every 12-24 hours	Most species, allergic feather picking, OCD, phobias Psittacine minimum 30 days for assessing effects
	Clomipramine	0.5-2.0 mg/kg by mouth every 12-24 hours	May cause regurgitation, drowsiness, death in birds with preexisting arrhythmias, dose has to be adjusted after 2-3 weeks
	Doxepin Nortriptyline	0.5-1.0 mg by mouth every 12 hours 16 mg/L drinking water	May cause sedation, dose may be increased at 14-day intervals Feather picking, decrease dose if hyperactivity develops

and cognitive ethology, a good level of welfare should be guaranteed to all avian patients with no differences between avian groups (Zucca, 2007). The emerging picture of the avian mind has a strong ethical implication on animal welfare, and owners should be informed of the "mental lives" of their avian pets because the health of the animal mind has the same importance as the animal's physical health.

ACKNOWLEDGMENTS

I would like to acknowledge Dr. Erik Jarvis of Duke University, North Carolina, and Zina Deretsky, National Science Foundation (Fig. 2-2); Professor Giorgio Vallortigara of the University of Trento, Italy (Fig. 2-7); Professor Nicola S. Clayton of the University of Cambridge, UK (Fig. 2-8); Professor Helmut Prior of the Goethe University Frankfurt, Germany; Professor Ariane Schwarz, Professor Onur Güntürkün of the Ruhr-University, Germany, *PLoS Biology* 6(8):e202. doi:10.1371/journal.pbio.0060202 (Fig. 2-9, *A* and *B*), the Alex Foundation/Professor Irene Pepperberg of the University of Harvard, Boston, MA (Fig. 2-10) and Professor Gavin Hunt of the University of Auckland, New Zealand (Fig. 2-12), for supplying and providing permission to reproduce the above listed images and Dr. Sara Tarabocchia for commenting on this manuscript.

REFERENCES

Auersperg AMI, von Bayern AMP, Gajdon GK, et al: Flexibility in problem solving and tool use of kea and New Caledonian crows in a multi access box paradigm, *PLoS ONE* 6(6):e20231, 2011.

Balcombe J: *Pleasurable kingdom: animals and the nature of feeling good*, London, 2006, Macmillan.

Balcombe J: Animal pleasure and its moral significance, *Appl Anim Behav Sci* 118:208–216, 2009.

Bang BG, Cobb S: The size of the olfactory bulb in 108 species of bird, *Auk* 85:55–61, 1968.

Boissy A, Manteuffel G, Jensen MB, et al: Assessment of positive emotions in animals to improve their welfare, *Physiol Behav* 92(3):375–397, 2007.

Bonadonna F, Sanz-Aguilar A: Kin recognition and inbreeding avoidance in wild birds: the first evidence for individual kin-related odour recognition, *Anim Behav* 84(3):509–513, 2012.

Brinkløv S, Fenton MB, Ratcliffe JM: Echolocation in oilbirds and swiftlets, *Front Physiol* 4:123, 2013.

Cambridge (The) Declaration of Consciousness: Francis Crick Memorial Conference: *Consciousness in human and non-human animals*. Available at http://fcmconference.org/img/CambridgeDeclarationOnConsciousness.pdf, 2012.

Carpenter JW: *Exotic animal formulary*, ed 4, St Louis, 2013, Saunders, Elsevier.

Clayton NS, Dickinson A: Episodic-like memory during cache recovery by scrub jays, *Nature* 395:272–274, 1998.

Davis CS: Parrot psychology and behavior problems, *Vet Clin N Am Small Anim Prac* 21(6):1281–1289, 1991.

Dharmaretnam M, Andrew RJ: Age- and stimulus-specific use of right and left eye by the domestic chick, *Anim Behav* 48:1395–1406, 1994.

Emery NJ: Are corvids "feathered apes"? Cognitive evolution in crows, jays, rooks and jackdaws. In Watanabe S, editor: *Comparative analysis of minds*, Tokyo, Japan, 2004, Keio University Press.

Emery NJ: Cognitive ornithology: the evolution of avian intelligence, *Philos Trans R Soc Lond B Biol Sci* 361(1465):23–43, 2006.

Emery NJ, Clayton NS: The mentality of crows: convergent evolution of intelligence in corvids and apes, *Science* 306(5703):1903–1907, 2004.

Garner JP, Meehan CL, Famula TR, et al: Genetic, environmental, and neighbor effects on the severity of stereotypies and feather picking in orange-winged Amazon parrots (*Amazona amazonica*): an epidemiological study, *Appl Anim Behav Sci* 96(1):153–168, 2006.

Garner JP, Meehan CL, Mench JA: Stereotypies in caged parrots, schizophrenia and autism: evidence for a common mechanism, *Behav Brain Res* 145(1–2):125–134, 2003.

Ghirlanda S, Vallortigara G: The evolution of brain lateralization: a game-theoretical analysis of population structure, *Proc Biol Sci* 271(1541):853–857, 2004.
Goodall J: *Through a window: 30 years observing the Gombe chimpanzees*, UK, 1991. Penguin edition.
Gottschaldt KM: Structure and function of avian somatosensory receptors. In King AS, McLelland J, editors: *Form and function in birds, vol 3*, London, UK, 1985, Academic Press.
Güntürkün O: Avian and mammalian "prefrontal cortices": limited degrees of freedom in the evolution of the neural mechanisms of goal-state maintenance, *Brain Res Bull* 66(4–6):311–316, 2005.
Hart NS, Partridge JC, Cuthill IC: Retinal asymmetry in birds, *Curr Biol* 10(2):115–117, 2000.
Healy K, McNally L, Ruxton GD, et al: Metabolic rate and body size are linked with perception of temporal information, *Anim Behav* 86(4):685–696, 2013.
Hunt GR: Manufacture and use of hook-tools by New Caledonian crows, *Nature* 379:249–251, 1996.
Jarvis ED, Güntürkün O, Bruce L, et al: Avian brains and a new understanding of vertebrate brain evolution, *Nat Rev Neurosci* 6:151–159, 2005.
Kotrschal K: *Im Egoismus vereint? Tiere und menschentiere–das neue weltbild der verhaltensforschung*, Munchen, Germany, 1995, Piper GmbH & Co.
Kreithen ML, Keeton WT: Detection of changes in atmospheric pressure by the homing pigeon, *Columba livia*, *J Comp Physiol* 89(1):73–82, 1974.
Lawton MPC: Behavioral problems. In Beynon PH, Forbes NA, Lawton MPC, editors: *Manual of psittacine birds*, Cheltenham, UK, 1996, British Small Animal Veterinary Association.
Lightfoot T, Nacewicz CL: Psittacine behavior. In Bradley Bays T, Lightfoot T, Mayer J, editors: *Exotic pet behavior*, St Louis, 2006, Elsevier.
Low R: *Papageien sind einfach anders—eigenheiten verstehen und verhaltensprobleme*, Stuttgart, Germany, 2001, *Lösen*, Eugen Ulmer Verlag.
Magat M, Brown C: Laterality enhances cognition in Australian parrots, *Proc Biol Sci* 276(1676):4155–4162, 2009.
Marler P: Social cognition: are primates smarter than birds? In Nolan V, Ketterson ED, editors: *Current ornithology*, New York, 1996, Plenum Press.
Marzluff JM, Miyaoka R, Minoshima S, et al: Brain imaging reveals neuronal circuitry underlying the crow "prefrontal cortices" left and right perceptual worlds, *Brain Lang* 73(2):189–219, 2012.
Meehan CL, Garner JP, Mench JA: Environmental enrichment and development of cage stereotypy in orange-winged Amazon parrots (*Amazona amazonica*), *Dev Psychobiol* 44(4):209–218, 2004.
Orosz SE: Diagnostic workup of suspected behavioral problems. In Luescher AE, editor: *Manual of parrot behavior*, New York, 2008, Blackwell Publishing.
Pepperberg IM: Evidence for numerical competence in an African grey parrot (*Psittacus erithacus*), *J Comp Psychol* 108:36–44, 1994.
Pepperberg IM: *The Alex studies*, Cambridge, MA, 1999, Harvard University Press.
Prior H, Schwarz A: Güntürkün O: Mirror-induced behavior in the magpie (*Pica pica*): evidence of self-recognition, *PLoS Biol* 6(8):e202, 2008.
Rogers LJ: *Minds of their own*, Sydney, Australia, 1997, Allen & Unwin.
Rogers LJ, Andrew RJ: *Comparative vertebrate lateralization*, New York, 2002, Cambridge University Press; re-issued in 2008.
Rogers LJ, Zucca P, Vallortigara G: Advantages of having a lateralized brain, *Proc Biol Sci* 271(Suppl 6):S420–S422, 2004.
Seibert LM, Sung W: Psittacines. In Tynes V, editor: *Behavior of exotic pets*, Hoboken, NJ, 2013, John Wiley & Sons.
Sovrano VA, Zucca P, Regolin L: *Il comportamento degli animali. Evoluzione, cognizione e benessere*, Roma, 2013, Manuali Universitari Scienze.
Steiger SS, Fidler AE, Valcu M, et al: Avian olfactory receptor gene repertoires: evidence for a well-developed sense of smell in birds? *Proc Biol Sci* 275(1649):2309–2317, 2008.
Tully TN: Formulary. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, 1997, WB Saunders.
Uexküll J Von: *Streifzüge durch umwelten von tieren and menschen*, Hamburg, Germany, 1934, Taschenbuch Verlang.
Vallortigara G: Comparative neuropsychology of the dual brain: a stroll through animals' left and right perceptual worlds, *Brain Lang* 73(2): 89–219, 2000.
Vallortigara G, Rogers LJ: Survival with an asymmetrical brain: advantages and disadvantages of cerebral lateralization, *Behav Brain Sci* 28(4):575–589, discussion 589–633, 2005.
Vallortigara G, Rogers LJ, Bisazza A: Possible evolutionary origins of cognitive brain lateralization, *Brain Res Rev* 30(2):64–175, 1999.
Watanabe S: Pigeons can discriminate "good" and "bad" paintings by children, *Anim Cogn* 13(1):75–85, 2010.
Watanabe S, Wakita M, Sakamoto J: Discrimination of Monet and Picasso in pigeons, *J Exp Anal Behav* 63:165–174, 1995.
Webster J: *Animal welfare: limping towards eden*, Hoboken, NJ, 2008, John Wiley and Sons.
Welle KR, Wilson L: Clinical evaluation of psittacine behavioral disorders. In Luescher AU, editor: *Manual of parrot behavior*, Oxford, UK, 2006, Blackwell Publishing.
Wilson L: *Managing Parrot behavior: behavior problems and the future of companion parrots*, Proceedings for the annual conference of the Mid-Atlantic States Association of Avian Veterinarians, 1999.
Wittling W: The right hemisphere and the human stress response, *Acta Physiol Scand Suppl* 640:55–59, 1997.
Wittling W: Brain asymmetry in the control of stress responses. In Boller F, Grafman J, editors: *Handbook of neuropsychology, vol 5, Emotional behavior and its disorders*, ed 2, Oxford, UK, 2001, Elsevier Health Sciences.
Wittling W, Schweiger E: Neuroendocrine brain asymmetry and physical complaints, *Neuropsychologia* 31:591–608, 1993.
Wuketits FM, Lorenz K: *Leben und werk eines grossen naturforschers*, München, Germany, 1990, Elsevier Health Sciences.
Zucca P: Anatomy: brain and behavior, peculiarities of the avian brain. In Cooper JE, editor: *Birds of prey: health and disease*, ed 3, Oxford, UK, 2002, Blackwell Publishing.
Zucca P: *Mind of the avian patient: cognition and welfare*, Proceedings of the 9th European Conference of the Association of Avian Veterinarians, Zurich, Switzerland, 2007.
Zucca P, Cerri F, Carluccio A, et al: Space availability influence laterality in donkeys (*Equus asinus*), *Behav Process* 88(1):63–66, 2011.
Zucca P, Sovrano VA: Animal lateralization and social recognition: quails use their left visual hemifield when approaching a companion and their right visual hemifield when approaching a stranger, *Cortex* 44:13–20, 2008.
Zucca P, Sovrano V, Bugnyar T: *Does preferential visual hemifield use allow inferring the emotional state of birds? The examples of quails and ravens*, Proceedings of the 10th European Conference of the Association of Avian Veterinarians, Antwerp, Belgium, 2009.

FURTHER READING

Bugnyar T, Kotrschal K: Observational learning and the raiding of food caches in ravens, *Corvus corax*: is it "tactical" deception? *Anim Behav* 64:185–195, 2002.
Doneley B: *Avian medicine and surgery in practice: companion and aviary birds*, Boca Raton, FL, 2010, CRC Press.
Griffin D: *Animal minds: beyond cognition to consciousness*, Chicago, IL, 2000, The University of Chicago Press.
Harrison GJ: Perspective on parrot behavior. In Ritchie B, Harrison GJ, editors: *Avian medicine: principles and application*, Greenacre, FL, 1994, Zoological Education Network.
Heinrich B: *Mind of the raven*, New York, 1999, Harper Collins Publishers.
Hugues HC: *Sensory exotica: a world beyond human experience*, London, UK, 2001, MIT Press.
Jarvis ED, Consortium ABN: Avian brains and a new understanding of vertebrate brain evolution, *Nat Rev Neurosci* 6:151–159, 2005.
Martin P, Bateson P: *Measuring behaviour: an introductory guide*, Cambridge, UK, 2007, Cambridge University Press.
Scheiber IBR, Weiß BM, Hemetsberger J, et al: *The social life of greylag geese: patterns, mechanisms and evolutionary function in an avian model system*, Cambridge, UK, 2013, Cambridge University Press.
Tully TN, Dorrestein GM, Jones A: *Handbook of avian medicine*, ed 2, Philadelphia, 2009, WB Saunders.
Wynne CDL: *Animal cognition, the mental lives of animals*, New York, 2001, Palgrave 200.
Wynne CDL: *Do animals think?* New Jersey, 2004, Princeton University Press.

3

Nutrition and Nutritional Management

Nature always springs to the surface and manages to show what she is. It is vain to stop or try to drive her back. She breaks through every obstacle, pushes forward, and at last makes herself a way.

Nicolas Boileau-Despréaux (1636-1711)

BASIC PRINCIPLES OF OPTIMAL NUTRITION

Janine Perlman

As is true of all animals, birds have evolved to eat and require particular types of foods. Virtually every aspect of birds' anatomy, physiology, and behavior helps them crave, seek, identify, procure, consume, digest, and assimilate their highly specific natural diet (Klasing, 1998).

As determined by that diet, birds are categorized into feeding guilds. Guild members share characteristics including gastrointestinal tract structure and function (Stevens and Hume, 2004), and diet-related physiology and biochemistry (e.g., Sabat *et al.*, 1998; Myers and Klasing, 1999). These evolved adaptations tightly constrain the types of foods birds can digest and assimilate to support optimal health.

Feeding guilds can be pictured as three trees: herbivores eat plant foods, faunivores eat animals, and omnivores eat from both kingdoms. These three "trunks" branch into generalist consumers that eat broadly, or specialist consumers that focus on particular kinds of foods. Specialists and generalists form a continuum, and between the two extremes, birds may be difficult to categorize.

Herbivorous specialists include frugivores (fruit eaters; some parrots and passerines), granivores (seed eaters; many columbids and some parrots and passerines), graminivores (grass eaters, may also be used for grain-seed eaters; many geese), nectarivores (sunbirds, lories/lorikeets, and hummingbirds), and folivores (browsing leaf eaters; plantcutters, ostriches, hoatzin, kakapo, and, seasonally, many grouse). Most herbivorous specialists eat mainly one plant part from a wide array of species. Many parrots are herbivorous generalists and may eat fruit, seeds, young leaves, buds, flowers and, in some cases, nectar.

Few birds are strictly herbivorous because plant-sourced foods do not contain sufficient levels of certain essential nutrients. To meet their needs for maintenance and demanding physiological states of growth, egg production, illness, injury, or molt, nearly all taxa consume some animal-sourced foods.

Faunivorous birds may also be specialists, including insectivores (most passerines), avivores (some falcons and accipters), mammalivores (some hawks and owls), piscivores (marine birds and osprey), and scavengers (vultures), or generalists (some hawks and marsh birds). Some faunivores specialize quite narrowly and eat mainly prey such as earthworms, mollusks, or caterpillars.

Feeding-guild membership is, in part, a function of life stage. For example, the young of nectarivorous hummingbirds, of many herbivorous ducks, and—regardless of adult diet—of virtually all passerines, require diets composed of invertebrates.

Food provides water, energy, vitamins, minerals, and essential fatty acids and amino acids. Birds experience dynamic changes in physiological state over a range of timescales. These changes compel equally dynamic adjustments in nutrient intake and thus in the specific foods a bird needs and chooses to eat (Murphy, 1994).

Birds have appetites for, and can precisely discern relative levels of, nutrients including energy, fat, protein, individual amino acids, thiamin, pyridoxine, ascorbic acid, sodium, phosphorus, calcium, zinc (compiled in Fernandez, 2008), and carotenoids (Senar et al., 2010). Additional specific appetites doubtless await discovery.

Recent studies also show that, contrary to earlier belief, birds possess high and complex taste acuity, with flavor responses that are specific to taxon and evolved diet (Roura *et al.*, 2013; Baldwin *et al.*, 2014).

Key to optimal captive feeding is the fact that, given free choice, birds use specific appetites to exquisitely regulate intake and meet their needs at any given time (e.g., Brown and Downs, 2003; Schaefer *et al.*, 2003; Wilkinson *et al.*, 2014).

Basic principles of nutritional biochemistry apply to all animals. Since the best-studied species is our own, it is instructive to examine the human literature where relevant. Although humans have eaten meat in recent evolutionary time, we evolved from hominids that were largely herbivorous. Human studies reflect this history; beyond the few dozen familiar essential nutrients, thousands of compounds, most of them phytochemicals (plant-sourced chemicals) (Scalbert *et al.*, 2011; Tomás-Barberón and Andrés-Lacueva, 2012; Benzie and Choi, 2014), are required for maximum wellness (Jensen *et al.*, 2014).

As additional essential nutrients continue to be identified, it has become clear that their salutary effects are negated when they are purified (Schreiner and Huyskens-Keil, 2006). *To support optimal health, nutrients must be consumed in their native context (i.e., in the physicochemical matrix that is unique to each whole, natural food)* (Jacobs and Tapsell, 2013; Liu, 2013).

These findings can be safely assumed to apply to other animals including birds whose evolved diets include significant amounts of plant-based foods.

FEEDING BIRDS IN CAPTIVITY

Translating the principles of optimal nutrition into captive feeding practice is not difficult, and the rewards are great. An informed naturalistic diet gives pleasure to bird and caregiver, strengthens the mutual bond, enhances the bird's health, and has the potential to add considerably to a captive bird's well-being (Watters, 2014).

Altricial nestlings have the most stringent nutritional demands and most vividly illuminate the need to provide birds their evolved diet. Nidicolous nestlings display the highest fractional growth rates of any

extant vertebrate (Ricklefs, 1973). Their growth is limited only by their ability to digest and absorb food (Lepczyk *et al.*, 1998). Thus their diet must superbly match their requirements for tissue accretion.

Almost all passeriform, apodiform, and other "near-passerine" nestlings are fed insects and spiders by their parents. These nestlings require the very high level and quality of protein offered by invertebrates. Psittacine hand-rearing formulations contain less than half the requisite protein, and it is also plant-sourced and of poorer quality than that found in insects. While current labeling encourages the use of these products for all taxa, they result in severe malnutrition in non-psittacines (MacLeod and Perlman, 2000; Fig. 3-1, *A*).

Studies of captive-reared nestling swifts and songbirds have also compared a diet comprising feeder insects versus either an "insect substitute" formulation based on companion carnivore kibble high in animal-sourced protein, or feeder rodents. The two latter diets contain the same levels of protein, fat, and carbohydrates as insects. The results are clear: nestlings fed insects (Fig. 3-1 *B, C*) exhibit very high survival and release rates and normal growth, development, and plumage compared with wild-reared conspecifics. Birds fed the "isonutritional" but unnatural diets have decreased survival and release rates and poor growth, development, and plumage (Fusté *et al.*, 2013, Birch and Perlman, unpublished data).

Thus unnatural diets, even with the same levels of conventional nutrients as the natural one, are inadequate to sustain health, or—in birds with the highest nutritional demands—life. These findings corroborate many studies in humans, and affirm the title of an influential review, "Food, not nutrients, is the fundamental unit in nutrition" (Jacobs and Tapsell, 2007).

Compared with optimally fed nestlings, those that survive poor diets have impaired immunity (Birkhead *et al.*, 1999; Hoi-Leitner *et al.*, 2001) and are considerably less likely to live to adulthood (Cichon and Dubiec, 2005) and to reproduce normally if they do survive (Blount *et al.*, 2006). Rehabilitation centers that feed insectivorous nestlings anything other than their evolved diet would seem to be wasting human resources and avian lives.

Formulations are necessary for two categories of nestlings: those whose parents regurgitate seeds (most parrots and fringillids) and those whose parents produce crop milk (MacLeod and Perlman, 2002; Dierenfeld *et al.*, 2009). For all such species, much remains to be learned about the nature of the parents' secretions before these formulations can be optimized.

While whole foods natural to the species are essential, diets must be informed and complete. Before the middle of the last century, uninformed attempts to provide naturalistic diets to captive exotic and wild birds were the rule. Incomplete diets such as those composed entirely of seed or meat were deficient in calcium and vitamins A and D, and often caused disease and early death.

After essential nutrients were discovered and chemically characterized, formulated "complete" diets became the norm for feeding livestock, including poultry. These products were, and continue to be, composed largely of grain and soy. They are made "complete" with additions of purified micronutrients.

Formulations for pets and exotic and wild captives soon followed, based on the same types of ingredients. The motto in animal science departments became "nutrients, not food." Although outdated, that approach still prevails in many settings. Indeed, one major manufacturer sells a single formulated diet that is labeled for use in zoo animals ranging from herbivorous hindgut fermenters to carnivores.

Among captive birds, formulations are often used for adult psittaciforms, whose natural diets may consist of fruit, nuts/seeds,

FIGURE 3-1 **(A),** Tree swallow (*Tachycineta bicolor*) fledgling fed a psittacine hand-rearing formulation during the latter 2 weeks of the nestling period. Feathers are poorly keratinized and disheveled because of structural defects. Swallows are aerial insectivores and must have excellent flying ability when they fledge. (Courtesy Veronica Bowers.) **(B),** Barn swallow (*Hirundo rustica*) nestling consuming mealworms. (Courtesy Veronica Bowers.) **(C),** Tree swallow and cliff swallow (*Petrochelidon pyrrhonota*) juveniles hand reared on an insect diet. (Courtesy Veronica Bowers.)

invertebrates, flowers, tender leaves, and in some cases nectar. Not surprisingly, formulations are far from optimal for these birds. Soy is poorly digested by birds (Parsons *et al.*, 1981; Elliston and Perlman, 2002; Choct *et al.*, 2010), and grain is a nutrient-poor dietary base for species that did not evolve to eat it (Cordain, 1999; Dewey, 2013). Pelleted diets for parrots have startlingly low bioavailability; only 50% of their protein is absorbed (Kalmar *et al.*, 2007) compared with >85% in naturalistic foods (Sales *et al.*, 2004).

Formulated products also have the major disadvantage of forcing birds to consume a monotonous diet that is inadequate or entirely lacking in most of the essential phytonutrients described above. And because birds' requirements are highly dynamic, all nutrient levels in formulations are inevitably nonoptimal, over significant periods of birds' lives, for all species. For certain species, nutrients including calcium and vitamin A are typically present in toxic excess (McDonald, 2003; de Matos, 2008). When birds cannot choose what they eat, they cannot avoid such toxicities, which also arise as a result of too-frequent quality control problems in formulations (Frederick *et al.*, 2003; http://www.fda.gov/animalVeterinary/safetyhealth/recallswithdrawals/default.htm).

A healthful and complete naturalistic diet for species of any taxon can be knowledgeably created by examining the primary literature (e.g., Witmer, 1996; Gilbert *et al.*, 2003; Brightsmith *et al.*, 2010). Collections of species accounts, notably *Birds of the World* (Oxford University Press; http://global.oup.com/academic/content/series/b/bird-families-of-the-world-bfw/?cc=&lang=en) and Birds of North America (American Ornithologists' Union; http://bna.birds.cornell.edu/bna/) are invaluable (Fig. 3-2, *A-D*).

FIGURE 3-2 **(A),** A selection of cultivated seeds, fruits, and vegetables for finches. (Courtesy Miriam Moyer.) **(B),** Cultivated foods *(bottom right)* are offered in a naturalistic environment that offers native wild foods consumed by American goldfinches (*Spinus tristis*). (Courtesy Miriam Moyer.) **(C),** Domestic ducks should be fed a variety of grains, lettuce, invertebrates, and free-choice oystershell for grit and calcium. High-protein fish-based koi pellets allow ducks to regulate their protein intake if invertebrates are not available *ad lib.* **(D),** Domestic ducks consume lettuce and invertebrates with great enthusiasm.

FIGURE 3-3 Cedar waxwing (*Bombycilla cedrorum*) with a fruit-based diet of cultivated and natural foods. Single birds of gregarious species also benefit from the presence of a mirror. (Courtesy Jayne Neville.)

Foraging enrichment is a crucial aspect of optimal feeding (Péron and Grosset, 2014). Birds actively seek food during much of their waking time, encountering continual challenges to their mental and physical agility. Foraging enrichment is more interesting to parrots, and more effective at preventing cage stereotypies, than other forms of enrichment (Meehan *et al.*, 2004). Possibilities for implementing this important captivity enhancement are limited only by the imagination of the caregiver (Fig. 3-3).

Although it cannot be relied on for vitamin D (see the following), natural full-spectrum light is important in optimal husbandry for reasons that include the full visibility and appeal of food (reviewed in Maddocks *et al.*, 2001; Schaefer *et al.*, 2008).

Studies that can inform best-feeding practices are emerging at an accelerating rate from an array of seemingly disparate fields. Even as knowledge advances, the fundamental principles of optimal feeding will remain unchanged because they are based on millions of years of evolution that have resulted in each bird's specific, dynamic requirements.

NUTRITIONAL MANAGEMENT

Captive diets for largely herbivorous psittaciforms are less often optimized than for birds of many other feeding guilds. In part this may result from the fact that both Eastern (Hasebe and Franklin, 2004; Perrin, 2009) and Western hemisphere parrots (Ragusa-Netto and Fecchio, 2006; Vaughan *et al.*, 2006) eat more widely between, and within, guilds than is often assumed (Péron and Grosset, 2014).

Seven categories of foods that parrots should be offered daily are presented in Box 3-1. Foods within each category should be rotated regularly. They must be presented as separate entities, and not as "cakes" or other mixes (Fig. 3-4). If the bird cannot choose foods individually, the diet becomes a formulation.

In addition to a selection of all the listed food types, nectarivores should have a 25% (Karasov and Cork, 1994) sucrose-in-water nectar replacer, also presented separately.

The proportion of foods needed and consumed from the previous categories is specific to species, physiological status, and season. Every effort should be made to provide a wide variety of foods belonging to the categories from which the species mainly eats, while also enticing the bird to sample rotating items from other categories. A sound feeding program is based on the biological reality that preferences reflect needs: preferences vary because needs vary over time.

In the rare case that a bird unsustainably restricts its food choices, the favored item(s) may be *carefully* limited as the bird is induced to try additional foods. The new foods and their procurement should be made as attractive as possible. Possibilities include the use of treat toys,

BOX 3-1 Daily Foods for Parrots

Fruits

Virtually all fruits safe for humans are safe for parrots. They should be fresh or frozen/thawed, with dried fruits offered less often. A list of possibilities may be found at http://en.wikipedia.org/wiki/List_of_culinary_fruits.

If seeds are included, be sure they are nontoxic (Bolarinwa *et al.*, 2014).

Do not feed avocado because of possible cardiotoxicity.

Non-Starchy Vegetables

Dark-green leafy types should be emphasized. Nontoxic wild species such as dandelion (*Taraxacum* spp.) and young leaves of wild lettuce (*Lactuca* spp.) add interest and nutrients.

Non-starchy vegetables are a subset of the list found at https://sites.google.com/site/worldvegetables/Home/vegetables.

Avoid bulbs (*Allium* spp.).

If significant amounts of Brassicaceae (mustard family; including broccoli, kale, cabbage, etc.) are consumed, they should be cooked to inactivate goitrogens.

Legumes

Offer only human-edible legumes, such as those listed at http://www.cropsreview.com/grain-legumes.html. Except for peas (*Pisum sativum*), most should be cooked to inactivate antinutrients and toxins.

Oily Seeds and Tree Nuts

Most may be offered either raw or roasted.

Oily seeds include flax, sunflower, safflower, and pumpkin. A list of nuts may be found at http://en.wikipedia.org/wiki/List_of_culinary_nuts.

Animal-Sourced Foods

Feeder insects such as mealworms (*T. molitor*), crickets (*Acheta domesticus*), cockroaches (various), and silkworm (*Bombyx mori*) larvae/pupae may be fed live or roasted.

Agricultural and seafood products should be thoroughly cooked. Examples include egg with shell, fish with bones, crustaceans with shell, and chicken with bone.

Premium feline kibble should be available *ad lib.*

Plain yogurt is safe. Cheese (unprocessed) is safe in moderation. Its high salt content may make it very attractive in the context of an otherwise low-sodium diet; a complete diet ensures that other sources of sodium are available.

Starchy Vegetables

Root vegetables are listed at http://en.wikipedia.org/wiki/List_of_root_vegetables.

In the absence of reliable information to the contrary, they should be cooked.

Avoid bulbs (*Allium* spp. and others).

Whole Grains

May be offered raw, dried, or cooked. A list can be found at http://www.cropsreview.com/cereal-crops.html.

Note that common terminology for edible plant food categories may not correspond to botanical classifications.

FIGURE 3-4 Examples of food types that should be offered to medium-sized parrots such as African greys (*Psittacus erithacus*) and *Cacatua* spp. The proportions of food types consumed will vary with species, individual, and physiological status. A wide variety of items should be emphasized in categories such as fruit and nuts, from which consumption is high.

FIGURE 3-5 Sturnids (mynas and starlings) eat mostly invertebrates, with a minor fraction of their diet comprised of fruit.

puzzle boxes, and other interesting and challenging presentations, and having the bird watch while the novel foods are enthusiastically consumed by the caregiver or avian companions.

The principles described for feeding parrots are applicable to all taxa. Raptors should be provided a variety of whole prey matching the natural diet. Granivores, including budgerigars, cockatiels, and most columbids and fringillids, should receive a wide array of grain and oil seeds, and should be offered at least one (rotating) item from each of the other previous categories daily. For omnivore-faunivores such as Sturnidae (mynas and starlings), a variety of animal-sourced foods and foods from other main dietary categories (e.g., fruit) should be available at all times, with items from additional categories rotated (Fig. 3-5). Feeder vertebrates and invertebrates should be fed their own high-quality evolved diets.

Taxon-specific needs must be understood and accommodated; for example, sturnids, muscicapids, and mimids are sucrose intolerant (Malcarney *et al.*, 1994), and fruits should be chosen accordingly. Detailed information on food composition may be found at the U.S. Department of Agriculture website, where sucrose levels in fruits are shown at http://tinyurl.com/krysngn. Some Passeriformes have lost the ability to synthesize ascorbate (Drouin *et al.*, 2011). Vitamin C should be added to captive diets for these species.

Diet-related iron storage diseases are surely uncommon in the wild, and in captive birds they appear to result from formulated feeds (Sheppard and Dierenfeld, 2002; Pereira *et al.*, 2010). Knowledgeably created naturalistic diets seem to be a preventive for these diseases.

Two micronutrients must be supplemented for captive self-feeding birds. *Calcium* carbonate must be provided *ad lib* in one or more forms and sizes recognized and ingestible by the species. Possibilities include cuttlefish bone and shells of poultry eggs, oysters, snails, and crustaceans. *Vitamin D* sufficiency can rarely be assured regardless of UV light exposure or its source; it must be provided in the diet. However, attempts to prevent deficiency can result in over-supplementation.

BOX 3-2 Fat-Soluble Vitamin Supplementation

5 cc omega-3 marine (fish body, not liver) oil
10,000 IU Vitamin D_3
10,000 IU Vitamin A
400 IU Vitamin E

Mix thoroughly, minimizing oxygenation; store refrigerated, in the absence of air, for up to 4 weeks.

Each vitamin can be bought as oil-based solution in gelatin capsules from manufacturers such as NOW at http://www.nowfoods.com/.

Feed 0.05 cc (1 drop) per 25 Kcal food.

Fortunately, *vitamins D and A given together in approximately equal amounts* (IU), as shown in Box 3-2, may be safely and effectively fed, even in very high amounts that would be toxic for either alone (Metz *et al.*, 1985; Perlman, 2011). The methods of supplying calcium and vitamins A and D shown in Box 3-2 reliably ensure healthful levels of these historically problematic micronutrients. Vitamin and mineral products labeled for bird/animal use are often poorly formulated and may suffer from inadequate quality control; individual supplements for humans are considerably safer.

Raw fish and mollusks, whether fresh or frozen, should be supplemented with 35 mg *thiamin* (Geraci, 1972) and 100 IU *vitamin E* (Engelhardt and Geraci, 1978) per kilogram food (as fed). Routine administration of *B-complex vitamins that include pyridoxine (B6)* is inadvisable because of a significant risk of toxicity (Samour, 2013; see Chapter 10). In cases of illness or starvation, hypothiaminosis is the most likely deficiency; thiamin may be safely orally supplemented, either alone or with other B vitamins present at appropriate levels (see Chapter 8).

Dirt/clay is consumed by most taxa and appears to provide numerous and various benefits (Gilardi *et al.*, 1999). Contaminant-free soil should be included in the habitat of captive birds.

Veterinarians are often asked to advise on the care of orphaned wild passerine and "near-passerine" nestlings. Unlike parrots and raptors,

FIGURE 3-6 (A-D), A brood of Bullock's orioles (*Icterus bullockii*) presented for rehabilitation soon after hatching, and hand reared on an insect diet. (Courtesy Veronica Bowers.)

these insectivores require very frequent feeding of insects such as mealworms and crickets, several times per hour, gradually decreasing to every 45 minutes by fledging (Fig. 3-6, *A-D*). Particular attention must be paid to establishing and maintaining hydration.

Insects must be supplemented with *2% elemental calcium* on a dry matter basis. A practical guideline is 5 mg elemental calcium per g bird per day. Allometric supplementation—moderately more calcium than this for smaller, more rapidly growing birds and moderately less for larger birds whose growth has slowed—better tailors intake to need.

Softgel capsules of oil-based $CaCO_3$ paste (human "absorbable calcium" products) make quantitation convenient. Taurine is essential for normal development (Arnold *et al.*, 2007), and ascorbic acid is helpful for captive and other stresses (McKee and Harrison, 1995); these may be added to the paste at 100 mg each, per 600 mg elemental calcium (the contents of 1 calcium capsule).

Outside of the few items discussed earlier, varied, naturalistic diets of whole foods contain all essential nutrients. "Complete and balanced" is the phrase used for formulations; if the caregiver provides a complete diet, the bird will balance it impeccably.

Sick and injured birds often crave and require considerably higher proportions of animal-sourced foods than they ordinarily consume. In extreme cases, herbivorous adults may "ontogenically regress" to accepting only a faunivorous diet.

Estimated *energy requirements* of captive birds are broadly useful. Recent analyses have led to revision of assumptions underlying metabolic rate allometry, including the traditional division between passerines and non-passerines (McKechnie and Wolf, 2004; Hudson *et al.*, 2013). Presently, no known equation is an excellent fit for all taxa, or for birds that, within a taxon, are at either size extreme. Further, individual physiological factors have a considerable impact on caloric needs. Thus the equations shown in Table 3-1 provide only approximations; frequent assessments of body condition and rate of weight change are indispensable guides to feeding.

SEASONAL VARIATIONS

The most dramatic seasonal variation in nutritional requirements is associated with reproduction. Breeding hens require greatly increased

TABLE 3-1 Estimated Daily Caloric (Kcal) Requirement of Birds

Adults at Maintenance	Young at Maximal Growth
$2.3 \times W^{0.65}$	$4.6 \times W^{0.65}$

Adapted from White *et al.*, 2006.
W, Body weight in grams.

intake of the wide array of nutrients (e.g., Blount *et al.*, 2004) used directly or indirectly to synthesize eggs. Caregivers who provide a complete, whole-foods diet witness increases in consumption by which the hen is, as ever, regulating her intake to precisely match her needs.

To a lesser degree, molt also increases requirements for a variety of nutrients (Murphy and King, 1992) including, for many species, carotenoids (Olson and Owens, 2005). These phytochemicals are obtained either directly from plants or as bioaccumulations in whole natural prey.

The majority of avian taxa migrate twice each year, over distances that range from local to transglobal, and exhibit corresponding dietary changes. Some specialists eat from the same category year round, while their specific foods change seasonally (Thompson and Furness, 1995), and sometimes dramatically (Piersma *et al.*, 1993).

Frugivore-insectivores eat large proportions of fruit before migrating, accreting stores of fat needed for the journey to a locale that once again allows them to eat mainly insects.

With changes in food availability, temperate-region birds that migrate only locally are likely to consume very different diets in winter than in summer. Within the set of foods comprising the bird's evolved feeding guild, the gastrointestinal tract (as well, presumably, as physiological and biochemical processes) adapts to seasonal dietary changes over days to weeks (e.g., van Gils *et al*, 2003).

Birds brought into captivity and given nonseasonal fare exhibit maldigestion (Levey and Karasov, 1989). Conversely, birds that are adapted to a captive diet, and then released to confront different food types in the wild, can face an insurmountable obstacle to survival. Thus it is crucial to ensure that the prerelease diet matches the one the bird will be consuming after release.

Scant literature exists on nonreproductive season-specific nutritional needs of birds in captivity. In some species, food preferences have been observed to change seasonally, and outdoor aviary birds are likely to require high-energy digestible foods in winter. Prudence dictates that for every species, seasonal changes should be incorporated into a captive feeding program that also emulates the natural diet in all other possible ways.

REFERENCES

Arnold KE, Ramsay SL, Donaldson C, et al: Parental prey selection affects risk-taking behaviour and spatial learning in avian offspring, *Proc Biol Sci* 274(1625):2563–2569, 2007.

Baldwin MW, Toda Y, Nakagita T, et al: Evolution of sweet taste perception in hummingbirds by transformation of the ancestral umami receptor, *Science* 345:929–933, 2014.

Benzie IF, Choi SW: Antioxidants in food: content, measurement, significance, action, cautions, caveats, and research needs, *Adv Food Nutr Res* 71:1–53, 2014.

Birkhead TR, Fletcher F, Pellatt EJ: Nestling diet, secondary sexual traits and fitness in the zebra finch, *Proc Biol Sci* 266(1417):385–390, 1999.

Blount JD, Houston DC, Surai PF, et al: Egg-laying capacity is limited by carotenoid pigment availability in wild gulls *Larus fuscus*, *Proc Biol Sci* 271:S79–S81, 2004.

Blount JD, Metcalfe NB, Arnold KE, et al: Effects of neonatal nutrition on adult reproduction in a passerine bird, *Ibis* 148:509–514, 2006.

Bolarinwa IF, Orfila C, Morgan MRA: Amygdalin content of seeds, kernels and food products commercially-available in the UK, *Food Chem* 152:133–139, 2014.

Brightsmith DJ, McDonald D, Matsafuji D, et al: Nutritional content of the diets of free-living scarlet macaw chicks in Southeastern Peru, *J Avian Med Surg* 24(1):9–23, 2010.

Brown KJ, Downs CT: Digestive efficiency of a generalist avian feeder, the Cape white-eye (*Zosterops pallidus*), *Comp Biochem Physiol A* 134:739–748, 2003.

Choct M, Dersjant-Li Y, McLeish J: Soy oligosaccharides and soluble non-starch polysaccharides: a review of digestion, nutritive and anti-nutritive effects in pigs and poultry, *Asian Austral J Anim Sci* 23(10):1386–1398, 2010.

Cichon M, Dubiec A: Cell-mediated immunity predicts the probability of local recruitment in nestling blue tits, *J Evol Biol* 18:962–966, 2005.

Cordain L: Cereal grains: humanity's double-edged sword. In Simopoulos AP, editor: *Evolutionary aspects of nutrition and health, diet, exercise, genetics and chronic disease, vol 84*, Karger, Basel, 1999, World Review of Nutrition and Dietetics.

de Matos R: Calcium metabolism in birds, *Vet Clin North Am Exot Anim Pract* 11(1):59–82, 2008.

Dewey KG: The challenge of meeting nutrient needs of infants and young children during the period of complementary feeding: an evolutionary perspective, *J Nutr* 143(12):2050–2054, 2013.

Dierenfeld E, Macek M, Snyder T, et al: A simple and effective egg-based hand-rearing diet for flamingos. In Clauss M, Fidgett A, Huisman T, et al, editors: *Zoo animal nutrition IV*, Fürth, Germany, 2009, Filander Verlag.

Drouin G, Godin J-R, Pagé B: The genetics of vitamin C loss in vertebrates, *Curr Genomics* 12:371–378, 2011.

Elliston EP, Perlman J: Meeting the protein requirements of adult hummingbirds in captivity, *J Wildlife Rehab* 25(2):14–19, 2002.

Engelhardt FR, Geraci JR: Effects of experimental vitamin E deprivation in the harp seal, *Phoca groenlandica*, *Can J Zool* 56(10):2186–2193, 1978.

Fernandez SC: Ph.D. thesis, http://tinyurl.com/phfuqur. 2008.

Frederick H, Dierenfeld E, Irlbeck N, et al: *Analysis of nectar replacement products and a case of iron toxicosis in hummingbirds*. Proceedings of the 5th Nutrition Advisory Group Conference, Minneapolis, MN, 2003.

Fusté E, Obon E, Olid E: Hand-reared common swifts (*Apus apus*) in a wildlife rehabilitation centre: assessment of growth rates using different diets, *J Zoo Aquarium Res* 1(2):61–68, 2013.

Geraci JR: Experimental thiamine deficiency in captive harp seals, *Phoca groenlandica*, induced by eating herring, *Clupea harengus*, and smelts, *Osmerus mordax*, *Can J Zool* 50(2):179–195, 1972.

Gilardi JD, Duffe SS, Munn CA, et al: Biochemical functions of geophagy in parrots: detoxification of dietary toxins and cytoprotective effects, *J Chem Ecol* 25:897–922, 1999.

Gilbert G, Tyler G, Smith KW: Nestling diet and fish preference of bitterns *Botaurus stellaris* in Britain, *Ardea* 91(1):35–44, 2003.

Hasebe M, Franklin DC: Food sources of the rainbow lorikeet *Trichoglossus haematodus* during the early wet season on the urban fringe of Darwin, Northern Australia, *Corella* 28(3):68–74, 2004.

Hoi-Leitner M, Romero-Pujante M, Hoi H, et al: Food availability and immune capacity in serin (*Serinus serinus*) nestlings, *Behav Ecol Sociobiol* 49(5):333–339, 2001.

Hudson LN, Isaac NJB, Reumann DC: The relationship between body mass and field metabolic rate among individual birds and mammals, *J Anim Ecol* 82:1009–1020, 2013.

Jacobs DR, Tapsell LC: Food, not nutrients, is the fundamental unit in nutrition, *Nutr Rev* 65(10):439–450, 2007.

Jacobs DR, Tapsell LC: Food synergy: the key to a healthy diet, *Proc Nutr Soc* 72:200–206, 2013.

Jensen K, Panagiotou G, Kouskoumvekaki I: Nutri Chem: a systems chemical biology resource to explore the medicinal value of plant-based foods, *Nucleic Acids Res*, Advanced Access, 2014.

Kalmar ID, Werquin G, Janssens GPJ: Apparent nutrient digestibility and excreta quality in African grey parrots fed two pelleted diets based on

coarsely or finely ground ingredients, *J Anim Physiol Anim Nutr* 91:210–216, 2007.
Karasov WH, Cork SJ: Glucose absorption by a nectarivorous bird: the passive pathway is paramount, *Am J Physiol* 267:G18–G26, 1994.
Klasing KC: *Comparative avian nutrition*, Wallingford, UK, 1998, CAB International.
Lepczyk CA, Caviedes-Vidal E, Karasov WH: Digestive responses during food restriction and realimentation in nestling house sparrows (*Passer domesticus*), *Physiol Zool* 71(5):561–573, 1998.
Levey DJ, Karasov WH: Digestive responses of temperate birds switched to fruit or insect diets, *Auk* 106:675–686, 1989.
Liu RH: Health-promoting components of fruits and vegetables in the diet, *Adv Nutr* 4:384S–392S, 2013.
MacLeod A, Perlman J: Dietary considerations for the hatchling/nestling passerine, *Natl Wildlife Rehab Assoc Q J* 18(4):2000.
MacLeod A, Perlman J: Cream of the crop: an improved hand-rearing diet for hatchling and nestling columbids, *J Wildlife Rehab* 25(1):12–17, 2002.
Maddocks SA, Church SC, Cuthill IC: The effects of the light environment on prey choice by zebra finches, *J Exp Biol* 204:2509–2515, 2001.
Malcarney HL, Martínez del Rio C, Apanius V: Sucrose intolerance in birds: simple non-lethal diagnostic methods and consequences for assimilation of complex carbohydrates, *Auk* 111(1):170–177, 1994.
McDonald D: Feeding ecology and nutrition of Australian lorikeets, *Semin Avian Exot Pet Med* 12(4):195–204, 2003.
McKechnie AE, Wolf BO: The allometry of avian basal metabolic rate: good predictions need good data, *Physiol Biochem Zool* 77(3):502–521, 2004.
McKee JS, Harrison PC: Effects of supplemental ascorbic acid on the performance of broiler chickens exposed to multiple concurrent stressors, *Poult Sci* 74(11):1772–1785, 1995.
Meehan CL, Garner JP, Mench JA: Environmental enrichment and development of cage stereotypy in orange-winged Amazon parrots (*Amazona amazonica*), *Dev Psychobiol* 44(4):209–218, 2004.
Metz AL, Walser MM, Olson WG: The interaction of dietary vitamin A and vitamin D related to skeletal development in the turkey poult, *J Nutr* 115(7):929–935, 1985.
Murphy ME: Dietary complementation by wild birds: considerations for field studies, *J Biosci* 19(4):355–368, 1994.
Murphy ME, King JR: Energy and nutrient use during moult by white-crowned sparrows, *Zonotrichia leucophrys gambelii, Ornis Scand* 23:304–313, 1992.
Myers MR, Klasing KC: Low glucokinase activity and high rates of gluconeogenesis contribute to hyperglycemia in barn owls (*Tyto alba*) after a glucose challenge, *J Nutr* 129(10):896–1904, 1999.
Olson VA, Owens IPF: Interspecific variation in the use of carotenoid-based coloration in birds: diet, life history and phylogeny, *J Evol Biol* 18:1534–1546, 2005.
Parsons CM, Potter LM, Brown RD Jr: True metabolizable energy and amino acid digestibility of dehulled soybean meal, *Poult Sci* 60(12):2687–2696, 1981.
Pereira LQ, Strefezzi RDF, Catão-Dias JL, et al: Hepatic hemosiderosis in red-spectacled Amazons (*Amazona pretrei*) and correlation with nutritional aspects, *Avian Dis* 54(4):1323–1326, 2010.
Perlman J: Feeding birds for optimal health, Association of Avian Veterinarians Clinical Forum, Feb-Apr 1-6, 2011.
Péron F, Grosset C: The diet of adult psittacids: veterinarian and ethological approaches, *J Anim Physiol Anim Nutr* 98(3):403–416, 2014.
Perrin MR: Niche separation in African parrots. In Harebottle DM, Craig AJFK, Anderson MD, Rakotomanana H, Muchai M, editors: Proceedings of the 12th Pan-African Ornithological Congress, 2009.
Piersma T, Koolhaas A, Dekinga A: Interactions between stomach structure and diet choice in shorebirds, *Auk* 110(3):552–564, 1993.
Ragusa-Netto J, Fecchio A: Plant food resources and the diet of a parrot community in a gallery forest of the southern Pantanal (Brazil), *Braz J Biol* 66(4):1021–1032, 2006.
Ricklefs RE: Patterns of growth in birds. II. Growth rate and mode of development, *Ibis* 115(2):177–201, 1973.
Roura E, Baldwin MW, Klasing KC: The avian taste system: potential implications in poultry nutrition, *Anim Feed Sci Technol* 180:1–9, 2013.
Sabat P, Novoa F, Bozinovic F, et al: Dietary flexibility and intestinal plasticity in birds: a field and laboratory study, *Physiol Zool* 71(2):226–236, 1998.
Sales J, De Schutter L, Janssens GPJ: The use of internal markers to determine metabolizable energy and digestibility of diets in the African Grey parrot (*Psittacus erithacus*), *Vlaams Diergeneeskundig Tijdschrift* 73:176–181, 2004.
Samour J: *Acute toxicity after administration of high doses of vitamin B6 (pyridoxine) in falcons*, Proceedings of the European College of Zoological Medicine Scientific Meeting, Wiesbaden, Germany, 2013.
Scalbert A, Andrés-Lacueva C, Arita M, et al: Databases on food phytochemicals and their health-promoting effects, *J Agric Food Chem* 59(9):4331–4348, 2011.
Schaefer HM, McGraw K, Catoni C: Birds use fruit colour as honest signal of dietary antioxidant rewards, *Funct Ecol* 22:30–310, 2008.
Schaefer HM, Schmidt V, Bairlein F: Discrimination abilities for nutrients: which difference matters for choosy birds and why?, *Anim Behav* 65:531–541, 2003.
Schreiner M, Huyskens-Keil S: Phytochemicals in fruit and vegetables: health promotion and postharvest elicitors, *Crit Rev Plant Sci* 25:267–278, 2006.
Senar JC, Møller AP, Ruiz I, et al: Specific appetite for carotenoids in a colorful bird, *PLoS ONE* 5(5):e10716, 2010.
Sheppard C, Dierenfeld E: Iron storage disease in birds: speculation on etiology and implications for captive husbandry, *J Avian Med Surg* 16(3):192–197, 2002.
Stevens EC, Hume ID: *Comparative physiology of the vertebrate digestive system*, ed 2, Cambridge, UK, 2004, Cambridge University Press.
Thompson DR, Furness RW: Stable-isotope ratios of carbon and nitrogen in feathers indicate seasonal dietary shifts in northern fulmars, *Auk* 112(2):493–498, 1995.
Tomás-Barberón FA, Andrés-Lacueva C: Polyphenols and health: current state and progress, *J Agric Food Chem* 60(36):8773–8775, 2012.
van Gils JA, Piersma T, Dekinga A: Cost–benefit analysis of mollusc-eating in a shorebird. II. Optimizing gizzard size in the face of seasonal demands, *J Exp Biol* 206:3369–3380, 2003.
Vaughan C, Nemeth N, Marineros L: Scarlet macaw, *Ara macao*, (Psittaciformes: Psittacidae) diet in Central Pacific Costa Rica, *Rev Biol Trop* 54(3):919–926, 2006.
Watters JV: Searching for behavioral indicators of welfare in zoos: uncovering anticipatory behavior, *Zoo Biol* 33:251–256, 2014.
White CR, Phillips NF, Seymour RS: The scaling and temperature dependence of vertebrate metabolism, *Biol Lett* 2(1):125–127, 2006.
Wilkinson SJ, Bradbury EJ, Bedford MR, et al: Effect of dietary nonphytate phosphorus and calcium concentration on calcium appetite of broiler chicks, *Poult Sci* 93(7):1695–1703, 2014.
Witmer MC: Annual diet of cedar waxwings based on US Biological Survey records (1885-1950) compared to diet of American robins: contrasts in dietary patterns and natural history, *Auk* 113(2):414–430, 1996.

THE IMPORTANCE OF DIET QUALITY

John Cooper

Food is a vital part of a bird's biological needs and essential to its health and welfare (Cooper, 2003). It is a long-established fact that the feeding of birds with diets that are inadequate in terms of essential nutrients or that present hazards on account of poor quality can cause disease or death.

Despite this, most proprietary bird diets are not subject to screening or health monitoring other than visual, naked eye, and basic manual checks. Little attention is paid to appearance, consistency, acceptability, and palatability and perhaps even less to the fact that such diets may harbor infectious agents, toxins, or adulterants. Greater quality control would appear to be desirable and could help ensure that products intended for both captive and wild (free-living) birds do not pose significant health risks.

For only a few species of bird are there reliable data on nutritional requirements (Jones, 2011), yet it is well-recognized that a diet that is

inadequate or unsatisfactory in quantity or quality, or both, may cause a bird to develop a deficiency or metabolic disease and compromise its welfare (Brue, 1994). Food items that are dusty or contain sharp or abrasive material may damage a bird's respiratory or alimentary tract. Dust and other debris can serve as fomites, transporting, for example, spores of the fungus *Aspergillus fumigatus*. In addition, the constituents of the diets can be a source of potentially pathogenic organisms, especially bacteria, yeasts, and protozoa, and toxins originating from fungi (e.g., mycotoxins) and other sources. These can cause ill health in the birds or, when placed outside, may affect avian and other species, sometimes including *Homo sapiens* (Cooper, 1990). At a time when there is particular concern about the health of garden birds (Kirkwood and Macgregor, 1998; Lawson *et al.*, 2001, 2006a; Pennycott *et al.*, 2005, 2006), the physical, chemical, and microbiological quality and safety of diets are increasingly relevant.

Food analysis is a specialized subject (Makowski *et al.*, 2010). It is concerned not only with the quantity and quality of ingredients but also—often important from a legal point of view—with providing evidence of contamination, adulteration, improper processing, decomposition, poisonous or deleterious materials, unacceptable additional contents, and signs of processing or manufacturing errors. Some types of analysis are specific and require special techniques, for example, the detection of mycotoxins. These are both toxic and carcinogenic. They can be present on various agricultural products (Whitaker *et al.*, 2010) and have been detected in free-living European passerine birds (Lawson *et al.*, 2006b).

The main techniques employed in large-scale (commercial) food analysis involve gross and hand lens examination, microscopy, toxicology, chemical analysis, scanning electron microscopy, x-ray microanalysis, Fourier transform infrared spectroscopy, and near-infrared (NIR) spectroscopy. However, as the subsequent section explains, samples can be satisfactorily investigated at a simple level, adequate as a routine screening exercise, using relatively inexpensive tests. The methods used are appropriate to a veterinary practice laboratory.

REFERENCES

Brue N: Nutrition. In Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Cooper JE: Birds and zoonoses, *Ibis* 132:181–191, 1990.

Cooper JE: *Captive birds in health and disease*, Fordingbridge, UK, 2003, World Pheasant Association and Hancock Publishing, Canada.

Jones AK: *Keeping parrots*, Marlborough, UK, 2011, The Crowood Press.

Kirkwood JK, Macgregor SK: Salmonellosis in provisioned free-living greenfinches (*Carduelis chloris*) and other garden birds, Chester, UK, 1998, Proceedings of the European Association of Zoo and Wildlife Veterinarians Second Scientific Meeting.

Lawson B, Cunningham A, Chantrey J, et al: Epidemic finch mortality, *Vet Rec* 159:367, 2006a.

Lawson B, MacDonald S, Howard T, et al: Exposure of garden birds to aflatoxins in Britain, *Sci Total Environ* 361:124–131, 2006b.

Lawson B, Hughes LA, Peters T, et al: Pulsed-field gel electrophoresis supports the presence of host-adapted *Salmonella typhimurium* strains in the British garden bird populations, *Appl Environ Microbiol* 77:8139–8144, 2001.

Makowski J, Vary N, McCutcheon M, et al: *Microscopic analysis of agricultural products*, ed 4, Urbana, IL, 2010, American Oil Chemists Society.

Pennycott TW, Lawson B, Cunningham AA, et al: Necrotic ingluvitis in wild finches, *Vet Rec* 157:360, 2005.

Pennycott TW, Park A, Mather HA: Isolation of different serovars of *Salmonella enterica* from wild birds in Great Britain between 1995 and 2003, *Vet Rec* 158:817–820, 2006.

Whitaker TB, Slate AB, Doko MB, et al: *Sampling procedures to detect mycotoxins in agricultural commodities*, Philadelphia, 2010, Springer Science and Business Media.

LABORATORY TESTING AND INVESTIGATION OF DIETS

Diets that are offered for sale as bird food range from pelleted preparations and mashes for poultry and game birds through seed mixes for wild birds and finches in captivity, to insects and sugar solutions for specialized species in zoos and captive-breeding programs. Carnivorous birds, such as raptors, require dead food of animal origin; this is not discussed here but reference should be made to Cooper (2008). Bird diets for passerine and psittacine species are available to the public from a variety of outlets, ranging from long-established bird food suppliers to market stalls.

This section describes a simple, laboratory-based screening procedure, referred to subsequently as a quality control (QC) program, that was first developed by the author in 2012 and is now proving valuable in providing information about the quality of seed and insect-based diets that are offered to captive and wild birds in the UK.

METHODS USED

The first requirement in developing this laboratory-based QC program was to obtain samples of proprietary diets of known quality and provenance to develop and assess procedures. A long-standing professional relationship with the British bird food company, John E Haith (Haith's), made possible the supply for investigation of a range of commercially produced diets. These were received by post or by hand and then examined in the author's laboratory (Fig. 3-7), with additional investigations at the University of Cambridge, Department of Veterinary Medicine, UK, when needed.

Initially each sample is examined visually using the naked eye, a mounted magnifying lens, and a dissecting (stereo) microscope (Fig. 3-8). Appearance, odor, and certain physical features are carefully recorded, by the same observer. Both reflected and transmitted light are used when using the lens and dissecting microscope. If appropriate, to help identify food components and to detect any possible contaminants or adulterants, the test diets or their constituents are also investigated using a compound microscope. Some components are stained using toluidine blue, iodine, or Sudan stains (Flint and Firth, 1988; Bundrett *et al.*, 1991; Webb and Cooper, 2013).

The next part of the investigation usually comprises a simple flotation test, whereby various fractions of the diet are separated on the

FIGURE 3-7 The author in his laboratory, examining a diet sample. Note that only basic facilities and equipment are required at this stage.

FIGURE 3-8 A key initial part of analysis is microscopic examination. Here a seed sample is investigated for quality using a stereomicroscope.

FIGURE 3-9 A flotation test on an uncleaned diet reveals dust and other particulate material in the supernatant.

basis of whether they float or sink in fluid or, in some cases, go into suspension. Samples can be taken of the fraction that has floated, the deposit, and the supernatant fluid and then subjected to more detailed investigation.

The volume of fluid and the amount of product required for the test depend upon the type of material under investigation; the latter can range from tiny canary seeds to various mixtures containing, for example, peanuts, sunflower seeds, dried mealworm (*Tenebrio molitor*) larvae, and pieces of pelleted diet. Flotation tests help in the detection and fractionation of dust and other particulate material (Fig. 3-9).

Aerobic culture for bacteria is performed on the surface, sometimes the contents, of each diet, using blood agar plates (Fig. 3-10).

FIGURE 3-10 Culture of a clean, dry sample at room temperature for 72 hours usually yields only a few bacterial and fungal colonies.

An aliquot of the product tested is kept in a tightly sealed container at room temperature for a further 4 weeks and checked with a lens at intervals to see if there is evidence of metamorphosed invertebrates, such as larvae or imagines of beetles (Coleoptera). Such invertebrates are not easily detected when they are present as ova.

A sample is also retained for 3 months in a freezer in case it is necessary to reexamine it at a later date—in the event of a complaint from a birdkeeper or an enquiry from a veterinarian, for example.

RESULTS AND CONCLUSIONS

This 3-year study has confirmed that useful information about the physical features of diets can be obtained using a relatively simple protocol and by recording results on a specially compiled report sheet.

The main features of the testing protocol are as follows:

a. Examination by naked eye, magnifying lens, and microscope
b. Flotation tests
c. Use of a compound microscope to examine certain samples, both stained and unstained
d. Culture on blood agar plates.

In the context of part a in the list above, it should be noted that it has proved particularly fruitful to combine visual (naked eye/mounted magnifying lens) examination with the use of a dissecting (stereo) microscope and to have a compound microscope as backup (Klein and Marquard, 2005). Such a multifaceted approach provides a rapid and apparently very reliable means of assessing the appearance and consistency of seeds and other dietary items, including mealworms and invertebrate derivatives, such as shed skins of mites. This method is also effective in detecting animate and inanimate contaminants, some of which may not be revealed in conventional analytical tests (Flint, 1994).

The methods described here are relatively simple to perform and, using standard equipment, can be carried out satisfactorily and inexpensively in a small laboratory. Some can be easily learned and put into practice by veterinary support staff and by appropriate personnel who care for birds and other animals in zoos and rehabilitation centers. Training in the examination of diets should form part of veterinary curricula, especially for students with an interest in a career in avian medicine (Fig. 3-11).

Basic investigations are usually adequate as a routine screening exercise; they can, if required, be adapted and expanded to provide a

FIGURE 3-11 Students at Cambridge Veterinary School (UK) learn how to test diets intended for captive birds and wildlife. (Courtesy Haith's Bird Food.)

more comprehensive quality control program for the evaluation of bird diets.

ACKNOWLEDGMENTS

I am grateful to Simon King and Margaret E. Cooper for their helpful comments on this section. The Directors of John E. Haith Ltd, Grimsby, North East Lincolnshire, UK, generously provided samples of their bird diets; this enabled working practices, protocols, and procedures to be established. Figure 3-5 is reproduced with permission of Haith's Bird Food. My thanks go to Madeline Fordham, Louise Grimson, and Rayna Skoyles of the University of Cambridge, Department of Veterinary Medicine, for their help and good humor in expertly processing a range of unconventional "diagnostic" samples.

REFERENCES

Bundrett MC, Kendrick B, Peterson CA: Efficient lipid staining in plant material with Sudan Red 7B or Fluoral Yellow 088 in polyethylene glycol-glycerol, *Biotech Histochem* 66:111–116, 1991.

Cooper JE: *Birds of prey: health & disease*, Oxford, UK, 2008, John Wiley and Sons.

Flint FO, Firth BM: Improved toluidine blue stain mountant for the microscopy of food products, *Analyst* 113:365–366, 1988.

Flint O: *Food microscopy: a manual of practical methods, using optical microscopy*. Microsc Handb 30, 1994.

Klein H, Marquard R: *Feed microscopy: atlas for the microscopic examination of feed containing vegetable and animal products*, Christchurch, NZ, 2005, Agrimedia.

Webb J, Cooper JE: Animal diets and foodstuffs. In Cooper JE, Cooper ME, editors: *Wildlife forensic investigation: principles and practice*, Boca Raton, FL, 2013, Taylor & Francis/CRC.

4

Capture and Handling

CAPTURE

Thomas A. Bailey

PHYSICAL CAPTURE

To handle the avian patient for a physical examination it must first be captured (Figs. 4-1 to 4-4). The method of capture depends on the species, the age, the level of tameness, the size of the cage/enclosure, and the environment.

Many patients are presented in small cages, and before capture is attempted all perches and food and water bowls should be removed. Small cage doors do not allow easy access and it may be more practical to remove the entire top of the cage in a darkened room. A paper towel or cloth may be used to serve as a visual barrier to enable the capture of many birds. Many tame cage birds may have been trained to hop on to your finger or wrist, after which they can be grasped from behind. Positive reinforcement techniques are commonly used in mammals managed in zoo and research establishments. Positive reinforcement programs have been developed for psittacines and trained behaviors in macaws include accepting liquid from a syringe, stepping onto a perch, stepping onto weighing scales, and allowing pressing of a syringe to the pectoral muscle area as a surrogate for an intramuscular injection (Daugette *et al.*, 2012). If owners or keepers are prepared to invest the time to train their birds, these techniques offer the opportunity to reduce the stress associated with capture and restraint. Trained raptors are best hooded before they are captured. However, some raptors, particularly trained imprint falcons used in breeding programs that are habituated by close interaction with skilled human handlers, are also trained to be manually caught for some procedures, such as being massaged for semen collection (males) or voluntarily inseminated (females), without the need for nets.

Army night vision goggles (Fig. 4-4) can be used to capture birds by hand in darkened rooms or aviaries at night. Birds housed in larger aviaries are often able to escape by flying or running and they may be captured using nets or corrals. A single bird in a small aviary can be captured by hand by one person if the bird is tame or by using a net handled by one or more people if the bird has a nervous temperament. Nets may be used either with or without a handle. Rims of nets should be padded to minimize the potential of causing traumatic injuries. This can easily be done by taping insulating foam to the rim of the net. Using a handle depends on the available space within the aviary. The catcher should push the bird into a corner before closing in and netting the bird. If the bird attempts to run or fly past the catcher, the net should be placed in front of it so the bird runs or flies into it. Care should be taken not to cause any injuries when netting flying birds. In all cases, if there is any doubt the catcher should allow the bird to pass by. Once netted the bird should be carefully removed and either held or placed in a box or carrier. While removing the bird from the net, special attention should be paid to the feet, head, and carpometacarpal joints to ensure that they are not entangled in the netting as the bird is pulled out. Using a towel can be helpful to keep larger birds under control.

In larger aviaries, flocks of cursorial birds (e.g., bustards) may be caught by making a corral from shade cloth. This should be hung or fastened to extensible metal poles and shaped into a blind-ended funnel with a wide mouth and a small circular catching area at the blind end. Some larger species, such as kori bustards (*Ardeotis kori*), may best be captured by cornering and grabbing them by hand. However, even with such large birds a net placed over their head and upper body makes capture easier and, therefore, less stressful for the bird.

Birds of prey, particularly falcons in large flight aviaries, can be caught in catching rooms (Fig. 4-5). These are rooms in which the birds become habituated to enter to receive food. On the day that the birds are to be caught, the entrance to the room is closed using a sliding door on a pulley system after the falcon has entered. The falcon can then be caught by net in the darkened catching room. Such large aviaries should also be built with canvas hangings that discourage the birds from hitting the aviary roof at high speed and damaging themselves (Fig. 4-6). Captive-bred falcons that will be sold into the commercial falconry market can also be habituated to falconry training while they are still maintained in large free-flight aviaries by placing food into the aviary through and on manikins that resemble the falconers who will be training them (Fig. 4-7).

Specialized texts should be referred to for the capture of ratites (Doneley, 2006). The capture and handling of the main avian groups maintained as exotic pets and in zoological collections are presented in Fowler and Miller (2003) and Girling (2013).

Readers interested in trapping wild birds are recommended to read Bub (1995). This book is a fascinating and thorough account of the methods used to trap all types of birds and is well illustrated with contemporary and historical images. Examples of devices for the capture of free-living birds include the following:

- Walk-in or swim-in traps (wildfowl)
- Cannon or rocket nets (wildfowl, gamebirds, and ostriches)
- Bal-chatri (raptors)
- Boma (ratites)
- Pop-up corral (ostriches)
- Dho-gazza (raptors)

Trapping-related injuries are not uncommon, and before attempting to trap free-living birds, veterinarians should be familiar with local wildlife regulations and should ensure familiarity with the particular trapping method to be used.

FIGURE 4-1 Larger species of bird can be pushed toward a corner where they can be captured.

FIGURE 4-2 Kori bustards (*A. kori*) may be captured when they pass between the handler and the side of an enclosure.

FIGURE 4-3 The use of sliding gates facilitates moving birds, such as houbara bustards (*Chlamydotis undulata*), from one quarter to another. Using this system, a bird can be singled out from a larger group to ease physical capture.

FIGURE 4-4 Army night vision goggles can be used to capture birds, such as this wild turkey (*Meleagris gallopavo*), by hand in darkened rooms or aviaries at night.

FIGURE 4-5 Falcon-catching pen.

FIGURE 4-6 Catching a falcon in the dark inside a catching room.

FIGURE 4-7 The "fake-sheikh." Falcons in a large flight pen are fed through this manikin of an Arab falconer. This is a method of habituating the falcons to associate the presentation of food with a human-shaped manikin. This is believed to contribute to the training of these birds for falconry.

REFERENCES

Bub H: *Bird trapping and bird banding. A handbook for trapping methods all over the world*, New York, 1995, Cornell University Press.

Daugette KF, Hoppes S, Tizard I, et al: Positive reinforcement training facilitates the voluntary participation of laboratory macaws with veterinary procedures, *J Avian Med Surg* 26(4):248–254, 2012.

Doneley B: Management of captive ratites. In Harrison G, Lightfoot T, editors: *Clinical avian medicine*, vol 2, Lake Worth, FL, 2006, Spix Publishing.

Fowler M, Miller E: *Zoo and wildlife medicine*, St Louis, 2003, Saunders.

Girling S: Avian handling and chemical restraint. In *Veterinary nursing of exotic pets*, Chichester, UK, 2013, Wiley-Blackwell.

FURTHER READING

Austin DH, Peoples TE, Williams LE: Procedures for capturing and handling live wild turkeys, *Southeastern Assoc Game Fish Commissioners* 26:222–236, 1972.

Bailey TA: *Diseases of and medical management of houbara bustards and other Otididae*, Abu Dhabi, 2008, Environmental Agency Abu Dhabi.

Bird DM, Bildstein KL: *Raptors: research and management techniques*, Hancock House, Blaine, WA, 2007, Raptor Research Foundation.

Cooper JE: Caged and wild birds. In Anderson RS, Edney AT, editors: *Practical animal handling*, Oxford, UK, 1991, Pergamon Press.

Fowler ME: *Restraint and handling of wild and domestic animals*, Ames, IA, 1995, Iowa State University Press.

Mullineaux E, Best D, Cooper JE: *BSAVA manual of wildlife casualties*, Gloucester, UK, 2003, British Small Animal Veterinary Association.

Pizzi R: Examination, triage and hospitalisation. In Chitty J, Lierz M, editors: *Manual of raptors, pigeons and waterfowl*, Cheltenham, UK, 2008, British Small Animal Veterinary Association Ltd.

Raftery A: Avian anaesthesia, *In Pract* 35:272–278, 2013.

Sonsthagen TF: *Restraint of domestic animals*, Goleta, CA., 1991, American Veterinary Publications.

CHEMICAL CAPTURE

Drugged baits were first used by J. L. Daude in 1942 to capture pest birds in France and are considered to be the most effective method for capturing free-living birds, particularly game birds and waterfowl (Jessup, 1982). Larger birds such as ratites may be chemically immobilized, under both captive and field conditions, using blow guns or pole syringes to deliver intramuscular drugs.

Avian veterinarians may be involved in the capture of free-living birds for the following reasons:

- Biomedical studies
- Disease control
- Game management
- Nuisance animal control
- Population control
- Fitting radio or satellite transmitters
- Ringing and biological studies
- Translocation

Baited food items include corn, eggs, and meat for the capture of granivorous Gruiformes and waterfowl and corvids and raptors, respectively (Jessup, 1982; Garner, 1988; Stouffer and Caccamise, 1991; Belant and Seamans, 1997; Hayes *et al.*, 2003).

Their use has been reported in the following species:

- American crows (*Corvus brachyrhynchos*)
- Canada geese (*Branta canadensis*)
- Doves (*Zenaidura macroura*)
- Ducks (*Anas platyrhynchos*)
- Harris hawk (*Parabuteo uncinatus*)
- Pheasants (*Phasianus colchicus*)
- Red-winged blackbirds (*Agelaius phoeniceus*)
- Sandhill cranes (*Grus canadensis*)

- Wild turkey (*Meleagris gallopavo*)
- Wood pigeons (*Columba palumbus*).

Dose rates for some oral drugs are presented in Table 4-1. Combinations of drugs have also been used (Jessup, 1982; Cyr and Brunet, 1992), for example, diazepam and α-chloralose in waterfowl (0.3-0.4 and 0.1-0.12 g per cup of bait, respectively), and α-chloralose and secobarbital in red-winged blackbirds (*A. phoeniceus*; 0.02-0.025 and 0.025-0.03 mg, respectively). Although oral ketamine has been used to successfully sedate an escaped raptor (Garner, 1988), it was not found to be effective for capturing turkeys (Clutton, 1998). The use of 1-2 grains of pentobarbital mixed with bread has also been reported to immobilize free-living ducks sufficiently for capture within 15-20 minutes (Harrison, 1986).

When drugged baits are used, it is difficult to control the dose and rate of absorption of drugs that have been ingested because of range of sizes and species, health status, and the weather and other environmental conditions. Complications can also occur in sedated individuals that are not captured, including:

- Hypothermia
- Hyperthermia
- Overdose
- Suffocation
- Aspiration pneumonia
- Drowning
- Predation
- Peer-inflicted trauma

Once the sedated birds have been caught, they may have to be confined to a recovery pen until the effects of the drug have worn off. If birds are overdosed, they can often be saved if an incision is made in the crop, the drugged bait is removed, and the crop is washed out (Jessup, 1982). Although it is impossible to control the amount of bait consumed, drugged baits are considered to cause less than 10% mortality when properly applied (Jessup, 1982). Before attempting oral baiting, veterinarians should be familiar with local wildlife regulations and the relevant literature.

Intramuscular ketamine has even been given by remote-controlled injector placed in the nest of breeding seabirds (Wilson and Wilson, 1989). African penguins (*Spheniscus demersus*), cape gannets (*Morus capensis*), bank cormorants (*Phalacrocorax neglectus*), and crowned cormorants (*P. coronatus*) anesthetized in this way were easily captured for biological studies.

Combinations of etorphine hydrochloride, acepromazine maleate, ketamine, medetomidine hydrochloride, and xylazine hydrochloride delivered intramuscularly by blow guns or pole syringes have been used to immobilize ostriches (*Struthio camelus*) and double-wattled cassowary (*Casuarius casuarius*; Robinson and Fairfield, 1974; Stoskopf *et al.*, 1982; Samour *et al.*, 1990; Ostrowski and Ancrenaz, 1995). Grobler and Begg (1997) reported the capture of three free-living kori bustards in the Kruger National Park using a dart gun and 1 mg of etorphine hydrochloride and 100 mg ketamine/5 mg xylazine to catch two birds (reversed with antidotes to etorphine and xylazine) and 30 mg/kg zolazepam/tiletamine (Zoletil) for one bird. Birds captured with Zoletil need to be kept in a quiet, dark, and undisturbed environment for at least 12 hours, and based on their experience, Grobler and Begg (1997) recommended using 20-25 mg/kg. Complications of chemical immobilization include hyperthermia, regurgitation, inhalation pneumonia, and myopathy. Dosages of chemical agents used to anesthetize ratites are dealt with in depth by Keffen (1993) and Tully and Shane (1996).

Intranasal administration of midazolam (2 mg/kg) has been used to sedate Amazon parrots to facilitate manual restraint for physical examination and diagnostic procedures (Mans *et al.*, 2012). Flumazenil can be used to antagonize the effects of midazolam. Likewise, administration of a low dose of intramuscular ketamine-medetomidine to a hooded, but unrestrained, falcon facilitates the restraint of the birds for inhalation anesthesia (Molero *et al.*, 2007). Similarly intramuscular xylazine (0.3-1.0 mg/kg) can be given to hooded raptors to enable the fitting of wing and tail guards before the birds are placed in transport boxes for shipment (Figs. 4-8 and 4-9). Atipamezole can be used to reverse the xylazine at the end of the procedure.

TABLE 4-1 Drugs Given as Oral Bait for Capturing Free-Living Birds

Agent	Species	Dose	Reference
α-Chloralose	Wild turkey	2.0 g pcb (per cup of bait)	Austin *et al.*, 1972; Williams *et al.*, 1973
	Sandhill cranes	0.45-0.5 g pcb	Williams and Phillips, 1973
	Canada geese	0.25 g pcb	Jessup, 1982
	American crows	0.035 g per egg	Stouffer and Caccamise, 1991
Ketamine	Harris hawk	100 mg/kg meat	Garner, 1988
Methoxymol	Wild turkey	4.0 g pcb	Jessup, 1982
	Doves	1.5-2.0 g pcb	
Methohexital	Doves	1.25 g pcb	Jessup, 1982
Sodium amobarbital	Mallards	900 mg	Gordon, 1977
Sodium secobarbital	Doves	1.25 g pcb	Jessup, 1982
Tribromoethanol	Wild turkey	10-11 g	Williams *et al.*, 1973
	Pheasant	40 g/kg corn	Fredrickson and Trautman, 1978

FIGURE 4-8 Heavily sedating the falcon (xylazine) enables it to be checked and fitted with feather protectors before shipment.

FIGURE 4-9 Sedated falcon (xylazine) being fitted with feather protectors before shipment.

REFERENCES

Austin DH, Peoples TE, Williams LE: Procedures for capturing and handling live wild turkeys, *Southeastern Assoc Game Fish Commissioners* 26:222–236, 1972.

Belant JL, Seamans TW: Comparison of three formulations of alpha-chloralose for immobilization of Canada geese, *J Wildl Dis* 33:606–610, 1997.

Clutton RE: Inefficacy of oral ketamine for chemical restraint in turkeys, *J Wildl Dis* 24:380–381, 1998.

Cyr A, Brunet J: Anesthetization of captive red-winged blackbirds with mixtures of alpha-chloralose and secobarbital, *J Zoo Wildlife Med* 24:80–82, 1992.

Fredrickson LF, Trautman CG: Use of drugs for capturing and handling pheasants, *J Wildl Manage* 42:690–693, 1978.

Garner MM: Use of an oral immobilizing agent to capture a Harris hawk (*Parabuteo uncinatus*), *J Raptor Res* 22:70–71, 1988.

Grobler DG, Begg S: *Chemical capture of kori bustard (*Ardeotis kori*), Newsletter of the World Association of Wildlife Veterinarians*, 1997.

Harrison GJ: Anesthesiology. In Harrison GJ, Harrison LR, editors: *Clinical avian medicine and surgery*, Philadelphia, 1986, WB Saunders.

Hayes MA, Hartup BK, Pittman JM, et al: Capture of sandhill cranes using alpha-chloralose, *J Wildl Dis* 39:859–868, 2003.

Jessup DA: Chemical capture of upland game birds and waterfowl: oral anesthetics. In Nielsen L, Haigh JC, Fowler ME, editors: *Chemical immobilization of North America wildlife*, Milwaukee, WI, 1982, Wisconsin Humane Society.

Keffen RH: The ostrich *Struthio camelus*: capture, care, accommodation, and transportation. In McKenzie AA, editor: *The capture and care manual*, Pretoria, South Africa, 1993, Wildlife Decision Support Services.

Mans C, Sanchez-Migallon Guzman D, Lahner LL, et al: Sedation and physiologic response to manual restraint after intranasal administration of midazolam in Hispaniolan Amazon parrots (*Amazona ventalis*), *J Avian Med Surg* 26(3):130–139, 2012.

Molero C, Bailey TA, Di Somma A: Anaesthesia of falcons with a combination of injectable anaesthesia (ketamine-medetomidine) and gas anaesthesia (isoflurane), *Exot DVM* 9(1):6–8, 2007.

Ostrowski S, Ancrenaz M: Chemical immobilisation of rednecked ostriches (*Struthio camelus*) under field conditions, *Vet Rec* 136:145–147, 1995.

Robinson PT, Fairfield J: Immobilization of an ostrich with ketamine HCl, *J Zoo Wild Anim Med* 5:11, 1974.

Samour JH, Irwin-Davies J, Faraj E: Chemical immobilisation in ostriches (*Struthio camelus*) using etorphine hydrochloride, *Vet Rec* 127:575–576, 1990.

Stoskopf MJ, Beall FB, Ensley PK, et al: Immobilization of large ratites: blue necked ostrich (*Struthio camelus austrealis*) and double wattled cassowary (*Casuarius casuarius*), with hematologic and serum chemistry data, *J Zoo Wild Anim Med* 13:160–168, 1982.

Stouffer PC, Caccamise DF: Capturing American crows using alpha-chloralose, *J Field Ornithol* 62:450–453, 1991.

Tully TN, Shane SM: *Ratite management, medicine and surgery*, Malabar, FL, 1996, Krieger Publishing.

Williams LE, Austin DH, Peoples TE, et al: *Capturing turkeys with oral drugs*, 1973, National Wild Turkey Symposium.

Williams LE, Phillips RW: Capturing sandhill cranes with alpha-chloralose, *J Wildl Manage* 37:94–97, 1973.

Wilson RP, Wilson M-PTJ: A minimal-stress bird-capture technique, *J Wildl Manage* 53:77–80, 1989.

FURTHER READING

Gordon B: The use of sodium amobarbital for waterfowl capture, *J Zoo Wild Anim Med* 8:34–35, 1977.

Loibl MF, Clutton RE, Marx BD, et al: Alpha-chloralose as a capture and restraint agent of birds: therapeutic index determination in the chicken, *J Wildl Dis* 24:684–687, 1988.

HANDLING

Thomas A. Bailey

IMMOBILIZATION

The main aims when restraining birds are to immobilize the wings and to control the legs and heads of species with powerful feet and beaks (Figs. 4-10 to 4-23). Time spent practicing techniques, along with a dose of patience, are essential prerequisites for minimizing the possibility of injury and stress to both the bird and handler. Equipment used to assist in the restraint of birds for physical examination is listed in Table 4-2.

The strategies for resisting human handling vary between bird species. Hawks generally tend to use their feet to resist the handler while falcons, imprinted birds of prey, vultures, some eagles, and some owls are likely to bite and "foot" the handler. Larger birds, such as swans, can cause injuries with their wings, while ratites have a dangerous kick. Knowing how the animal is likely to resist may assist the handler in making split-second decisions necessary to restrain a bird safely. Recommended techniques for the handling and restraint of different groups of birds are given in Table 4-3. Further specialized information on handling techniques for different species of birds may be gleaned from the texts listed in the bibliography.

Restraining devices made up of medium-weight canvas and Velcro straps have been designed and successfully used in bustards (Figs. 4-10, 4-24, and 4-25) and other species such as swans and small to medium-sized birds of prey (Harris and Brown, 2003). Falconers refer to these devices as "casting jackets." These devices protect the birds from trauma within transport boxes or crates and protect the integrity of the feathers.

FIGURE 4-10 Restraining a houbara bustard (*C. undulata*) using a body harness. These are manufactured from a medium-weight canvas cloth and Velcro bands. These devices are commonly used to restrain large waterfowl, such as swans, and some birds of prey.

FIGURE 4-11 Restraint technique for a medium-sized houbara bustard with a falconry hood in place.

FIGURE 4-12 Correct procedure for holding the hind limbs of a bustard—placing one or two fingers between them.

FIGURE 4-13 Restraint technique for a large kori bustard (*A. kori*) with a cloth hood in place.

FIGURE 4-14 Superficial pressure damage to the skin on the medial aspect of the hocks of a kori bustard after incorrect handling.

FIGURE 4-15 Correct method of restraint of an African grey parrot (*Psittacus erithacus*). (Courtesy A. Jones.)

FIGURE 4-16 Potentially dangerous birds such as this golden eagle (*Aquila chrysaetos*) should be restrained with gloves. In addition, female handlers should also wear a leather apron whenever handling large raptors. (Courtesy A. Jones.)

FIGURE 4-17 Wrapping a psittacine in a paper towel while recovering from anesthesia. (Courtesy A. Jones.)

FIGURE 4-18 Saker falcon (*F. cherrug*) with an adapted hood to prevent self-inflicted injuries. (Courtesy Dr. J. Samour.)

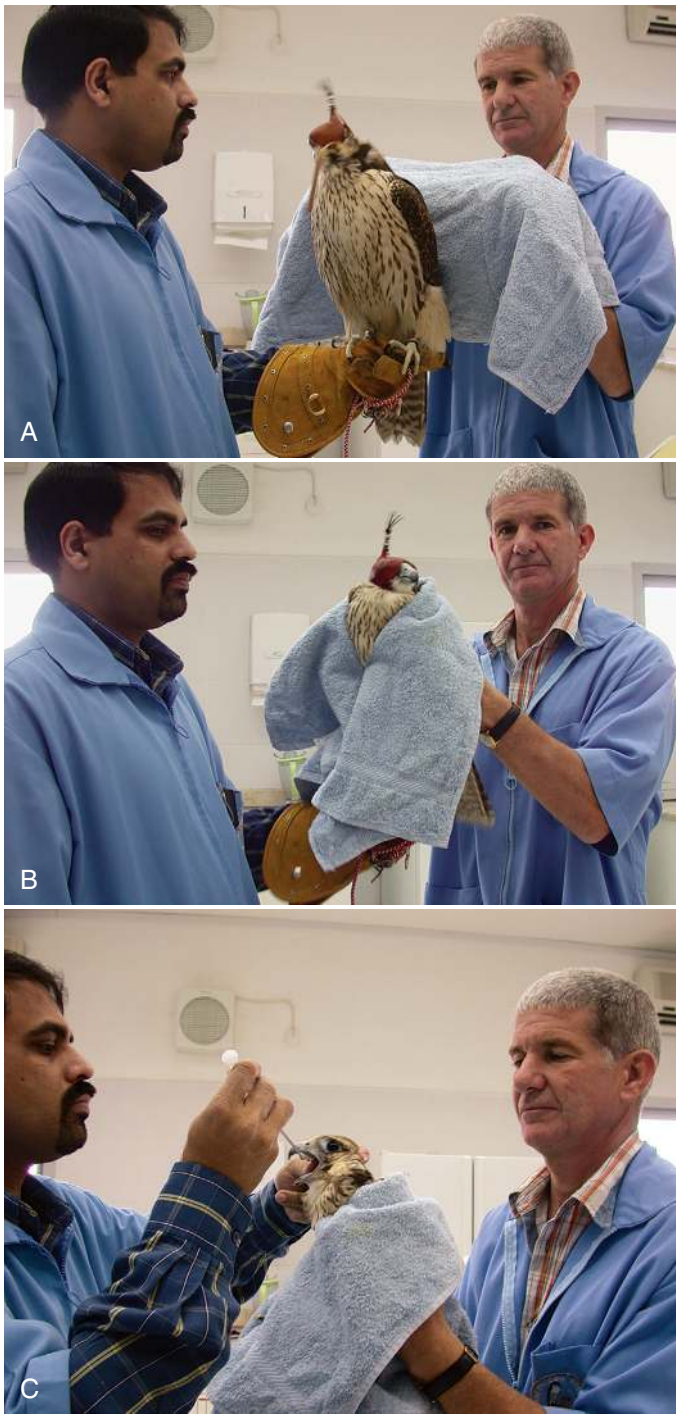


FIGURE 4-19 Restraining a hooded falcon using a towel. **(A),** The assistant is holding the hooded falcon on his gloved hand. The operator holds a soft towel ready to place it around the body of the bird. **(B),** The operator wraps the towel around the body of the bird and holds the falcon firmly. **(C),** The assistant has removed the glove from his hand and can now proceed with administering oral medication to the falcon.

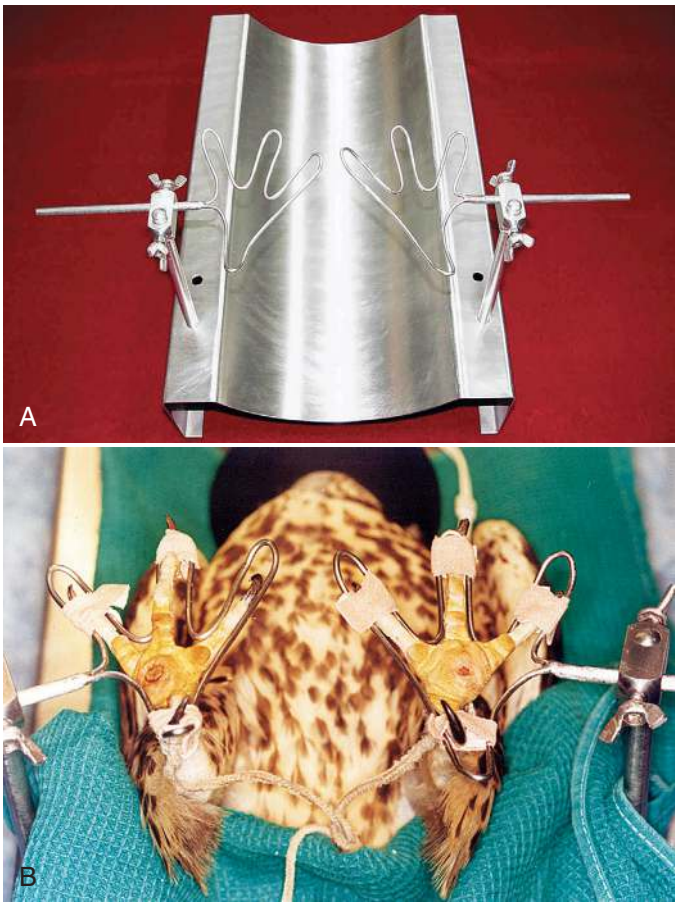


FIGURE 4-20 **(A),** Customized restraint device for surgery on the avian foot. **(B),** The device used to immobilize the feet of a falcon prior to bumblefoot surgery. (Courtesy Dr. J. Samour.)

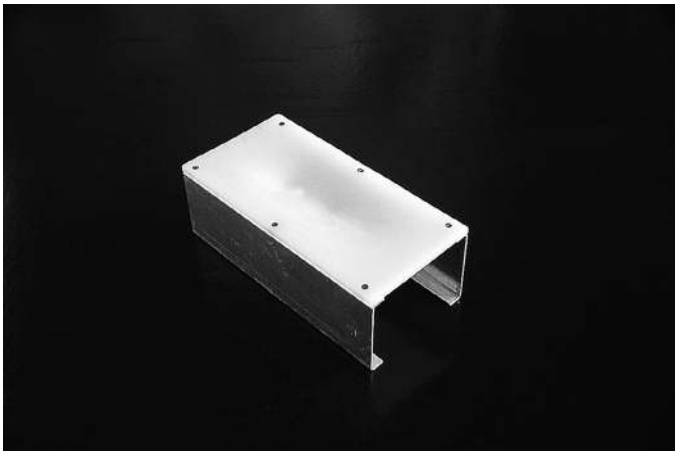


FIGURE 4-21 Operating table (20 cm × 15 cm × 10 cm) used for surgical procedures in small birds (<50 g). Note that the sides are made of aluminum and the top is made of Perspex molded to accommodate the shape of the body. The table can be placed over a heating pad to maintain a suitable temperature during surgery. (Courtesy Dr. J. Samour.)

TABLE 4-2 Equipment That May Facilitate the Handling and Restraint of Birds

Equipment	Purpose	Comments
Cardboard tubing	Minimizes struggling and facilitates weighing and other procedures	Often used by field biologists; the bird appears quieter and less easily stressed
Cloth bag, sack, stocking, or pillow case	As above	Care must be taken not to asphyxiate or damage the bird; if cloth material is used it should be washed and autoclaved between each bird to prevent transmission of infections
Cork or rubber tubing	Can be placed over bills that have a sharp tip to reduce potential damage to handler's face	
Ear protectors	To prevent hearing loss that can occur from repeated exposure to screaming patients	
Elastic bands and sticky tape	To seal beak and protect the handler	Remember that the bird can still stab and to remove the band or tape before release
Foot wraps	Immobilize the feet of birds of prey	Place a roll of cotton or gauze on the footpad and wrap the feet with a nonadhesive wrap to immobilize the talons
Forked stick or handlebars	To fend off large ostriches or other ratites	
Gloves	Reduction of damage to handler	Avoid unless essential; should not be used to restrain psittacines or passerines; use thin gloves wherever possible; elbow length gloves can be useful for large, aggressive birds
Harness and other devices	To restrain birds to minimize struggling and facilitate procedures	The "Guba" is a design used to restrain falconers' birds, while "swan jackets" have been designed for restraining large waterfowl
Hoods	To cover the head of a diurnal bird to minimize struggling and facilitate procedures	A standard method of quieting and restraining falconers' birds and can be used to advantage in many other species; a loose cloth bag or sock can be used if a well-fitted hood is not available; falconry hoods must fit the bird well
Padded examination table	Birds should be examined on a soft surface to prevent trauma as the bird struggles	Blankets, towels, covered cushions, or foam may be used
Padded sheets of plywood or Plexiglas	Such shields are used to allow handlers to move large, fractious ratites	Handlers should be prepared to withstand solid blows to the board
Paper or cloth towels	To wrap around birds to facilitate handling and permit restraint	Paper towels are better because they can be discarded after use; material should be washed and autoclaved between each bird, as above
Safety glasses	To protect the face and eyes of the handler	May be considered when dealing with aggressive birds such as storks or herons
Stanchions or "plucking boxes"	Used to restrain ostriches for feather harvesting	

Modified from Cooper JE: Caged and wild birds. In Anderson RS, Edney AT, editors: *Practical animal handling*, Oxford, UK, 1991, Pergamon Press, Oxford.

Birds are highly sensitive to stress, and incorrect handling can cause:

- Temporary or permanent limb paresis or paralysis
- Hyperthermia
- Fractures of legs or wings
- Skin lacerations, bruising, and feather loss
- Luxation of the tibiotarsal bones
- Dislocation of the cervical vertebrae
- Compression of the flexible trachea and internal organs
- Progression of a disease process and even death.

Before attempting to catch and handle small and obviously sick birds, it is wise to warn the owner that there is a risk that the bird may suddenly die of heart failure. Birds that are brought in from the wild are unaccustomed to humans, and because of this, handlers must consider the effects of stress and minimize restraint time.

TRANSPORT

Birds should be transported in a secure, darkened, and well-ventilated container. Containers should have ventilation holes low on their sides (to minimize light at eye level) and have a new piece of carpet, rubber matting (which can be disinfected and reused), or similar material on the floor to allow the bird adequate grip. Straw, peat, or hay should be avoided as bedding because of the risk of contamination with spores of *Aspergillus* sp. The container must be free of sharp edges or protrusions that could cause injury. Padding the ceiling and sides of a container can reduce injuries. The bird should be maintained at an ambient temperature of 21.1° to 26.6° C (70° to 80° F) and should never be left unattended. The size of the container should not permit wing flapping but must allow the bird room to stand up in a natural position and turn around.

The transport requirements of birds vary greatly among different groups. Pigeons and smaller waterfowl can be carried in small disposable cardboard boxes. Long-legged birds such as flamingos must not only be able to stand up in transit but must also have their bodies supported ventrally, for example, by a sling, to prevent collapse. Swans and large geese may be restrained in purpose-made restraints. Free-living raptors can be transported in small, strong cardboard boxes. Falconers' birds can be transported hooded on cadges or on the fist of the falconer. Passerines and psittacine birds can be transported in their

TABLE 4-3 Methods of Handling and Restraint of Various Groups of Birds

Bird Group	Handling Technique	Additional Comments
Small passerines	Place the head between two fingers so that the body rests in the palm of the hand, or it can be restrained by holding the head gently between the thumb and first finger	May stab or bite with beak; thin gloves will help to minimize effect; elastic band or sticky tape may be used to seal beak
Large passerines	Hold with two hands, round wings	
Small psittacines	As for small passerines	
Large psittacines	As for large passerines	
Small and medium-sized birds of prey	As for large passerines; falconry hoods are very helpful in blocking visual stimuli and have a calming effect	Raptors will often grasp air with their feet when restrained and it is important not to allow them to puncture themselves with their talons (use foot wraps)
Large birds of prey	As for small and medium birds of prey; can use a cloth towel to grasp round wings; alternatively catch while perching by seizing legs and quickly turning the bird upside down: the wings will usually be extended but can be readily folded into the body	Use heavy gloves and wear appropriate falconers' equipment; vultures may regurgitate food from their crop when handled
Pigeons and doves	As for small and large passerines; pigeon fanciers prefer to hold birds with one hand around the base of the tail	Rarely bite or scratch; inclined to defecate during handling; feathers easily lost
Small waterfowl	Can be restrained by their wings or by grasping the back and wings and using the thumb and fingers to restrain the feet	Heavy-bodied species should not be carried by using the wings or feet alone
Large waterfowl	The base of both wings should be grasped with one hand while the other hand and arm supports the body; these birds may be carried under one arm, with their head facing to the back; the arm is wrapped around the wings and a hand is used to support the body and control the legs	Some geese have sharp claws and powerful legs, causing scratches; swans and geese may flap wings, which may deliver painful blows and prove difficult to restrain; these species should not be carried by the wings alone because temporary or permanent brachial paralysis may ensue
Gamebirds	In the larger species the base of the wing is fixed with one hand and the legs are controlled with the other hand; the abdomen should be supported from below	Never restrain gamebirds by the feathers alone—the whole body must be secured to prevent a shock molt; cocks with spurs can injure handlers and the beak also serves as a weapon
Waders, herons, storks, flamingos, cranes	As above depending on size; grasp neck of herons, storks, and cranes first to restrain head; when the bird is picked up, the legs should be extended parallel to the ground; it is important to place one or two fingers or a rolled towel between the hocks to prevent injuries	May stab with beak; protect eyes and exposed skin; handle with care because long legs and wings are prone to damage; storks and cranes have strong legs and will kick; the margins of the blunt bill of the flamingo are serrated and can lacerate fingers or arms; storks may regurgitate food when handled
Bustards	As above depending on size	Rarely bite or scratch; inclined to defecate during handling; feathers easily lost; handle with care because long legs are prone to damage, including fractures; some species have strong legs and will kick
Gulls, terns, petrels, shearwaters	As above depending on size	Gulls very likely to stab with beak; always use an elastic band; all of this group inclined to vomit during handling and fulmars may regurgitate oil
Ratites	Small or immature ratites can be caught by grasping the legs firmly and picking the bird off the ground; large ratites are handled by catching the head and pulling it forward and down until the vision of the animal is blocked; additional handlers grasp the wings from the sides and place pressure in a downward direction to prevent the bird from jumping; more steady pressure will cause the bird to sit down	Darkness (hooding or subdued lighting) is one of the best restraint techniques, which may be used in ratites of all sizes; manual restraint of ratites is potentially dangerous to both the handler and the animal; ratites can react rapidly when frightened and can jump and flail with their legs; male ostriches can be more dangerous during the breeding season
Hummingbirds	Most easily restrained and transported wrapped in cloth jackets with their heads protruding so that they can be fed	
Penguins	*Spheniscus* penguins and most crested penguins should be restrained by grasping them suddenly by the neck and hoisting them into the air at the length of the arm; the feet can be controlled with the other hand; from this position the bird can be supported on the lap of the handlers and examined thoroughly	These birds can pummel an aggressor with their powerful flippers, which can be painful

Modified from Cooper JE: Caged and wild birds. In Anderson RS, Edney AT, editors: *Practical animal handling*, Oxford, UK, 1991, Pergamon Press, Oxford.

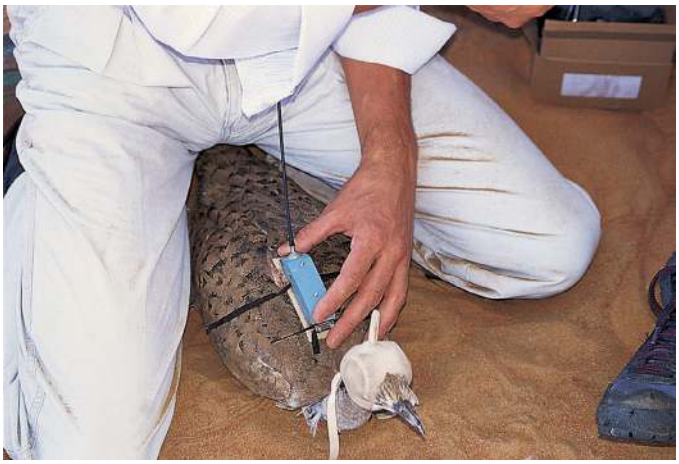


FIGURE 4-22 Free-living birds, such as this houbara bustard, may need to be restrained to assist field biologists when placing satellite transmitters. (Courtesy Dr. J. Samour.)

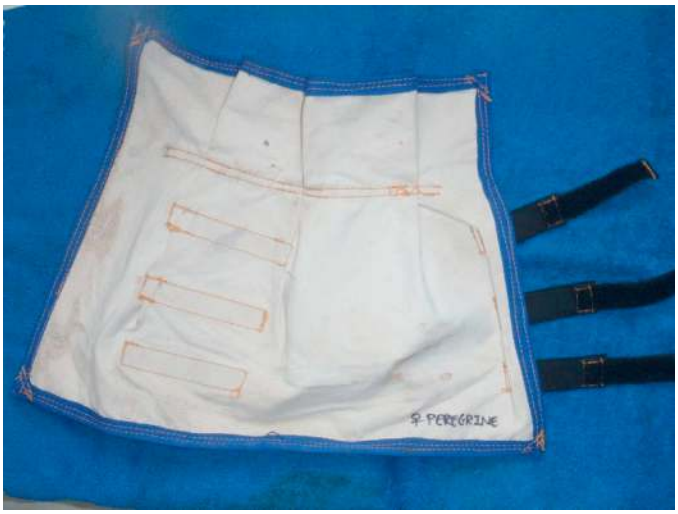


FIGURE 4-24 A casting jacket.

FIGURE 4-23 Correct handling of a flamingo.

FIGURE 4-25 Falcon in a restraint or casting jacket being measured.

cages but should have the water dish emptied. Toys and all but one perch should be removed, and a blanket can be placed over the cage to provide darkness. Crates for transporting wildfowl need to have good ventilation and a receptacle for water (if the trip is for more than a few hours). Adult ratites can be transported in a shipping crate or an enclosed horse trailer (Fig. 4-26, *A-C*), while young ratites can be transported in pet carriers. Transporting at night is recommended for birds such as ratites because they are calmer and subject to less thermal stress. Further specialized information on handling different species of birds may be gleaned from the texts listed in the bibliography.

Containers that have been previously used to transport birds must be cleaned and disinfected before reuse. Wooden crates are not ideal for transporting birds because they are difficult to disinfect. Carrier specifications and labeling guidelines for international air transportation of birds are set by the International Air Transport Association (IATA, 2015; www.iata.org/; Figs. 4-27 and 4-28). Extremes of environmental conditions can cause morbidity and mortality during transportation. Hyperthermia is probably the greatest risk to birds when they are transported and in the care of transportation companies. Temperature-monitoring data loggers (e.g., DS1921 iButton, Revolution Education Ltd.; www.rev-ed.co.uk) can readily be obtained and fitted to transportation boxes (Fig. 4-29, *A* and *B*). This enables retrospective assessment of the environmental conditions that occurred during transportation (Fig. 4-30). Such information can be vital if investigating mortality incidents, but is also invaluable in demonstrating the danger points during the transportation process that may need modifying. As an example, when I was involved in shipping falcons from the UK to the Middle East the use of data loggers showed that the loading and unloading periods were the times when environmental temperature was uncontrolled and the birds were at risk for developing hyperthermia. Liaison with the airlines enabled better care of the transported falcons at these danger points.

FIGURE 4-26 (A-C), Sequence of images showing the loading of an ostrich into refrigerated transport trailer.

FIGURE 4-27 Correct labeling of a transport box with a "this way up sticker."

FIGURE 4-28 Correct labeling of a transport box with a "live animal sticker."

FIGURE 4-29 **(A)** and **(B),** Series of iButton images showing size and location on transport box.

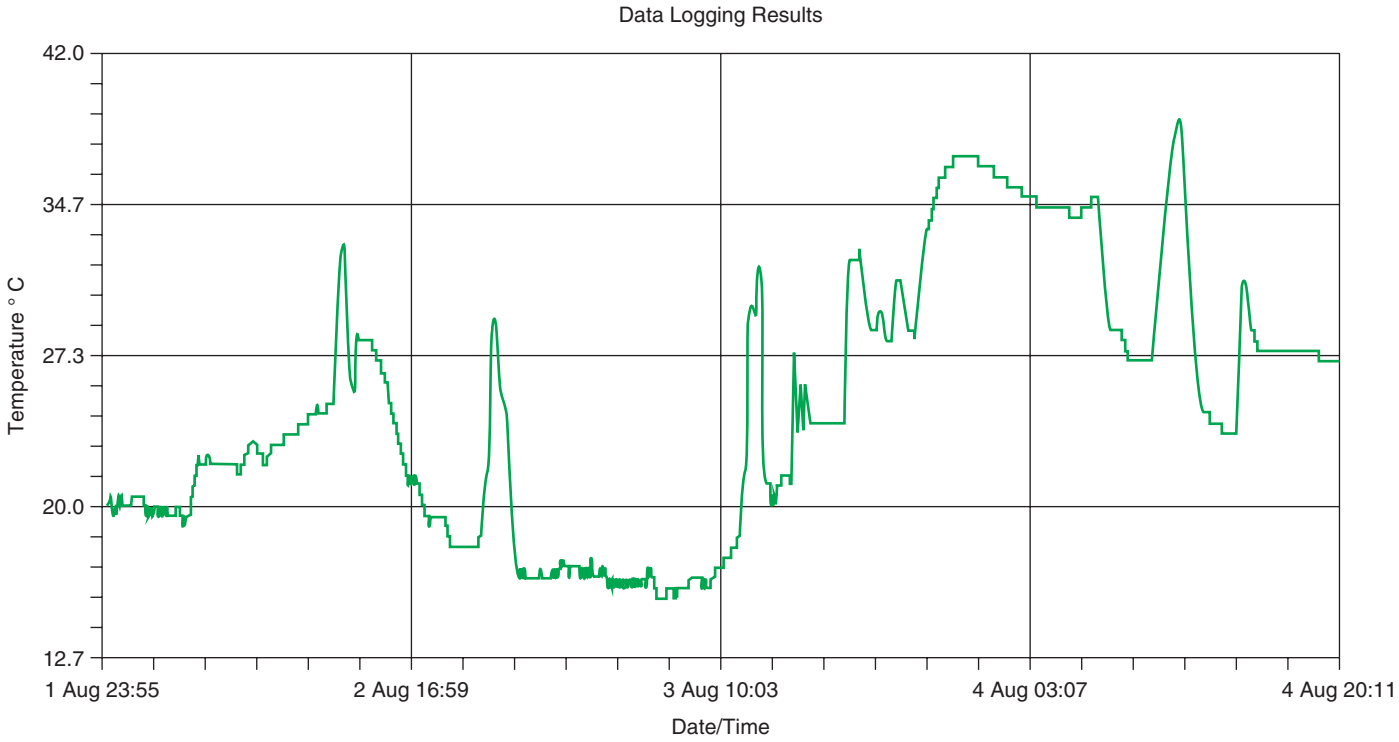


FIGURE 4-30 Temperature data logger results from the transport box of a falcon sent by air from the UK to the Middle East.

REFERENCES

Cooper JE: Caged and wild birds. In Anderson RS, Edney AT, editors: *Practical animal handling*, Oxford, UK, 1991, Pergamon Press.

Harris JM, Brown B: *A restraint and transportation device for raptors*, Pittsburgh, PA, 2003, Proceedings of the Association of Avian Veterinarians.

IATA: *Live animal regulations*, Geneva, 2015, International Air Transport Association. http://www.iata.org/index.asp.

FURTHER READING

Cooper JE, Hutchinson MF: *Manual of exotic pets*, Cheltenham, UK, 1985, British Small Animal Veterinary Association.

Fowler ME, Miller E: *Zoo and wild animal medicine*, ed 5, Philadelphia, 2003, Elsevier Science.

Girling S: Avian handling and chemical restraint. In *Veterinary nursing of exotic pets*, Chichester, UK, 2013, Wiley-Blackwell.

Jensen JM: Ratite handling and restraint. In Fowler ME, editor: *Zoo and wild animal medicine, current therapy 3*, Philadelphia, 1993, WB Saunders.

Mouser M: Restraint and handling of the emu. In Tully TN, Shane SM, editors: *Ratite management, medicine and surgery*, Malabar, FL, 1996, Krieger Publishing.

Pizzi R: Examination, triage and hospitalisation. In Chitty J, Lierz M, editors: *Manual of raptors, pigeons and waterfowl*, Cheltenham, UK, 2008, British Small Animal Veterinary Association Ltd.

Ritchie BW, Harrison GJ, Harrison LR: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Sonsthagen TF: *Restraint of domestic animals*, Goleta, CA, 1991, American Veterinary Publications.

Wade JR: Restraint and handling of the ostrich. In Tully TN, Shane SM, editors: *Ratite management, medicine and surgery*, Malabar, FL, 1996, Krieger Publishing.

White J: Raptor restraint, *J Assoc Avian Vet* 4:91–92, 1990.

5

The Clinical Examination

GENERAL CONSIDERATIONS

Robert Doneley

Regardless of the reason for presentation, the clinical examination of the avian patient is the single most important aspect of avian medicine and surgery. Without a detailed and thorough clinical examination, the clinician has no basis for diagnostic testing or treatment. Junior clinicians are more likely to perform a complete physical examination, but not understand the meaning of all the biologic information they collect. Conversely, more senior clinicians may rely too much on experience and neglect the rest of the examination, focusing only on what has been identified in the history or distant examination. To minimize the effect of both behaviors, it is important that a thorough examination, following a set pattern, is conducted (Fig. 5-1).

Underpinning the importance of the clinical examination is an understanding of the *masking phenomenon*—the innate desire of a bird to hide, or mask, signs of illness from potential predators (including their human carers). Wherever possible, a sick bird will assume a "normal" appearance, even if only for a few moments. This instinctive behavior has been a part of avian behavior for millions of years, and a few generations of captivity will not alter this. A bird that can no longer mask signs of illness is severely unwell and starting to decompensate.

To complicate this, many bird owners are not familiar with early signs of illness in birds—the subtle changes in a bird's behavior, appetite, droppings, and vocalization—that indicate the bird is unwell. When these two factors are combined with a common attitude of many bird owners—"Wait a couple of days and see if it gets better"—many birds presented to veterinarians are critically ill. This makes a thorough clinical examination even more important.

It is important that an understanding of the masking phenomenon is communicated to reception and nursing staff in the veterinary clinic. Often the first contact with a veterinary clinic is via the receptionist, and it is important that reception staff are trained to recognize when a bird needs to be seen urgently, and then communicate this sense of urgency to the client. Similarly, nursing staff must be trained to see past the bird's ability to mask clinical signs and recognize changes in a bird's condition while it is hospitalized (Fig. 5-2).

When an appointment has been made, reception staff need to ensure that the client brings in any relevant information, including digital photographs of the bird's cage or aviary, previous medical records, current medications, details of diet, and breeding records. In a critically ill patient, having this information readily at hand can make a significant difference in short-term outcomes and decision making. If possible, the bird should be brought into the clinic in its own cage, allowing the clinician to better assess the husbandry and nutrition provided by the bird's carer.

On initial presentation the clinician needs to quickly ascertain if the bird requires urgent supportive care—warmth, fluids, oxygen—before obtaining a history or performing a physical examination (Fig. 5-3). A bird that is fluffed and sitting on the floor of the cage with its eyes closed, or is severely dyspnoeic, is a patient in need of urgent attention. (Well-trained nursing staff will show their mettle in this situation, often providing urgent supportive care while the clinician discusses details with the client.) Often the single most important supportive care that can be given to a critically ill patient is active warming and oxygen before any other intervention is attempted (see Chapter 8).

However, most of the time the patient does not require such urgent care, and the clinical examination can proceed in a methodic and thorough manner. The hierarchy of different parts of the examination can be illustrated in a diagnostic pyramid (Fig. 5-4). As demonstrated in this diagram, a detailed history of the patient, followed by the reason for presentation, form the foundation stones of the diagnostic process and should never be overlooked, even in critical cases. This is followed by a physical examination—distant and then close—before diagnostic testing is used to confirm a suspected diagnosis. As each stage is performed, the clinician's "Index of suspicion" narrows, with the list of differential diagnoses shortening as each stage is completed.

EXAMINATION ROOM EQUIPMENT

Appropriate equipment for use in the examination room (Fig. 5-5) 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), or a container in which to weigh smaller birds. Larger scales may be needed to weigh large birds such as raptors or waterfowl.
- A training perch for the bird to perch on while being examined.
- Clinical equipment including a stethoscope, a focal light source, magnifying loupes, needles and syringes, blood collection bottles, and culture swabs.
- Alcohol for wetting feathers down for a closer examination of the skin and underlying fat and muscle.
- Treats to reward pet birds and make the experience more enjoyable (or at least, less stressful).

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 (Fig. 5-6).

Ensure that the room is escape-proof and clinic staff will not enter the room unexpectedly. Avoid stressful sights and sounds, such as dogs, cats, and other potential predators.

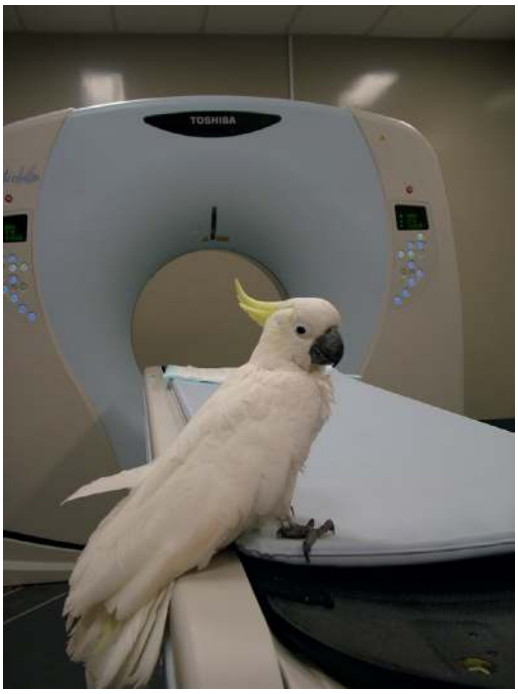


FIGURE 5-1 Advanced imaging techniques, such as computed tomography, are an adjunct to a thorough physical examination rather than a replacement for it.

FIGURE 5-3 In some cases supportive care, such as aggressive heating, is needed before moving on to a physical examination.

FIGURE 5-2 Birds will mask signs of illness as long as possible. This parrot, despite its apparently good appetite, died shortly after this photo was taken.

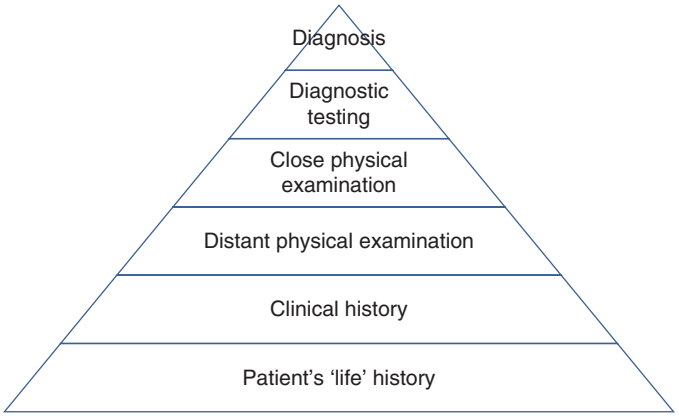


FIGURE 5-4 The diagnostic pyramid.

FIGURE 5-5 The avian examination room, not very different from that found in any veterinary clinic, has everything needed close at hand. There is also a station with a microscope that is fitted with a camera connected to a medical monitor to show clients parasitology and cytology findings. This is a highly popular educational system among clients and should also form part of public awareness programs. (Courtesy Dr. Jaime Samour.)

FIGURE 5-6 When handling birds, it is often wise to remember that many are closely bonded to their owners; heavy-handed restraint of these birds is often inappropriate.

MEDICAL RECORDS

Robert Doneley

Medical records are maintained for three reasons: to preserve a database of previous clinical findings for future reference and comparison; to prompt the clinician's memory on details when a patient has not been seen for weeks, months, or years; and to demonstrate a clear (and defensible) thought process when the records are reviewed by another party, whether they be another clinician or a court of law. They should be generated at the time of clinical examination or shortly after, and in such a manner that the record is regarded as accurate and trustworthy.

The production and maintenance of comprehensive, legible records:

- promote good medicine,
- establishe and demonstrate a valid veterinarian-client-patient relationship,
- add value to a practice by demonstrating goodwill,
- are an effective public relations tool (for clients and other veterinary practices), and
- provide a medical defense when needed.

Whether the record is handwritten or computerized, the use of a template encourages a methodic and organized approach to the clinical examination, assisting the clinician in both organizing his/her thoughts and ensuring that nothing is overlooked. Templates such as SOAP allow specific templates to be used within a general template, adding to the thoroughness of the examination and its documentation.

The SOAP template is becoming universally accepted and many recent (and not so recent) graduates are familiar and comfortable with the concept of its use. The acronym SOAP stands for:

- ***Subjective:*** What the client tells the clinician. A template under this heading could include the patient's life history and the clinical history of the presenting problem(s).
- ***Objective:*** What is seen and measured by the clinician. It includes physical examination findings and the results of laboratory tests and diagnostic imaging. Again, this can be recorded on a template as a checklist.
- ***Assessment:*** The collation and interpretation of the information obtained in the previous two steps. It may include a list of identified problems, the differential diagnoses, and a tentative or confirmed diagnosis.
- ***Plan:*** The actions that need to be taken to resolve the problem. It may include further testing, medications, surgery, client communications, discharge instructions, and follow-up appointments.

After an initial record is created on the first visit, a separate record should be created for each subsequent day in the hospital, any revisits, or any visits for other problems. Some aspects of previous records (e.g., weight gains or losses, appetite and thirst, and changes in body condition) should be referenced in subsequent records.

It is also important that records contain communications between the client and the clinic, including recommended treatments that were declined by the client, telephone calls made by either party, cost estimates given to the client, and notification of patient progress and laboratory results to the client.

The medical record should also contain copies of laboratory and radiology reports or provide information as to where these records are held.

Local veterinary boards should be consulted regarding the required content of medical records and the length of time inactive records have to be kept.

THE PATIENT'S 'LIFE' HISTORY

Robert Doneley

Before a clinician can begin to understand a disease problem, he/she must first understand the patient itself. Without knowing the bird's species, age, sex, diet, husbandry, reproductive history, and previous health problems, it is impossible to determine where a problem originated and how to prevent it from recurring.

SIGNALMENT

There are many different species of birds presented to veterinary clinics, with a large deal of geographic variation (e.g., in Australia, macaws and African grey parrots *[Psittacus erithacus]* are uncommon, although Australian species are very common; in other countries the reverse is true). Ideally the clinician should have a good working knowledge of commonly kept species in the area, as it is not unusual for the client to be unaware of some basic facts about their own bird (such as species, sex, and age).

The patient's signalment can provide valuable clues to the reason for presentation. Some species of birds have specific dietary and husbandry requirements or are predisposed to certain diseases. They may have certain behavioral characteristics or physical characteristics not seen in other species. Knowing the species, and its associated unique features, can offer the clinician vital clues to likely problems (Fig. 5-7).

The age of the bird can offer other clues. As a general rule, juvenile birds, which are rapidly growing but often not fully immunocompetent, are more likely to suffer from infectious diseases and nutritional deficiencies, although adult birds are more likely to suffer from neoplasia, chronic malnutrition, and degenerative conditions such as cardiovascular disease, arthritis, and hepatic lipidosis (Fig. 5-8). These age-related differences offer the clinician vital clues.

Knowing the sex of a bird can be vital, as many conditions such as behavioral problems and reproductive-related diseases can be linked to the bird's sex. For example, coelomic hernias are rare in male birds but are relatively common in females; yolk-related peritonitis is obviously only seen in hens; cocks do not become egg-bound. Unless the species is sexually dimorphic, it may be unwise to accept the owner's assertion of the bird's sex unless the owner has proof of sex identification such as a history of egg laying or a certificate of sex identification (surgical or DNA) that can be correlated with the bird.

FIGURE 5-7 To the untrained and unfamiliar clinical eye, the juvenile gyr (*Falco rusticolus*) falcon depicted in this image is in the agonal stage of a terminal sickness and is close to collapse from its perch. In reality, the falcon is just sleeping. Most falcons tuck their head at the back between the cover feathers between the scapular areas when they sleep. However, some gyr falcons very often bend their neck downward and sleep in this position. (Courtesy Dr. Jaime Samour.)

FIGURE 5-8 Knowing the normal. This cockatiel's (*Nymphicus hollandicus*) owner thought the feathering was due to aging. In fact, it is associated with chronic liver disease and possibly hypothyroidism.

Being able to accurately identify the bird, particularly in a collection, can be important for many reasons (Fig. 5-9). Ideally the bird will be microchipped; alternatively, it may have a leg band (preferably a closed ring rather than a split ring). If there is no means of identification, this fact needs to be placed on the patient's record and the owner counseled about the advisability of permanent identification.

THE BIRD'S ORIGINS

The length of time the bird has been with the client can give clues to the bird's problems and its source.

Birds that have been in the owner's possession for many years, with no recent exposure to other birds, are less likely to have contracted an infectious disease than a recently obtained bird that may have had close

FIGURE 5-9 In a falcon hospital setting, in common with any other avian clinical facility, the attending reception staff has to be familiar with the species and the approximate age of the different patients. The falcon in the photograph offers a significant challenge as it is technically impossible to determine the specie and the age as the plumage is completely white. However, this is not a prize-winning individual but the product of unscrupulous falcon dealers who have mastered over the years the ability to bleach the plumage and sell the falcons as unusual and unique specimens at exorbitant prizes. (Courtesy Dr. Jaime Samour.)

contact with other birds and, as such, have possibly been exposed to infectious diseases.

With experience, the clinician will be able to identify "problem sources" of birds in the local area (i.e., a certain breeder or a pet shop). Developing a working knowledge of the quality of the sources of pet birds in the local area can be a key element of patient evaluation. Wild-caught birds, although less common in recent times, still appear occasionally in the pet market or avicultural collections. The behavior and diseases of such birds may well be linked to their source.

The health of juvenile birds is dependent, to a large extent, on the health and nutrition of the parents. When assessing a juvenile bird, it is often of great assistance to know if the bird was parent-reared or hand-reared. If parent-reared, the health and diet of the parents should be ascertained. If hand-reared, the experience and skill of the person doing the hand-rearing, as well as the diet been used, are important elements of the history. It is also important to know if the bird was hand-reared in a collective nursery, meaning chicks from different sources are being reared in the one facility (with a higher probability of the spread of diseases such as polyomavirus and chlamydia), or in a closed nursery.

HUSBANDRY

Aspects of captive bird husbandry are discussed in Chapter 1. Clinicians should be familiar with common husbandry methods and the terminology associated with them. Knowing the difference between a full-flight aviary and a suspended-wire aviary gives the clinician a good background on how birds are kept and engenders a better relationship between the clinician and the client.

Questions that need to be asked regarding husbandry include:

- How has the bird been managed? Is this an aviary bird, a companion bird, a racing bird, a hunting bird, or a zoologic specimen? An assessment of a companion bird's husbandry must include the cage, the environment around the cage, and the bird's interaction with

its environment. If the patient is a racing or hunting bird, its training regimen must be assessed.
- Ascertain whether the cage in the examination room is the bird's permanent cage, or simply a transport cage. If the latter, ask the owner to describe the permanent cage, or to bring along photographs or videos of it.
- If this is a pet bird, where is the cage located in the house? Is it exposed to toxins such as burning nonstick cookware, cigarette smoke, or certain household plants? Does the bird get any privacy? Does it feel secure where it is positioned? Is it kept isolated, away from family activities? Does it get a good night's sleep, or is it forced to stay up with its owner watching television all night? Does the bird get access to direct, unfiltered sunlight on a daily basis? If the bird is kept outside, is it safe from predators and from vectors of diseases such as sarcocystis?
- Does the bird come out of its cage? How long does it have outside the cage each day? Are its wings clipped? Is it supervised when out of the cage? Does it interact with other animals and birds?
- Does the client have other birds? Any other pets?

Often the answers to these questions will generate further questions, or the answers will be revisited as more information comes to light.

NUTRITION

Underlying many health problems in birds is a common thread of malnutrition. For many generations bird owners have accepted as fact that companion and aviary birds eat seed, and that is all they need. Parrots, as with many other animals, have a preference for 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." Raptors, pigeons, finches, and other species face their own peculiar challenges. Nutrition and nutritional requirements are discussed in Chapter 3.

It is important to ascertain:
- What does the owner feed the bird and what does the bird actually eat? There is often a major difference between the two. The answer to the first question reveals what the owner knows about their bird's nutritional requirements; the answer to the second is often a vital clue to the bird's health status. The clinician needs to be aware that there are major species differences in dietary requirements with, for example, some species having a higher requirement for fat than others (e.g., macaws versus Amazons, black cockatoos versus white cockatoos). There is no one diet to suit all avian species, any more than there is one diet to suit all mammalian species.
- How much food has been consumed? (Some birds, on an excellent diet, will still eat too much and gain excessive weight.)
- Is the food prepared fresh daily?
- Are dishes cleaned each day?
- Does the bird dunk food into its water dish, creating a nutrient-rich broth ideal for bacterial contamination?
- Does the bird get any treats, such as food off the owner's plate?
- Are vitamin and mineral supplements being offered?

Pediatric cases open another spectrum of questions. What hand-rearing formula is being used? How is it prepared, and are the manufacturer's recommendations being followed? Is anything extra being added to an already balanced diet? At what temperature is it being fed? How much is being fed, and how often? How is the chick being fed—syringe, crop tube, or spoon? How are these utensils cleaned and disinfected? Pediatrics are discussed in Chapter 15.

BEHAVIOR

A behavioral history is becoming increasingly more important as pet birds move out of their cages and more into their owners' lives. Just as countless dogs and cats are euthanized every year because of behavioral problems, many birds suffer the same fate, or are transferred from household to household, for the same reasons. Bird behavior is determined to a large extent by the interaction between the bird, its owners, and its environment; questioning must therefore focus on these areas. This is discussed in more detail in Chapter 2.

PREVIOUS MEDICAL HISTORY

It is important to know what the patient's medical history is. Has the bird been ill before? Who evaluated it, what did they diagnose, and how was it treated? Has it had remedies supplied by pet shops or breeders? Has the bird been presented for a second opinion or a specialist referral?

It is also important to know if other birds in the household or collection have been unwell, or if there have been any recent deaths.

If appropriate, request permission to obtain copies of medical records from the previous (or referring) veterinarian.

THE CURRENT PROBLEM

Robert Doneley

The reason the client has brought their bird to the veterinarian is paramount to that client and often all they wish to discuss. The clinician must control this discussion, as otherwise vital clues in the patient's life history will be overlooked. But when the clinician is satisfied that they understand how the bird lives, it is necessary to understand why the bird has been presented.

Questions that must be asked include:
- Why has the bird been presented? It may have been presented for a purchase examination, reproductive evaluation, poor performance, injury, or illness. This needs to be determined and then details of the problem elicited.
- Are other birds affected? If the presented bird is ill, it is important to know if other birds are similarly affected, indicating the possibility of an infectious disease. It should not be assumed this is the case as nutritional problems can be widespread through a collection.
- When did the problem start? Bearing in mind the discussion earlier regarding the ability of birds to mask signs of illness, it needs to be determined whether this problem is acute or chronic. In turn, this helps shape the clinician's problem list and differential diagnoses.
- What has the owner done about the problem before presentation? Knowing if the client has attempted treatment, either by themselves or at the direction of another veterinarian, and how the bird responded to that treatment will provide the clinician with a great deal of information that will affect their decision-making process.
- What effect is the problem having on the bird's appetite, thirst, droppings, and behavior? Changes—either an increase or decrease—in these functions of life can provide more clues to the clinician to assist in determining a diagnosis and treatment plan.

THE PHYSICAL EXAMINATION

Robert Doneley

THE DISTANT EXAMINATION

Although a history is being taken, the clinician should take the opportunity to perform a distant examination of the patient (i.e., without

actually touching the bird). Allowing the bird to "settle in" to the examination room and relax allows the clinician to observe the bird's behavior when it isn't masking clinical signs, examine the bird's droppings, and assess its husbandry by looking at the cage. There is a lot of information obtainable in a distant examination; the clinician simply needs to take the time to observe and interpret.

THE CAGE

Aspects of captive bird husbandry are discussed in Chapter 1; the clinician, if unfamiliar with this aspect of avian medicine, should read this carefully before examining birds. The author finds the cage to be a valuable insight into the owner's understanding of husbandry.

If it is a carry cage, rather than the bird's living cage, an idea of husbandry can be obtained by viewing the construction and cleanliness of the cage. Food remnants, dried fecal material, and other stains within the cage reflect poorly on the owner's concepts of bird management.

If it is the bird's living cage, it should be carefully assessed, not just for an indication of the owner's standards of husbandry, but also to obtain clues such as exposure to potential toxins (e.g., metallic toys), foreign bodies (e.g., cotton fiber toys), and reproductive cues (e.g., nest boxes and other potential nest sites). Examining the contents of the feed containers will provide evidence as to what the bird actually eats (in contrast to what the owner states it is fed). Other evidence such as molted feathers, blood stains on perches or the floor, vomitus, and molding food remnants will give the clinician more clues as to potential problems.

THE BIRD

After a few minutes of casual observation, the sick bird often reverts to behavior and clinical signs it may have tried to mask when first placed on the examination table. In particular, the clinician should look for:

- "Sick bird look": Eyes closed, feathers fluffed, bilateral wing droop, and the head turned back over the shoulders. These are nonspecific indicators of ill health (Fig. 5-10).
- Dyspnea: Mouth-breathing, exaggerated sternal lifting, tail-bobbing, audible respiratory noises, and panting (Fig. 5-11)
- Lameness, either bilateral or unilateral
- Dyschezia (straining to defecate)
- Staining of the feathers on the ventral tail and around the vent
- Unilateral or bilateral wing droop
- Feather loss, color change, or untidy plumage
- Evidence of bleeding
- Obvious tumors and masses

THE DROPPINGS

Birds' droppings are made up of three components: feces, urates, and urine. 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 range from brown to green. The urates should be a crisp white and slightly moist. Be aware that droppings passed more than a few hours previously will have greenish urates because biliverdin leaches out of the feces. The urine should only extend a couple of millimeters past the dropping. Do not confuse true polyuria with so-called "excitement" or "stress" polyuria: the excess urine produced by an excited or nervous bird. Lorikeets, because of their liquid diet, will produce large amounts of urine, which should not be mistaken for polyuria. A close examination of the droppings is a valuable starting point to a clinical examination (Fig. 5-12, *A-F*).

Some abnormalities commonly encountered include:

- Diarrhea: unformed fecal portion
- Undigested food in feces
- Very bulky droppings indicate maldigestion, malabsorption, reproductively active hens, abdominal growth, or pelleted diets
- Melena or very dark droppings can indicate anorexia or intestinal hemorrhage
- Malodorous droppings are often associated with bacterial/fungal overgrowth
- Green urates (biliverdinuria) are often indicative of liver disease
- Pink/red urates (hematuria/hemoglobinuria) are seen in cases of renal disease often associated with lead poisoning, especially in Amazons and galahs
- Yellow urates can be associated with anorexia
- Orange urates indicate that a vitamin B injection may have been given in the last few hours
- Thick, pasty urates are seen in dehydrated birds

FIGURE 5-10 The "sick bird look"—eyes sunken and half closed, feathers fluffed, sitting quietly—is a general sign of illness.

FIGURE 5-11 This severely dyspnoeic duck requires urgent stabilization before a clinician can attempt a physical examination.

FIGURE 5-12 **(A),** Normal droppings from a healthy parrot, showing formed feces, normal white urates, and a small amount of urine. **(B),** Hematuria is a sign of severe renal disease, often suggestive of lead toxicosis. **(C),** Biliverdinuria and polyuria suggestive of liver disease **(D),** Large, bulky droppings are associated with a space-occupying mass in the coelom, such as egg laying. **(E),** Melena suggests severe gastrointestinal diseases. **(F),** Undigested seed passed in the droppings indicates dysfunction of the proventriculus and ventriculus.

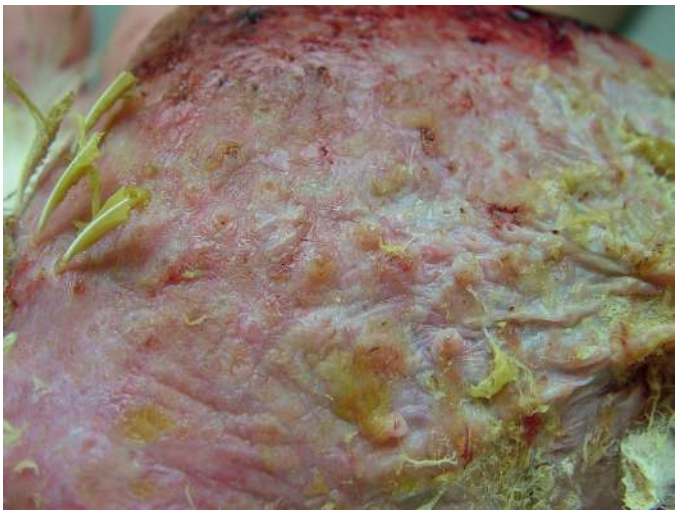


FIGURE 5-13 Dermatitis and folliculitis in a cockatoo after a dog attack.

FIGURE 5-14 Bruising of the unfeathered skin on the mandible of a macaw caused by restraint.

- Polyuria
- Anuria
- Discolored urine has similar causes as discolored urates
- Fresh blood indicates either a cloacal problem or oviductal disease

A thorough, systematic physical evaluation of the patient is essential to obtaining clues about the bird's problem and diagnosis (Fig. 5-13). Clinicians should develop a thorough examination protocol that they are comfortable with and use it for every patient, regardless of the reason for presentation.

HANDLING AND RESTRAINT

Handling and restraint of birds is discussed in detail in Chapter 4. Some salient points to be considered are:

- The use of heavy gloves is unacceptable when working with small birds such as parrots, and usually inadvisable in larger birds. Instead, if needed, the clinician should use a small hand towel to gently envelop and then restrain the bird.
- It is often inappropriate to use heavy-handed restraint on a companion bird. Having the owner remove the bird from the cage and place it on a perch for examination is much less stressful for the bird, the owner, and the clinician (Fig. 5-14).
- As the oils on human skin can be detrimental to the feathers of many species, a light dusting of talcum powder on the clinician's hands is appropriate before beginning an examination. This is particularly necessary for those birds that do not produce powder down (e.g., Amazons, eclectus parrots, and lorikeets). When handled frequently without talcum powder, the green feathers on these birds begin to acquire a black discoloration that can become quite unsightly.
- At all times the clinician must be aware of the bird and how it is handling the stress of restraint and examination. 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.

WEIGHT RECORDING

A bird's bodyweight is an often reliable guide to its physical condition, especially when recorded on each visit or each day when hospitalized

FIGURE 5-15 Weighing the patient is a key element of every physical examination. This cockatiel (*Nymphicus hollandicus*), weighing 140 g, is morbidly obese and the physical examination will be partly focused on problems associated with this condition.

(Fig. 5-15). It also allows the clinician to accurately calculate drug dosage rates and to monitor the patient's response to therapy. Over a period of time, the clinician will also develop a working knowledge of expected body weights for different species. It should be noted that bodyweight is not an absolute indicator of body condition—there are both small and large specimens of every species that fall outside the normal range, despite being healthy—so it needs to be combined with an assessment of the bird's body condition (see subsequent information).

The bird's weight should be recorded to the nearest gram. A good quality digital scale, readily available in many electronics or

kitchenware stores, is an essential item in the examination room. Larger birds can be weighed by having them stand on a T-perch mounted on the digital scale. Smaller birds, such as budgerigars, canaries, and finches, may be placed inside a metal or plastic container similarly mounted on the scale.

The patient's weight must be recorded on its medical records in order to demonstrate to clients their pet's weight gain or loss, and to provide part of that patient's minimum database.

BODY CONDITION SCORING

Body condition scoring, as used in small animal medicine, has been applied to birds by the palpation of the pectoral muscles and allocating a numeric score based on the muscle and fat coverage over the sternum. This system fails to take into account two significant attributes of birds:

- There are species differences in the amount of pectoral musculature, especially between flighted vs nonflighted birds.
- Fat is not only stored over the pectoral muscles; it is deposited in the coelom, over the flanks, around the thoracic inlet and the back of the neck, and on the back near the tail.

Any system of body condition scoring needs to take these attributes into account; it must also be repeatable among different clinicians; be consistent among different bird species; and be universally accepted. At the time of writing, several systems have been proposed but none are accepted by all clinicians. Until that is achieved, the author prefers to use a verbal description (e.g., thin, obese), rather than a numeric score, to describe the body condition of a bird (Fig. 5-16).

AUSCULTATION

When to auscultate is the prerogative of the clinician, but may be better performed before the bird is handled for too long and becomes tachycardic from excitement or stress. The heart rate is usually rapid, although that of some larger pet birds (e.g., cockatoos) can be surprisingly slow compared with wilder birds. Murmurs, arrhythmias, and bradycardia are occasionally detectable. Lung and air sac noises can be auscultated, and occasionally friction rubs associated with air sacculitis can be detected when auscultating along the back on either side of the spine.

SYSTEMATIC PHYSICAL EXAMINATION

FEATHERS, SKIN, AND ADNEXA EXAMINATION

Andrés Montesinos

The feathers should be carefully examined as a whole and then individually. Examine the coverts, contour feathers, primary and secondary remiges, and rectrices. Describe any patterns of feather loss or feather abnormalities. Keep in mind that normal avian skin is incompletely covered by feathers. The clinician has to look for signs of discoloration (Fig. 5-17), excessive wear (e.g., birds kept in small or wrongly designed cages can result in feathers brushing against the wall of the cage), abnormal morphology, and parasites. Feather color is dietary dependent in species with carotenoid pigmentation (e.g., canaries and flamingos). Proper development of yellow, orange, and red pigments are all affected by the amount and type of supplements provided in the diet. Vitamin A is frequently added to the diet of these birds but remains unavailable when bound to indigestible cellulose (e.g., carrots). Beta carotene and canthaxanthin are commonly added coloring agents.

The period between feather molts varies among species, but excessively worn feathers suggest either improper husbandry or a prolonged molt cycle. First examine the bird's behavior, cage, and perch to determine whether the feathers are being traumatized (Fig. 5-18). Next investigate whether the bird is provided with water for grooming (e.g., baths, water mist, and rain). Some species of passerines utilize wet foliage for bathing. Unplanted aviaries or badly designed cages may cause these birds to abandon or modify normal grooming habits. Because molting is a complex process that may be affected by nutritional or other systemic factors, further investigation requires an analysis of the diet and reproductive history, as well as routine clinical pathologic testing (Fig. 5-19). Molt sequences vary among species and may appear abnormal to those unfamiliar with certain patterns. For example, some jays simultaneously molt all the contour feathers of the head and should not be considered pathological. Feather loss from the head and the neck of canaries is thought to be induced by malnutrition. Affected birds are typically egg-laying females. This condition should not be confused with aggressive feather plucking, which is performed by dominant males on females and subordinate males. Such behavior typically occurs in socially stressed populations (overcrowded

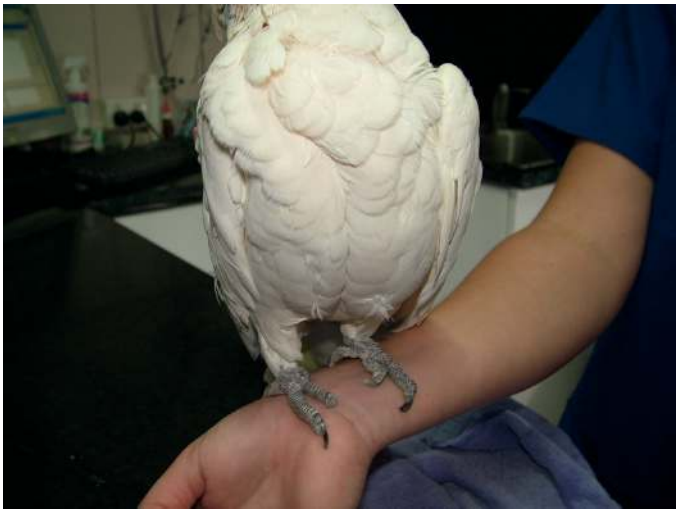


FIGURE 5-16 This obese cockatoo shows excessive body condition as evidenced by a 'cleavage' between the pectoral muscles.

FIGURE 5-17 Black feathers in an Amazon (*Amazona* sp.) parrot. A possible cause of this finding is petting the parrot with greasy hands.

FIGURE 5-18 The lack of feather in the nape region of finches point to cage mate aggression.

FIGURE 5-20 Male of eclectus parrot (*Eclectus roratus*) suffering advanced circovirus infection.

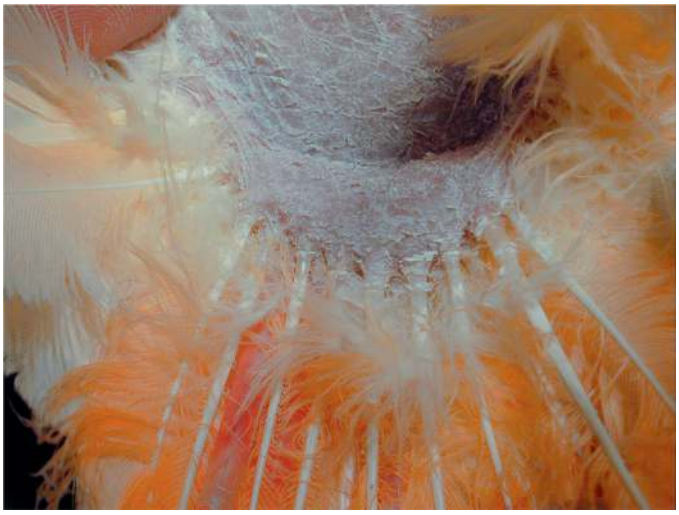


FIGURE 5-19 The featherless area under the crest of cockatoos is a good place to inspect the skin in this genus of birds.

FIGURE 5-21 The presence of abnormal colored feathers, like pink feathers in African grey parrots (*Psittacus erithacus*), could be related to malnutrition or circovirus infections among other causes.

cages) of finches, lovebirds, and other species. Affected areas include the back, the head, and (less commonly) the trunk.

Feather loss patterns, such as excessive molting, can be caused by thyroid abnormalities or light cycle influences. Feather loss can also be caused by bacterial or viral (circovirus, polyomavirus) folliculitis, pulpitis, or dermatitis. Failure to molt can be caused by thyroid abnormalities, light cycle influences, obesity (e.g., in Amazon parrots), or again, infections by circovirus (Figs. 5-20 and 5-21). In a physical examination, the clinician may find feathers chewed in areas the bird can reach with its beak; these findings suggest feather damaging behavior. Feather picking can be caused by behavioral or infectious causes, including parasitic (e.g., external parasites and also giardiasis), bacterial, viral, or fungal etiologies; musculoskeletal defects; fractures; trauma; pain; poor nutrition, leading to dry and flaky skin; or barbering by the bird's mate or cage mates. Behavioral feather picking is most common in some species like African grey parrots (*Psittacus erithacus*), cockatoos, conures, and Harris's hawks (*Parabuteo unicinctus*) (Fig. 5-22). Birds with behavioral problems can only damage the feathers it can reach with its beak; the head remains normal.

Stress bars or stress lines are seen in both wild and captive individuals. These may be translucent or appear as linear areas of depigmentation. Lesions are created during feather development and may be linked to restraint, illness, malnutrition, use of some drugs during the growing period of the affected feather (e.g., fenbendazole), or environmental stress. Retained feather sheaths and incomplete development of the barbs and barbules is referred to as 'straw feathers' and may be induced by viral infections or be genetically mediated in some breeds of canaries. Cockatoos and Grey parrots that are unable to groom normally (e.g., with spinal disease or an Elizabethan collar in place) will often develop "straw feathers" over their thighs; these are powder down feathers usually chewed by the bird during normal grooming.

FIGURE 5-22 Feather picking of the ventral covert feathers in a young peregrine falcon (*Falco peregrinus*). This is very likely a brood or incubation patch. Female falcons, at the beginning of the breeding season, pluck the feathers in preparation to incubate eggs even if they are still too young to breed.

FIGURE 5-23 Flaky skin in the breast area of an African grey parrot (*Psittacus erithacus*).

Feather cysts appear as lumps involving one or more feather follicles, which typically affect the wings, the back, and the chest. Heredity may also be linked to the development of feather cysts in Norwich, Gloucester, crested, crest-bred, and dimorphic new color canaries (*Serinus canaria*), but in other species may be induced by chronic trauma over the feathers.

The skin over the most of a bird's body is thin, soft, dry, and relatively translucent. Small portions of discarded feather sheaths are normally found on the skin and should not be confused with dry, flaky skin (Fig. 5-23). Uric acid deposit may be noted under the skin in cases

FIGURE 5-24 Constrictive rings of skin in the digits of a 6-week-old eclectus parrot chick (*Eclectus roratus*). This syndrome has been associated with low humidity in the brooder, but other causes should be investigated.

FIGURE 5-25 Constrictive lesions caused by debris accumulation under the ring in a Lady Gouldian finch (*Erythrura gouldiae*).

of gout. Examination of the subcutaneous tissues can be enhanced by wetting the overlaying feathers with warm water or alcohol.

Scales covering the legs and feet should be smooth, soft, and uniform. Individual digits should be flexible and free of defects (Figs. 5-24 and 5-25). Examination of every digit should be conducted and is best performed with a magnifying loupe or similar device. Devitalization may occur secondary to stricture (e.g., linear foreign bodies, scar formation, or poxvirus), bacterial infections, self-mutilation, ergot poisoning, and frostbite.

In birds with darkly pigmented scales, as the scales separate because of swelling, the resulting lesions appear as areas of depigmentation (e.g., as caused by cutaneous poxvirus infections, insect bites, and granulomas). Proliferative lesions on the legs and feet may be caused by papillomavirus or *Cnemidocoptes* mites (Figs. 5-26 to 5-28). Corkscrew-shaped nails can also result from a *Cnemidocoptes* infestation.

FIGURE 5-26 View of the lesions of a *Cnemidocoptes* infestation on the face of a budgerigar (*Melopsittacus undulatus*). The tunnels in the proliferative tissue create a characteristic honeycomb appearance.

FIGURE 5-28 Proliferative (wart-like) lesions on the face skin of an African grey parrot (*Psittacus erithacus*). The skin biopsies revealed papilloma virus particles and secondary bacterial infection.

FIGURE 5-27 Papilloma-like lesions on the feet of macaws have been related to local herpesvirus infections.

FIGURE 5-29 Type I pododermatitis in an obese Amazon parrot (*Amazona* sp). Plantar skin surface should always be examined in a complete physical examination of a bird.

Examine the plantar aspects of both feet. Incipient pododermatitis lesions appear as small pink areas. These progress in severity and can be graded (I to VI) to describe the level of pathology more accurately (Fig. 5-29). Obtain a good dietary history and details of the bird's caging and husbandry. Improper nutrition, obesity, and orthopedic injuries may contribute to the development of this problem. Proper perching is also very important. Perches should vary in size to prevent birds from always bearing weight on the same surface of the foot. Abnormal or overgrown nails may result in self-inflicted injuries that can progress to pododermatitis.

Examine the bilobed uropygial or preen gland and its wick, which is located on the dorsal surface of the tail in the species that have this gland (Figs. 5-30 and 5-31). The preen gland is especially well developed in most aquatic species, such as Sphenisciformes, Podicipediformes, and gulls (Laridae). It is absent in Struthioniformes, Rheiformes, Casuariiformes, and bustards (Otididae). The gland is either absent or very small in some members of the Caprimulgiformes, Columbiformes, and Piciformes. Within the Psittaciformes, Amazon parrots (*Amazona* sp.), Hyacinth macaws (*Anodorhynchus hyacinthinus*), and palm cockatoos (*Probosciger aterrimus*) do not have an uropygial gland. Its presence is inconsistent in many other psittacine birds, but the gland is always present in budgerigars and African grey parrots. This gland is susceptible to infection secondary to blockage of either a duct or the central papilla where the wick is located. If the gland is found to be asymmetric, hyperemic, or engorged on examination, then impaction, abcessation, tumor, or chronic dermatitis are suspect. The most common tumors associated with the preen gland are squamous cell carcinomas, adenomas, and papillomas.

FIGURE 5-30 The uropygial gland should be inspected in all of the birds who possess one. This parrot pecked the feather surrounding the obstructed gland.

FIGURE 5-31 Uropygial gland adenoma in a budgerigar (*Melopsittacus erithacus*) before excision surgery.

Some gallinaceous birds have unique skin appendages. Domestic fowl and jungle fowl possess marked, unpaired, corneous combs consisting of a wide intermediate layer, which is formed of a fibrillar network filled with mucous-like substances that affect elastic stability to the comb. The intermediate layer is covered by the strongly vascularized corium and the epidermis. The paired wattles of the throat are similar in structure to the comb. Like the comb, the size of the wattles is influenced by hormones, and both are better developed in cocks than in hens.

FURTHER READING

Briscoe JA, Morris DO, Rosenthal KL, et al: Evaluation of mucosal and seborrheic sites for staphylococci in two populations of captive psittacines, *J Am Vet Med Assoc* 234(7):901–905, 2009.

Garner MA, Club SL, Mitchell MA, Brown L: Feather-picking psittacines: histopathology and species trends, *Vet Pathol* 45(3):401–408, 2008.

Rubisteins J, Lightfoot T: Feather loss and feather destructive behavior in pet birds, *Vet Clin North Am Exot Anim Pract* 17(1):77–102, 2014.

Shawkey MD, Hill GE: Feathers at a fine scale, *Auk* 121(3):652–655, 2004.

FIGURE 5-32 Featherless area between the eye and the nares opening are very suggestive of sinus inflammation.

THE HEAD REGION AND OROPHARYNX

Andres Montesinos

The head should be examined directly from the front of both the unrestrained and restrained bird, taking note of overall symmetry, especially of the eyes, nares, cere, beak, and ears. Both before and after the examination, observe the patient from a concealed vantage point. Before examination, the bird should appear alert and aware of its surroundings. Partially closed eyes, fluffed feathers, and drooping wings are all general signs of poor health. Begin the examination by looking directly along the length of the bird's bill and comparing the symmetry of the right and left sides of the head. Repeat the process while looking down on the dorsal aspect of the skull.

The eye should be clear and free of discharge (Fig. 5-32). A serosanguinous or frank bloody discharge from the eyes occasionally occurs during handling of African grey parrots (*Psittacus erithacus*) and Major Mitchell's cockatoos (*Lophochroa leadbeateri*), among others. A more detailed description of the eye examination can be found later in this chapter.

The nares should be symmetric, oval to round in shape, and of appropriate size for the bird. They should also be free of discharge and organic debris that could eventually accumulate and cause formation of nasal rhinoliths. The cere should be smooth, dry, firm, and of uniform color. Cere and nare asymmetry could indicate a parasitic infection with *Cnemidocoptes* mites, an allergic reaction to the environment, or an insect bite. A browning of the cere, or brown cere hypertrophy, is known to occur in budgerigars (Fig. 5-33). This change may be from pink to brown in females and is of no consequence. In males, the change from blue to brown cere is thought to be abnormal and is associated with gonadal tumors (e.g., Sertoli tumors). Moist, matted feathers around the nares may indicate nasal discharge, but if this matting progresses further up to the crown and beyond, regurgitation should be considered. Moist feathers around the ears may indicate aural discharge or regurgitation in a bird that spits up and shakes its head.

Avian bills vary greatly in size, shape, and color. Normally, the keratin of the beak should be smooth and free of any cracks or deviations. An overgrown or flaky beak may be indicative of poor nutrition, liver disease, or improper management (Fig. 5-34). In addition to a balanced diet, captive psittacine birds need access to hard wood and

leather toys to chew for proper and safe beak wear. Captive raptors also need bones to gnaw in order to keep the beak in shape; also, some birds of prey possess a tomial tooth as part of the normal anatomy of the beak. Normal adult cockatoos and African grey parrots have powder down on their beaks. Psittacine beak and feather disease virus (PBFDV) affects the powder-producing feathers, resulting in shiny, powderless beaks. Beaks should grow straight to attain proper occlusion. Asymmetry, malocclusion, or malalignment of the beak could indicate development malformations (e.g., scissor beak in macaws), past trauma to a fully formed beak, grooves in the beak secondary to rhinitis, a deep-seated fungal or bacterial infection, or beak necrosis secondary to psittacine circovirus (Figs. 5-35 and 5-36).

The ears canals should be open and free of discharge, odors, and parasites. A small amount of normal flaky desquamated material is frequently evident within the opening (Fig. 5-37). Young macaws (such as the Buffon's *Ara ambiguus* and the blue-throated *Ara glaucogularis*)

FIGURE 5-33 Brown hypertrophy of the cere in a female budgerigar (*Melopsittacus undulatus*).

FIGURE 5-34 Close view of the irregular overgrowth of the beak keratin in a Lady Gouldian finch (*Erythrura gouldiae*) caused by fungal infection.

FIGURE 5-35 Deformed beak and narina of a You-You (*Poicephalus senegalus*) affected by chronic rhinitis.

FIGURE 5-36 Scissor beak is a common malformation seen in young macaws and could be related to several etiologies such as metabolic bone disease, bad nutrition, or improper hand-feeding technique.

FIGURE 5-37 The presence of dry exudates surrounding the ear opening should direct the clinician to a close inspection of the ears.

and lovebirds (*Agapornis* sp.) are prone to ear infections that can occur secondary to pinpoint ear canal apertures or to fungal infections. This is a common developmental abnormality in these species and is easy to correct if caught in the early stages. The ear canals are carefully stretched mechanically with a fine pair of hemostats, thus avoiding fluid and wax buildup and subsequent infection. When dealing with avian wildlife, the ear opening is often the place to look for a tick infestation. In owls, the ear opening can be looked at as part of the eye examination. The infraorbital sinus of the bird extends rostrally and caudally from the ear canal; hence, an infraorbital sinusitis can also cause ear infection.

To examine inside the mouth, one should use a gauze swab to pry the maxilla and mandible apart gently (especially when dealing with parrots), or the bill or beak may be opened with the fingers. Swollen or blunted choanal papillae can be cultured with this technique, as can other lesions found in the oral cavity and oropharynx. The mouth, tongue, oropharynx, and choana are best examined with a small, bright light source. The oral mucosa is typically pink and moist, but could be black in some avian species. The tongue should be firm, smooth, and dry; the oropharynx should be uniformly pigmented and moist. These areas should be free of debris, discharge, abnormal growths, and plaques (Fig. 5-38). Abnormal growths can be neoplastic; adenocarcinoma is the most common tumor found in the avian oral cavity. White or yellowish plaques in the mouth can occur secondary to several conditions such as:

- Hypovitaminosis A with subsequent squamous metaplasia (Fig. 5-39)
- Candidiasis
- Poxvirus
- Trichomoniasis (Fig. 5-40)
- Capillariasis
- Mycobacteriosis
- Neoplasia
- Herpesvirus (Fig. 5-41)

The choana (the slit in the roof of the mouth) connects the nasal cavity to the opening of the bird's trachea and with the mouth. Discharge from the choanal slit is therefore indicative of an upper respiratory tract infection.

FIGURE 5-38 The mouth and oropharynx of all the birds presented in the clinic should be inspected carefully. The presence of white plaques in the oral mucosa could be related to numerous issues.

FIGURE 5-39 Granulomatous lesions found in the tongue of a Brazilian Amazon (*Amazona brasiliensis*) parrot. Bacterial infections or hypovitaminosis A should be considered as the cause of these findings.

FIGURE 5-40 Supraorbital sinusitis in a canary finch. Trichomoniasis, hypovitaminosis A, and mycoplasmosis should be considered as possible causes.

FIGURE 5-41 Yellowish lesions found in the mouth of a European eagle owl (*Bubo bubo*) associated with herpesvirus infection.

FURTHER READING

Welle KR: Maximizing avian wellness examination, *J Exot Pet Med* 20(2):86–97, 2011.

Worell AB: Dermatological conditions affecting the beak, the claws, and feet of captive avian species, *Vet Clin North Am Exot Anim Pract* 16(3):777–799, 2013.

THE EYE AND EYELIDS

Alejandro A. Bayón del Rio

INTRODUCTION

Basically, the morphology of the bird's eyes, as well as their physiology, are similar to that of mammals, though certain particularities exist that must be considered in order to carry out a correct interpretation of the ocular examination (Kern and Colitz, 2013). In addition, it is important to consider that systemic diseases with ocular signs are just as important in birds as in mammals. Generally, to allow a precise diagnosis, the same methods and equipment for ocular examination that are used in mammals are also used in birds, though there are limitations with the small ocular size of some birds.

CHARACTERISTICS OF THE BIRD'S VISUAL FUNCTION

The sensory organ of vision in birds is highly specialized for adjustment to their living conditions, their visual acuity being 2 to 8 times higher than that of mammals. Their visual fields are over 360 degrees, the range of stereopsis is 0 to 70 degrees, the maximum spatial frequency (skill to distinguish a certain movement in simple images) is over 160 images/second (compared with 10 to 15 in humans), and they have a minimal detection of movements over 15 degrees/hour (movements that are performed in a very slow way) (Bennett and Cuthill, 1994; Korbel, 2002).

The perception of ultraviolet light is a common skill in diurnal birds caused by the rods in the retina having a special sensitivity to ultraviolet light. This ability plays a very important role in features such as communication, camouflage, and orientation (Bennett and Cuthill, 1994).

ANATOMY AND OCULAR PHYSIOLOGY

Orbit and Globe

The globe is large compared with body size, with a posterior segment disproportionately larger than the anterior segment (Fig. 5-42). The back part of the globe fits narrowly in the orbit, though many temporal and dorsal zones are unprotected by bone. The diameter of the equator of the globe exceeds the diameter of the anterior bony orbital rim in many species. The orbits are separated only by a thin bony structure or a septum of connective tissue (Korbel, 2000).

The shape of the eyes is supported by the 10 to 18 scleral ossicles in the intermediate segment, as well as by the hyaline cartilage present in the posterior segment of the sclera. There are three basic shapes of the eyeball (Kern, 1989; Williams, 1994):

- Flat: Presenting with a short anteroposterior axis, flat or concave ciliary region, convex cornea, and hemispherical posterior segment, this is typical of the majority of birds.
- Globose: The ciliary region protrudes further from the posterior segment though remaining concave. It is typical for many diurnal birds because they need high resolution for long distances (diurnal birds of prey, insectivores, ravens).

FIGURE 5-42 The skull of an owl illustrating the orbit with a tubular-shaped exposed eye.

- Tubular: The intermediate concave segment elongates anteroposteriorly, forming a tube, before joining the posterior segment at a sharp angle. The cornea is at the front. This shape is typical of owls (see Fig. 5-42).

The extraocular muscles are rudimentary; thus, the ocular motility is limited in comparison with mammals. There are four straight muscles, two oblique, one pyramidalis, and one quadratus (they replace the retractor bulbi muscle in mammals, which are innervated by cranial nerve VI and move the nictitating membrane). The muscles mentioned earlier are innervated by the oculomotor nerve (sixth cranial pair), and the function is to move the nictitating membrane. Some parts of the lengths of the first through sixth cranial nerves have an intraorbital course, with the segment of the optical nerve being quite short. The vascular plexus is in the ventrolateral zone of the orbit (Kern, 1989; Williams, 1994; Kern, 1999).

The infraorbital sinus and part of the cervicocephalic air sac system are situated laterally, subcutaneous to the nasal area and rostroventral to the eyes, in several groups of birds (e.g., psittacines, storks). The sinus can be connected with pneumatized sections of the skull bones that spread toward the upper parts of the beak, jaw, and orbit (Kern, 1989; Williams, 1994).

Eyelids and Ocular Annexes

Birds have an upper and a lower eyelid, plus a nictitating membrane. The lower eyelid is more mobile than the upper one, which allows it to cover a larger part of the eye during blinking. It also has a fibroelastic tarsal plate. Near the palpebral margin there are modified feathers for protection or for tactile function. Upon hatching, eyelids are well developed and the palpebral fissure is open in precocial birds, although the lids are sealed together and incompletely developed in altricial birds. The time of opening of eyelids in altricial birds is variable: in the case of cockatoos, it happens between 10 and 17 days after hatching; in macaws, from 17 to 26 days. The palpebral separation starts centrally, progressing medially and laterally. There are no meibomian glands at the tarsal edge of the eyelids (Kern, 1989; Williams, 1994).

The nictitating membrane is well developed and mobile; it is thin and translucent and moves over the globe from a dorsonasal position toward a ventrotemporal position, dragged by the pyramidal muscle that originates from the back of the sclera, turning around the optic

nerve and passing through a sling formed by the quadratus muscle. The temporal top edge of the nictitating membrane is firmly adherent to the underlying sclera and is associated with the conjunctiva, and the pyramidalis tendon is inserted along the lower nasal edge. The free edge of the nictitating membrane has a marginal pigmented fold or edging that facilitates the distribution of tears over the ocular surface during blinking (Kern, 1989; Williams, 1994).

The Harderian gland, placed near the base of the nictitating membrane, is the main source of tears in birds. A wide canal runs from the gland and opens inside the conjunctival sac between the eyeball and the nictitating membrane. The lymphoid tissue associated with the conjunctiva, together with the Harderian gland, plays an important role in the humoral immunologic defense of the ocular surface. The lacrimal gland is positioned inferotemporal to the globe (absent in penguins and owls). In some birds, such as budgerigars, a nasal or salt gland lies in the orbit dorsomedial to the globe. The duct of this gland penetrates the frontal bone and enters the nasal cavity.

Anterior Segment

The cornea in birds is histologically similar to those of mammals. The lens is soft and almost spherical in nocturnal birds, or has a flattened anterior aspect in diurnal species, including companion birds. An annular pad lies under the lens capsule in the equatorial region, and can be separated from the center of the lens during cataract surgery. The power of the lens can be increased by contraction of the midstriate ciliary muscles (Brücke and Crampton muscles), which compress the annular cushion (Kern, 1989; Williams, 1994). The ciliary processes are attached to the equatorial lens capsule. The Crampton muscle has connections with the peripheral cornea, being able to produce changes in the corneal curvature when it contracts. In several diving birds, the changing shape of the lens, produced by miosis, is part of the accommodation. The iris is generally brown, though other colors can also be present. The stromal pigments of the iris are composed of carotenoids, purines, and pteridines; in some species, the coloration of the iris can change with age and sex. In red-tailed falcons, there is a color shift from yellowish to gray when the birds reach 4 years of age; in araraunas, the iris changes from brown to gray when the birds are 1 year old; in Amazons, the iris changes from brown to red or orange when they grow up; in cockatoos, there is sexual dimorphism—the females have a red-colored iris and males have dark brown or black, and in young cockatoos, the iris is brown in both sexes.

The iris muscles are mainly striated, with smooth muscles appearing only in smaller proportion. This allows voluntary contraction of the pupil. The striated circumferential muscle seems to be the primary pupillary sphincter in all species. The circular pupil responds rapidly to accommodation and voluntary control, as seen, for instance, in stress during handling. There is a direct pupillary reflex but no consensual one, caused by the complete decussation of the optical nerve axons. A small degree of anisocoria can be normal. The iridocorneal angle is well developed (Fig. 5-43) (Kern, 1989; Mikaelian *et al.*, 1994; Williams, 1994).

Posterior Segment

The vitreous body is large and transparent. The fundus is normally gray or reddish in color with the choroidal vessels not always visible. The optic disc is long and oval, but it is often difficult to observe on ophthalmoscopy because of the pecten. The pecten is a vascular prominence emerging from the retina and protruding into the vitreous, of variable comb-like shape and black in color (Fig. 5-44). It is involved in the nutrition of the retina and plays a role in intraocular acid-base balance and production of intraocular fluids, and mechanically shakes the vitreous fluid during ocular movements, facilitating the movement of fluids inside the eye (Bellhorn, 1997).

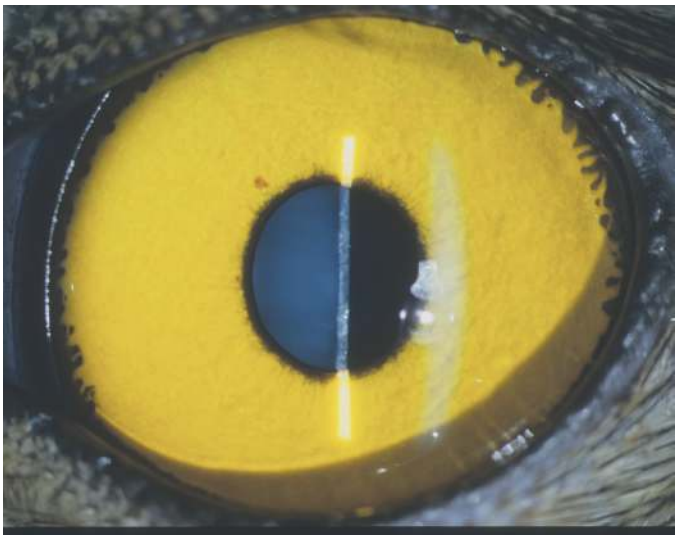


FIGURE 5-43 Iridocorneal angle.

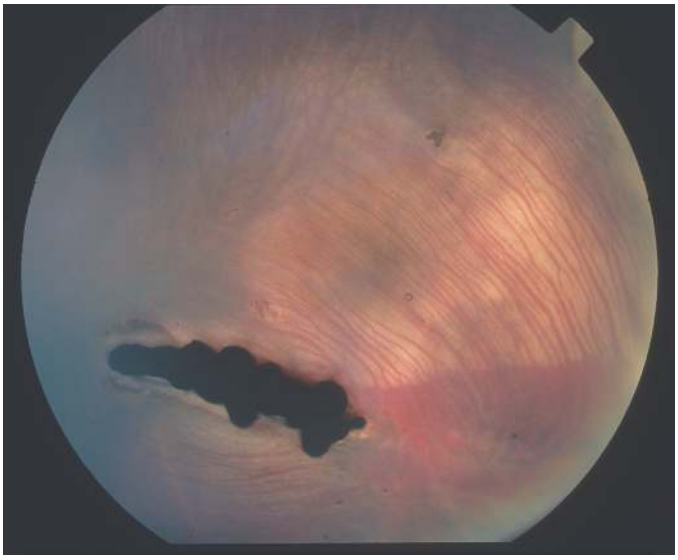


FIGURE 5-44 Typical fundus oculi of a nocturnal raptor: the choroidal vessels with whitish sclera in between and the ventronasal periphery of the pecten.

The retina is avascular and without a tapetum. The type of photoreceptors and the density vary among birds, but generally rods and cones or double cones with oil droplets are present. According to the specialization of the retina to enhance the resolution or sharpness, birds can be classified as:

- Afoveate: these have an area centralis or visual line usually present when there is absence of fovea, seen in the majority of the domestic birds and some birds of land and water.
- Monofoveate: having a central (majority of birds) or temporal (e.g., owls, swifts) fovea with or without a visual line around the fovea.
- Bifoveate: with a principal central fovea and temporal subsidiary, with or without a visual line of enhancement of the sharpness between the fovea (e.g., falcons, eagles, several Passeriformes, others that hunt during flight).

Color vision is well developed and several species can detect nearby ultraviolet light (Korbel, 2000; Murphy *et al.,* 1985).

OPHTHALMOLOGIC EXAMINATION

The ophthalmologic examination in birds is similar to what is performed in mammals, with some particularities derived from the anatomic and physiologic differences (Kern and Colitz, 2013). In general, the ocular examination will follow a general examination of the bird. First a complete clinical history must be gathered, as well as a study of the habitat and nutrition. While the clinical history is being taken, it is recommended that the bird should be kept in its cage to evaluate its visual acuity and state of alertness, as well as its general behavior while being under the control of the owner.

Examination

Ideally, the ophthalmologic examination should be carried out without the use of sedatives or anesthetic agents because they can interfere with behavior, lacrimation, and reflexes.

The examination of the anterior segment and periocular structures is carried out by means of a beam of light, otoscope by magnifying glass, direct ophthalmoscope (with a lens of +25D to +40D), and a slit-lamp biomicroscope (Fig. 5-45). The latter is necessary for examining the small eyes of some species of birds because it allows magnification and visualization of small injuries. Likewise, expert help in handling birds is useful to hold the animal while the clinician works the equipment (Kern, 1989).

Ocular Reflexes

In birds, the palpebral reflex is evaluated by touching skin areas on the lateral and medial edge of the eyelid. The nictitating membrane goes over the cornea quickly and completely in normal birds; both eyelids will move easily, though the lower one covers a larger area of the corneal surface than the upper one. The globe does not retract and the eyelids may not close completely in normal birds. The direct pupillary light reflex may be evaluated by the use of a beam of bright light in a dimly lit room (Kern, 1989; Kern, 1999; Williams, 1994).

Spontaneous pupil movements may happen in situations of excitement because of the voluntary control. Indirect or consensual reflexes are not expected in birds because of the complete decussation of the fibers of the optical nerve. However, sometimes small responses can be provoked because of the fact that the eyes are separated by a very thin septum. The menace reaction is weak in birds with normal vision, thus it has very little diagnostic importance. The conjunctional movements of the eyes are minimal in the majority of birds because of the limited ocular motility (Kern, 1989).

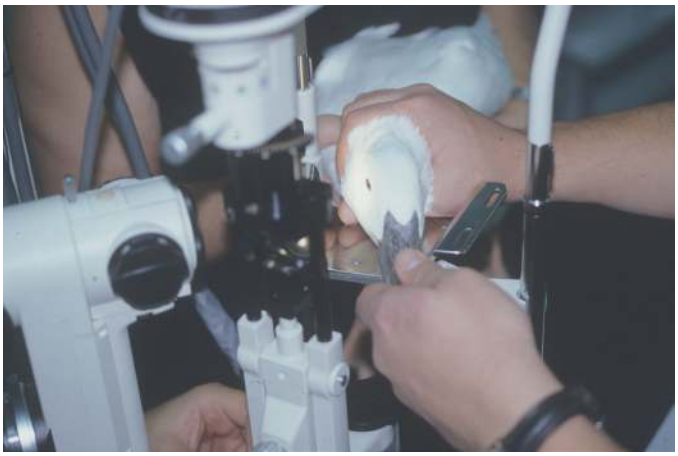


FIGURE 5-45 Biomicroscopy examination in a duck.

The corneal reflex, though it is not routinely examined in birds, is observed by means of blinking, movement of the nictitating membrane, and negative response to external stimuli; however, the eyeball does not retract because of the absence of the retractor muscles of the globe.

Schirmer Tear Test

It is carried out mainly in birds of great size. In a study carried out in 255 birds of 42 species, Schirmer tear test values have been obtained for Psittaciformes, being 3.2 to 7.5 mm/min without topical anesthesia and 1.7 to 4.5 mm/min with topical anesthesia; in Falconiformes, 4.1 to 14.4 mm/min without anesthesia and 2 to 4.2 mm/min with topical anesthesia; in Accipitriformes, 10.7 to 11.5 mm/min without anesthesia and 3.6 to 5.9 mm/min with topical anesthesia (Korbel and Leitenstorfer, 1998; Williams, 1994).

Staining

Topical fluorescein sodium, together with a cobalt blue light, reveals damage to the cornea or possible obstructions in the lachrymal system. Staining with rose bengal is carried out for the diagnosis of keratitis (it stains keratinized epithelium) (Williams, 1994).

Cytology and Culture

These procedures may be necessary when an infectious or parasitic problem is suspected (mites in the periocular structures), and there is an ocular discharge. If ocular mucus or mucopurulent secretion is observed, a sterile sample for isolation of bacteria, mycoplasma, or virus is indicated. For the isolation of *Chlamydia* it is preferable to gather samples from the choana, trachea, or cloaca (Kern, 1989).

The most frequent flora present in the conjunctival sac in psittacines includes gram-positive bacteria *(Staphylococcus epidermidis, Staphylococcus aureus, β-Hemolytic streptococcus, Corynebacterium* sp.); gram-negative bacteria are rare and only reported to be isolated from 1% of samples (Zenoble *et al.,* 1983).

Corneal cytology is indicated in superficial erosions and progressive ulceration associated with infiltrations of inflammatory cells. To take a sample for cytology, a topical anesthetic is instilled (1 or 2 drops). After 60 seconds, the sample can be obtained, then the smear stained with Giemsa or DiffQuick stains. One should be aware that in birds the topical anesthetic can cause systemic toxicity and even general anesthesia.

Tonometry

The equipment used to perform tonometry tests are Tonopen (applanation), Tonovet (rebound) (Fig. 5-46), and Schiötz tonometer (indentation). In birds, the normal values of intraocular pressure are as follows: In turkeys, 25 mm Hg (applanation); in birds of prey, 11 to 16 mm Hg; and in psittacines, 20 to 25 mm Hg (applanation). In the study carried out by means of Tonopen in 275 normal birds of 39 species, the range of values was between 9.2 and 16.3 mm Hg. The reproducibility of the values is obtained with corneal diameters ≥ 9 mm, being limited in corneal diameters < 5 mm (Korbel, 1993; Stiles *et al.* 1994). In large birds and by means of the Schiötz tonometer, values of 15 to 17 mm Hg have been obtained in falcons and 20 mm Hg in domestic chickens. The Tonovet has been used in another study with 31 birds of prey, obtaining a range of measures of 9 mm Hg in small raptors and 40 mm Hg in large birds of prey (Bayón *et al.,* 2006). Compared with applanation tonometry, rebound tonometry significantly overestimated intraocular pressure in Eurasian eagle owls (*Bubo bubo*) (Jeong *et al.,* 2007).

FIGURE 5-46 Tonometry with Tonovet.

Direct and Indirect Ophthalmoscopy

The mydriasis necessary for ophthalmoscopy in birds can be obtained by means of general anesthesia with ketamine or by topical or 20 mg/mL intracameral tubocurarine (d-tubocurarine chloride, Sigma Chemical, Colo.) (Arnall, 1961; Korbel, 2000). A study in raptors proved the efficiency of three curariform agents, obtaining the following results (Mikaelian *et al.,* 1994). Vecuronium bromide topically (2 drops every 15 minutes): the maximum reaction appears immediately after application, and is effective for 4 hours. Alcuronium chloride causes mydriasis of 3 hours' duration, though in the study the majority of the birds presented with palpebral paralysis and even paralysis of the neck and hind limbs in some birds. Pancuronium bromide produces a very slight reaction.

Direct ophthalmoscopy is used most frequently, though it is not the best to use on exotic birds because of the often small size of the eyes and because the head of the clinician must be close to the bird. When an injury is suspected, the indirect ophthalmoscope can be used because it allows exploration of a very wide area of the fundus (with a reversed image) at a larger distance from the clinician (Fig. 5-47). The lens required depends on the size of the birds, from 20D to 30D in large birds, and up to 90D in small species. The fundus of both eyes must be compared, especially when some anomalies are present. The optic nerve head is most often hidden under the pecten.

Retinography

Retinography allows the examiner visualization of the back of the eye only in large species; in very small eyes, the focal distance is not suitable.

Electroretinography

Electroretinography is used for assessing retina function. It is carried out on animals anesthetized by means of an intramuscular combination of ketamine-medethomidine or by means of inhalation anesthesia, such as isoflurane, with the same equipment used in mammals with contact lens, Dawson Trick Litzkow (DTL) fiber electrode (in very small eyes), and dermal electrodes (Fig. 5-48) (Bayón *et al.,* 2007). The protocol is similar to the one established for dogs (Narfström *et al.,* 2002).

Bidimensional and Doppler Ultrasonography

In ophthalmology, this noninvasive technique is used fundamentally for ocular biometry and when the opacification of anterior structures

FIGURE 5-47 Indirect ophthalmoscopy.

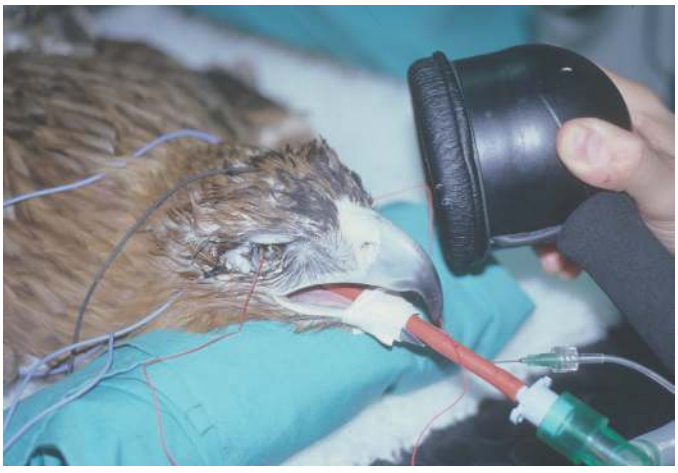


FIGURE 5-48 Electroretinography in a raptor, showing placement of electrodes.

(i.e., cornea, anterior chamber, lens) prevents the visualization of deeper structures (i.e., vitreous body and retina) (Fig. 5-49). Likewise, it offers information about diseases of the orbit (e.g., neoplasia, foreign bodies). It can be carried out with general equipment or special equipment for ophthalmology using linear transducers of high frequency (7.5 to 11 MHz). Color Doppler ultrasonography allows evaluation of vascularization of the ocular structures (Bayón *et al.,* 2007).

Radiology

Radiology is used in ophthalmology before other visual techniques (e.g., ultrasonography, axial computerized tomography, and magnetic resonance) for the evaluation of the orbit and cranium (Kern, 1989).

Axial Computerized Tomography

Axial computerized tomography provides detailed images of the structures contained in the orbit (e.g., eyeball extraocular muscles, optic

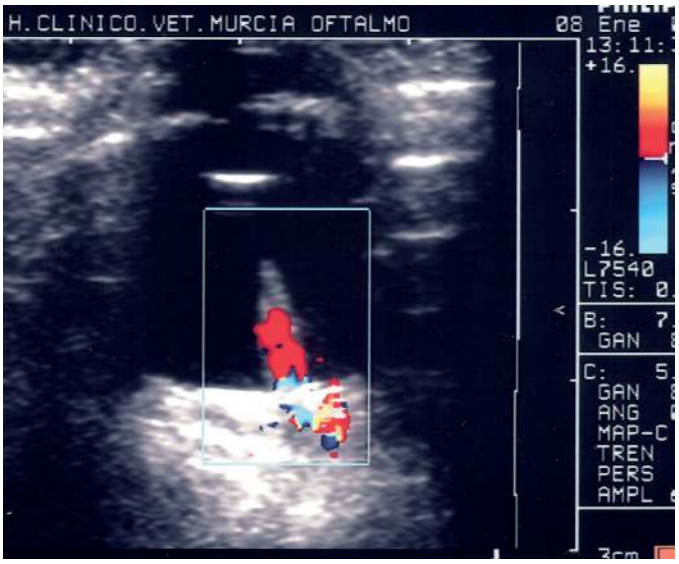


FIGURE 5-49 Color Doppler ultrasonography showing blood flow in the pecten.

nerve), as well as the bones. It gives important information for the diagnosis of orbital neoplasms and inflammatory and traumatic diseases.

Magnetic Resonance

In small animals, magnetic resonance is used fundamentally in neuro-ophthalmology because of the good resolution that it provides for the evaluation of soft tissues.

REFERENCES

Arnall L: Anesthesia and surgery in cage and aviary birds. II. A regional outline of surgical conditions, *Vet Rec* 73:173–178, 1961.

Bayón A, Almela RM, Talavera J: Avian ophthalmology, *Eur J Companion Anim Pract* 17:253–265, 2007.

Bayón A, Vecino E, Albert A, et al: Evaluation of intraocular pressure obtained by two tonometers and their correlations with corneal thickness obtained by pachymetry in raptors, Proceedings ESVO-ECVO Meeting, pp 154, 2006.

Bellhorn RW: Retinal nutritive systems in vertebrates, *Semin Avian Exotic Pet Med* 6:108–118, 1997.

Bennett AT, Cuthill IC: Ultraviolet vision in birds: what is its function? *Vision Res* 34:1471–1478, 1994.

Jeong MB, Kim YJ, Yi NY: Comparison of the rebound tonometer (TonoVet®) with the applanation tonometer (TonoPen XL®) in normal Eurasian eagle owls (*Bubo bubo*), *Vet Ophthalmol* 10:376–379, 2007.

Kern J: Disorders of the special senses. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, 1989, WB Saunders, pp 563–589.

Kern TJ: Exotic animal ophthalmology. In Gelatt KN, editor: *Veterinary ophthalmology*, Philadelphia, 1999, Williams & Wilkins, pp 1273–1305.

Kern TJ, Colitz MH: Exotic animal ophthalmology. In Gelatt KN, editor: *Veterinary ophthalmology*, Hoboken, 2013, Wiley & Blackwell, pp 1750–1819.

Korbel RT: Tonometry in avian ophthalmology, *J Assoc Avian Vet* 7:44, 1993.

Korbel RT: Disorders of the posterior eye segment in raptors—Examination procedures and findings. In Lumeij JT, Remple JD, Redig PT, et al, editors: *Raptor biomedicine III*, Lake Worth, 2000, Zoological Education Network, pp 179–193.

Korbel RT: Avian ophthalmology principles and application. In *Proceedings WSAVA-FECAVA-AVEPA Congress*, 2002, pp 214–217.

Korbel R, Leitenstorfer P: The modified Schirmer tear test in birds—A method for checking lacrimal gland function, *Tierarztl Prax Ausg K Kleintiere Heimtiere* 26:284–294, 1998.

Mikaelian I, Paillet I, Williams D: Comparative use of various mydriatic drugs in kestrels (*Falco tinnunculus*), *Am J Vet Res* 55:270, 1994.

Murphy CJ, Kern TJ, Loew E, et al: Retinal dysplasia in a hybrid falcon, *J Am Vet Med Assoc* 187:1208–1209, 1985.

Narfström K, Ekesten B, Rosolen SG, et al: Guidelines for clinical electroretinography in the dog, *Doc Ophthalmol* 105:83–92, 2002.

Stiles J, Buyukmihci NC, Farver TB: Tonometry of normal eyes in raptors, *Am J Vet Res* 55:477–479, 1994.

Williams D: Ophthalmology. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, 1994, Wingers Publishing, pp 673–694.

Zenoble RD, Griffith RW, Clubb SL: Survey of bacteriologic flora of conjunctiva and cornea in healthy psittacine birds, *Am J Vet Res* 44:1966–1967, 1983.

PHYSICAL EXAMINATION

THE NECK

Robert Doneley

The neck should be gently palpated and turned to assess mobility. The cervical vertebrae are flexible and generally held in an "S" position, allowing the neck to turn and stretch in a variety of directions. In most birds, the feathers over the neck are arranged in pterylae, allowing easy visualization of the skin, trachea, and esophagus. (The exceptions are waterfowl and pigeons, where the feathers cover the entire skin of the neck without being arranged into pterylae.) The use of internal light and wetting of the skin with alcohol can allow transillumination of the trachea when looking for foreign bodies or strictures.

Abnormalities that may be detected include bruising, subcutaneous emphysema, and kinking of the neck (wry neck). The latter occurs with a unilateral contracture of the muscles and tendons of the neck, and may become permanent with the development of fibrosis.

THE CROP

Robert Doneley

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, especially in sick birds, as fluid or ingesta in the crop can be pushed back up the neck into the oropharynx, resulting in sneezing, aspiration, and death.

In most birds, the crop is usually empty or near-empty in the examination room. The crop can be gently kneaded between the thumb and forefinger. The crop mucosa should feel smooth and thin. There may be some food present in the most pendant part of the crop. Abnormal findings include a thickened crop mucosa or an overly pendulous crop when empty. Doughy material, fluid, or gas filling the crop (especially when associated with a sour smell from the mouth) suggests crop stasis. Occasionally ingluviliths (crop stones: concretions of food and other material) or other foreign bodies (e.g., fibrous twine, long grass, or grit) can be detected.

If abnormalities are detected, a crop wash or radiographs can be used to better evaluate the size and contents of the crop.

THE BODY

Robert Doneley

The bird's body should be examined and gently palpated from the thoracic inlet to the base of the tail.

Palpation of the skin over the trunk occasionally reveals subcutaneous emphysema. Although this is normal in species such as pelicans,

FIGURE 5-50 Kyphosis in an African grey parrot (*Psittacus erithacus*); note the hunched back and the tail directed vertically to the floor of the cage. Although a common congenital condition, this case was acquired kyphosis caused by vertebral aspergillosis.

in most species it is often the result of trauma, surgery, or infection rupturing air sacs and allowing the escape of air under the skin.

The ventral coelomic wall, between the end of the sternum and the pubic bones, is usually concave. Convexity can be caused by either internal distention of the coelom or extracoelomic masses. Internal distention of the coelom can be because of fat, organ enlargement, ascites, neoplasia, intestinal gas, or the presence of an egg. External distention can be caused by subcutaneous fat, neoplasia (especially lipomas and xanthomas), or hernias. Diagnostic imaging, endoscopy, or ultrasound may be required to distinguish between internal and external coelomic distention, and between different causes of both.

Deeper coelomic palpation, especially in thinner birds, can detect the presence of an egg, neoplastic masses, or hepatomegaly. Care must be taken not to confuse the ventriculus (gizzard) with a mass or egg. This palpation may elicit pain or discomfort in some birds, suggesting a painful internal lesion such as caused by pancreatic disease.

The bird's spine should be carefully palpated for evidence of scoliosis, lordosis, or kyphosis. As the notarium and synsacrum are predominantly fused, flexibility of the spine cannot be assessed as it is in dogs and cats. The area most likely to be affected by spinal deformities are the lumbar and coccygeal regions (Fig. 5-50).

The carina of the sternum should be palpated for evidence of distortion, trauma, or congenital defects such as sterna bifida. 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 pectoral muscle mass should be palpated and examined for muscle wastage, excessive fat deposition, bruising, granulomas, or other lesions. As mentioned earlier, the amount of muscle mass is often dependent on the bird's flight capability, and clinicians will need to be familiar with differences in normal pectoral musculature between species such as parrots and chickens. As most birds catabolize protein before fat when they are in a catabolic state, the pectoral muscles waste quickly in these conditions. Known to bird fanciers as "going light," this is not a disease in its own right, but rather an indication of catabolism for more than a few days.

The area between the vent and the tail should be carefully examined. A common problem in cockatiels is splitting of the skin in this area ("tail split"). This is associated with a poor diet and a poor wing clip. The wing clip causes the bird to land awkwardly, pushing its tail up as it does so. Malnutrition causes the skin to lose its elasticity, resulting in it splitting from side to side. Affected birds are usually presented for bleeding and feather picking in this area.

The pericloacal area is also a common site to locate hernias in hens, often containing the oviduct or even cystic ovaries. These need to be distinguished from lipomas or xanthomas.

THE WINGS

Robert Doneley

Each wing should be carefully extended and flexed to assess mobility and should be compared with the other wing. The bones and joints should be palpated for instability, swelling, or crepitus. Recent trauma may be evident as blue-black bruising of the soft tissue. After 2 to 3 days, this bruising will begin to turn green; this is still bruising, and should not be mistaken for infection or necrosis (birds lack biliverdin reductase; biliverdin is therefore the primary hemoglobin breakdown product). White deposits in the soft tissues around the joints may indicate the presence of articular gout. Healed fractures may be quite stable, but still prevent normal flight or wing carriage.

Neoplastic masses and xanthomas are relatively common findings, and can be located anywhere along the wing. Feather cysts are sometimes found at the base of the primary and secondary feathers and can be quite large and inflamed.

Swellings and fractures should be evaluated radiographically, as should an unexplained wing droop. The bones of the pectoral girdle are covered in relatively thick muscles, and fractures of the coracoids are often not detectable by palpation alone.

The patagial membranes should be evaluated for loss of elasticity, trauma, or scarring and for the presence of a tattoo on the prepatagial membrane (indicating the bird has been surgically sexed).

The wing tips should be examined for evidence of trauma associated with inappropriate wing trimming or wing strike against the side of an enclosure.

THE LEGS

Robert Doneley

As with the wings, each leg should be flexed and extended and then carefully palpated to detect abnormalities such as fractures, swelling, bruising, healed calluses, and angular deformities of the long bones. Each leg should be compared with the other in all aspects: symmetry, length, alignment, and swelling. Angular limb deformities can be detected by flexing the tibiotarsus-tarsometatarsus joint; the dorsal aspect of the foot should naturally align with the cnemial (tibial) crest. If the foot comes up lateral (most common) or medial (uncommon) to the crest, rotation of the tibiotarsus or tarsometatarsus may be present.

Joints should also be examined for swelling, instability, or the deposition of chalky white uric acid crystals (i.e., articular gout). The gastrocnemius tendon courses over the caudal aspect of the tibiotarsus-tarsometatarsus joint, lying within a groove. It should be palpated for swelling or luxation.

The toes should be examined for abnormalities including:

- missing digits or nails,
- annular constrictions, (see Figs. 5-24 and 5-25)
- 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, and
- excessively long or twisted nails.

The plantar surface of each foot should be examined and the condition of the metatarsal pad and digital pads noted. 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 raptors, but can be seen in any bird where a unilateral lameness causes more 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.

THE VENT

Andres Montesinos

The vent should be examined for symmetry, integrity, and hygiene. A neurologic examination should be performed on the cloacal lips to assess the cloacal reflex. The cloaca should also be examined internally by circumferential eversion with a moistened cotton-tipped swab. Normal mucosa is a bright pink color, but in anemic individuals, the tissue takes on a much paler appearance. The everted cloacal tissue should be checked for lacerations, swelling, or papillomatosis. A single acetic acid (i.e., vinegar) drop reliably turns papillomas frosty white (Figs. 5-51 and 5-52). The cloaca should then be flushed with water or saline in order to eliminate the vinegar. The cloaca also has to be palpated for the presence of cloacoliths, soft-shelled eggs, or other physical abnormalities. A relatively common functional abnormality of the cloaca is prolapsed cloaca (Fig. 5-53). This syndrome is most common in male cockatoos and egg-bound female birds of any species (Fig. 5-54). The origin in male birds is unknown, but hormonal changes are suspected.

In some avian species, mostly passerines, the vent is surrounded by a small tuft of feathers. The presence of accumulated excrement on these feathers suggests evidence of diarrhea or polyuria. To inspect the mucosa in these very small birds, the clinician can manually evert the caudal aspect of the cloaca by flexing the tail dorsally and either gently spreading or compressing the margins of the vent between the thumb and the forefinger. In the smallest species, a premoistened cotton-tipped urethral swab can be introduced into the cloaca. As the swab is slowly withdrawn, the mucosa is everted. At the caudal end of the ductus deferens is the seminal glomus that functions in the storage of sperm. During the breeding season, the left and the right glomera enlarge, pushing the vent caudally and causing the pericloacal area to appear swollen. This projection is called the cloacal promontory or the cloacal protuberance. When present, this finding makes gender determination easier (e.g., in canaries).

Ducks, geese, and ratites have an erectile, intromittent phallus inside the cloaca. In birds this organ has a reproductive function only and is not involved with the urinary system. During copulation, the erect phallus of the male bird everts and enters the cloaca of the female; after copulation, the phallus normally retracts back into the male's proctodeum. Phallic prolapse can result from mechanical damage, infection, intracoelomic-occupying space masses, or hypersexuality. Prolonged phallus eversion outside of the cloaca may lead to phallus necrosis, and amputation of this organ may be needed.

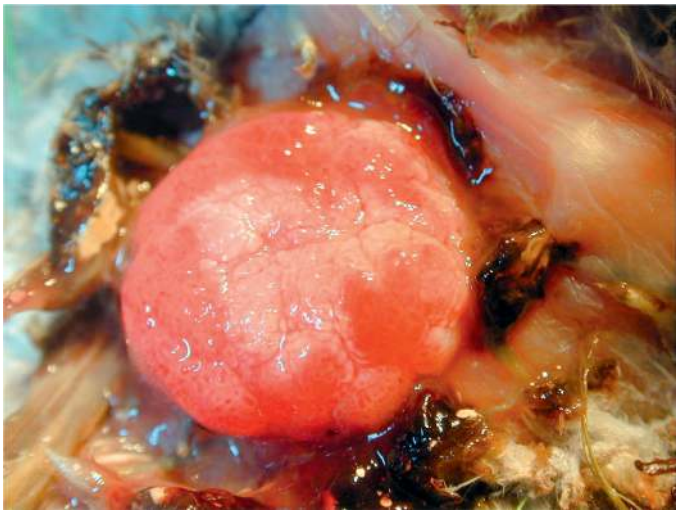


FIGURE 5-51 Close view of the reaction to acetic acid of papillomatous tissue in the cloaca of a macaw.

FIGURE 5-52 Bleeding and prolapsed tissue from the cloaca of an Amazon parrot (*Amazona* sp.). Papillomas can be revealed using acetic acid.

FIGURE 5-53 Distended and prolapsed cloaca in an African grey parrot (*Psittacus erithacus*). Early resolution of the prolapse is mandatory, but then a complete investigation should be done in order to discover the cause of the condition.

FIGURE 5-54 Chronic egg laying females are prone to cloacal prolapse, as can be seen in this African grey parrot (*Psittacus erithacus*).

FURTHER READING

Brandau J, Breaufrere H: Clinical update and treatment of selected gastrointestinal diseases in avian species, *J Exot Pet Med* 22(2):101–117, 2013.

Rosen LB: Avian reproductive disorders, *J Exot Pet Med* 21(2):124–131, 2012.

NEUROLOGIC ASSESSMENT

Robert Doneley

Birds presented for neurologic issues (e.g., abnormal posture, paresis, paralysis, or a weak grip) or the potential for neurologic damage (e.g., limb fractures) require a thorough neurologic assessment (Fig. 5-55). This assessment should be performed methodically and logically. The order in which the assessment is performed depends on the clinical condition and cooperation of the patient. The degree of cooperation of the patient should be considered when interpreting responses to neurologic tests.

The first part of the neurologic assessment is to step back and observe the bird's posture, mentation, flight, gait, and behavior; then catch the bird and physically examine it. In particular, evaluate the tone of pectoral and hind limb musculature. Muscle atrophy is suggestive of reduced innervation. Palpate the skeletal system for evidence of crepitus or other abnormalities.

A systematic evaluation of the nervous system is then conducted.

Cranial Nerve Assessment

Start by examining the eyes. The pupil size should be symmetric. If not, also consider intraocular inflammation, ocular structural lesions, or sympathetic neuropathy affecting cranial nerve III. Horner syndrome, enophthalmos, upper eyelid ptosis and slight elevation of the lower lid, piloerection of feathers on the affected side of the head, pupil constriction, and narrowing of the palpebral fissure have been reported with intracranial lesions or lesions affecting the cervical sympathetic tract or brachial plexus. Note that miosis and third eyelid protrusion are not as obvious in birds because of the presence of striated muscle in the pupil.

FIGURE 5-55 Head tilt in an Australian kestrel (*Falco cenchroides*). This is not a specific syndrome: diagnosis will require a physical examination and diagnostic testing.

Nystagmus, the periodic, rhythmic, and involuntary movement of both eyeballs in unison in either a vertical, horizontal, or rotary direction, indicates damage to cranial nerve VIII, a cerebellar lesion, or increased intracranial pressure. Strabismus, an involuntary deviation of one eye, can occur with damage to cranial nerves III, IV, or VI and vestibular lesions.

A menace response, in evaluating cranial nerves II and VII, can be provoked by bringing the hand toward each eye. Normal responses include eye-blink, pulling the head away, or an attempt to bite the hand. A symmetric eye blink normally occurs when the cornea or lateral canthus of the eye is gently touched with a moist cotton swab. If not, consider an abnormality affecting cranial nerves V and VII. The pupillary light response evaluates cranial nerves II and III. It is often considered unreliable in birds because, although there is complete decussation of the optic nerves at the chiasma—and therefore no consensual pupillary light response—the thin bones of the avian skull can allow a light shone in one eye to stimulate a response in the other eye.

The beak and oropharynx can also be examined. Reduced beak strength when biting or eating indicates possible damage to cranial nerves V, IX, X, XI, and XII. The tongue should be positioned and move normally. Abnormal tongue movement or a deviated tongue indicates damage to cranial nerves IX to XII.

Torticollis, caused by cervical muscle contraction, has been seen with lesions involving cranial nerve XI.

Peripheral Nerve Assessment

Spinal reflexes, although difficult to assess objectively in birds, can help determine if a lesion is centrally or peripherally located. Spinal reflexes require reflex arcs only to be intact—no other parts of the central nervous system are involved. Spinal reflex tests include:

a. Body balancing. With wings held in to the body, suspend the bird vertically and head down, then quickly rotate up to horizontal position and observe fanning of the tail feathers. Dip the bird forward again, back to vertical position, and observe the tail flick up. This reflex functions to maintain balance.
b. Wing withdrawal. Lightly touch a wing and observe it being pulled away. This is a segmental reflex which, if present, indicates that the

FIGURE 5-56 This umbrella cockatoo (*Cacatua alba*) is displaying a proprioceptive deficit of the legs.

FIGURE 5-57 The neurologic signs (loss of proprioception and seizuring) displayed by this galah (*Eolophus roseicapilla*) cockatoo were caused by lead toxicosis.

reflex arc and associated spinal cord segment in the cervicothoracic cord are intact. Damage may still exist higher in the central nervous system.

c. Leg withdrawal. Lightly touch a leg and observe it being pulled away. This is a segmental reflex; if present, it indicates that the reflex arc and associated segment in the thoracolumbar spinal cord are intact.
d. Vent reflex. Touch the vent mucosa with a fine object and observe it close tightly. This is also a segmental reflex, indicating that the reflex arc and associated lumbosacral spinal cord segment are intact.

Reactions, on the other hand, require reflex pathways (i.e., ascending and descending fiber tracts in the spinal cord and higher centers) to be present and functional. It is important to look closely for subtle asymmetric deficits. Comparison of spinal reflexes and reactions helps to localize lesions. If a reflex exists, but its corresponding reaction does not, a lesion exists within the central nervous system rostral to the segment involved in the reflex arc. Reactions include:

a. Proprioception can be assessed by examining the wings and legs (Figs. 5-56 and 5-57). Note the resting wing carriage. Pull one wing out of its resting position and note the time taken for its return. Birds with normally innervated wings will quickly correct displacement. Then assess the legs by knuckling the toes under the foot so that the bird is standing on the palmar aspect of the foot. This is quickly corrected in normal birds.
b. Unilateral and bilateral wing fanning should be assessed while the bird is moved up, down, and from side to side while the feet and lower back are restrained. Forceful, rhythmic fanning out of the wings should be evident and, in the bilateral test, wing movements should be simultaneous. Unilateral wing fanning will occur only in the normally innervated wing, although in the bilateral test, a lesion causing loss of afferent stimulus on one side will be compensated for by afferent stimulus from the other side and normal wing movements may occur.
c. Placing: With wings held into the body and the legs hanging freely, approach a horizontal surface (e.g., a desk top). As soon as any part of the foot touches the surface, both feet should swiftly position themselves accurately on the surface to support the bird's weight.

The differentiation between pain perception and withdrawal reflex is critical. Movement of a pinched limb does not indicate that the patient is able to feel the stimulus. Some type of conscious recognition of the stimulus is required (e.g., vocalization, attempts to escape, or biting). This part of the examination is best kept till last, so as not to influence the patient's behavior to other segments of the neurologic examination. Loss of pain perception is a poor prognostic sign. Deficits in a particular wing may only be obviously detected if its counterpart is restrained.

CONCLUSION

A thorough physical examination, complimented by a detailed patient history, provides the clinician with a starting point for the selection of diagnostic tests and treatment. The results of the examination, along with the history, should be comprehensively recorded in the patient's medical file in a format that allows other clinicians to follow the record and understand the thought processes behind it.

6

Clinical and Laboratory Diagnostic Examination

GENERAL PRINCIPLES

Jaime Samour

Accurate diagnosis of disease in the living bird depends on a series of carefully carried out investigations. Observation of the sick or injured bird should follow careful analysis of the history and other relevant records. Ideally the bird should be observed in its own surroundings without being aware of the observer. Clinical examination, which implies handling and restraint, follows observation. When the bird is in the hand, it is possible to supplement clinical examination with a range of clinical diagnostic tests.

In this section, clinical diagnostic techniques that may be carried out routinely are discussed. They primarily involve the taking of blood or other samples for laboratory investigation.

The samples that are regularly taken from birds for diagnostic purposes are as follows:

- Feces
- Urates
- Blood
- Other "normal" body products (e.g., semen)
- Biopsies
- Swabs
- Aspirates
- Feathers
- Skin scrapings

Each of the samples mentioned previously, plus others that are not listed, may be used for a variety of investigations. The tests to be carried out may dictate how the sample is taken, how it is preserved, how it is transported, and how it is processed. It is therefore an important rule before taking samples to plan carefully and to be certain that appropriate materials and facilities are available. In recent years there has been an exponential increase in our understanding of the normal parameters of wild birds, particularly in captivity, and as a result, interpretation of findings in samples has become easier and more reliable. Blood is the classic example. Techniques for the examination of avian blood have been improved beyond measure. Normal values for hematology and blood biochemistry have been established for many species and, even where these are not available, extrapolation from other species, particularly related ones, can be used successfully (Gascoyne *et al.*, 1994). There is a need to build a larger database on normal values of birds; the taking of samples for diagnostic purposes, in addition to its role in diagnosis, may also assist in this respect.

The following rules apply generally to samples for clinical diagnostic investigation:

- As a general rule, be prepared to take blood and other samples from every avian case. It is better to be prepared to take samples and then be unable to do so than to have to subject a bird to subsequent handling because equipment and materials were not available on the first occasion. Have bottles, slides, and specimen containers ready before clinical examination commences.
- Use the best-quality equipment because poor samples can yield erroneous results. Anticoagulant bottles, for example, should be recently purchased and should have been stored properly, particularly in hot climates. Microscope slides should have been cleaned and polished beforehand.
- When sampling birds, follow standard techniques and ensure the sampling is performed efficiently and humanely. This may mean limiting how much blood is taken from a particular individual, especially if the bird is in poor condition. An in-house code of practice for sampling has much to commend it.
- Ensure that all samples taken are properly labeled and recorded. Various techniques may facilitate this; for example, the use of frosted-glass microscope slides will facilitate labeling because a pencil can be used. Make sure that the slide is labeled on the same side as the sample. **Label bottles, not lids.** Such precautions are not only important from the point of view of prompt diagnosis and reducing the risk of transposing samples, but may also be relevant if there is a court case or other inquiry into the circumstances of the case or the way in which samples were prepared.
- Monitor the bird carefully after sampling, not only because this is good practice in terms of the well-being of the bird, but also because it may provide further information on the condition of the bird. For example, prolonged bleeding time after blood sampling may be suggestive of dicoumarol poisoning.
- Be aware of the possible risks to human health when taking samples and follow appropriate guidelines. If, for example, a bird is believed to have a zoonotic infection (e.g., chlamydiosis), take samples under a hood or ensure that those involved are wearing appropriate protective clothing. Do not unnecessarily expose staff or owners to hazards.

BIOMEDICAL SAMPLING

Christudas Silvanose

Table 6-1 sets out the protocol for collection, transportation, and processing of all types of samples.

BLOOD SAMPLING

Jaime Samour

Blood testing is one of the most frequently used clinical laboratory diagnostic assays used in veterinary clinical practice. Blood from birds, like blood from mammals, can yield a surprising amount of valuable information. The following assays are commonly carried out using blood samples:

- Hematology: A qualitative and quantitative assessment of blood cells and other components
- Biochemistry: Assay of various substances, normal and abnormal, in the blood
- Parasitology: Detection of protozoal parasites or other blood parasites (e.g., microfilaria)

TABLE 6-1 Protocols for the Collection, Transportation, and Processing of Samples

Specimen	Investigation	Transportation	Medium Method
		VIROLOGY	
Lesion, discharge, and organs	Direct microscopy Culture PCR	Smear or swab Viral transport media (BD UVT), at 4-10° C Swabs kept in dry sterile tubes at 4-10° C	Electron microscopy Embryonated egg culture, cell culture Use dacron or synthetic swabs Do not use calcium alginate swabs
		MYCOLOGY	
Skin scraping	Direct microscopy Culture	Sterile container at 4-10° C (39.2-50° F) Saline swab Sterile container at 4-10° C	Smear with 10% potassium hydroxide (KOH) or lactophenol aniline blue preparation Sabouraud agar, dermatophyte agar CHROMagar Malassezia
		PROTOZOOLOGY	
Feces/cloaca/ oropharynx swab	Direct microscopy Culture	Normal saline at 20-30° C (68-86° F) Normal saline at 20-30° C	Smear with normal saline preparation for trophozoites; smear with Lugol iodine or Dobell and O'Connor iodine solution for *Amoeba* cysts; potassium dichromate incubation for *Coccidia* oocysts Balamuth media for *Amoeba* spp. Unipath or Clausen media for *Trichomonas* spp.
Blood	Direct microscopy Culture	Alcohol-fixed smear Blood in ACD or CPD	May-Grünwald-Giemsa–stained smear NNN or Tobie or Wenyon media for *Trypanosoma* spp. Broth containing serum, erythrocytes, inorganic salts, amino acids, and various growth factors for *Plasmodium* spp. Cell or embryonated egg cultures for *Toxoplasma gondii*
		BACTERIOLOGY	
Aspirated fluid, bronchial wash, swabs/ postmortem samples	Direct microscopy *Mycobacterium* spp. Aerobic culture Anaerobic culture *Chlamydia* culture *Mycoplasma* culture *Haemophilus* spp. culture *Campylobacter* spp. *Salmonella* spp.	Flame fixed smear Amies charcoal media at 4-10° C (39.2-50° F) Amies charcoal media Spencer and Johnson media at 4-10° C PPLO broth or Trypticase soy broth with bovine, 2SP and calf serum, at 4-10° C Amies charcoal media at 20-25° C (68-77° F) Clark or Cary-Blair transport media Selenite broth or Cary-Blair transport media	Gram-stained smear Fluorescent microscopy for *Chlamydia* spp. Ziehl-Neelsen staining technique for *Mycobacterium* spp. Löwenstein-Jensen media Blood agar and MacConkey or EMB agar RCM, *Clostridium* agar, and blood agar with anaerobic incubation Cell culture *Mycoplasma* media Chocolate agar with X, Y factor, and 10% carbon dioxide incubation *Campylobacter* selective media or Butzler selective media. APW and TCBS for *Vibrio* spp. XLD agar, MacConkey agar, Salmonella Chromogenic agar
Swab from egg shell	Aerobic culture Anaerobic culture	Amies charcoal media Amies charcoal media	Blood agar and MacConkey or EMB agar RCM, *Clostridium* agar and blood agar with anaerobic incubation
Infertile egg contents	Direct microscopy Aerobic culture Anaerobic culture *Chlamydia* culture *Mycoplasma* culture	Sterile container Flame-fixed smear Amies charcoal media Amies charcoal media Spencer and Johnson media at 4-10° C (39.2-50° F) PPLO broth or trypticase soy broth with bovine, 2SP and calf serum	Gram-stained smear Fluorescent microscopy for *Chlamydia* spp. Blood agar and MacConkey or EMB agar RCM, *Clostridium* agar and blood agar with anaerobic incubation Cell culture *Mycoplasma* media
Animal feed	Aerobic culture Anaerobic culture	Amies charcoal media Amies charcoal media	Blood agar and MacConkey or EMB agar RCM, *Clostridium* agar, and blood agar with anaerobic incubation
Swab from incubators and hatchers	Aerobic culture Anaerobic culture	Amies charcoal media at 4-10° C (39.2-50° F) Amies charcoal media	Blood agar and MacConkey or EMB agar RCM, *Clostridium* agar and blood agar with anaerobic incubation

ACD, Acid citrate dextrose; APW, alkaline phosphate water; BD UVT, BD Universal Viral Transport media; CPD, citrate phosphate dextrose; EMB, eosin methylene blue; KOH, potassium hydroxide; NNN, Novy-MacNeal-Nicolle medium; PCR, polymerase chain reaction; PPLO, pleuropneumonia-like organisms; RCM, reinforced clostridial medium; SP, sucrose phosphate broth; TCBS agar, thiosulfate-citrate-bile salts-sucrose agar; XLD agar, xylose lysine deoxycholate agar.

- Toxicology: Assay of toxic or abnormally elevated substances in the blood (may overlap with biochemistry)
- Microbiology: Detection of bacteria and other organisms in the blood and possible culture or passage to tissue culture or other animals
- DNA and chromosomal studies
- Blood gases (e.g., P_{O_2} and P_{CO_2} levels)
- Other

Each of these techniques may require specific sampling or preservation techniques and, as outlined earlier, this must be considered before the sample is taken. The development of microtechniques in recent years has meant that relatively small samples can be accurately analyzed. This means that, often, one modest blood sample can be used for a variety of tests. However, care has to be taken to ensure that the right sample is preserved in the right anticoagulant (if appropriate) and that confusion does not occur.

COLLECTION OF BLOOD SAMPLES

The total blood volume of a bird is approximately 10% of its bodyweight. Therefore a 30-g bird will have approximately 3 mL of blood of which, in a healthy bird, up to 10% (0.3 mL) can be safely removed without any detrimental effect. This volume has to be reduced in sick birds. It is possible to run a full hematology profile on 0.3 mL of blood.

Blood collected from a bird should be of venous origin and in most birds can be taken from a basilic vein (*vena cutanea ulnaris superficialis*, Fig. 6-1), which crosses the ventral surface of the humeral-radioulnar joint (elbow) immediately under the skin; the jugular vein (*vena jugularis dextra*): usually the right, which is larger than the left (Fig. 6-2); or the caudal tibial vein (*vena metatarsalis plantaris superficialis*), which is located on the medial side of the tibiotarsus above the tarsal joint (Fig. 6-3). Samples can be collected from most species of birds under manual restraint and in the dorsal position. In larger species (e.g., ostriches, *Struthio camelus*), a suitable restraining device may be required (Figs. 6-4 and 6-5). Other methods include clipping of a toenail, although blood from this site (i.e., from a capillary bed) often yields abnormal cell distributions and artifacts and should only be used when the other methods of collecting venous blood have failed. In smaller birds, where it is not possible to remove more than one drop of blood, this can be used to prepare a blood smear.

As an example, the technique for blood collection from the basilic vein for hematology analysis is described. While the bird is in the dorsal position, extend the right wing fully and prepare the medial aspect of the humeral area using cotton wool and surgical spirit. The application of pressure with the thumb at the proximal end of the humerus will raise the basilic vein, rendering it clearly visible running along the external lateral aspect of the humerus. A volume of 0.3 to 0.5 mL of blood can be obtained using 1- or 3-mL disposable syringes and 25- or 23-gauge × ⅝-inch disposable needles, depending on the size of the bird. Bend the needle to an angle of about 25 to 30 degrees and insert it gently into the vein. Begin collecting the sample but try to avoid exerting high negative pressure because this will invariably cause the vein to collapse. It is highly recommended, while collecting the sample, to maintain steady pressure at the proximal end of the humerus to ensure a well-defined vein. Bird skin is fairly delicate and damages easily. Bleeding and hematomas can easily occur, so care has to be taken with the technique, especially in small birds, in which the loss of a few drops of blood can have a significant effect on total blood volume. Collect samples into commercially available tubes containing the anticoagulant agent ethylenediaminetetraacetic acid (EDTA; 1.5 mg/mL of blood) or lithium heparin. EDTA samples are preferred for general hematological analysis because it is not possible to estimate fibrinogen or to obtain an accurate white cell count on heparinized samples. In certain bird species (Corvidae, Gruidae, Struthionidae, Alcedinidae), mixing of blood with EDTA causes progressive hemolysis of the red cells; it is necessary to use lithium heparin as the anticoagulant.

FIGURE 6-2 Blood sample collection from the jugular vein (*vena jugularis dextra*) of a saker falcon (*Falco cherrug*).

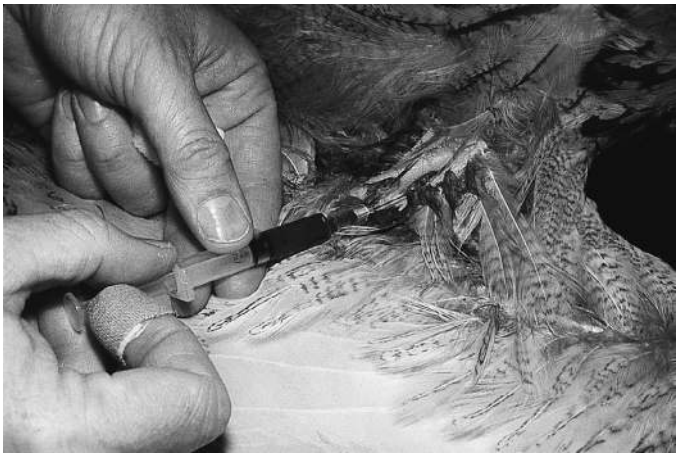


FIGURE 6-1 Blood sample collection from a basilic vein (*vena cutanea ulnaris superficialis*) of a European eagle owl (*Bubo bubo*).

FIGURE 6-3 Blood sample collection from caudal tibial vein (*vena metatarsalis plantaris superficialis*) of a kori bustard.

FIGURE 6-4 Custom-built restraining device specifically designed for subadult and adult ostriches (*Struthio camelus*). This device is made up of a frame built entirely with welded galvanized pipe of different diameters and walls and doors made up using ¾ inch- (19 mm-) thick marine plywood screwed to the frame. The interior walls and doors are padded using high density 3 inch- (76 mm-) thick foam covered with commercial grade knitted shade cloth. The front and back upper doors are open or closed depending on the height of the ostrich and the type of procedure to be carried out. Please note the wide band fixed on the interior side of the device. This is made of the same shade cloth and is used as a restraining belt to stop the ostrich from jumping.

FIGURE 6-5 Custom-built device used for restraining a subadult (10- to 12-month-old) ostrich (*Struthio camelus*) while the clinician collects a blood sample from the right jugular vein (*vena jugularis dextra*). The head of the ostrich has been covered with a cloth sleeve to ease the restraining procedure.

TRANSPORTATION OF SAMPLES

All samples collected should be well labeled, and sample submission forms should contain the following information:

- Name of owner
- Name/reference number of the bird
- Species, age, and sex
- Clinical history, presenting signs, current treatments, and differential diagnosis
- Date of sampling and time of collection
- Type of sample collected and, if applicable, anticoagulants used
- Details of site or sites from where the sample has been collected, with a diagram, if necessary for biopsies
- Indication of tests or examination required
- Name of veterinary surgeon

Note: If the samples are to be sent to a commercial laboratory it is necessary to pack them in compliance with local postal regulations.

SPECIAL CONSIDERATIONS

- The containers should be secure, leak proof, and protected from breakage, and also accessible to the staff receiving the sample.
- It is important that there is no contamination of documents enclosed or of anyone who comes into contact with the sample in the post office or in the laboratory.
- The sample should be sealed with waterproof tape to prevent any leakage.
- The container should be wrapped in an absorbable material such as cotton wool to soak up any potential leakage and help protect the container from any damage.
- The container should be double wrapped in leakproof plastic bags with the laboratory form attached in a separate bag.
- The sample should then be placed in a plastic clip-down container, lightweight metal container, or cardboard or polystyrene box.
- The sample or samples should then be placed in a "Jiffy" bag or other post office–approved packaging and correctly addressed.
- In the United Kingdom (UK), for example, it is necessary to label the package with hazard tape and the following message: "PATHOLOGICAL SPECIMEN—FRAGILE WITH CARE." This can then be sent by first-class post or dispatched by courier.

PROCESSING OF HEMATOLOGY SAMPLES

Hematology samples should be processed in the laboratory on the same day of collection if possible; if not, then blood films should be made and air-dried at the time of collection. These will keep for up to 72 hours without fixation, although it is essential that the films do not come into contact with any form of moisture. The techniques used to do full blood counts and fibrinogen are based on those used for mammals. Some modifications have been made to account for the fact that the red blood cells (RBCs) are relatively large and contain nuclei. It is useful to prioritize the order in which a sample is processed when there is a limited sample volume available.

PRIORITIES WHEN PROCESSING A BLOOD SAMPLE

These are listed in order of priority.

- *Blood smear*: A small drop of blood on a microscope slide to make a smear. Stain immediately. This enables the morphology of the white blood cells (WBCs) and RBCs to be examined.
- *PCV/Hct*: A filled capillary tube can be spun down and the packed cell volume (PCV) or hematocrit (Hct) measured.

- *WBC*: 50 µL of blood into 0.95 mL of 1% ammonium oxalate solution.
- *Hemoglobin*: 20 µL of blood into 4 mL of 0.04% ammonia solution.
- *RBC*: 20 µL of blood into 4 mL of RBC diluting fluid (formol citrate solution) into a hemocytometer.

HEMATOLOGY ANALYSES

Helene Pendl, Jaime Samour

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 diagnostic support in avian medicine. Hematology assays seldom provide an etiologic diagnosis, but they remain, nevertheless, indispensable diagnostic tools to evaluate health and disease in individuals, to monitor the response and progress of patients to therapeutic regimen, and to offer a prognosis.

The routine collection and processing of blood samples allows the evaluation of hematological responses to disease. In addition, the creation of hematology databases is important in establishing reference values for various avian species. Significant advances have been made in the use of hematology assays in the differential diagnosis of pathologic conditions in avian species in the past 15 years. This appears to have developed parallel to other areas such as nutrition, anesthesia, surgery, and therapeutics.

The processing of hematology samples has also been enhanced in recent years. In the past, automatic analyses of avian blood samples were basically limited to total red blood cell (RBC) counts using the cell counters that were available and making manual adjustments of the thresholds and current aperture settings (Coulter Counter ZF, Beckman Coulter Inc., Fullerton, Calif., USA). 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 (e.g., Cell Dyn hematology analyzers, Abbott Laboratories, Chicago, Ill., USA; Sysmex hematology analyzers, Sysmex America Inc., Lincolnshire, Ill., USA). This methodology is based on the measurement of scattered laser light, which fluctuates with the size of the cell, the complexity of the cell (i.e., overall shape, nucleus to cytoplasm ratio, granulation), and the size and shape of the nucleus after blood cells are exposed to a laser beam. This unit produces a graphic display containing a total optical white blood cell (WBC) count, WBC differential count expressed in percentage and absolute values, total RBC count, hemoglobin measurement by the cyanmethemoglobin method, thrombocyte count, and WBC count by cell-lysing impedance measurement of cell nuclei (Fudge, 1995). As with mammals, this method performs reasonably well with physiologic blood panels from healthy birds. However, it will reveal unreliable results in case of pathologic hemograms because the cytomorphologic changes, such as left shift and toxicity, result in a higher resemblance of different cell types to each other. Therefore, even in mammalian patients, a manual blood film review is recommended to confirm numeric results from the automated counting, assess cytomorphology, and search for hemoparasites, microorganisms, and cell inclusions (Allison and Meinkoth, 2007). In birds an additional obstacle has to be overcome; species-specific and individual variability of cell morphology is already present under healthy conditions. Problematic areas include the differentiation of heterophils and eosinophils; the differentiation of thrombocytes, erythrocytes, and small lymphocytes; and the differentiation of mononuclear cells into small lymphocytes, large lymphocytes, and monocytes. Consequently, extensive calibration of the software has to be carried out for each species based on time-consuming repeated manual assessments on a significant number of samples, which may outweigh the time-saving benefits of the automated count.

Recent refinement of this methodology has been carried out for chicken blood (Seliger *et al.*, 2012) by using flow cytometry in combination with an anti-CD45 monoclonal antibody and selected subset-specific markers. EDTA–blood samples are diluted, spiked with fluorescence beads, and incubated with a mixture of fluorochrome conjugated chicken leukocyte–specific antibodies. Results show that the total leukocyte, thrombocyte, monocyte, T-cell, B-cell, and heterophilic granulocyte numbers can be determined by flow cytometry in a single step without prior cell lysis, cell separation, or cell washing steps. Large sample numbers can be analyzed within hours, even from shipped specimens as blood samples can be fixed before cell staining. Comparison of this technique with conventional microscopy revealed superior precision. Currently, this methodology is available for chicken blood only; the applied tools show poor cross reactivity with other species. Future studies should facilitate the development of appropriate tools for other avian species to make this technique broadly applicable.

This section intends to combine the practicalities of a manual and an atlas by including hematological techniques and photographic identification of blood cells. The photographic section illustrates normal RBCs, WBCs, and thrombocytes, and includes some of the most common hemopathologic responses (Figs. 6-6 to 6-72).

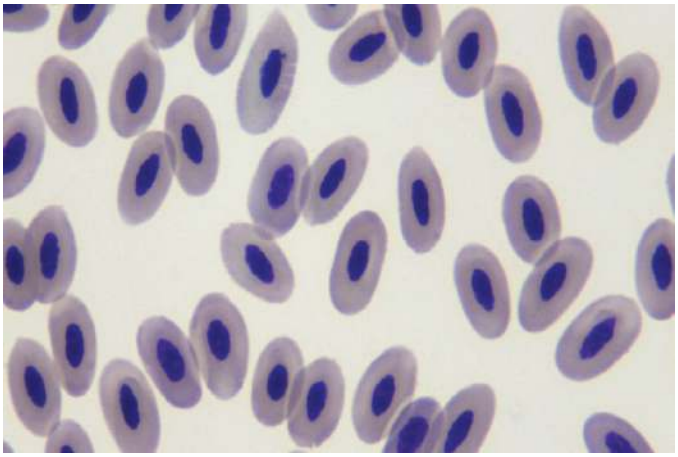


FIGURE 6-6 Normal erythrocytes of a saker falcon (*Falco cherrug*). The erythrocytes in birds are oval in shape with oval nuclei containing dense chromatin clumps. (Modified Wright-Giemsa stain.)

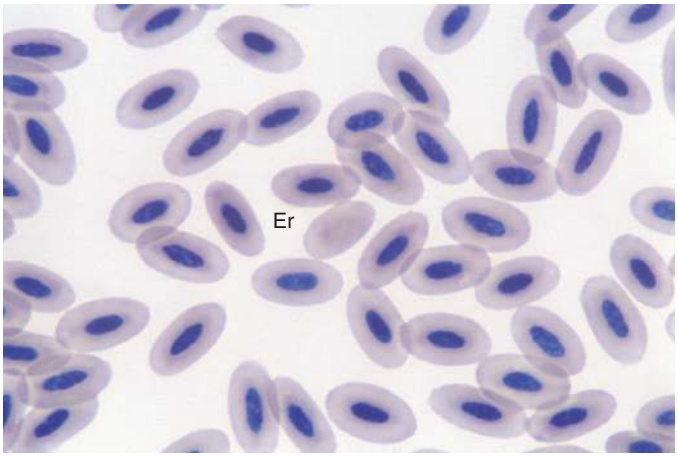


FIGURE 6-7 An erythroplastid form (Er) of a saker falcon (*Falco cherrug*). The presence of a small number of anucleated erythrocytes is relatively normal during the examination of a blood film. (Modified Wright-Giemsa stain.)

Text continued on p. 88

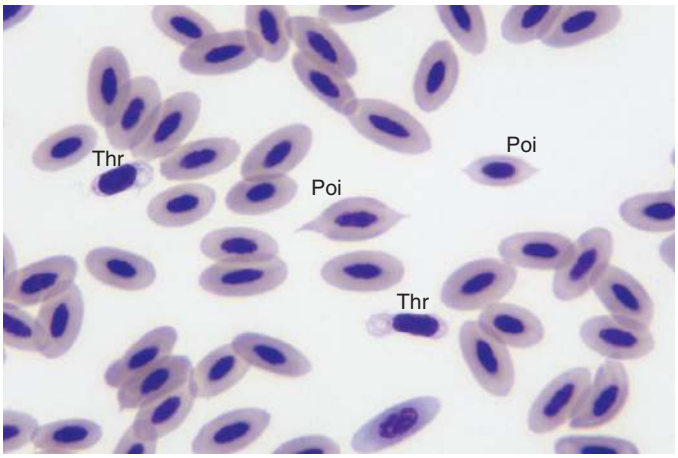


FIGURE 6-8 Poikilocytes (Poi) and thrombocytes (Thr) of a saker falcon (*Falco cherrug*). (Modified Wright-Giemsa stain.)

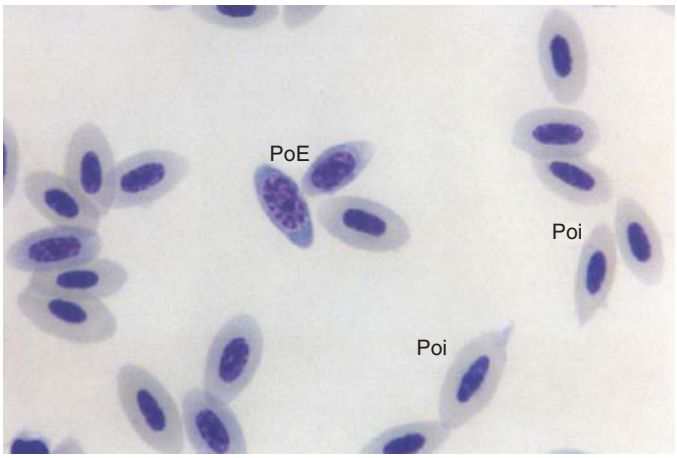


FIGURE 6-9 Polychromatic erythroblasts (PoE) and poikilocytes (Poi) of a saker falcon (*Falco cherrug*). The size of the nucleus decreases and the amount of cytoplasm increases as the cells reach maturity. (Modified Wright-Giemsa stain.)

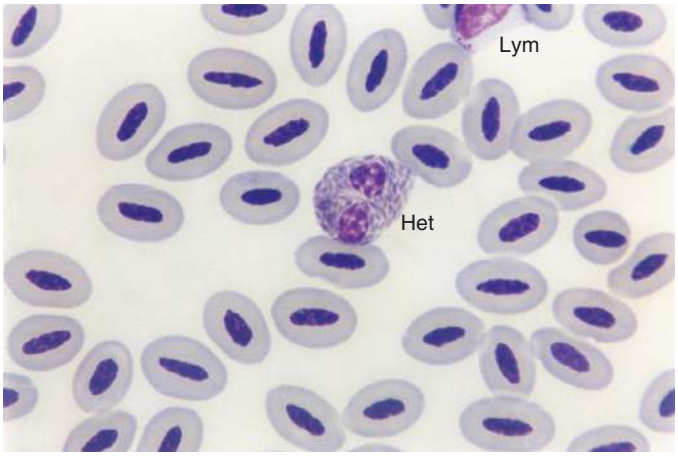


FIGURE 6-10 Normal heterophil (Het) and normal lymphocyte (Lym) of a houbara bustard (*Chlamydotis undulata*). The avian heterophil is characterized by the presence of eosinophilic rod-shaped granules within the cytoplasm. (May-Grünwald-Giemsa stain.)

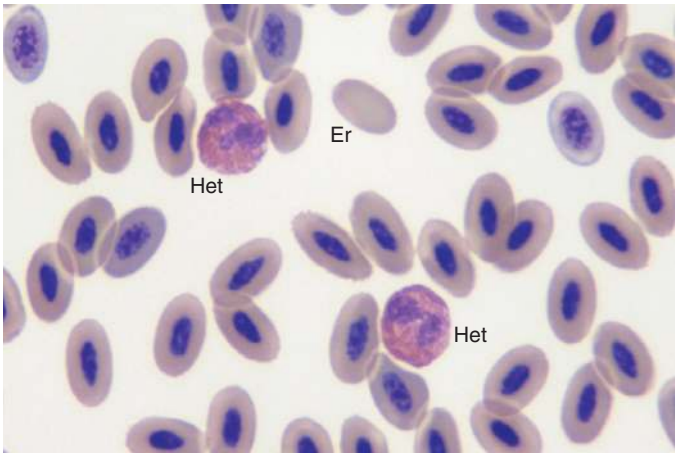


FIGURE 6-11 Normal heterophils (Het) and an erythroplastid form (Er) of a saker falcon (*Falco cherrug*). (Modified Wright-Giemsa stain.)

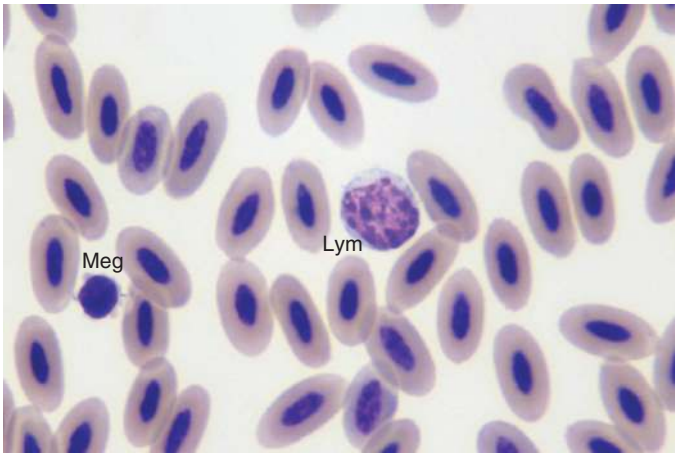


FIGURE 6-12 Normal lymphocyte (Lym) and megathrombocyte (Meg) of a peregrine falcon (*Falco peregrinus*). The lymphocyte is a mononuclear cell with a relatively large nucleus. These cells are often found compressed among two or three erythrocytes. (Modified Wright-Giemsa stain.)

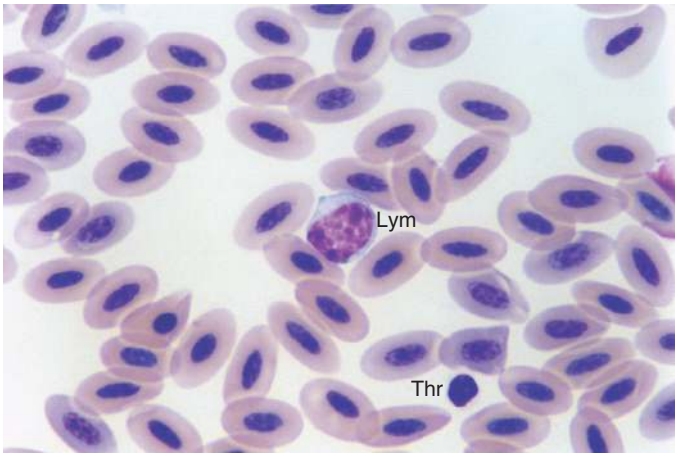


FIGURE 6-13 Normal lymphocyte (Lym) and normal thrombocyte (Thr) of a saker falcon (*Falco cherrug*). Thrombocytes in birds are characterized by the presence of strongly basophilic nuclei with a highly condensed chromatin and a vacuolated cytoplasm. (Modified Wright-Giemsa stain.)

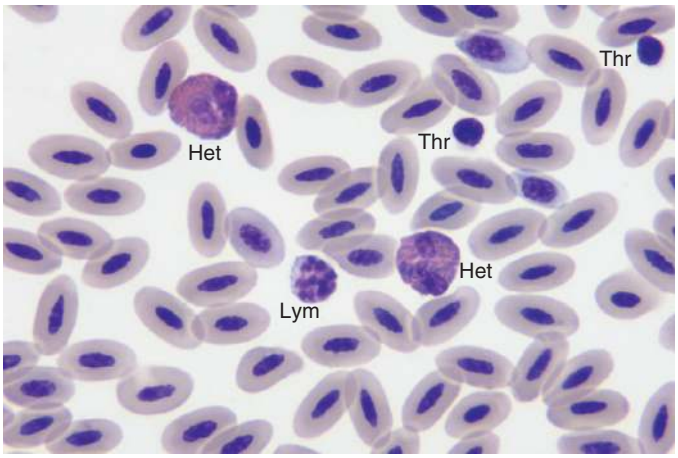


FIGURE 6-14 Normal heterophils (Het), normal lymphocyte (Lym), and normal thrombocytes (Thr) of a saker falcon (*Falco cherrug*). (Modified Wright-Giemsa stain.)

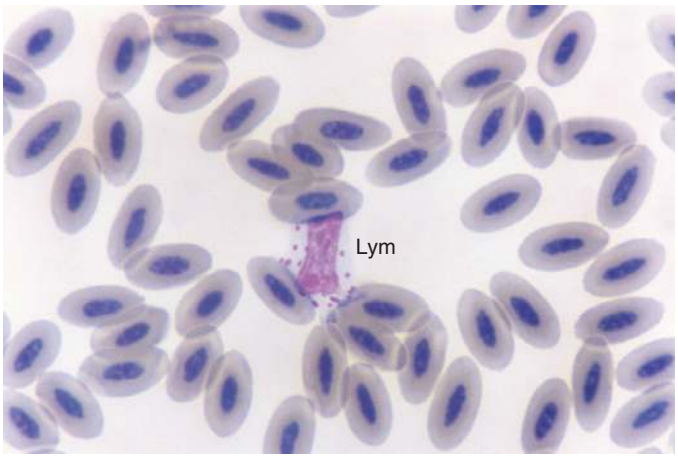


FIGURE 6-15 A lymphocyte with azurophilic granules (Lym) of a saker falcon (*Falco cherrug*). These granules can be found in blood smears from healthy individuals. However, the presence of granules in the cytoplasm has been associated with viral diseases, in particular Newcastle disease. (Modified Wright-Giemsa stain.)

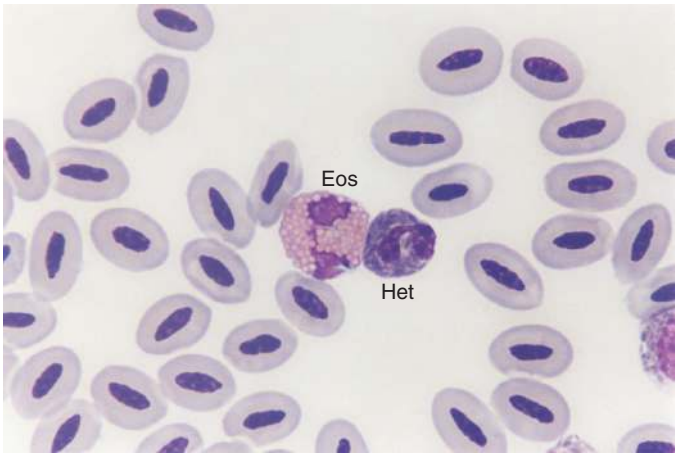


FIGURE 6-16 Normal eosinophil (Eos) and normal heterophil (Het) of a kori bustard (*Ardeotis kori*). The eosinophil of this bustard species is characterized by the presence of large, symmetric, round, red-brick-colored intracytoplasmic granules. (May-Grünwald-Giemsa stain.)

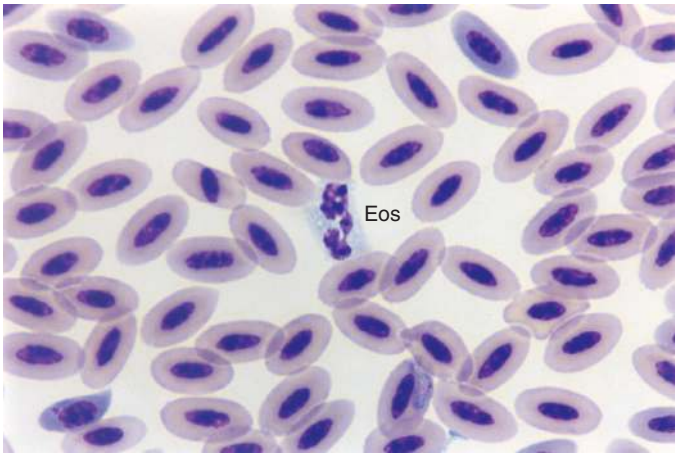


FIGURE 6-17 Normal eosinophil (Eos) of a saker falcon (*Falco cherrug*) stained with Diff Quick stain. The granules within the cytoplasm are not stained, giving the impression of numerous vacuoles.

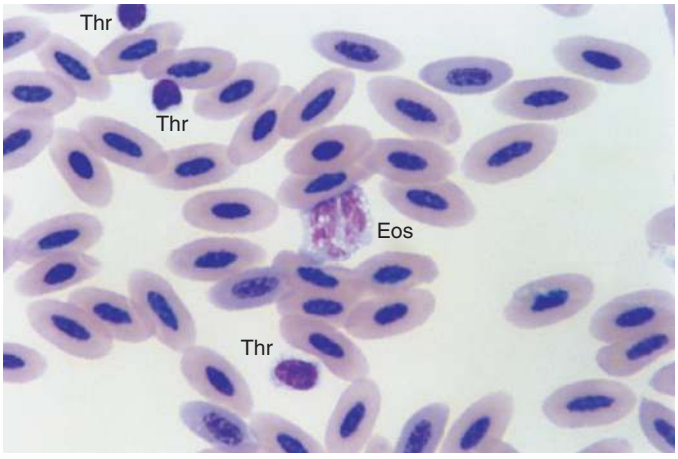


FIGURE 6-18 Normal eosinophil (Eos) and normal thrombocytes (Thr) of a saker falcon (*Falco cherrug*) stained with May-Grünwald-Giemsa stain. The granules are not stained, giving the false impression of vacuoles within the cytoplasm.

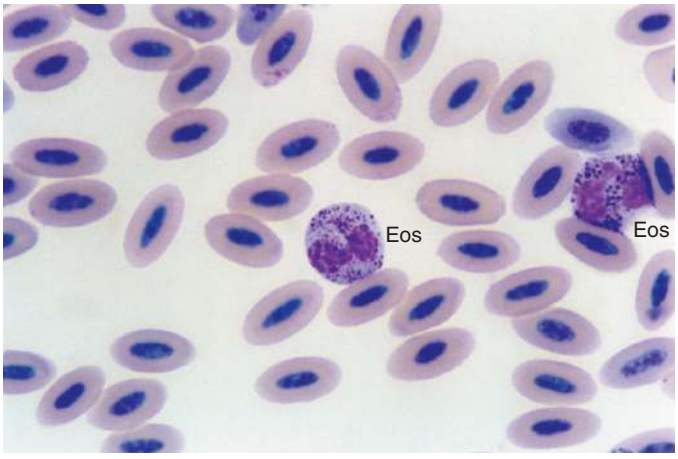


FIGURE 6-19 Normal eosinophils (Eos) of a saker falcon (*Falco cherrug*). This staining method, as described in the text, provides an excellent granular definition making the positive identification of these cells easy. (Modified Wright-Giemsa stain.)

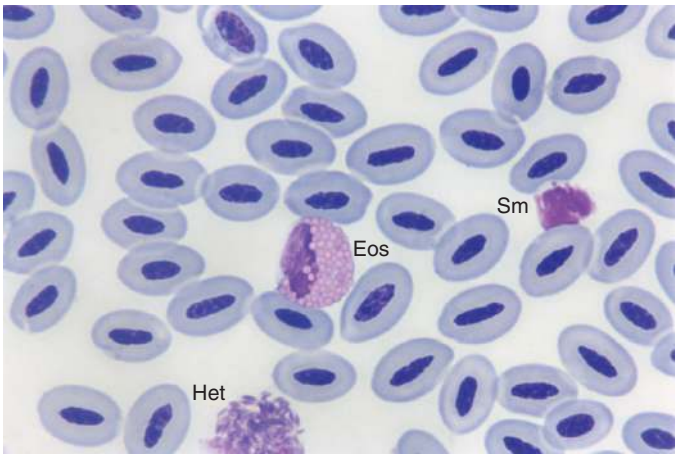


FIGURE 6-20 Normal eosinophil (Eos), normal heterophil (Het), and a smudged cell (Sm) of a kori bustard (*Ardeotis kori*). The so-called "smudged cells" are very often the remains of the nuclei of damaged erythrocytes. This tends to occur during the preparation of the blood film as a result of mechanical damage. (May-Grünwald-Giemsa stain.)

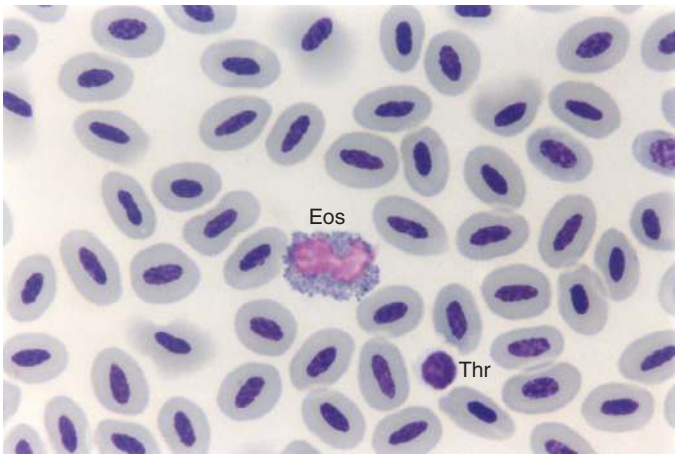


FIGURE 6-21 A slightly disrupted eosinophil (Eos) and a normal thrombocyte (Thr) of a lesser sulfur-crested cockatoo (*Cacatua sulphurea*). The medium-sized round granules are pale blue in color. (May-Grünwald-Giemsa stain.) The basophilic color of the eosinophil granules is characteristic of most psittacine birds.

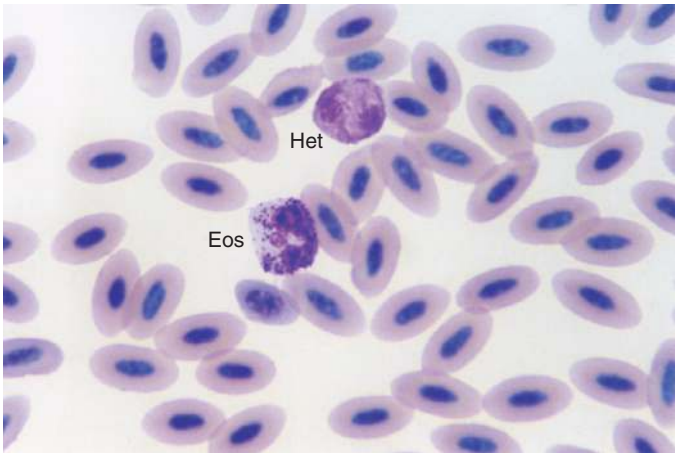


FIGURE 6-22 Normal eosinophil (Eos) and normal heterophil (Het) of a peregrine falcon (*Falco peregrinus*). (Modified Wright-Giemsa stain.)

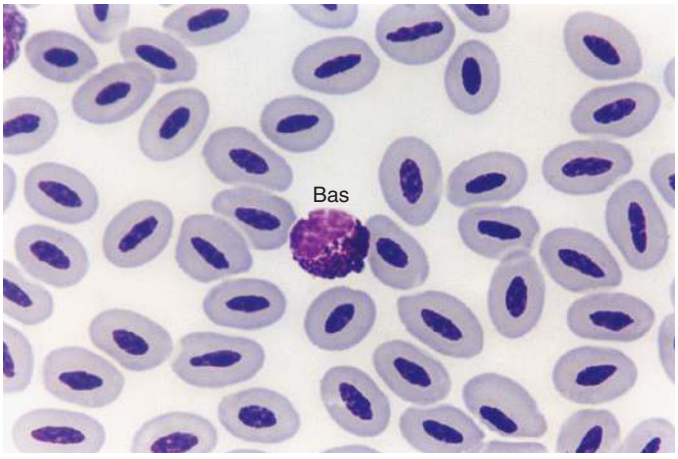


FIGURE 6-23 Normal basophil (Bas) of a houbara bustard (*Chlamydotis undulata*). The avian basophil is the smallest of the granulocytes and is characterized by the presence of strongly basophilic granules obscuring the cytoplasm and the nucleus of the cell. (May-Grünwald-Giemsa stain.)

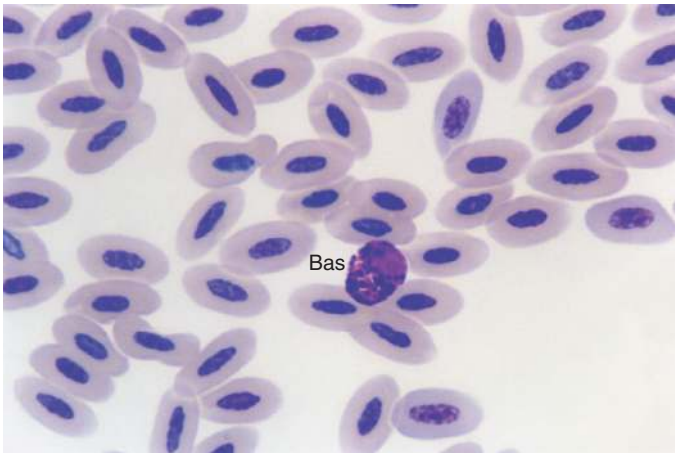


FIGURE 6-24 Normal basophil (Bas) of a saker falcon (*Falco cherrug*). (Modified Wright-Giemsa stain.)

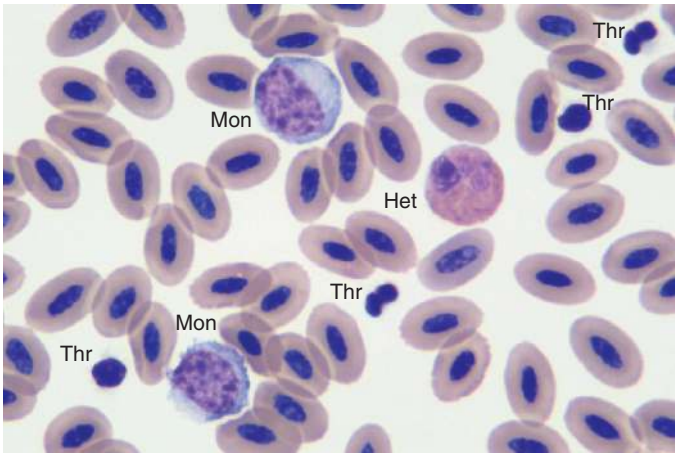


FIGURE 6-25 Normal monocytes (Mon), normal heterophil (Het), and normal thrombocytes (Thr) of a saker falcon (*Falco cherrug*). The avian monocyte is characterized by its large size. The nucleus is commonly round or kidney-bean-shaped; the cytoplasm stains pale blue with a fine granular appearance and very often contains small- to medium-sized vacuoles. (Modified Wright-Giemsa stain.)

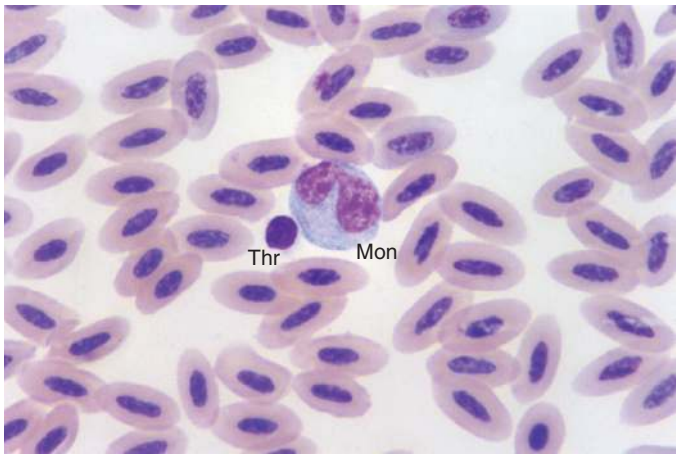


FIGURE 6-26 Normal monocyte (Mon) and normal thrombocyte (Thr) of a saker falcon (*Falco cherrug*). (Modified Wright-Giemsa stain.)

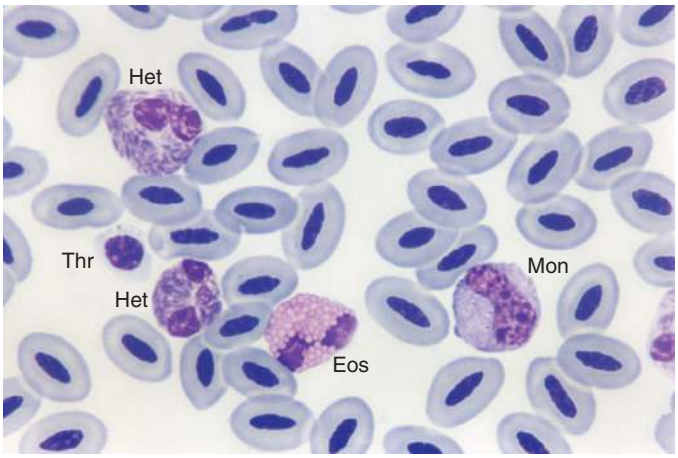


FIGURE 6-27 Normal eosinophil (Eos), normal heterophils (Het), normal monocyte (Mon), and normal thrombocyte (Thr) of a kori bustard (*Ardeotis kori*). (May-Grünwald-Giemsa stain.)

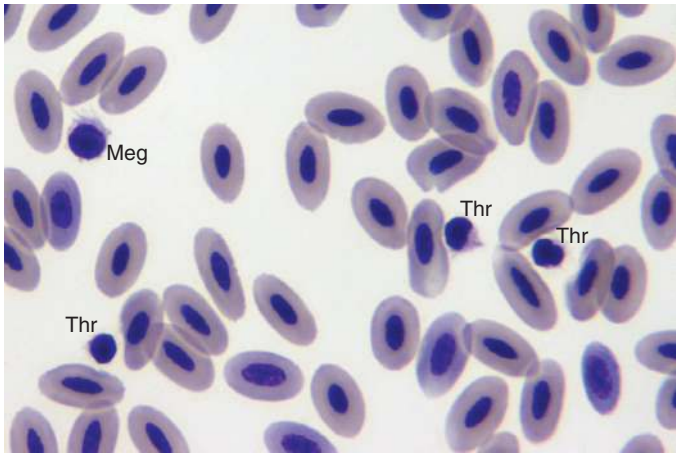


FIGURE 6-28 Normal thrombocytes (Thr) and one megathrombocyte (Meg) of a saker falcon (*Falco cherrug*). (Modified Wright-Giemsa stain.)

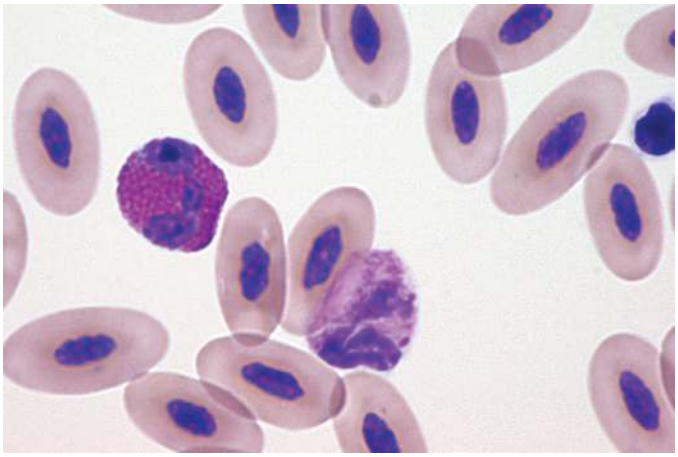


FIGURE 6-29 Common buzzard (*Buteo buteo*): position 9, eosinophil; position 5, heterophil with artificial degranulation because of improper alcohol fixation; position 3, thrombocyte. 1000× magnification. (Wright-Giemsa stain.)

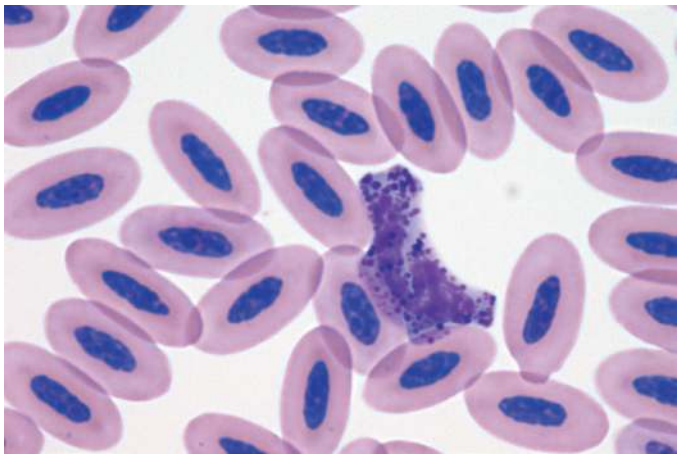


FIGURE 6-30 Saker falcon (*Falco cherrug*), eosinophil with species-specific morphologic appearance different from basophils. 1000× magnification. (Wright-Giemsa stain.)

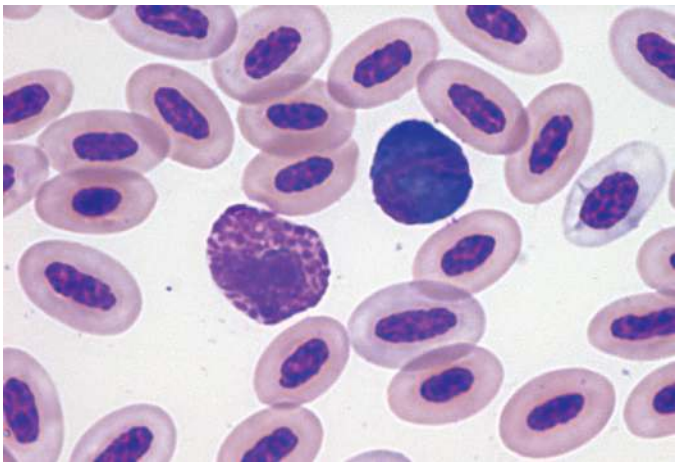


FIGURE 6-31 Bank's cockatoo (*Calyptorhynchus banksii*): position 8, heterophil; position 11, eosinophil with basophilic granulation typical for *Psittaciformes;* erythrocytic hypochromasia and anisocytosis. 1000× magnification. (Wright-Giemsa stain.)

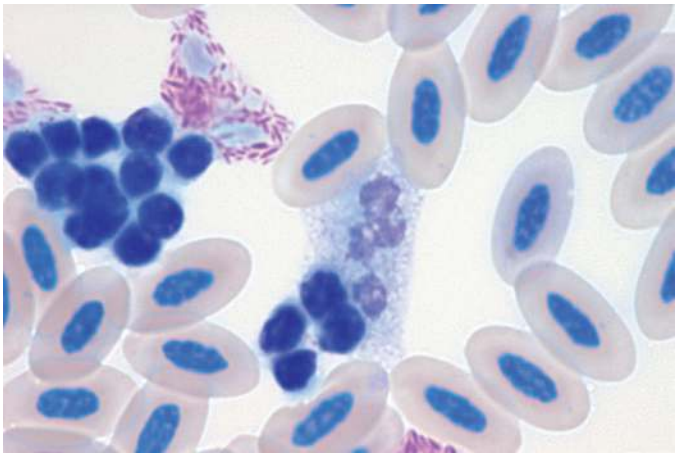


FIGURE 6-32 Oystercatcher (*Haematopus ostralegus*): center, eosinophil with fine, light blue to colorless granules; positions 10 and 11, heterophils with artificial understaining of the nucleus; positions 10 and 6, aggregated thrombocytes; typical pole bodies visible in the aggregate at position 6. 1000× magnification. (Wright-Giemsa stain.)

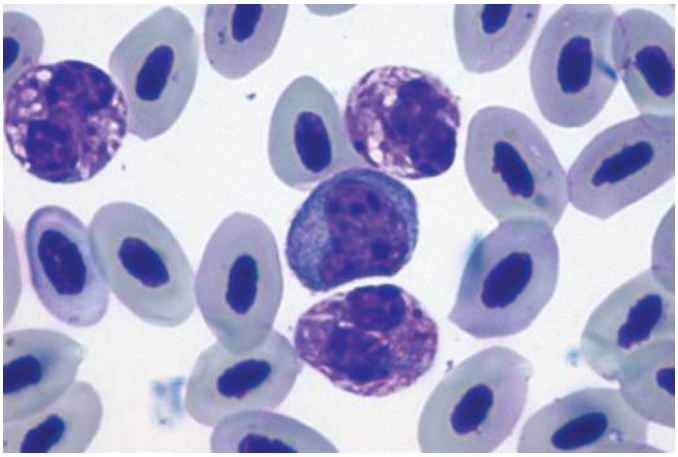


FIGURE 6-33 Blue-fronted Amazon (*Amazona aestiva*), three heterophils with morphology frequently seen in quick stains: intensive stain with loss of subcellular detail, center; reactive monocyte with visible Golgi apparatus (i.e., translucent cytoplasmic area close to the nuclear indentation). 1000× magnification. (Diff Quik stain.)

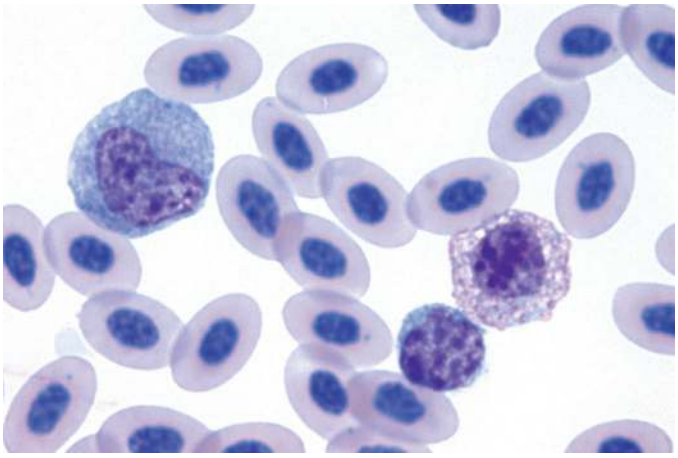


FIGURE 6-34 Domestic chicken (*Gallus gallus*): position 11, monocyte; position 4, basophil with degranulation and decoloration of granules because of improper alcohol fixation; position 5, small lymphocyte. 1000× magnification. (Wright-Giemsa stain.)

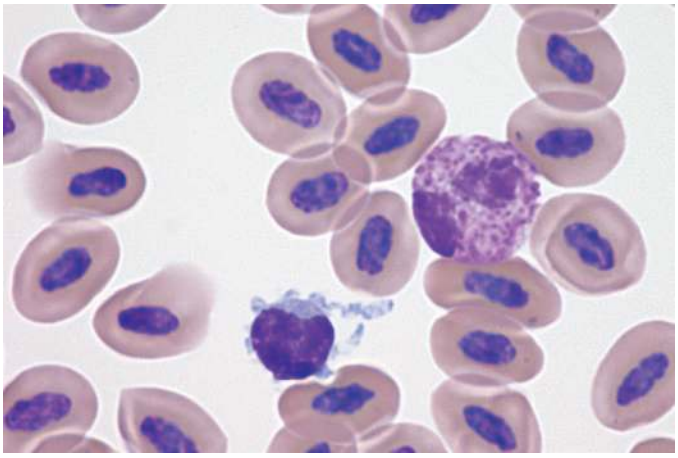


FIGURE 6-35 Green-cheeked Amazon (*Amazona autumnalis*): position 6, small lymphocyte with cytoplasmic bleb formation; position 3, heterophil with incomplete staining of granules, only the central body of each granule is well stained. 1000× magnification. (Wright-Giemsa stain.)

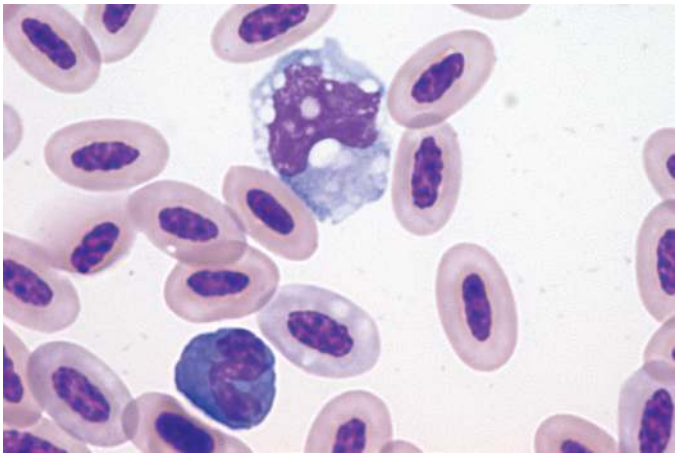


FIGURE 6-36 Bank's cockatoo (*Calyptorhynchus banksii*): position 12, monocyte with cytoplasmic vacuolation; position 7, eosinophil; erythrocytic hypochromasia. 1000× magnification. (Wright-Giemsa stain.)

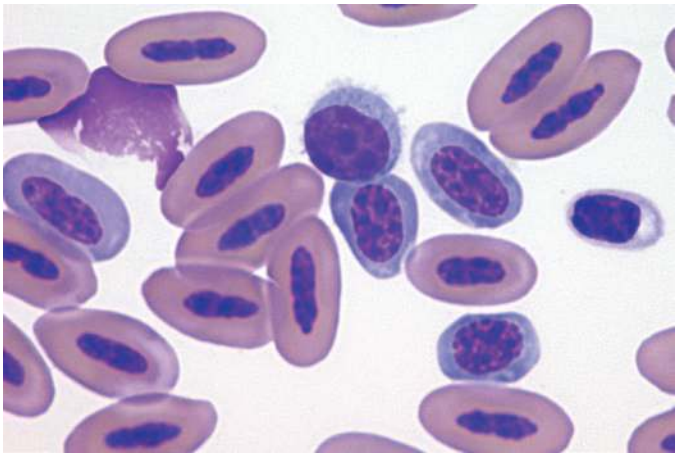


FIGURE 6-37 Blue-fronted Amazon (*Amazona aestiva xanthopteryx*) with moderate regenerative left shift (Hct 52%, PI 3 four late polychromatic erythrocytes visible): position 12, reactive small lymphocyte with bleb formation; position 3, thrombocyte. 1000× magnification. (Wright-Giemsa stain.)

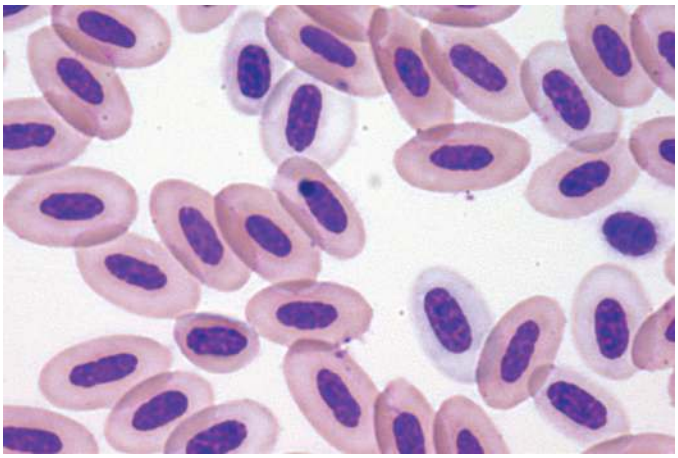


FIGURE 6-38 Red-fronted parrot (*Poicephalus gulielmi*) with hypochromic, depressive anemia (hct 29%, PI = 1): hypochromasia of erythrocytes, slight anisocytosis with microcyte at position 8; right shift (i.e., immature stages missing); position 3, thrombocyte. 1000× magnification. (Wright-Giemsa stain.)

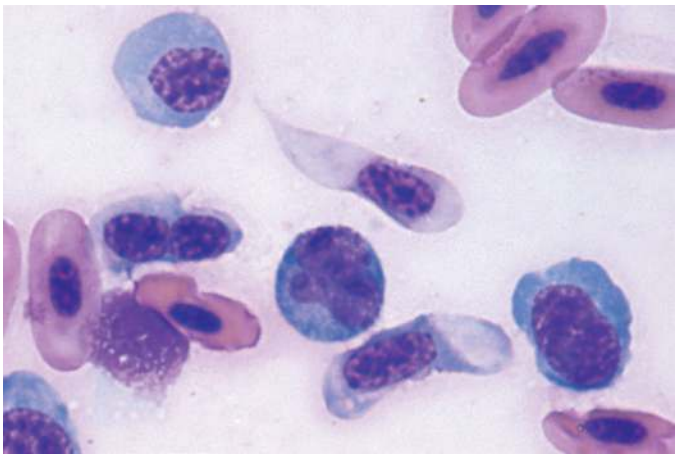


FIGURE 6-39 Sao Tome barn owl (*Tyto alba thomensis*) with massive degenerative anemia (hct 16%, PI = 5). Except for the lymphocyte with an abnormal trilobed nucleus in the center, all other basophilic cells belong to the erythropoietic line and present with more or less abnormal cytomorphology; the amitotic cell division at position 9 indicates an extramyeloid, precipitous proliferation of red cells in response to peripheral tissue hypoxia. 1000× magnification. (Wright-Giemsa stain.)

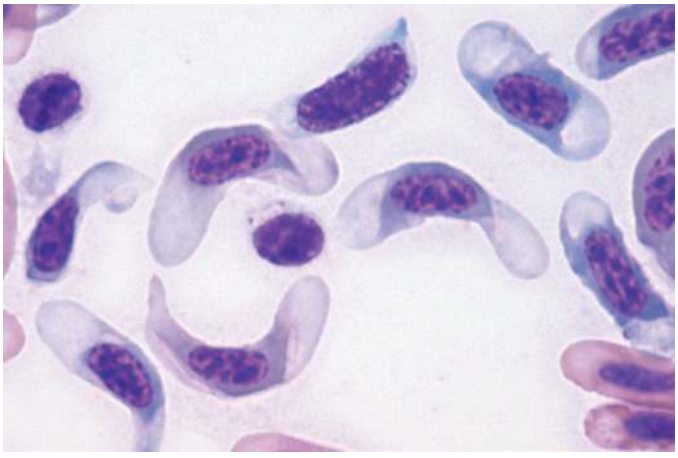


FIGURE 6-40 Same bird as in Fig. 6-3c; prominent hypochromasia and cytoplasmic poikilocytosis. Position 11 and center: two thrombocytes with well visible pole bodies. 1000× magnification. (Wright-Giemsa stain.)

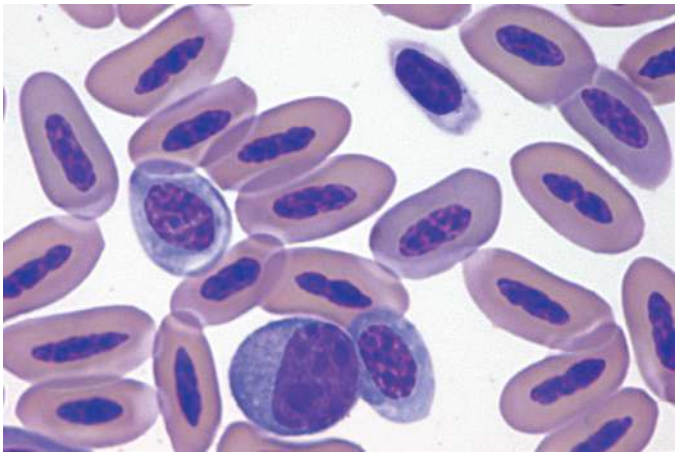


FIGURE 6-41 Blue-fronted Amazon (*Amazona aestiva xanthopteryx*) (hct 52%) with physiologic red cell morphology. Position 1, thrombocyte; position 6, large lymphocyte or monocyte; positions 9 and 5, late polychromatic erythrocytes. 1000× magnification. (Wright-Giemsa stain.)

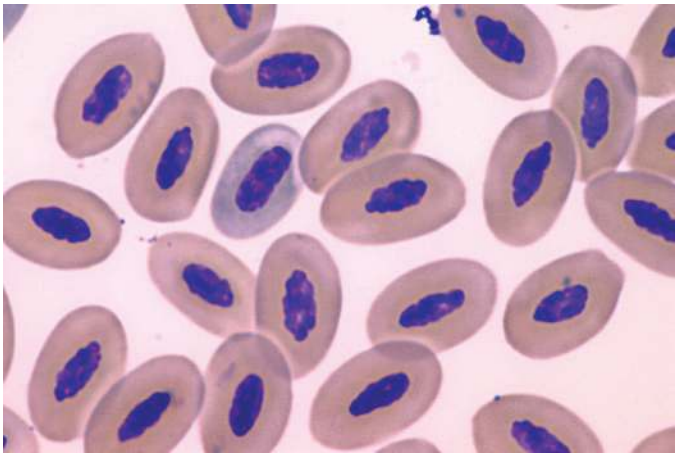


FIGURE 6-42 Blue-fronted Amazon (*Amazona aestiva xanthopteryx*) (hct 60%) with hyperchromic erythrocytes under hypoxic conditions; note the slight thickening of the erythrocytes. 1000× magnification. (Wright-Giemsa stain.)

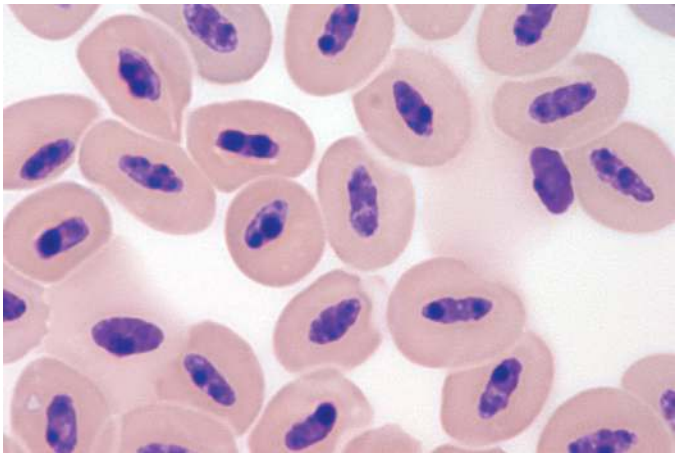


FIGURE 6-43 Blue-fronted Amazon (*Amazona aestiva xanthopteryx*) (hct 92%) with hyperchromic erythrocytes under hypoxic conditions; note the prominent thickening of the erythrocytes and tendency of hemolysis. 1000× magnification. (Wright-Giemsa stain.)

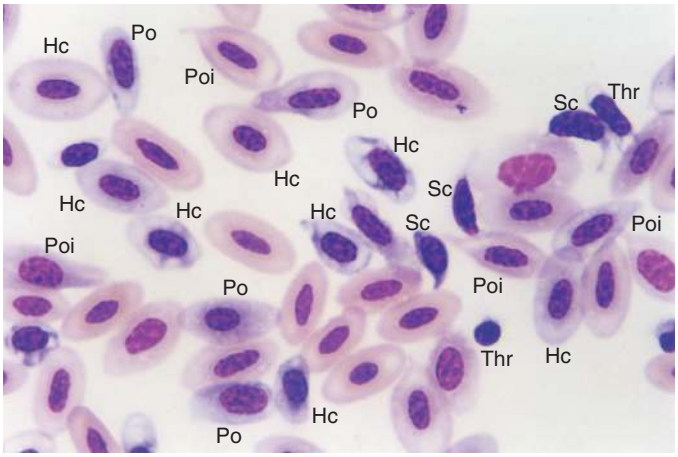


FIGURE 6-44 Red cells from a saker falcon (*Falco cherrug*) with severe anemia showing hypochromasia (Hc), poikilocytes (Poi), polychromasia (Po), a number of sickle cells (Sc), and some thrombocytes (Thr). (Modified Wright-Giemsa stain.)

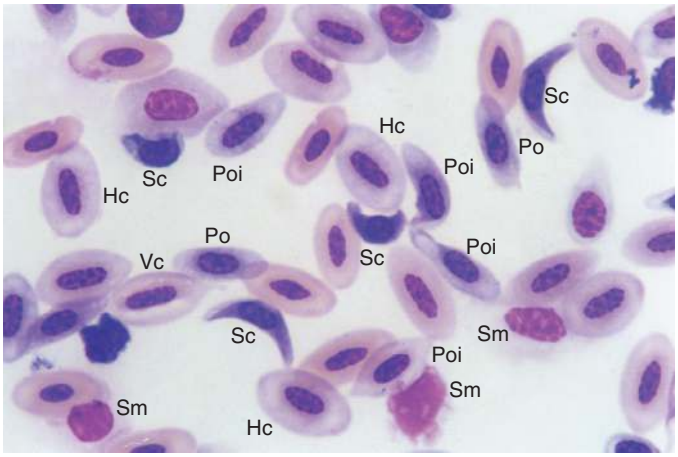


FIGURE 6-45 A blood film from a saker falcon (*Falco cherrug*) with severe sickle cell anemia showing many sickle cells (Sc), hypochromasia (Hc), poikilocytes (Poi), polychromasia (Po), vacuolation (Vc), and some smudged cells (Sm). (Modified Wright-Giemsa stain.)

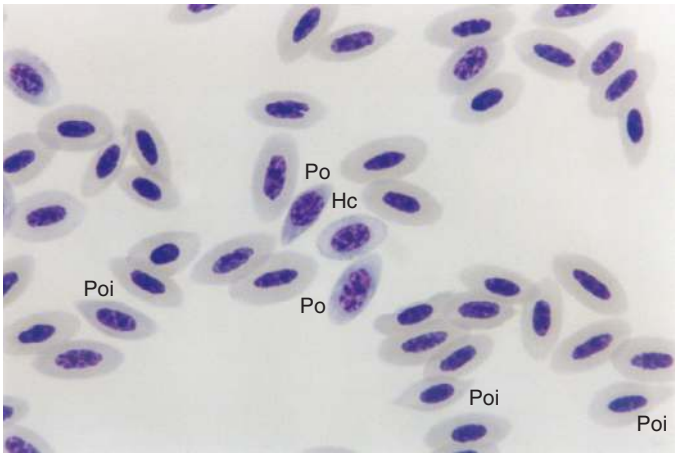


FIGURE 6-46 Poikilocytes (Poi), polychromasia (Po), and hypochromasia (Hc) in a saker falcon (*Falco cherrug*). This bird was undergoing a moderate infection with the intracytoplasmic parasite *Babesia shortii*. (Modified Wright-Giemsa stain.)

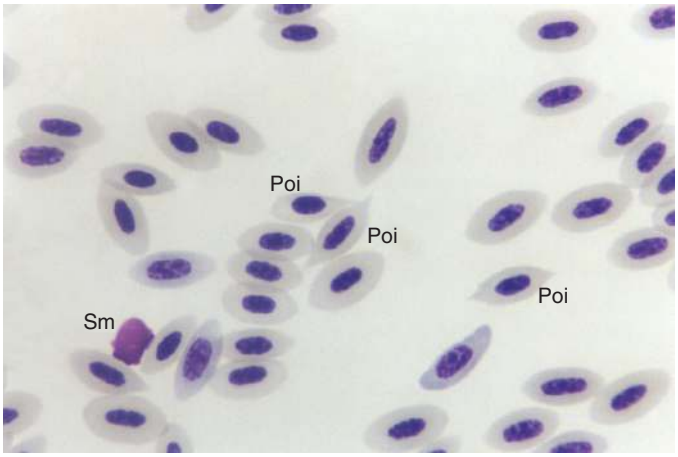


FIGURE 6-47 Poikilocytes (Poi) and a smudged cell (Sm) of a saker falcon (*Falco cherrug*). (Modified Wright-Giemsa stain.)

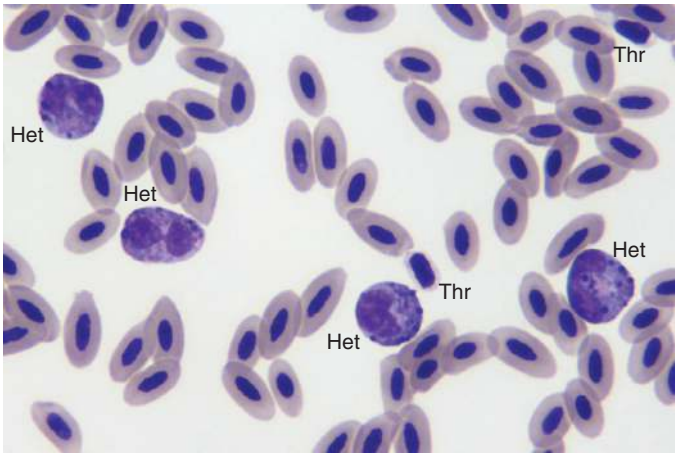


FIGURE 6-48 Toxic heterophils (Het) and thrombocytes (Thr) of a peregrine falcon (*Falco peregrinus*). Loss of lobulation (left shift) of the nucleus and degranulation. The granules are large, round, and strongly basophilic. (Modified Wright-Giemsa stain.)

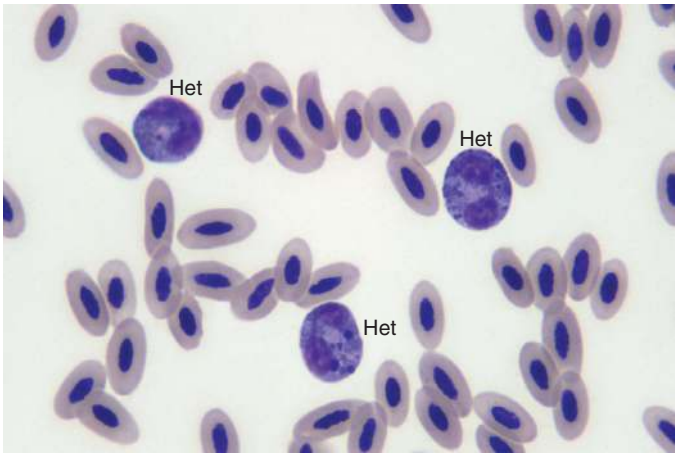


FIGURE 6-49 Toxic heterophils (Het) of a gyr falcon (*Falco rusticolus*). Severe toxic changes include loss of lobulation (left shift) of the nucleus and severe degranulation. The granules are large, round, and strongly basophilic. The falcon was undergoing a severe infection with the fungus *Aspergillus fumigatus*. (Modified Wright-Giemsa stain.)

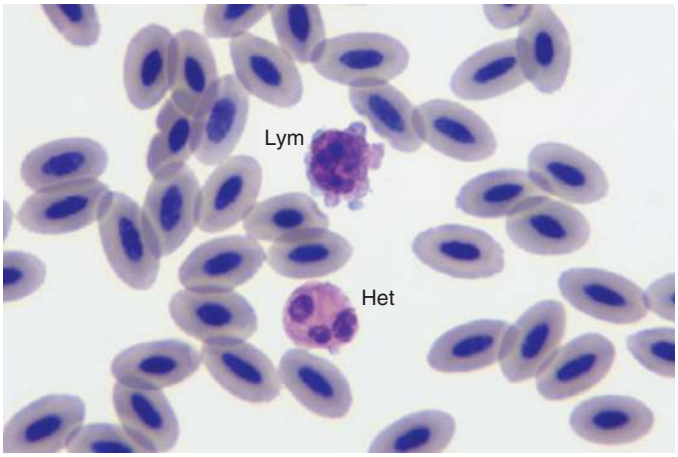


FIGURE 6-50 Reactive lymphocyte (Lym) of a gyr falcon (*Falco rusticolus*). The cytoplasm is deeply basophilic with several cytoplasmic projections (i.e., scalloped cytoplasmic margin). The nucleus is round, centrally located, and dark purple in color. A normal heterophil (Het) can also be seen. (Modified Wright-Giemsa stain.)

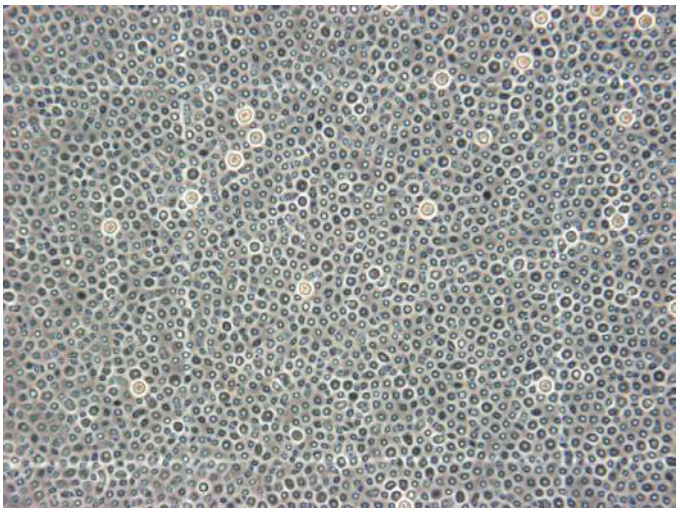


FIGURE 6-52 Microscopic view of a loaded improved Neubauer counting chamber ready for leukocyte count. The white cells or leukocytes can be observed as white shiny cells under phase contrast microscopy (400×).

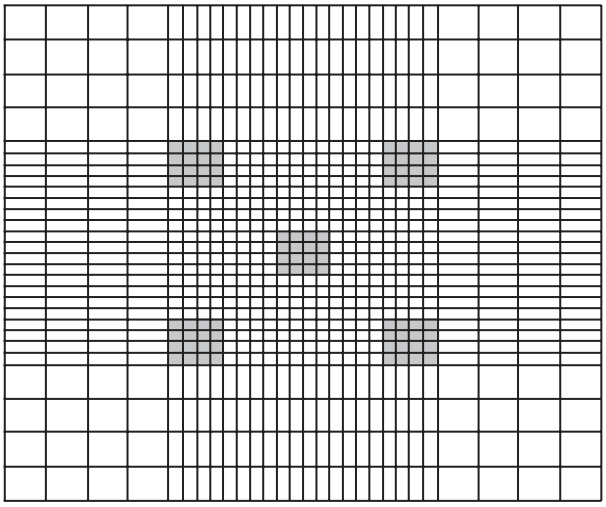


FIGURE 6-54 Diagram of counting grid of the improved Neubauer hemocytometer. The 5 × 16 shaded squares are used for the red blood cell (RBC) count.

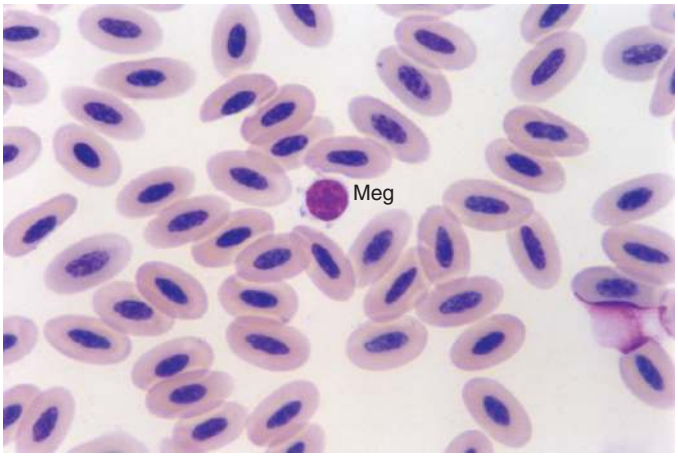


FIGURE 6-51 Megathrombocyte (Meg) of a saker falcon (*Falco cherrug*). The presence of the so-called megathrombocytes is usually associated with chronic inflammation. (Modified Wright-Giemsa stain.)

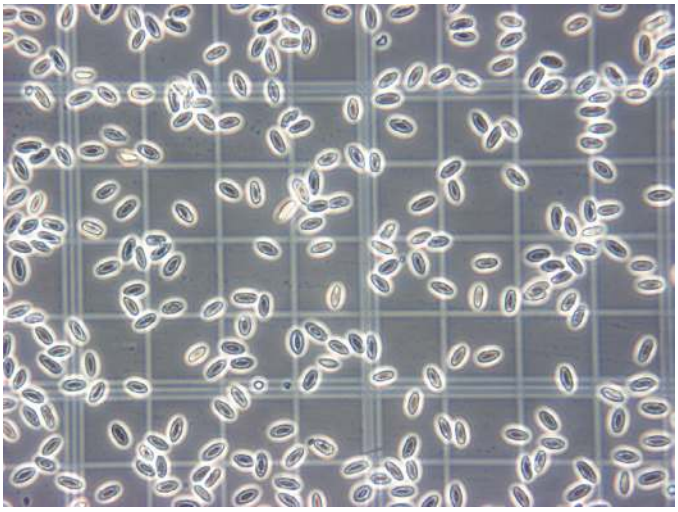


FIGURE 6-53 Microscopic view of a loaded improved Neubauer counting chamber ready for erythrocyte count under phase contrast microscopy. Counting is usually conducted following the "L" rule. Cells that touch the central line of the small squares to the left and bottom are counted (400×).

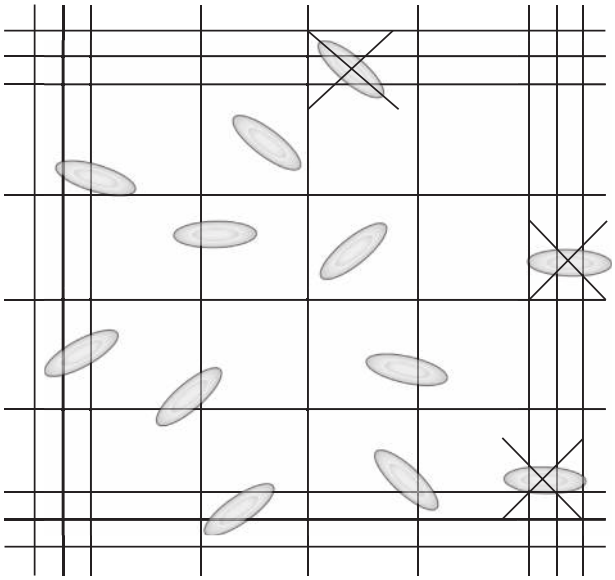


FIGURE 6-55 Diagram illustrating the position of the cells counted. Area enlarged from Fig. 6-54.

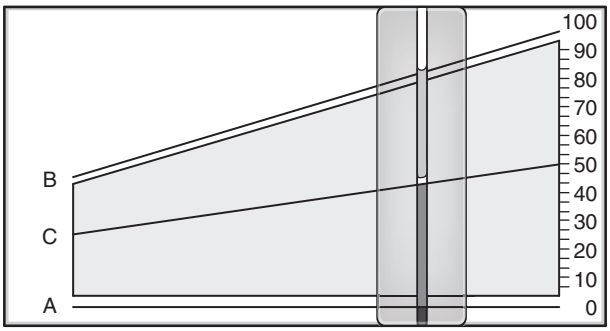


FIGURE 6-56 Diagram of hematocrit reader illustrating the method for the estimation of the packed cell volume: PCV%—hematocrit: L/L.

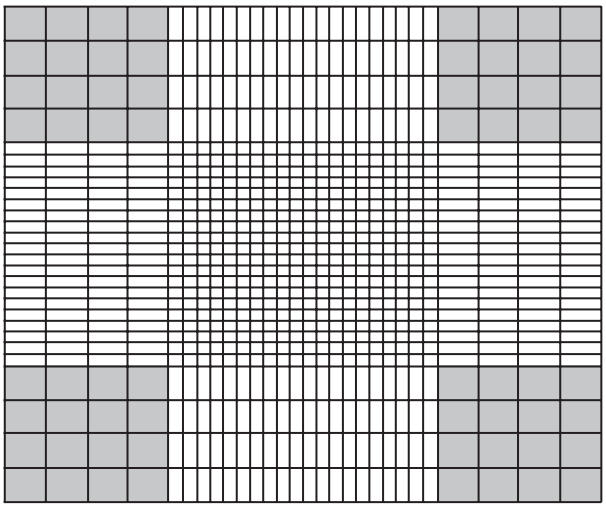


FIGURE 6-57 Diagram of counting grid of the improved Neubauer hemocytometer. The four large shaded squares in the corners are used for the white cell (WBC) count.

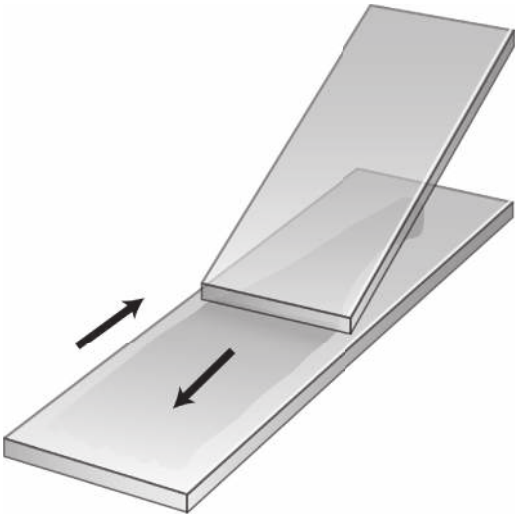


FIGURE 6-58 Diagram illustrating the preparation of a blood smear using the slide-to-slide technique. Move the spreader slide backward to touch the drop of blood gently, allowing it to run across the edge of the slide. Move forward to make the smear. Move slowly if blood runs slowly; move fast if blood runs fast.

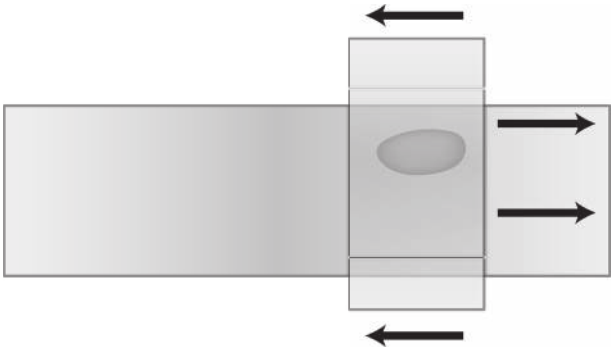


FIGURE 6-59 Diagram illustrating the preparation of a blood smear using the coverslip-to-slide technique. Place coverslip on the drop of blood. Apply gentle pressure downward. Move slide and coverslip in opposite directions to make the smear.

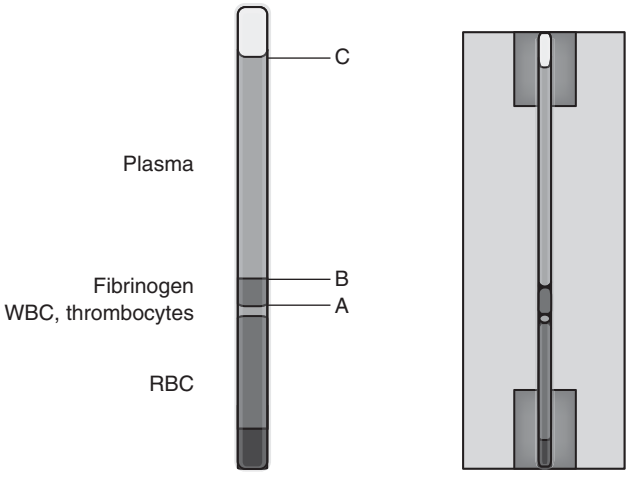


FIGURE 6-60 On the left, the diagram shows the different measurements taken for the estimation of the fibrinogen; on the right, shown is a modified microscope slide for holding the microcapillary tube during reading.

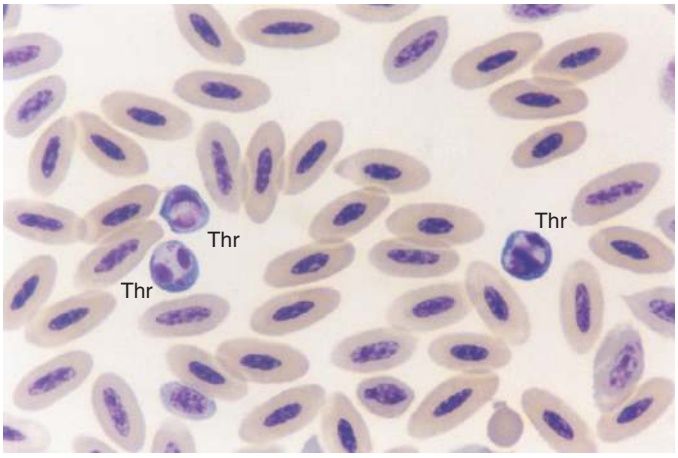


FIGURE 6-61 Abnormal thrombocytes (Thr) of a saker falcon (*Falco cherrug*). This abnormality was related to a severe hepatic disorder. (Modified Wright-Giemsa stain.)

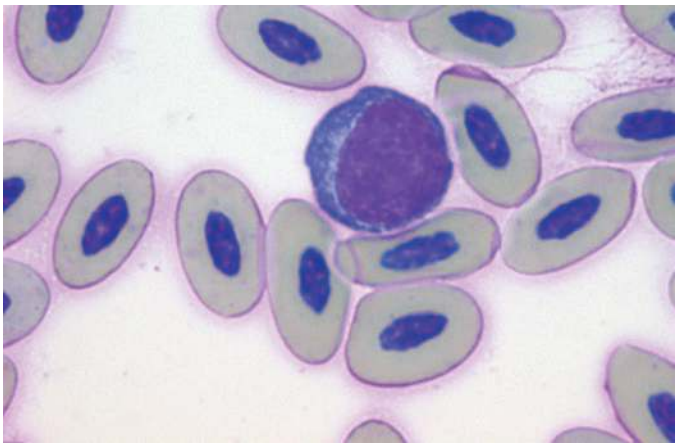


FIGURE 6-62 Japanese crane (*Grus japonica*). Normocytosis (7000 cells/μL) with left shift of both the heterophilic and mononuclear lines; poorly differentiated mononuclear cell; slightly overstained sample. 1000× magnification. (Wright-Giemsa stain.)

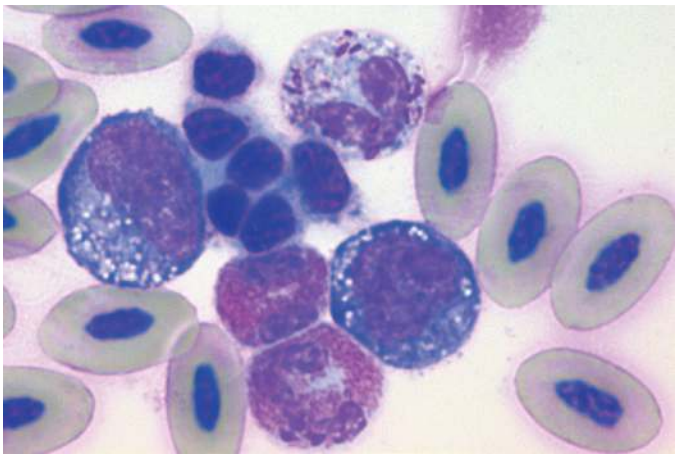


FIGURE 6-63 Japanese crane (*Grus japonica*). Normocytosis (7000 cells/μL) with left shift of both the heterophilic and mononuclear line. Position 9 and 4, two blastoid, mononuclear cells with delicate vacuolation; position 1, heterophil with grade 2 toxicity; position 6, two eosinophils; center, thrombocyte aggregate, partially with cytoplasmic bleb formation; slightly overstained sample. 1000× magnification. (Wright-Giemsa stain.)

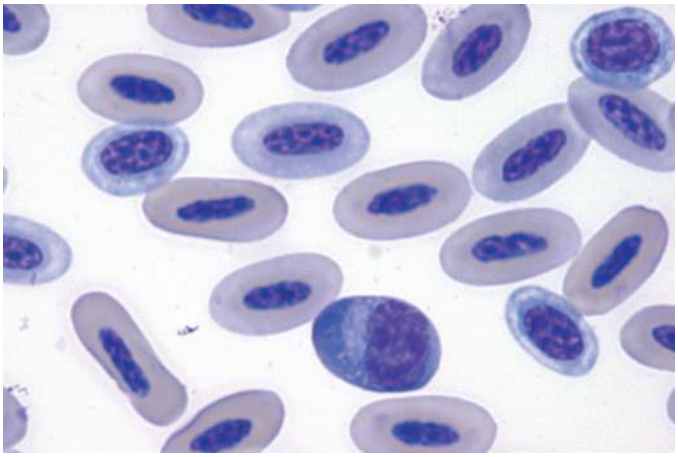


FIGURE 6-64 Sun conure (*Aratinga solstitialis*): position 6, reactive lymphocyte of plasma cell type with eccentric nucleus deep basophilic cytoplasm and well-visible Golgi apparatus (translucent area) close to the nucleus; regenerative left shift of erythrocytes (PI 3) with polychromasia and anisocytosis. 1000× magnification. (Diff Quik stain.)

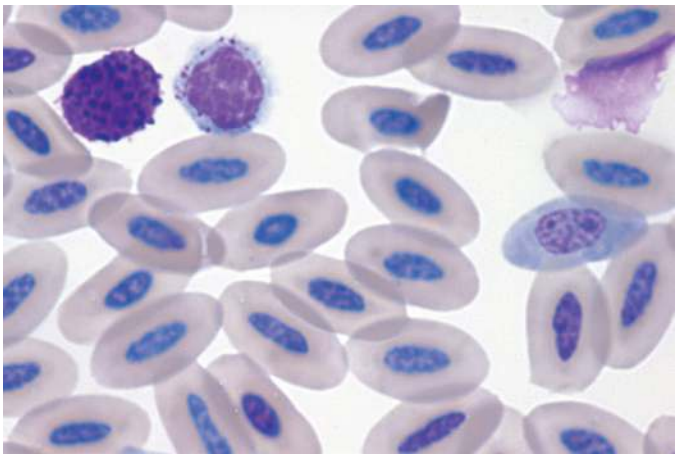


FIGURE 6-65 Turkey (*Meleagris gallopavo*). Position 11: left, basophil; right, magenta body-carrying lymphocyte possibly consistent with natural killer (NK) cells. Low physiologic numbers. 1000× magnification. (Wright-Giemsa stain.)

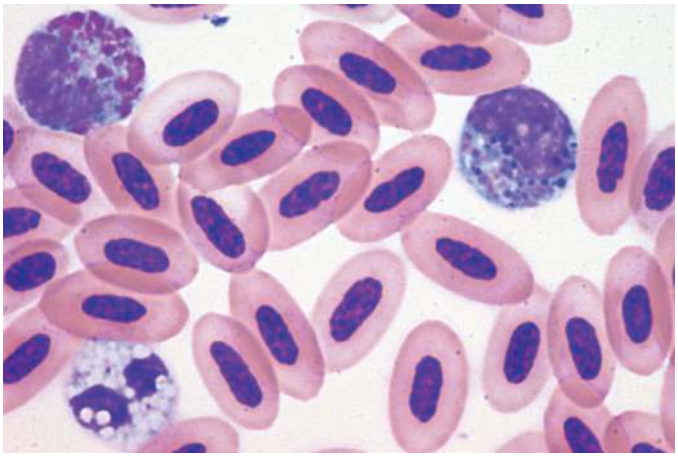


FIGURE 6-66 Jardine's parrot (*Poicephalus gulielmi*) with toxic left shift of heterophils and erythrocytic hypochromasia. Position 11, heterophil with toxicity grade 1, increased cytoplasmic basophilia, and granular swelling; position 1, heterophil with immature basophilic granules beside brick-red mature granules indicating the stage of metamyelocyte; position 7, eosinophil with colorless granules. 1000× magnification. (Wright-Giemsa stain.)

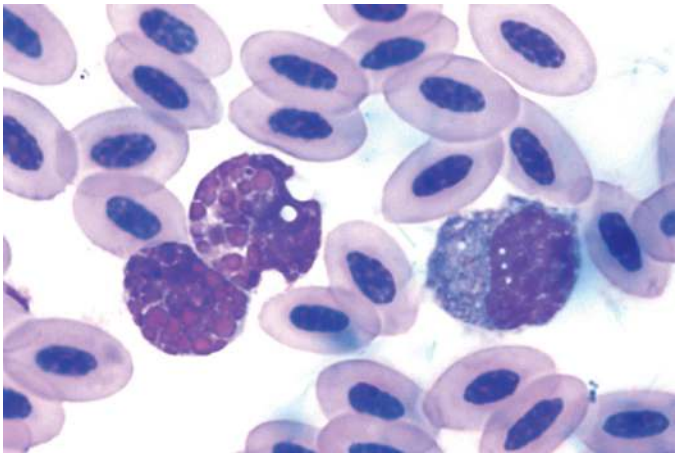


FIGURE 6-67 Saker falcon (*Falco cherrug*) with septicemia. Position 9, heterophils with toxic changes grade 3, and prominent granular swelling with cytoplasmic basophilia and vacuolation; position 3, poorly differentiated (blastoid) mononuclear cell. 1000× magnification. (Wright-Giemsa stain.)

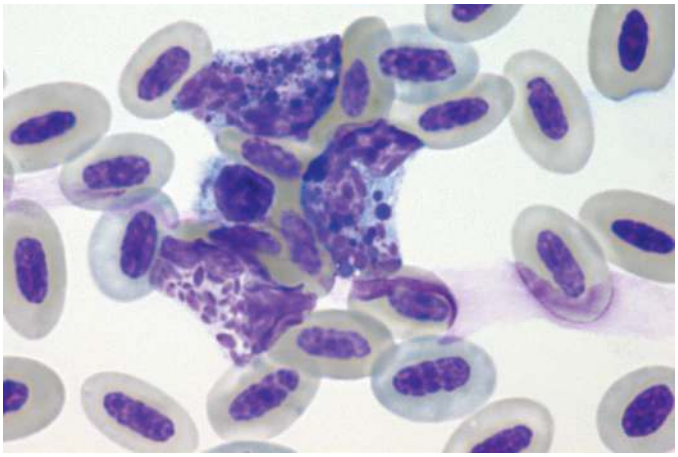


FIGURE 6-68 Blue-fronted Amazon (*Amazona aestiva*). Direct comparison of almost physiologic, mature heterophil (position 7) to two metamyelocytes (position 12 and 3); megathrombocyte (center). 1000× magnification. (Wright-Giemsa stain.)

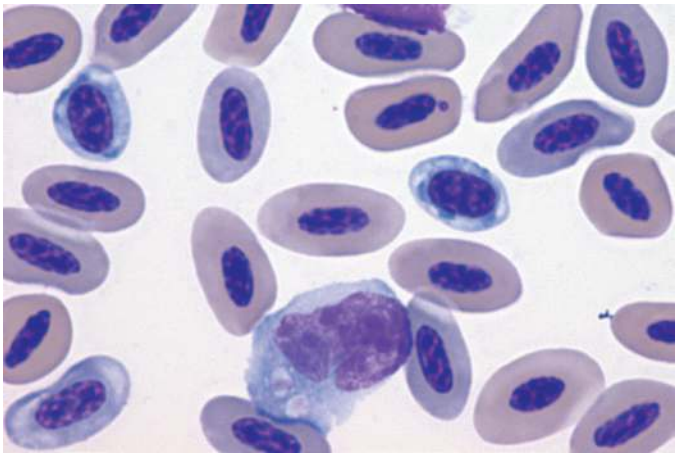


FIGURE 6-69 Sun conure (*Aratinga solstitialis*) with septicemia; showing monocyte with two phagolysosomes containing light basophilic material. 1000× magnification. (Diff Quik stain.)

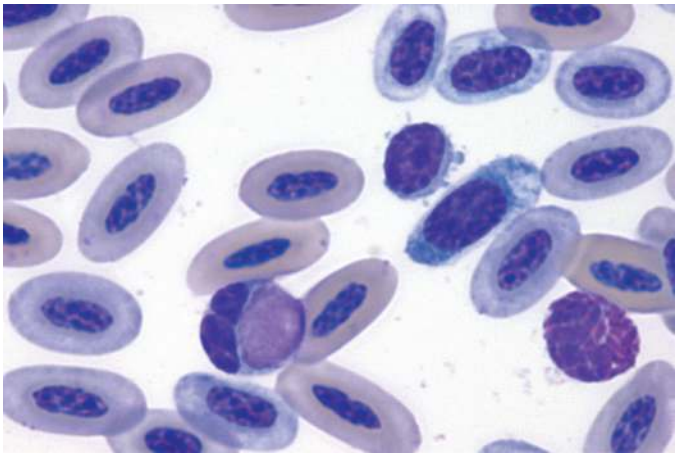


FIGURE 6-70 Sun conure (*Aratinga solstitialis*) with septicemia. Position 7, phagocyte of unknown type with large, light, basophilic phagocytized structure; positions 12-1, reactive small lymphocyte with bleb formation and several polychromatic erythrocytes; position 5, normal heterophil. 1000× magnification. (Diff Quik stain.)

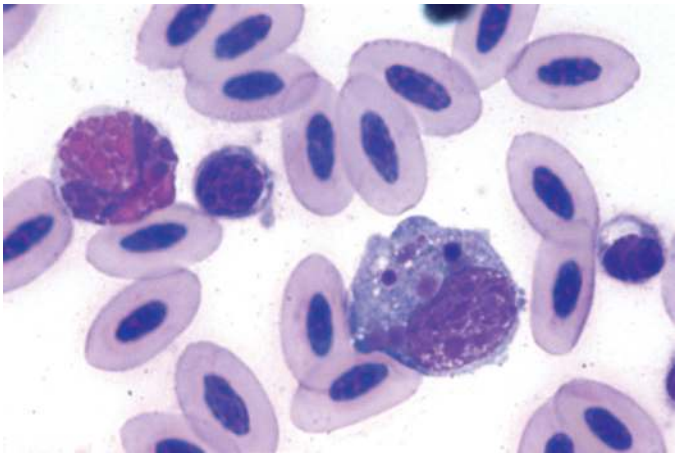


FIGURE 6-71 Saker falcon (*Falco cherrug*) with septicemia. Position 5, monocyte with several phagocytized round structures; position 9, heterophil with toxic swelling of granules and increased cytoplasmic basophilia and megathrombocyte with a single cytoplasmic bleb. 1000× magnification. (Wright-Giemsa stain.)

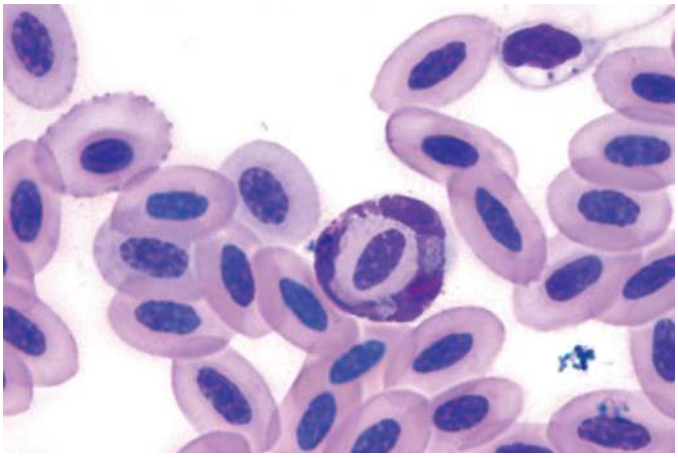


FIGURE 6-72 Saker falcon (*Falco cherrug*) with septicemia and erythrophagocytosis by heterophil. Position 1, thrombocyte with two well visible pole bodies. 1000× magnification. (Wright-Giemsa stain.)

LABORATORY TECHNIQUES

Red Blood Cell Count (RBC × 10^{12}/L)

The total RBC count is in itself an important hematology assay, but it is also essential for the estimation of the mean corpuscular volume (MCV) and the mean corpuscular hemoglobin (MCH). Many laboratories prefer to estimate RBC count using an automatic system because this is more precise than manual methods. The method described subsequently refers to a manual technique.

Working Solutions

BD Unopette 365851 Red Blood Count Manual Hematology Test (Becton Dickinson Co., NJ, USA). The Unopette 365851 system is probably the most popular method used for manual RBC count in avian species. This system uses 10 µL of whole blood into 1.9 mL of 0.85% saline, resulting in a 1:200 dilution. The use of this system will not be described in this section.

The other common systems are based on using either Natt and Herrick's solution, formol citrate solution, or Dacie's fluid, depending on whether the examination will be carried with or without phase contrast microscopy.

Natt and Herrick's Solution—without Phase Contrast Microscopy

Ingredient	Amount
NaCl	3.88 g
Na_2SO_4	2.5 g
Na_2HPO_4 $12H_2O$	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.

Formol Citrate Solution (Dacie's Fluid)—with Phase Contrast Microscopy

Ingredient	Amount
Formaldehyde 40%	10 mL
Trisodium citrate	31.3 g
Distilled water	to 1000 mL

Note: Refrigerate at 8-12° C.
Dacie's formol citrate solution is the least-known diluting fluid, but is the diluting fluid we use and recommend.

Materials and Equipment

- 5-mL disposable sample tube with lid
- Automatic dispenser 0 to 50 mL
- Micropipette 20 mL and tip
- Roller mixer
- Plain microcapillary tube
- Petri dish (8.5 cm diameter)
- Filter paper (8.5 cm diameter)
- Swab sticks
- Distilled water
- Improved Neubauer hemocytometer and coverslip
- Microscope, preferably with phase contrast facility
- Laboratory tissues

Method

Dispense 4 mL of formol citrate or Natt and Herrick's solution into the sample tube. Pipette 20 µL of blood from the sample, wipe the outside of the pipette tip, and dispense into the tube. Place tube on a roller mixer for 3 minutes. Clean the hemocytometer using a dry, clean, lint-free cloth or a tissue. Fix the coverslip firmly, making sure that Newton's rings (colored interference patterns) are present on either side of the counting chamber.

Take a small aliquot of the diluted sample using a capillary tube and fill the hemocytometer. Do not over- or underfill the chamber or allow any bubbles to be admitted during this process. Line a Petri dish with the filter paper and wet the paper slightly using distilled water. Break off two pieces from the swab sticks and place the pieces (6 cm long) on either side at the bottom of the Petri dish. Store the loaded hemocytometer on the sticks within the wetted Petri dish to avoid dehydration of the sample. Wait 5 minutes and count cells in 5 × 16 squares in the center of the counting grid (80 small squares).

$$n = \text{Number of cells counted, then: } \frac{n}{20} = \text{RBC} \times 10^{12}/\text{L}.$$

Improved Neubauer Counting Chamber

The total RBC count is performed by counting the number of cells contained in the 25 groups of 16 small squares at the four corner and central squares in the central area of the chamber. These squares are separated by closely ruled triple lines, illustrated in the drawing as thick lines (see Fig. 6-54).

Counting System

Count cells that touch the center triple line (seen here as a thick 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. 6-53 and 6-55).

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. Commercial laboratories estimating hemoglobin using an automatic hematology analyzer have to take into consideration the photometric interference of the free nuclei after lysing of the erythrocytes. 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. The method described subsequently relies on the use of a hemoglobinometer or a colorimeter.

Materials and Equipment

- Automatic dispenser 0 to 50 mL
- 5-mL disposable sample tube with lid
- Micropipette 20 mL and tip
- Roller mixer
- Toothpicks
- Cuvette 10 mm^2
- Laboratory lens tissue
- Hemoglobinometer

Working Solution

Ammonia Solution

Ingredient	Amount
Ammonia solution	4 mL (0.88 specific gravity)
Distilled water	to 1000 mL

Note: Store in refrigerator at 8-12° C.

Method

Label the sample tubes using a permanent marker. Use an automatic dispenser to transfer 4 mL of ammonia solution into the sample tube. Wait for 5 minutes to allow the working solution to reach room temperature. Aspirate 20 µL of whole blood from the storage tube using a micropipette, wipe the side of the 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 because this will cause capillary shift of blood into the tissue. Avoid immersing the pipette tip

into the diluting fluid; this is poor laboratory practice. Place the sample tube in a roller mixer and wait for 3 minutes. Decant approximately 3.5 mL of the diluted blood into the cuvette. Remove cell nuclei jelly using toothpicks. Do not touch the clear reading walls of the cuvette with your bare fingers. Clean the clear reading walls of the cuvette using laboratory lens tissue. Zero the hemoglobinometer using ammonia solution as a blank. Reading is expressed as hemoglobin (Hb) g/dL.

Dedicated Hemoglobinometers

Hemoglobin can also be estimated as azide methemoglobin by using a dedicated hemoglobinometer system (HemoCue AB, Ängelholm, Sweden; Hemo-Vet, EKF Diagnostics, Barleben/Magdeberg, Germany). These systems include reagent-preloaded microcuvettes and a photometer. The readings are carried out at 570 nm and 880 nm to compensate for turbidity within the sample.

Packed Cell Volume Estimation (PCV%) Hematocrit (Hct L/L)

PCV is an important hematology assay because it provides an easy and objective way of estimating the number of blood cells in the sample (see Fig. 6-73). The Hct is essential for the calculation of the MCV and mean corpuscular hemoglobin concentration (MCHC). In avian species, PCV and Hct are best estimated using the microhematocrit method described subsequently. The use of plain microcapillary tubes is preferable because the same tube can be used subsequently to estimate fibrinogen.

Materials and Equipment

- Plain microcapillary tubes
- Cristaseal or any other suitable plastic sealant
- Microhematocrit centrifuge
- Microhematocrit reader

Method

Fill the microcapillary tube to approximately three quarters of its length. Seal the dry end using the plastic sealer compound. Position the capillary tube correctly within the rotor and centrifuge at 10,000 *g* for 5 minutes. Determine PCV on an Hct reader. Position the capillary tube on the acrylic holder of the reader. Align, at the distal end of the tube, the demarcation line between the sealing compound and the RBCs with line A of the Hct reader. By sliding the tube holder to the right or to the left, align the marginal meniscus at the top of the plasma column with line B of the Hct reader. Position line C at the interface of the buffy layer and RBCs and read the Hct value on the scale (see Fig. 6-56).

Red Cell Indices

MCV is the expression of the average volume of individual erythrocytes calculated with the following formula:

$$\text{MCV} = \frac{\text{Hct} \times 10}{\text{RBC}} = \text{MCV (femtoliters [fL])}$$

MCH is the expression of the average hemoglobin content of a single erythrocyte and is calculated with the following formula:

$$\text{MCH} = \frac{\text{Hb} \times 10}{\text{RBC}} = \text{MCH (picograms [pg])}$$

MCHC is the expression of the volume within the erythrocyte mass occupied by the hemoglobin and is calculated with the following formula:

$$\text{MCHC} = \frac{\text{Hb} \times 100}{\text{Hct}} = \text{MCHC (g/L)}$$

White Blood Cell Count (WBC × 10^9/L)

Working Solutions

(Avian) Leukopet (Unopette 5877 Replacement)—Vetlab Supply (www.vetlab.com). The Unopette 365877 system was originally developed for the estimation of eosinophils in human hematology, but it has proved useful for determining the total WBC count in avian species. It has been replaced by the (Avian) Leukopet system since 2007. The kit contains 50 prefilled tubes containing 0.775 of 1% phloxine B diluent, a 25 µL Minipet, and disposable tips. First, 25 µL of whole blood is placed into one tube, resulting in a 1:32 dilution; this is dispersed onto a Neubauer hemocytometer with improved ruling. The properly filled and covered chamber has to be stored in a humidity chamber, such as a covered Petri dish equipped with a wet piece of cotton swab, for at least 10 minutes to allow full cell sedimentation before the count is started (Wiskott, 2002). Absolute heterophil and eosinophil numbers in the dilution are determined by counting all round, bright-shining, red-orange cells in both sides of the hemocytometer chamber (i.e., in 18 large squares).

Second, relative heterophil and eosinophil numbers are determined by performing a differential count in a stained blood film. Finally, the total WBC count is recalculated by using the following formula.

$$\text{TWBC [cells/µL]} = \frac{\text{Avian leukopette count x1778}}{(\text{heterophils} + \text{eosinophils})\,[\%]}$$

This chamber count is easy to perform because the cells of interest are clearly colored and cannot be confused with other blood cells. However, a greater margin of error in terms of falsely low counts has to be expected in cases with low granulocyte numbers. Another disadvantage of this method is that a differential count is required to obtain the total WBC count. Furthermore, separate preparations are necessary to determine the total thrombocyte count.

Natt and Herrick's (For Use without Phase-Contrast Microscopy). (See formula in RBC method earlier).

1% Ammonium Oxalate Solution (For Phase-Contrast Microscopy)

Ingredient	Amount
Ammonium oxalate	10 g
Distilled water	to 1000 mL

Note: The method described next is based on the use of ammonium oxalate solution, which is the method we use and recommend.

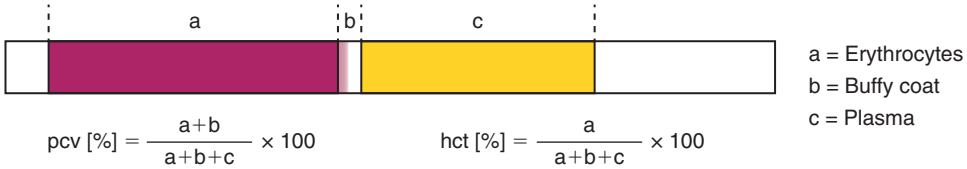


FIGURE 6-73 Cellular and liquid portions of a spinned hematocrit tube.

Material and Equipment

- 3-mL disposable sample tube with lid
- Automatic dispenser 0 to 50 mL
- Micropipette 100 mL and tip
- Roller mixer
- Plain capillary tube
- Petri dish (8.5 cm diameter)
- Filter paper (8.5 cm diameter)
- Swab sticks
- Improved Neubauer hemocytometer and coverslip
- Distilled water
- Microscope, preferably with phase contrast facility
- Laboratory tissues

Method

Dispense 1.9 mL of 1% ammonium oxalate solution into the sample tube. Pipette 100 µL of blood from the sample, wipe the outside of the pipette tip, and dispense into the tube. Place the tube on a roller mixer for 3 minutes. Clean the hemocytometer using a dry, clean, lint-free cloth or a tissue. Fix the coverslip firmly, making sure that Newton's rings (colored interference patterns) are present on either side of the counting chamber.

Take a small aliquot of the diluted sample using a capillary tube and fill the hemocytometer. Do not over- or underfill the chamber or allow any bubbles to be admitted during this process. Line a Petri dish with the filter paper and wet the paper slightly using distilled water. Break off two pieces from the swab sticks and place the pieces (6 cm long) on either side at the bottom of the Petri dish. Store the loaded hemocytometer on the sticks within the wetted Petri dish to avoid dehydration of the sample. Wait 5 minutes and count the cells in the four outer large squares of the counting grid (a total of 64 small squares) (see Figs. 6-52 and 6-57).

$$n = \text{number of cells counted, then: } \frac{n}{20} = \text{WBC} \times 10^9/\text{L}.$$

Estimated White Blood Cell Counts from the Blood Film

Estimations from the blood film require a technically perfect blood film containing a monolayer area. Practice-specific reference ranges are recommended as the magnification and the size of the view field of the microscope influences counting results. Two techniques are commonly used and are performed by counting all leukocytes in 20 consecutive view fields. The method according to Campbell (1995) uses the oil-immersion (1000×) objective, whereas the method according to Lane (1996) is performed with the 40× objective (Box 6-1). The result is multiplied by 875 for Campbell's and by 100 for Lane's method to obtain a total estimated count. The factor consists of constants such as the average number of erythrocytes per field at a normal Hct. In case the Hct value is outside the physiologic range of 40% to 50%, the result from Campbell's estimation has to be corrected by multiplication with the current Hct divided by 45%. The usefulness of estimated counts is controversial because the estimates are considered too imprecise for clinical use. It is true that estimation methods yield significantly higher counts compared with hemocytometer-based counts. Precision and correlation between the methods worsens with increasing leukocyte numbers, resulting in wider reference ranges for estimations (Wiskott, 2002). At the estimation method by Lane, this imprecision is considered in the interpretation by reporting a range spread in addition to the obtained estimated value. Compared with conventional hemocytometer techniques, however, estimations are extremely time, cost, and work saving. Moreover, after fixated and stained, the samples will stay in the same condition for a prolonged time. This allows an evaluation at a later time, which is especially useful under field conditions. With increasing experience, quick rough estimations can be performed simultaneously with assessment of cell morphology and can be reported as "normal," "increased," or "decreased," which is often all that is required for clinical interpretation or quality control (Allison and Meinkoth, 2007).

BOX 6-1 Methods for Estimated Total White Blood Cell (TWBC) Counts from the Stained Blood Film

Estimations from Blood Films

Method by Campbell (1995) Using Oil Immersion Magnification

TWBC/µLest = Number of leukocytes in 20 fields × 875 corrected if PCV outside the range of 40% to 50%: TWBCcorr = TWBC/µLest × observed PCV/45%

Method by Lane (1996) Using 40× Magnification

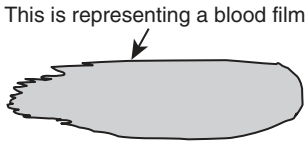
TWBC/µLest = Number of leukocytes in 20 fields × 100 report of number with range spread

TWBCest [cells/µL]	Range Spread
<25,000	2000
25,000-40,000	4000
40,000-65,000	5000
65,000-140,0000	10,000
>140,000	20,000

Example: TWBCest [cells/µL] = 40,000; range spread = 5000; range = 35,000-45,000 [cells/µL]

Differential White Cell Count Percentage and Absolute Number

To avoid frustrating repetitions because of miscount, it is highly recommendable to assess the cytomorphology of the particular sample before performing a differential count. Several consecutive fields need to be assessed to be able to identify at least one cell per cell type. Ideally, various gradually different forms of the same type are grouped into cell lineages. For the differential WBC count and absolute WBC count, the film should be examined thoroughly under high power magnification (1000×) using oil immersion. The recommended topographic site is on the shoulder of the blood film (see below), as this is the area where the blood cells are in one layer and are slightly segregated, thus facilitating examination. When using the slide-to-slide technique for blood film preparation (see later), the leukocytes tend to demix, with heterophils and monocytes located preferentially at the margins and small lymphocytes in the center. To avoid related counting errors, it is advisable to scan the slide in a continuous manner perpendicular to the blood film direction from edge to edge.



In general terms, 100 WBCs 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 WBC count is expressed as a percentage of individual cell groups. The percentage of each cell group

is then converted into absolute numbers by reference to the total WBC count using the following formula:

$$\frac{\text{Percentage of WBCs counted} \times \text{total WBC}}{100} = \text{absolute No} \times 10^9/\text{L}.$$

Fixation and Staining of the Blood Film

Fixation. It is commonly accepted that blood films can be prepared, fixed, and stained at a later date. This is incorrect. The best staining results are achieved with native, air-dried blood films prepared and stained as soon as possible after blood collection. If storage and shipment before staining cannot be avoided, the samples should be placed in a dark, dust-free, and dry slide storage box, ideally equipped with desiccation granulate. Air-dried blood films are prone to hemolysis when exposed to humidity, which is a particular problem in hot and humid environment or under cold and freezing conditions. Therefore a set of alcohol-fixated substitutes should be prepared to be on the safe side. Fixation should be performed immediately after preparation. Blood films should never be exposed to direct sunlight, moisture of any kind, or vapor from chemicals—formaldehyde in particular, because this would invariably affect cell morphology. For the same reason, blood films should never be stored in the refrigerator because humidity will cause hemolysis with certainty. In general, freshly prepared blood films should be immersed in absolute methanol (95%, acetone free) within a Coplin jar for 5 to 10 minutes and air dried. This should be done immediately after preparation. If necessary, fixed blood films can then be stored as mentioned earlier and stained at a later date. 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 to preserve the integrity of these structures. Improper alcohol fixation, such as the use of ethanol instead of methanol or aged fixatives with suboptimal alcohol concentration, will result in loss and decoloration of the granules. 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 (e.g., Wright-Giemsa stain, Leishman stain) and are used at full strength so films are fixed and stained at the same time. In case of storage within a Coplin jar, the fixative has to be checked frequently and replaced as soon as it begins to show chemical fatigue. This will depend on the number of slides fixed and the environmental conditions within the laboratory.

Staining. Most Romanowsky stains used for the staining of human and mammalian blood films are suitable for the staining of avian blood films. However, the results obtained with the various stains may be slightly different and the selection of stains is generally accepted as a matter of personal preference. Stains commonly used include Wright stain, Giemsa stain, Wright-Giemsa stain, Leishman stain, Wright-Leishman stain, May-Grünwald stain, and May-Grünwald-Giemsa stain. Commercially available quick-stain kits are widely used because of their time and cost effectiveness. They are easy and fast to perform, and will usually be used rapidly enough not to age. They are based on highly concentrated dyes with a high alcohol content, which allows them to penetrate membranes within seconds to stain the cells and subcellular structures. This rapid reaction frequently causes membrane damages, which result in a more or less severe loss of subcellular structures such as heterophilic granules, cell inclusions, and hematozoa. Basophils exhibit signs of disintegration in the majority of cases, even when using conventional stains, and sometimes disappear completely in quick-stained samples.

Automatic slide stainers facilitate the staining of a relatively large number of blood films at the same time, producing consistent results and eliminating variations that may occur with manual techniques. Needless to say, this kind 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 because of careless preparation and improper methodology. Examples include understaining of cell nuclei because of an aged Giemsa component or an increased pH of the buffer, overstaining because of inadequate staining time, and the presence of precipitates because of dust deposition or residues of color powder.

The grade of staining of heterophilic granules is variable. Occasionally, only the central bodies of the granules are stained with the usual staining protocol and appear as eosinophilic, brick-red dots in the cytoplasm. Prolonged staining time will result in full coloration of the granules in these cases.

The staining method currently used and recommended by the authors is a slightly modified Wright-Giemsa staining procedure, which is described here.

Working Stain

Ingredient	Amount
Wright stain powder	3 g
Giemsa stain powder	0.3 g
Glycerol	5 mL
Absolute methanol	to 1000 mL (acetone free)

Note: Filter and store.

Method. Prepare thin blood smears. Place them on a staining rack. Flood the smear with Wright-Giemsa stain and allow to stand for 3 minutes. Add an equal amount of Sørensen's buffer pH 6.5 to 6.8, depending on the batch of stain. Mix gently by blowing with a pipette until a metallic green sheen forms on the surface; allow to stand for 6 minutes. Rinse with buffer; allow to stand for 1 minute for differentiation. Wash copiously with buffer. Wipe the back of the smear with a tissue to remove excess stain. Prop in the rack until dry.

Note: This technique is modified from Campbell, 1995.

The placement of a coverslip over the blood smear using a commercially available mounting medium is optional. However, the optical quality of the stained blood film is increased, enhancing visualization for optimal examination and photography. Additional advantages include prevention of scratching during transport, protection against damage during excessive manipulation (e.g., use as teaching material), and conservation in high quality for years.

Morphologic and Staining Characteristics of Red Blood Cells, White Blood Cells, and Thrombocytes

Adequate knowledge of the morphology and staining characteristics of the different blood cells is of the utmost importance for the differentiation and classification of the different blood cells.

The most notable figure in the world of biological stains was Paul Ehrlich (1854-1915). He first invented a triacid stain that allowed differentiation and classification of WBCs into the grouping widely used today. This stain was replaced by an eosin and methylene blue stain

invented by Dimitri Leonidovich Romanowsky (1861-1921), which was subsequently modified by physicians such as Richard May (1863-1936), Gustav Giemsa (1867-1948), and James Homer Wright (1871-1928). In general, the widely known "Romanowsky stains" contain blue azure, which reacts with acid groups, including those of nucleic acids and proteins of the nucleus and cytoplasm; and eosin Y, which has an affinity for basic groups, in particular those 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. Tables 6-2 and 6-3 outline the morphologic and staining characteristics of different avian leukocytes. For corresponding information on erythrocytes please refer to the section "Interpretation of the Hematology Findings."

Preparation of the Blood Smear

Method. Blood films can be made from a drop of fresh nonanticoagulated 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 and the coverslip-to-slide technique. The most popular method among avian clinicians is the coverslip-to-slide technique because RBC smudging is generally minimized.

Slide-to-Slide Technique. It is highly recommended to use one-end frosted microscopic slides to write down the ID of the sample using a pencil. Wipe slides clean with a lint-free cloth or lens tissue. Use a plain microcapillary tube to withdraw a small amount of fresh nonanticoagulated 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 an angle of about 45 degrees. It is needless to say that the selected slide should be free from any indentation. To test this, pass the spreading edge over the edge of a finger nail. Move the spreader slide gently backward to touch the drop of blood and allow the blood to run across the edge of the slide. Drive the slide gently forward with a steady but firm movement to create a uniform smear. It is always good practice to make two to three good-quality blood films (see Fig. 6-58).

TABLE 6-2 Morphological and Staining Characteristics of Avian Granulocytes

Criteria	Heterophil	Eosinophil	Basophil
Plasma	Colorless; eosinophilic tinge in the case of granular disintegration	Colorless to basophil	Colorless; basophil or eosinophil
Granulation	Elliptic, eosinophilic, occ. brick-red tinge, signs of degranulation or isolated stain of central bodies (round granules)	Round (rods in Anseriformes), colorless, eosinophilic to basophilic, clearly visible, no central bodies, larger and more variable than in basophils	Basophil to reddish white caused by tendency of disintegration; rarely, densely packed with small dark-violet granules (blackberry)
Nucleus	Lobulated, less clearly structured	Lobulated, intensive coloration, clearly structured	Nonlobulated, roundish, poorly structured
Key features	Similar for all species, low in contrast, susceptible to artifacts	Species-specific variability, rich in contrast, little susceptibility to artifacts	Technique-dependent variability, tendency for disintegration, very susceptible to artifacts

Modified according to Campbell and Ellis, 2007; Lucas and Jamroz, 1961.

TABLE 6-3 Morphological and Staining Characteristics of Avian Mononuclear Cells and Thrombocytes

Criteria	Monocyte	Lymphocyte	Thrombocyte
Size	Medium to large	Small, almost without plasma to large with abundant plasma	Small to medium, occ. in case of aggregation only nuclei visible
Form	Round, irregular form in the case of contact to adjacent cells		Round to oval, polymorphic in aggregations
Plasma	Blue-gray, foamy, occ. orange-red tinge close to the nucleus, vacuoles, formation of protoplasmic blebs (sign of activation)	Homogeneous, basophil, occ. vacuoles, formation of protoplasmic blebs (sign of activation)	Colorless to slightly gray, fine reticular structure, undulated membrane (sign of activation)
Granulation	Dust-like azurophilic granules close to the nucleus	Azurophilic granules or magenta bodies (sign of activation)	Reddish to violet pole bodies close to the nucleus
Nuclear position	Rather excentric	Rather central	No preference
Nucleo : cytosomal ratio	Moderate to high	Low to moderate	Low to moderate
Nuclear form	Rather irregular, occ. kidney shaped with more or less deep, smooth indentation	Roundish, occ. sharp indentation caused by artificial folding	Round to oval, polymorphic in the case of aggregation
Nuclear structure	Small chromatin clumps integrated into nuclear reticulum, light appearance	Coarse chromatin clumps within the nuclear reticulum, very dense in small lymphocytes	Dense to pyknotic, dark appearance
Key features	Large, delicately structured cell with clearly visible details	Medium- to large-size cell with coarse structures and clearly visible details	Small- to medium-size pyknotic cell with poorly defined structures

Modified according to Campbell and Ellis, 2007; Lucas and Jamroz, 1961.

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 (see Fig. 6-59).

Thrombocyte Count (10^9/L)

Method

Count thrombocytes while performing the differential WBC count. The absolute number of thrombocytes in the sample is subsequently calculated by using the following formula:

$$\frac{\text{Number of thrombocytes counted}}{100} \times \text{WBC count} = \text{Thrombocytes } 10^9/\text{L}.$$

Fibrinogen Estimation (g/L)

Material and Equipment

- Microcapillary tube rack for use in waterbath
- Microhematocrit centrifuge
- Water bath at 56° C ±1° C
- Microcapillary tube holder
- Microscope with measuring eyepiece and stage Vernier scale
- Timer

Method

After the measurement of the PCV, place microcapillary tubes in the rack and immerse in waterbath at 56° C for 3 minutes. Make sure the entire plasma column is immersed. Remove the capillaries and centrifuge again at 10,000 *g* for 5 minutes. Place in the microcapillary tube holder and, using the measuring eyepiece and the stage Vernier of the microscope, take readings at the upper and lower limit of the protein layer and at the upper limit of the plasma column (see Fig. 6-60).

The fibrinogen is estimated using the following formula:

$$\frac{B - A}{C - A} \times 100 = \text{fibrinogen (g/L)}.$$

Note: It is essential to perform this test on samples collected into EDTA because the test is invalidated on samples stored in heparin or on samples containing clots.

INTERPRETATION OF THE HEMATOLOGY FINDINGS

Helene Pendl

General Considerations

Assessment of the hemogram gives information on the status of circulating blood cells at a single time point and allows conclusions on the current general health status of the patient. Aspects to consider when interpreting a hematologic result include the comparison with reference ranges, physiologic and technical causes, the severity or frequency of changes, the tendency of development, the function of the cells affected, and correlations to other results (Pendl, 2008). Hematology is a very sensitive but poorly specific diagnostic tool. Factors such as age, gender, hormonal status, and environmental conditions all have an effect on the hemogram already under physiologic conditions. Thus, apart from hemoparasitemias and leukemic neoplasias, etiologic diagnoses are rarely made from hematology only. Different techniques and definitions of reference groups additionally contribute to a high variability of reference values for birds given in literature. To improve interpretation, practice specific reference ranges should be established. Serial blood tests at a distance of 2 to 3 days are helpful to obtain information on the progress of a disease, the prognosis, or the efficacy of a treatment. In the case of very valuable birds, individual profiles generated by regular health checks are recommended.

Systematic Approach to Evaluation and Interpretation

Final interpretation of results should be performed stepwise in a logical order. First, numeric and morphologic findings are summarized to a hematologic diagnosis with special attention to cell function (Campbell and Ellis, 2007). In a second step, the hematologic diagnosis is combined with results from other investigations to generate a list of differential diagnoses in descending order of probability. This separation of the hematologic diagnosis from the etiologic differential diagnosis is essential to maintain a broad assessment of the situation.

Changes to the Erythrogram

Erythrocytic alterations always reflect changes of erythropoietic activity and are strongly related to tissue oxygen tension (Po_2). Chronic tissue hypoxia increases erythropoiesis and may be caused by atmospheric hypoxia in high altitudes (Calle and Stewart, 1987; Sturkie and Griminger, 1986), cardiovascular or respiratory disease (Fudge and Reavill, 1993; Taylor and Hunter, 1991; VanDerHeyden, 1994), vitamin or mineral deficiency (Blalock and Thaxton, 1984; Julian *et al.,* 1986), hemorrhage, and hemolysis. At a progressed stage, the first three possibilities result in an absolute polycythemia with an elevated hematocrit (Hct), whereas blood loss and hemolysis lead to anemia with decreased Hct. Chronic iron or protein deficiency, such as in chronic emaciating disease, will lead to a decreased erythropoietic activity with subsequent depressive anemia. Table 6-4 summarizes possible changes of the erythrogram.

Numeric Changes: Changes in the Spinned Hematocrit Tube

The Hct is defined as the percentage of the red-colored erythrocyte precipitate to the total blood column in a spinned Hct tube. The subsequent whitish buffy coat consists of leukocytes and thrombocytes followed by the transparent plasma column. A portion of approximately 1% buffy coat of the total column is considered physiologic for adult healthy individuals of most avian species (Fig. 6-73). Hct values between 35% and 55% for healthy adult birds are considered physiologic for most species (Campbell and Ellis, 2007). Because of the physiologic presence of up to 10% immature erythrocytes in the circulating blood, a smooth transition can be seen between the red column and the buffy coat.

Quantitative changes in the Hct are of either cellular (absolute) or plasmatic (relative) origin and can be differentiated by measuring the total protein content (Campbell and Ellis, 2007). A decrease in Hct value reflects an anemic situation; a rise points to a polycythemic situation. A buffy coat above 1.5% indicates leukocytosis or thrombocytosis; values above 10% are strongly suggestive for a leukemic neoplasia. Qualitative changes can be observed in the transition area between the two cell columns. An abrupt change from red to white indicates a right shift of erythrocytes; a broadened light-red transition area indicates a left shift. In severe cases the latter can result in a pinkish buffy coat in total with a complete mixture of immature erythrocytes, leukocytes, and thrombocytes, rendering a clear measurement of the Hct impossible. Physiologic seasonal and diurnal variations are described for RBC parameters (Gylstorff, 1983). Androgens, thyroxin, and prolactin have a positive effect on erythropoiesis, whereas estrogens have a negative effect. This results in a more or less profound sexual dimorphism in Hct, total RBC counts (TRBC), and Hb values, and especially higher values in stages with higher metabolic turnover (e.g., reproduction, migration). In the laying hen, the negative effect of estrogen is counterbalanced with the positive effect of thyroxin (Gylstorff, 1983; Sturkie and Griminger, 1986). Chicks physiologically display lower

TABLE 6-4 Changes of the Erythrogram: Findings—Hematologic Diagnosis—Etiologies

Finding	Hematologic Diagnosis		Possible Etiologies (Example)
Hct > 55%, PI = 2-3, discontinuous left shift, thickening of cells	Increased erythropoiesis	Polycythemia	Primary, (*polycythemia vera*) rare; secondary, adaptation to high altitudes, deficient oxygen binding to erythrocytes, chronic respiratory or cardiocirculatory disease (pneumonia, cardiomyopathies, atherosclerosis, ascites, rickets, abdominal masses, induration of liver tissue)
Hct < 35%, PI = 2-5, depending on stage and severity of disease		Hemorrhagic anemia	Trauma (injuries, blood-sucking parasites), coagulopathies (factor deficiencies, hepatopathies, toxins), infections, septicemias, toxemias, ulcers, neoplasias (secondary tissue hemorrhages, coagulopathies)
Hct < 35%, PI = 4-5		Hemolytic anemia	Congenital defects, hemoparasites, septicemias, intoxications, immune-mediated disease, neoplasias, burns
Hct < 35%, PI = 1-2, hypochromasia and microcytia possible	Reduced erythropoiesis	Depressive anemia	Iron/hemoglobin deficiency (malnutrition, chronic emaciating disease, lead/zinc intoxication), lack of erythropoietic factors (hepatopathies, nephropathies, hypothyroidism, hyperestrogenism, deficiency of vitamin B_{12}/folic acid), bone marrow damage (infections, toxins, therapeutics, neoplasias)

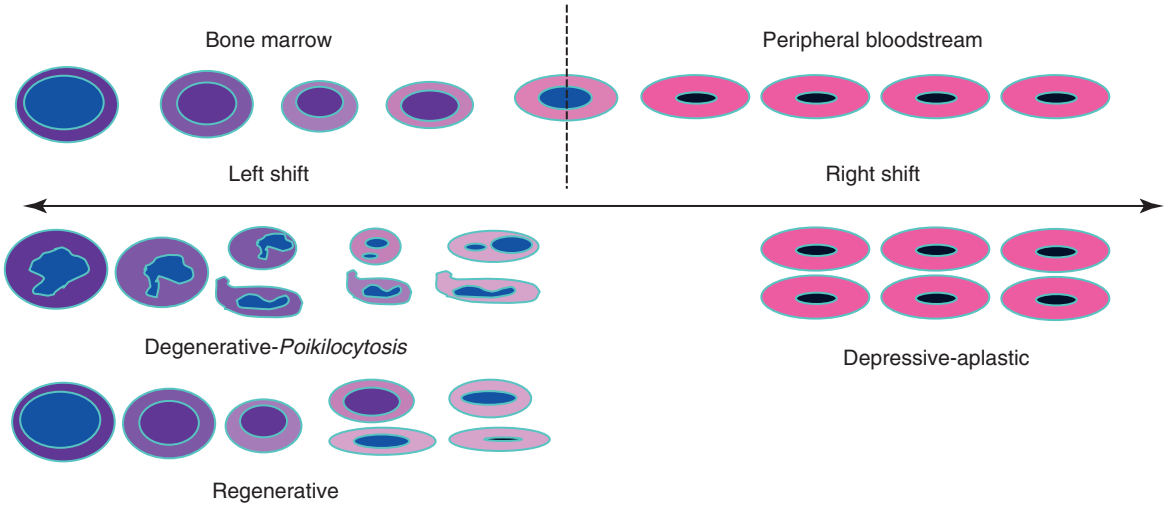


FIGURE 6-74 Definition of left and right shift in the erythropoetic line. *Dotted line* = border between forms present in the circulation and forms present in the bone marrow. Left shift = displacement of the borderline to the left. Right shift = displacement of the border line to the right.

total Hct, TRBC, and Hb values, and higher portions of immature cells, which adapt to adult values when sexual maturity is reached. The degree and duration of this adaptation is species specific and is completed more rapidly in precocial than in altricial species. Even in species with long nestling periods (e.g., *Ara* sp.), this process is finished within 6 weeks after hatching at the latest (Clubb *et al.*, 1991; Gylstorff, 1983; Hauska and Gerlach, 1995; Howlett *et al.*, 2002; Joyner *et al.*, 1992).

The clear blood plasma column in the centrifuged Hct tube may have different colors, mainly influenced by lipochromes (carotenoids) ingested through the diet. The intensity of plasma and feather color varies with the amount of carotenoid intake. A reddish tinge is characteristic for birds with bright red plumage such as the red ibis *(Eudocimus ruber)* or the greater flamingo *(Phoenicopterus ruber ruber)* and must not be confused with hemolysis. Yellow is frequently seen in granivorous species (Finger and Burkhardt, 1994; Slappendel, 1989) and in eclectus parrots *(Eclectus* sp.), for which a higher nutritional need for vitamin A is discussed. A rare green lipochrome occasionally causes green plasma colorations in toucans *(Ramphastos* sp.) (Finger and Burkhardt, 1994) and hawk-headed parrots *(Deroptyus acciptrinus)* and may be misinterpreted as biliverdinemia. In contrast to jaundice in mammals, disturbances in hemoglobin metabolism present as greenish biliverdinemia, which always is a sign of a severe condition (Campbell and Ellis, 2007). A milky-white appearance is typical for lipemia. This can occur postprandially or because of an impaired lipid metabolism (Campbell and Ellis, 2007). If present in a female bird in conjunction with a depressive anemia, hyperestrogenism because of a physiologic reproductive stage or pathologic alterations of the reproductive tract should be considered.

Morphologic Changes of Erythrocytes in the Stained Blood Film

Left and Right Shift: Polychromatic Index. Blood loss and hemolysis result in a left, depressive anemia in a right shift of erythrocytes in the blood film. The terms *right* and *left shift* refer to the portion of precursor cells within the circulating blood cells. The morphologic alterations can be of regenerative, degenerative, depressive, or aplastic nature (Fig. 6-74). A right shift describes a lack of immature progenitors and can change gradually from *depressive* to *aplastic*. A concurrent hypochromasia and microcytia may be present. In case of a left shift, an increased amount of immature progenitor cells is visible. The left shift is classified as *regenerative* if the immature forms display

physiologic and morphologic characteristics of the erythropoetic cell line. During their maturation, the primarily round cells with dark cytoplasm and large nuclei differentiate into cells with a progressively elliptic form of the nucleus and cytoplasm. The plasma color changes from basophilic over polychromatic *gray* to eosinophilic. The nucleo to cytosomal ratio decreases with advancing maturation. Increased variability in size is known as *anisocytosis*; heterogeneity in color is defined by the term *polychromasia;* and lack of coloration is called *hypochromasia*. Abnormal alterations of nuclear and cytoplasmic shape are subsumed under the term *poikilocytosis* and indicate a degenerative left shift (see Fig. 6-74). The presence of irregular nuclear and cytoplasmic shapes and the presence of lysed cells, mitoses (not shown), and amitotic divisions strongly suggest a degenerative background in this case.

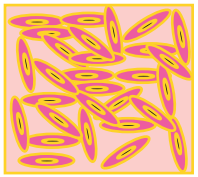
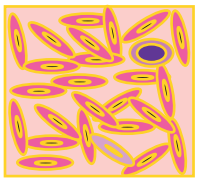
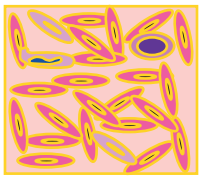
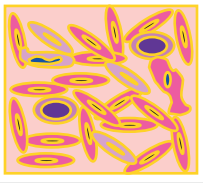
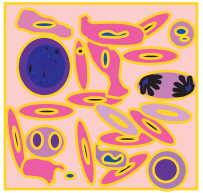
The polychromatic index (PI) established by Dein is a valuable tool to assess erythrocyte morphology (Dein, 1983) (Table 6-5). Using a semiquantitative scale from 1 to 5, the proportion of immature red cells is estimated in the monolayer of the blood film (Pendl, 2008). All immature developmental stages are subsumed under the umbrella term *polychromatic cell*. According to the variable characteristics of the different maturation stages, polychromatic cells display a very heterogenous morphology. The PI grades correlate well with the subjective first impression, allowing an assessment at first sight.

Polycythemia and Hyperchromic Normocythemia

Polycythemia is defined as a relative or absolute increase of erythrocyte numbers above physiologic ranges. Absolute polycythemia can be distinguished morphologically from the relative form (hemoconcentration) by the presence of a discontinued left shift (VanDerHeyden, 1994). The blood film contains an increased number of round-shaped early polychromatic cells with large nuclei, but a physiologic amount of late polychromatic cells. Intermediate stages are virtually absent. The PI is between 2 and 3. Absolute polycythemias can be of primary (i.e., neoplastic) or secondary origin because of chronic tissue oxygen deficiency (Jain, 1993; VanDerHeyden, 1994). The first, also called erythroblastosis or *polycythemia vera,* is a well-known myeloproliferative disorder in poultry and belongs to the avian leukosis/sarcoma group (Löliger, 1992). Secondary polycythemias, as mentioned earlier, always develop under chronic hypoxic conditions regardless of cause.

A respiratory syndrome resembling human interstitial lung disease (ILD) has been reported in psittacines, specifically in blue and yellow macaws (*Ara ararauna*) (Fudge and Reavill, 1993; Taylor and Hunter, 1991) and Amazon parrots (Amann *et al.,* 2007; Pendl and Reball, 2004; Zandvliet *et al.,* 2001). In contrast to the true polycythemia in the macaws, the affected Amazons frequently develop a hyperchromic normocythemia characterized by physiologic total

TABLE 6-5 Polychromatic Index

Fig.	Index	[%]1	Morphologic Criteria	Interpretation
	1	0	"Homogeneous": Erythrocytes homogenous in shape and texture; almost no polychromatophilic cells	Physiologic or depression of erythropoiesis suspected
	2	<10	"Slightly Irregular": Erythrocytes not entirely homogenous; few polychromatophilic cells present	Physiologic
	3	10-20	"Moderately Irregular": Many forms present from polychromatophilic through mature erythrocytes	Moderate regenerative response/ left shift
	4	20-50	"Severely Irregular": Significant numbers of polychromatophilic cells	Marked regenerative response/left shift
	5	>50	"Extremely Irregular": Large numbers of polychromatophilic cells; significant poikilocytosis	Severe regenerative to degenerative response/left shift: binucleation, abnormal nuclear divisions, mitotic figures, nuclear and cytoplasmic poikilocytosis

(Modified from Dein, 1983.)

erythrocyte numbers with an increase in cell size (MCV > 200 fl) resulting in an increase of the PCV to sometimes more than 90%. The hemoglobin content may exceed 20 g/L and the RBC cytomorphology in the blood film appears to be rounded because of prominent extension of the cytoplasm, which is packed with hemoglobin (Pendl and Reball, 2004; Taylor and Hunter, 1991; Zandvliet *et al.,* 2001). Because circulating avian erythrocytes still contain nuclei, they are capable of hemoglobin synthesis. This allows them to increase their hemoglobin content under hypoxic conditions (Gylstorff, 1983).

Depressive Anemia

Reduced erythropoietic activity leads to a depressive anemia. Depressive anemias are classified as *substrate deficient* (iron, hemoglobin deficiency), *hypoproliferative* (lack of erythropoietic factors) or *hypoplastic-aplastic* forms (direct damage of bone marrow). Morphologically, a depressive anemia is characterized by a PI between 1 and 2. Persistent values around 3 with a low Hct are also considered depressive, because the regenerative activity does not countervail the loss of cell mass in these cases. Hypochromasia is frequently seen in iron or protein deficiencies; macrocytic erythrocytes are typical for vitamin B_{12}—or folic acid—(B_9) deficiency (Gylstorff, 1983). Autoagglutination of erythrocytes usually displays as numerous small cell aggregates in the area of the monolayer. True rouleaux formation as seen in mammals is infrequent. The most common cause for high antibody titers causing autoaggluntination in exotic bird species is an infection with a highly antigenic agent such as mycobacteriosis, chlamydiosis, or aspergillosis in an active stage of immune response.

Changes to the Leukogram

General Considerations

The WBC count is the most common parameter to assess immunocompetence under clinical conditions. It has to be emphasized, however, that the WBC count only provides information on the circulating cellular immunity (O'Neal and Ketterson, 2012) without giving information on its functionality (Demas *et al.,* 2011). To capture the complexity of immune competence in total, various measures of immunity are necessary (Demas *et al.,* 2011). Therefore, WBC counts today are increasingly performed in combination with other tests (Demas *et al.,* 2011), such as concentrations of plasma proteins like acute phase proteins (innate response), cytokines (innate and acquired response), and immunoglobulins (acquired responses). Functionality of immune responses can be assessed with challenge tests using an infectious or noninfectious antigen as a trigger and measuring immune reactions postchallenge (Dietert *et al.,* 1994).

Cell Function: The Immune System of Birds

Like in mammals, immune reactions in birds consist of a primary innate immune response frequently followed by an adaptive immune response. Because of the high metabolic rate, however, these two processes take place rapidly and may cause simultaneous changes in the blood panel. Heterophils, monocytes, and thrombocytes are the principal phagocytes of the immune system. Together with natural killer (NK) cells, they form the main cellular components of the innate immune response. These cells are fully active at the time of hatching.

In contrast, all lymphocytes (except for the NK Cells) undergo a development and maturation process after hatching, either in the thymus or the bursa of Fabricius, before populating secondary lymphatic organs such as the spleen and mucosal-associated lymphatic tissue (MALT). Lymphocytes represent the main cellular part of the secondary, acquired immune system, which acts through antigen-specific mechanisms. Recurrent stimulation with a certain pathogen amplifies the scale and speed of this specific reaction (Schmidt *et al.,* 2003). The development of the adaptive immune system is approximately completed with the onset of the involution of the thymus and the bursa of Fabricius. In altricial species, thymus involution occurs at the stage of independent food intake, whereas in precocial species it takes place at the time of sexual maturity. Bursal involution is accomplished around the time of sexual maturation in both groups (Clubb *et al.,* 1991; Joyner *et al.,* 1992; Lane *et al.,* 1988; VanDerHeyden, 1986).

AGE-RELATED CHANGES IN THE LEUKOGRAM

Helene Pendl, Jaime Samour

As a reflection of this development, differential counts of galliform, anseriform, and columbiform species change within the first weeks of life from a predominantly heterophilic to a predominantly lymphocytic picture. In Psittaciformes and Falconiformes, this change is less pronounced because these species pertain a more or less heterophilic differential count through adulthood. The total leukocyte counts can transiently exceed the physiologic ranges for adult birds and decline with progressing age (Clubb *et al.,* 1991; Howlett *et al.,* 2002).

Gradual Transition from Physiologic to Pathologic Blood Panels

Because of the dynamic nature of immune reactions, the transition from *physiologic* to *pathologic* is gradual. During the course of a disease, variations in the blood panel have to be expected. Physiologic results do not necessarily rule out a pathologic condition, but simply reflect a missing hematologic response. Depending on the stage of an infection with *Aspergillus* sp., for example, all types of hemograms from highly reactive (active response, exacerbation, accessible antigen) to completely unremarkable (antigen compartmented in granulomas, silent pause) can be obtained.

Numeric Changes

Cell counts need to be read in absolute values. For example, a relative differential count of 90% heterophils and 10% lymphocytes in an African grey parrot with an absolute leukocyte count of 60,000 cells/µL indicates a true heterophilia. In the case of 10,000 cells/µL, a lymphopenia is present. The first points toward a heavy reaction of the innate immune system such as in the case of acute or subacute inflammation. The second indicates a lymphocyte depression possibly caused by an immunosuppressive pathogen or transport- and treatment-induced stress.

Heterophils

Because heterophils are the key phagocytes in the first line of immune defense, heterophilias first and foremost indicate an increased demand for phagocytosis. Etiologically, this may be correlated to a bacterial infection but can also be seen in cases of increased phagocytosis of cell debris, such as in wound healing or loss of tissue caused by toxic, metabolic, neoplastic, and infectious causes other than bacteria. Chronic stress of various origins induces an increase of heterophil numbers. This can be detected in the differential count as an elevated ratio of heterophils to lymphocytes (H:L-ratio) and serves as a stress parameter in poultry (Lentfer *et al.,* 2015). In the majority of cases, a heteropenia is of artifactual origin. Pathologic decreases are usually accompanied by a prominent left shift and represent either a reduced granulopoiesis caused by compromised bone marrow function or an overwhelming demand, such as in septicemias or toxemias. Prominent leukopenias with heteropenia and overwhelming secondary septicemias are seen in *Circovirus* infections in young African grey parrots *(Psittacus erithacus)* and are caused by atrophy and necrosis of the granulopoietic lines in the bone marrow (Schmidt *et al.,* 2003;

Schoemaker *et al.*, 2000). The virus attacks and kills B cells and causes lymphocytolysis and extensive necrosis of bursal follicles. Consequently, these birds are profoundly immunosuppressed both in the innate and the acquired system. A breakdown of myelopoiesis has also been reported in cases of intoxication with benzimidazole anthelmintics. Clinical signs are usually seen within 48 hours after treatment and include acute death, secondary septicemia, heteropenia to agranulocytosis, and sometimes anemia (Wiley, 2009).

Mononuclear Cells and Thrombocytes

Just like heterophilias, a monocytosis indicates increased phagocytic activity, but may also point toward antigenic challenge, fibrosis, granuloma, and giant cell formation. Because monocytes play a role both in primary and secondary immune responses, their evaluation is unsuitable for the temporary staging of a disease process. Marked lymphocytosis with or without lymphoblasts and/or signs of activation point toward a lymphoid neoplasia or a leukemoid inflammatory reaction with pronounced antigenic challenge. Lymphocytosis is rarely seen in species with a predominantly heterophilic leukogram (see earlier) and is always a sign of a specific immune reaction. Lymphopenia may indicate an increased migration to extravasal compartments such as in primary stages of infections or true suppression of lymphocyte proliferation. Many infectious agents employ immune evasive mechanisms to survive the innate immune responses of the host. Microbes causing a persistent infection additionally need to be capable to counteract acquired immune responses. Often these mechanisms target lymphoid cells in a direct or indirect manner, resulting in a lymphopenia or impaired lymphocyte function. Environmental toxins such as crude oil (Briggs *et al.*, 1996), organochlorides, and mycotoxins can cause severe lymphocyte depletion and lymphoid cell destruction (Koutsos and Klasing, 2014). Lymphoid depletion of the thymus, bursa, and spleen resulting in a leukopenia and lymphopenia have been reported in poultry for both ochratoxin (OTA) (Stoev, 2010) and cyclopiazonic acid (CPA) (Kamalavenkatesh *et al.*, 2005), two mycotoxins produced by several *Aspergillus* spp. Furthermore, OTA is hepatotoxic and nephrotoxic. Similar pathophysiologic mechanisms may also apply for aspergillosis in noncommercial avian species; in clinically overt stages of chronic aspergillosis, pet birds frequently display a true lymphopenia along with heterophilia and monocytosis. Hepatopathies and nephropathies may also be present. A correlation to mycotoxins in these species, however, still needs experimental proof. Sublethal doses of lead cause impairment of immune function in humans, mammals (Dietert and Piepenbrink, 2006), and birds (Gao *et al.*, 2007; Kendall *et al.*, 1996; Redig *et al.*, 1991). Because lead is rather noncytotoxic (i.e., it produces only modest changes to immune cell populations and lymphoid organs), diagnosis of chronic low dose lead intoxication from white blood cells (WBCs) is hardly possible. Besides their function in hemostasis, avian thrombocytes have a conspicuous capability of phagocytosis (Grecchi *et al.*, 1980; Gylstorff, 1983; Wigley *et al.*, 1999). Therefore a thrombocytosis either points toward elevated coagulative or phagocytic activity. Thrombocytopenia is, in the majority of cases of artifactual origin due to thrombocyte aggregation. True thrombocytopenias with a left shift (see later) are always a sign of a serious condition with exhaustion of mature cell pools.

Eosinophilia and Basophilia

Eosinophilias and basophilias are difficult to interpret in birds because the function of these cells is not completely understood. Chicken eosinophils resemble mammalian eosinophils morphologically and are discernible from other granulocytes in a stained blood film. However, they may represent an entirely different type of cell; to date, neither eosinophil-attracting chemokines, their cognate receptors, nor their encoding genes have been detected (Kaiser and Staeheli, 2014). Automated methods for cell differentiation based on fluorescence-activated cell sorting (FACS) fail to distinguish eosinophils and basophils in chickens (Seliger *et al.*, 2012). Cytomorphology of eosinophils is highly variable among avian orders, which additionally supports the hypothesis of a possible inhomogeneous group of cells of unknown type. Furthermore, experiments that trigger eosinophilia in mammals produce inconsistent results in birds, making eosinophils an unreliable indicator for intestinal parasitism and hypersensitivity reactions in birds (Campbell, 1995; Fudge, 2000). Some species show remarkably high eosinophil (family Buteoidae), or basophil (cockatiels, *Nymphicus hollandicus*), counts without any signs of disease. Clinical and experimental findings in chickens suggest that eosinophils participate in delayed rather than in immediate hypersensitivity reactions (Maxwell, 1987). Blood eosinophilias have been observed in various infectious diseases with profound tissue damage and often chronic granulomatous or fibrous inflammatory reactions, such as mycoplasmosis, mycobacteriosis, streptococcosis, staphylococcosis, listeriosis, erysipeloid, and infection with West Nile virus (WNV). In pet birds, an empirical correlation of eosinophilia and basophilia with damage of epithelia with direct contact to the environment has been observed. Examples include diseases of the skin, the respiratory tract, and to a lesser extent, the gastrointestinal (GI) tract. Related conditions include smoke inhalation, feather picking, self-mutilation, cannibalism, flying accidents, carnivore attacks, drug injections, and postsurgical recovery (Fudge, 2000).

Cytomorphologic Changes

Morphologic changes in monocytes, lymphocytes, and thrombocytes include cytoplasmic basophilia, vacuolation, and bleb formation (i.e., constriction of vesicles from the cell membrane). Frequently the Golgi apparatus can be detected as a light-blue area close to the nucleus. In general, cytoplasmic basophilia and loss of coarse nuclear chromatin structure indicate an increased cell metabolism, which may either point to an increased reactivity of mature cells or to an increased amount of immature cells in the peripheral circulation (i.e., a left shift). Pathologic blood panels frequently display a left shift in several cell lines, which hampers evaluation of blood films as discrimination of thrombocytes, lymphocytes, and polychromatic erythrocytes gets challenging. In cases with a massive immune response, the blood film may even mimic a leukemoid neoplastic process with high leukocyte counts and poorly differentiated (i.e., blastoid) mononuclear cells that are impossible to differentiate into lymphocytes and monocytes.

Morphologic characteristics to differentiate between reactive and immature thrombocytes are poorly defined in literature. An increased presence of immature thrombocytes reflects a regenerative reaction to meet higher demands (Campbell and Ellis, 2007). There are certain pathologic conditions in which the presence of enlarged thrombocytes, commonly referred to as megathrombocytes, in the blood film appears to be a characteristic hemoresponse. For instance, in the houbara bustard (*Chlamydotis undulata macqueenii*), the mean thrombocyte measurements in birds undergoing chronic inflammation (e.g., severe shoulder injury as a result of repeated crashing against the wall of an enclosure) were 9.22 ± 0.21 mm length and 8.10 ± 0.19 mm width compared with 5.47 ± 0.12 mm length and 4.96 ± 0.10 mm width in clinically normal birds (D'Aloia *et al.*, 1994).

The role of magenta body–carrying lymphocytes as indicators for a pathologic condition is unknown. According to more recent human literature, these cells could represent cytotoxic T-cells of the large granular lymphocyte (LGL) type and may be identical with natural killer (NK) cells (Langenkamp, 2005). In chickens, magenta body–carrying lymphocytes account for 5% of the total leukocyte count in

healthy individuals. They demonstrate spontaneous lytic capabilities toward tumor cells, play a pivotal role in the natural defense against microbial infections (Herberman and Ortaldo, 1981; Sharma and Okazaki, 1981), and their presence seems to correlate with a shortened lifespan and cases of avian leukosis (Lucas and Jamroz, 1961).

Morphologic changes of granulocytes mainly affect heterophilic granulocytes and are commonly summarized under the term *toxic left shift*. First signs of toxicity are characterized by cytoplasmic basophilia and swelling of the granula. Alterations continue with a loss of granular structures, the appearance of vacuoles, and finally result in cell death with karyorrhexis or karyolysis (grade 4) (Campbell and Ellis, 2007). These toxic changes are usually accompanied by a left shift of the heterophilic line. The immature cells present with a basophilic cytoplasm and a variable segmentation of the nucleus. In addition to eosinophilic, elliptic, and mature granules, basophilic and round immature forms can be found in the cytoplasm. These granules usually represent less than 50% of all granules, which defines the cells as intermediate to late stages of metamyelocytes (Lucas and Jamroz, 1961). Characteristic signs to distinguish between a toxic morphology and a heterophilic left shift are controversially discussed in literature. Although toxic features are separately dealt with from immature features, the descriptions of their morphologic characteristics are almost identical (Campbell and Ellis, 2007). Because both phenomena usually occur simultaneously, the subsumption under the term *toxic left shift* is justified from the clinicopathologic standpoint.

Phagocytosis by heterophils, monocytes, and thrombocytes is rarely seen in blood films. It indicates an intravascular immune reaction usually caused by a septicemic condition or immune-mediated disease.

Conclusive Remarks: The Value of Cytomorphologic Assessment

Compared with numeric parameters, changes in cell morphology are less affected by physiologic variation and environmental influences. Thus the presence of cells with morphologic abnormalities is a more reliable index of inflammation than cell counts (Allison and Meinkoth, 2007). True abnormalities need to be distinguished from artifacts caused by staining and fixation. Because immune reactions are also happening under healthy conditions, a substantial amount of cells must be altered to be considered as significant for a pathologic condition. The benefit of the assessment of cytomorphology lies in the specification of numeric findings. A hematocrit (Hct) of 30% in an Amazon parrot in conjunction with a polychromatic index (PI) value between 1 and 2 indicates a depressive anemia, which usually resolves spontaneously when the primary cause is treated. A PI of 3 indicates a regenerative anemia caused by blood loss and possibly points to an ongoing hemorrhage. An acute life-threatening finding is a PI of 5, which requires immediate measures to stabilize the patient.

As for leukocytes, a normocytosis of 7000 cells/µL and physiologic values for the differential count and physiologic cellular morphology does not point to an increased immune reactivity. In contrast, identical numeric results combined with a toxic left shift of heterophils and activation of mononuclear cells in the blood film indicate a massive immune reaction with possible damage of the hematopoietic tissues. A left shift always has to be considered a sign of a severe imbalance between cell production and demand. Immature cells do not have the full spectrum of defense mechanisms present in mature cells. Thus the immune reaction may be inadequate or insufficient, which predisposes the patient for opportunistic secondary infections. Increasing grading of toxicity and immaturity and declining total cell counts point to a septicemic or toxemic situation with breakdown or severe damage of hematopoiesis with a guarded prognosis.

ACKNOWLEDGMENTS

The authors would like to thank the editors of *Clinical Avian Medicine* (2006), Spix Publishing, for allowing the authors to reproduce parts of the chapter on the diagnostic value of hematology.

FURTHER READING

Campbell TW: *Avian hematology and cytology*, ed 2, Ames, 1995, Iowa State University Press.

Campbell TW, Ellis C: *Avian and exotic animal hematology and cytology*, ed 3, Ames, 2007, Blackwell Publishing Professional.

Dein FJ: Hematology. In Harrison GJ, Harrison LR, editors: *Clinical avian medicine and surgery*, Philadelphia, 1986, WB Saunders, pp 174–191.

Dorrestein GM: Cytology and haemocytology. In Beynon PH, Forbes NA, Lawton MPC, editors: *Manual of psittacine birds*, Gloucestershire, 1996, British Small Animal Veterinary Association, pp 38–48.

Gascoyne SC, Bennet PM, Kirkwood JK, Hawkey CM: Guidelines for the interpretation of laboratory findings in birds and mammals with unknown reference ranges: plasma biochemistry, *Vet Rec* 134:7–11, 1994.

Gulland FMD, Hawkey CM: Avian haematology, *Veterinary Annual* 30:126–136, 1990.

Hawkey CM, Samour JH: The value of clinical hematology in exotic birds. In Jacobson ER, Kollias GV Jr, editors: *Contemporary issues in small animal practice*, New York, 1988, Churchill Livingstone, pp 109–142.

International Council for Standardization in Haematology: Guidelines for the evaluation of blood cell analysers including those used for differential leucocyte and reticulocyte counting and cell marker applications, *Clin Lab Haematol* 16:157, 1994.

Jain CJ: *Essentials of veterinary hematology*, Philadelphia, 1993, Lea & Febiger.

Jennings IB: Haematology. In Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *Manual of raptors, pigeons and waterfowl*, Cheltenham, 1996, British Small Animal Veterinary Association, pp 68–78.

Lucas AH, Jamroz C: *Atlas of avian hematology* (vol 25), Ames, 1961, United States Department of Agriculture.

REFERENCES

Allison RW, Meinkoth JH: Hematology without the numbers: in-clinic blood film evaluation, *Vet Clin North Am Small Anim Pract* 37(2):245–266, 2007.

Amann O, Kik MJ, Passon-Vastenburg MH: Chronic pulmonary interstitial fibrosis in a blue-fronted Amazon parrot (*Amazona aestiva aestiva*), *Avian Dis* 51(1):150–153, 2007.

Blalock TL, Thaxton JP: Hematology of chicks experiencing marginal vitamin B_6 deficiency, *Poult Sci* 63(6):1243–1249, 1984.

Briggs KT, Yoshida SH, Gershwin ME: The influence of petrochemicals and stress on the immune system of seabirds, *Regul Toxicol Pharmacol* 23(2):145–155, 1996.

Calle PP, Stewart CA: Hematologic and serum chemistry values for captive Hyacinth macaws (*Anodorhynchus hyazinthinus*), *J Zoo Wildl Med* (2–3):98–99, 1987.

Clubb SL, Schubot RM, Joyner K, et al: Hematologic and serum biochemical reference intervals in juvenile cockatoos, *J Assoc Avian Vet* 5:16–21, 1991.

D'Aloia MA, Samour JH, Howlett JC: Haemopathological responses to chronic inflammation in the houbara bustard (*Chlamydotis undulata macqueenii*), *Comparative Haematology International* 4:203–206, 1994.

Dein FJ: Avian hematology: erythrocytes and anemia, *Proceedings of the Annual Meeting of the Association of Avian Veterinarians*, pp 10–23, 1983.

Demas GE, Zysling DA, Beechler BR, et al: Beyond phytohaemagglutinin: assessing vertebrate immune function across ecological contexts, *J Anim Ecol* 80(4):710–730, 2011.

Dietert RR, Golemboski KA, Austic RE: Environment-immune interactions, *Poult Sci* 73(7):1062–1076, 1994.

Dietert RR, Piepenbrink MS: Lead and immune function, *Crit Rev Toxicol* 36(4):359–385, 2006.

Finger E, Burkhardt D: Biological aspects of bird colouration and avian colour vision including ultraviolet range, *Vision Res* 34(11):1509–1514, 1994.

Fudge AM: Clinical application of laser flow cytometry to avian hematology analysis. *Proceedings of the Association of Avian Veterinarians*, pp 17–18, 1995.
Fudge AM: Disorders of avian leukocytes. In Fudge AM, editor: *Laboratory medicine avian and exotic pets* (vols 19–25), Philadelphia, 2000, WB Saunders.
Fudge AM, Reavill DR: Pulmonary artery aneurysm and polycythaemia with respiratory hypersensitivity in a blue and gold macaw (*Ara ararauna*), *Proceedings of the European Conference of Avian Medicine and Surgery*, pp 382–387, 1993.
Gao D, Mondal TK, Lawrence DA: Lead effects on development and function of bone marrow-derived dendritic cells promote Th2 immune responses, *Toxicol Appl Pharmacol* 222(1):69–79, 2007.
Grecchi R, Saliba AM, Mariano M: Morphological changes, surface receptors and phagocytic potential of fowl mono-nuclear phagocytes and thrombocytes in vivo and in vitro, *J Pathol* 130(1):23–31, 1980.
Gylstorff I: Blut. Blutbildung und Blutkreisauf. In Mehner A, Hartfiel W, editors: *Handbuch der geflügelphysiologie*, Jena, 1983, Fischer, pp 280–395.
Hauska H, Gerlach H: The development of the red blood cell pattern of growing parrot nestlings, *Proceedings of the Conference of the European Committee of the Association of Avian Veterinarians*, pp 183–186, 1995.
Herberman RB, Ortaldo JR: Natural killer cells: their roles in defenses against disease, *Science* 214(4516):24–30, 1981.
Howlett JC, Bailey TA, Samour JH, et al: Age-related hematologic changes in captive-reared houbara, white-bellied, and rufous-crested bustards, *J Wildl Dis* 38(4):804–816, 2002.
Joyner KL, De Berger N, Lopez EH, et al: Health parameters of wild psittacines in Guatemala: a preliminary report, *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp 287–303, 1992.
Julian RJ, Summers J, Wilson JB: Right ventricular failure and ascites in broiler chickens caused by phosphorus-deficient diets, *Avian Dis* 30(3):453–459, 1986.
Kaiser P, Staeheli P: Avian cytokines and chemokines. In Schat KA, Kaspers B, Kaiser P, editors: *Avian immunology*, ed 2, London, 2014, Academic Press Elsevier, pp 189–204.
Kamalavenkatesh P, Vairamuthu S, Balachandran C, et al: Immunopathological effect of the mycotoxins cyclopiazonic acid and T-2 toxin on broiler chickens, *Mycopathologia* 159(2):273–279, 2005.
Kendall RJ, Lacher TE, Bunck C, et al: An ecological risk assessment of lead shot exposure in non-waterfowl avian species: upland game birds and raptors, *Environ Toxicol Chem* 15(1):4–20, 1996.
Koutsos EA, Klasing KC: Factors modulating the avian immune system. In Schat KA, Kaspers B, Kaiser P, editors: *Avian immunology*, ed 2, London, 2014, Academic Press Elsevier, pp 299–313.
Lane R: Avian hematology: Basic cell identification and WBC count determination, and clinical pathology. In Rosskopf WJ, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, 1996, Williams and Wilkins, pp 739–772.
Lane RA, Rosskopf W, Allen KL: Avian pediatric hematology: preliminary studies of the transition from the neonatal hemogram to the adult hemogram in selected psittacine species: African greys, Amazons, and macaws. *Proceedings of the Annual Meeting of the Association of Avian Veterinarians*, pp 231–238, 1988.
Langenkamp U: *Signaltransduktion in NK-Zellen während der natürlichen Toxizität*. (Dr. med.), Lübeck, 2005, Universität zu Lübeck.
Lentfer TL, Pendl H, Gebhardt-Henrich SG, et al: H/L ratio as a measurement of stress in laying hens—methodology and reliability, *Br Poult Sci* 56(2):157–163, 2015.
Löliger HC: Aviäre Onkovirosen. In Heider G, Monreal G, editors: *Krankheiten des wirtschaftsgeflügels. Ein handbuch für wissenschaft und praxis. Band 1: allgemeiner teil und spezieller teil 1* (vol 1), Jena, Stuttgart, 1992, Fischer, pp 742–743.
Maxwell MH: The avian eosinophil—a review, *Worlds Poult Sci J* 43:190–207, 1987.
O'Neal DM, Ketterson ED: Life-history evolution, hormones, and avian immune function. In Demas GE, Nelson RJ, editors: *Ecoimmunology*, New York, 2012, Oxford University Press, pp 8–44.
Pendl H: Für Studium und Praxis: Möglichkeiten und Grenzen einer praxisnahen Hämatologie beim Vogel—Teil 1: Methodische Einführung und Befunde beim klinisch gesunden Vogel, *Tierärztliche Praxis Kleintiere* 36(4):290–298, 2008.
Pendl H, Reball H: Hyperchrome Normämien bei Amazonen—hämatologisches Bild. *Tagung über Vogelkrankheiten, Deutsche Veterinärmedizinische Gesellschaft, Fachgruppe Geflügelkrankheiten* (WVPA), Institut für Geflügelkrankheiten der Ludwig-Maximilians-Universität München, Germany, 2004.
Redig PT, Lawler EM, Schwartz S, et al: Effects of chronic exposure to sublethal concentrations of lead acetate on heme synthesis and immune function in red-tailed hawks, *Arch Environ Contam Toxicol* 21(1):72–77, 1991.
Schmidt RE, Reavill DR, Phalen DN: *Pathology of pet and aviary birds*, Ames, 2003, Iowa State Press.
Schoemaker NJ, Dorrestein GM, Latimer KS, et al: Severe leukopenia and liver necrosis in young African grey parrots (*Psittacus erithacus erithacus*) infected with psittacine circovirus, *Avian Dis* 44(2):470–478, 2000.
Seliger C, Schaerer B, Kohn M, et al: A rapid high-precision flow cytometry based technique for total white blood cell counting in chickens, *Vet Immunol Immunopathol* 145(1–2):86–99, 2012.
Sharma JM, Okazaki W: Natural killer cell activity in chickens: target cell analysis and effect of antithymocyte serum on effector cells, *Infect Immun* 31(3):1078–1085, 1981.
Slappendel RJ: Wet lab: avian hematology. *Proceedings of the 2nd Symposium on Avian Medicine and Surgery*, Utrecht, the Netherlands. 1989.
Stoev SD: Studies on some feed additives and materials giving partial protection against the suppressive effect of ochratoxin A on egg production of laying hens, *Res Vet Sci* 88(3):486–491, 2010.
Sturkie PD, Griminger P: Body fluids: blood. In Sturkie PD, editor: *Avian physiology*, ed 5, New York, 1986, Springer, p 112.
Taylor M, Hunter B: A chronic obstructive pulmonary disease of blue and gold macaws, *J Assoc Avian Vet* 5(2):71, 1991.
VanDerHeyden N: The hematology of nestling raptors and psittacines. *Proceedings of the Annual Meeting of the Association of Avian Veterinarians*, pp 347–353, 1986.
VanDerHeyden N: Evaluation and interpretation of the avian hemogram, *Seminars of Avian and Exotic Pet Medicine* 3:5–13, 1994.
Wigley P, Hulme SD, Barrow PA: Phagocytic and oxidative burst activity of chicken thrombocytes to *Salmonella, Escherichia coli* and other bacteria, *Avian Pathol* 28(6):567–572, 1999.
Wiley JL, Whittington JK, Wilmes CM, Messick JB: Chronic myelogeneous leukemia in a great horned owl (*Bubo virginianus*), *J Assoc Avian Vet* 23(1):36–43, 2009.
Wiskott M: *Vergleich verschiedener Methoden zur Leukozytenzählung bei Vögeln*. (Dr. med.vet.), Vienna, 2002, University of Vienna.
Zandvliet MM, Dorrestein GM, Van Der Hage M: Chronic pulmonary interstitial fibrosis in Amazon parrots, *Avian Pathology* 30(5):517–524, 2001.

BIOCHEMISTRY ANALYSES

Thomas A. Bailey

In this section, the biochemical tests that are commonly used to evaluate avian health are reviewed. Clinical signs in birds may be nonspecific, and often only limited information is gleaned from the physical examination. Blood chemistry assays are a component of the clinical laboratory support that is required in the differential diagnosis of many diseases. Interpreting what a list of chemistry values from a sick bird really means can be confusing and, although biochemistry results are not usually diagnostic, they may be helpful in ruling out conditions or indicating the severity of organ pathology (Fudge, 1997). Relating changes in chemistry values to organ pathology is difficult, because with the exception of studies on pigeons (Lumeij, 1987) and bustards (Bailey *et al.*, 1999b), there have been few detailed biochemical investigations in nondomestic avian species concerning tissue enzyme profiles, age-related changes, and changes after experimental organ damage.

SAMPLE COLLECTION AND STORAGE

Techniques for blood collection and the volume of blood that can safely be collected have already been discussed. Although the anticoagulant of choice for most laboratory tests is lithium heparin, there are some exceptions, and the recommended samples that should be collected for different biochemical tests are listed in Table 6-6. Sample handling difficulties can result in adverse effects on biochemical results. It is known that potassium values can be effected by hemolysis or delays before centrifugation. Similarly blood that remains in contact with erythrocytes will have a decreased glucose concentration over time. Pond *et al.*, (2012) recently demonstrated that centrifugation method did not have any effect on plasma quality for biochemical analysis.

NORMAL BIOCHEMISTRY REFERENCE RANGES

Published normal biochemistry ranges of blood enzymes, metabolites, electrolytes, and trace elements for a selection of healthy adult and juvenile avian species are presented in Appendix 3. These values may prove useful for the interpretation of some laboratory findings. However, it is important for the reader to be aware that many of these "normal" ranges are derived from single studies in captive collections, and for some species, only small numbers of birds were involved. True population values can only be determined from larger samples that represent different diets, climates, housing environments, exercise levels, genders, and age groups (Merritt *et al.*, 1996). Unfortunately such studies are rare and avian veterinarians must make do with ranges derived from small numbers of birds. To generate meaningful reference

TABLE 6-6 Recommended Blood Samples for Avian Biochemistry Tests

Test	Plasma*	Serum	Other	Sampling Comments
Alkaline phosphatase (ALKP)	√	√		
Alanine aminotransferase (ALT)	√	√		Hemolysis causes elevated activities
Ammonia			EDTA	Analyze samples immediately; ammonia is released by catabolism of many substances (e.g., urea)
Amylase	√	√		
Aspartate aminotransferase (AST)	√	√		
Bicarbonate	√	√		
Bile acids	√	√		Birds should be fasted for 12-24 h before sampling
Bilirubin	√	√		
Calcium	√	√		Calcium-binding anticoagulants (e.g., EDTA) will cause artificially low values
Chloride	√	√		
Cholesterol	√	√		
Creatine kinase (CK)	√	√		Citrate and fluoride inhibit CK activity
Copper	√			
Creatinine	√	√		
Delta-aminolevulinic acid dehydratase	√	√		
Gamma glutamyl transferase (GGT)		√	EDTA	Heparin interferes with test reactants and citrate, oxalate and fluoride artificially depress activity
Glutamate dehydrogenase (GLDH)	√	√		
Glucose	√	√		Analyze samples within 2 h to minimize effect of glycolysis by erythrocytes
Iron	√	√		Avoid hemolysis. Avoid citrate, oxalate, and EDTA because they bind iron
Lactate dehydrogenase (LDH)	√	√		Hemolysis causes elevated activities
Magnesium	√			
Phosphorus	√	√		Avoid hemolysis. Citrate, oxalate, and EDTA interfere with analysis
Potassium	√	√		Levels are elevated by hemolysis. Separate samples within minutes for accurate results. Hyperlipemia and hyperproteinemia cause artificially low values
Selenium		√		
Sodium	√	√		Hyperlipemia and hyperproteinemia cause artificially low values
Total protein	√	√		Plasma contains fibrinogen and in pigeons the concentration of total protein in plasma is higher than serum
Triglycerides	√	√		
Urea	√	√		
Uric acid	√	√		
Zinc		√		

*Plasma from lithium heparin tubes.
EDTA, Ethylenediaminetetraacetic acid.

intervals, the following factors must be defined: clinical health, age, sex, husbandry, geographic location, season, reproductive cycle, breed, fasting status, stress, exercise, and medications (Cray, 2012).

In addition to seeing how many birds were sampled, to calculate a "normal range" clinicians should critically assess what statistics were used to analyze the data. Unfortunately, many published reference ranges have been derived using inappropriate statistics. Many biochemistry data do not conform to a gaussian (normal) distribution and nonparametric statistics are needed to establish reference ranges (Lumeij, 1987; Lumeij *et al.*, 1988a; Lumeij *et al.*, 1988b). Reference ranges are established statistically to produce a 95% confidence interval. In the case of normally distributed data, this is a 95% confidence interval of the mean; in the case of data that is not normally distributed, a 95% confidence interval of the median is more appropriate. What this means is that 5% (i.e., 1 in 20) of healthy birds will have values that fall outside a given "normal" reference range.

The reader is recommended to consult the literature to gain a deeper insight into theories and pitfalls of establishing normal reference ranges (Lumeij, 1987; Lumeij *et al.*, 1988a; Lumeij *et al.*, 1988b; Hochleithner, 1994; Cray, 2012). These days, there are many statistics books that are intellectually digestible for nonstatisticians (Petrie and Watson, 1999; Petrie and Sabin, 2000).

ENZYME PROFILES

Table 6-7 reviews causes of increases in enzyme activities and also summarizes the tissue distribution of some enzymes in birds. This table may be of assistance in interpreting changes of plasma enzyme levels seen in clinical practice.

Elevations in plasma enzyme activities are related to leakage of enzymes from damaged cells (Lumeij, 1987; Lumeij *et al.*, 1988a). Interpretation of elevated plasma enzyme levels can only be performed if the enzyme profiles of various organs of the species under investigation are known because the distribution of enzymes is markedly different between different organs and animal species. The clinical enzymology characteristics of many domestic mammals are well known, but studies of enzyme patterns in avian tissues for diagnostic purposes have been limited to a few species (Cornelius *et al.*, 1958; Bogin and Israeli, 1976; Bogin *et al.*, 1976; Lumeij and Wolfswinkel, 1987; Lumeij *et al.*, 1988a; Lumeij *et al.*, 1988b; Bailey *et al.*, 1999b).

It should be noted that not all elevations in plasma enzyme activities indicate a disease process, and tissue enzyme profiles can only serve as a rough guide to the interpretation of plasma enzyme activity. For example, although creatine kinase (CK) appears to be a specific and sensitive indicator of muscle cell damage in both mammals (Chalmers and Barrett, 1982) and birds (Lumeij *et al.*, 1988a; Lumeij *et al.*, 1988b), it is known that CK and lactate dehydrogenase (LDH) levels dramatically increase in healthy bustards that are handled (Bailey *et al.*, 1997). Consequently, consideration should be given to previous episodes of handling when interpreting plasma CK and LDH values. Similarly, Dorrestein *et al.* (1986) induced muscle damage in pigeons by injecting doxycycline in the pectoral muscle and found good correlation between CK levels and the severity of the injury caused by the injection. When birds are known to have been recently injected intramuscularly, elevated plasma CK, aspartate aminotransferase (AST), and LDH activity should be interpreted with caution. Plasma sorbitol dehydrogenase activity has been recently shown to be a specific indicator of liver injury (Williams *et al.*, 2012). Other causes of biochemical artifacts are discussed in the accompanying tables and include bacterial contamination of samples, unseparated blood, hemolysis, and various anticoagulants.

METABOLITES AND MINERALS

Analysis of metabolites in the blood provides information on the functional capacity of organs that are involved in different metabolic pathways. Commonly measured metabolites include plasma ammonia, bile acids, inorganic phosphate, urea, and uric acid. The macrominerals (calcium, phosphorus, potassium, sodium, and chloride) and microminerals (magnesium, zinc, iron, copper, and selenium) also serve

TABLE 6-7 Activity of Enzymes in Avian Tissues and Causes of Increases in Avian Species

Enzyme	Activity in Pigeon* and Bustard† Tissues	Causes of Increase in Avian Species
ALT	Present in most tissues including the duodenum, pancreas, liver, proventriculus, heart, and skeletal muscle	Nonspecific cell damage. Only rarely increased in avian liver disease
AST	Present in most tissues including liver, heart, skeletal muscle, brain, kidney, duodenum, and pancreas. In bustards the highest levels are in the proventriculus, heart, and skeletal muscle	Mainly liver (e.g., fatty liver), heart, or muscle disease. Vitamin E/Se deficiency, IM injections. AST has a longer half-life than LDH and levels remain elevated for a few days longer after cellular damage has stopped
ALKP	Mostly in duodenum, kidney. Low levels in liver	Increased cellular activity, not necessarily damage. Higher in juveniles. Increases seen in egg-laying, fractures, neoplasia, and infection
CK	Present in most tissues including the duodenum, pancreas, kidney, liver, proventriculus, skeletal muscle, heart muscle, and brain	Muscle damage, IM injections, neuropathies, physical capture, surgery, vitamin E/Se deficiency, lead toxicity
GGT	Biliary and renal tubular epithelium	Not a sensitive indicator of hepatocellular damage
GLDH	Mitochondrial enzyme in most tissues. Liver, kidney, and brain	Hepatocellular necrosis and severe liver disease
LDH	Present in most tissues including the duodenum, pancreas, skeletal muscle, heart muscle, liver, bone, kidney, and red blood cells. Highest levels in bustards are in the proventriculus and heart muscle	Hemolysis and liver (e.g., fatty liver), heart or muscle disease, IM injections. This enzyme has a short half-life and concentrations decline rapidly after organ damage

*Lumeij, 1987.
†Bailey *et al.*, 1999b.
Source: extracted and modified from Hochleithner, 1994; Fudge, 1997; Lumeij, 1987; Bailey *et al.*, 1999b.
ALKP, Alkaline phosphatase; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatine kinase; GGT, gamma glutamyl transferase; GLDH, glutamate dehydrogenase; IM, intramuscular; LDH, lactate dehydrogenase; Se, selenium.

important metabolic functions and are crucial for maintenance, growth and reproduction. Tables 6-8 and 6-9 summarize causes of changes in metabolic and electrolyte tests in avian species. Once again, normal physiologic variations need to be taken into account when interpreting plasma metabolite levels. Not all elevations indicate a disease process: for example, significantly elevated plasma bile acid, uric acid, and urea concentrations occur postprandially in raptors (Lumeij and Remple, 1991; Lumeij and Remple, 1992). Other physiologic variations in metabolite levels are discussed in the accompanying tables.

Mineral deficiency or excess can cause disease, so animal health evaluation often requires the determination of mineral status. Mineral status can be determined by analysis of serum, bone, tissues (e.g., liver), and feed (Scheideler *et al.*, 1994). The normal mineral concentration

TABLE 6-8 Causes of Changes in Metabolic Tests in Avian Species

Metabolite	Physiology	Causes of Increase	Causes of Decrease
Albumin	Albumin functions primarily as an osmotic pressure regulator and a transport protein; typically comprises 45-70% of avian serum protein. Accurate albumin determination can only be calculated through electrophoresis and many of the values presented in the appendices were determined by wet and dry chemistry assays and consequently should not be considered to provide an accurate measurement of albumin. Albumin levels should be assessed in context of the albumin : globulin ratio		Decreased synthesis caused by chronic liver disease, chronic inflammation; increased loss caused by renal disease, parasitism, or overhydration
Ammonia	Most absorbed from the alimentary tract, some derived from protein catabolism in skeletal muscle. In healthy birds, ammonia is converted into uric acid and urea in the liver and blood levels are low	Decreased liver function; ammonia poisoning	
Amylase	Produced in the pancreas, liver, and small intestine	Elevations associated with acute pancreatitis and enteritis	
Bile acids	Synthesized in the liver from cholesterol and act primarily as emulsifying agents in fat digestion and absorption. With the ingestion of food, bile is carried via the bile duct into the small intestine. Over 90% of bile acids are reabsorbed from the gastrointestinal tract and return via the portal circulation to the liver, where they are recycled. This is the "enterohepatic cycle." If liver function is impaired, bile acids are not properly reabsorbed and consequently the amount of excreted bile acids entering the circulation increases. Measurement of bile acid concentration is considered to be the most sensitive and most specific test available for determining liver dysfunction in birds and mammals	Reduced liver function (e.g., fatty liver disease), postprandial increase in some species	
Bilirubin	In birds the major bile pigment is biliverdin and biliverdin is not converted into bilirubin. Consequently, low or negligible concentrations are detected in the serum of healthy birds	Liver disease (rarely), chlamydiosis	
Calcium	Major constituent of bone. Involved in the transmission of nerve impulses, permeability and excitability of membranes, activation of enzyme systems, calcification of shells, and contraction of the uterus before egg laying. Blood calcium levels are directly linked to albumin levels. Calcium exists as three fractions in avian serum: as the ionized salt, as calcium bound to proteins, and as complexed calcium (Stanford, 2003). The ionized calcium is physiologically active but the protein-bound calcium is inactive. Consequently, the measurement of ionized calcium is currently considered to be the most accurate reflection of the calcium status of avian patients (Stanford, 2003)	Hyperproteinemia, dietary excess of vitamin D, dehydration, osteolytic bone tumor, ovulating hens	Hypocalcemic syndrome in some parrots, age-related in young birds, hypoalbuminemia
Cholesterol	Major lipid that is the precursor of steroid hormones and bile acids. Obtained from animal protein sources, as well as being synthesized by the liver	Hypothyroidism, liver disease, bile duct obstruction, starvation, high-fat diet, atherosclerosis	Liver disease, aflatoxicosis, low dietary fat, *Escherichia coli* endotoxemia
Creatinine	Derived from catabolism of creatine in muscle tissue and excreted by the kidneys. Does not provide an accurate assessment of avian renal function	Severe kidney damage, egg peritonitis, chlamydiosis, renal trauma, nephrotoxic drugs, feeding high-protein diets	Heavy metal toxicity

Continued

TABLE 6-8 Causes of Changes in Metabolic Tests in Avian Species—cont'd

Metabolite	Physiology	Causes of Increase	Causes of Decrease
Delta-aminolevulinic acid dehydratase	Delta-aminolevulinic acid dehydratase (ALAD) is an enzyme that is affected by the presence of heavy metals. Blood ALAD levels are decreased in heavy metal toxicity		
Glucose	Required as an energy source and must be maintained at adequate levels in the plasma. Blood levels are maintained by the conversion of liver glycogen. All plasma glucose is filtered from the blood through renal glomeruli and reabsorbed in the tubules	Higher in many juvenile birds, circadian rhythm, increases after feeding, stress, diabetes mellitus	Hepatic dysfunction, septicemia, aspergillosis, neoplasia, anorexia
Phosphorus	Inorganic phosphorus is derived from the diet and is a major constituent of bone, as well as playing a role in the storage, release, and transfer of energy in acid-base metabolism. Elevations of phosphorus are uncommon in birds	Severe renal damage, hypervitaminosis D, nutritional hyperparathyroidism	Hypovitaminosis D, malabsorption, long-term glucocorticoid therapy
Total protein	Most plasma proteins are synthesized in the liver (not immunoglobulins and protein hormones). Proteins form the basis of organ and tissue structure	Chronic infections, lymphoproliferative disease, dehydration, in females normal increase before egg laying	Chronic hepatopathy, malabsorption, wasting diseases, blood loss, enteropathy, parasitism, renal disease, starvation, malnutrition, overhydration, age-related in young birds
Triglycerides	Major storage form of lipids and important energy source. Synthesized in the intestinal mucosa and liver from components of fat digestion	Egg-related peritonitis, hyperadrenocorticism, starvation of obese birds, liver disease, gonadal disease, can also be iatrogenic after administration of androgens, estrogens and glucocorticoids	
Urea	Formed by protein breakdown in the liver and excreted by glomerular filtration from the kidney. Tubular reabsorption occurs and is dependent on the state of hydration. In dehydrated birds, urea is reabsorbed; in hydrated birds, most filtered urea is excreted	Dehydration, urethral obstruction	
Uric acid	Major product of nitrogen catabolism. Synthesized in the liver and renal tubules and eliminated by secretion into the renal tubules. By the time plasma uric acid levels are elevated, significant tubular damage has occurred. Grain-eating birds have lower uric acid levels than carnivorous birds	Ovulation, postprandial increase hypovitaminosis A–induced renal damage, dehydration, renal infection, renal intoxication, hypervitaminosis A, hypervitaminosis D_3, nephrotoxic drugs, gout (articular)	Juvenile birds have lower levels; severe liver disease

Source: Extracted and modified from: Hochleithner, 1994; Fudge, 1997; Harris, 2000; Montesinos *et al.*, 2013, Nemetz, 2013.

TABLE 6-9 Causes of Changes in Electrolyte Tests in Avian Species

Metabolite	Physiology in Avian Species	Causes of Increase	Causes of Decrease
Chloride	Major extracellular anion. Osmotically active constituent of plasma. Changes rarely seen in avian samples	Dehydration	
Potassium	Only 2% of the body's potassium is in the extracellular fluid: the remaining 98% is kept within the cells by potassium pumps	Severe tissue damage, renal failure, adrenal disease, acidosis, dehydration, hemolytic anemia	Chronic diarrhea, diuretic therapy, alkalosis
Sodium	Present in extracellular fluid and responsible for determining extracellular fluid volume and osmotic pressure	Salt poisoning, excess water loss, decreased water intake	Renal disease, diarrhea, overhydration
Bicarbonate	Alterations of bicarbonate are characteristic of acid-base balance	Increase caused by metabolic acidosis	Decrease caused by metabolic alkalosis

Source: Extracted and modified from Hochleithner, 1994.

ranges in the blood or tissues of healthy animals must be known to determine mineral status. Trace minerals, including copper, manganese, selenium, and zinc function as accessory factors to enzymes and are required in small amounts in the diet (National Research Council, 1980; National Research Council, 1994). Trace minerals have been extensively studied in the blood and tissues of domestically farmed animals such as poultry, and health examination of flocks frequently involves an assessment of mineral status. Some field studies on free-living birds have also used plasma mineral and biochemical values to evaluate health and nutritional status of wild populations, as well as to providing comparative data useful to clinicians working with captive birds of the same species (McDonald *et al.*, 2010). Although the collection of blood samples is a practical and minimally invasive technique for screening nondomestic birds, further studies are warranted to correlate tissue (e.g., liver) and blood levels. For example, liver levels are considered to be the most reliable indicator of copper status in domestic species (Keen and Graham, 1989). Table 6-10 presents a summary of the physiology and effects of toxicity and deficiency in avian species of some minerals for which blood or tissue levels in birds have been published.

VITAMINS

Vitamins are defined as natural food components that are present in minute quantities, are organic in nature, and are essential for normal metabolism and health (Brue, 1994). They cause specific and characteristic deficiency symptoms when they are limited in the diet. A summary of the physiology and effects of changes in vitamin levels in avian species is reported in Table 6-11.

The diagnosis of vitamin deficiencies in birds has tended to be diagnosed on the basis of clinical signs and response to supplementation. However, now that tests measuring vitamin levels in tissues and blood are becoming more widespread, the ability of veterinarians to diagnose deficiencies and to provide more rational supplementation will undoubtedly improve. Plasma vitamin E concentrations have been measured in a wide range of captive avian species (Gulland *et al.*, 1988; Dierenfeld 1989; Schweigert *et al.*, 1991; Dierenfeld and Traber, 1992; Dierenfeld *et al.*, 1993; Anderson *et al.*, 2002), but blood levels of other vitamins have not been so widely reported. Blood vitamin levels in some avian species are presented in Appendix 3.

ACID-BASE BALANCE

The diagnosis of acid-base disturbances and electrolyte imbalances in humans and many domestic animals is well documented, but little information has been published about birds. Venous heparinized blood is the sample most commonly used for blood gas analysis in birds, and ideally determination should be carried out as rapidly as possible in-house. The advent of lower cost and portable units such as the I-STAT blood analyzer (Abbott Laboratories, Chicago, Ill., USA) makes blood gas a more practical analysis for clinicians. For assessment of acid-base status, pH, P_{CO_2} and H_{CO_3} levels are considered the most appropriate parameters (Martin, 1994). The assessment of the acid-base balance of sick birds is important when determining the most appropriate type of solution to use in fluid therapy. For example, lactated Ringer solution is more appropriate in birds that are acidotic, and a 5% dextrose saline solution is more appropriate for birds with alkalosis (McKinney, 2003). Blood gas values for some avian species are presented in Appendix 3. Further work is warranted to establish reference ranges and interpretive guidelines in birds.

AGE-RELATED BIOCHEMISTRY CHANGES

Investigations in many juvenile avian species, including psittacines (Clubb *et al.*, 1990; Joyner and Duarte, 1994), storks (Montesinos *et al.*, 1997), and bustards (Bailey *et al.*, 1998a, 1998b, 1999a) have reported age-related changes in biochemistry values. These studies demonstrate significant differences in many chemistry values, including glucose, total protein, alkaline phosphatase (ALKP), aspartate aminotransferase (AST), lactate dehydrogenase (LDH), and calcium, between healthy adult and juvenile birds. Calcium, total protein, and AST levels tend to be significantly lower in the plasma of juvenile birds compared with adults. The requirement of protein, a major constituent of tissues, for growth may explain the low circulating levels in juvenile birds. High plasma ALKP is seen in juvenile birds and is considered to be associated with normal bone growth and development. As an example, Figs. 6-75 and 6-76 show the changes in plasma calcium and ALKP in growing kori bustards.

URINALYSIS

Urinalysis is indicated if renal disease is suspected. Very few investigations have defined normal parameters of avian urine (Rosskopf *et al.*, 1986; Halsema *et al.*, 1988; Huchzermeyer, 1998; Tschopp *et al.*, 2007). Tissue enzyme studies have shown that avian kidney tissues contain high concentrations of glutamate dehydrogenase (GLDH), gamma glutamyl transferase (GGT), ALKP, CK, LDH, AST, and alanine aminotransferase (ALT) (Lumeij and Wolfswinkel, 1987; Lumeij *et al.*, 1988a, Bailey *et al.*, 1999b). In mammals, it is known that after renal damage these enzymes are largely excreted via the urine (Keller, 1981),

TABLE 6-10 Physiology and Effects of Toxicity and Deficiency in Avian Species of Some Minerals

Trace Element	Physiology in Avian Species	Signs of Toxicity	Signs of Deficiency
Copper	Component of important enzymes and involved in hematopoiesis and in absorption and transfer of iron and hemoglobin synthesis. Serum copper levels are also useful in suspected cases of deficiency because low levels are considered to be diagnostic. The normal range of copper in the blood of most healthy animals is between 50 and 150 mg/dL, although birds, fish, and marsupials are characterized by copper levels that are half these values (Keen and Graham, 1989). Published sera levels of copper in birds include: kori bustards 67.8-101.6 mg/dL, ratites 15-28 mg/dL, and Hispaniola Amazons (*Amazona ventralis*) 6.5-18 mg/dL (Bailey *et al.*, 2004; Angel, 1996; Osofsky *et al.*, 2000)	Chick mortality, gizzard erosion, and anemia. May induce selenium deficiency	Anemia, reduced feather pigmentation, bone demineralization, heart disorders, abnormal feather growth, and ataxia and paralysis of chicks
Selenium	Essential for enzyme activity and other biochemical processes. An essential component of glutathione peroxidase, which inhibits the formation of peroxidases	Poor reproductive performance, embryonic deaths and deformities	Simultaneous deficiency of selenium and vitamin E results in specific deficiency diseases
Zinc	Essential for enzyme activity and other biochemical processes. The most widely used method for assessing zinc status is the measurement of plasma levels (Keen and Graham, 1989). Typical plasma or serum levels of zinc in most species range from 50-150 mg/dL (Keen and Graham, 1989), and the normal range for zinc in Hispaniola Amazons (*Amazona ventralis*) is 125-229 mg/dL (Osofsky *et al.*, 2000)	Leg paralysis and bone demineralization. High levels may result in secondary selenium deficiency	Embryonic abnormalities and reduced hatchability, scaling of the skin, poor feather development, impaired reproduction, shortened and thickened long bones, and enlarged hock joints. Zinc absorption is reduced by high dietary levels of calcium and phytate phosphorus
Magnesium	Essential for normal physiologic processes such as cellular respiration and enzyme activity and is involved in bone and egg shell formation	Altered bone calcification and mortality in young birds	Reduced hatchability, chick mortality, and neuromuscular convulsions. Magnesium absorption is reduced by high dietary levels of calcium and phosphorus

Source: Extracted and modified from Keen and Graham, 1989; Anderson *et al.*, 2002; National Research Council, 1994; Friend and Franson, 1999.

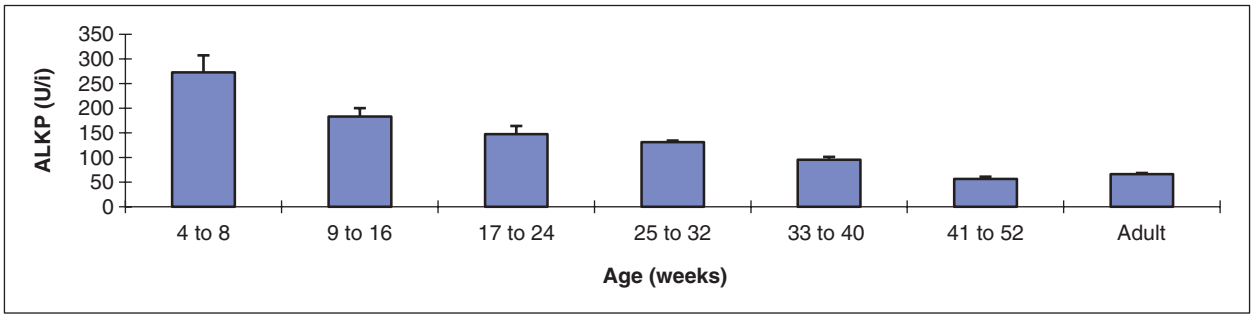


FIGURE 6-75 Plasma alkaline phosphatase levels (U/i) in kori bustards. ALKP, Alkaline phosphatase.

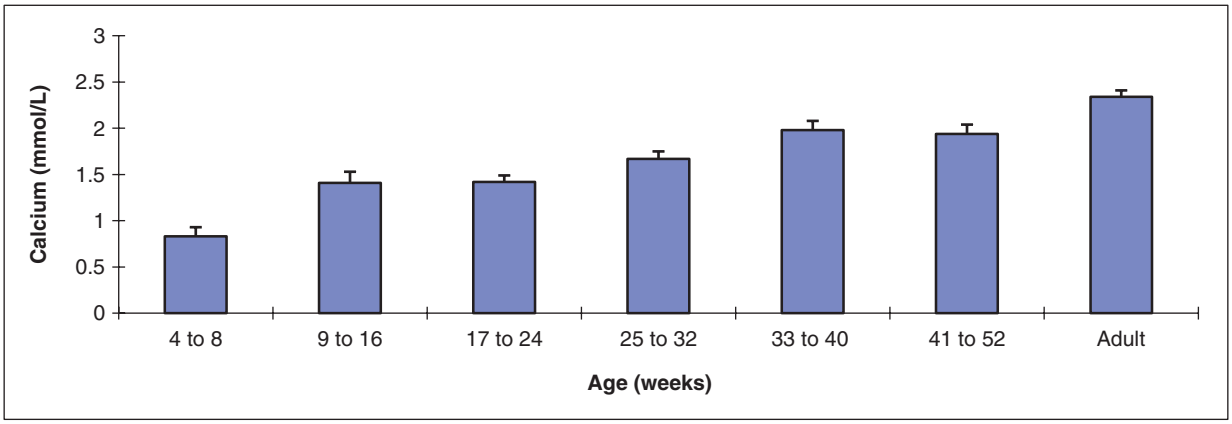


FIGURE 6-76 Plasma calcium levels (U/i) in kori bustards.

TABLE 6-11 A Summary of the Physiology and Effects of Changes in Vitamin Levels in Avian Species

Vitamin	Physiology in Avian Species	Causes and Effects of Changes in Vitamin Levels
A	Fat-soluble vitamin essential for growth and differentiation of epithelial tissues, mucopolysaccharide formation, stability of cell membranes, growth of bones, and normal reproduction. Also improves the immune system. Stored in the liver and has the potential to act as a cumulative toxicant. Deficiencies can result from insufficient dietary fat, insufficient antioxidant protection, or disorders that interfere with fat digestion or absorption. Liver disease may reduce the bird's ability to store vitamin A	**Deficiency**—Embryo mortality and abnormalities; susceptibility to respiratory infections; visual disorders; squamous metaplasia of mucous membranes; hyperkeratosis; decreased testis size and testosterone levels; urate deposits in the kidneys and ureters; egg binding; poorly formed eggs **Toxicity**—Bone abnormalities; spontaneous fractures; conjunctivitis; enteritis; suppressed keratinization; internal hemorrhages; fatty liver and kidneys; secondary deficiencies of other fat-soluble vitamins
D_3	Fat-soluble vitamin essential for the absorption of calcium and consequently for normal bone and eggshell formation. It is destroyed by excess radiation with ultraviolet light and oxidation in the presence of rancidifying fatty acids. There are two forms of this vitamin: ergocalciferol (D_2), a plant derivative, and cholecalciferol (D_3), produced in the bird's body. Vitamin D_3 is synthesized in avian skin exposed to ultraviolet light and is 30-40 times more potent than vitamin D_2. A dietary source of vitamin D_3 is needed by animals that do not have access to ultraviolet light	**Deficiency**—Thin, soft-shelled eggs; embryonic abnormalities and mortality; metabolic bone disease; leg weakness; seizuring; pathologic bone fractures; poor feathering. Can be induced by high dietary vitamin A or E levels **Toxicity**—Reduced fertility; decreased eggshell quality; soft tissue calcification; renal and artery calcification; bone demineralization; muscular atrophy
E	Fat-soluble vitamin that provides natural antioxidation protection for cells, fatty acids, and other fat-soluble vitamins. Working in conjunction with vitamin E are several metalloenzymes that incorporate manganese, zinc, copper, iron, and selenium. The selenium-containing glutathione peroxidase is the most important of these enzymes. Because of their similar activity, selenium and vitamin E tend to have a sparing effect on each other. Vitamin E is active in several metabolic systems, including cellular respiration, normal phosphorylation reactions, ascorbic acid synthesis, and sulfur amino acid synthesis. It also has effects on immunity by increasing phagocytosis and antibody production, as well as stimulating macrophage and lymphocyte activity	**Deficiency**—Low fertility; embryonic mortality; low hatchability; immunosuppression; testicular degeneration; and specific clinical abnormalities such as encephalomalacia, exudative diathesis, and muscular myopathies. May be predisposed by giardiasis **Toxicity**—Enlarged fatty livers; waxy feathers. High levels can cause secondary deficiency signs of bone demineralization or blood clotting failure if vitamins D_3 and K are marginal
K	Fat-soluble vitamin essential for normal blood clotting. It comes from three sources: green plants, bacteria, and synthetic forms. The microbial synthesis in the intestinal tract is significant in most species. The requirements of this vitamin vary according to the extent to which different species use the synthesized vitamin K and to which they practice coprophagy. Destroyed by oxidation, alkaline conditions, strong acids, ultraviolet light, and some sulfur drugs. Vitamin K also requires the presence of dietary fats and bile salts for absorption from the gut, so decreased pancreatic and biliary function can impair normal absorption	**Deficiency**—Embryonic mortality; hemorrhaging; anemia; altered bone metabolism. Can be induced by high dietary levels of vitamins A or E or by prolonged antibiotic treatment **Toxicity**—High levels can cause chick mortality and anemia
B_1	Thiamine is a water-soluble vitamin essential for enzyme activity and cellular respiratory control, as well as being involved in nerve activity. It is common in plant and animal food sources but generally at low concentration. Several compounds in nature possess antithiamine activity. These include amprolium, which inhibits thiamine absorption from the intestine, thiaminases, which are found in raw fish, and thiamine antagonists such as tannic acid. Thiamine is not stored in the body for a long time	**Deficiency**—Embryonic mortality; muscular paralysis; ataxia; convulsions; neurologic signs; organ atrophy **Toxicity**—Not studied in birds. High levels in mammals can cause depression of the respiratory center and blockage of nerve transmission
B_2	Riboflavin is a water-soluble vitamin essential for enzyme activity, carbohydrate utilization, cellular metabolism and respiration, uric acid formation, amino acid breakdown, and drug metabolism. It is destroyed by ultraviolet light and alkaline solutions. Very little riboflavin is stored in the body and it is rapidly excreted	**Deficiency**—Embryonic abnormalities and mortality; chick mortality; curled toe paralysis and other neuromuscular disorders; dermatitis; poor feather pigmentation; splayed legs; fatty liver **Toxicity**—Not reported in birds. Toxicity not thought to be a risk because it is not well absorbed from the gut

Continued

TABLE 6-11 A Summary of the Physiology and Effects of Changes in Vitamin Levels in Avian Species—cont'd

Vitamin	Physiology in Avian Species	Causes and Effects of Changes in Vitamin Levels
B_6	Pyridoxine is a water-soluble vitamin involved in a number of enzyme systems as a coenzyme. It is required in all areas of amino acid utilization, the synthesis of niacin, and the formation of antibodies. It is destroyed by oxidation	**Deficiency**—Reduced hatchability; ataxia; neuromuscular disorders; perosis; hemorrhaging; gizzard erosion **Toxicity**—Acute death in falcons after 20 mg/kg IM injection has been reported (see Chapter 10)
B_{12}	Cyanocobalamin is a product of bacterial biosynthesis and therefore must be obtained by consuming a bacterial source or animal tissues that accumulate the vitamin. It is a critical component of many metabolic pathways and is involved in the synthesis of nucleic acids and protein, as well as carbohydrates and fats. Most vitamin B_{12} in the body is found in the liver, with secondary stores in the muscles. Vitamin B_{12} is stored efficiently, with a long biological half-life of 1 year in humans	**Deficiency**—Embryo abnormalities and mortality; chick mortality; gizzard erosion; poor feathering **Toxicity**—Not reported in birds
Biotin	Water-soluble vitamin that is an active part of four different carboxylase enzymes in the body involved in the metabolism of energy, glucose, lipids, and some amino acids. It is destroyed by strong acids and bases, oxidizing agents, and the protein avidin in raw egg albumin. Biotin is widely distributed in foods at low concentrations. The synthesis of biotin by intestinal microflora may be important	**Deficiency**—Embryo abnormalities and mortality; poor growth; dermatitis; perosis and leg abnormalities; fatty liver–kidney syndrome **Toxicity**—Not reported in birds
Choline	Water-soluble vitamin that has four important metabolic functions: (1) as a component of phospholipids and therefore in maintaining cell integrity, (2) maturation of the cartilage matrix of bone, (3) fat metabolism in the liver, and (4) acetylated to form the neurotransmitter acetylcholine. Although most animals synthesize choline, young animals cannot synthesize enough to meet the demands for growth	**Deficiency**—Reduced hatchability; perosis and enlarged hocks; hepatic steatitis; fatty liver syndrome **Toxicity**—Not reported in birds
Folic acid	Water-soluble vitamin involved in amino acid metabolism and bioconversion and in the synthesis of nucleotides. It is involved in red blood cell maturation, white cell production, functioning of the immune system, and uric acid formation. It is also essential for normal growth. Some sulfur drugs increase folic acid requirements. Zinc deficiency can decrease the absorption of folic acid by reducing activity of the mucosal enzyme that creates an absorbable form of folic acid. Enzyme inhibitors are present in some foods such as cabbage, oranges, beans, and peas	**Deficiency**—Embryo abnormalities and mortality; perosis; macrocytic anemia; poor feathering; loss of feather pigmentation **Toxicity**—Not reported in birds
Niacin	Water-soluble vitamin that is an important component of coenzymes NAD and NADP, which are involved in carbohydrate, fat, and protein metabolism	**Deficiency**—Dermatitis; perosis; stomatitis; enlarged hocks; anemia; digestive disorders; general muscular weakness **Toxicity**—Coarse, dense feathering and anteriorly directed short legs in chickens
C	Ascorbic acid has not been demonstrated to be a required nutrient for most avian species. It is easily manufactured in the liver and kidneys of birds but biosynthesis can be inhibited by deficiencies of vitamins A, E, and biotin. Ascorbic acid is involved in the synthesis of collagen, is an excellent antioxidant, and can regenerate vitamin E	**Deficiency**—Signs of vitamin C deficiency have not been documented in birds
Pantothenic acid	Water-soluble vitamin that is a structural component of coenzyme A, one of the most critical coenzymes in tissue metabolism. As such it is involved in fatty acid biosynthesis and degradation, and the formation of cholesterol, triglycerides, phospholipids, and steroid hormones. It is destroyed by heat, acids, and bases	**Deficiency**—Embryonic mortality; dermatitis; perosis; poor feathering; poor growth; fatty liver–kidney syndrome; ataxia; reduced semen volume and fertility **Toxicity**—Not reported in birds

Adapted from: Anderson (1995), Brue RN (1994), and McWhirter P (1994).
NAD, Nicotinamide adenine dinucleotide; NADP, nicotinamide adenine dinucleotide phosphate.

and biochemical analysis of avian urine may be a valid diagnostic assay that warrants more consideration than it has received to date.

The main problem with birds is collecting uncontaminated samples. Ostriches are the only bird to deposit urine separately from their feces, which enables the collection of clean samples of urine without being contaminated with protein from feces (Huchzermeyer, 1998; Mushi *et al.*, 2001). Under experimental conditions, samples have been collected from pigeons fitted with a cloacal cannula or from birds placed in holding cages with mesh floors, the samples from whom could be collected onto plastic sheeting. Transient polyuria can be induced in many species by administering water by crop tube and, in some groups of birds, such as raptors, collection of urine in a clinical setting is a

comparatively straightforward technique (Tschopp *et al.*, 2007). In falcons, a normal mute (intestinal and urinary tract output in raptors) consists of a dark black center (feces) surrounded by a pure chalky white urate mass, sometimes with a larger ring of clear urine. The liquid (urine) part of a fresh mute can readily be aspirated, centrifuged, and the supernatant analyzed using either a commercial dip stick or a standard biochemistry analyzer. Tschopp *et al.* (2007) found increased levels of GGT and total protein in sick falcons compared with healthy falcons (Table 6-12). Reference values of urinalysis in healthy falcons are presented in Appendix 3. Sometimes the only laboratory evidence of renal disease may be the presence of casts and urine sediment. Therefore samples should be carefully examined for the presence of these.

ELECTROPHORESIS

Evaluation of protein distribution by electrophoresis (EP) allows the early detection of inflammatory and humoral responses and is a well-established aid to diagnosis of many diseases of humans and animals (Kaneko, 1997; Cray and Tatum, 1998). Serum protein electrophoresis (SPE) has gained importance in bird medicine during the last decade, and like other biochemical parameters, interpretation patterns must be based on species-specific reference ranges because of differences in fractions between different avian species (Cray *et al.*, 2007; Kostka and Janeczek, 2013). Several attempts have been made to establish reference ranges for various bird species and EP patterns for common avian diseases (Quesenberry and Moroff, 1991; Blanco and Hofle, 2003; Gelli *et al.*, 2005; Spagnolo *et al.*, 2006, 2008; Kummrow *et al.*, 2012). However, lack of data for many bird species and inconsistency in methodology and interpretation still make it difficult for clinicians to include EP as part of routine diagnostic procedures in birds (Rosenthal *et al.*, 2005). Additionally the EP pattern may differ according to the method used (Cray *et al.*, 2011) and published EP patterns in birds can be inconsistent. Ceron *et al.* (2011) considers that laboratory-specific reference ranges are important because of the variation that can result from the use of different equipment and methods.

Plasma or serum can be used for EP, but it must be remembered that the fibrinogen in plasma samples can often obscure the electrophoretogram in the β-γ region (Thomas, 2000; Gelli *et al.*, 2005) (Figs. 6-77 and 6-78). Appropriate reference ranges for either serum or plasma should be referred to depending on the sample submitted for analysis. Hemolysis and lipemia can also interfere with EP results (Ceron *et al.*, 2011).

TABLE 6-12 Urinalysis in Avian Species

Parameter	Normal Physiology	Causes of Change in Avian Species
Color and consistency	Urine usually clear, exceptions include ratites and Anseriformes, which have opaque, cloudy urine	The color of the urine can change after ingestion or injection of water-soluble vitamins (e.g., vitamin B). Lead intoxication can cause chocolate-milk–colored urine and urates. Severe liver diseases (e.g., Pacheco disease and virus, chlamydiosis, falcon herpes virus) can increase the secretion of biliverdin, resulting in lime-green urine and urates
Specific gravity	Varies with the state of hydration. Measured with a refractometer. Values of 1.005-1.02 are considered normal	Any disease characterized by polyuria and polydipsia. Increased water loss without increased solute loss results in a low specific gravity and occurs in intravenous fluid therapy, hyperthyroidism, liver disease, pituitary neoplasia, and glucocorticoid therapy
pH	Most pet birds have a urinary pH between 6 and 8. pH is related to the diet: carnivores tend to have acidic urine and granivores more alkaline urine	Birds with urine pH less than 5 are considered acidotic
Urinary protein	Trace protein, probably because of fecal contamination, can be detected in the urine of the majority of birds	In raptors protein levels have been reported as being twice as high in urine from sick birds (e.g., aspergillosis, parasitic diseases, lead toxicosis, amyloidosis) compared with healthy birds
Glucose	Avian urine should not normally contain glucose. Trace levels may be detected in normal birds because of fecal contamination	In raptors glucose levels have been reported as being higher in urine from sick birds (e.g., aspergillosis, parasitic diseases, lead toxicosis, amyloidosis) compared with healthy birds
Blood	Commercial test strips can differentiate between hematuria and hemoglobinuria	Blood in the urine may originate from the cloaca, urinary, reproductive, or alimentary tracts. The diet should be taken into consideration: most raptors are positive for blood because of their meat diet
GGT	Avian kidney tissues have been shown to have high activity of many enzymes, including GGT	In raptors increased levels of GGT have been reported in urine from sick birds (e.g., aspergillosis, parasitic diseases, lead toxicosis, amyloidosis), whereas serum GGT levels were in the normal range for these birds. More work is needed on the clinical significance of urinary enzymes
Chloride	Chloride levels in urine depend mainly on the concentration of sodium chloride in food and also on the state of hydration, which is influenced by climatic factors	Very few studies are available on chloride levels in the urine of birds. Urinalysis results from farmed healthy ostriches showed that ranges of chloride were much higher (up to 400 times higher) than values in falcons

Source: Extracted and modified from Hochleithner, 1994; Tschopp *et al.*, 2007.
GGT, Gamma glutamyl transferase.

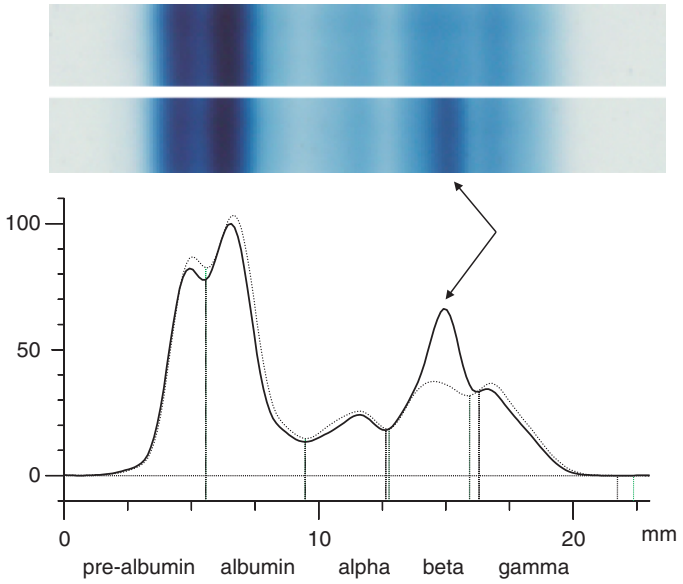


FIGURE 6-77 Comparison of protein electrophoresis of plasma (*solid, lower band*) and serum (*dotted, upper band*) from a clinically healthy hybrid falcon in a high-resolution agarose gel (SAS-1 SP-24 SB). The clearly elevated peak and a band in the plasma pattern represent the beta-globulin fraction (*arrows*). (From Kummrow M, Vorbruegen S, Silvanose C, et al: Serum protein electrophoresis in healthy and *Aspergillus* sp. infected falcons, *J Avian Med Surg* 26(4):213–220, 2012.)

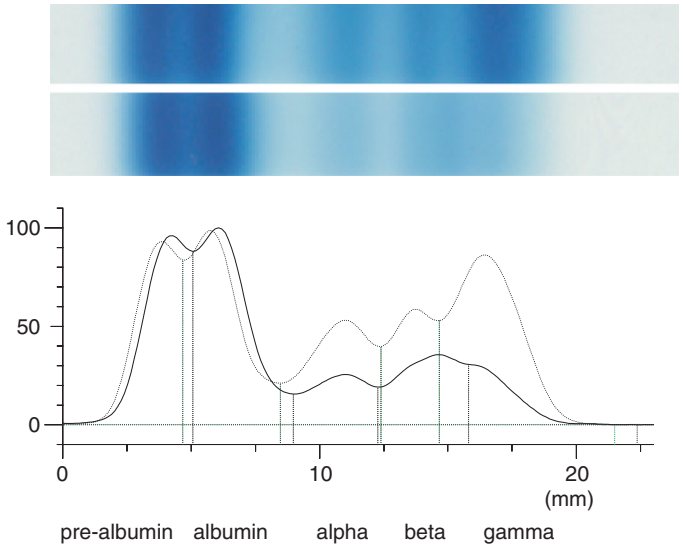


FIGURE 6-78 Comparison of serum protein electrophoresis patterns of a clinically healthy hybrid falcon (*solid, lower band*) and a gyrfalcon with aspergillosis (*dotted, upper band*) in a high-resolution agarose gel (SAS-1 SP-24 SB). (From Kummrow M, Vorbruegen S, Silvanose C, et al: Serum protein electrophoresis in healthy and *Aspergillus* sp. infected falcons, *J Avian Med Surg* 26(4):213–220, 2012.)

Abnormal electrophoretic patterns may precede seroconversion, detection of pathogens, or biochemical and hematologic values. Consequently, EP may be useful as an accessory tool to indicate avian diseases such as aspergillosis, delineating the need for more comprehensive diagnostic techniques (Kumrow *et al.*, 2012). A summary of the conditions associated with changes in EP fractions in avian species is reported in Table 6-13. For the monitoring of recovery and success of therapy, EP may be helpful when individual cases are followed serially during treatment, but further studies are needed to demonstrate the usefulness of EP in routine avian medicine.

TABLE 6-13 Causes of Changes in Plasma Electrophoresis Values in Avian Species

Change in Fraction	Change Associated with:
Decreased albumin	Decreased production (e.g., hepatic insufficiency), increased loss (enteritis), increased use (chronic inflammation)
Elevated α-globulins	Acute inflammation, infection, female reproductive activity
Elevated β-globulins	Acute inflammation, infection
Elevated γ-globulins	Chronic inflammation, infection

Source: Dorrestein, 2008.

REFERENCES

Anderson S: Bustard micronutrient review, *NARC External Report* No. 4., 1995.

Anderson SJ, Dawodu A, Patel M, et al: Plasma concentrations in six species of bustards (Gruiformes: Otididae), *J Wildl Dis* 38:414–419, 2002.

Angel CR: Serum chemistries and vitamin D metabolites in ostriches, emus, rheas and cassowaries. *International Conference on Improving Our Understanding of Ratites in a Farming Environment*, Manchester, pp 122–124, 1996.

Bailey TA, Silvanose CD, Combreau O, Howlett JC: Normal blood concentrations of copper, magnesium and zinc in stone curlew and five species of bustards in the United Arab Emirates. *Proceedings of the European Association of Zoo and Wildlife Veterinarians Meeting*, Ebeltoft, pp 297–301, 2004.

Bailey TA, Wernery U, Howlett J, et al: Normal blood chemistry and age-related changes in the white-bellied bustard (*Eupodotis senagalensis*), with some clinical observations, *Comparative Haematology International* 8:61–65, 1998a.

Bailey TA, Wernery U, Howlett J, et al: Age-related plasma chemistry changes in the buff-crested bustard (*Eupodotis ruficrista gindiana*), *J Vet Med* 45:635–640, 1998b.

Bailey TA, Wernery U, Howlett J, et al: Age-related plasma chemistry changes in houbara *(Chlamydotis undulata)* and kori bustards *(Ardeotis kori)*, *J Wildl Dis* 35:31–37, 1999a.

Bailey TA, Wernery U, Howlett J, et al: Diagnostic enzyme profile in houbara bustard tissues (*Chlamydotis undulata macqueenii*), *Comparative Haematology International* 9:36–42, 1999b.

Bailey TA, Wernery U, Naldo J, Samour JH: Concentrations of creatine kinase and lactate dehydrogenase in captive houbara bustards (*Chlamydotis undulata macqueenii*) following capture, *Comparative Haematology International* 7:113, 1997.

Blanco JM, Hofle U: Plasma protein electrophoresis as diagnostic and prognostic tool in raptors, *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp 256–261, 2003.

Bogin E, Avidar Y, Israeli B: Enzyme profile of turkey tissues and serum, *J Vet Med* 23:858–862, 1976.

Bogin E, Israeli B: Enzyme profile of heart and skeletal muscles, liver and lung of roosters and geese, *J Vet Med* 23:152–157, 1976.

Brue RN: Nutrition. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine and surgery: principles and applications*, Lake Worth, 1994, Wingers Publishing, pp 63–95.

Ceron JJ, Caldin M, Martinez-Subiela S: Answers to some common questions on serum protein electrophoresis, *Vet Rec* 168(17):453–454, 2011.

Chalmers GA, Barrett MW: Capture myopathy. In Hoff GL, Davis JW, editors: *Noninfectious diseases of wildlife*, Ames, 1982, Iowa State University Press, pp 84–89.

Clubb SL, Schubot RM, Joyner K, et al: Hematologic and serum biochemical reference ranges in juvenile macaws, cockatoos and eclectus parrots. *Proceedings of the Association of Avian Veterinarians*, Phoenix, pp 58–59. Association of Avian Veterinarians, Bedford, TX, 1990.

Cornelius CE, Bishop J, Switzer J, et al: Serum and tissue transaminase activities in domestic animals, *Cornell Vet* 49:116–126, 1958.

Cray C: Reference intervals: new guidelines for an essential resource, *Vet Rec* 171:215–216, 2012.

Cray C, King E, Rodriguez M, et al: Differences in protein fractions of avian plasma among three commercial electrophoresis systems, *J Avian Med Surg* 25(2):102–110, 2011.

Cray C, Rodriguez M, Zaias J: Protein electrophoresis of psittacine plasma, *Vet Clin Pathol* 36:64–72, 2007.

Cray C, Tatum LM: Application of protein electrophoresis in avian diagnostics, *J Avian Med Surg* 12:4–10, 1998.

Dierenfeld ES: Vitamin E deficiency in zoo reptiles, birds, and ungulates, *J Zoo Wildl Med* 20:3–11, 1989.

Dierenfeld ES, Sheppard CD, Langenberg J, et al: Vitamin E in cranes: reference ranges and nutrient interactions, *J Wildl Dis* 29:98–102, 1993.

Dierenfeld ES, Traber MG: Vitamin E status of exotic animals compared with livestock and domestics. In Packer L, Fuchs J, editors: *Vitamin E in health and disease*, New York, 1992, Marcel Dekker, pp 345–370.

Dorrestein GM: Clinical pathology and post-mortem examination. In Chitty J, Lierz M, editors: *BSAVA manual of raptors, pigeons and passerine birds*, Cheltenham, 2008, British Small Animal Veterinary Association, pp 3–96.

Dorrestein GM, De Bruijne JJ, Buitelaar MN: Bioavailability, tissue distribution, muscle injury and effects on serum enzyme activity after parenteral administration of doxycycline to pigeons, PhD thesis, University of Utrecht, Netherlands, 1986.

Friend M, Franson JC: *Field Manual of Wildlife Diseases*, 1999, US Department of the Interior and US Geological Survey, Wildlife Health Center, 6006 Schroeder Road, Madison, WI 53711 pp 317–337.

Fudge A: Avian clinical pathology: hematology and biochemistry. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, 1997, WB Saunders, pp 142–157.

Gelli D, Franceschini F, Bedin M, et al: Serum biochemical and electrophoresic patterns in the Eurasian buzzard *(Buteo buteo):* reference values, *Proceedings of the Annual Conference of the European Association of Avian Veterinarians*, pp 166–170, 2005.

Gulland FMD, Ghebremeskel K, Williams G, Olney PJS: Plasma vitamins A and E, total lipid and cholesterol concentrations in captive jackass penguins (*Spheniscus demersus*), *Vet Rec* 123:666–667, 1988.

Halsema WB, Alberts H, deBruijne JJ, Lumeij JT: Collection and analysis of urine in racing pigeons (*Columba livia domestica)*, *Avian Pathology* 17:221–225, 1988.

Hochleithner M: Biochemistries. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine and surgery: principles and applications*, Lake Worth, 1994, Wingers Publishing, pp 223–245.

Huchzermeyer FW: *Diseases of ostriches and other ratites*, Onderstepoort, 1998, Agricultural Research Council, Republic of South Africa, p 48.

Joyner KL, Duarte JPS: Hematology and serum biochemistry values in captive juvenile yellow-naped amazons (*Amazona auropalliata*). *Proceedings of the Association of Avian Veterinarians*, Reno, pp 185–188, 1994.

Kaneko JJ: Serum proteins and the dysproteinemias. In Kaneko JJ, Harvey JW, Bruss ML, editors: *Clinical biochemistry of domestic animals*, ed 5, San Diego, 1997, Academic Press, pp 117–138.

Keen CL, Graham TW: Trace elements. In Kaneko JJ, editor: *Clinical biochemistry of domestic animals*, San Diego, 1989, Academic Press, pp 753–795.

Keller P: Enzyme activities in the dog: tissue analyses, plasma values, and intracellular distribution, *Am J Vet Res* 42:575–582, 1981.

Kostka V, Janeczek F: Diagnostic value of protein electrophoresis in large psittacines, *International Conference on Avian, Herpetological and Exotic Mammal Medicine,* Germany, pp 421–422, 2013.

Kummrow M, Vorbruegen S, Silvanose C, et al: Serum protein electrophoresis in healthy and *Aspergillus* sp. infected falcons, *J Avian Med Surg* 26(4):213–220, 2012.

Lumeij JT: A contribution to clinical investigative methods for birds, with special reference to the racing pigeon, *Columba livia domestica*, PhD thesis, University of Utrecht, Netherlands. 1987.

Lumeij JT, de Bruijne JJ, Slob A, Rothuizen J: Enzyme activities in tissues and elimination half-lives of homologous muscle and liver enzymes in the racing pigeon (*Columba livia domestica*), *Avian Pathology* 17:851–864, 1988a.

Lumeij JT, Meidam M, Wolfswinkel J, et al: Changes in plasma chemistry after drug-induced liver disease or muscle necrosis in racing pigeons (*Columba livia domestica*), *Avian Pathology* 17:865–874, 1988b.

Lumeij JT, Remple JD: Plasma urea, creatinine and uric acid concentrations in relation to feeding in peregrine falcons *(Falco peregrinus)*, *Avian Pathology* 20:79–83, 1991.

Lumeij JT, Remple JD: Plasma bile acid concentrations in response to feeding in peregrine falcons *(Falco peregrinus)*, *Avian Dis* 36:1060–1062, 1992.

Lumeij JT, Wolfswinkel J: Tissue enzyme profile of the budgerigar, *Melopsittacus undulatus*. In: A contribution to clinical investigative methods for birds, with special reference to the racing pigeon, *Columba livia domestica*, pp 71–78, PhD thesis, University of Utrecht, Netherlands, 1987.

Martin L: pH, P_aCO_2, electrolytes and acid–base status. In *All you really need to know to interpret arterial blood gases*, Baltimore, 1994, Lippincott Williams & Wilkins, pp 107–129.

McDonald DL, Jaensch S, Harrison GJ, et al: Health and nutritional status of wild Australian psittacine birds: an evaluation of plasma and hepatic mineral levels, plasma biochemical values, and fecal microflora, *J Avian Med Surg* 24(4):288–298, 2010.

McKinney PA: Clinical applications of the I-Stat blood analyser in avian practice. *Proceedings of the European Association of Avian Veterinarians*, Tenerife, pp 341–345, 2003.

McWhirter P: Malnutrition. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine and surgery: principles and applications*, Lake Worth, 1994, Wingers Publishing, pp 842–861.

Merritt EL, Fritz CL, Ramsay EC: Hematologic and serum biochemical values in captive American flamingos (*Phoenicoterus ruber ruber*), *J Avian Med Surg* 10:163–167, 1996.

Montesinos A, Ardiaca M, Bonvehi C, et al*:* Fasting ammonia levels and bile acid levels in diagnosis of hepatic disease in Amazon parrots (*Amazona* sp.). *International Conference on Avian, Herpetological and Exotic Mammal Medicine,* Germany, p 418, 2013.

Montesinos A, Sainz A, Pablos MV, et al: Hematological and plasma biochemical reference intervals in young white storks, *J Wildl Dis* 33:405–412, 1997.

Mushi EZ, Binta MG, Isa JW: Biochemical composition of urine from farmed ostriches (*Struthio camelus*) in Botswana, *J S Afr Vet Assoc* 72:46–48, 2001.

National Research Council: *Mineral tolerance of domestic animals*, Washington, D.C., 1980, National Academy Press.

National Research Council: *Nutrient requirements of poultry*, ed 9, Washington, D.C., 1994, National Academy Press.

Nemetz L: The relevance of plasma triglyceride determination and hypertriglyceridemia in birds. *International Conference on Avian, Herpetological and Exotic Mammal Medicine,* Germany, pp 419–420, 2013.

Osofsky A, Jowett PL, Hosgood G, Tully T: Normal blood concentrations for lead, zinc, iron and copper in Hispaniolan amazons (*Amazona ventralis*). *Proceedings of the Association of Avian Veterinarians*, Portland, pp 243–244, 2000.

Petrie A, Sabin C: *Medical statistics at a glance*, Oxford, 2000, Blackwell Science, p 174.

Petrie A, Watson P: *Statistics for veterinary and animal sciences*, Oxford, 1999, Blackwell Science, p 299.

Pond J, Thompson S, Hennen M, et al: Effects of ultracentrifugation on plasma biochemical values of prefledged wild peregrine falcons *(Falco peregrinus)* in northeastern Illinois, *J Avian Med Surg* 26(3):140–143, 2012.

Quesenberry K, Moroff S: Plasma electrophoresis in psittacine birds. *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp 111–115, 1991.

Rosenthal KL, Johnston MS, Shofer FS: Assessment of the reliability of plasma electrophoresis in birds, *Am J Vet Res* 66:375–378, 2005.
Rosskopf WJ, Woerpel RW, Lane RA: The practical use and limitations of the urinalysis in diagnostic pet avian medicine: with emphasis on the differential diagnosis of polyuria, the importance of cast formation in the avian urinalysis and case reports. *Proceedings of the Association of Avian Veterinarians*, Miami, pp 61–73, 1986.
Scheideler SE, Wallner-Pendleton EA, Schneider N, Carlson M: Determination of baseline values for skeletal (leg bone) growth, calcification and soft tissue mineral accretion. *Proceedings of the Association of Avian Veterinarians*, Reno, pp 111–120, 1994.
Schweigert FJ, Uehlein-Harrell S, Hegel GV, Wiesner H: Vitamin A (retinol and retinyl esters), a-tocopherol and lipid levels in plasma of captive wild mammals and birds, *J Vet Med A* 38:35–42, 1991.
Spagnolo V, Crippa V, Marzia A, et al: Hematologic, biochemical and protein electrophoretic values in captive tawny owls *(Strix aluco)*, *Vet Clin Pathol* 37:225–228, 2008.
Spagnolo V, Crippa V, Marzia A, Sartorelli P: Reference intervals for hematologic and biochemical constituents and protein electrophoretic fractions in captive common buzzards *(Buteo buteo)*, *Vet Clin Pathol* 35:82–87, 2006.
Stanford M: Measurement of ionised calcium in African grey parrots (*Psittacus erithacus*): the effect of diet. *Proceedings of the European Association of Avian Veterinarians*, Tenerife, pp 269–275, 2003.
Thomas JS: Protein electrophoresis. In Feldman BF, Zinkl JG, Jan NC, editors: *Schalm's veterinary haematology*, ed 5, Philadelphia, Pennsylvania, 2000, Williams and Wilkins, pp 899–903.
Tschopp R, Bailey TA, Di Somma A, Silvanose C: Urinalysis as a non-invasive procedure in *Falconidae*, *J Avian Med Surg* 21(1):1–7, 2007.
Williams SM, Holthaus L, Barron HW, et al: Improved clinicopathologic assessments of acute liver damage due to trauma in Indian ring-necked parakeets (*Psittacula krameri manillensis*), *J Avian Med Surg* 26(2):67–75, 2012.

FURTHER READING

Cray C, Wack A, Arheart KL: Invalid measurement of plasma albumin using bromocresol green methodology in penguins (*Spheniscus* spp, *J Avian Med Surg* 25(1):14–22, 2011.
Harris DJ: Clinical tests. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry KE, editors: *Avian medicine and surgery*, Philadelphia, 2000, WB Saunders, pp 43–51.
Lierz M, Hafez HM: Sex-related differences in plasma chemistry reference values in stone curlews (*Burhinus oedicnnemus*), *Vet Rec* 157:91–92, 2005.
Lumeij JT, Remple JD, Remple CJ, Riddle KE: Plasma chemistry in peregrine falcons *(Falco peregrinus)*: reference values and physiological variations of importance for interpretation, *Avian Pathology* 27:129–132, 1998.
Stanford M: Measurement of 25-hydroxycholecalciferol in captive grey parrots (*Psittacus e. erithacus*), *Vet Rec* 153:58–59, 2003.

BLOOD GASES AND CRITICAL CARE HEMATOLOGY AND CHEMISTRY ANALYSES

Hugues Beaufrère

INDICATIONS, SAMPLE COLLECTION, AND ANALYSIS

Arterial blood gas analysis is typically performed when an oxygenation problem is suspected and to help with the diagnosis of causes of hypoxia or assess response to treatment. Due to the small size of most bird species, the difficulties of collecting arterial blood samples, and the potential adverse effects of bleeding and hematoma, arterial blood sampling is uncommonly done in clinical cases. However, arterial blood samples are relatively simple to collect from larger birds and may be useful in these species. Acid-base disorders can be assessed on both arterial and venous blood samples, and because venous samples are much easier to collect, it is generally done in the latter. Reference values are similar between arterial and venous blood gases except for the oxygenation parameters (P_{CO_2} is slightly higher and pH slightly lower in venous samples). Venous blood gases and electrolytes analyses are indicated when fluid therapy replacement is implemented to select the appropriate fluid type and develop a fluid therapy plan. It may also help in the diagnosis of the origin and mechanism of fluid and electrolyte imbalances. Blood gas analysis is also frequently performed during anesthetic monitoring. Vertebrates maintain body pH and electrolytes within a very narrow range, and even small departures in homeostasis of these parameters may have devastating consequences.

Blood samples for blood gases are typically obtained using commercial heparinized syringes to have an appropriate titration of heparin, which is especially important for electrolyte measurements such as ionized calcium (Fig. 6-79). Syringes must be made airtight until analysis so as not to cause contamination with room air. Analysis should be performed within 15 minutes or the sample should be placed on ice for analysis within 1 hour. Because avian erythrocytes are nucleated, the rate of O_2 consumption may be high; therefore, arterial sample analysis should be prompt. In addition, the analysis should be

FIGURE 6-79 Example of a blood gas collection system with a pre-heparinized syringe with lyophilized heparin and a cap to prevent mixing with room air.

performed at avian body temperature, which is usually higher than mammals and is not routinely measured in clinical cases in birds (38 to 42° C).

A variety of blood gas analyzers are available. Large radiometers (e.g., ABL800 FLEX, Radiometer Medical, Akandevej, Denmark) are more precise and typically offer a panel of electrolytes that allow a thorough interpretation of acid-base disorders and their mechanisms (Fig. 6-80) (Table 6-14). These panels usually contain all blood gas parameters, most electrolytes, and other key analytes in critical care such as hemoglobin, glucose, and lactates. The I-Stat (Abbott Laboratories, Princeton, N.J., USA) is a popular point-of-care blood gas and electrolyte analyzer commonly used in bird studies and has been validated in a few experiments (Fig. 6-81) (Paula *et al.*, 2008; Montesinos and Ardiaca, 2013; Steinmetz *et al.*, 2007; Rettenmund *et al.*, 2014; Martin *et al.*, 2010). It uses a very low sample size of about 0.1 mL and is likely more easily available for private practitioners than costly reference blood gas analyzers. Different cartridges are available but a full panel of electrolytes and blood gas parameters is not available currently (see Table 6-14). For instance, most cartridges (except the EC8+) lack the chlorides, which prevent the calculation of the strong ion difference (SID) and anion gap (AG) and further categorization of metabolic acidosis and alkalosis. Likewise, most cartridges lack the lactates and P_{O_2}. The metabolic status of avian patients can also be assessed on a standard biochemistry panel as total C_{O_2} and electrolytes are frequently included. The total C_{O_2} is mainly influenced by the bicarbonates because the contribution of P_{CO_2} is low. However, this approach precludes evaluation of the respiratory component and a more thorough assessment of metabolic abnormalities, but it can give a good ballpark assessment.

FIGURE 6-80 Example of a reference blood gas analyzer, the ABL 800 FLEX (Radiometer Medical).

TABLE 6-14 Blood Gas and Electrolyte Profile of a Reference Blood Gas Analyzer and a Point of Care Blood Gas Analyzer with Selected Cartridges

Analytes	ABL 800 FLEX	I-Stat EC8+	I-Stat CG8+	I-Stat EG7+	I-Stat EG6+
Glucose	■	■	■		
Na^+	■	■	■	■	■
K^+	■	■	■	■	■
Cl^-	■	■			
pH	■	■	■	■	■
P_{CO_2}	■	■	■	■	■
P_{O_2}	■		■	■	■
HCO_3^-	■	■	■	■	■
Base excess	■	■	■	■	■
S_{O_2}	■		■	■	■
Hb	■	■	■	■	■
Lactates	■				
Ca^{2+}	■		■	■	
Urea		■			

FIGURE 6-81 Example of a point-of-care blood gas analyzer, the I-Stat (Abbott Laboratories).

INTERPRETATION OF AVIAN BLOOD GASES AND ELECTROLYTES

Approach to Interpretation

Because this topic may be extremely complex, only the essentials are presented herein; the reader is invited to consult additional references on blood gas interpretation for further details (DiBartola *et al.*, 2012; Johnson and Autran de Morais, 2012; DiBartola, 2012).

Reference intervals for blood gas and electrolytes are fairly similar in birds as compared with mammals (see Appendix 3). However, according to a large number of studies, birds tend to have a slightly higher venous pH of around 7.4 to 7.5 with a lower P_{CO_2} of around 30 to 35 mm Hg than domestic mammals (Zehnder *et al.*, 2014; Powell, 2015; Paula *et al.*, 2008; Heatley *et al.*, 2015; Steinmetz *et al.*, 2007; Montesinos and Ardiaca, 2013; Martin *et al.*, 2010; Stämpfli *et al.*, 2006; Rettenmund *et al.*, 2014). It is unknown if this is a result of respiratory alkalosis caused by stress-induced hyperventilation or a true representation of normal avian values. Birds sampled with various techniques including remote sampling from catheters showed an overall consistent trend of a higher pH and lower P_{CO_2} than mammals. However, a few studies showed a blood pH closer to 7.35 in some psittacine species (Montesinos and Ardiaca, 2013; Paula *et al.*, 2008). As a result, the interpretation of avian blood gas should take into account this higher pH and lower P_{CO_2}. Lactates may normally be high (up to 8-10 mmol/L) in birds on the radiometer panel because of manual restraint or flight activities before sampling and should not be interpreted with the same cut-off values as in domestic carnivores.

The approach to interpretation is identical to that of mammals, with the specific physiology and diseases of birds in mind (Fig. 6-82). First, the pH is assessed to determine whether an acidemia or alkalemia is present. Then the metabolic (bicarbonates, controlled by the kidneys) and the respiratory (P_{CO_2}, controlled by the lungs) components are evaluated with the factor trending in the direction of the pH considered the primary disorder. Base excess is typically −4 to 4 and is also used to detect metabolic disorders. Primary disorders include metabolic acidosis, metabolic alkalosis, respiratory acidosis, and respiratory alkalosis. Compensation is subsequently assessed to determine the presence of mixed or complex disorders (if the compensation is higher or lower than expected for the magnitude of changes in the primary disorder, expected values for compensation are unknown in birds but mammalian values may be used). A normal pH does not

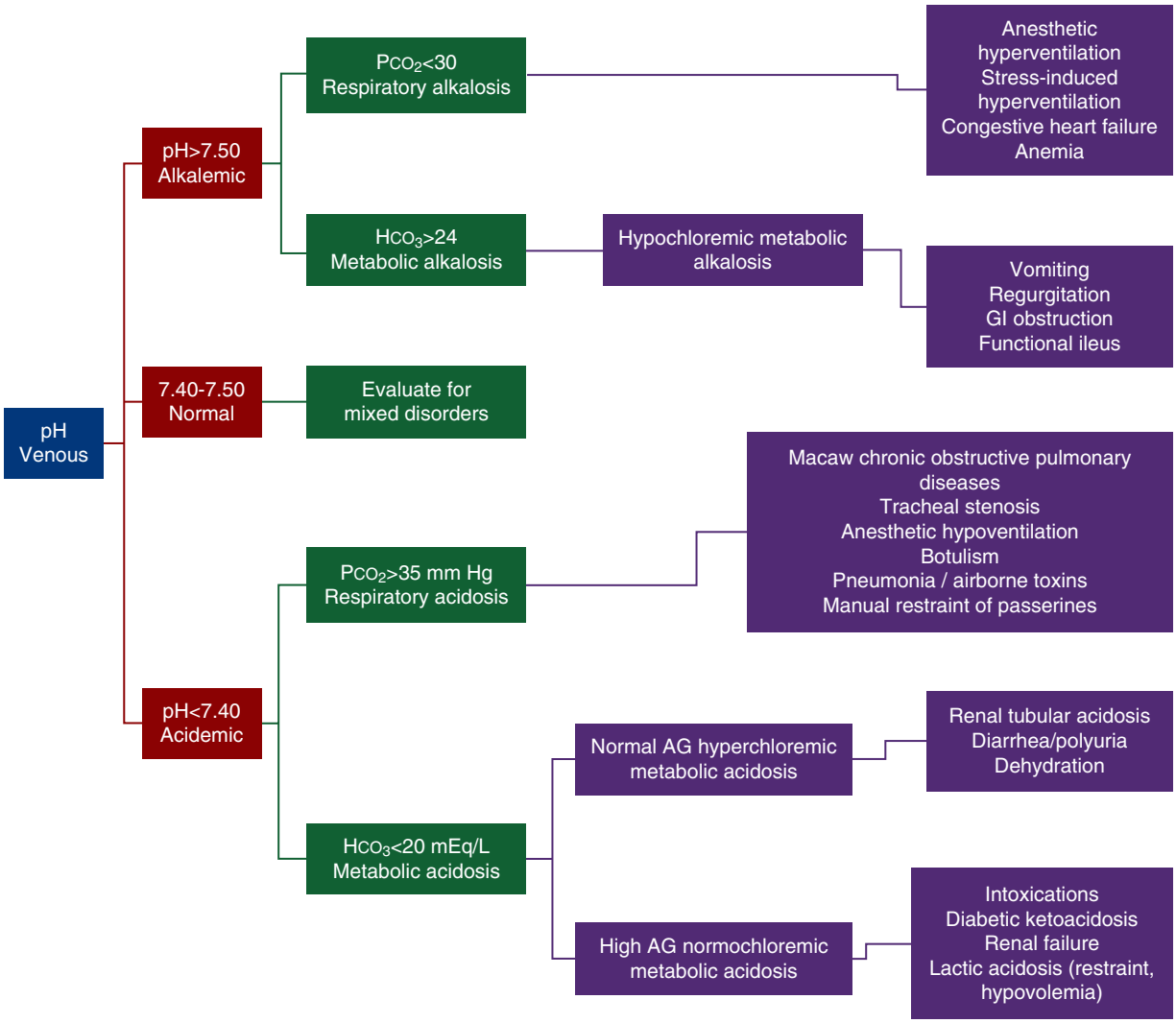


FIGURE 6-82 Simplified algorithm for the interpretation of acid-base disorders in birds with most frequent causes.

imply the patient is normal because mixed disorders may occur. The electrolytes are then evaluated, in particular the chlorides, potassium, and sodium. The AG (Na+K-Cl-Hco_3) and the SID (Na+K+Ca-Cl-lactates) are calculated, and the values are generally similar to mammals. In mammals, the AG is roughly equal to 16 mEq/L and is primarily used to differentiate between types of metabolic acidosis. In birds the AG appears to be slightly lower at around 10 to 15 mEq/L but is frequently increased by the lactates produced during restraint (Stämpfli *et al.,* 2006; Heatley *et al.,* 2015). The SID is frequently simplified to SID = Na-Cl, because they are the main components of the equation, and is roughly equal to 36 mEq/L. The SID was found to be roughly similar in pigeons as compared with mammals (Stämpfli *et al.,* 2006). The SID is used to assess the relative loss or gain of electrolytes compared with each other and may further refine the acid-base disorders assessment.

Hypoxemia

Hypoxemia is a lack of oxygen in the blood and can be caused by anemia, ventilation/perfusion mismatching, hypoventilation, cardiac failure, hemorrhage, and toxicities. Po_2 varies with the fraction of inspired o_2 (Fio_2). As in mammals, the normal arterial Po_2 of birds is typically around 95 to 100 mm Hg and the So_2 is around 97% to 100% (Paula *et al.,* 2008; Powell, 2015). Under anesthesia, Po_2 may be around 300 to 500 mm Hg. Hypoxia is defined with an arterial Po_2 < 80 mm Hg. The Po_2/FiO2 ratio (Fio_2 to fraction of inspired o_2) is helpful in determining the severity of hypoxemia, with a ratio < 300 indicating lung injuries and respiratory distress. Pulse oximetry may also be used to measure the So_2 but accuracy is variable in birds because interpretations are based on mammalian hemoglobin dissociation curves (Schmitt *et al.,* 1998).

A rough idea of oxygenation may also be obtained from venous samples. In mammals a Po_2 value lower than 30 mm Hg and an So_2 lower than 50% suggest hypoxia. Venous Po_2 is also affected by anything that decreases oxygen delivery to tissues, such as hypotension or heart failure. Assessment of the Pco_2 will also give an idea of ventilatory disorders. Lactates may also be elevated in the case of increased anaerobic metabolism.

The reader is invited to consult the section on oxygen therapy in Chapter 8 for further details on the treatment of hypoxia.

Metabolic Acid-Base Disorders

Metabolic acidosis is characterized by low blood bicarbonate levels and is traditionally classified into normal and increased AG metabolic acidosis. A high AG suggests the presence of nonmeasured anions, typically an organic acid that can cause metabolic acidosis such as uric acid, lactate, ketones, phosphates, and some toxic compounds. A low AG with concurrent elevation of chlorides (and low SID) suggests a loss of bicarbonates (chlorides increase to maintain electroneutrality) or a relative loss of sodium. In mammals expected respiratory compensation is a 0.7 mm Hg decrease in Pco_2 for each 1 mEq/L decrease in bicarbonates. The SID may also be used to characterize metabolic acid-base disorders. Strong anions (e.g., chlorides) are considered acids and strong cations (e.g., sodium) are considered bases. A low SID is caused by high chlorides in relation to sodium and thus leads to a hyperchloremic metabolic acidosis. A high SID is caused by low chlorides in relation to sodium and thus leads to a hypochloremic metabolic alkalosis. With some complex cases of metabolic acidosis, a complete biochemistry panel is necessary to help with the interpretation and the diagnosis of causes.

In birds common causes of high AG metabolic acidosis include renal failure (caused by the failure to excrete acid anions such as phosphates or uric acid) and lactic acidosis. Metabolic acidosis from the production of lactates during struggling also frequently occurs. Common causes of normal AG metabolic acidosis include renal failure (only the tubules; this is renal tubular acidosis), diarrhea, polyuria, and dehydration. It is unclear whether the high uric acid seen in birds with renal failure influences blood pH and promotes acidemia or whether the severe acidemia frequently seen with hyperuricemic birds is mainly caused by the failure of renal excretion of other organic acids as seen in mammals. Considering that uric acid is a relatively weak acid, that its concentration is measured in umol/L, and that metabolic acidosis primarily caused by hyperuricemia has not been demonstrated in birds and humans with gout, it seems less plausible that uric acid levels may have any substantial effect on avian blood pH except in extreme cases. Fluid therapy usually consists of reestablishing normal volemia and infusing balanced crystalloid solutions such as Plasmalyte A 7.4.

Metabolic alkalosis is characterized by an increase in bicarbonates. In birds, almost all cases are hypochloremic metabolic alkalosis caused by selective loss of chlorides (increased SID) into or from the GI system. Common causes are regurgitation, vomiting, third spacing, and mechanical or functional ileus (including lead-induced ileus). Fluid therapy usually consists of reestablishing normal volemia and infusing acidifying crystalloid solutions with high chloride content such as NaCl 0.9%.

CASE STUDY 1: BLUE AND GOLD MACAW

A blue and gold macaw was presented with severe lethargy, was unable to stand, and had bloody diarrhea and increased respiratory rate. Physical examination revealed severe dehydration. Initial blood gas and electrolyte panel is shown subsequently.

Several abnormalities were detected: low values for blood glucose, sodium, bicarbonates, Pco_2, pH, and hemoglobin. The bird had a moderate to severe acidemia and a marked metabolic acidosis with appropriate respiratory compensation. The AG was 18.6 mEq/L, which is approximately normal to slightly elevated. The chlorides were normal but the SID was 20 mEq/L, which, considering the hyponatremia, points toward a selective loss of sodium relative to chlorides (hyperchloremic metabolic acidosis).

The biochemistry profile revealed a severe hyperuricemia and inversed calcium to phosphorus ratio. The complete blood cell count revealed marked heterophilic leukocytosis with degenerate left shift.

The acidemia was likely a result of mixed acidotic disorders caused by renal failure, hyperuricemia, hyperphosphatemia, and the loss of sodium through diarrhea. The low blood glucose and marked leukocytosis with degenerative left shift were suggestive of sepsis, which was later confirmed on the blood culture. The initial treatment included balanced crystalloid fluids (Plasmalyte A 7.4) and IV antibiotics. The tachypnea was likely caused by respiratory compensation for the acidemia. The anemia was likely caused by the blood loss through intestinal bleeding. The bird was later diagnosed with a nonobstructive intestinal neoplasia with subsequent septicemia.

Analytes	Values	Analytes	Values
Glucose (mmol/L)	9.3	Base excess	–20.4
Na^+ (mmol/L)	126	Hco_3^- (mmol/L)	6.8
K^+ (mmol/L)	5.3	So_2 (%)	49.5
Cl^- (mmol/L)	106	Hb (g/dL)	8.5
pH	7.15	Lactates (mmol/L)	1.4
Pco_2 (mm Hg)	20.4	Ca^{2+} (mmol/L)	1.06
Po_2 (mm Hg)	62.1		

CASE STUDY 2: YELLOW-NAPPED AMAZON PARROT

A yellow-napped Amazon parrot was presented with lethargy, regurgitation, and lack of fecal production. Initial blood gas and electrolyte panel is shown subsequently.

Several abnormalities were detected: low chlorides, high pH, high Pco_2, high bicarbonates, high base excess, and low hemoglobin. The bird had a moderate alkalemia and a marked metabolic alkalosis with respiratory compensation. The AG was 15. The SID was 55 and indicated a loss of chlorides in excess of the sodium, which suggested a hypochloremic metabolic alkalosis.

Radiographs revealed massive distention of the intestines, which, along with the acid-base findings and the clinical signs, suggested GI obstruction. The bird was treated with NaCl 0.9% IV and an exploratory coeliotomy diagnosed an obstructive mass in the colon. The anemia was likely caused by GI bleeding.

Analytes	Values	Analytes	Values
Glucose (mmol/L)	12.7	HCO_3^- (mmol/L)	42.6
Na^+ (mmol/L)	145	Base excess	17.8
K^+ (mmol/L)	2.6	So_2 (%)	54.8
Cl^- (mmol/L)	90	Hb (g/dL)	9.1
pH	7.55	Lactates (mmol/L)	2.6
Pco_2 (mm Hg)	49.4	Ca^{2+} (mmol/L)	1.15
Po_2 (mm Hg)	42.1		

Respiratory Acid-Base Disorders

Respiratory disorders are frequently seen as a result of stress and manual restraint in birds. The stress of restraint and sampling may lead to respiratory acidosis from the inhibition of breathing (Harms and Harms, 2012). Conversely, restraint may cause respiratory alkalosis from stress-induced hyperventilation (Heatley *et al.*, 2015). Improper ventilation under anesthesia may also lead to hypercapnia or hypocapnia.

Diseases that may cause respiratory acid-base disorders in birds are similar to those of mammals and commonly include congestive heart failure, anemia, chronic obstructive respiratory disease (e.g., in macaws), and primary respiratory diseases such as pneumonia and airborne toxins (e.g., polytetrafluoroethylene [PTFE or Teflon] or smoke inhalation injury).

REFERENCES

DiBartola SP: Metabolic acid-base disorders. In DiBartola SP, editor: *Fluid, electrolyte, and acid-base disorders in small animal practice*, ed 4, St. Louis, 2012, Elsevier, pp 253–286.

DiBartola SP, Autran de Morais H, Constable PD: Strong ion approach to acid-base disorders. In DiBartola SP, editor: *Fluid, electrolyte, and acid-base disorders in small animal practice*, ed 4, St. Louis, 2012, Elsevier, pp 316–329.

Harms CA, Harms RV: Venous blood gas and lactate values of mourning doves (*Zenaida macroura*), boat-tailed grackles (*Quiscalus major*), and house sparrows (*Passer domesticus*) after capture by mist net, banding, and venipuncture, *J Zoo Wildl Med* 43(1):77–84, 2012.

Heatley JJ, Cary J, Kingsley L, et al: Midazolam sedates passeriformes for field sampling but affects multiple venous blood analytes, *J Vet Med Res Rep* 6:1–9, 2015.

Johnson RA, Autran de Morais H: Respiratory acid-base disorders. In DiBartola SP, editor: *Fluid, electrolyte, and acid-base disorders in small animal practice*, ed 4, St. Louis, 2012, Elsevier, pp 287–301.

Martin MP, Wineland M, Barnes HJ: Selected blood chemistry and gas reference ranges for broiler breeders using the I-STAT® handheld clinical analyzer, *Avian Dis* 54(3):1016–1020, 2010.

Montesinos A, Ardiaca M: Acid-base status in the avian patient using a portable point-of-care analyzer, *Vet Clin North Am Exot Anim Pract* 16(1):47–69, 2013.

Paula VV, Fantoni DT, Otsuki DA, Auler JOC Jr: Blood-gas and electrolyte values for Amazon parrots *(Amazona aestiva)*, *Pesquisa Veterinária Brasileira* 28(2):108–112, 2008.

Powell FL: Respiration. In Scanes CG, editor: *Sturkie's avian physiology*, ed 6, San Diego, 2015, Academic Press, pp 301–336.

Rettenmund CL, Heatley JJ, Russell KE: Comparison of two analyzers to determine selected venous blood analytes of quaker parrots (*Myiopsitta monachus*), *J Zoo Wildl Med* 45(2):256–262, 2014.

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.

Stämpfli H, Taylor M, McNicoll C, et al: Experimental determination of net protein charge, [A]tot, and Ka of nonvolatile buffers in bird plasma, *J Appl Physiol* 100(6):1831–1836, 2006.

Steinmetz HW, Vogt R, Kästner S, et al: Evaluation of the I-STAT portable clinical analyzer in chickens (*Gallus gallus*), *J Vet Diagn Invest* 19(4):382–388, 2007.

Zehnder AM, Hawkins MG, Pascoe PJ: Avian anatomy and physiology. In West G, Heard D, Caulkett N, editors: *Zoo animal & wildlife immobilization and anesthesia*, ed 2, Ames, 2014, Wiley-Blackwell, pp 391–398.

ASPIRATES

Morena Bernadette Wernick

In birds the coelomic cavity is subdivided into five distinct peritoneal cavities: right and left dorsal hepatic (RDHPC, LDHPC), right and left ventral hepatic (RVHPC, LVHPC), and the intestinal peritoneal cavity (IPC). Pleural and pericardial reflections create three additional coelomic cavities: the right and left pleural cavities (RCP, LCP) and the pericardial cavity (PC). In any bird, both pleura and peritoneum produce only a very small amount of fluid to facilitate organ movement (Campbell, 2007). Normally this amount of fluid is too small to be collected. However, in the case of disease, fluids may accumulate, especially in one or more peritoneal cavities, causing severe coelomic distention. Large amounts of fluids can then compress the bird's complex air sac system, consequently resulting in severe dyspnea. This may happen because of inflammatory conditions, disruption of vessels, or neoplastic diseases (Campbell, 2007).

Transudates with low specific gravity (< 1.017), low cell counts (< 1000 cells/µL), and low protein concentrations (< 2.5 g/dL) often occur in birds with chronic liver disease because of portal hypertension or in the case of cardiac disease. By contrast, exudates with high specific gravity (> 1.025), high cell counts (> 5000 cells/µL), and high protein contents (> 3 g/dL) often develop because of inflammatory conditions (e.g., egg coelomitis). In any case, aspirates can yield valuable information that may assist in diagnosis and improve the patient's clinical condition (Fig. 6-83).

Aspirates should not be taken until every patient has undergone a thorough clinical examination. Radiography and ultrasound are helpful to evaluate the patient's general condition. To minimize the risk of further damage to the bird, sedation or anesthesia may be necessary to perform aspiration in anxious, stable patients. The equipment for aspiration of fluids consists essentially of a syringe and needle or butterfly needle. Care must be taken not to rapidly remove a large amount of fluid from a lesion because this may have severe systemic effects. Most often, only a small amount of fluid is aspirated

FIGURE 6-83 Skin growth on the leg of an orange-winged Amazon parrot (*Amazona amazonica*). Fine-needle aspiration revealed unstained rods positive to Ziehl-Neelsen stain. A positive PCR for *Mycobacterium avium* was obtained from a biopsy of the lesion. (Courtesy Andrés Montesinos.)

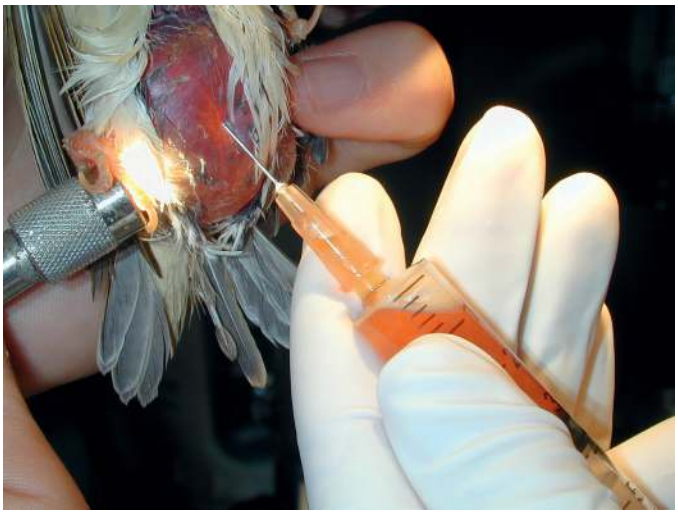


FIGURE 6-84 Canary (*Serinus canaria*) presented with a severe case of ascites. A large volume of fluid was aspirated through the abdominal wall using a wide-gauge needle and disposable syringe. Please note the blunt-tipped instrument pressing against the abdomen to ease the aspiration procedure. (Courtesy Andrés Montesinos.)

for complete cytology and/or microbiology test. The size of the needle and syringe is dependent on the patient's size but also varies upon individual preference. As a general rule, the needle should be as short as possible because longer needles may damage other tissues, but must be compatible with obtaining the sample. In birds, 21- to 25-gauge needles are appropriate for aspiration of samples. Very narrow gauge needles should not be used because there is the danger of cell lysis and the appearance of artifacts, as known from hematology. Additionally, semisolid fluids or fluids containing solid material may not be aspirated with such needles.

Before sampling, surgical preparation of any aspiration site is indispensable. To obtain a sample from the coelomic cavity in avian patients, a needle is inserted immediately distal to the sternum (Campbell, 2007). As the ventriculus lies on the left ventral side just caudal to the sternum, the needle should point to the right side of the coelomic cavity to avoid any puncture. After insertion of the needle for a few millimeters and application of pressure by drawing back the plunger on the syringe, fluid can be carefully aspirated (Fig. 6-84). Changes in direction may be necessary until fluid can be aspirated. After aspiration, every patient must be carefully monitored, in particular for signs of dyspnea or signs of leakage through the needle hole. Prophylactic treatment with antibiotics and analgesics is most often mandatory.

Apart from the coelomic cavity, other sample sites for aspirates in birds include swollen joints (collection of synovial fluid) or fluid-filled sinuses in the case of sinusitis (left or right infraorbital sinus).

To obtain synovial fluid, flexion of the affected joint is necessary to be able to insert the needle through the joint capsule into the joint cavity (Campbell, 2007). As blood contamination commonly occurs during this procedure, samples should be placed into ethylenediaminetetraacetic acid (EDTA) tubes to prevent clotting.

Aspiration of the left or right infraorbital sinus is best performed by inserting a needle parallel to the skin at the commissure of the beak, directed vertically under the jugal bone, midway between eye and external nares (Campbell, 2007). Care must be taken not to penetrate the ocular orbit. As an alternative, the paraorbital sinus just below the eye may be used for sampling (Campbell, 2007).

When aspirates have been obtained into the needle, smears for cytology or cultures for microbiology can be created. Of course, it is also possible to perform other tests if samples are sufficiently large. Even if aspiration appears to be unsuccessful, this is not necessarily the case. The tip of the needle or the terminal few millimeters may both contain material from the lesion. For this reason, the needle should nevertheless be used for laboratory tests, even though no fluid could be sampled. If bacteriologic culture is needed, the tip of the needle can be used to plate out directly on to blood agar or other media. It is also possible to remove the needle and to flush through a small volume (0.1 mL maximum) of sterile saline to wash out the tip of the needle and to flush out any material that may be present.

REFERENCE

Campbell TW: Comparative Cytology. In Campbell TW, Ellis CK, editors: *Avian and exotic animal hematology and cytology*, ed 3, Ames, 2007, Blackwell Publishing, pp 139–221.

BIOPSIES

Morena Bernadette Wernick

The term biopsy refers to a procedure that helps obtain tissue for microscopic examination or other diagnostic tests, with the goal to establish a precise diagnosis for the patient and to enhance understanding of the development of specific diseases. Taking biopsies is a common practice in small animal veterinary medicine and has become an increasingly important part of avian diagnostic medicine. A variety of organs and tissues of birds can be biopsied (Fig. 6-85). Biopsy collection includes not only surgical excision or removal of tissue (e.g., punch biopsy, incisional biopsy, excisional biopsy), but also fine-needle aspiration (aspiration biopsy, needle core biopsy), brushing, washing, scraping or taking impression smears (touch imprints). The choice of technique depends highly on the anatomic location of the lesion, the analysis to be performed, the bird's general condition (whether suitable for anesthesia), and the clinician's own preference. In general, biopsy techniques can be divided into two major groups: (1) pretreatment

biopsies and (2) excisional biopsies. Pretreatment biopsies help obtain information about specific lesions before definitive treatment, whereas excisional biopsies enable histopathologic diagnosis after surgical removal of lesions (e.g., removal of suspected neoplasia). In the latter case, biopsies also offer the possibility to evaluate completeness of excision (Ehrhart and Withrow, 2007). A list of organs and tissues of birds suitable for biopsy is given in Table 6-15.

Interpretation of removed tissues is highly dependent on both the quality and quantity of submitted samples. The site of biopsy often requires careful thought and planning. Frequently, the assistance of other techniques such as radiography or ultrasound is needed to ascertain the optimum location for sampling. Most often, the border between normal and abnormal tissue is best for histopathology to examine the difference between both tissues and to determine the degree of invasiveness in case of neoplasia. Care must then be taken not to incise normal tissue that cannot be resected or is needed for reconstruction of the surgical defect, as a spill of cancerous cells may occur (Ehrhart and Withrow, 2007). Biopsies need to be sufficiently deep to be able to provide relevant information. Because some lesions may be inhomogeneous and may contain different areas of necrosis and inflammation, several samples can help improve the likelihood of obtaining an accurate diagnosis compared with examination of one single sample. Samples that contain irrelevant material such as blood clots and debris should be avoided. Because electrocautery and surgical lasers are commonly used in avian medicine but both deform cellular architecture, biopsies should only be obtained by blade removal (Ehrhart and Withrow, 2007).

FIGURE 6-85 Bone marrow aspiration from the tibiotarsus in an Amazon (*Amazona* sp.) parrot. (Courtesy Hugues Beaufrère.)

Handling of samples before fixation and processing must be carried out very carefully. Sharp or squeezing instruments such as scissors and forceps may damage or destroy specimens when used inappropriately. Particular care must be taken if the biopsy material is needed fresh, for example for microbiology, biochemistry, or clinical tests, as well as for fixing for light or electron microscopy. In such circumstances, multiple biopsies should be taken and each biopsy should be placed in a separate container. It is beneficial to prepare impression smears (touch imprints) of all samples before fixation. Imprints should be obtained from freshly cut surfaces that are relatively dry and free of blood and several imprints should be made on each slide (Campbell, 2007).

All biopsy requests should always be accompanied by a thorough anamnesis about the patient and by all relevant information concerning the sampled lesion (location, thickness, consistency, adherence to the skin or surrounding tissues) because this will be of tremendous

TABLE 6-15 Biopsy Sites and Techniques in Birds

Organ/Tissue	Technique(s)	Comments
Skin, including feather follicles	Surgical excision or skin biopsy punch Needle biopsy; scraping; plucking of feathers may provide small numbers of cells Exfoliative	Avoid aggressive preoperative disinfection, which may affect biopsy Postoperative treatment of biopsy wound may be necessary Particularly useful if lesion is ulcerated
Muscle and fat (external)	Surgical excision; needle biopsy	Bleeding often marked, but rarely of consequence
Oral cavity and cloaca	Surgical excision; exfoliative	Some lesions (e.g., cloacal papillomas) may bleed heavily Electrosurgical excision or cryosurgery will minimize hemorrhage, but may damage cell architecture of the biopsy specimen
Upper gastrointestinal tract	Endoscopy; biopsy forceps; exfoliative	Crop biopsies should be taken in the left lateral area of the crop
Lower intestinal tract	Endoscopy; biopsy forceps; exfoliative	
Kidney	Endoscopy; biopsy forceps	
Female reproductive tract (oviduct)	Endoscopy; biopsy forceps; exfoliative	
Male reproductive tract (testes)	Endoscopy; biopsy forceps; exfoliative	
Respiratory tract (lungs)	Endoscopy; biopsy forceps	
Respiratory tract (air sacs)	Endoscopy; biopsy forceps Needle biopsy; surgical excision; exfoliative	
Liver	Endoscopy; biopsy forceps; needle biopsy; wedge biopsy sample	Sampling by endoscopy, ultrasound-guided fine-needle aspiration, or directly by incision through skin and abdominal musculature caudal to sternum, lateral to midline
Bone	Needle biopsy; bone punch (trephine); surgical excision	Bone punches are expensive
Bone marrow	Needle biopsy	

TABLE 6-16 Handling and Processing of Biopsies from Birds

Type of Biopsy	Procedure	Comments
Surgical excision (total) or incision (partial removal)	Touch imprints from freshly cut surface on glass slides, then: 1) half in 10% buffered formal saline (BFS) or other fixative for histopathology and/or electron microscopy 2) half kept fresh for microbiology and other procedures	Touch imprints should be relatively dry and free of blood Choice of fixative will depend on techniques to be followed Heavily keratinized material (e.g., tarsometatarsal skin) may need to be softened before histologic processing
Skin biopsy punch	Follow surgical excision or incision procedure	See surgical excision or incision comments Can be used for horny structures (e.g., hornbill beaks) after drilling. Shorter and wider biopsies compared with fine-needle aspiration biopsies. Sutures may be placed if intact skin was opened
Endoscopic biopsy Biopsy forceps	Sample lifted out from forceps cup using a 23-gauge needle and placed on lens tissue moistened with sterile saline Selected biopsies can then either be submitted fresh for microbiology or (still wrapped in lens tissue) fixed in 10% BFS or other fixative	Possible to gain biopsies from respiratory, gastrointestinal, and urogenital systems Small, easily damaged and lost samples Should be counted, dealt with rapidly, kept moist, and not handled unnecessarily
Fine-needle aspiration biopsy	**Aspiration technique:** Use of hypodermic needle (22-gauge, 1-inch, range of 25-20 gauge acceptable), syringe (3 mL or larger), and glass microscope slides Preparation of the skin using alcohol Stabilization of the lesion and insertion of the needle with attached syringe Retraction of syringe plunger to provide 0.5-1 cc of vacuum Needle is advanced and retracted at different angles Deposition of aspirated sample onto glass microscope slide (Campbell, 2007) **Nonaspiration technique:** Preparation of tissue as previously described Insertion of a needle into the lesion without the use of an attached syringe After sampling, attachment of a syringe to expel the sample from the needle lumen onto a glass slide (Campbell, 2007)	Cost-effective procedure Can be performed on different tissues or internal masses Minimal trauma to the patient Method can be repeated if necessary Sample is small, friable and easily lost Reduction of blood contamination in highly vascularized tissues
Bone biopsy punch	See fine-needle aspiration biopsy punch procedure	Decalcification is usually necessary.
Bone marrow aspiration	Use of pediatric bone marrow biopsy needles or spinal needles containing a stylet	Commonly used sites include the medial or cranial aspect of the proximal tibiotarsus and the sternum (Galliformes)

help for the pathologist to provide accurate and clinically relevant information.

A suggested approach for handling and processing of biopsies from birds is given in Table 6-16.

Several problems may occur when taking biopsies in avian patients. This includes extensive bleeding after sampling, chronic changes after tissue damage (e.g., adhesions), infections (e.g., internal granulomas, abscesses), spill of neoplastic cells, or the perforation of organs and tissues. Therefore a thorough examination and observation of the patient before and after sampling is crucial.

REFERENCES

Campbell TW: Comparative cytology. In Campbell TW, Ellis CK, editors: *Avian and exotic animal hematology and cytology*, ed 3, Ames, 2007, Blackwell Publishing, pp 139–221.

Ehrhart NP, Withrow SJ: Biopsy Principles. In Withrow SJ, Vail DM, editors: *Withrow and MacEwen's small animal clinical oncology*, ed 4, St. Louis, 2007, Saunders Elsevier, pp 147–153.

TRACHEAL WASH AND AIR SAC FLUSHING

Morena Bernadette Wernick

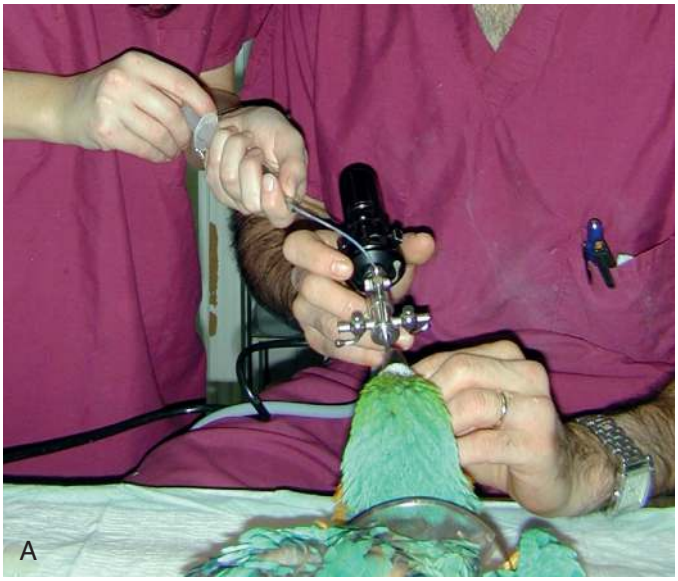
The avian trachea stretches from the glottis to the syrinx, which is located at or near the junction of the trachea and bronchi (Campbell, 2007). It has complete, calcified rings and is lined by a ciliated, pseudostratified columnar epithelium containing secreting goblet cells. The trachea of some birds, such as toucans, deviates ventrally cranial to the thoracic inlet.

Tracheal wash samples are indicated in birds with suspected respiratory disease and may be of paramount importance in the diagnosis of etiologic agents in respiratory tract infection. Flushing most often requires general anesthesia or at least light sedation for the patient's safety. Rarely, flushing can be performed in well-restrained, nonanesthetized birds. Flushing under light sedation or in a conscious patient offers the advantage that the bird is still able to cough and therefore able to clear the airways of remaining fluid (Campbell, 2007) (Fig. 6-86, *A*).

For flushing, a plastic or rubber catheter/tube (small enough to pass down the trachea) is inserted via the open glottis into the trachea to the level of the syrinx. A small amount of sterile saline (1-2 mL/kg) is

quickly infused into the area and immediately reaspirated into the tube. If the bird is awake, care must be taken to utilize an oral speculum to keep parts of the collection tube from being bitten off (Campbell, 2007). In anesthetized larger birds, the catheter or tube can be passed through an inserted sterile endotracheal tube or endoscope working channel. To prevent contamination in the oral cavity, the tip of the collection tube should not touch any parts of the oral mucosa. In some species, such as penguins, the trachea bifurcates at a short distance in the cervical region. Therefore tracheal flushing may result in the sampling of only one side (Campbell, 2007).

Similar techniques can be employed to collect samples of the lower respiratory tract. Because most species of birds have a unique respiratory system with nine air sacs, endoscopy allows thorough examination of the whole respiratory system and the GI and genital tract. Endoscopic air sac flushing can easily be performed on an anesthetized bird in right or left lateral recumbency. The choice and preparation of the endoscopy site correlates to already described methods (see Chapter 6, Endoscopy). As mentioned earlier, sterile saline can be infused through a sterile tube, placed into the biopsy channel of an endoscope, and infused into the air sacs. Care must be taken to elevate the bird's head and cranial aspect of the body after infusion to prevent saline from entering the lungs (Campbell, 2007). Aspirated fluid is then used for cytology or bacteriologic examination (Fig. 6-86, *B*).



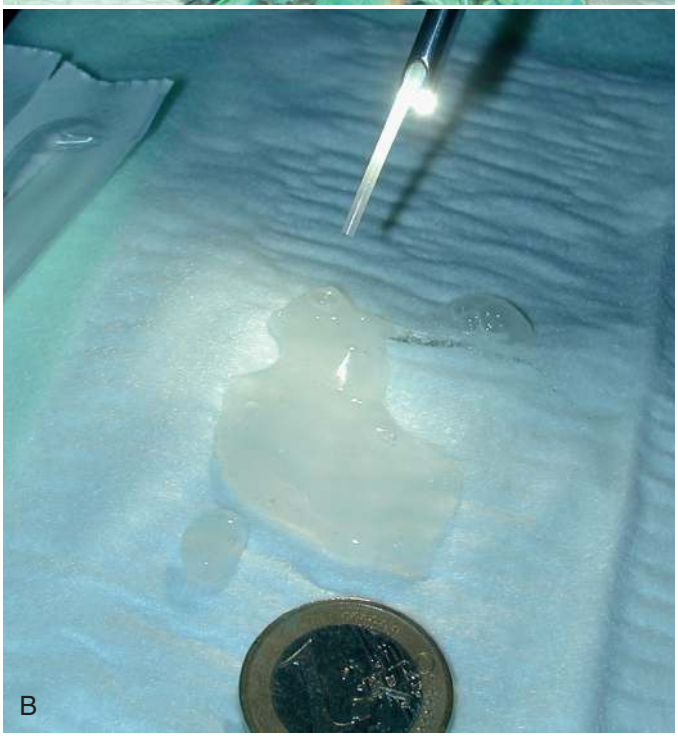


FIGURE 6-86 (A), Performing a tracheal wash in a blue and gold macaw (*Ara ararauna*). **(B),** Aspirated fluid can be used for cytology or bacteriologic examination. (Courtesy Andrés Montesinos.)

REFERENCE

Campbell TW: Comparative Cytology. In Campbell TW, Ellis CK, editors: *Avian and exotic animal hematology and cytology*, ed 3, Ames, 2007, Blackwell Publishing, pp 139–221.

CROP FLUSHING

Morena Bernadette Wernick

Examination of the oral cavity and the crop should be included in every routine physical examination of any bird. Crop aspirates are commonly indicated, especially in patients with a history of regurgitation, vomiting, or in the case of any crop abnormalities (e.g., delayed crop emptying, crop dilation). Wet mounts help identify motile *Trichomonas* sp. (*Trichomonas gallinae*) or other protozoa, yeast (both budding and with pseudohyphae), or bacteria, which may otherwise be difficult to diagnose.

Crop aspirates can be obtained by carefully inserting a sterile, round-ended plastic or rubber tube or a stainless steel gavage tube through the oral cavity and esophagus into the crop of the conscious bird. Because there is the risk to damage or even puncture the thin esophageal wall, the bird's head and neck should be extended during the procedure (Campbell, 2007). To ensure proper tube placement, the tube should be palpated during its passage through the esophagus and then be fixed with one hand on the bird's head to avoid uncontrolled movements. The crop content can then be gently aspirated into a sterile syringe attached to the free end of the tube. Care should always be taken not to create too much negative pressure on the crop mucosa because this can cause extensive damage (e.g., ischemic lesions) (Campbell, 2007). A crop wash can be achieved, flushing the crop with a small amount (5-10 mL/kg body weight) (Campbell, 2007) of sterile 0.9% saline at room temperature and immediately aspirating the fluid for cytologic evaluation using the previously mentioned technique (Fig. 6-87).

FIGURE 6-87 Crop flushing in a cockatiel (*Nymphicus hollandicus*). A metal gavage tube is carefully inserted through the oral cavity and the esophagus into the crop. A crop wash is achieved by flushing the crop with a small amount of sterile saline.

REFERENCE

Campbell TW: Comparative Cytology. In Campbell TW, Ellis CK, editors: *Avian and exotic animal hematology and cytology*, ed 3, Ames, 2007, Blackwell Publishing, pp 139–221.

SKIN AND FEATHER EXAMINATION

Rolf K. Schuster

The more commonly found arthropod ectoparasites affecting the skin and feathers of birds are mites, but lice, fleas, ticks, and flies may also be seen. The symptoms can include feather damage and loss, skin irritation, and pruritus. Some of the more common ectoparasites are detailed in Table 6-17.

The integument of avian species is generally much thinner and more delicate than that of mammals. It is attached to muscles in only a few places but has extensive attachments to the skeleton (e.g., feet and skull). As with mammals it consists of three layers: the epidermis, the dermis (containing connective tissue, blood vessels, nerve endings, feather follicles, and feather-erecting muscles), and the subcutis (containing fat). The subcutis and dermis do not contain much elastic fiber and therefore are not very elastic and tear easily. Skin scrapings are carried out to determine whether fungal or parasitic (mite) infections of the superficial skin layers are present (see the section on Arthropods in Parasites in Chapter 14). These samples should be taken from a suspected area, although severely traumatized areas of skin should be avoided.

OBTAINING A SKIN SCRAPING

Superficial Skin Scrapings

- Moisten the skin with cotton wool soaked in mineral oil.
- Tense the skin between a finger and thumb.
- The skin can then be gently scraped with a dull scalpel blade; include scrapings from the edge of a lesion.

Deeper Scrapings (Some Mites Dwell in the Subcutis)

- Moisten the area to be scraped with a little 10% potassium hydroxide (KOH).
- Tense the skin between a finger and thumb.
- Gently (remembering that avian skin is quite delicate) scrape the lesion until pinpoints of blood appear.

In both cases transfer the material collected onto a glass slide and cover with a cover slip (or put into a suitable container). Too much material on the slide will make identification more difficult. Gentle warming helps the KOH to clear the keratin and debris so that a systematic search can be made for parasites and fungal spores. The KOH helps "clear" the parasite, making the features more identifiable. The slide can be examined under the microscope on low power.

EXAMINATION OF FEATHERS

Identification of ectoparasites can prove difficult because detailed clinical examination can fail to confirm the presence of ectoparasites. It may be necessary to take feather and/or feather stub samples to investigate whether arthropods such as mites are in residence. Biting lice usually can be seen with the naked eye. The presence of mites can be established by examining feathers under the stereoscopic microscope. A treatment of feathers and stubs with keratolytic substances dissolves keratin but does not affect the chitin of arthropods.

Place feathers or feather stubs for maceration in sodium hydroxide (10% aquatic solution) in a V-bottomed container such as a 30-mL universal container; keep at 37° C (98.6° F) overnight. Microscopic examination of the sediment should reveal ectoparasites or body parts against nondescript material. When feathers and stubs are placed in a sealed plastic envelope for at least 24 hours, the parasites tend to migrate from the feathers and become caught in the folds of the

TABLE 6-17 Ectoparasites in Birds

Parasite	Symptoms	Identification
Soft ticks (*Argas* spp.)	Loss of condition, anemia	Nocturnal parasites; can be found in cracks and cleaves in poultry houses
Hard ticks (Ixodidae spp.)	Usually no symptoms	Birds usually infested by tick larvae on the body, around eyes and beak
Red mite (*Dermanyssus gallinae*)	Restlessness, loss of condition, anemia	Nocturnal parasites; can be found in cracks and cleaves in poultry houses
Poultry mites (*Ornithonyssus* spp.)	Loss of condition, anemia	Mites usually can be found on the host
Quill mites (Syringophilidae spp.)	Feather damage, usually apathogenic	Invade the calamus of feathers, difficult to see
Chigger or harvest mites (Trombiculidae spp.)	Blistering can occur around the point of attachment	Orange larvae between feathers on belly and legs
Feather mites (Analgoidea spp.)	Usually apathogenic, feather damage	Can be seen on the surface of feathers, feather quills, or on skin
Burrowing mites (Knemidocoptidae spp.)	Skin irritation, honey comb–like lesions, scaly leg, scaly face	Skin scrapings, maceration in 10% KOH solution
Fowl cyst mite (*Laminosioptes cysticola*)	Cyst formation in subcutaneous tissues	Biopsy of cysts
Bugs (*Cimex* spp.)	Restlessness, anemia	Nocturnal parasites; can be found in cracks and cleaves in poultry houses
Biting lice (Mallophaga spp.)	Restlessness, pruritus	On feathers and on skin; nits are on feather barb or base of quill
Mosquitoes, midges, black flies (*Nematocera* spp.)	Pruritus, restlessness, toxicosis (vector role!)	
Fly larvae (Sarcophagidae spp., Calliphoridae spp., Neottiophilidae spp.)	Myiasis	Fly larvae in skin and subcutaneous tissues
Louse flies (Hippoboscidae spp.)	Pruritus, restlessness, toxicosis (vector role!)	In plumage on belly and neck
Fleas (Ceratopyllus spp., *Echidnophaga*)	Pruritus, restlessness	In bird nests and on the body, females stick tight around eyes and beaks

KOH, Potassium hydroxide.

envelope. The ectoparasites can be examined through the plastic under low power on the microscope. Feather stubs should also be examined because some species of mite live in the shaft of the feather. For quill mites, the shaft can be split lengthways and placed in 70% ethanol and examined as previously explained. For further details on examination of the skin and feather follicles, see details in the section on Biopsies in this chapter.

ACKNOWLEDGEMENTS

The author would like to acknowledge Judith Howlett for her contribution to this section in a previous edition of this book.

SWABS

Christudas Silvanose

Swabs are pieces of absorbent material commonly attached to long wooden or metal sticks and are used in medicine to collect microbiology samples for either a culture or for the preparation of smears for cytology (Figs. 6-88 and 6-89). Other techniques can also be used (e.g., washings, brushings, and scrapings). This was discussed under the section on biopsies previously.

Swabs can be taken for a variety of purposes including bacteriology, mycology, virology, mycoplasmology, and cytology tests.

FIGURE 6-88 A cloacal swab being collected from a saker falcon (*Falco cherrug*) under isoflurane anesthesia.

There are two important considerations when collecting a swab:

- The type of swab used
- The area that is swabbed

The type of swab can greatly influence the results and thus the action to be taken. There are many types of swabs, and although the majority consist essentially of a wooden or metal (e.g., aluminum) stick and a cotton-based tip, there are many variants. The swabs most likely to be used in avian practice are:

- Dry cotton wool swabs
- Cotton wool swabs in transport medium (e.g., Stuart's transport medium)
- Alginate-coated swabs
- Any of the previously mentioned items designed specifically for pediatric use or for sampling narrow orifices (e.g., nasopharyngeal swabs)

Each of these tools has some advantages. Under most circumstances, dry cotton wool swabs are satisfactory, particularly if the sample is being taken from an extensive lesion (see later) and is likely to be plated or processed rapidly in-house. Sometimes the efficacy of such dry swabs, in terms of picking up organisms and the latter surviving, is enhanced if the dry swab is first immersed in sterile saline. Swabs in transport medium are to be preferred when samples are not to be examined immediately and particularly when proper storage may be difficult (e.g., in field work or in countries where refrigeration and other facilities are not available). Again, there are many types of transport medium, each with its own particular features, but in general it can be assumed that Stuart's transport medium will prove satisfactory for the storage of many types of bacteria for substantial periods of time. Transport media are also available for more specific purposes, such as transportation of suspect viruses or mycoplasmas. Even if a transport medium is used, every care must be taken to store and transport samples carefully. They should be handled gently (not dropped or shaken) and in general are best kept at normal refrigerator temperature (4° C [39.2° F]) until processing can be carried out.

COLLECTION OF MICROBIOLOGICAL SWABS

For consistent laboratory results it is important to avoid errors, including:

- Sampling from the incorrect site
- Nondiagnostic sample
- Contamination of the swab from environmental factors (e.g., accidentally touching the swab)

FIGURE 6-89 Oropharyngeal swabs can be collected relatively easily from a live buff-crested bustard (*Eupodotis ruficrista*).

TABLE 6-18 Antibiotic Sensitivity Tests*

Antibiotics	Concentration	Isolates
Amoxicillin	25 µg	All pathogenic gram-negative and gram-positive bacteria
Amoxicillin–clavulanic acid	30 µg	All pathogenic gram-negative and gram-positive bacteria
Ampicillin	10 µg	Gram-positive pathogenic bacteria
Ampicillin	30 µg	Gram-negative pathogenic bacteria
Carbenicillin	100 µg	*Pseudomonas* spp. and other gram-negative pathogens
Chloramphenicol	10 µg	Gram-negative pathogenic bacteria
Chloramphenicol	30 µg	Gram-negative pathogenic bacteria
Enrofloxacin	25 µg	All pathogenic gram-negative and gram-positive bacteria
Erythromycin	15 µg	All pathogenic gram-positive bacteria
Gentamicin	10 µg	All pathogenic gram-negative and gram-positive bacteria
Piperacillin	100 µg	*Pseudomonas* spp. and other gram-negative pathogens
Penicillin-G	1 unit	Gram-positive pathogenic bacteria
Penicillin-G	2 units	Gram-negative pathogenic bacteria
Sulfamethoxazole	25 µg	All pathogenic gram-negative and gram-positive bacteria
Sulfonamide	300 µg	*Pseudomonas* spp. and other gram-negative pathogens
Tetracycline	30 µg	All pathogenic gram-negative and gram-positive bacteria
Ticarcillin	75 µg	*Pseudomonas* spp. and other gram-negative pathogens

*Antibiotic sensitivity tests include media-nutrient agar, Mueller-Hinton agar, blood agar, Kirbey-Bauer disc diffusion, metheselene technique, and broth dilution technique.

- Sampling during antibiotic therapy
- Using inappropriate materials, including the wrong transport medium
- Contact with inhibitory chemicals (i.e., disinfectants)

Antibiotic sensitivity tests are set out in Table 6-18.

The different recommended protocols for the collection, transportation, and processing of samples are outlined in Table 6-19.

UPPER RESPIRATORY TRACT

Sampling is recommended if a bird is presenting any of the following signs:

- Pharyngitis
- Coughing
- Sneezing
- Oral odor

Swab any obvious oral lesions, but otherwise swab the choanal slit.

TABLE 6-19 Some Staining Techniques in Avian Cytology

Stain	Use	Comments
Romanowsky stains (e.g., Giemsa, Wright, Wright-Giemsa, May-Gründwald-Giemsa)	All cell types including blood; will also stain organisms such as hemoparasites and *Chlamydia*	Best to air-dry; variable results depending on type of stain and skill of technician Stained preparations retain color if kept in dark
Commercial quick stains (e.g., "Dif-Quik," "Rapidiff," "Hemacolor," "Aviacolor")	Most cell types but especially blood and bone marrow	No fixation needed. Rapid—can be examined within a few minutes Stained preparations tend to fade
Gram	Bacteria Myelin	Standard procedure
Ziehl-Neelsen (Z-N)	*Mycobacterium* spp. Other acid-fast organisms (e.g., *Cryptosporidium)*	Modified Z-N (Macchiavello) will detect *Chlamydia* and some mycoplasmas
Sudan III or oil-red-O	Detection of fat (lipid)	Useful because in histologic sections the fat has been removed and cannot, therefore, be demonstrated directly
New methylene blue	Fungal hyphae Fibrin Certain bacteria	Can combine with other stains (e.g., eosin)

Method

The bill or beak may be opened manually with the fingers or by using gauze bandages on the upper and lower beak. In some birds with a strong bite, such as psittacines, an oral metal speculum can be used, or a towel acts as a soft gag.

Nasal discharges are not necessarily good samples even if the discharge is associated with upper respiratory tract infection. (Microscopic examination may show the discharge to be full of bacteria, which can be a primary or secondary infection.) Ocular or conjunctival swabs are not always of clinical value. Ocular signs in upper respiratory tract infections are often caused by *Chlamydia* or *Mycoplasma* spp.; therefore, sample the choana.

A noninvasive method to sample sinuses is to instill sterile normal saline into the nares. Allow the saline to permeate through the sinuses; drainage will occur through the choanal slit. Swab this site as described previously.

LOWER GASTROINTESTINAL TRACT

It is best to sample very fresh feces for a culture; a cloacal swab may be collected as an alternative. This may not represent the lower GI tract because the cloaca may be dry and relatively devoid of bacteria. The swab should be wetted with sterile saline or Ringer solution; this can help with the insertion and with the recovery of organisms. With regard to the size of the swab, it may be beneficial to use a small swab, such as those used in ear, nose, and throat (ENT) medicine with diminutive patients.

FURTHER READING

Fudge AM: Avian microbiology. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, 1996, Williams & Wilkins, pp 795–806.

Gaskin JM: Microbiologic techniques in avian medicine. In Jacobson ER, Kollias GV Jr, editors: *Contemporary issues in small animal practice*, New York, 1988, Churchill Livingstone, pp 159–175.

Koneman EW, Allen SD, Janda WM, et al: Diagnosis of infections caused by viruses, *Chlamydophila*, *Rickettsia* and related organisms. In *Color atlas and textbook of diagnostic microbiology*, ed 4, Philadelphia, 1992, JB Lippincott, pp 966–1048.

Koneman EW, Allen SD, Janda WM, et al: Introduction to microbiology part 1: the role of the microbiology laboratory in the diagnosis of infectious diseases. Guidelines to practice and management. In *Color atlas and textbook of diagnostic microbiology*, ed 4, Philadelphia, 1992, JB Lippincott, pp 1–46.

Van Cutsem J, Rochette F: Diagnostic methods. In Van Cutsem J, Rochette F, editors: *Mycoses in domestic animals*, Beerse, 1991, Janssen Research Foundation, pp 23–43.

CYTOLOGY

John E. Cooper, Christudas Silvanose

INTRODUCTION

Cytology is the study of cells and has an important role in avian medicine:

a. In its own right, cytology is a rapid and inexpensive technique that can be used in the clinic and in the field (Campbell, 1993, 1994, 1995; Cooper, 2002, 2009, 2013; Corr *et al.*, 2002; Latimer *et al.*, 1988; Silvanose and Bailey, 2008).
b. Cytology is an important adjunct to other disciplines, especially histopathology but also bacteriology and parasitology (Cooper, 1994; Thrall *et al.*, 2012; Rosenthal *et al.*, 2004; Teachout, 2005).

Cytology can either provide a diagnosis itself or supplement/complement one made using other methods. It has several advantages over histology in addition to its speed and cheapness. For example, some tumors show better detail in cytologic preparations, shrinking artifact is not present, and microorganisms such as protozoa are often easily seen. On the other hand, there are drawbacks to cytology; for instance, there is rarely any identifiable tissue architecture, thus limiting or preventing comment on invasion and determining margins in neoplasia and precluding comment on the precise relationship of inflammation to the lesion.

The key to successful cytologic investigation is accurate sampling and tissue preparation (Pinches, 2005a, 2000b, 2000c). As in hematology (itself a form of cytology), the essential prerequisite is a monolayer (Hawkey and Dennett, 1989). A useful analogy is an egg, where the yolk is the nucleus and the albumen is the cytoplasm. In cytologic preparations, the cells should be like a fried egg—well-spread and thin.

SAMPLING AND PROCESSING

Specimens from birds for cytologic examination can be conveniently divided initially into:

- Fluids, such as serous exudates of peritoneal effusions, which are best taken by syringe/needle and then spread on a slide in a similar way to blood.
- Solids, such as the cut surfaces of a tumour or granuloma, which are best sampled either in situ or after removal (Cooper, 1994) as imprints (touch preparations or impression smears), having first reduced the amount of blood on the cut surface by blotting on filter paper.
- Samples from oropharynx, trachea, nares, and cloaca are collected by sterile swab and rotated/rolled on to a slide.
- Washed samples require cytocentrifugation to increase smear cellularity.

At least two preparations, preferably more, should always be taken even if not all are stained and examined. It is far better to have an excess of preparations than to rely on only one smear and to have misgivings about sending it to a colleague for a second opinion and thus risk its being lost or broken.

Fixation may or may not be necessary or desirable, depending on the staining technique to be used. If in doubt, if the sample is to be processed within 24 hours, it should be air-dried—but that this can cause crenation of cells. Keep slides well away from formalin because it can interfere with staining procedures. Various stains can be employed—see Table 6-19.

Unstained preparations can also be of value. Wet mounts may demonstrate, for example, ciliated host cells or parasites, and an unstained smear may reveal fat (adipocytes) or cholesterol crystals.

Microscopy may reveal five main categories of structures—see Table 6-20.

Important pathologic changes, which for detection may require examination of many fields and involve several cell types, include:

- Acute inflammation
- Chronic inflammation
- Nonmalignant proliferation
- Malignant proliferation (neoplasia)

Inflammatory (acute and chronic) and neoplastic responses can sometimes be confused. Some examples, with means of differentiation, are given in Table 6-21.

The oral cavity is a useful guide to avian health and, even if gross lesions are not visible, should routinely be swabbed. Some frequently observed cytologic changes are listed subsequently in Tables 6-22 to 6-27.

TABLE 6-20 Categories of Structures

Cell/Structure	Comments
Normal host cells	May show an increase in numbers (e.g., lymphoid hyperplasia of the spleen, proliferation of epithelium), or be present in abnormal sites (e.g., heterophilic infiltration of the liver)
Abnormal host cells	May be indicative of pathology but may also be artifacts caused by poor sample collection, transportation, or processing
Pathologic host cells	Pathologic host cells may show discrete individual changes (e.g., degeneration, vacuolation, metaplasia, neoplasia, or be part of a pattern) involving different types of cells. The size of cells may be important (measure with graticule and compare with cells of known size, e.g., erythrocytes). Giant cells and inclusion bodies may be a feature.
Extrinsic cells	These are cells that are not derived from the host (patient) but may be relevant to diagnosis (e.g., parasites, inhaled material, foreign bodies).
Contaminants	Be wary of plant and other contaminants, especially when working in the field when several samples are being collected or processed at the same time—transportation of cells can occur.

TABLE 6-21 Type of Cytologic Response

Response	Cell Type	Significance/Comments
Inflammatory	Heterophils (normal alone)	Acute inflammation
	Heterophils (degenerate)	Infection, usually bacteria
	Mixed heterophils, lymphocytes	Chronic or subacute infection
	Macrophages in abundance (sometimes giant cells)	Fungal, *Mycobacterium,* foreign body reactions
Neoplastic	General features of neoplasia are populations of similar cells with individual differences, including variable nuclear: cytoplasmic ratio, prominent nuclei and nucleoli, sometimes abnormal/multiple nuclei; increase in mitotic index	
	Spindle-shaped cells that exfoliate poorly	Sarcoma
	Round/oval cells, often in patterns	Carcinoma
	Round/oval cells, lymphoblast-like	Lymphoid neoplasm (e.g., leukemia)
	Mixed cells (but with neoplastic features)	Poorly differentiated neoplasm
	Squamous epithelial cells in large numbers but few features of neoplasia	Papilloma (or tissue hyperplasia)

TABLE 6-22 Common Cytologic Findings in Oral Cavity and Intestine

Sample Site	Findings	Possible Diagnosis
Oral cavity	Superficial squamous epithelial cells, small numbers of mixed commensal flora (bacteria) present	Normal cytology
	Excess exfoliation of keratinized squamous cells	Hypovitaminosis A
	Excess exfoliation of keratinized squamous cells with colonization by thin bacterial rods	Contamination with *Pseudomonas* or other environmental organisms
	Excess exfoliation of keratinized squamous cells with budding cells of *Candida* spp. present	Ill-health, low condition, reduced immunity, or prolonged administration of antibiotic treatment
	Budding cells of *Candida* spp., with pseudohyphae present	Candidiasis
	Inflammatory cells with colonization of bacteria. a) Cocci in clusters: *Staphylococcus* sp. b) Chain-forming cocci: *Streptococcus* sp. c) Cocco-bacillary (short) rods: *Pasteurella* sp. d) Thin bacterial rods: *Pseudomonas* sp.	Stomatitis
	Inflammatory cells with flagellate protozoa present	Trichomoniasis (trichomonosis)
	Parabasal cells, basal cells, and inflammatory cells with bacteria present	Stomatitis: erosions or ulcers
	Intermediate and basal squamous cells with Bollinger bodies and Borrel bodies present	Pox
	Capillaria eggs; excessive exfoliation of superficial, intermediate, and basal cells and inflammatory cells	Capillariasis
Intestine	Epithelial cells with mixed normal flora bacteria present	Normal cytology
	Exfoliation of columnar squamous cells with predominance of one type of bacterium	Enteritis
	a) Thick and long rods with often chain formation: *Clostridium* sp.	Clostridiosis
	b) Short rods: *Salmonella* sp.	Salmonellosis
	Large numbers of coccidial oocysts, columnar cells, erythrocytes (RBCs), and inflammatory cells. Excess mucus	Coccidiosis
	Cestode or trematode eggs and RBCs present	Helminthiasis

RBCs, Red blood cells.

- Acute bacterial infection: Predominance of single type of bacterium, > 70% heterophils, < 30% macrophages
- Chronic bacterial infection: Predominance of single type of bacterium, > 70% macrophages, < 30% heterophils
- Chronic active bacterial infection: Predominance of single type of bacterium colonization; mixed population of heterophils and macrophages approximately 1 : 1 ratio

GENERAL POINTS

i. Always first examine the entire slide at low magnification. Search carefully; there may only be a few cells present.

ii. Look at *all* cytologic preparations that are available. Significant findings may be restricted to only one of the slides (usually the last one to be read!).

iii. Avoid trying to interpret (a) areas that are thick and overstained or (b) cells that are damaged by processing.

iv. Always try to quantify cellularity. Remember that some cells (e.g., epithelium) exfoliate more readily than do others (e.g., fibroblasts). The numbers of cells may therefore vary, depending on the type involved.

v. Record all findings, even if, at the time, they appear to be irrelevant.

Although interpretation is based primarily upon clinical/postmortem history coupled with analysis of cytologic findings, it is *vital* to compare

TABLE 6-23 Common Cytologic Findings in Respiratory Tract

Sample Site	Findings	Diagnosis
Trachea	Small numbers of lining columnar squamous cells and small numbers of mixed commensal flora bacteria present	Normal cytology
	Inflammatory cells with colonization of bacteria. a) Cocci in clusters: *Staphylococcus* sp. b) Chain-forming cocci: *Streptococcus* sp. c) Coccobacillary (short) rods: *Pasteurella* sp. or *Bordetella* sp. d) Thin bacterial rods: *Pseudomonas* sp. e) Cytoplasmic inclusions: *Chlamydia/Mycoplasma* spp.	Tracheitis
	Fungal hyphae, spores, giant cells, goblet cells, and mixed inflammatory cells	Aspergillosis
	Cryptosporidia oocysts, mucus, inflammatory cells, and cellular debris	Cryptosporidiosis
	Syngamus sp. eggs, excessive exfoliation of columnar cells and inflammatory cells	Syngamosis
	Serratospiculum sp. eggs and superficial squamous cells	Serratospiculosis
	Exfoliation of tracheal cells, exfoliation of cilia, karyorrhexis.	Viral tracheitis
Lungs/Air sac	Lung imprints: RBCs seen without bacteria. Air sac walls; squamous cells	Normal cytology
	Inflammatory cells with colonization of bacteria. a) Cocci in clusters: *Staphylococcus* sp. b) Chain-forming cocci: *Streptococcus* sp. c) Coccobacillary (short) rods: *Pasteurella* sp. or *Bordetella* sp. d) Thin bacterial rods: *Pseudomonas* sp. e) Cytoplasmic inclusions: *Chlamydia/Mycoplasma* sp.	Air sacculitis
	Mixed population of heterophils and macrophages, with bacterial colonisation in lung samples	Pneumonia
	Fungal hyphae, spores, giant cells, and mixed inflammatory cells present	Aspergillosis
	Cryptosporidial oocysts, mucus, inflammatory cells, and cellular debris present	Cryptosporidiosis
	Serratospiculum sp. eggs	Serratospiculosis
	Air sac lesions showing ghost cells, macrophages, and giant cells (acid-fast bacterial rods in Z-N stain)	Mycobacteriosis (tuberculosis)

RBC, Red blood cell; Z-N, Ziehl-Neelsen.

TABLE 6-24 Common Cytologic Findings in Conjunctiva

Sample Site	Findings	Diagnosis
Conjunctiva	Conjunctiva; columnar cells or squamous cells present	Normal cytology
	Predominance of one type of bacterium and inflammatory cells a) Cocci in clusters: *Staphylococcus* sp. b) Chain-forming cocci: *Streptococcus* sp. c) Coccobacillary (short) rods: *Pasteurella* sp. or *Bordetella* sp. d) Thin bacterial rods: *Pseudomonas* sp. or *Aeromonas* sp. e) Cytoplasmic inclusions: *Chlamydia/Mycoplasma* spp. f) Bacterial rods, Chinese-letter pattern: *Corynebacterium* sp.	Conjunctivitis
	Squamous cells with Bollinger bodies present	Avian pox
	Squamous cells, inflammatory cells, and cryptosporidial oocysts present	Cryptosporidiosis
	Helminths, whole or portions present	Ocular trematode or nematode infection

TABLE 6-25 Common Cytologic Findings in Aspirated Fluids

Sample Site	Findings	Diagnosis
Aspirated fluids	Mesothelial cells and RBCs present	Normal cytology
	Reactive mesothelial cells, bacteria, and inflammatory cells present a) Cocci in clusters: *Staphylococcus* sp. b) Chain-forming cocci: *Streptococcus* sp. c) Rods: *E. coli/Pseudomonas* sp.	Inflammation
	Synovial cells with Bollinger bodies present	Avian pox
	Serratospiculum sp. eggs	Serratospiculosis
	Urate (crystal) deposition	Visceral or articular gout

RBC, Red blood cell.

TABLE 6-26 Common Cytologic Findings in Internal Organs

Sample Site	Findings	Diagnosis
Heart	RBCs without bacteria	Normal cytology
	Inflammatory cells including heterophils and macrophages, bacteria present	Pericarditis
	a) Chain-forming cocci: *Streptococcus* sp.	Mycobacteriosis (tuberculosis)
	d) Thick rods: *Clostridium* sp.	
	c) Ghost cells: *Mycobacterium* sp. (acid-fast bacterial rods in Z-N stain)	
	Urate crystals	Visceral gout
	Microfilaria	Filariasis
Kidney	Renal squamous cells	Normal cytology
	Reactive renal cells, inflammatory cells, and bacteria	Septicemia
	a) Chain-forming cocci: *Streptococcus* sp.	
	b) Thick rods: *Clostridium* sp.	
	Plasma cells, reactive renal cells	Avian leucosis
	Plasma cells, reactive renal cells.	Septicemic avian pox
	Urate crystals	Visceral gout
Liver	Hepatocytes and RBCs	Normal cytology
	Heterophils, macrophages, Kupffer cells and bacteria	Bacterial hepatitis
	a) Chain-forming cocci: *Streptococcus* sp.	
	b) Thick rods: *Clostridium* sp.	
	c) Short rods: *Salmonella* sp.	
	d) Cytoplasmic inclusions: *Chlamydia* sp.	
	Hepatocytes with vacuolation (lipid)	Fatty liver/lipidosis
	Hepatocytes and fibrous tissue	Amyloidosis
	Hepatocytes with intranuclear inclusions	Herpes virus infection
	Hepatoytes with reactive changes, lymphoid cells	Avian leucosis
	Hepatocytes with reactive changes, numerous Kupffer cells, and ghost cells (acid-fast bacterial rods in Z-N stain)	Mycobacteriosis (tuberculosis)
Spleen	Lymphoid cells	Normal cytology
	Plasma cells, reactive lymphoid cells, and bacteria	Septicemia
	a) Chain-forming cocci: *Streptococcus* sp.	
	b) Thick rods: *Clostridium* sp.	
	c) Short rods: *Salmonella* sp.	
	Plasma cells, reactive lymphoid cells, and cytoplasmic inclusions	Chlamydiosis
	Plasma cells, reactive lymphoid cells with perinuclear inclusions	Herpes virus infection
	Plasma cells, reactive lymphoid cells	Avian leucosis
	Plasma cells, reactive lymphoid cells, and ghost cells (acid-fast bacterial rods in Z-N stain)	Mycobacteriosis (tuberculosis)
	Plasma cells, reactive lymphoid cells with karyorrhexis	Septicemic avian pox

RBC, Red blood cell; Z-N, Ziehl-Neelsen.

TABLE 6-27 Common Cytologic Findings in Skin

Sample Site	Findings	Diagnosis
Skin	Keratinized squamous cells	Normal cytology
	Bacteria and inflammatory cells	Bacterial dermatitis, abscess or bumblefoot (pododermatitis)
	Squamous cells with Bollinger bodies	Avian pox
	Louse, mite, flea, or nymph of ticks (or parts thereof)	Ectoparasitic infection
	Fungal filaments and keratinized squamous cells	Mycotic dermatitis

the latter with results of other investigations (e.g., microbiology, hematology, clinical chemistry, and possibly histopathology and electronmicroscopy).

INTERPRETATION

Correct interpretation requires:

a. A sound knowledge of normal host cell morphology and the appearance of microorganisms and metazoan parasites (Cousquer, *et al.*, 2010).
b. An understanding of pathologic changes, especially at the cellular level (e.g., pyknosis, karyorrhexis, inclusion-body formation).
c. Recognition that artifactual changes may mimic lesions or parasites. For example, stain sediment may resemble bacteria, and talc crystals may resemble deposits in the kidney. Keratin may be

deposited on the preparation from the clinician's or technician's fingers. Colored material from the ultrasound gel may give an unusual color to material on the slide.

d. An appreciation of the limitations of cytologic techniques and of our poorly developed understanding of the relevance of some changes, especially in nonmammalian species.

Interpretation is based on:

a. Clinical/postmortem history
b. Assessment of cytologic findings by microscopy

Slides should be stored in the dark after examination and oil should not be wiped off from preparations where there is no coverslip because this can damage the cells and structures.

EXAMPLES OF AVIAN CYTOLOGIC FINDINGS

These are illustrated in Figs. 6-90 to 6-99.

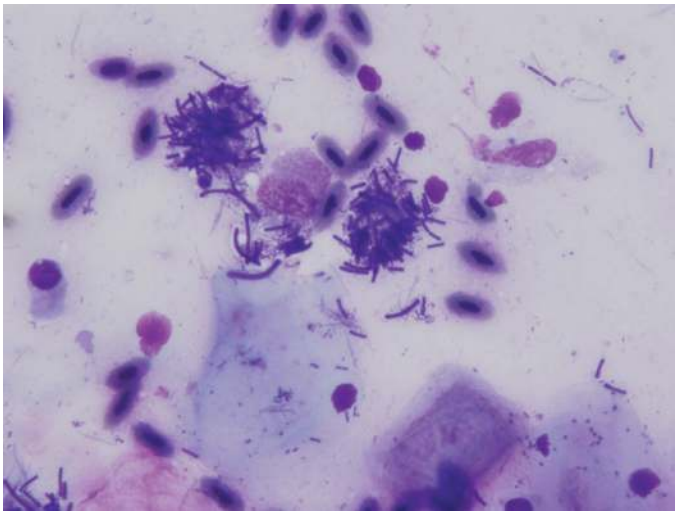


FIGURE 6-90 Smear from the choana of a saker falcon showing *Macrorhabdus ornithogaster* (Neat stain, 1000×).

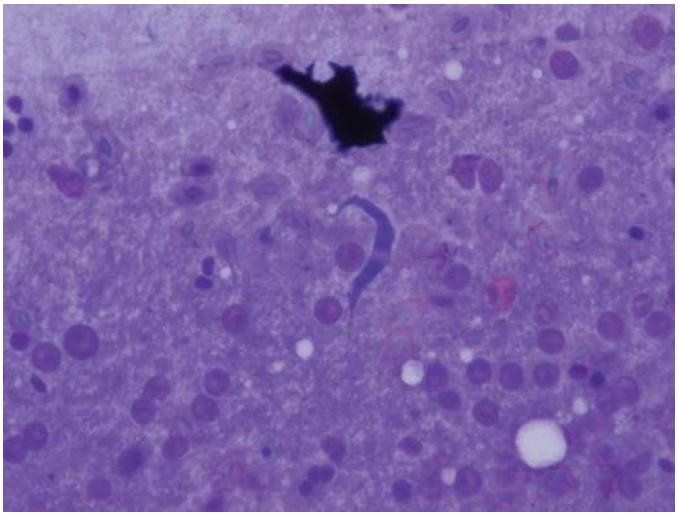


FIGURE 6-91 Liver imprint of an eagle showing a *Trypanosoma* sp. (Neat stain, 1000×).

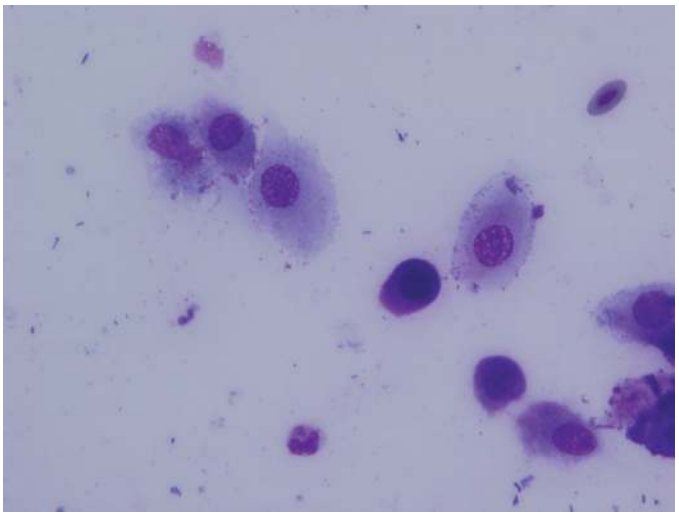


FIGURE 6-92 Smear from the conjunctiva of a gyr falcon showing *Chlamydia* inclusions (Neat stain, 1000×).

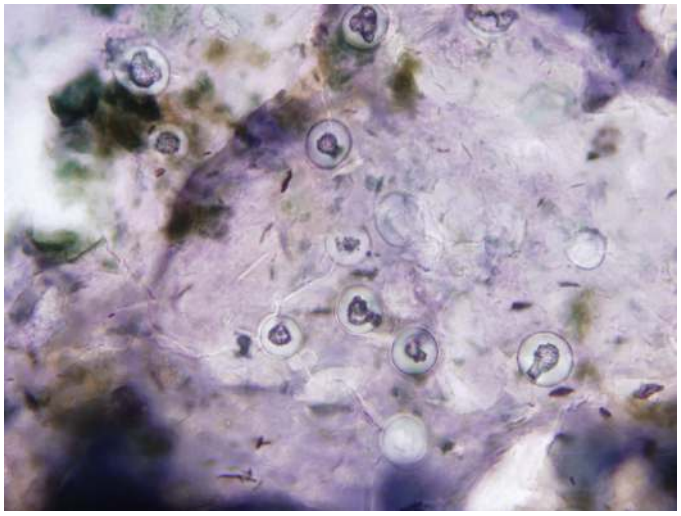


FIGURE 6-93 Biopsy smear from the air sac of a gyr falcon during antifungal treatment showing hulle cells of an *Aspergillus* sp. (Neat stain, 1000×).

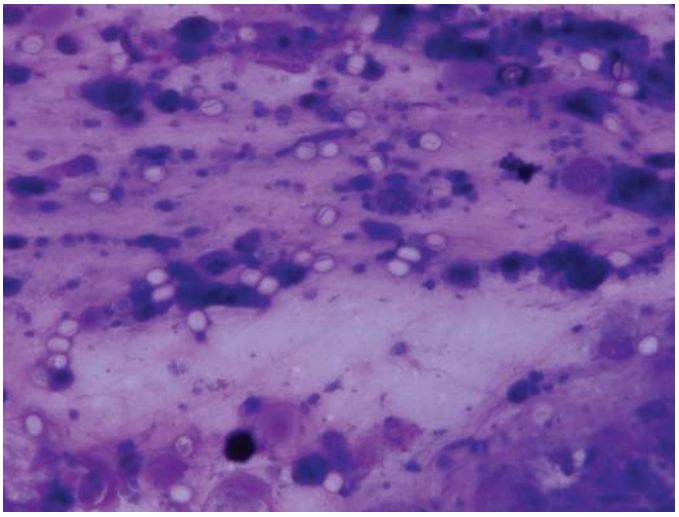


FIGURE 6-94 Smear from the trachea of a peregrine falcon showing a *Cryptosporidium* sp. (Neat stain, 1000×).

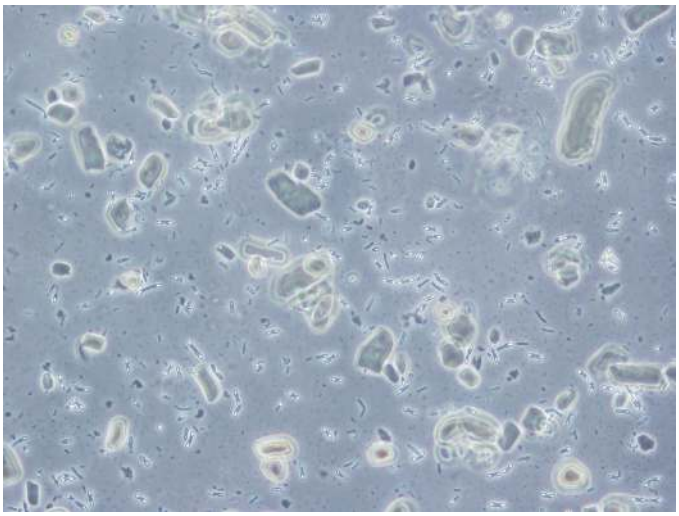


FIGURE 6-95 Direct phase contrast microscopy of feces of a gyr falcon showing sporulated *Clostridium* bacteria (400×).

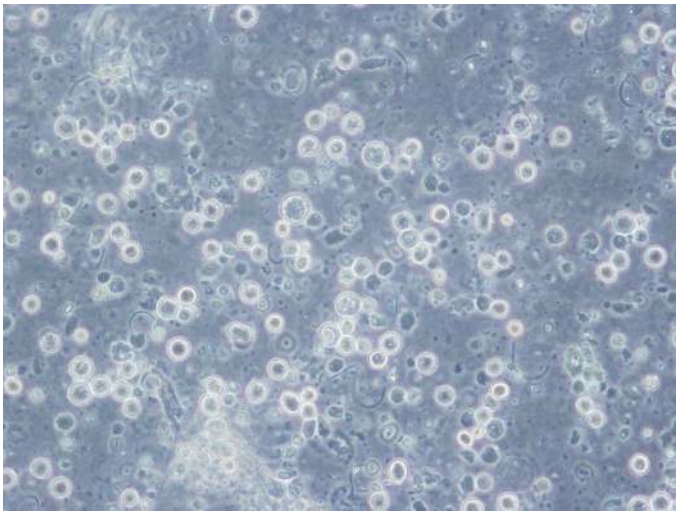


FIGURE 6-96 Direct phase contrast microscopy of feces of a peregrine falcon showing *Cryptococcus* sp. (400×). Note the pleomorphic appearance that provides the differential diagnosis from coccidial species.

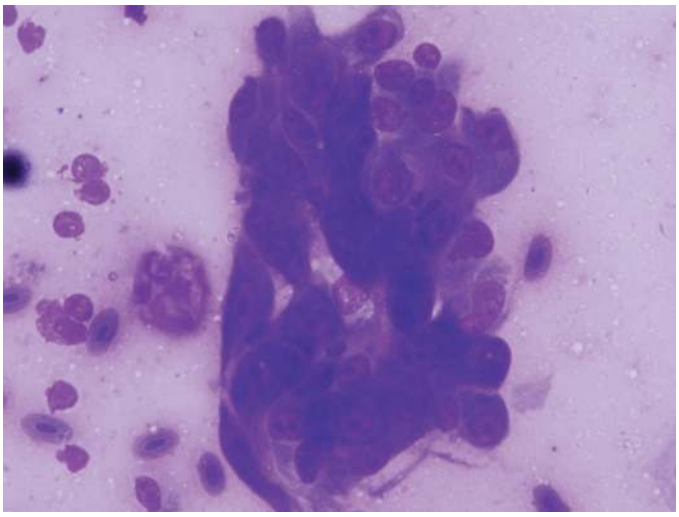


FIGURE 6-97 Liver imprint of a trumpeter hornbill with a herpes infection, showing hepatocytes with metaplasia, nuclear pleomorphism, dispersed chromatin, and multiple nucleoli (Neat stain, 1000×).

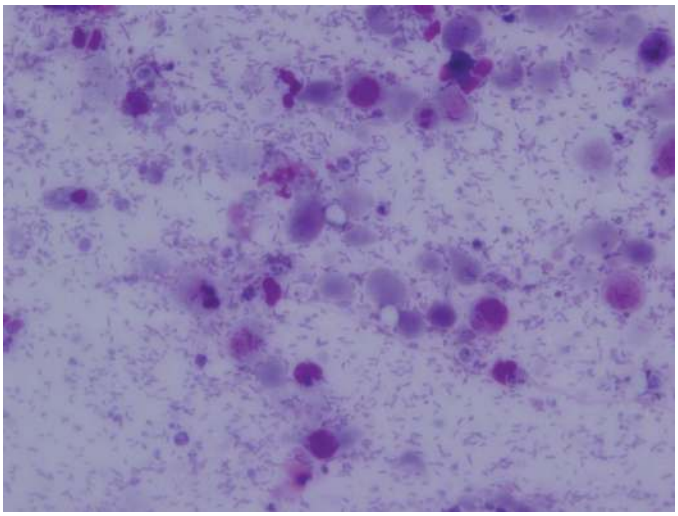


FIGURE 6-98 Smear from the trachea of a peregrine falcon showing mixed infection with a *Pseudomonas* sp. and a *Cryptosporidium* sp. (Neat stain, 1000×).

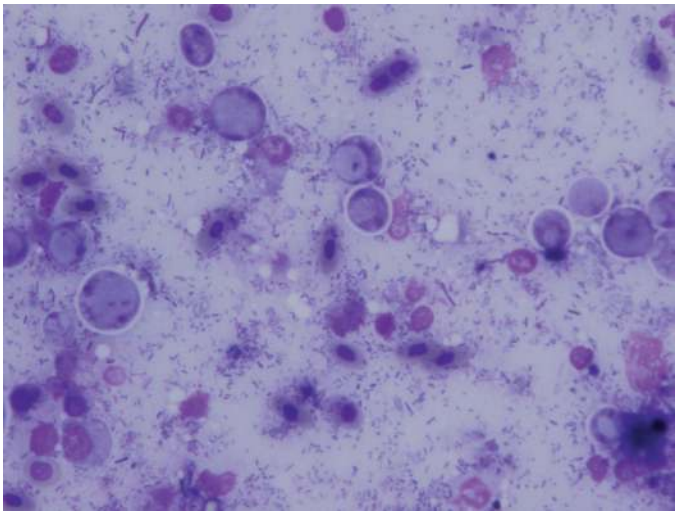


FIGURE 6-99 Lung imprint of a partridge showing mixed bacterial and a protozoal (probably a *Histomonas* sp.) infection (Neat stain, 1000×).

NEGATIVE FINDINGS

Failure to make a diagnosis or provide a helpful interpretation of findings can be caused by various factors; some related to the specimen itself, others to poor technique. For instance, exfoliation may be minimal or nonexistent if the lesion is composed only of mesenchymal cells. If the tissue is highly vascular, or too large a needle is used to obtain an aspirate, only blood may be visible on the slide. Inadequate blotting of touch preparations may mean that they too deposit excess blood on the slide and significant cells are rendered invisible.

ACKNOWLEDGEMENTS

We are grateful to Dr. Jaime Samour for his invitation to contribute again to his book. We thank numerous colleagues, past and present, in Arabia, Europe, Africa, and the Caribbean, who have participated in our studies and shared interests in avian cytology.

REFERENCES

Campbell TW: Cytodiagnosis in raptor medicine. In Redig PT, Cooper JE, Remple JD, Hunter DB, editors: *Raptor biomedicine*, Minneapolis, 1993, University of Minnesota Press, pp 199–222.

Campbell TW: Cytology. In Ritchie B, Harrison G, Harrison L, editors: *Avian medicine: principles and application*, Lake Worth, 1994, Wingers Publishing, p 101.

Campbell TW: *Avian hematology and cytology*, ed 2, Ames, 1995, Iowa State University Press.

Cooper JE: Biopsy techniques, *Seminars in Avian and Exotic Pet Medicine* 3(3):161–165, 1994.

Cooper JE: *Birds of prey: health & disease*, Oxford, 2002, Blackwell.

Cooper JE: In-practice and field techniques for the investigation of parasitic infections, *J Exot Pet Med* 18(4):280–290, 2009.

Cooper JE: Field techniques in exotic animal medicine, *J Exot Pet Med* 22(1):1–64, 2013.

Corr SA, Maxwell M, Gentle MJ, Bennett D: Preliminary study of joint disease in poultry by the analysis of synovial fluid, *Vet Rec* 152:549–554, 2002.

Cousquer GO, Cooper JE, Cobb MA: Conjunctival flora in tawny owls (*Strix aluco*), *Vet Rec* 166:652–654, 2010.

Hawkey CM, Dennett TB: *A colour atlas of comparative veterinary haematology*, London, 1989, Wolfe.

Latimer KS, Goodwin MA, Davis MK: Rapid cytologic diagnosis of respiratiory cryptosporidiosis in chickens, *Avian Dis* 32:826–830, 1988.

Pinches M: First steps in cytology; non inflammatory cell types, *UK VET* 10(5):78–81, 2005a.

Pinches M: First steps in cytology; the examination, and beyond, *UK VET* 10(4):89–92, 2005b.

Pinches M: Increasing information yield in cytology, *UK VET* 10(3):96–98, 2005c.

Rosenthal KL, Morris DO, Mauldin EA, et al: Cytologic, histologic, and microbiologic characterization of the feather pulp and follicles of feather-picking psittacine birds: a preliminary study, *J Avian Med Surg* 18(3):137–143, 2004.

Silvanose CD, Bailey TA: Bustard diagnostic cytology. In Bailey TA, editor: *Diseases and medical management of houbara bustards and other Otididae*, Abu Dhabi, 2008, Emirates Printing Press, pp 97–116.

Teachout DJ: Cytological sample analysis and interpretation: National Wildlife Rehabilitators' Association: topics in wildlife medicine, *Clin Pathol* 1:43–53, 2005.

Thrall MA, Weiser MG, Allison R, Campbell T: *Veterinary hematology and clinical chemistry*, ed 2, Hoboken, 2012, Wiley-Blackwell.

RADIOGRAPHY

Jesus Naldo, Miguel Saggese

Radiography is an essential and practical clinical diagnostic procedure in avian medicine that is applicable to the diagnosis of musculoskeletal disorders and diseases of the coelomic cavity. It is one of the most important diagnostic tools because of the availability of rapid interpretation and the ability to perform it on patients of different sizes. It is useful as a primary diagnostic technique and also as an adjunct to other procedures, such as endoscopy and hematology, in making a differential diagnosis. In addition, radiography can prove valuable for the monitoring of progression of diseases and in evaluating the efficiency of therapeutic regimens. Radiologic techniques in avian practice have made great progress with the introduction of high-frequency ultralight radiographic units, cassettes with high-definition screens, fast films, automatic developers, and more recently, digital radiology systems. With the advent of safe and efficient inhalation anesthetic agents, such as isoflurane, radiography in birds is now an uneventful procedure.

RADIOLOGY UNITS

In avian radiography the radiology unit should be capable of producing at least 300 milliamperes (mA), the exposure time capability should be 0.17 (1/60) seconds or shorter, the peak kilovoltage (kVp) settings should have a range of 40 to 90 kVp, and kVp settings should be adjustable in 2 kVp increments. Short exposure times (0.15-0.05 seconds or shorter) should be used to minimize motion blur caused by the high respiratory rate and generalized muscle tremors that are common in small- and medium-sized birds. Low kVp techniques (40-60 kVp) are preferred for most film screen systems because they produce a high degree of contrast and a wide gray scale range. The recommended focal-film distance is between 80 and 100 cm. Grids should not be used in birds.

Portable units are most widely used in general veterinary practice and are suitable for avian radiography. They possess a number of advantages, which include:

- They are less expensive than other types of units.
- They can operate from a 13-A or 15-A electrical point.
- They can be easily dismantled and transported by car.
- They are lightweight and easily maneuvered.

SCREENS, CASSETTES, AND FILMS

High-definition or fine-grain screens in cassettes are now more widely used in avian practice than nonscreen films because of the following features:

- They produce more fine detail than fast screens.
- They require less amperage than nonscreen film but more amperage than fast screens.

Rare earth intensifying screens with single-emulsion films will give detailed results. However, they require a longer exposure time compared with double emulsion film-cassette combinations.

The choice of film depends on the detail required in the radiograph and the nature of the examination. There are three types of screen film:

- Standard: A fine-grain medium-speed film that is good for use with high-definition intensifying screens. Excellent for avian radiography and extremities of larger species.
- Fast: The speed is almost twice that of standard. Good for veterinary radiography.
- Ultrafast: Needs a shorter exposure time; suitable to use in avian radiography but has a short storage life.

DIGITAL RADIOLOGY UNITS

Digital radiographic image capture, such as direct digital and computed radiography, is slowly replacing film-screen systems in veterinary medicine and will eventually predominate. Initial replacement of traditional radiographic equipment to digital may be expensive. Digital radiology is especially useful in avian clinics and hospitals with large case loads and practitioners or academicians involved in avian research and/or publishing case reports.

Nonscreen film and high-detail film-screen systems produce images with superior detail compared with digital systems. However, digital systems have some advantages over film systems (Silverman and Tell, 2009):

- They have a higher image contrast range, which results in improved image quality.
- They produce images that can be electronically manipulated.
- They do not require film processing.
- They are immediately viewable.
- They result in fewer repeat exposures caused by incorrect exposure factors and film processing errors.

- They allow their electronic transference.
- There is a reduced exposure to radiation.

Digital systems often use 10% to 15% higher kVp and mAs than film-screen systems.

RESTRAINT AND POSITIONING

Adequate restraint for radiography is critical if high-quality diagnostic radiographs are to be obtained. Physical restraint is stressful and there is a high probability of worsening the condition of the bird and causing dislocations or even bone fractures. More importantly, with physical restraint there is an increased possibility of radiation exposure to staff.

Birds are ideally fasted before radiographic examinations. Birds weighing less than 100 grams are fasted for 2 hours and larger birds are fasted for 3 to 5 hours before the radiographic procedures. The decision to withhold food in a clinical situation is complex because avian patients, especially those that are debilitated, are easily compromised by food deprivation.

Inhalation anesthesia with isoflurane (IsoFlo, Abbott Laboratories, North Chicago, Ill., USA) is the safest method of restraining birds. Before radiographic examination, birds are anesthetized with a combination of isoflurane and oxygen administered by a face mask. Birds under anesthesia for more than 15 minutes are intubated with an uncuffed endotracheal tube. Anesthesia is induced with 5% isoflurane and maintained with 2% to 3% isoflurane combined with oxygen at 0.5 L/min.

Positioning of the patient is very important to produce a good diagnostic radiograph. Survey radiographs of the whole body in the ventrodorsal and lateral projections are taken of each bird. Detailed radiograph of an extremity (e.g., head, neck, wing, foot) is taken when indicated.

Ventrodorsal radiographs are suitable for assessing the following:

- Variations in symmetry of the relative positions of the organs
- Heart and liver shadows
- Abdominal air sacs
- Wing bones
- Pectoral girdle and pelvis
- Hip, femur, tibiotarsus, and tarsometatarsus
- Skull and cervical spinal column

Lateral radiographs are suitable for assessing the following:

- Skull
- Spinal column and synsacrum
- Ribs and sternum
- Heart and major blood vessels
- Lung structure and main bronchus
- Spleen, kidneys, and gonads
- GI tract

Adequate positioning can be achieved with the following procedures (Helmer, 2006; Krautwald-Junghanns, 2007; Krautwald-Junghanns and Trinkaus, 2000; Krautwald-Junghanns and Pees, 2009; Krautwald-Junghanns *et al.*, 2011; Pees, 2008; Samour and Naldo, 2007; Silverman and Tell, 2009).

Positioning for Imaging the Body

In the **ventrodorsal view** (Fig. 6-100):

- The bird is placed on dorsal recumbency.
- The keel should be superimposed over the vertebral column.
- Both wings are slightly extended laterally and secured with radiolucent tape.
- Both legs are pulled backward, positioned symmetrically, and secured with radiolucent tape on the tarsometatarsus.
- The x-ray beam is centered midline on the caudal portion of the sternum.

FIGURE 6-100 Positioning technique for ventrodorsal body radiograph of a violet turaco (*Musophaga violacea*) under isoflurane anesthesia. The bird is placed on dorsal recumbency. The keel should be superimposed over the vertebral column. Both wings are slightly extended laterally and secured with radiolucent tape. Both legs are pulled backward, positioned symmetrically, and secured with radiolucent tape on the tarsometatarsus. The x-ray beam is centered midline on the caudal portion of the sternum.

- The x-ray beam field includes the coelom, head, and extremities for small birds. For medium and large birds, the x-ray field includes the body, proximal extremities, and caudal cervical regions.
- Metallic "R" and "L" markers are placed on the cassette indicating the laterality of the patient.

In the **lateral view** (Fig. 6-101):

- The bird is usually placed in left to right lateral recumbency.
- The hip and shoulder joints should be superimposed.
- The wings should be extended dorsally, with the lower wing placed slightly cranial to the upper wing to permit differentiation of right from left.
- The upper wing is secured with radiolucent tape across the carpometacarpal joints.
- Foam padding should be placed in between the wings to prevent overextension.
- Both legs can be extended caudally or the dependent leg can be positioned cranially to the contralateral leg and secured at the tarsometatarsus with radiolucent tape.
- The x-ray beam is centered on the midline cranial to the caudal tip of the sternum.
- The x-ray beam field includes the entire bird for small birds. For medium and large birds, the x-ray field includes the body, proximal extremities, and caudal cervical regions.
- A metallic "R" marker is placed on the cassette indicating that the right side is dependent.

FIGURE 6-101 Positioning technique for lateral body radiograph of a violet turaco (*Musophaga violacea*) under isoflurane anesthesia. The bird is usually placed in left to right lateral recumbency. The hip and shoulder joints should be superimposed. The wings should be extended dorsally, with the lower wing placed slightly cranial to the upper wing to permit differentiation of right from left. The upper wing is secured with radiolucent tape across the carpometacarpal joints. Both legs can be extended caudally or the dependent leg can be positioned cranially to the contralateral leg and secured at the tarsometatarsus with radiolucent tape. The x-ray beam is centered on the midline cranial to the caudal tip of the sternum.

Positioning for Imaging the Head

In the **ventrodorsal view** (Fig. 6-102):

- The bird is placed on dorsal recumbency.
- A radiolucent tape is placed onto the ventral aspect of the rhinotheca to hyperextend the maxilla at an angle closer to the cassette.
- The x-ray beam is centered between the eyes on the midline.
- The x-ray beam field includes the entire head and the cervical vertebrae.
- Metallic "R" and "L" markers are placed on the cassette indicating the laterality of the patient.

In the **dorsoventral view** (Fig. 6-103):

- The bird is placed on ventral recumbency.
- A radiolucent tape is placed onto the ventral aspect of the rhinotheca to hyperextend the mandible at an angle closer to the cassette.
- The x-ray beam is centered between the eyes on the midline.
- The x-ray beam field includes the entire head and the cervical vertebrae.
- Metallic "R" and "L" markers are placed on the cassette indicating the laterality of the patient.

In the **lateral view** (Fig. 6-104):

- The bird is placed on right lateral recumbency with the head resting on the cassette.
- A radiolucent tape is used to secure the maxilla and mandible.
- The x-ray beam is centered ventral to the eye.
- The x-ray beam field includes the entire head and the cervical vertebrae.
- A metallic "R" marker is placed on the cassette indicating that the right side is dependent.

In the **bisecting angle view(s)** (Fig. 6-105):

- The bird is placed in dorsal or lateral recumbency.

FIGURE 6-102 Positioning technique for ventrodorsal radiograph of the head of a violet turaco (*Musophaga violacea*) under isoflurane anesthesia. The bird is placed on dorsal recumbency. A radiolucent tape is placed on the ventral aspect of the rhinotheca to hyperextend the maxilla at an angle closer to the cassette. The x-ray beam is centered between the eyes on the midline.

FIGURE 6-103 Positioning technique for dorsoventral radiograph of the head of a violet turaco (*Musophaga violacea*) under isoflurane anesthesia. The bird is placed on ventral recumbency. A radiolucent tape is placed on the ventral aspect of the rhinotheca to hyperextend the mandible at an angle closer to the cassette. The x-ray beam is centered between the eyes on the midline.

FIGURE 6-104 Positioning technique for lateral radiograph of the head of a violet turaco (*Musophaga violacea*) under isoflurane anesthesia. The bird is placed on right lateral recumbency with the head resting on the cassette. A radiolucent tape is used to secure the maxilla and mandible. The x-ray beam is centered ventral to the eye.

FIGURE 6-105 Positioning technique for rostrocaudal radiograph of the head of a violet turaco (*Musophaga violacea*) under isoflurane anesthesia. The bird is placed on dorsal recumbency. The head of the bird is laid at an angle of 90 degrees to the cassette with its beak closed or slightly opened. The head is held in this position by a piece of radiolucent tape placed on the base of its neck. The x-ray beam is centered at the tip of the beak.

- The head of the bird is placed at an angle of 45 degrees to the cassette with its beak closed or slightly opened.
- The head is held in this position by a piece of radiolucent tape placed along the sagittal axis.
- The x-ray beam is centered at the level of the tip of the beak.
- The x-ray beam field includes the entire head and the cervical vertebrae.
- Metallic "R" and "L" markers are placed on the cassette indicating the laterality of the patient.

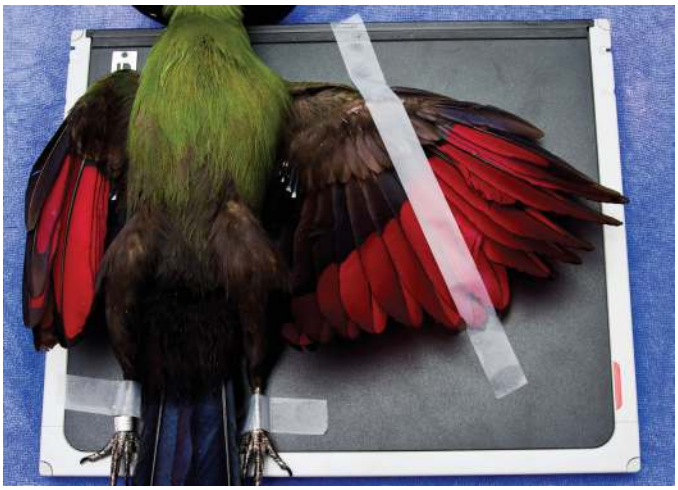


FIGURE 6-106 Positioning technique for mediolateral radiograph of the wing of a violet turaco (*Musophaga violacea*) under isoflurane anesthesia. The bird is placed on dorsal recumbency on the side of the cassette. The keel should be superimposed over the vertebral column. Both legs are pulled backward, positioned symmetrically, and secured with radiolucent tape on the tarsometatarsus. The wing is fully extended laterally from the pectoral girdle and taped directly to the radiographic cassette. The x-ray beam is centered in the middiaphyseal region of the radius and ulna.

Positioning for Imaging the Wing

In the **mediolateral view** (Fig. 6-106):

- The bird is placed on dorsal recumbency on the side of the cassette.
- The keel should be superimposed over the vertebral column.
- Both legs are pulled backward, positioned symmetrically, and secured with radiolucent tape on the tarsometatarsus.
- The wing is fully extended laterally from the pectoral girdle and taped directly to the radiographic cassette.
- The x-ray beam is centered in the middiaphyseal region of the radius and ulna.
- The x-ray beam field includes the entire wing, including the scapulohumeral joint.
- The appropriate metallic "R" or "L" marker is placed on the cassette indicating whether the image is of the right or left wing.

In the **caudocranial view** (Fig. 6-107):

- The caudocranial view of the wing may be beneficial particularly to evaluate fractures of the wing or damage to the clavicle, coracoid, scapula, or humerus. It also can be very useful to evaluate placement of internal and external skeletal fixators during orthopedic surgery.
- The anesthetized bird is held in an inverted position with the head directed toward the floor and long axis of the bird's body perpendicular to the surface of the x-ray table.
- The wing is fully extended and the cranial edge of the wing is placed on the film cassette.
- The x-ray beam is centered in the middiaphyseal region of the radius and ulna.
- The x-ray beam field includes the entire wing, including the scapulohumeral joint.

FIGURE 6-107 Positioning technique for caudocranial radiograph of the wing of a violet turaco (*Musophaga violacea*) under isoflurane anesthesia. The bird is held in an inverted position with the head directed toward the floor and long axis of the bird's body perpendicular to the surface of the x-ray table. The x-ray beam is centered in the middiaphyseal region of the radius and ulna.

FIGURE 6-108 Positioning technique for "stressed" radiograph of the wings of a Western plantain-eater (*Crinifer piscator*) under isoflurane anesthesia. The bird is placed on dorsal recumbency. Both legs are pulled backward, positioned symmetrically, and secured with radiolucent tape on the tarsometatarsus. Both wings are fully extended laterally, "stressed" cranially, positioned symmetrically, and secured with radiolucent tape on the metacarpals.

- The appropriate metallic "R" or "L" marker is placed on the cassette indicating whether the image is of the right or left wing.

  In the **"stressed" view** (Fig. 6-108):
- Detailed radiograph of the wing in "stressed" position may be beneficial to evaluate fractures or damage to the humerus, clavicle, coracoid, or scapula.
- The bird is placed on dorsal recumbency.
- Both legs are pulled backward, positioned symmetrically, and secured with radiolucent tape on the tarsometatarsus.
- Both wings are fully extended laterally, "stressed" cranially, positioned symmetrically, and secured with radiolucent tape on the metacarpals.

FIGURE 6-109 Positioning technique for dorsoplantar radiograph of the hind limb of a Western plantain-eater (*Crinifer piscator*) under isoflurane anesthesia. The bird is placed on dorsal recumbency. The leg is pulled backward and secured with radiolucent tape on the tarsometatarsus. The x-ray beam is centered on the middiaphyseal region of the tibiotarsus. The x-ray beam field includes the entire limb of interest including the coxofemoral joint.

- The x-ray beam is centered midline on the cranial portion of the sternum.
- The x-ray beam field includes the neck, anterior body, and both wings.
- Metallic "R" and "L" markers are placed on the cassette indicating the laterality of the patient.

Positioning for Imaging the Hindlimb

In the **dorsoplantar view** (Fig. 6-109):

- The bird is placed on dorsal recumbency.
- The leg is pulled backward and secured with radiolucent tape on the tarsometatarsus.
- All digits are secured with radiolucent tape.
- The x-ray beam is centered on the middiaphyseal region of the tibiotarsus.
- The x-ray beam field includes the entire limb of interest including the coxofemoral joint.
- For comparison with the contralateral leg, both legs are pulled backward, positioned symmetrically, and secured with radiolucent tape on the tarsometatarsus.
- The appropriate metallic "R" or "L" marker is placed on the cassette indicating whether the image is of the right or left leg.

  In the **mediolateral view** (Fig. 6-110):
- The bird is placed in lateral recumbency with the leg of interest in the dependent position.
- The leg is taped at the distal aspect of the tarsometatarsus.
- All digits are secured with radiolucent tape.

FIGURE 6-110 Positioning technique for mediolateral radiograph of the hind limb of a Western plantain-eater (*Crinifer piscator*) under isoflurane anesthesia. The bird is placed in lateral recumbency with the leg of interest in the dependent position. The leg is taped at the distal aspect of the tarsometatarsus. All digits are secured with radiolucent tape. The nondependent leg is extended caudally to separate the legs and minimize superimposition. The x-ray beam is centered on the intertarsal joint. The x-ray beam field includes the entire limb of interest including the coxofemoral joint.

FIGURE 6-111 Positioning technique for dorsoplantar radiograph of the foot of a Western plantain-eater (*Crinifer piscator*) under isoflurane anesthesia. The bird is placed on dorsal recumbency. The leg is pulled backward and secured with radiolucent tape on the tarsometatarsus. All digits are fully extended and secured with radiolucent tape. The x-ray beam is centered on the condyles of the tarsometatarsal bone. The x-ray beam field includes all of the phalanges.

- The nondependent leg is extended caudally to separate the legs and minimize superimposition.
- The x-ray beam is centered on the intertarsal joint.
- The x-ray beam field includes the entire limb of interest including the coxofemoral joint.
- The appropriate metallic "R" or "L" marker is placed on the cassette indicating whether the image is of the right or left leg.

Positioning for Imaging the Foot

In the **dorsoplantar view** (Fig. 6-111):

- The bird is placed on dorsal recumbency.
- The leg is pulled backward and secured with radiolucent tape on the tarsometatarsus.
- All digits are fully extended and secured with radiolucent tape.
- The x-ray beam is centered on the condyles of the tarsometatarsal bone.
- The x-ray beam field includes all of the phalanges.
- For comparison with the contralateral foot, both feet are pulled backward, positioned symmetrically, and secured with radiolucent tape on the tarsometatarsus.
- The appropriate metallic "R" or "L" marker is placed on the cassette indicating whether the image is of the right or left foot.

In the **mediolateral view** (Fig. 6-112):

- The bird is placed in lateral recumbency with the leg of interest in the dependent position.
- The leg is taped at the distal aspect of the tarsometatarsus.
- All digits are fully extended and secured with radiolucent tape.
- The nondependent foot is extended caudally to separate the feet and minimize superimposition.
- The x-ray beam is centered on the condyles of the tarsometatarsal bone.
- The x-ray beam field includes all of the phalanges.
- The appropriate metallic "R" or "L" marker is placed on the cassette indicating whether the image is of the right or left foot.

In the **caudoplantar view** (Fig. 6-113):

- A caudoplantar view of the foot is beneficial to evaluate the digits, metatarsophalangeal joints, and the sesamoid bone between the metatarsophalangeal joint of digit 2 and the flexor tendons in some raptor species. It is particularly useful in assessing chronic bumblefoot infection.
- The bird is placed on ventral recumbency over a rolled towel.
- The foot is positioned with the plantar surface as close as possible to the cassette.
- All digits are secured with radiolucent tape.
- The x-ray beam is centered at the point of the metatarsophalangeal joint of digit 1 (hallux).

Survey radiographs of hooded birds of prey that cannot be anesthetized for a particular reason (e.g., anesthetic risks—recently fed, too stressed, dyspneic—or simply because the owner has refused anesthesia) can be taken with the bird standing on a perch. The diagnostic

FIGURE 6-112 Positioning technique for mediolateral radiograph of the foot of a Western plantain-eater (*Crinifer piscator*) under isoflurane anesthesia. The bird is placed in lateral recumbency with the leg of interest in the dependent position. The leg is taped at the distal aspect of the tarsometatarsus. All digits are fully extended and secured with radiolucent tape. The nondependent leg is extended caudally to separate the legs and minimize superimposition. The x-ray beam is centered on the condyles of the tarsometatarsal bone. The x-ray beam field includes all of the phalanges.

FIGURE 6-113 Positioning technique for caudoplantar radiograph of the foot of a gyr falcon (*Falco rusticolus*) under isoflurane anesthesia. The bird is placed on ventral recumbency over a rolled towel. The foot is positioned with the plantar surface as close as possible to the cassette. All digits are secured with radiolucent tape. The x-ray beam is centered at the point of the metatarsophalangeal joint of digit 1.

value of this technique is very limited and it can be used only on selected cases (e.g., some musculoskeletal disorders, lead pellets or fragments in the ventriculus, impaction, and detection of a passive induced transponder [PIT]).

Ventrodorsal radiographs with limited diagnostic value can be taken in depressed or stuporous birds by working in complete darkness or by dimming the lights of the radiology room. The patient is gently positioned on dorsal recumbency on the cassette or table. Wings and legs are stretched out and immobilized with radiolucent tape. Further immobilization can be achieved by covering the head with a light towel or, in the case of raptors, with a hood of appropriate size.

Standing position:

- Radiographs can be obtained in the ventrodorsal or lateral positions.
- A cassette should be placed on the holder or the stand positioned as closed as possible to the patient.
- Rotate the head of the radiographic machine to center the horizontal beam over the patient and collimate to reduce scatter.
- Maintain the required exposure settings (Table 6-28).

CONVENTIONAL RADIOGRAPHY

For conventional radiography a portable radiographic unit (GIERTH HF80/15 plus, Mikasa X-ray Co., Ltd., 13-2, 3-Chome Hongo, Bunkyo-ku, Tokyo, Japan) can be used. This unit has an x-ray tube voltage of 50 to 80 kV, fixed 15 mA current, and exposure time of 0.02 to 1.99 seconds. Screen films (MG-SR, Konica Medical Film, Konica Corp. No. 26-2, Nishishinjuku 1-Chome, Shinjuku-ku, Tokyo 163-0512, Japan) and cassettes (HR-Regular, Veterinary X-Rays, Seer Green, Beaconsfield, Bucks HP9 2QZ, England) are commonly used. The exposure settings for conventional radiography used by the authors are described in Table 6-28.

MAGNIFICATION RADIOGRAPHY

Magnification or augmented radiography will enhance visualization of special areas of interest (e.g., the infraorbital sinus, limbs, joints). The cost of magnification is a reduction in the spatial resolution or image sharpness, which is inversely proportional to the degree of magnification (Tell *et al.*, 2003). The exposure settings for magnification radiography used by the authors are described in Table 6-29.

Indications:

- Evaluating the nature and extent of craniofacial soft tissue or musculoskeletal abnormalities
- Evaluating sinuses
- Evaluating ocular and ear abnormalities
- Evaluating limbs and joints

Tabletop technique (Fig. 6-114):

- The film cassette is placed on the top of the table.
- The object to film distance (OFD) is increased by placing the patient on foam blocks.
- The focal to film distance (FFD) is decreased by lowering the tube housing closer to the film cassette.

TABLE 6-28 Avian Radiographic Techniques—Conventional Radiography*

Subject	Bodyweight (Grams)	kV	mA	Time (Seconds)	FFD
Whole body, proximal limbs	2500-3500	60	15	0.04	26″
Head, distal limbs	2500-3500	55-60	15	0.04	23.5″-26″
Whole body	1400-1500	55-60	15	0.04	23.5″
Whole body	800-1300	55	15	0.04	23.5″
Whole body	<800	50	15	0.04	23.5″
Extremities: head, feet, wing	1000-1500	55	15	0.04	23.5″
Extremities: head, feet, wing	<1000	50	15	0.04	23.5″

*Radiographs were recorded on standard double emulsion films (MG-SR, Konica Medical Film, Japan) in high-definition screens in cassettes (HR-Regular, Veterinary X-Rays, UK).
The Atomscope HF 80 Portable X-ray equipment has a constant setting of 15 mA.
FFD, Focal to film distance; kV, kilovoltage; mA, milliampere.

TABLE 6-29 Avian Radiographic Techniques—Magnification Radiography*

Subject	Bodyweight (Grams)	kV	mA	Time (Seconds)	OFD	FFD
Whole body	1400-1500	55-60	15	0.04	12″	20″
Whole body	800-1300	55	15	0.04	12″	20″
Whole body	<800	50	15	0.04	12″	20″
Extremities: head, feet, wing	1000-1500	55	15	0.04	12″	20″
Extremities: head, feet, wing	<1000	50	15	0.04	12″	20″

*Radiographs were recorded on standard double emulsion films (MG-SR, Konica Medical Film, Japan) in high-definition screens in cassettes (HR-Regular, Veterinary X-Rays, UK).
The Atomscope HF 80 Portable X-ray equipment has a constant setting of 15 mA.
FFD, Focal to film distance; kV, kilovoltage; mA, milliampere; OFD, object to film distance.

FIGURE 6-114 Positioning technique for obtaining a magnified view of the hips of a saker falcon (*Falco cherrug*). The object to film distance (OFD) was 12 inches and the focal to film distance (FFD) was 20 inches. The OFD was increased by placing the falcon on a foam block.

RADIOGRAPHIC CONTRAST STUDY

Contrast studies may be performed for the examination of the following:

- Organ size
- Organ shape
- Organ position
- Abnormal contents
- Neoplasia
- Outline of an organ against neighboring organs
- Determination of organ function
- Thickness and condition of the wall of hollow structures

Common contraindications of contrast studies are:

- Organ perforation or suspicion of being perforated
- Known susceptibility of the patient to hypersensitivity reactions
- Repeated exposition to contrast medium (increases risk of hypersensitivity reactions)
- Concomitant use of anesthesia
- Intravenous administration of iodine contrast medium is also not recommended in patients with shock, emaciation, and in cases of kidney disorders.

Gastrointestinal Contrast Studies

Indications:

- Chronic regurgitation
- Persistent diarrhea
- Constipation

- Abnormal palpation
- Abdominal enlargement
- Abnormalities in the gastrointestinal tract observed on survey radiographs

A survey radiograph must always be taken before beginning a GI tract contrast study. If birds are not severely ill, they should be fasted for a period of time according to the species' size, nutritional status, and energy demands, and before the administration of the contrast medium to judge transit time. In dehydrated birds adequate fluid replacement must be achieved before the administration of the contrast medium. Silverman and Tell (2009) recommend that in general birds should be anesthetized for the survey radiographs, then administered the contrast medium while still under anesthesia and subsequent images acquired, if the patient's health allows, before the patient is allowed to recover from anesthesia.

A study in psittacines showed no significant differences in the progression of barium sulfate between radiographs collected with isoflurane and manual restraint alone (Lennox and Crosta, 2005).

The common techniques used in avian GI tract radiography include:

- GI contrast with 25% to 45% barium sulfate administered directly into the esophagus with a dose of 20 mL/kg bodyweight. The contrast medium will be in the proventriculus and ventriculus within a few minutes and will reach the intestines in 30 to 60 min (Tables 6-30 and 6-31). If the area of interest is the lower GI tract, the contrast medium can be administered directly to the ventriculus (Krautwald-Junghanns and Pees, 2009; Krautwald-Junghanns *et al.*, 2011; Pees, 2008).
- Double contrast with 25% barium sulfate (positive contrast medium) in a dose of 10 mL/kg bodyweight given either orally or cloacally. Air (negative contrast medium) is introduced immediately after the administration of barium sulfate at 20 mL/kg bodyweight. This technique is intended to speed the progression of the contrast medium through the digestive tract. This technique is useful for demonstration of the thickness and condition of the wall of the GI tract and for demonstration of the cloaca (Krautwald-Junghanns, 2007; Krautwald-Junghanns *et al.*, 2011; Krautwald-Junghanns and Pees, 2009; Silverman and Tell, 2009).
- Nonionic and water-soluble iodine compounds, such as iodixanol and iotrolan, may be used as an alternative to barium sulfate suspension for GI tract contrast studies. They should be used with precautions in suspected cases of intestinal perforation because iodine is less likely than barium sulfate to cause peritonitis. They are administered in a dose of 10 mL/kg bodyweight (solution containing 250 mg iodine/mL). Iodine compounds have a lesser opacity and rapid transit time through the avian GI tract compared with barium (Krautwald-Junghanns *et al.*, 2011; Pees, 2008; Romagnano and Love, 2000; Smith and Smith, 1997).

Radiographic abnormalities that may be defined by GI contrast studies (McMillan, 1994) include:

- Change in location, size, or shape of abdominal organs
- Differentiation between the GI tract and other organs
- Increased or decreased motility
- Increased or decreased luminal diameter
- Mucosal irregularities
- Filling defects
- Changes in wall thickness
- Dilution of contrast with mucus or fluid
- Presence of radiolucent foreign bodies

TABLE 6-30 Barium Sulfate Transit Times*

Subject	Stomach	Small Intestines	Large Intestines	Cloaca
Canary	5	10-15	15-30	30-90
Indian hill mynah	5	10-15	15-30	30-90
Racing pigeon	5-10	10-30	30-120	120-240
Hawk	5-15	15-30	30-90	90-360
Budgerigar	5-30	30-60	60-120	120-240
African grey parrot	10-30	30-60	60-120	120-230
Amazon parrot	10-60	60-120	120-150	150-240
Pheasant	10-45	45-120	120-150	150-240

*Time in minutes for barium sulfate administered by crop gavage to reach and fill various portions of the gastrointestinal tract.
(Modified from McMillan MC: Imaging techniques. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application,* Lake Worth, FL., USA, 1994, Wingers Publishing, pp 246–326.)

TABLE 6-31 Barium Sulfate Transit Time*

Subject	Stomach	Intestines	Cloaca
Canary	5-10	10-20	30-45
Pigeon	5-15	30-60	60-120
Raptor	10-15	30-120	90-120

*Time in minutes for barium sulfate (20 mL/kg bodyweight) administered by crop gavage to reach and fill various portions of the gastrointestinal tract.
Modified from Pees M (2008).

Positive Pressure Insufflation Contrast Radiography (Fig. 6-115)

In an anesthetized, intubated bird, air sacs can be insufflated manually to increase total air sac space, thus using air as a negative contrast medium to improve visualization of internal organs and their borders on radiographs (Sherrill *et al.*, 2001).

- When anesthetized, birds are intubated with a semiflexible silicone endotracheal tube and maintained at a surgical plane of anesthesia.
- A rebreathing circuit fitted with a ventilation bag (1 L) and a manometer (units in cm of water) is used.
- Whole body radiographs are taken without and then with positive pressure insufflation (PPI).
- PPI at 20 cm H_2O pressure is applied manually to the ventilating bag of a closed rebreathing circuit while the radiograph is taken.

Urography

There are very few indications for urography in birds because gross and histologic characteristics of the kidney cause the resulting images to provide relatively little useful information. However, it can be used for identification of intraparenchymal renal masses or cysts, ureteral obstructions, and abnormalities in renal excretion (Helmer, 2006).

Potential indications for urogenital tract contrast studies:

- Polyuria/polydipsia of renal origin
- Changes in the size and form of the kidneys
- Nonspecific clinical signs of leg paresis or joint swelling

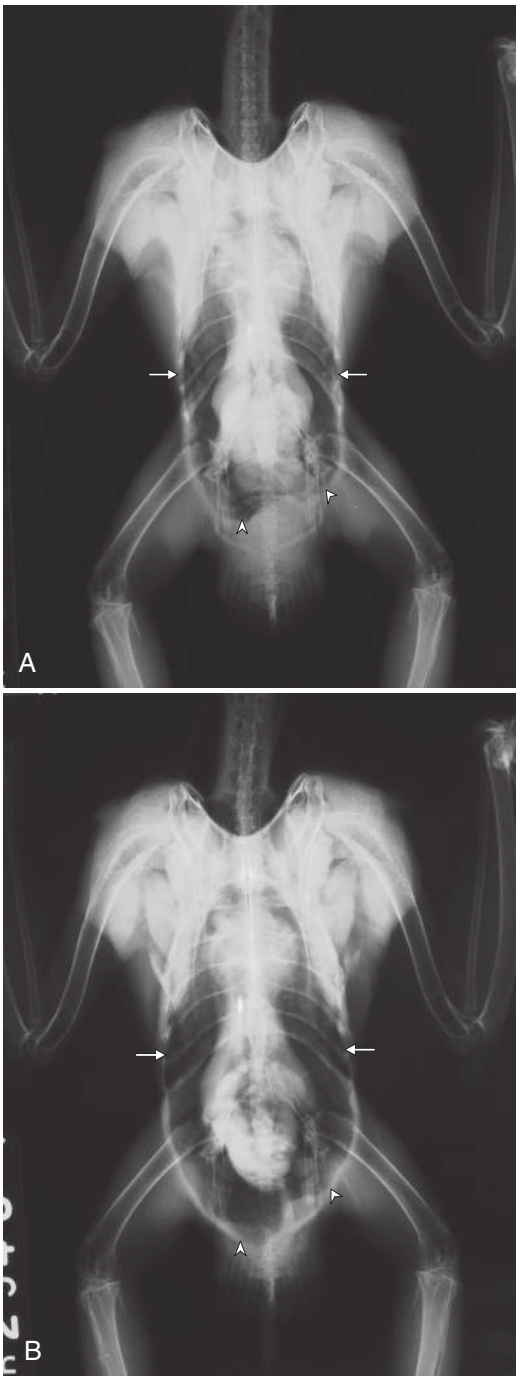


FIGURE 6-115 **(A)** Ventrodorsal survey radiograph and **(B)** ventrodorsal positive-pressure insufflation (PPI) radiograph of an anesthetized, intubated saker falcon (*Falco cherrug*). Note enhanced visualization of internal structures, including the thoracic (*arrows*) and abdominal (*arrowheads*) air sacs, as a result of PPI.

- To differentiate the kidney from the surrounding tissues or organs (e.g., ovarian cysts, tumors of the gonads)

For urogenital tract contrast radiography the agent used is an organic iodine compound with 300 to 400 mg iodine/mL. It must be warmed to body temperature and then slowly administered intravenously to the anesthetized bird at a dose of 2 mL/kg bodyweight. The heart, aorta, and lung arteries are already depicted 10 seconds after injection. The contrast agent reaches the kidneys and ureters after 30 to 60 seconds and the rectum and cloaca after 2 to 5 minutes (Krautwald-Junghanns *et al.*, 2011).

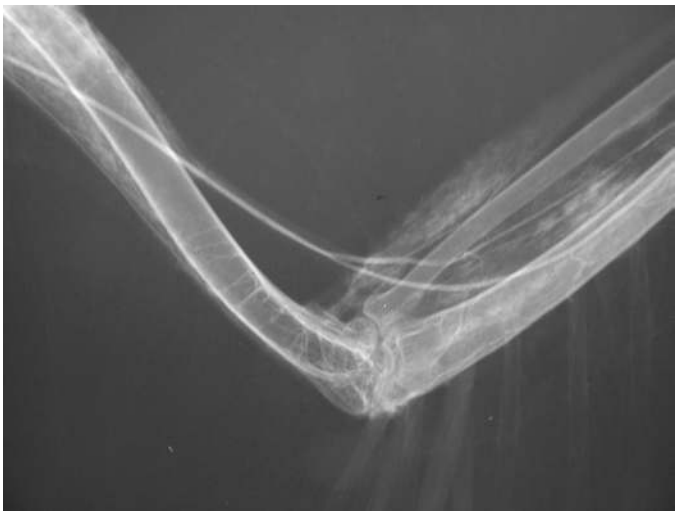


FIGURE 6-116 Angiography of the patagium of a Eurasian buzzard (*Buteo buteo*). The radiograph was obtained using 100 kVp, 300 mA, and 5/10 s. (Courtesy M. Delogu.)

Angiography (Fig. 6-116)

Angiography could be an important diagnostic tool for the detection of cardiovascular disease in birds. Angiography is useful for the assessment of heart size and function in those cases that cannot be adequately diagnosed using echocardiography (Krautwald-Junghanns *et al.*, 2011). It has been used in the diagnosis of aneurysm of the right coronary artery in a white cockatoo (*Cacatua alba*) (Vink-Nooteboom *et al.*, 1998) and atherosclerosis of the aorta and brachiocephalic arteries in a severe macaw (*Ara severa*) (Phalen *et al.*, 1996). Fluorescein angiography was used to examine the vascular tunic and blood supply of the eyes of various raptors (Korbel *et al.*, 2000). Angiography is indicated when no suitable coupling sites are available for the ultrasonographic transponder (Krautwald-Junghanns *et al.*, 2011).

Angiography should always be done under general anesthesia. The iodine-based contrast agent (e.g., iopamidol, 250 mg iodine/mL solution) at a dose of 2 to 4 mg/kg bodyweight should be slowly injected intravenously into the jugular or basilic vein (Krautwald-Junghanns *et al.*, 2011).

Myelography

Abnormalities that can be detected by myelography include spinal cord compression, spinal trauma, or space-occupying masses. Patients must be anesthetized for this procedure. A 25-gauge spinal needle is carefully inserted at the thoracosynsacral junction and 0.8 to 1.2 mL/kg of nonionic iodinated material is injected into the subarachnoid space (Harr *et al.*, 1997). Alternatively, contrast material can be injected directly into the cerebellar medullary cistern (McMillan, 1994). Immediately after the injection, the patient should be held in the upright position for 5 minutes before the myelogram can be performed (Krautwald-Junghanns *et al.*, 2011). In practice, myelographic investigations in birds are rarely attempted because of the high degree of risk with the procedure. Currently, magnetic resonance imaging (MRI) is a better diagnostic method available to diagnose spinal injuries (Krautwald-Junghanns *et al.*, 2011).

RADIOGRAPHIC INTERPRETATION

A systematic approach to film interpretation is important to reach a correct diagnosis. In evaluating films do not just focus on the most obvious lesion and perhaps overlook more subtle changes. A useful technique is to first assess the film for overall quality from a technical viewpoint, then work sequentially through each body system. The authors use an organ-by-organ system approach, proceeding from cranial to caudal, evaluating the head and neck, skeletal system, respiratory, cardiovascular, GI, other coelomic organs, and genitourinary systems.

Silverman and Tell (2009) recommends the following analysis:

- Skeletal: Skull, spine, pectoral girdle, pelvic girdle, wings, legs
- Cardiovascular: Heart, greater vessels
- Respiratory: Nasal sinuses, mouth, trachea, syrinx, lungs, air sacs
- GI: Mouth, crop, esophagus, proventriculus, ventriculus, intestines, cloaca
- Genitourinary: Pelvic region, kidneys, abdomen, cloaca
- Accessory organs: Liver, spleen

The radiographs are scrutinized according to the following criteria (McMillan, 1994):

- Size of the organ
- Density of organs and parts of organs
- Structure of organs
- Evaluation of the contents of the GI tract

INTERPRETATION OF RADIOGRAPHIC FINDINGS

Abnormal radiographic findings and their indications are given in Table 6-32. Figs. 6-117 to 6-128 illustrate some abnormal radiologic findings.

Text continued on p. 147

TABLE 6-32 Abnormal Radiographic Findings

Radiologic Signs	Indications
RESPIRATORY SYSTEM	
Increased radiodensity of the tracheal lumen (tracheal masses)	Hypovitaminosis A Bacterial infection (Pseudomoniasis) Mycotic infection (Aspergillosis) Parasitic infection (Trichomonosis) Pox virus infection Foreign bodies (seeds) Endotracheal tube injury
Displacement or abnormal position of the trachea	Presence of abnormal masses in the ventral cervical area
Increased radiodensity of the tracheal rings or wall of the tracheobronchial syrinx	Calcification of the trachea or tracheobronchial syrinx in older birds
Mottled tracheobronchial syrinx shadow with overdistention of the abdominal air sacs (air trapping)	Stenosis caused by a mycotic granuloma on the tracheobronchial syrinx, pseudomoniasis, or trichomonosis
Overdistention of the axillary portion of the clavicular air sacs	Stenosis of the lower respiratory tract
Homogeneous increased radiodensity of the lung field	Bacterial pneumonia
Nonhomogeneous increased radiodensity of the lung field	Mycotic pneumonia
Irregular, focal dense areas in the lungs	Mycotic, mycobacterial granuloma
Increased radiodensity in the heart-lung area often concentrated around the main bronchus	Chronic bronchitis, chronic bronchopneumonia
Increased radiodensity and rounding of the caudal lung field	Congestion of the caudal part of the lung in chronic disease
Thickening of the air sac walls (cavern formation)	Chronic mycotic infection
Increased radiodensity of the air sac walls	Caused by crystalline deposits or calcification of the air sac walls
Homogeneous increased radiodensity of the thoracic and abdominal air sacs	Fat deposits on the air sacs, air sacculitis of uncertain origin (e.g., bacterial, viral, chlamydial, or mycotic infections)
Nonhomogeneous increased radiodensity of the thoracic and abdominal air sacs	Chronic mycotic air sacculitis
Solitary or multiple, focal increased radiodensities in the thoracic and abdominal air sacs	Mycotic or mycobacterial granuloma, abscesses, neoplasia
Rounding of the caudal parts of the abdominal air sacs	Chronic air sacculitis
Compression of the thoracic and abdominal air sac field Overall homogeneous "ground glass" increase in density of both thoracic and abdominal cavities	Seen as secondary to mass lesions in the caudal coelomic cavity (distention of the alimentary tract, neoplasms or egg binding) Peritonitis Ascites

TABLE 6-32 Abnormal Radiographic Findings—cont'd

Radiologic Signs	Indications
GASTROINTESTINAL SYSTEM	
Thickening of the wall of the esophagus/crop and proventriculus	Vitamin A deficiency (often associated with an enlarged kidney shadow) Chronic inflammation caused by *Candida* spp. infection or a worm infection
Distention and/or impaction of the crop	Overeating of grit, improper hand-rearing technique, ingestion of foreign materials, secondary to enlarged thyroids, lead toxicity, obstruction in the proventriculus, ventriculus, and upper intestines
Dilatation of the gastrointestinal tract	Neurogenic infections, neurotoxic poisons, food impaction, ileus of the distal segments
Dilatation of the proventriculus	Yeast infection (e.g., *Macrorhabdus ornithogaster*), mycotic infection (e.g., *Candida* spp.), parasitic infection (e.g., *Porrocaecum* sp.), heavy metal toxicity, impaction, foreign body, normal baby bird
Thickening of the proventricular wall	Parasitic infection (e.g., tapeworms)
Severe dilatation of the proventriculus, retarded passage, thinning of the proventricular walls, and atrophy and deformation of the ventriculus	Proventricular dilatation disease, candidiasis
Gas-filled dilatation of the ventriculus	Lead toxicosis, Newcastle disease
Thickening of the wall of the ventriculus	Newcastle disease
Increased density, gas filling, and distention of the ventriculus	Parasitic infection Bacterial infection
Gas-filled ventriculus	Normal in game birds and some waterfowls
Excessive grit in the ventriculus and intestines	Deficiency disease or disturbance in the crop, ventriculus, or intestines
Heavy metal particles that are visible as radiopaque foreign bodies in the ventriculus	Lead shot, paint flakes, wire
Dorsocranial or dorsocaudal displacement of the ventriculus	Enlarged liver
Ventrocranial or ventrocaudal displacement of the ventriculus	Enlarged kidney, spleen, or gonad
Ventrocranial displacement of the ventriculus	Enlarged intestinal loops, egg in the oviduct, ovarian cysts
Gas-filled intestines	Bacterial infection; functional ileus; luminal or extraluminal mass obstruction; aerophagia secondary to severe respiratory distress, heavy metal toxicity, or gas anesthesia
Dilatation of the duodenal loop or any other loop of the intestine with increased radiodensity	Massive prepatent worm infection, bacterial or mycotic infection, pancreatitis, neoplasia, luminal or extraluminal mass obstruction
Dilatation of the cloaca	Cloacitis, neoplasms, cloacolith, proventricular dilatation disease, retained soft shell egg, traumatic dilatation, idiopathic dilatation
LIVER AND SPLEEN	
Decreased hepatic radiopacity	Hepatic lipidosis
Focal irregularities of the hepatic contour	Granulomatous lesions
Reduced size of the liver shadow (microhepatica). The liver shadow is separated from the heart in the hourglass shadow	Generalised emaciation, poor nutrition, pesticide toxicity, may occur normally in macaws
Enlarged liver shadow	*Chlamydia psittaci* infection, Pacheco disease, herpes virus hepatitis, other viral diseases, neoplasia, metabolic diseases, parasitic diseases, intoxication
Ground-glass appearance of the entire coelomic cavity. The lungs and air sacs are compressed (ascites)	Liver cirrhosis, hemochromatosis, neoplasia, congestive heart failure, viral infections, bacterial endocarditis and myocarditis
Severe spleen enlargement	*Chlamydia psittaci* infection (accompanied with air sacculitis and lung consolidation), tuberculosis, yersiniosis, neoplasia
Enlarged spleen shadow with enlargement of the liver and kidneys	Tuberculosis, viral diseases

Continued

TABLE 6-32 Abnormal Radiographic Findings—cont'd

Radiologic Signs	Indications
UROGENITAL SYSTEM	
Radiodense crystalline deposits in the kidneys	Renal gout, dehydration, chronic bacterial infection
Enlarged kidney shadows with or without increased density	Neoplasia, cysts, *Chlamydia psittaci* infection, bacterial diseases, metabolic diseases, postrenal obstruction, heavy metal toxicity, vitamin A deficiency
Enlargement of the cranial pole of the kidney	Kidney enlargement as described in previous box, adrenal enlargement, gonadal enlargement
Increased density without enlargement of the kidneys	Gout, dehydration, vitamin A deficiency
Egg in the oviduct	Egg binding if combined with specific symptoms (weakness, pressing, dyspnea)
An area of increased density caused by fragmented egg shell dorsal and caudal to the intestinal mass	Salpingitis
Diffused increased in radiodensity of the caudal coelomic cavity and no differentiation of various organs is possible (abdominal effusion)	Ovarian neoplasia, egg yolk–related peritonitis,
CARDIOVASCULAR SYSTEM	
Increased apex to base dimension, enlarged vascular structures, prominence to the left atrial segment, abnormal shape of the heart shadow (cardiomegaly)	Valvular disease, endocarditis, chronic anemia, compression from extrinsic masses, hemochromatosis
Globoid enlargement of the heart shadow	Pericardial effusion that may be caused by *Chlamydia psittaci* infection, polyomavirus, tuberculosis, polyomavirus, sarcocystosis, or neoplasia
Enlargement and/or increased radiodensity of the heart shadow	Pericarditis, epicarditis, marked fatty deposition
Alteration of the heart's shape and contour (dilatation of one chamber)	Maybe genetic abnormality
Not well-defined shadow of the cranial end of the heart	Bronchitis, bronchopneumonia
Reduced size of the heart shadow (microcardia). May have radiolucent gap between heart and liver	Hypovolemia, nutritional inadequacy
Calcification of major vessels and lung fields	Arteriosclerosis (in very old psittacines and birds of prey)
SKELETAL SYSTEM	
Increased radiodensity of the infraorbital sinus. Osteolytic changes may be present on the surrounding bones	Rhinitis, sinusitis
Decreased skeletal radiodensity, deformities of the long bones, ribs and spine, and/or fractures of the metaphyses	Metabolic bone disease (osteoporosis, osteomalacia, rickets, fibrous osteodystrophy, nutritional secondary hyperparathyroidism); twisting and bending deformities of the long bones
Multiple osteolytic and sclerotic changes in the medullary cavity of the long bones	Mycobacterial infections
Homogeneous increased in medullary bone density (polyostotic hyperostosis)	In female birds before egg production
Irregular increased in medullary bone density	Pathologic high estrogen level associated with laminated eggs, gonadal tumors, or cysts
Increased bone radiodensity associated with periosteal proliferation and swelling of surrounding soft tissue	Osteomyelitis
Collapsed joint space, periarticular proliferation of bone, and soft tissue swelling	Arthritis
Arthritic and osteolytic changes of the digits, joints, and tarsometatarsus	Septic bumblefoot

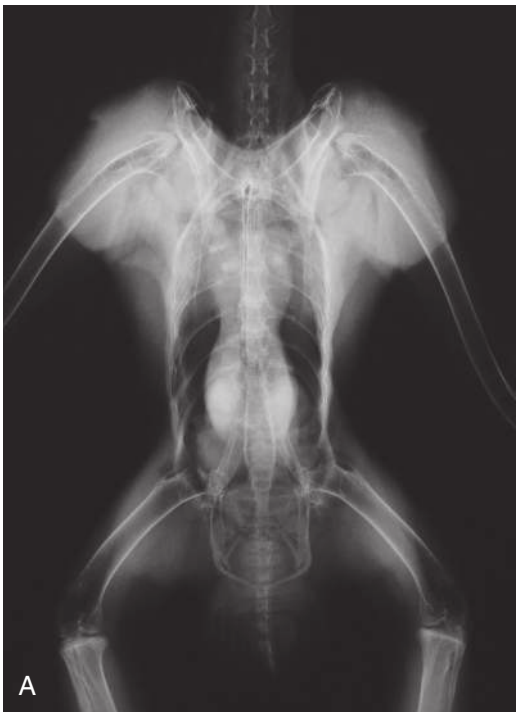
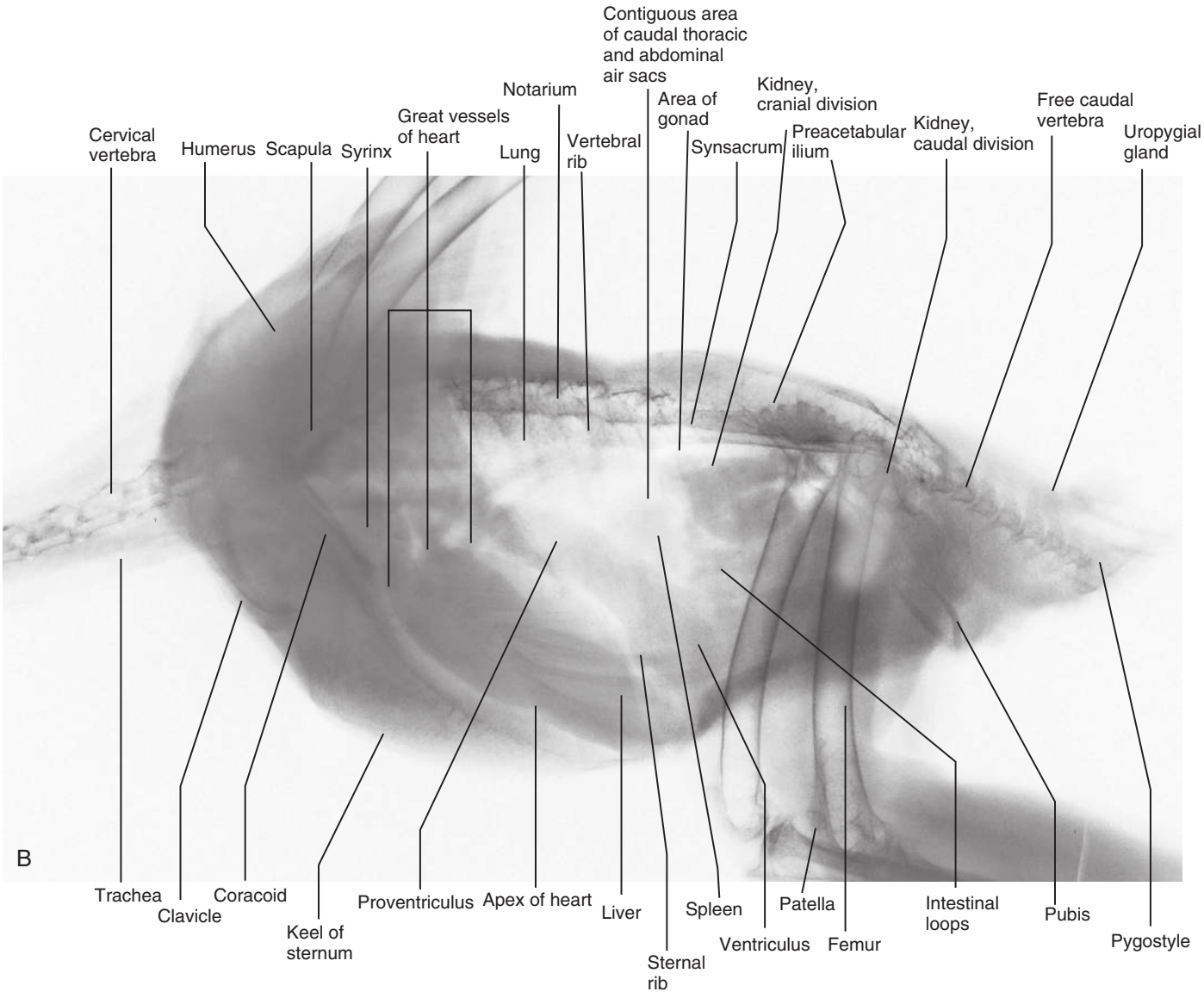


FIGURE 6-117 **(A),** Ventrodorsal radiograph of the body of a clinically normal red kite (*Milvus milvus*). **(B),** Digitized picture of the same bird to illustrate the different body parts.



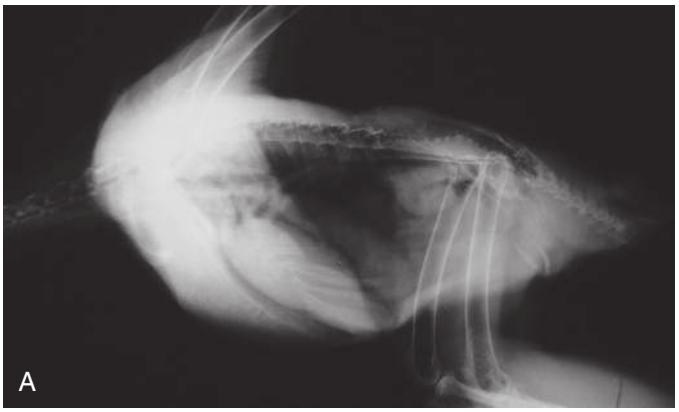
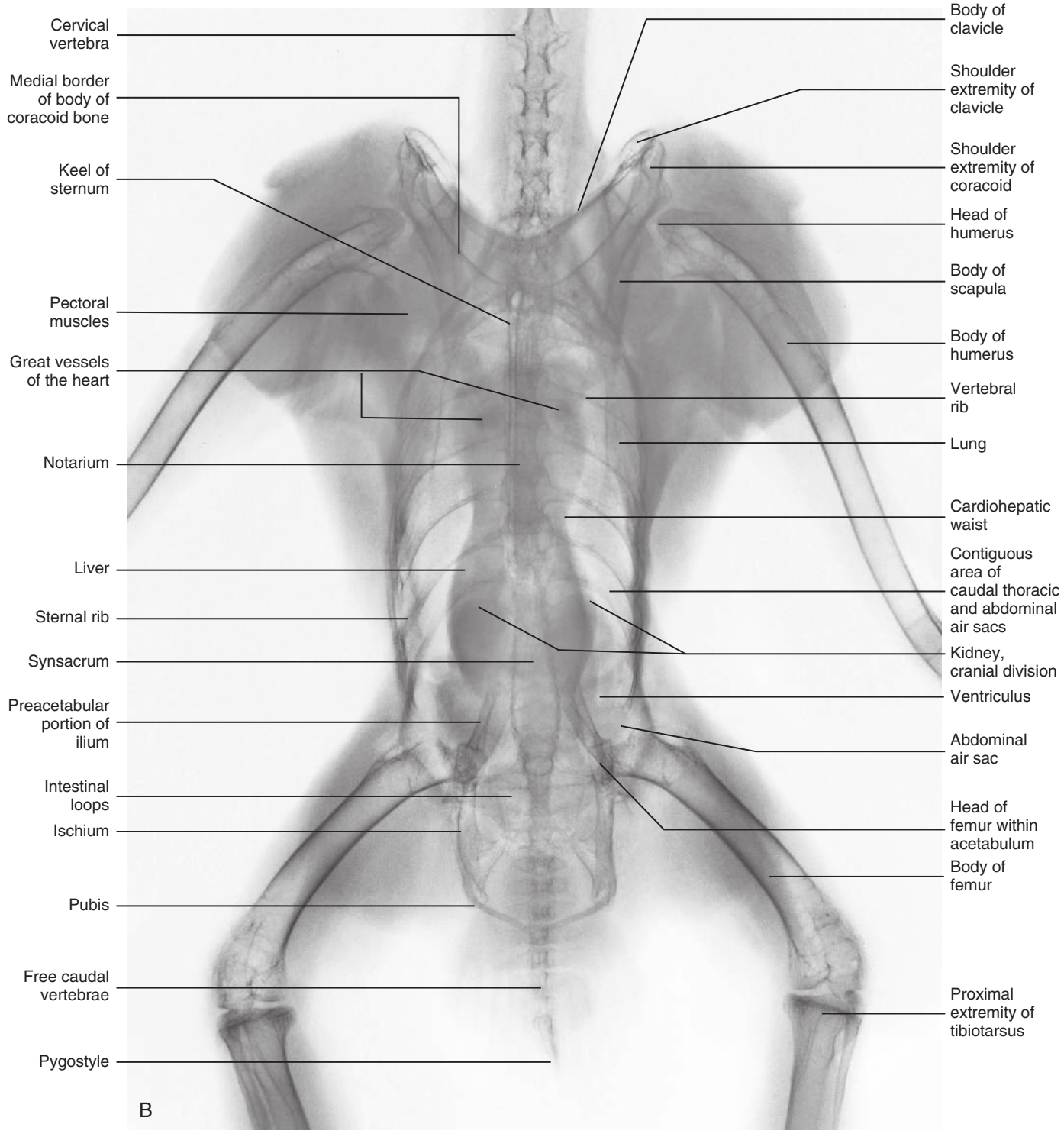


FIGURE 6-118 **(A),** Lateral (Le-Rt) radiograph of the body of a clinically normal red kite (*Milvus milvus*). **(B),** Digitized picture of the same bird to illustrate the different body parts.



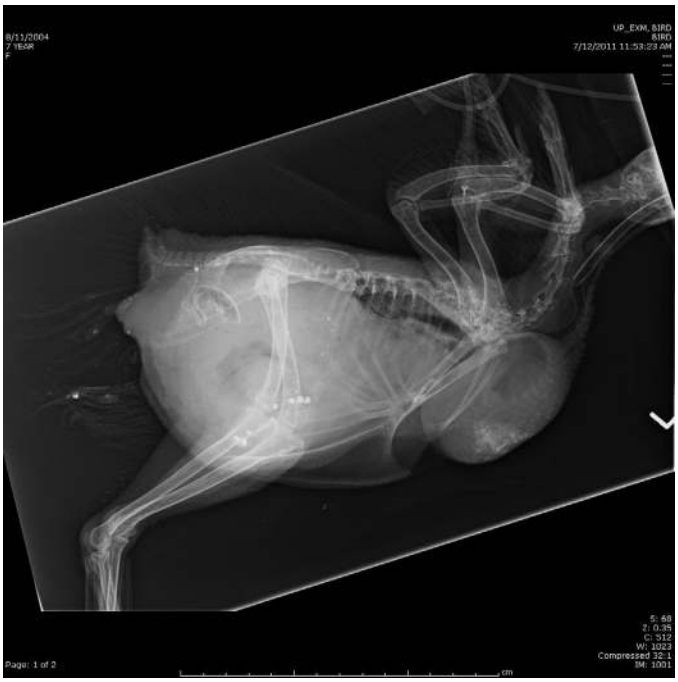


FIGURE 6-119 Impacted crop content in a domestic chicken (*Gallus gallus*) associated with systemic illness resulting from a retained and collapsed egg in the oviduct. (Courtesy Robert Doneley.)

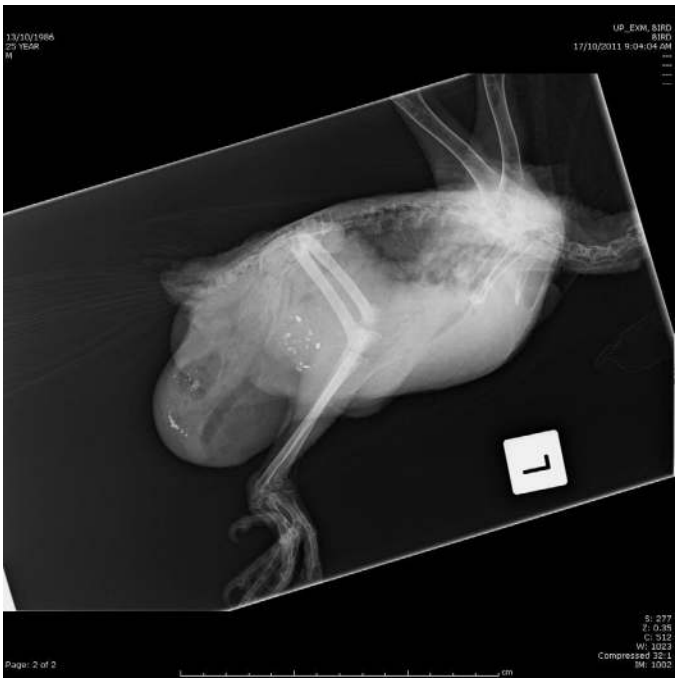


FIGURE 6-120 Hernia in a cockatoo. This medical condition is relatively common in overweight, unfit, reproductively active hens. (Courtesy Robert Doneley.)

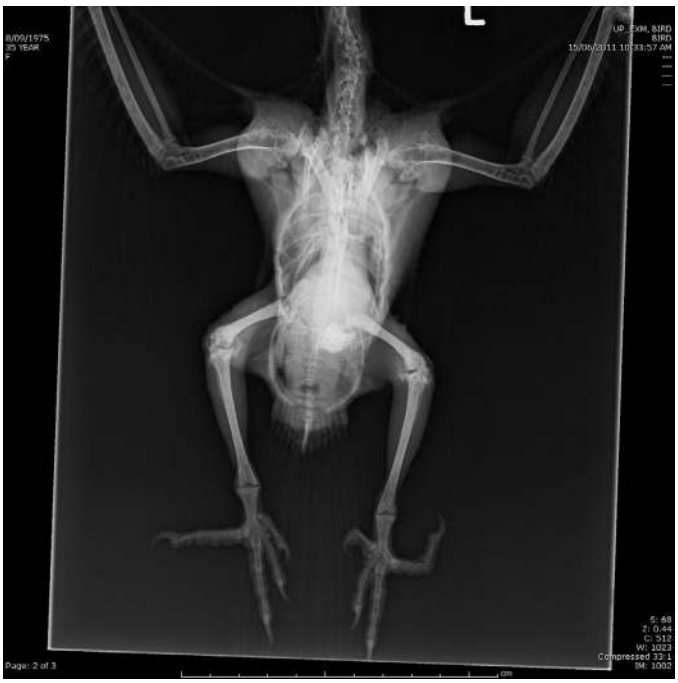


FIGURE 6-121 Severe bilateral arthritis in the knee joints of a galah cockatoo (*Eolophus roseicapilla*). (Courtesy Robert Doneley.)

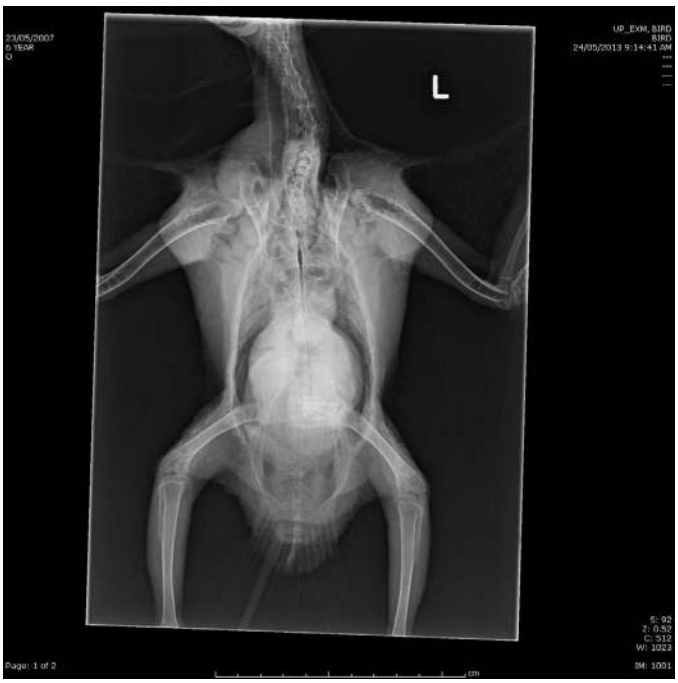


FIGURE 6-122 Pronounced hepatomegaly in a cockatoo. (Courtesy Robert Doneley.)

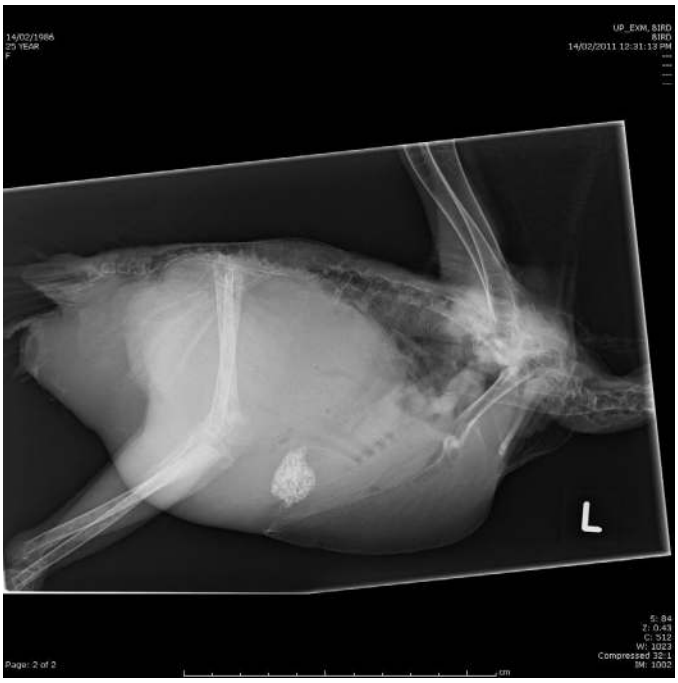


FIGURE 6-123 Diffused coelomitis in a cockatoo. (Courtesy Robert Doneley.)

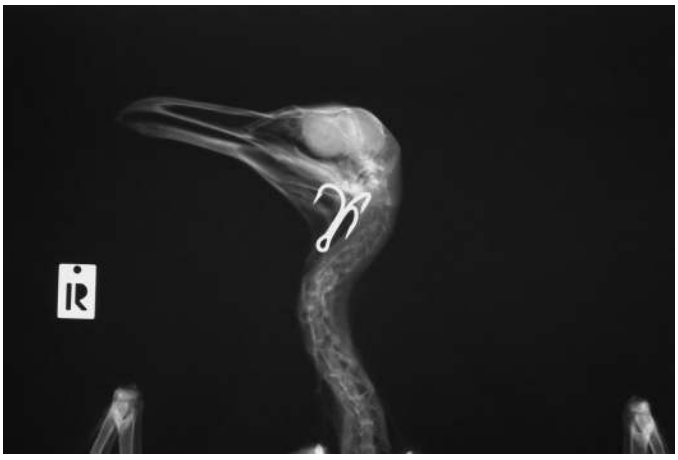


FIGURE 6-124 Ingested three-pronged fishing hook in a sea gull. Gulls and other sea shore bird species (e.g., pelicans, cormorants) will eat unattended or discarded baited fishing hooks left behind around piers and marinas by irresponsible fishermen. (Courtesy Dr. Jaime Samour.)

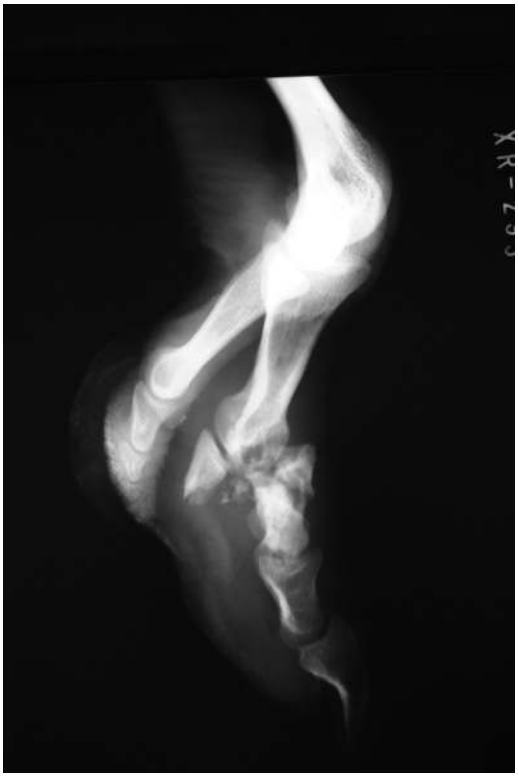


FIGURE 6-125 Fracture of the second phalanx of a digit in a Masai ostrich (*Struthio camelus masaicus*) with multiple fragments. (Courtesy Dr. Jaime Samour.)

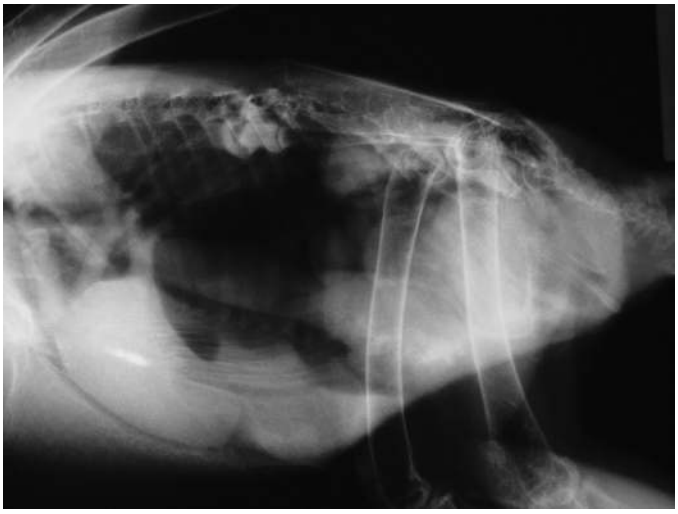


FIGURE 6-126 Osteoarthritis of the free thoracic vertebra in a gyr falcon (*Falco rusticolus*). These types of radiologic findings are typical of collision injuries resulting in compression of the vertebral column at this level. Commonly this leads to paresia or paralysis of the hind limbs. (Courtesy Dr. Jaime Samour.)

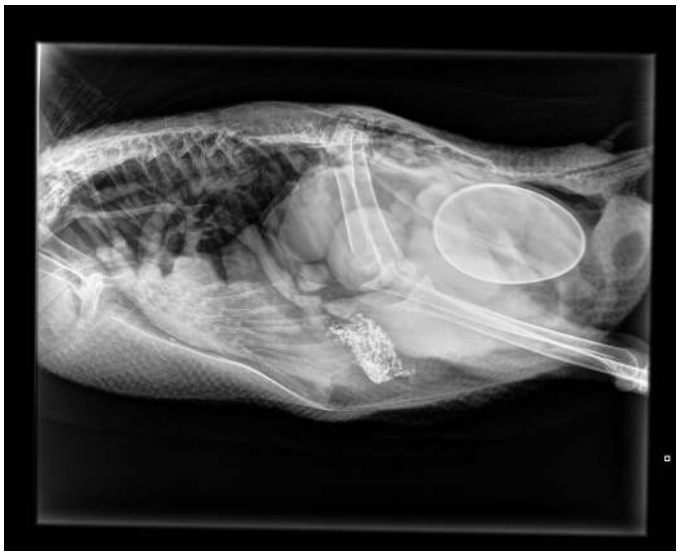


FIGURE 6-127 Ovarian follicles at different stages of development and a fully formed egg in a domestic goose (*Anser anser domesticus*). Laterolateral view. (Courtesy the Clinic for Birds, Reptiles, Amphibians and Fish, JLU Giessen, Germany.)

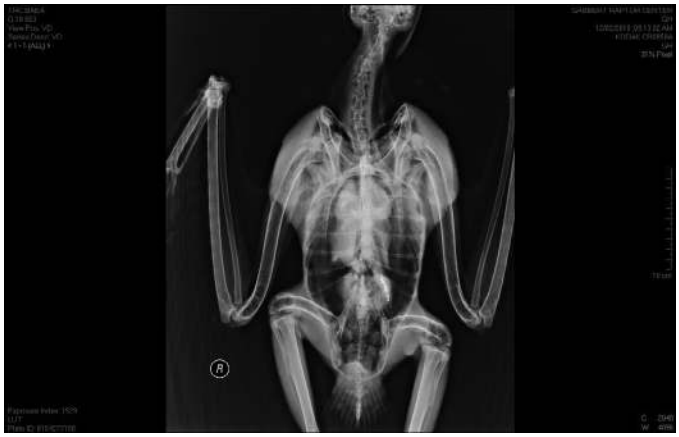


FIGURE 6-128 Metallic foreign body in the gastrointestinal (GI) tract of a bald eagle (*Haliaetus leucocephalus*). (Courtesy The Raptor Center, University of Minnesota, USA.)

REFERENCES

Harr KE, Kollias GV, Rendano V, et al: A myelographic technique for avian species, *Vet Radiol Ultrasound* 38:187–192, 1997.

Helmer P: Advances in diagnostic imaging. In Harrison GJ, Lightfoot TL, editors: *Clinical avian medicine* (vol 2), Palm Beach, 2006, Spix Publishing, Inc., pp 653–659.

Korbel RT, Nell B, Redig PT, et al: Video fluorescein angiography in the eyes of various raptors and mammals. *Proceedings Association of Avian Veterinarians*, 2000, Portland, Oregon, pp 89–95.

Krautwald-Junghanns ME: Aids to diagnosis. In Coles BH, editor: *Essentials of avian medicine and surgery*, ed 3, Oxford, 2007, Blackwell Publishing Ltd., pp 56–102.

Krautwald-Junghanns ME, Pees M: Imaging techniques. In Tully TN, Dorrestein GM, Jones AK, editors: *Handbook of avian medicine*, ed 2, Philadelphia, 2009, Saunders Elsevier, pp 85–100.

Krautwald-Junghanns ME, Pees M, Reese S, et al: *Diagnostic imaging of exotic pets*, Hannover, 2011, Schlütersche.

Krautwald-Junghanns ME, Trinkaus K: Imaging techniques. In Tully TN, Lawton MPC, Dorrestein GM, editors: *Avian medicine*, Oxford, 2000, Butterworth-Heinemann, pp 52–73.

Lennox AM, Crosta L: The effects of isoflurane anaesthesia on gastrointestinal transit time. *Proceedings European Association of Avian Veterinarians*, 2005, Arles, France, pp 207–210.

McMillan MC: Imaging techniques. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, 1994, Wingers Publishing Inc., pp 246–326.

Pees M: Radiography. In Chitty J, Lierz M, editors: *Manual of raptors, pigeons and passerine birds*, Gloucester, 2008, British Small Animal Veterinary Association, pp 114–120.

Phalen DN, Hays HB, Filippich LJ, et al: Heart failure in a macaw with atherosclerosis of the aorta and brachiocephalic arteries, *J Am Vet Med Assoc* 209(8):1435–1440, 1996.

Romagnano A, Love NE: Imaging interpretation. In Olsen GH, Orosz SE, editors: *Manual of avian medicine*, St. Louis, 2000, Mosby, pp 391–423.

Samour JH, Naldo JL: *Anatomical and clinical radiology of birds of prey: including interactive advanced anatomical imaging*, Philadelphia, 2007, Saunders Elsevier, pp 3–7.

Sherrill J, Ware LH, Lynch WE, et al: Contrast radiography with positive-pressure insufflation in northern pintails (*Anas acuta*), *J Avian Med Surg* 15(3):178–186, 2001.

Silverman S, Tell LA: *Radiology of birds: an atlas of normal anatomy and positioning*, St. Louis, 2009, Saunders Elsevier, pp 4–14.

Smith BJ, Smith SA: Radiology. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, 1997, WB Saunders, pp 170–199.

Tell L, Silverman S, Wisner E: Imaging techniques for evaluating the head of birds, reptiles and small exotic mammals, *Exotic DVM* 5(2):31–37, 2003.

Vink-Nooteboom M, Schoemaker NJ, Kik MJ, et al: Clinical diagnosis of aneurysm of the right coronary artery in a white cockatoo (*Cacatua alba*), *J Small Anim Pract* 39(11):533–537, 1998.

IMAGE-INTENSIFIED FLUOROSCOPY

Bárbara Arca-Ruibal

Fluoroscopy is an imaging technique that uses X-rays to produce real-time video images. In fluoroscopy, after the X-rays pass through the patient, instead of being captured by film, they go through an image intensifier and are converted into light. This light is then captured by a television camera and displayed on a video monitor. As opposed to traditional radiography, fluoroscope images of radiodense tissues or objects appear black and radiolucent ones appear white.

FLUOROSCOPY EQUIPMENT AND ADVANTAGES

Fluoroscopy units used in avian practice consist of two components: a C-arm and a video processing unit. The C-arm houses the X-ray generator, the X-ray detector, the image intensifier, and the video camera. The C-arm is the mobile part that can be positioned around the patient. The video processing unit allows for the manipulation of the image according to different settings. Images of different qualities can be viewed by adjusting the number of video frames averaged (noise suppression). The lowest noise suppression settings are desirable for cinematic viewing, which is important in motion studies. Higher noise suppression settings are required for high quality still images. Fig. 6-129 shows the difference in quality between two noise suppression settings.

Fluoroscopy systems work at greatly reduced levels of radiation exposure and scatter. However, standard radiology safety measures should be followed and operators should always wear lead aprons.

Even though fluoroscopy provides less-detailed images it has many advantages over conventional radiography. Fluoroscopy images can be reviewed in situ, and certain dynamic body processes can be observed in "real-time." Fluoroscopy is also minimally invasive because physical restraint and anesthesia of the patient are not necessary. Therefore it can be used to evaluate the function of the GI tract with food and with circulatory and respiratory compromised patients.

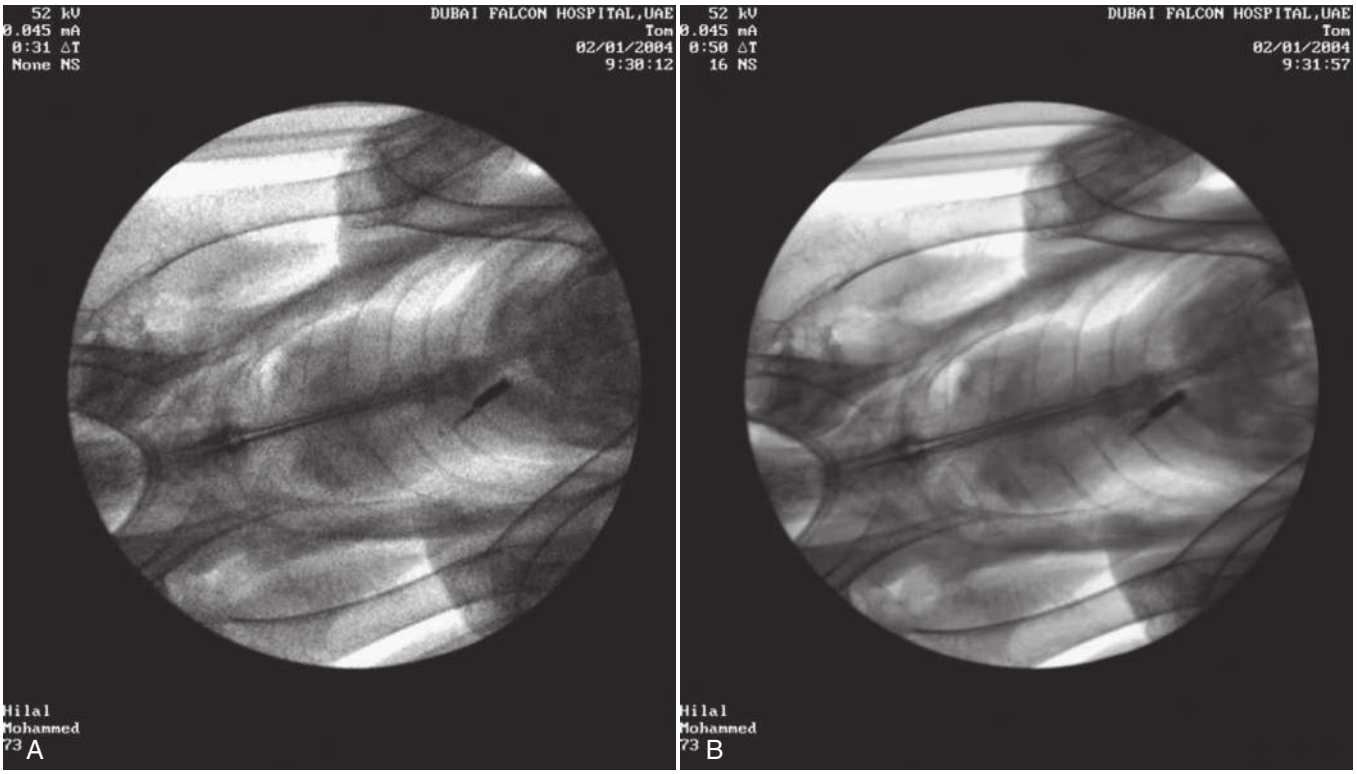


FIGURE 6-129 Falcon, anteroposterior projection. Hepatomegaly. In the sequence, it is possible to see the improvement of image quality using the noise suppression feature. Note the grainy quality of the image with no noise suppression **(A)** and the improved quality using noise suppression **(B)**.

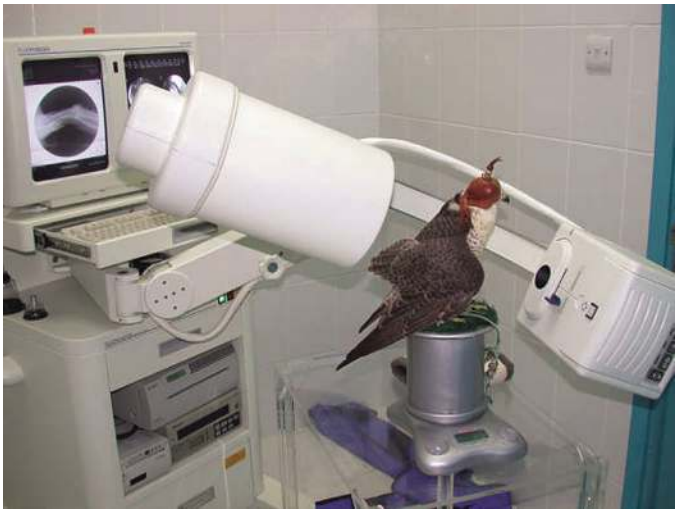


FIGURE 6-130 Falcon with a dropped wing standing on a perch for fluoroscopy examination. Trained falcons that are hooded will quietly stand on a perch while fluoroscopy investigations are undertaken. Fluoroscopy is useful in the preliminary screening of cases without using anesthesia.

Avian patients can be placed in cardboard or plastic boxes, on perches, or on acrylic plastic tables and positioned within the C-arm to reduce stress during examinations (Fig. 6-130). Fluoroscopic examinations can be recorded and digitally stored for review at a later stage.

The main barrier for widespread use of fluoroscopy has been the cost of new equipment; however, over the last few years equipment has become more affordable through refurbished or retired units from human medicine (Ford, 2006).

FLUOROSCOPY APPLICATIONS IN AVIAN MEDICINE

Investigation of the Motility of the Gastrointestinal Tract

Fluoroscopy is considered the best technique to monitor the GI motility in birds (McMillan, 1994; Beaufrère *et al.*, 2010a). Examples of its use in birds include screening and evaluation of proventricular dilation disease in psittacines (Storm and Greenwood, 1993; Degernes *et al.*, 1999); contrast studies of the psittacine GI tract (Taylor *et al.*, 1999; Vink-Nooteboom *et al.*, 2003); location of string foreign bodies in a juvenile umbrella cockatoo (*Cacatua alba*) (Oglesbee and Sreinohrt, 2001); diagnosis of megacloaca in a Moluccan cockatoo (*Cacatua moluccensis*) (Graham *et al.*, 2004); and investigation of proventricular obstruction in an eclectus parrot (*Eclectus roratus*) (De Voe *et al.*, 2003). It has also been used as a research tool to investigate the effects of metoclopramide on GI tract motility in Hispaniolan parrots (*Amazona ventralis*) (Bowman *et al.*, 2002).

Considering the significant species and individual variation in transit times of the GI tract in birds, it is important to be aware of normal transit times for a given species. Normal reference ranges for GI motility in Hispaniolan parrots have been established by Beaufrère *et al.*, (2010a), as well as a measurement method of the proventriculus and ventriculus size by using femoral heads units as a standard reference unit. GI motility patterns and transit times have also been studied in American kestrels (*Falco sparverius*) (Duke *et al.*, 1997) and in great horned owls (*Bubo virginianus*) (Rhoades and Duke, 1977). In falcons a wide range of transit times has been observed even in healthy individuals (Garcia-Martinez *et al.*, 2007), with some of the healthy birds having a delayed emptying of the crop.

Fig. 6-131 presents a sequence of images from a falcon GI tract study before and after barium administration at different times.

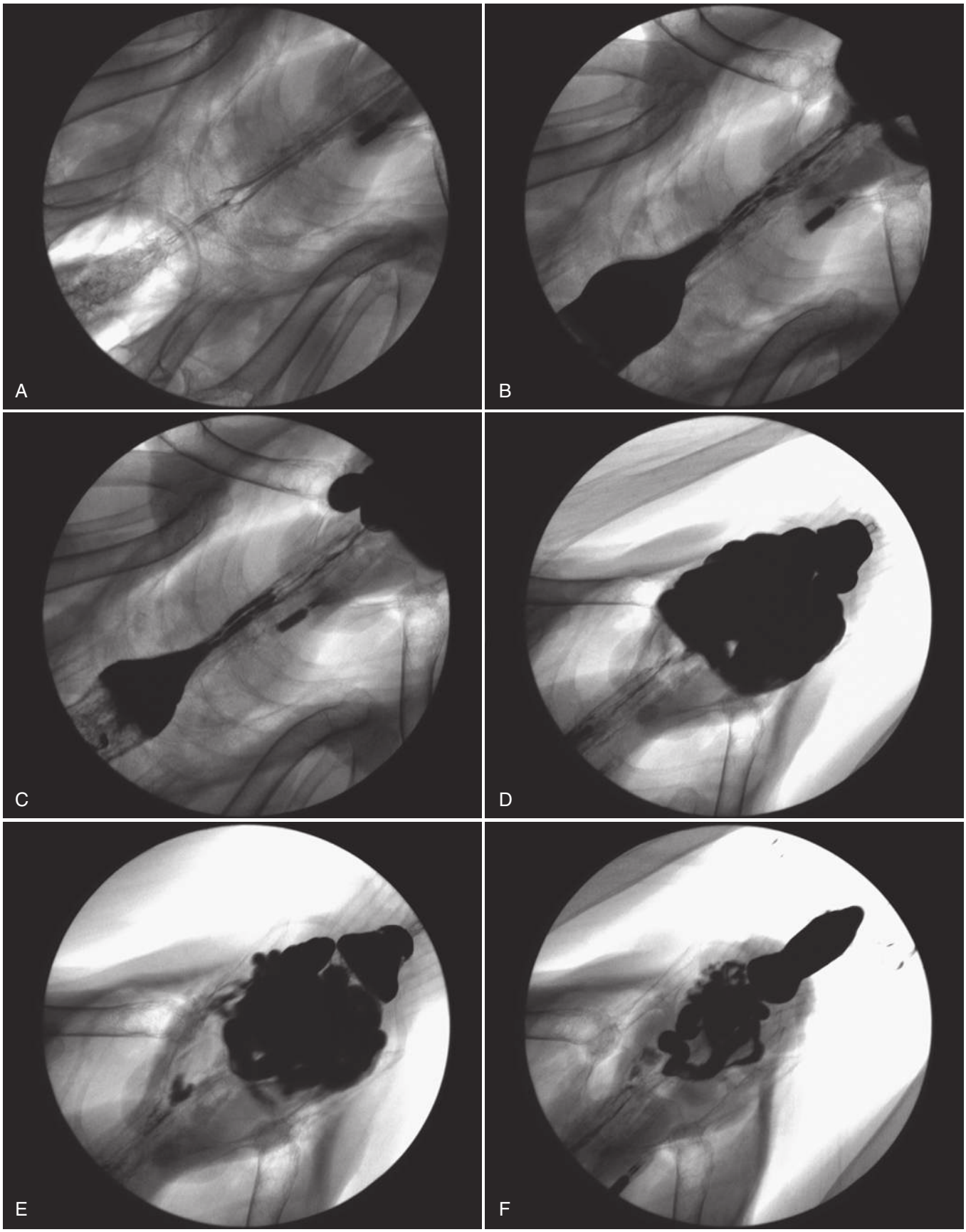


FIGURE 6-131 Series of images before and after administration of barium in a falcon: **(A),** Before administration; **(B),** At 1 to 3 minutes; **(C),** At 15 minutes; **(D),** At 30 minutes; **(E),** 1 hour; **(F),** 2 hours;

Continued

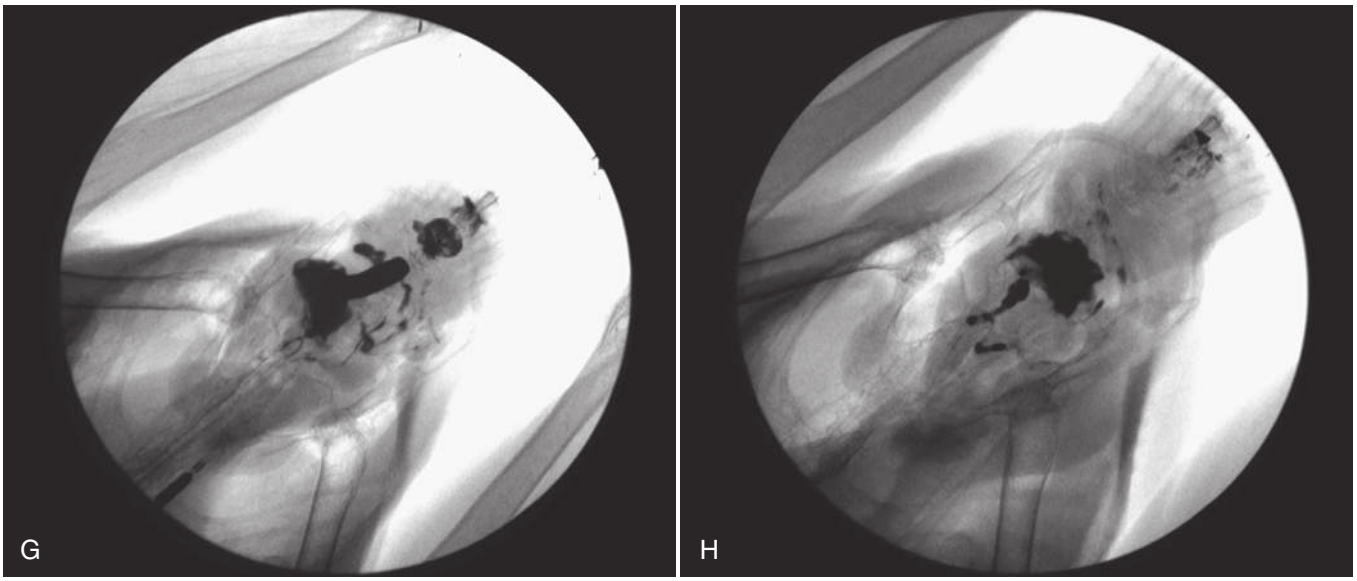


FIGURE 6-131, cont'd **(G),** 4 hours; **(H),** 8 hours after administration.

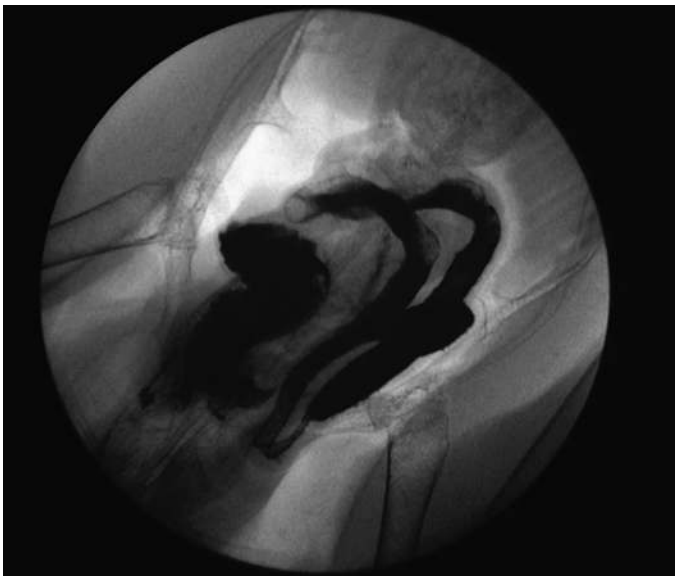


FIGURE 6-132 Houbara bustard, ventrodorsal projection. Gastrointestinal contrast with barium sulfate at 30 hours. In the houbara bustard contrast media is retained in the ceca from 6 to 30 hours onward.

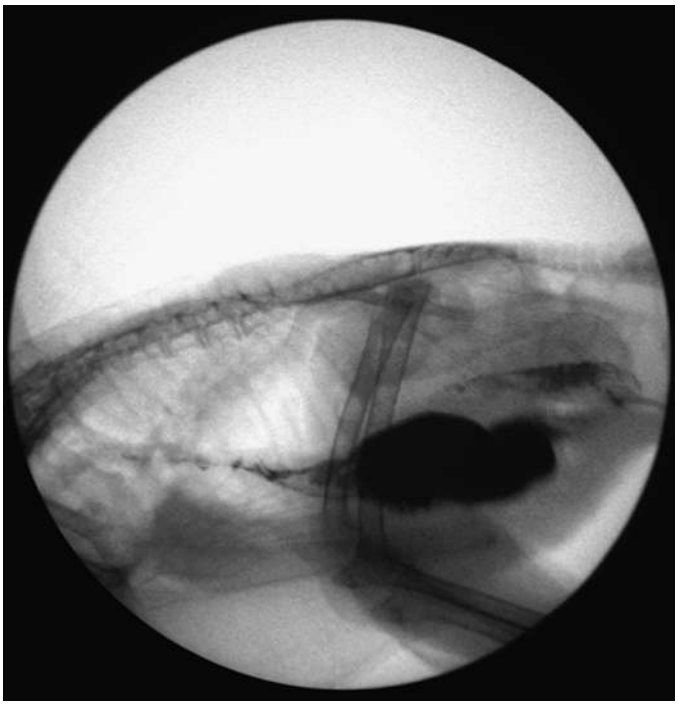


FIGURE 6-133 Stone curlew (*Burhinus oedicnemus*), lateral projection. Gastrointestinal contrast with barium sulfate. Stone curlews are susceptible to gastric candidiasis and delayed gastric transit time can be an indication of candidiasis in this species.

GI motility in birds is affected by other factors such as size, diet, stress, use of anesthetic drugs, environmental temperature, age, time of the day, medications, fasting, nutritional status, and manual restraint (McMillan, 1994; Denbow, 2000; Lennox *et al.*, 2002) that should be taken into consideration during GI fluoroscopy studies.

Fig. 6-132 shows retention of barium in the ceca of a houbara bustard (*Chlamydotis undulata macqueenii*) 30 hours after administration, a normal finding in this species. Fig. 6-133 shows barium in the GI tract in a stone curlew (*Burhinus oedicnemus*), a species that is susceptible to gastric candidiasis. In this species, a delayed GI transit time can be an indication of candidiasis.

Orthopedics

Orthopedics is another major application of fluoroscopy in avian medicine. Suspected fractures can be preliminarily diagnosed with the advantage of the patient not having to be restrained (see Fig. 6-130). Intraoperative imaging during orthopedic surgery to assist in the realignment of fractures and placement of orthopedic implants is another important fluoroscopy application (Fig. 6-134), in addition to its use for the monitoring of bone healing and to check the placement of intraosseous catheters (Fig. 6-135).

Cardiovascular Diseases

Fluoroscopic angiography has been used to aid in the diagnosis of cardiovascular diseases in birds; however, reports are still limited

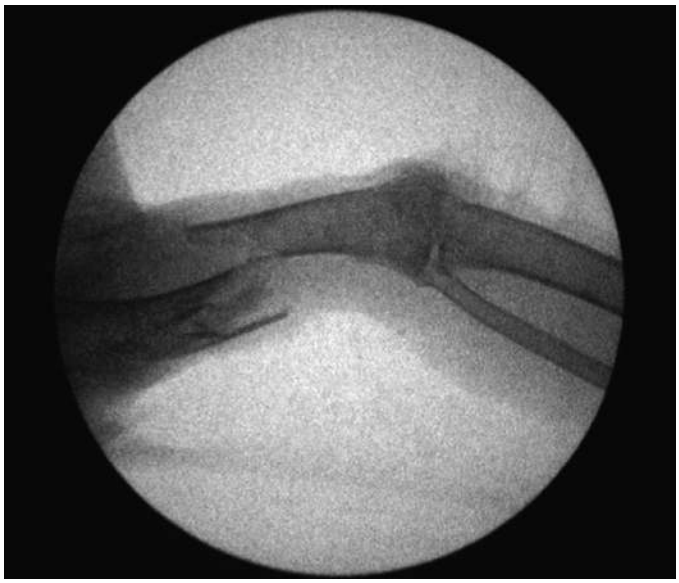


FIGURE 6-134 Falcon, anteroposterior projection. Fractured humerus.

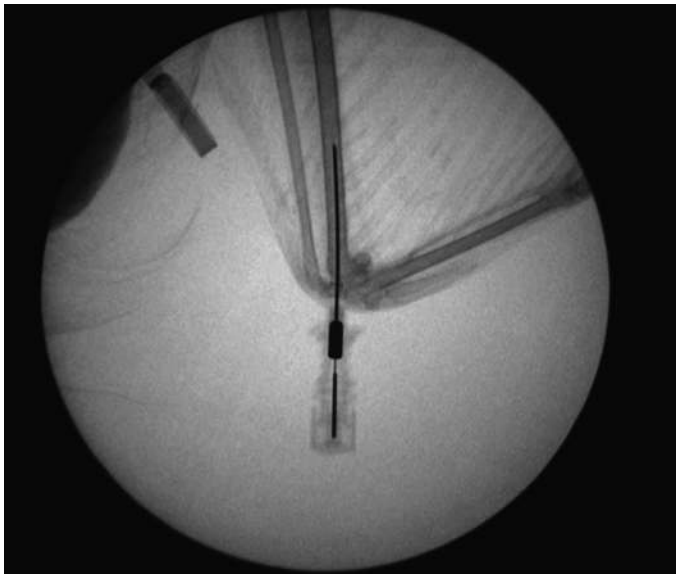


FIGURE 6-135 Fluoroscopy provides a very rapid way of checking the placement of intraosseous catheters.

(Beaufrère *et al.*, 2010b). Fluoroscopy angiography allows for the evaluation of the heart and vascular tree in real time after injection of a nonionic iodinated contrast agent. Digital subtraction angiography helps visualize arteries that are not seen by conventional angiography because of the size or density of the surrounding tissues. For a detailed description of both angiographic techniques in birds the reader is referred to Beaufrère *et al.* (2010b).

Other Applications

Fluoroscopy can be used in avian obstetrics, particularly in the investigation of dystocia and egg retention. Examples of other applications of fluoroscopy in avian medicine include the screening of birds for ventricular foreign bodies (Fig. 6-136); hepatomegaly (Fig. 6-137; see

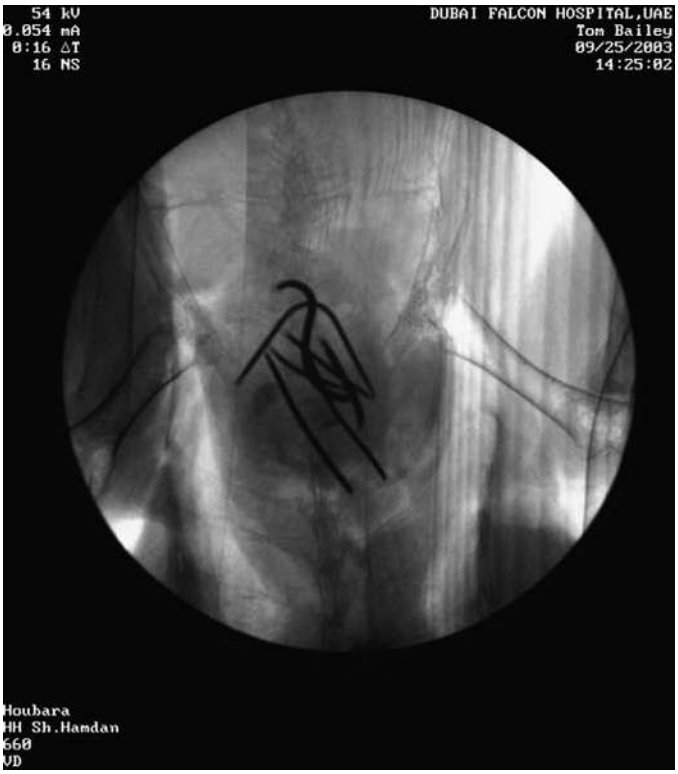


FIGURE 6-136 Houbara bustard (*Chlamydotis undulata*), anteroposterior projection. Radiopaque particles (wire) in the ventriculus.

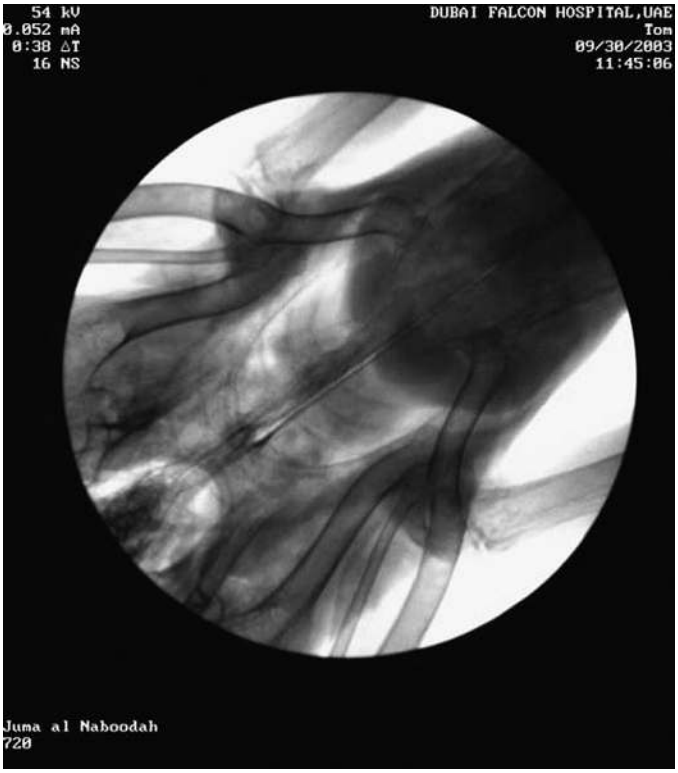


FIGURE 6-137 Falcon, anteroposterior projection. Hepatomegaly. Histopathology of liver biopsies confirmed amyloidosis.

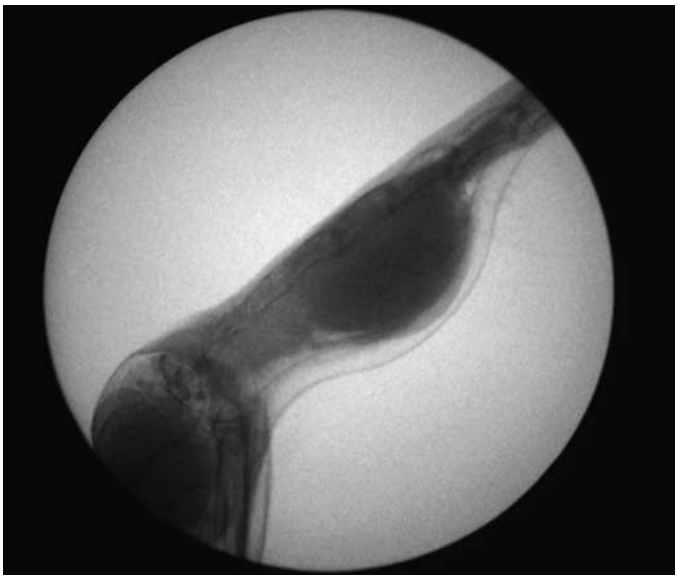


FIGURE 6-138 Houbara bustard, lateral projection. Soft tissue radiodensities in the esophagus because of *Trichomonas* sp. infection.

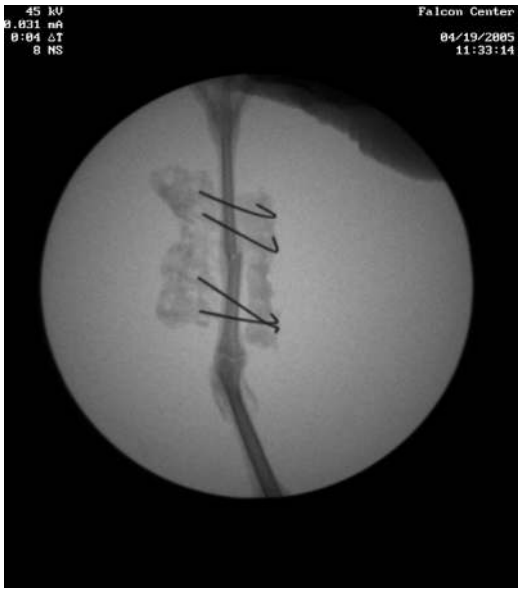


FIGURE 6-140 Stone curlew, anteroposterior projection. Repair of fractured tibiotarsus using type II external fixators.

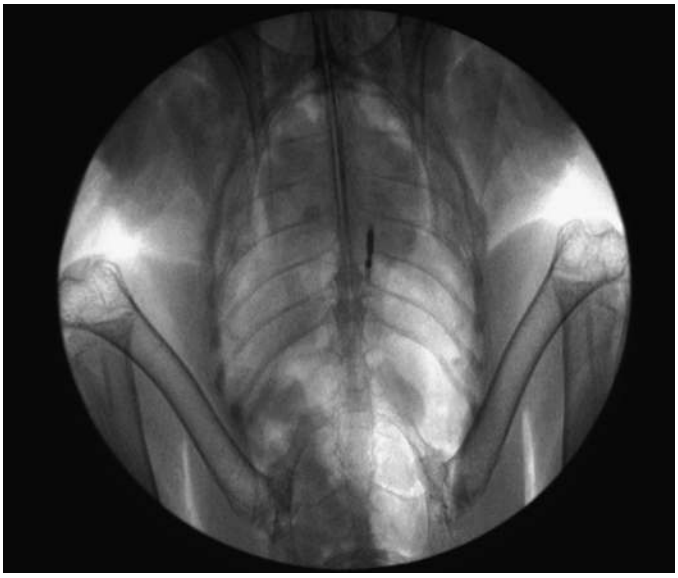


FIGURE 6-139 Falcon, anteroposterior projection. Asymmetric shadow over the air sacs; asymmetric irregular area of increased density in the lungs; and aspergillosis.

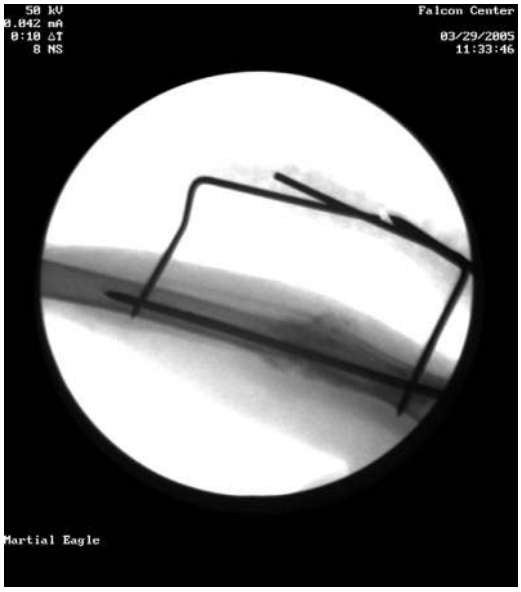


FIGURE 6-141 Fluoroscopy of the right tibiotarsus of a martial eagle 39 days after orthopedic surgery. Notice the presence of bridging callus.

also Fig. 6-129); esophageal granulomas (Fig. 6-138); and consolidated granulomas of the lower respiratory tract (Fig. 6-139). Fluoroscopy does not, however, provide sufficiently fine-resolution images for detecting early pathological changes in the lower respiratory tract (Figs. 6-140 and 6-141).

ACKNOWLEDGMENTS

The author would like to acknowledge the contribution to this section in the previous edition by Thomas A. Bailey, Antonio Di Somma, and Celia Garcia Martinez.

REFERENCES

Beaufrère H, Nevarez J, Taylor WM, et al: Fluoroscopic study of the normal gastrointestinal motility and measurements in the Hispaniolan Amazon parrot (*Amazona ventralis*), *Vet Radiol Ultrasound* 51:441–446, 2010a.

Beaufrère H, Pariaunt R, Rodriguez D, et al: Avian vascular imaging: a review, *J Avian Med Surg* 24:174–184, 2010b.

Bowman MR, Part JA, Ziegler LE, O'Brien R: Effects of metoclopramide on the gastrointestinal tract motility of Hispaniolan parrots (*Amazona ventralis*), *Proceedings of the American Association of Zoo Veterinarians* 1:117, 2002.

De Voe R, Degernes L, Karli K: Dysplastic koilin causing proventricular obstruction in an eclectus parrot (*Eclectus roratus*), *J Avian Med Surg* 17:27–32, 2003.
Degernes LA, Fisher PE, Diaz DE: Gastrointestinal scintigraphy in psittacines. *Proceedings of the Association of Avian Veterinarians*, 1999, New Orleans, pp 93–94.
Denbow DM: Gastrointestinal anatomy and physiology. In Whittow G, editor: *Sturkie's avian physiology*, San Diego, 2000, Academic Press, pp 299–326.
Duke GE, Reynhout J, Tereick AL, et al: Gastrointestinal morphology and motility in American kestrels receiving high or low fat diets, *Condor* 99:123–131, 1997.
Ford S: Surgical microscopy and fluoroscopy in avian practice, *J Exot Pet Med* 15:91–97, 2006.
Garcia-Martinez C, Bailey TA, Di Somma A: Radiography and image-intensified fluoroscopy of barium passage through the gastrointestinal tract of falcons. *Proceedings of the European Association of Avian Veterinarians*, 2007, Zurich, pp 508–511.
Graham JE, Tell LA, Lamm MG, Lowenstine LJ: Megacloaca in a Moluccan cockatoo (*Cacatua moluccensis*), *J Avian Med Surg* 18:41–49, 2004.
Lennox AM, Crosta L, Buerkle M: The effects of isoflurane anesthesia on gastrointestinal transit time. *Proceedings of the Association of Avian Veterinarians*, 2002, Monterey, pp 53–55.
McMillan MC: Imaging techniques. In Ritchie BW, Harrison GJ, Harrison LF, editors: *Avian medicine: principles and application*, Lake Worth, 1994, Wingers Publishing, pp 246–326.
Oglesbee B, Sreinohrt L: Gastrointestinal string foreign bodies in a juvenile umbrella cockatoo, *Compend Contin Educ Vet* 23:946–946, 2001.
Rhoades DD, Duke GE: Cineradiographic studies of gastric motility in the great horned owl (*Bubo virginianus*), *Condor* 79:328–334, 1977.
Storm J, Greenwood AG: Fluoroscopic investigation of the avian gastrointestinal tract, *J Avian Med Surg* 7:192, 1993.
Taylor M, Dobson H, Hunter B, et al: The functional diagnosis of avian gastrointestinal disease—an update, *J Avian Med Surg* 20:85, 1999.
Vink-Nooteboom M, Lumeij JT, Wolvekamp WT: Radiography and image-intensified fluoroscopy of barium passage through the gastrointestinal tract in six healthy Amazon parrots *(Amazona aestiva)*, *Vet Radiol Ultrasound* 44:43–48, 2003.

ULTRASONOGRAPHY

Maria Elisabeth Krautwald-Junghanns, Michael Pees

Although there are limiting factors for the use of ultrasound in birds (the small size of the objects, limited coupling possibilities and anatomic peculiarities, and above all the air sac system), because of the technical progress over the last few years, ultrasonography in birds has become a valuable and important diagnostic tool. Today, various studies on the use of ultrasonography have been published covering the examination of the heart, the liver, the spleen, and the GI and urogenital system. For some indications, especially the examination of the cardiovascular and the urogenital system, ultrasonography provides unique information and is the first diagnostic choice. For some disease processes (e.g., pericardial effusion) it is even the only definite possibility for intra vitam diagnosis.

Whereas the sonographic presentation of the inner organs in healthy birds may sometimes be difficult, the situation in diseased birds is often completely different. Organ enlargements, displacement of the air sacs, and fluid accumulations facilitate the coupling of the transducer and improve the image quality.

TECHNICAL EQUIPMENT

The small size of a bird's organs and—concerning echocardiography—high heart rates make special demands on the technical equipment. However, most modern ultrasound devices should be able to produce diagnostic images.

The following requirements should be met when using ultrasound in birds:

- Electronic probes with small coupling surfaces (microcurved probes are preferable; phased array probes are not recommended)
- Probes with examination frequencies of at least 8 MHz up to 14 MHz
- An appropriate possibility to record images and motion loops

The size of the probe is critical, especially in small birds. Best results are obtained using scanners developed for human pediatric medicine or for operative or gynecologic use. For small birds, a stand-off might be useful for the examination.

For birds up to 1000 g in body mass, a scanner frequency of 8 MHz is recommended for proper visualization of the cardiac structures. Higher frequencies may be beneficial but lead to a decrease in frame rate and maximum examination depth. Recording of digital motion loops (video tape sequences will not reproduce the high frame rate) is recommended because the assessment and morphometry can be done after the examination without stress for the bird.

For echocardiographic examinations, the following points are important:

- A minimum of 100 frames per second
- A Doppler function (color and spectral Doppler)
- An electrocardiography (ECG) trigger function might be useful but is not mandatory

The high frame rate is necessary to get images from defined cardiac stages such as systole and diastole. In birds with heart rates up to 600 beats/min, meaning 10 beats/sec, a frame rate of 100 images/s provides 10 images/cardiac cycle. Although there is only limited experience with the use of Doppler in birds, ultrasound devices used for cardiology should provide both spectral Doppler and color Doppler function; these techniques will become more important in the future. The trigger function is useful to correlate cardiac images and measurements with certain stages of the ECG, therefore being able to identify the corresponding cardiac stage (end-diastolic and end-systolic). If not available, the loop function can be used to scroll to the respective cardiac stage (e.g., maximum contraction for end-systolic assessment).

PATIENT PREPARATION, APPROACHES, AND EXAMINATION PROCEDURE

Because the filled GI tract may disturb the penetration of the ultrasound waves and therefore the visualization of organs beyond the intestines (especially with the ventromedian approach, see later), birds should be fasted before examination. For psittacines, 2 to 4 hours are recommended; pigeons should be fasted for 12 hours and raptors up to 48 hours.

In general, no anesthesia is necessary for the ultrasonographic examination. For the examination of the circulatory system, stress may be problematic for the interpretation of the results in awake birds, but on the other hand, anesthesia may also affect the heart rate and contractility. Therefore, for B-mode examinations, anesthesia is recommended only for stress-sensitive birds, but spectral Doppler examinations should be done under general anesthesia to reduce the influence of handling and fixation to the blood velocity.

The patient may be held by an assistant or by the owner either in dorsal or lateral (using the flank area) recumbency or in a standing position. In patients with clinical signs of cardiac disease or dyspnea, dorsal recumbency may cause severe circulatory problems and therefore should be avoided. Generally, it is advisable to hold the bird as upright as possible.

Because of the anatomy of the avian patient, the possibilities of placing the transducer in contact with the skin are limited. Because

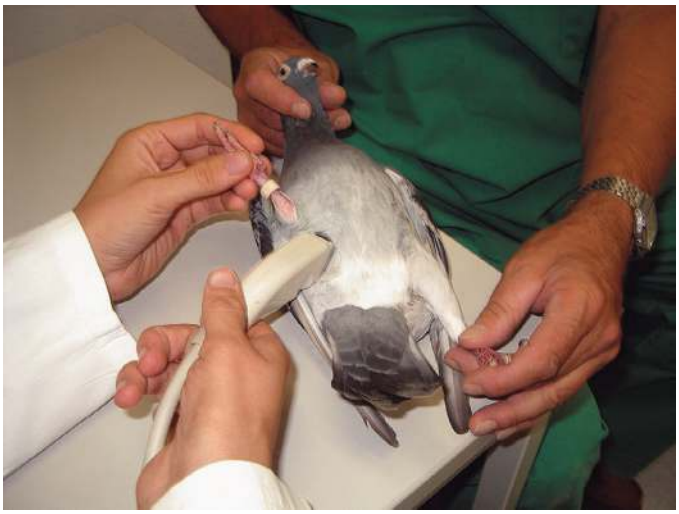


FIGURE 6-142 Pigeon (*Columba livia*), parasternal approach. For this approach, the leg is pulled either backward or forward. It is only possible in birds with enough space between the last rib and the pubic bones.

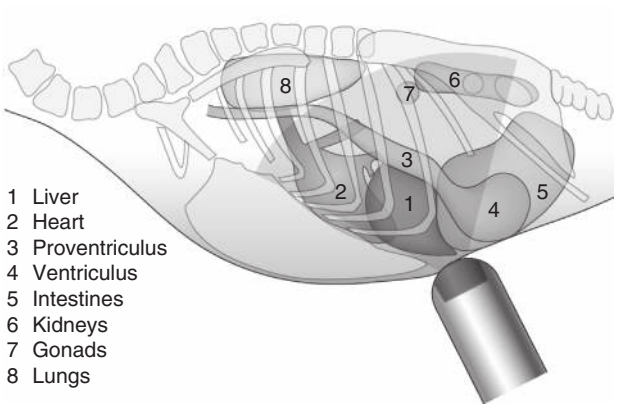


FIGURE 6-143 Ventromedian approach, schematic view. The homogenous tissue of the liver serves as an acoustic window to visualize the heart.

intimate contact between the transducer and skin is necessary for optimal image quality, there are only two approaches suitable: the ventromedian approach behind the sternum and the parasternal approach behind the last rib. The ventromedian approach is the main approach. The transducer is coupled in the median directly behind the caudal end of the sternum. The parasternal approach is usable in birds with sufficient space between the last rib and the pelvic bones (e.g., pigeons and some raptor species). The scanner is coupled on the right side of the bird because on the left side the gizzard might disturb the penetration of the ultrasound waves. The leg is pulled either backward or forward and the probe has to be pressed slightly to the body wall to compress the underlying air sacs (Fig. 6-142).

Feathers impede the contact between scanner and skin and therefore reduce the image quality. Whether they have to be plucked or if it is sufficient to part them depends on the species. In pigeons, chickens, and birds of prey, some feathers normally have to be plucked. In most psittacines, however, separating the feathers is sufficient in most cases. For the ventromedian approach, in psittacines a featherless area behind the sternum (breeding spot) can be used. In waterfowl, plucking of the feathers should be done very carefully because the birds might lose their ability to swim. Finally, sufficient quantities of a commercially available water-soluble acoustic gel should be applied to ensure a good contact between the transducer and the skin. These gels are well tolerated and can be removed from feathers and skin easily.

In ultrasonographic diagnostics, assessment of the results is much more subjective and much more dependent on personal experience and personal examination technique than in radiography. Therefore for the routine ultrasonographic evaluation, a fixed pattern for the examination should be used. A recommended method for the examination of the coelomic cavity is to start with the evaluation of the liver followed by the heart, the GI, and finally the urogenital system.

With the ventromedian approach, the transducer is directed cranially to visualize the liver tissue (Fig. 6-143). After identifying the liver, the transducer is swept from lateral to medial until the whole liver has been examined. If indicated, ultrasound-guided liver biopsies may be taken.

For examination of the heart, the transducer is directed craniodorsally until the heart is identified; the liver works as an acoustic window. When the heart has been visualized, the transducer is swept laterally to examine section by section. First, the sagittal view (perpendicular to the sternum) is examined; afterward the transducer is rotated 90 degrees to have a second plane of view. Before taking measurements, the probe has to be adjusted until the maximum extent of the ventricles is shown. With the parasternal approach, additional transverse sections of the heart can be obtained.

The transducer is swept to the left side to start the examination of the GI tract. The gizzard is easy to identify because of its large muscles and its large content of grit stones in granivorous birds. Using frequencies of at least 12 MHz, the gizzard wall and the koilin layer can be assessed. The proventriculus can be seen occasionally on the right side; the small intestines can only be demonstrated clearly with high examination frequencies (at least 10 MHz). Furthermore the wall, content, and peristalsis of the small intestines can be recognized; this is particularly easy for the duodenal loop including the pancreas. The cloaca is seen in the caudal abdomen.

Finally, the urogenital system laying behind the intestines is examined starting with the presentation of the kidneys. The kidneys should be scanned in a cross section to identify the tissue (see later). After identification, the whole extent of the organ can be demonstrated in a longitudinal section. The presentability of the testes and the ovaries depends on the status of sexual activity; immature and inactive gonads are normally not visible in the ultrasound image.

ORGANS AND ORGAN SYSTEMS

Liver

The most frequent indication for the ultrasonographic examination of the liver is an enlargement of the hepatic silhouette in the radiographic examination. The ultrasonographic appearance of the liver parenchyma is of average echogenicity and coarsely granular, but with a uniform texture throughout (Fig. 6-144). The edges of the liver appear sharp, but because only parts of the liver can be assessed at the same time, measurements of the liver size are difficult. Intrahepatic vessels are sometimes visible as anechoic channels. In birds with a gallbladder, this is a smooth, clearly defined, round or oval structure with thin walls and anechoic contents. It is located caudally to the right liver lobe.

In birds with liver disease, common alterations found in the ultrasonographic examination include:

- Enlarged (or reduced) size
- Irregular, swollen edges (Fig. 6-145)
- Decreased or increased echogenicity of the parenchyma or focal parenchymal lesions
- Dilated and/or congested liver vessels (see Fig. 6-145)

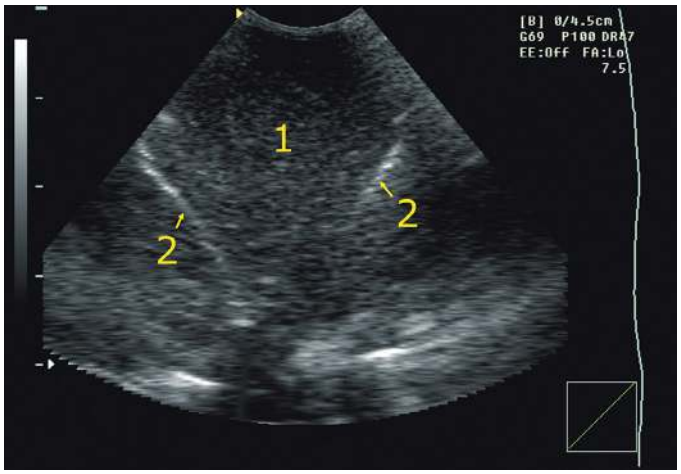


FIGURE 6-144 African gray parrot (*Psittacus erithacus*), sonographic examination, ventromedian approach; normal liver. The liver tissue (*1*) is delicately granulated and of average echogenicity. The borders between the liver lobes are visible as hyperechoic lines (*2*).

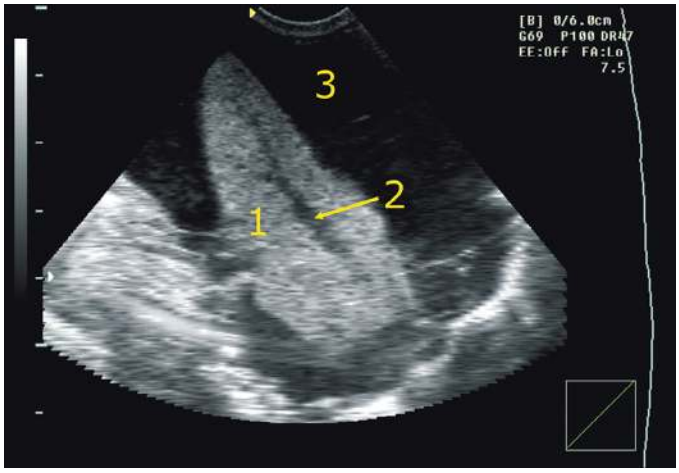


FIGURE 6-145 African gray parrot (*Psittacus erithacus*), sonographic examination, ventromedian approach. The liver (*1*) is swollen, dilated vessels (*2*) are visible, and ascites (*3*) is present.

- Liver cysts (sharply defined, anechoic mass with marked posterior acoustic enhancement)
- Fluid (e.g., ascites) in the coelomic cavity (see Fig. 6-145)

Focal lesions are easy to identify because they interrupt the uniform appearance of the liver parenchyma. Neoplastic alterations often appear as focal echogenic areas, whereas necrosis often produces hypoechoic areas. The identification of diffuse parenchymal lesions (e.g., fatty liver alterations) is more difficult. Although the echogenicities of inflammation, neoplasia, calcification, and granuloma are different, it is not possible to predict the histologic nature of a lesion from the ultrasonographic appearance. Only tentative diagnoses are possible and biopsy is required for the definitive diagnosis. Ultrasound-guided biopsies may be taken easily from defined regions of the liver parenchyma according to the procedure in mammals.

Circulatory System

The great advantage of ultrasound in examining the avian heart is the presentation of the inner structures and therefore the possibility to assess both the morphologic and the functional status. However,

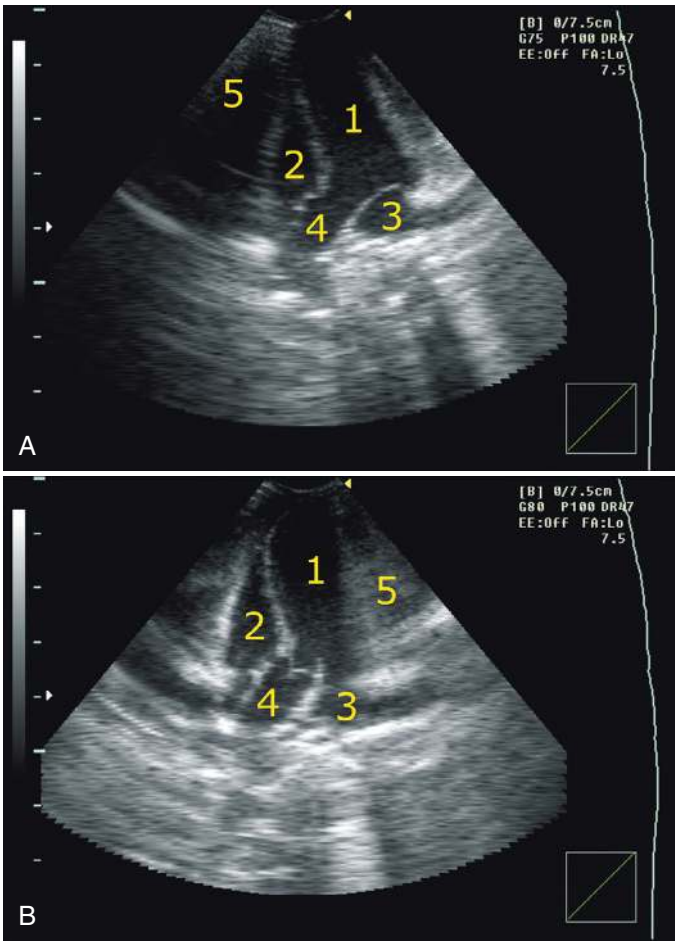


FIGURE 6-146 European buzzard (*Buteo buteo*), sonographic examination, ventromedian approach; normal heart. **(A)** systole, **(B)** diastole. The left (*1*) and right (*2*) ventricle and the left atrium (*3*) are visible. The valves of the aortic root (*4*) are closed during diastole, whereas during systole, the atrioventricular valves are closed. (*5*) liver tissue.

because of the anatomic peculiarities of the avian heart, the protocol (standardized views) recommended for echocardiography in mammals cannot be used in birds. M-mode, a valuable tool for assessment of wall thickness and contractility in mammals, is not useful in birds because the avian heart is only visualized in longitudinal and semi-transverse views. To date, B-mode (two-dimensional [2D] echocardiography) in birds is an established examination technique and reference values have been reported for several species (Table 6-33). Also, Doppler echocardiography has been tested successfully in birds. Flow patterns could be shown in color mode and velocities could be measured in the areas of the atrioventricular (AV) openings and the aortic root. Systematic examinations and reference values using pulsed-wave spectral Doppler are available for some species but the use of color Doppler is only documented in some case reports.

The body mass and the external palpable sternal length are useful parameters to set the measurements in relation to the bird's size. An ECG should be derived to trigger the cardiac images to an end-systolic and end-diastolic stage.

B-Mode (Two-Dimensional Echocardiography)

Using 2D echocardiography, the inner structures of the heart can be assessed subjectively and by taking measurements (Figs. 6-146 to 6-148). The size of the ventricles, the wall thickness of the

TABLE 6-33 2D Echocardiography, Important Measured and Calculated Parameters in Birds (Mean ± SD)

Parameter	Ventromedian Approach *Psittacus Erithacus* (Pees *et al.*, 2004)	*Amazona* spp. (Pees *et al.*, 2004)	*Cacatua* spp. (Pees *et al.*, 2004)	Diurnal Raptors* (Boskovic *et al.*, 1999)	Parasternal Approach Pigeons (Krautwald-Jugghanns *et al.*, 1995)
Body mass (g)	493 ± 55	353 ± 42	426 ± 162	720 ± 197	434 ± 52
LEFT VENTRICLE					
Length systole (mm)	22.5 ± 1.9	21.1 ± 2.3	19 ± 1.3	14.7 ± 2.8	17.9 ± 1.0
Length diastole (mm)	24 ± 1.9	22.1 ± 2.2	19.9 ± 1.6	16.4 ± 2.7	20.1 ± 1.4
Width systole (mm)	6.8 ± 1	6.7 ± 1.2	6.4 ± 1.7	6.3 ± 1.1	5.2 ± 0.4
Width diastole (mm)	8.6 ± 1	8.4 ± 1	8.3 ± 1.5	7.7 ± 1.2	7.4 ± 0.6
Width fractional shortening (%)	22.6 ± 4.4	22.8 ± 4.2	25.6 ± 7	Not given	27.2 ± 4.5
RIGHT VENTRICLE					
Length systole (mm)	9.2 ± 1.4	9.4 ± 1.8	10.3 ± 1.2	12.7 ± 2.7	Not given
Length diastole (mm)	11.5 ± 1.9	10.3 ± 1.3	11.3 ± 2.3	13.9 ± 2.5	9.9 ± 0.8
Width systole (mm)	2.8 ± 0.9	3.1 ± 0.7	2.3 ± 0	2.1 ± 0.6	Not given
Width diastole (mm)	4.8 ± 1.1	5.2 ± 1.3	3.5 ± 0.5	2.5 ± 0.8	4 ± 0.5
Width fractional shortening (%)	40.8 ± 11.9	34.1 ± 3.7	33.3 ± 10.3	Not given	Not given
INTERVENTRICULAR SEPTUM					
Thickness systole (mm)	2.9 ± 0.5	2.2 ± 0.1	1.9 ± 0.3	1.9 ± 0.6	3.8 ± 0.1
Thickness diastole (mm)	2.5 ± 0.3	2.1 ± 0.4	1.7 ± 0.4	1.9 ± 0.5	3.3 ± 0.2

*Including *Buteo buteo, Accipiter nisus, Accipiter gentilis,* and *Milvus milvus*.

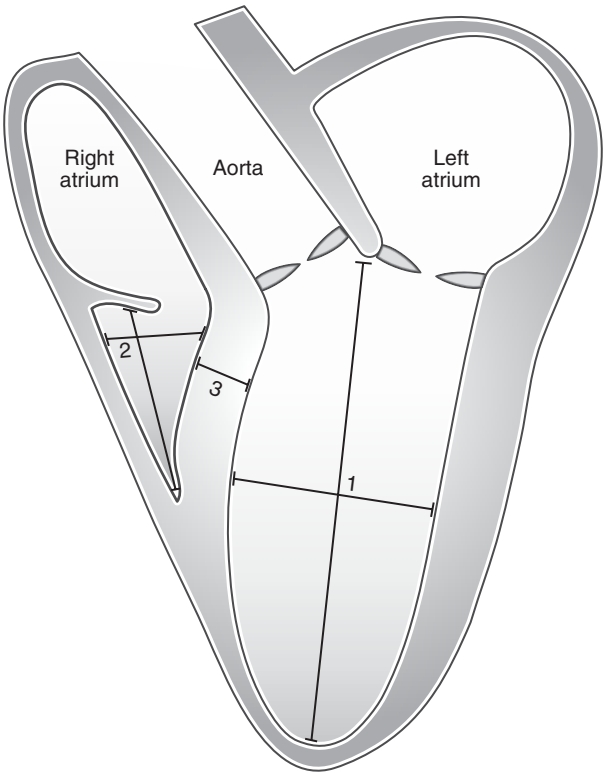


FIGURE 6-147 Schematic view of the avian heart, horizontal view, measurement points. Reference values are given in Table 6-33 for the length and the width of the left ventricle (*1*), the right ventricle (*2*), and the interventricular septum (*3*).

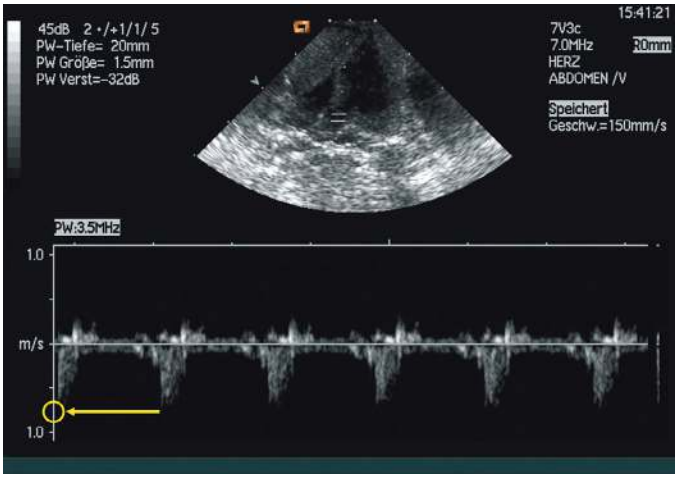


FIGURE 6-148 Carrion crow (*Corvus corone*), sonographic examination, ventromedian approach, spectral Doppler; normal heart. Within the two-dimensional image, a gate shows the area for the spectral Doppler measurements. The velocity is shown against the time, in this case approx. 80 cm/s *(yellow line)*.

interventricular septum, and the contractility of the ventricles are important parameters to evaluate the cardiac morphology and function. Additionally, the left AV valves, the aortic valves, and the right muscular AV valve can be assessed depending on the image quality. Signs of congestion (hydropericardium, ascites, and congestion of liver vessels) are easy to recognize.

Measurements are taken from the 2D image following the "inner edge method." In addition to the size, the contractility (fractional shortening [FS]) of the ventricles is of special importance. It is calculated using the formula FS% = (diastolic value – systolic value) × 100/diastolic value. Because of the sickle moon shape of the right ventricle in the avian heart, the contractility of this chamber is much higher than that of the left one.

Doppler Echocardiography

Spectral Doppler is used for determining the velocity of the blood flow (inflow, outflow), which is displayed as a 2D graph against time (see Fig. 6-148). Reference values are available for the diastolic inflow into the left and right ventricle and the systolic outflow into the aorta (Table 6-34).

Color Doppler shows the velocity of the blood flow in colors overlying the 2D image (Fig. 6-149). It can be used for positioning of the gate for spectral Doppler echocardiography but has also been reported to be useful in detecting valvular insufficiencies and aneurysms. Because the frame rate decreases considerably in most ultrasound devices when using color Doppler, its value is limited in avian echocardiography at present.

In birds with cardiovascular disease, common alterations found in the ultrasonographic examination include:

- Arrhythmias
- Enlarged (or reduced) ventricles (Fig. 6-150)
- Increased or decreased thickness of the walls
- Increased or decreased contractility of the ventricles
- Alterations of the myocardium or the endocardium
- Alterations of the pericardium, including pericardial effusion (see Fig. 6-150; Fig. 6-151)
- Alterations of the valves (thickening, insufficiency) (see Fig. 6-150)
- Increased or decreased cardiac blood flow velocities
- Ascites and liver congestion (see Fig. 6-150; Fig. 6-152)

The most frequent pathologic echocardiographic findings are hydropericardium and hypertrophy/dilatation of the right ventricle. Both alterations are often caused by right-sided congestive heart failure. In these cases, the right ventricle is often nearly as large as the left one; the walls are significantly thickened. In birds with

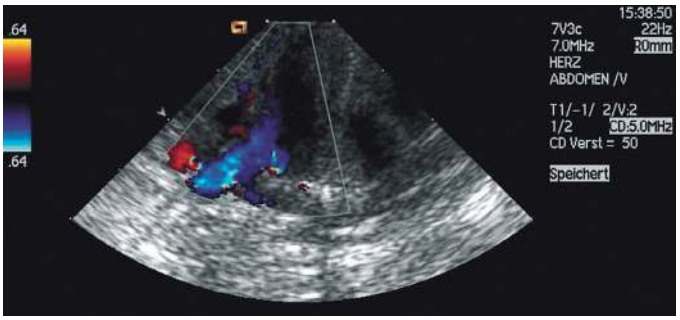


FIGURE 6-149 Carrion crow (*Corvus corone*), sonographic examination, color Doppler, ventromedian approach; normal heart. The outflow into the aorta is shown as blood flowing away from the transducer (*blue*).

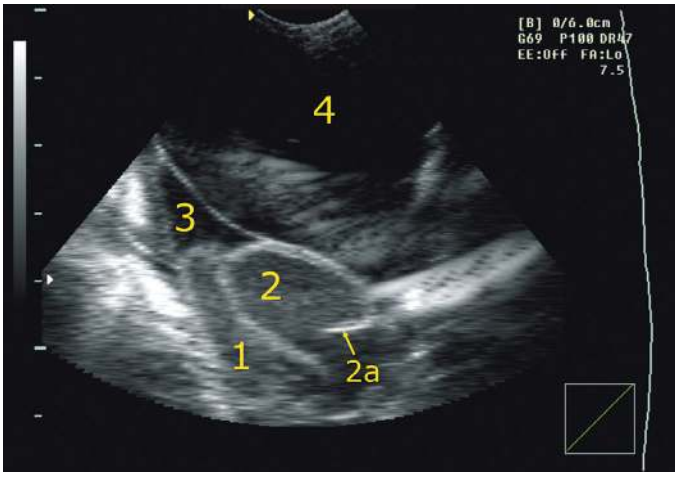


FIGURE 6-150 African gray parrot (*Psittacus erithacus*), sonographic examination, ventromedian approach; right-sided cardiac insufficiency. The right ventricle (*2*) is larger than the left ventricle (*1*); the muscular atrioventricular valve (*2a*) is thickened. Pericardial effusion (*3*) and ascites (*4*) are present.

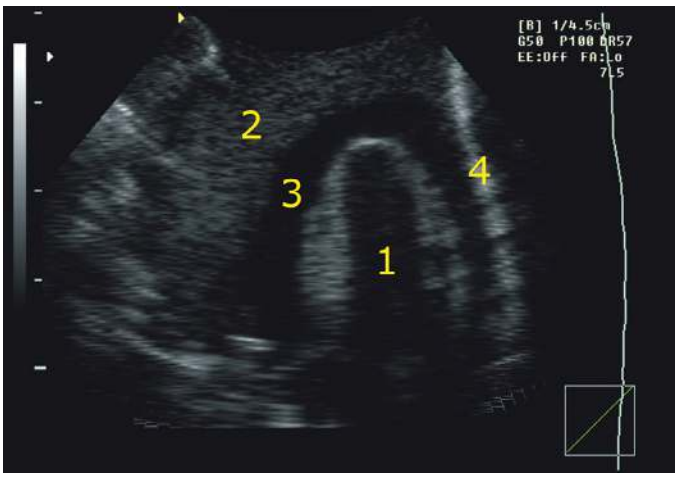


FIGURE 6-151 African gray parrot (*Psittacus erithacus*), sonographic examination, ventromedian approach; chlamydiosis. Pericardial effusion (*3*) is visible as anechoic area between the heart (*1*) and the liver (*2*). (*4*) sternum.

TABLE 6-34 Velocities of Intracardial Blood Flow in Some Bird Species (Anesthetized)

Parameter	*Amazona* spp. (Pees *et al.*, 2005)	*Ara* spp. (Carrani *et al.*, 2003)	*Cacatua Galerita* (Carrani *et al.*, 2003)	*Psittacus Erithacus* (Carrani *et al.*, 2003)	*Falco* spp. (Straub *et al.*, 2001)	*Buteo Buteo* (Straub *et al.*, 2001)
Diastolic inflow left ventricle (m/s)	0.18 ± 0.03	0.54 ± 0.07	0.32 ± 0.15	0.39 ± 0.06	0.21 ± 0.03	0.14 ± 0.01
Diastolic inflow right ventricle (m/s)	0.22 ± 0.05	Not given	Not given	Not given	0.21 ± 0.04	0.14 ± 0.02
Systolic outflow aortic root (m/s)	0.83 ± 0.08	0.81 ± 0.16	0.78 ± 0.19	0.89 ± 0.13	0.95 ± 0.07	1.18 ± 0.05

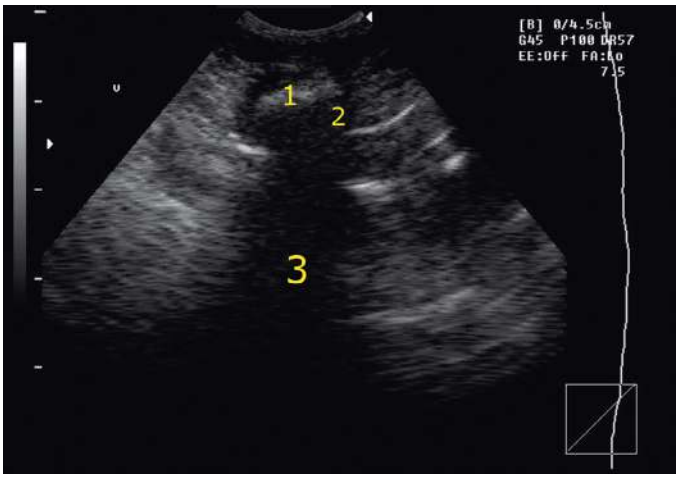


FIGURE 6-152 Monk parakeet (*Myiopsitta monachus*), sonographic examination, ventromedian approach; normal gizzard. The content of the gizzard is hyperechoic (*1*), whereas the muscular wall is hypoechoic (*2*). Because of the total reflections of the grit, there are no structures visible beyond the gizzard (acoustic shadowing, *3*).

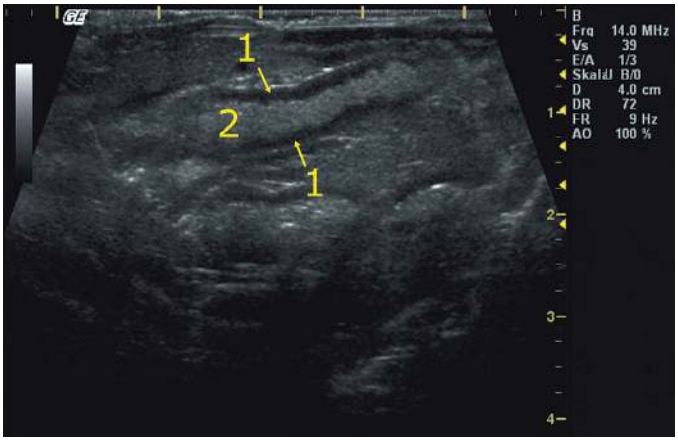


FIGURE 6-153 Channel-billed toucan (*Ramphastos vitellinus*), sonographic examination, ventromedian approach; enteritis. Different layers of the thickened wall (*1*) are visible; (*2*) intestinal content. (Courtesy I. Kiefer, Leipzig.)

hydropericardium, an anechoic area is visible between the heart and the pericardium. Increase in blood pressure in the large circulatory cycle often leads to liver congestion (dilated liver vessels visible) and to ascites. Hypertrophy of the right muscular AV valve is often associated with hypertrophy of the right ventricle. Alterations of the left ventricle are seen less frequently. They may be combined with thickened AV valves indicating valvular damage and insufficiency. Left-sided congestive heart failure is normally combined with right-sided alterations because of congestion in the small circulatory cycle.

Gastrointestinal Tract

The gizzard is easy to identify because of its large muscles and its large content of grit in granivorous birds. With higher probe frequencies, the koilin layer can be assessed. Because of the total reflexion of the content, a typical acoustic shadowing can be seen behind the gizzard (see Fig. 6-152). The proventriculus can be demonstrated when enlarged whereas the identification of the normal-sized organ is difficult. The intestinal layers and the content can be demonstrated with probes that have a frequency of at least 12 MHz (Fig. 6-153). Peristalsis can be also recognized with lower frequencies. The cloaca is easily demonstrated in the caudal abdomen. A retrograde filling of the cloaca with fluid may help assess the mucosa.

In birds with GI disease, common alterations found in the ultrasonographic examination include:

- Increased or decreased peristalsis
- Enlarged or reduced size of the organs
- Increased wall thickness (e.g., because of inflammation)

Because there have still been no studies of the use of ultrasound for the examination of the GI tract in diseased birds, assessment is based on the subjective experience of the examiner. Therefore at present the value of ultrasound for the examination of the GI tract in birds is limited in comparison with other imaging techniques.

Urogenital System

Because of its anatomy, the reproductive tract in the avian species is not as easy to examine as in mammals. The GI tract and the air sacs impede the examination. At the moment, sex determination with ultrasonography is only possible in female birds with large follicles or eggs present or in larger birds, such as ratites, with the use of intracloacal scanners.

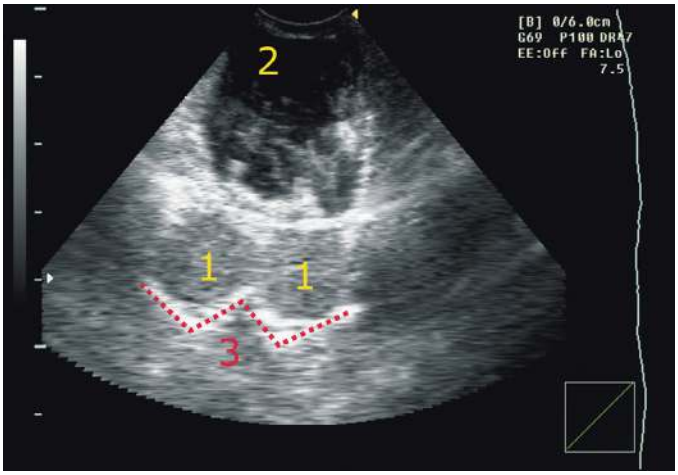


FIGURE 6-154 African gray parrot (*Psittacus erithacus*), sonographic examination, ventromedian approach; nephrosis because of toxicosis. The increased size of the kidneys (*1*) is visible; ascites (*2*) is present. (*3*) W-shaped reflection of the vertebral column and the pelvic bones.

In birds with urogenital tract disease, common changes found at ultrasonographic examination include:

- Organ enlargements, including signs of neoplastic alteration (e.g., necrotic areas) (Figs. 6-154 to 6-157)
- Cystic alterations of the ovary and the kidneys (see Fig. 6-156)
- Eggs without calcified shell (Fig. 6-158)
- Eggs with broken calcified shell
- Thickening of the oviduct (inflammatory processes) (Fig. 6-159)
- Inflammation products within the oviduct (laminated eggs) (see Fig. 6-159)

Kidneys

Sonographic demonstration of the normal kidney by transcutaneous ultrasonography is not possible in most cases. This is because of its position along the vertebral column within the depressions of the pelvis and the surrounding abdominal air sacs. However, in cases of kidney enlargement, the air sacs are distended and it is possible to visualize the kidneys. Both the size and the parenchyma can be assessed without difficulty not only in large birds but also in smaller ones

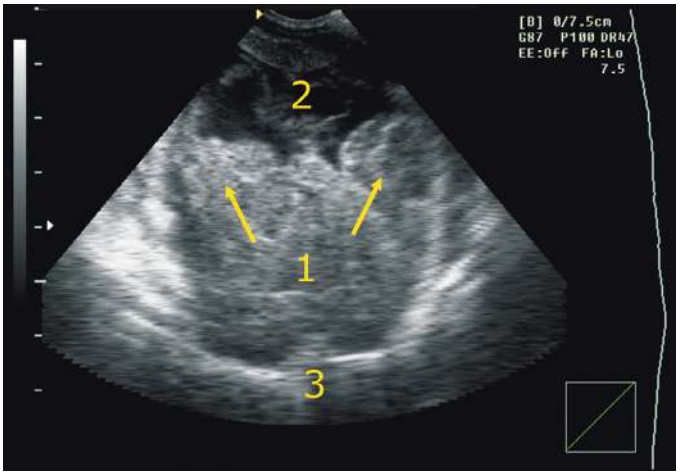


FIGURE 6-155 Yellow-crowned Amazon (*Amazona ochrocephala*), sonographic examination, ventromedian approach; kidney neoplasia. The kidney tissue (*1*) forms a solid mass of inhomogeneous echogenicity. Other organs are displaced (*arrows*); ascites (*1*) is present. (*3*) W-shaped reflexion of the vertebral column and the pelvic bones.

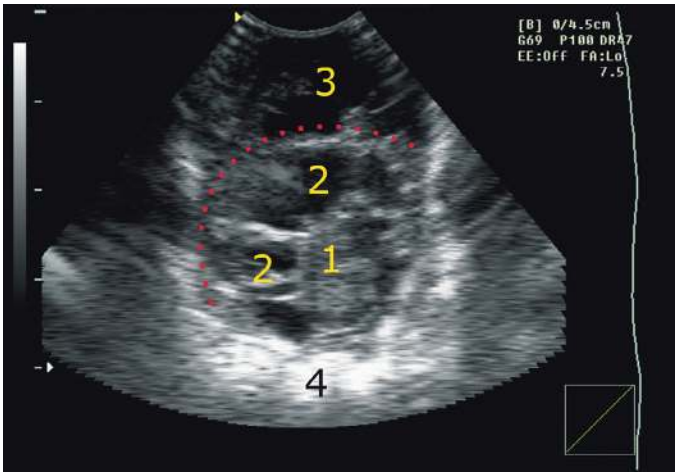


FIGURE 6-157 Budgerigar (*Melopsittacus undulatus*), sonographic examination, ventromedian approach; testicular neoplasia. The neoplasia is presented as a solid mass (*1*) with several anechoic areas (*2*, necroses); (*3*) ascites; (*4*) reflection of the vertebral column.

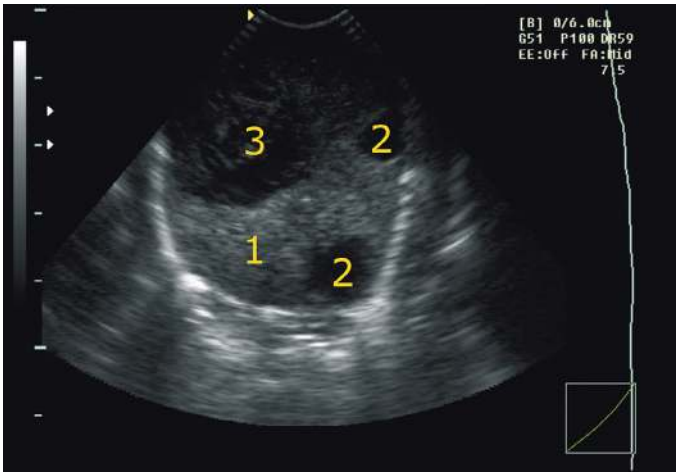


FIGURE 6-156 Little corella (*Cacatua sanguinea*), sonographic examination, ventromedian approach; kidney cysts and kidney bleeding. The cysts (*2*) are visible as anechoic areas within the kidney tissue (*1*). The bleeding was demonstrated as an anechoic area containing movable hyperechoic structures (coagula).

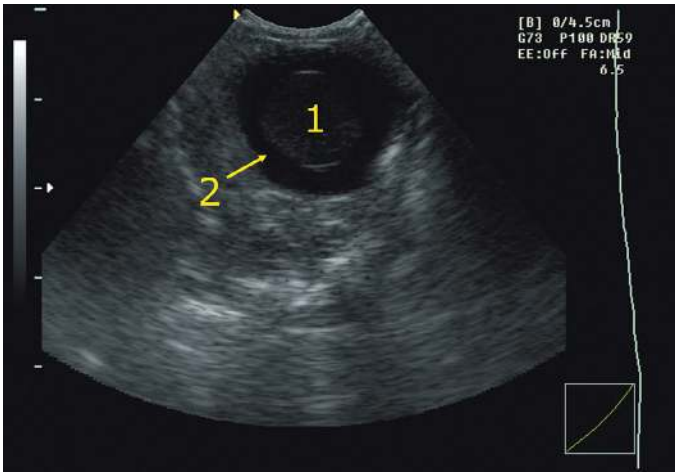


FIGURE 6-158 Peach-faced lovebird (*Agapornis roseicollis*), sonographic examination, ventromedian approach; uncalcified egg. The yolk (*1*) is demonstrated as a central area of average echogenicity; the albumin (*2*) presents as an anechoic area around the yolk. This case is not a pathologic alteration but a normal stage of egg development.

(e.g., budgerigars). In a cross section (i.e., with the plane of the beam perpendicular to the spinal column), the kidneys are visualized laying in a W-shaped total reflexion caused by the pelvic/spinal bones (see Figs. 6-154 and 6-155). In this view, the kidneys can be compared with one another. In this section view, the kidneys appear as round to oval and their size can be measured. By turning the transducer about 90 degrees, the kidneys can be examined in their long axis (longitudinal to the spinal column). The echotexture of an enlarged inflamed kidney is homogenous and rather anechoic, with no recognizable inner structure. Neoplasms are visible as voluminous and often rounded single masses, frequently with diffuse inhomogeneous echotexture and hypoechoic areas (see Fig. 6-155). Cysts of the kidneys are easy to detect by means of ultrasound. They appear sonographically as clearly defined, rounded anechoic structures (see Fig. 6-156), often with marked distal acoustic enhancement. If a differentiation between renal and ovarian cysts is not possible, an ultrasound-guided puncture of the fluid-filled cavities can be performed and the fluid can be examined.

Uric acid depositions and calcifications cause reflections (i.e., increased echogenicity); the renal tissue appears more inhomogeneous. However, diagnosis of renal gout by means of ultrasound is difficult: other techniques (e.g., radiology, endoscopy, blood chemistry, and biopsy) should be taken into account before making a diagnosis.

Gonads

Sonographic demonstration of testes is only successful in the case of highly sexually active birds. The parenchyma of this organ shows a delicately granulated structure of average echogenicity. Neoplasia, inflammatory processes, and other changes, in conjunction with enlargement of the organ, are observable sonographically. Neoplastic tissue is often presented as a rounded single mass that is demarcated from surrounding structures (see Fig. 6-157). However, it is not possible to associate the masses definitely with the testes. Ultrasound-guided biopsy for histopathology is possible but difficult (risk of internal bleeding).

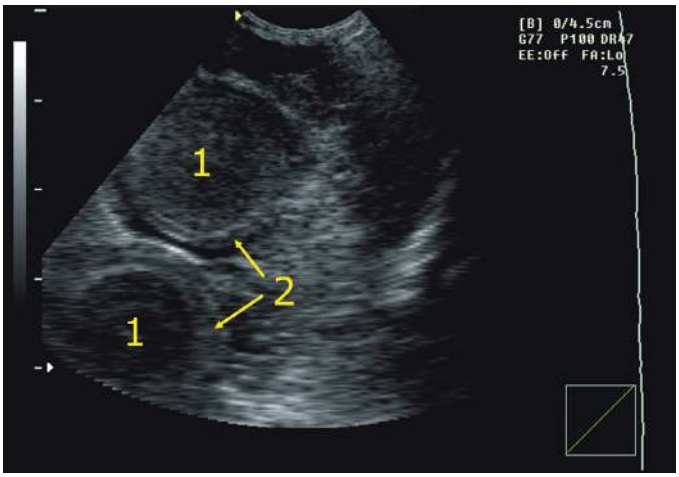


FIGURE 6-159 African gray parrot (*Psittacus erithacus*), sonographic examination, ventromedian approach; laminated egg. The wall of the oviduct (*2*) is thickened; the content (*1*) is demonstrated with alternating echogenicity ("onion layers").

Ovary

Sonographic demonstration of the ovary is successful in most active hen birds with a bodyweight of more than 70 g. The picture of active ovaries is characterized by the presence of follicles of different sizes representing various stages of development. Developing follicles are first seen as round areas with an indistinct, anechoic, or hypoechoic inner structure. In advanced stages of development the follicles exhibit the more echoic content of yolk. More distally in the oviduct, in the magnum, the ova exhibit a distinct separation between the echogenic yolk and a surrounding poorly echoic perimeter of albumin. The hyperechoic shell, added in the uterus, is easily recognizable.

Ovarian neoplasias are distinctly demonstrable by means of sonography. Accompanied by massive enlargement of the affected organ, the well-defined structures appear as large rounded masses of mixed echogenicity, seen as marked focal or diffuse inhomogeneous echo-texture. Because of their massive extension, the origin of the tumors cannot be determined. Ovarial cysts appear sonographically as clearly defined, rounded anechoic compartments, showing the phenomenon of distal acoustic enhancement.

Oviduct

Irrespective of its functional state, the unchanged oviduct is often sonographically indistinguishable from the surrounding abdominal structures (i.e., intestines). The active oviduct can be distinguished because of the presence of eggs and the lack of contractility (in comparison to the intestines).

Advanced inflammatory processes of the oviduct are recognizable by increased thickness of the oviduct wall. If laminated eggs are present, their echogenicity depends on the kind of effusion. Mostly, laminated eggs are presented with changing hypoechoic and hyperechoic areas around a central point. This is because of the different densities of the deposited material, which gives the laminated egg the sonographic appearance of onion layers (see Fig. 6-159).

Sometimes, cysts of the rudimentary right oviduct may be detected. They show the same sonographic picture as ovarian cysts. Abnormal eggs are detected most frequently in suspected cases of egg binding. Thin-shelled or noncalcified eggs (see Fig. 6-159), malformed eggs, and eggs with destroyed shells are sonographically assessable. Because of their high echogenicity, roughness of the eggshell cannot be demonstrated in most cases.

Other Organs

Spleen

Because of the position of the spleen, the parasternal approach is preferable for the examination. Sonographic demonstration of the normal organ is not possible. In birds with splenomegaly, the spleen might be identified as a round or oval structure of average to high echogenicity. Differentiation between neoplastic and inflammatory changes can be attempted using color Doppler assessment.

Eye

Ultrasonography of the avian eye is useful for diagnosis of ocular changes, especially when direct visualization of the inner eye is not possible, for instance in cases of opacity of the lens. A-mode ultrasonography is mainly used for biometry of the eyeball. 2D B-mode ultrasonography supplies more information about ocular structures and pathologic changes in the eye. After application of local anesthetic the transducer can be placed directly on the cornea. Acoustic gels are only required if the transducer is placed on the closed eyelids. The anterior chamber and the vitreous are physiologically free of echogenic structures. See references in the Further reading list for detailed information on ultrasonographic examination of the avian eye.

REFERENCES

Boskovic M, Krautwald-Junghanns M-E, Failing K, Schneider M: Möglichkeiten und Grenzen echokardiographischer Untersuchungen bei Tag- und Nachtgreifvögeln (Accipitriformes, Falconiformes, Strigiformes), *Tierärztliche Praxis* 27:334–341, 1999.

Carrani F, Gelli D, Salvadori M, et al: A preliminary echocardiographic initial approach to diastolic and systolic function in medium and large parrots. *Proceedings of the Association of Avian Veterinarians*, 2003, Tenerife, pp 145–149.

Krautwald-Junghanns ME, Schulz M, Hagner D, et al: Transcoelomic two-dimensional echocardiography in the avian patient, *J Avian Med Surg* 9(1):19–31, 1995.

Pees M, Straub J, Krautwald-Junghanns ME: Echocardiographic examinations of 60 African grey parrots and 30 other psittacine birds, *Vet Rec* 155: 73–76, 2004.

Pees M, Straub J, Schumacher Juergen, et al: Pilot study of echocardiographic studies using color- and pulsed-wave spectral Doppler methods in blue-crowned amazons (Amazona ventralis) and blue-fronted amazons (Amazona a. aestiva), *Dtsch Tierarztl Wochenschr* 112(2):39–43, 2005.

Straub J, Pees M, Schumacher J, Krautwald-Junghanns ME: Doppler-echocardiography in birds. *Proceedings of the Association of Avian Veterinarians*, 2001, Munich, pp 92–94.

FURTHER READING

Gumpenberger M, Korbel RT: Comparative aspects of diagnostic imaging in avian ophthalmology using ultrasonography and computed tomography. *Proceedings of the Association of Avian Veterinarians*, 2001, Munich, pp 99–102.

Krautwald M-E, Pees M, Reese S, Tully T, editors: *Diagnostic imaging of exotic pets*, Hannover, 2011, Schlütersche Verlagsanstalt, pp 1–136.

Krautwald-Junghanns ME, Pees M: Ultrasonography of the avian liver and gall bladder. *Proceedings of the 27th Annual Association of Avian Veterinarians Conference*, 2006, San Antonio, pp 53–58.

Krautwald-Junghanns ME, Stahl A, Pees M, et al: Sonographic investigations of the gastrointestinal tract of granivorous birds, *Vet Radiol Ultrasound* 43:576–578, 2002.

Krautwald-Junghanns M-E, Zebisch K, Enders F, et al: Diagnosis of liver disease in birds by radiography and ultrasonography—under special consideration of ultrasound-guided liver biopsy, *Seminars in Avian and Exotic Pet Medicine* 10(4):153–161, 2001.

ADVANCED CLINICAL IMAGING

Nico J Schoemaker, Yvonne RA van Zeeland

Of the advanced diagnostic imaging techniques, computed tomography (CT) has proven its value with many potential applications in the field of avian medicine. The other techniques (i.e., MRI, scintigraphy, single photon emission computed tomography [SPECT], and positron emission tomography [PET]) are currently far less commonly used, but show future potential. In this section a description of the techniques and their clinical application are discussed.

COMPUTED TOMOGRAPHY

CT is currently an established advanced diagnostic technique in avian medicine to diagnose disease, trauma, and organ abnormalities in a variety of avian orders, including psittaciformes, falconiformes, anseriformes, and columbiformes (Bartels *et al.*, 2000; Gumpenberger and Henninger, 2001; Krautwald-Junghanns *et al.*, 1993, 1998; Martel *et al.*, 2005; Orosz and Toal, 1992). When comparing CT imaging to conventional radiography, the former allows visualization of the various tissues and structures without interference of superimposition (Fig. 6-160). Combined with the superior resolution of the CT images, structures are now visualized that previously remained undetected. In smaller-sized patients (e.g., budgerigars), however, resolution of the regularly used CT scanners may be too limiting to be of diagnostic value in evaluating presence of abnormalities.

To produce CT images a CT scanner is used, which consists of a moving table on which the patient is placed, and an immobile gantry in which the rotating x-ray tube and a ring of x-ray detectors are placed (Fig. 6-161). Other CT scanners, however, have a sliding gantry that moves across the patient. The initial CT scanners produced a single slice per rotation. Scanning times started to decrease with the introduction of helical (spiral) CT scanners in which the x-ray tube circles around the patient in a continuous movement combined with simultaneous movement of the table. With these scanners an entire bird could be scanned within 2 minutes. The more advanced multislice helical scanners may produce up to 64 slices per rotation, which further decreases scanning times but also increases resolution as the sequential slices are placed closer together. Despite the procedure being of short duration, sedation or anesthesia is generally required to achieve appropriate positioning and prevent movement of the patient during the scanning period (Jenkins, 1991; Krautwald-Junghanns and Pees, 2010; Mackey *et al.*, 2008). Birds are preferably placed in ventral recumbency to facilitate breathing and perpendicular to the gantry to enable scanning in a transverse plane (see Fig. 6-161). To aid in the positioning, materials such as towels, foam pads, or IV fluid bags can be used. Although sandbags can be used for this purpose as well, they are

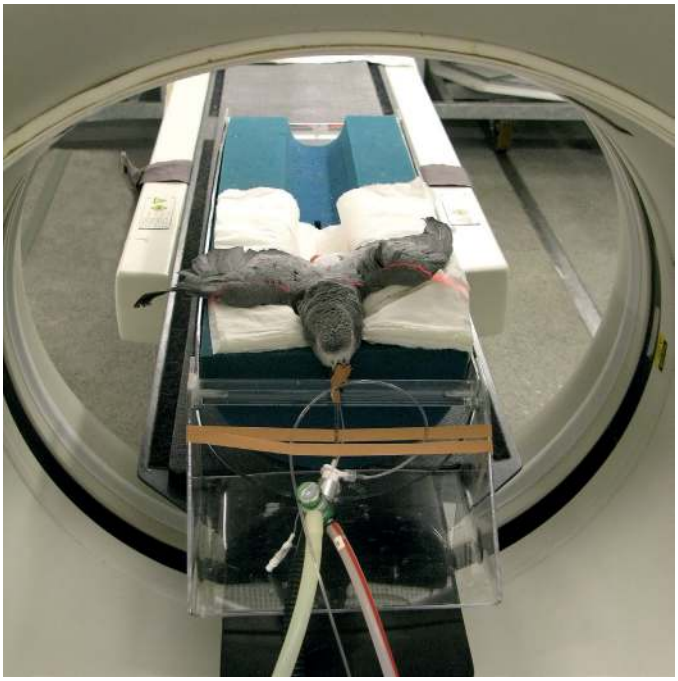


FIGURE 6-161 A 5-year-old female grey parrot (*Psittacus erithacus*) is anesthetized with isoflurane and placed in ventral recumbency on a sliding table that moves into the gantry of a helical CT scanner.

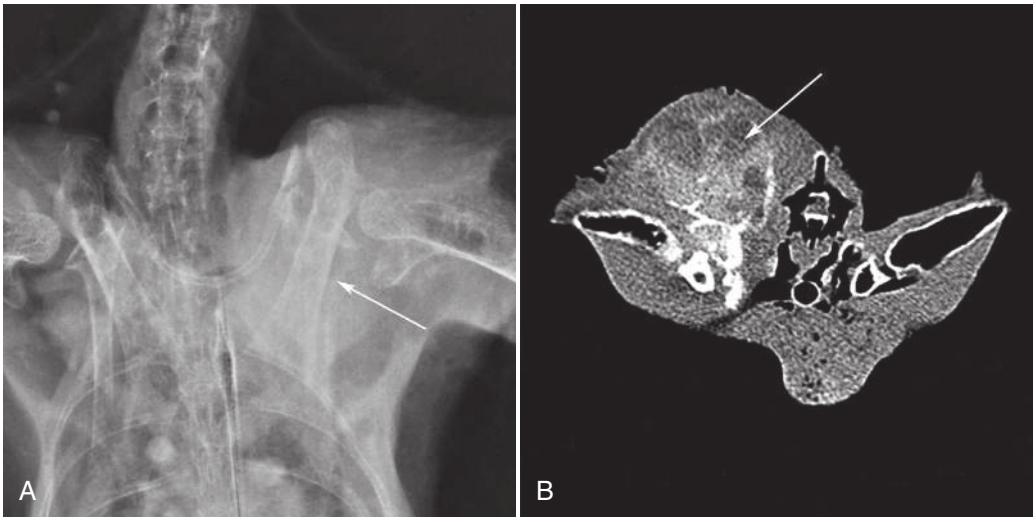


FIGURE 6-160 A ventrodorsal radiograph **(A)** and a transverse CT image **(B)** at the level of the scapula of a 6-month-old scarlet macaw (*Ara macao*) with a giant cell tumor of the left scapula. Note the osteolysis of the scapula that may easily be overlooked on the radiograph *(arrow)*, but really stands out on the CT (*arrow*). (Photos courtesy the Division of Diagnostic Imaging, Faculty of Veterinary Medicine, Utrecht University.)

generally not recommended because they may interfere with the images (Silverman and Dennison, 2010).

Although a wide variety of organ systems may be evaluated by using CT, the main clinical indications in avian medicine are currently the assessment of known or suspected abnormalities in the skeletal structures and respiratory tract (Krautwald-Junghanns and Pees, 2010). Soft tissue structures with abnormal vascularization because of disease (e.g., neoplasia, inflammation) can best be differentiated from normal tissue by administering IV contrast media whereby the abnormal tissues either show a decreased (unvascularized) or increased (vascularized) uptake of contrast medium (Figs. 6-162 and 6-163). Although noniodinated contrast media may be administered, iodinated media are most commonly used with doses of up to 2 mL/kg of a 250 to 300 mg of iodine/mL solution (Silverman and Dennison, 2010). A definite determination of the underlying pathology of the abnormalities detected on the CT images requires either a fine-needle aspiration of the lesion (which can be performed CT-guided; Fig. 6-164) or a diagnostic endoscopy, during which the lesion can be visualized and biopsied (Mackey *et al.*, 2008).

Image Processing

In each image different values of gray are seen representing different anatomic tissues and fluids. The varying gray values, which are named Hounsfield units (HU), have a quantitative meaning according to the amount of x-rays attenuated by the tissues. The HUs have been defined such that HU of air and water are −1000 and 0, respectively (Bushberg *et al.*, 2012). Darker (hypo-attenuating) areas in the image, representing, for example air (HU: −1000) and fat (HU: −50-−100), thus have a negative HU value whereas lighter (hyperattenuating) areas, representing, for example soft tissues (HU ~ +100) and mineralized structures (HU up to +3000), have a positive HU value.

The standard format used to store CT images is DICOM (digital imaging and communications in medicine), created by the American College of Radiology and the National Electrical Manufactures

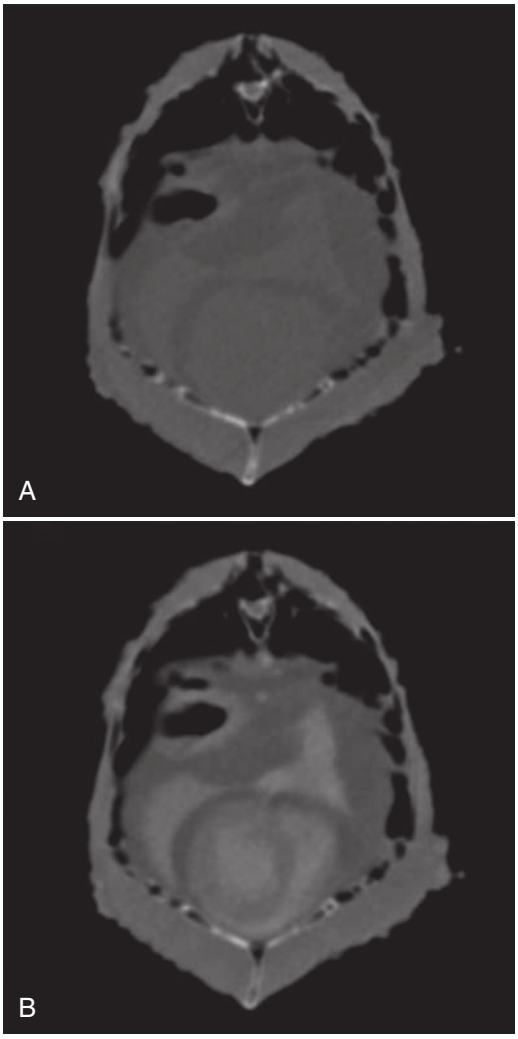


FIGURE 6-162 Transverse CT images of a 17-year-old female Moluccan cockatoo (*Cacatua molucensis*) that was presented with lethargy and a distended coelom. In image **(A)** the contours of the heart and liver can vaguely be distinguished. After intravenous administration of a contrast medium, the liver, proventricular wall, and heart can more easily be distinguished from the ascites. (Photos courtesy the Division of Diagnostic Imaging, Faculty of Veterinary Medicine, Utrecht University.)

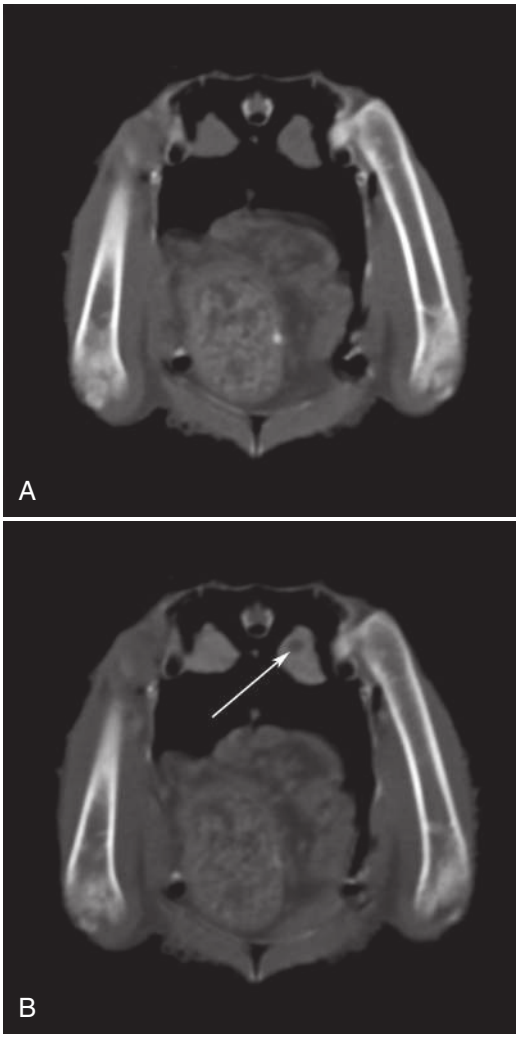


FIGURE 6-163 Transverse CT images of a 4-year-old male green-winged macaw (*Ara chloroptera*) that was presented for CT for evaluation of the heart and lungs. As a coincidental finding, a kidney cyst *(arrow)* was detected after the administration of an intravenous contrast medium **(B),** but this cyst was not visible on the image obtained without contrast medium **(A)**. (Photos courtesy the Division of Diagnostic Imaging, Faculty of Veterinary Medicine, Utrecht University.)

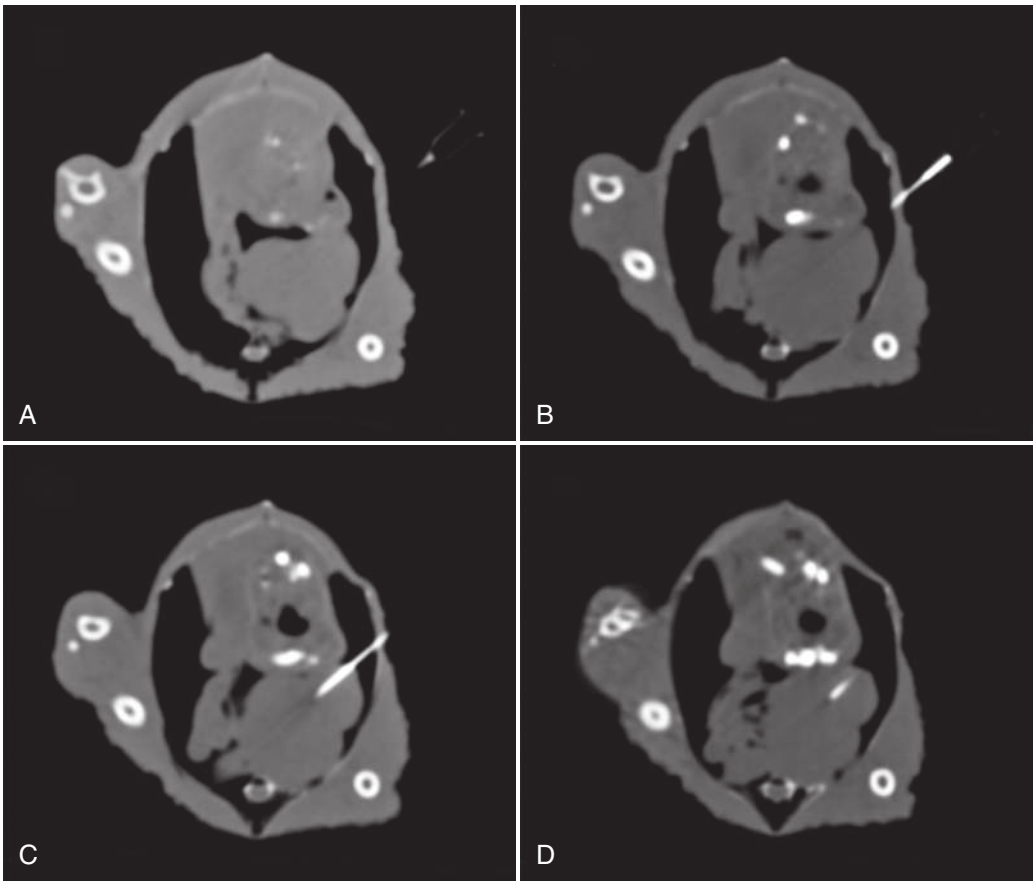


FIGURE 6-164 A series of four consecutive transverse CT images of a 14-year-old female African grey parrot (*Psittacus erithacus*) that underwent CT scanning because of seizures. As an incidental finding a mass with a diameter of 2.4 cm was found in the left caudal thoracic air sac. A CT-guided fine-needle aspiration biopsy was taken, confirming this mass to be an ovarian tumor. (Photos courtesy the Division of Diagnostic Imaging, Faculty of Veterinary Medicine, Utrecht University.)

Association (NEMA). Although software programs such as Adobe Photoshop can be used to view individual images, specific DICOM programs are needed to make optimal use of all the information stored within the DICOM files. The most popular freeware DICOM program is OsiriX (www.osirix-viewer.com), which runs on the Macintosh platform. A small selection of other freeware programs are Osiris (http://www.softpedia.com/get/Science-CAD/Osiris-Viewer.shtml), ezDICOM (http://www.mccauslandcenter.sc.edu/mricro/ezdicom/), and MRIcron (http://www.mccauslandcenter.sc.edu/mricro/mricron/index.html). With these programs multiplanar reconstructions (MPR) and three-dimensional (3D) image reconstructions and renderings are possible (Bushberg *et al.*, 2012). With multiplanar reconstruction, the organs and structures can be reviewed in three different planes (i.e., sagittal, longitudinal, transverse; see Fig. 6-165). Although all images are in 2D, the combination of the three planes allows for a 3D reconstruction in the mind of the person evaluating the images. These software programs also allow for the performance of different types of measurements (e.g., size, volume, density).

Applications

Although large abnormalities in the respiratory tract may be identified by conventional radiographs, many early and smaller lesions may be missed or misinterpreted (Krautwald-Junghanns *et al.*, 1993; Romagnano and Krautwald-Junghanns, 1997; Westerhof *et al.*, 2005). CT images allow for cross-sectional evaluation of the homogenous lungs, thereby providing detailed information about alterations of the respiratory tract in birds such as increased (multi)focal radiopacity (e.g., in case of mycotic or mycobacterial pneumonia) or decreased volume of the lung parenchyma (e.g., as is typical for lung fibrosis) (Fig. 6-166) (Amann *et al.*, 2007a; Krautwald-Junghanns, 1997; Krautwald-Junghanns *et al.*, 1998; Romagnano and Krautwald-Junghanns, 1997; Westerhof *et al.*, 2005). In addition to imaging abnormalities in the lung parenchyma, CT also enables visualization of abnormalities of the trachea (e.g., intraluminal or extraluminal masses, strictures, stenosis, or displacement), syrinx, bronchi, and air sacs (e.g., overinflation, air sacculitis, empyema, and compression by ascites, eggs, or intracoelomic masses) (Fig. 6-167) (Krautwald-Junghanns, 1997; Krautwald-Junghanns *et al.*, 1993; Krautwald-Junghanns and Pees, 2010).

Conventional radiographs are usually adequate for diagnosing the majority of cases that affect the skeletal system (e.g., fractures). In the case of spinal fractures, however, more than half of these types of fractures may be missed; however, CT offers an increased sensitivity for visualization and identification (Whittington *et al.*, 2008). Other examples of areas where CT shows superior imaging quality over conventional radiographs are the shoulder joint (see Fig. 6-160), pelvis (where much superimposition of overlying soft tissue structures and wings or legs is present; Fig. 6-168), and skull (in particular the hyoid

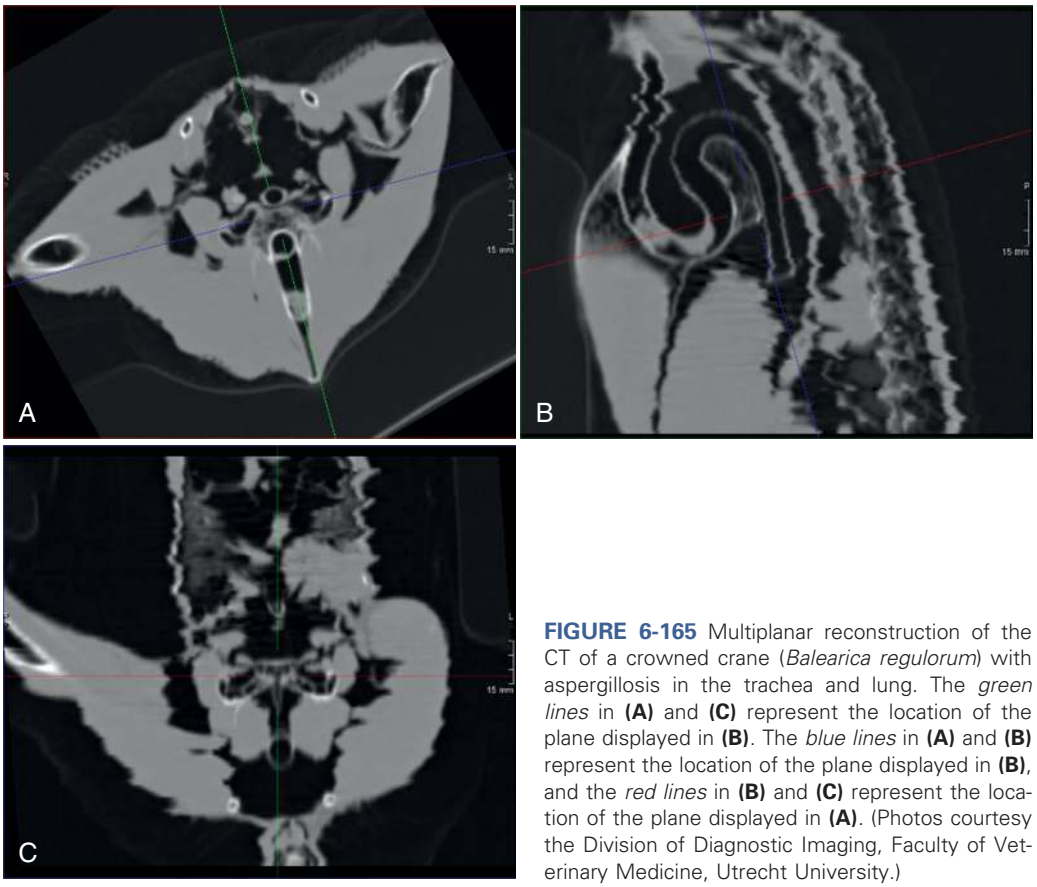


FIGURE 6-165 Multiplanar reconstruction of the CT of a crowned crane (*Balearica regulorum*) with aspergillosis in the trachea and lung. The *green lines* in **(A)** and **(C)** represent the location of the plane displayed in **(B)**. The *blue lines* in **(A)** and **(B)** represent the location of the plane displayed in **(B)**, and the *red lines* in **(B)** and **(C)** represent the location of the plane displayed in **(A)**. (Photos courtesy the Division of Diagnostic Imaging, Faculty of Veterinary Medicine, Utrecht University.)

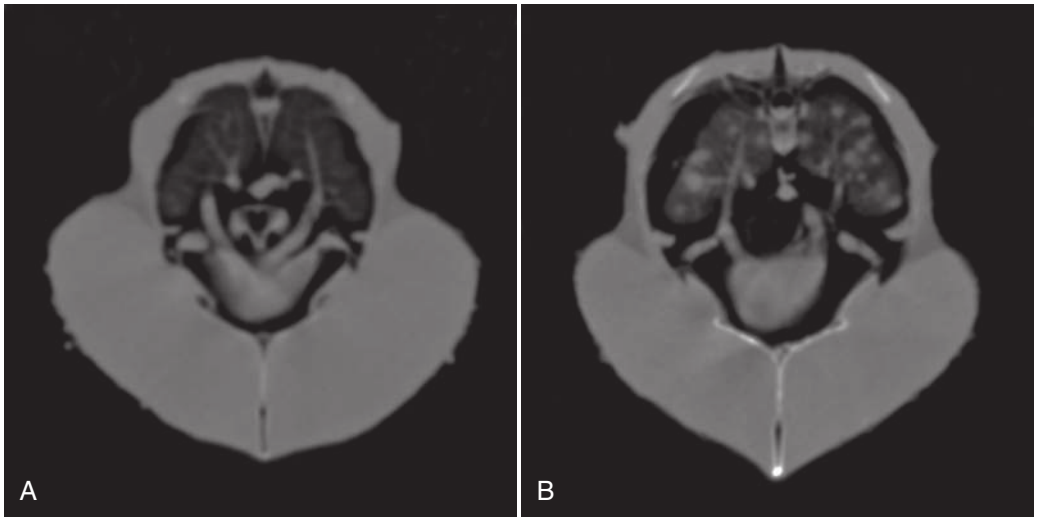


FIGURE 6-166 A selection of transverse CT images of birds with a variety of lung pathology. The following diagnoses were made: normal lung **(A)**, multifocal mycotic pneumonia **(B)**; lung cyst

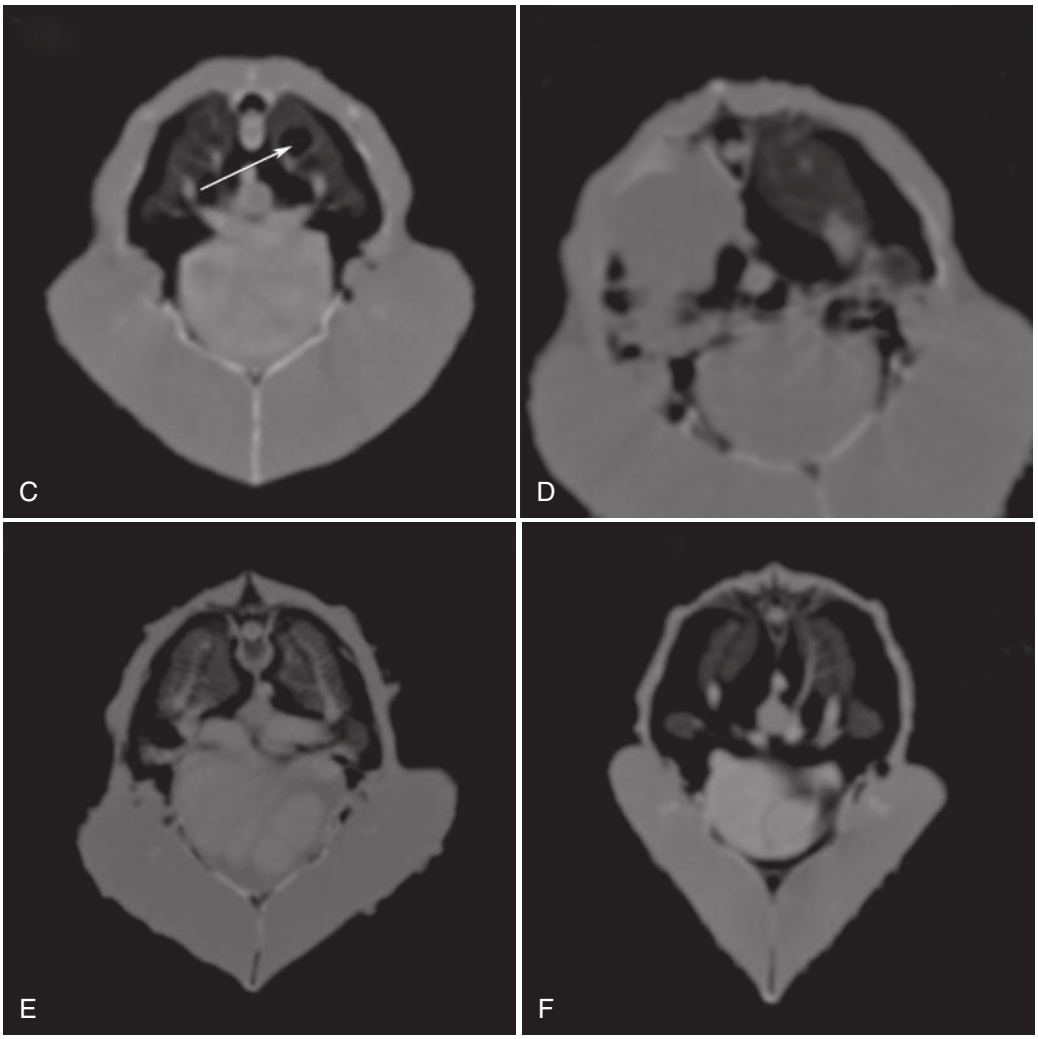


FIGURE 6-166, cont'd **(C)**; lung tumor **(D)**; lung edema **(E)**; lung fibrosis **(F)**. (Photos courtesy the Division of Diagnostic Imaging, Faculty of Veterinary Medicine, Utrecht University.)

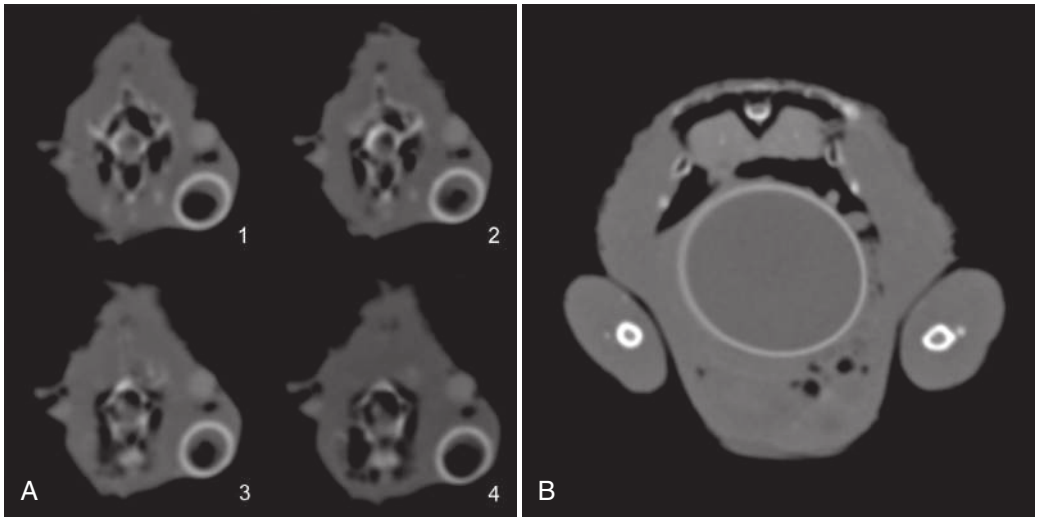


FIGURE 6-167 A selection of abnormalities that can be diagnosed with the aid of CT imaging. **(A),** A series a 4 consecutive CT images show a partial tracheal obstruction. **(B),** The lining of an egg is seen. Obviously, this would have been visible on a regular radiograph as well.

Continued

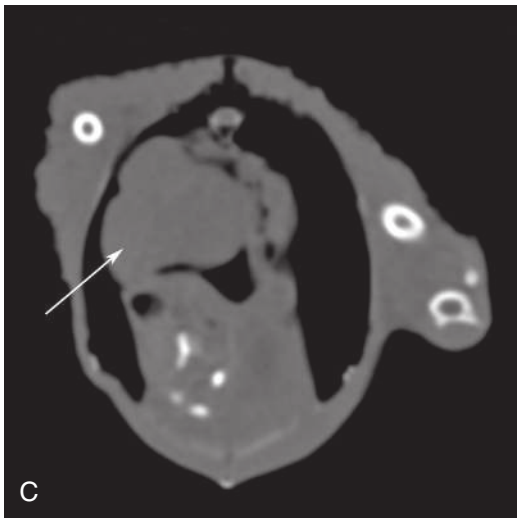


FIGURE 6-167, cont'd (C), A mass in the left dorsal part of the coelom was found *(arrow)*. With a fine-needle aspiration (see Fig. 6-164 where the bird was placed in dorsal recumbency) this mass was confirmed to be an ovarian tumor. (Photos courtesy the Division of Diagnostic Imaging, Faculty of Veterinary Medicine, Utrecht University.)

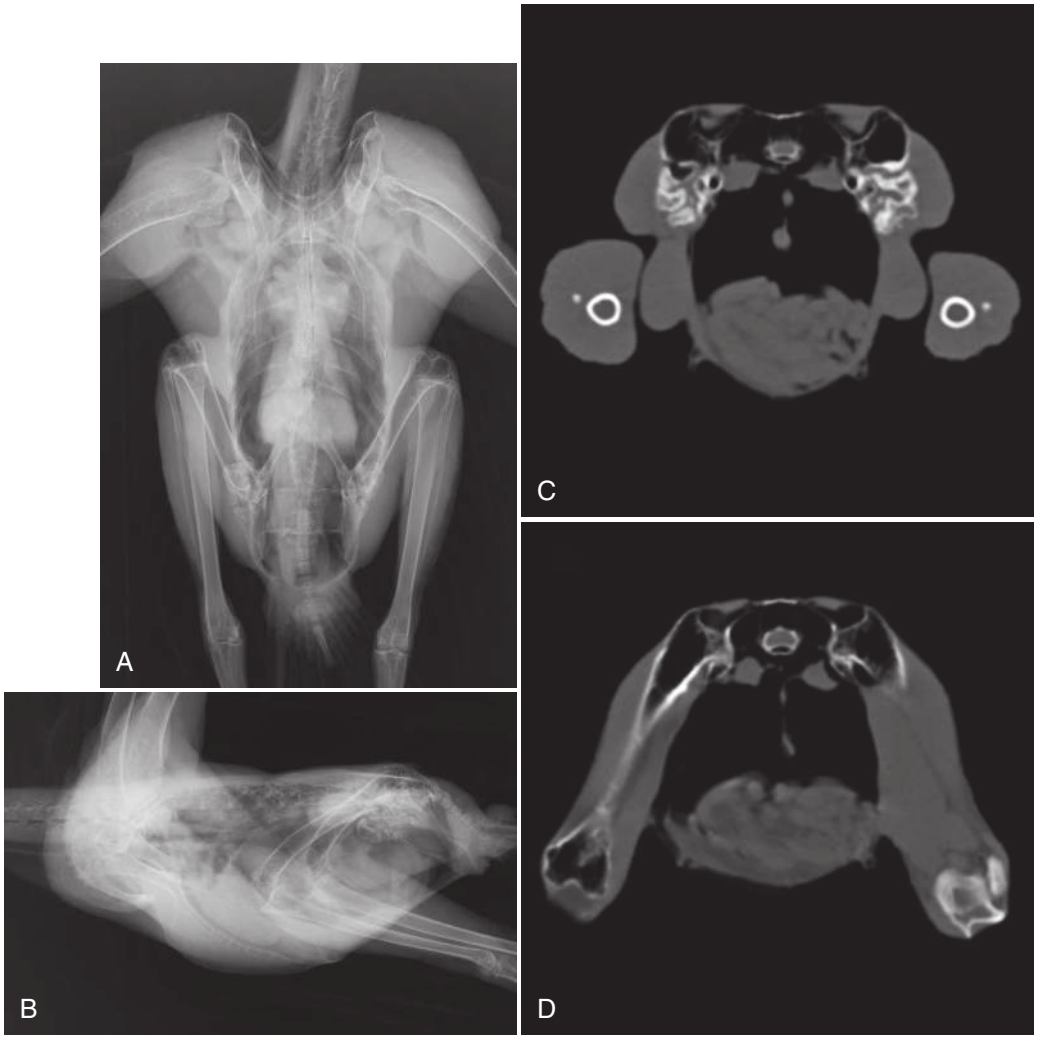


FIGURE 6-168 A 4-year-old, male hooded vulture (*Necrosyrtes monachus*) was presented with bilateral lameness. On radiographs no clear abnormalities could be noticed, aside from the fact that the legs could not be extended to allow for correct positioning **(A, B)**. On the transverse CT images it became clear that a significant amount of bone formation was present around the hip joints **(C)**. As a comparison, a CT scan was performed on another hooded vulture from the same owners, which clearly demonstrated what a normal hip joint should look like **(D)**. (Photos courtesy the Division of Diagnostic Imaging, Faculty of Veterinary Medicine, Utrecht University.)

bone and beak apparatus) (Amann *et al.*, 2007b; Gumpenberger *et al.*, 2001; Krautwald-Junghanns and Pees, 2010; van Zeeland *et al.*, 2009).

Enlargement of various intracoelomic organs, such as the liver, kidney, and spleen, may easily be detected with CT imaging, especially when contrast medium is used (Gumpenberger *et al.*, 2001). CT may furthermore be useful in determining the size and extent of masses of various origins and locations, thereby also helping determine and plan the possibilities for therapeutic intervention and the associated prognosis (Amann *et al.*, 2007b; Beaufrère *et al.*, 2010; Graham *et al.*, 2003; Spaulding and Loomis, 1999).

MAGNETIC RESONANCE IMAGING

MRI is a more recent cross-sectional imaging modality that is superior in soft-tissue imaging compared with CT. This imaging modality uses a powerful magnetic field in combination with high-frequency radio waves to align protons (i.e., nuclei of hydrogen atoms) in the body and obtain signals resulting from the change in movement of these atoms (Wallack, 2007). By changing the parameters of the MRI scanner, different tissue weightings are obtained, which are subsequently used to analyze the tissue composition. The three types of weighting that are commonly used are the T1 weighted images (T1) in which fat can be differentiated from water, with water showing a darker and fat a lighter color; T2 weighted images (T2) whereby fat shows a darker and water a lighter color; and the less-commonly-used (Ludewig and Krautwald-Junghanns, 2010; Wallack, 2007) proton-density-weighted images (PDW), in which tissues with a high protein density are seen brightest. Combining the results of the various types of MRI images is often considered useful to identify the origin of the tissue involved.

In clinical veterinary medicine, most MRI scanners have a magnetic field that ranges from 0.2 to 1.5 Tesla (T). The investigation time of an MRI with a magnetic field of less than 1.5 T, however, is so long (> 45 minutes) that CT is frequently still preferred in avian medicine when a more advanced MRI scanner is not available. Additionally, the relatively small size of most birds in combination with the relatively low spatial resolution of most systems (< 0.5 T) limit the diagnostic value that MRI has in avian patients, especially because the minimal slice thickness needs to be 1 to 3 mm to achieve a usable signal-to-noise ratio with such machines.

The use of a powerful magnetic field furthermore warrants some specific precautions before the scan. First, special anesthetic machines and monitoring equipment are needed because nothing magnetic may be taken into the scanner room. In addition, the metal microtransponders that are commonly placed in the pectoral muscle to allow identification of birds may result in signal extinction and artifacts in MRI scanners of 0.2 T. For MRI scanners with a magnet of 1.5 T, the signal is generally strong enough to overcome the noise from the minor amounts of metal in these transponders, thereby no longer posing a problem.

Applications

MRI is considered particularly useful for visualizing and evaluating the various parts of the central nervous system (CNS) in birds, including the cerebral hemispheres (Fig. 6-169), cerebellum, optic chiasm, brainstem, and spinal cord (Romagnano *et al.*, 1996). Among the diseases that have been diagnosed on MRI examination are (viral) encephalitis, lead poisoning, vestibular disease, hydrocephalus, ischemic stroke, spinal cord trauma, and a peripheral nerve sheath tumor (Beaufrère *et al.*, 2011; Delk *et al.*, 2014; Fleming *et al.*, 2003; Keller *et al.*, 2011; Redig *et al.*, 2010; Romagnano *et al.*, 1996; Stauber *et al.*, 2007; Wernick *et al.*, 2014; Whittington *et al.*, 2008). MRI is also an excellent diagnostic tool for evaluating the eye, orbit, and sinuses; identifying, localizing, and characterizing lesions in these tissues; and planning the proper therapeutic approach (Morgan *et al.*, 1994; Pye *et al.*, 2000). MRI furthermore has potential value in visualizing the GI tract, liver, spleen, and urogenital tract (Enders *et al.*, 2001; Ludewig and Krautwald-Junghanns, 2010; Romagnano *et al.*, 1996).

SCINTIGRAPHY

Scintigraphy is a 2D nuclear imaging technique with which the physiologic or pathologic function of a specifically targeted organ system or tissue is visualized. This is achieved by administering radiopharmaceuticals (i.e., technetium, iodine, thallium, gallium, xenon, and krypton) (Barber and Roberts, 1983). The radiation emitted after the decay of these compounds is subsequently captured by a gamma or scintillation camera and used to form images.

In avian medicine scintigraphy has demonstrated its potential use in avian patients with a variety of orthopedic problems (Goggin *et al.*, 2005), as well as its usefulness in the therapeutic monitoring of patients with various types of neoplasia, including diagnosing the presence of metastasis (Jones *et al.*, 2001; Lung and Ackerman, 1993; Wiley *et al.*, 2009).

SINGLE PHOTON EMISSION COMPUTED TOMOGRAPHY

SPECT is a technique that is similar to scintigraphy. It does, however, use a rotating gamma camera that enables the object to be reconstructed into a 3D image. The information is typically presented as cross-sectional slices but reconstructions can be made in different planes by manipulating the data. Although its use has been described in veterinary medicine (LeBlanc *et al.*, 2014; Martlé *et al.*, 2013), no applications have been reported in avian medicine.

POSITRON EMISSION TOMOGRAPHY

PET is similar to SPECT with the exception that with SPECT the emitted gamma radiation is measured directly, but PET tracers emit positrons that cause two gamma rays to be emitted in opposite directions. By detecting these "coincident pairs" a more accurate localization of the origin of radiation can be achieved (Myers and Hume, 2002). The fact that most PET systems are sold integrated with a CT scanner further enhances the exact localization of the lesion (Bushberg *et al.*, 2012).

The most commonly used radiopharmaceutical agent in PET imaging is ^{18}F-fluorodeoxyglucose. The characteristics of this specific tracer make the technique particularly important in the diagnosis of brain disease and monitoring of cancer treatments.

A number of applications in clinical avian medicine have been described, which include the use of PET for planning and monitoring cancer therapy, determining the "activity" of lesions/nodules, and diagnosing fractures of the skull and/or luxations of facial bones (Grunkemeyer *et al.*, 2010; Souza *et al.*, 2006, 2008).

CONCLUSION

With the advancement of imaging modalities the possibilities for diagnosing disease have increased tremendously. CT imaging should be considered by each practitioner as a readily available technique to noninvasively visualize respiratory lesions. Now that 1.5T MR scanners are being installed at veterinary diagnostic imaging centers, this technique should certainly also be considered for the avian patient. The use of scintigraphy, SPECT, and PET imaging will remain restricted to

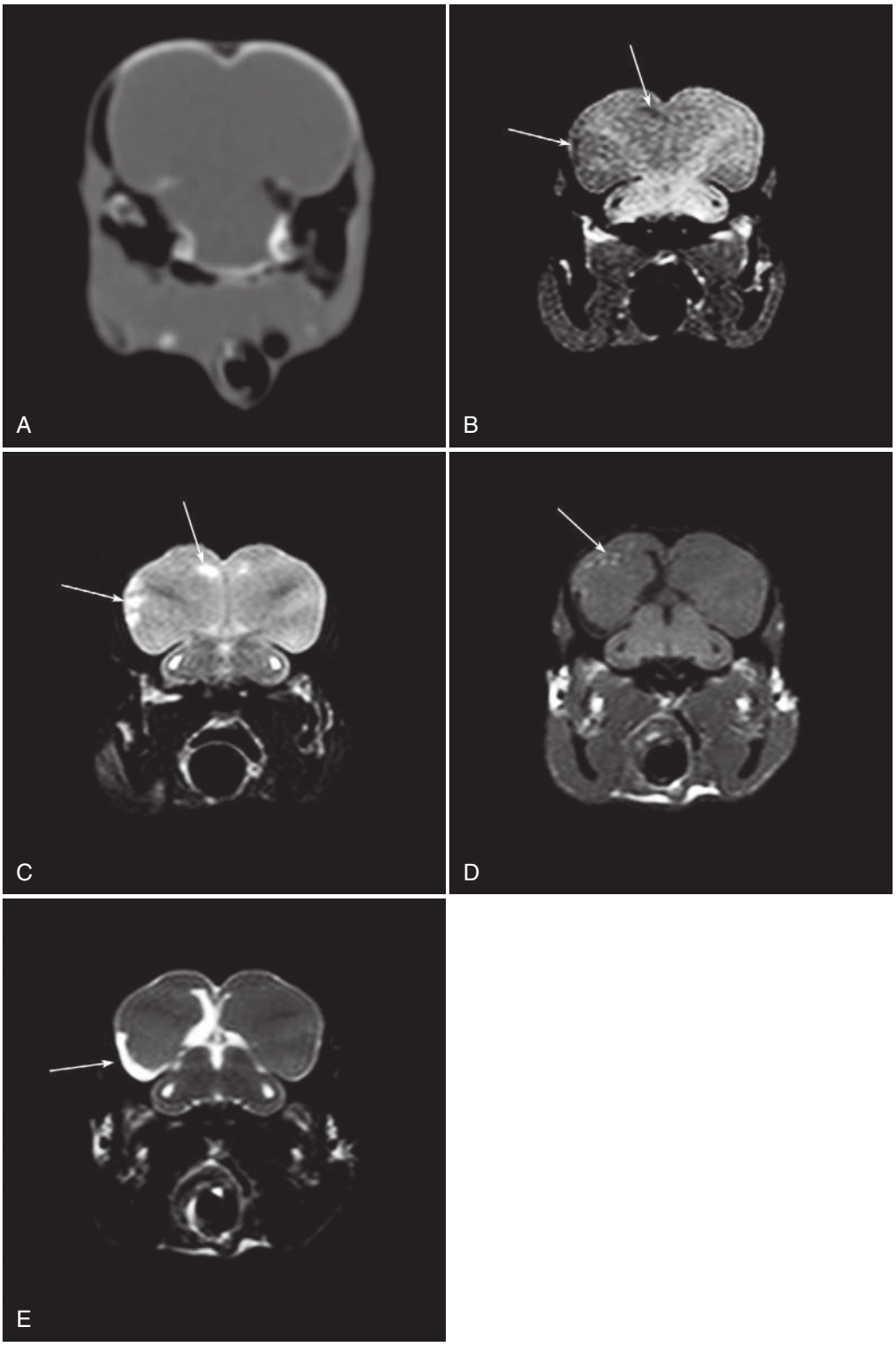


FIGURE 6-169 Advanced diagnostic imaging of a 22-year-old, male African grey parrot (*Psittacus erithacus*) that was presented with weakness in the legs, falling off the perch, and signs of aphasia. On the transverse CT image of the brain **(A)** no abnormalities can be seen. On the MRI images, however, multiple focal reactions in the right hemisphere can be seen *(arrows:* black on T1 **[B]** and white on T2 **[C]**). Although the parrot improved during the following 11 weeks, it continued to have episodes of weakness. On the T1 weighted MRI image, signs of recent hemorrhages could be seen (**D:** *arrow*), but on the T2 weighted image an accumulation of fluid around the right hemisphere could be detected (**E:** *arrow*). (Photos courtesy the Division of Diagnostic Imaging, Faculty of Veterinary Medicine, Utrecht University.)

university clinics for some time but should certainly be considered as advanced imaging techniques that may potentially be used in specific cases.

REFERENCES

Amann O, Kik MJL, Passon-Vastenburg MHAC, et al: Chronic pulmonary interstitial fibrosis in a blue-fronted amazon parrot (*Amazona aestiva aestiva*), *Avian Dis* 51:150–153, 2007a.

Amann O, Meij BP, Westerhof I, et al: Giant cell tumor of the bone in a scarlet macaw (*Ara macao*), *Avian Dis* 51:146–149, 2007b.

Barber D, Roberts R: Imaging: nuclear, *Vet Radiol Ultrasound* 24:50–57, 1983.

Bartels T, Krautwald-Junghanns ME, Portmann S, 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 Pathology* 29:101–108, 2000.

Beaufrère H, Castillo-Alcala F, Holmberg DL, et al: Branchial cysts in two Amazon parrots (*Amazona* species), *J Avian Med Surg* 24:46–57, 2010.

Beaufrère H, Nevarez J, Gaschen L, et al: Diagnosis of presumed acute ischemic stroke and associated seizure management in a Congo African grey parrot, *J Am Vet Med Assoc* 239:122–128, 2011.

Bushberg JT, Seibert JA, Leidholdt EM Jr, et al: *The essential physics of medical imaging*, Philadelphia, 2012, Wolters Kluwer Health/Lippincott Williams & Wilkins.

Delk KW, Mejia-Fava J, Jiménez DA, et al: Diagnostic imaging of peripheral vestibular disease in a Chinese goose (*Anser cygnoides*), *J Avian Med Surg* 28:31–37, 2014.

Enders F, Jurina K, Straub J, et al: Magnetic resonance imaging in birds. *Proceedings of the European Association of Avian Veterinarians*, Munich, Germany, 2001, pp 87–91.

Fleming GJ, Lester NV, Stevenson R, et al: High field strength (4.7T) magnetic resonance imaging of hydrocephalus in an African grey parrot (*Psittacus erithacus*), *Vet Radiol Ultrasound* 44:542–545, 2003.

Goggin JM, Hoskinson JJ, Carpenter JW, et al: Scintigraphic assessment of digital extremity perfusion in 17 patients, *Vet Radiol Ultrasound* 38:211–220, 2005.

Graham JE, Wetrner JA, Lowenstine LJ, et al: Periorbital liposarcoma in an African grey parrot (*Psittacus erithacus*), *J Avian Med Surg* 17:147–153, 2003.

Grunkemeyer VL, Jones MP, Greenacre CB, et al: Humeral air sac cystadenocarcinoma in a Moluccan cockatoo (*Cacatua moluccencis*) monitored via serial ^{18}F-fluorodeoxyglucose integrated positron emission tomography—computed tomography scans. *Proceedings of the Association of Avian Veterinarians*, San Diego, California, 2010, pp 343–344.

Gumpenberger M, Henninger W: The use of computed tomography in avian and reptile medicine, *Seminars in Avian and Exotic Pet Medicine* 10:174–180, 2001.

Jenkins J: Use of computed tomography (CT) in pet bird practice. *Proceedings of the Association of Avian Veterinarians*, Chicago, 1991, pp 276–279.

Jones MP, Orosz SE, Richman LK, et al: Pulmonary carcinoma with metastasis in a Moluccan cockatoo (*Cacatua moluccencis*), *J Avian Med Surg* 15:107–113, 2001.

Keller KA, Sanchez-Migallon Guzman D, Muthuswamy A, et al: Hydrocephalus in a yellow-headed amazon parrot (*Amazona ochrocephala oratrix*), *J Avian Med Surg* 25:216–224, 2011.

Krautwald-Junghanns ME: *Computertomographie des aviären Respirationstraktes*, Reihe: Akademisches Forum, Berlin, Germany, 1997, Blackwell Wissenschafts-Verlag.

Krautwald-Junghanns ME, Kostka VM, Dörsch 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* 12:149–157, 1998.

Krautwald-Junghanns ME, Pees M: Computed tomography (CT). In Krautwald-Junghanns ME, Pees M, Reese S, Tully T, editors: *Diagnostic imaging of exotic pets*, Hannover, 2010, Schlütersche, pp 54–63.

Krautwald-Junghanns ME, Schuhmacher F, Tellehlm B: Evaluation of the lower respiratory tract in psittacines using radiology and computed tomography, *Vet Radiol Ultrasound* 34:382–390, 1993.

LeBlanc AK, Peremans K: PET and SPECT imaging in veterinary medicine, *Semin Nucl Med* 44:47–56, 2014.

Ludewig E, Krautwald-Junghanns ME: Magnetic resonance imaging (MRI). In Krautwald-Junghanns ME, Pees M, Reese S, Tully T, editors: *Diagnostic imaging of exotic pets*, Hannover, 2010, Schlütersche, pp 64–69.

Lung N, Ackerman N: Scintigraphy as a tool in avian orthopedic diagnosis. *Proceedings of the Association of Avian Veterinarians*, Nashville, Tennessee, 1993, p 45.

Mackey EB, Hernandez-Divers SJ, Holland M, et al: Clinical technique: application of computed tomography in zoological medicine, *J Exot Pet Med* 17:198–209, 2008.

Martel A, van Caelenberg A, Gielen I, et al: Computed tomographic (CT) anatomy of the pigeon (*Columba livia*). *Proceedings of the Association of Avian Veterinarians*, 2005, Monterey, pp 373–374.

Martlé V, Peremans K, van Ham L, et al: High-resolution micro-SPECT to evaluate the regional brain perfusion in the adult beagle dog, *Res Vet Sci* 94:701–706, 2013.

Morgan RV, Donnell RL, Daniel GB, et al: Magnetic resonance imaging of the normal eye and orbit of a screech owl (*Otus asio*), *Vet Radiol Ultrasound* 35:362–367, 1994.

Myers R, Hume S: Small animal PET, *Eur Neuropsychopharmacol* 12:545–555, 2002.

Orosz SE, Toal RL: Tomographic anatomy of the golden eagle (*Aquila chrysaetos*), *J Zoo Wildl Med* 23:39–46, 1992.

Pye GW, Bennet RA, Newell SM, et al: Magnetic resonance imaging in psittacine birds with chronic sinusitis, *J Avian Med Surg* 14:243–256, 2000.

Redig PT, Nicolas de Francisco O, Armièn AG, et al: Use of MRI and histological methods for detection of lead-induced lesions in the CNS of bald eagles. *Proceedings of the Association of Avian Veterinarians*, 2010, San Diego, pp 33–34.

Romagnano A, Krautwald-Junghanns ME: Respiratory radiology and imaging. *Proceedings of the Association of Avian Veterinarians*, Minneapolis, Minnesota, 1997, pp 17–21.

Romagnano A, Shiroma JT, Heard DJ, 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.

Silverman S, Dennison S: Imaging in special species. *Proceedings of the Association of Avian Veterinarians*, 2010, San Diego, pp 237–246.

Souza MJ, Greenacre CB, Jones MP, et al: Clinical use of MicroPET and CT imaging in birds. *Proceedings of the Association of Avian Veterinarians*, San Antonio, Texas, 2006, pp 13–16.

Souza MJ, Newman SJ, Greenacre CB, et al: Diffuse intestinal T-cell lymphosarcoma in a yellow-naped Amazon parrot (*Amazona ochrocephala auropalliata*), *J Vet Diagn Invest* 20:656–660, 2008.

Spaulding K, Loomis M: Principles and applications of computed tomography and magnetic resonance imaging in zoo and wildlife medicine. In Fowler M, Miller R, editors: *Zoo and wild animal medicine: current therapy 4*, Philadelphia, 1999, WB Saunders, pp 83–88.

Stauber E, Holmes S, Deghetto DL, et al: Magnetic resonance imaging is superior to radiography in evaluating spinal cord trauma in three bald eagles (*Haliaeetus leucocephalus*), *J Avian Med Surg* 21:196–200, 2007.

Wallack S: Basic magnetic resonance imaging principles used for evaluating animal patients with neurologic disease, *Vet Clin North Am Exot Anim Pract* 10:909–925, 2007.

Wernick MB, Dennler M, Beckmann K, et al: Peripheral nerve sheath tumor in a sub-adult golden eagle (*Aquila chrysaetos*), *J Avian Med Surg* 28:57–63, 2014.

Westerhof I, Schoemaker NJ, Barthez P, et al: Spiral computed tomography in respiratory diseases in birds. *Proceedings of the European Association of Avian Veterinarians*, 2005, Arles, pp 27–28.

Whittington JK, Osterbur KA, O'Dell-Anderson K: Imaging modalities and limitations in diagnosing spinal fractures of birds. *Proceedings of the Association of Avian Veterinarians*, 2008, Savannah, pp 353–359.

Wiley JL, Whittington JK, Wilmes CM, et al: Chronic myelogenous leukemia in a great horned owl (*Bubo virginianus*), *J Avian Med Surg* 23:36–43, 2009.

Zeeland van YRA, Schoemaker NJ, Veraa S, et al: Femoral head resection in a hooded vulture (*Necrosyrtus monachus*). *Proceedings of the Association of Avian Veterinarians*, Milwaukee, Wisconsin, 2009, pp 349–350.

ENDOSCOPY

Jaime Samour

Endoscopy (Greek: *endon* = within + *skopein* = to examine) is the direct visual inspection of any cavity of the body and organs using an endoscope (Table 6-35). Endoscopy was first used in avian medicine as a means of determining the sex of monomorphic species. One of the earliest reports concerning the use of an endoscope for sex determination in birds during the 1940s and 1950s was made by Hauser (1977). Dr. Hauser, a physician from Tacoma, Washington, was a devoted and passionate aviculturalist. On his farm he kept and bred cranes, waterfowl, and gallinaceous birds. In this report he described the technique of cloacal examination using a handheld, battery-operated otoscope commonly used in human practice. During the late 1960s and 1970s, human otoscopes and proctoscopes were used by field biologists and zoologists for sex determination in penguins through cloacal examination (Ainley, 1970; Le Resche, 1971; Sladen, 1978). Bailey (1953) was probably the first to report the use of an otoscope inserted into the body through a small incision to determine sex and assess reproductive status. Subsequently, endoscopic examination of the gonads through a small laparotomy and the use of otoscopes became an established procedure in avian medicine (Risser, 1971; Ingram, 1977, 1978, 1980; Harlin, 1996). However, it was not until 1977 that the first report on sex determination in avian species using rigid endoscopes appeared in the literature (Satterfield and Altman, 1977). The technique was based on the use of endoscopes fitted with a rigid rod-lens system and the illumination was provided through a fiberoptic cable attached to a powerful light source. Nowadays, endoscopy is a very well-established medical procedure in avian medicine as a diagnostic technique for sex determination of monomorphic species and to carry out minor surgical procedures (Bush, 1978, 1980; Bush *et al.*, 1978; Harrison, 1978, 1986; Satterfield, 1980; Burr *et al.*, 1981; McDonald, 1982, 1987, 1996; Jones *et al.*, 1984; Kollias, 1988; Taylor, 1989, 1990, 1992, 1994; Samour, 1991; Lierz, 2006; Lierz 2008) (Figs. 6-170 and 6-171).

EQUIPMENT AND INSTRUMENTATION

Handheld battery-operated otoscopes and tubular endoscopes are still favored by many practitioners. These represent a more modest investment and offer the advantage that they can be used under field conditions. However, rod-lens endoscopy technology is far superior, providing a higher-resolution image, a wider angle of view, and increased illumination. Moreover, high-quality still photography and video imaging are more viable propositions through rod-lens endoscopes, although this is also possible with some of the most sophisticated tubular endoscopes.

Great advances have been made in the past 25 years in the production of endoscopy equipment suitable for avian use. The choice of equipment is directly related to the different applications and the size of the avian patient. Table 6-36 lists equipment and instruments for avian endoscopy.

TABLE 6-35 Common Endoscopy Applications in Avian Medicine

Application	Anatomic Site
Otoscopy or auriscopy	External auditory canal
Rhinoscopy	Cranial sinuses
Pharyngoscopy	Oropharynx
Tracheoscopy	Trachea
Ingluvioscopy	Crop
Esophagoscopy	Esophagus
Gastroscopy	Proventriculus, ventriculus
Celoscopy/laparoscopy	Coelomic cavity
Cloacoscopy	Cloaca

CLINICAL AND SURGICAL APPLICATIONS

Clinical Examination

Indications for clinical endoscopy examination are given in Table 6-37. The different techniques for endoscopic examination of the avian patient are facilitated by the existence of air sacs, the cloaca, and, in most species, a crop. Rigid endoscopes are ideal for examining the coelomic cavity, the upper digestive and respiratory systems, and the cloaca. Flexible endoscopes are also useful and offer several advantages over rigid endoscopes in the clinical examination of the upper digestive and respiratory tracts. For instance, the retrieval of foreign objects from the gizzard of birds such as penguins, waterfowl, and birds of prey is greatly facilitated by using a flexible endoscope. Handheld

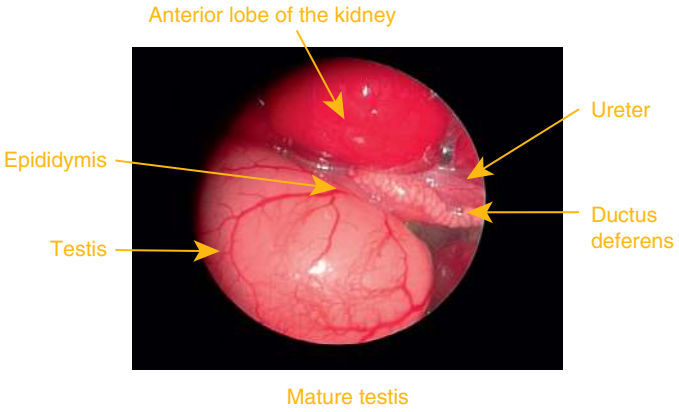


FIGURE 6-170 Endoscopy view of a well-developed testis and ductus deferens of an adult domestic pigeon (*Columba livia*) during the nuptial phase of the gonadal cycle.

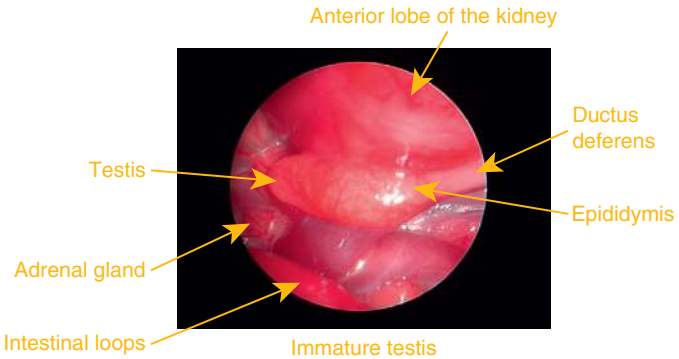


FIGURE 6-171 A similar view of the testis of an immature domestic pigeon (*Columba livia*).

TABLE 6-36 Equipment and Instruments for Avian Endoscopy

Equipment	Specifications	Description
DIAGNOSIS AND EXAMINATION		
Rigid endoscope (selfoscope) angle of view	1.2, 1.7 mm outer diameter	67-114 mm working length; 0 degrees and 30 degrees
Rigid endoscope (needlescope, arthroscope)	1.9, 2.2, 2.5, 2.7, 3, 4, 5 mm outer diameter	170-190 mm working length; 0-degree and 30-degree angle of view
Flexible endoscope	1.6, 2.4, 3, 3.5, 5 mm outer diameter	200, 255, 365, 380, 450 mm working length; 0-degree angle of view; 0.6, 1.2, 2.2 mm working channel
Proctoscopes	12, 18, 21 mm outer diameter	100-120 mm working length
Fiberoptic cable	1800, 2300 mm length	1-3.5 mm fiber bundle diameter
Light source	Halogen or xenon high-intensity light; lamps 150, 250, 300, 400 W	Adjustable light intensity, standby lamp, endoflash
Second viewer or teaching attachment		Rigid or flexible attachment to allow viewing by second operator
PHOTOGRAPHY AND VIDEO IMAGING		
Still camera	35-mm SLR camera, automatic	Camera adaptor for rigid and flexible endoscopes
Video camera	Camera controller; PAL and NTSC color systems; lens 21-38 mm focal distance	Camera adaptor for rigid and flexible endoscopes
Color video monitor	9, 14, 20" screen; resolution 450-700 lines	Full-size image, 4 or 16 split, thermo sublimations print system
Color video printer	S-VHS or Betacam SP video recorder	Video recorder
BIOPSY AND SURGERY		
Operating rigid endoscopes	2.7, 5, 10 mm diameter, straight, obliquely offset, or right-angled eyepiece endoscopes	250-300 mm working length; 0- degree angle of view; 1.8-5 mm working channel
Rigid instruments	Biopsy forceps, grasping forceps, injector, scissors, needle holder, clamps	Conventional, bipolar
Flexible instruments	Biopsy forceps (fenestrated, ellipsoid, alligator), cytology brush, grasping forceps (basket, sharp-toothed), injector	Conventional
Irrigation and suction pump	Irrigation and suction tubes	Vacuum 65 kPa ± 10%, pressure 200 kPa ± 10%, aspiration capacity 3.5 L/m
Radiosurgery system	3.8 or 4 MHz dual radiofrequency unit, foot pedal, monopolar, and bipolar leads	
Laser system	Diode and Co_2 units, 400- and 600-mm conical and flat-tipped diode laser fibers, semirigid Co_2 laser ceramic probes	
CLEANING AND MAINTENANCE		
Automatic cleaning and disinfecting units		For rigid and flexible endoscopes
Automatic maintenance units		For flexible endoscopes
Trays	Disinfecting, sterilization, storage	For rigid endoscopes
Brushes	Cleaning	Working channels for rigid and flexible endoscopes

kPa, Kilopascal; MHz, megahertz; NTSC, National Television System Committee; PAL, Phase Alternating Line; S-VHS, super video home system; SLR, single-lens reflex; SP, superior performance.

battery-operated otoscopes and tubular endoscopes are ideal for examination of the upper digestive and respiratory systems and the cloaca in the most common cage and aviary birds (Fig. 6-172). Proctoscopes or vaginoscopes, commonly used in human medicine, that are fitted with fiberoptic lighting are the instruments of choice for cloacal examination in penguins, waterfowl, and ratites.

Surgical Applications

There is a vast range of surgical procedures carried out in human medicine that use endoscopy techniques, thus minimizing trauma and recovery time. In avian medicine the benefits of conducting surgery through endoscopes are just beginning to be explored. However, some advances have been made in recent years. The removal, with the aid of

TABLE 6-37 Indications for Clinical Endoscopy Examination

Endoscopy Examination	Indication
Otoscopy or auriscopy	Ectoparasites, foreign bodies, infections
Rhinoscopy	Trichomonosis, candidiasis, other infections
Pharyngoscopy	Trichomonosis, candidiasis, general infections, foreign bodies
Tracheoscopy	Trichomonosis, aspergillosis, gapeworms, foreign bodies
Ingluvioscopy	Trichomonosis, foreign bodies, general infections, retained pellets (mostly birds of prey)
Esophagoscopy	Trichomonosis, foreign bodies
Gastroscopy	Foreign bodies, impactions
Celoscopy/ laparoscopy	Sex determination, assessment of gonadal activity, monitoring of gonadal cycle, age determination, retrieval of *Serratospiculum* spp. filarial worms, diagnosis and treatment of *Aspergillus* spp. lesions, diagnosis of tuberculosis and neoplasm, surgery, biopsies, general clinical diagnosis
Cloacoscopy	Sex determination, impactions, uroliths, prolapse, infections

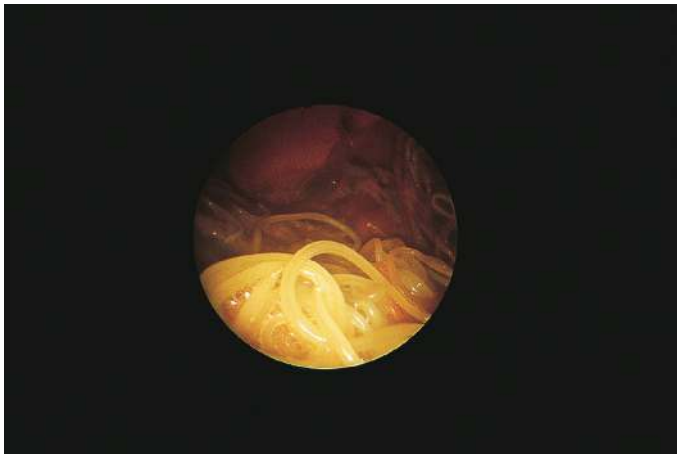


FIGURE 6-172 Large number of *Serratospiculum seurati* filarial worms in the caudal thoracic air sac of a saker falcon (*Falco cherrug*).

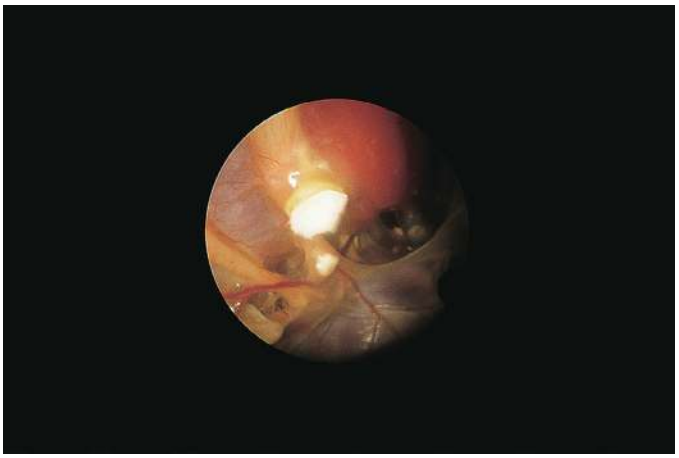


FIGURE 6-173 Typical *Aspergillus fumigatus* plaques within the coelomic cavity of a peregrine falcon (*Falco peregrinus*).

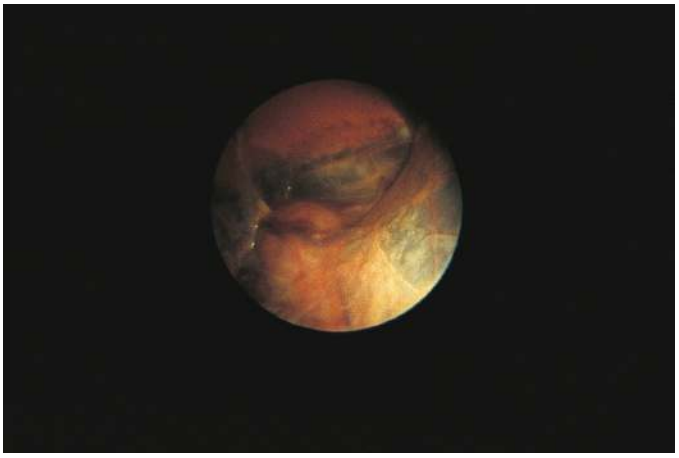


FIGURE 6-174 Air sacculitis in a gyrfalcon (*Falco rusticolus*) associated with *Aspergillus flavus* infection.

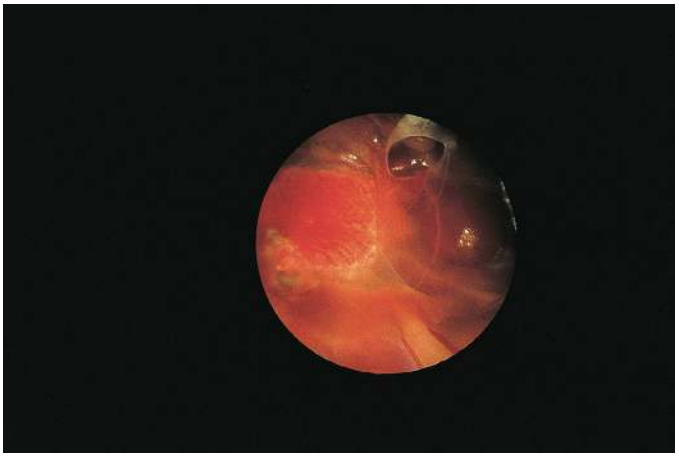


FIGURE 6-175 Severe pneumonia in a peregrine falcon. Most of the lung shows extensive congestion.

endoscopy, of *Serratospiculum seurati* adult filarial worms from the coelomic cavity of falcons (Fig. 6-173) is routinely practiced in falcon hospitals in the United Arab Emirates (Samour, 1996). Many avian practitioners treat lesions of aspergillosis on the air sacs or the coelomic cavity topically using antifungal agents in addition to parenteral therapy (Fig. 6-174). Subsequently, the lesions are removed through endoscopy. Other conditions are illustrated in Figs. 6-175 to 6-177.

Growing concern has been expressed in many countries around the world about the use of hybrid falcons in the sport of falconry. It has been suggested that a hybrid falcon could accidentally escape and crossbreed with free-living birds of closely related species. Hybrids of gyr (*Falco rusticolus*) and saker (*Falco cherrug*) falcons, as well as peregrine (*Falco peregrinus*) and gyr falcons are among the most popular used in falconry. There have been several independently confirmed cases of crossbreeding between accidentally released hybrids and free-living falcons in both North America and Europe. Vasectomy has been carried out in domestic pigeons *(Columba livia)* using endoscopy techniques (Samour, 2010). This surgical procedure consisted of sectioning

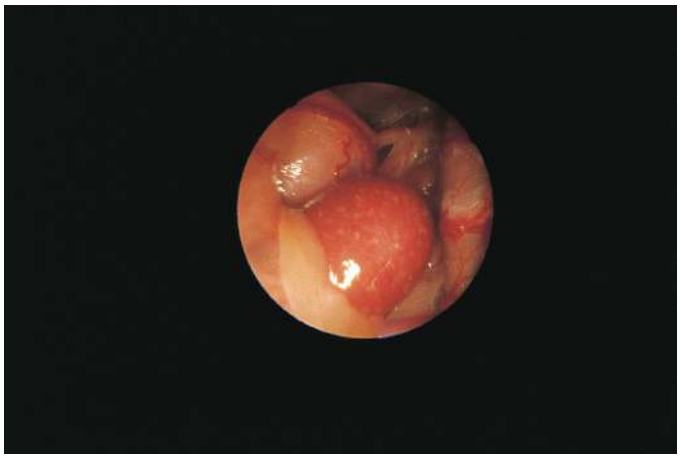


FIGURE 6-176 An enlarged spleen with "mottled" appearance in a houbara bustard (*Chlamydotis undulata*). Structural changes of the spleen have often been observed in clinically normal individuals of this species during routine endoscopy examinations. Part of an intestinal loop is visible above the spleen.

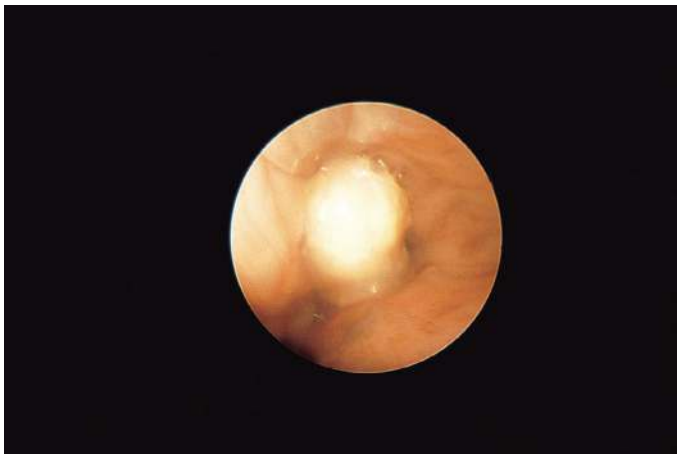


FIGURE 6-177 Large caseous mass in the esophagus of a kori bustard (*Ardeotis kori*) produced by *Trichomonas gallinae*.

and removing a 10- to 20-mm section of the vas deferens of adult, mature birds. In the adult bird, the identification and sectioning of the vas deferens is relatively easy, in particular during the breeding cycle. Great care has to be exercised during the manipulation of the surgical instruments (e.g., biopsy forceps) because of the close proximity of the vas deferens to the ureter and common iliac vein (Fig. 6-178). A technique to vasectomize immature Japanese quails *(Coturnix japonica)* was described (Jones and Redig, 2003). The single-entry endoscopy-assisted technique consisted in sectioning the vas deferens at its proximal end as it leaves the epididymis—where there is no close association with the ureter—using a biopsy forceps (Figs. 6-179 to 6-181). The vas deferens was then gently separated from the ureter by pulling carefully and completely sectioned in the middle by gentle traction. Surgical techniques to sterilize male and female birds through celiotomy have been previously described by Bennett (1993), Heidenreich (1997), and Forbes (2008a, 2008b). Endoscopy-guided surgical procedures to sterilize male (Jones and Redig, 2003; Samour, 2010) and female birds have also been described (Pye *et al.*, 2001a, 2001b; Lierz, 2004; Hernandez-Divers, 2005; Lierz and Hafez, 2005; Lierz, 2006; Lierz, 2008). It is now possible, for instance, to perform a salpingohysterectomy or a vasectomy in birds using one-, two- or three-point entry. A single-entry technique involves the use of an endoscope, commonly a 2.7-mm telescope, inserted through an operating sheath. When the vasectomy has been detected, a flexible or rigid biopsy forceps is then introduced through the port of the sheath and directed toward the surgical site. With this technique, the surgeon holds the sheath and endoscope with one hand while manipulating the forceps with the other one (Samour 2010). Using the two-point entry technique, the operator holds the endoscope with one hand while manipulating the electrosurgical unit or surgical instruments with the other one at a different entry point. Using the three-point surgical entry technique, the endoscope can be placed on a sand bag or handled by an assistant while the operator manipulates the surgical instruments and the electrosurgical unit (Lierz, 2004; Hernandez-Divers, 2005; Lierz and Hafez, 2005; Lierz, 2006; Lierz, 2008), which have been inserted via different surgical sites. Endoscopes can also be held in position using a custom-made or commercially available angle-poise device.

Experimental trials have been carried out sterilizing male birds through endoscopy-assisted injections directly into the testes using zinc gluconate neutralized by arginine (Wilson *et al.*, 2004). Two different doses, a high and a lower, were tried in this study. The authors found undesirable effects, including mortality, associated with the high dose. The lower dose did not produce any undesirable effects but there was histologic evidence suggesting the retention of reproductive ability (Wilson *et al.*, 2004). There is an obvious need for further research with this and other novel chemical products that may become available in the future.

One of the most popular surgical applications using an endoscope is endoscopy-guided biopsy collection (Taylor, 1994; McDonald, 1996; Lierz 2006; Lierz 2008). A novel endoscopy-assisted testicular biopsy technique was recently described in Psittaciformes as a means of assessing fertility (Crosta *et al.*, 2002). A similar endoscopy-assisted testicular biopsy technique involving aspiration and cytology to assess the reproductive status in male swift parrots *(Lathamus discolor)* has also been described (Gartrell, 2002). Both techniques may prove useful for establishing the reproductive potential of individuals in captive breeding programs. The indications and different techniques for the collection of biopsies are covered earlier in this chapter.

Other endoscopic surgical techniques in the avian patient include the removal of tracheal foreign bodies (Clayton and Ritzman, 2005) and in situ suction and removal of impacted and soft-shelled eggs within the oviduct (Crosta and Timossi, 2005). A staged endoscopy-assisted technique for the removal of a large foreign body lodged in the ventriculus of a gyr falcon was recently described (Lloyd, 2009). Endoscopy-assisted removal of the mass was carried out through five different ingluviotomies conducted over a period of 64 days (Lloyd, 2009).

SEX DETERMINATION OF MONOMORPHIC SPECIES

Celoscopy (Laparoscopy) for Sex Determination

The preoperative considerations for celoscopy in the avian patient are similar to those for general surgery. A full clinical examination is essential, in addition to information relating to age, diet, housing, and general management. Old and obese birds improperly housed and fed on inadequate diets are high anesthetic risks and individuals from the Falconiformes and Psittaciformes are particularly so.

The selection of anesthetic technique varies according to the species and the circumstances confronting the avian practitioner. Injectable anesthetic agents have been successfully used in more than 10,000

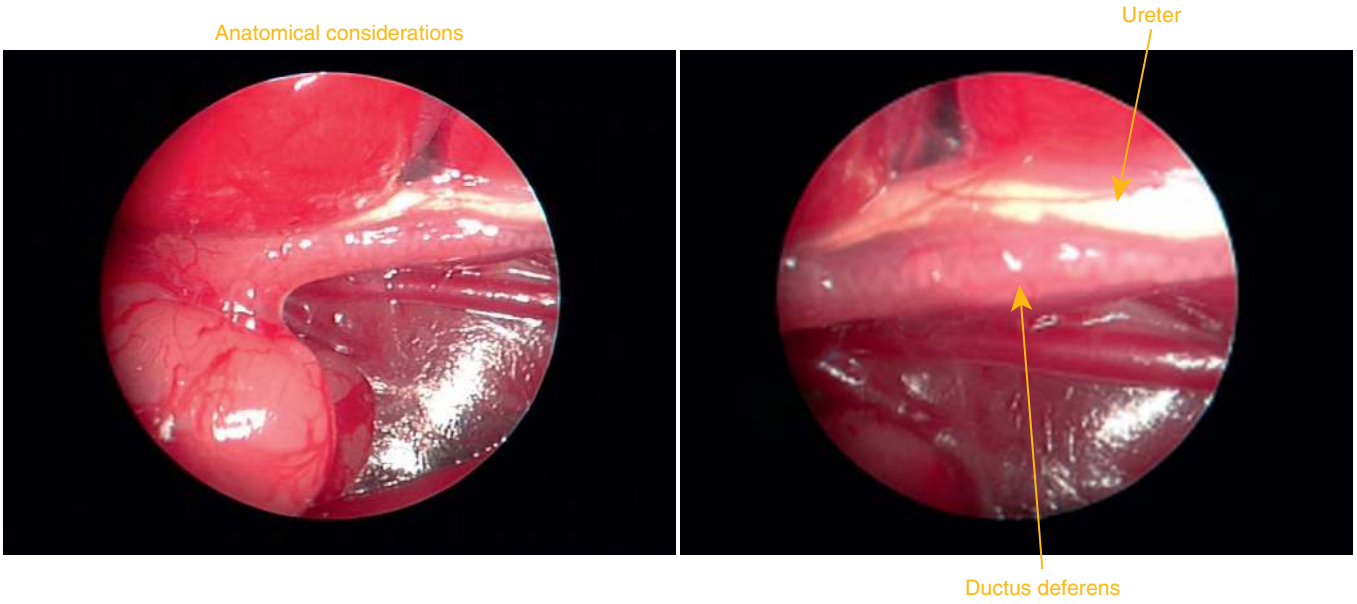


FIGURE 6-178 The main surgical concern with internal vasectomy in avian species is the close parallel association of the ductus deferens and the ureter as can be observed in this view.

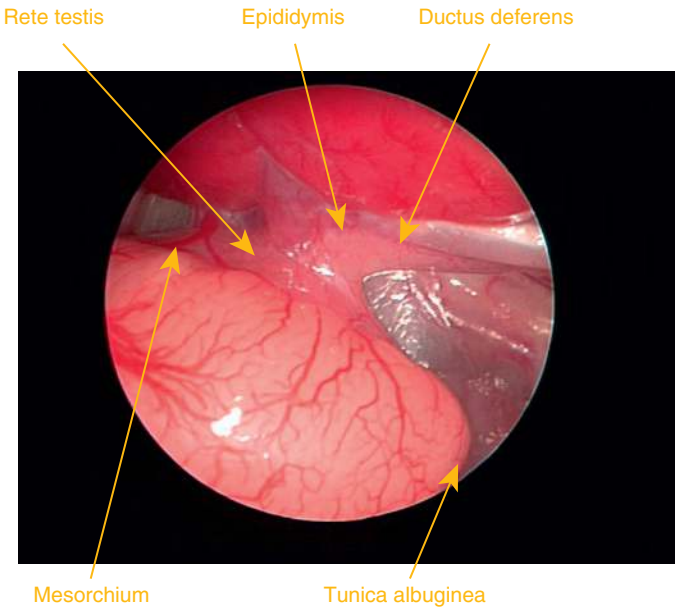


FIGURE 6-179 Close-up view of the rete testis and the base of the epididymis. This is the anatomic site indicated for performing internal vasectomy in avian species without the risk of severing or damaging the ureter.

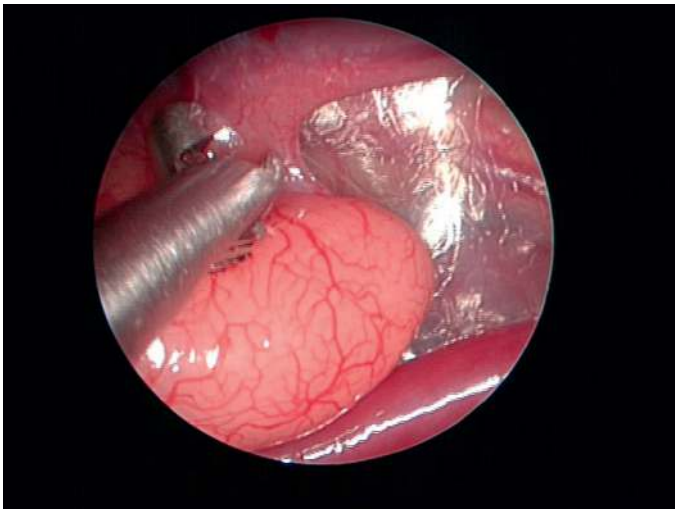


FIGURE 6-180 The biopsy forceps is directed toward and positioned carefully at the base of the epididymis. The epididymis is then grasped and cut by applying gentle traction.

endoscopy examinations in more than 350 different avian species (Jones *et al.*, 1984; J. Samour, unpublished data). The anesthetics and anesthetic combinations used include ketamine hydrochloride, ketamine hydrochloride in combination with xylazine hydrochloride, and alphaxalone-alphadolone. This last has been the anesthetic of choice for long-legged birds, such as cranes, storks, and flamingoes, and other species such as touracos, vultures, and hornbills (Samour *et al.*, 1984a). Gaseous anesthetic agents are also routinely used in birds. Currently, isoflurane is the preferred agent of many avian practitioners (Harlin, 1996; Lawton, 1996; McDonald, 1996; Rosskopf and Woerpel, 1996). This anesthetic agent offers several advantages when used with the avian patient, including a high safety margin, rapid induction, and fast recovery.

In birds weighing less than 250 g the use of a small operating table 250-mm long, 150-mm wide, and 100-mm high is recommended. Good options have a top made of plastic molded to accommodate the body of the bird and sides made of aluminum, finishing in an "L"-shaped base 10-mm wide. The table should be placed on a heating pad at 37° C (98.6° F). This provides a suitable temperature of around 28° C (82.4° F) through heat conduction. Larger birds can be laid on

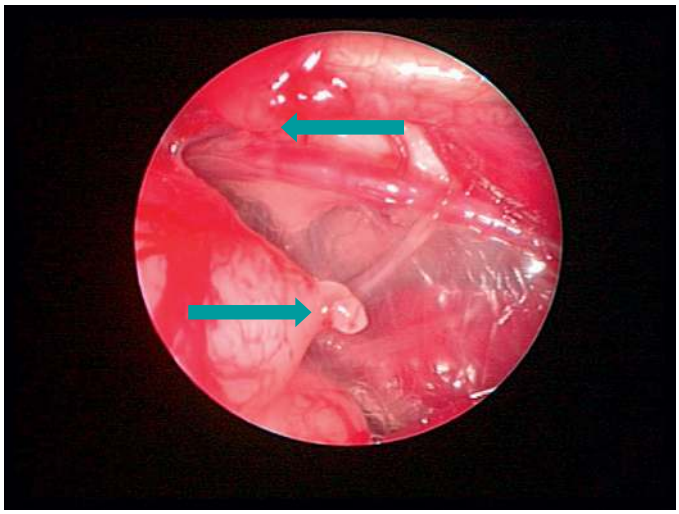


FIGURE 6-181 The epididymis has been severed *(arrows)* and a small portion (6 to 8 mm) has been removed. The procedure is then repeated in the contralateral side. Hemorrhage is minimal if the procedure has been conducted correctly.

FIGURE 6-183 A veterinary surgeon carrying out an endoscopy examination on a bird. The surgeon can share the endoscopy findings with the assistant and/or visitors by showing the images on the high-definition monitor obtained through a video camera attached to the endoscope. The high-quality images are a great teaching tool and the photographs obtained can be incorporated into the clinical record.

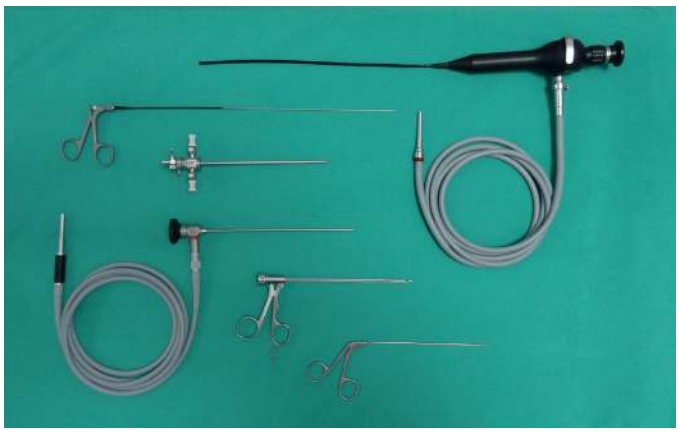


FIGURE 6-182 Rigid and flexible endoscopes together with flexible and rigid biopsy forceps routinely used to collect samples for microbiology and histopathology. (Courtesy Dr. Melodiya Magno.)

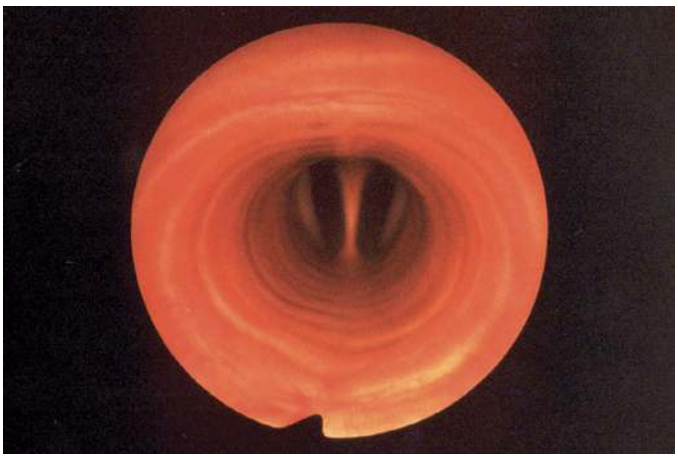


FIGURE 6-184 An endoscopy view of the normal trachea and the tracheobronchial syrinx in a saker falcon (*Falco cherrug*). The tracheobronchial syrinx is an anatomic site commonly affected with aspergillosis, pseudomoniasis, and trichomonosis infections. The examination of these anatomic sites is imperative with patients presenting with dyspnea, stridor, and wet rale.

a towel placed directly on the heating pad. Figs. 6-182 to 6-183 illustrate the equipment commonly used for avian endoscopy.

The avian patient is positioned in right lateral recumbency with the wings folded in the normal anatomic position or extended fully dorsally and secured to the table using masking tape. The left leg is fully extended forward and secured with masking tape and the lateral aspect of the abdomen is prepared for surgery. The area should be plucked of all feathers and scrubbed and disinfected with a suitable disinfectant agent. A sterile drape should be placed over the bird. Transparent drapes offer the advantage of allowing the surgeon to observe the surgical reference points clearly, in contrast to conventional cloth types of drapes. The surgical approach varies according to the species. When the left leg is fully extended, it is possible to identify a triangle formed by the last rib, the proximal femur, and the cranial edge of the pubis. The surgical approach recommended for most species is the proximal area of this triangle. This approach is particularly recommended in birds with a large ventriculus, such as birds of prey. Several other techniques have been proposed by many different authors and these have been adequately covered by Taylor (1994). Fig. 6-184 illustrates the procedure in progress.

Endoscopy examination of the gonads has been carried out in individuals weighing between 28 g and 12 kg by the author over the years. The size of the endoscope should be directly related to the size of the bird. For instance, in small birds weighing less than 100 g an endoscope of 1.9-mm diameter is suitable, while in larger birds weighing 500 g a 3-mm endoscope is more appropriate. In large birds, such as cranes, flamingoes, and storks, a 5-mm endoscope is ideal. A small incision (of the same diameter as the endoscope to be used) is made in the skin. The trocar and cannula are then inserted into the cavity. If the bird is restrained manually, the role of the assistant holding the anesthetized bird is extremely crucial at this point, in particular when

using conventional cloth drapes. The bird has to be held square on the table with the back completely straight. Any change of the positioning could result in an accident when the trocar is driven into the cavity. As a practical rule, the trocar and cannula should be inserted at a 45-degree angle to the table and 45 degrees to the back of the bird. Blunt-ended trocars are preferable in avian endoscopy. A common accident for beginners, and even experienced practitioners, is to lose the sense of direction and puncture the liver or, more commonly, the ventriculus. This may happen if the assistant has inadvertently rotated the bird in either direction or the surgeon has not been able to identify correctly the reference points for the surgical approach. The latter is a very common occurrence with obese birds. When the instruments are correctly placed, the trocar is withdrawn and the endoscope is inserted through the cannula. It is a poor practice to introduce the telescope without the aid of the cannula because the fragile seal or the lens at the terminal end of the telescope could be damaged. In addition, the use of the cannula offers the advantage of allowing the operator to introduce and withdraw the telescope repeatedly for cleaning.

If the endoscope is correctly placed, the surgeon should be looking into the caudal thoracic air sac (Figs. 6-185 and 6-186). Cranially, it should be possible to see the lung ventrally to the left, the ventriculus partially covered by the liver, and, to the right, the wall of the left abdominal air sac. Figs. 6-187 and 6-188 are cranial thoracic air sac views. The attention should be directed to the abdominal air sac for the examination of the gonads. In most species, only the left ovary is functional in the female bird. In contrast, both testes are present in the male bird. The gonads (Figs. 6-189 to 6-193) are usually located at the anterior base of the cranial lobe of the kidney, forming a triangle with the adrenal gland at the front. One of the greatest advantages of sex determination by direct visualization of the gonads is that it is possible to gather other useful information in addition to determining the sex of the bird. The gonads undergo dramatic seasonal changes throughout the year. Changes in the size, color, and appearance of the gonads are all important key features for the assessment of reproductive status.

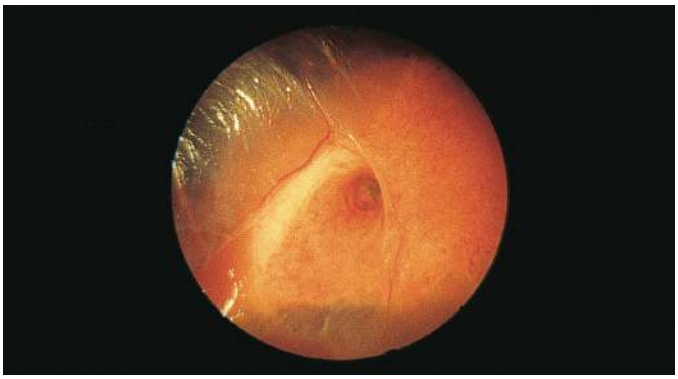


FIGURE 6-187 Ostium of the cranial thoracic air sac in a houbara bustard.

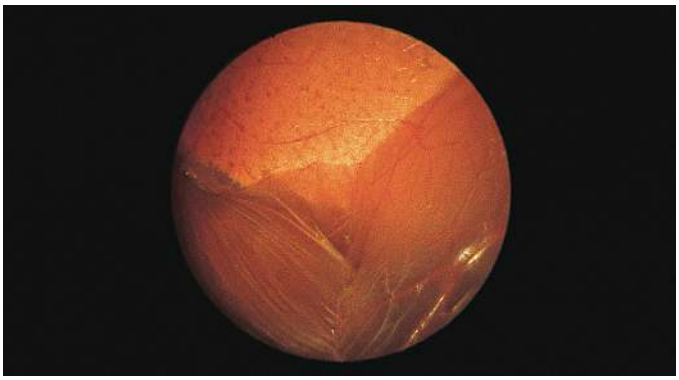


FIGURE 6-185 An anterior upper view of the caudal thoracic air sac. The lung can be seen at the cranial end of the air sac.

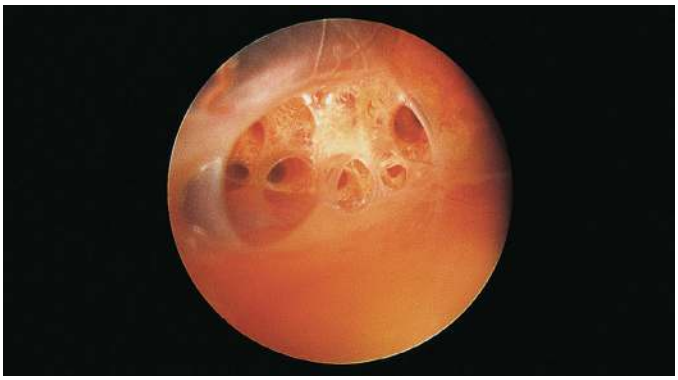


FIGURE 6-188 Ostium of the caudal thoracic air sac in a gyrfalcon.

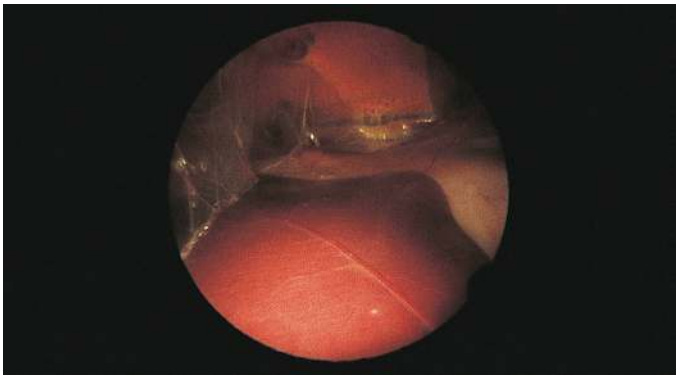


FIGURE 6-186 A posterior lower view of the caudal thoracic air sac. Part of the liver can be seen partially covering the ventriculus in a clinically normal peregrine falcon.

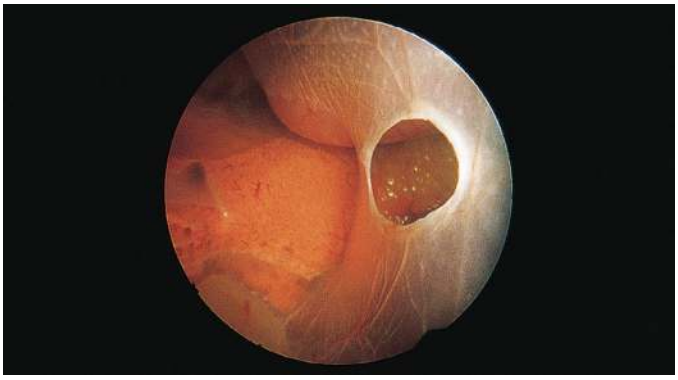


FIGURE 6-189 A small perforation has been made on the wall of the abdominal air sac to examine and obtain a photograph of the immature ovary in a houbara bustard as part of a research project.

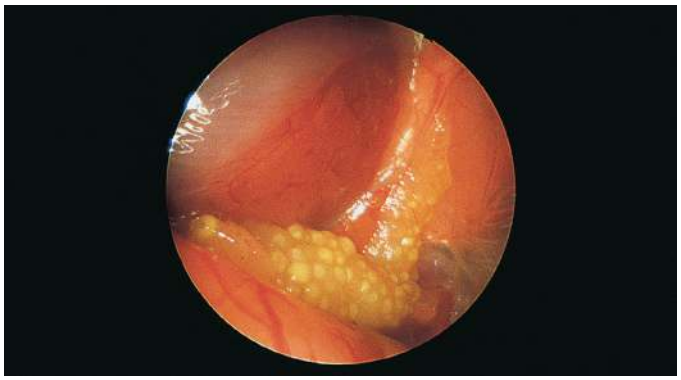


FIGURE 6-190 Kidney, adrenal gland, and immature ovary of an 8-month-old stone curlew (*Burhinus oedicnemus*). Note the V shape of the immature gonad.

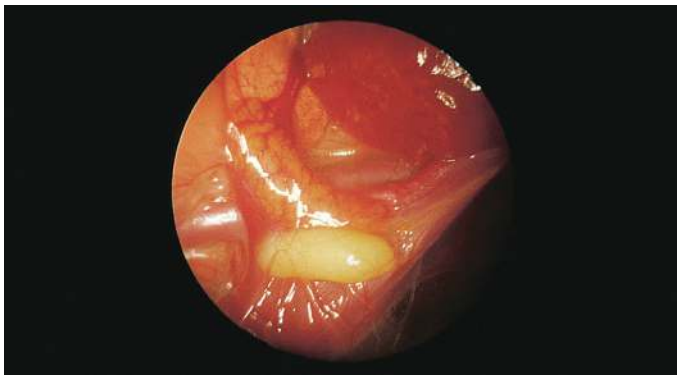


FIGURE 6-191 Kidney, adrenal gland, and immature testis of an 8-month-old stone curlew.

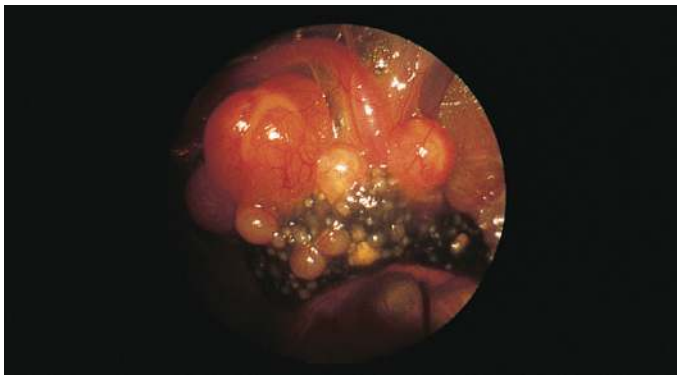


FIGURE 6-192 Melanistic ovary of a lesser sulfur-crested cockatoo (*Cacatua sulphurea*) at the onset of the breeding season. Note the small- and medium-sized developing follicles.

The gonads of some avian species, such as cockatoos, toucans, touracos, and many others are pigmented as a result of melanin deposits. Immature and sexually inactive individuals of these species display a wide range of gonad coloration, from pale gray, pale blue-green, and pale green through to dark metallic green and black. The color of the gonads changes with seasonally related morphologic changes. For instance, the weight of the testes in a male bird may increase 10 to 500 times (Johnson, 1986). In a particular study, the seasonal developmental changes of the ovary were monitored in houbara bustards (*Chlamydotis undulata*) using photography and assigning a particular score according to the different structural anatomic features (J. Samour, unpublished data).

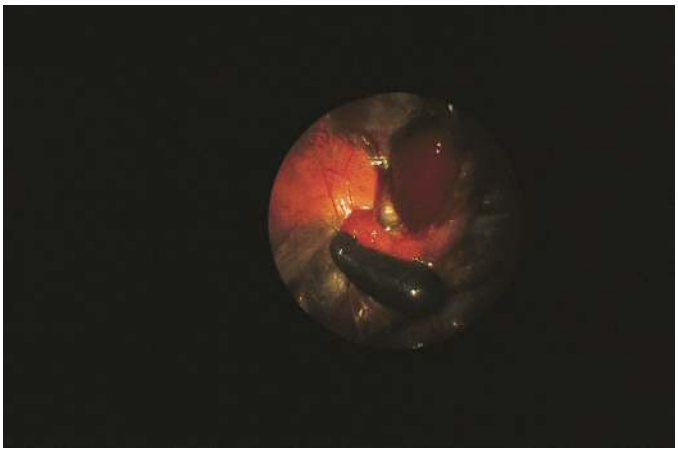


FIGURE 6-193 Melanistic and inactive testis of a lesser sulfur-crested cockatoo outside the breeding season.

Cloacoscopy for Sex Determination

Cloacoscopy (cloacal examination) has proved to be a useful technique to determine the sex of penguins (Samour *et al.*, 1983) and young ratites (Samour *et al.*, 1984b). In the case of penguins, the bird is held upside down with its head and neck between the knees of, and its back toward, the seated operator. The legs and wings are restrained by a handler. While holding the tail, the operator inserts the lubricated proctoscope into the cloaca to a depth of 65 mm in king (*Aptenodytes patagonica*), 40 mm in gentoo (*Pygoscelis papua*), and 25 mm in Humboldt (*Spheniscus humbolti*), rockhopper (*Eudyptes crestatus*), and blackfooted (*Spheniscus demersus*) penguins. The obturator is then withdrawn and the dorsal wall of the cloaca carefully examined. The avian cloaca is divided into four different regions: the proctodeum, urodeum, coprodeum, and colorectum. The examination is directed toward the urodeum. Around the midline of this area, there are two pairs of papillae. The inner pair make up the ureteric papillae, which are similar in length and morphology in both sexes. The outer pair represent the openings of the vasa deferentia. In the male bird, these papillae are well developed and the same size as the inner pair. In contrast, in the female bird, the papillae of the outer pair are smaller and substantially shorter. Additionally in females, the oviductal opening is clearly visible on the left side of the urodeum. During the laying season, the mucous membrane around the opening of the oviduct is swollen and pink-red in color.

The technique for sex determination in young ratites is very similar to that for penguins. The bird is restrained in a similar way and the choice of proctoscopes is related to the size of the bird. Through the proctoscope, the operator will easily be able to identify the phallus in males. At this age, it is not possible to identify the oviductal opening of young female birds. Therefore in females the sex is established only by the absence of the phallus. In monomorphic species, such as rheas and emus, and in juvenile and subadult individuals of dimorphic species such as ostriches, sex determination is carried out through digital examination of the cloaca (Samour *et al.*, 1984; Fowler, 1996).

REFERENCES

Ainley DG: PhD thesis, Johns Hopkins University, 1970.

Bailey RE: Surgery for sexing and observing gonad condition in birds, *Auk* 70:497–499, 1953.

Bennett RA: A review of avian soft tissue surgery. *Proceedings of the Association of Avian Veterinarians*, 1993, Nashville, pp 65–71.

Burr EW, Huchzermeyer FW, Riley AE: Laparoscopic examination to determine sex in monomorphic avian species, *J S Afr Vet Assoc* 52(1):45–47, 1981.

Bush M: Laparoscopy in zoological medicine, *J Am Vet Med Assoc* 9:1081–1087, 1978.

Bush M: Laparoscopy in birds and reptiles. In Harrison RM, Wildt DE, editors: *Animal laparoscopy*, Baltimore, 1980, Williams & Wilkins, pp 186–197.

Bush M, Kennedy S, Wildt DE, Seager SWJ: Sexing birds by laparoscopy, *Int Zoo Yearb* 18:197–199, 1978.

Clayton LA, Ritzman TK: Endoscopic-assisted removal of a tracheal seed foreign body in a cockatiel (*Nymphicus hollandicus*), *J Avian Med Surg* 19:14–18, 2005.

Crosta L, Gerlach H, Bürkle M, Timossi L: Endoscopic testicular biopsy in Psittaciformes, *J Avian Med Surg* 16:106–110, 2002.

Crosta L, Timossi L: Imaging techniques in avian obstetrics. *Proceedings of the European Association of Avian Veterinarians*, 2005, Arles, pp 301–306.

Forbes NA: Soft tissue surgery. In Samour J, editor: *Avian medicine*, London, 2008a, Mosby Elsevier, pp 154–167.

Forbes NA: Soft tissue surgery. In Chitty J, Lierz M, editors: *BSAVA manual of raptors, pigeons and passerine birds*, Quedgeley, 2008b, British Small Animal Veterinary Association, pp 143–156.

Fowler ME: Clinical anatomy of ratites. In Tully TN, Shane SM, editors: *Ratite management medicine and surgery*, Malabar, 1996, Krieger Publishing Company, pp 1–11.

Gartrell BD: Assessment of the reproductive state in male swift parrots (*Thamus discolor*) by testicular aspiration and cytology, *J Avian Med Surg* 16:211–217, 2002.

Harlin RW: Otoscopic sexing. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, 1996, Williams & Wilkins, pp 697–698.

Harrison GJ: Endoscopic examination of avian gonadal tissue, *Vet Med Small Anim Clin* 73:479–484, 1978.

Harrison GJ: Endoscopy. In Harrison GJ, Harrison LR, editors: *Clinical avian medicine and surgery*, Philadelphia, 1986, WB Saunders, pp 224–244.

Hauser WMP: Game bird breeders, *Aviculturists, Zoologists and Conservationists Gazette* 26:6–11, 1977.

Heidenreich M: *Birds of prey: medicine and management*, Oxford, 1997, Blackwell Science, pp 196–199.

Hernandez-Divers SJ: Minimally invasive endoscopic surgery of birds, *J Avian Med Surg* 19:107–120, 2005.

Ingram KA: Laparotomy technique to determine sex of psittacine birds. *Proceedings of the American Association of Zoo Veterinarians*, 1977, Honolulu, pp 40–44.

Ingram KA: Laparotomy technique for sex determination of psittacine birds, *J Am Vet Med Assoc* 176:1244–1246, 1978.

Ingram KA: Otoscopic technique for sexing birds. In Kirk RW, editor: *Current veterinary therapy VII*, Philadelphia, 1980, WB Saunders, pp 656–658.

Johnson AL: Reproduction in the female and male. In Sturkie PD, editor: *Avian physiology*, ed 4, New York, 1986, Springer Verlag, p 404.

Jones DM, Samour JH, Knight JA, Ffinch JM: Sex determination of monomorphic birds by fiber-optic endoscopy, *Vet Rec* 115:595–598, 1984.

Jones RG, Redig PT: Endoscopy guided vasectomy in the immature Japanese quail (*Coturnix coturnix japonica*). *Proceedings of the European Association of Avian Veterinarians*, 2003, Tenerife, pp 117–123.

Kollias GV Jr: Avian endoscopy. In Jacobson ER, Kollias GV Jr, editors: *Contemporary issues in small animal practice*, New York, 1988, Churchill Livingstone, pp 75–104.

Lawton MPC: Anesthesia. In Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *BSAVA manual of raptors, pigeons and waterfowl*, Cheltenham, 1996, British Small Animal Veterinary Association, pp 79–88.

Le Resche RE: PhD thesis, Johns Hopkins University, Baltimore, 1971.

Lierz M: Endoskopie. In Pees M, editor: *Leitsymptome bei papageien und sittichen*, Stuttgart, 2004, Enke Verlag, pp 185–194.

Lierz M: Diagnostic value of endoscopy and biopsy. In Harrison GJ, Lightfoot TL, editors: *Clinical avian medicine* (vol 2), Palm Beach, 2006, Spix Publishing Inc., pp 631–652.

Lierz M: Endoscopy, biopsy and endosurgery. In Chitty J, Lierz M, editors: *BSAVA manual of raptors, pigeons and passerine birds*, Quedgeley, 2008, British Small Animal Veterinary Association, pp 128–142.

Lierz M, Hafez HM: Endoscopy guided multiple entry surgery in birds. *Proceedings of the European Association of Avian Veterinarians*, 2005, Arles, pp 184–189.

Lloyd C: Staged endoscopy ventricular foreign body removal in a gyr falcons *(Falco rusticolus)*, *J Avian Med Surg* 23(4):314–319, 2009.

McDonald SE: Surgical sexing of birds by laparoscopy, *Californian Vet* 5:16–22, 1982.

McDonald SE: Endoscopic examination. In Burr EW, editor: *Companion bird medicine*, Ames, 1987, Iowa State University Press, pp 166–174.

McDonald SE: Endoscopy. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, 1996, Williams & Wilkins, pp 699–717.

Pye GW, Bennett RA, Plunske R, et al: The effect of endoscopy salpingohysterectomy in juvenile cockatiels (*Nymphicus hollandicus*) on ovulation. *Proceedings of the European Association of Avian Veterinarians*, 2001a, Munich, pp 66–69.

Pye GW, Bennett RA, Plunske R, Davidson J: Endoscopic salpingohysterectomy of juvenile cockatiels (*Nymphicus hollandicus*), *J Avian Med Surg* 15:90–94, 2001b.

Risser AC: A technique for performing laparotomy on small birds, *Condor* 73:376–379, 1971.

Rosskopf WJ Jr, Woerpel RW: Practical anesthesia administration. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, 1996, Williams & Wilkins, pp 664–671.

Samour JH: Avian endoscopy. In Brearley MJ, Cooper JE, Sullivan M, editors: *A colour atlas of small animal endoscopy*, London, 1991, Wolfe Publishing, pp 97–109.

Samour JH: Veterinary medicine, falcons and falconry in the Middle East. *Proceedings of the Association of Avian Veterinarians*, 1996, Tampa, pp 233–239.

Samour JH, Jones DM, Knight JA, et al: Comparative studies of the use of some injectable anesthetic agents in birds, *Vet Rec* 115:6–11, 1984a.

Samour JH, Markham J, Nieva O: Sexing ratite birds by cloacal examination, *Vet Rec* 115:167–169, 1984b.

Samour JH, Stevenson M, Knight JA, Lawrie AJ: Sexing penguins by cloacal examination, *Vet Rec* 113:84–85, 1983.

Samour JS: Vasectomy in birds: a review, *J Avian Med Surg* 24(3):169–173, 2010.

Satterfield WC: Diagnostic laparoscopy in birds. In Kirk RW, editor: *Current veterinary therapy VII*, Philadelphia, 1980, WB Saunders, pp 659–661.

Satterfield WC, Altman RB: Avian sex determination by endoscopy. *Proceedings of the American Association of Zoo Veterinarians*, 1977, Honolulu, pp 45–48.

Sladen WJL: Sexing penguins by cloacoscope, *Int Zoo Yearb* 18–77, 1978.

Taylor M: A morphologic approach to the endoscopic determination of sex in juvenile macaws, *J Assoc Avian Vet* 3:199–201, 1989.

Taylor M: Endoscopy. *Proceedings of the Association of Avian Veterinarians*, 1990, Phoenix, pp 319–324.

Taylor M: *Endoscopy: laboratory manual*, Lake Worth, 1992, Association of Avian Veterinarians, pp 1–10.

Taylor M: Endoscopic examination and biopsy techniques. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, 1994, Wingers Publishing, pp 327–354.

Wilson GH, Richey L, McBride M, et al*:* Chemical castration of pigeons via endoscopic intratesticular injection. *Proceedings of the Association of Avian Veterinarians*, 2004, New Orleans, pp 91–93.

Anesthesia and Analgesia

GENERAL ANESTHESIA

Martin P. C. Lawton

The routine use of isoflurane in avian practice has almost made anesthesia of birds a predictably safe and uneventful procedure. There is, however, more to anesthesia than masking a bird down with isoflurane. The unique anatomy and physiology of the bird affects the design and use of anesthetic circuits, intubation, or placement of an air sac tube and the method of resuscitation should an emergency occur. The aims of anesthesia should be to provide a smooth, reliable induction with adequate restraint, muscle relaxation, and analgesia followed by a fast, but full, uneventful recovery (Lawton, 1996a,b). Recent publications provide updated and comprehensive information on anesthesia in birds (Edling, 2006; Heatley, 2008; Tully *et al.*, 2009).

ANATOMICAL AND PHYSIOLOGICAL CONSIDERATIONS

Anatomical Considerations

Only the most important characteristics of avian anatomy that have a direct bearing on the management and maintenance of anesthesia are discussed here. For a more detailed description of the anatomy and physiology of the avian respiratory see King and McLelland (1975) and McLelland (1990).

Trachea

The avian trachea has complete interlocking rings, which are cartilaginous in some species and ossified in others. This has implications when intubation of birds is necessary, as cuffed endotracheal tubes, if used, could damage these complete rings (Fitzgerald and Blais, 1993). Unlike mammals, birds can still vocalize even when intubated because of the location of the syrinx at the tracheal bifurcation (Heard, 1997). Some Anseriformes and other species do have diverticulum and bulbous expansions, while others have complicated tracheal loops or even a double trachea (penguins; Edling, 2003), which can lead to complications with dead space within the trachea.

Air Sacs

The Class *Aves* has a unique respiratory system, employing air sacs that act as bellows and reservoirs when breathing (Fig. 7-1). Most birds have nine air sacs, some of which pneumatize bones, while some leave the coelomic cavity and terminate subcutaneously (Fedde, 1986). As a generalization, there are usually the following air sacs: paired cranial thoracic, caudal thoracic, abdominal and clavicular, and a single cervical. The air sacs are avascular and contribute less than 5% toward respiratory gas exchange (Edling, 2003, 2006).

The position of a bird under anesthesia will affect the ability of the air sacs to work normally. In dorsal recumbency, the weight of the abdominal organs will cause a partial collapse of the abdominal and thoracic air sacs. Intermittent positive pressure ventilation (IPPV) can help overcome the effects on respiration of dorsal positioning.

Lungs

The avian lungs are relatively rigid and do not move appreciably during respiration. There is no diaphragm, therefore, lungs do not collapse when the thoracic (coelomic) cavity is entered surgically (or endoscopically). The avian lung is a "flow-through" system. The entrances between the air sacs and the lungs are either via ostia or sometimes via large tubes called the saccobronchi. The primary bronchus also has an extrapulmonary portion that extends through the lung to the abdominal air sac (Edling, 2003, 2006). This arrangement of lungs interconnecting with the air sacs allows the bird to be artificially ventilated either via the trachea or via an abdominal air sac tube (see the following).

The lungs are divided into paleopulmonic and neopulmonic areas. During inspiration the air flow is divided between these two areas, but gaseous exchange mainly occurs in the paleopulmonic area with only a limited amount occurring in the neopulmonic area. Air that passes through the paleopulmonic area enters the cervical, clavicular, or cranial thoracic air sacs, whereas air that goes through the neopulmonic area passes into the caudal thoracic and abdominal air sacs. There has been some debate as to whether inspired air passes over the lung tissue twice (James *et al.*, 1976) or only once (Scheid and Piiper, 1971; Fitzgerald and Blais, 1993). During both inspiration and expiration, although air moves bidirectionally through the neopulmonic area, air will only travel unidirectionally (caudal to cranial) through the paleopulmonic area (Fedde, 1986). The unidirectional flow through the paleopulmonic parabronchi is thought to be from the aerodynamic shape and angle of the bronchi and cranial air sacs creating a flow resistance within the lungs, as no valves have been grossly identified.

Physiological Considerations

Trachea

The tracheal length and volume is greater in birds than in mammals of equal body mass. The tracheal dead space is 4.5 times that of mammals. Birds compensate by increasing their tidal volume and decreasing respiratory frequency as compared with a mammal of equal size (Fedde, 1986). This increased tidal volume must be maintained during anesthesia to prevent hypocapnia associated with the increased dead space volume of the trachea and any endotracheal tube used.

Inspiration and Expiration

Avian inspiration occurs when the inspiratory muscles increase the body volume by movement of the thoracoabdominal body wall (in particular, the sternum is moved downward and the ribs move outward). This increase in the body volume results in a negative pressure buildup within the air sacs, causing air to be "sucked in" through the nares and mouth and pass through the lungs and into the air sacs. On handling or positioning a bird before surgery, any pressure to the ribs and, in particular, the sternum may affect the ability to breathe because the necessary body volume changes may not occur.

Expiration occurs when the expiratory muscles cause a reduction in the size of the body volume by compression of the thoracic skeleton

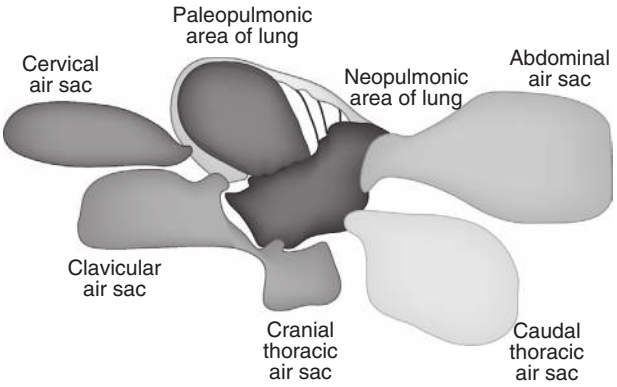


FIGURE 7-1 Relationship of air sacs to paleopulmonic and neopulmonic area of lungs.

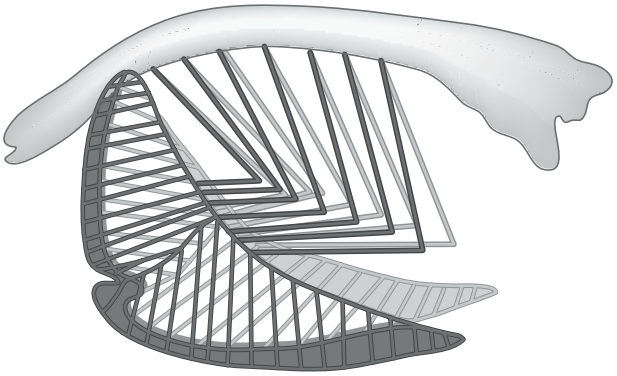


FIGURE 7-2 Demonstration of the movement of the keel and ribs during respiration. The light-shaded area represents expiration while the dark-shaded area represents inspiration.

(Fig. 7-2) and is not passive, unlike the situation in mammals. The reduction of the body volume causes an increased pressure within the air sacs, forcing gas from the air sacs back into the lungs and then out via the mouth or nares. The air sacs thus act as bellows. On the relaxation of the respiratory muscles, the sternum is midway between its inspiratory and expiratory position. A deeply anesthetized bird may not generate sufficient muscular contractions to allow adequate "pumping" of air back through the lungs. Sinn (1994) advised the routine use of positive IPPV (at a frequency of 20 to 40/min at 15 mm Hg) to overcome any possibility of hypocapnia and to maintain adequate oxygenation.

Gaseous Exchange

Gaseous exchange within the avian lung relies on a cross-current exchange system in which the air within the air capillaries flows at right angles to the flow of blood through the blood capillaries. This cross-current exchange system is more effective than that found in mammalian lungs. The cross-current flow causes a potential increase in the partial pressure of carbon dioxide (P_{CO_2}) expired and an increase in P_{O_2} within the blood. Avian lungs are considered to be 10 times more effective than mammalian lungs (James *et al.*, 1976).

In birds, CO_2 is mainly in the form of hydrogen bicarbonate in the plasma, with only small amounts as dissolved or plasma-bound CO_2. Carbonic anhydrase is responsible for the production of the bicarbonate and the subsequent dissociation of the hydrogen ion. It appears that CO_2 has no direct effect on the oxygen affinity of hemoglobin other than through the metabolic release of hydrogen ions. In the lungs, the hydrogen bicarbonate enters the red blood cells and the resulting metabolism releases the CO_2, which is then breathed out. A small change in the P_{CO_2} leads to a large change in the blood CO_2.

Ventilation Triggers

Ventilation during anesthesia is affected by a number of physiological factors that should be considered in an anesthetized patient. As with most taxa, inhalation of CO_2 stimulates ventilation. There are known receptors in the carotid bodies and the intrapulmonary chemoreceptors and CO_2 directly stimulating the nervous system (Fedde, 1986). The carotid bodies are responsible for controlling ventilation when there is a reduction in the Pa_{O_2}, hypoxia, or an increase in the Pa_{CO_2}. Pain will also stimulate respiration. Increases in body temperature will cause a thermal polypnea but not usually hyperventilation. Under anesthesia, subjecting the larynx and trachea to cold gases is known to slow breathing and may even produce apnea. Temperature also has an influence on the O_2 affinity of hemoglobin: an increased temperature in active tissue favors a release of O_2 from the hemoglobin, but if the bird cools down too much under anesthesia, then the release of oxygen is affected because the binding with hemoglobin is increased.

Hypoglycemia

Birds are very prone to hypoglycemia when anesthetized. It is not recommended that small birds be fasted before gaseous induction, but where possible induction should be performed when the crop (where present) is empty. It is often overlooked that the total time a bird is fasted would not just include any period before anesthesia (if any), but also the time up until the bird has fully recovered and is willing to eat. Cooper (1989) stated that small birds should never be deprived of food for longer than 3 hours. Fasting may also reduce hepatic detoxification of certain anesthetic agents (Carter-Storm, 1988). Regurgitation is seldom a problem in granivorous psittacine birds, unlike waterfowl or frugivorous birds, in which a period of fasting has been recommended (Mandelker, 1987).

VOLATILE ANESTHESIA

There are a number of anesthetic agents that have historically been used for induction and maintenance of birds. Ether, one of the older volatile anesthetic agents, can be dismissed on the basis that it is unsafe because the safety margin is below that of more modern anesthetic agents because there is a risk of explosion and it irritates the mucosa. Although methoxyflurane has been used in the past with very good results, the lack of availability and the requirement of a dedicated vaporizer, together with the disadvantage of the hangover effect (50% being metabolized), has virtually removed its use from avian practice. Two agents likely to be used routinely in a practice situation are halothane and isoflurane, and these are compared in Table 7-1. Rosskopf and Woerpel (1996) considered that, if veterinary surgeons are unwilling to invest in isoflurane and the necessary equipment to use it, they should refer the case for surgery to a practice that is properly equipped. Recent studies have shown that isoflurane has very little effect on natural physiological processes, such as gastrointestinal transit time (Lennox *et al.*, 2002); therefore, it can be used in situations where previous stressful restraint was required (such as for barium meal radiography of the gastrointestinal tract).

Sevoflurane is part of the next generation of gaseous anesthetic agents and is proving even safer than isoflurane. This agent is considered by many to be the gaseous anesthetic of choice for birds. Sevoflurane has an even lower blood gas partition coefficient than isoflurane (0.69), giving a marginally shorter recovery time than isoflurane. It is thought that sevoflurane will increase the chance of success in critical or prolonged procedures (Edling, 2003, 2006; Klaphake *et al.*, 2006).

TABLE 7-1 Comparison of Isoflurane and Halothane

	Isoflurane	Halothane
Safety margin This is the ratio of lethal dose to anesthetizing dose (Dohoo, 1990)	5.7 This safety margin alone makes other agents obsolete (Rosskopf *et al.*, 1992)	3.0 High concentrations of anesthetic agent are "held" in the air sacs after induction, which may lead to fatalities
Blood gas partition coefficient The higher the value, the greater the solubility in blood and tissue	1.4 at 37°C Very low solubility allows rapid induction and rapid recovery, with less retention in the body tissues compared with halothane	2.3 at 37°C Higher solubility gives more potential for redistribution from the body compartments back into circulation after induction, and a slower recovery than isoflurane
Degree of metabolism Any metabolism will slow the speed of elimination from the body, and often metabolites can cause a "hangover" effect	0.3% Virtually no metabolism allows excretion solely by expiration; 2% isoflurane has been used for prolonged anesthesia and recovery was still rapid, occurring within 6 minutes and full recovery considered to occur within 21 minutes (Clutton, 1986)	155%-20% Because of increased distribution in body tissues (associated with the higher blood gas partition coefficient) and increased metabolism, there is a slower recovery than with isoflurane; recovery is delayed if there is any underlying liver disease
Muscular relaxation	Very good	Poor
Analgesia	Good	Poor
Respiratory effects	Little respiratory depression	Marked respiratory depression
Cardiac effects	Possibility of slight myocardial depression, which results in little or no change in the heart rate (Jenkins, 1993)	Moderate myocardial depression; there is catecholamine sensitization
Contraindications	None reported	Hepatic dysfunction, cardiovascular disease or catecholamine release
Overdose	There is usually apnea before cardiac arrest and this allows a good change of prompt artificial ventilation, leading to a full recovery	Apnea and cardiac arrest usually occur simultaneously, making artificial ventilation and a full recovery more difficult than with isoflurane

The efficiency of sevoflurane as an anesthetic agent in birds has been assessed in parrots. A recent study evaluated the minimum anesthetic concentration of sevoflurane in thick-billed parrots (*Rhynchopsitta pachyrhyncha*; Phair, 2012). This study concluded that the minimum anesthetic concentration in the thick-billed parrot, determined by mechanical stimulation, was equal to that determined in chickens and humans (Phair, 2012). In addition, the effect of premedication using ketamine hydrochloride alone or in combination with diazepam on anesthesia with sevoflurane has also been evaluated in the blue-fronted amazon parrot (*Amazona aestiva*; de Paula *et al.*, 2013). The results of this study suggested that ketamine alone or in combination with diazepam provided a good quality sedation easing the handling procedure and thus reducing stress on the birds (de Paula *et al.*, 2013). Despite these advances the cost difference between sevoflurane and isoflurane may make its routine use hard to justify.

Equipment

Although it is possible to use volatile anesthetic agents in a Boyle's bottle or by the primitive method of placing a soaked cotton wool swab directly into a chamber with a bird, this is not advised. The use of a dedicated vaporizer is recommended to allow an exact concentration to be given (irrespective of temperature or air pressure, within certain ranges). Ideally, the anesthetic machine should be on a mobile trolley with shelves for placing the monitoring equipment, such as respiratory and cardiac monitors, together with drugs to deal with an emergency (Fig. 7-3). It is not possible to use the same vaporizer for both halothane and isoflurane because of the differences in these volatile fluids, unless the vaporizer is cleaned and recalibrated before each change. An anesthetic machine with a "Selectatec" fitting will allow easy changing between dedicated vaporizers should this be required (e.g., for different classes).

FIGURE 7-3 Anesthetic trolley with isoflurane vaporizer, respiratory monitor, cardiac monitor, and emergency drug box.

There are several advantages using IPPV (Fig. 7-4) for any anesthetized bird because this allows control over not only the rate and depth of respiration but also oxygenation and the prevention of hypercapnia. The effect of IPPV on the depth of anesthesia during and after isoflurane anesthesia was studied in the sulfur-crested cockatoo (*Cacatua galerita*; Chemonges, 2014). This study concluded that IPPV increases the depth of anesthesia in a rate- and dose-related manner and prolongs recovery (Chemonges, 2014).

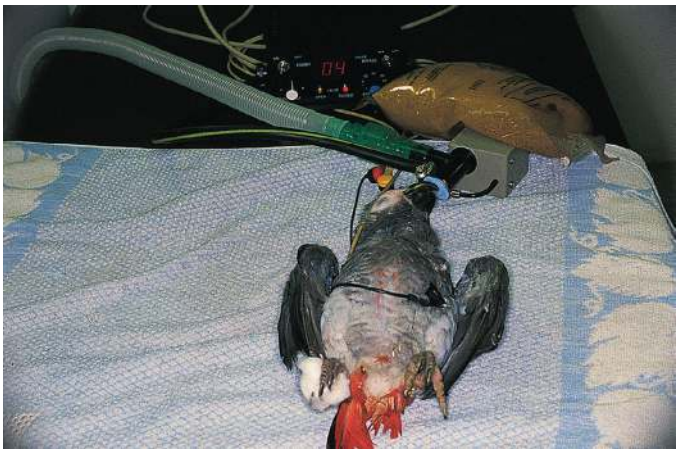


FIGURE 7-4 Intermittent positive pressure ventilation of an African grey parrot (*Psittacus erithacus*).

FIGURE 7-5 Face mask induction of an African grey parrot with isoflurane anesthesia.

Whether using IPPV, intubation, and a suitable circuit or just a face mask, the flow rate of oxygen to the lungs must be kept high to prevent hypercapnia. The gaseous flow rate should be a minimum of three times the normal minute volume (i.e., approximately 3 mL/g bodyweight—a 400-g Amazon parrot (*Amazona* sp.) needs 1.2L/min), although I use 2 to 3 L/min irrespective of size.

Face Mask

Isoflurane allows a relatively easy method of induction by face mask (other than for diving birds), reducing many of the complications of handling and injecting and the stresses that are involved with these procedures. Therefore, the most basic anesthetic circuit consists of a vaporizer, a source of carrier gas (usually oxygen), and a face mask. To keep the bird relaxed and prevent flapping, the patient should be adequately restrained, for instance, in a towel (Figs. 7-5 and 7-6). Face masks can be purchased or self-made from disposable items such as syringe cases (for small birds) or soft-drink bottles (for macaws or long-beaked birds; Figs. 7-7 and 7-8). The advantages of using disposable face masks are the elimination of the risk of spread of infection between birds and it is often a more suitable mask for an avian patient than those currently on the market. If a disposable face mask is not used, then it is important that the mask is cleaned well and ideally sterilized before using it on another patient. A face mask does have

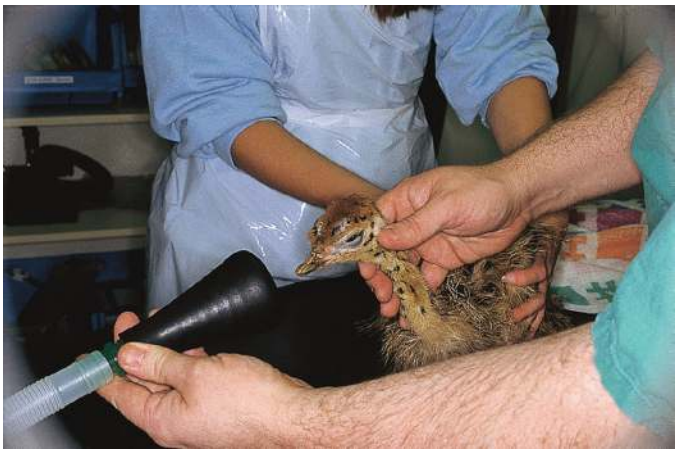


FIGURE 7-6 Use of a Hall's mask for induction of an ostrich chick (*Struthio camelus*).

FIGURE 7-7 A cut-down soft drink bottle, with the edges protected with sticking plaster, makes an ideal mask for a blue and gold macaw (*Ara ararauna*).

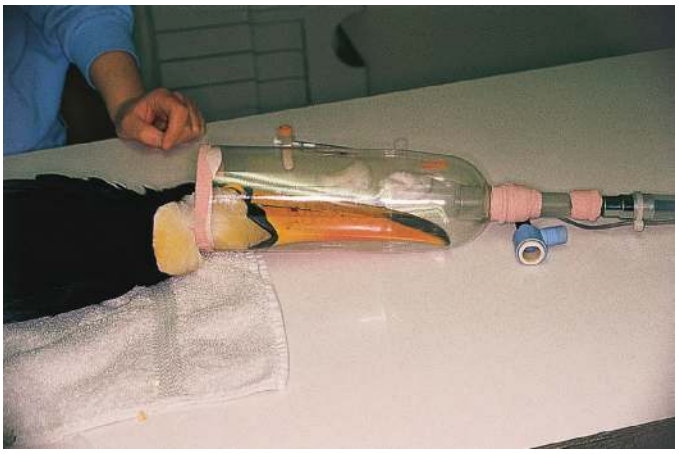


FIGURE 7-8 A soft drink bottle makes an extended face mask for long-billed birds such as toucans (*Ramphastos* sp.).

disadvantages, especially if examining or operating around the head, although a mask can be adapted for this (Fig. 7-9).

Anesthetic Chambers

For birds that are likely to be highly stressed by the handling required for masking down, the use of an anesthetic chamber is advised

FIGURE 7-9 A syringe face mask can be cut to allow access to the eye area while still maintaining anesthesia without intubation.

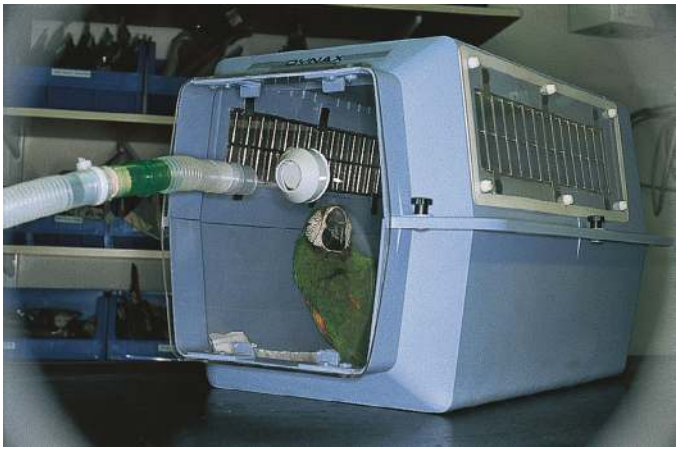


FIGURE 7-10 An adapted cage with Perspex makes a suitable anesthetic induction chamber.

(Fig. 7-10). Birds tend not to be distressed by being in an anesthetic chamber, especially if the volatile anesthetic agent is isoflurane. Anesthetic chambers can be very simple, such as a cage and a bag placed over it into which the volatile anesthetic is introduced, or dedicated purpose-built chambers fit into an anesthetic machine with scavenging capacity. The main disadvantages of using an anesthetic chamber are usually cost and the slightly increased length of time before the intubation can be performed when compared with a bird that is masked down.

Endotracheal Intubation

Once a patient is induced, although it is possible to maintain anesthesia just with a face mask (Fig. 7-11), endotracheal intubation should be considered except for the shortest procedures. An airway should be provided to allow maintenance of the bird under anesthesia but also for ventilation should apnea occur. Intubation of birds is easy, because of the forward-placed glottis behind the base of the tongue (Figs. 7-12 and 7-13). With the mouth held open and, in the case of psittacines, the tongue gently pulled forward, a suitably sized tube can be introduced through the glottis (Fig. 7-14). Even small budgerigars or cockatiels may be intubated using cut-down cannulas or catheters (Fig. 7-15), although these small-diameter tubes may become blocked

FIGURE 7-11 An African grey parrot maintained under anesthesia with just a face mask.

FIGURE 7-12 An anesthetized African grey parrot showing glottal opening and base of tongue.

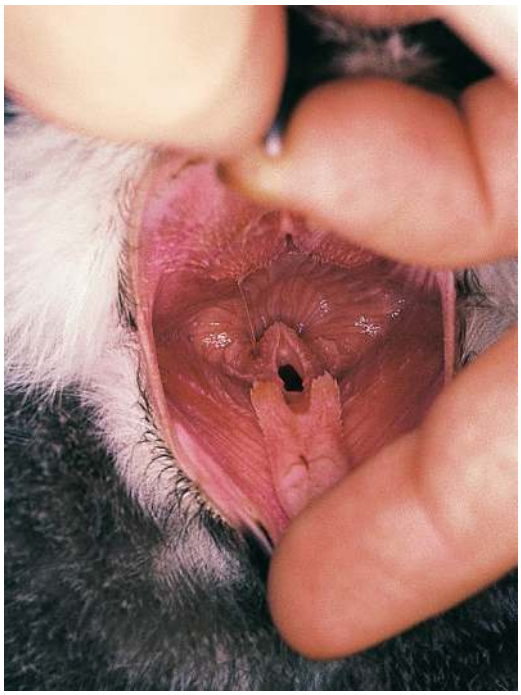


FIGURE 7-13 The glottal opening of a European eagle owl (*Bubo bubo*). Compare this with Fig. 7-12.

FIGURE 7-14 Intubation of an anesthetized African grey with a Bethune 2.5mm endotracheal tube.

FIGURE 7-15 A cut-down intravenous catheter makes an ideal endotracheal tube for a budgerigar.

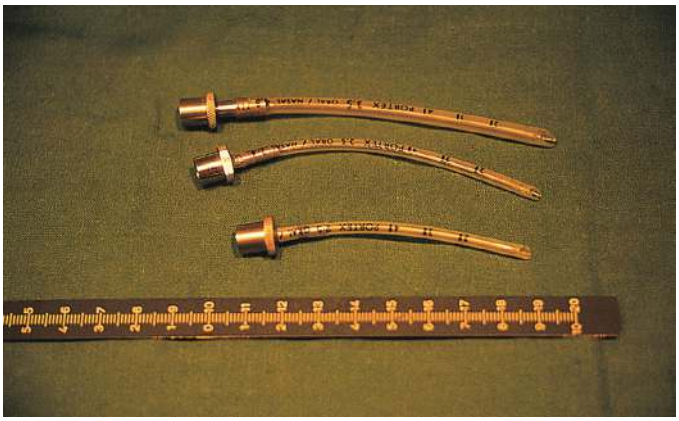


FIGURE 7-16 A selection of Bethune endotracheal tubes suitable for birds.

with respiratory secretions. Because of the great differences in the size of birds, a wide range of tubes also have to be available, which can be purpose-made tubes or premade out of cut-down intravenous catheters or urinary cannulas (Figs. 7-16 and 7-17). Most catheters and cannulas have lure fittings and can be connected to an anesthetic

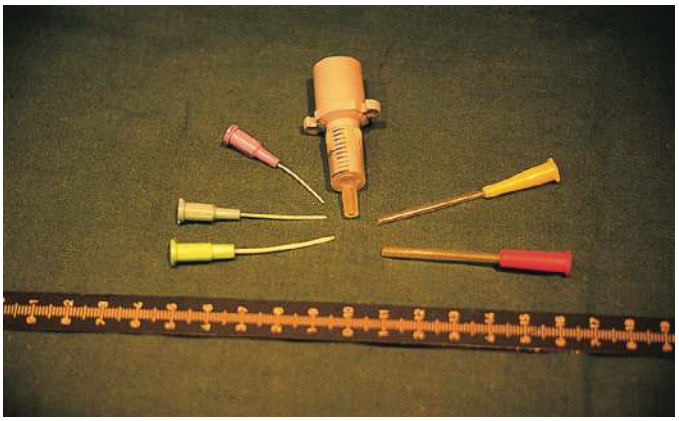


FIGURE 7-17 Prepared intravenous cannulas or urinary catheters with a syringe adaptor.

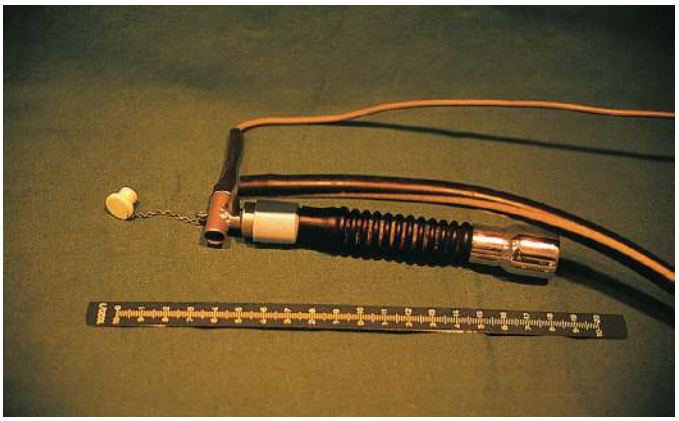


FIGURE 7-18 A Bethune anesthetic circuit with scavenging and respiratory monitor probe attached.

circuit (Fig. 7-18) by using a cut-down 2-mL syringe fitted on to an endotracheal adaptor (usually 8.5 mm). Once intubated, a bird should be maintained on a Bethune or Ayre's T-piece system (Fig. 7-19). Circle circuits should not be used for the avian patient because it is not able to exert sufficient force to open the valves. Intubation will allow ventilation of the bird should this prove necessary, and also allows scavenging of waste gases, an increasing requirement under most countries' legislation. Scavenging of waste gases is difficult, if not impossible, with an open face mask unless a more expensive active scavenging system, such as Fluvac (designed for a face mask) is used.

Air Sac Intubation

When undertaking surgery around the beak, head, or face, a mask or even an endotracheal tube is likely to restrict access. The presence of air sacs and the unique air flow from the abdominal and caudal thoracic air sacs into the lungs means that when a tube is placed into one of the air sacs, anesthetic gases can be introduced (Figs. 7-20 and 7-21). The site for the placement of an air sac tube is a matter of preference but is usually similar to the site chosen for endoscopic examination. Traditionally, this is the left side just behind the ribs, although Sinn (1994) suggested the use of short endotracheal tubes or rubber tubes into the clavicular or caudal thoracic air sacs. The placement of the air sac tube is usually performed after induction by injection, face mask, or anesthetic chamber. In cases of severe airway obstruction it is possible to place the tube in a physically restrained conscious bird. The

FIGURE 7-19 The Bethune circuit attached to a Bethune endotracheal tube. Note the reduction of dead space between the circuit and bird.

FIGURE 7-20 Placement of an air sac tube in a scarlet macaw (*Ara macao*).

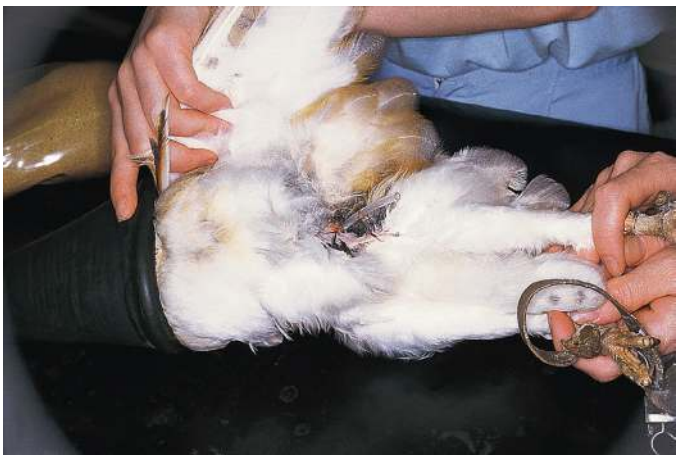


FIGURE 7-21 A restrained owl with head in mask breathing 100% oxygen while an air sac tube is placed.

placement of the tube in a conscious bird is quick and appears to cause little discomfort or distress. In emergency situations, to reduce the risks that are associated with handling respiratory distress, the bird may be restrained with its head in a mask into which 100% oxygen is delivered (Lawton, 1996b).

As large a tube as possible (French gauge 14) should be placed attached to the anesthetic circuit. IPPV via the placed tube is required while the bird is under anesthesia because birds with air sac intubation will usually stop breathing spontaneously as a result of the expulsion of all carbon dioxide from the respiratory system (Korbel *et al.*, 1993). Ventilated birds will not breathe again spontaneously until after perfusion via the air sac is terminated and the blood carbon dioxide levels rise. The tube can be removed postoperatively or left in situ in cases of dyspnea (e.g., after surgery to the neck or in cases of aspergillomas in the syrinx).

INJECTABLE ANESTHESIA

If an injectable agent is to be used, the bird should be accurately weighed. Without an accurate weight, it is not possible to calculate an accurate dose, and an overdose and even a fatality could occur. Where induction with a volatile anesthetic agent is performed, it is less stressful to weigh the bird after induction and should be done before the administration of any other agent. A recent publication assesses the effects of anesthesia with a combination of xylazine, diazepam, and ketamine on the heart rate, respiratory rate, and cloacal temperatures in the domestic fowl (*Gallus domesticus*; Mostachio *et al.*, 2008). A practical application of the use of an injectable anesthetic was recently described when a combination of propofol and a mixture of local anesthetics were used to implant satellite transmitters in bar-tailed godwits (*Limosa lapponica*) and bristle-thighed curlews (*Numenius tahitiensis*; Mulcahy *et al.*, 2011). Injectable anesthetic agents are listed in Table 7-2.

Intranasal Inoculation Anesthesia

Intranasal anesthesia is a novel and practical method for inducing anesthesia in birds, and it is currently receiving much attention. Recently, an experimental work was carried out in domestic pigeons (*Columba livia*) to assess the efficiency of inducing sedation using midazolam alone or in combination with dexmedetomidine using the intranasal route. The effect was reversed by the administration of atipamezole (Hornak *et al.*, 2015). This study concluded that the use of midazolam alone did not produce adequate sedation. However, the combination of midazolam and dexmedetomidine appeared to provide a more effective degree of immobilization lasting 20 to 30 min at the dose rate of 5 mg/kg midazolam and 80 µg/kg dexmedetomidine. Adequate reversal occurred after the intranasal administration of 250 µg/kg of atipamezole, but full recovery was delayed over 10 minutes (Hornak *et al.*, 2015). It has to be noted that the combination of midazolam and dexmedetomidine caused significant depression on heart rate, respiratory rate, and cloacal temperature on the subjects, persisting until the end of the procedure (Hornak *et al.*, 2015). A similar study was carried out in ring-necked doves (*Streptopelia* sp.) comparing the effect of intranasal or intramuscular administration of isomer S+ ketamine and midazolam (Beier *et al.*, 2013). Each dove received 20 mg/kg of S+ ketamine and 3.5 mg/kg of midazolam intramuscularly. The same dose was administered intranasally 2 weeks later. This study determined that the administration of intranasal S+ ketamine and midazolam was an acceptable method to induce anesthesia in ring-necked doves (Beier *et al.*, 2013). In a study with budgerigars (*Melopsittacus undulatus*; Bigham, 2013), each bird received xylazine intranasally at the dose rate of 25.6 ± 2.2 mg/kg, or diazepam at the dose rate of 13.6 ± 1.1 mg/kg, or midazolam at the dose rate of 13.2 ± 1.3 mg/kg (Bigham, 2013). The results of this study suggested that the intranasal administration of midazolam or diazepam provided adequate sedation for diagnostic and minor procedures. However, the use of xylazine at the dose rate used in this study is not recommended

because the quality of sedation was insufficient to perform any clinical procedure (Bigham, 2013; Table 7-3).

ANESTHESIA MONITORING

Despite the considered safety of isoflurane, there is no excuse for complacency over monitoring during anesthesia. The depth of anesthesia may only be correctly controlled if the bird is carefully and continuously monitored. Monitoring of birds should be approached in exactly the same way as monitoring of any mammalian species, although it is considered to be more challenging (Flammer, 1989).

Reflexes

In birds the best reflexes to monitor are the palpebral, corneal, cere, toe pinch, and wing twitch. As the bird becomes more deeply anesthetized, the standard reflexes usually slow and decrease in strength and

TABLE 7-2 Injectable Anesthetic Agents

Agent	Dose and Route	Comments on Use	Disadvantages
Alphaxalone/ alphadolone	5-10 mg/kg IV; 36mg/kg IM, IP	Alphaxalone/alphadolone was considered a relatively good anesthetic agent (Harcourt-Brown, 1978); there is a wide safety margin but only a short length of action (Mandelker, 1987); the large volumes required make IV the preferred route; there are now better alternatives to this agent	Following IV administration there is often a transient apnea (Cooper and Frank, 1973, 1974), which can be alarming; IP or IM routes produce immobilization but poor analgesia (Cooper and Frank, 1973, 1974); there are reports of deaths when used in red-tailed hawks (Cooper and Redig, 1975)
Ketamine	20-50 mg/kg SC, IM, or IV In waterfowl 18 mg/kg, with further 9 mg/kg incremental doses as necessary, was reported as producing good immobilization (Borzio, 1973); Forbes (1991) recommended a sliding dosage: 30 mg/kg for up to 150 g bodyweight, 20 mg/kg for 200-400 g, 10 mg/kg for up to 1 kg but only 5 mg/kg for birds over 2 kg	First reported use in birds in 1972 (Mandelker, 1972); historically, ketamine was the drug of choice; it is now used less often in avian practice, although it is useful for reducing stress when handling larger species such as swans or other waterfowl; it has been used orally (Garner, 1988) for immobilizing a captive-bred hawk that had flown off and was avoiding recapture; the dose used was 100 mg/kg in a 30 g piece of meat, although it took up to 2 hours to have the desired effect; this route may also be used for catching ducks on a pond, free-ranging peacocks, etc.; ketamine may give up to 30 minutes anesthesia, with full recovery taking up to 3 hours (Ensley, 1979); the speed of recovery is dose dependent, which is inversely proportional to the body size (Boever and Wright, 1975); large waterfowl tend to recover more slowly than other birds because of their decreased metabolism	Ketamine by itself is a good sedative but a poor anesthetic, with poor muscle relaxation and little analgesia, although there is little respiratory or cardiovascular depression (Flammer, 1989); with ketamine, hippus (rhythmic contraction and dilation of the pupil) is seen until the bird becomes deeply anesthetized (Lawton, 1984); there is often wing flapping during recovery, even when used in combination with tranquilizers and this may continue for several minutes (Mandelker, 1987); the kidneys eliminate ketamine; toxicity may be noted in debilitated or dehydrated birds, and those with renal dysfunction; IV fluids can hasten recovery from ketamine by causing diuresis; doses of 35 mg/kg IV may cause immediate cardiac arrest or prolonged apnea followed by cardiac arrest in a number of raptors, and others that survive having convulsions after induction (Redig and Duke, 1976)
Ketamine/ diazepam or midazolam	Ketamine 10-30 mg/kg IV and diazepam 1-1.5 mg/kg IM or 0.2 mg/kg SC, IM	These are good combinations allowing a smooth induction and recovery when compared to ketamine by itself. The benefit of midazolam is that it can be mixed in the same syringe as ketamine, while diazepam has to be given as a separate injection	Mandelker (1988) considered these as the most effective combinations available but, with the introduction of medetomidine, which can be reversed, this no longer true
Ketamine/ medetomidine	1.5-2 mg/kg ketamine + 60-85 mg/kg medetomidine IM (reversed by atipamezole 250-380 mcg/kg IM)	The addition of medetomidine provides sedative and analgesic properties, with good muscle relaxation but no arrhythmias or respiratory depression (Jalanka, 1989). This combination is particularly good for waterfowl	Medetomidine has hypotensive, bradycardic and hypothermic effects
Ketamine/ xylazine	4.4 mg/kg ketamine + 2.2 mg/kg xylazine IV (then reversed by yohimbine 0.1 mg); atipamezole 250-380 mcg/kg IM can be used to reverse the effects of xylazine	The synergistic action of the combination of xylazine with ketamine produces smooth induction and improved muscle relaxation without difficulties in recovery due to residual ketamine effect (Degernes *et al.*, 1988); Petruzzi *et al.* (1988) found that 18.5 mg/kg ketamine and 1.5 mg/kg xylazine to be effective in raptors	Unreversed, there is a prolonged recovery and postoperative depression that may result in the bird being unable to perch properly or unable to feed, leading to hypothermia, hypoglycemia, and even death (Lawton, 1984); Lumeij (1993) also reported two deaths postoperatively (24 and 50 hours) in goshawks, which were attributed to severe sinus bradycardia

TABLE 7-2 Injectable Anesthetic Agents—cont'd

Agent	Dose and Route	Comments on Use	Disadvantages
Propofol	1.33-14 mg/kg IV	Very high safety margin and easily metabolized; a very smooth, rapid induction with good muscle relaxation with a short duration of 2-7 minutes (Heard, 1997)	High cost; propofol is metabolized far too quickly in birds to be realistically used by itself as an agent for surgery; the combination of propofol and isoflurane may lead to difficulties in keeping the bird anesthetized; intravenous propofol is considered to be more stressful than mask induction with isoflurane (Lawton 1996a, 1996b)
Tiletamine/ zolazepam	5-10 mg/kg IM	Tiletamine is a phencyclidine derivative that is more potent than ketamine; this combination provides good immobilization and is considered safe (Kreeger *et al.*, 1993)	Tiletamine causes convulsions unless given with a sedative, thus, the manufactured combination
Xylazine	1-20 mg/kg IM or IV (reversed with yohimbine hydrochloride, 0.1-0.2 mg/kg IV or atipamezole 250-380 mcg/kg IM)	Seldom used as a sole agent	Xylazine by itself is unreliable, causes bradycardia and AV block and is extremely respiratory depressant (Mandelker, 1987); the bradycardic effects can be reduced if atropine is used; Raptors may show a hypersensitivity to external stimuli, including increased trembling, vocalization, and labored respiration, and higher dosages did not increase the depth of sedation (Freed and Baker, 1989)

IM, Intramuscular; IP, intraperitoneal; IV, intravenous; SC, subcutaneous.

TABLE 7-3 Intranasal Agents

Agent	Dose and Route	Comments on Use	Disadvantages
Midazolam in combination with dexmedetomidine in domestic pigeons (*C. livia*)	5 mg/kg IN midazolam and 80 μg/kg IN dexmedetomidine; reversal with atipamezole 250 μg/kg IN	Adequate immobilization for 20-30 minutes	Delayed recovery after reversal with atipamezole; the combination caused significant depression on heart rate, respiratory rate, and cloacal temperature (Hornak *et al.*, 2015)
S+ ketamine in combination with midazolam in ring-necked doves (*Streptopelia* sp.)	20 mg/kg of S+ ketamine and 3.5 mg/kg midazolam IN	Adequate sedation	Beier *et al.*, 2013
Xylazine, diazepam, midazolam in budgerigars (*Melopsittacus undulatus*)	25.6 ± 2.2 mg/kg xylazine or 13.6 ± 1.1 mg/kg diazepam, or 13.2 ± 1.3 mg/kg midazolam	Adequate sedation using diazepam or midazolam	The use of IN xylazine is not recommended at such dose rates since it does not produce adequate sedation (Bigham, 2013)

IN, Intranasal(ly).

will eventually disappear. The toe (Fig. 7-22), cere, and wing reflexes disappear as the bird enters a medium plane of anesthesia. The corneal reflex (Fig. 7-23) is usually the last reflex to be abolished and shows that the bird is very deeply anesthetized (Lawton, 1996a). The tone of the jaw should also be assessed: it becomes less tense as the bird enters a medium plane of anesthesia.

Circulatory Volume

Birds are thought to be better able to tolerate blood loss than mammals (Heard, 1997), although hemorrhage is still a problem, especially in small birds. The amount of blood loss during surgery should be carefully monitored (if necessary by measuring swabs) and fluid therapy or even a blood transfusion should be considered. In an emergency situation pigeon blood can be used for most species, although there are always risks involved in this procedure, not least from viral infections.

The weight of the bird before and after surgery will allow assessment of fluid loss. Although many different figures for fluid

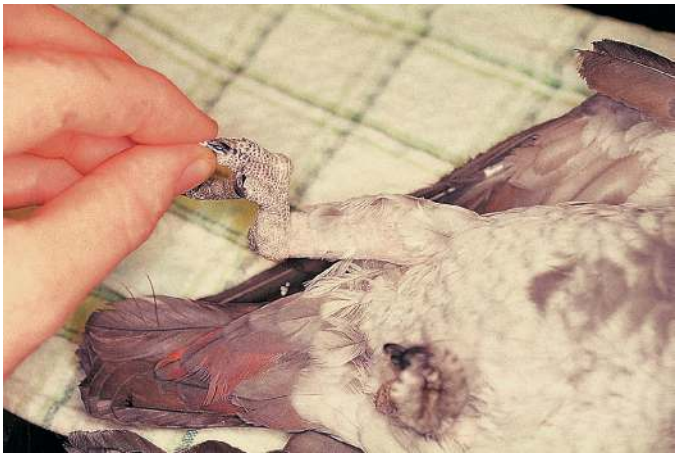


FIGURE 7-22 Demonstration of the toe pinch reflex in an African grey parrot.

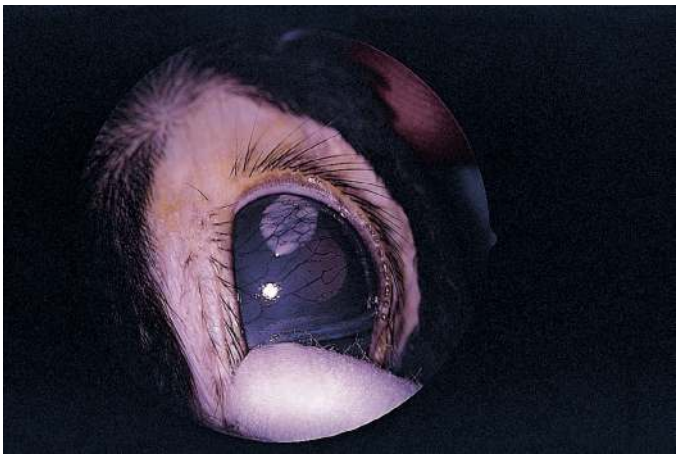


FIGURE 7-23 The corneal reflex is one of the last reflexes to be abolished under anesthesia.

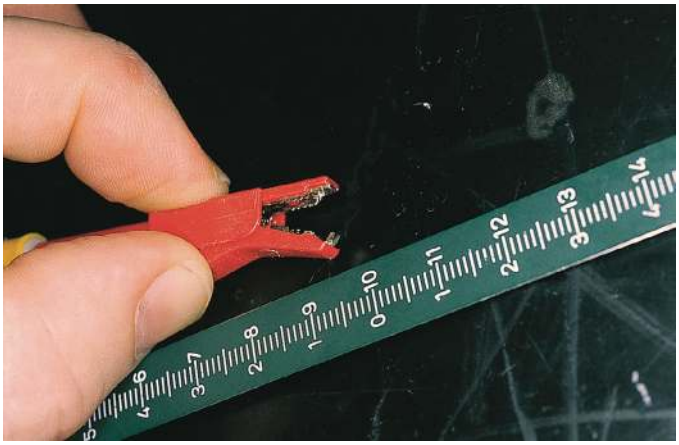


FIGURE 7-24 Suitable small ECG clamp, which is relatively atraumatic.

FIGURE 7-25 Placement of an ECG clamp onto the wing, at elbow level, in an African grey.

FIGURE 7-26 The standard placement of electric leads for ECG monitoring in an African grey: one on each wing and one on the right hind leg.

FIGURE 7-27 The appearance of the ECG trace from an anesthetized African grey parrot.

requirements exist, Sinn (1994) suggested that the normal daily requirement is ~5% of bodyweight in milliliters, while up to 10% of bodyweight in milliliters may be required for a dehydrated bird. If a daily intake of less than 5% is achieved, supplementation should be considered.

Pain

The response of the bird during surgery to painful stimuli will often show as a change in respiration, heart rate, or movement. The control of pain both during and after anesthesia is to be recommended. Analgesics (especially when anesthetic agents with poor analgesic properties are used) allow a more stable maintenance of anesthesia and reduce the possibility of surgical shock. Suitable analgesic agents are listed later in this chapter.

Electrocardiogram

Figures 7-24 to 7-28 illustrate electrocardiogram (ECG) equipment and lead placements. Where possible, the use of a cardiac monitor is recommended, although an esophageal stethoscope can be useful. Cardiac monitors are essential when certain anesthetic agents, such as xylazine, are used, to indicate whether atrioventricular block occurs. The standard lead placements are over the distal lateral tarsometatarsus and the carpal joints of each wing (Burtnick and Degernes, 1993) using atraumatic clamps or silver needles (Figs. 7-25 and 7-26). As an aid to the assessment of pain, the heart rate is dramatically effective (Figs. 7-27 and 7-28). It is not uncommon for a cockatiel, on feeling pain, to increase its heart rate from 300 to over 700 beats/min (Lawton, 1996a). The heart rate should never fall below 120 beats/min (Doolen and Jackson, 1991).

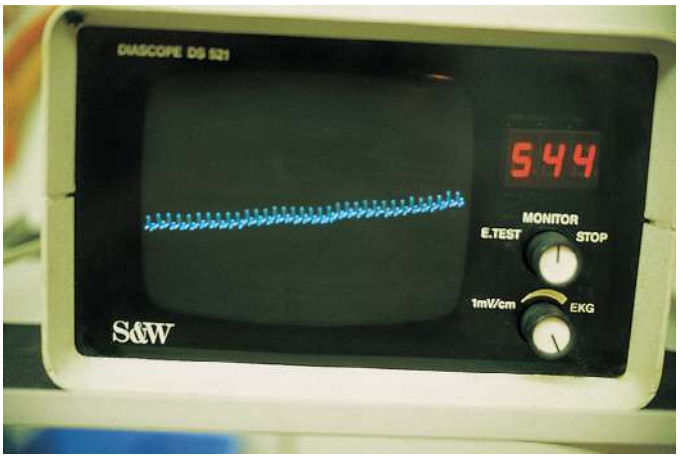


FIGURE 7-28 The same ECG trace for an African grey parrot as shown in Fig. 7-27, but after a response to pain. The ECG trace is a very good method of establishing the depth of anesthesia in avian patients.

FIGURE 7-30 Postoperatively an African grey parrot is disconnected from the anesthetic circuit, but a respiratory monitor probe is placed by the tube to continue monitoring respiration until the bird is fully recovered.

FIGURE 7-29 Careful monitoring of an anesthetized owl with respiratory monitor and continuous visual monitoring.

FIGURE 7-31 An African grey parrot recovering from anesthesia is placed in a cage with an infrared lamp close by to provide extra heat. Care has to be taken not to overheat the bird.

Doppler flow apparatus can also be used and may give an audible signal of arterial flow, as well as monitoring heart rate and rhythm (Heard, 1997). Some manufacturers produce a cloacal probe, or alternatively, a pediatric probe may also be used.

Respiration Monitor

Electronic monitoring of respiration is considered the best indicator of the depth and stability of anesthesia in the absence of response to pain. The pattern of respiration is also important: it should be stable and continually monitored during anesthesia (Figs. 7-29 and 7-30). A sudden change in pattern, especially in the depth of respiration (from shallow to deep), may indicate that the bird's plane of anesthesia is lightening or that the bird is feeling pain. As the bird enters a deeper plane of anesthesia, the rate and depth usually decrease. Depending on the bird's body size, the respiration rate should not fall below 25 to 50 beats/min (Doolen and Jackson, 1991); below this, there is a risk of hypercapnia. The respiratory rate of any anesthetized bird should never fall below its normal resting rate (Coles, 1985).

The majority of respiratory monitors work on thermal changes between inspired and expired gases. This can lead to difficulty in measuring small birds, especially when the flow rates of the cold carrier gases are high. A sensitive cardiac monitor (especially if it has an amplifier) will pick up the movement of the respiratory muscles and further assesses respiration. Pulse oximeters with a cloacal probe are useful for assessing the oxygenation of the blood and also the rate of respiration. IPPV allows the anesthetist to provide a defined suitable rate and depth of respiration, thus, removing the requirement of further monitoring. This is particularly useful in a patient with underlying respiratory disease.

Respiration should be carefully monitored (even if just by watching the movement of the sternum) until the bird is fully recovered from the anesthetic. If isoflurane is used, consideration should be given to holding the bird until it is recovered enough to perch (or to be released), allowing continuous observation. When injectable agents have been used, it may be necessary to strap or wrap the bird to prevent bruising or damage of the head or wings; this is particularly important when ketamine has been used (alone or in combination).

Temperature

Warmth should be provided before induction, during anesthesia, and in the recovery period (Fig. 7-31). Sick or anesthetized birds may not be able to maintain their core body temperature adequately (Figs. 7-32

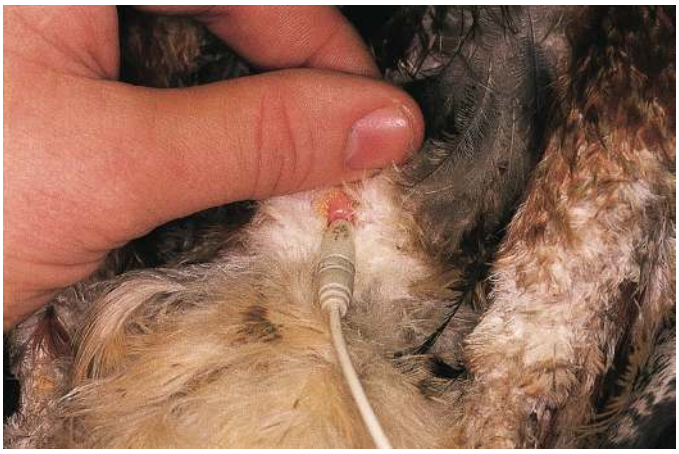


FIGURE 7-32 Placement of a temperature probe into the vent of a Harris hawk (*Parabuteo unicinctus*).

FIGURE 7-33 An anesthetized Harris hawk showing the monitoring of core body temperature via a cloacal probe.

and 7-33). Sick birds attempting to maintain their high core temperature may become hypoglycemic as a result of hypothermia. Hypothermia can cause peripheral vasoconstriction, bradycardia, hypotension, and, when severe, ventricular fibrillation (Heard, 1997).

Anesthetizing a bird and placing it onto a cold operating table may result in a rapid fall in body temperature. The core body temperature of birds is usually between 40°C and 44°C (104 to 111.2°F; Carter-Storm, 1988), with smaller birds at 41°C (105.8°F; Cooper, 1989). Excessive removal of feathers, preoperative washing, or application of surgical spirit at the site of surgery will result in lost insulation and heat loss. Anesthetized birds should be placed onto a towel or insulated Vetbed; the use of heating pads or lights can also help reduce heat loss but care must be taken to prevent overheating or burns. Bubble wrap or "space" sheets can also be used for wrapping most of the bird to prevent unnecessary heat loss. The use of OpSite (Smith and Nephew) would reduce the need to pluck a bird bald yet maintain an adequately clear surgical site. Cold anesthetic gases also have a chilling effect on the bird, but there is little that can be done to prevent this other than keeping the overall duration of anesthesia time as short as possible.

The cloacal temperature should be monitored during anesthesia (Doolen and Jackson, 1991). Likewise, a continuous assessment of hemorrhage should be done during surgery to prevent surgical shock and resultant hypothermia.

FIGURE 7-34 A veterinary technician in the process of anesthetizing a gyrfalcon (*Falco rusticolus*) within a field hospital. (Courtesy Dr. J. Samour.)

FIGURE 7-35 The same falcon as in Fig. 7-34. Note that the hood has been left on the falcon while its head is within the face mask. This facilitates handling and restraint during the induction period. (Courtesy Dr. J. Samour.)

Anesthesia Under Field Conditions

Today, modern hunting parties from the Middle East carry fully equipped field hospitals in which falcons can be safely anesthetized for different medical procedures. Figs. 7-34 and 7-35 illustrate the administration of anesthesia using isoflurane to a gyrfalcon (*Falco rusticolus*) under field conditions.

REFERENCES

Beier SL, Rosa AC, Oleskovicz N, et al: Anesthetic effects of intranasal or intramuscular administration of S+ ketamine and midazolam in ring necked dove (*Streptopelia* sp), *Pesquisa Vet Brasil* 33(4):517–522, 2013.

Bigham AS: Comparison of intranasal administration of xylazine, diazepam, and midazolam in budgerigars (*Melopsittacus undulatus*): clinical evaluation, *J Zoo Wildlife Med* 44(2):241–244, 2013.

Boever WJ, Wright W: Use of ketamine for restraint and anesthesia of birds, *Vet Med/Small Anim Clinician* 70:86, 1975.

Borzio F: Ketamine hydrochloride as an anesthetic for wildfowl, *Vet Med/Small Anim Clinician* 35:1364, 1973.

Burtnick NL, Degernes LA: Electrocardiography of fifty-nine anesthetized convalescing raptors. In Redig PT, Cooper JE, Remple JD, et al, editors: *Raptor biomedicine*, Minneapolis, MN, 1993, University of Minnesota Press.

Carter-Storm A: Special considerations for general anesthesia of birds, *Clin Insight* 2(3):61, 1988.

Chemonges S: Effect of intermittent positive pressure ventilation on depth of anesthesia during and after isoflurane anaesthesia in sulphur crested cockatoos (*Cacatua galerita*), *Vet Med Intl* 2014:250523, 2014.

Clutton RE: Prolonged isoflurane anesthesia in the golden eagle, *Zoo Anim Med* 17:103, 1986.

Coles BH: *Avian medicine and surgery*, ed 2, Oxford, UK, 1985, Blackwell Scientific.

Cooper JE: Anaesthesia of exotic species. In Hilbery ADR, Waterman AE, Brouwer GJ, editors: *Manual of anaesthesia of small animal practice*, ed 3, Cheltenham, UK, 1989, British Small Animal Veterinary Association.

Cooper JE, Frank LG: The use of the steroid anesthetic CT 1341 in birds, *Vet Rec* 92:474, 1973.

Cooper JE, Frank LG: The use of the steroid anesthetic CT 1341 in birds, *Raptor Res* 8:20, 1974.

Cooper JE, Redig PT: Unexpected reaction to the use of CT 1341 by red tailed hawks, *Vet Rec* 97:352, 1975.

de Paula VV, Oysuki DA, Aulet JO, et al: The effect of premedication with ketamine, alone or with diazepam, on anesthesia with sevoflurane in parrots (*Amazona aestiva*), *BMC Vet Res* 9(1):6148–9-142, 2013.

Degernes LA, Kreeger TJ, Redig PT: Ketamine-xylazine anesthesia in red-tailed hawks with antagonism by yohimbine, *J Wildlife Dis* 24:322, 1988.

Dohoo SE: Isoflurane as an inhalational anesthetic agent in clinical practice, *Can Vet J* 31:847, 1990.

Doolen MD, Jackson L: Anesthesia in caged birds, *Iowa State Univ Vet* 53(2):76, 1991.

Edling TM: *Inhalation anesthesia, monitoring and anesthetic pain management*, Proceedings of the Association of Avian Veterinarians, Pittsburgh, PA, 2003.

Edling TM: Anesthesia and monitoring. In Harrison GJ, Lightfoot TL, editors: *Clinical avian medicine*, Palm Beach, FL, 2006, Spix Publishing.

Ensley P: Cage bird medicine and husbandry, *Vet Clin North Am Small Anim Pract* 9:391, 1979.

Fedde MR: Respiration. In Sturkie PD, editor: *Avian physiology*, ed 4, New York, 1986, Springer-Verlag.

Fitzgerald G, Blais D: Inhalation anesthesia in birds of prey. In Redig PT, Cooper JE, Remple JD, et al, editors: *Raptor biomedicine*, Minneapolis, MN, 1993, University of Minnesota Press.

Flammer K: Update on avian anesthesia. In Kirk RW, Bonagura JD, editors: *Current veterinary therapy X*, Philadelphia, PA, 1989, WB Saunders.

Forbes NA: Birds of prey. In Beynon PH, Cooper JE, editors: *Manual of exotic pets*, Cheltenham, UK, 1991, British Small Animal Veterinary Association.

Freed D, Baker B: Antagonism of xylazine hydrochloride sedation in raptors by yohimbine hydrochloride, *J Wildlife Dis* 25:136, 1989.

Garner MM: Use of an oral immobilizing agent to capture a Harris' hawk (*Parabuteo unicinctus*), *J Raptor Res* 22:70, 1988.

Harcourt-Brown NH: Avian anaesthesia in general practice, *J Small Anim Pract* 19:573, 1978.

Heard DJ: Anesthesia and analgesia. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Heatley JJ: Anesthesia and analgesia. In Chitty J, Lierz M, editors: *BSAVA manual of raptors, pigeons and passerine birds*, Quedgeley, Gloucester, UK, 2008, British Small Animal Veterinary Association.

Hornak S, Liptak T, Ledecky V, et al: A preliminary trial of the sedation induced by intranasal administration of midazolam alone or in combination with dexmedetomidine and reversal by atipamezole for a short-term immobilization in pigeons, *Vet Anaesthes Analges* 42(2):192–196, 2015.

Jalanka HH: Chemical restraint and reversal in captive markhors (*Capra falconeri megaceros*): a comparison of two methods, *J Zoo Wildlife Med* 20:413, 1989.

James AE, Hutchings G, Bush M, et al: How birds breathe: correlation of radiographic with anatomical and pathological studies, *J Am Radiol Soc* 17:77, 1976.

Jenkins JR: Post-operative care of the avian patient, *Semin Avian Exot Pet Med* 2:97–102, 1993.

King AS, McLelland J: *Outline of avian anatomy*, London, 1975, Baillière Tindall.

Klaphake E, Schumacher J, Greenacre C, et al: Comparative anesthetic and cardiopulmonary effects of pre- versus postoperative butorphanol administration in Hispaniolan Amazon parrots (*Amazona ventralis*) anesthetized with sevoflurane, *J Avian Med Surg* 20(1):2–7, 2006.

Korbel R, Milovanovic A, Erhardt W, et al: *Aerosaccular perfusion with isoflurane—an anesthetic procedure for head surgery of birds*, Proceedings of the European Association of Avian Veterinarians, Utrecht, The Netherlands, 1993.

Kreeger TJ, Degernes LA, Kreeger JS, et al: Immobilization of raptors with tiletamine and zolazepam (Telazol). In Redig PT, Cooper JE, Remple JD, et al, editors: *Raptor biomedicine*, Minneapolis, MN, 1993, University of Minnesota Press.

Lawton MPC: Avian anaesthesia, *Vet Rec* 115:71, 1984.

Lawton MPC: Anaesthesia. In Beynon PH, Forbes NA, Lawton MPC, editors: *Manual of psittacine birds*, Cheltenham, UK, 1996a, British Small Animal Veterinary Association.

Lawton MPC: Anaesthesia. In Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *Manual of raptors pigeons and waterfowl*, Cheltenham, UK, 1996b, British Small Animal Veterinary Association.

Lennox AM, Crosta L, Buerkle M: *The effects of isoflurane anesthesia on gastrointestinal transit time*, Proceedings of the Association of Avian Veterinarians, Monterey, 2002.

Lumeij JT: Effects of ketamine-xylazine anesthesia on adrenal function and cardiac induction in goshawks and pigeons. In Redig PT, Cooper JE, Remple JD, et al, editors: *Raptor biomedicine*, Minneapolis, MN, 1993, University of Minnesota Press.

Mandelker L: Ketamine hydrochloride as an anesthetic for parakeets, *Vet Med/Small Anim Clinician* 68:55, 1972.

Mandelker L: Anesthesia and surgery. In Burr EW, editor: *Companion bird medicine*, Ames, IA, 1987, Iowa State University Press.

Mandelker L: Avian anesthesia, part II: injectable agents, *Compan Anim Pract* 2(10):21, 1988.

McLelland J: *A colour atlas of avian anatomy*, London, 1990, Wolfe Publishing.

Mostachio GQ, De-Oliveira LD, Carciofi AC, et al: Effects of anesthesia with a combination of intramuscular xylazine-diazepam-ketamine on heart rate, respiratory rate and cloacal temperature in roosters, *Vet Anaesthes Analges* 35(3):232–236, 2008.

Mulcahy DM, Gartrell B, Gill RE, et al: Coelomic implantation of satellite transmitters in the bar tailed godwit (*Limosa lapponica*) and the bristle-thighed curlew (*Numenius tahitiensis*) using propofol , bupivacaine, and lidocaine, *J Zoo Wildlife Med* 42(1):54–64, 2011.

Petruzzi V, Coda S, Ximenes LA, et al: L'associazione ketamina-xilazina nell' anesthesia generale dei rapaci. Valutazione di alcuni parametri vitali, *Documenti Veterinari* 6:59–62, 1988.

Phair KA, Larsen RS, Wack RF, et al: Determination of the minimum anesthetic concentration of sevoflurane in thick-billed parrots (*Rhynchopsitta pachyrhyncha*), *Am J Vet Res* 73(9):350–1355, 2012.

Redig PT, Duke GE: Intravenously administered ketamine HCl and diazepam for anesthesia of raptors, *J Am Vet Med Assoc* 169(9):886, 1976.

Rosskopf WJ Jr, Woerpel RW: Practical anesthesia administration. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, MD, 1996, Williams & Wilkins.

Rosskopf WJ Jr, Woerpel RW, Reed S, et al: Anesthetic agents: anesthesia administration for pet birds, *Vet Pract Staff* 4(2):34, 1992.

Scheid P, Piiper J: Direct measurement of the pathway of the respired gas in duck lungs, *Respir Physiol* 11:308, 1971.

Sinn LC: Anesthesiology. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and applications*, Lake Worth, FL, 1994, Wingers Publishing.

Tully TN, Dorrestein G, Jones A: *Handbook of avian medicine*, ed 2, Philadelphia, PA, 2009, WB Saunders.

ANALGESIA

David Sanchez-Migallon Guzman, Michelle G. Hawkins, Joanne Paul-Murphy

RECOGNITION OF PAIN

Pain is defined as the sensory and emotional experience associated with actual or potential tissue damage. Pain is subjective and has an emotional component that can be difficult for us to translate because most avian species lack facial expression and do not share verbal language with humans. Nociception is the transduction, conduction, and central nervous system processing of signals generated by the stimulation of nociceptors. It is the physiologic process that, when performed to completion in a conscious animal, results in the perception of pain.

Behavioral changes associated with pain in birds can be subtle (Fig. 7-36). Behavioral changes do manifest differently among different species of birds, and observers must become familiar with the full range of normal behaviors for the species and the individual. Birds tend to respond to noxious stimuli with a fight-or-flight response (i.e., escape reactions, vocalization, excessive movement) and/or conservation–withdrawal responses (i.e., no escape attempts or minimal vocalization and immobility) (Fig. 7-37). In domestic chickens, removal of feathers caused a progression of behavioral changes from an initial alert-agitated response to periods of crouching immobility following successive removal of feathers (Gentle and Hunter, 1991). Movement, head motions, and beak clacks were all significantly reduced in red-tailed hawks hospitalized with recent orthopedic injuries, compared with birds without recent orthopedic injuries (Mazor-Thomas, *et al.*, 2014). Social interactions might decrease in birds that have social systems. Birds in pain may display guarding behavior to protect a painful area. Grooming activity may decrease when a bird is in pain; conversely, over grooming and feather-destructive behavior have been associated with chronic pain, which may include neuropathic pain.

Pain scales and score sheets are tools increasingly used to assess pain in animals, especially when specifically designed for a given species under well-defined conditions. Using pain scales requires understanding normal and pain-related behavior for the species and individual. Pain score sheets can help improve uniformity and efficacy of pain scoring using behavioral analysis. Score-sheet descriptions of behavior must be refined, and terms must be clearly defined to reduce observer bias and interobserver variability. In lieu of species-specific pain score sheets, a generic pain scale of 1 to 10 is useful for assessing bird's pain and the response to treatment and recovery from a painful condition. In a study using pigeons with experimental fractures, two numeric pain scales (scores from 0 to 5) were sensitive and specific for detecting pain. The first evaluated the position of the fractured limb (from 0, bearing the same weight on both feet, to 5, lying on the floor) and the second evaluated the overall assessment of the bird (from 0, no sign of pain, to 5, unable to stand, lying on the floor; Desmarchelier *et al.*, 2012a). Effective analgesia is expected to show a marked, easily discernable change in posture or behaviors that will effect a reliable change in the subjective pain score. If no change in pain score occurs, then the drugs, dosage, or frequency of administration should be reevaluated for that patient.

TREATMENT OF PAIN

General Approaches

Pre-emptive analgesia with administration of analgesics before tissue injury will prevent central and peripheral sensitization. Preemptive analgesia with opioid drugs, nonsteroidal antiinflammatory drugs (NSAIDs), and/or local anesthetics can block transmission of sensory noxious stimuli to the central nervous system, which will reduce the overall potential for inflammation and pain and improve the patient's short-term and long-term recovery. Multimodal protocols with opioids, NSAIDs, local anesthetics, and/or other drugs acting at different points in the nociceptive system provide a greater effect and potentially less toxicity than individual drugs given alone.

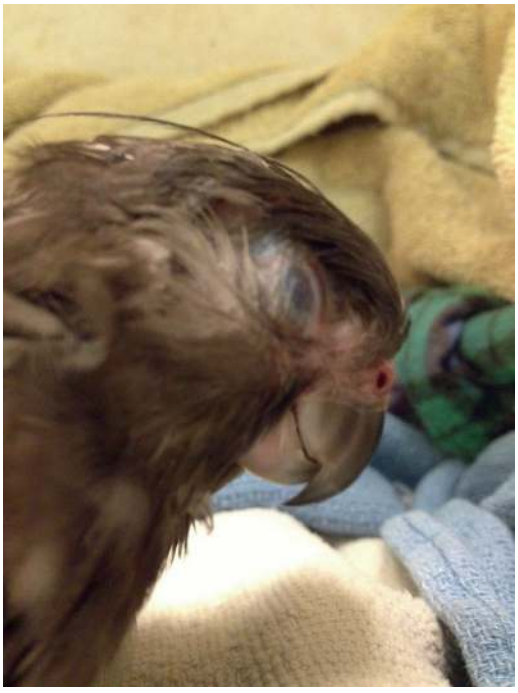


FIGURE 7-36 Cockatiel (*Nymphicus hollandicus*) 2 hours after surgical excision of the uropygial gland with closed eyes and the head withdrawn into the body consistent with signs of pain. The bird appeared brighter and more active shortly after administration of butorphanol.

FIGURE 7-37 American kestrel (*F. sparverius*) in a testing box with a thermal perch. This species has been used as a model for other raptor species, using the thermal antinociceptive model to evaluate the potential analgesics effects.

OPIOID DRUGS

Opioids are used for moderate to severe pain, such as traumatic or surgical pain. Opioids reversibly bind to specific receptors in the central and peripheral nervous system. These drugs are categorized as either agonists, partial agonists, mixed agonist/antagonists, or antagonists based upon their ability to induce an analgesic response once bound to a specific receptor. The action of opioid drugs on these receptors activates G-proteins, leading to a reduction in transmission of nerve impulses and inhibition of neurotransmitter release (Bovill, 1997). Agonist drugs have a linear dose–response curve that may be titrated to reach the desired effect, whereas the agonist/antagonist drugs may reach a ceiling effect after which increasing the dose does not appear to provide additional analgesia. During anesthesia, opioids are used to provide perioperative analgesia that may reduce the concentrations of volatile anesthetics (i.e., gas anesthesia-sparing effects). The most common adverse effects reported with opioids are cardiac and/or respiratory depression. In many cases, these drugs may be reversed with antagonists, which will also terminate analgesia. The application and dosages of several opioid formulations have been scientifically evaluated and clinically applied in birds (Table 7-4).

Few studies have been conducted in birds to evaluate distribution, quantity, and function of each opioid receptor type, and although marked species variability is assumed, further studies are necessary. In pigeons, the regional distribution of μ, κ, and δ receptors in the forebrain and midbrain were similar to mammals, but the κ and δ receptors were more prominent in the pigeon forebrain than μ receptors and 76% of opioid receptors in the forebrain were determined to be κ-type (Mansour *et al.*, 1988). In one-day-old chicks, marked dissimilarities to this distribution suggest either age- or species-related

TABLE 7-4 Opioid Drugs Evaluated in Avian Species by Either Pharmacokinetic or Pharmacodynamic Studies

Drug	Dosage mg/kg	Species	Comments	References
Butorphanol	1, 3, and 6 IM	American kestrel	Failed to increase foot withdrawal thermal thresholds; instead caused hyperesthesia or hyperalgesia and agitation in males receiving 6 mg/kg; do not recommend in American kestrels	Sanchez-Migallon Guzman *et al.*, 2014a
	2, 4 IV	Guinea fowl	Resulted in arrhythmias and hypotension at 4 mg/kg in anesthetized guinea fowl; one bird died	Escobar *et al.*, 2012
	2 IV	Domestic chickens	Remained above target plasma concentration for analgesia in mammals for ~2 hours	Singh *et al.*, 2011
	5 IV, IM, PO	Hispaniolan Amazon parrots	Remained above target plasma concentration for ~2 hours after IV and IM administration; oral administration had poor oral bioavailability (<10%)	Sanchez-Migallon Guzman *et al.*, 2011a
	2 IM	Hispaniolan Amazon parrots	No significant cardiorespiratory depression with sevoflurane anesthesia	Klaphake *et al.*, 2006
	2, 5 IM	Hispaniolan Amazon parrots	Failed to increase thermal and electrical withdrawal thresholds after 2 mg/kg IM; serum target plasma concentrations maintained at 2 hours following 5 mg/kg	Sladky *et al.*, 2006
	1 IM	Cockatoos African grey parrots Blue-fronted Amazon parrots	Reduced isoflurane MAC in the cockatoos and African greys, but not Amazon parrots	Curro 1994, Curro *et al.*, 1994
	1 IM	African grey parrots	Increased electrical withdrawal threshold after 1 mg/kg in over 50% of the birds tested	Paul-Murphy *et al.*, 1999
Nalbuphine	12.5, 25, and 50 IM; 12.5 IV	Hispaniolan Amazon parrots	Resulted in increased thermal thresholds for 3 hours at 12.5 mg/kg; higher doses did not increase analgesic time	Sanchez-Migallon Guzman *et al.*, 2011b; Keller *et al.*, 2011
Nalbuphine sustained-release	37.5, 33.7 IM	Hispaniolan Amazon parrots	Remained above target plasma human concentrations for 24 hours after 37.5 mg/kg; Increased thermal withdrawal threshold after 33.7 mg/kg for 12 hours	Sanchez-Migallon Guzman *et al.*, 2013a,b
Hydromorphone	0.1, 0.3, and 0.6 IM	Cockatiels	Failed to increased thermal withdrawal thresholds; some birds had mild sedation at the highest dosages	Sanchez-Migallon Guzman *et al.*, 2014e
	0.1, 0.3, and 0.6 IM; 0.6 IV	American kestrels	Increased thermal withdrawal thresholds for 3-6 hours; some birds moderate sedation at the highest dosages	Sanchez-Migallon Guzman *et al.*, 2013c, 2014c

Continued

TABLE 7-4 Opioid Drugs Evaluated in Avian Species by Either Pharmacokinetic or Pharmacodynamic Studies—cont'd

Drug	Dosage mg/kg	Species	Comments	References
Fentanyl	Targeted controlled infusions	Hispaniolan Amazon parrots	Reduced isoflurane MAC in a dose-dependent manner, with significant effects on heart rate and blood pressure	Hawkins, *et al.*, 2014
	Targeted controlled infusions 10-30 μg/kg/h IV	Red-tailed hawks	Reduced isoflurane MAC 31%-55% in a dose-related manner, without significant effects on heart rate, blood pressure, $PaCO_2$, or PaO_2	Pavez *et al.*, 2011
	0.02 IM, 0.2 SC	White cockatoos	Increased withdrawal thermal thresholds at 0.2 mg/kg withdrawal thresholds to electrical and thermal stimuli; 0.02 mg/kg did not affect either threshold; 0.2 mg/kg affected both withdrawal thresholds only some birds; hyperactivity in first 15-30 minutes	Hoppes *et al.*, 2003
	0.05-1.0 IA	Chickens	No significant effect on induced arthritis	Gentle *et al.*, 1999
Buprenorphine	0.6, 1.2, and 1.8 IM	Cockatiels	Failed to increased thermal withdrawal thresholds; some birds had mild sedation at the highest dosages	Sanchez-Migallon Guzman *et al.*, 2014d
	0.1, 0.3, and 0.6 IM	American kestrels	Increased thermal withdrawal thresholds for 6 hours	Ceulemans *et al.*, 2014; Gustavsen, *et al.*, 2014
	0.1 IM	African grey parrots	Failed to achieve target plasma concentrations and to increase withdrawal electrical threshold	Paul-Murphy, *et al.*, 1999, 2004
	0.25, 0.5 IM	Domestic pigeons	Increased latency withdrawal to electrical stimulus for 2-5 hours	Gaggermeier, *et al.*, 2003
	0.05, 1.0 IA	Chickens	No significant effect on induced arthritis	Gentle *et al.*, 1999
Buprenorphine sustained-release formulation	1.8 IM	American kestrels	Increased thermal withdrawal threshold for 12-24 hours	Sanchez-Migallon Guzman, personal communication
Tramadol	10 PO	Penguin		Kilburn *et al.*, 2014
	5, 15, and 30 PO	American kestrel	Increased thermal withdrawal thresholds at 5 mg/kg; higher doses resulted in less effect	Sanchez-Migallon Guzman *et al.*, 2014b
	10, 20, and 30 PO; 30 PO	Hispaniolan Amazon parrots	Remained above target plasma concentrations for ~8 hours after 30 mg/kg PO; increased thermal withdrawal threshold at 30 mg/kg but not at 10 or 20 mg/kg; 30 mg/kg needed for significant antinociceptive effects	Sanchez-Migallon Guzman *et al.*, 2012; Souza *et al.*, 2012, 2013
	11 PO, IV	Red-tailed hawks	Remained above target plasma concentrations for ~4 hours (but only three birds in study); recommended 15 mg/kg PO q 12 hours	Souza *et al.*, 2011
	7.5 PO	Indian peafowl	Remained above target plasma concentrations for ~12 hours in most birds	Black *et al.*, 2010
	4 IV, 11 PO	Bald eagles	Recommended 5 mg/kg PO q 12 hours based on plasma concentrations	Souza *et al.*, 2009

IA, Intraarticular; IM, intramuscular; IP, intraperitoneal; IV, intravenous; MAC, minimum anesthetic concentration; PO, *per os* (by mouth); SC, subcutaneous.

differences (Csillag *et al.*, 1990). Some of these dissimilarities might account for the varying responses to different opioid drugs identified between bird species.

Butorphanol is a mixed κ-opioid receptor agonist and μ-opioid receptor antagonist opioid drug. This opioid has historically been one of the most commonly utilized opioid analgesics in birds, despite many studies suggesting it requires very frequent dosing. Isoflurane-sparing studies using 1 mg/kg intramuscular (IM) butorphanol in three psittacine species showed species variability either in response to the drug itself or to the dose administered (Curro, 1994; Curro *et al.*, 1994). In studies using withdrawal thresholds with electrical stimulus (Paul-Murphy *et al.*, 1999) or thermal stimulus in psittacines, a wide range of dosages ranging from 1 to 6 mg/kg IM proved effective, but a ceiling effect appeared to be achieved between 3 and 6 mg/kg. Based on these studies, dosages of 1 to 4 mg/kg IM, IV (intravenous), or SC (subcutaneous) have been recommended in psittacines for the treatment of moderate to severe pain. In contrast, in studies with American kestrels (*F. sparverius*) using thermal stimulus (Sanchez-Migallon Guzman *et al.*, 2014a) butorphanol tartrate failed to increase foot withdrawal thermal thresholds at dosages ranging from 1 to 6 mg/kg IM. Because of these findings, butorphanol is not recommended in kestrels for pain management, and additional studies in this and other raptorial species are needed to provide further recommendations. There is evidence in psittacines to suggest that butorphanol is safe when

administered perioperatively and does not produce respiratory depression following 2 mg/kg IM (Klaphake *et al.*, 2006), while studies in other species have raised concerns about cardiovascular effects (Escobar *et al.*, 2014). Butorphanol pharmacokinetic (PK) studies have been published in several species of birds including raptors (Riggs *et al.*, 2008; Sanchez-Migallon Guzman *et al.*, 2014a), chickens (Singh *et al.*, 2010), and psittacines (Sanchez-Migallon Guzman *et al.*, 2011a). From all of these studies, it can be concluded that butorphanol has a very short half-life and requires frequent administration q 2 to 3 hours at the dosages recommended. It is also important to highlight that in the species studied, butorphanol has poor oral bioavailability (e.g., <10%) and this route of administration is not recommended (Sanchez-Migallon Guzman *et al.*, 2011a). Because of this short half-life and duration of action, efforts have been made in developing and investigating slow-release formulations of butorphanol (Sladky *et al.*, 2006; Paul-Murphy *et al.*, 2009a,b), but it is currently not commercially available. Two of the authors (D.S.M.G. and J.P.M.) use butorphanol as an IV continuous rate infusion (CRI) when needed in psittacines at 1 mg/kg/h, but studies are needed to determine appropriate rate recommendations.

Nalbuphine is a κ-opioid receptor agonist and μ-opioid receptor antagonist, with a similar mechanism of action to butorphanol. It is used as an analgesic in the treatment of moderate to severe pain in humans and has a relatively low incidence of respiratory depression that does not increase with additional dosing. Nalbuphine has shown antinociceptive efficacy in Amazon parrots using withdraw thresholds with thermal stimulus at 12.5, 25, and 50 mg/kg IM without causing sedation (Sanchez-Migallon Guzman *et al.*, 2011b). In this study, the highest dosages did not show additional benefit when compared with 12.5 mg/kg, suggesting a ceiling effect. PK studies in Amazon parrots have shown a short half-life comparable to butorphanol, (Keller *et al.*, 2011) and frequent administration q 2 to 3 hours would also be required for this formulation. Efforts have also been made in developing and investigating slow-release formulations of nalbuphine (Sanchez-Migallon Guzman *et al.*, 2013a,b), but as with butorphanol, a slow-release formulation is not currently available.

Morphine is a μ-receptor agonist that has been infrequently used in avian medicine because some of the early studies with this drug in domestic fowl yielded conflicting results. They also suggested that very high dosages were required to reduce response to toe pinch (Schneider, 1961) or a noxious electrical stimulation (Bardo and Hughes, 1978). Further investigations using noxious thermal stimulation with chickens revealed strain-dependent effects (Fan *et al.*, 1981) with morphine administration resulting in analgesia in some strains and hyperalgesia in others (Hughes, 1990). A study in chickens using the isoflurane-sparing technique found that increasing doses of morphine caused a decrease in the isoflurane minimum anesthetic concentration (Concannon *et al.*, 1995). The PK profile of morphine at 2 mg/kg IV has (Singh *et al.*, 2010) a short half-life in chickens, similar to other opioids in other species of birds.

Hydromorphone is a μ-receptor agonist with a potency ~5 to 7 times that of morphine, and with similar potency to oxymorphone. Hydromorphone has gained attention in avian medicine following the results of recent studies. In a study using withdrawal thresholds with thermal stimulus in American kestrels, hydromorphone was effective at dosages ranging from 0.1 to 0.6 mg/kg IM (Sanchez-Migallon Guzman *et al.*, 2013c) with effects lasting up to 6 hours at the highest dosage evaluated. Moderate to severe sedation was also noted in some birds at 0.6 mg/kg, and these birds appeared agitated when handled. A similar study in cockatiels, evaluating the same dosages of hydromorphone, had very different results, and hydromorphone failed to increase the thermal withdrawal thresholds or cause sedation (Sanchez-Migallon Guzman *et al.*, 2014e). Hydromorphone PK in cockatiels and kestrels had a similar profile, with good IM bioavailability and a very short half-life (Sanchez-Migallon Guzman *et al.*, 2014e). Based on the results of these studies, hydromorphone would be recommended in kestrels for treatment of moderate to severe pain but cannot be recommended in psittacines before additional studies are done.

Fentanyl is a short-acting μ-receptor agonist not commonly used in avian medicine until recently. Fentanyl has shown efficacy using thermal and electrical withdrawal thresholds at relatively high SC dosages in white cockatoos (*C. alba*). Many birds appeared hyperactive for the first 15 to 30 minutes after receiving a high dose (Hoppes *et al.*, 2003). The PK profile of fentanyl in white cockatoos had a very short half-life (Hoppes *et al.*, 2003). Fentanyl as an IV CRI in red-tailed hawks decreased the concentration of isoflurane needed to respond to an electrical noxious stimulus and maintained cardiovascular stability (Pavez *et al.*, 2011). In HAPS, Hispaniola amazon parrot (*Amazona ventralis*), approximately 20 times higher dosing is necessary to achieve the same reductions in isoflurane anesthetic concentrations, causing minor cardiovascular and respiratory effects, which could be potentially significant in clinical cases. Based on the results of these studies, fentanyl is recommended by the authors in red-tailed hawks for moderate to severe pain as a CRI, but the cardiovascular effects in psittacines at these dosages warrants further investigation before recommending it.

Buprenorphine is a partial μ-receptor agonist with κ-receptor activities that are less well defined. Buprenorphine has a slow onset of action with longer duration than other opioid drugs and a unique pharmacological profile. In African grey parrots (*Psittacus erithacus*) and cockatiels using a noxious stimulus, buprenorphine at dosages ranging from 0.1 to 1.8 mg/kg IM failed to increase withdrawal thresholds or cause sedation (Paul-Murphy *et al.*, 1999; Sanchez-Migallon Guzman *et al.*, 2014d). In Columbiformes using electrical stimulus at a dosage of 0.25 to 5 mg/kg IM (Gaggermeier *et al.*, 2003) and in kestrels using a thermal stimulus at a dosage of 0.1 to 0.6 mg/kg (Ceulemans *et al.*, 2014) buprenorphine was effective in increasing the withdrawal thresholds and caused sedation. In a similar study, a commercially available slow-release formulation of buprenorphine resulted in thermal antinociception in American kestrels for almost 24 hours following 1.8 mg/kg IM administration (Sanchez-Migallon Guzman *et al.*, 2015). Interestingly, the PK profile of buprenorphine has been evaluated in kestrels and African grey parrots and does not show major differences between the species evaluated (Paul-Murphy *et al.*, 2004; Gustavsen *et al.*, 2014). Based on the results of these studies, hydromorphone would be recommended in kestrels and pigeons for treatment of moderate pain but cannot be recommended in psittacines before additional studies are done.

Tramadol is a weak μ-receptor agonist opioid derivative that also inhibits the uptake of serotonin and norepinephrine (Scott and Perry, 2000). Most of the effects on the μ-receptor are attributed to the O-desmethyl metabolite (M1) and conversion to the M1 metabolite has been demonstrated in the avian species studied (Souza *et al.*, 2009, 2011, 2012; Black *et al.*, 2010; Kilburn *et al.*, 2014). In humans, less respiratory depression and constipation are seen with tramadol than with other μ-agonist opioids, but there are no data on these potential adverse effects in birds. The biggest advantage over other opioid drugs is that tramadol can be administered orally. The PK profile has been evaluated in raptors, psittacines, peafowl, and penguins with important differences in the oral bioavailability. These differences in oral bioavailability can explain the difference in dosage recommendation between the species (Souza *et al.*, 2009, 2011, 2012; Black *et al.*, 2010; Kilburn *et al.*, 2014). For example, oral bioavailability of tramadol was 97.94% in American bald eagles (Souza *et al.*, 2009), while only 23.48% in Hispaniolan Amazon parrots (Souza *et al.*, 2012). In both Amazon

parrots and kestrels, tramadol has shown efficacy, in increasing thermal withdrawal thresholds but at significantly different dosages, and can be recommended in both species for the treatment of mild to moderate pain (Sanchez-Migallon Guzman *et al.*, 2012, 2014b).

NONSTEROIDAL ANTIINFLAMMATORY DRUGS

NSAIDs are used to relieve musculoskeletal and visceral and acute and chronic pain (Figs. 7-38 and 7-39). The pharmacological activity of NSAIDs has been reviewed elsewhere and the mechanism of action is thought to be similar when administered to birds (Bergh and Budsberg, 2005; Papich, 2008). A broad tissue distribution of cyclooxygenase (COX) has been demonstrated in chickens (Mathonnet *et al.*, 2001). The relative expression of COX-1 and COX-2 enzymes varies between species and both enzymes are important in avian pain and inflammation (Table 7-5).

The most common adverse effects of NSAIDs include effects on the gastrointestinal system, renal system, and coagulation. NSAIDs have been recently implicated in humans and mammals with an increased risk of myocardial infarction and delays in bone healing (Gerstenfeld *et al.*, 2003; Dajani and Islam, 2008), but these effects have not been substantiated in birds. The kidney uses both COX-1 and COX-2 for prostaglandin synthesis, and renal injury occurs when renal prostaglandin synthesis is inhibited. Therefore, in conditions of relative intravascular volume depletion and/or renal hypoperfusion such as dehydration, hemorrhage, hemodynamic compromise, hypotension associated with anesthesia, heart failure, and renal disease, interference with COX-2 activity can have significant deleterious effects.

FIGURE 7-38 Yellow-headed Amazon parrot (*Amazona oratrix*) with a tibiotarsal fracture surgically repaired with an external skeletal fixator-intramedullary pin tie-in. Management of postoperative pain with κ-opioid agonists μ-antagonists (e.g., butorphanol, nalbuphine) or tramadol and NSAIDS is recommended in this species.

Carprofen is considered a weak COX inhibitor at therapeutic doses, yet exhibits good antiinflammatory activity. This weak inhibition of both COX isoforms may explain its apparent wide margin of safety, and it may achieve its therapeutic effects partially through other pathways (Lees and Landoni, 2002). Carprofen is available in tablets and as an injectable formulation. It has been shown to improve lameness in chickens with spontaneous arthritis in a dose-dependent manner using relatively very high dosages parenterally, but the safety of these dosages has not been evaluated (McGeowen *et al.*, 1999; Hocking *et al.*, 2005; Swarup *et al.*, 2007; Caplen *et al.*, 2013). In Amazon parrots with experimental arthritis, carprofen did not improve significantly weight-bearing at 3 mg/kg IM (Paul-Murphy *et al.*, 2009b). In pigeons, there is some evidence suggesting hepatic toxicity of this drug and caution is recommended when administered in these species (Zollinger *et al.*, 2011). The PK profile of carprofen has not been evaluated in avian species, and little is known about bioavailability and half-life of this drug in birds. Further studies are needed to determine appropriate dosages, dosing routes, dosing frequency, and safety of carprofen in different species of birds.

Meloxicam is a COX-2-selective NSAID that has become the most widely used antiinflammatory medication in avian medicine. It is currently available as oral tablets, oral suspension, and injectable formulations. In studies using an experimental model of arthritis in Amazon parrots, meloxicam showed efficacy in improving weight-bearing at 1 mg/kg IM q 12 hours (Cole *et al.*, 2009), while in pigeons using an experimental fracture model 2 mg/kg PO q 12 hours was required to be effective (Desmarchelier *et al.*, 2012b). The PK profile has been evaluated in a large number of species that includes raptors, psittacines, Columbiformes, Anseriformes, Galliformes, and ratites (Baert and De Backer, 2003; Wilson *et al.*, 2004; Naidoo *et al.*, 2008; Montesinos *et al.*, 2011; Lacasse *et al.*, 2013; Molter *et al.*, 2013). In the species in which PK has been determined, the oral bioavailability ranges from approximately 60% to 70%. In addition, the adverse effects of meloxicam have been evaluated in species of psittacines (Pereira and Werther, 2007; Montesinos *et al.*, 2009; Dijkstra *et al.*, 2015), Galliformes (Sinclair *et al.*, 2012), and raptors (Summa, personal

FIGURE 7-39 Peregrine falcon (*F. peregrinus*) with a tibiotarsal fracture surgically repaired with an external fixator. Management of postoperative pain with μ-opioid agonists (e.g., hydromorphone, buprenorphine) or tramadol and NSAIDs is recommended in this species.

TABLE 7-5 NSAIDs Evaluated in Avian Species by Either Pharmacokinetic, Pharmacodynamics, or Safety Studies

Drug	Dosage mg/kg	Species	Comments	References
Carprofen	3 IM	Hispaniolan Amazon parrots	Arthritis pain did not improved significantly	Paul-Murphy *et al.*, 2009b
	2, 5, and 10 IM	Domestic pigeon	Elevation on AST, ALT; muscle and liver lesions following administration q 24 hours for 7 days	Zollinger *et al.*, 2011
	30 IM	Domestic chickens	Improved lameness of arthritic bird	Hocking *et al.*, 2005
	1 IM	Domestic chickens	Improved locomotion of lame birds	McGeowen *et al.*, 1999
Meloxicam	0.5 IV, PO	Red-tailed hawks and great-horned owls		Lacasse *et al.*, 2013
	2 IM	Japanese quail	Did not cause significant renal lesions but resulted in muscle necrosis following injections q 12 hours for 14 days	Sinclair *et al.*, 2012
	0.5, 2 PO	Domestic pigeon	Increased weight-bearing on fracture limb after 2 mg/kg q 12 hours	Desmarchelier *et al.*, 2012b
	0.5, 1 IM, IV	African grey parrots	Remained above target plasma concentrations for 24 hours after 1 mg/kg for 7 days; 0.5 mg/kg IM q 12 hours for 14 days did not cause renal lesion on biopsy	Montesinos *et al.*, 2009, 2011
	0.5, 1 IM, IV, PO	Hispaniolan Amazon parrots	Remained above target plasma concentrations for 12 hours after 1 mg/kg IM; increased weight-bearing on arthritic limb after 1 mg/kg IM	Cole *et al.*, 2009; Molter *et al.*, 2013
	2 PO	Gyps vultures	No abnormalities on hematology or chemistry after administration for 7 days	Swarup *et al.*, 2007
	0.1 IM	Budgerigars	Glomerular congestion following administration q 24 hours for 7 days	Pereira and Werther, 2007
	0.5 IM	Ring-necked parakeets		Wilson *et al.*, 2004
	0.5 IV	Chickens, ostriches, ducks, turkeys, pigeons	Significant differences in drug half-life between species	Baert and De Backer, 2003
Flunixin meglumine	5 IV	Budgerigars and Patagonian conures		Musser *et al.*, 2013
	5 IM	Mallard ducks	Duration of action of 12 hours based on thromboxane plasma concentrations	Machin *et al.*, 2001
	5.5 IM	Budgerigars	Significant renal lesions following administration q 24 hours for 7 days	Pereira and Werther, 2007
	1.1 IV	Domestic chickens, ostriches, ducks, turkeys, domestic pigeons	Significant differences in half-life between species	Baert and De Backer, 2003
	3 IM	Domestic chickens	Improved lameness on arthritic birds	Hocking *et al.*, 2005
Ketoprofen	2.0 IV, IM, PO	Japanese quail		Graham *et al.*, 2005
	12 IM	Domestic chickens	Improved lameness on arthritic birds	Hocking, *et al.*, 2005
	2.5 IM	Budgerigars	Significant renal lesions following administration q 24 hours for 7 days	Pereira and Werther, 2007
	5 IM	Mallard ducks	Duration of action of 12 hours based on thromboxane plasma concentrations	Machin and Livingston, 2002
	2-5 PO, IM, IV	Eiders	Resulted in high percentage of mortality in male eiders	Mulcahy *et al.*, 2003
Piroxicam	0.5 PO	Brolga cranes	Recommended q 24 hours	Keiper and Hartup, 2014

Note: PO, *per os* (by mouth).

communication, 2015). In these studies, there were minimal to no renal, gastrointestinal or hemostatic adverse effects reported at the dosages evaluated. Adverse effects have also been evaluated in Asian vultures (*Gyps bengalensis*, *G. indicus*, and *G. tenuirostris*), in which renal toxicity has been a reported with other NSAIDs (Oaks *et al.*, 2004; Naidoo *et al.*, 2010) but not with meloxicam (Swarup *et al.*, 2007). A survey to determine NSAID toxicity in captive birds treated in zoos reported no fatalities when meloxicam was administered to over 700 birds from 60 species (Cuthbert *et al.*, 2007). Despite these reports, the general recommendations regarding safety of NSAIDs in birds still need to be considered when using meloxicam. Because of the efficacy, PK, and safety data available, meloxicam is the NSAID of choice to treat perioperative and chronic pain.

Flunixin meglumine is a nonselective COX inhibitor. It has shown efficacy at reducing lameness in chickens with arthritis when administered at 3.0 mg/kg IM (Hocking *et al.*, 2005). In a study using inflammatory mediators as an indication of efficacy in ducks, ketoprofen at 5 mg/kg IM decreased thromboxane for ~12 hours (Machin *et al.*, 2001). The PK profile has been evaluated in psittacines, Anseriformes, Galliformes, Columbiformes, and ratites (Baert and De Backer, 2003). There are significant concerns regarding the renal toxicity of flunixin meglumine in birds, and even at relatively low dosages it appears to

result in some degree of renal damage as reported in studies in quails (Klein *et al.*, 1994) and budgerigars (Pereira and Werther, 2007). Based on these findings, the administration of flunixin meglumine should be done cautiously in avian species and administration of other NSAIDs might be preferable from a safety standpoint.

Ketoprofen is a potent, nonselective COX-1 inhibitor that was used extensively in avian medicine until the appearance in the market of other NSAIDs like carprofen and meloxicam. In a study using inflammatory mediators as an indication of efficacy in Anseriformes, ketoprofen at 5 mg/kg IM decreased thromboxane plasma concentrations for ~12 hours (Machin *et al.*, 2001). The PK profile of ketoprofen has been evaluated in Galliformes, with low oral (24%) and IM (54%) bioavailability and a short half-life in quail (Graham *et al.*, 2005). In a clinical study, ketoprofen at 2 to 5 mg/kg IM administered to free-ranging spectacled eiders (*Somateria fischeri*) and king eiders (*S. spectabilis*), resulted in renal tubular necrosis, acute rhabdomyolysis, and mild visceral gout causing death in 4/10 male spectacled eiders and 5/6 male king eiders 1 to 4 days after surgery (Mulcahy *et al.*, 2003). Because of these results, caution is advised when administering ketoprofen to Anseriformes and other avian species.

Piroxicam is a nonselective NSAID used for its anti-inflammatory properties and its value as a chemopreventative and antitumor agent. It has a much higher potency against COX-1 than COX-2. It has been used clinically for long-term treatment of chronic arthritis in cranes (Hanley *et al.*, 2005), and recent PK profile studies in cranes suggest a dosage of 0.5 mg/kg PO q 24 hours (Keiper and Hartup, 2014). Despite the high incidence of adverse effects of piroxicam in humans, there are no reports of its toxicity in birds. Piroxicam warrants further research before its use can be recommended in other avian species.

Diclofenac (DF) has been linked to the massive mortalities reported in multiple vulture species on the Asian subcontinent, leading to banning of DF on the Indian subcontinent. Common findings of diffuse visceral gout and proximal convoluted tubular damage indicated that the site of toxicity was the kidneys or the renal supportive vascular system (Oaks *et al.*, 2004; Meteyer *et al.*, 2005; Swan *et al.*, 2006; Naidoo and Swan, 2009). The effect of DF on inhibition of renal prostaglandins and subsequent renal portal valve closure was proposed to cause the severe renal ischemia and nephrotoxicity (Meteyer *et al.*, 2005), but additional studies determined that vulture susceptibility to DF results from a combination of increases in reactive oxygen molecules (such as oxygen ions and peroxide), interference with uric acid transport, and duration of exposure (Naidoo and Swan, 2009). Additionally, the terminal half-life of DF in vultures (14 hours) is much longer than chickens (2 hours), exposing vultures to toxic effects of DF for prolonged periods (Naidoo *et al.*, 2008).

LOCAL ANESTHETICS

Local anesthetics reversibly bind to Na^+ channels and block impulse conduction. Local anesthetics will be absorbed by the vasculature in the region being blocked. Systemic uptake of the local anesthetics can be rapid in birds, and metabolism may be prolonged, increasing the potential for toxic reactions (Table 7-6). Dosage recommendations have been lower for birds than mammals because anecdotal evidence has suggested that birds may be more sensitive to the adverse effects of these drugs. Toxic effects reported in birds include fine tremors, ataxia, recumbency, seizures, stupor, cardiovascular effects, and death. No adverse cardiovascular effects were identified in a study in chickens under isoflurane anesthesia when lidocaine was administered at 6 mg/kg IV (Brandao *et al.*, 2014).

Lidocaine is available as a commercial preparation of 2% (20 mg/mL) and has a relatively short duration of action. Based on empirical use, the recommended dosage is 2 to 4 mg/kg by one of the authors (D.S.M.G.). For small birds, the commercial preparation may need to be diluted 1:10 or more to achieve an effective volume for the block, but it is unknown whether dilution allows either appropriate tissue drug concentrations for analgesia or the expected duration of analgesia to occur. The PK profile of lidocaine following 2.5 mg/kg IV administration in anesthetized chickens showed a short half-life and appears to share similar mechanisms of metabolism and elimination to mammalian species reported (Da Cunha *et al.*, 2012).

Bupivacaine is available commercially as 0.25, 0.5, and 0.75% solutions (2.5, 5, and 7.5 mg/mL, respectively), and the lower concentration may not need dilution for birds. It has been used conservatively in birds because of concerns for toxic effects. Based on empirical use, the recommended maximum dosage of bupivacaine is 2 mg/kg by one of the authors (D.S.M.G.). It has shown efficacy in chickens following intraarticular administration (3 mg in 0.3 mL saline) to treat arthritic pain (Hocking *et al.*, 1997) and topically for pain associated with amputated beaks (1:1 mixture of bupivacaine and dimethyl sulfoxide) to improve feed intake (Glatz *et al.*, 1992). The PK profile has been determined in ducks at 2 mg/kg SC suggesting a shorter duration of action than in mammals but with potential for delayed toxicity (Machin *et al.*, 2001).

Local line or splash blocks are the most common methods of regional infiltration used in birds. The subcutaneous space in most

TABLE 7-6 Local Anesthetic Evaluated in Avian Species by Either Pharmacokinetic, Pharmacodynamic or Safety Evaluations

Drug	Dosage mg/kg	Species	Comments	References
Lidocaine	6 IV	Domestic chicken	No clinically relevant effects on HR or MAP	Brandao *et al.*, 2014
	2.5 IV	Domestic chicken		
	2 PN	Hispaniolan Amazon parrot	High failure rate in brachial plexus block	da Cunha *et al.*, 2013
	15 PN	Mallard ducks	Ineffective in providing regional analgesia in brachial plexus block	Brenner *et al.*, 2010
	20 PN	Domestic chickens	High failure rate in brachial plexus block	Figueiredo *et al.*, 2008
Bupivacaine	2, 8 PN	Mallard ducks	Ineffective in providing regional analgesia in brachial plexus block	Brenner *et al.*, 2010
	5 PN	Domestic chickens	High failure rate in brachial plexus block	Figueiredo *et al.*, 2008
	2 PN	Mallard ducks		Machin and Livingston, 2001
	3 PN	Domestic chickens	Effective in treating arthritic pain	Hocking *et al.*, 1997

Note: PN, perineurally; HR, heart rate; MAP, mean arterial pressure.

avian species is very thin so a small gauge needle is recommended to make several SC injections into the operative area. Brachial plexus blockade has been described in a variety of avian species but neither bupivacaine (2 and 8 mg/kg) or lidocaine (15 mg/kg) with epinephrine perineurally effectively blocked nerve transmission in the brachial plexus in ducks (Brenner *et al.*, 2010) or in chickens with lidocaine (20 mg/kg) or bupivacaine (5 mg/kg) with epinephrine for brachial plexus blockade using a nerve locator (Figueiredo *et al.*, 2008). In Amazon parrots, neither palpation- nor ultrasound-guided brachial plexus blockade was found to result in an effective block using lidocaine at 2 mg/kg perineurally (da Cunha *et al.*, 2013). Sciatic-femoral nerve block has also been described in falcons and was considered a feasible technique by applying lidocaine at 2 mg/kg perineurally with the aid of a nerve locator (d'Ovidio, *et al.*, 2014). The safe use of transdermal patches and creams, epidural infusions, spinal blocks, and IV blocks have not yet been reported in birds.

Other Drugs

Gabapentin, a GABA analog, has been used to treat neuropathic pain in humans. Its exact mechanism of action is unknown, but its therapeutic action on neuropathic pain is thought to involve voltage-gated N-type calcium ion channels. As in humans and other mammals gabapentin is generally considered to be synergistic with other drugs and may not obtain the effect expected as a sole agent (Doneley, 2007; Shaver *et al.*, 2009). To date, there are limited reports of use of gabapentin as part of a multimodal therapeutic plan for suspected neuropathic pain in birds. The PK profile of gabapentin has been studied in Amazon parrots (Baine *et al.*, 2013), and 10 mg/kg PO every 12 hours was suggested as a starting dosage. Increasing stepwise dosages might be necessary as it is generally necessary in other species.

Dietary Supplements

Omega-3 polyunsaturated fatty acids like eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) have anti-inflammatory effects that might reduce the pain associated with osteoarthritis. The total EPA and DHA dosages are primary factors to consider with the omega-3 to omega-6 fatty acid ratio a lesser factor. Other omega-3 fatty acids (e.g., plant-based omega-3 fatty acid, α-linoleic acid) do not have similar effects. There have been no studies in birds evaluating the effects and optimal dosage of EPA and DHA in osteoarthritic pain.

Glucosamine, *methylsulfonylmethane*, and *chondroitin sulfate* may have benefits in treating osteoarthritis through anti-inflammatory effects, but there are no studies in birds evaluating dosages and potential adverse effects.

Polysulfated glycosaminoglycans have also been used anecdotally in the management of degenerative joint disease in birds, but fatal coagulopathies in different avian species (one Coraciiformes, two raptors, and one psittacine) following IM administration have been reported (Anderson *et al.*, 2013).

Physical Rehabilitation

Treatment for pain in humans and companion mammal species may include physical modalities, manual therapy, and therapeutic exercise. The application of adjunctive therapy should be considered for acute and chronic pain in birds, although evidence-based information is not yet available for birds. Therapeutic exercises, such as static weight-bearing, can be generally used in the acute phase of an injury with gradual progression of difficulty as healing occurs. Physical modalities such as thermotherapy and low-level laser are used to diminish pain. Thermotherapy can have analgesic effects after the inflammatory effect has subsided. Low-level laser therapy has been shown to decrease indicators of neuropathic pain. Manual physical therapeutic techniques for joint mobilization can decrease pain but trigger point-pressure techniques might induce central sensitization (Mathews *et al.*, 2014).

Supportive Care

Cold compress during acute injury can reduce swelling and provide analgesia. Cold compress generally needs to be in place for 15 to 20 minutes to be effective. Warm compress is generally more comfortable after the acute phase has passed, but can aid tissue relaxation and be used as a precursor to massage or stretching. Warm compress generally needs to be in place for 10 to 15 minutes (Anderson *et al.*, 2013; Mathews *et al.*, 2014).

The environment may affect pain, and stress and anxiety can have a modulatory effect. The bird should be in an environment where it is emotionally and physically comfortable. Human presence or absence, decreasing light and noise, and separating other animals from the bird can reduce stress and anxiety during the pain period. When handling and moving an animal, avoid painful areas (surgical/trauma site, osteoarthritic joints, etc.), even when the animal is anesthetized or sedated, to avoid inflicting a painful stimulus that can begin a new pain cascade (Mathews *et al.*, 2014).

REFERENCES

Anderson K, Garner MM, Reed HH, et al: Hemorrhagic diathesis in avian species following intramuscular administration of polysulfated glycosaminoglycan, *J Zoo Wildl Med* 44(1):93–99, 2013.

Baert K, De Backerd P: Comparative pharmacokinetics of three non-steroidal anti-inflammatory drugs in five bird species, *Comp Biochem Physiol Toxicol Pharmacol* 134(1):25–33, 2003.

Baine K, Jones M, Cox S, Martín-Jiménez T: *Pharmacokinetics of gabapentin in Hispaniolan Amazon parrots (Amazona ventralis)*, Proceedings of the Association of Avian Veterinarians, 2013.

Bardo MT, Hughes RA: Brief communication. Shock-elicited flight response in chickens as an index of morphine analgesia, *Pharmacol Biochem Behav* 9(1):147–149, 1978.

Bergh MS, Budsberg SC: The coxib NSAIDs: potential clinical and pharmacologic importance in veterinary medicine, *J Vet Intern Med* 19(5):633–643, 2005.

Black PA, Cox SK, Macek M, et al: Pharmacokinetics of tramadol hydrochloride and its metabolite *O*-desmethyltramadol in peafowl (*Pavo cristatus*), *J Zoo Wildl Med* 41(4):671–676, 2010.

Bovill JG: Mechanisms of actions of opioids and non-steroidal anti-inflammatory drugs, *Eur J Anaesthesiol* 15:9–15, 1997.

Brandao J, da Cunha AF, Pypendop B, et al: Cardiovascular tolerance of intravenous lidocaine in broiler chickens (*Gallus gallus domesticus*) anesthetized with isoflurane, *Vet Anaesthes Analges* 2014. doi: 10.1111/vaa.12226.

Brenner DJ, Larsen RS, Dickinson PJ, et al: Development of an avian brachial plexus nerve block technique for perioperative analgesia in mallard ducks (*Anas platyrhynchos*), *J Avian Med Surg* 24(1):24–34, 2010.

Caplen G, Colborne GR, Hothersall B, et al: Lame broiler chickens respond to non-steroidal anti-inflammatory drugs with objective changes in gait function: a controlled clinical trial, *Vet J* 196(3):477–482, 2013.

Ceulemans SM, Sanchez-Migallon Guzman D, Olsen GH, et al: Evaluation of thermal antinociceptive effects after intramuscular administration of buprenorphine hydrochloride to American kestrels (*Falco sparverius*), *Am J Vet Res* 75(8):705–710, 2014.

Cole GA, Paul-Murphy J, Krugner-Higby L, et al: Analgesic effects of intramuscular administration of meloxicam in Hispaniolan parrots (*Amazona ventralis*) with experimentally induced arthritis, *Am J Vet Res* 70(12):1471–1476, 2009.

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(6):806–811, 1995.

Csillag A, Bourne RC, Stewart MG: Distribution of mu, delta, and kappa opioid receptor binding sites in the brain of the one-day-old domestic

chick (*Gallus domesticus*): an in vitro quantitative autoradiographic study, *J Comp Neurol* 302(3):543–551, 1990.
Curro T, Brunson D, Paul-Murphy J: Determination of the ED50 of isoflurane and evaluation of the analgesic properties of butorphanol in cockatoos (*Cacatua* spp.), *Vet Surg* 23:429–433, 1994.
Curro TG: *Evaluation of the isoflurane-sparing effects of butorphanol and flunixin in psittaciformes*, Proceedings of the Association of Avian Veterinarians, 1994, pp 17–19.
Cuthbert R, Parry-Jones J, Green RE, et al: NSAIDs and scavenging birds: potential impacts beyond Asia's critically endangered vultures, *Biol Lett* 3(1):90–93, 2007.
da Cunha AF, Messenger KM, Stout RW, et al: Pharmacokinetics of lidocaine and its active metabolite monoethylglycinexylidide after a single intravenous administration in chickens (*Gallus domesticus*) anesthetized with isoflurane, *J Vet Pharmacol Ther* 35(6):604–607, 2012.
da Cunha AF, Strain GM, Rademacher N, et al: Palpation- and ultrasound-guided brachial plexus blockade in Hispaniolan Amazon parrots (*Amazona ventralis*), *Vet Anaesthes Analges* 40(1):1–7, 2013.
Dajani EZ, Islam K: Cardiovascular and gastrointestinal toxicity of selective cyclo-oxygenase-2 inhibitors in man, *J Physiol Pharmacol* 59(2):17–133, 2008.
Desmarchelier M, Troncy E, Beauchamp G, et al: Evaluation of a fracture pain model in domestic pigeons (*Columba livia*), *Am J Vet Res* 73(3):353–360, 2012a.
Desmarchelier M, Troncy E, Fitzgerald G, et al: Analgesic effects of meloxicam administration on postoperative orthopedic pain in domestic pigeons (*Columba livia*), *Am J Vet Res* 73(3):361–367, 2012b.
Dijkstra B, Sanchez-Migallon Guzman D, Gustavsen K, et al: Renal, gastrointestinal, and hemostatic effects of meloxicam in Hispaniolan Amazon parrots (*Amazona ventralis*), *Am J Vet Res* 76(4):308–317, 2015.
Doneley B: *The use of gabapentin to treat presumed neuralgia in a little corella (Cacatua sanguinea)*, Proceedings of the Australian Association of Avian Veterinarians, 2007.
d'Ovidio D, Noviello E, Adami C: Nerve stimulator-guided sciatic-femoral nerve block in raptors undergoing surgical treatment of pododermatitis, *Vet Anaesthes Analges* 2014. doi: 10.1111/vaa.12204.
Escobar A, Valadao CA, Brosnan RJ, et al: Effects of butorphanol on the minimum anesthetic concentration for sevoflurane in guineafowl (*Numida meleagris*), *Am J Vet Res* 73(2):83–188, 2012.
Escobar A, Valadão CA, Brosnan RJ, et al: Cardiopulmonary effects of butorphanol in sevoflurane-anesthetized guineafowl (*Numida meleagris*), *Vet Anaesthes Analges* 41(3):284–289, 2014.
Fan S, Shutt AJ, Vogt M: The importance of 5-hydroxytryptamine turnover for the analgesic effect of morphine in the chicken, *Neuroscience* 6(11):2223–2227, 1981.
Figueiredo JP, Cruz ML, Mendes GM, et al: Assessment of brachial plexus blockade in chickens by an axillary approach, *Vet Anaesthes Analges* 35(6):511–518, 2008.
Gaggermeier B, Henke J, Schatzmann U: *Investigations on analgesia in domestic pigeons (C. livia, Gmel., 1789, var. dom.) using buprenorphine and butorphanol*, Proceedings of the European Association of Avian Veterinarians, 2003.
Gentle MJ, Hocking PM, Bernard R, et al: Evaluation of intraarticular opioid analgesia for the relief of articular pain in the domestic fowl, *Pharmacol Biochem Behav* 63(2):339–343, 1999.
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.
Gerstenfeld LC, Thiede M, Seibert K, et al: Differential inhibition of fracture healing by non-selective and cyclooxygenase-2 selective non-steroidal anti-inflammatory drugs, *J Orthopaed Res* 21(4):670–675, 2003.
Glatz PC, Murphy LB, Preston AP: Analgesic therapy of beak-trimmed chickens, *Aust Vet J* 69(1):18, 1992.
Graham JE, Kollias-Baker C, Craigmill AL, et al: Pharmacokinetics of ketoprofen in Japanese quail (*Coturnix japonica*), *J Vet Pharmacol Ther* 28(4):399–402, 2005.
Gustavsen KA, Sanchez-Migallon Guzman D, Knych HK, et al: Pharmacokinetics of buprenorphine hydrochloride following intramuscular and intravenous administration to American kestrels (*Falco sparverius*), *Am J Vet Res* 75(8):711–715, 2014.
Hanley CS, Thomas NJ, Paul-Murphy J, et al: Exertional myopathy in whooping cranes (*Grus americana*) with prognostic guidelines, *J Zoo Wildl Med* 36(3):489–497, 2005.
Hawkins MG, Pascoe PJ, DiMaio Knych HK, et al: *Effect of fentanyl citrate target-controlled-infusions on isoflurane MAC and cardiovascular function in Hispaniolan Amazon parrots (Amazona ventralis)*, Proceedings of the Association of Avian Veterinarians, 2014.
Hocking PM, Gentle MJ, Bernard R, et al: Evaluation of a protocol for determining the effectiveness of pretreatment with local analgesics for reducing experimentally induced articular pain in domestic fowl, *Res Vet Sci* 63(3):263–267, 1997.
Hocking PM, Robertson GW, Gentle MJ: Effects of non-steroidal anti-inflammatory drugs on pain-related behaviour in a model of articular pain in the domestic fowl, *RTes Vet Sci* 78(1):69–75, 2005.
Hoppes S, Flammer K, Hoersch K, et al: Disposition and analgesic effects of fentanyl in white cockatoos (*Cacatua alba*), *J Avian Med Surg* 7(3):124–130, 2003.
Hughes RA: Strain-dependent morphine-induced analgesic and hyperalgesic effects on thermal nociception in domestic fowl (*Gallus gallus*), *Behav Neurosci* 104(4):619–624, 1990.
Keiper NL, Hartup BK: *Pharmacokinetics of piroxicam in brolgas*, Proceedings of the Association of Avian Veterinarians, 2014.
Keller DL, Sanchez-Migallon Guzman D, Klauer JM, et al: Pharmacokinetics of nalbuphine hydrochloride after intravenous and intramuscular administration to Hispaniolan Amazon parrots (*Amazona ventralis*), *Am J Vet Res* 72(6):741–745, 2011.
Kilburn JJ, Cox SK, Kottyan J, et al: Pharmacokinetics of tramadol and its primary metabolite O-desmethyltramadol in African penguins (*Spheniscus demersus*), *J Zoo Wildl Med* 45(1):93–99, 2014.
Klaphake E, Schumacher J, Greenacre C, et al: *J Avian Med Surg* 20(1):2–7, 2006.
Klein PN, Charmatz K, Langenberg J: *The effect of flunixin meglumine (banamine) on the renal function of northern bobwhite quail (Colinus virginianus): an avian model*, Proceedings of the American Association of Zoo Veterinarians, 1994, pp 128–131.
Lacasse C, Gamble KC, Boothe DM: Pharmacokinetics of a single dose of intravenous and oral meloxicam in red-tailed hawks (*Buteo jamaicensis*) and great horned owls (*Bubo virginianus*), *J Avian Med Surg* 27(3):204–210, 2013.
Lees P, Landoni MF: Pharmacodynamics and enantioselective pharmacokinetics of racemic carprofen in the horse, *J Vet Pharmacol Ther* 25(6):433–448, 2002.
Machin KL, Livingston A: *Plasma bupivicaine levels in mallard ducks (Anas platyrhynchos) following a single subcutaneous dose*, Procedings of the American Association of Zoo Veterinarians, 2001.
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.
Machin KL, Tellier LA, Lair S, et al: Pharmacodynamics of flunixin and ketoprofen in mallard ducks (Anas platyrhynchos), *J Zoo Wildl Med* 32(2):222–229, 2001.
Mansour A, Khachaturian H, Lewis ME, et al: Anatomy of CNS opioid receptors, *Trends Neurosci* 11(7):308–314, 1988.
Mathews K, Kronen PW, Lascelles D, et al: Guidelines for recognition, assessment and treatment of pain: WSAVA Global Pain Council members and co-authors of this document, *J Small Anim Pract* 55(6):E10–E68, 2014.
Mathonnet M, Lalloue F, Danty E, et al: Cyclo-oxygenase 2 tissue distribution and developmental pattern of expression in the chicken, *Clin Exp Pharmacol Physiol* 28(5–6):425–432, 2001.
Mazor-Thomas JE, Mann PE, Karas AZ, et al: Pain-suppressed behaviors in the red-tailed hawk, *Appl Anim Behav Sci* 152:83–91, 2014.
McGeowen D, Danbury TC, Waterman-Pearson AE, et al: Effect of carprofen on lameness in broiler chickens, *Vet Rec* 144(24):668–671, 1999.
Meteyer CU, Rideout BA, Gilbert M, et al: Pathology and proposed pathophysiology of diclofenac poisoning in free-living and experimentally exposed oriental white-backed vultures (*Gyps bengalensis*), *J Wildl Dis* 41(4):707–716, 2005.

Molter CM, Court MH, Cole GA, et al: Pharmacokinetics of meloxicam after intravenous, intramuscular, and oral administration of a single dose to Hispaniolan Amazon parrots (*Amazona ventralis*), *Am J Vet Res* 74(3):375–380, 2013.

Montesinos A, Ardiaca M, Juan-Selles C, et al: *Evaluation of the renal effects of meloxicam in African Grey parrots (Psittacus erithacus erithacus)*, Proceedings of the European Association of Avian Veterinarians, Antwerp, Belgium, 2009.

Montesinos A, Ardiaca M, Diez-Delgado I, et al: *Pharmacokinetics of parenteral and oral meloxicam in African grey parrots (Psittacus erithacus erithacus)*, Proceedings of the European Association of Avian Veterinarians, 2011.

Mulcahy DM, Tuomi P, Larsen RS: Differential mortality of male spectacled eiders (*Somateria fischeri*) and king eiders (*Somateria spectabilis*) subsequent to anesthesia with propofol, bupivacaine, and ketoprofen, *J Avian Med Surg* 17(3):117–123, 2003.

Musser JM, Heatley JJ, Phalen DN: Pharmacokinetics after intravenous administration of flunixin meglumine in budgerigars (*Melopsittacus undulatus*) and Patagonian conures (*Cyanoliseus patagonus*), *J Am Vet Med Assoc* 242(2):205–208, 2013.

Naidoo V, Swan GE: Diclofenac toxicity in *Gyps* vulture is associated with decreased uric acid excretion and not renal portal vasoconstriction, *CompBiochem Physiol C Toxicol Pharmacol* 149(3):269–274, 2009.

Naidoo V, Wolter K, Cromarty AD, et al: The pharmacokinetics of meloxicam in vultures, *J Vet Pharmacol Ther* 31(2):128–134, 2008.

Naidoo V, Wolter K, Cromarty D, et al: Toxicity of non-steroidal anti-inflammatory drugs to *Gyps* vultures: a new threat from ketoprofen, *Biol Lett* 6(3):339–341, 2010.

Oaks JL, Gilbert M, Virani MZ, et al: Diclofenac residues as the cause of vulture population decline in Pakistan, *Nature* 427(6975):630–633, 2004.

Papich MG: An update on non-steroidal anti-inflammatory drugs (NSAIDs) in small animals, *Vet Clin North Am Small Anim Pract* 38(6):1243–1266, 2008.

Paul-Murphy J, 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.

Paul-Murphy J, Hess J, Fialkowski JP: Pharmokinetic properties of a single intramuscular dose of buprenorphine in African grey parrots (*Psittacus erithacus erithacus*), *J Avian Med Surg* 18(4):224–228, 2004.

Paul-Murphy JR, Krugner-Higby LA, Tourdot RL, et al: Evaluation of liposome-encapsulated butorphanol tartrate for alleviation of experimentally induced arthritic pain in green-cheeked conures (*Pyrrhura molinae*), *Am J Vet Res* 70(10):1211–1219, 2009a.

Paul-Murphy JR, Sladky KK, Krugner-Higby LA, et al: Analgesic effects of carprofen and liposome-encapsulated butorphanol tartrate in Hispaniolan parrots (*Amazona ventralis*) with experimentally induced arthritis, *Am J Vet Res* 70(10):1201–1210, 2009b.

Pavez JC, Hawkins MG, Pascoe PJ, et al: Effect of fentanyl target-controlled infusions on isoflurane minimum anaesthetic concentration and cardiovascular function in red-tailed hawks (*Buteo jamaicensis*), *Vet Anaesthes Analges* 38(4):344–351, 2011.

Pereira M, Werther K: Evaluation of the renal effects of flunixin meglumine, ketoprofen and meloxicam in budgerigars *(Melopsittacus undulatus*), *Vet Rec* 160(24):844–846, 2007.

Riggs SM, Hawkins MG, Craigmill AL, et al: Pharmacokinetics of butorphanol tartrate in red-tailed hawks (*Buteo jamaicensis*) and great horned owls (*Bubo virginianus*), *Am J Vet Res* 69(5):596–603, 2008.

Sanchez-Migallon Guzman D, Flammer K, Paul-Murphy JR, et al: Pharmacokinetics of butorphanol after intravenous, intramuscular, and oral administration in Hispaniolan Amazon parrots (*Amazona ventralis*), *J Avian Med Surg* 25(3):185–191, 2011a.

Sanchez-Migallon Guzman D, KuKanich B, Keuler NS, et al: Antinociceptive effects of nalbuphine hydrochloride in Hispaniolan Amazon parrots (*Amazona ventralis*), *Am J Vet Res* 72(6):736–740, 2011b.

Sanchez-Migallon Guzman D, Souza MJ, Braun JM, et al: Antinociceptive effects after oral administration of tramadol hydrochloride in Hispaniolan Amazon parrots (*Amazona ventralis*), *Am J Vet Res* 73(8):1148–1152, 2012.

Sanchez-Migallon Guzman D, Braun JM, Steagall PV, et al: Antinociceptive effects of long-acting nalbuphine decanoate after intramuscular administration to Hispaniolan Amazon parrots (*Amazona ventralis*), *Am J Vet Res* 74(2):196–200, 2013a.

Sanchez-Migallon Guzman D, KuKanich B, Heath TD, et al: Pharmacokinetics of long-acting nalbuphine decanoate after intramuscular administration to Hispaniolan Amazon parrots (*Amazona ventralis*), *Am J Vet Res* 74(2):191–195, 2013b.

Sanchez-Migallon Guzman D, Drazenovich TL, Olsen GH, et al: Evaluation of thermal antinociceptive effects after intramuscular administration of hydromorphone hydrochloride to American kestrels (*Falco sparverius*), *Am J Vet Res* 74(6):817–822, 2013c.

Sanchez-Migallon Guzman D, Drazenovich TL, KuKanich B, et al: Evaluation of thermal antinociceptive effects and pharmacokinetics after intramuscular administration of butorphanol tartrate to American kestrels (*Falco sparverius*), *Am J Vet Res* 75(1):11–18, 2014a.

Sanchez-Migallon Guzman D, Drazenovich TL, Olsen GH, et al: Evaluation of thermal antinociceptive effects after oral administration of tramadol hydrochloride to American kestrels (*Falco sparverius*), *Am J Vet Res* 75(2):117–123, 2014b.

Sanchez-Migallon Guzman D, KuKanich B, Drazenovich TL, et al: Pharmacokinetics of hydromorphone hydrochloride after intravenous and intramuscular administration of a single dose to American kestrels (*Falco sparverius*), *Am J Vet Res* 75(6):527–531, 2014c.

Sanchez Migallon-Guzman D, Houck E, Beaufrere H, et al: *Evaluation of the thermal antinociceptive effects of buprenorphine hydrochloride in cockatiels (Nymphicus hollandicus)*, Proceedings of the Association of American Association of Zoo Veterinarians, 2014d.

Sanchez Migallon-Guzman D, Houck E, Beaufrere H, et al: *Evaluation of the thermal antinociceptive effects of hydromorphone hydrochloride in cockatiels (Nymphicus hollandicus)*, Proceedings of the Association of Avian Veterinarians, 2014e.

Sanchez-Migallon Guzman D, Knych H, Olsen GH, et al: *Evaluation of the thermal antinociceptive effects and pharmacokinetics of a sustained-release buprenorphine formulation in American kestrels (Falco sparverius)*, Proceedings of the Association of Avian Veterinarians, 2015, pp 13–14.

Schneider C: Effects of morphine-like drugs in chicks, *Nature* 191:607–608, 1961.

Scott LJ, Perry CM: Tramadol: a review of its use in perioperative pain, *Drugs* 60(1):139–176, 2000.

Shaver SL, Robinson NG, Wright BD, et al: A multimodal approach to management of suspected neuropathic pain in a prairie falcon (*Falco mexicanus*), *J Avian Med Surg* 23(3):209–213, 2009.

Sinclair KM, Church ME, Farver TB, et al: Effects of meloxicam on hematologic and plasma biochemical analysis variables and results of histologic examination of tissue specimens of Japanese quail (*Coturnix japonica*), *Am J Vet Res* 73(11):1720–1727, 2012.

Singh PM, Johnson C, Gartrell B, et al: Pharmacokinetics of morphine after intravenous administration in broiler chickens, *J Vet Pharmacol Ther* 33(5):515–518, 2010.

Singh PM, Johnson C, Gartrell B, et al: Pharmacokinetics of butorphanol in broiler chickens, *Vet Rec* 168:588, 2011.

Sladky K, Krugner-Higby L, Meek-Walker E, et al: Serum concentrations and analgesic effects of liposome-encapsulated and standard butorphanol tartrate in parrots, *Am J Vet Res* 67(5):775–781, 2006.

Souza MJ, Gerhardt L, Cox SK: Pharmacokinetics of repeated oral administration of tramadol hydrochloride in Hispaniolan Amazon parrots (*Amazona ventralis*), *Am J Vet Res* 74(7):957–962, 2013.

Souza MJ, Sanchez-Migallon Guzman D, Paul-Murphy JR, et al: Pharmacokinetics after oral and intravenous administration of a single dose of tramadol hydrochloride to Hispaniolan Amazon parrots (*Amazona ventralis*), *Am J Vet Res* 73(8):1142–1147, 2012.

Souza MJ, Martin-Jimenez T, Jones MP, et al: Pharmacokinetics of intravenous and oral tramadol in the bald eagle (*Haliaeetus leucocephalus*), *J Avian Med Surg* 23(4):247–252, 2009.

Souza MJ, Martin-Jimenez T, Jones MP, et al: Pharmacokinetics of oral tramadol in red-tailed hawks (*Buteo jamaicensis*), *J Vet Pharmacol Ther* 34(1):86–88, 2011.

Summa N, Fitzgerald G, Larrat S, et al: *Evaluation of toxicity of meloxicam in American Kestrels (Falco sparverius)*, Proceedings of the International Conference Avian Herpetological and Exotic mammal Medicine and Surgery, 2015, p 335.

Swan GE, Cuthbert R, Quevedo M, et al: Toxicity of diclofenac to *Gyps* vultures, *Biol Lett* 2(2):279–282, 2006.

Swarup D, Patra RC, Prakash V, et al: Safety of meloxicam to critically endangered *Gyps* vultures and other scavenging birds in India, *Anim Conserv* 10:192–198, 2007.

Wilson GH, Hernandez-Divers S, Budsberg SC, et al: *Pharmacokinetics and use of meloxican in psittacine birds*, Proceedings of the Association of Avian Veterinarians, 2004.

Zollinger TJ, Hoover JP, Payton ME, et al: Clinicopathologic, gross necropsy, and histologic findings after intramuscular injection of carprofen in a pigeon (*Columba livia*) model, *J Avian Med Surg* 25(3):173–184, 2011.

HYPOTHERMIA

David Sanchez-Migallon Guzman

Hypothermia is a critical complicating factor of avian anesthesia. Hypothermia develops because of heat loss through convection, conduction, radiation, and evaporation and occurs more rapidly in avian species because of the larger body surface relatively to the body mass. Along with the loss of physiologic responses under anesthesia to reduce core temperature, hypothermia is also induced during surgical preparation by removal of large areas of feathers and the use of alcohol or other fluids to prepare the area for surgery or the exposure of cavities and organs. Hypothermia has a number of adverse physiologic effects that increases with its severity including hypoxia, metabolic acidosis, cardiac arrhythmias, and reversible platelet dysfunction. It also results in decreased minute ventilation and tidal volume, decreases anesthetic requirement, and metabolism that can result in prolonged recovery.

The core temperatures of pigeons with no external heat support were found to drop 2.5 to 8°C during as little as 30 minutes of gas anesthesia (Phalen *et al.*, 1996). The treatment of hypothermia should focus on preventing further loss of body temperature and providing thermal support until the body reaches normal temperature. Radiant heat sources, water blankets, and forced air warmer systems (e.g., Bair Hugger Warming System) are routinely used to maintain core body temperature and should be used in the case of hypothermia to increase the body temperature. The most effective method for maintaining the temperature has been found to be the forced-air warming device while covering the bird with a transparent drape, when compared with water blanket or radiant heat source with a drape (Rembert *et al.*, 2001). The administration of IV fluids warmed at body temperature during surgical procedures using a fluid warmer (e.g., Vet Temp Fluid Warmer) might prevent further loss of body temperature and aid with recovery of normothermia. The type of anesthetic delivery system, with or without heated air, does not appear to affect the core body temperature (Boedeker *et al.*, 2005). The use of uncovered electrical heating pads is discouraged because of the potential for thermal injury. "Hot-water gloves," while a common practice for avian veterinarians, should be used carefully for the same reason. The use of warm incubators at ~25°C (77°F) is recommended until the animal is fully recovered.

ANESTHETIC EMERGENCIES

David Sanchez-Migallon Guzman

Emergencies during anesthesia should be expected and planned for with careful monitoring and support during anesthesia of the nervous system, cardiovascular system, respiratory function, and body temperature. It is recommended that emergency drug dosages are calculated and one to two doses be predrawn before induction and that the emergency equipment (e.g., endotracheal tubes, oxygen, IV catheters and materials for securing them, ventilatory support, and emergency drugs) is prepared and accessible for use.

BRADYPNEA AND HYPOVENTILATION

During anesthetic procedures, some degree of respiratory depression should be expected, which will increase over time. Because of that, tracheal intubation with IPPV is recommended at 2 breaths per minute in spontaneously ventilating birds, and 10 to 20 breaths per minute in birds with significant bradypnea. If used in combination with a capnograph, IPPV rate should be adjusted to maintain $ETCO_2$ between 30 and 45 mm Hg. The anesthetist should also assess the frequency and range of sternal motion and reservoir bag movement. A change in respiratory pattern involving increased respiratory effort may indicate obstruction of the endotracheal tube, a particular concern in very small patients. If an obstruction is suspected, repositioning of the bird with the neck extended and/or suction through the endotracheal tube is recommended to resolve any possible blockage. Normal ventilation is associated with little to no respiratory tract noise.

RESPIRATORY ARREST

If respiratory arrest occurs, tracheal intubation and intermittent positive-pressure ventilation (IPPV) is required at 10 to 20 breaths/min in birds. The level of gas anesthesia may be reduced, if indicated, but not necessarily discontinued. If used in combination with a capnograph, IPPV rate should be adjusted to maintain $ETCO_2$ between 30 to 45 mm Hg. The ventilatory support should be continued throughout the anesthetic procedure or until spontaneous ventilation resumes. If corrected immediately, the prognosis for respiratory arrest is good. The use of doxapram as a treatment for apnea is controversial and is not recommended (Gunkel and Lafortunem, 2005).

BRADYCARDIA AND HYPOTENSION

Bradycardia and hypotension (defined as systolic blood pressure [BP] <90 mm Hg) develop secondary to several conditions during anesthesia, including cardiovascular depression caused by the anesthetic agent, hypothermia, vagal response due to severe pain, and hypovolemia related to dehydration or blood loss. The anesthetist must assess whether it represents a true emergency (e.g., continues blood loss during a procedure) or is a transient response (e.g., vagal response due to pain during fracture manipulation). The level of inhalant anesthesia should be reduced if bradycardia and hypotension are thought to be caused by a high anesthetic plane. The administration of IV fluids is indicated if the bradycardia and hypotension are attributed to hypovolemia or for immediate correction of hypotensive state. The anticholinergic agents such as atropine (fast acting, short lasting) or glycopyrrolate (slow acting, long lasting) can be used to increase the heart but might result in sinus tachycardia and increase myocardial work and oxygen consumption. An IV or intraosseous (IO) administration of warmed crystalloid fluids at10 mL/kg/h or 10 to 15 mL as a bolus over 5 to 10 minutes is indicated for hypovolemic birds and can be repeated if necessary to increase systolic BP to more than 90 mm Hg. Synthetic colloid solutions (i.e., hetastarch [Abbott Laboratories, North Chicago, IL], 5 mL/kg IV over 5 to 10 minutes) may be used in hypoproteinemic birds or birds that have suffered acute blood loss during surgery. Blood transfusions may also be indicated for acute blood loss at a rate of 5 mL/kg/h.

Dobutamine administered at 5 to 15 μg/kg/min and dopamine at 5 to 10 μg/kg/min have shown to help correct severe hypotension in Hispaniolan Amazon parrots caused by anesthesia maintained with 2.5% isoflurane (Schnellbacher *et al.*, 2012).

CARDIAC ARREST

If cardiac arrest develops, continue ventilatory support as explained above in the respiratory arrest section. The solid keel interferes with effective cardiac massage, although in the event of cardiac arrest, attempts should be made to compress the sternum at a rate of 60 to 100 compressions per minute. Emergency drugs, including epinephrine (1:1000; 0.1 mg/kg IV, IO, or a double dosage intratracheal IT) and atropine (0.02 mg/kg IV, IO, or a double dosage IT), should be administered for two to three rounds. The use of vasopressin at a dosage of 0.8 U/kg IV, IO, or a double dosage IT in addition or instead of epinephrine has been suggested (Lichtenberger, 2007). The anesthetist should assess the efforts during cardiopulmonary resuscitation to see if there is spontaneous pulse. These efforts should be continued for up to 5 minutes. The prognosis for recovery is poor for avian patients that go into cardiac arrest, and although rare, some birds might recover.

REFERENCES

Boedeker NC, Carpenter JW, Mason DE: Comparison of body temperatures of pigeons (*Columba livia*) anesthetized by three different anesthetic delivery systems, *J Avian Med Surg* 19(1):1–6, 2005.

Gunkel C, Lafortunem M: Current techniques in avian anesthesia, *Sem Avian Exot Pet Med* 14(5):263–276, 2005.

Lichtenberger M: Shock and cardiopulmonary-cerebral resuscitation in small mammals and birds, *Vet Clin North Am Exot Anim Pract* 10(2):275–291, 2007.

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* 10(3):174–178, 1996.

Rembert MS, Smith JA, Hosgood G, et al: Comparison of traditional thermal support devices with the forced-air warmer system in anesthetized Hispaniolan Amazon parrots (*Amazona ventralis*), *J Avian Med Surg* 15(3):187–193, 2001.

Schnellbacher RW, da Cunha AF, Beaufrere H, et al: Effects of dopamine and dobutamine on isoflurane-induced hypotension in Hispaniolan Amazon parrots (*Amazona ventralis*), *Am J Vet Res* 73(7):952–958, 2012.

8

Medical, Nursing, and Cosmetic Procedures

MEDICAMENT ADMINISTRATION

Hugues Beaufrère

Medication may be administered to birds by any of the following major routes: parenteral, oral, nebulization, and topical. Each has its own advantages and disadvantages that should be considered before a particular route is chosen (Table 8-1).

PARENTERAL ADMINISTRATION

The parenteral route is the method of choice in treating seriously ill birds that require immediate, direct, and aggressive therapy (Figs. 8-1 to 8-6). An exact dose can be administered with minimal stress and blood levels can be rapidly achieved. In addition, parenteral routes should be chosen for drugs with poor oral bioavailability (e.g., amikacin, butorphanol, amphotericin B) and for patients with gastrointestinal diseases that may preclude enteral drug absorption. The main routes of administration used in birds are intramuscular (IM), intravenous (IV), subcutaneous (SC), intraperitoneal, intrasinusal, intraosseous (IO), intratracheal, and intranasal. Doses of drugs administered should be based on species-specific pharmacokinetic data. When not available, some degree of extrapolation is needed, which is easily done for drugs eliminated by glomerular filtration and with no hepatic biotransformation. For other types of drugs (most of them), clinicians should acknowledge that interspecies differences are unpredictable and do not necessarily follow allometric scaling (Hunter and Isaza, 2008).*

A variety of agents can be administered to birds by injection. IM administration of most drugs induces muscle trauma, which in turn may elevate nonspecific enzymes on the biochemistry panel (e.g., AST, CK) for several days and also result in pain. Local irritation is dependent on the type of drug (especially the pH), the volume administered, and the frequency of administration. It is generally recommended to alternate IM sites whenever possible.

Newer methods of drug administration derived from research use may also soon find clinical applications. For instance, osmotic pumps (Alzet) are small sterile containers that can be loaded with a set volume of drugs. Upon implantation under the skin or in a body cavity, it will slowly and continuously release drugs over a predetermined period of time. Although this has been widely used in a variety of research applications, it may also prove extremely useful in avian medicine in certain situations.

There are also certain general considerations that must be kept in mind, regardless of the route used or the medication applied. These include the following:

- *The choice of needle (size and length) is important*. A needle causes tissue damage and, although this may appear to be slight, it can be very significant, especially in small birds. Generally, a needle with a narrow gauge with as short a length as is consistent with efficient administration of the drug is desirable. However, it should be noted that thin needle may prove unsatisfactory for thick viscous compounds and a needle that is too short may make it impossible to place a drug depot (and this may be irritant) deep into the musculature. For most small- to medium-sized birds, needle sizes of 27 to 25 gauge appear satisfactory for IM injections.
- *The site chosen must take into consideration the particular circumstances*. For example, irritant injections will cause muscle lesions. Therefore, it must be assumed that there may be some impairment of flight if the pectoral muscles are used or impairment in walking/running if leg muscles are used. A useful guide is to inject such compounds into the muscle mass, which is less likely to cause inconvenience or adverse effect to the bird. Thus, terrestrial birds, such as quail, which prefer to walk rather than fly, should generally have injections into their pectoral muscles, whereas a falcon, which depends greatly on its powers of flight, may be injected more safely in the leg muscles. However, the presence of large pectoral muscles makes the injection into these muscles both convenient and well tolerated by most birds.
- *The volume of the injected medication must be considered*. As a general guideline, the maximum volumes that can be given by IM injection per site are as follows: macaw and cockatoo, 1 mL; Amazon or African grey parrot, 0.5 mL; cockatiel and small conure, 0.2 mL; budgerigar, canary, and finch, 0.1 mL (Rupley, 1997). A volume up to 1.5 mL per site can be administered in birds weighing more than 1500 g. Use several injection sites if larger volumes are to be administered. Multiple injections in the same side of the breast or the use of irritating drugs may result in muscle necrosis or atrophy.
- *SC injections* are preferable when large volumes are injected. However, part of the medication may leak out and irritating drugs may cause skin necrosis and ulceration. These drugs may be diluted in 0.9% saline before injection.
- *IV injections* are given mainly during critical care treatment. Rapid therapeutic levels are achieved when this route is used. IV or IO catheterization allows repeated injections of multiple drugs and continuous rate infusions (CRIs) without having to manually restrain the birds. Potential side effects include histamine release upon injection and anaphylactic reactions. Drugs with short half-lives should be given as CRIs (e.g., dopamine, fentanyl). Single-dose drug IV administration is also possible when an IV catheter is not used. In this situation, it is recommended as a one-time occurrence to limit the formation of hematomas with repeated venipuncture.
- *IT injection* is used to deliver drugs to the lungs and airways of birds (Jenkins, 1997). It is an effective route for administering amphotericin B to birds with aspergillosis. Volumes up to 2 mL/kg of water-soluble medication may be administered safely using a

*http://www.ncbi.nlm.nih.gov/pubmed/19110691

TABLE 8-1 Different Routes of Injection and Recommended Sites

Route of Administration	Site(s) of Injection
Intramuscular	Pectoral muscle Thigh muscles
Intravenous	Basilic vein Right jugular vein Medial metatarsal vein
Subcutaneous	Inguinal web Interscapular area Axillary region
Intraperitoneal	Peritoneal cavity
Intranasal	Nares
Intrasinusal	Infraorbital sinuses
Intraosseous	Proximal tibiotarsus Distal ulna
Intratracheal	Oropharynx Tracheal cartilage rings

FIGURE 8-1 Venipuncture of the basilic vein of a salmon-crested cockatoo (*Cacatua moluccensis*) under isoflurane anesthesia. The same site is used for administering intravenous medications.

small-diameter metal feeding needle. The medication is injected into the trachea with some force, and then the bird is released and allowed to recover. Alternatively, the drug may be administered under anesthesia using the channels or the injection needle of a rigid endoscope.

- *Infraorbital sinus injection* is useful for flushing and administering medication to birds with sinusitis. It can also be used to dislodge exudates and foreign bodies from the sinuses or to obtain samples for cytology, culture, and sensitivity testing. Sterile water and an antibiotic or antifungal solution may be used for treatment of sinusitis. Do not use irritating solutions for sinus flushing (Rupley, 1997) because severe sinusitis may ensue.
- *A nasal flush* may be used for both therapeutic and diagnostic purposes in infraorbital sinus infections. Antibiotics and antifungals recommended for nebulization can be used in a lower dose. The amounts of fluids for nasal flushing are 1 to 3 mL for a budgerigar and up to 10 to 15 mL for a large macaw or cockatoo

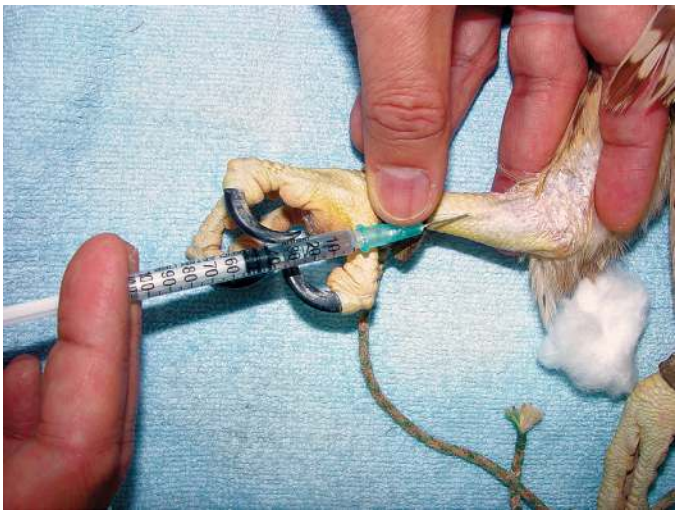


FIGURE 8-2 Intravenous injection into the medial metatarsal vein of a lanner falcon (*Falco biarmicus*).

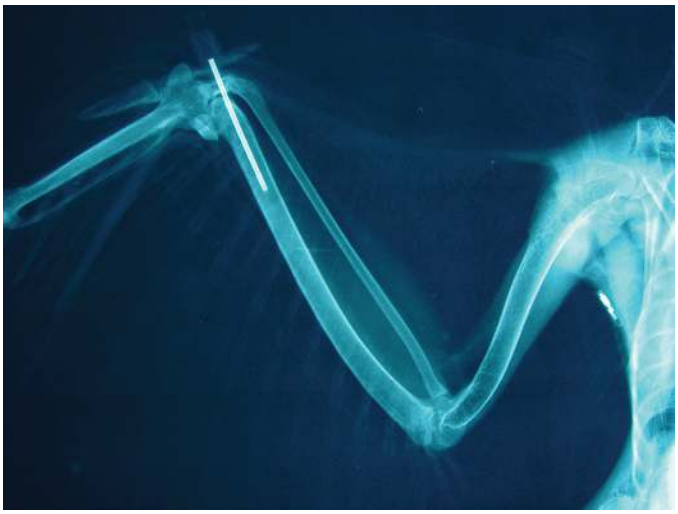


FIGURE 8-3 Intraosseous administration into the distal ulna of a saker/gyr hybrid falcon (*Falco cherrug–F. rusticolus*).

FIGURE 8-4 Nasal flushing in a peregrine falcon.

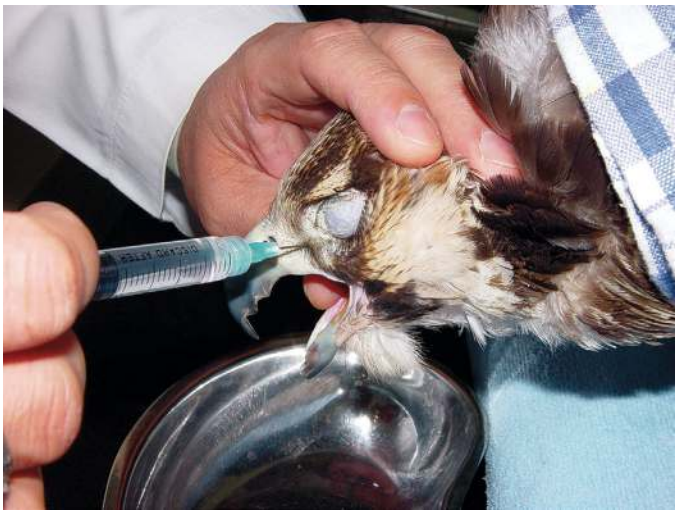


FIGURE 8-5 Sinus flushing in a peregrine falcon.

FIGURE 8-6 Air compressor, nebulizer, and nebulizing chamber used for small birds.

(Jenkins, 1997; Rupley, 1997). A volume of up to 40 mL on each naris has been used to dislodge large amounts of rhinoliths and debris in falcons. It is best to hold the bird's head down while flushing to prevent fluid aspiration into the trachea.

- *Endoscopic-guided administration.* A variety of medications may also be administered during endoscopic procedures. These techniques have been used in the treatment of air sac aspergillosis with local instillation of antifungals into the air sacs or injections into fungal granulomas.

ORAL ADMINISTRATION

Administrating drugs or other compounds orally has much to commend it, especially its ease of administration. Oral administration can include (1) incorporation of the compound in the food or water, (2) administration into the oral cavity with a syringe, and (3) administration by crop or stomach tube. The practicalities of the latter are discussed later, but first some general points about oral administration are given in the following list.

- Administration in food or water, although apparently simple, can present problems. Some birds rarely, if ever, drink and thus administration in drinking water may prove futile. For instance, tetracyclines administered in water have proven to reach low plasma levels in budgerigars whereas they are usually appropriate in cockatiels. Birds that are unwell may not feed or may take in very little food, thus, compounds/drugs in the food may not be ingested, let alone absorbed. Even if a bird is drinking and eating, it may not take in adequate medication because it is aware of the appearance or the taste of the compound in the food. Among birds, carnivorous and piscivorous birds usually take medications hidden in food items easily. However, this strategy is usually not optimal in granivorous and frugivorous species. Giving drugs in food or drinking water may be the only feasible treatment in large collections of birds, in particular, passerine birds.
- Even if the compound is ingested, apparently in adequate amounts, absorption from the gastrointestinal tract may be insufficient to produce adequate blood levels of that agent (this is not applicable to drugs that are intended only for action within the intestinal tract). The absorption may be inadequate under any circumstances or may be reduced because the crop, stomach, or intestines are full of ingesta. Even the efficacy of drugs intended for use within the intestine may be reduced if there are large quantities of ingesta present.

It will be appreciated, therefore, that oral administration of drugs in food items, although very tempting, and sometimes very useful, is not without its drawbacks (Table 8-2). Before embarking on this course of action, veterinarians should check the previous points and be reassured that the therapy is likely to prove effective. Putting drugs in food can be done in a number of ways. Birds fed a proprietary mash or pelleted diet can have the compound included either at the source (the manufacturer) or mixed at a later stage by the owner or veterinary surgeon. For birds that eat fruit, it may be possible to inject the compound into a particularly favored item. Birds that eat meat or whole animals can have certain agents hidden within the meat or the body of the prey.

When it comes to administration of drinking water, assuming that the bird is a species that regularly drinks, the question of appearance and palatability comes to the forefront. Some drugs color the water and this may discourage a bird from drinking or reduce its intake. Likewise, the taste of a drug may reduce palatability. The effect of color can be minimized by providing the bird with water in a dark brown or similarly colored container for a few days before medication starts. Palatability is less easy to solve but, in some cases, adding sugar or another sweet substance to the water may help disguise the taste of the medication, but it may promote bacterial overgrowth.

Clearly, oral administration in food is not a panacea. In the past, a standard treatment for sick cage birds in many veterinary practices was "oxytetracycline in the drinking water." Over the years many authors have drawn attention to the unreliability of this approach, and the modern avian veterinarian is well aware that he must be more enterprising if drugs are to be effective.

The best way to orally administer drugs is direct administration into the oral cavity of a manually restrained bird, which remains the method of choice for most pet birds. In hospitalized birds, crop administration is also convenient and may be combined with nutritional support (see the following section, Tube Feeding and Nutritional Support).

Oral drugs may be available in different strengths and formulations. In general, liquid suspensions or solutions are more convenient to administer in the majority of birds, and drugs not commercially available in liquid must be compounded. Veterinary compounding must be based on knowledge of the effect of compounding techniques and storage time and conditions on the stability of the drugs. However, pills and tablets may be easily administered to some bird species such as birds of prey and waterfowl.

TABLE 8-2 Oral Administration: Advantages and Disadvantages

Route of Administration	Advantages	Disadvantages
Directly by mouth—capsules/tables	Can be administered by the owner Provides a definite drug volume administered to individual bird	Can be stressful for both the patient and the person giving the medication Limited to tame and relatively docile birds Absorption into the gut can be unpredictable Time-consuming in large numbers of birds
Fluids and suspensions via crop or stomach tube	Very useful for gastrointestinal-tract–associated conditions Allows accurate dosing Fast arrival time into the gut Good absorption	Can be stressful because of repeated handling Risk of regurgitation and aspiration Time-consuming in large numbers of birds
Medicated feed	Handling of birds not required No stress Birds will self-medicate several times daily Food consumption is more consistent than water consumption Very practical in large numbers of birds Useful for prevention, follow-up treatment, or treatment of gastrointestinal-tract–associated conditions Reliable gut concentrations or blood levels	Unsatisfactory in anorectic birds Food intake may be affected by the taste Unpredictable interaction between drug and food Depending on the type of drug used, there may be insufficient absorption to reach therapeutic blood levels
Medicated drinking water	Handling of birds not required No stress Birds will self-medicate several times daily Probably the only possible route when medicating large flocks or wild birds Reduces bacterial contaminants in drinking water Helps to control the multiplication of bacteria in the oropharynx	Intake and absorption of antibiotics administered in drinking water are unreliable, especially in species adapted to conditions that require very little drinking Some drugs are not stable or soluble in water Therapeutic blood levels are rarely achieved Inadequate intake of antibiotics may lead to drug-resistant bacteria

TABLE 8-3 Topical Therapy: Advantages and Disadvantages

Topical Medicaments	Advantages	Disadvantages
Ophthalmic ointments	Remain in the eyes and exert their therapeutic action longer than eye drops or solutions Can be squeezed directly into the nostrils of birds to treat sinusitis	Excessive amount of ointment contaminating skin of eyelids may cause irritation and self-mutilation
Creams and ointments	Useful for localized infections, such as dermatitis or skin trauma, that do not warrant systemic injections	Can cause clogging of the feathers May lead to self-mutilation May cause toxicosis if ingested when the bird preens Steroid-containing creams can cause polydipsia if used excessively
Ear and eye drops	Useful in treating rhinitis by instillation of the external nares Preferable to ointments because this will limit damage to feathers	Short duration of therapeutic action
Aerosol antibiotic/anthelmintic sprays	Does not cause clogging of the feathers	The color of the spray stains the feathers making them look unpleasant
Powders	Useful for the treatment of ectoparasites	
Antibiotic ointment/DMSO impregnated gauze swab	Useful in treating localized soft tissue swelling on the foot	
AIPMA beads	Useful in treating bumblefoot and other infected, ischemic wounds Higher local concentrations of antibiotic can be achieved without relying on vascular supply and tissue integrity	

AIPMA, Antibiotic-impregnated polymethyl methacrylate; DMSO, dimethyl sulfoxide.

TOPICAL ADMINISTRATION

Topical drug administration gives the advantage of direct application to the site of the insult, with advantages and disadvantages as outlined in Table 8-3. The main disadvantage of such an approach is the short duration of the action of topical medications because of systemic absorption. Ointments or topical cream manufactured for use in humans or domestic mammals should be used cautiously in birds because the dose administered may be considerably higher than expected. For instance, some corticosteroid ointments may have lasting toxic effects on birds and should not be used under any circumstances.

The use of antibiotic-impregnated polymethyl methacrylate (AIPMA) beads, after surgical debridement, offers an effective method for the delivery of antibiotics to an infected ischemic site (Klemm,

1993). This technique has been used in birds for the treatment of osteomyelitis, cellulitis (Wheler *et al.*, 1996), and bumblefoot (Remple and Forbes, 2000). Some antibiotics that can be incorporated in AIPMA beads include piperacillin, rifampin, amoxicillin, clindamycin, enrofloxacin, and gentamicin (Remple and Forbes, 2000). Several other compounds may be mixed with active drugs to obtain sustained-release formulations. One such example is poloxamer gel (Pluronic F-127), which is a thermoreversible polymer that gellifies at body temperature and is compatible with most antimicrobials and other drugs. It can be applied directly in infected sites for longer duration of injectable antibiotics (e.g., pododermatitis, granulomas) or as an ointment.

Wounds are treated with a variety of ointments, depending on their nature. Most ointments have one or several active drug ingredients. Honey and silver are particularly popular in infected wounds because of their long-lasting effects of reducing the frequency of bandage changes and the fact that avian wounds are poorly exudative.

ACKNOWLEDGMENT

The author would like to acknowledge the contribution of Jesus Naldo to this section in the previous edition.

REFERENCES

Hunter RP, Isaza R: Concepts and issues with interspecies scaling in zoological pharmacology, *J Zoo Wildl Med* 39(4):517–526, 2008.

Jenkins JR: Hospital techniques and supportive care. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Klemm KW: Antibiotic bead chains, *Clin Orthop Relat Res* 295:63–76, 1993.

Remple JD, Forbes NA: Antibiotic-impregnated polymethyl methacrylate beads in the treatment of bumblefoot in raptors. In Lumeij JT, Remple JD, Redig PT, et al, editors: *Raptor biomedicine III*, Lake Worth, FL, 2000, Zoological Education Network.

Rupley AE: *Manual of avian practice*, Philadelphia, PA, 1997, WB Saunders.

Wheler CL, Machin KL, Lew LJ: *Use of antibiotic-impregnated polymethacrylate beads in the treatment of chronic osteomyelitis and cellulitis in a juvenile bald eagle (Haliaeetus leucocephalus)*, Proceedings of the Association of Avian Veterinarians, Tampa, FL, 1996.

FURTHER READING

Dorrestein GM: Principles of therapy. In Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *Manual of raptors, pigeons and waterfowl*, Cheltenham, UK, 1996, British Small Animal Veterinary Association.

Dorrestein GM: Nursing the sick bird. In Tully TN, Lawton MPC, Dorrestein GM, editors: *Avian medicine*, Oxford, UK, 2000, Butterworth-Heinemann.

Flammer K: Antimicrobial therapy. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Forbes NA, Lawton MPC: Examination, basic investigation and principles of therapy. In Beynon PH, Forbes NA, Lawton MPC, editors: *Manual of psittacine birds*, Cheltenham, UK, 1996, British Small Animal Veterinary Association.

Rosskopf WJ, Woerpel RW: Practical avian therapeutics with dosages of commonly used drugs. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, MD, 1996, Williams & Wilkins.

NEBULIZATION IN BIRDS

Hugues Beaufrère

The goals of aerosol therapy are to deliver airborne therapeutics directly to diseased respiratory tissues and to moisten airways to increase clearance of exudates and necrotic debris (Tully and Harrison, 1994). Local therapeutic delivery may be indicated when vascularization is decreased in diseased tissues or in low-perfused respiratory areas such as the air sacs. Nebulization allows high local concentration of therapeutics while maximizing its efficacy and minimizing systemic absorption, reducing potential for toxicity, and reducing drug biotransformation. Humidification of the mucociliary escalator may also improve its efficiency.

Depending on particle sizes and airway diameters, aerosolized therapeutics may only reach the upper respiratory system, the larger airways, or lungs and air sacs. Birds' airways have the smallest diameters in vertebrates with parabronchi diameters ranging from 0.5 (e.g., hummingbirds) to 2 mm (e.g., chickens, penguins) and air capillaries ranging from 3 (e.g., passerines) to 10 µm (e.g., swans, coots; King and McLelland, 1984). In comparison, mammalian alveoli have an approximate diameter of 30 to 150 µm (smallest is in mice). Birds with smaller airways require smaller particle sizes, and particle diameters of 0.5 µm are generally recommended (Quesenberry and Hillyer, 1994; Tully and Harrison, 1994). During celioscopy amphotericin B coating caudal air sacs of birds that have been nebulized is sometimes seen, suggesting that nebulized drugs also reach the caudal thoracic and abdominal air sacs in diseased birds.

Aerosol therapy includes humidification, vaporization, and nebulization. The first two methods produce particles that are too large to reach the lower respiratory system but may be adequate for upper respiratory or tracheal diseases. The latter produces particles small enough to be deposited into smaller pulmonary airways. Nebulization can be achieved with a variety of devices using different technology such as air-jet nebulizers and ultrasonic nebulizers (including vibrating mesh technology; Watts *et al.*, 2008). Air-jet nebulizers are noisier and heavier but are less expensive and have a greater ability to nebulize viscous liquid than ultrasonic nebulizers (Fig. 8-7). Pressurized metered-dose inhaler aerosols (such as classically available for albuterol) are a type of air-jet nebulizer and delivers 1- to 2-µm particles (Taylor *et al.*, 1993). Drugs used for nebulization should ideally be hydrosoluble, of low viscosity, and display adequate nebulization characteristics. Drugs that are commonly nebulized include 0.9% saline, antibiotics, antifungals, antiparasitics, bronchodilators, and mucolytic agents (Table 8-4). Nebulized drugs may induce bronchospasm and patients should be assessed before and after administration (e.g., *N*-acetyl cysteine).

FIGURE 8-7 Air-jet nebulizer commonly used in avian medicine.

TABLE 8-4 Drugs Commonly Used for Nebulization in Birds

Drug	Dose
Antibiotics	
Amikacin	5-6 mg/mL sterile water/saline
Gentamicin	3-6 mg/mL sterile water/saline
Cefotaxime	10 mg/mL saline
Doxycycline	13 mg/mL saline
Tobramycin	1 mg/mL saline
Enrofloxacin	10 mg/mL saline
Piperacillin	10 mg/mL saline
Antifungals	
Amphotericin B	0.1-5 mg/mL sterile water
Enilconazole	10 mg/mL sterile water/saline
Clotrimazole	10 mg/mL in propylene glycol
Terbinafine	1 mg/mL sterile water
Voriconazole	10 mg/mL saline
Bronchodilators	
Aminophylline	3 mg/mL sterile water/saline
Terbutaline	0.01 mg/kg in 5-10 mL saline
Other	
N-acetyl cysteine	22 g/mL sterile water
F10 disinfectant	10 mL of 1:250 dilution

From Hawkins M, Barron H, Speer B, et al: Nebulization agents used in birds. In Carpenter J, Marion C, editors: *Avian and exotic formulary*, ed 4, St Louis, MO, 2013, Elsevier Saunders and Tully T, Harrison G: Pneumonology. In Ritchie B, Harrison G, Harrison L, editors: *Avian medicine: principles and applications*, Palm Beach, FL, 1994, Wingers Publishing.

Nebulization time is typically 15 to 30 minutes and frequency is typically once to three times daily. In birds, the respiratory surface and functional efficiency of avian lungs may lead to greater systemic absorption of the nebulized drugs than in other species. This may be advantageous in certain circumstances when obtaining drug plasma therapeutic levels without having to restrain the bird. However, plasma concentrations achieved are usually low and of low duration. For instance, the pharmacokinetics of the these nebulized drugs have been performed in birds: terbinafine, voriconazole, oxytetracycline, and ceftriaxone (Emery *et al.*, 2012; Beernaert *et al.*, 2009; Dyer and Van Alstine, 1987; Junge *et al.*, 1994; Van Alstine and Dyer, 1985).

REFERENCES

Beernaert LA, Baert K, Marin P, et al: Designing voriconazole treatment for racing pigeons: balancing between hepatic enzyme auto induction and toxicity, *Med Mycol* 47(3):276–285, 2009.

Dyer D, Van Alstine W: Antibiotic aerosolization: tissue and plasma oxytetracycline concentrations in parakeets, *Avian Dis* 31: 677–679, 1987.

Emery LC, Cox SK, Souza MJ: Pharmacokinetics of nebulized terbinafine in Hispaniolan Amazon parrots (*Amazona ventralis*), *J Avian Med Surg* 26(3):161–166, 2012.

Hawkins M, Barron H, Speer B, et al: Nebulization agents used in birds. In Carpenter J, Marion C, editors: *Avian and exotic formulary*, ed 4, St Louis, MO, 2013, Elsevier Saunders.

Junge R, Naeger L, LeBeau M, et al: Pharmacokinetics of intramuscular and nebulized ceftriaxone in chickens, *J Zoo Wildl Anim Med* 25(2):224–228, 1994.

King A, McLelland J: Respiratory system. In King A, McLelland J, editors: *Birds: their structure and function*, Eastbourne, UK, 1984, Bailliere Tindall.

Quesenberry K, Hillyer E: Supportive care and emergency therapy. In Ritchie B, Harrison G, Harrison L, editors: *Avian medicine: principles and applications*, Lake Worth, FL, 1994, Wingers Publishing.

Taylor RH, Lerman J, Chambers C, et al: Dosing efficiency and particle-size characteristics of pressurized metered-dose inhaler aerosols in narrow catheters, *Chest* 103(3):920–924, 1993.

Tully T, Harrison G: Pneumonology. In Ritchie B, Harrison G, Harrison L, editors: *Avian medicine: principles and applications*, Palm Beach, FL, 1994, Wingers Publishing.

Van Alstine WG, Dyer DC: Antibiotic aerosolization: tissue and plasma oxytetracycline concentrations in turkey poults, *Avian Dis* 29(2):430–436, 1985.

Watts AB, McConville JT, Williams RO: Current therapies and technological advances in aqueous aerosol drug delivery, *Drug Dev Industr Pharm* 34(9):913–922, 2008.

PARENTERAL FLUID THERAPY

Hugues Beaufrère

APPLIED PHYSIOLOGY OF AVIAN BODY FLUIDS

To understand and properly apply fluid therapy principles to avian patients, a good knowledge of fluid physiology is of great importance. While birds share the same body fluid dynamics principles as mammals, several important differences exist and can be clinically significant. Knowledge of both general principles of veterinary fluid therapy and avian-specific osmoregulatory and vascular physiology are required to provide a sound and effective fluid therapy plan.

The distribution of fluids in the avian body is similar to mammals (DiBartola *et al.*, 2012). About 60% to 70% of a bird's body weight is made of water with a one third extracellular and two thirds intracellular (Goldstein and Skadhauge, 2000). The extracellular fluid is composed of interstitial and intravascular fluid (plasma). Plasma represents about 3.5% to 6.5% of the total avian body weight (hence blood is about 10%; Goldstein and Skadhauge, 2000). In fact, blood volume varies by bird species (e.g., 5% of body weight in pheasants, 7.5% in quails, 10.5% in galahs). Body fluids contain a variety of solutes that get exchanged between fluid compartments through biological membranes with different permeability to solutes and electrolytic transport pumps. The extracellular fluid predominantly contains sodium and chloride, whereas the intracellular fluid mainly contains potassium, phosphorus, and magnesium (DiBartola *et al.*, 2012). Other solutes are in low concentrations in both compartments. A variety of osmoregulatory mechanisms keep body fluid composition within tight limits as part of the overall homeostasis of the avian organism. The vascular system perfuses all cells of the body as a transport mechanism for fluids, dissolved respiratory gases, metabolites, proteins, and blood cells. Avian plasma has a slightly higher osmolality than mammals at about 300 to 340 mOsm/kg depending on the species (Goldstein and Skadhauge, 2000; Beaufrère *et al.*, 2011). Large molecules, such as proteins in bird's plasma, are responsible for 5% of plasma osmolality and for retaining fluids in the intravascular space (colloidal pressure), especially at capillary beds, according to the Starling mechanism. Reported avian colloid osmotic pressure ranged from 9 to 20 mm Hg depending on species and reports (Cornelius *et al.*, 1982; Keil *et al.*, 1991; Peltonen and Sankari, 2011).

A bird's main osmoregulatory organs are the kidneys, coprodeum and colon, and salt glands (species dependent; Braun, 2015). The

kidneys are typically composed of the cranial, middle, and caudal renal lobes, which contain about 30% looped nephrons and 70% loopless nephrons. A renal portal system made of a vascular circle of veins containing a renal portal valve is present ventral to the kidneys. It perfuses the tubules directly from venous blood draining from the lower part of the body. Birds are uricotelic, which means the end product of protein catabolism is uric acid, which is produced by the liver. Uric acid is of low toxicity compared with urea and does not diffuse through biological membranes. Uric acid is excreted predominantly by tubular secretion at 90%. The low toxicity of uric acid combined with its excretion by tubules means that birds do not need to maintain a constant glomerular filtration rate (GFR) like mammals to eliminate toxic nitrogenous waste. Indeed, birds modulate their GFR depending on hydration status but preserve tubular perfusion through the renal portal system. Once uric acid is excreted into the tubules, it combines with mucopolysaccharides and electrolytes to form microspheres that allow it to exist in supersaturated suspension without precipitation and bind electrolytes (Braun, 2015). Avian kidneys have a moderate ability to concentrate urine, but postrenal handling of urine in the coprodeum and colon allows further reabsorption of water and electrolytes and further concentration of the urine. Salt glands are very effective osmoregulatory organs and are present in most marine birds, some desert-dwelling birds, and other birds (Braun, 2015). They may be the primary osmoregulatory organ in these bird species, thus, their function should not be overlooked. Although avian kidneys typically concentrate urine less than mammals, it is only one aspect of avian osmoregulation, and birds' overall water conservation mechanisms tend to be superior to most mammals.

The avian water requirement in species commonly seen in clinics has been poorly investigated but is roughly estimated to be 50 to 100 mL/kg/day. Neonates and passerine birds tend to have higher maintenance requirements (Quesenberry and Hillyer, 1994; Steinohrt, 1999).

TECHNICAL ASPECTS OF FLUID THERAPY IN BIRDS

Fluids may be given through the oral, SC, IV, and IO routes. Other routes are described but are less practical and not typically used in practice. The choice of routes of fluid administration and the type of fluid depend on several criteria, such as the degree of dehydration; inciting cause of dehydration; species; demeanor of the bird; degree of depression; and clinical, hematologic, and biochemical endpoints. Table 8-5 gives an overview of the advantages and disadvantages of different routes. For oral fluid therapy, the reader is invited to consult the corresponding section.

The SC route is typically used in birds for maintenance therapy, as a vehicle for tissue-irritating drugs (e.g., enrofloxacin), or for replacement therapy in mildly dehydrated birds. Peripheral vasoconstriction that occurs during more severe dehydration or hypovolemia precludes its use in these situations. In addition, hypertonic and colloid fluids and dextrose higher than 2.5% cannot be administered. Some birds such as the brown pelicans (*Pelecanus occidentalis*) and the turkey vulture (*Cathartes aura*) have extensive subcutaneous air connected to various air sacs (typically the interclavicular air sac); therefore, SC administration should be used cautiously in these species. SC fluid is typically administered in the inguinal, axillary, or interscapular area using large needles for quick administration (Fig. 8-8).

The IV route is the route of choice as replacement therapy can be fine-tuned to the various electrolytic and acid-base disorders and permits rapid dissemination throughout the body (Fig. 8-9). All types of fluids can be given intravenously. When IV access is available, all therapeutics may be given intravenously and constant rate infusion of drugs is possible. The main disadvantage associated with IV catheterization is its low acceptance from birds resulting in difficulties in

FIGURE 8-8 Subcutaneous fluid injection in the inguinal web in a canary.

TABLE 8-5 Selected Routes of Fluid Administration in Birds

Routes	Sites	Advantages	Disadvantages	Fluid Type
SC	Inguinal web Interscapular Axillary area	Least invasive Well tolerated	Only for mild dehydration Limited volume (10 mL/kg/site)	Isotonic Hypotonic
IV	Ulnar vein Medial metatarsal vein Jugular vein	IV access for drugs Intravascular fluid Rapid dissemination More precise dosing	Low tolerance Significant bleeding if removed by bird	Isotonic Hypotonic Hypertonic Colloid Blood
IO	Ulna Tibiotarsus	See IV No bleeding if removed by bird Ideal when veins are too small or collapsed	Low tolerance Painful Potential for osteomyelitis Fluid extravasation with high rates	Isotonic Hypotonic (Hypertonic) (Colloid) Blood

IO, Intraosseous; IV, intravenous; SC, subcutaneous.

maintaining access, especially in Psittaciformes. However, it is well tolerated in most Galliformes, Anseriformes, hooded Falconiformes, and many others. Significant bleeding may occur if the bird bites the IV line. Small antisiphon valves (NP Medical Inc.) may be placed proximally so bleeding does not occur in case the line is sectioned by the bird. Most common sites include the ulnar vein (Fig. 8-10), the medial metatarsal vein (Fig. 8-11), and the jugular vein. Administration requires the use of fluid pumps or syringe pumps in smaller species to give a precise rate (see Fig. 8-9). In low-resource settings or in field situations, spring-loaded syringe infusers (e.g., Springfusor) may be used but the rate is predetermined. IV fluid administration is recommended for moderate to severe dehydration and during surgery. It is routinely performed in birds down to 100 g of body weight. Except in moribund or cooperative birds, sedation is required, and the author typically uses a combination of midazolam 1 to 3 mg/kg and butorphanol 2 mg/kg in Psittaciformes. The midazolam can be reversed with flumazenil at 0.05 mg/kg.

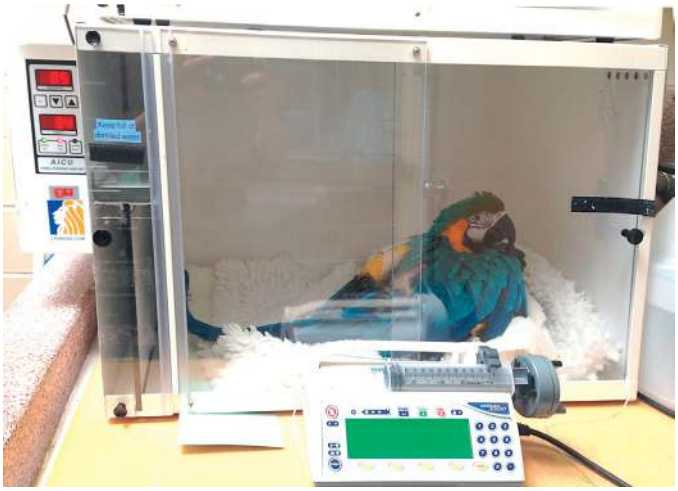


FIGURE 8-9 Blue and gold macaw receiving fluid replacement therapy through an ulnar vein catheter and programmable syringe pump. Preprogrammed boluses are entered in the syringe pump in case a fluid bolus must be quickly administered. The bird is kept in a temperature-controlled incubator.

The IO route is similar to the IV route and is easier and faster to secure. It can be placed in birds of all sizes. Studies have shown that it is almost identical to IV access (Lamberski and Daniel, 1992; Aguilar *et al.*, 1993) It is typically placed either in the distal ulna or proximal tibiotarsus. Pneumatic bones should be avoided. In some birds, such as Cathartiformes, the ulna is pneumatized, which precludes the placement of an IO catheter at that location (Stringfield, 2012). Placement in the ulna is favored because correct placement can be verified by injecting a small fluid bolus or a bubble of air and observing it flowing in the ulnar vein. Also birds are bipedal, and pain induced to the leg may cause lameness and discomfort while standing. For the ulnar placement, a dorsal approach may be used where the manus is slightly pronated, then a small number of feathers are plucked in the area, and a 22-gauge 1.5-inch long spinal needle is inserted just distal to the dorsal condyle. In smaller birds, a 25- or 26-gauge hypodermic needle can be used, but a core of bone may obstruct the needle, which will need to be replaced. Alternatively, a small wire stylet may be used during placement. For tibiotarsal placement, the knee is flexed, the patellar tendon is pushed medially, and the spinal needle is inserted into the bone. Radiographs are not needed to confirm placement but may still be performed if needed. Fluid accumulation in soft tissue indicates misplacement. Once in place, antiseptic ointment should be applied to the site and it should be bandaged and treated like an IV catheter. Local lidocaine infusion may be used before placement. Sedation is required except in lethargic birds. Birds are not routinely anesthetized for IO catheterization because they tend not to be good candidates for anesthesia. All types of fluids may be infused; hypertonic solutions and colloids tend to cause painful reactions and are best diluted.

All administered fluids must be warmed to avian body temperature (38° to 40°C). This requires storing fluid packages in incubators and using in-line fluid warmers if available (Fig. 8-12).

TYPES OF FLUIDS AND INDICATIONS

Fluids are classified as crystalloids or colloids. Crystalloids are further divided into hypotonic, isotonic, and hypertonic. Table 8-6 lists the

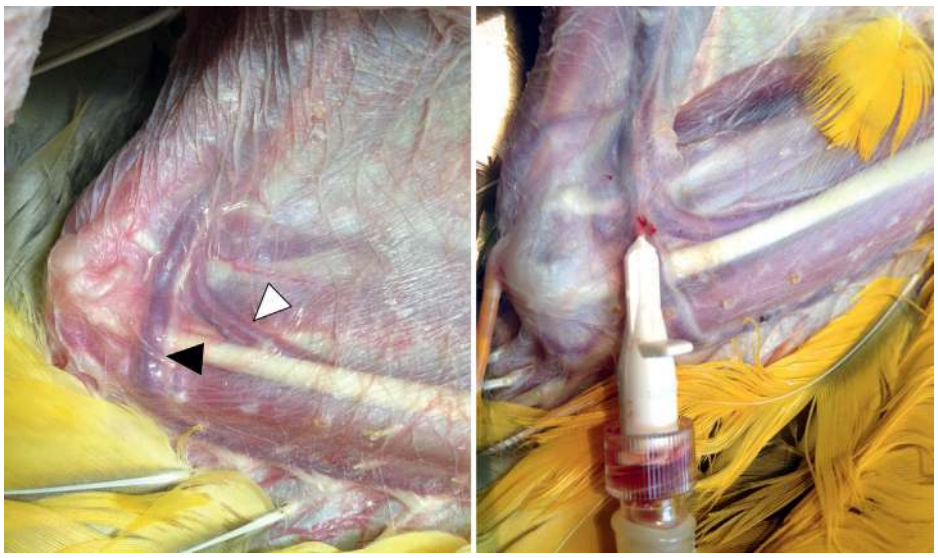


FIGURE 8-10 Placement of an intravenous 26-gauge catheter in a severely dehydrated blue and gold macaw. Left, the ulnar vein (*black arrowhead*) and the superficial ulnar artery (*white arrowhead*) are easily seen through the thin skin.

FIGURE 8-11 Placement of an intravenous 26-gauge catheter in a dehydrated, yellow-crowned Amazon parrot.

FIGURE 8-12 This Exo Terra reptile egg incubator is an inexpensive and efficient fluid-warming storage unit.

composition of common crystalloid fluids used in avian medicine and their characteristics. Prepackaged fluids have been developed for mammalian usage, which have lower plasma osmolality than birds. As a consequence, fluids classified as isotonic may be slightly hypotonic for birds (e.g., lactated Ringer's solution [LRS]). Crystalloid fluids may or may not be buffered. Depending on their composition and characteristics, crystalloids are either maintenance or replacement fluids. Maintenance fluids are seldom used in avian medicine, and they approximate the normal daily requirements of fluids and electrolytes for animals unable to drink. Replacement fluids approximate the extracellular fluid composition in electrolytes and are used for correcting losses of water and electrolytes. Crystalloids should be considered as interstitial rehydrators because only 25% of fluids remain in circulation after a short period of time. LRS, Normosol-R, and Plasma-Lyte A are balanced buffered solutions that more closely approximate the extracellular fluid composition; therefore, they can be used in most situations. Saline 0.9% is not buffered and unbalanced and is typically restricted for patients with metabolic alkalosis. LRS is hypoosmolar to avian plasma and should not be used at a high IV rate (e.g., resuscitation), but it is well suited for SC administration because it may be absorbed more readily than isotonic solutions. Plasma-Lyte A 7.4 is the closest fluid to avian plasma composition in pH, osmolality, and electrolyte concentrations. Hypertonic saline can be considered as an intravascular expander because it leads to rapid intravascular expansion equivalent to colloids at one fourth the volume. It is used in resuscitation and in combination with crystalloids and colloids. Dextrose 5% is primarily used as a carrier for CRIs of drugs or as a source of pure water.

Colloids are large molecules that do not readily diffuse across membranes (Table 8-7). They increase the plasma colloidal pressure attracting interstitial fluids into the intravascular space. As such, they can be considered intravascular expanders and are typically used when the colloidal pressure is low, in hypotension and hypovolemia, in hypoproteinemia, in significant hemorrhages, and in fluid resuscitation. Main contraindications include coagulopathy, pneumonia, congestive heart failure, and renal failure. Blood is also considered a colloid while providing blood cells and coagulation factors. Blood is typically transfused whole in birds and collected from the same species of birds. The half-life of infused blood is about a week. Transfusing from other bird species is not indicated because transfused blood cells are rapidly destroyed (Sandmeier *et al.*, 1994; Degernes *et al.*, 1999). Oxyglobin, a hemoglobin-based oxygen carrier colloid, has been discontinued.

With IV or IO catheters, various additives may be added to the fluids or in a separate IV lines with a dedicated syringe pump

TABLE 8-6 Selected Crystalloid Fluids and Their Characteristics

Crystalloids	pH	mOsm/L	Na	Cl	K	Ca	Buffer	Tonicity
LRS	6.5	272	130	109	4	3	Lactate	Isotonic, slightly hypotonic for birds
Plasma-Lyte A	5.5	312	140	103	10	5	Acetate	Isotonic
Plasma-Lyte A 7.4	7.4	294	140	98	5	0	Acetate	Isotonic
Plasma-Lyte M in 5% dextrose	5.5	377	40	40	16	5	Acetate and lactate	Isotonic
Normosol-R	6.4	296	140	98	5	0	Acetate	Isotonic
Hartmann's	6.3	279	131	112	5	2	Lactate	Isotonic, slightly hypotonic for birds
0.9% NaCl	5.0	308	154	154	0	0	None	Isotonic
0.45% NaCl	5.0	154	77	77	0	0	None	Hypotonic
3% NaCl	5.0	1026	513	513	0	0	None	Hypertonic
7.5% NaCl	5.0	2566	1283	1283	0	0	None	Hypertonic
5% dextrose	4.0	252	0	0	0	0	None	Hypotonic

TABLE 8-7 Selected Colloidal Fluids and Their Characteristics

Colloids	MW (kDa)	mOsm/L	Half Life	Dose
6% hetastarch	670	310	25 h	20 mL/kg/day 5 mL/kg bolus, can be repeated twice
10% pentastarch	200	326	2.5 h	20 mL/kg/day 5 mL/kg bolus, can be repeated twice
	NA	300-340	8-10 days	Full volume administered over 1-4 h

MW, Molecular weight.

(Table 8-8). Most replacement fluids are deficient in potassium and should be supplemented after initial stabilization or hypokalemia may develop after a few days.

THE FLUID THERAPY PLAN

The objectives of fluid replacement therapy are to replace fluid deficits to correct perfusion and hydration without inducing fluid overload, to anticipate fluid loss and provide maintenance needs, and to correct electrolytes and acid-base abnormalities. In addition, the fluid therapy plan must be monitored and reassessed and underlying conditions must be diagnosed and treated.

The first step is to determine the presence and estimate the degree of dehydration and other homeostatic abnormalities. Overall dehydration is harder to assess in birds than in mammals (Steinohrt, 1999). Typical clinical signs encountered with dehydration in birds are presented in Table 8-9. Unfortunately, indirect blood pressure measurement is unreliable in small- to medium-sized birds, so hypotension may have to be estimated based on physical examination in these species (Acierno *et al.*, 2008; Johnston *et al.*, 2011). However, arterial pressure may be monitored using an arterial catheter in larger birds (Fig. 8-13; Schnellbacher *et al.*, 2014). If available, blood gas and electrolyte measurements are important and critical in fluid selection.

Fluid therapy is classically divided into three stages: resuscitation, rehydration, and maintenance. If the bird is unstable and has signs of hypoperfusion, shock, or active hemorrhage, then emergency fluid resuscitation is required. Fortunately, birds are more resistant to hypovolemic shock than mammals and do not go into decompensated shock below 60% of intravascular volume loss (Djojosugito *et al.*, 2002; Lichtenberger *et al.*, 2002; Lichtenberger, 2007). Fluids classically used in resuscitation include isotonic-balanced crystalloids such as Plasma-Lyte A 7.4 and/or a combination of hypertonic saline (7.5% NaCl), crystalloids, and colloids. To use the least amount of fluids to reach the desired effect and to achieve fast intravascular volume expansion and reverse the hypotensive state, it is recommended to use 3 mL/kg of 7.5% NaCl mixed with 5 mL/kg of colloid given over 10 minutes followed by crystalloids bolus at 10 mL/kg (Lichtenberger, 2007). Crystalloid boluses may be repeated every 10 to 15 minutes until improvement of clinical markers is seen. If treating large birds, indirect blood pressure may be measured using a Doppler unit. Atropine (0.2 mg/kg IV) and epinephrine (0.02 mg/kg IV) may also be used if

FIGURE 8-13 Intraarterial 26-gauge catheter placed into the deep radial artery in a gyrfalcon in preparation for surgery. This approach is blind, based on anatomic landmarks of the wing.

TABLE 8-8 Selected Fluid Additives Commonly Used in Birds

	Dose	Comments
Additives		
Dextrose 50%	Dilute to 2.5-5% 0.5 mL/kg bolus over 15 minutes	Treatment hypoglycemia and metabolic support
KCl	Maintenance: total dose of 15-20 mEq/L Moderate hypokalemia: 40 mEq/L Severe hypokalemia: 60 mEq/L	Correct fluid deficiency Treatment of hypokalemia Do not exceed 0.5 mEq/kg/h
$NaHCO_3$	0.3*W_{kg}*base excess over 30-60 minutes, may give half initially	Metabolic acidosis
Calcium gluconate 10%	0.5-1.5 mL/kg bolus over 15-20 minutes 1-5 mg elemental calcium/kg/h	Treatment of ionized hypocalcemia or hyperkalemia Use NaCl 0.9% as fluid or precipitation may occur
Insulin	0.5 U/kg + 2 g of dextrose/insulin units	Hyperkalemia
Constant Rate Infusion		
Hydromorphone	0.1 mg/kg/h	Pain management (various)
Butorphanol	1 mg/kg/h	Pain management (parrots)
Fentanyl	10 to 30 mcg/kg/h	Pain management (hawks)
Dopamine	5-15 µg/kg/min	Hypotension

TABLE 8-9 Signs of Dehydration in Birds

	Clinical Endpoints
Clinical signs of dehydration (interstitial loss) 5%	Subclinical dehydration History of fluid loss Skin slow to return when tenting of skin over keel or eyelids
7-8%	Lethargy Dry and tacky mucous membranes Strings of thick mucus in oral cavity
10-12%	Skin very slow to return when tenting skin over keel and eyelids Sunken eyes
15%	Comatose, marked weakness
Clinical signs of hypovolemia (intravascular loss)	Altered consciousness Tachycardia Low ulnar vein refilling time (>1 to 2 seconds) Poorly palpable pulse Hypothermia Hypotension
Common laboratory findings with dehydration (depending on comorbid conditions, laboratory changes may be inconsistent)	Increased PCV (Packed cell volume) Increased TS (Total solids) and TP (Total protein) Increased urea Increased uric acid Increased electrolyte concentrations Increased plasma osmolality Increased blood lactate (from hypoperfusion) Increased blood glucose (from increased sympathetic tone) Altered plasma pH

nonresponsive. At this stage, bloodwork, electrolytes, and blood gas analysis may help assess other causes of nonresponsive shock (e.g., hypoglycemia, hypocalcemia). If severe hemorrhage occurred or severe anemia is present, birds may benefit from homologous transfusions. The transfusion (usually 10% of body weight taken from a donor bird because a higher volume is generally not feasible unless several donors are available) is typically administered over 1 to 4 hours and the use of a pediatric microfilter is recommended (Jankowski and Nevarez, 2010).

For the rehydration phase of the fluid therapy plan, once perfusion has been restored and the degree of dehydration estimated, the rate of fluids should be calculated. Usually 50% to 100% of estimated loss may be replenished within the first 24 hours, and maintenance requirements and anticipated losses should be added to this. In general, the more rapid the fluid loss, the more rapid the replacement should be, especially when prerenal azotemia has been identified. Consequently, total fluid deficit may be replenished in 4 to 10 hours if acute dehydration is suspected. The choice of fluid type is usually guided by the acid-base and electrolytic abnormalities. Most avian patients are in metabolic acidosis with just dehydration or various illnesses, which makes Plasma-Lyte A 7.4 the fluid of choice. Alternatively, LRS may be used, but it is slightly hypotonic to avian plasma. Except for rapid rates, potassium may be supplemented to the fluids (see Table 8-8). Other additives may be added to the infusion depending on identified abnormalities (see Table 8-8). It should be noted that potassium artifacts are common on avian biochemistry analysis so one should exert caution before treating hypokalemia. For vomiting and loss of KCl and metabolic alkalosis, 0.9% NaCl is the fluid of choice. Calculations are just rough estimates and monitoring of the response to fluid therapy is important. Body weight gives a good estimate of the amount of rehydration. Because fluid deficits and requirements are difficult to estimate and clinical endpoints challenging to assess in birds, rehydration may continue for another 24 to 48 hours using a lower rate. Because of the low tolerance of avian patients for IV and IO catheters, the maintenance phase is typically performed subcutaneously and the catheters removed. Refer to the clinical example below.

Fluid rates under anesthesia ("surgical rate") are typically 10 mL/kg/h to treat the isoflurane-induced hypotension and fluid loss from to oxygen flow and evaporation through the surgical sites.

CLINICAL EXAMPLE

A 500-g African grey parrot is dehydrated at 10% with mild signs of hypovolemia and mild polyuria. Plasma pH is 7.2, HCO_3^- is 15 mmol/L, P_{CO_2} is 24 mm Hg, Na is 145 mmol/L, Cl is 114 mmol/L, K is

3.4 mmol/L, uric acid is 1000 µmol/L, urea is 1.1 mmol/L, PCV is 35, TS is 3.4 g/L, and glucose is 21 mmol/L.

- Fluid deficits = 500*0.1 = 50 mL given over 24 hours
- Anticipated loss ≈20% maintenance = 0.2*50*0.5 = 5 mL/day
- Maintenance = 50 mL/kg/day = 50*0.5 = 25 mL/day
- Fluid rate: 50 + 5 + 25 = 80 mL for the first 24 hours → 80/24 = 3.3 mL/h

The fluid rate may also be calculated quickly by using a multiple of the hourly maintenance rate of fluids, which is approximately equivalent to that calculated previously.

- Fluid rate at three times maintenance: 2*0.5*3 = 3 mL/h
- If the bird is urinating excessively, the rate should be reduced.

Because a mild metabolic acidosis is present, Plasma-Lyte A 7.4 is used as a replacement crystalloid fluid.

An initial fluid bolus of 10 mL/kg = 5 mL should be given over 5 to 10 minutes because signs of hypoperfusion and prerenal azotemia are present. This may be repeated once and pentastarch at 5 mL/kg = 2.5 mL added if necessary.

Adding dextrose to the fluids is generally not recommended as most avian patients are normoglycemic or slightly hyperglycemic, except if hypoglycemia is documented.

If an IV or IO fluid line cannot be maintained because of the bird's behavior, the daily total amount of fluid should be divided into six to eight boluses:

- 80/8 = 6.3 mL bolus over 5 to 10 minutes

When catheters cannot be placed because of specific situations, SC administration of the total daily amount should be performed:

- 80/4 = 20 mL in each inguinal area twice a day

The next day, the rate may be decreased to (50/2 + 5 + 25)/24 = 2.3 mL/h. As complete rehydration is not always achieved in 24 hours and clinical endpoints are harder to monitor in birds, ¼ to ½ of fluid deficits may still be given over another 24 hours.

If still acidemic, a bolus of bicarbonates at 0.3*0.5*10 = 1.5 mEq may be given over 15 to 20 minutes.

Acid-base, electrolytes, packed cell volume, total protein, urea, and uric acid should be rechecked within the next 48 hours to assess the results of the fluid therapy. Frequent rechecks of blood parameters are not always feasible in birds because of their small size.

REFERENCES

Acierno MJ, da Cunha A, Smith J, et al: Agreement between direct and indirect blood pressure measurements obtained from anesthetized Hispaniolan Amazon parrots (*Amazona ventralis*), *J Am Vet Med Assoc* 233(10):1587–1590, 2008.

Aguilar RF, Johnston GR, Callfos CJ, et al: Osseuous-venous and central circulatory transit times of technetium-99m pertechnetate in anesthetized raptors following intraosseous administration, *J Zoo Wildl Med* 24(4):488–497, 1993.

Beaufrère H, Acierno M, Mitchell M, et al: Plasma osmolality reference values in African grey parrots (*Psittacus erithacus erithacus*), Hispaniolan Amazon parrots (*Amazona ventralis*), and red-fronted macaws (*Ara rubrogenys*), *J Avian Med Surg* 25(2):91–96, 2011.

Braun EJ: Osmoregulatory systems of birds. In Scanes CG, editor: *Sturkie's avian physiology*, ed 6, San Diego, CA, 2015, Academic Press.

Cornelius ST, Klugman KP, Hattingh J: Effects of haemorrhage on colloid osmotic pressure in the pigeon, *Comp Biochem Physiol A Comp Physiol* 71(2):337–339, 1982.

Degernes LA, Harrison LD, Smith DW, et al: Autologous, homologous, and heterologous red blood cell transfusions in conures of the Genus *Aratinga*, *J Avian Med Surg* 13(1):10–14, 1999.

DiBartola SP, Wellman ML, Kohn CW: Applied physiology of body fluids in dogs and cats. In DiBartola SP, editor: *Fluid, electrolyte, and acid-base disorders in small animal practice*, ed 4, St Louis, MO, 2012, Elsevier.

Djojosugito AM, Folkow B, Kovách AG: The mechanisms behind the rapid blood volume restoration after hemorrhage in birds, *Acta Physiol Scand* 74(1):114–122, 2002.

Goldstein DL, Skadhauge E: Renal and extrarenal regulation of body fluid composition. In Whittow GC, editor: *Sturkie's avian physiology*, ed 5, San Diego, CA, 2000, Academic Press.

Jankowski G, Nevarez J: Evaluation of a pediatric blood filter for whole blood transfusions in domestic chickens (*Gallus gallus*), *J Avian Med Surg* 24(4):272–278, 2010.

Johnston MS, Davidowski LA, Sangeeta R: Precision of repeated, doppler-derived indirect blood pressure measurements in conscious psittacine birds, *J Avian Med Surg* 25(2):83–90, 2011.

Keil R, Gerstberger R, Simon E: Effects of changes in intravascular oncotic pressure on renal responses to volume loading in the saltwater-acclimated Pekin duck (*Anas platyrhynchos*), *J Comp Physiol [B]* 161(2):179–187, 1991.

Lamberski N, Daniel GB: Fluid dynamics of intraosseous fluid administration in birds, *J Zoo Wildl Med* 23(1):47–54, 1992.

Lichtenberger M: Shock and cardiopulmonary-cerebral resuscitation in small mammals and birds, *Vet Clin N Am Exot Anim Pract* 10(2):275–291, 2007.

Lichtenberger M, Orcutt C, DeBehnke D: Mortality and response to fluid resuscitation after acute blood loss in mallard ducks (*Anas platyrhinchos*), *Proceedings of the Annual Conference of the Association of Avian Veterinarians* 23:65–70, 2002.

Peltonen LM, Sankari S: Ott's protein osmotic pressure of serum and interstitial fluid in chickens (*Gallus gallus*): effect of age and gender, *J Exp Biol* 214(4):599–606, 2011.

Quesenberry KE, Hillyer EV: Supportive care and emergency therapy. In Ritchie BW, Harrison LR, Harrison GJ, editors: *Avian medicine: principles and applications*, Lake Worth, FL, 1994, Wingers Publishing.

Sandmeier P, Stauber EH, Wardrop KJ, et al: Survival of pigeon red blood cells after transfusion into selected raptors, *J Am Vet Med Assoc* 204(3):427–429, 1994.

Schnellbacher R, da Cunha A, Olson EE, et al: Arterial catheterization, interpretation, and treatment of arterial blood pressures and blood gases in birds, *J Exot Pet Med* 23(2):29–141, 2014.

Steinohrt LA: Avian fluid therapy, *J Avian Med Surg* 13(2):83–91, 1999.

Stringfield C: The California condor (*Gymnogyps californianus*) Veterinary Program: 1997-2000. In Miller RE, Fowler M, editors: *Fowler's zoo and wildlife animal medicine current therapy*, vol 7, St Louis, MO, 2012, Elsevier Saunders.

ORAL FLUID THERAPY

Janine Perlman

"If the gut works, use it" often applies to fluid therapy, as well as to food. For treatment of dehydration, if the patient's condition permits, the preferred route for fluid therapy may be *per os*. Oral administration can be less painful and stressful than other routes, and may be safer and at least as effective (Martin and Kollias, 1989; Hartling *et al.*, 2006). Contrary to earlier belief, oral fluid therapy may be indicated for mild, moderate, and severe dehydration (Atia and Buchman, 2009). If fluids are to be administered orally, the bird should be conscious; upright; or supported at a 45-degree angle and free of seizures, regurgitation, and gastrointestinal stasis (Quesenberry and Hillyer, 1994).

The cause and nature of the patient's fluid loss may have some bearing on the choice of therapeutic solution (Steinohrt, 1999), but in most cases oral fluids should be hypotonic (Hunt *et al.*, 1992; Duggan *et al.*, 2004) and should contain sodium and glucose (Schultz and Zalusky, 1964; Martin and Kollias, 1989).

Fluids should be warmed to 39° to 40°C using a water bath. Human medical products for oral rehydration in pediatric diarrhea such as Pedialyte, Equalyte, and Dioralyte are good choices. A mixture of one volume of a balanced IV salt solution (LRS, Normosol-R, Plasma-Lyte, Hartmann's solution, and Darrow's solution) with one volume of 5%

dextrose may also be used, with Darrow's solution notably offering potassium. LRS should not be used in cases of lactic acidosis.

Although systematic studies have not been performed with marine birds, hypotonic salt solutions may be contraindicated for patients with active salt glands (Frankfurter *et al.*, 2012). They should receive an isotonic balanced salt solution with added potassium (final concentration 35 mOsm) and 2.5% to 5% glucose.

Sports drinks vary widely and many formulations are contraindicated, so this class of product is not recommended.

A key to successful oral treatment of moderate to severe dehydration appears to be aggressive therapy administered over as long as 3 days. Usual frequency/volume guidelines for parenteral fluid administration can be considerably exceeded using the oral route, to the patient's benefit.

Gavage technique is described in the next section Tube Feeding and Nutritional Support. To minimize risk of aspiration, the initial fluid bolus should be 2% of body weight (BW), carefully instilled into the crop or stomach. This should be repeated every 15 minutes. In most cases, the volume can quickly be increased to 5% of body weight, with intervals increased to 90 minutes.

For mild dehydration, after dilute urine is produced, a 5% BW fluid gavage should be repeated at least one to two times. For moderate and severe dehydration, this instillation should continue every 90 minutes. After it is indicated that the patient is fully hydrated over a period of at least 6 hours, frequency can be significantly tapered, but high fluid intake may be advisable through 72 hours of treatment to ensure renal reperfusion and recovery (K. Robertson, personal communication).

Dehydrated altricial nestlings capable of begging will often accept fluids dropped or "fed" from a syringe, without the necessity of gavage. Some fledged and adult birds with dehydration behaviorally regress to begging, and, for initial dosing, may also voluntarily take considerable amounts of fluid offered by hand.

ADDITIONS TO ORAL FLUIDS

Water-soluble vitamins (*B-complex* and *C*) should be added to oral fluids for patients whose history of illness, injury, stress, or starvation is likely to have resulted in depletion of these micronutrients. Pyridoxine (vitamin B_6; Samour, 2013) and niacin can be toxic at high levels, and should be present at no more than 10 and 20 mg/L, respectively.

Glutamine may be helpful for restoration of gut function in cases of gastroenteritis or starvation (Bardhan, 2007). It should be dissolved in hot water and added at a concentration of 15 g/L.

Formulation of a rehydration solution for emaciated patients is detailed in the next section.

REFERENCES

Atia AN, Buchman AL: Oral rehydration solutions in non-cholera diarrhea: a review, *Am J Gastroenterol* 104:2596–2604, 2009.

Bardhan PK: Improving the ORS: Does glutamine have a role?, *J Health Popul Nutr* 25(3):263–266, 2007.

Duggan C, Fontaine O, Pierce NF, et al: Scientific rationale for a change in the composition of oral rehydration solution, *JAMA* 291:2628–2631, 2004.

Frankfurter G, Ziccardi MH, Massey JG: Effects of freshwater housing and fluid types on aquatic bird serum electrolyte concentrations, *J Zoo Wildl Med* 43(4):852–857, 2012.

Hartling L, Bellemare S, Wiebe N, et al: Oral versus intravenous rehydration for treating dehydration due to gastroenteritis in children, *Cochrane Database Syst Rev* (3):CD004390, 2006.

Hunt JB, Thillainayagam AV, Salim AF, et al: Water and solute absorption from a new hypotonic oral rehydration solution: evaluation in human and animal perfusion models, *Gut* 33:1652–1659, 1992.

Martin HD, Kollias GV: Evaluation of water deprivation and fluid therapy in pigeons, *J Zoo Wildl Med* 20(2):173–177, 1989.

Quesenberry KE, Hillyer EV: Supportive care and emergency therapy. In Ritchey BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Samour J: *Acute toxicity after administration of high doses of vitamin B6 (pyridoxine) in falcons*, Proceedings of the European College of Zoological Medicine Scientific Meeting, Wiesbaden, Germany, 2013.

Schultz SG, Zalusky R: Ion transport in isolated rabbit ileum. II. The interaction between active sodium and active sugar transport, *J Gen Physiol* 47:1043–1059, 1964.

Steinohrt LA: Avian fluid therapy, *J Avian Med Surg* 13(2):83–91, 1999.

TUBE FEEDING AND NUTRITIONAL SUPPORT

Janine Perlman

NUTRITIONAL REQUIREMENTS

Before feeding commences, normal body temperature and adequate hydration must be achieved. The nutritional requirements of patients needing enteral feeding are governed by the principles of feeding guilds and evolved diets described earlier (see Chapter 3). Birds' evolved responses to trauma and/or infection also result in increased requirements for animal-sourced nutrients.

Immune responses and tissue repair mobilized by animals in critical condition require large amounts of high-quality protein (Hoffer and Bistrian, 2012) and other micronutrients found only in fauna. Indeed, debilitated adult herbivorous birds able to self-feed have been observed to "revert" to eating solely animal-sourced foods like those they required during growth (Perlman, 2011; M. Gibson, personal communication), marking a complete transition from their usual feeding guild.

In addition to an increase in animal-sourced food, injured and ill birds require more calories. For healthy captive adults, daily caloric expenditure may be estimated using the equation $2.3 \times W^{0.65}$, where W is body weight in grams. Injured and/or ill patients are likely to be in a hypermetabolic state, in which energy requirements are increased in the range of 40% (Young *et al.*, 1985) to 70% (Frankenfield *et al.*, 1994). These guidelines are only approximate; the patient's actual needs must be assessed with body condition and prefeeding weight recorded every 24 hours, while overall condition, hydration status, and fecal quality are continually monitored.

The stresses and demands of illness, injury, captivity, and restraint increase requirements for a range of micronutrients. Ascorbic acid (vitamin C) is essential in stressful conditions (McKee and Harrison, 1995). Thiamine deficiency is common in critical-care patients. High-dose supplementation of multiple micronutrients (Manzanares *et al.*, 2012) including selenium (Alhazzani *et al.*, 2013) improve survival.

Starvation and emaciation present a somewhat different set of issues from those of trauma and illness. Reanalysis of previous studies suggests that "hypometabolism of starvation" is likely artifactual, and if the actual (emaciated) body weight is used, calculated energy requirements may also be close to actual (McCue, 2010).

Starvation causes gastrointestinal tract atrophy. Emaciated patients cannot mechanically or enzymatically break down ordinary particle sizes and amounts of food. Small food-particle size and small meal volumes are required until gut mass and function are restored.

Refeeding syndrome (RFS) presents an additional hazard in treating emaciation. RFS is manifested in hypophosphatemia, hypokalemia, and hypomagnesemia, as well as hypothiaminosis and other deficiencies. It appears to result from sudden large increases in insulin; thus,

BOX 8-1 Oral Rehydration/Therapeutic Solution for Emaciation

- 1 L Pedialyte or similar human pediatric oral rehydration solution
- 1.5 g KCl
- 1.2 g K_2HPO_4
- 1.3 g magnesium citrate
- 1000 mcg sodium selenite
- 500 mg thiamine
- 1 high-potency multivitamin/multimineral (human) supplement without iron
- 10 g L-glutamine
- 10 g L-arginine
- 1 capsule of multistrain (human) probiotic

This solution has inorganic ion (electrolyte) concentrations of 57 mOsm Na^+, 53 mOsm K^+, 55 mOsm Cl^-, 6.5 mOsm PO_4^{-3}, 5.5 mOsm SeO_3, and 3.5 mOsm Mg^{2+}, for a total of 187 mOsm.

When more than half of the bird's volumetric intake is whole (isotonic) food, this solution should be made with 1 L of water rather than Pedialyte. Mixed with water, the inorganic ion concentration is 87 mOsm.

All powders can be weighed, uniformly mixed, and stored dry and refrigerated. Add 27.5 g to 1 L of pediatric oral rehydration solution or water (see text). Powder mix shelf-life is limited only by the expiration date of the probiotic.

Adapted from Stanga Z, Brunner A, Leuenberger M, et al: *Eur J Clin Nutr* 62:687–694, 2008 and Boateng AA, Sriram K, Meguid MM, et al: *Nutrition* 26:156–167, 2010.

significant amounts of carbohydrates, especially for faunivores, are contraindicated. Raptors may be particularly vulnerable to RFS (Stevens, 1996).

Emaciated birds are typically significantly dehydrated. Compared with other routes of administration, oral rehydration reduces risk of fluid overload and hypernatremia. Specific additions to fluids (Box 8-1) are helpful for both gastrointestinal tract recovery and RFS prevention.

TUBE-FEEDING TECHNIQUE

The two goals of tube feeding are to assiduously avoid harm—including aspiration, trauma to bill, oropharynx and gastrointestinal tissues, and unnecessary stress—and to gavage sufficient food to minimize the frequency of the procedure. The first goal must be paramount.

Parrots and other species that can bite with sufficient force to injure a finger or puncture a rubber feeding tube are often gavaged using stainless-steel feeding tubes. Videos by Dr. Ross Perry demonstrating use of this equipment are available at https://www.youtube.com/watch?v=sKayj-s1Sxs and https://www.youtube.com/watch?v=WjCzaQvdTr4.

Practitioners who treat other taxa find that soft, rubber feeding tubes (human urinary catheters) have advantages compared with feeding needles:

- There is little danger of traumatizing esophagus or crop.
- Tube diameter can be significantly larger than the glottis, making accidental intubation of the airway impossible.
- Rather than laterally crossing the oropharynx, the tube can be both placed and inserted down the same (right) side (and in anatids and strigiforms also the left, because these taxa do not possess a crop), thus avoiding the possibility of covering the glottis.
- The tube's length permits access to the stomach when needed, even in long-necked species.
- The bird's body can be allowed to maintain a normal angle relative to the ground, and the neck does not need to be unnaturally extended. Postural normality is less stressful, allows easier swallowing to aid the tube's insertion, and decreases the risk of regurgitation when the bird is returned to its housing.

Rubber feeding tubes also can be used for parrots and other species with high bite pressure, if the beak is held open using a speculum or a tool designed for tube-feeding chelonians (see https://www.facebook.com/pages/The-Little-Jimmy/431659853622333).

The feeding tube should be measured along the bird's ventrum from bill tip to caudal end of the target organ, whether crop or proventriculus (which can be relatively accessible even in some birds possessing a crop; Ziswiler and Farner, 1972). It should be inserted with gentle even pressure, using the bird's swallowing to assist. It should not be forced. Birds are generally comfortable if the tube is properly placed. Sudden struggling may indicate pain or glottis occlusion.

Assuming that the bird has already tolerated oral fluid boluses of 5% body weight, this volume of food can also be gavaged. Some species can hold somewhat more. Emaciated patients are exceptions (see the following section).

A planned daily feeding schedule incorporates estimated caloric requirement, Kcal/cc of food, and volume per feeding. However, it is determined in detail by the bird's ability to hold and assimilate food, fecal characteristics, and changes in weight.

In cases of emaciation, it is imperative that meals are small and frequent. Initial feedings should be no more than 10% of calculated daily requirement, with total daily caloric intake increasing from approximately 60% of requirement to 100% over several days, as tolerated.

Food should be gavaged at body temperature. It is crucial to instill the meal slowly, while carefully watching for swallowing, which can indicate food welling up toward the oropharynx. Food reaching the vicinity of the glottis presents a high risk of aspiration. This phenomenon can virtually always be avoided with sufficient care.

ENTERAL NUTRITIONAL FORMULATIONS

Severely debilitated patients should be given food that matches their actual nutritional requirements as closely as possible. Injured and ill birds are in particular need of the nutrients found in their whole evolved foods (see Chapter 2), rather than highly processed, unnatural food derivatives and purified micronutrients comprising commercial critical-care formulations.

Commercial tube-feeding products are commonly soy based. This is far from optimal (Donoghue, 1994), in part because its digestibility by birds is poor (Parsons *et al.*, 1981; Elliston and Perlman, 2002; Choct *et al.*, 2010). Trials have shown that another common ingredient in commercial products, dried egg, is also indigestible by birds of multiple taxa (J. Perlman; M. Moyer, personal communication). Because of their low nutrient density and potential to increase insulin, grains should be included in tubed diets only if the patient is granivorous.

The protein in extruded diets (pellets and crumbles) for parrots is poorly assimilated even by healthy birds (Kalmar *et al.*, 2007), and this should not be used for assisted feeding of critical-care patients. Emaciated patients benefit from partially hydrolyzed, but not truly elemental, diets (Vazquez *et al.*, 1985; Poullain *et al.*, 1989; Chambon-Savanovitch *et al.*, 2001).

In contrast to nutrient-impoverished "elemental" products, a highly nutritious, partially hydrolyzed naturalistic diet can be prepared using pancreatin and/or pancrelipase (Fieker *et al.*, 2011). Depending on the bird's evolved need for fat, medium-chain triglycerides, which require less digestion than ordinary fats, may be added. In most patients, however, gastrointestinal digestive-enzyme activity is sufficient for feeding of whole food, if the particle size is small (i.e., puree).

Tube-feeding diets should be based on foods natural to the bird and what it has recently been eating. Bulk (chemically indigestible components) in the natural diet aids in normal bowel function and helps minimize diarrhea.

Raw and canned/jarred foods are more nutritious and more easily digested than dry extruded products. If the bird has recently been eating raw food, its use may result in less diarrhea than cooked products.

A food processor or blender assures ease and reliability of preparation. For example, raptors might be fed pureed neonate mice or other raw meat (preferably with bone). Cooked foods could include all-poultry jarred baby food or canned all-meat chicken, duck, or rabbit food for dogs and cats. Other types of formulations, including small-animal prescription diets, are not optimal.

Fresh or frozen seafood or canned whole fish (with attention to appropriate sodium levels) can be fed to piscivorous birds. Raw fish should be supplemented with the micronutrients described in Chapter 3.

Fresh mealworms, frozen/thawed crickets, and dried flies may be blended (with measured water and calcium) to make an insect puree. This can be stored frozen as ice cubes, and thawed and fed to insectivores, or added to other diets.

Pure nut butters with no additives are convenient for appropriate patients. A wide selection of nut butters is available made from raw or roasted nuts. If left sitting for a considerable period or centrifuged, the oil separates and can be removed (and replaced with water), permitting nonfat-nutrient enrichment.

Basic purees can be made, for example, from oily seeds (or nut butters) and fruit for granivorous-frugivorous parrots; grains, oily seeds, and greens for cockatiels; grains for columbids; and leafy greens (mainly dark lettuce) with modest amounts of grain, for wild geese in summer. Attention should be paid to assuring an abundance of high-quality protein within the parameters of the natural diet.

The increased protein requirements of critical-care omnivores and herbivores can be met by adding animal-sourced foods to the above-mentioned purees at a proportion approximately threefold higher than the bird's baseline intake (Hoffer and Bistrian, 2012). The choice of insect or vertebrate puree added to species-specific, blended whole natural foods should be based on the patient's natural and recent diet. All diets should be supplemented with micronutrients as described in Chapter 3.

Tube-fed mixes must comprise 25% dry matter and 75% water by weight, and have an energy density of 1.0 to 1.5 Kcal/cc. This dry matter : water ratio is crucial for maintenance of hydration, and ensures the correct caloric density for virtually all whole-food diets.

REFERENCES

Alhazzani W, Jacobi J, Sindi A, et al: The effect of selenium therapy on mortality in patients with sepsis syndrome: a systematic review and meta-analysis of randomized controlled trials, *Crit Care Med* 41(6):1555–1564, 2013.

Boateng AA, Sriram K, Meguid MM, et al: Refeeding syndrome: treatment considerations based on collective analysis of literature case reports, *Nutrition* 26:156–167, 2010.

Chambon-Savanovitch C, Felgines C, Walrand S, et al: A pancreatic extract-enriched diet improves the nutritional status of aged rats, *J Nutr* 131(3):813–819, 2001.

Choct M, Dersjant-Li Y, McLeish J, et al: Soy oligosaccharides and soluble non-starch polysaccharides: a review of digestion, nutritive and anti-nutritive effects in pigs and poultry, *Asian Aust J Anim Sci* 23(10):1386–1398, 2010.

Donoghue S: Nutritional support of hospitalised dogs and cats, *Aust Vet J* 71:332–336, 1994.

Elliston EP, Perlman J: Meeting the protein requirements of adult hummingbirds in captivity, *J Wildl Rehab* 25(2):14–19, 2002.

Fieker A, Philpott J, Armand M: Enzyme replacement therapy for pancreatic insufficiency: present and future, *Clin Exp Gastroenterol* (4):55–73, 2011.

Frankenfield DC, Wiles CE 3rd, Bagley S, Siegel JH: Relationships between resting and total energy expenditure in injured and septic patients, *Crit Care Med* 22(11):1796–1804, 1994. 18(5):398–403, 1994.

Hoffer LJ, Bistrian BR: Appropriate protein provision in critical illness: a systematic and narrative review, *Am J Clin Nutr* 96(3):591–600, 2012.

Kalmar ID, Werquin G, Janssens GPJ: Apparent nutrient digestibility and excreta quality in African grey parrots fed two pelleted diets based on coarsely or finely ground ingredients, *J Anim Physiol Anim Nutr* 91:210–216, 2007.

Manzanares W, Dhaliwal R, Jiang X, et al: Antioxidant micronutrients in the critically ill: a systematic review and meta-analysis, *Crit Care* 16:R66, 2012.

McCue MD: Starvation physiology: reviewing the different strategies animals use to survive a common challenge, *Comp Biochem Physiol A Mol Integr Physiol* 156(1):1–18, 2010.

McKee JS, Harrison PC: Effects of supplemental ascorbic acid on the performance of broiler chickens exposed to multiple concurrent stressors, *Poult Sci* 74(11):1772–1785, 1995.

Parsons CM, Potter LM, Brown RD Jr: True metabolizable energy and amino acid digestibility of dehulled soybean meal, *Poult Sci* 60(12):2687–2696, 1981.

Perlman J: Feeding birds for optimal health, *Association of Avian Veterinarians Clinical Forum*, 1–6, 2011.

Poullain M-G, Cezard J-P, Roger L, et al: Effect of whey proteins, their oligopeptide hydrolysates and free amino acid mixtures on growth and nitrogen retention in fed and starved rats, *J Parenter Enteral Nutr* 13(4):382–386, 1989.

Stanga Z, Brunner A, Leuenberger M, et al: Nutrition in clinical practice—the refeeding syndrome: illustrative cases and guidelines for prevention and treatment, *Eur J Clin Nutr* 62:687–694, 2008.

Stevens L: *Avian biochemistry and molecular biology*, Cambridge, UK, 1996, Cambridge University Press.

Vazquez JA, Morse EL, Adibi SA: Effect of starvation on amino acid and peptide transport and peptide hydrolysis in humans, *Am J Physiol* 249(5):G563–G566, 1985.

Young B, Ott L, Norton J, et al: Metabolic and nutritional sequelae in the non-steroid treated head injury patient, *Neurosurgery* 17:784–791, 1985.

Ziswiler V, Farner DS: Digestion and the digestive system. In Farner DS, King JR, Parkes KC, editors: *Avian Biol*, vol 2, New York, 1972, Academic Press.

INTENSIVE CARE UNITS

Hugues Beaufrère

Incubators or "intensive care unit" (ICU) cages are hospitalization cages with built-in heating, humidification, and medical oxygen systems. Several products are available on the veterinary market at different costs and with different specificities (Table 8-10). Of note is the Lyon Pro-Care therapy door, which transforms a regular hospitalization cage into an incubator with oxygen capabilities (Fig. 8-14). Some incubators can also be placed on carts for transportation in different areas of the hospital. Temperature and humidity are usually controlled with an electronic panel, but the authors recommend placing thermometer and hygrometer gauges inside to verify that the set temperature is correct (some incubators may become less accurate over time). Except for advanced and costly ICU cages, most provide oxygen using a flow meter with no oximetry measurements inside to adapt the environmental percentage of oxygen; thus, verifying the oxygen content with an oximeter initially at a different flow rate is advisable. In some incubators, small openings are available to pass fluid and oxygen lines. In addition, door-within-door designs allow catching birds or providing food and water without opening the main door and losing all the oxygen (Fig. 8-15). Some ICU cages also measure CO_2 or extract CO_2 gas produced by the animal. Nebulization units are

TABLE 8-10 Selected Incubators and Small Intensive Care Unit Cages Used in Birds

Product	Characteristics	Company Website
Pro-Care 27	Heat, oxygen, nebulization	www.lyonelectric.com
Pro-Care Therapy door	Heat, oxygen, nebulization	www.lyonelectric.com
AICU	Heat	www.lyonelectric.com
TLC-40 and TLC-50	Heat	www.brinsea.com
Vetario S50 and T40	Heat	www.vetario.com
Nursery hospital brooder	Heat	http://www.petiatric.com/browseproducts/Nursery-Hospital-2-Mechanical-Brooder.HTML
Rcom Brooder intensive care unit	Heat	www.r-com-hatcher.com
Snyder Intensive care unit	Heat, oxygen, CO_2 monitoring	www.snydermfg.com

FIGURE 8-14 Animal Intensive Care Unit by Lyon Electric. These incubators come in two sizes, are made of metal, and are stackable. A small opening on the back can accommodate an oxygen line.

available in some units, but the particle size is typically not small enough for birds' lower respiratory diseases but may help with overall humidification of the air. Inexpensive incubators may also be made using glass or Plexiglas aquaria placed on heating blankets (Fig. 8-16).

Incubators are typically used with any sick bird because they often benefit from supplemental heat and oxygen, at least during initial stabilization. Settings are approximately 30°C (86°F; corresponding to the thermoneutral zone of most avian species) and 70% humidity. Please see the following sections for further details. Incubators may not accommodate large birds such as large raptors and waterfowl, so infrared heat lamps or raising the ambient temperature in the aviary or hospital room is a more convenient way to thermally support these species.

FIGURE 8-15 Pro-Care 27 (Lyon Electric). This popular incubator allows the use of oxygen therapy, nebulization of large particles, and heat support. Its door-within-door design is convenient to catch small-to-medium-sized birds and providing food and water without losing oxygen.

FIGURE 8-16 Pro-Care Therapy door (Lyon Electric). This therapeutic door allows the transformation of a regular hospitalization cage into an ICU cage with oxygen and heat support.

THERMAL SUPPORT

Hugues Beaufrère

Birds have a higher core body temperature than mammals of approximately 38° to 39° C at rest and 40° to 42° C when active; Prinzinger *et al.*, 1991). Domestic poultry and pigeons have higher temperatures than most birds (approximately 41° to 42° C; Yahav, 2015). As mammals, birds regulate their temperature within a narrow range by various homeothermic mechanisms (Yahav, 2015). Some birds are heterothermic (may be able to increase or lower their temperature by a few degrees) as an adaptation to conserve water or reduce energy expenditure such as the torpor seen in some birds (e.g., hummingbirds and nightjars) or the controlled hypothermia seen in multiple passerine birds and mousebirds (Prinzinger *et al.*, 1991). Countless feather muscles regulate the thickness of the air layer maintained between the skin and the down and covert feathers. This thick feathery air layer is a formidable insulation cover. As such, hyperthermic birds tend to appear slim with wings apart from the body and hypothermic birds appear fluffed up with wings pressed against the body. Birds also shiver to produce heat (Dawson and Whittow, 2000; Yahav, 2015). On the other hand, observable mechanisms used by birds to cool themselves include panting, gular fluttering (e.g., pelicans, owls), cloacal evaporation (e.g., Inca dove), heat transfer at bare skin areas, and urohidrosis (e.g., New World vultures) among others (Dawson and Whittow, 2000; Yahav, 2015).

Cloacal temperature is not routinely performed during physical examination because the stress of transport, examination, and restraint may increase body temperature and confound its interpretation in relation to disease states. Contrary to some beliefs, birds experience fever with an increased temperature as seen in mammals (D'Alecy and Kluger, 1975; Maloney and Gray, 1998). In addition, a low cloacal temperature is always meaningful and indicative of hypothermia, which can be associated with hypotensive shock and hypovolemia.

Because birds have a high metabolism, providing heat support as part of the overall supportive care has the potential to greatly reduce energy expenditure (homeothermy is very energy-consuming) and help stabilize avian patients, which in turn may reduce oxygen consumption and daily caloric needs (translating into a lower amount of assisted feeding and decreased weight loss in anorexic patients). Also, most companion bird species are tropical species adapted to warm and humid environments; thus, these conditions should be replicated in sick patients. Marked feather loss is also common in pet birds because of feather-damaging behavior, which implies greater heat loss. Because of their high body area-to-volume ratio, birds tend to lose heat quickly, especially under general anesthesia with the added cooling effect of the oxygen flow, aseptic preparation of the skin for surgery (alcohol should be avoided in small birds), vasodilatory effects of anesthetic drugs, and inhibition of thermoregulatory mechanisms. On the other hand, birds are easier to warm up. Some arctic species may show the opposite trend and become hyperthermic upon restraint, anesthesia, or when heat support is provided. In addition, birds restrained in towels may become hyperthermic in 15 minutes (Greenacre and Lusby, 2004). For periods longer than 15 minutes, sedation is recommended and may alleviate iatrogenic hyperthermia (Mans *et al.*, 2012).

A variety of warming solutions are available and each may transmit heat to patients through one of three mechanisms including conduction (e.g., warming blankets), convection (e.g., Bair Hugger), and radiation (e.g., heating bulbs).

Conscious sick birds must be placed in incubators or heated areas. A variety of "intensive care units" are available with humidity and temperature control (see previous section). Less expensive alternatives

FIGURE 8-17 Heat bulbs that could be used in thermal support. From left to right are a ceramic bulb, a light-emitting basking light, and an infrared heat bulb. The latter is particularly useful for large birds (e.g., swans) that cannot be placed in incubators.

may be used such as ceramic and light-emitting heat lamps (Fig. 8-17). For birds (e.g., swans) that are too large to be placed in incubators, an infrared heat lamp works best. The temperature should ideally be set up within the thermoneutral zone of the species. This zone corresponds to the temperature where thermal balance is reached and no energy is spent for thermogenesis or cooling, birds are at their basal metabolism, and hyperthermia does not occur (Dawson and Whittow, 2000; Yahav, 2015). The thermal neutrality zone is approximately 25° to 30° C (77° to 85° F) for most birds. It may be higher in tropical species and passerines and lower in arctic species and poultry. Above this zone, high temperature may have negative effects by exacerbating fever and evaporative water loss in already dehydrated birds. All feeding formulas and fluids given to birds should be warmed up to avian body temperature before administration. Intravenous and intraosseous fluid should also be kept warm by placing a warmed oat bag on the syringe or using an in-line electrical fluid warmer. Fluid pumps may also be placed inside incubators to keep fluids warm in cooperative patients. On the other hand, some heterothermic species, such as hummingbirds, decrease their body temperature at night to conserve energy (Prinzinger *et al.*, 1991; Yahav, 2015). Preventing this adaptive mechanism by providing constant high ambient temperature may lead to greater daily energy expenditures in these species. If the patients are severely hypothermic, they should actively be warmed up using heating blankets, hair dryers, or forced-air warmer systems (e.g., Bair Hugger).

Under anesthesia, hypothermia may be prevented by a variety of warming devices including an electrical warming blanket, circulating hot water blanket, warming polymeric fabric blanket (e.g., HotDog, Augustine Biomedical, Eden Prairie, MN), forced-air warmer system (e.g., Bair Hugger, 3M, St. Paul, MN), self-manufactured oat or rice bags, and warm fluid bags or hot water-containing gloves (Figs. 8-18 to 8-22). A study in Amazon parrots showed that the Bair Hugger was more effective than a circulating water blanket and infrared heat emitter at preventing heat loss under anesthesia (Rembert *et al.*, 2001). Another study in pigeons demonstrated that the HotDog system was better at preventing heat loss than the Bair Hugger device (van Zeeland *et al.*, 2012). Incoming oxygen and anesthetic gases can also be warmed by expiratory gas in a tube-within-tube design of anesthetic circuits or other systems. Also, care should be taken to pluck the minimum area needed for surgery. The use of alcohol-based antiseptics should be avoided in small birds, plastic draping may be used to more efficiently conserve heat (VSP surgical drapes; Veterinary Specialty Products, Shawnee, KS), and surgical time should be kept to a minimum.

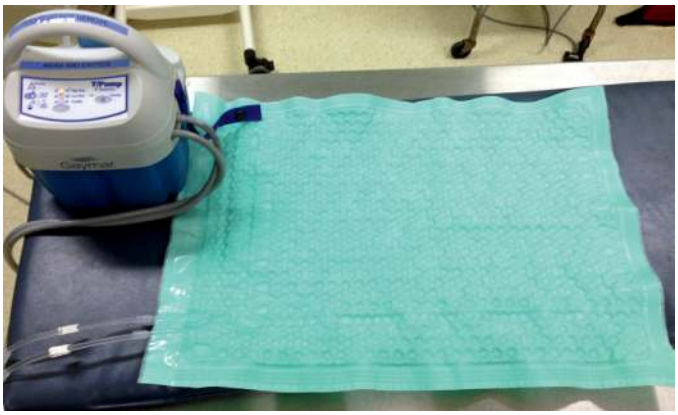


FIGURE 8-18 Recirculating hot water blanket.

FIGURE 8-19 HotDog warming polymeric fabric blanket.

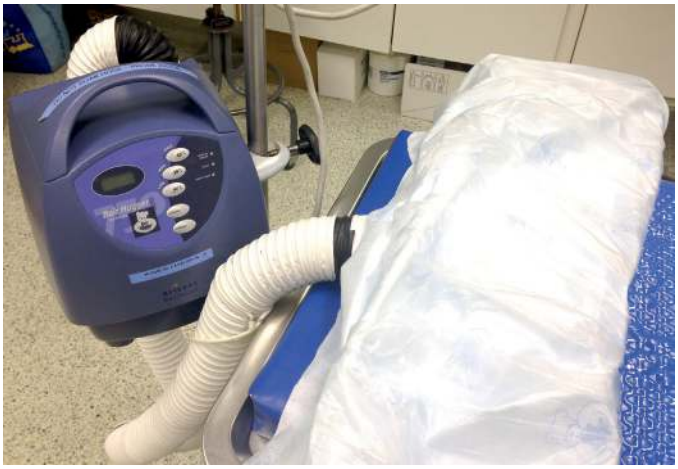


FIGURE 8-20 Bair Hugger forced-air warming device.

FIGURE 8-21 "Oat bags" may be warmed in a microwave and are very effective and cost-effective at providing heat support.

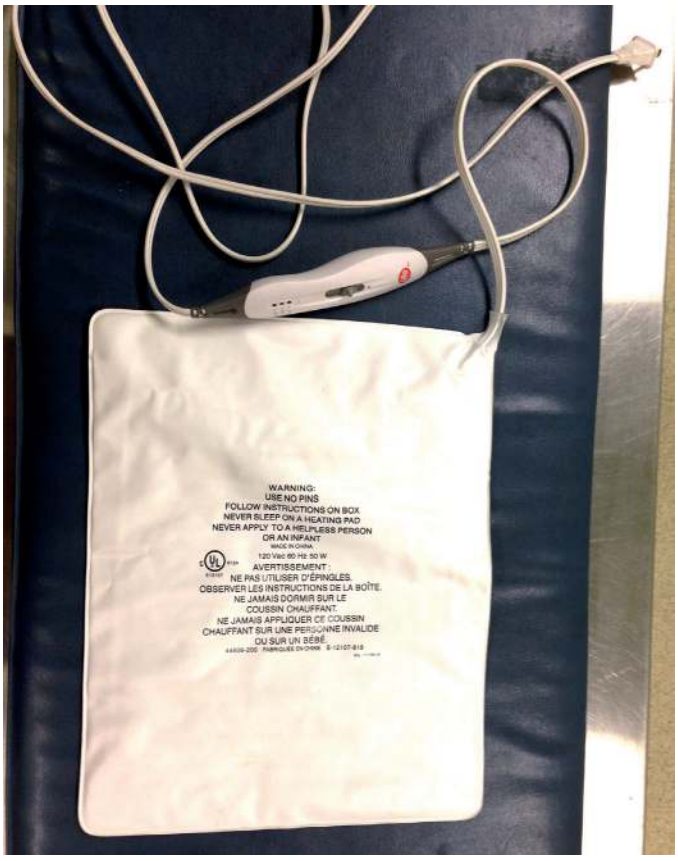


FIGURE 8-22 Electric warming blanket.

REFERENCES

D'Alecy LG, Kluger MJ: Avian febrile response, *J Physiol* 253(1):223–232, 1975.

Dawson WR, Whittow GC: Regulation of body temperature. In Whittow GC, editor: *Sturkie's avian physiology*, ed 5, San Diego, CA, 2000, Academic Press.

Greenacre CB, Lusby AL: Physiologic responses of Amazon parrots (*Amazona* species) to manual restraint, *J Avian Med Surg* 18(1):19–22, 2004.

Maloney SK, Gray DA: Characteristics of the febrile response in Pekin Ducks, *J Comp Physiol [B]* 168(3):177–182, 1998.

Mans C, Sanchez-Migallon Guzman D, Lahner LL, et al: Sedation and physiologic response to manual restraint after intranasal administration of midazolam in Hispaniolan Amazon parrots (*Amazona ventralis*), *J Avian Med Surg* 26(3):130–139, 2012.

Prinzinger R, Preßma A, Schleuche E: Body temperature in birds, *Comp Biochem Physiol A Physiol* 99(4):499–506, 1991.

Rembert MS, Smith JA, Hosgood G, et al: Comparison of traditional thermal support devices with the forced-air warmer system in anesthetized Hispaniolan Amazon parrots (*Amazona ventralis*), *J Avian Med Surg* 15(3):187–193, 2001.

van Zeeland YRA, Cardona T, Schoemaker NJ: Maintenance of core body temperature in anaesthetised pigeons (*Columba livia* domestica): a comparison of two thermal devices, *Vet J* 194(3):429–432, 2012.

Yahav S: Regulation of body temperature: strategies and mechanisms. In Scanes CG, editor: *Sturkie's avian physiology*, ed 6, San Diego, CA, 2015, Academic Press.

OXYGEN THERAPY IN BIRDS

Hugues Beaufrère

The goal of oxygen supplementation is to increase the oxygen concentration of inspired air (fraction of inspired O_2: Fi_{O_2}) to improve blood oxygenation and increase tissue delivery of O_2. General hypoxia may be caused by anoxic hypoxia (low Fi_{O_2}, hypoventilation, diffusion impairment, and ventilation/perfusion mismatching), anemic hypoxia (anemia, methemoglobinemia, and CO poisoning), stagnant hypoxia (low blood flow from cardiac failure or hemorrhage), and histiocytic hypoxia (cyanide poisoning; Manning, 2002). Common indications of oxygen therapy include severe anemia, hemodynamic compromise, and hypoxemia (decreased blood oxygen concentration) from pulmonary and obstructive airway diseases (Hopper, 2010).

Oxygen therapy in avian patients is typically implemented using an oxygen cage because it is low stress and noninvasive. The author strongly encourages veterinarians to measure the Fi_{O_2}, which can be achieved in oxygen cages using an oxygen sensor, to verify its measurement before administering supplemental oxygen. For instance, Fi_{O_2} measured by the authors is 60% in the Lyon Pro-Care Large Exotic Animal Care Unit and approximately 40% in the Lyon Small Animal Care Unit with oxygen at 6 to 8 L/min. The former has a door-within-a-door system, which allows manipulation of the animal without massive loss of O_2 from the cage. An Fi_{O_2} of 25% to 45% may be reached using flow-by oxygen and 35% to 60% using facemask (Manning, 2002; Hopper, 2010). It is ideal to monitor the Sp_{O_2} or Pa_{O_2} during oxygen supplementation, but this is rarely practical in most birds.

Response to therapy should also be used for monitoring and to titrate the oxygen flow. The response to oxygen therapy may be poor depending on the disease. For instance, a poor to fair response may be seen in diseases producing a low-ventilation/perfusion mismatch (perfusion predominates with decreased supply in O_2 at the exchange surface) such as pulmonary edema, pneumonia, asthma, pulmonary neoplasia, and atelectasis. In extreme cases of these diseases (severe pneumonia), oxygen therapy may not be efficacious because blood makes no contact with ventilated lungs. Response is good in diseases with a high-ventilation/perfusion mismatch (low perfusion in normally ventilated lungs) such as pulmonary thromboembolism (Manning, 2002). Improving cardiac function and fluid resuscitation are best to correct stagnant hypoxemia. Although the degree of response to oxygen therapy may be variable, regardless of the underlying conditions, any patient with acute respiratory distress and signs of hypoxia (cyanosis, dyspnea, tachypnea, and open-mouth breathing) may benefit from supplemental oxygen.

Oxygen therapy seems extremely beneficial to birds that have higher metabolic demand for oxygen and may benefit more from increased Fi_{O_2} because of their more efficient gas exchange surface.

The most common complication associated with oxygen therapy is oxygen toxicity because oxygen is a potent oxidizing agent. The degree of toxicity is related to the level of oxygen and duration and the lung is the most vulnerable organ to oxygen radicals. General guidelines recommend not to supplement oxygen at 60% to 100% for more than 24 to 48 hours in mammals (Manning, 2002; Hopper, 2010). Depletion of endogenous antioxidant levels may also promote oxygen toxicity at lower oxygen percentages. A study demonstrated oxygen toxicity in canaries and budgerigars on 68% to 100% after 3 to 8 days (Stauber, 1991). In another experiment, budgerigars were found to have pulmonary lesions 3 hours after exposure to 100% oxygen (Jaensch *et al.*, 2001).

REFERENCES

Hopper K: Oxygen therapy. In Ettinger S, Feldman E, editors: *Textbook of veterinary internal medicine*, ed 7, St Louis, MO, 2010, Saunders Elsevier.

Jaensch S, Cullen L, Raidal SR: Normobaric hyperoxic stress in budgerigars: enzymic antioxidants and lipid peroxidation, *Comp Biochem Physiol C Toxicol Pharmacol* 128(2):173–180, 2001.

Manning AM: Oxygen therapy and toxicity, *Vet Clin North Am Small Anim Pract* 32(5):1005–1020, 2002.

Stauber E: Effects of increased concentration of inspired oxygen, *Proc Eur Assoc Avian Vet* 105–114, 1991.

METABOLIC DRUG SCALING

Thomas A. Bailey

Medication and vaccination of nondomestic birds is often accomplished by extrapolating dosage regimens from other species, if such data exist, but the effectiveness and safety of this method is not always considered satisfactory (Dorrestein, 1991). Anatomic and physiologic differences account for some of the difficulties of extrapolating drug dosages for birds from dosages prescribed for mammals (Dorrestein, 1991). As knowledge of drug disposition and metabolism expands, it is becoming clear that large differences exist in dosage, administration interval, and organ distribution, not only between birds and mammals but among different avian species (Dorrestein, 1993; Baggot, 1995). Ideally, drug administration should be based on knowledge of the pharmacokinetics of that drug in the species to which it is being administered. Although some pharmacokinetic studies have been performed in birds, for the majority of drugs used in everyday clinical practice, no studies have been performed. Even in the future, it is unrealistic to expect pharmacokinetic data derived from studies in birds to be available for more than a handful of drugs, so it is often necessary to make estimates of appropriate drug doses and dose frequencies. Beyond the pharmacologic differences among species, the diversity of birds also ranges in size from the tiny bee hummingbird (*Mellisuga helenae*) weighing 2 g to large ratites, such as ostriches (*Struthio camelus*), weighing 145 kg.

Allometric scaling is a method for examining the structural and functional consequences of changes in size or scale among otherwise

similar organisms (Schmidt-Nielson, 1984). It has been found that many physiologic variables can be related to bodyweight (W) by mathematical relationships; for example, rates of production and consumption vary with $W^{0.5}$ and the timespans of biological processes vary with $W^{0.25}$ (Kirkwood, 1983). The use of allometric principles to scale physiologic parameters between animals of various sizes is now well established and is widely used in estimating the energy requirements and food needs of captive and free-living wildlife (Kirkwood and Bennett, 1992; Kirkwood, 1996). The uptake, distribution, and elimination of drugs administered to animals all involve physiologic processes that can be scaled allometrically (Sedgwick, 1993). Thus, when using novel drugs for which no other avian dose is known, it is possible to use allometric equations to assist in calculating doses. The reader is recommended to consult the literature to gain a deeper insight into the theories, applications, and pitfalls of using metabolic scaling to extrapolate doses between different species (Kirkwood and Bennett, 1992; Pokras *et al.*, 1993; Sedgwick, 1993; Hunter and Isaza, 2008; Hunter 2010; Toutain *et al.*, 2010). Caution is recommended and Hunter and Isaza (2008) list examples of pharmaceutical agents that are good or poor candidates for allometric scaling. Gentamicin is an example of a drug that is scalable (Kirkwood and Merriam, 1990). Many drugs are not suitable to be allometrically scaled because there are well-documented differences in hepatic drug metabolism between species. These are differences in phase I and phase II metabolism, cytochrome P450 isoenzyme expression, and other species-specific differences in hepatic physiology that make extrapolating pharmacokinetic parameters for metabolized drugs challenging (Hunter and Isaza, 2008). Only a few studies have been performed assessing the drug-metabolizing enzyme systems of birds, including one on the houbara bustard (Bailey *et al.*, 1998a), and there is a need for additional investigations in this field.

Although allometric equations can appear to be intimidating, it is important to be able to use them to extrapolate drug doses from one species to another. Once one is familiar with the worksheet approach described by Sedgwick (1993), it is relatively straightforward to write the calculations in one of the commonly used spreadsheet or database programs and doses can then be rapidly calculated in the clinic. Such information can provide a starting point for a dose for an unfamiliar medicine and is better than guessing. Although the size range of birds is not as vast as other groups of animals, when we consider the size difference in the Otididae between a newly hatched red-crested bustard chick (*Eupodotis ruficrista*) weighing 30 g and a large, adult kori bustard (*Ardeotis kori*) weighing 15 kg we are still dealing with a 500-fold difference. Insufficient attention has been paid to selecting doses to treat chicks and juvenile birds that are a small fraction of the weight of adult birds, and greater attention should be paid to using metabolic scaling to guide the calculation of size-appropriate doses and dose intervals. Papers by Sedgwick (1993) and Pokras *et al.* (1993) have been used in the following two examples.

The specific minimum energy cost (SMEC) for any animal can be calculated as follows:

$$\text{SMEC} = K\,(W_{kg}^{0.75}/W_{kg}) = K\,(W_{kg}^{-0.25})$$

where K = energy constant (Table 8-11).

The SMEC dose for a drug is calculated by dividing the dose rate (mg/kg) of the control animal by the control's SMEC (Boxes 8-2 and 8-3). Treatment frequency is the number of times a drug dose is administered to a patient in a day (24 hours) when a treatment regimen is a multiple of one. The SMEC frequency is the treatments frequency of a control animal divided by the SMEC (Boxes 8-2 and 8-3). Two worksheet examples are provided. The first shows how to extrapolate an established treatment regimen for azithromycin from humans (control species) in which the dose rate has been established after pharmacologic trials for different size and age classes of bustards. The second shows how to extrapolate an established treatment regimen for enrofloxacin from adult houbara bustards (control species) in which the dose rate has been established after pharmacologic trials (Bailey *et al.*, 1998b) for different size and age classes of bustards. The best data for selection of a control animal from which to extrapolate treatment regimens come from pharmacokinetic studies performed in the control species. Of the two examples, clearly we would have more confidence in the values of enrofloxacin for our three different types of bustard, which were extrapolated from pharmacokinetic investigations in houbara bustards (Bailey *et al.*, 1998b), than the values for azithromycin that were extrapolated from humans to bustards. Azithromycin is used at a dose of 43 mg/kg every 24 hours in psittacines (Rupiper *et al.*, 2000), which is not so wildly different from the results of the allometric calculations. Even with the limitations of allometric scaling, I think that the results of these examples help demonstrate that drug doses should be interpreted with caution and show that a drug dose used safely in adult birds may have a different effect on a chick.

TABLE 8-11 Hainsworth's Energy Groups

Group	Constant (K)	Mean Core Temperature (°C)
Passerine bird	129	42
Nonpasserine bird	78	40
Placental mammal	70	37

From Sedgwick, 1993.

BOX 8-2 Example 1: SMEC Dose and SMEC Frequency Scaling Worksheet Extrapolating Standard Dosage Regimens for Azithromycin from Humans to Three Different Age and Size Classes of Bustard

Control Species SMEC Calculations:

The dose rate for azithromycin in a human (bodyweight 70 kg) is 500 mg (7 mg/kg) every 24 hours

Control species: human (weight W_{kg} 70 kg)

Dose rate is 500 mg (7 mg/kg) every 24 hours

SMEC = $K\,(W_{kg}^{-0.75}/W_{kg}) = K\,(W_{kg}^{-0.25}) = 24.2$

SMEC dose is the dose rate divided by SMEC = 7/24.2 = 0.3

SMEC dose = 0.3

Frequency (number of treatment intervals per 24 hours) = 24/24 = 1

SMEC frequency is the frequency divided by SMEC = 1/24.2 = 0.04

SMEC frequency = 0.04

Species, Age and Weight	Buff-Crested Bustard Chick (0.05 kg)	Adult Male Houbara Bustard (1.5 kg)	Adult Male Kori Bustard (15 kg)
SMEC	$78(0.05^{-0.25}) = 165$	$78(1.5^{-0.25}) = 70.5$	$78(15^{-0.25}) = 40$
Dose rate calculations	$165 \times 0.3 = 49.5$	$70.5 \times 0.3 = 21.2$	$40 \times 0.3 = 12$
Dose frequency calculations	$165 \times 0.04 = 6.6$	$70.5 \times 0.04 = 2.8$	$40 \times 0.04 = 1.6$
$40 \times 0.3 =$ Regimen	50 mg every 4 h	21 mg every 8 h	12 mg every 12 h

BOX 8-3 Example 2: SMEC Dose and SMEC Frequency Scaling Worksheet Extrapolating Standard Dosage Regimens for Enrofloxacin Established for Adult Houbara Bustards to Three Different Age and Size Classes of Bustard

Control Species SMEC Calculations

The dose rate for enrofloxacin in an adult houbara bustard (bodyweight 1.5 kg) is 10 mg/kg every 12 hours (Bailey *et al.*, 1998b)
Control species: houbara bustard (weight W_{kg} 1.5 kg)
Dose rate is 10 mg/kg every 12 hours
SMEC = $K(W_{kg}^{-0.75}/W_{kg}) = K(W_{kg}^{-0.25}) = 70.5$
SMEC dose is the dose rate divided by SMEC = 10/70.5 = 0.15
SMEC dose = 0.15
Frequency (number of treatment intervals per 24 hours) = 24/12 = 2
SMEC frequency is the frequency divided by SMEC = 2/70.5 = 0.03
SMEC frequency = 0.03

Species, Age and Weight	Buff-Crested Bustard Chick (0.05 kg)	Adult Female Buff-Crested Bustard (0.5 kg)	Adult Male Kori Bustard (15 kg)
SMEC	$78(0.05^{-0.25}) = 165$	$78(0.5^{-0.25}) = 93$	$78(15^{-0.25}) = 40$
Dose rate calculations	$165 \times 0.15 = 25$	$93 \times 0.15 = 14$	$40 \times 0.15 = 6$
Dose frequency calculations	$165 \times 0.03 = 4.9$	$93 \times 0.03 = 2.8$	$40 \times 0.03 = 1.2$
$40 \times 0.3 =$ Regimen	25 mg every 6 h	14 mg every 8 h	6 mg every 24 h

REFERENCES

Baggot JD: Pharmacokinetics: disposition and fate of drugs in the body. In Adams HR, editor: *Veterinary pharmacology and therapeutics*, ed 7, Ames, IA, 1995, Iowa State University Press.

Bailey TA, John A, Mensah-Brown EP, et al: Drug metabolizing enzyme systems in the houbara bustard (*Chlamydotis undulata macqueenii*), *Comp Biochem Physiol C Pharmacol Toxicol Endocrinol* 120(3):365–372, 1998a.

Bailey TA, Sheen RS, Silvanose C, et al: Clinical pharmacology and pharmacokinetics of enrofloxacin after intravenous, intramuscular and oral administration in houbara bustard (*Chlamydotis undulata macqueenii*), *J Vet Pharmacol Ther* 21:288–297, 1998b.

Dorrestein GM: The pharmacokinetics of avian therapeutics, *Vet Clin North Am Small Anim Pract* 21:1241–1263, 1991.

Dorrestein GM: Antimicrobial drug use in pet birds. In Prescott JF, Baggot JD, editors: *Antimicrobial therapy in veterinary medicine*, Ames, IA, 1993, Iowa State University Press.

Hunter RP: Interspecies allometric scaling, *Handb Exp Pharmacol* 199:139–157, 2010.

Hunter RP, Isaza R: Concepts and issues with interspecies scaling in zoological pharmacology, *J Zoo Wildl Med* 39(4):517–526, 2008.

Kirkwood J: Nutrition of captive and free-living wild animals. In Kelly NC, Wills JM, editors: *Manual of companion animal feeding*, Cheltenham, UK, 1996, British Small Animal Veterinary Association.

Kirkwood JK: Influence of body size in animals on health and disease, *Vet Rec* 113:287–290, 1983.

Kirkwood JK, Bennett PM: Approaches and limitations to the prediction of energy requirements in wild animal husbandry and veterinary care, *Proc Nutr Soc* 51:117–124, 1992.

Kirkwood JK, Merriam J: Variation in plasma half-life of gentamicin between species in relation to bodyweight and taxonomy, *Res Vet Sci* 49(2):160–165, 1990.

Pokras MA, Karas AM, Kirkwood JK, et al: An introduction to allometric scaling and its uses in raptor medicine. In Redig PT, Cooper JE, Remple JD, Hunter DB, editors: *Raptor biomedicine*, Minneapolis, MN, 1993, University of Minnesota Press.

Rupiper DJ, Carpenter JW, Mashima TY: Formulary. In Olsen GH, Orosz SE, editors: *Manual of avian medicine*, St Louis, MO, 2000, Mosby.

Schmidt-Nielson K: *Scaling: why is animal size so important?*, New York, 1984, Cambridge University Press.

Sedgwick CJ: Allometric scaling and emergency care: the importance of body size. In Fowler ME, editor: *Zoo and wildlife medicine: current therapy 3*, Philadelphia, PA, 1993, WB Saunders.

Toutain PL, Ferran A, Bousquet-Mélou A: Species differences in pharmacokinetics and pharmacodynamics, *Handb Exp Pharmacol* 199:19–48, 2010.

BANDAGES AND DRESSINGS

Judith C. Howlett

Bandage application is a skill in which veterinarians and veterinary technicians should become proficient. A bandage should be comfortable for the patient, look professional, and serve the purpose for which it is designed. Several principles must be followed to avoid complications. For wound management, bandages should be sufficiently padded, applied evenly and snugly and in three layers, and placed to avoid the newly formed granulation tissue or epithelium. There are many different bandages and dressings available from simple to sophisticated, some of which are listed in the following sections (Figs. 8-23 to 8-25).

FUNCTIONS OF DRESSINGS AND BANDAGES

Protection

- Protect wounds after surgery and prevent desiccation
- Provide thermal insulation
- Prevent interference with wounds from beak and claw
- Protect wounds from pathogenic organisms

Pressure

- As a first-aid measure to avert hemorrhage and edema and reduce dead space
- Reduce swelling after trauma or surgery

Support

- As a first-aid measure to minimize further damage from a simple fracture or luxation
- Immobilize the affected part and therefore relieve pain after surgery or trauma
- Maintain intravenous and intraosseous catheters
- Redistribute weight loading for wounds on weight-bearing surfaces (e.g., pododermatitis)

Absorption, Moist Environment, and Holding in Place

- Dressings absorb exudate and help debride the wound surface.
- Dressings help to maintain a moist environment to encourage granulation and reepithelialization as quickly as possible.
- Correct bandaging keeps a dressing in place.

Comfort

- Provide comfort for the patient

FIGURE 8-23 Dressing and bandage applied to the wing of a houbara bustard (*Chlamydotis undulata*) after pinioning was performed.

FIGURE 8-24 The same bird as in Fig. 8-23, showing the correct way to "anchor" the bandage to the wing to stop it from slipping. The author currently uses a cohesive bandage, such as Vetrap instead of Sleek, as the preferred choice for the tertiary layer.

OTHER CHARACTERISTICS OF AN IDEAL DRESSING

- Low-adherence or nonadherent (except for wet-to-dry bandages)
- Requiring infrequent changing
- Free from particulate contaminants
- Safe to use (nontoxic, nonsensitizing, and nonallergenic)
- Comfortable and moldable
- Good absorption characteristics (exuding wounds)
- Impermeable to microorganisms
- Sterile
- Available in a suitable range of sizes/forms
- Cost-effective

PROCESS OF SELECTION

The correct dressing for wound management depends not only on the type of wound but also on the stage of the healing process. When selecting a dressing good knowledge of wound healing processes is essential in addition to an awareness of the properties of the dressing available. Successful wound management requires these two factors to be considered together. Many of the principles and techniques of

FIGURE 8-25 A simple bandage applied to the wing of a saker falcon after surgery to immobilize a fractured radius. In this case, a single intramedullary pin was fixed together with the bandage. This bandage consists of two rings made of 1-inch masking used to hold the primary feathers in place and to stop the falcon from opening its wing. This is the bandage of choice used on the wings of falcons after surgery at our hospital to reduce and immobilize fractures of the ulna and/or the radius.

wound management and bandaging in mammals apply to birds, although of course anatomic differences have to be considered. In most birds, the majority of wounds exhibit very little to no exudation and often appear dry. Wound dressing materials for humans are being continually developed with increased knowledge of wound-healing processes. The new dressings keep wounds moist and prevent scab formation, which significantly increases the rate of reepithelialization. Adaptation of these products in avian medicine has significantly improved wound management and healing.

After initial assessment of wound type and appropriate stabilization (if necessary) of the bird, it is essential to perform wound lavage to debride both visible and microscopic debris before applying any dressings or ointments. In human medicine, normal saline or even plain water is the preferred fluid for wound irrigation because it is not toxic to the tissues. Use of antibiotics in wound flushing is controversial and strong solutions of antiseptics such as chlorhexidine can be toxic to healing tissues, an anachronism to be avoided. However, 0.05% chlorhexidine aqueous solution is minimally toxic for tissues and provides effective antisepsis. Some dressings are marketed specifically as wound-cleaning agents. Studies have shown that wound cleaning does not actually remove bacteria, it merely redistributes them. After wound lavage, debridement of nonviable tissue can be performed.

Honey, an ancient remedy for the treatment of infected wounds, has recently been "rediscovered" by the medical profession. After 10 years' research in Australian, New Zealand, and UK hospitals in 2006, Medihoney launched two new honey-based wound care products. There are a number of published reports describing the effectiveness of honey in

reducing infection from wounds, with no adverse effects, and there is also evidence to suggest that honey may actively promote healing. Honey is particularly effective in infected wounds with no exudation.

It is good practice to use a standardized wound-assessment tool to ensure that valid, reliable, and consistent information is documented.

WOUND ASSESSMENT

Wound assessment should include:

- Location of wound
- Cause of wound
- Form
- Etiology
- Tissue type
  - Necrotic (usually black, covered with devitalized epidermis)
  - Sloughy (contains a layer of viscous fluid with dead cells—yellow in color)
  - Clinically infected/malodorous (yellow/green in color)
  - Granulating (highly vascular granulation tissue—red in appearance)
  - Epithelializing (shows evidence of pink wound margin)
- Size

MANAGEMENT OBJECTIVES OF WOUND TREATMENT

- *Dry necrotic wound*. Debride and provide a moist wound environment.
- *Sloughy wound*. Cleanse, debride, absorb, fill in dead space, and provide a moist wound environment.
- *Highly exuding wound*. Control large amounts of exudate to help prevent maceration while maintaining a moist wound environment.
- *Cavity wound*. Protect, hydrate, and fill in dead space.
- *Granulating/epithelializing wound*. Protect, fill in dead space, and provide a moist wound environment.
- *Skin tear*. Protect, fixate, absorb, and provide a moist wound environment.
- *Surgical*. Protect, absorb, and provide a moist wound environment.

The three main layers of bandaging and dressings are shown in Table 8-12.

- *Primary or dressing layer*: Dressing in contact with the wound that should be sterile; stay in place against the wound with patient movement; and provide a moist wound climate and assist debridement, encourage granulation, and reepithelialization.
- *Secondary layer*: For absorbing fluids and wound exudates, padding the wound from trauma, supporting or immobilizing a limb, and protecting underlying fractures.
- *Tertiary layer*: Holds other layers in place, provides pressure, and keeps inner layers protected from the environment.

CARE OF BANDAGES

- Bandages should be protected from dirt and wet.
- Good observation is necessary to check for movement of the bandage or dressings and the presence of any sores, unpleasant odors, discharges, and discoloration.

TABLE 8-12 Materials for Dressings

Dressing—Make and Manufacturer	Description and Application
Primary Layer	
Adhesive Dressings	Wet-to-dry bandaging
Fine mesh and open-weave pads and gauze swabs	Warm saline-soaked gauze swabs with daily changes can be used in the first 3 to 4 days in open, severely contaminated wounds to encourage debridement and removal of necrotic tissue These can be followed by hydroactive dressings Disadvantages of wet-to-dry dressings are that the moist environment may encourage the growth of bacteria and that regular dressing change may disrupt the healing process With the advent of hydrogels and hydrocolloids they are now obsolete
Antimicrobial Dressings Actisorb Plus (Johnson & Johnson) Actisorb Silver 220 (Johnson & Johnson)	Suitable for discharging purulent and contaminated wounds Contains activated charcoal and silver, which inhibits bacterial growth The dressing creates a favorable environment for effective wound healing by adsorbing and killing microorganisms that contaminate and infect wounds Activated charcoal also binds bacterial endotoxins
Acticoat with Silcryst (Smith & Nephew)	Acticoat with Silcryst nanocrystals works like Actisorb Silver 220
Inadine (Johnson & Johnson)	A topical antimicrobial wound dressing impregnated with an ointment containing 10% povidone–iodine (PVP-I) The povidone molecule provides sustained release of iodine
Silvasorb sheet	Continuous antibacterial protection for up to 7 days, nonadherent dressing
Calcium Alginate Dressings Sorbsan (Pharma-Plast Ltd., Steriseal Division) Sorbsan Plus (Pharma-Plast Ltd.) 3M Tegagen Alginate Dressing (3M Health Care Ltd.) Kaltostat (ConvaTec Ltd.) Kaltogel (ConvaTec Ltd.) Algisite M (Smith & Nephew) Melgisorb (Mölnlycke)	Highly absorbent biodegradable alginate dressings are derived from seaweed and are applied to cleanse a variety of secreting lesions; high absorption is achieved via a strong hydrophilic gel that limits wound secretions and minimizes bacterial contamination Alginate fibers trapped in a wound are readily biodegraded These cavity dressings are presented in a variety of forms (rope, ribbon filler) depending on product area of use: sloughy wounds, cavity wounds, not suitable for dry necrotic wounds or infected wounds; most require a secondary dressing

TABLE 8-12 Materials for Dressings—cont'd

Dressing—Make and Manufacturer	Description and Application
Collagen Dressings Collamend dressings and particles (Genitrex Animal Health and Nutrition)	Contains collagen and can be used with hydrogels and MVP dressings Suitable for use with degloved wounds, burns, and lacerations Particles make excellent contact with wound surface, can absorb 60 times their own weight in fluid, help remove exudate and infectious materials from wound, and act as an enzymatic debriding agent Dressings are porous collagen membranes that can be used on any wound type at any stage of healing Produces fluid containing growth factors Interacts with the wound bed to form an optimal environment for wound healing Provides a matrix for cell epithelialization
Honey Medihoney Antibacterial Wound Gel Medihoney Antibacterial Medical Honey	Antibacterial wound gel and antibacterial medical honey are both packaged in single-use tubes The gel has a high viscosity and is recommended for use with ulcers, surgical sites, and burns Deep wounds, sinuses, and necrotic and surgical wounds are best treated with the honey
Hydrocellular (Foam) Dressings Allevyn (Smith & Nephew) Allevyn Cavity Wound Dressing® (Smith & Nephew) Tielle (Johnson & Johnson) Lyofoam (Seton Healthcare Group) Mepilex (Mölnlycke)	These products consist of hydrophobic polyurethane foam or polyurethane foam film with or without adhesive borders The side of the dressing that comes in contact with the skin has been heat treated to collapse the cells of the foam and enable it to absorb liquid by capillarity The dressings are freely permeable to gases and water vapor but resist the penetration of aqueous solutions and wound exudate When used the dressing absorbs blood or other tissue fluids and the aqueous component is lost through evaporation via the back of the dressing The dressing maintains a moist, warm environment at the surface of the wound, which is conducive to the formation of granulation tissue and reepithelialization Most foams are suitable for light to medium exuding wounds They can be held in place with tape or a bandage: a secondary dressing is not usually required Not recommended for dry or superficial wounds
Hydrocolloid or Hydroactive Dressings Granuflex (ConvaTec) DuoDerm (ConvaTec) DuoDerm ExtraThin (ConvaTec) Comfeel Hydrocolloid Dressing (Coloplast) 3M Tegasorb Hydrocolloid Dressing (3M Health Care Ltd.) 3M Tegasorb Thin Hydrocolloid Dressing (3M Health Care Ltd.) Replicare Ultra (Smith & Nephew)	Semiflexible opaque membranes are impermeable to moisture vapor and act as a physical barrier on a necrotic wound and help it become rehydrated The necrotic tissue eventually separates leaving behind yellow, partially liquefied material known as slough The dressings adhere to the normal skin but not to wounds and form a gelatinous mass over the wound that creates a good atmosphere for healing Hydrocolloids promote the formation of granulation tissue and provide pain relief by covering nerve endings with gel and exudate These dressings have been successfully used in a variety of avian species and are particularly useful for extensive wounds with an excessive exudate production Also for wounds that are slow to heal and those in need of debridement DuoDerm Extra Thin has been successfully used on chronic scalp traumas and held in place with dabs of Vetbond (3M) tissue glue DuoDerm Extra Thin has limited absorbency and used in treatment of lightly exuding wounds It can also be used as a secondary dressing over hydrogels and alginates DuoDerm can be changed on a weekly basis once healing processes are underway In their intact state most hydrocolloid dressings are impermeable to water vapor but as the gelling process takes place the dressing becomes progressively more permeable Hydrocolloids are not suitable for infected wounds
Hydrogels Intrasite Gel (Smith & Nephew) Granugel (ConvaTec) Nu-Gel (Johnson & Johnson) Purilon hydrogel (Coloplast) Vetalintex wound hydrogel (Robinson Animal Healthcare) Silvasorb gel	Hydrogels' basic structure consists of 2%-3% gel-forming polymer such as sodium carboxymethylcellulose, modified starch, or sodium alginate; 20% propylene glycol; and 80% water Gel is placed on the wound and covered with a suitable secondary layer (e.g., MVP or thin hydrocolloid dressings) that prevents loss of moisture from the gel or absorption by the outer layer Water is donated by the gel to the dead tissue and it becomes rehydrated and thus more easily removed Hydrogels are suitable for use on dry, "sloughy," or necrotic wounds and lightly exuding wounds They are suitable for all stages of wound healing except infected or heavily exuding wounds

Continued

TABLE 8-12 Materials for Dressings—cont'd

Dressing—Make and Manufacturer	Description and Application
Low-adherence Dressings Melolin (Smith & Nephew) Mepitel (Mölnlycke Health Care) Mepore (Mölnlycke) Mesorb (Mölnlycke) N-A Ultra (Johnson & Johnson Medical) Release (Johnson & Johnson) Tricotex (Smith & Nephew)	Low-adherence dressings are the current-day alternative to traditional dry dressings such as cotton wool, gauze, and lint N-A Ultra is claimed to be truly nonadherent; the other dressings are considered low adherence Most are suitable on dry or lightly exuding wounds Mepitel, Mesorb, and Mepore can be used on medium to heavily exuding wounds, although a secondary dressing may be required to absorb excess exudate
Polysaccharide Dressings Debrisan beads Debrisan paste (Pharmacia & Upjohn Ltd.)	Consist of pale dextranomer 0.1-0.3 mm diameter beads When introduced into an exuding wound 1 g of beads will absorb up to 4 g of exudate When applied to relatively small sloughy wounds the beads absorb fluid and progressively move bacteria and cellular debris away from the wound surface Not for use on dry or lightly exuding wounds
Iodosorb (Smith & Nephew)	Consists of hydrophilic beads of cadexomer (a modified starch hydrogel, which is biodegradable) impregnated with elemental iodine Suitable for infected exuding cavities Dressings need to be changed regularly if wound heavily exuding, indicated by loss of color of the iodine
Iodoflex (Smith & Nephew)	Consists of a sterile cadexomer iodine paste sandwiched in protective gauze and changed when there is a loss of color
Polyurethane matrix dressing Cutinova Hydro (Smith & Nephew) Hydro-Selective Dressing	Cutinova Hydro is a recently introduced dressing developed as a successor to the hydrocolloids It has been designed to offer the benefits of a hydrocolloid with none of the drawbacks Its special structure offers a unique mode of action, absorbing water from wound fluid but leaving essential wound-healing agents behind in the wound Cutinova Hydro therefore combines all the proven benefits of clean, moist wound healing with the ability to leave growth factors, essential agents in wound healing, and other natural proteins on the wound bed
Tulle (nonmedicated) dressings Jelonet (Smith & Nephew) Paratulle (Seton)	Can be used for clean superficial wounds Tulles contain different weights of paraffin per unit area Paraffin reduces the adherence of the dressing to the wound but requires frequent changes to stop it drying out and being incorporated into granulation tissue A secondary dressing is always required
Tulle (Medicated) Dressings Fucidin Intertulle (Leo Laboratories Ltd.) Tulle (medicated) dressings (Smith & Nephew) Serotulle (Seton) Clorhexitulle (Roussel) Sofra-Tulle (Roussel)	Medicated tulle dressings are often used inappropriately for infected superficial wounds Bactigras, Clorhexitulle, and Serotulle are similar, containing 0.5% chlorhexidine These dressings are suitable if the use of an antiseptic product is deemed necessary The use of Fucidin Intertulle and Sofra-Tulle is declining in health care because both products contain topical antibiotics and lanolin, which carry the risk of skin sensitization
Vapor-permeable Adhesive Film (MVP) Dressings Bioclusive (Johnson & Johnson) OpSite Flexigrid (Smith & Nephew) 3M Tegaderm Transparent Film Dressing (3M Health Care Ltd.) Tegaderm Plus (3M) Mefilm (Mölnlycke)	MVP dressings are slim, flexible, and transparent polyurethane membranes with an adhesive backing They are permeable to oxygen but not to water or bacteria, allowing accumulation of fluid exudate under the dressing The maintenance of an aerobic environment under the dressing prevents scab formation and promotes more rapid epithelialization, while preventing wound desiccation and reducing pain associated with lack of moisture and raw nerve endings Both these and hydrocolloid membranes are indicated for a variety of avian wounds, but MVP dressings are more suited to areas that are difficult to bandage (e.g., head wounds) because of the superior adhesive quality and flexibility of the material Dressings are changed every 2-3 days initially, and more frequently if there is excessive exudate production resulting in leakage of fluid from under the dressing Tegaderm Plus is coated with a layer of acrylic adhesive that contains 2% available iodine in the form of an iodophor; when in contact with the skin it slowly releases the iodine

TABLE 8-12 Materials for Dressings—cont'd

Dressing—Make and Manufacturer	Description and Application
Secondary Layer	
Padding	Plenty of padding should be used, especially on pressure points and areas easily traumatized (e.g., wing tips)
Absorbent cotton	
Softband	
Orthoband	
Bandaging	A cotton bandage that has now been superseded by a conforming bandage
White open-weave bandage	
Conforming Bandage	Can be applied firmly over the initial dressing and padding and as the name suggests conforms to the area bandaged
Crinx (Smith & Nephew)	Several layers may be applied
Bioband (Leatherite PTY Ltd., Australia)	Bioband is an antimicrobial-impregnated bandage that prevents the growth of gram-positive and gram-negative bacteria and reduces the risk of the bandage becoming a source of external contamination
Vet-Band (Millpledge Veterinary)	
Vetband (Smith & Nephew)	
Tertiary Layer	
Elastic Adhesive Bandage	Usually applied as the external layer to give extra support and hold the other bandage in place
Elastoplast (Smith & Nephew)	Care should be taken not to wrap it too tightly or to attach it to too many feathers or skin
Treatplast (Animalcare)	
Veterinary Flexoplast (Robinson)	
Cohesive Bandage	Consist of water-vapor–permeable, nonwoven polyester fabric containing longitudinal strands of polyester elastane
3M Vetrap Bandaging Tape (3M Health Care Ltd.)	The fabric is coated with a self-adherent substance that gives the bandage the ability to stick to itself and not to the skin
Coflex (Valley Vet Supply)	Care is needed in its application, however, because of the loss of the potential for movement between turns of the bandage to equalize the pressure on local areas
Coban (3M)	High tension carries the risk of creating a tourniquet effect
Wrapz (Millpledge Veterinary)	
Co-Form (Millpledge Veterinary)	
Easifix	

- The bird should be prevented from interfering with the bandage; in psittacines it may be necessary to use an Elizabethan collar or similar neck-restraining device.
- Most dressings are suitable to be left in situ for 3 to 7 days and should be left undisturbed to maintain constant temperature, humidity, and reduce bacterial access. If wounds are infected or the dressing becomes contaminated, it is then essential to change them.

REMOVAL OF DRESSINGS

- Bandages should be removed using round-ended scissors or bandage shears; care needs to be taken not to cut the skin or interfere with the healing process of the wound.
- The actual dressing removal will depend on type used; flushing with saline may be necessary.
- Contaminated dressings should be suitably disposed of and the scissors used in the procedure should be washed and sterilized.
- Hands should be washed with an antiseptic solution before applying a new dressing.

The range of dressings available is diverse, and depending on their structure and composition, dressings may be used to absorb exudate, combat infection, relieve pain, promote autolytic debridement, and provide and maintain a moist environment at the wound surface to promote granulation tissue and the process of epithelialization. Some dressings simply absorb exudate and may be suitable for use with a variety of different wounds. Others have a very clearly defined specialized function and have a more limited range of indications. Wound healing is a dynamic process; no one dressing is suitable for all wound types and few are suitable for the treatment of a single wound during all the stages of the healing process. Good wound management requires a flexible approach in the selection of dressings and understanding of the healing processes. Without taking this knowledge into consideration, the process becomes rather capricious and potentially ineffective.

ACKNOWLEDGMENTS

The author and the editor are most grateful to Dr. Hugues Beaufrère for his assistance in reviewing this section.

FURTHER READING

Cousquer G: Wound management in the avian wildlife casualty, *World Wide Wounds*: http://www.worldwidewounds.org, 2005.

Degernes LA: Trauma medicine. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Fowler A: *How to Manage Open Wounds in Wildlife*, National Wildlife Rehabilitation Conference. http://www.nwrc.com.au/forms/anne_fowler_1.pdf, 2005.

Merck Veterinary Manual: Emergency medicine and critical care: wound management, http://www.merckvetmanual.com/, 2006.

Molan PC: Honey as a topical antibacterial agent for the treatment of infected wounds, *World Wide Wounds*: http://www.worldwidewounds.org, 2001.

Morgan DA: Wound management products on the Drug Tariff, *Pharmaceut J* 263:820–825, 1999. Available online at: http://www.pjonline.com/Editorial/19991120/education/wounddressings.html.

Redig PT: Avian emergencies. In Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *Manual of raptors, pigeons and waterfowl*, Cheltenham, UK, 1996, British Small Animal Veterinary Association.
Stewart J: Next generation products for wound management, *World Wide Wounds*: http://www.worldwidewounds.org, 2002.
Thomas S: A structured approach to the selection of dressings, *World Wide Wounds*: http://www.worldwidewounds.org, 1997.

USEFUL WEBSITES

3M Health Care Ltd.: www.3m.com/healthcare.
Andover Health Care: www.andoverhealthcare.com.
Coloplast: http://www.coloplast.co.uk/.
ConvaTec Ltd.: www.convatec.com.
CPD Solutions Practical Wound Management for Nurses Session 2 Debridement and Open Wound Management: http://www.veterinarywebinars.com/assets/Wound_Debridement_Open_Wound_Management_Session_2_Notes.pdf.
Dechra Veterinary Products: http://www.dechra.co.uk/.
Dermasciences: http://dermasciences.com/medihoney.
Johnson & Johnson: http://www.jnj.com/home.htm.
Kruuse: http://www.kruuse.com.
Medihoney™ Dressings Made Easy—Wounds UK, Volume 7, November 4, 2011: http://www.wounds-uk.com/made-easy/medihoney-dressings-made-easy—products-for-practice.
Medihoney: www.dermasciences.com/medihoney.
Millpledge: http://www.millpledge.com.
Mölnlycke Health Care: http://www.molnlycke.com/index.asp?id=3142&lang=2.
Robinson Animal Healthcare: www.robinsonhealthcare.com.
Smith & Nephew Medical Ltd.: www.smith-nephew.com.
Smiths Medical: http://www.smiths-medical.com/veterinary/.
Systagenix: http://www.systagenix.co.uk/our-products/.
Vet Wound Library: http://www.vetwoundlibrary.com/news.
Veterinary Wound Management Society: www.vwms.net.
World Wide Wounds: www.worldwidewounds.org.
Wounds International: http://www.woundsinternational.com.

PROTECTIVE FOOT CASTING

Jaime Samour

Protective foot casts are indicated to reduce pressure-related trauma to the newly created wound in the postoperative care of pododermatitis or bumblefoot. Halliwell (1981) first proposed the application of a thermoplastic tape (Hexcelite) to immobilize the limbs of birds of prey. Remple and Remple (1987) described a casting method using the same thermoplastic tape as an adjunctive therapy to bumblefoot surgery in falcons. After surgery, a dressing and a bandage are applied to the distal tarsometatarsus and the foot using an elastic nonadhesive bandage (Vetrap, 3M). Lighter, individual bandages are also placed around the first phalanx of each toe. The cast consists of one piece of thermoplastic tape wrapped around the tarsometatarsus and extending forward to the first phalanx of each toe. Narrow strips of nonadhesive tape are used to fix each toe to the cast. A double-thickness piece of the thermoplastic tape is used to make a bridge under the foot, leaving a gap under the plantar surface of the foot. This piece is then secured on both sides of the tarsometatarsus. Before application, the thermoplastic tape should be immersed in hot water to make it pliable and easy to manipulate.

Remple (1993) proposed a new foot-casting method for the postsurgical management of bumblefoot. Preformed "shoes" or plantar casts were made using a commercially available styrene plastic polymer commonly used for car body repairs (Bondo). Casts of different sizes and shapes were prepared using precast molds. A large hollowed area is left at the center of the cast to ensure adequate protection of the newly operated site. After surgery the foot and the toes, up to the first phalanx, are bandaged using an elastic nonadhesive tape (Vetrap). The cast is then glued on to the bandage.

Riddle and Hoolihan (1993) designed a form-fitting composite-casting method for the legs and wings of birds. This casting method was mainly used in the postoperative care of falcons operated on for bumblefoot. After surgery, the first phalanx of each toe was bandaged using an elastic nonadhesive bandage. A small amount of fast-setting epoxy glue was applied to the ventral surface of each toe. Then a small ball was made of cotton wool and wrapped in the same nonadhesive tape to form a cylinder. This was then fixed onto the plantar area of the foot at the point of the base of the first phalanx of each toe. The cylinder was secured to the foot using the same nonadhesive tape. Fresh epoxy glue was coated onto the cylinder and was secured to the foot using the same nonadhesive tape. Fresh epoxy glue was coated onto the whole bandage and the cylinder. The sections of the bandage around the tarsometatarsus and the distal end of the toes were left without glue as a soft buffer layer between cast and skin. The bottom section of the cylinder was cut to provide a window to the plantar area of the foot and to allow periodic inspection and redressing of the wound.

Harcourt-Brown (1996) described the use of an adherent hydrocolloidal dressing in combination with a plastic casting material in the postoperative care of bumblefoot. More recently, Remple (2005) proposed the use of a silicone-composite dental material to produce a form-fitting and flexible protective cast to aid healing in the postoperative care of bumblefoot. This technique produces a much softer shoe than any of the techniques previously described (Remple and Remple, 1987; Remple, 1993; Riddle and Hoolihan, 1993; Harcourt-Brown, 1996). The author favors the use of protective shoes manufactured from a thick soft rubber commonly used to make beach sandals. These can be manufactured in different sizes and stored (Samour, 2005). Other surgeons prefer to use padded rings (donut-shaped) fitted to the feet of the bird using nonadhesive elastic bandages (N. Forbes, personal communication). The use of protective casts in the postoperative management of bumblefoot is not only limited to raptors. More recently, the use of protective shoes manufactured from neoprene was described in penguins (Reidarson *et al.*, 1999). The shoes included a high heel to prevent slippage on icy surfaces and were fastened to the foot of the birds using Velcro straps. A similar technique can probably be used in the postoperative care of bumblefoot in other species, such as flamingos, waders, and shore birds and large waterfowl.

REFERENCES

Halliwell WH: New thermoplastic casting material and its application to birds of prey. In Cooper JE, Greenwood AG, editors: *Recent advances in the study of raptor diseases*, Keighley, West Yorkshire, UK, 1981, Chiron Publications.
Harcourt-Brown NH: Foot and leg problems. In Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *Manual of raptors, pigeons and waterfowl*, Quedgeley, Gloucester, UK, 1996, British Small Animal Veterinary Association.
Reidarson TH, McBain J, Burch L: A novel approach to the treatment of bumblefoot in penguins, *J Avian Med Surg* 13(2):124–127, 1999.
Remple JD: Raptor bumblefoot: a new treatment technique. In Redig PT, Cooper JE, Remple JD, et al, editors: *Raptor biomedicine*, Minneapolis, MN, 1993, University of Minnesota Press.
Remple JD: *Use of a composite silicone dental impression material to create a form-fitting, flexible, support cushion to facilitate wound healing in*

bumblefoot, Proceedings of the European Association of Avian Veterinarians, Arles, France, 2005.
Remple JD, Remple CJ: Foot casting as adjunctive therapy to surgical management of bumblefoot in raptorial species, *J Am Anim Hosp Assoc* 23:633–639, 1987.
Riddle KE, Hoolihan J: Form-fitting, composite-casting method for avian appendages. In Redig PT, Cooper JE, Remple JD, et al, editors: *Raptor biomedicine*, Minneapolis, MN, 1993, University of Minnesota Press.
Samour J: Clinical management of captive raptors. In Harrison GJ, Lightfoot T, editors: *Clinical avian medicine*, Lake Worth, FL, 2005, Spix Publishing.

COLLARS

Morena Bernadette Wernick

Collars are useful devices commonly used to prevent birds from feather plucking, self-mutilation, destroying bandages, or disturbing newly operated sites. They are used to create a physical barrier between the sharp beak and the affected area. A variety of collars is commercially available in different sizes and is commonly made from clear vinyl or acrylic.

Elizabethan collars or "inverted" Elizabethan-style collars are comfortable to wear and allow freedom of movement to the patient. They have a disk-shaped appearance and can easily be made of cut-away sections of radiographic film or from any lightweight and rigid, but flexible, material. Several modified versions of the traditional Elizabethan collar have been introduced to the market.

Stultiens-type collars or other tubular-shaped collars provide a comfortable fit around the neck and appear to be well tolerated by most patients (Figs. 8-26 and 8-27). They are commercially available in different sizes, but can also be made from pipe-insulating foam. For this purpose, a tube can be cut out of the foam to a length equal to the straight length of the neck and be taped around the neck (Chitty, 2005). Combinations of Elizabethan collars and tubular-shaped collars are possible and beneficial in certain cases.

Spherical plastic collars made of two interlocking sections are also commercially available and provide a comfortable fit around the neck. They can be easily fixed and removed by the pet owners.

Care must be taken with every patient whenever a collar has been fitted. Because collars may be extremely stressful for birds, patients should be observed at least 6 hours after application to assert proper fit and adaptation (McCluggage, 1997). Hospitalization may be necessary in some cases until the patient gets used to its collar. It is essential to screen food and water intake during this time and to reposition food and water bowls in a way that they are easily reachable for the patient.

FIGURE 8-26 Stultiens-type collar on an African grey parrot (*Psittacus erithacus*). (Courtesy Andrés Montesinos.)

FIGURE 8-27 Tubular shaped collar on a gray-headed lovebird (*Agapornis canus*). (Courtesy Andrés Montesinos.)

REFERENCES

Chitty J: Feather and skin disorders. In Harcourt-Brown N, Chitty J, editors: *BSAVA manual of psittacine birds*, Cheltenham, UK, 2005, British Small Animal Veterinary Association.
McCluggage DM: Bandaging. In Altmann RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

PRODUCT REFERENCE LIST

Avian Spherical CollarR, GHN Inc., Fort Myers, FL 33912-4919: http://www.aviancollar.com.
Veterinary Specialty Products Inc., Mission, KS 66201: http://www.vet-products.com.
Hexcelite, Hexcel Medical, 6700 Sierra Lane, Dublin, CA 94566: www.excel.com.
Veterinary Thermoplastic, Imex Veterinary Inc., Longview, TX 75604: www.imexvet.com.
Sam Splint, Moore Medical Corporation, New Britain, CT 06050-2620: www.mooremedical.com.

WING CLIPPING, PINIONING, AND FEATHER-FOLLICLE EXTIRPATION

Morena Bernadette Wernick

Wing clipping, pinioning, and feather-follicle extirpation are techniques designed to prevent birds from flying. These procedures are performed easily, but which is the best method to follow is controversial.

Although wing clipping should not be used as a long-term treatment in parrots (e.g., for the convenience of the owner; Chitty, 2005), it is still indicated in waterfowl, cranes, storks, or flamingos housed in open paddocks or in highly nervous terrestrial species kept in large aviaries. There are several ways to perform wing clipping. Most often, the primary feathers of only one wing are simply cut short using a pair of strong scissors or nail clippers. The advantage of a unilateral cut is its effectiveness at deflighting a bird. The main disadvantage of this method is that birds can get severely unbalanced after the procedure and seem to have a higher risk of injury from a spiraling fall compared with birds with a bilateral (two-winged) cut (Chitty, 2005). In both methods (one-winged and two-winged), some recommend leaving the first outer primary feathers intact, because these will give the closed wing a more natural appearance and the bird more balance. Other methods only remove the outer primary flight feathers. With this method, the bird obviously appears wing clipped (disturbing cosmetic appearance), and some people argue that it does not respect the natural molting sequence of the feathers, and since the molting sequence varies from species to species, this has to be considered. It is suspected that the missing outer feathers will not guard the new feathers, which are then possibly at a higher risk of damage (Chitty, 2005). Excessive trimming should be avoided at all times, because wing trimming should only stop the bird from having a strong and sustainable flight, not stop the bird from flying at all. In the worst case, extensive wing trimming can result in severe accidents if a bird is unable to escape from cage companions or to move freely within the enclosure. When wing trimming is performed, every feather should be cut singly (Chitty, 2005) and only fully grown feathers should be cut. Cutting of "green" or "blood" feathers will result in profuse bleeding. Feathers should be clipped just proximal to the distal tip of the covert feathers (Bennett and Baumgartner, 2014). As they tend to splinter, only strong scissors or nail clippers should be used for trimming (Figs. 8-28 to 8-30).

Wing clipping only impairs flight for a relatively short period and has to be done on a regular basis, depending on the species. Therefore, techniques that are more permanent are described to deflight birds in zoologic collections and bird gardens. Methods described include patagiectomy (Mangili, 1971; Robinson, 1975a), tenotomy (Schroeder and Kock, 1940; Geron, 1981), tenectomy of the extensor tendons (Degernes and Feduccia, 2001), radial neurectomy, fusing the carpal joint by cerclage (Sedgewick, 1967), and pinioning (Robinson, 1975b; Fletcher and Miller, 1980; Chitty, 2005). Pinioning is the method most used worldwide, especially in waterfowl.

Ideally, pinioning should be conducted when the birds are approximately 1 week old. The process at this age is simple and done using a pair of scissors. The wing is cut at the proximal end of the metacarpal bone and surgical glue or a hemostatic clip is applied to the wound if hemorrhage occurs. Anesthesia and good analgesia is recommended. Usually, pinioning is only performed on one wing.

In older birds, pinioning is a much larger surgical procedure (Williamson and Russell, 1971). Surgery must be performed under general anesthesia and good analgesia is mandatory. The area around the metacarpal joint is aseptically prepared and a circular incision is made on the skin, approximately 2 to 5 cm from the joint, depending on the size of the bird. Larger blood vessels are ligated before severing the muscle and tendons. The bones are then cut using an orthopedic saw. The skin is stitched and a dressing and a bandage are applied to the tip of the wing. Alternatively, the wing tip may be amputated by disarticulation of the carpal joint instead of severing the wing tip just below the joint. The alula is left in place to provide a more natural appearance (Fig. 8-31).

Another pinioning method, described by Lewandowski and Sikarskie (1996), applies a castration rubber band just above the incision site and cuts the skin and underlying tissues, including the bones, with a double-action bone cutter.

More recently, feather-follicle extirpation has been recommended as the method of choice for deflighting birds. Surgical excision,

FIGURE 8-28 Wing clipping in a cockatiel (*Nymphicus hollandicus*). Here the primary feathers of one wing are cut short, using a pair of strong scissors or nail clippers. The main disadvantage of this method is that birds can become severely unbalanced after the procedure and they seem to have a higher risk of injury from a spiraling fall compared with birds with bilateral clipping, which is now recommended by some clinicians. (Courtesy Bob Doneley.)

FIGURE 8-29 The primary feathers in the cockatiel (*Nymphicus hollandicus*) have been cut. Excessive trimming should be avoided at all times, since wing trimming should only stop the bird from having a strong and sustainable flight, but should not stop the bird from flying at all. (Courtesy Bob Doneley.)

FIGURE 8-30 Significant blood loss in a blue and yellow macaw (*Ara ararauna*) after the owner attempted to perform a wing clip and cut through a blood feather. (Courtesy Neil Forbes)

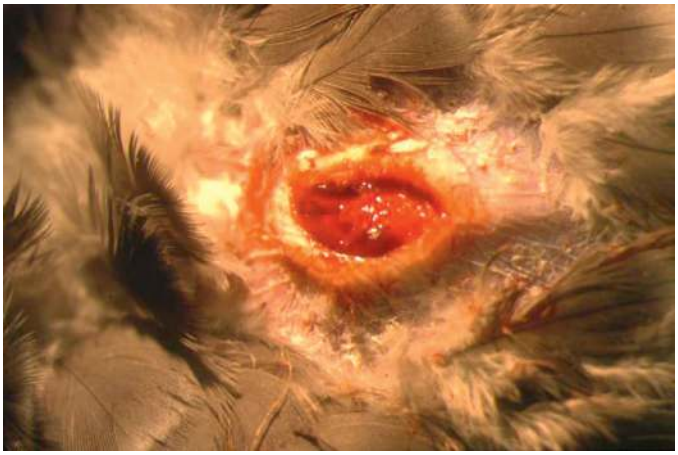


FIGURE 8-31 Another example of the negative side of wing clipping in parrots. This is a large ulcer over the keel of an African grey parrot (*Psitttacus erithacus*) also resulting from repeated crashing to the floor. In parrots, wing clipping should not be used as a long-term treatment and/or for the convenience of the owner. (Courtesy Neil Forbes.)

electrosurgical fulguration, cryosurgery, and the use of lasers have been described. Diode lasers, in particular, seem to be promising and can be used to extirpate the primary remiges because the heat from the laser light energy causes thermal necrosis of the feather follicle (D'Agostino *et al.*, 2006; Krawinkel *et al.*, 2008; Shaw *et al.*, 2012; Bennett and Baumgartner, 2014). The main advantage of this method is that birds do not need any bandages or topical or systemic antibiotic treatment. Bleeding and tissue damage are minimal and the birds can be taken back to their group immediately after recovering from anesthesia. As for all procedures, good analgesia is mandatory throughout the first postoperative days.

REFERENCES

Bennett RA, Baumgartner K: Avian deflighting techniques. In Miller RE, Fowler ME, editors: *Fowler's zoo and wildlife animal medicine*, *vol* 8, St Louis, MO, 2014, Elsevier Saunders.

Chitty J: Basic techniques. In Harcourt-Brown N, Chitty J, editors: *BSAVA Manual of psittacine birds*, ed 2, Gloucester, UK, 2005, British Small Animal Veterinary Association.

D'Agostino JJ, Snider T, Hoover J, et al: Use of laser ablation and cryosurgery to prevent primary feather growth in a pigeon (*Columba livia*) model, *J Avian Med Surg* 20:219–224, 2006.

Degernes LA, Feduccia A: Tenectomy of the supracoracoideus muscle to deflight pigeons (*Columba livia*) and cockatiels (*Nymphicus hollandicus*), *J Avian Med Surg* 15:10–16, 2001.

Fletcher KC, Miller BW: A technique for cosmetic pinioning of adult birds, *Vet Med Small Anim Clin* 75:1898–1904, 1980.

Geron R: Inhibition of flight in captive birds by tenotomy of the extensor pollicis brevis, *Int Zoo Yearbook* 21(1):172–173, 1981.

Krawinkel P, Weber H, Schauerte N, et al: *Extirpation of feather follicles—a practical, acceptable, and permanent method to prevent zoo birds from flying?* Proceedings of the European Association of Zoo and Wildlife Veterinarians, 7th Scientific Meeting, 2008.

Lewandowski AH, Sikarskie JG: *Pinioning. a quick and simple technique*, 1st International Conference of Zoo and Avian Medicine, 1996.

Mangili G: Unilateral patagiectomy: a new method of preventing flight in captive birds, *Intl Zoo Yearbook* 13(1):87–94, 1971.

Robinson PT: Unilateral patagiectomy: a technique for deflighting large birds, *Vet Med Small Anim Clin* 70(2):143–145, 1975a.

Robinson PT: Pinioning young birds with hemostatic clips, *Vet Med Small Anim Clin* 70:1415–1417, 1975b.

Schroeder CR, Kock K: Preventing flight in birds by tenotomy, *J Am Vet Med Assoc* 97:160–170, 1940.

Sedgewick CJ: Deflighting pet birds, *Mod Vet Pract* 48:38–40, 1967.

Shaw SN, D'Agostino JJ, Davis MR, et al: Primary feather follicle ablation in common pintail (*Anas acuta acuta*) and a white faced whistling duck (*Dendrocigna viduata*), *J Zoo Wildl Med* 4(32):342–346, 2012.

Williamson WM, Russell WC: Prevention of flight in older captive birds, *J Am Vet Med Assoc* 159:596–598, 1971.

FURTHER READING

Ellis DH, Dein FJ: Surgical techniques, part E: flight restraint. In Ellis DH, Gee GF, Mirande CM, editors: *Cranes: their biology, husbandry, and conservation*, Washington, DC, 1996, National Biological Service.

Miller JC: The importance of immobilizing wings after tenectomy and tenotomy, *Vet Med Anim Clin* 68:35–38, 1973.

Olsen JE: Anseriformes. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and applications*, Lake Worth, FL, 1994, Wingers Publishing.

CLAW AND TALON TRIMMING

Morena Bernadette Wernick

Claws or talons (raptors) of birds surround the phalanx distalis of each digit and are composed of two layers: a hard, keratinized casing, covering the dorsal and lateral aspects, and the ventral surface is formed of a softer "solear" structure. As the dorsal ridge grows faster than the ventral plate, claws have a curved appearance (Orosz, 1997). Underneath the keratinized layer, dermis and subcutis provide neurovascular supply. Claws protect the phalanx from damage and serve species specifically as a tool for grasping, digging, landing, climbing, hunting, and defense (Hirschberg, 2008). Their appearance varies from species to species, from the blunter and short claws in Passeriformes to the very sharp and curved talons of raptors. As a unique characteristic, chicks of the hoatzin (*Opisthocomus hoazin*), order Opisthocomiformes, possess claws on two of their wing digits. In addition, several raptor species possess rudimentary claws at the tips of their wings (e.g., golden eagles, gyrfalcons, and other species).

Overgrown claws or talons are frequently seen in captive birds from several orders in veterinary practice, but in particular from birds of the

FIGURE 8-32 Overgrown and deformed talons in a saker falcon (*Falco cherrug*). Regular trimming and reshaping would prevent such deformities. (Courtesy Dr. Jaime Samour.)

FIGURE 8-33 Grinding tools (Dremel) are useful for trimming the claws of psittacine birds. As grinders usually produce heat, bleeding is less likely because nails are often automatically cauterized. (Courtesy Andrés Montesinos.)

orders Psittaciformes, Passeriformes, and Falconiformes (Fig. 8-32). Often, these birds are presented with very long and sometimes even curled claws. In contrast, birds from other orders (e.g., Columbiformes) rarely require claw trimming. Reasons for overgrowth include insufficient wear associated with perches that are too small or too wide in diameter and perching surfaces that are too soft. Other overgrowth conditions from failure to perch properly include medical problems like pododermatitis or osteoarthritis. Also viral diseases (psittacine beak and feather disease), liver disease, or malnutrition may disturb the normal keratin metabolism and provoke overgrowth.

Nail clippers, metal files (larger birds), fine-grain nail files (smaller birds), or grinding tools (Dremel) may be used for claw trimming (Figs. 8-33 to 8-36). To reshape the talons of birds of prey, utility knives with curved blades and a combination of flat and round metal files are beneficial. Usually, it is only necessary to trim the claw tips—3 mm in smaller species and up to 5 to 8 mm in larger species. By using nail clippers, it seems to be advantageous to cut the claw from side to side to squeeze the claw over the artery to reduce hemorrhage and prevent

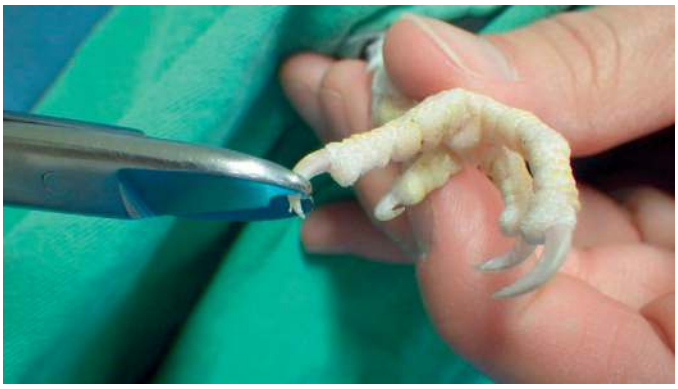


FIGURE 8-34 Nail cutters are ideal for trimming the talons of birds of prey and the claws of other birds. (Courtesy Peter Sandmeier.)

FIGURE 8-35 Fine-grain nail files give the final touch in claw/talon trimming and reshaping. (Courtesy Dr. Jaime Samour.)

FIGURE 8-36 A utility knife (commonly used to cut carpets) fitted with a curved blade is a very useful for reshaping the talons of falcons. (Courtesy Dr. Jaime Samour.)

claw splitting (Chitty, 2008). During the trimming process, hemorrhages may occur if the claws or talons are cut too short. Additionally, especially in overgrown claws, the artery often nearly reaches the end of the nail. Bleeding is less likely with the use of grinding tools (e.g., Dremel) because grinders usually produce heat that automatically

cauterizes the nail. If hemorrhages of the claws and talons occur, silver nitrate pencils or powders, potassium permanganate crystals or powders, thermocautery, or electrocautery may be used to stop extensive bleeding. With the help of an experienced veterinary technician, claw or talon trimming can be done without general anesthesia in most birds within a few minutes. General anesthesia is required in nervous and uncooperative patients or if the required trimming is too extensive and hemorrhage is unavoidable (Chitty, 2008; Jones, 2009). In such cases, adequate analgesia is mandatory.

Very often, clinicians are confronted with detached, broken, or shed claws or talons. Because claws or talons grow continuously, injuries affecting the zone of proliferation may cause severe abnormalities in the development of the protective outer keratin layers. Even small injuries may generate extensive bleeding. Special care must be taken if the claw is detached and the underlying phalangeal bone and its germinal epithelium are exposed. After cleansing and drying of the bone, a protective cover can be made using four to five layers of cyanoacrylate glue mixed with antibiotic powder (e.g., clindamycin, piperacillin) and talcum powder (Molnar and Ptacek, 2001). Alternatively, cyanoacrylate glue and fine sodium bicarbonate powder can be used to the same effect (Samour, 2005). The artificial claw is usually shed after a few weeks. If a claw is detached and glue material is not available, the bone must be cleaned, disinfected, and covered with an appropriate wound dressing (e.g., hydrocolloid dressing; Chitty, 2008). Good analgesia and the application of antibiotics are mandatory. Dressings should be changed after 48 hours and afterward every 5 to 7 days. Regrowth of a new claw will usually take 4 to 8 months, depending on the species.

REFERENCES

Chitty J: Basic techniques. In Chitty J, Lierz M, editors: *BSAVA manual of raptors, pigeons and passerine birds*, Quedgeley, Gloucester, UK, 2008, British Small Animal Veterinary Association.

Hirschberg RM: Anatomy and physiology. In Chitty J, Lierz M, editors: *BSAVA manual of raptors, pigeons and passerine birds*, Quedgeley, Gloucester, UK, 2008, British Small Animal Veterinary Association.

Jones AK: The physical examination. In Tully TN, Dorrestein G, Jones AK, editors: *Avian medicine*, Philadelphia, PA, 2009, Saunders Elsevier.

Molnar L, Ptacek M: *Traumatic injuries of beak and talon of captive raptors*, Proceedings of the European Association of Avian Veterinarians, Munich, 2001.

Orosz S: Anatomy of the integument. In Altmann RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Samour J: Clinical management of captive raptors. In Harrison GJ, Lightfoot T, editors: *Clinical avian medicine*, Lake Worth, FL, 2005, Spix Publishing.

BEAK TRIMMING

Morena Bernadette Wernick

The avian beak is composed of the upper (maxilla) and lower (mandible) jaw bones and their keratinized sheaths (rhamphotheca). The rhamphotheca can further be divided into the maxillary keratin sheath (rhinotheca) and the mandibular keratin sheath (gnatotheca). Both rhinotheca and gnatotheca possess median dorsal and, respectively, ventral borders (culmen and gonys; Lumeij, 1994). In the embryo, the culmen of most birds has a pointed protuberance (egg tooth) to break out of the shell, which drops off after hatching (Denbow, 2000). Falcons possess a "falcon tooth" or "tomial tooth" on the upper beak. Cutting edges of both rhinotheca and gnatotheca are called the tomia.

Histologically, the rhamphotheca is composed of modified epidermis, with a thickened and hardened stratum corneum, containing free calcium phosphate, crystals of hydroxyapatite, and keratin. Especially at the tip of the beak, the stratum corneum is very thick and hard. Underneath the epidermis, the dermis becomes continuous with the periosteum of the maxilla or mandible (Orosz, 1997). Within the dermal layer, sensory nerve organs (Herbst corpuscles) cause the tissue to be sensitive to pressure and vibration (Lintner, 2013).

The beaks of caged and aviary birds and birds of prey are prone to overgrowth, most often because of lack of wear (e.g., from inappropriate indoor environment, soft food; Fig. 8-37). Also, congenital defects like scissors beak (lateral deviation of the maxilla) or mandibular prognathism can prevent normal beak function (Fig. 8-38). Embryonic nutritional deficiencies may lead to beak malformation in gallinaceous birds (e.g., deficiencies in folic acid, biotin, pantothenic acid; Lumeij, 1994). Traumatic injuries (punctures, lacerations, cracks, splits, and avulsions) can lead to severe beak malformations or even necrosis of the beak in all bird species.

A variety of infectious pathogens (bacteria, fungi, viruses, or parasites) may affect the germinative layers of the beak. Severe sinus infections of the infraorbital sinuses (e.g., *Pseudomonas* spp., *Mycoplasma* spp., *Mycobacterium* spp., *Chlamydia psittaci*) can descend to the keratin of the beak and even to the delicate bony structures of the beak. Fungal infections, associated with crumbling of the beak's keratin, include *Candida* spp. and *Aspergillus* spp.

FIGURE 8-37 A saker falcon (*Falco cherrug*) with an extremely long beak. It is not uncommon for beaks in such birds to break during feeding. (Courtesy Dr. Jaime Samour).

FIGURE 8-38 Scissors beak and overgrowth of the beak in a cockatiel. The lateral deviation of the rhinotheca and the overgrowth inhibits proper occlusion.

FIGURE 8-39 Cnemidocoptes infestation in a budgerigar (*Melopsittacus undulatus*). Please note the typical "honey-combed" crusts.

Cnemidocoptic mite infestation from *Cnemidocoptes pilae* (psittacine and passerine birds) causes development of "honey-combed" crusts and provokes intensive overgrowth of the beak (Fig. 8-39). Because of mite tunneling through the keratin and damage of keratin layers, parts of the beak may even break. Also, metabolic disease (primarily liver disease) may provoke extensive overgrowth of the maxilla. Therefore, a biochemistry panel should always be performed to exclude any important health issues.

The beak can be trimmed using instruments for nail trimming. Nail clippers are very useful in most species to shorten overgrown tissue. Pediatric 4- to 6-inch bone rongeurs can be used to carefully remove small amounts of tissue and to separate layers of horn (Lintner, 2013). In the author's experience, it is beneficial to start beak trimming at the lateral edges of the premaxilla on both sides until the tip of the beak is reached. Along with nail clippers or rongeurs, nail files in small bird species and a combination of flat and round metal files in large bird species are helpful to grind down overgrown tissue. To shorten the tip, special care must be taken not to evoke excessive bleeding by cutting straight across. Instead, nail files or a Dremel hand drill can be used to carefully smooth the tip. As Dremel hand drills with suitable attachments for small and large birds may produce substantial damage to the very sensible structures of the beak because of friction heat, these tools should only be applied with special care. After the maxilla has been reshaped, the mandible should be trimmed to allow realigning of the occlusal surfaces. Finally, excessive material on the surface of the maxilla can be removed. The beak should be thoroughly cleaned with water. There is no need to cover the beak with any topical products. After trimming, parrots should be encouraged to chew hard material (e.g., branches of fruit trees).

The tip of the beak of captive birds of prey used for falconry should be clipped at the start of the molting season. *Coping* is the ancient falconry term for trimming and reshaping of talons and beaks in Falconiformes (Samour, 2005).

For lateral deviation of the maxilla (scissors beak), a variety of possible treatments have been described in the literature regarding the age of the affected bird. During rearing, manual pressure on the deviated maxilla by hand or by taping the maxilla back to its normal position between feeds may reverse the acquired deformities (Harcourt-Brown, 2005). In older birds, the mandible can be covered with a thick mass of dental composite shaped into a groove that guides the maxilla back in a normal position (Harcourt-Brown, 2005). A more significant deviation may be corrected using trans-sinus pinning techniques (Speer, 2002). Techniques using dental composite have also been described for correction of mandibular prognathism (Schnellbacher, *et al.*, 2010).

As the lateral aspects of the beak are prone to cracks and splits, periodic filling is sometimes necessary. Periodontal dressings can be used to cover larger defects until healing to prevent any further damage. In addition, systemic antibiotics and analgesics have to be added to the therapy to support the healing process. Several other techniques to repair cracks, fissures, and fractures of the beak have been described in the literature (Rosskopf and Woerpel, 1996; Altman, 1997; Clipsham, 1997). The techniques include repairing the beak using pins, stainless wire, and acrylic and epoxy resins.

REFERENCES

Altman RB: Beak repair, acrylics. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Clipsham R: Beak repair, rhamphorthotics. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Denbow M: Gastrointestinal anatomy and physiology. In Whittow GC, editor: *Sturkie's avian physiology*, London, 2000, Academic Press.

Harcourt-Brown N: Orthopaedic and beak surgery. In Harcourt-Brown N, Chitty J, editors: *BSAVA manual of psittacine birds*, Quedgeley, Gloucester, UK, 2005, BSAVA.

Lintner ML: *The beak trim*, Proceedings of the Association of Avian Veterinarians, 2013, Jacksonville, Florida, 2013.

Lumeij LT: Gastroenterology. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Orosz S: Avian dermatology. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Rosskopf WJ, Woerpel RW: Beak repair and surgery. In Rosskopf WJ Jr, Woerpel RW, editors: *Disease of cage and aviary birds*, ed 3, Baltimore, MD, 1996, Williams & Wilkins.

Samour J: Clinical management of captive raptors. In Harrison GJ, Lightfoot T, editors: *Clinical avian medicine*, Lake Worth, FL, 2005, Spix Publishing.

Schnellbacher RW, Stevens AG, Mitchell MA, et al: Use of a dental composite to correct beak deviation in psittacine species, *J Exot Pet Med* 19(4):290–297, 2010.

Speer BL: *Trans-sinus pinning to address scissor-beak deformities in psittacine species*, Proceedings of the Annual Conference of the Association of Avian Veterinarians Australian Committee, 2002.

FEATHER REPAIR

Jaime Samour

The integrity of the primary feathers (remiges) and tail feathers (rectrices) is of the utmost importance for flight performance in species destined for release back into the wild or for birds of prey used in the ancient sport of falconry. Invariably, feathers tend to suffer bends or fractures during captivity in rescue and rehabilitation centers, from poor aviary or holding cage design, or in crash landings or fighting with quarry during training or hunting, or from inadequate transport and handling practices.

Feather repair or *imping*, a medieval falconry term, is the art of repairing bent or fractured feathers. Modern imping techniques involve total or partial feather replacement or splinting. For a total and partial feather replacement, it is necessary to procure a feather from the same species, side (e.g., wing feather), size, sex, age, color, and markings. Rescue and rehabilitation centers, falconry enthusiasts, and medical facilities devoted to raptor medicine usually maintain a collection of

molted feathers and feathers obtained from carcasses. It is recommended to perform feather examination and feather repair procedures under general inhalant anesthesia.

MATERIALS AND INSTRUMENTS USED FOR IMPING

- Scissors: small, sharp, fine-pointed, 130 and 160 mm long
- Guillotine nail cutter: medium and large (e.g., cat and dog size)
- Imping needles: made from steel hairpins, long 50 mm × 1.5 mm, medium 40 mm × 1.5 mm, short 30 mm × 1.5 mm, fine 25 mm × 1 mm
- Hair clips (aluminum): 90 mm long
- Nail files: coarse and fine
- Cyanoacrylate glue: 2-g tube
- Epoxy glue: fast-setting (5 min), twin tubes, 4.2 g
- Sodium bicarbonate powder: fine
- Pliers: curved, fine-tipped, 130 mm long
- Wire cutters: 200 mm long
- Flat metal file: 150 mm long, fitted with a plastic handle
- Barbecue skewers or bamboo pegs of different diameters
- Knitting needles with aluminum pegs: no. 14 (2 mm), 13 (2.2 mm), 12 (2.5 mm), 11 (3 mm), 10 (3.2 mm), and 9 (3.7 mm)
- Utility knife: fine-pointed interchangeable blades
- Cardboard cards: thin, square 5 × 5 cm

BENT FEATHER REPAIR

The treatment to correct bent feathers varies according to the severity of the damage. Usually in mild cases bent feathers can be straightened by applying steam directly onto the shaft of the feather, for a couple of minutes, from a boiling kettle. In more severe cases, it is necessary to apply hot water directly onto the affected area and straighten by digital manipulation. Moderate or severe bending might occur at different levels of the shaft. Bent feathers are repaired using the splinting technique (Figs. 8-40 to 8-47). The bend is straightened on its dorsoventral or laterolateral axis with a pair of fine-tipped, curved electrician pliers. The ventral aspect of the feather shaft is then split 12 to 15 mm in either direction from the bend. A small amount of cotton wool is placed in the newly created groove and secured firmly with

FIGURE 8-40 Materials and instruments used by the author for feather repair.

FIGURE 8-41 Ventral view of the right wing of an adult female saker falcon (*Falco cherrug*) showing the first three feathers (Arab falconry classification) with partial fractures resulting in severe bends at the midshaft. This type of fracture commonly occurs when the falcon strikes the perch or the ground during a fight with its quarry.

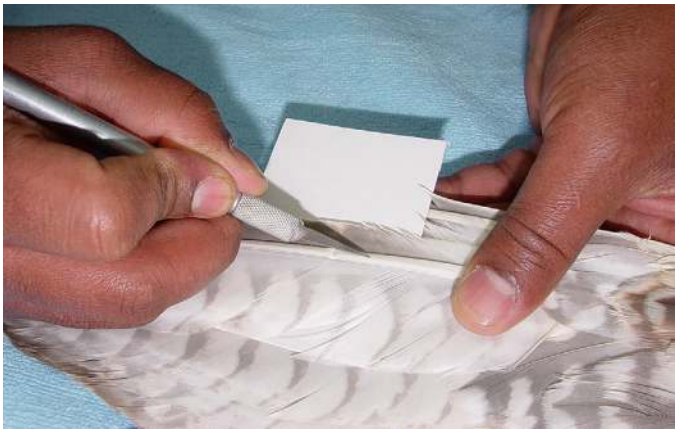


FIGURE 8-42 The first step consists of making a longitudinal incision over the fracture extending about 10 mm in either direction of the fracture. The incision should include only the upper layer of the feather shaft.

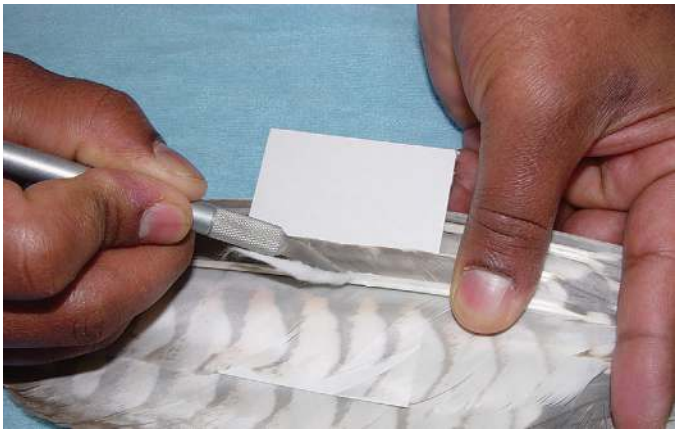


FIGURE 8-43 The bend is straightened up on its dorsoventral and laterolateral aspects using a pair of fine-tipped curved pliers. An elongated wad of cotton wool is snugly inserted into the feather shaft using the blunt side of a utility knife.

FIGURE 8-44 A small amount of methacrylate glue is placed directly on the incision, impregnating the wad of cotton wool. Pressure is then applied laterally over the incision using a pair of fine-tipped curved pliers until the glue sets. When dried the glue-impregnated cotton wool provides a strong inner reinforcement to the damaged feather shaft.

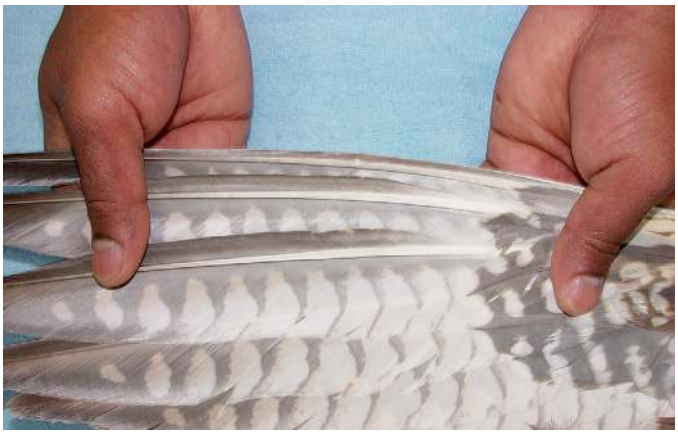


FIGURE 8-47 The feathers are now repaired with a strong reinforcement splint formed over the original fracture. The external splint is translucent, making coloring unnecessary.

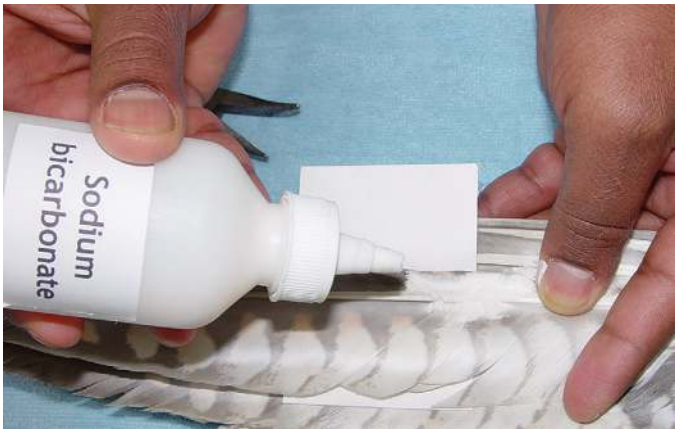


FIGURE 8-45 The surface of the feather shaft around the incision is roughened using a fine nail file. A thin layer of methacrylate glue is applied over the area. A small amount of sodium bicarbonate is sprinkled directly onto the glued surface. The powder binds with the glue, creating a strong cement-like layer over the bend.

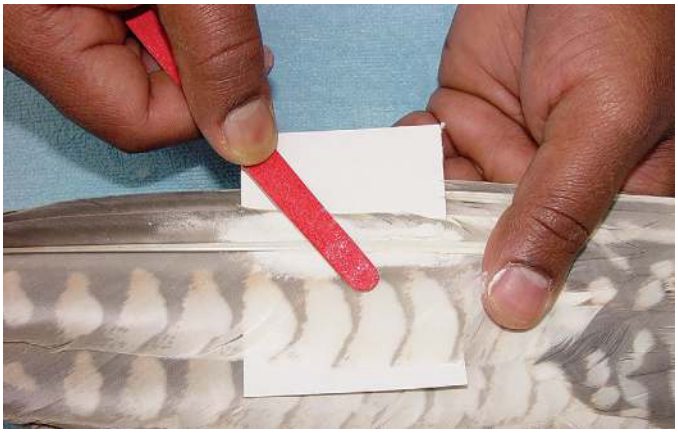


FIGURE 8-46 The upper surface of the newly created layer is filed using a fine nail file. The procedure can be repeated two or three times to create a thicker layer, should this prove necessary.

cyanoacrylate glue. The glue, when combined with the cotton wool, creates a strong inner reinforcement mesh. The ventral surface of the feather shaft around the bend is roughened with a fine nail file. A thin layer of cyanoacrylate glue is smeared onto the site approximately 10 mm on either side of the bend. A small amount of sodium bicarbonate is sprinkled directly onto the freshly glued surface. 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 surface and the edges of the newly created layer are filed with a fine nail file. The external splint is translucent, making the need for coloring unnecessary. Very often during feather repair a small amount of cyanoacrylate glue may spill onto the barbules, creating an undesirable unilateral or bilateral crust around the repair site. The excess cyanoacrylate glue can be removed using a dedicated glue solvent (e.g., Glue Remover, Henkel Loctite Ltd., Winsford, Cheshire, UK). Such products can be applied directly onto the crust and work it through the barbules using a fine hypodermic needle and a brush.

PARTIAL FEATHER REPLACEMENT

Partial feather replacement is indicated if the fracture has occurred at the midshaft or at the distal end of the feather. If the fracture is complete and the feather fragment is missing, a similar fragment must be procured from a donor feather. Conversely, if the fragment is still available, this can then be reattached. In both cases, the ends of the fragments are smoothed out with fine-pointed scissors and a fine nail file to make a near perfect joint. A previously prepared imping needle, made from a steel hair fastener, of suitable length and diameter is carefully inserted in both fragments to make a narrow channel. A fine-diameter drill bit can also be used for the same purpose. The needle is then fixed to the fragment with a small amount of cyanoacrylate glue. The fragment is attached to the rest of the feather and checked for correct alignment. Additional glue is then applied to the free end of the needle of the fragment, which is then attached to the rest of the feather (Figs. 8-48 and 8-49).

Pressure should be applied over the imping site with fine-tipped electrician's pliers for approximately 30 s to allow the glue to set. The dorsal and ventral aspects of the fracture line are then filed with a fine

FIGURE 8-48 This falcon suffered fractures of the first three primary (Arab falconry) feathers, with loss of the distal fragments. Similar fragments must be procured from donor feathers to maintain bilateral symmetry and to ensure adequate flying performance.

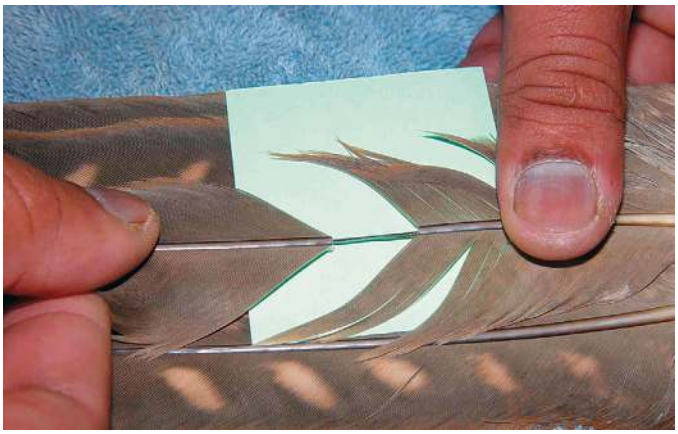


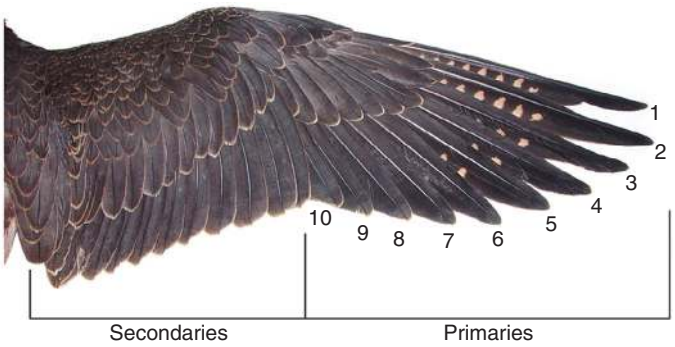
FIGURE 8-49 This peregrine falcon (*Falco peregrinus*) suffered a fracture of a deck feather, with loss of the distal fragment. The fragment was fixed using a previously prepared imping needle manufactured from a steel hair fastener.

nail file. In partial replacement, it is strongly recommended that a ventral external splint, fabricated from multiple layers of cyanoacrylate glue and sodium bicarbonate, is applied in addition to the method described earlier to produce a more satisfactory and efficient result. The dorsal aspect of the imping site can be colored, if necessary, with a marker pen. The main disadvantage of this method is that the feather tends to break near either tip of the rigid imping needle when subjected to stress. Cutting up the tips of the imping needle after making the channel considerably reduces the incidence of splitting or fracturing at either end of the imping needle.

TOTAL FEATHER REPLACEMENT

Total feather replacement is indicated when the feather is fractured at the proximal section of the feather shaft (Figs. 8-50 to 8-59). After examination and determining the number of feathers for replacement, the area should be prepared. First, the covert feathers are deflected backward and held in place using 1-inch 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. The new feather is placed in

FIGURE 8-50 Materials, instruments, and equipment used by the author for total feather replacement.



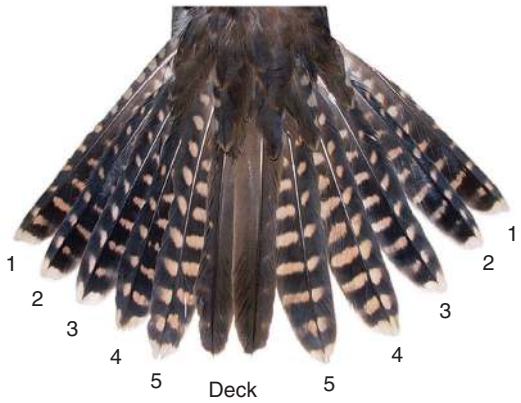


FIGURE 8-51 Arab falconry classification of wings and tail feathers.

FIGURE 8-52 Ventral view of the left wing of an adult female saker falcon showing a fracture of the second feather (Arab falconry classification), with loss of the distal one third segment.

FIGURE 8-53 A suitable feather is procured, taking into consideration the species, sex, age, and size of the individual and the color and markings of the feather. The feather is measured and cut making sure bilateral symmetry is maintained.

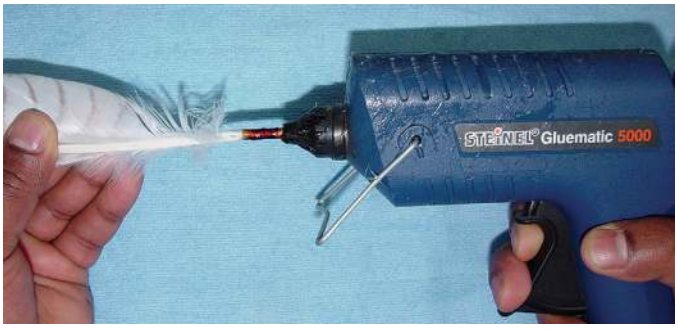


FIGURE 8-54 A small amount of hot glue is applied directly into the feather shaft of the newly selected feather. An aluminum imping needle of suitable diameter is promptly inserted into the shaft, as the glue tends to set in seconds.

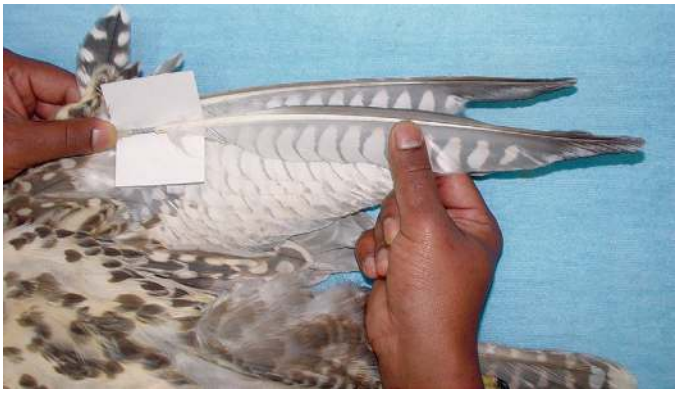


FIGURE 8-55 The feather is then inserted into the shaft to double check the length and correct angle.

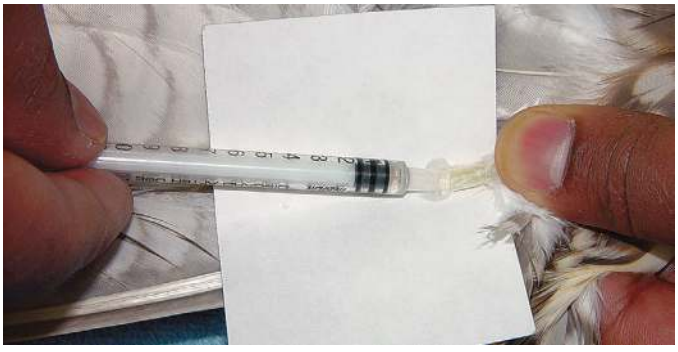


FIGURE 8-56 Rapid-setting epoxy glue is then prepared and inserted into the shaft using a tuberculin syringe. Note that a small card has been placed underneath the working site to avoid smearing glue on the adjacent feathers.

FIGURE 8-57 The new feather is then placed into the shaft and any surplus of epoxy glue is wiped off using cotton buds.

FIGURE 8-58 A small amount of sodium bicarbonate is sprinkled onto the working site to bind with any glue residue.

FIGURE 8-59 Hair clips are placed to ensure that the feathers remain in the correct position while the glue sets.

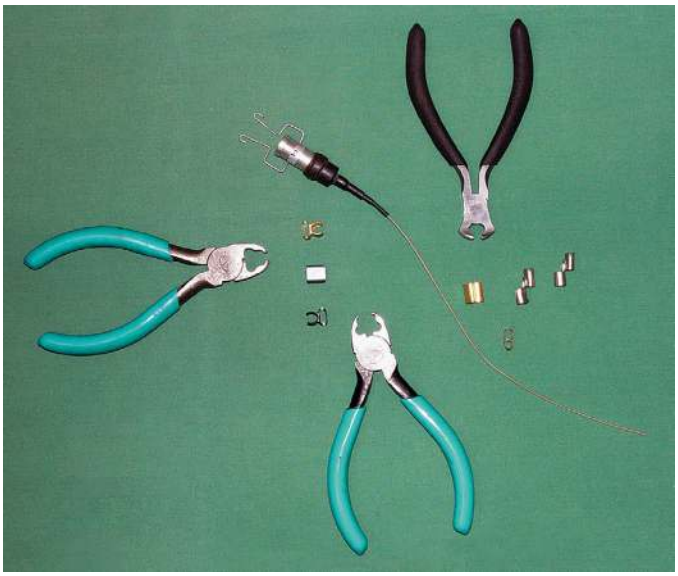


FIGURE 8-60 Radio transmitters and the use of telemetry equipment are now widely used in the sport of falconry. A tail kit integrates a small, battery-operated radio transmitter fitted with a long aerial mounted on a stainless-steel clip and a tail feather clamp. Different types of feather clamps and fixing tools, together with a radio transmitter, are shown here.

position to assess the length making sure that bilateral symmetry is maintained with the opposite wing when replacing a wing feather or the opposite side when 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. For instance, 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; Fig. 8-51). This general ornithological knowledge is essential to performing 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 injected with a 1 mL tuberculin syringe into the shaft, making sure the feather is properly aligned. Fractures at the proximal end of growing feathers (e.g., "green" or "blood" feathers) are corrected by first applying a plug made of cotton wool for a few days and waiting until the feather follicle has stopped bleeding. Small crocodile forceps, commonly used to retrieve foreign objects from the ear, are suitable for removing the cotton plug. When the bleeding has stopped and the feather shaft is judged to be fully grown, a new feather is attached using the technique described earlier.

A small piece of cardboard should be placed under the imping site to prevent the glue from smearing onto adjacent feathers. Any glue residue around the imping site should be cleaned using cotton buds and a small amount of sodium bicarbonate is then applied to bind with any excess glue and to complete the cleaning process. The wing or the tail should then be closed in the natural anatomic position and all the feathers held in place with hair clips until the glue is set.

It is paramount to ensure that bamboo pegs made from barbecue skewers are immersed in water before use, as dried, old bamboo pegs are very brittle. Ideally, bamboo pegs should be made from freshly cut green bamboo stems if these are readily available. Conversely, lightweight aluminum pegs can be used for the same purpose. These can be manufactured from knitting needles commonly found in tailors and handicraft shops and are available in different diameters. These are the pegs the author prefers to use. Such aluminum pegs offer several advantages. First, knitting needles are cheap and readily available on the market. Second, pegs made from these needles can be cut and shaped using common hardware tools, e.g., strong wire cutters and flat files. Third, and most important, the pegs can be bent to ensure adequate alignment. A useful and comprehensive review of the different techniques used for feather repair and feather replacement has been described by Remple (2003) and Samour (2005). A novel technique concerning feather cysts and repair under the skin has also been described by Remple (2003).

FIGURE 8-61 Fixing a Marshall tail feather clamp to a peregrine falcon using a dedicated Marshall fixing tool. The clamp is fixed onto a central deck feather. Some operators prefer to add a small amount of epoxy glue to ensure an adequate grip.

Figures 8-60 to 8-62 illustrate a related technique for fitting telemetry equipment to the tail feathers of a falcon to locate it while free flying.

FIGURE 8-62 The radio transmitter has been fixed on the tail of the falcon. The use of telemetry equipment allows the falconer to locate the falcon if it flies away during a training session or while on a hunting trip.

REFERENCES

Remple JD: *Feather tricks: practical pearls for the avian practitioner*, Proceedings of the European Association of Avian Veterinarians, Tenerife, Spain, 2003.

Samour J: Clinical management of captive raptors. In Harrison GJ, Lightfoot T, editors: *Clinical Avian Medicine*, Lake Worth, FL, 2005, Spix Publishing.

BEAK REPAIR

Melodiya Nyela Magno, Jaime Samour

The beak, rostrum (Latin meaning beak), or bill is an integral part of the integument of birds. Apart from some reptiles (e.g., Cheloniidae), the bird belongs to the only group in the Animal Kingdom that has evolved an edentulous beak adopting different sizes and shapes related to its function.

The different shapes of the beak include:

- Flat (ducks)
- Crossed (crossbills)
- Chisel (woodpeckers)
- Conical (cardinals)
- Long curved (Ibis)
- Probe (hummingbirds)
- Strong curved (birds of prey)
- Strong hooked (parrots)
- Long tapered (storks)
- Short slender (doves)
- Narrow long colorful (Ramphastidae)
- Long strong with casque (most Bucerotidae)

The different functions of the beak include:

- Feeding
- Pecking (cranes)
- Cracking delicate (finches)
- Cracking strong (parrots)
- Probing (snipes)
- Filtering (flamingos)
- Tearing and shredding (birds of prey)
- Spearing (herons)
- Scooping (pelicans)
- Grooming
- Killing
- Fighting
- Courtship
- Handling objects
- Heat exchange (Ramphastidae)
- Sexual dimorphism (American avocet—*Recurvirostra americana*)

Because of the complex structure and diverse functions, beaks are prone to trauma and deformities.

TRAUMA

Most injuries of the beak are divided into fractures (simple, depressed, avulsion, and with bone deficit) and luxations (palatine bone luxation mainly seen in macaws).

Fractures are the most common type of injuries of bird beaks. These may be the result of fights with companions or accidents within the aviary. Several techniques have been described using Steinmann pins and cerclage stainless-steel wires. The most commonly used procedure involves the placement of two or more pins on opposite sides of the fractured fragments and then placing a cerclage wire in a figure of eight looped around the pins. Polymethyl methacrylate bone cement is then applied to the site to hold the pin and wires firmly.

In old, contaminated injuries, repair should be delayed until the area is adequately cleaned and necrotic material is debrided. It may be necessary to repeat this procedure over several sessions until all necrotic material is removed and healthy tissue is observed underneath. The surrounding area is then roughened using a Dremel hand drill. The procedure may produce significant damage to the very sensitive structures of the beak because of friction heat if not used with great care. Dental acrylic is applied over the defect onto a dust-free, clean, roughened area. The tissue will granulate and reepithelialize under the acrylic. The acrylic can then be stained using commercially available stain kits for a more natural appearance.

Eventually, the acrylic patch will slough off because the normal growth of the beak leaves healthy tissue underneath. If a portion of the beak is missing, plastic mesh splinting tape is used to provide scaffolding when the acrylic is applied. The beak is cleaned and roughened before placing strips of plastic mesh over the defect and securing them using cement or resin. Once the defect is covered, acrylic is used to cover the mesh and normal surrounding areas. Conversely, methacrylate glue also can be applied to the surface of the deficit or over the plastic mesh and then sprinkled liberally with fine-grain baking soda (sodium bicarbonate) creating a cement-like layer. The procedure can be repeated several times until a suitable thickness is reached. This technique is widely used in falcon medicine to create a cap over the exposed phalangeal terminal end, after claw detachment, thus arresting hemorrhage and preventing further trauma.

Luxation of the palatine bone occurs mainly in macaws and is characterized by permanent hyperextension of the premaxilla. This may occur when the palatine bone locks onto the vomer preventing the bird from closing its beak. Reduction is accomplished by inserting a Steinmann pin through the base of the premaxilla to use as a handle. The beak is hyperextended freeing the palatine bone from the vomer. Ventral pressure is then applied to the pin and the upper beak gently closed. To prevent relapse, the jugal bone is sutured to the infraorbital rim.

DEFORMITIES

Scissors beak (lateral deviation of the premaxilla to the right or to the left) and mandibular prognathism (mandible longer than the premaxilla) are two types of beak deformity commonly observed in birds. Scissors beak is more often seen in macaws and mandibular prognathism is more often seen in cockatoos. Causes of scissors beak include the method used for hand-feeding, malnutrition, genetics, incubation faults, upper respiratory tract infections, and trauma.

Scissors beak can be corrected using physical therapy if instituted early in the development of the chick and when the bone is still soft and pliable. The procedure involves frequent application of gentle pressure toward the opposite side of the abnormal deviation. This is commonly performed at feeding time. If the bird is too old or physical therapy fails to achieve its objective, a more invasive procedure would be required. There are two basic methods used to correct scissors beak, namely the ramp prosthesis and the trans-sinus pinning techniques. The ramp prosthesis involves the placement of an acrylic ramp onto the surface of the lower beak exerting an opposing force to the deviation of the upper beak. The trans-sinus pinning technique involves the placement of a Steinmann pin transversely into the frontal bone making a hook or a loop at the end of the pin and on the opposite side of the deviation. A relatively deep furrow is made on the tip of the beak. A rubber band is then placed from the hook of the pin to the furrow made on the beak securing this with cement. This system results in constant tension gradually correcting the deviation. This is commonly left for 2 to 3 months

Mandibular prognathism can be corrected either by using a technique usually referred to as scaffolding or by applying a prosthesis. The scaffolding is used primarily in older birds with severe deformities. The scaffolding is made using nonthreaded Steinmann pins, which are placed similarly to trans-sinus pinning. Both sides of the pin are bent symmetrically rostrally and dorsally close to their exit with hooks added at the terminal ends. A second pin is inserted at the distal end of the beak also with hooks at the end. Rubber bands are then connected to the hooks correcting the deviation. The prosthesis is used to create a functional cap extending distally from the cere exerting pressure at the occlusal ledge of the maxilla. The lower beak should not extend out and beyond the prosthesis.

Excessive growth is a deformity of the beak that is corrected using hand saws fitted with fine metal blades, a Dremel hand drill, and fine-grain hand files for the final touches. In large birds with solid beaks (e.g., hornbills) and in extreme cases, the use of an orthopedic oscillating saw may be necessary. The methods used to reshape the beak have been described in the section "Beak Trimming" early in this chapter. Also see additional images of beak trimming and other beak deformities in the same section. Examples of beak repair are shown in Figures 8-63 to 8-65.

For specific information on the techniques and the materials used for the repair of beak injuries and beak deformities the reader is referred to Rosskopf and Woerpel, 1996; Altman, 1997; Clipsham, 1997; Olsen 2003; Harcourt-Brown, 2005; Bennett, 2013; and van Zeeland, 2014.

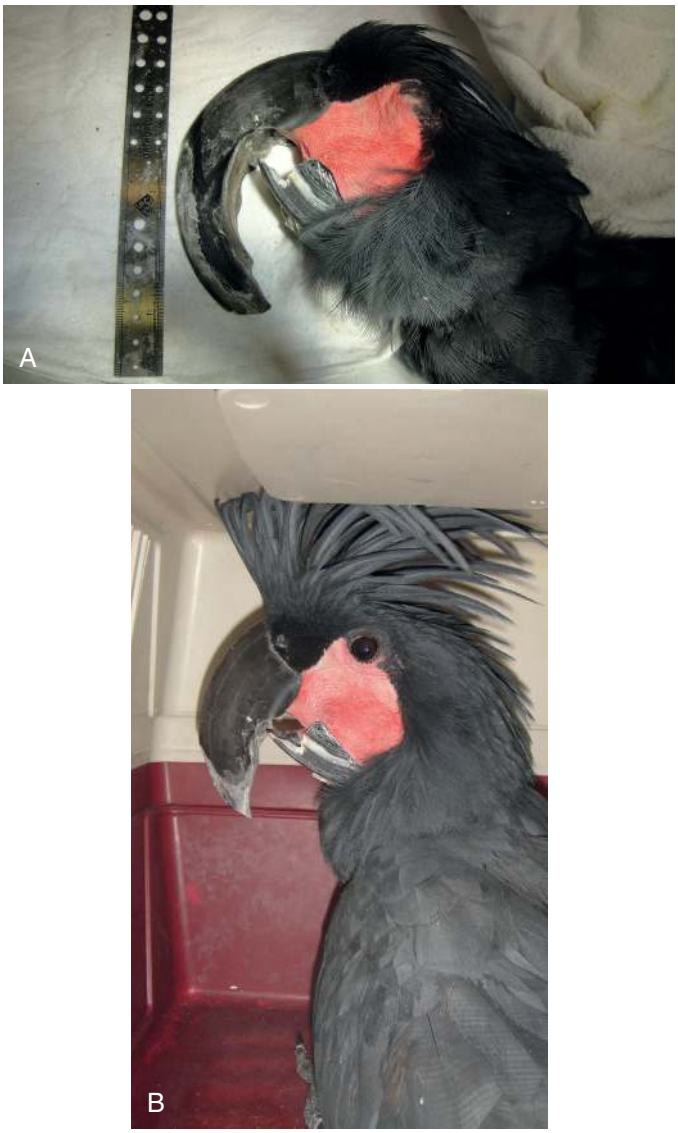


FIGURE 8-63 **(A),** Excessive growth of the upper beak of a palm cockatoo (*Probosciger aterrimus*). This bird was housed in a breeding aviary and the keeping staff failed to detect the condition until it was very advanced. **(B),** The same cockatoo after the beak was cut and reshaped using a Dremel hand drill. In such extreme cases, several sessions over a period of time are sometimes necessary until normality is restored.

FIGURE 8-64 **(A),** Female rhinoceros hornbill (*Buceros rhinoceros*) with a severely damaged casque attained during a fight with a male companion. **(B),** Close-up view of the beak showing the fracture site with loss of fragments, multiple cracks, and a long fissure.

FIGURE 8-64, cont'd **(C),** The casque of hornbills is hollow, therefore, to repair the fracture the cavity was filled using multiple pellets of a thermoplastic resin. The area was then smeared with cyanoacrylate glue and liberally sprinkled with sodium bicarbonate to provide a smooth finish.

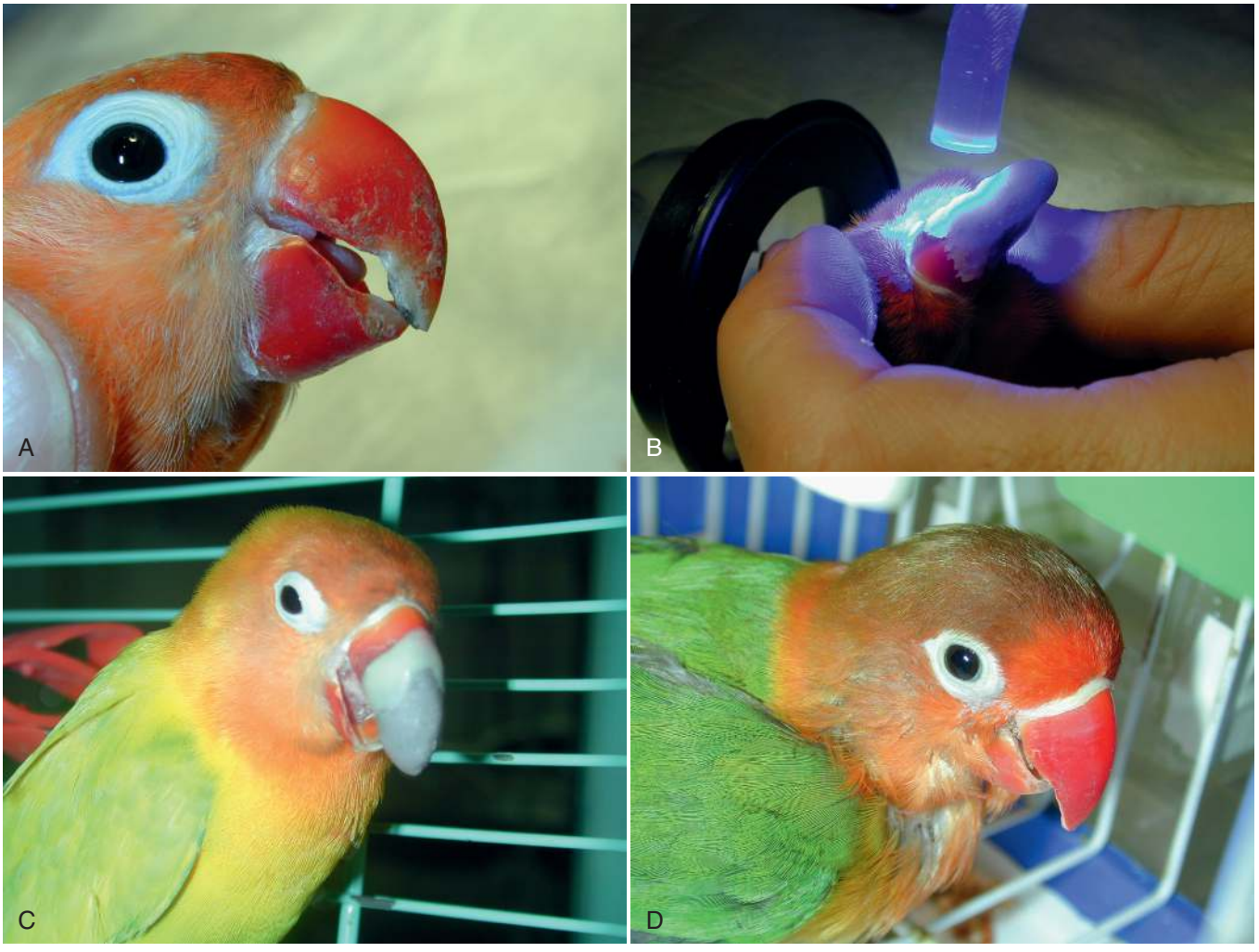


FIGURE 8-65 **(A),** A lovebird (*Agapornis fischeri*) with mild prognathism. The beak was carefully reshaped using a Dremel drill. **(B),** A small hole was drilled in the upper beak to insert a stainless-steel wire. A prosthesis was fixed on the upper beak anchoring it to the wire previously placed. This prosthesis was made using a dental photosensitive polymer. **(C),** The lovebird with the completed prosthesis. This was replaced 2 weeks later. **(D),** The prosthesis was removed from the lovebird after 1 month displaying a normal beak and free from prognathism. (Courtesy A. Montesinos.)

REFERENCES

Altman RB: Beak repair, acrylics. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Bennett AR: *Avian beak surgery*, Proceedings of the Western Veterinary Conference, Exp 29, 2013.

Clipsham R: Beak repair, rhamphorthotics. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Harcourt-Brown N: Orthopaedic and beak surgery. In Harcourt-Brown N, Chitty J, editors: *BSAVA manual of psittacine birds*, Quedgeley, Gloucester, UK, 2005, BSAV.

Olsen GH: Oral biology and beak disorders of birds, *Vet Clin North Am Exot Anim Pract* 6:505–521, 2003.

Rosskopf WJ, Woerpel RW: Beak repair and surgery. In Rosskopf WJ Jr, Woerpel RW, editors: *Disease of cage and aviary birds*, ed 3, Baltimore, MD, 1996, Williams & Wilkins.

van Zeeland YRA: *Surgery in pet birds part II—orthopedic surgery*, Internacionalizace výuky veterinární medicíny jakocesta na evropský trh práce projekt è, Brno, Czech Republic, 2014.

9

Trauma-Related Medical Conditions

EYE AND EYELID INJURIES AND OCULAR DISEASES

Alejandro A. Bayón del Rio

Over the past 25 years, birds have become an important part of veterinary ophthalmology consultations, not only because they are frequently kept as pets, but also because there is an increasing awareness of the environment and the conservation of nature and its species. Good vision is especially important in birds because of the direct influence on flight, feeding, and breeding. Prevalence of ocular diseases in birds has been reported to be 7.6%, and in the case of birds of prey, it is even higher, up to 14% to 26% (Korbel, 2000; Bayón *et al.*, 2005).

OCULAR DISEASES

Congenital Diseases

Palpebral malformations described (though infrequent) have been partial agenesis of the top eyelid in birds of prey (peregrine falcon—*Falco peregrinus*; Kern *et al.*, 1985) and ankyloblepharon and cryptophthalmos (merging of the eyelid margins; Fig. 9-1) in nymphs (Buyukmihci *et al.*, 1990).

The presence of microphthalmia can be the result of congenital malformation or acquired phtisis bulbi. Bilateral microphthalmia has been described in ducks; bilateral anophthalmia in budgerigars; and microphthalmia with the presence of cataracts, retinal dysplasia, and retinal detachment in birds of prey (Buyukmihci *et al.*, 1988; Kern, 1989). Phtisis bulbi is frequently observed secondary to uveitis, but is less evident in mammals because of the presence of scleral ossicles. Therefore the differentiation between microphthalmia and phtisis depends on the clinical history and the ocular exploration (Kern, 1989; Williams, 1994).

Corneal dermoids were reported in a goose in which feathers grew out of the aberrant dermal tissue on the lateral aspect of the globe. Unilateral corneoconjunctival dermoid was successfully removed from a blue-fronted Amazon parrot (*Amazona aestiva*; Williams, 1994). Ectropion and secondary exposure keratitis has been reported in cockatiels (*Nymphicus hollandicus*).

Exophthalmos

Orbital diseases that cause exophthalmos are infrequent in birds. If present, the anterior displacement of the globe can be from orbital trauma (fractures of the cranium), inflammation (orbital post-traumatic hemorrhage—very infrequent in birds compared with mammals), inflammation of Harderian gland in psittacines, infections such as orbital abscesses that spread from the paranasal sinuses in Amazon (*Amazona* sp.) and African grey parrots (*Psittacus erithacus*), neoplasias such as lymphoreticular neoplasms, adenocarcinoma and osteosarcoma in budgerigars (*Melopsittacus undulatus*), glioma of the optical nerve, sarcomas, chromophobe pituitary adenoma, and medulloepithelioma in nymphs (Arnall, 1961; Rambow *et al.*, 1981; Dukes and Pettit, 1983; Paul-Murphy *et al.*, 1985).

Treatment in cases of trauma and inflammatory diseases should include systemic corticosteroid and antiinflammatory drugs and systemic and topical antibiotics (bacitracin-neomycin-polymixin B). If an orbital abscess is suspected systemic antibiotics should be administered for 14 days. If this therapy fails to improve the condition, an alternative antibiotic or reassessment of the etiology of the exophthalmos should be considered (Kern, 1989; 1999).

If neoplasia is suspected or proved by biopsy or aspirate tests, exenteration of the orbit is recommended, with enucleation and removal of all orbital soft tissues. Before surgery, the likelihood of metastasis or an association with primary systemic disease or neoplasia elsewhere should be assessed. Workup should include radiology, hematology, and serum chemistry testing (Kern, 1989; 1999).

Periocular Swelling

Local or diffuse periocular swelling can progress from pathologies that involve the eyelids, conjunctiva, infraorbital sinus, or nasal gland (in birds that have them). Eyelid disorders can be traumatic or infectious and include lacerations, hemorrhages, and abrasions. The most frequently described causes of infectious eyelid diseases are blepharoconjunctivitis from *Staphylococcus* in Amazon parrots (Shimakura *et al.*, 1981); fibrinopurulent blepharoconjunctivitis secondary to *Escherichia coli*, *Streptococcus* spp. (Cheville *et al.*, 1988), *Pasteurella multocida* (Olson, 1980), and *Actinobacillus* spp. in waterfowl and *Plasmodium spp.* in canaries; bilateral supraorbital abscesses from *Pseudomonas* spp. in Amazon parrots (Tully and Carter, 1993) and poxvirus in dove, Amazon parrot (Graham and Halliwell, 1981; MacDonald *et al.*, 1981; Poonacha and Wilson, 1981), canaries (Fig. 9-2; Johnson and Castro, 1986), mines, parrots (Poonacha and Wilson, 1981), and birds of prey; and blepharitis from parvovirus in geese and protuberant lesions for *Knemidokoptes pilae* in parakeets. Vitamin A deficiency can cause conjunctival hyperkeratosis and swelling of the eyelids similar to that caused by poxvirus (Jacobson *et al.*, 1983; Williams, 1994).

Treatment of poxvirus lesions should include topical antibiotic ophthalmic ointments to reduce the incidence of secondary infections with bacteria or fungus. Systemic antibiotics may also be required in severely affected birds. Early lesions should be flushed with dilute antiseptic solutions. Once scabs have formed they should not be removed. It may be beneficial to soften the scabs, however, with hot or cold compresses soaked in nonirritating baby shampoo. It has been reported that prophylactic vitamin A supplementation of exposed birds decreases the severity of infection (Kern, 1989; Williams, 1994).

Infraorbital sinusitis is frequent in psittacines, causing swelling of the medial and ventromedial areas to the eyeball. It is generally associated with diseases of the respiratory system. The inflammation of the salt gland is seen as puffiness over the globe and can be caused by ingestion of water with high levels of sodium (Kern, 1989).

FIGURE 9-1 Cryptophthalmos.

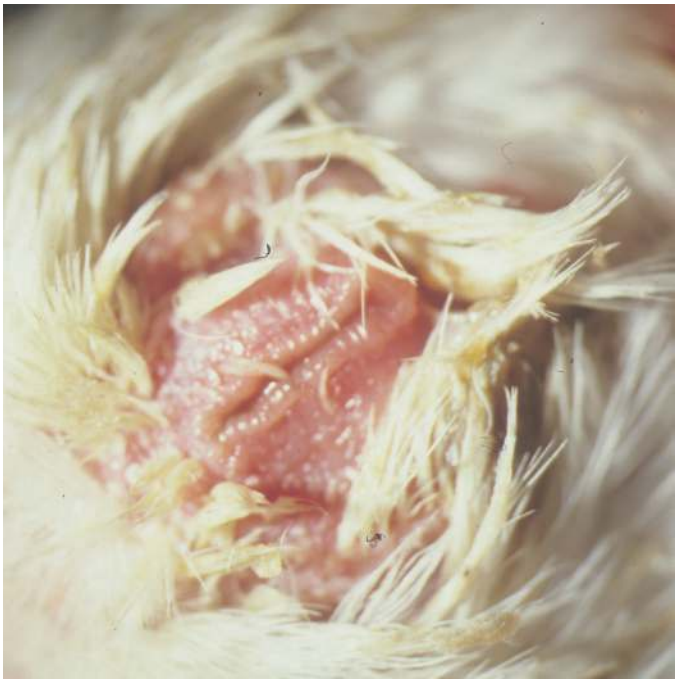


FIGURE 9-2 Poxvirus in a canary (*Serinus canaria*).

Palpebral and conjunctival neoplasms are not frequent. A benign tumor of basophil cells has been reported in an African grey parrot, histiocytic sarcoma in an owl, mastocytoma and cystadenoma in yakos, and subconjunctival hibernoma in a goose (Williams, 1994).

Conjunctivitis, Keratoconjunctivitis, and Keratitis

Conjunctivitis can be classified clinically into three groups. The first group is caused by strictly local factors, such as localized conjunctival infection or foreign bodies. The second group is caused when conjunctivitis is a manifestation of periorbital or orbital disease, which is mainly related to sinusitis. In the third group conjunctival hyperemia is caused by septicemia. Almost any organism causing systemic infection can result in conjunctivitis. A careful examination of the bird for upper respiratory disease is mandatory in determining the causes of ocular discharge or conjunctival hyperemia. Exposure to cigarette smoke, chemical fumes, and other airborne environmental toxins should always be considered in the differential diagnostics of conjunctivitis, with or without signs of upper respiratory disease (Williams, 1994).

FIGURE 9-3 African grey parrot (*Psittacus erithacus*) with chlamydial conjunctivitis.

Conjunctivitis in passerines can be caused by Newcastle virus, paramyxovirus, poxvirus, cytomegalovirus, *Streptococcus* spp., Erysipelothrix rhusiopathiae, *Clostridium botulinum*, *Mycobacterium avium* serotype 2, *E. coli*, *Pseudomonas aeruginosa*, *Bordetella avium*, *Chlamydia psittaci* (Fig. 9-3), *Mycoplasma* spp., *Candida albicans*, *Aspergillus* spp., herpesvirus, adenovirus, and pneumovirus.

The most frequent parasites are spirurids (*Ceratospira* and *Oxyspirura*) in psittacines and mynahs and trematodes (*Philophthalmus gralli*), nematodes (*Thelazia* in Senegal parrot; *Poicephalus senegalus*), and *Setaria* in passerines. Conjunctivitis can also be secondary to poor hygienic sanitary conditions from ammonia in feces (Kern, 1989; Williams, 1994).

Treatment of conjunctivitis consists of the administration of topical antibiotics (bacitracin-polymixin B, neomycin, tetracycline, and chloramphenicol) for 14 days. When respiratory signs also exist an antibiotic should be administered parenterally. If there are parasites, they can be removed with forceps under topical anesthesia. If this is impossible, the use of ivermectin must be considered.

Keratoconjunctivitis, a frequent ocular disease in psittacines, can be caused by chlamydiosis, trauma in the cage, and vitamin A deficiency. Corneal crystalline deposits of unknown etiology have been found in 8.7% of nymphs, parakeets, and Amazons on necropsy and observed in Amazon parrots with poxvirus. Bilateral deposits of cholesterol have been observed in the corneal stroma in falcons. Punctate keratitis associated with sinusitis has been described in Amazons in which the lesions were bilateral and the most common presenting signs were blepharospasm and clear ocular discharge (Kern, 1989; Williams, 1994). Keratitis can be difficult to resolve, but, as a rule, topical antibiotics and corneal bandaging techniques provide a sterile environment and time for the corneal epithelium to heal. By extrapolation from other species, anticollagenases should be used in deep ulcers, especially in hotter climates, where corneal melting may cause rupture of the globe (Bayón *et al.*, 2007). Also, antibiotic treatment and the use of topical nonsteroidal antiinflammatories can be useful in punctate keratitis (Kern, 1989).

Uveitis

The principal causes of uveitis in birds include trauma, infections, immune-mediated inflammation, and neoplasia (Schmidt *et al.*, 1986; Tsai *et al.*, 1993; Davidson, 1997; Seruca *et al.*, 2011). A blunt or sharp trauma can cause anterior and/or posterior uveitis, frequently associated with hemorrhage (Fig. 9-4). In several studies performed in birds of prey hyphema and hypopyon were the most frequent clinical findings (Fig. 9-5), and fibrin clots, iridocyclo-dialysis (tearing of the iris), lens injuries, and fractures of scleral ossicles also can be present. Anterior uveitis can also develop secondary to corneal ulcers as in mammals (Korbel, 2000; Bayón *et al.*, 2005).

The most important infectious etiologies of uveitis are those secondary to viruses that affect birds, such as encephalomyelitis, Marek's disease, and poxvirus. Septicemia from any bacterial infection (*P. multocida*, *Salmonella*, and *Mycoplasma gallisepticum*) also may cause uveitis. Mycotic endophthalmitis has been associated with disseminated aspergillosis and candidiasis in budgerigars. Toxoplasmosis has caused chorioretinitis and blindness in canaries and raptors. Clinical signs of the anterior uveitis in birds include photophobia, blepharospasm, corneal edema, Tyndall effect, vitreous opacity, hypotony or secondary glaucoma, miosis, dyscoria, thickening or discoloration of the iris, rubeosis iridis, and anterior or posterior synechiae. In posterior uveitis, diffuse or focal retinal edema, hemorrhages near the pecten, retinal detachment, and vitreous opacity can be present. The visual function can be diminished or abolished.

Sequelae of chronic uveitis include diffuse corneal edema, posterior synechiae causing pupillary occlusion and iris bombe, anterior and posterior synechiae (Fig. 9-6), secondary glaucoma (Fig. 9-7), cataracts, and retinal atrophy or detachment and blindness (Davidson, 1997).

Iatrogenic uveitis and reduction in intraocular pressure can be mistaken for each other clinically. The latter can appear as decubitus during anesthesia in psittacines and birds of prey, caused by pressure applied to the eye provoking a forced drainage of the aqueous humor (Kern, 1989).

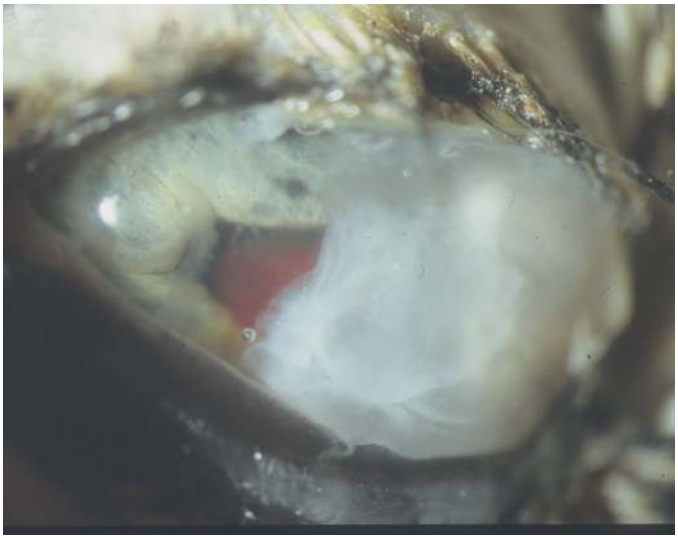


FIGURE 9-4 Hyphema in the anterior chamber and corneal perforation in a raptor.

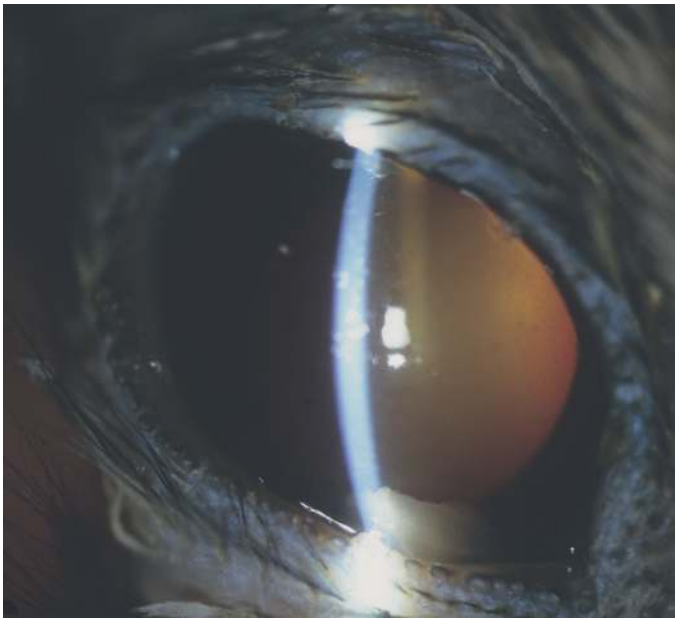


FIGURE 9-5 Traumatic uveitis and hypopyon.

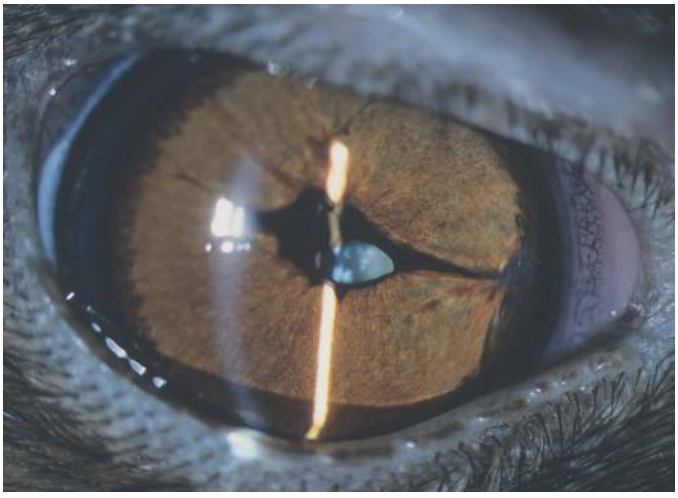


FIGURE 9-6 Anterior synechia, cataract, and uveitis in an eagle.

FIGURE 9-7 Traumatic buphthalmos in an eagle.

Treatment of uveitis should include elimination of the cause, control of inflammation, preservation of the pupil, and prevention and treatment of secondary glaucoma. Prescribe topical antibiotics and topical and systemic antiinflammatory agents (steroids and nonsteroids) for symptomatic treatment of inflammation. It is very important to watch for systemic side effects (polyuria, polydipsia, etc.). Mydriasis cannot be achieved by topical therapy (e.g., atropine) in birds, and pupil preservation is best accomplished by adequate control of inflammation (Kern, 1989; Williams, 1994).

Glaucoma

Glaucoma can be secondary to uveitis and hyphema (Fig. 9-7). It has been provoked, experimentally, in birds maintained in constant light or darkness. Primary glaucoma is not described in birds because of the width of the iridocorneal angle. Buphthalmia is not very severe in glaucoma because of the inflexibility of the sclera because of the ossicles. The safety and efficacy of topical and oral medications routinely prescribed for mammals for glaucoma are unproven for birds. In severe cases, enucleation of the eye or placement of an intrascleral prosthesis is the treatment of choice in birds (Kern, 1989; Williams, 1994).

Cataracts

Cataracts in birds are congenital and secondary to nutritional deficiencies, trauma, age (senile cataracts), and retinal degeneration (Bellhorn, 1973; Kern, 1989; Millichamp, 1991; Williams, 1994).

Ocular development anomalies such as microphakia, development of lenses with abnormal material (lentoids, as well as dysplasia and detachment of retina), and hypoplasia of the optic nerve have been described in birds of prey. Hereditary cataracts have also been described in canaries (Fig. 9-8) with an autosomal recessive model of transmission (Yorkshire and Norwich canaries). Other etiologies of cataracts include avian encephalomyelitis, maternal vitamin E deficiency, trauma, dinitrophenol (chicks), and chronic uveitis (Kern, 1989; Williams, 1994).

Lens opacities can be capsular, cortical, and/or nuclear. Hypermature cataracts can differ from incipient ones by their shrinking, capsular wrinkling and the presence of a deeper anterior chamber. Luxation or subluxation of the lens can accompany cataracts primarily or as a secondary form. Treatment of cataracts is, as in mammals, done by phacoemulsification, except in very small eyes (Kern *et al.*, 1984). In birds with very small eyes, complete blindness is not an exclusive reason for euthanasia, because a canary blind from bilateral cataracts can lead a normal life in its cage providing the cage interior is not modified.

Retinopathy and Optical Neuropathy

Retinal diseases include congenital anomalies, degeneration, inflammation, and detachment. Congenital retinal dysplasia has been described in birds of prey (falcons, fundamentally; Murphy *et al.*, 1985). Idiopathic degeneration has been reported in a budgerigar (Tudor, 1978). Trauma is the most frequent reason for injuries of the posterior segment (Fig. 9-9; Millichamp, 1991), especially in birds of prey, although it can be associated with bacteremia or viremia. The chorioretinitis lesions caused by toxoplasmosis are easy to identify in birds of prey (Fig. 9-10; Korbel, 2000).

Optic neuropathy can be associated with congenital anomalies (hypoplasia of the optic nerve associated with cataract), trauma

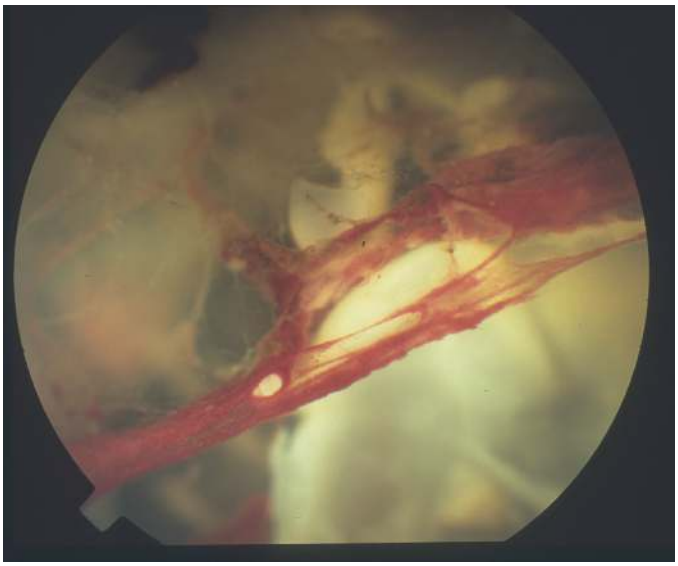


FIGURE 9-9 Intravitreal hemorrhage and white fibrin clots in a raptor.

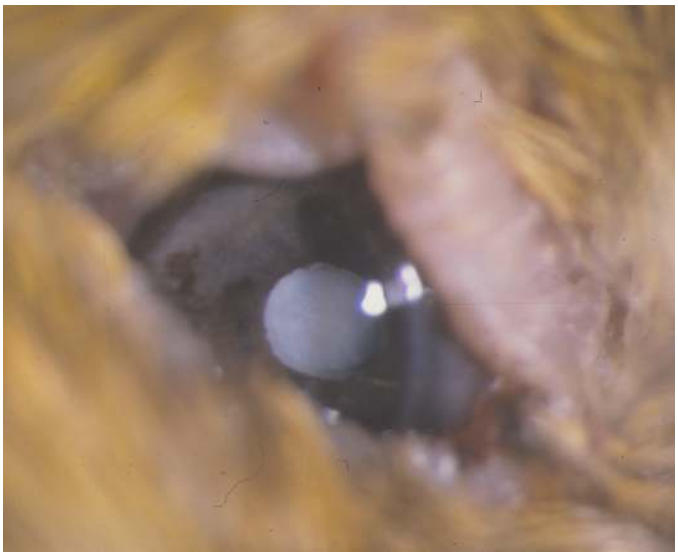


FIGURE 9-8 Typical cataract in a canary (*Serinus canaria*).

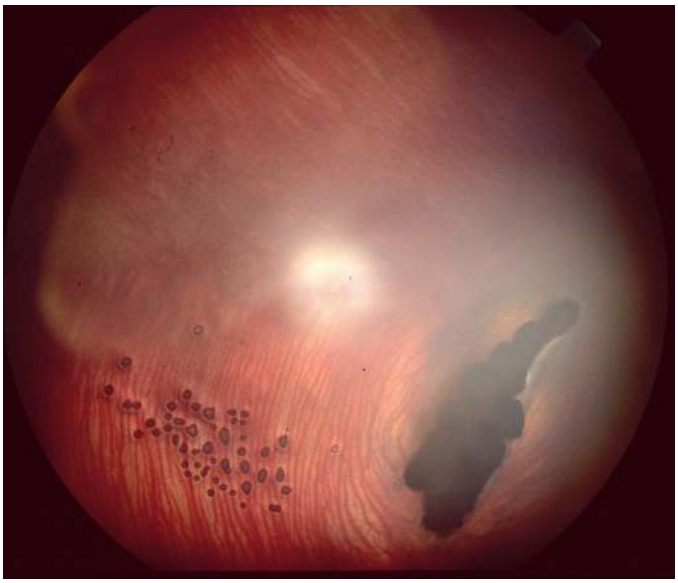


FIGURE 9-10 Chorioretinitis secondary to toxoplasmosis in an owl.

(frequently provoking neuritis), and neoplasia (chromophobe adenoma of the pituitary gland causing compression of the optic nerve, atrophy, and blindness in parakeets and nymphs). The focal or multifocal retinopathies and the optic neuropathies do not interfere with vision, with only minor change in direct pupillary light reflexes. Wide lesions in the retina and optic nerve cause loss of vision, variable degrees of mydriasis, and a deficiency in the direct pupillary light reflex. Unilateral lesions cause anisocoria. Cataracts frequently develop secondary to retinal degeneration. The signs observed in the retina include depigmentation, hyperpigmentation, and loss of the choroidal vascular patterns. Intraretinal hemorrhages and hemorrhage around the pecten can frequently be observed after ocular trauma, as well as retinal edema and detachment, which appear as gray and slightly elevated areas. Acute traumatic retinopathy and/or optic neuropathy are treated systemically with broad-spectrum antibiotics and antiinflammatory doses of corticosteroids (Kern, 1989; Williams, 1994; Korbel, 2000; Bayón *et al.*, 2005).

Blindness with Normal Pupil Sizes and Responses

Malformations, trauma, infections (bacterial, viral, parasitic, and fungal), and intoxications (i.e., hexachlorophene causing reversible blindness in parakeets) can cause central nervous system signs (Kern, 1989). Clinical signs include variable unilateral or bilateral blindness with normal resting pupil sizes and pupillary light responses. Other reported signs are disorientation, seizures, and abnormal behavior. Therapy should be oriented to treat etiology (i.e., antiinflammatory corticosteroid after trauma).

REFERENCES

Arnall L: Anesthesia and surgery in cage and aviary birds. II. A regional outline of surgical conditions, *Vet Rec* 73:173–178, 1961.

Bayón A, Albert A, Almela RM, et al: Ocular disorders in raptors in a 3-year-period (2002-2004). *Proceedings ESVO-ECVO Meeting*, pp 117, 2005.

Bayón A, Almela RM, Talavera J: Avian ophthalmology, *EJCAP* 17:253–265, 2007.

Bellhorn RW: Ophthalmologic disorders of exotic and laboratory animals, *Vet Clin North Am* 3:345–356, 1973.

Buyukmihci NC, Murphy CJ, Paul-Murphy J, et al: Eyelid malformation in four cockatiels, *J Am Vet Med Assoc* 196:1490–1492, 1990.

Buyukmihci NC, Murphy CJ, Schulz T: Developmental ocular disease of raptors, *J Wildl Dis* 24:207–213, 1988.

Cheville NF, Tappe J, Ackermann M, et al: Acute fibrinopurulent blepharitis and conjunctivitis associated with *Staphylococcus hyicus*, *Escherichia coli*, and *Streptococcus sp.* In chickens and turkeys, *Vet Pathol* 25:369–375, 1988.

Davidson MG: Ocular consequences of trauma in raptors. In Murphy CJ, Paul-Murphy J, editors: *Seminars in avian exotic pet medicine—ophthalmology*, Philadelphia, PA, 1997, WB Saunders.

Dukes TW, Pettit JR: Avian ocular neoplasia—a description of spontaneously occurring cases, *Can J Comp Med* 47:33–36, 1983.

Graham DD, Halliwell WH: Viral diseases of birds of prey. In Fowler ME, editor: *Zoo and wild animal medicine*, Philadelphia, PA, 1981, WB Saunders.

Jacobson ER, Mladinich CR, Clubb S, et al: Papilloma-like virus infection in an African gray parrot, *J Am Vet Med Assoc* 183:1307–1308, 1983.

Johnson BJ, Castro AE: Canary pox causing high mortality in an aviary, *J Am Vet Med Assoc* 189:1345–1347, 1986.

Kern J: Disorders of the special senses. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1989, WB Saunders.

Kern TJ: Exotic animal ophthalmology. In Gelatt KN, editor: *Veterinary ophthalmology*, Philadelphia, PA, USA, 1999, Lippincott Williams & Wilkins, pp 1273–1305.

Kern TJ, Murphy CJ, Heck WR: Partial upper eyelid agenesis in a peregrine falcon, *J Am Vet Med Assoc* 187:1207, 1985.

Kern TJ, Murphy CJ, Riis RC: Lens extraction by phacoemulsification in two raptors, *J Am Vet Med Assoc* 185:1403–1406, 1984.

Korbel RT: Disorders of the posterior eye segment in raptors—examination procedures and findings. In Lumeij JT, Remple JD, Redig PT, et al, editors: *Raptor biomedicine III*, Lake Worth, FL, 2000, Zoological Education Network.

MacDonald SE, Lowenstine LJ, Ardans AA: Avian pox in blue-fronted Amazon parrots, *J Am Vet Med Assoc* 179:1218, 1981.

Millichamp NJ: Exotic animal ophthalmology. In Gelatt KN, editor: *Veterinary ophthalmology*, ed 2, Philadelphia, 1991, Lea & Febiger.

Murphy CJ, Kern TJ, Loew E, et al: Retinal dysplasia in a hybrid falcon, *J Am Vet Med Assoc* 187:1208–1209, 1985.

Olson LD: Ophthalmia in turkeys infected with *Pasteurella multocida*, *Avian Dis* 25:423–430, 1980.

Paul-Murphy JR, Lowestine L, Turrel JM, et al: Malignant lymphoreticular neoplasm in a African gray parrot, *J Am Vet Med Assoc* 187:1216–1217, 1985.

Poonacha KB, Wilson M: Avian pox in pen-raised bobwhite quail, *J Am Vet Med Assoc* 179:1264, 1981.

Rambow VJ, Murphy JC, Fox JG: Malignant lymphoma in a pigeon, *J Am Vet Med Assoc* 179:1266–1268, 1981.

Schmidt RE, Becker LL, Mc Elroy JM: Malignant intraocular medulloepithelioma in two cockatiels, *J Am Vet Med Assoc* 189:1105–1106, 1986.

Seruca C, Molina-López R, Peña T: Ocular consequences of blunt trauma in two species of nocturnal raptors (*Athene noctua* and *Otus scops*), *Vet Ophthalmol* 15:236–244, 2011.

Shimakura S, Sawa H, Yamashita T, et al: An outbreak of ocular disease caused by staphylococcal infection in Amazon parrots (*Amazona aestiva*) imported into Japan, *Jpn Vet Sci* 43:273–275, 1981.

Tsai SS, Park JH, Hirai K, et al: Eye lesions in pet birds, *Avian Pathol* 22:95–112, 1993.

Tudor DC: Retinal atrophy in a parakeet, *Vet Med Small Anim Clin* 73:1456, 1978.

Tully TN, Carter TD: Bilateral supraorbital abscesses associated with sinusitis in an orange-winged Amazon parrot (*Amazona amazonica*), *J Assoc Avian Vet* 7:157–158, 1993.

Williams D: Ophthalmology. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

SPINAL INJURIES

Jesus Naldo

Spinal injuries are usually mechanical damage to the vertebral column that could lead to compression of the spinal cord resulting in temporary or permanent changes in the normal motor, sensory, or autonomic functions. These can range from mild overextension of the spine to subluxations and fractures. In birds, spinal injuries are commonly the result of trauma. Gunshot wounds, collisions with vehicles, crashing against glass windows or poles within aviaries, and severe crash landings have all been implicated. Radiography, magnetic resonance imaging, computed tomography scans, and scintigraphy are all useful advanced imaging techniques for identifying these lesions.

The intervertebral discs of birds differ from those of mammals. They consist of a fibrocartilaginous central region surrounded by a "C"-shaped synovial cavity that extends around the dorsal and lateral margins of the disc. A fibrocartilaginous, wedge-shaped meniscus protrudes into the joint cavity from the dorsal and lateral margins (Emerson, *et al.*, 1990). This zygapophyseal joint has hyaline cartilage with an intervening synovial cavity. With intervertebral disc rupture, this meniscus is driven into the spinal canal along with the fibrocartilaginous disc material (Bennett, 1994).

The junction of the fixed synsacrum with the more flexible portion of the thoracolumbar spine is a location susceptible to mechanical stress and vertebral subluxation (Bennett, 1994). This condition is commonly diagnosed in falcon species (Fig. 9-11, *A, B*). In broiler chickens, a congenital defect of the vertebral facets of T6 and T7 allows ventral displacement of T7, producing spondylolisthesis and varying degrees of spinal cord compression (Wise, 1970).

Neck dislocation or fracture is the loss of continuity of the cervical section of the spine. Neck fractures are debatable subjects as clinicians suggest fractures are not possible in birds because of the compact anatomic structure of the avian cervical vertebrae. Injuries that may result in dislocation or fracture to the cervical vertebral column are traumatic in origin and usually associated with birds crashing into the fence or the roof of their enclosures. Both neck fractures and dislocations have been diagnosed in a collection of bustards (Figs. 9-12 to 9-14). In most cases, affected birds were found dead beside the fence or wall of their aviary. External examination usually revealed bruising on the skin of the neck and swelling of the subcutaneous tissue from hematoma. Bruising on the head is sometimes an accompanying finding. Initial diagnosis of dislocation or fracture was made through physical examination of the cervical vertebral column. Cervical dislocations and fractures were later confirmed through radiographic and postmortem examinations.

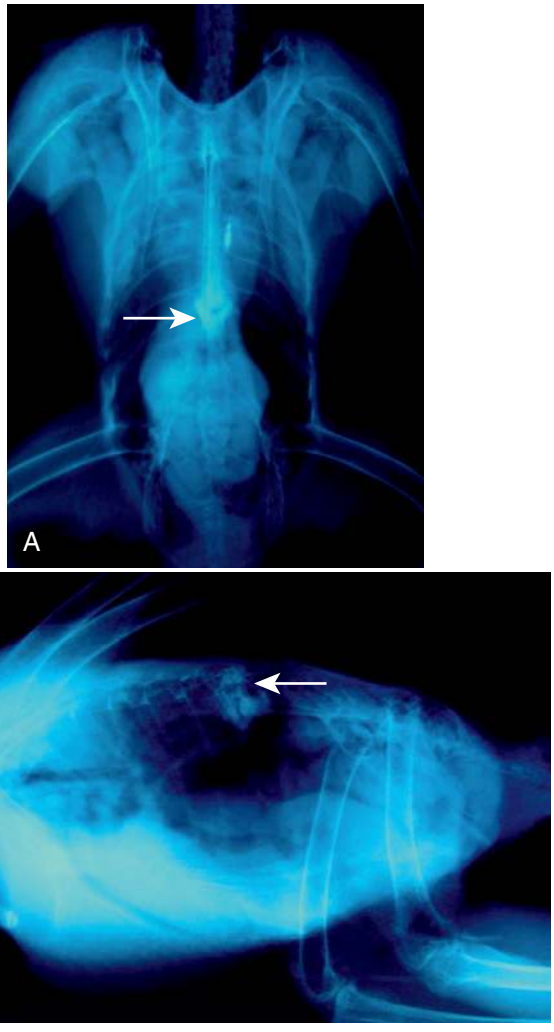


FIGURE 9-11 **(A),** Ventrodorsal survey radiograph of a saker falcon (*Falco cherrug*) that was presented because of lameness of both legs. There is an increased radiopacity in the thoracosynsacral junction (*arrowhead*). Such abnormality is commonly related to trauma. **(B),** Lateral survey radiograph of the same falcon. There is abscess formation subsequent to trauma in the thoracosynsacral junction (*arrowhead*) leading to spinal luxation.

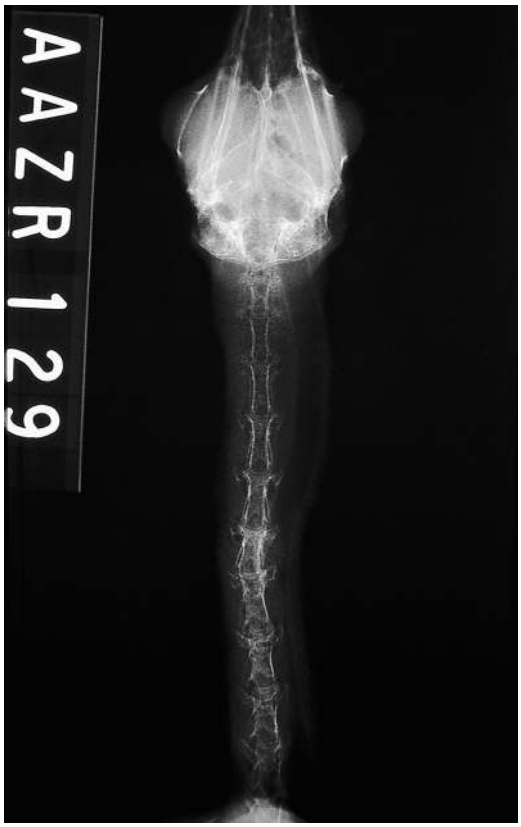


FIGURE 9-12 Ventrodorsal radiograph of an adult houbara bustard (*Chlamydotis undulata*) with dislocation on the middle portion of the cervical vertebral column.

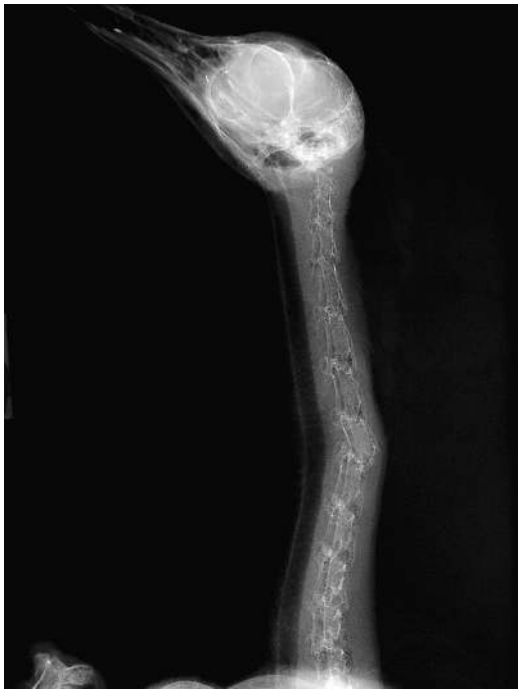


FIGURE 9-13 Lateral radiograph of the bird in Figure 9-3.

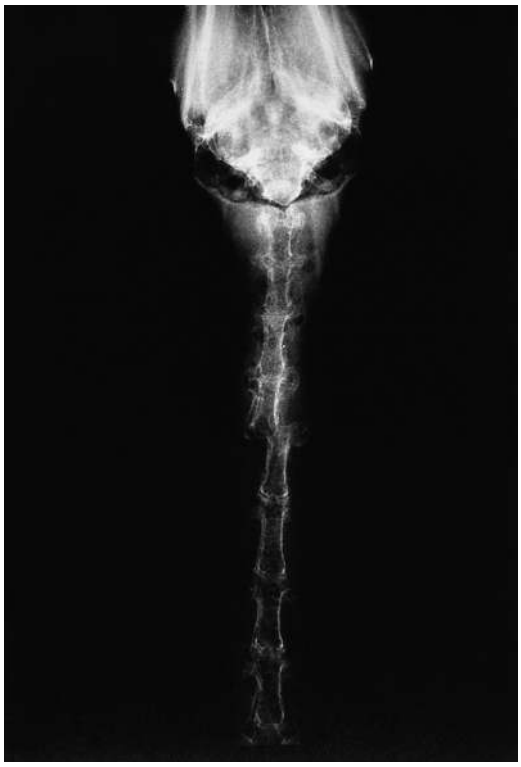


FIGURE 9-14 Houbara bustard (*Chlamydotis undulata*), adult. Dislocation between the 4th and 5th cervical vertebrae.

REFERENCES

Bennett RA: Neurology. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Emerson CL, et al: Ruptured intervertebral disc in a juvenile king penguin, *J Zoo Wildl Med* 21(3):345–350, 1990.

Wise DR: Spondylolisthesis ("kinky back") in broiler chickens, *Res Vet Sci* 11:447–451, 1970.

KEEL INJURIES

Melinda Cowan, Deborah Monks

Keel injuries are described as the loss of continuity of the skin and, very often, the adjacent pectoral muscles around the carina (Figs. 9-15 to 9-19). Severe injuries can also lead to trauma to the underlying bone of the region.

Blunt trauma and self-mutilation are the most common causes of keel injuries. The most common cause of keel trauma in captive parrots is improper wing trimming of the remiges, resulting in poor aerodynamic control. These birds repeatedly crash into walls, floors, and other surfaces, and split the skin overlying the carina. A number of factors have been implicated in this problem and include extensive or unilateral trimming of the primary flight feathers, trimming of the secondary flight feathers, and clipping of tail feathers. Young birds that have not developed flight skills before wing trimming are particularly at risk of injury. It is now thought that birds should have bilateral wing trims, starting with the outer primary feathers, and leaving as many of the secondary feathers as possible to allow the bird to "glide" to the ground rather than crash or spiral out of control.

In other species, such as bustards, storks, and cranes, keel injuries can be associated with repeated crashing against fences or walls of enclosures. Birds of prey used in the sport of falconry are very often involved in "crash landings." During training exercises, a falcon or a hawk may fail to grasp the lure offered very close to the ground or miss catching prey during hunting and crash onto the floor. Alternatively, they may crash into other structures.

When repairing these wounds, it is crucial that the underlying etiology is addressed. For instance, if a keel wound is from an improper wing trim, then simply suturing the wound (and not addressing the underlying repetitive trauma) is inviting surgical

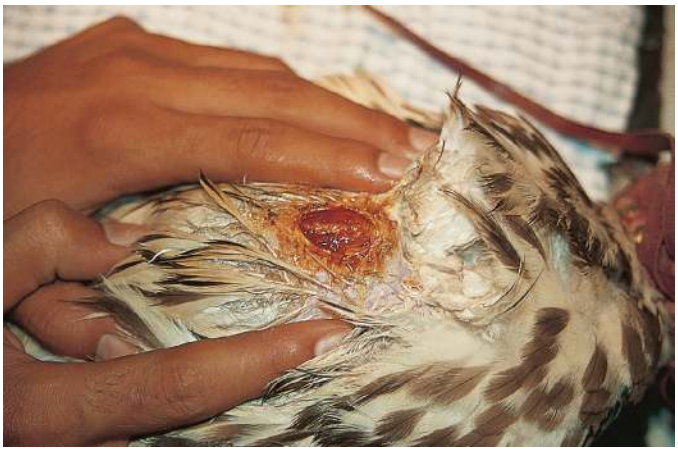


FIGURE 9-15 Recent open keel injury on a saker falcon (*Falco cherrug*) after a "crash landing." The bird suffered a severe cut on the skin and underlying tissues when it hit rocky ground.

FIGURE 9-16 Chronic open wound with associated fibrosis and infection on the keel region of a houbara bustard (*Chlamydotis undulata*). The lesion was caused by repeatedly crashing against the fence of the enclosure.

FIGURE 9-17 Cockatiel (*Nymphicus hollandicus*) attacked by a cat. Along with normal wound management, antibiosis is crucially important in this case.

FIGURE 9-19 Young Alexandrine parrot (*Psittacula eupatria*) with a keel injury and subcutaneous abscess secondary to an improper wing trim.

FIGURE 9-18 Bird after an intramuscular injection of doxycycline, which caused significant muscle necrosis.

dehiscence. If a keel injury is from flight-or-fight responses in caged bustards, then the husbandry needs to be examined to provide more security to the birds.

Self-mutilation seems to be mainly diagnosed in captive parrots, and in many cases wounds are observed around the keel region. The sharp beak of these species can inflict significant damage to soft tissue, and, in severe cases, underlying bone. Although many consider this a behavioral issue, a thorough clinician should always perform a full diagnostic evaluation to rule out physical or internal disease. While the primary etiology is being elucidated, it is usually necessary to protect the area using bandaging or collaring or both.

WING TIP INJURIES

Melinda Cowan, Petra Zsivanovits, Deborah Monks

Wing tip injuries are also a common occurrence in many bird species and are normally associated with trauma. The following sections are common classifications of wing tip injuries.

WOUNDS AND ULCERS

In aviary or free-flying birds, these injuries are normally caused by birds crashing into the fences or walls of their enclosures. In companion birds these injuries often occur from impact with cage bars, as sequelae to night frights, being captured, or other panic reactions. In birds that have excessively trimmed distal remiges, the exposed wing tip is predisposed to trauma. Repeated injury to the same site may lead to ulcerative wounds, fibrosis, and in extreme cases ankylosis of the carpal joint. Primary treatment consists of standard wound management including debridement, tension relief, and suturing (where possible). These injuries are often highly vascular and bandages assist wound management and provide protection. It is important to remember to anchor the bandage to the feathers, because bandages on the wing tip tend to slip off. It is also essential to correct the main cause of the injuries by minimizing disturbance and padding the walls of cages and enclosures. Care must be taken to avoid excessive tension on the wound, because this will predispose the bird to dehiscence and lymphatic drainage may be compromised, causing swelling that can further reduce blood supply to traumatized tissue. Secondary intention healing has to suffice in cases in which primary closure is not possible. Appropriate bandaging is essential throughout this process. To reduce the risk of joint fibrosis, regular frequent physiotherapy of the joint is required when using long-term bandaging (Figs. 9-20 to 9-22).

FIGURE 9-20 Carpal joint injury showing ulceration and associated mild fibrosis in a houbara bustard (*Chlamydotis undulata*).

FIGURE 9-21 Chronic carpal joint injury in a houbara bustard. Note the tumor-like growth on the ventral aspect of the joint from proliferative fibrosis. These types of lesions are also caused by repeated trauma to the wing tip by crashing against the wall or fences of enclosures.

FIGURE 9-22 Acute carpal joint injury in a houbara bustard. The wound is open and bleeding severely. Such injuries are susceptible to myiasis in tropical countries and should be sutured and a dressing and bandage applied.

FIGURE 9-23 "Blain" or bursitis of the carpal joint in a saker falcon (*Falco cherrug*). Lesions of this type are caused by repeated injury to the ventral aspect of the wing.

LUXATIONS AND FRACTURES

Fractures and luxations of the distal thoracic limb are covered in the orthopedic section (see Chapter 12). Luxations and fractures of the carpal joint are often the result of severe trauma (Figs. 9-20 to 9-22). Luxations of the carpal joint are best treated using external fixation devices. Chronic luxations are very often difficult to correct and usually result in partial or total ankylosis. Martin *et al.* (1993) reviewed eight cases of elbow luxation in birds of prey. More recently, Ackermann and Redig (1997) published a very useful paper on the surgical repair of elbow luxation in raptors. Fractures of the carpometacarpus are common in many species of bird, and this type of fracture is very often associated with injuries to the wing tip when juvenile birds begin experimenting with newly feathered wings. This is a common occurrence that goes largely undiagnosed in large terrestrial species such as cranes, storks, and bustards (J. Naldo, personal communication). Fractures of this type are best corrected by reduction and immobilization using an external splint, as suggested by Coles (1985), or a suitable bandage (Degernes, 1994; McCluggage, 1996, 1997).

BURSITIS

Inflammation of the synovial capsule of the carpal joint is often diagnosed in tethered birds of prey (Fig. 9-23; Cooper, 1978; Simpson, 1996). "Blain" is the old term used by falconers to describe this condition. Bursitis of the carpal joint is the result of repeated injury to the ventral aspect of the wing against the floor when attempting to escape from the approaching handler. The treatment of bursitis includes antibiotic and antiinflammatory therapy and poultices and dressings combined with suitable bandages.

EDEMA AND DRY GANGRENE SYNDROME

This syndrome is characterized by edema, transudate around the base of the distal primary feathers, and avascular necrosis–related clinical signs (Figs. 9-24 to 9-26). It is most commonly diagnosed in birds of prey. The exact etiology is unknown but cold weather appears involved, because the syndrome is usually diagnosed in temperate countries during the winter and early spring. However, frostbite alone does not appear to explain this lesion. Birds of prey kept tethered to perches, and therefore with forced inactivity, are more commonly affected compared with other birds kept in free-flying aviaries (Forbes, 1991; Simpson, 1996).

FIGURE 9-24 Severe injury to the wing tip on a Princess parrot (*Polytelis alexandrae*) from inadequate wing trimming and housing the parrot in a small cage. (Courtesy Bob Doneley.)

FIGURE 9-25 Early changes in the wing tip of a Harris hawk (*Parabuteo unicinctus*) affected by wing tip edema and dry gangrene syndrome. The condition is characterized by the formation of edema in the wing tips progressing gradually to dry gangrene if not treated promptly. (Courtesy Neil Forbes.)

Treatment involves trying to restore adequate blood circulation and the administration of antiinflammatory compounds, vasodilators such as pentoxifylline, and analgesia. Antibiotics can be used with secondary infection. Prognosis is usually reserved, as many affected birds slough the distal wing tip (Forbes, 1991; Simpson, 1996).

FROSTBITE

Frostbite can also cause a lesion similar to that described in the previous section and also lead to necrosis of the wing tip. Management, as with the aforementioned syndrome, revolves around three goals: maintaining blood supply to the affected region using COX-1 inhibiting antiinflammatory drugs and vasodilators, analgesia, and managing infection should it occur (Wellehan, 2003). It is advisable to wait at least 4 to 6 weeks before making the decision to amputate ischemic or necrotic tissue, as tissue changes can occur slowly.

FIGURE 9-26 More advanced case of edema and dry gangrene syndrome in a Harris hawk (*Parabuteo unicinctus*). This condition tends to affect first-year birds more often and commonly occurs in the winter. In the UK and other European countries with harsh winter temperatures, species more often affected include peregrine (*Falco peregrinus*), lanner (*F. biarmicus*), and lugger (*F. jugger*) falcons and Harris hawks. (Courtesy Neil Forbes.)

REFERENCES

Ackermann J, Redig P: Surgical repair of elbow luxation in raptors, *J Avian Med Surg* 11:247–254, 1997.

Coles BH: *Avian medicine and surgery*, Oxford, UK, 1985, Blackwell Science.

Cooper JE: *Veterinary aspects of captive birds of prey*, Saul, Gloucestershire, 1978, Standfast Press.

Degernes LA: Trauma medicine. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Forbes NA: Wing tip edema and dry gangrene in birds, *Vet Rec* 129(3):58, 1991.

Martin HD, Bruecker KA, Herrick DD, et al: Elbow luxations in raptors: a review of eight cases. In Redig PT, Cooper JE, Remple JD, Hunter DB, editors: *Raptor biomedicine*, Minneapolis, MN, 1993, University of Minnesota Press.

McCluggage DM: Bandaging and collaring. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, MD, 1996, Williams & Wilkins.

McCluggage DM: Bandaging. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Simpson GN: Wing problems. In Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *Manual of raptors, pigeons and waterfowl*, Cheltenham, UK, 1996, British Small Animal Veterinary Association.

Wellehan JFX: Frostbite in birds: pathophysiology and treatment, *Comp Cont Educ Pract Vet* 25(10):776–781, 2003.

WOUNDS

Deborah Monks, Melinda Cowan

Wounds are very common in birds. Inquisitive species such as parrots interact with toys, other birds, and predators, often leading to trauma. Hunting birds commonly present with lacerations from prey or impact injuries from the chase. Birds such as bustards and quail often startle, leading to self-trauma from impact with caging. Wild birds can suffer from entanglement injuries. Wounds may be open, closed, or have components of both (Figs. 9-27 to 9-36; Table 9-1).

FIGURE 9-27 Lanner falcon (*Falco biarmicus*) with a large transversal wound across the occipital area of the head. The wound occurred during an encounter with a larger falcon within a hunting vehicle. The borders of the incision were slightly swollen because of proliferation of granulating tissue. (Courtesy Dr. J. Samour.)

FIGURE 9-28 Large wound on the internal aspect of the tibial area of a leg in a saker falcon (*Falco cherrug*). Fights between falcons are not uncommon, particularly when they try to steal the food of eating companions. (Courtesy Dr. J. Samour.)

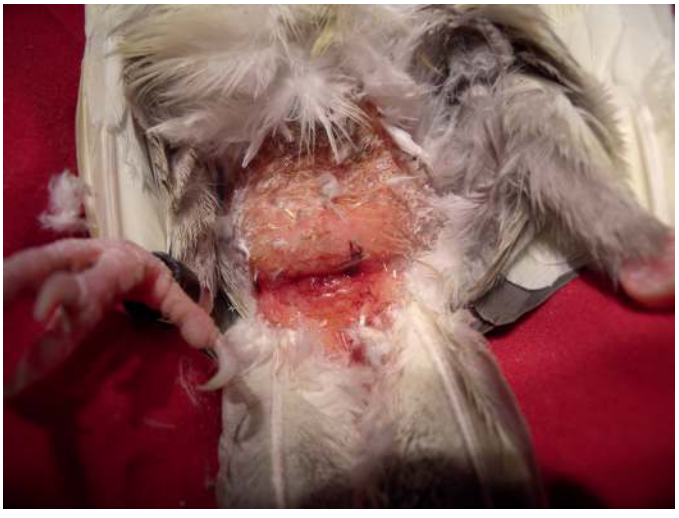


FIGURE 9-29 Parrot with a pygostyle split injury caused by an excessively severe wing trim. These lesions can extensively dissect into surrounding tissue.

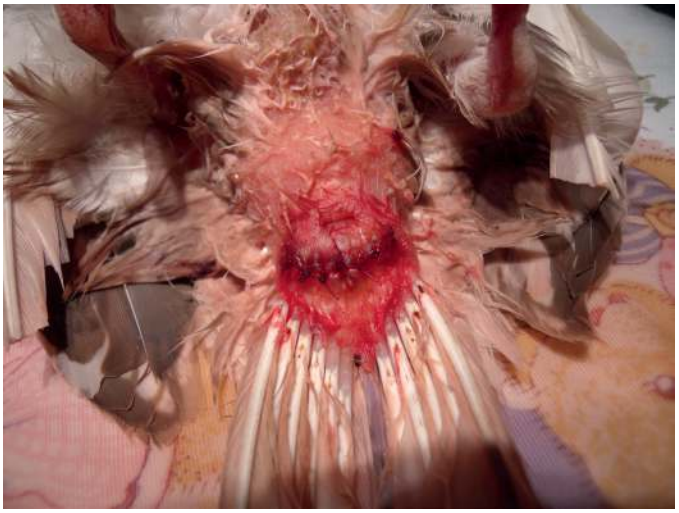


FIGURE 9-30 Same parrot as in Figure 9-29 with injury debrided and stitched. Simple interrupted sutures are recommended to assist with accurate reduction of dead space. If the repetitive crashing of the bird is not addressed by either imping new feathers or by changing the husbandry and handling techniques of the owner, then the wound will dehisce secondary to continual falling.

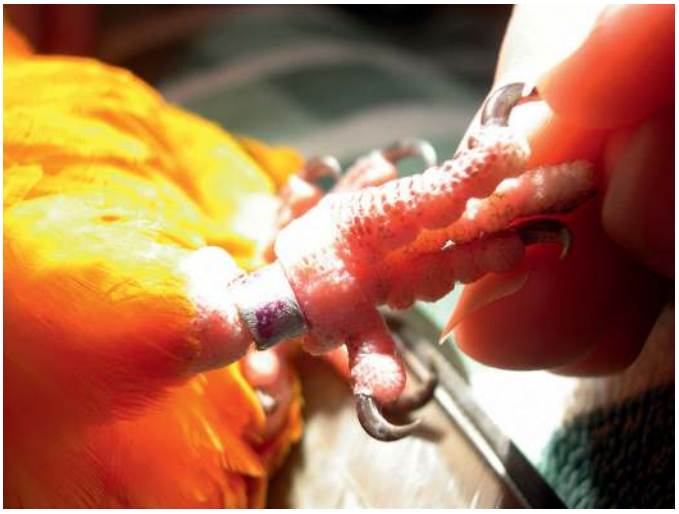


FIGURE 9-31 Conure with a limb constriction secondary to a split ring becoming more crimped. Ring constrictions from split rings are easier to manage, as the ring can usually be removed with little trauma to the limb. Ring constrictions resulting from swelling around entire nonsplit rings are more difficult. When the limb is swollen, as in this case, removing the ring without causing iatrogenic trauma can be challenging. Some practices use high speed burs to cut the rings in two places and protect the underlying tissue by sliding aluminum foil between the ring and the skin. Care should be taken using plier-type tools, as twisting of the ring is common, which can lead to bone fracture. Once the ring is removed, the wound is treated normally.

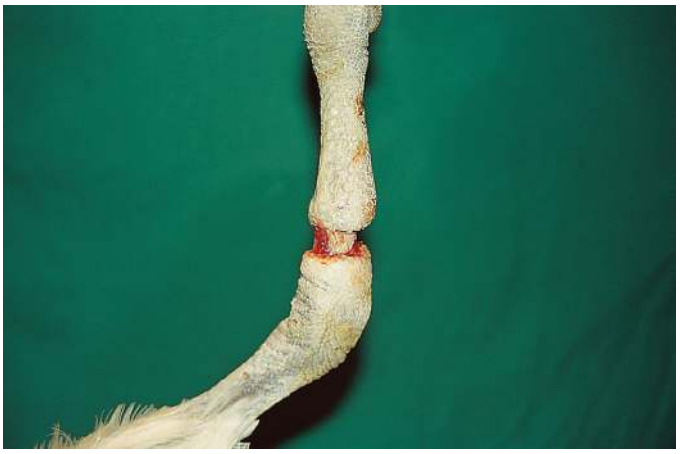


FIGURE 9-32 Ring constriction lesion on the leg of a kori bustard (*Ardeotis kori*) caused by the use of a small metal band. (Courtesy Dr. J. Samour.)

FIGURE 9-33 Australian white ibis (*Threskiornis molucca*) with fishing line entangled around the foot and digits. Some of the distal digit is necrotic. (Courtesy Hammy Forrest.)

FIGURE 9-34 Avascular necrosis secondary to pox.

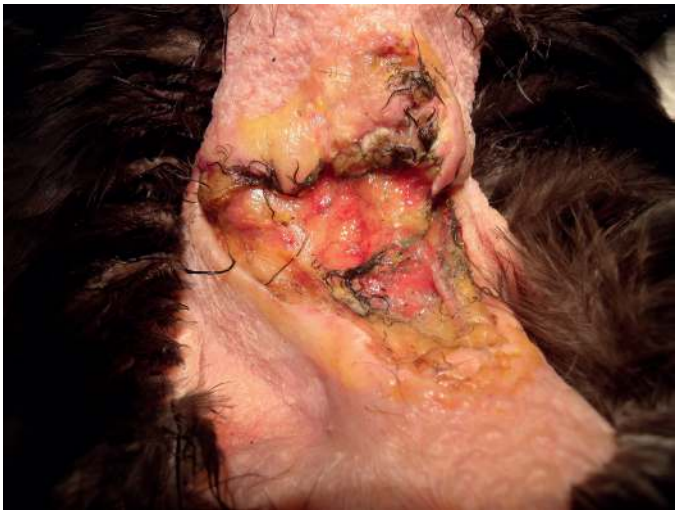


FIGURE 9-35 Wound on a chicken's (*Gallus domesticus*) neck secondary to a dog attack. These wounds require debridement and a closure with as little tension as possible. Antibiosis is important, as infection is common.

TABLE 9-1 Wounds: The Main Types

Type of Wound	Cause	Treatment
Incision/laceration	Surgery, tearing, fighting	Control bleeding Clean, debride, and suture, if possible If primary closure not possible, then consider tension-relieving suture to reduce area requiring secondary granulation May need more than one procedure to get primary closure
Puncture	Penetration via sharp object; may penetrate deeply	Control bleeding Clean and lavage copiously Before lavage, ensure that wound does not enter the coelomic cavity and communicate with the air sac system Often leave open to facilitate more aerobic cleaning
Contusion	Wound with bruising	If swelling is increasing, consider bandaging to assist stabilization Watch for distal extremities losing blood supply with swelling
Myiasis (fly strike)	Fly eggs laid onto broken, dirty, or macerated integument Larvae hatch and begin to feed on debris	Removal of larvae, using mechanical methods such as irrigation and direct removal Use of insecticidal compounds such as ivermectin (topically or systemically) or fipronil (spray formulation only, used topically) to destroy any remaining larvae Lavage remaining tissue with gentle disinfectant Analgesic drugs are essential, and antimicrobial agents are usually used
Abrasion	Superficial loss of epidermis	Dressing to keep tissue moist while granulation and epithelialization occurs

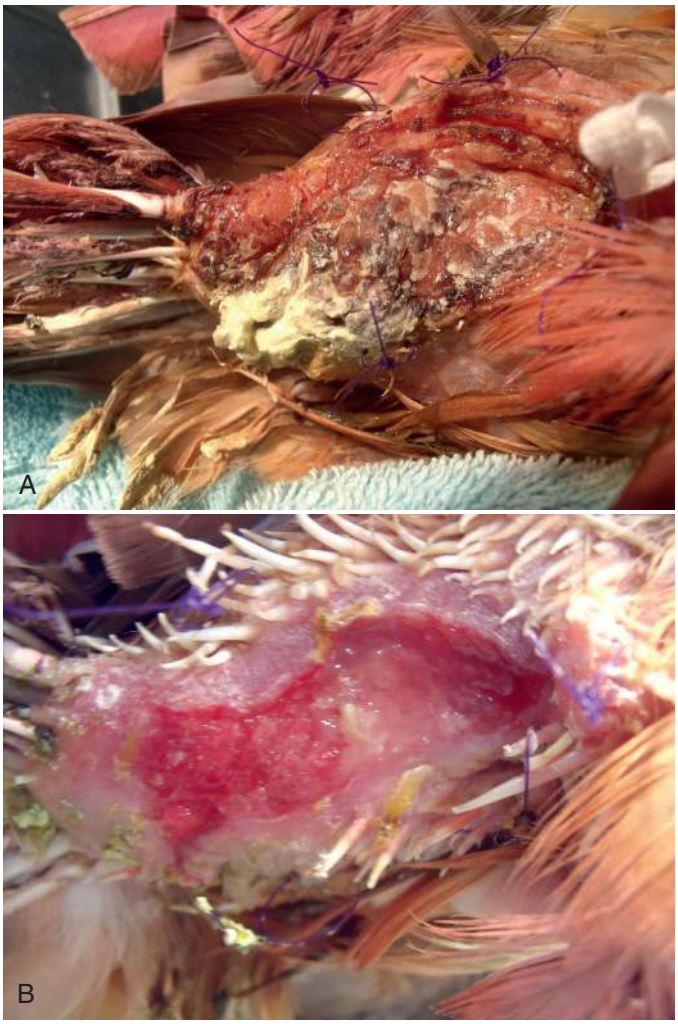


FIGURE 9-36 **(A)** and **(B),** Galah/corella cross parrot extensively self-mutilated. Although the cause was not determined on extensive workup, the wound was managed aggressively over a number of weeks and completely healed. The sutures visible are for anchoring bandage material.

HEALING OF WOUNDS

Wounds heal by one of two methods: first-intention healing or second-intention healing. First-intention healing can only take place in incised wounds where the edges are close together. This can be achieved by suturing or bandaging while healing takes place (see Bandages and dressings in Chapter 8). Wound healing in avian species is very similar to mammals and has three phases: inflammatory, proliferative, and remodeling. The initial injury fills with a blood clot and an acute inflammatory reaction occurs in the surrounding tissue. Leukocytes phagocytize necrotic material, debris, and bacteria. Collagen is synthesized by fibroblasts, which are attracted to the site by cytokines released from macrophages. Fibrin that forms within the clot provides a platform for the migration fibroblasts and endothelial cells.

The proliferation of endothelial cells and fibroblasts characterize the proliferative phase. Epithelialization and contraction of the wound occur in this phase. The collagen bed is replaced with stronger fibers in the remodeling phase of where the normal strength and tension of the injured tissue is reestablished. Second-intention healing is characterized by the formation of granulation tissue within a wound where there is extensive tissue loss and the wound edges are not opposable. Second-intention healing also occurs when there is a marked inflammatory response, such as occurs with infected wounds.

Local factors that inhibit wound healing include contamination with foreign material, infection, extensive necrosis, poor regional vasculature, tension, and excessive motion. The skin over the skull, sternum, and extremities are firmly attached to the bone and wounds in these locations can therefore be affected by many of these factors. As a result, wounds in these regions can be difficult to heal. Malnutrition, dehydration, hypoproteinemia, anemia, or other conditions resulting in a poor systemic state also impact wound healing. The administration of corticosteroids in avian species can significantly impair wound healing and are not recommended.

TREATMENT OF WOUNDS

Treatment of wounds comprises the following steps (Box 9-1):

I. Control hemorrhage.
II. Assess the systemic state of the patient.
  A. Treat immediate life-threatening conditions such as shock, excessive blood loss, dehydration, and sepsis.
  B. Perform a thorough physical examination (including neurologic and orthopedic evaluation) and identify any additional injuries.
  C. Provide analgesia.
  D. Consider radiography, hematology, and biochemistry, if indicated.
III. Assess wound.
  A. Decide if wound can be immediately closed. Infected, heavily contaminated wounds or those with extensive tissue loss or necrosis should initially be managed as an open wound.
IV. Formulate a management plan.
  A. Remove contamination.
    1. Lavage is best performed with Ringer's lactated solution using an 18-gauge needle and 20- to 30-mL syringe volume to achieve an ideal pressure of 7 to 8 psi.
    2. Collect culture and sensitivity samples and start empirical antibiosis if indicated.
  B. Debridement of necrotic tissue.
    1. Selective surgical removal of devitalized tissue may need to be performed several times throughout treatment. If the demarcation between healthy and necrotic tissue is unclear, surgery can be postponed for 3 to 5 days until the margins become apparent. This is especially true in cases of burns, including neonatal crop burns.
    2. Bandaging can be useful for the debridement of wounds with a small amount of necrotic tissue.
  C. Bandaging and wound dressings.
    1. The roles of a primary bandage layer include removal of necrotic tissue, delivery of medications, transmission of exudate, and occlusion.
      a. To prevent disruption to the feather integrity, water-based topical medications are recommended.
      b. The function of adherent dressings is debridement of the wound, whereas nonadherent dressings are used to help prevent dehydration and disruption of the wound bed during healing.
      c. Wound medications that have been used in avian species include medical grade honey and silver sulfadiazine.
    2. The role of the secondary bandage is to absorb exudate and protect the wound.

BOX 9-1 Treatment of Common Traumatic Wound Injuries in Avian Species

Blood Feather Damage

It is advisable to remove damaged feather from the affected follicle. This can result in damage to the germinal epithelium, which is more likely if done repeatedly or roughly, and may result in permanent dystrophic changes to that feather. Pulling a blood feather is a painful procedure, and the wing needs to be firmly anchored to avoid iatrogenic damage. Any bleeding can be controlled with gentle pressure. In emergency situations, small gauge ligatures may stop the bleeding until the feather is assessed.

Beak or Nail Injuries

Treat with a cauterizing agent such as a silver nitrate stick or an electrosurgical electrode. Severely torn nails may require amputation. Molnar and Ptacek (2001) described the repair of talon injuries in raptors using cyanoacrylate glue, talcum powder, and an antibiotic mixture to make a "talon cap" over the distal process of the digit. A similar technique has been used to protect torn talons and beak injuries in falcons using cyanoacrylate glue and sodium bicarbonate (J. Samour, personal communication). Parrots may still traumatize the nail after this technique, so additional bandaging is recommended.

Oropharynx Injuries

May need to anesthetize the bird and suture the wound directly or use electrocautery. Topical epinephrine may be useful, as the region is highly vascular. Caution is required, as excessive collateral trauma during the repair will impair postoperative feeding.

Teeth or Claw-Related Injuries

Irrigation, cleaning, and drainage should be done. Bacteria, especially *Pasteurella* spp., commonly infect these wounds and can cause a fatal septicemia. This occurs in bites from dogs, cats, and especially rodents. Many birds that are bitten by rodents become septicemic within 48 to 72 hours, even if antibiotics have been administered immediately after the attack.

Head Wounds

Usually caused by trauma and can result in extensive loss of the skin on the scalp. Head wounds can be difficult to heal because of the lack of subcutaneous tissue and subsequent poor blood supply to the skin. The use of hydrocolloid or hydroactive dressings will speed up healing. These may need to be sutured in place. The healing process has a tendency to "stall," leaving deficits of open bone, so gentle repeated debridement is advisable. Cotton tips are useful for this, and anesthesia is not always required. Continued exposure of dead bone can lead to full-thickness necrosis and infectious meningitis.

Infected Joints

Tenosynovitis, arthritis, and osteomyelitis are infectious causes of lameness in cursorial birds. Infection of joints, commonly the tibiotarsal-tarsometatarsal joint, can result after a traumatic wound or from hematogenous spread. Radiographs are indicated before beginning treatment, and treatment protocols for septic arthritis should be aggressive. Joint lavage, antibiotic therapy, nonsteroidal antiinflammatory drugs, analgesics, antibiotic-impregnated polymethacrylate beads, topical DMSO, and intraarticular injection of antibiotics have been used. The methods for making these antibiotic beads are described and discussed by Remple and Forbes (2000).

3. The frequency of bandage changes depends on the type of wound and stage of healing, although they are typically changed every 1 to 3 days.

D. Reconstructive surgery.

1. The main surgical considerations for primary closure in avian species are gentle tissue handling and minimizing tension.

Many wound management techniques used with avian wounds have been adapted from studies performed in domestic mammals. The use of skin flaps and grafts have been sporadically reported in avian species to treat large defects in the integument. Axial flaps, advancement flaps, free grafts, and xenogeneic grafts from porcine small intestinal submucosa have been described (Gentz Y Linn, 2000; Hernandez-Divers et al 2003; Stroud et al, 2003).

REFERENCES

Gentz EJ, Linn KA: Use of a dorsal cervical single pedicle advancement flap in three birds with cranial skin defects, *J Avian Med Surg* 14:31–36, 2000.

Hernandez-Divers SJ, Hernandez-Divers SM: Xenogeneic grafts using porcine small intestinal submucosa in the repair of skin defects in 4 birds, *J Avian Med Surg* 17:224–234, 2003.

Molnar L, Ptacek ML: Traumatic injuries of beak and talons of captive raptors. *Proceedings of the European Association of Avian Veterinarians*, Munich, pp. 246–247, 2001.

Remple JD, Forbes NA: Antibiotic impregnated polymethylacrylate beads in the treatment of bumblefoot in falcons. In Lumeij JT, Remple JD, Redig PT, et al, editors: *Raptor biomedicine III*, Lake Worth, FL, 2000, Zoological Education Network.

Stroud PK, Amalsadvala T, Swaim SF: The use of skin flaps and grafts for wound management in raptors, *J Avian Med Surg* 17:78–85, 2003.

FURTHER READING

Burke HF, Swaim SF, Amalsadvala T: Review of wound management in raptors, *J Avian Med Surg* 16:180–191, 2001.

10

Management-Related Medical Conditions

BUMBLEFOOT (PODODERMATITIS)

Petra Zsivanovits, Deborah Monks

Bumblefoot, or pododermatitis, is a disease syndrome involving disruption to the plantar epithelium and/or deeper structures of the feet. Multiple species such as raptors, waterfowl, psittacines, poultry, or Passeriformes can be affected. While the ultimate step in the course of disease is invariably ischemic necrosis, the predisposing causes vary according to species.

The predisposing factors in raptors include the following:

- Excessive load bearing on feet from obesity.
- Asymmetric weight distribution between the legs. Potential causes include surgery, pain, trauma, and bone or joint deformity.
- General inactivity. Blood supply to the feet is highly dependent on the level of exercise. Studies in raptors demonstrate the increase of blood flow and temperature rise of 10° C after flight that returned to normal after 20 minutes (Mueller *et al.*, 2000).
- Unsuitable perches. Too big or too small for the bird's feet and/or all perches presented in the same size and diameter force the foot to take up the bird's weight continuously in the same spot, leading to pressure sores. Very wide and flat perches may lead to excessive weight bearing on the toe pads, whereas very small perches may cause too much pressure on the metatarsal pads.
- Abrupt cessation of flying postseason in falconry birds (Fig. 10-1). A study in falcons showed that a threefold likelihood to develop bumblefoot with this abrupt method compared with a gradually slowing down of the training over 3 to 4 weeks (9% to 3%; Lierz, 2003). The sudden inactivity together with a diet high in energy and proteins results in edema in the distal extremities. Often the birds gain weight quickly, adding more pressure to the feet and closing the vicious circle.

The result of bumblefoot is ischemic insult to the tissue with subsequent inflammation and necrosis. Further trauma or microtrauma (by overlong talons or bite wounds or penetrating trauma) or fragile skin from poor skin hygiene or malnutrition can allow the entry of pathogens and the resultant infections.

The consequences of these factors are similar: the development of microepithelial damage, localized impairment of the immune system and invasion of opportunistic pathogens from erosions and ulcers, and localized ischemia with inflammation and necrosis. The devitalized tissue has a reduced immune response to opportunistic pathogens and limited healing capacity. Histologically epithelium degeneration; the spread of bacteria, yeast, or fungi; hyperkeratosis; necrotic foci; and thrombosis of the capillary vessels can be seen.(Harcourt-Brown, 2000; Cooper, 2002).

There are several classification systems for bumblefoot. The authors favor the following (Oaks, 1993):

- Classification I: Showing reddening and smoothing of the callused skin of the bottom side of the feet. There might also be slight discoloration (Fig. 10-2).
- Classification II: Showing hyperkeratosis, beginning skin necrosis, and distinct discoloration, as well as the development of scabs and mild swelling around the lesion (Fig. 10-3).
- Classification III: Leading to a hyperkeratotic/necrotic core penetrating the skin and inflammation of the subcutaneous tissue, resulting in a caseous abscess with distinct swelling (Fig. 10-4).
- Classification IV: Involving tendon sheaths and tendons resulting in ascending infection to the intertarsal joints.
- Classification V: Affecting the bones in the form of osteolysis/osteomyelitis or septic arthritis of the tarsometatarsal or phalangeal joints.
- Birds suffering from grade IV to V bumblefoot might also present swelling on the dorsal aspect of the feet from severe infection/inflammation of the tissue and abscess development between the digits (Fig. 10-5).

Therapeutic principles are listed in Box 10-1. For any cases greater than Classification III, radiographic assessment is crucial to determine the prognosis of the case. For most birds with bumblefoot grade V, euthanasia is a serious consideration. Infection and inflammation often cause irreparable damage or rupture of the flexor tendons, septic arthritis, or osteomyelitis. Endocarditis might be a sequela of the severe infection.

The combination of medical treatment, surgical debridement, and husbandry correction promises the best chance of success (Figs. 10-6 to 10-8). The aim of treatment is to fight infection locally and/or systemically, to stimulate increased blood supply to the foot, and to debride infected and purulent tissue within the lesion. Antibiotic therapy should comply with culture and sensitivity testing, although often clindamycin, marbofloxacin, or lincomycin are used initially as drugs with good bone affinity and gram-negative efficacy. Necrosis and swelling from infection reduces blood supply to the lesion, limiting immune factors and delivery of systemic antibiotics. This results in granuloma formation, which further shields the pathogen from systemic therapeutics. Antibiotic-impregnated beads or suspensions designed for bovine intramammary treatment can be used to maintain high local levels of antibiosis within the surgical site (Remple et al, 2000). Systemic antibiosis and antiinflammatory drugs/analgesia and prophylactic antifungal drugs (if indicated for the species) should complete the medical management (Harcourt-Brown, 1996).

Bandages fulfill multiple functions of skin protection, holding ointments and dressings in place to assist softening of crusts, and the improvement of blood supply to the affected area. This is achieved by padding the foot such that the surrounding tissues are weight bearing, but the affected area is not. Various options for such foot casting were proposed using plastic casting materials or constructions out of thermoplastic tape with a hole in the middle, bandage donuts, sleeping mat padding with holes, etc. (Figs. 10-9 and 10-10). Care must be taken not to bandage the foot too firmly to avoid constriction and to manage those bandages appropriately (clean sand-free housing of the patient and regular bandage changes). DMSO solution can help reduce the

FIGURE 10-1 The plantar aspect of the foot of a normal peregrine falcon (*Falco peregrinus*). The reticulate scales of the digital and metatarsal pads have a well-defined papilliferous appearance and the integument is a homogenous yellow color.

FIGURE 10-2 Classification I. The integument coloration of the immature saker falcon (*Falco cherrug*) is usually blue, but this bird has also had early bumblefoot: the digital and metatarsal pads have flattened, the reticulate scales have become flattened and smooth, and there is a reactive hyperemia in these areas.

FIGURE 10-3 Classification II. Early proliferative changes are seen in the foot of a peregrine falcon and are greatest at points of maximum weight bearing. Although the skin has lost its suppleness and there is thickening of the digital pads, as well as the obvious changes on the metatarsal pad, subcutaneous infection has not yet occurred.

FIGURE 10-4 Classification III. A single proliferative lesion has thickened, forming a "corn." Separation has occurred between the surrounding thinned epidermis and the hyperkeratinized corn and has allowed infection through the integument. An abscess is forming in the plantar surface of the foot.

FIGURE 10-5 Classification IV/V. In cases where the changes seen in Figure 10-4 are not noticed (most often seen when a bird is placed in a large aviary to go through its annual molt), the bird can have advanced changes when brought for treatment. This bird has been lying in sternal recumbency for some time before presentation; the feet have become very swollen and contain pus.

FIGURE 10-6 A female saker falcon with bumblefoot has had the purulent material and infected scar tissue removed. A purse string suture has been applied to reduce the deficit. The toes have been bandaged in preparation for a foot cast using several layers of a soft, conforming bandage and a single layer of zinc oxide tape.

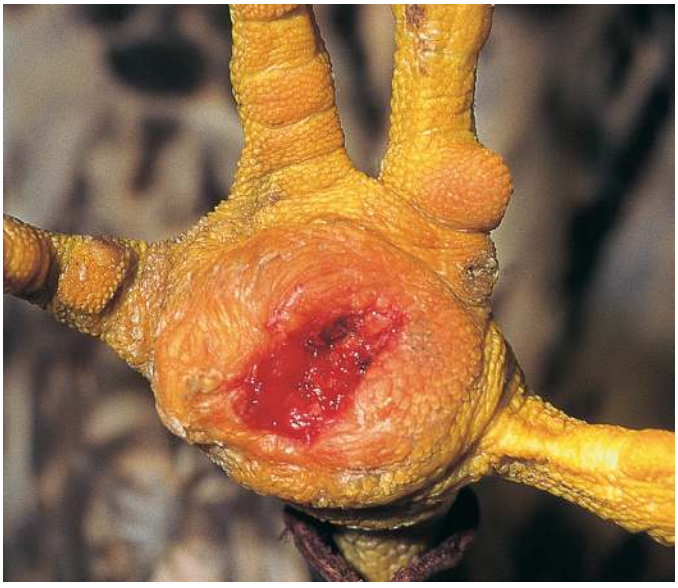


FIGURE 10-7 An adult saker falcon was presented with two scabbed and swollen feet. Changes had been present for many months. The base of the foot was distorted with scar tissue. The scab has been removed in this view.

BOX 10-1 Therapeutic Principles When Approaching Raptor Bumblefoot

Classification I: Antibiotics, antiinflammatory medications, husbandry adjustment

Classification II: Antibiotics, antiinflammatory medications, bandages, husbandry adjustment

Classification III: Antibiotics, antiinflammatory medications, bandages, surgery, husbandry adjustment—radiographs recommended

Classification IV: Antibiotics, antiinflammatory medications, bandages, surgery, husbandry adjustment—with a guarded prognosis—radiographs essential.

Classification V: Antibiotics, antiinflammatory medications, bandages, surgery, husbandry adjustment—very poor prognosis—radiographs essential and euthanasia.

inflammation and increase blood circulation. A drawing ointment can also be useful for encapsulation and abscess formation before surgery.

The principle of surgery is to remove all nonviable, caseous, and necrotic tissue. With a defined abscess this might be straightforward; however, often the inflammation extends diffusely into the surrounding tissue making a separation challenging. In many cases, surgery is delayed for several days to allow the action of antibiosis and antiinflammatories. This often assists the identification of viable versus nonviable tissue. If pieces of the abscess capsule are left behind there is the risk of a recurrence. Attention is necessary to remove all affected tissue but not to damage the tendons. In advanced cases these might be very fragile; however, any devitalized ligaments or tendons must be removed. Therefore in advanced cases there is the risk of loss of digital function. Avian pus is normally firm with a caseous appearance. Turbid, yellowish-white viscous liquid during surgery can indicate involvement of joint synovia or tendon sheaths. The use of antibiotic-impregnated polymethylmethacrylate beads within the debrided wound cavity can be beneficial to healing. Multiple surgeries might be necessary to resolve the problem. Treating bumblefoot with or without surgery is always a long-term commitment over several weeks or even months, requiring owner compliance.

Husbandry adjustment is crucial in the management of bumblefoot. Perches need to vary in diameter size and angle as well as surface/texture so that the pressure is not put on the same spot of the foot all the time. Swing perches not only reduce the impact of landing but also force the bird to balance, encouraging movement of the leg and feet. To counterbalance the contribution of a hard and even perch surface one might use AstroTurf as a cover. The spikes of AstroTurf provide a

FIGURE 10-8 Surgical removal of all abnormal tissue shown in Figure 10-7 allowed reconstruction of a more normal plantar aspect of the foot. Skin sutures were placed to obliterate dead space and to oppose skin edges.

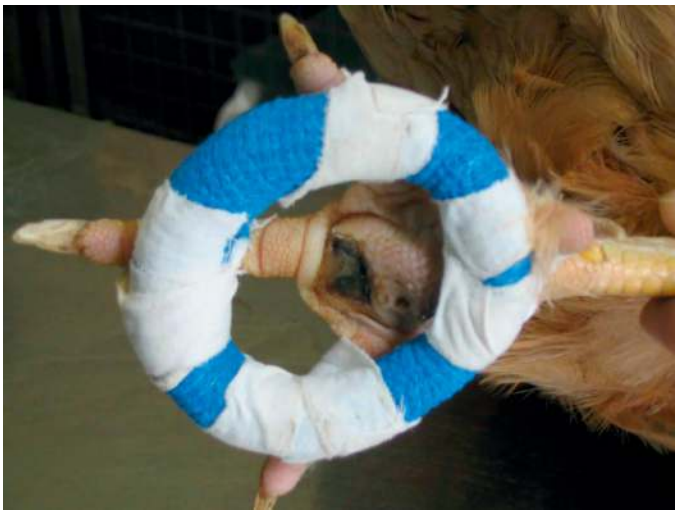


FIGURE 10-9 Donut type bandages are widely used to protect the sole of the foot on recently operated bumblefoot cases or to arrest the development of early lesions into severe forms. (Courtesy Bob Doneley.)

continuous massage to the plantar surface of the feet and assist in spreading the weight equally. However, in some grades of AstroTurf there are shorter, harder spikes that need to be removed to avoid puncturing the foot.

Thorough hygiene of the cages, aviaries, and perches should be practiced to minimize pathogen contamination. The same is true for well-balanced nutrition with particular consideration of vitamins (including vitamin A) and mineral nutrients. Weight control is crucial in cases of obesity. Furthermore, birds in better condition are more likely to exercise. Increasing exercise is probably the single most important action. Falconry birds can be taken back into training. If flight training is not a possibility so-called high jumps in the aviary are a good alternative. For parrots and other pet birds, spacious cages and aviaries should be provided and various measures for environmental enrichment to motivate flight should be available.

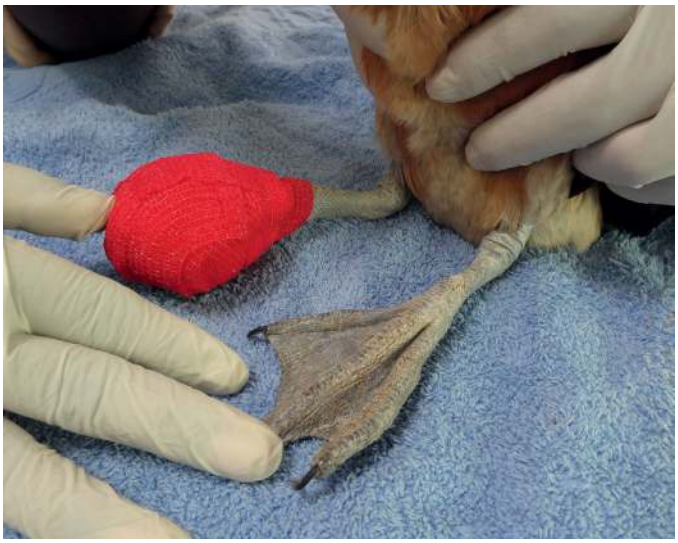


FIGURE 10-10 Ball bandages are still one of the favored nursing bandaging techniques used worldwide to protect the digits or the sole of the foot from recent bumblefoot surgery, digit amputation, wounds, pox lesions, and other trauma. (Courtesy Melodiya Magno.)

RAPTORS

Falconiformes seem to more susceptible to bumblefoot than accipiters. Falconiformes, such as the gyr and saker falcons, are more likely to develop bumblefoot than peregrines (13% susceptibility compared with 5%; Lierz, 2003). The larger size of gyr and saker falcons was mentioned as a possible predisposing factor. Often the lesions are contaminated with bacteria such as *Staphylococcus* spp. or *Escherichia coli.* Husbandry adjustments including gradual cessation of the training and the use of AstroTurf or similar rough surfaces for the perches play a pivotal role. Bumblefoot is mainly found in captive raptors—if a free-ranging bird is affected, there is usually another primary disease. Bumblefoot has been recognized among falconers since their first days in the Middle Ages; however, management remains challenging.

WATERFOWL

In waterfowl, classifications I to IV are the most common presentations (Fig. 10-11). Surgery can prove difficult as the altered tissue is often firmly integrated. Also, because of the weight of the birds and their preference for water, the management of bandages can be challenging. Relapses after surgery are quite common. Medical treatment should include vitamin A supplementation and very regular bathing of the feet in iodine or chlorhexidine solution. Husbandry is a major aspect in the management of bumblefoot in waterfowl, especially abrasion of the epithelium, puncture wounds, or bruising of the feet by rough and hard surfaces. Hard surfaces such as concrete must be replaced by soft substrates such as sand or grass. Good hygiene is elementary. Furthermore, waterfowl must always have access to a pond with clean water for swimming and bathing, but that must be balanced against maintaining bandage integrity.

PARROTS AND PASSERIFORMES

Most often bumblefoot lesions of grade I to III are found in these birds (Fig. 10-12). The main causative factors include immobility from bad husbandry or chronic disease, obesity, or asymmetric weight bearing, along with inappropriate perches. Particularly in finches, overgrown talons can result in improper weight distribution on the sole of the feet.

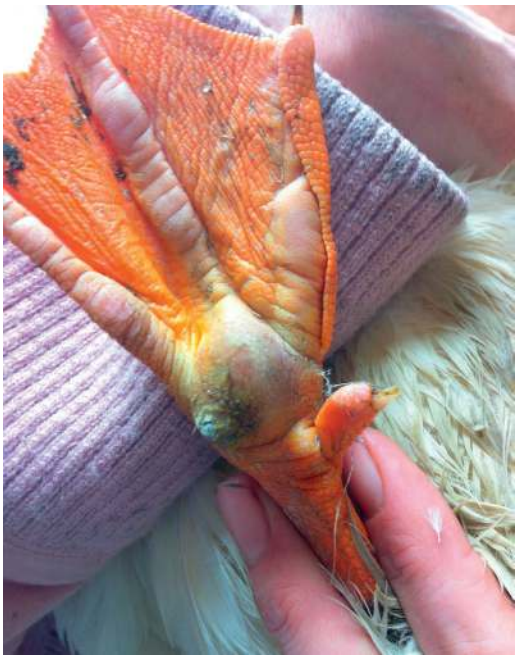


FIGURE 10-11 Bumblefoot in a duck with classification III. Please note the necrotic core on the lateral aspect of the foot penetrating the skin and related inflammation resulting in a caseous abscess. (Courtesy Bob Doneley.)

FIGURE 10-12 Bumblefoot in an amazon parrot, classification II.

Malnutrition (particularly vitamin A deficiency) may cause dry and hyperkeratotic skin and at the same time weaken any immune response. In these species, bumblefoot might affect not only the plantar sole of the feet but often also the plantar aspect of the tarsal joints. Next to medical treatment, bandages combined with repeated surgical debridement of the affected area are an approved method. It is worthwhile for patient and pet owner to patiently and persistently continue with the treatment because several cases are known in which continuous bandaging for up to a year resulted in complete and permanent resolving of the lesions. Husbandry adjustment should include an appropriate nutritional audit of the patient and proper perching. Perches covered with sandpaper or sandpaper on the floor of the cage should be avoided. It might be necessary to provide perches with padding (wrapping with bandages or carpeting material) to initially protect the feet. A good variety of perches added to swing perches should be available, including safe, bark-covered natural branches. Dietary correction and the prevention of excessive body weight needs to be emphasized.

ACKNOWLEDGMENTS

The authors would like to acknowledge the contribution of Nigel Harcourt-Brown to this section in the previous edition of this book.

REFERENCES

Cooper JE: Foot conditions. In *Birds of prey: health and disease*, Oxford, UK, 2002, Blackwell Science.

Harcourt-Brown NH: Foot and leg problems. In Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *Manual of raptors, pigeons and waterfowl*, Cheltenham, UK, 1996, British Small Animal Veterinary Association.

Harcourt-Brown NH: *Birds of prey: anatomy, radiology, and clinical conditions of the pelvic limb*, CD-ROM, Lake Worth, FL, 2000, Zoological Education Network.

Lierz M: Aspects of the pathogenesis of bumblefoot in falcons. *Proceedings of the Association of Avian Veterinarians*, Tenerife, pp 178–184, 2003.

Mueller MG, Wernery U, Koesters J: Bumblefoot and lack of exercise among wild and captive-bred falcons tested in the United Arab Emirates, *Avian Dis* 44:676–680, 2000.

Oaks JL: Immune and inflammatory responses in falcon staphylococcal pododermatitis. In Redig PT, Cooper JE, Remple JD, et al, editors: *Raptor biomedicine*, Minneapolis, MN, 1993, University of Minnesota Press.

Remple JD, Forbes NA: Antibiotic impregnated polymethylacrylate beads in the treatment of bumblefoot in falcons. In Lumeij JT, Remple JD, Redig PT, et al, editors: *Raptor biomedicine III*, Lake Worth, FL, 2000, Zoological Education Network.

FEATHER-DAMAGING BEHAVIOR IN PSITTACINE BIRDS

Yvonne van Zeeland

Feather-damaging behavior (FDB), also referred to as feather-destructive behavior, feather plucking, feather picking, or pterotillomania (Harrison, 1986; Orosz, 2006; Lumeij and Hommers, 2008), is one of the most common and frustrating conditions to deal with in captive parrots. It has been estimated that approximately 10% to 15% of captive psittacine birds chew, pluck, bite, or pull their feathers (Grindlinger and Ramsay, 1991; Gaskins and Bergman, 2011; Kinkaid *et al.*, 2013), inflicting localized or generalized damage to their plumage in the areas that can be accessed using their beak (i.e., neck, chest, flanks, inner thighs, wings; Harrison, 1986; Nett and Tully, 2003). Although in many cases the consequences of this self-inflicted feather damage may be solely esthetic, medical issues may also arise because of alterations to the birds' thermoregulatory abilities and metabolic demands, hemorrhage, and/or (secondary) infections (Galvin, 1983; Rosskopf and Woerpel, 1996; Nett and Tully, 2003).

SPECIES, AGE, AND GENDER PREDILECTIONS

Although noted in all psittacine birds, FDB is most commonly reported in African grey parrots (*Psittacus erithacus*) and cockatoos (*Cacatua* spp.), with prevalence of approximately 40% reported in these species (Briscoe *et al.*, 2001; Seibert, 2006; Kinkaid *et al.*, 2013; Gaskins and

Hungerford, 2014; Jayson *et al.*, 2014). In contrast, FDB appears less common in Amazon parrots (*Amazona* spp.), cockatiels (*Nymphicus hollandicus*), and budgerigars (*Melopsittacus undulatus*; Briscoe *et al.*, 2001; Seibert, 2006; Kinkaid *et al.*, 2013). Other predisposing factors include age and sex, with FDB suggested to occur more often in adolescent and adult female birds (Garner *et al.*, 2006; van Zeeland *et al.*, 2009; Kinkaid *et al.*, 2013). Furthermore, FDB appears to occur in those individuals demonstrating a *proactive coping strategy* (i.e., an active, fight–flight behavioral response, which is characterized by higher levels of aggression and territorial control, fast and superficial exploration of the environment, and an overall rigid and routine-like behavior (van Zeeland *et al.*, 2013b).

FDB is often self-inflicted, but in rare instances, group-housed birds may also pluck their cage mates or offspring. In these situations, the head and face generally appear to be the primary target area (Fig. 10-13; Wedel, 1999; Lightfoot and Nacewicz, 2006).

ETIOLOGIC CONSIDERATIONS FOR FEATHER-DAMAGING BEHAVIOR

FDB is considered a multifactorial disease, in which various medical, genetic, neurobiologic, and socioenvironmental factors may play a role (Table 10-1; van Zeeland *et al.*, 2009). Numerous medical conditions have been associated with FDB, but a true causal relationship is often lacking. In short, any disease causing pain, discomfort, irritation, and/or pruritus may result in development of FDB. This may include primary feather and skin diseases and systemic diseases (Table 10-1; Rosenthal, 1993; van Zeeland *et al.*, 2009). In cases of systemic disease, feather damage may be either diffuse and generalized or localized directly over the region of discomfort. Renal disease, for example, appears to induce FDB in the region of the synsacrum (Pollock, 2006; Burgos-Rodriguez, 2010), whereas hepatic disease may either induce feather damage that is limited to the ventral portion of the body or follows a more diffuse, generalized pattern (Davies, 2000; Grunkemeyer, 2010).

In addition to true medical conditions, environmental factors may also induce FDB. A small cage or poor cage design, for example, may result in damage to the feathers, particularly the primaries and tail

FIGURE 10-13 Feather loss is usually the result of self-inflicted damage. Typically, these birds have a normally feathered head. Occasionally, feather loss may also be inflicted by a cage mate, as was the case in this green-winged macaw (*Ara chloroptera*), in which the baldness remained localized to the head.

TABLE 10-1 Suspected Medical, Socioenvironmental, and Neurobiologic Causes of Feather-Damaging Behavior in Parrots

Medical	Socioenvironmental	Neurobiologic Factors
Ectoparasites (e.g., *Knemidokoptes*, feather or quill mites, lice)	Hand-rearing and imprinting on humans	Abnormal repetitive behavior resulting from neurotransmitter deficiencies and/or excesses (e.g., dopamine, serotonin, endorphins), similar to obsessive-compulsive or impulsive disorders
Bacterial or fungal dermatitis and/or folliculitis (including *Staphylococcus*, *Aspergillus*, *Candida*, *Malassezia*)	Restrictive environment, lack of sufficient stimulation, boredom	Suspected involvement of the corticostriatal circuits (based on findings in other species)
Polyomavirus	Inability to perform species-specific behaviors, e.g., foraging	Genetic factors also considered to play a role
Psittacine beak and feather disease	Presence of aversive stimuli, resulting in, e.g., anxiety or stress	
Nutritional deficiencies (e.g., hypovitaminosis A) and/or dietary imbalances	Sudden changes to the environment resulting in stress	
Airborne and/or topical toxins, including cigarette smoke, scented candles, air fresheners, hand lotions, and creams	Small cage or poor cage design with little space for the parrot to move around	
Low humidity levels, lack of bathing opportunities	Overcrowding	
Skin neoplasia (e.g., xanthoma, lipoma, squamous cell carcinoma)	Social isolation	
Hypersensitivity, skin allergy	Lack of a sexual partner, sexual frustration	
Airsacculitis, pneumonia	Abnormal photoperiod resulting in, e.g., sleep deprivation	
Chlamydiosis	Attention-seeking behavior, reinforced by actions of the owner/caretaker	
Proventricular dilatation disease	Tension release, coping strategy to deal with stressful situations	
Liver and/or renal disease		
Hypocalcaemia		
Endocrine disease (e.g., hypothyroidism, diabetes mellitus)		
Reproductive disease (e.g., egg binding, cystic ovaries)		
Heavy metal toxicosis (e.g., lead, zinc)		
Gastrointestinal disorders such as colic, endoparasitism (particularly Giardiasis in cockatiels)		
Obesity		
Orthopedic disorders (e.g., osteosarcoma, fracture, osteomyelitis)		
Trauma		
Poor wing trim		

feathers. As a result, the bird may remove the damaged feathers, which should actually be considered normal behavior. Other environmental risk factors implicated in FDB include nutritional deficiencies and dietary imbalances, airborne and topical toxins, and low humidity levels (Koski, 2002; van Zeeland *et al.*, 2009).

Several theories exist concerning the motivational and etiologic backgrounds of nonmedical FDB. Confinement and limited or no access to essential stimuli may, for example, result in inability to engage in species-specific behavior, inducing stress, boredom, and/or frustration. Particularly the lack of social interaction with humans or other birds or a sexual partner (especially in reproductively active birds) and deprivation of locomotor activities and/or foraging opportunities are thought to play a role (Meehan *et al.*, 2003a,b; Seibert, 2006; Jayson *et al.*, 2014). Of the latter, the lack of foraging opportunities has clearly been linked to FDB (Meehan *et al.*, 2003a; Lumeij and Hommers, 2008). Although the onset of FDB may in part result from an altered time budget (i.e., foraging times decrease from approximately 5 to 8 hours in the wild to approximately 1 hour in captivity; Snyder *et al.*, 1987; van Zeeland *et al.*, 2013c), several studies have demonstrated that parrots are motivated to forage and will *contrafreeload* (i.e., work for food even when identical food is freely available), suggesting that foraging may be a "behavioral need" (Coulton *et al.*, 1997; Joseph, 2010; van Zeeland *et al.*, 2010).

Aside from the lack of appropriate stimuli, the presence of aversive stimuli and/or sudden changes in the environment (lack of predictability) may result in anxiety and/or stress and subsequent FDB (Westerhof and Lumeij, 1987; Rosenthal, 1993). Birds may also experience stress because of abnormal photoperiods (resulting in either sleep deprivation or prolonged sleep periods), overcrowding, and/or lack of routine (Harrison, 1986, Gaskins and Hungerford, 2014). As a result, FDB may develop an attempt to cope with these negative affective states, with FDB serving as a tension-release mechanism resulting in dearousal by redirection of the motivation toward the feathers (*redirected behavior*) or performance of comforting grooming behavior (*displacement activity*; Delius, 1988; Spruijt *et al.*, 1993). In all of the aforementioned examples, FDB may be considered as *maladaptive behavior*, resulting from attempts to cope with an inadequate environment (from lack of appropriate stimuli, presence of aversive stimuli, or a combination of both). Furthermore, owners should be aware that they may also affect the onset and maintenance (or deterioration) of FDB, as their actions may actually reinforce the behavior (if attention and interaction are indeed considered desirable by the parrot).

With prolonged deprivation of appropriate stimuli or continued presence of aversive stimuli, the behavior may eventually develop into abnormal repetitive behavior (ARB), which includes both stereotypic behavior and impulse control disorders. In ARBs, a changed neurochemistry and neuroanatomy may lead to persistence of the behavior, even in absence of the original stressors or environmental deficits (Garner, 2006). However, not only the current living environment is thought to play a role in this process; early living environment (particularly the method used to raise the parrots [i.e., hand-rearing versus parent-raising]) is also shown to have a considerable effect on the behavioral development of the individual and the occurrence of abnormal behavior such as FDB (Collette *et al.*, 2000; Fox and Millam, 2004; Schmid, 2004; Fox, 2006; Schmid *et al.*, 2006). It has therefore been suggested that FDB may also represent *malfunctional behavior*, resulting from abnormal brain development and altered neurochemistry. Although the latter should not necessarily be considered as a separate entity compared with maladaptive behavior, the ability to make a distinction between the two may be important when considering the success of future therapeutic interventions. Whereas maladaptive behavior may benefit from changes to the environment that help to optimize the bird's living conditions, *malfunctional behavior* may show a lack of response to these measures and will more likely require the use of psychopharmaceutic drugs to reduce the behavior.

DIAGNOSTIC APPROACH TO FEATHER-DAMAGING BEHAVIOR

As a general rule, it first needs to be established whether the feather damage is inflicted by the bird (or its cage mate) or because of a medically or environmentally related condition that causes loss or damage to the bird's plumage irrespective of its behavior. It may, however, not always be possible to reliably determine whether the damage is indeed self-inflicted. First, birds are often left unobserved throughout a specific portion of the day, which limits the owner's ability to properly observe the bird's behavior. Second, it is often difficult for the owner to distinguish FDB from normal preening behavior (van Hoek and King, 1997).

If one is able to positively identify that the feather damage or loss is self-inflicted, the next challenge is to identify whether the condition primarily originates from a medical condition; results from husbandry, management, and/or nutrition-related issues; or if it should be regarded as a primary behavioral problem (i.e., psychogenic FDB). For this purpose, a thorough history and complete physical examination are deemed essential. During the physical examination, the self-inflicted nature of the feather damage and/or loss may be confirmed by the absence of feather abnormalities on the head, which is inaccessible to the bird's own beak (Fig. 10-14; Harrison, 1986).

In addition to taking a history and a physical examination, a thorough dermatologic examination of the skin and feathers is warranted, after which diagnostic skin and feather samples may be collected

FIGURE 10-14 African grey parrot (*Psittacus erithacus erithacus*) with feather-damaging behavior. Note the normally feathered head, which is typical for a bird with self-inflicted damage to the feathers.

(Table 10-2). If an underlying systemic cause is suspected, diagnostic workup may be further expanded with, e.g., a hematologic and/or biochemical blood panel, urinalysis, diagnostic imaging, and/or endoscopy (Table 10-2; Lamberski, 1995). Intradermal skin testing for diagnosis of allergic skin disease has been described, but thus far has been found unreliable in part due to the bird's diminished reaction to histamine (Colombini *et al.*, 2000; Nett *et al.*, 2003). Definite diagnosis of allergic skin disease may therefore be difficult, although the collection of paired skin biopsies from affected and unaffected areas of the same patient may identify the presence of inflammation consistent with delayed-type hypersensitivity reaction (Rosenthal *et al.*, 2004; Garner *et al.*, 2008).

If the abovementioned tests fail to identify a medical problem, a psychological or behavioral origin of the disorder becomes likely. It then becomes important to identify the potential underlying triggers (antecedents) and reinforcing factors (consequences) that may have contributed to the onset and maintenance of FDB (Friedman *et al.*, 2006b). The latter is, however, often difficult and time-consuming and may be limited by reliability and accuracy of the owner's observations and his willingness to learn and commit to behavioral enrichment and modification techniques. Applied behavior analysis, which focuses on the environmental determinants of behavior and the behavioral adaptations that occur through the process of learning, may be particularly helpful to generate hypotheses regarding the underlying motives for the behavior and test and evaluate these. Such *functional assessments* are generally considered the most useful for accurately assessing *what* is going on and *why*, which are two of the key factors in the development of a successful and effective behavioral intervention plan (S.G. Friedman *et al.*, 2006; Friedman, 2007).

THERAPEUTIC CONSIDERATIONS FOR FEATHER-DAMAGING BEHAVIOR

The therapeutic approach to FDB in the individual bird will largely depend on the findings of the history, physical examination, and diagnostic tests. An initial therapeutic plan will often be aimed at correction of the diet and modification of the bird's housing and living conditions to address any environmental factors that may be involved.

If any medical issues are encountered, these should be appropriately addressed, and this may include the use of topical and/or systemic antibiotics, antifungals, and antiparasitic drugs to treat any underlying parasitic or infectious disease. If there are suspected

TABLE 10-2 Diagnostic Tests That May Be Performed in Birds with Feather-Damaging Behavior

Diagnostic Test	Indications
CBC and biochemistry	Hepatopathy, nephropathy, generalized infection or inflammatory process, diabetes mellitus, hypocalcemia
Toxicology	Suspected lead or zinc toxicosis; collect heparinized whole blood (lead) or plasma/serum in nonrubber plastic or glass tubes
TSH stimulation test	Hypothyroidism
Fecal examination (cytology including gram stain, wet mount, and/or flotation)	Giardiasis (common in cockatiels), helminth infection, coccidiosis (rare in parrots), candidiasis, *Macrorhabdus ornithogaster* infection (avian gastric yeast), bacterial overgrowth, or dysbacteriosis
Radiology	Heavy metal intoxication, reproductive disorder (e.g., egg binding); hepatomegaly, splenomegaly, or renomegaly; proventricular dilatation disease; pneumonia; airsacculitis; neoplastic conditions; musculoskeletal disease (e.g., osteoarthritis, osteomyelitis, fractures, osteosarcoma)
Ultrasound	Hepatomegaly, reproductive disorders (e.g., egg peritonitis, cystic ovary), neoplastic conditions, cardiac disease, ascites
Endoscopy	Air sacculitis, hepatopathy or nephropathy, splenomegaly, pancreatic disorders, reproductive disease
Skin scrapings	Ectoparasites, in particular mites (e.g., *Knemidokoptes*, *Metamichrolichus nudus*); be careful not to tear the skin!
Impression smear, swab cytology, or tape strip	Bacterial or fungal dermatitis, dermatophytosis, *Malassezia*, *Candida*, ectoparasites (e.g., feather mites, lice), pox virus
Fine-needle aspirate	Skin neoplasia, xanthomatosis, feather follicle cyst, hematoma, bacterial dermatitis or abscess
Feather digest (using potassium hydroxide)	Ectoparasites (quill mites)
Feather pulp cytology	Bacterial or fungal folliculitis, PBFD or polyomavirus infection, quill mites
Culture and sensitivity testing	Bacterial or fungal infection (e.g., dermatitis, folliculitis)
Skin and/or feather follicle biopsy (histopathology)	Various infectious, inflammatory, and/or neoplastic skin diseases, e.g., PBFD, polyomavirus, bacterial and fungal folliculitis, quill mite infestation, xanthomatosis, squamous cell carcinoma, feather follicle cysts; a biopsy of an area that the bird is unable to reach may help to differentiate between psychogenic and inflammatory causes (i.e., no abnormalities versus presence of inflammation in the control biopsy) Note: biopsies may also be collected from other tissues and organs to detect abnormalities found with other diagnostic tests
Intradermal skin testing	Hypersensitivity reactions, allergic skin disease; thus far not found to be reliable due to the bird's diminished reaction to histamine
Tests for specific causative agents	PCR testing on whole blood, feather pulp or tissue for PBFD PCR testing on fecal swab or tissue for presence of polyomavirus PCR on cloacal swab/feces and/or serologic testing for avian bornavirus PCR on conjunctival/choanal/cloacal swab and/or serologic testing for *Chlamydia psittaci* Serology or galactomannan assay for *Aspergillus* (relative low sensitivity and specificity)

CBC, Complete blood count; PBFD, psittacine beak and feather disease; PCR, polymerase chain reaction; TSH, thyroid-stimulating hormone.

allergies, antihistamines (e.g., hydroxyzine hydrochloride, 2 mg/kg by mouth every 8 hours) and/or corticosteroids may be considered in addition to dietary and/or environmental modifications that aim to decrease or eliminate exposure to the suspected allergen(s), although one should always be hesitant to use corticosteroids in birds because of the potential for profound immunosuppression and development of secondary infections (Krinsley, 1993).

Promoting a more stimulating environment with social contact, perches, chewing toys, puzzle feeders, and other forms of environmental enrichment should be considered an important part of the treatment regimen to reduce FDB (van Zeeland *et al.*, 2009). In particular, foraging enrichment has been shown to effectively reduce FDB (Coulton *et al.*, 1997; Meehan *et al.*, 2003a; Lumeij and Hommers, 2008). Providing such enrichment may be as simple as providing complicated food items such as corn on the cob, pineapples, or pomegranates; providing food in larger chunks or pellets; using multiple feeding stations; scattering the food through the enclosure; and/or mixing food with inedible items (Fig. 10-15; Rozek *et al.*, 2010; van Zeeland *et al.*, 2013c). Owners may also use paper bags, cardboard boxes, plastic bottles, and other materials to create their own foraging toys, or, alternatively, buy one or more of the more complicated foraging devices and puzzle feeders that have become commercially available (Fig. 10-16).

In addition to providing environmental enrichment, behavior modification techniques such as differential reinforcement of alternative behavior may be used to alter the behavior of the bird (Davis, 1999; Seibert, 2006Friedman *et al.*, 2006a). Furthermore, training may also help provide a mentally stimulating challenge or task for the bird, provided the owner is able and willing to use the techniques in a proper and consequent manner.

Other treatments used to treat FDB include the use of Elizabethan collars and neck braces (Fig. 10-17); fabric "ponchos," "jackets," or "vests" (Fig. 10-18); and/or local application of foul-tasting substances. These interventions, however, are primarily aimed at preventing the symptoms rather than eliminating the underlying cause; therefore they are only recommended as a temporary measure to stop birds from automutilating and/or break the cycle of habitual FDB. To help facilitate the placement of a collar, it may be helpful to administer an intramuscular tranquilizer such as midazolam (0.3 to 0.5 mg/kg).

Pharmacologic intervention is considered helpful in patients that appear refractory to treatment with behavior modification therapy and environmental changes. Options include (1) anxiolytic drugs such as diazepam (Seibert, 2007); (2) antipsychotic drugs such as the dopamine antagonist haloperidol (Iglauer and Rasim, 1993; Lennox and VanDerHeyden, 1993, 1999); (3) tricyclic antidepressants such as amitriptyline, clomipramine, and doxepin (Ramsay and Grindlinger, 1994; Seibert *et al.*, 2004; Seibert, 2007); (4) serotoninergic reuptake inhibitors such as paroxetine and fluoxetine (Mertens, 1997; Seibert, 2007; van Zeeland *et al.*, 2013a); and (5) opioid antagonists such as naltrexone (Turner, 1993; Seibert, 2007; Table 10-3). In cases of suspected sexual or hormonally related FDB (e.g., seasonal occurrence and presence of [hyper]sexual and nesting behavior) treatment may be initiated with a depot gonadotropin-releasing hormone such as deslorelin or leuprolide acetate or medroxyprogesterone acetate (Iglauer and Rasim, 1993).

PROGNOSIS AND MONITORING

Because of the inability to determine the antecedents and consequences associated with FDB, the chronicity and/or ritualization of the behavior, and the overall lack of scientific evidence regarding the efficacy of the various therapeutic interventions, management of the condition often proves to be challenging. To evaluate whether therapeutic interventions elicit any effect, continued monitoring and adequate follow-up of the patient are essential. Although direct behavioral observations are possible (Colombini *et al.*, 2000), these rarely appear reliable. In contrast, feather scoring systems, which measure FDB indirectly by assessing plumage condition, pose a reliable and practical alternative, thus helping to monitor changes in the plumage condition (and thus effects of specific interventions) throughout time (Table 10-4;

FIGURE 10-15 Foraging enrichment is easily provided by mixing food with inedible items such as marbles **(A)** or providing larger sized food particles **(B)**.

FIGURE 10-16 A variety of commercially available foraging enrichments (puzzle feeders) that may help promote foraging activity and increase foraging time in captive parrots.

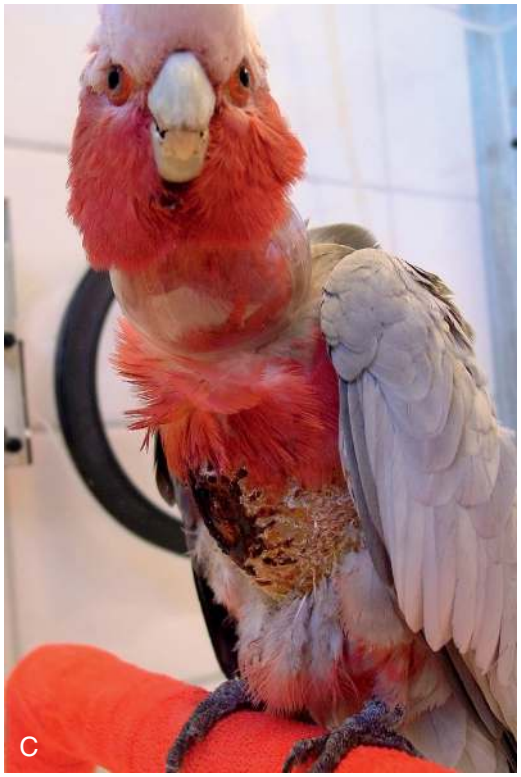


FIGURE 10-17 Collars are available in various shapes and sizes **(A, B)** and are used to prevent birds from reaching the skin and feathers. However, they do not eliminate the underlying cause of feather-damaging behavior. Their use should therefore be limited to birds that automutilate, such as the rose-breasted cockatoo (*Eolophus roseicapillus*) in **(C)**. Alternatively, they may sometimes be useful to help break the behavior if it has become a "habit."

FIGURE 10-18 Although socks **(A)** or custom-designed jackets **(B)** are another form of symptomatic treatment, they may pose a friendlier and, therefore, a more suitable alternative to the use of collars to help prevent the bird from automutilating and/or plucking itself.

TABLE 10-3 List of Psychotropic Drugs That May Be Used in Parrots with Feather-Damaging Behavior

Drug	Mode of Action	Suggested Dose
Amitriptyline	Tricyclic antidepressant; antihistamine	1-5 mg/kg per mouth every 12-24 h
Buspirone	Anxiolytic drug, used in the treatment of anxiety disorders	0.5 mg/kg every 12 h
Clomipramine	Tricyclic antidepressant; antihistamine; used, e.g., in the treatment of ICD/OCD, depression and/or anxiety disorders	0.5-1 mg/kg per mouth every 12-24 h
Diazepam	Benzodiazepine; tranquilizer; used in treatment of anxiety or panic disorders	0.5-0.6 mg/kg IM/IV every 8-24 h
Doxepin	Tricyclic antidepressant; antihistamine	0.5-1 mg/kg by mouth every 12 h
Fluoxetine	Selective serotonin reuptake inhibitor; antidepressant; used in the treatment of depression, posttraumatic stress, and panic disorders and ICD/OCD	0.4-4 mg/kg by mouth every 12-24 h
Haloperidol	Dopamine antagonist; antipsychotic drug	0.1-2 mg/kg by mouth every 12-24 h
Leuprolide acetate	Synthetic GnRH agonist depot drug; may be used in cases of FDB with suspected hormonal component	0.1 mg/kg IM every 24 h
Medroxyprogesterone acetate	Progesterone derivative; was used for reproductive-related FDB in the past, but not recommended because of severe side effects	5-50 mg/kg SC or IM every 4 to 6 weeks
Naltrexone	Opiate receptor antagonist, used in the treatment of addictions	1.5 mg/kg by mouth every 8-12h
Paroxetine	Selective serotonin reuptake inhibitor; antidepressant; used in the treatment of depression, posttraumatic stress, and panic disorders and ICD/OCD	2-4 mg/kg by mouth every 12-24 h

FDB, Feather-damaging behavior; GnRH, gonadotrophic-releasing hormone; ICD/OCD, impulsive and obsessive-compulsive disorders; IM, intramuscular; IV, intravenous; SC, subcutaneous.
Note: Most of the dosages mentioned in this table have been derived from case reports and/or anecdotal evidence. As information on pharmacokinetics, pharmacodynamics, efficacy, and toxicity is currently lacking for most of these drugs, no specific recommendations can be made at this stage.

TABLE 10-4 Feather Scoring System for Monitoring Plumage Condition in Parrots

(A) Score Determination Table for Coverts and Down Feathers; Used For Chest/Neck/Flank, Back, Legs, Dorsal and Ventral Surface of the Wings

	Down Feathers			
Coverts	**No Down Removed**	**<50% of Down Removed**	**>50% of Down Removed**	**All Down Removed**
All coverts intact	100	85	70	60
Fraying or breakage	95	80	65	55
<25% of coverts removed	90	75	60	50
25-50% of coverts removed	80	65	50	40
50-75% of coverts removed	70	55	40	30
75-90% of coverts removed	60	45	30	20
>90% of coverts removed	50	35	20	10

van Zeeland YRA, Bergers MJ, van der Valk L, et al: Evaluation of a novel feather scoring system for monitoring feather damaging behaviour in parrots, *Vet J* 196:247–252, 2013d.

The percentage of damage to the covert and down feathers is assessed for each body part separately.

Deduct 10 points from the score if skin damage is present.

Total body plumage score (0 – 100)* = 0.25 × chest/flank + 0.17 × back + 0.10 × legs + 0.28 × dorsal wings + 0.20 × (ventral wings).

To determine the total body plumage score, the scores for each body part are corrected for their relative body surface percentage, similar to scoring systems used in human burn victims. These percentages (expressed as percentage of the total body surface area excluding the surface area of the head and unfeathered parts of the legs) were determined in six African grey parrots. Mean (±SD) values for the various body parts were 25 ± 1.2% (chest/neck/flank), 17 ± 1.5% (back), 10 ± 1.2% (legs), 28 ± 2.2% (dorsal wing surface, up to the level of the tertiaries), and 20 ± 1.9% (ventral wing surface, up to the level of the tertiaries).

(B) Score Determination for Flight Feathers; Used for Tail, Primary, and Secondary Feathers (Wings)

Score	**Description**
0	Flight feather with signs of fraying and/or breakage over >50% of the original length
1	Flight feather with signs of fraying and/or breakage over <50% of the original length
2	Flight feather with little or no damage present

Damage to individual flight feathers is assessed.

Total flight feather score (0 – 100)* = (primary + secondary feathers left wing) + (primary + secondary feathers right wing) + (tail feathers).

The maximum score is dependent on total number of flight feathers of the bird. In general, each wing has 10 primary feathers and 10 secondary feathers (remiges), whereas the tail has 10 to 12 flight feathers (rectrices). As each individual flight feather is awarded a score from 0 to 2, the score will range from 0 to 40 for each wing and from 0 to 20 (or 0 to 24 in the case of 12 tail feathers) for the tail, respectively.

Meehan *et al.*, 2003a; van Zeeland *et al.*, 2013d). When evaluating the parrot's plumage, intervals of at least 4 weeks (but preferably longer) should be taken into account to ensure sufficient time has elapsed for feathers to regrow.

REFERENCES

Briscoe J, Wilson L, Smith G: Non-medical risk factors for feather picking in pet parrots. *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, Orlando, FL, pp 131, 2001.

Burgos-Rodríguez AG: Avian renal system: clinical implications, *Vet Clin North Am Exot Anim Pract* 13:393–411, 2010.

Collette J, Millam J, Klasing K, et al: Neonatal handling of amazon parrots alters the stress response and immune function, *Appl Anim Behav Sci* 66:335–349, 2000.

Colombini S, Foil CS, Hosgood G, et al: Intradermal skin testing in Hispaniolan parrots (*Amazona ventralis*), *Vet Dermatol* 11:271–276, 2000.

Coulton L, Waran N, Young R: Effects of foraging enrichment on the behaviour of parrots, *Anim Welf* 6:357–363, 1997.

Davies RR: Avian liver disease: etiology and pathogenesis, *Semin Avian Exot Pet Med* 9:115–125, 2000.

Davis C: Basic considerations for avian behavior modification, *Semin Avian Exot Pet Med* 8:183–195, 1999.

Delius JD: Preening and associated comfort behavior in birds, *Ann NY Acad Sci* 525:40–55, 1988.

Fox R: Hand-rearing: behavioral impacts and implications for captive parrot welfare. In Luesher AU, editor: *Manual of parrot behaviour*, Oxford, UK, 2006, Blackwell Publishing.

Fox RA, Millam JR: The effect of early environment on neophobia in orange-winged amazon parrots (*Amazona amazonica*), *Appl Anim Behav Sci* 89:117–129, 2004.

Friedman SG: A framework for solving behavior problems: functional assessment and intervention planning, *J Exot Pet Med* 16:6–10, 2007.

Friedman SG, Edling TM, Cheney CD: Concepts in behavior: section I, the natural science of behavior. In Harrison GJ, Lightfoot TL, editors: *Clinical avian medicine*, Palm Beach, FL, 2006a, Spix Publishing.

Friedman SG, Martin S, Brinker B: Behavior analysis and parrot learning. In Luescher AU, editor: *Manual of parrot behavior*, Oxford, UK, 2006b, Blackwell Publishing.

Galvin C: The feather picking bird. In Kirk RW, editor: *Current veterinary therapy VIII. Small animal practice*, Philadelphia, PA, 1983, WB Saunders.

Garner J: Perseveration and stereotypy: systems-level insights from clinical psychology. In Rushen J, Mason GJ, editors: *Stereotypic animal behaviour: fundamentals and applications to welfare*, Oxfordshire, UK, 2006, CAB International.

Garner JP, Meehan CL, Famula TR, et al: Genetic, environmental, and neighbor effects on the severity of stereotypies and feather picking in

orange-winged amazon parrots (*Amazona amazonica*): an epidemiological study, *Appl Anim Behav Sci* 96:153–168, 2006.
Garner MM, Clubb SL, Mitchell MA, et al: Feather-picking psittacines: histopathology and species trends, *Vet Pathol* 45:401–408, 2008.
Gaskins LA, Bergman L: Surveys of avian practitioners and pet owners regarding common behavior problems in psittacine birds, *J Avian Med Surg* 25:111–118, 2011.
Gaskins LA, Hungerford L: Nonmedical factors associated with feather picking in pet psittacine birds, *J Avian Med Surg* 28:109–117, 2014.
Grindlinger HM, Ramsay E: Compulsive feather picking in birds, *Arch Gen Psychiatry* 48(9):857, 1991.
Grunkemeyer VL: Advanced diagnostic approaches and current management of avian hepatic disorders, *Vet Clin North Am Exot Anim Pract* 13:413–427, 2010.
Harrison GJ: Disorders of the integument. In Harrison GJ, Harrison LR, editors: *Clinical avian medicine and surgery*, Philadelphia, PA, 1986, WB Saunders.
Iglauer F, Rasim R: Treatment of psychogenic leather picking in psittacine birds with a dopamine antagonist, *J Small Anim Pract* 34:564–566, 1993.
Jayson SL, Williams DL, Wood JL: Prevalence and risk factors for feather plucking in African grey parrots (*Psittacus erithacus erithacus* and *Psittacus erithacus timneh*) and cockatoos (*Cacatua* spp.), *J Exot Pet Med* 23:250–257, 2014.
Joseph L: Contrafreeloading and its benefits to avian behavior. *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, San Diego, CA, pp 399–401, 2010.
Kinkaid HMY, Mills DS, Nichols SG, et al: Feather-damaging behaviour in companion parrots: an initial analysis of potential demographic risk factors, *Avian Biol Res* 6:289–296, 2013.
Koski MA: Dermatologic diseases in psittacine birds: an investigational approach, *Semin Avian Exot Pet Med* 11:105–124, 2002.
Krinsley M: Use of dermcaps liquid and hydroxyzine HCl for the treatment of feather picking, *J Assoc Avian Vet* 7:221, 1993.
Lamberski N: A diagnostic approach to feather picking, *Semin Avian Exot Pet Med* 4:161–168, 1995.
Lennox A, VanDerHeyden N: Haloperidol for use in treatment of psittacine self-mutilation and feather plucking. *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, Nashville, TN, pp 119–120, 1993.
Lennox A, VanDerHeyden N: Long-term use of haloperidol in two parrots. *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, Bedford, TX, pp 133–137, 1999.
Lightfoot T, Nacewicz CL: Psittacine behaviour. In Bradley-Bays T, Lightfoot T, Mayer J, editors: *Exotic pet behavior: birds, reptiles and small mammals*, St Louis, MO, 2006, WB Saunders.
Lumeij JT, Hommers CJ: Foraging "enrichment" as treatment for pterotillomania, *Appl Anim Behav Sci* 111:85–94, 2008.
Meehan C, Garner J, Mench J: Isosexual pair housing improves the welfare of young amazon parrots, *Appl Anim Behav Sci* 81:73–88, 2003a.
Meehan C, Millam J, Mench J: Foraging opportunity and increased physical complexity both prevent and reduce psychogenic feather picking by young amazon parrots, *Appl Anim Behav Sci* 80:71–85, 2003b.
Mertens P: Pharmacological treatment of feather picking in pet birds. *Proceedings of the 1st International Conference on Veterinary Behavioural Medicine*, Birmingham, UK, pp 209–213, 1997.
Nett CS, Hodgin E, Foil C, et al: A modified biopsy technique to improve histopathological evaluation of avian skin, *Vet Dermatol* 14:147–151, 2003.
Nett CS, Tully T: Anatomy, clinical presentation, and diagnostic approach to feather-picking pet birds, *Compend Contin Educ Pract Vet* 25:206–219, 2003.
Orosz SE: Diagnostic workup of suspected behavioral problems. In Luescher AU, editor: *Manual of parrot behavior*, Oxford, UK, 2006, Blackwell Publishing.
Pollock C: Diagnosis and treatment of avian renal disease, *Vet Clin North Am Exot Anim Pract* 9:107–128, 2006.
Ramsay EC, Grindlinger H: Use of clomipramine in the treatment of obsessive behavior in psittacine birds, *J Assoc Avian Vet* 8:9–15, 1994.
Rosenthal K: Differential diagnosis of feather-picking in pet birds. *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, Nashville, TN, pp 108–112, 1993.
Rosenthal KL, Morris DO, Mauldin EA, et al: Cytologic, histologic, and microbiologic characterization of the feather pulp and follicles of feather-picking psittacine birds: a preliminary study, *J Avian Med Surg* 18:137–143, 2004.
Rosskopf W, Woerpel R: Feather picking and therapy of skin and feather disorders. In Rosskopf W, Woerpel R, editors: *Diseases of cage and aviary birds*, Baltimore, MD, 1996, Williams & Wilkins.
Rozek JC, Danner LM, Stucky PA, et al: Over-sized pellets naturalize foraging time of captive orange-winged amazon parrots (*Amazona amazonica*), *Appl Anim Behav Sci* 125:80–87, 2010.
Schmid R: *The influence of the breeding method on the behaviour of adult African grey parrots*. PhD thesis, Universität Bern, Switzerland, 2004.
Schmid R, Doherr MG, Steiger A: The influence of the breeding method on the behaviour of adult African grey parrots (*Psittacus erithacus*), *Appl Anim Behav Sci* 98:293–307, 2006.
Seibert LM: Feather-picking disorder in pet birds. In Luescher AU, editor: *Manual of parrot behavior*, Oxford, UK, 2006, Blackwell Publishing.
Seibert LM: Pharmacotherapy for behavioral disorders in pet birds, *J Exot Pet Med* 16:30–37, 2007.
Seibert LM, Crowell-Davis SL, Wilson GH, et al: Placebo-controlled clomipramine trial for the treatment of feather picking disorder in cockatoos, *J Am Anim Hosp Assoc* 40:261–269, 2004.
Snyder NF, Wiley JW, Kepler CB: *The parrots of luquillo: natural history and conservation of the Puerto Rican parrot*, Los Angeles, 1987, The Western Foundation of Vertebrate Zoology.
Spruijt B, Van Hooff J, Gispen W: Ethology and neurobiology of grooming behavior, *Physiol Rev* 72:825–852, 1993.
Turner R: Trexan (naltrexone hydrochloride) use in feather picking in avian species. *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, Nashville, TN, pp 116–118, 1993.
van Hoek CS, King CE: Causation and influence of environmental enrichment on feather picking of the crimson-bellied conure (*Pyrrhura perlata perlata*), *Zoo Biol* 16:161–172, 1997.
van Zeeland YRA, Spruit BM, Rodenburg TB, et al: Feather damaging behaviour in parrots: a review with consideration of comparative aspects, *Appl Anim Behav Sci* 121:75–95, 2009.
van Zeeland YRA, Schoemaker NJ, Haritova A, et al: Pharmacokinetics of paroxetine, a selective serotonin reuptake inhibitor, in grey parrots (*Psittacus erithacus erithacus*): influence of pharmaceutical formulation and length of dosing, *J Vet Pharmacol Ther* 36:51–58, 2013a.
van Zeeland YRA, Schoemaker NJ, Lumeij JT: Contrafreeloading in grey parrots. *Proceedings of the Annual Conference of Avian Veterinarians*, San Diego, CA, pp 9, 2010.
van Zeeland YRA, van der Aa MMJA, Vinke CM, et al: Behavioural testing to determine differences between coping styles in grey parrots (*Psittacus erithacus erithacus*) with and without feather damaging behavior, *Appl Anim Behav Sci* 148:218–231, 2013b.
van Zeeland YRA, Bergers MJ, van der Valk L, et al: Evaluation of a novel feather scoring system for monitoring feather damaging behaviour in parrots, *Vet J* 196:247–252, 2013d.
van Zeeland YRA, Schoemaker NJ, Ravesteijn MM, et al: Efficacy of foraging enrichments to increase foraging time in grey parrots (*Psittacus erithacus erithacus*), *Appl Anim Behav Sci* 149:87–102, 2013c.
Wedel A: Verhaltensstörungen. In Wedel A, editor: *Ziervögel-Erkrankungen, haltung, futterung*, Vienna, Austria, 1999, Parey-Verlag.
Westerhof I, Lumeij J: Feather picking in the African grey parrot. *Proceedings of the 1st European Symposium on Birds' Diseases*, Beerse, Belgium, pp 98–103, 1987.

CAPTURE PARESIA

Thomas A. Bailey

This section is concerned with capture paresia, which is an important cause of mortality in birds during capture and translocation episodes

(Spraker *et al.*, 1987). Although a similar condition is a well-recognized complication in the capture of wild ungulates (Harthoorn and Young, 1974; Harthoorn, 1976; Wallace *et al.*, 1987; Robinson *et al.*, 1988; Spraker, 1993), it has received less attention in birds.

Capture paresia is known by a spectrum of names such as muscular dystrophy, capture disease, capture myopathy, degenerative polymyopathy, overstraining disease, white muscle disease, leg paralysis, muscle necrosis, idiopathic muscle necrosis, exertional rhabdomyolysis, stress myopathy, transit myopathy, diffuse muscular degeneration, and white muscle stress syndrome (Spraker, 1993).

DEFINITION

Paresis is defined by Blood and Studdert (1988) as "slight or incomplete paralysis, and includes animals that can make purposeful attempts to rise without being able to do so, those that are able to rise with assistance, those that are able to rise and walk with major difficulty including frequent falling, and those able to stand and walk without assistance, but with slight errors."

SPECIES AFFECTED

Capture paresia and other similar degenerative myopathies have been described in the following species:

- Greater flamingos (*Phoenicopterus ruber roseus*) and lesser flamingos (*Phoeniconaias minor*; Young, 1967)
- Secretary birds (*Sagittarius serpentarius*; Heerden, 1977)
- Ostriches (*Struthio camelus*), emus (*Dromaius novaehollandiae*), and rheas (*Rhea americana*; Heerden, 1977; Rae, 1992; Stewart, 1994; Tully *et al.*, 1996)
- Bar-tailed godwits (*Limosa lapponica*; Minton, 1980)
- Sandhill (*Grus canadensis*) and whooping cranes (*G. americana*; Windingstad *et al.*, 1983; Gainer, 1988; Carpenter *et al.*, 1991)
- Pileated woodpeckers (*Dryocopus pileatus*; Ruder *et al.*, 2012)
- Canada geese (*Branta canadensis*; Chalmers and Barrett, 1982)
- Free-living turkeys (*Meleagris gallopavo*; Atkinson and Forrester, 1987; Spraker *et al.*, 1987; Jessup, 1993)
- East African crowned cranes (*Balearica regulorum gibbericeps*; Brannian *et al.*, 1981)
- Houbara (*Chlamydotis undulata macqueenii*), kori (*Ardeotis kori*), little and rufous-crested bustards (*Eupodotis ruficrista*, Fig. 10-19; Bailey *et al.*, 1996a,b; Marco *et al.*, 2006)
- Domestic turkey (*M. gallopavo*; Cardona *et al.*, 1992)

FIGURE 10-19 Recumbent houbara bustard (*Chlamydotis undulata*) with capture-related paresia.

PATHOGENESIS

The exact pathogenesis of capture paresia is not clear, but it involves anaerobic metabolism during intense muscular activity (Harthoorn, 1976; Wallace *et al.*, 1987). Lactic acid produced in muscle causes local and systemic acidosis, resulting in the lesions and clinical signs of paresia (Harthoorn, 1976; Chalmers and Barrett, 1982). Low pH at the tissue level results in increased permeability of cell membranes and cell lysis, releasing intracellular enzymes including creatine kinase (CK), lactate dehydrogenase (LDH), and aspartate aminotransferase (AST) into the blood (Harthoorn, 1975; Chalmers and Barrett, 1982). Elevated concentrations of CK and AST in serum or plasma thus reflect damage to skeletal and cardiac muscle. Elevation of serum CK concentration appears to be the most sensitive and specific index of muscle damage in both mammals (Chalmers and Barrett, 1982) and birds (Franson, 1982; Lumeij, 1988a,b, 1993; Cardona *et al.*, 1992). It should be noted that not all elevations in plasma CK activities are an indication of disease; for example, it is known that CK levels dramatically increase in healthy bustards that are handled (Bailey *et al.*, 1997). Elevations of AST, alanine aminotransferase, CK, LDH, and serum potassium have been reported in whooping cranes (*G. americana*) with capture paresia (Hanley *et al.*, 2005).

CLINICAL SIGNS AND HISTORY

A number of factors considered to predispose birds to capture paresia include the following:

- Strenuous pursuit during capture operations
- Prolonged handling
- Translocation
- Poor transport conditions
- Possible vitamin E and selenium deficiencies
- Intercurrent disease
- Hot weather
- Cramping of the limbs

The clinical signs of capture paresia include the following:

- Depression
- Limb paresia or paralysis
- Hock-sitting
- Lateral or sternal recumbency with reluctance to rise or move
- Death during or after capture, handling or translocation

Acute death occurs in many cases and is thought to be caused by myocardial necrosis and trauma, while necrosis of the muscles of the thighs and flank causes limb paralysis (Young, 1967). The generally excitable disposition of ratites predisposes these animals to myopathy, especially in poorly managed facilities. Dietary factors are important when investigating outbreaks of myopathy. Myopathy in pelicans (*Pelecanus occidentalis*) has been reported following vitamin E deficiency caused by feeding rancid food (Shivaprasad *et al.*, 2002). Monensin toxicity has been linked to degenerative myopathy in ostriches (Baird *et al.*, 1997).

DIFFERENTIAL DIAGNOSIS

Other causes of hindlimb paresia or paralysis must be considered and ruled out in the differential diagnosis of this condition. A full list of the possible causes of paresia and paralysis in birds is presented in Table 10-5.

DIAGNOSIS

Diagnosis of capture paresia is based on consideration of the history, clinical signs, and detection of elevated plasma levels of CK, AST, and LDH.

TABLE 10-5 Differential Diagnosis of the Causes of Paresia and Paralysis in Birds

History	Possible Cause
Traumatic	Vertebral fractures or luxations Multiple fractures Pelvic fractures Dislocations or sprains
Infectious	Neuritis (peripheral nerve) Encephalitis or encephalomyelitis Intervertebral abscess Septicemia with Spinal Infection Nephritis Viral infections including paramyxovirus group 3, reovirus, papovirus, Pacheco's virus Bacterial infection, including aspergillosis, involving the central nervous system
Metabolic/ nutritional	Suspected vitamin E/selenium deficiency Multiple fractures secondary to metabolic bone disease
Reproductive	Obturator paralysis from difficult delivery Egg binding Broken leg from calcium deficiency Ectopic egg
Neoplastic	Renal adenocarcinoma Fibrosarcoma Other neoplasia or space-occupying lesion
Poisons	Botulism Lead toxicosis Furazolidone and ionophore toxicity
Miscellaneous	Cloacal lithiasis

POSTMORTEM CHANGES

The following macroscopic findings have been observed in birds examined postmortem that died from capture paresia (Young, 1967; Windingstad *et al.*, 1983; Spraker *et al.*, 1987; Carpenter *et al.*, 1991; Cardona *et al.*, 1992; Rae, 1992):

- Small to large white or pale foci and streaks on the myocardium, muscles of the hindlimbs, and the pectoral muscles
- Ruptured muscles
- Hemorrhages in the musculature of the thighs and flank
- Petechiae in the myocardium

Histopathology is important in diagnosing this condition because macroscopically visible lesions may not always be detected on postmortem examination (Gainer, 1988). Microscopically the main changes include necrosis of myocardial and skeletal muscle. Signs of renal failure may also be detected. The microscopic changes associated with this condition are fully described elsewhere (Young, 1967; Windingstad *et al.*, 1983; Spraker *et al.*, 1987; Carpenter *et al.*, 1991; Rae, 1992).

TREATMENT

The primary goal of treatment is the control of shock and hyperthermia. This is done by using the following:

- Intravenous and oral sodium bicarbonate to correct acidosis
- Fluid therapy to restore blood pressure and volume
- Parenteral vitamin E and selenium and multiple vitamins
- Corticosteroids
- Cooling the bird if it is hyperthermic
- Possible cardiac and respiratory stimulants

FIGURE 10-20 Slings can be made to support birds with paresia.

Attempts have been made to support affected birds in slings (Fig. 10-20) and to provide physiotherapy in the form of massage and placing limbs in lukewarm water. Mild cases of paresia may recover, but the prognosis is poor for severe cases. Businga et al. (2007) described the successful treatment of three wild greater sandhill cranes (*G. canadensis tabida*). Their treatment consisted of corticosteroids, selenium/vitamin E, parenteral fluids, and gavage feedings. Physical therapy consisted of assisting the cranes to stand and walk two to eight times a day, massaging leg muscles, and moving limbs manually through the range of motion.

PREVENTION

Capture myopathy is difficult to treat and every effort should be made to prevent the problem. Recommendations for minimizing the problem include the following:

- Supplemental vitamin E and selenium before episodes of capture, handling, and/or translocation (Mushi *et al.*, 1998)
- Capturing birds on days that have acceptable environmental conditions
- Keeping handling times and struggling to a minimum and avoiding hyperthermia
- Using proven capture techniques for the species to be caught
- Being aware that certain species, such as free-living turkeys, appear to be more susceptible to this condition (Spraker *et al.*, 1987)
- Transporting birds in well-ventilated containers
- Conditioning and training groups of animals, which can reduce the mortality associated with older methods of capture that involve them in exertion

REFERENCES

Atkinson CT, Forrester DJ: Myopathy associated with megaloschizonts of *Haemoproteus meleagridis* in a wild turkey from Florida, *J Wildl Dis* 23:495–498, 1987.

Bailey TA, Nicholls PK, Samour JH, et al: Post-mortem findings of bustards in the United Arab Emirates, *Avian Dis* 40:296–305, 1996b.

Bailey TA, Samour JH, Naldo J, et al: Causes of morbidity in bustards in the United Arab Emirates, *Avian Dis* 40:121–129, 1996a.

Bailey TA, Wernery U, Naldo J, et al: Concentrations of creatine kinase and lactate dehydrogenase in captive houbara bustards (*Chlamydotis undulata macqueenii*) following capture, *Comp Haematol Int* 7:113, 1997.

Baird GJ, Caldow GL, Peek IS, et al: Monensin toxicity in a flock of ostriches, *Vet Rec* 140:624–626, 1997.

Blood DC, Studdert VP: *Baillière's comprehensive veterinary dictionary*, London, 1988, Baillière Tindall.
Brannian RE, Graham DL, Creswell J: Restraint associated myopathy in East African crowned cranes. *Proceedings of the American Association of Zoo Veterinarians*, Seattle, WA, pp 21–23, 1981.
Businga NK, Langenberg J, Carlson L: Successful treatment of capture myopathy in three wild greater sandhill cranes (*Grus canadensis tabida*), *J Avian Med Surg* 21(4):294–298, 2007.
Cardona CJ, Bickford AA, Galey FD, et al: A syndrome in commercial turkeys in California and Oregon characterized by rearlimb necrotizing myopathy, *Avian Dis* 36:1092–1101, 1992.
Carpenter JW, Thomas NJ, Reeves S: Capture myopathy in an endangered sandhill crane (*Grus canadensis pulla*), *J Zoo Wildl Med* 22:488–493, 1991.
Chalmers GA, Barrett MW: Capture myopathy. In Hoff GL, Davis JW, editors: *Non-infectious diseases of wildlife*, Ames, IA, 1982, Iowa State University Press.
Franson JC: Enzyme activities in plasma, liver, and kidney of black ducks and mallards, *J Wildl Dis* 18:481–485, 1982.
Gainer RS: Capture mortality of a young whooping crane (*Grus americana*). *Proceedings of the American Association of Zoo Veterinarians and American Association of Wildlife Veterinarians*, Toronto, p 57, 1988.
Hanley CS, Thomas NJ, Paul-Murphy J, et al: Exertional myopathy in whooping cranes (*Grus americana*) with prognostic guidelines, *J Zoo Wildl Med* 36(3):489–497, 2005.
Harthoorn AM: Operative factors in capture myopathy. *Proceedings of the World Veterinary Congress*, Thessaloniki, Greece, 1975.
Harthoorn AM: *The chemical capture of animals*, London, 1976, Baillière Tindall.
Harthoorn AM, Young E: A relationship between acid–base balance and the acute phase of capture myopathy (so-called over-straining disease) in zebra (*Equus burchelli*) and an apparent therapy, *Vet Rec* 95:337–342, 1974.
Heerden JV: Leg paralysis in birds, *Ostrich* 48:118–119, 1977.
Jessup DA: Translocation of wildlife. In Fowler ME, editor: *Zoo and wild animal medicine current therapy 3*, Philadelphia, PA, 1993, WB Saunders.
Lumeij JT: Enzyme activities in tissues and elimination half-lives of homologous muscle and liver enzymes in the racing pigeon (*Columba livia domestica*), *Avian Pathol* 17:851–864, 1988a.
Lumeij JT: Changes in plasma chemistry after drug-induced liver disease or muscle necrosis in racing pigeons (*Columba livia domestica*), *Avian Pathol* 17:865–874, 1988b.
Lumeij JT: Avian plasma chemistry in health and disease. *Proceedings of the European Association of Avian Veterinarians*, Utrecht, the Netherlands, pp 558–566, 1993.
Marco I, Mentaberre G, Ponjoan A, et al: Capture myopathy in little bustards after trapping and marking, *J Wildl Dis* 42(4):889–891, 2006.
Minton CDT: Occurrence of "cramp" in a catch of bar-tailed godwits (*Limosa lapponica*), *Wader Study Group Bull* 28:15–16, 1980.
Mushi EZ, Isa JEW, Chabo RG, et al: Selenium-vitamin E responsive myopathy in farmed ostriches (*Struthio camelus*) in Botswana, *Avian Pathol* 27:326–328, 1998.
Rae M: Degenerative myopathy in ratites. *Proceedings of the Association of Avian Veterinarians*, New Orleans, pp 328–335, 1992.
Robinson WF, Wyburn RS, Grandage J: The skeletal system. In Robinson WF, Huxtable CRR, editors: *Clinicopathologic principles for veterinary medicine*, Cambridge, UK, 1988, Cambridge University Press.
Ruder MG, Noel BL, Bednarz JC, et al: Exertional myopathy in pileated woodpeckers (*Dryocopus pileatus*) subsequent to capture, *J Wildl Dis* 48(2):514–516, 2012.
Shivaprasad HL, Crespo R, Puschner B, et al: Myopathy in brown pelicans (*Pelecanus occidentalis*) associated with rancid feed, *Vet Rec* 150:307–311, 2002.
Spraker TR: Stress and capture myopathy in artiodactylids. In Fowler ME, editor: *Zoo and wild animal medicine current therapy 3*, Philadelphia, PA, 1993, WB Saunders.
Spraker TR, Adrian WJ, Lance WR: Capture myopathy in wild turkeys (*Meleagris gallopavo*) following trapping, handling and transportation in Colorado, *J Wildl Dis* 23:447–453, 1987.
Stewart J: Ratites. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.
Tully TN, Hodgin C, Morris M, et al: Exertional myopathy in an emu (*Dromaius novaehollandiae*), *J Avian Med Surg* 10:96–100, 1996.
Wallace RS, Bush M, Montali RJ: Deaths from exertional myopathy at the National Zoological Park from 1975 to 1985, *J Wildl Dis* 23:454–462, 1987.
Windingstad RM, Hurley SS, Sileo L: Capture myopathy in a free-flying greater sandhill crane (*Grus canadensis tabida*) from Wisconsin, *J Wildl Dis* 19:289–290, 1983.
Young E: Leg paralysis in the greater flamingo and lesser flamingo, *Int Zoo Yearb* 7:226–227, 1967.

TOXICOLOGY

Jaime Samour

Toxicology is the area of medical science that studies the actions of chemical compounds on biological systems. These compounds are normally referred to as toxicants or toxic agents, poisons, and toxins. The term *toxin* should be used exclusively to define a protein produced by a biological organism such as a higher plant, certain animals, and pathogenic bacteria that is toxic to other living organisms. Toxicosis, poisoning, and intoxication relate to the disease caused by the action of the chemical compound. Toxic agents may be exogenous or endogenous. Exogenous agents originate from outside the body. These include (1) man-made or synthetic compounds, such as pesticides, and (2) naturally occurring or natural compounds, such as those found in toxic plants and fungi. Endogenous toxic agents originate from within the body and include mainly toxins produced by pathogenic bacteria or fungi.

Captive birds are particularly susceptible to various toxicoses because of their inquisitive nature. It is common for pet birds to fly freely within the house and have easy access to household goods such as detergents, pesticides, disinfectants, toxic plants, and inhalant agents that could be harmful to them. In bird gardens, zoologic collections, parks, and farms, birds can be exposed to building materials, toxic plants, or items thrown by visitors that could be detrimental to their health. Free-living birds very frequently come into contact with toxic agents such as pesticides, fertilizers, and herbicides in farm and cultivated lands; lead from shotgun pellets or fishing weights in ponds, lakes, and rivers; and a myriad potentially harmful compounds in industrial areas, construction sites, and trash dumps.

Avian toxicology is a vast area and to include a full description of all agents potentially toxic to birds is beyond the scope of this book. This section deals with the most common toxicoses reported by clinicians in avian species. For further information on avian toxicology the reader is referred to Petrak, 1982; Cooper, 1985; Harrison, 1986; Osweiler, 1986; Rosskopf and Woerpel, 1986; Humphreys, 1988; LaBonde, 1991, 1996a; Lumeij *et al.*, 1993; Porter, 1993; Dumonceaux and Harrison, 1994; Bauck and LaBonde, 1997; and Lang, 1997.

AMMONIUM CHLORIDE TOXICOSIS

Ammonium chloride (NH_4Cl) or ammonium muriate is an inorganic salt commercially available as hygroscopic, colorless crystals or as a white crystalline powder with a cool saline taste. The dose producing a 50% probability of death (LD_{50}) in the rat is 1650 mg/kg. Its application in both human and veterinary medicine is primarily for acidifying the urine and increasing the rate of urine flow, but it is also widely used as a secretory expectorant and cilia augmentor. This is probably achieved by directly or indirectly increasing the beat frequencies of the cilia in the respiratory tract, but the exact mode of action or the mechanism involved is poorly understood (Brander *et al.*, 1991).

In domestic animals and humans, following ingestion ammonium chloride is metabolized in the liver and converted into urea and hydrochloric acid, resulting in severe acidosis. Excretion takes place via the urinary pathway (Gilman *et al.*, 1985). Birds are uricotelic, excreting the end product of nitrogen metabolism as uric acid. This is synthesized in the liver and excreted by glomerular filtration but mainly by tubular secretion (King and McLelland, 1984). Thus in mammals, and presumably in birds, when a high dose of ammonium chloride is administered orally or in the presence of liver insufficiency, acute hyperammonemia is experienced. As a result, the levels of NH_3 are too high for the liver to detoxify and subsequently act as a cytotoxic agent, mainly in the brain. Sometimes, depending on the dose ingested and the digestive process, carbamates appear as toxic metabolites acting as reversible inhibitors of cholinesterase (Forth *et al.*, 1983).

In the countries around the Persian Gulf, ammonium chloride is best known as *schnather*, an Arabic word widely used by falconers and by people trading in traditional Arab medicine, who normally sell it in the form of crystals. During the initial phase of the hunting period (November), a considerable number of falconers in the Gulf routinely administer ammonium chloride to the falcons under their charge to improve their hunting ability. It is also administered to particular birds that fail to kill or show interest in prey during their first hunting trip. The method normally requires two handlers, one for casting the falcon and the second for forcing a small (10 to 25 mm diameter) crystal of ammonium chloride down into the crop of the immobilized bird. It is also common practice to wrap several small crystals of ammonium chloride in a piece of cotton cloth, forming a small sac, tied at one end with a piece of thin string about 25 cm long. When the small sac is force fed, the other end of the string is left protruding from the mouth so it can be used to retrieve the sac after a few minutes. The theory behind this procedure is that the chemical action of ammonium chloride will remove the "fat deposits within the stomach," resulting in a hungrier bird that is more interested in hunting.

Clinical Signs

Two or three minutes after the administration of the ammonium chloride the falcon usually vomits violently, bringing up large quantities of a thick, green-yellow mucus (Fig. 10-21), sometimes with whitish strands and the partially dissolved crystal. Nevertheless, falconers are very familiar with the toxic effects of this substance and know, probably from previous painful experience, that if the bird is not able to vomit the ingested crystal within 5 to 10 minutes it will undoubtedly die. In this respect, I have witnessed the death of several birds within 15 minutes following ingestion. Sometimes a large ingested crystal breaks down into smaller fragments within the crop, resulting only in partial vomition of the ammonium chloride crystal originally swallowed, an event that usually goes unnoticed by the falconer. In this case the bird soon becomes lethargic and anorexic; loses weight rapidly; and begins passing characteristic, dark metallic-green mutes. During the terminal phase, the bird is unable to stand on its block, remaining on the floor most of the time, and its breathing becomes dyspneic. This is followed by a short period, usually 4 to 8 hours, characterized by fits and opisthotonus followed by death. The clinical signs develop over 3 to 7 days, depending on the total amount of ammonium chloride ingested, although there have been occasions in which the bird died up to 2 weeks later.

FIGURE 10-21 Ammonium chloride toxicosis in a peregrine falcon (*Falco peregrinus*). Three minutes after force feeding the toxic agent, the bird started vomiting. Note the wall smeared with green mucus.

Pathologic Changes

Gross pathologic changes observed at postmortem examinations of affected birds included generalized congestion of the mucosa and the presence of dark, metallic-green mucus along the entire digestive tract. The liver was friable and of a uniform dark, metallic-green color. The kidneys showed mild perirenal edema and mild to severe cortical and medullary congestion. Histopathological findings were nonspecific. The livers showed moderate to severe congestion and golden brown pigment (possibly hemosiderin) within Kupffer cells and macrophages. Other lesions were variable. In some birds these included perivascular cuffing by plasma cells and other mononuclear cells. Subcapsular, well-scattered small foci of early coagulative necrosis and vacuolation of the hepatocyte cytoplasm were also found. Sometimes the necrosis was associated with mononuclear cell infiltration. It is possible that some of these lesions were secondary to bumblefoot. The kidneys showed mild to severe tubular nephrosis. Many tubules were dilated and/or partially occluded with acidophilic or slightly basophilic amorphous material, probably from urate nephrosis.

Physiology and Pathologic Considerations

The biochemical action of ammonium chloride as an acidifier may be responsible for the stimulation of appetite at the central nervous system level. In domesticated animals, it is known that groups of neurons in the lateral hypothalamus promote hunger and that noradrenergic and cholinergic transmitter systems are also implicated in the stimulation of appetite (Klemm, 1984). Probably, and perhaps more correctly, ammonium chloride may be responsible for a chronic chemical irritation of the hunger terminals in the upper digestive tract. In Falconiformes, the taste buds are located on the base of the tongue. There are 30 to 40 axons connecting each taste bud to the central nervous system through the glossopharyngeal nerve (King and McLelland, 1984). The autonomic terminals in the crop, esophagus, and gizzard may also be stimulated by this chemical action, sending constant messages of hunger to the hypothalamus.

Falconers often report that, following administration of ammonium chloride, the falcon looks more alert during hunting and is hungry all the time. It is difficult to assess the validity of this statement but, despite its apparently favorable effect, ammonium chloride remains a highly toxic agent responsible for the death of a significant number of falcons.

ACKNOWLEDGMENT

I am grateful to the *Veterinary Record* for allowing me to reproduce part of Samour JH, Bailey TA, Keymer IF: Use of ammonium chloride in falconry in the Middle East, *Vet Rec* 137:269-270, 1995.

LEAD TOXICOSIS

Lead toxicosis or plumbism is the most common heavy metal toxicity in free-living and captive birds, and is perhaps the most frequent form of intoxication in avian species worldwide. Birds can ingest lead deliberately, when waterfowl ingest shotgun pellets as grit, or accidentally, when captive birds of prey ingest hidden shotgun pellets from a bird or a mammal that has been shot. Lead is relatively insoluble, but small amounts are absorbed throughout the gastrointestinal tract causing a wide variety of clinical signs. These may include acute or chronic forms and the severity usually depends on the amount of lead ingested. Bailey *et al.* (1995) reported lead toxicosis in a flock of houbara bustards (*Chlamydotis undulata*) after ingestion of lead-based paint flakes.

Lead toxicosis is still a major hazard in reintroduction programs of endangered raptor species. For instance, the most common cause of death among California condors (*Gymnogyps californianus*) reintroduced into the Grand Canyon region of Northern Arizona is lead toxicosis. This is produced through the ingestion of lead bullet fragments left in the carcasses of killed and unrecovered animals (Hunt *et al.*, 2009). Crop distention and stasis appear to be common sequelae of led toxicosis in Californian condors. A novel and practical technique for feeding tube placement as part of the therapeutic management of lead toxicosis in this species was recently described (Aguilar *et al.*, 2012). A useful retrospective study to determine risk factors and seasonal trends in lead toxicosis in bold (*Haliaeetus leucocephalus*) and golden (*Aquila chrysaetos*) eagles in the United States inland Pacific Northwest region was recently published (Stauber *et al.*, 2010). A practical method for lead pellet or lead fragment retrieval in falcon species also was recently published (Samour and Naldo, 2005). For further information on lead toxicosis in avian species, refer to Redig *et al.*, 1980; MacDonald *et al.*, 1983; Harrison, 1986; Dement *et al.*, 1986; Mautino, 1990; Dumonceaux and Harrison, 1994; LaBonde, 1996b; and Bauck and LaBonde, 1997. Table 10-6 shows the most common sources of lead and toxic-related clinical signs in birds.

Diagnosis

Figures 10-22 to 10-27 illustrate clinical and postmortem signs of and diagnosis and treatment for lead poisoning.

The diagnosis of lead toxicosis in birds is based on the following:

- Clinical history
- Radiology (presence of radiopaque foreign bodies in the crop and gastrointestinal tract)
- Blood chemistry (increased AST, LDH, and CPK)
- Hematology (low hemoglobin and red blood cell [RBC] count, intracytoplasmic vacuoles, and basophilic stippling)
- Blood analysis (blood levels of 20 mg/dL are indicative of lead toxicity and levels of 50 mg/dL or more confirm the diagnosis)
- Tissue analysis (tissue wet levels of 3 and 6 ppm are indicative of lead toxicity and wet levels of 6 ppm or more confirm the diagnosis)

TABLE 10-6 Sources and Clinical Signs Related to Lead Toxicosis

Source	Clinical Signs
Lead-based paint, lead-free paint with leaded drying agents, fishing weights, shotgun pellets, batteries, linoleum, plaster, masonry putty, petrol fumes, lead-coated household and industrial items	General signs: Weakness, weight loss, lethargy, anorexia Hematological signs: basophilic stippling, red blood cell intracytoplasmic vacuolization Renal signs: polyuria, hematuria, hemoglobinuria Gastrointestinal signs: diarrhea, dark feces, ileus of upper digestive tract, regurgitation Neurologic signs: ataxia, head tremors, circling, head tilt, dropped wings, paresia, hyperesthesia, paralysis, blindness, convulsions

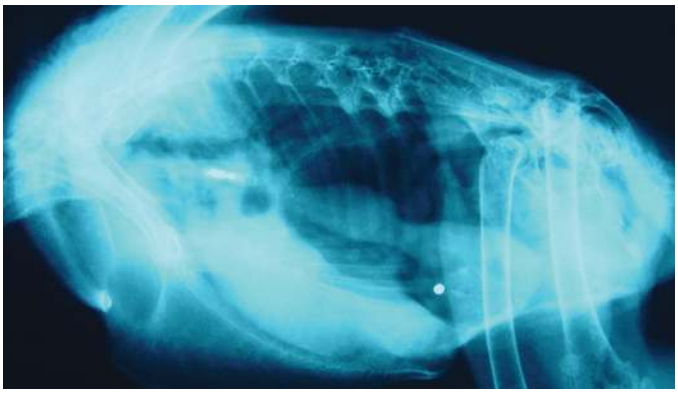


FIGURE 10-22 Laterolateral radiograph of a saker falcon (*Falco cherrug*) showing a lead pellet from a shotgun within the ventriculus. In the Middle East, as in other countries, falcons are commonly intoxicated with lead after eating small birds shot with air rifles or shotguns that have lead pellets or lead pellet fragments in their bodies.

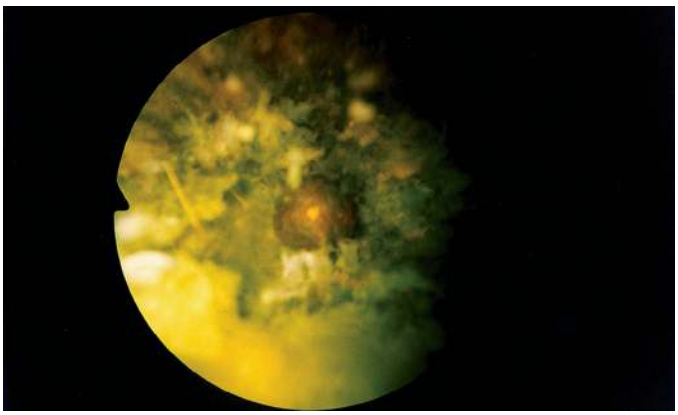


FIGURE 10-23 Endoscopic view of the lead pellet within the ventriculus of the same falcon, as in Fig. 10-22.

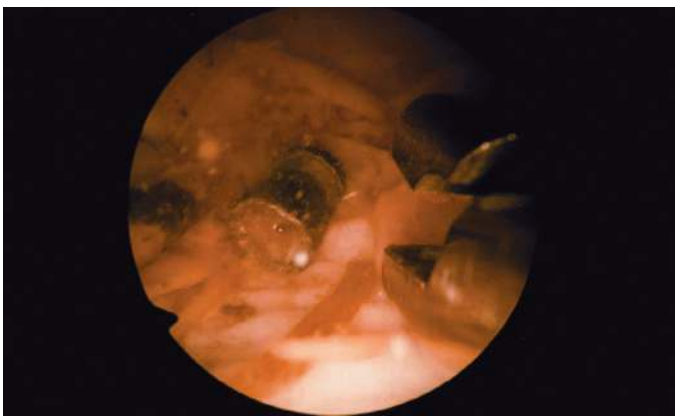


FIGURE 10-24 A pellet from an air rifle within the ventriculus of a gyrfalcon (*Falco rusticolus*). Endoscopic-assisted pellet removal using either flexible or rigid endoscopes is sometimes necessary when stomach flushing fails to dislodge them from the ventriculus.

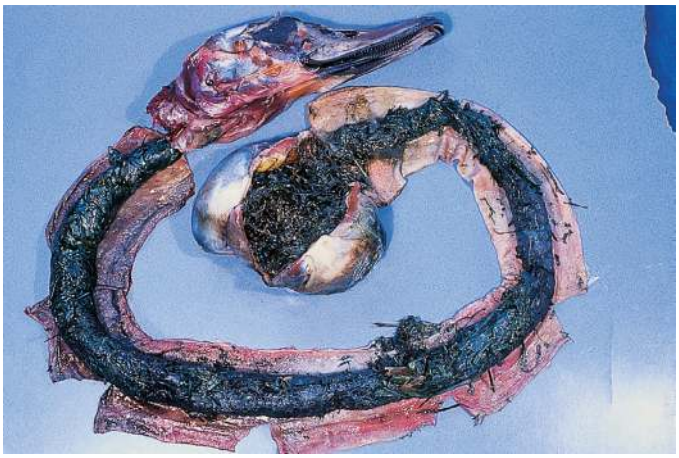


FIGURE 10-25 Mute swan with esophagus, proventriculus, and gizzard opened to show impaction with vegetable matter. (Courtesy A. Hunt.)

FIGURE 10-26 A similar case of lead poisoning in a goshawk (*Accipiter gentilis*). (Courtesy N. A. Forbes.)

Traditionally, blood lead levels in veterinary medicine have been estimated using graphite furnace atomic absorption spectrometry. However, a new electrochemical method (LeadCare System) has recently been introduced for use with avian species (Samour and Naldo, 2002). This system relies on electrochemistry and a sensor to detect lead levels in whole blood. Most of the lead is carried in the erythrocytes, and when whole blood is mixed with the treatment reagent the lead present in the erythrocytes is removed and made available for detection. When a test is run, the analyzer causes the lead to collect on the sensor. After a short time, the analyzer removes the lead, measures it, and converts the results into a displayed blood lead level. The results are expressed in mg/dL. The upper measuring range of the analyzer is 65 mg/dL. Results greater than this value are expressed as "high."

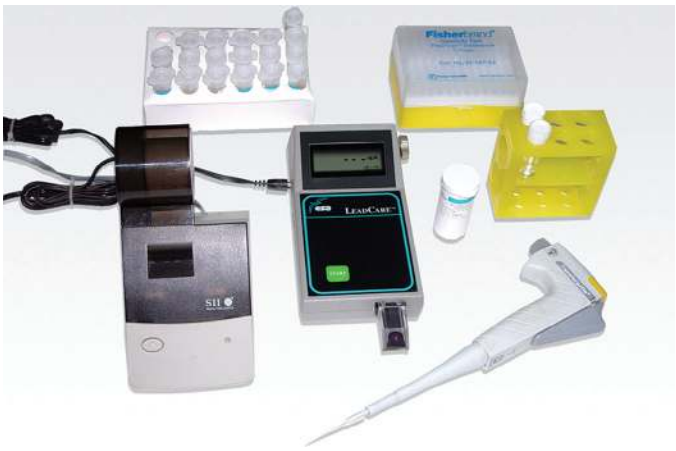


FIGURE 10-27 The LeadCare System is widely used in the diagnosis of lead toxicosis in birds. It relies on electrochemistry and a sensor to detect lead levels in whole blood.

Treatment

Primary Treatment

Primary treatment includes removal of foreign bodies (crop or gastric lavage, endoscopy, and surgical).

Chelative Therapy

- Calcium disodium ethylene diamine tetraacetate ($CaNa_2$ EDTA) 10 to 40 mg/kg intramuscularly (IM) twice daily. A recent publication reported the use of a 25% solution of $CaNa_2$ EDTA in falcons at the dose rate of 100 mg/kg, undiluted IM twice a day for 5 to 25 consecutive days without observing any deleterious effect (Samour and Naldo, 2004)
- D-penicillamine (PA) 55 mg/kg by mouth twice daily for 10 days
- Dimercaprol (BAL) 2.5 mg/kg IM every 4 hours for 2 days, followed by twice daily administration until the clinical signs are resolved
- Dimercaptosuccinic acid 25 to 35 mg/kg by mouth twice daily 5 days a week for 3 to 5 weeks.

Support Therapy

Support therapy consists of intravenous or subcutaneous glucose/electrolyte fluids, corticosteroids, antibiotics, vitamin B_{12}, vitamin B_1, antifungal agents, and magnesium sulfate.

ZINC TOXICOSIS

Zinc toxicosis or "new wire disease" is a relatively common toxic condition mainly affecting captive birds. Newly manufactured galvanized wire and newly manufactured galvanized watering and feeding containers are usually the main source of zinc toxicosis in captive birds. Some galvanized coatings may contain up to 99.9% zinc and others may have 98% zinc and 1% lead (Howard, 1992). Some coins also have a high percentage of zinc.

In one instance, a mallard duck (*Anas platyrhynchos*) was brought to a clinic for clinical examination. The bird was weak, lethargic, and unable to walk. An x-ray showed a large, irregularly shaped foreign body in the gastric area. Surgery was performed and a stack of 12 worn out British 2 pence coins was retrieved from its gizzard. Tissues were not analyzed but a presumptive diagnosis of zinc toxicosis was made (J. Samour, unpublished observation). Zinc toxicosis has been recorded in a black bustard (*Eupodotis afra*; Lloyd, 1992) and in a hyacinth macaw (*Anodrhynchus hyacinthus*; Romagnano *et al.*, 1995). More

recently, zinc toxicosis was diagnosed in a flock of orange-bellied parrots (*Neophema chrysogaster*) housed in a newly built aviary. The parrots did not show any sign of disease before death. Most birds were found dead with no obvious histologic lesions. Affected birds had a mean zinc level of 154.3 mg/g in the kidneys, 289.8 mg/g in the liver, and 723.6 mg/g in the pancreas (Holz *et al.*, 2000). A recent report gave a detailed description of the diagnosis and treatment of zinc toxicosis in a wattled crane (*Bugeranus carunculatus*) from a zoologic collection (Barrows *et al.*, 2005). The clinical signs of zinc toxicosis are very similar to those described for lead. For additional information on zinc toxicosis in other species of birds, the reader is referred to Harrison, 1986; Howard, 1992; Dumonceaux and Harrison, 1994; LaBonde, 1996a; Bauck and LaBonde, 1997; and Van Sant, 1997. Table 10-7 presents the most common sources of zinc and toxicity-related signs.

Diagnosis

Zinc toxicosis is usually diagnosed by

- Clinical history
- Radiology
- Blood analysis (zinc levels of 200 mg/dL are suggestive of zinc toxicosis)
- Tissue analysis (pancreatic tissue zinc levels greater than 1000 mg/g are suggestive of zinc toxicosis)

Treatment

Treatment of zinc toxicosis is very similar to the treatment described for lead toxicosis.

Primary Treatment

Primary treatment is removal of foreign bodies (crop or gastric lavage, endoscopy, and surgical).

Chelative Therapy

- $CaNa_2EDTA$ 10 to 40 mg/kg IM twice daily
- PA 55 mg/kg by mouth twice daily for 10 days
- BAL 2.5 mg/kg IM every 4 hours for 2 days, followed by twice daily administration until the clinical signs are resolved

Note: $CaNa_2$ EDTA or PA are useful chelative agents, but BAL is perhaps the most indicated agent in the case of zinc toxicosis.

Support Therapy

Support therapy is as described for lead toxicosis.

COPPER TOXICOSIS

There are scant reports in the literature concerning copper toxicosis in avian species. This may be because copper is less widely used around birds or bird facilities than lead or zinc. Frank and Borg (1979) reported a liver copper level greater than 3000 mg/kg and a kidney copper level greater than 50 mg/kg in a case involving a mute swan (*Cygnus olor*) displaying typical clinical signs associated with copper toxicosis. I witnessed a case of copper toxicosis in a brown kiwi (*Apteryx australis*) in a zoologic collection after the ingestion of three segments, 3 to 5 cm long, of electrical copper wire left in the enclosure by electricians on routine maintenance work. The liver copper level in the kiwi was nearly 3500 mg/kg (J. Samour, unpublished observation). For more information concerning copper toxicosis in other species of birds the reader is referred to Dumonceaux and Harrison, 1994. The potential sources of copper and related clinical signs of copper toxicosis are listed in Table 10-8.

TABLE 10-7 Sources and Clinical Signs Related to Zinc Toxicosis

Source	Clinical Signs
Galvanized wire and mesh, galvanized feeding and watering containers, some coins	Lethargy, weight loss, anemia, regurgitation, polydipsia, polyuria, hyperglycemia, ataxia, convulsions

Diagnosis

Copper toxicosis can be diagnosed by

- Clinical history
- Radiology
- Tissue analysis

Treatment

Chelative Therapy

PA 52 mg/kg/day by mouth every 6 days has been recommended for mammals.

Support Therapy

Support therapy is as described for lead toxicosis.

BOTULISM

Botulism (Figs. 10-28 and 10-29) is a toxic neuroparalytic disease produced by the ingestion of toxins of *Clostridium botulinum*. There are at least seven types of toxin produced by the different strains of *C. botulinum*. Type C is responsible for most of the toxicosis reported in birds worldwide. Types A and E, which are more relevant to human botulism, have also been implicated in toxicity outbreaks in birds. *C. botulinum* is an anaerobic, spore-forming, motile, and gram-positive bacillus commonly found associated with putrefying vegetable matter in marshes and wetlands and decomposing animal carcasses. It can also be found in badly stored and decomposing grain, silage, and hay.

Outbreaks of botulism have been widely documented in waterfowl, but seagulls, terns, and other aquatic species can be affected. Botulism

TABLE 10-8 Sources and Clinical Signs Related to Copper Toxicosis

Source	Clinical Signs
Electrical wire, some coins, excessive copper dietary supplementation, antialgae agents (copper sulfate)	Anemia, weakness, weight loss, lethargy Postmortem finding: metal-black liver appearance

FIGURE 10-28 A great black-headed gull (*Larus ichthyaetus*) affected with *Clostridium botulinum* toxicosis. Note the typical clinical sign of wing paralysis manifested through the "dropped wing" appearance. Gulls are scavengers and are very often affected with *C. botulinum* toxicosis when feeding from trash dumps near human habitation. (Courtesy Dr. U. Wernery.)

FIGURE 10-29 Typical wasp-waist appearance of a white mouse after intraperitoneal inoculation with *Clostridium botulinum* toxin. (Courtesy Dr. U. Wernery.)

FIGURE 10-30 Postmortem examination of a gyrfalcon (*Falco rusticolus*). The falcon died of enterotoxemia caused by *Clostridium perfringens*. Note the highly congested intestine. (Courtesy Dr. U. Wernery and Dr. J. Kinne.)

has also been documented in captive ostriches (Shakespeare, 1995). In the Middle East, this disease has been observed in feral domestic pigeons (*Columba livia*) and collared doves (*Streptopelia decaocto*) after eating proprietary pellets that had been badly stored in a silo (J. Samour, unpublished observation). This toxic condition is more frequently reported during the hottest months of the year because of the increased alkalinity of stagnant water and the anaerobic conditions created on the substrate of ponds and marshes. The toxins of *C. botulinum* affect the releasing mechanism of acetylcholine at the terminal sections of the peripheric nerves, causing an acute, flaccid, and descending paralysis. Other clinical signs include dyspnea, hypersalivation, nasal and ocular discharge, and diarrhea. For more information on botulism in avian species, refer to Bennett, 1994; LaBonde, 1996a; and Gerlach, 1997 (Figs. 10-30 and 10-31).

Diagnosis

Botulism can be diagnosed by

- Clinical history
- Culture
- Tissue toxin analysis (submit frozen liver and kidneys)
- Water and feed toxin analysis (submit frozen)
- Mouse inoculation neutralization assay

Treatment

Primary Therapy

Primary therapy includes antitoxin administration (0.05 to 1 mL/day).

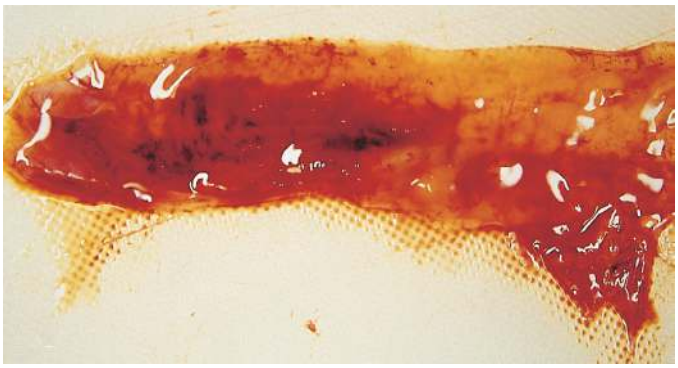


FIGURE 10-31 Intestinal mucosa of the gyrfalcon in Fig. 10-30. Note the extensive hemorrhagic changes as a results of severe enteritis. (Courtesy Dr. U. Wernery and Dr. J. Kinne.)

Support Therapy

- Drenches, cathartics, and laxatives
- Tube feeding
- Intravenous or subcutaneous glucose/electrolyte fluids, antibiotics, and vitamins B_{12} and B_1

MYCOTOXICOSIS

Mycotoxicosis is a general term used to describe a series of toxic conditions caused by the ingestion of feed contaminated with the toxins of different saprophytic and phytopathogenic fungi and molds. These toxins are normally referred to as mycotoxins. Mycotoxins are secondary metabolites that are not produced for the benefit of the fungus or mold.

Fungi and molds commonly grow on basic feed ingredients and pelletized commercial feed if stored for a long period of time and under suboptimal temperature and relative humidity conditions. Some fungi even grow on the crop itself when the environmental conditions are suitable. Dumonceaux and Harrison (1994) provided a more detailed account of mycotoxicosis in birds. An account of the effect of mycotoxins (aflatoxin B_1 and vomitoxin) in young ostrich chicks was described by Scheideler and Kunze (1997). As outlined by Pier (1990), aflatoxin is toxic to ducklings in an oral LD_{50} dose of 0.36 mg/kg and to chicks in an oral LD_{50} dose of 6.5 mg/kg.

In a recent study, aflatoxin B_1, dissolved in dimethyl sulfoxide, was administered to three groups of pigeons (*C. livia*) at the dose rate of 3 mg/kg daily for 2, 4, and 6 consecutive days. Mortality was observed in all three groups of pigeons, but more significantly in the group receiving aflatoxin for 6 days. Significant hepatic changes were observed in pigeons from each group. A fourth group was given 3 mg/kg for 2 consecutive days to induce acute hepatic damage without causing death of the participating pigeons. Even though the authors observed increased liver enzyme activity suggesting hepatocellular damage, bile acid analyses and hepatobiliary scintigraphy did not indicate significantly decreased hepatic function (Hadley *et al.*, 2010).

The most important mycotoxins relevant to birds produced by fungi or molds are listed in Table 10-9.

Diagnosis

Mycotoxicosis can be diagnosed by

- Clinical history
- Postmortem changes
- Histopathologic analysis
- Toxin quantitative analysis in food and gastrointestinal contents

TABLE 10-9 Relevant Mycotoxins in Avian Medicine Produced by Fungus or Mold

Mycotoxin	Fungus or Mold	Clinical and Pathologic Signs
Aflatoxin B_1	*Aspergillus parasiticus, A. flavus, A. fumigatus*	Anorexia, lethargy, CNS signs, sudden death, hepatitis, splenitis, pancreatitis
Ochratoxin A	*Aspergillus ochraceus, Penicillium citrinum, P. viridicatum*	CNS signs, hepatotoxic and nephrotoxic signs, immune system and bone marrow suppression
Vomitoxin (deoxynivalenol)	*Fusarium roseum, Gibberella zeae*	Vomiting, regurgitation, diarrhea
Trichothecenes, satratoxins, T_2 toxin, diacetoxyscirpenol	*Stachybotrys atra, Fusarium roseum,* F. *scirpi, F. tricinctum, F. equiseti, F. culmorum*	Necrotic ulcerative lesions of the upper digestive tract, flaccid neck and wing paralysis, contact dermatitis, distal necrosis

CNS, Central nervous system.

Treatment

Attempting to treat clinical cases affected with aflatoxicosis is usually unrewarded. However, with other mycotoxicosis, if not chronic or severe, the cases usually resolve when the source of toxicity is withdrawn, with the aid of support therapy.

PHARMACOLOGIC COMPOUNDS TOXICOSIS

There are numerous pharmacologic compounds that are potentially toxic to birds. In most cases, the toxic effect of a particular compound is related to the administration of a dose higher than recommended. Conversely, the toxicity may be from the administration of a particular compound for a period of time longer than normally recommended (Harrison, 1986; Dumonceaux and Harrison, 1994; LaBonde, 1996a; Bauck and LaBonde, 1997). A recent study reported suspected fenbendazole toxicosis in two vulture species and Marabou storks. A group of 10 African white-backed vultures (*Gyps africanus*), three lappet-faced vultures (*Torgos tracheliotus*), and six Marabou storks (*Leptoptilos crumeniferus*) were routinely treated for gastrointestinal parasites using 47 to 60 mg/kg for 3 consecutive days. Six white-backed and one lappet-faced vultures and one Marabou stork died after a short period of depression and anorexia. Hematology analyses revealed profound leukopenia in all cases. On histology examination, severe necrotizing enteritis, bacterial hepatitis, and evidence of septicemia were found (Bonar *et al.*, 2003).

The past 10 years have seen a sharp decline in the population of at least three vulture species in the Indian subcontinent. The species involved include the long-billed vulture (*G. indicus*), slender-billed vulture (*G. tenuirostris*). and Oriental white-backed vulture (*G. Bengalensis*; Prakash, 1999; Gilbert *et al.*, 2002). This crisis prompted an international response to investigate the cause of the decline.

Extensive health screening failed to detect a common cause for the extensive mortality. Postmortem examination, however, found severe visceral gout in over 85% of the carcasses examined. Extensive toxicologic investigation for heavy metals, pesticides, and herbicides did not reveal any significant finding. On further toxicologic examination, the nonsteroidal antiinflammatory drug diclofenac, in residues of 0.051 to 0.643 mg/g of kidney, was detected in 25 of 25 vultures that had visceral gout (Green *et al.*, 2004; Oaks *et al.*, 2004; Risebrough, 2004). The control group, consisting of vultures that had died of other causes, proved negative for the presence of diclofenac. In 2005 the government of India announced the phasing out of diclofenac for veterinary use within 6 months (Anonymous, 2006).

Table 10-10 shows the clinical and pathologic signs associated with the use of certain pharmacologic compounds in birds.

TABLE 10-10 Potential Toxic Effects of Common Pharmacologic Compounds in Birds

Pharmacologic Compound	Clinical and Pathologic Signs
Antibiotics	
Cephalosporins	Nephrotoxic, hepatotoxic
Chloramphenicol	Nephrotoxic
Gentamicin	Nephrotoxic
Doxycycline	Tissue necrosis, cartilage abnormalities in growing birds
Ticarcillin	In combination with tobramycin may be hepatotoxic
Oxytetracyclines	Tissue necrosis, inflammation, nephrotoxic, prolonged use may depress gut flora
Trimethoprim–sulfa drug combinations, furazolidone	Regurgitation, general depression, gastrointestinal tract stasis
Tylosin	Convulsions
Antifungal	
Amphotericin B	Nephrotoxic, hepatotoxic, vomiting, convulsions
Flucytosine	Anemia, bone marrow depression, leukopenia
Anthelmintics/Antiparasitics	
Fenbendazole	CNS signs
Ivermectin	Lethargy, depression and death in some small psittacines when administered IM
Levamisole	Regurgitation, ataxia, dyspnea, hepatotoxic
Praziquantel	General depression, death
Antiprotozoal	
Dimetridazole	CNS signs, hepatotoxic
Vitamins	
A	Osteodystrophy, parathyroid hyperplasia, dermatitis
D_3	Mineralization of organs, nephrosis, increase serum calcium level
B_6	Hepatotoxicity, nephritis, general depression, vomiting, anorexia, death
Anticoccidial/Antiprotozoal	
Monensin	Ataxia, dyspnea, degenerative myopathy, death

CNS, Central nervous system; IM, intramuscular.

PYRIDOXINE (VITAMIN B_6) TOXICOSIS

Clinical Case

A group of 12 of 16 5- to 6-month-old, female gyr falcons (*Falco rusticolus*) died following the IM injection of 1 mL of a vitamin B complex preparation containing 20 mg thiamine (B_1), 5 mg riboflavin-6 phosphate (B_2), 200 mcg hydroxocobalamin (B_{12}), 60 mg nicotinamide (B_3), 17 mg d-panthenol (B_5), and 20 mg pyridoxine hydrochloride (B_6) per 1 mL suspended in water for injection. The reason behind the administration of such a large volume of this vitamin B complex preparation was to provide 20 mg/kg of vitamin B_1 to each falcon in accordance to the dose of vitamin B complex widely recommended in the avian medicine literature. The falcons were all clinically normal and in top physical condition after undergoing a 6-week falconry training program. The mean bodyweight of the falcons was 1300 g.

After the injection, clinical symptoms began developing gradually in severity starting within 30 min with the passing of pistachio green–colored urates, regurgitation, and vomiting, progressing rapidly to a complete lack of appetite, total absence of preening and interacting activity, almond-shaped eyes, collapse, and death. Death occurred between 24 to 36 hours after injection. Three birds showed vomiting and frothing of partially digested blood, clonic spasms, and convulsions before death. Gross postmortem findings included hepatitis, splenitis, and nephritis, and, in three cases, moderate to severe hemorrhagic gastroenteritis. Histopathology findings revealed acute changes including widespread hemorrhages in the liver with vacuolization and lytic necrosis of hepatocytes, loss of nuclei, degeneration, and necrosis of individual cells; the spleen was also hemorrhagic, completely depleted of lymphoid tissue, with loss of PALS, necrosis of the arteries and empty sinusoids with scattered histiocytes and red blood cells (RBCs); and the kidneys showed hyperemia with the tubules largely degenerated or necrotic with pycnotic nuclei. Anaerobic bacteriology cultures made from swabs of intestinal content obtained from the three individuals displaying central nervous system signs revealed mild to moderate growths of *C. perfringens.* Four individuals showed mild to moderate clinical symptoms but recovered within 48 hours through intensive support therapy (Samour, 2013).

Comparative Posology Assessment

Comparative analysis of the preparation used showed the content of the B complex to be similar to those available in injectable multivitamin preparations widely used in falcons across the Middle East. For instance, in one of the most commonly used injectable products, the content of vitamin B_1 is 10 mg, B_2 is 5 mg, B_6 is 3 mg, B_{12} is 25 mcg, B_3 is 35 mg, and B_5 is 25 mg per 1 mL. Vitamin B_1 and B_{12} preparations are available on their own in veterinary formulations and are widely used as part of support therapy strategies in falcon medicine at much higher doses across the Middle East. In this respect, doses of vitamin B_1 from 25 mg up to 50 mg/kg IM and doses of vitamin B_{12} from 50 mcg up to 100 mcg/kg IM/kg are routinely used by clinicians in falcons without observing any deleterious effect.

It was therefore assumed that the content of vitamin B_1 and vitamin B_{12} in the preparation could not have been implicated in the death of the falcons. Vitamins B_2, B_3, B_5, and B_6 are not available on their own in veterinary formulations, but they all form an integral part of the composition in multivitamin preparations commonly used in falcons. These have been used extensively at similar or slightly higher dose rates with the exception of vitamin B_6, as the content of this vitamin was over six times higher in the B complex preparation reputedly responsible for the death of the falcons. To the author's knowledge, there are no reports of toxicity on the use of vitamins B_1, B_2, B_{12}, B_3, and B_5 at such dose rates in the avian medicine literature. Therefore the suspicion for the death of the 12 falcons focused on the high content of vitamin B_6 in the preparation used. This suspicion was further reinforced after becoming aware of a similar case of acute toxicity in domestic pigeons (*C. livia*) following the administration of 1 tablet by mouth of a human vitamin B complex preparation containing 100 mg of vitamin B_1, 150 mg of B_6, and 15 mcg of B_{12}. This study concluded that vitamin B_6 was responsible for the death of the pigeons after each of the vitamins or the combination of the three vitamins was administered by mouth to various groups of pigeons. The authors suggested that the lethal dose of B_6 was estimated at 90 to 100 mg by mouth per pigeon or 200 mg/kg by mouth (Peeters *et al.*, 1977).

Experimental Studies

To confirm this hypothesis, vitamin B_6 (BP grade) in powder form was diluted in water for injection and administered IM to three groups of male falcons. Each falcon in Group 1 received 20 mg/kg IM, in Group 2 each falcon received 15 mg/kg IM, and in Group 3 each falcon received 10 mg/kg IM. All falcons in the three groups died within 24 hours. In a separated trial, two additional groups of four falcons in each group were administered a human medicine oral B complex preparation containing 100 mg of vitamin B_1, 200 mcg of B_{12}, and 200 mg of B_6 per tablet. Each falcon in Group 1 received 75 mg/kg B_6 by mouth and in Group 2 each falcon received 50 mg/kg B_6 by mouth. Similarly, all falcons in both groups died within 24 to 36 hours.

Gross postmortem findings included moderate to severe congestion of the lungs and enlarged pale liver. The most relevant histopathology findings included marked congestion and perivascular hemorrhages of the lungs, acute moderate to severe degeneration of the liver, and mild to marked demyelinization of the brain. The histopathological findings in the two groups of falcons were similar to those observed in the group of 12 falcons that died previously and are consistent with severe and acute toxic changes.

C. perfringens was isolated from liver and intestinal content from most individuals. The isolates were α toxin producers as shown by ELISA testing. As in the previous cases, the presence of *C. perfringens* was considered secondary to the primary cause of death (Samour, 2013).

An additional study was carried out in two groups of female gyr falcons, four in each group. All falcons in Group 1 received 5 mg/kg B_6 IM and in Group 2 each falcon received 25 mg/kg B_6 by mouth. None of the participating falcons in the two groups became ill or died. These appear to be the maximum safe doses of vitamin B_6 for clinically normal falcons.

This report highlights the need to more carefully examine the recommended dose rates of the different vitamins contained in B complex preparations used in falcons. This may also be applicable to other avian species.

ACKNOWLEDGMENTS

I would like to thank Janine Perlman, Victoria Brookes, Greg Harrison, Gerry Dorrestein, Ulrich Wernery, Joerg Kinne, Vijay Baskar, Tony Gestier, Ben Minter, Peter Coutteel, Harald Kuspert, and to all those who contributed to this study.

PESTICIDE TOXICOSIS

Pesticide is a broad term that encompasses groups of chemicals commonly used to eradicate unwanted and destructive animals and plants. These different chemical compounds can be classified under three main groups: insecticides, rodenticides, and herbicides.

Most cases of pesticide toxicosis in birds occur by negligence or by accident. Very often the instructions of manufacturers are not properly followed and the pesticide is applied directly to birds or in aviaries. In this respect, I have witnessed several cases in which bird owners trying to treat their birds for flea or lice infestation had sprayed the birds directly with products commonly used for flies and cockroaches. Conversely, pesticides are commonly applied or placed in areas where birds have direct access to and ingest them, either in their pure form or through contaminated food or water.

The clinical signs vary significantly depending on the compound and degree of toxicosis. The most commonly encountered clinical signs include gastrointestinal disorders, such as anorexia, regurgitation, vomiting and diarrhea, central nervous system signs, convulsions, dyspnea, cyanosis, and death.

The diagnosis is usually based on clinical history and forensic examination for a particular toxic compound. The treatment of severe cases of pesticide toxicosis is usually unrewarded, but in some cases health can be restored to normal by withdrawing the toxic source and by additional support therapy.

A series of clinical cases of possible cholecalciferol rodenticide toxicosis involving several avian species housed in a zoologic collection was recently described (Swenson and Bradley, 2013). The cholecalciferol rodenticide had been placed around bird enclosures as part of a vermin control performed at the premises. On several occasions keeping staff was able to observe the actual cholecalciferol pellets within the bird enclosures presumably brought in and left on the ground by rodents. The presumptive diagnosis was based on the anamnesis, abnormal blood chemistry values, and mineralization of tissues. The authors concluded that although cholecalciferol is not expected to produce relay toxicosis in birds, primary toxicosis should be suspected in the presence of unspecific clinical signs and the use of cholecalciferol in and around the premises (Swenson and Bradley, 2013).

For more detailed accounts of pesticide toxicosis in birds, the reader is referred to Harrison, 1986; Lumeij *et al.*, 1993; Porter, 1993; Dumonceaux and Harrison, 1994; LaBonde, 1996a; and Bauck and LaBonde, 1997. Tables 10-11 to 10-13 illustrate the most common pesticides that are potentially toxic to birds.

TABLE 10-11 Common Insecticides Potentially Toxic to Birds

Toxic Compound	Source/Action
Chlorinated hydrocarbons	Currently used around live animals: lindane, methoxychlor, toxaphene Toxic for use around live animals: aldrin, dieldrin, benzene hexachloride, chlordane, endrin
Organophosphates	Dichlorvos, malathion, parathion, diazinon, fenthion, trichlorfon, coumaphos, acephate, chlorpyrifos, dimethoate
Carbamates	Carbaryl, carbofuran, methomyl, propoxur, bendiocarb
Compounds of plant origin	Pyrethrins: pyrethrum is a widely used insecticide extracted from the flowers of *Chrysanthemum cinerariaefolium* Pyrethroids: synthetic preparations made from pure pyrethrins including allethrin, cypermethrin, decamethrin, fenvalerate, fluvalinate, permethrin, tetramethrin

TOXIC PLANTS

There is widespread controversy in the literature as to which species of plants and which part of the plant offer a definite risk of toxicity to birds. There also appears to be a discrepancy as to which bird species are susceptible to toxicosis after the ingestion of certain plants.

A large number (greater than 579) of greater flamingos (*P. rubber*) died during the summer of 2001 in Doñana National Park in Spain. Other species of waterfowl also perished in the same outbreak. The suspected cause was the sudden presence of a dense water bloom of cyanobacteria but mainly *Microcystis aeruginosa* and *Anabaena flos-aquae* (Alonso-Andicoberry *et al.*, 2002).

A series of cases of necrotic dermatitis from photosensitization in waterfowl associated with consumption of perennial rye grass (*Lolium perenne*) was recently reported (Rostami *et al.*, 2011). Affected birds included a number of mute (*C. olor*) and whopper (*C. cygnus*) swans and mixed breed domestic geese (*Anser domesticus*). The enclosure housing the birds had been planted with perennial rye grass 3 years previously and the birds were seen grazing on a daily basis. The swans and geese had a history of developing similar lesions from March to September the previous 3 years, which had resolved spontaneously by the end of the year. Other waterfowl species housed in the same enclosure were not affected presumably because of different feeding habits. Affected birds showed vesicular and necrotic dermatitis involving the feet, eyelids, and beak. All birds recovered completely after affected individuals were removed from the enclosure (Rostami *et al.*, 2011).

Most of the plant species listed as toxic to birds have been widely documented as toxic to mammals, especially herbivores, or to humans. However, there are only a handful of clinical cases in which toxicosis

TABLE 10-12 Common Rodenticides Potentially Toxic to Birds

Toxic Compound	Source/Action
Anticoagulant rodenticides	Warfarin, brodifacoum, coumafuryl
Pyriminil	Vacor
Zinc phosphide	Used widely since affected rodents commonly die in the open
Crimidine	Castrix

TABLE 10-13 Common Herbicides Potentially Toxic to Birds

Toxic Compound	Source/Action
Aliphatic herbicide	Glyphosate
Plant hormone herbicides	2-4-dichlorophenoxyacetic acid, 2,4,5-trichlorophenoxyacetic acid, 2-methyl-4 chlorophenoxyacetic acid, 2,2-dichloropropionic acid
Triazine compounds	Atrazine, cyanazine, prometryn, propazine, metribuzin, simazine
Thiocarbamate compounds	Barban, chlorpropham, diallate, pebulate, triallate, vernolate
Phenylurea compounds	Diuron, fenuron, linuron, monolinuron, norea
Pentachlorophenol	Also used as fungicide, insecticide, wood preservative and molluscicide

from toxic plants has been diagnosed (Harrison, 1986; Dumonceaux and Harrison, 1994; LaBonde, 1996a; Bauck and LaBonde, 1997). A recent report documented toxicosis in a 9-month-old budgerigar (*Melopsittacus undulatus*) after ingestion of freshly harvested crown vetch (*Coronilla varia*) leaves. The bird eventually recovered after supportive care and activated charcoal. Crown vetch contains the potent neurotoxin nitrotoxin B-nitropropionic acid (Campbell, 2006). Table 10-14 lists the plant species mentioned in the literature as potentially toxic to birds.

OTHER TOXIC COMPOUNDS

There is a wide range of household and industrial products that are potentially toxic to birds (Fig. 10-32). Sporadic reports appear in the literature with definite accounts of toxicosis from particular compounds (Dumonceaux and Harrison, 1994; LaBonde, 1996a; Lang, 1997). Table 10-15 illustrates some of the most common toxicoses related to various miscellaneous compounds.

Figure 10-33 illustrates an unusual case of toxicosis in a saker falcon (*Falco cherrug*) bitten on the hind talon by a sand snake (*Psammophis schokari*). Snake bite is commonly reported in domestic mammals but, to my knowledge, has never been documented in a bird.

Recently an interesting incident of suspected chocolate toxicity was reported in an adult, male African grey parrot (*Psittacus erithacus*). The parrot was taken to a veterinarian approximately 12 hours after ingesting a large chocolate donut. The bird died 24 hours after presentation. The gross postmortem changes observed and subsequent

TABLE 10-14 Plants Potentially Toxic to Birds

Scientific Name	Common Name	Scientific Name	Common Name
Amarylidaceae	Amaryllis	*Convallaria majalis*	Lily of the valley
Rhododendron occidentale	Azalea	*Lobelia* spp.	Lobelia
Persea americana	Avocado	*Astragalus* and *Oxytropis* spp.	Locoweed
Poinciana gilliesii	Bird of paradise	*Kalanchoe* spp.	Maternity plant
Acepodium podograria	Bishop's weed	*Cannabis sativa*	Marijuana
Robinia pseudoacacia	Black locust	*Phoradendron villosum*	Mistletoe
Microcystis aeruginosa	Blue-green algae	*Asclepias* spp.	Milkweed
Buxus sempervirens	Boxwood	*Prunus caroliniana*	Mock orange
Arctium minus	Burdock	*Aconitum* spp.	Monkshood
Ranunculaceae	Buttercup	*Ipomoea* spp.	Morning glory
Caladium spp.	Caladium	*Kalmia latifolia*	Mountain laurel
Trichodesma incanum	Camel bush	*Narcissus* spp.	Narcissus
Ricinus communis	Castor bean	*Solanum* spp.	Nightshade
Prunus spp.	Cherry	*Quercus* spp.	Oak
Montana rubens	Clematis	*Nerium oleander*	Oleander
Sesbania vesicaria	Coffee bean	*Petroselinum sativum*	Parsley
Caltha palustris	Cowslip	*Lolium perenne*	Perennial rye grass
Coronilla varia	Crown vetch	*Philodendron scandens*	Philodendron
Datura spp.	Datura	*Euphorbia pulcherrima*	Poinsettia
Daphne spp.	Daphne	*Phytolacca americana*	Pokeweed
Dieffenbachia spp.	Dieffenbachia	*Conium maculatum*	Poison hemlock
Hedera helix	English ivy	*Solanum tuberosum*	Potato (shoots)
Alocasia and *Colocasia* spp.	Elephant's ear	*Abrus precatorius*	Precatory bean
Claviceps purpurea	Ergot	*Ligustrum vulgare*	Privet
Digitalis purpurea	Foxglove	*Rhododendron simsii*	Rhododendron
Conium maculatum	Hemlock	*Rheum rhaponticum*	Rhubarb
Hyacinthus orientalis	Hyacinth	*Abrus precatorius*	Rosary pea
Hydrangea spp.	Hydrangea	*Symplocarpus foetidus*	Skunk cabbage
Iris spp.	Iris	*Ornithogalum umbellatum*	Snowdrop
Arisaema spp.	Jack-in-the-pulpit	*Nicotiana* spp.	Tobacco
Solanum pseudocapsicum	Jerusalem cherry	*Parthenocissus* spp.	Virginia creeper
Datura stramonium	Jimsonweed	*Wisteria* spp.	Wisteria
Juniperus virginiana	Juniper	*Taxus media*	Yew
Delphinium spp.	Larkspur		

Adapted from Harrison GJ: Toxicology. In Harrison GJ, Harrison LR, editors: *Clinical avian medicine and surgery*, Philadelphia, PA, 1986, WB Saunders and LaBonde J: Toxic disorders. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, MD, 1996a, Williams & Wilkins.

FIGURE 10-32 An immature tern partially covered with crude oil. Oil spills are very common around coastal areas of the world and every year are responsible for the death of hundreds of seashore birds. Death usually occurs as a result of the feathers becoming coated with oil and birds are unable to move freely and to feed. Death may also occur because of hypothermia and by direct ingestion of oil.

FIGURE 10-33 An unusual case of a saker falcon bitten on the back talon by a sand snake (*Psammophis schokari*), a mildly poisonous snake inhabiting areas of sparse vegetation in the Middle East. The falcon was bitten during a training exercise when it unknowingly landed close to the spot where the snake was hiding. The falcon was taken for veterinary attention 12 hours after the incident, but it was too late and the bird died immediately after admission.

TABLE 10-15 Miscellaneous Compounds Potentially Toxic to Birds

Toxic Compound	Source
Ethylene glycol	Antifreeze compounds
Chocolate	Commercially available
Nicotine	Tobacco products, tobacco smoke
Ammonia, chlorine, sodium hydroxide	Disinfectants, cleaners
Selenium sulfide	Dog shampoo
Arsenic	Contaminated mineral block
Carbon monoxide	Automobile engine, combustion oven and domestic oven/cooker fumes
Mercury	Mirror linings
Sodium chloride	Household salt, rock salt
Silicone	Peat moss
Nitrates	Fertilizers
Polytetrafluoroethylene gas	Overheated Teflon-lined cooking pans, ironing board covers, some heating elements, some lampshades
Petroleum	Crude petroleum or its derivatives

histopathology findings in the parrot were consistent with changes observed in canines after ingesting a lethal dose of theobromine, the methylxanthine found in chocolate (Cole and Murray, 2005; Fig. 10-33).

REFERENCES

Aguilar RF, Yoshicedo JN, Parish CN: Ingluviotomy tube placement for lead-induced crop stasis in the California condor (*Gymnogyps californianus*), *J Avian Med Surg* 26(3):176–181, 2012.

Alonso-Andicoberry C, García-Villada L, Lopez-Rodas V, et al: Catastrophic mortality of flamingos in a Spanish national park caused by cyanobacteria, *Vet Rec* 151:706–707, 2002.

Anonymous: *Action plan for vulture conservation in India*, New Delhi, India, 2006, Ministry of Environment and Forests, pp 1–18.

Bailey TA, Samour JH, Naldo J, et al: Lead toxicosis in captive houbara bustards (*Chlamydotis undulata macqueenii*), *Vet Rec* 137:193–194, 1995.

Barrows M, Hartley M, Pittman JM: Zinc toxicosis in a wattled crane (*Bugeranus carunculatus*). *Proceedings of the European Association of Avian Veterinarians*, Arles, France, pp 491–494, 2005.

Bauck L, LaBonde J: Toxic diseases. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Bennett RA: Neurology. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Bonar CJ, Lewandowski AH, Schaul J: Suspected fenbendazole toxicosis in 2 vulture species (*Gyps africanus, Torgos tracheliotus*) and marabou storks (*Leptoptilos crumeniferus*), *J Avian Med Surg* 17:16–19, 2003.

Brander GC, Pugh DM, Bywater RJ, et al: Drugs used in the modification of cell, tissue, organ and system function. In Daykin PW, editor: *Veterinary applied pharmacology and therapeutics*, ed 5, London, 1991, Baillière Tindall.

Campbell TW: Crown vetch (*Coronilla varia*) poisoning in a budgerigar (*Melopsittacus undulatus*), *J Avian Med Surg* 20(2):97–100, 2006.

Cole G, Murray M: Suspected chocolate toxicity in an African gray parrot (*Psittacus erithacus*). *Proceedings of the Association of Avian Veterinarians*, Monterey, pp 339–340, 2005.

Cooper JE: *Veterinary aspects of captive birds of prey*, ed 2, Saul, Gloucestershire, UK, 1985, Standfast Press.

Dement SH, Chisolm JJ, Barber JC, et al: Lead exposure in an "urban" peregrine falcon and its avian prey, *J Wildl Dis* 22:238–244, 1986.

Dumonceaux G, Harrison G: Toxins. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Forth W, Heuschler D, Rummel W: *Infusionstherapie. Allgemeine und spezielle pharmakologie und toxicologie*, ed 4, Mannheim, Germany, 1983, Bibliographisches Institut.

Frank A, Borg K: Heavy metals in tissue of the mute swan *(Cygnus olor)*, *Acta Vet Scand* 20:447–465, 1979.

Gerlach H: Anatiformes. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Gilbert M, Virani MZ, Watson RT, et al: Breeding and mortality of Oriental white-backed vulture *Gyps bengalensis* in Punjab Province, Pakistan, *Bird Conserv Int* 12:311–326, 2002.

Gilman GA, Goodman LS, Rall TW, et al: Ammonium and acid-forming salts. In Hardman JG, Limbird LE, editors: *Goodman and Gilman's pharmacological basis of therapeutics*, ed 7, New York, 1985, Macmillan.
Green RE, Newton I, Shultz S, et al: Diclofenac poisoning as a cause of vulture population decline across the Indian subcontinent, *J Appl Ecol* 41:793–800, 2004.
Hadley TL, Grizzle J, Rotstein DS, et al: Determination of an oral aflatoxin dose that acutely impairs hepatic function in domestic pigeons (*Columba livia*), *J Avian Med Surg* 24(3):210–221, 2010.
Harrison GJ: Toxicology. In Harrison GJ, Harrison LR, editors: *Clinical avian medicine and surgery*, Philadelphia, PA, 1986, WB Saunders.
Holz P, Phelan J, Slocombe R, et al: Suspected zinc toxicosis as a cause of sudden death in orange-bellied parrots (*Neophema chrysogaster*), *J Avian Med Surg* 14:37–41, 2000.
Howard BR: Health risks of housing small psittacines in galvanized wire mesh cages, *J Am Vet Med Assoc* 200:1667–1674, 1992.
Humphreys DJ: *Veterinary toxicology*, ed 3, London, UK, 1988, Baillière Tindall.
Hunt WG, Parish CN, Orr K, et al: Lead poisoning and the reintroduction of the California condor in Northern Arizona, *J Avian Med Surg* 23(2):145–150, 2009.
King AS, McLelland J: Special sense organs. In King AS, McLelland J, editors: *Birds: their structure and function*, London, UK, 1984, Baillière Tindall.
Klemm WR: Behavioral physiology. In Swenson M, editor: *Duke's physiology of domestic animals*, ed 10, Ithaca, NY, 1984, Cornell University Press.
LaBonde J: Avian toxicology, *Vet Clin North Am Small Anim Pract* 21:1329–1342, 1991.
LaBonde J: Toxic disorders. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, MD, 1996a, Williams & Wilkins.
LaBonde J: Medicine and surgery of Anseriformes. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, MD, 1996b, Williams & Wilkins.
Lang T: Selected toxins in birds. *Proceedings of the Association of Avian Veterinarians*, Reno, Nevada, pp 287–292, 1997.
Lloyd M: Heavy metal ingestion: medical management and gastroscopy foreign body removal, *J Assoc Avian Vet* 6:25, 1992.
Lumeij JT, Westerhof I, Smit T, et al: Diagnosis and treatment of poisoning in raptors from the Netherlands: clinical case reports and review of 2,750 post-mortem cases 1975-1988. In Redig PT, Cooper JE, Remple JD, et al, editors: *Raptor biomedicine*, Minneapolis, MN, 1993, University of Minnesota Press.
MacDonald JW, Randall CJ, Ross HM, et al: Lead poisoning in captive birds of prey, *Vet Rec* 113:65–66, 1983.
Mautino M: Avian lead intoxication. *Proceedings of the Association of Avian Veterinarians*, Phoenix, AZ, pp 245–247, 1990.
Oaks JL, Gilbert M, Virani MZ, et al: Diclofenac residues as the cause of vulture population decline in Pakistan, *Nature* 427:630–633, 2004.
Osweiler GD: Household and commercial products. In Kirk RW, editor: *Current veterinary therapy IX*, Philadelphia, PA, 1986, WB Saunders.
Peeters N, Viaene N, Devriese L: Acute toxicity of high doses of vitamin B6 (Pyridoxine) in pigeons, *Vlams Diergen Tijds* 46(4):268–269, 1977.
Petrak ML: *Diseases of cage and aviary birds*, Philadelphia, PA, 1982, Lea & Febiger.
Pier AC: Mycotoxins and mycotoxicoses. In Biberstein EL, Zee C, editors: *Review of veterinary microbiology*, Oxford, UK, 1990, Blackwell Science.
Porter SL: Pesticide poisoning in birds of prey. In Redig PT, Cooper JE, Remple JD, et al, editors: *Raptor biomedicine*, Minneapolis, MN, 1993, University of Minnesota Press.
Prakash V: Status of vultures in Keoladeo National Park, Bharatpur, Rajasthan, with special reference to population crash in *Gyps* species, *J Bombay Nat Hist Soc* 96:365–378, 1999.
Redig PT, Stowe CM, Barnes DM, et al: Lead toxicosis in raptors, *J Am Vet Med Assoc* 177:941–943, 1980.
Risebrough R: Fatal medicine for vultures, *Nature* 427:596–597, 2004.
Romagnano A, Grindem CB, Degernes L, et al: Treatment of a hyacinth macaw with zinc toxicosis, *J Avian Med Surg* 9:185–189, 1995.
Rosskopf WJ Jr, Woerpel RW: Heavy metal intoxication in caged birds: parts I and II. In *Exotic animal medicine practice, the compendium collection*, Trenton, NJ, 1986, Veterinary Learning Systems.
Rostami A, Madani SA, Vajhi A: Necrotic dermatitis in waterfowl associated with consumption of perennial rye grass (*Lolium perenne*), *J Avian Med Surg* 25(1):44–49, 2011.
Samour J: Acute toxicity after administration of high doses of vitamin B6 (Pyridoxine) in falcons. *Proceedings of the European College of Zoological Medicine Scientific Meeting*, Wiesbaden, Germany, 2013.
Samour JH, Naldo J: Diagnosis and therapeutic management of lead toxicosis in falcons in Saudi Arabia, *J Assoc Avian Vet* 16:16–20, 2002.
Samour JH, Naldo J: The use of Ca Na_2 EDTA in the treatment of lead toxicosis in falcons. *Proceedings of the Association of Avian Veterinarians*, New Orleans, pp 125–129, 2004.
Scheideler SE, Kunze K: A summary of mycotoxin (Aflatoxin B1 or vomitoxin) challenge in young ostrich chicks. *Proceedings of the Association of Avian Veterinarians*, Reno, Nevada, pp 167–168, 1997.
Shakespeare AS: *Compendium on Continuing Education for the Practicing Veterinarian*, 1440, 1995.
Stauber E, Finch N, Talcott PA, et al: Lead poisoning of bald eagles (*Haliaeetus leucocephalus*) and golden (*Aquila chrysaetos*) eagles in the US inland pacific Northwest region—an 18 year retrospective study—1991–2008, *J Avian Med Surg* 24(4):279–287, 2010.
Swenson J, Bradley GA: Suspected cholecalciferol rodenticide toxicosis in avian species at a zoological institution, *J Avian Med Surg* 27(2):136–147, 2013.
Van Sant F: Zinc and clinical disease in parrots. *Proceedings of the Association of Avian Veterinarians*, Reno, Nevada, pp 387–391, 1997.

FURTHER READING

Antopol W, Tarlov IM: Experimental study of the effects produced by large doses of vitamin B6, *J Neuropathol Exp Neurol* 1:330–336, 1942.
Baird GJ, Caldow GL, Peek IS, et al: Monensin toxicity in a flock of ostriches, *Vet Rec* 140:624–626, 1997.
Berger A, Schaumburg HH: More on neuropathy from pyridoxine abuse, *N Engl J Med* 311:986–987, 1984.
Heard GS, Annison EF: Gastrointestinal absorption of vitamin B-6 in the chicken (*Gallus domesticus*), *J Nutr* 116:107–120, 1986.
National Academy of Science: *Nutrient requirements of laboratory animals*, ed 2, Committee on Animal Nutrition. National Research Council, Washington DC, 1972, National Academy Press.
National Academy of Science: *Vitamin tolerance of animals*, Subcommittee on Vitamin Tolerance, Committee on Animal Nutrition, National Research Council, Washington DC, 1987, National Academy Press.
Parry GJ, Bredsen DE: Sensory neuropathy with low-dose pyridoxine, *Neurology* 35:1466–1468, 1985.
Samour J, Naldo JL: Lead toxicosis in falcons: method for lead retrieval, *Semin Avian Exot Pet Med* 14(2):143–148, 2005.
Scheineder RA: *Pyridoxine (Vitamin B6) toxicity: literature review*. Physiology Division Biomedical Laboratory, Aerospace Medical Division, Wright-Peterson Air Force Base, Ohio, 1964.
Unna K, Antopol W: Toxicity of vitamin B6, *Proc Soc Exper Biol Med* 43:116–118, 1940.

VETERINARY CARE OF OILED BIRDS

Kerri Morgan, Michael Ziccardi

There is increasing global awareness of the environmental impact of oil spill events. Because of their dependence on the air–sea interface, pelagic avian species are at high risk of contamination during a maritime oil spill, with significant consequences for the oiled bird's ability to survive in the harsh marine environment. Even in relatively mild environmental conditions, long-term survival of significantly oiled birds can be very low because of required changes to their behavior. Coastal and estuarine birds are at risk as oil comes ashore, impacting

feeding, roosting, and breeding habitats. Terrestrial species and those inhabiting inland waterways are also at risk from inland spills or from marine spills affecting their terrestrial habitat.

EFFECTS OF OIL ON BIRDS

The negative effects of oil on birds have been well documented (e.g., Stowe and Underwood 1984; Underhill *et al.*, 1999; Goldsworthy *et al.*, 2000; Balseiro *et al.*, 2005), although the true impact of oil spills at a population level are poorly understood because of lack of data and the highly mobile nature of birds (Heubeck *et al.*, 2003). Individual birds may be directly contaminated via external oiling, ingestion of oil, respiration of volatile components, or through contamination of embryos through the egg shell (Leighton, 1993). Alternatively, individuals or populations may be indirectly impacted through mechanisms such as contamination of food sources, habitat degradation, and disruption of breeding through response activities (Eppley and Rubega, 1990; Henkel *et al.*, 2014).

External contamination with oil is the most visible and important direct effect of oil on birds, leading to high mortality (Fig. 10-34; Leighton, 1993). Feather barbules become matted with oil resulting in disruption of the critical feather structure and leading to an inability of the bird to repel water, thermoregulate, and fly (Fig. 10-35; Leighton, 1993). Consequently, birds typically develop hypothermia and those unable to get to land often drown at sea.

Contact of oil with skin and mucous membranes may cause skin and eye lesions and burns (Oiled Wildlife Care Network, 2000). Volatile products such as diesel are highly irritant to mucous membranes and the respiratory tract, resulting in inhalant pneumonia and emphysema (Oiled Wildlife Care Network, 2000). Internal effects of oil occur via ingestion while swimming or feeding or through preening activities (Leighton, 1993). Direct exposure of the gastrointestinal tract to oil may lead to disruptions in nutrient absorption and subsequent diarrhea, anorexia, regurgitation, and hemorrhage (Leighton, 1993; Tseng, 1999), often resulting in dehydration and emaciation (Balseiro *et al.*, 2005). Hypoglycemia may occur from a decrease in glucose uptake

FIGURE 10-34 External contamination with oil exerts a smothering effect on seabirds, resulting in loss of waterproofing and thermoregulatory ability with associated high mortality.

FIGURE 10-35 Oiled feathers progressing through various stages of wash, illustrating the clumping of feather barbules **(A)** through to oil free where feathers regain a more normal appearance **(B** through **D)**.

(Leighton, 1993; Alonso-Alvarez *et al.*, 2007). Absorbed polycyclic aromatic hydrocarbons from petroleum products can exert systemic toxic effects depending on congener, level of exposure, and presence of functional metabolic processes (Balseiro *et al.*, 2005). Oxidative damage to red blood cells can lead to the development of hemolytic anemia (Newman *et al.*, 1999; Balseiro *et al.*, 2005), which may result in severe hemosiderosis of viscera such as the liver, spleen, and kidney (Balseiro *et al.*, 2005). Urate depositions on these organs may indicate renal disease either secondary to dehydration or through nephrotoxic effects of compounds within oil (Balseiro *et al.*, 2005). Oil may also be hepatotoxic, although there is some debate in the literature about this (Leighton 1993). Immunosuppression may result from either direct toxicity from the oil or secondary to stress related to rehabilitation (Briggs *et al.*, 1996), often manifesting clinically as aspergillosis (Fig. 10-36; Carrasco *et al.*, 2001, Balseiro *et al.*, 2005; Gartrell *et al.*, 2013). Short- and long-term reproductive impairment has been observed as a sequel to oil spill events (e.g., Ainley *et al.*, 1981; Barros *et al.*, 2014). Nesting birds may contaminate eggs from oil on their plumage, leading to embryo mortality (Leighton, 1993). Other reasons for reproductive impairment include a reduction in egg production, fertility, and hatching success; egg shell thinning; embryo toxicity and teratogenic malformations; abnormal behavior and disruption to pair bonds; and the inability of parents to meet energetic demands of the chicks (Sievwright, 2014).

THE VETERINARIAN'S ROLE IN CARING FOR OILED BIRDS

The process of caring for oiled birds follows six basic steps: recovery of oiled wildlife from the environment and transport to a rehabilitation center or field stabilization site, processing and intake, stabilization, cleaning, prerelease conditioning, and release back to the wild (Massey, 2006). This section concentrates on the role of the veterinarian in caring for oiled birds. As a member of a large team of oiled wildlife responders, the veterinarian plays a crucial role in establishing animal health and welfare protocols specific to the incident and providing advice throughout the operation on animal and human health-related and management-related issues as well as technical expertise within the primary rehabilitation center and any field stabilization sites. Specifically, the review of safety plans to ensure potential zoonotic diseases are addressed is critically important. Protocols regarding quarantine and hygiene procedures also require input from the veterinarian at all stages during the response.

Appropriate personal protective equipment must be worn at all times when handling birds, including eye protection, protective clothing, and nitrile gloves (Fig. 10-37). Latex gloves are oil permeable so they are unsuitable for handling oiled animals.

Recovery and Transportation of Oiled Birds

To minimize any further impact of oiling, the goal of an oiled wildlife response when rehabilitation is an agreed course of action is to capture and transport oiled animals to a care facility as soon as possible. There is usually a single rehabilitation facility (Fig. 10-38), but depending on the geographic location of the spill, there may be one or more stabilization sites in the field. Field stabilization is typically used if the anticipated transport times to the rehabilitation facility exceeds 1 to 2 hours or if the affected species is of particular importance or sensitivity (Oiled Wildlife Care Network, 2000).

FIGURE 10-37 Oiled wildlife responders wearing correct personal protective equipment. (Courtesy University of California, Davis.)

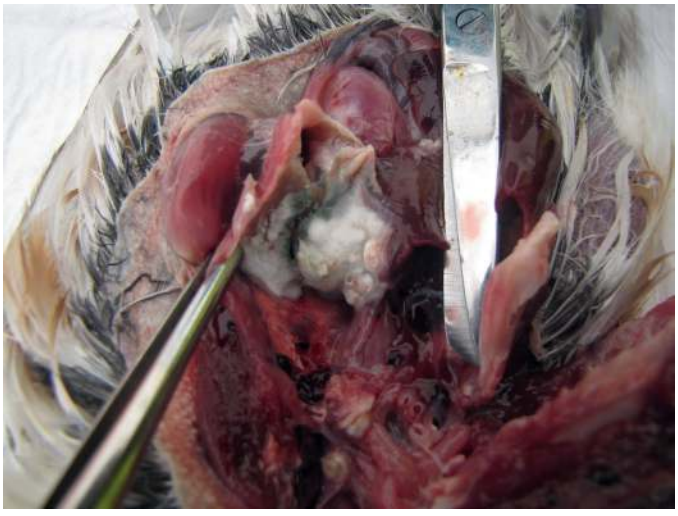


FIGURE 10-36 A granuloma caused by *Aspergillus* spp. in the air sac of a New Zealand dotterel (*Charadrius obscurus aquilonius*) that died during rehabilitation.

FIGURE 10-38 Oiled wildlife rehabilitation facilities used during a response may be permanent rehabilitation centers or temporary facilities such as this one, which has been constructed using large tents and containerized wash units.

FIGURE 10-39 Cardboard or corflute portapets are routinely used for transporting oiled birds. Note the use of wood to separate boxes to ensure adequate circulation of air during transport. (Oiled Wildlife Care Network, 2000.)

On capture, birds may be transported in pillowcases for short periods of time before transfer to a solid-sided but well-ventilated transport box, such as airline kennels, cardboard or corflute portapets, or cardboard boxes (Fig. 10-39).

Field stabilization techniques include assessment and triage of birds; the administration of oral water or electrolyte solutions at 30 mL/kg every 1.5 to 2 hours; an attempt to restore normal core body temperature; and removal of large amounts of oil from nares, eyes, and glottis (Tseng, 1999; Oiled Wildlife Care Network, 2000).

Boxes should be placed in a sheltered area until transport to the rehabilitation facility, preferably in an enclosed, ventilated van (Oiled Wildlife Care Network, 2000). During transport, boxes should be separated to ensure adequate circulation of air, and particular attention to ventilation must be taken when the oil product is fresh and volatile (e.g., diesel; Massey, 2006). As oiled birds have lost their ability to thermoregulate, the provision of an ambient temperature during transportation of approximately 21°C (70°F) is critical (Massey 2006), although wet birds may require temperatures closer to 27°C (80°F; Oiled Wildlife Care Network, 2000). Special care must be taken in very hot climates to ensure birds do not overheat. Animals should be regularly monitored during periods of transport of more than 1 hour, under the direction of the veterinarian, and may require repeat fluid therapy during transport (Oiled Wildlife Care Network, 2000).

Intake

Upon arrival at the rehabilitation center, birds undergo processing and intake. The intake team should include an experienced veterinarian, and a triage process should be established to ensure those individuals in need get priority care.

During this stage, birds are given individual identification, usually through placement of temporary numbered plastic leg bands. Flipper bands are usually used for penguins (Fig. 10-40). Implantation of a subcutaneous microchip should be considered as a backup in case of band loss during rehabilitation, and it can also be used for postrelease monitoring if appropriate (Sievwright, 2014). A medical record is initiated, and in some countries, photographs and oiled feathers are collected as evidence (Massey, 2006). Cloacal temperature should be evaluated, with normal temperatures ranging from 39°C to 41°C (102°F to 105°F; Oiled Wildlife Care Network, 2000). If the bird is hypothermic or hyperthermic, the veterinarian should consider immediate action to stabilize core body temperatures. Hypothermic birds

FIGURE 10-40 A little blue penguin (*Eudyptes minor*) with a temporary flipper band used for identification during rehabilitation.

FIGURE 10-41 Modified pet driers are used to blow warm air into net-bottomed pens to provide thermal support to oiled or freshly washed birds. (Oiled Wildlife Care Network, 2000.)

can be warmed through using warm heat packs, forced warm air, or heat lamps (Fig. 10-41). In severe instances, incubators can be used, but they should be used sparingly because of lack of available ventilation. Hyperthermic birds may be treated by misting with cool water, application of alcohol pads to the foot area, or immersion of the bird in cool water (Tseng, 1999).

A thorough veterinary examination should be undertaken with special attention paid to potential ocular injuries as a result of the oiling; signs of contact dermatitis, especially within the axillary and inguinal regions; and other overt signs of acute oil exposure such as respiratory and neurologic effects (Massey, 2006). Fractures and

FIGURE 10-42 Blood samples are taken to assess packed cell volume and total plasma protein upon intake to the rehabilitation center.

FIGURE 10-43 Thermal support can be provided using a ducted gas system, allowing both heating and ventilation. Heat lamps can provide additional point source heating; however, care must be taken to avoid thermal burns.

wounds should also be evaluated, and triage should be considered at this point depending on caseload and available capacity of personnel and facilities. The bird's weight and an estimation of body condition should be recorded, as well as quantity and location of external oiling (Oiled Wildlife Care Network, 2000). Blood samples should be taken for immediate determination of packed cell volume (PCV) and total plasma protein (TPP; Fig. 10-42). Veterinary intervention is required if the bird has a PCV of less than 15% or a TPP of less than 10g/L (Oiled Wildlife Care Network, 2000). If the bird appears significantly debilitated and/or anorexic, blood glucose levels can also be evaluated, although alterations in glucose are typically only observed in oiled birds immediately before death. Any oil within the nares or oropharynx should be wiped using a swab, and eyes should be flushed with saline to remove any residual contamination by oil or sand.

In consultation with the veterinarian, triage protocols should be established for each incident as these are often circumstance dependent and are particularly important when resources are limited. In larger responses, a "herd-health" approach is adopted, with less focus on the treatment of individual birds. Triage should take into account the medical status of the individual and the conservation status of the affected species (McConnell *et al.*, 2009). In general, priority is given to those with the best chance of survival from a medical point of view (body temperature of 39° C to 40° C and no concurrent injuries), and those species with highest conservation value. Birds with the least chance of survival are those with severe injuries, a PCV of less than 20%, and a consistently low body temperature of 37° C to 37.5° C (Walraven, 2004). Euthanasia is most commonly performed at this stage of the rehabilitation process and should be performed by a veterinarian through an intravenous overdose of barbiturates. Alternatively, an overdose of inhalational agents may be used. Intraperitoneal, intracranial, or other routes of administration of barbiturates are not suitable by themselves for euthanasia because of the pain associated with these methods (Finlayson and Morgan, 2014), although they can be combined with a deep anesthetic in certain circumstances.

At the conclusion of the intake process, oral fluids are administered at 50 mL/kg by mouth (if neurologic evaluation reveals that risk of regurgitation is slight), and the bird is moved into the stabilization area.

Stabilization

The aim of stabilization (also called prewash rehabilitation) is to provide the bird with the best chance of surviving the stressful wash process. Stabilization includes reestablishment of a normal body temperature, correction of dehydration, and the provision of nutritional support. The length of this phase is dependent on the condition of the individual bird and can take anywhere from a minimum of 2 days to more than a week (Massey, 2006). Those birds captured later in the response generally take longer to stabilize because they are more significantly debilitated before capture (Massey, 2006).

The correction of hypothermia is ideally achieved through the use of a holding facility with appropriate ambient temperature supplemented with an external heat source when necessary. To reach a constant ambient temperature of approximately 27° C, an adequately designed room with forced-air heating is ideal (Fig. 10-43), but warmed, forced air injected either from above or below individual pens can be used and is preferable to point sources of heat such as heat lamps because of the risk of thermal burns.

Oral water or electrolyte solutions should be initiated at intake and continued during the stabilization phase until fluid deficiencies are corrected. Again, a herd-health approach is taken, with the assumption that the average oiled bird is 8% to 10% dehydrated (Tseng, 1999). Once rehydrated, nutritional support is provided through gavage of high-calorie slurries up to four times daily, alternating with electrolytes (Tseng, 1999; Oiled Wildlife Care Network, 2000), or diluted slurries up to eight times daily. Depending on the species, recommended volumes for gavage are up to 50 mL/kg per feeding, with initial volumes around half of this until the bird can tolerate the procedure (Tseng, 1999). Depending on the bird species, shallow pans with water and nonoily small fish may be offered *ad lib* (Oiled Wildlife Care Network, 2000).

Birds are regularly assessed to determine adequate restoration of physiologic parameters for eligibility to move to the cleaning process. Blood values determined at intake are reassessed every second day. The Oiled Wildlife Care Network, University of California Davis, has set the following minimum criteria for wash: the bird has been hospitalized for more than 48 hours, it is alert and responsive, PCV is greater than or equal to 30%, and TPP is greater than or equal 25 g/L (Massey, 2006). In the case of contamination with highly toxic products such as diesel or jet fuel, contaminants may be removed through a "quick wash" procedure shortly after admission (Oiled Wildlife Care Network, 2000).

FIGURE 10-44 An oiled pied cormorant (*Phalacrocorax varius*) undergoing a wash procedure using warm, softened freshwater and 1% to 2% detergent.

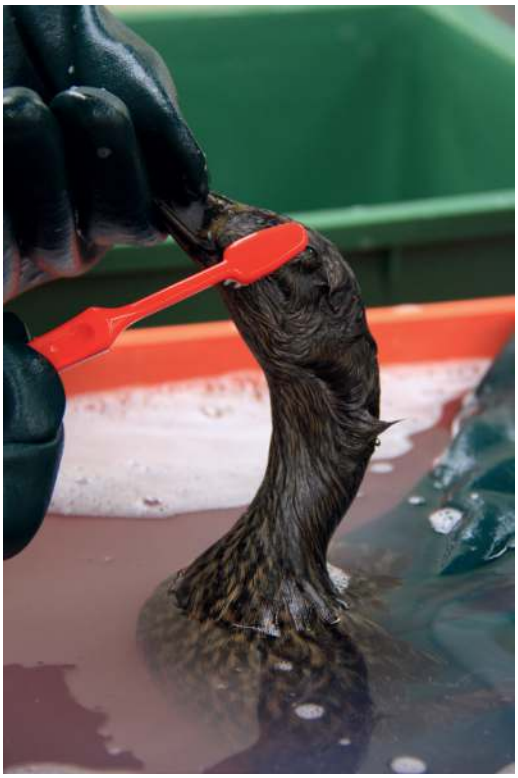


FIGURE 10-45 Soft toothbrushes are used to clean around the delicate head area.

Wash

Wash should only be done after nutritional slurries are provided that morning to minimize risk of hypoglycemia during the procedure. Thorough cleaning of oiled birds requires washing in sequential tubs of warmed (40°C to 41°C), softened freshwater (two to three grains of hardness) with the addition of diluted dish detergent (1% to 2%; (Fig. 10-44). Soft toothbrushes or waterpicks are used around the delicate head area (Fig. 10-45). Weathered oil may need to undergo preconditioning with warmed canola oil or methyl oleate (35°C to 38°C) before cleaning. Thorough rinsing at a pressure of 40 to 60 pounds per square inch with warmed softened freshwater is essential to remove any trace of residual soap (Fig. 10-46; Massey, 2006). This entire process can take more than 60 minutes, depending on the size of the bird, how heavily oiled the bird is, and the expertise of the wash team (Oiled Wildlife Care Network, 2000). Wash may need to be suspended mid procedure if birds are "crashing," and emergency procedures may be necessary at that point to address hypothermia. However, if at all possible, completion of wash procedures should be accomplished.

FIGURE 10-46 Detergent is thoroughly rinsed from the clean bird using warmed, softened freshwater at a pressure of 40 to 60 pounds per square inch.

Prerelease Conditioning

The goal of prerelease conditioning is to allow the restoration of waterproofing, as well as the return of normal behavioral function and body condition. Feather realignment is achieved through encouragement of preening activities by providing a pool for increasing periods of time for pelagic birds (Fig. 10-47), or appropriate aviaries with access to water for nonobligate waterbirds. As birds are not waterproof immediately after cleaning, close supervision is required until they start to reestablish their waterproofing (Fig. 10-48). At this stage, birds are usually fed whole fish or whole food items as close to their normal diet as possible. Some species may require force feeding, and others may be free fed in shallow pans (Tseng, 1999). The addition of multivitamin tablets is essential. As birds near release, salt gland activity should be encouraged in marine species through the provision of either dietary salt, the addition of salt in pools, or the careful administration of sodium chloride via an electrolyte solution at 250 mg/kg at least once every other day.

Release

Birds are ready for release once these criteria are met: the individual bird is healthy and waterproof, the environment is clean, and there is minimal risk of re-oiling. Well-established criteria are used to assess

FIGURE 10-47 A cleaned little blue penguin (*Eudyptes minor*) is swum to encourage preening to realign feather barbules disrupted during the wash process.

FIGURE 10-48 Cleaned little blue penguins utilizing a haul-out area in the pool. These birds are not yet waterproofed, with separation of feathers and penetration of water to the skin.

FIGURE 10-49 Soft-sided net-bottomed pens allow minimal contact with feces and urates and provide an even weight distribution on the bird's ventral surface.

FIGURE 10-50 Keel "donuts" are attached to highly susceptible individuals to prevent pressure sores. (Oiled Wildlife Care Network, 2000.)

the readiness of individual birds (Oiled Wildlife Care Network, 2000; Walraven, 2004). These include the exhibition of normal behavior (feeding, swimming, and diving), body weight is within 10% of normal for that species, the bird is 100% waterproof, PCV and TPP are within normal limits, and all medical problems are resolved. Before release, temporary identification bands should be removed and, if appropriate, replaced with permanent bands to enable postrelease monitoring.

PREVENTION OF DISEASE SECONDARY TO CAPTIVITY

Throughout the stabilization, cleaning, and prerelease conditioning phases, veterinary input is required to prevent and treat husbandry-related disease. Throughout the rehabilitation process, itraconazole should be administered at a dose proven to be preventive in the species of concern (20 mg/kg once daily in alcids, 10 mg/kg for penguins) for aspergillosis prophylaxis, combined with 10 to 15 air changes per hour (Tseng, 1999). Pododermatitis is reduced through the use of net-bottomed cages during the stabilization and postwash rehabilitation phases (when housed indoors) to minimize contact with feces and urates (Fig. 10-49). These also act to ensure even weight distribution on the bird's ventral surface. Special consideration should be taken to the keel, which is prone to pressure sores. These may be further prevented through the use of keel "donuts" on thin or emaciated birds that are not standing (Fig. 10-50).

REFERENCES

Ainley DG, Grau CR, Roudybush TE, et al: Petroleum ingestion reduces reproduction in Cassin's auklets, *Mar Pollut Bull* 12(9):314–317, 1981.

Alonso-Alvarez C, Pérez C, Velando A: Effects of acute exposure to heavy fuel oil from the Prestige spill on a seabird, *Aquat Toxicol* 84:103–110, 2007.

Balseiro A, Espí A, Márquez I, et al: Pathological features in marine birds affected by the Prestige's oil spill in the north of Spain, *J Wildl Dis* 41:371–378, 2005.

Barros A, Alvarez D, Velando A: Long term reproductive impairment in a seabird after the Prestige oil spill, *Biol Lett* 10:20131041, 2014.

Briggs KT, Yoshida SH, Gershwin ME: The influence of petrochemicals and stress on the immune system of seabirds, *Reg Toxicol Pharmacol* 23:145–155, 1996.

Carrasco L, Lima JS Jr, Halfen DC, et al: Systemic aspergillosis in an oiled magallanic penguin (*Spheniscus magellanicus*), *J Vet Med Ser B* 48(7):551–554, 2001.

Eppley Z, Rubega M: Indirect effects of an oil spill: reproductive failure in a population of South Polar skuas following the "Bahia Paraiso" oil spill in Antarctica, *Mar Ecol Prog Ser* 67:1–6, 1990.

Finlayson S, Morgan K: Humane euthanasia methods for pet birds and wildlife, *VetScript* 27(8):22–24, 2014.

Gartrell BD, Collen R, Dowding JE, et al: Captive husbandry and veterinary care of northern New Zealand dotterels (*Charadrius obscurus aquilonius*) during the CV Rena oil-spill response, *Wildl Res* 40:624–632, 2013.

Goldsworthy SD, Gales RP, Giese M, et al: Effects of the Iron Baron oil spill on little penguins (*Eudyptula minor*). I. Estimates of mortality, *Wildl Res* 27:559–571, 2000.

Henkel JR, Sigel BJ, Taylor CM: Oiling rates and condition indices of shorebirds on the northern Gulf of Mexico following the Deepwater Horizon oil spill, *J Field Ornithol* 85(4):408–420, 2014.

Heubeck M, Camphuysen KCJ, Bao R, et al: Assessing the impact of major oil spills on seabird populations, *Mar Pollut Bull* 46:900–902, 2003.

Leighton F: The toxicity of petroleum oils to birds, *Environ Rev* 2:92–103, 1993.

Massey J: Summary of an oiled bird response, *J Exot Pet Med* 15(1): 33–39, 2006.

McConnell HM, Morgan KJ, Gartrell BD, et al: Prioritising the treatment of oiled wildlife in New Zealand by conservation status. Effects of Oil on Wildlife, Tallinn, Estonia, 2009.

Newman SH, Mazet JK, Ziccardi MH, et al: Haematological changes and anaemia associated with captivity and petroleum exposure in seabirds, *Comp Haematol Int* 9:60–67, 1999.

Oiled Wildlife Care Network: *Protocols for the care of oil-affected birds, oiled wildlife care network*, 2000, University of California, Davis.

Sievwright K: *Post-release survival and productivity of oiled little blue penguins (Eudyptula minor) rehabilitated after the 2011 C/V Rena oil spill*, 2014, Massey University, Masters of Science, p 153.

Stowe T, Underwood L: Oil spillages affecting seabirds in the United Kingdom, 1966–1983, *Mar Pollut Bull* 15(4):147–152, 1984.

Tseng F: Considerations in care for birds affected by oil spills, *Semin Avian Exot Pet Med* 8(1):21–31, 1999.

Underhill LG, Bartlett PA, Baumann L, et al: Mortality and survival of African penguins (*Spheniscus demersus*) involved in the Apollo Sea oil spill: an evaluation of rehabilitation efforts, *Ibis* 141:29–37, 1999.

Walraven E: *Rescue and rehabilitation of oiled birds*, Sydney, Australia, 2004, Zoological Parks Board of New South Wales.

11

Soft Tissue Surgery

Neil A. Forbes

EQUIPMENT

For avian soft tissue surgery to be performed without undue risk of failure, the following equipment should always be available:

- Surgical instruments with miniaturized tips and standard preferably counterbalanced handles should be used. Atraumatic (e.g., Harris ring tip) forceps and hemoclips are essential for many soft tissue procedures. Illuminated magnification (which not only allows you to see more detail, but also reduces hand tremor during surgery) is essential for accurate safe surgery. Sterile cotton buds for soft tissue handling and hemostasis are invaluable.
- Adhesive, waterproof transparent drapes are useful to conserve body temperature and permit patient observation. An ergonomic, seated surgical position with forearm support should be used.
- Correct use of radiosurgery, with a frequency of 3.8 to 4.0 MHz, and monopolar and bipolar forceps, with minimal sized electrodes and contact time, such that lateral tissue damage and subsequent delayed tissue healing are minimized.
- Laser (light amplification by the stimulated emission of radiation) surgery is now more readily available and affordable. Tissues may be cut or ablated (vaporized) using contact (least collateral damage—typically 300 to 600 μm) or noncontact (when visualization is improved, although lateral damage tends to be slightly greater) modes. Using either technique, blood vessels of up to 2 mm diameter may be incised in the absence of any hemorrhage. Laser surgery can be used endoscopically.

PREPARATION

The patient must be assessed regarding energy and nutritional status, as well as circulatory fluid or blood deficit, and any abnormalities corrected. Intraoperative and postoperative hypothermia, analgesia, sepsis, and shock must be controlled. Presurgical starvation should be sufficient purely to ensure an empty crop.

Sufficient feathers are removed to enable adequate sterile access to the operative site; adjacent feathers are retracted with adhesive tape. In the author's practice, skin preparation is performed using iodine-based alcoholic tincture disinfectant. An aerosol surgical adhesive is applied to the skin and a sterile transparent drape is applied.

SURGERY OF THE SKIN AND ADNEXA

Feather Cysts (Folliculoma)

Feathers cysts are in growing feathers, which result in inflammatory swellings filled with feather material. They are most common at the sites of insertion of flight feathers, arising subsequent to infection or trauma. Feather cysts are common in canaries and are considered to be hereditary. The entire cyst including the dermal papilla (in flight feathers this is situated in the periosteum on the ventral aspect of the wing) must be surgically removed.

Uropygial (or Preen) Gland

The uropygial or preen glands are situated on the caudodorsal body wall, just proximal to the insertion of the tail flight feathers. The uropygial gland is the most prominent epidermal gland in birds and produces a waxy oily secretion via two or more ducts. This oil is spread through the plumage during preening. The uropygial gland may suffer from ductal blockage, gland abscess, or neoplasia (Fig. 11-1). Blockage is often resolved by application of gentle digital pressure, resulting in a jet of thick waxy and oily secretion.

Infection and neoplasia can be difficult to differentiate as both result in significant pain, swelling, and an inflammatory response. Adenoma, adenocarcinoma, and squamous cell carcinoma occur (Forbes *et al.*, 2000). A biopsy should always be taken for a differential diagnosis. Abscesses are treated by curettage and culture and sensitivity testing with appropriate topical and systemic antibiosis. Self-trauma over and around the preen gland is a common feature in psittacines. Application of a collar during healing, environmental enrichment, and surgical removal of the preen gland can all prove efficacious.

Preen gland neoplasia requires careful surgical excision. The gland itself benefits from a significant blood supply and radiosurgery is invaluable. The gland is bordered ventrally by fibrinous connective tissue that attaches firmly to the dorsal surface of the pygostyle and caudal vertebrae. Surgical removal must extend to the connective tissue layer, which is relatively avascular compared with the gland. The two sides of the gland are separated by a central septum. In early cases, it is possible to remove just the affected side, preserving preen gland function. During surgery, the skin overlying the gland should be preserved to enable postoperative closure.

TREATING SOFT TISSUE WOUNDS AND INJURIES

Birds typically have thin skin with minimal soft tissue structures (especially the extremities). In birds desiccation and devitalization of subcuticular tissues following loss of skin integrity is common. Closing the skin or covering it with hydrocolloidal dressings to prevent desiccation is necessary. Tissue damage/necrosis/organic contamination or significant bacterial or fungal infection precludes first-intention healing (Scott Echols, 2008). Extensive debridement (e.g., wet-to-dry dressings) and irrigation facilitates primary intention closure. The most common site for skin deficit is the cranium. These are closed with single pedicle or bipedicle cervical grafts moving loose skin from the

FIGURE 11-1 Uropygial or preen gland adenocarcinoma in a cockatiel (*Nymphicus hollandicus*).

FIGURE 11-2 The propatagium of a falcon. Please note the propatagial ligament (ligamentum propatagiale longus) enclosed within the leading edge.

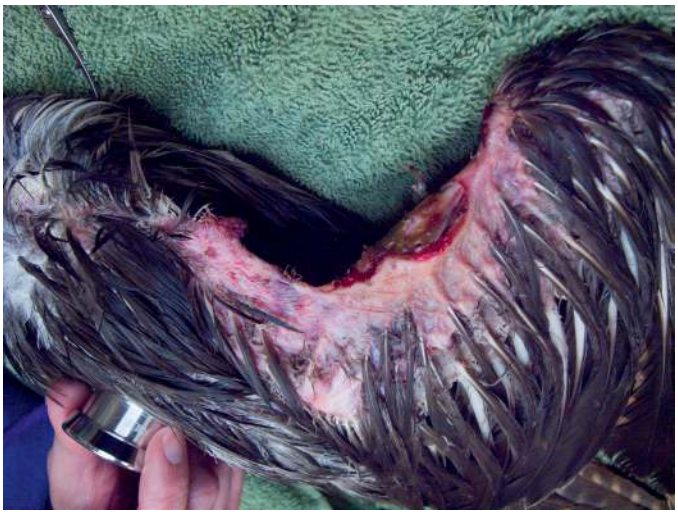


FIGURE 11-3 Propatagium following significant trauma. Tissues retracted against the radius in a saker falcon (*Falco cherrug*).

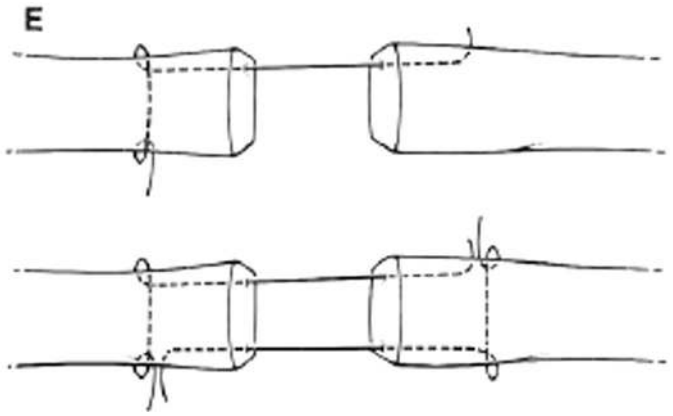


FIGURE 11-4 Kessler suture pattern. This is a tension suture used for apposition of severe tendons ends.

neck up over the deficit. Avian skin is typically thin and free skin grafts tend to be unsuccessful. Psittacine wounds are generally best protected or covered, although parrots are generally reluctant to tolerate bandaging. Hydrocolloidal dressings (e.g., Granuflex, ConvaTec, UK) may be sewn over a wound to stimulate healing and provide wound protection.

Propatagial Repair

The propatagium (airfoil on the anterior edge of the wing attaching at the shoulder proximally, carpus distally, and elbow caudally) is essential for birds to fly. It is a delicate structure with limited vascular supply. Direct blunt or electrical trauma or avascular necrosis consequent to bandaging can lead to significant loss of tissue and the ability to fly. The propatagium comprises two layers of skin (reinforced by an elastic web), one dorsally and one ventrally, with a propatagial ligament (ligamentum propatagiale longus) enclosed within the leading edge. The ligament has collagen sections at either end with an elasticated section in the middle (pars elastica; Fig. 11-2).

The propatagium receives its blood supply from the radial artery and branches of the subscapular artery, which runs just caudal to the propatagial ligament. In the event of injury, the propatagial ligament retracts (because of the elasticated nature of its central section), which is beneficial as the ends retract within moist tissue and both desiccation and devitalization are prevented. With the ligament no longer intact, the propatagium will tend to retract back against the humerus and radius (Fig. 11-3).

The free ends of the propatagial ligament must be located and exteriorized. Any defect or deficit of the propatagial ligament should be removed and the fresh ends joined using a Kessler or similar suture technique (Harcourt-Brown, 2000) (Figs. 11-4 and 11-5). Any deficit in dermal tissue must then be closed around the ligament. Following this the dorsal and ventral propatagial surfaces must also be reconstructed, using tension-sparing methods (e.g., horizontal or vertical mattress sutures; Fig. 11-6).

A moderate shortening of the propatagial ligament can be tolerated by most birds because the structure stretches postoperatively to accommodate for any shortening. Post surgery, a single piece of hydrocolloid dressing, cardboard, or radiology film is shaped to cover the dorsal, over the leading edge, and ventral propatagium (Fig. 11-7). This material is sewn dorsal to ventral and remains in place for at least 6 weeks.

NEOPLASMS

Birds suffer from a range of cutaneous, subcutaneous, and internal neoplasms. These should be approached similarly to those in other species. Masses may be aspirated for cytologic examination, biopsies

FIGURE 11-5 Post ligament repair.

FIGURE 11-6 After propatagial repair.

FIGURE 11-7 Radiography film over propatagial repair to prevent stretching.

FIGURE 11-8 Xanthoma on the wing tip in a cockatiel (*Nymphicus hollandicus*).

collected, or masses removed and submitted for histopathology (Forbes *et al.*, 2000).

Lipomas

Lipomas are benign tumors of fat tissue—a common finding in many psittacine species, particularly budgerigars (*Melopsittacus undulatus*)—and are most common over the cranial sternum. Nutritional manipulation should be considered before surgery. Birds on millet or seed-based diets should be converted to a reduced-fat diet. The addition of L-carnitine to the diet may obviate surgery (De Voe *et al.*, 2003). Lipomas often have a significant singular blood supply and removal is not difficult, but care must be taken to maintain hemostasis.

Xanthomas

Xanthomas are non-neoplastic masses, typically found on extremities, especially following trauma or hemorrhage. They are intradermal deposits of cholesterol clefts with an associated foreign body reaction. However, this author has removed confirmed xanthoma from an infraorbital sinus and two from the tracheal lumen (Monks *et al.*, 2006). They may appear yellowish, as cutaneous or subcutaneous plaques, as diffuse thickening, or as lobulated masses and occasionally ulcerating. Xanthomas tend to be highly vascularized and invasive (Fig. 11-8). Dietary fat reduction (convert from seed- or nut-based diets) may be helpful, but total surgical removal when initially diagnosed (i.e., when as small as possible) is preferable.

Squamous Metaplasia

Squamous metaplasia of the submandibular salivary glands is a common sequel to vitamin A deficiency, typically seen in aged parrots on chronically deficient (seed-based) diets (Fig. 11-9). The patient should be treated medically with vitamin A, nonsteroidal antiinflammatory drugs, and antibiosis before surgical removal, because intraoperative hemorrhage can be a complication. Diet should be altered to include more highly colored fresh fruits and vegetables, particularly sweet corn and apricots.

HYPERINFLATION OF THE CERVICOCEPHALIC AIR SAC

This condition is seen as generalized or localized subcutaneous emphysema (Fig. 11-10). Etiology is associated with trauma or chronic respiratory disease (e.g., chlamydiosis or chronic poor air quality, such as tobacco smoke). The author's preferred technique is to sear an aperture

FIGURE 11-9 Squamous metaplasia of submandibular salivary gland subsequent to hypovitaminosis A in a blue-fronted Amazon parrot (*Amazona aestiva*).

FIGURE 11-10 Subcutaneous inflation over cervical air sac in an Amazon parrot (*Amazona* sp.).

through the overlying skin to release the air. The burning delays skin healing to enable the perforation in the air sac wall to heal before skin healing.

GASTROINTESTINAL TRACT TECHNIQUES

Tongue

Because of the way psittacines use their tongues and chew at solid, hard, abrasive, and fragmentary objects, penetrations, lacerations, and foreign bodies in the psittacine tongue do occur. Any recurrent or nonhealing lesion of the tongue should be fully investigated with this in mind. Differential diagnoses include *Cryptococcus neoformans* and mycobacterial infections. Other differentials for tongue pathology include candidiasis, trichomoniasis, or bacterial granuloma. Noninfectious differentials include hypovitaminosis A (cysts or abscesses), lymphoreticular neoplasia, cystadenoma, and squamous cell carcinoma.

Proximal Esophagus

Esophageal stricture formation may occur after infections (trichomoniasis, capillariasis, and candidiasis), iatrogenic trauma during tube

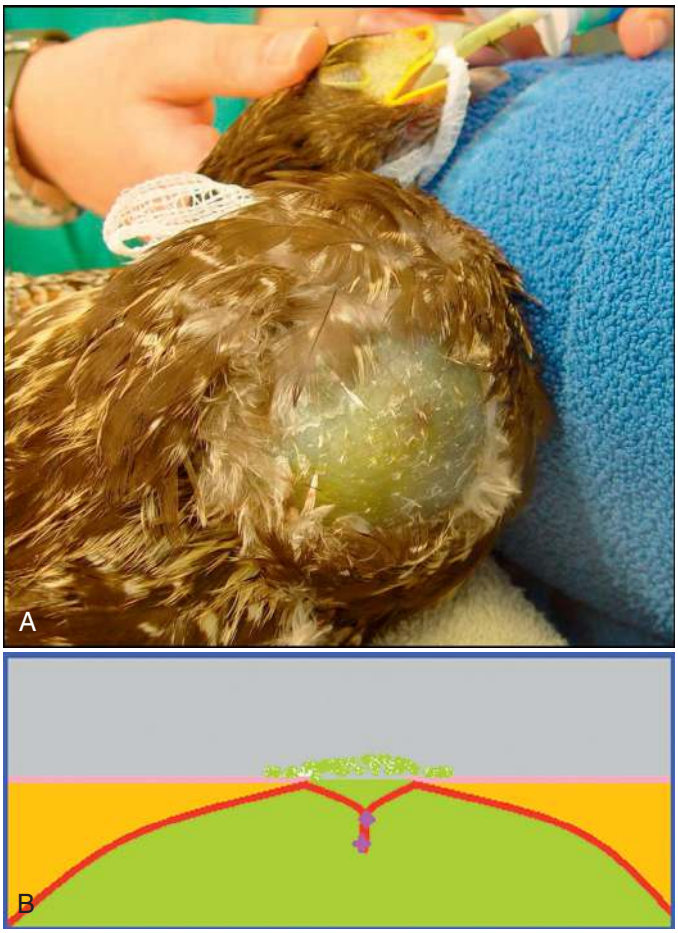


FIGURE 11-11 (A), Harris hawk (*Parabuteo unicinctus*) with severe sour crop. **(B),** Repair technique for the crop.

feeding, thermal or caustic trauma, foreign body ingestion, or iatrogenic surgical trauma. Where strictures occur, the eliciting cause must be determined and addressed. If necessary, a pharyngostomy tube (see later section) may be placed during supportive and medical care. If a stricture remains it may be relieved by serial mechanical dilation, which is achieved by passing tubes or cannulae of increasing size down the esophagus periodically over a period of several weeks.

Ingluviotomy

Commonly indicated for treatment of sour crop (Fig. 11-11, *A*), an ingluviotomy is done to retrieve crop calculi, ingluvioliths, or foreign bodies (which are not accessible per os) or to retrieve proventricular or ventricular foreign bodies (using micromagnets [glued in place within plastic tubes], lavage, or endoscopy) and for the placement of an ingluviotomy or proventriculotomy tube or the collection of crop wall biopsies. A whole range of materials may be found, including rolled up hay, newspaper, or other nest material; whole peanuts, grapes, or olives; and sand. These may be hard and inert or may be susceptible to putrefaction leading to toxemia.

To perform the ingluviotomy, the bird is placed in dorsal or lateral recumbency and intubated, with the head elevated above the level of the crop. A probe is placed per os into the crop to delineate the position of the organ. The skin is incised over the proximal crop wall (at a position above normal crop filling level) and the crop wall is localized and separated from the overlying skin. An incision site is selected to avoid large blood vessels and not to interfere with postoperative feeding or

tube placement. Stay sutures are placed in the crop and an incision one third to one half the length of the skin incision is made (as it will stretch to equal that of the skin). Crop closure is achieved with 4-0 to 6-0 synthetic monofilament absorbable material using a single or double continuous inversion pattern (Fig. 11-11, *B*), followed by separate skin closure.

Crop Biopsy

Crop biopsy remains a safe and minimally invasive antemortem diagnostic technique for psittacine proventricular dilation syndrome (PPDS). However, the method is only 68% sensitive, but 100% specific (Gregory *et al.*, 1996). The collection site should be in the left lateral (nondependent) area of the crop. The sensitivity is further maximized (up to 76%) by harvesting a section of crop wall (full-thickness biopsy—0.5 to 1.0 × 0.5 to 1.0 cm) to include a visible blood vessel. Differential diagnoses for PPDS include heavy metal (Pb, Zn, Cu, and Fe) poisoning; enteric papillomatosis; foreign body ingestion; severe parasitic enteritis; mycobacteriosis; bacterial, *Macrorhabdus ornithogaster*, or fungal ventriculitis; ventricular foreign bodies; and proventricular or ventricular neoplasia or papilloma (Antinoff, 2001; Van Sant, 2001).

Treatment of Crop Burns

Hand-reared psittacines, fed excessively hot or inadequately mixed food (typically microwaved food with "hot spots") may suffer crop burns. Birds may present with delayed crop emptying or a wet skin patch over the crop (often 4 to 7 days after the incident). Necrosis may also occur in adult birds following the consumption of caustic substances. Necrosis of the crop wall and skin leads to fistula formation. Surgical repair should be delayed (4 to 5 days), until necrotic material can be clearly differentiated from viable tissue. It is essential that nutritional support is provided and that secondary infections (bacterial or fungal) are prevented. Ingluviotomy feeding may be necessary (see later section). By the time a fistula has formed, the crop wall will be adhered to the skin. Following anesthetic induction and intubation, the skin is surgically separated from the crop wall. The crop wall is then closed, using a double inversion pattern 4-0 to 6-0 synthetic monofilament absorbable material, followed by a separate skin closure.

Crop or Esophageal Lacerations

Crop or esophageal lacerations may be iatrogenic following traumatic gavage feeding or subsequent to external trauma (e.g., talon punctures from a raptor). Crop perforations are often not recognized at the time of trauma but are obvious when a significant subcutaneous buildup of fetid material has developed. A significant active inflammatory reaction will be present. Surgical exploration, closure of the crop wound, drainage (ingluviotomy tube placement if required), fluid therapy, analgesia, and antiinflammatory and antibiotic therapy may be required before surgical skin closure some days later.

Pharyngostomy, Esophagostomy, or Ingluviostomy Tube Placement

Tube placement is required in situations where food must bypass the mouth, proximal or distal esophagus, or crop (Huynh *et al.*, 2014). Such conditions include orthopedic conditions of the beak and head or trauma, infection, neoplasia, severe parasitic infestations, or strictures affecting any part of the gastrointestinal tract between the mouth and the proventriculus or simply in a bird that is so weak it is unable to feed itself or where the use of an ingluviotomy tube is less stressful to the patient than repeated catching and manual restraint. When performing this procedure, the bird is anesthetized, intubated, and placed in lateral recumbency.

FIGURE 11-12 Feeding a falcon via ingluviotomy tube.

A metal feeding tube is placed by mouth and tented in an appropriate position in the cervical esophagus (proximal to the crop). The skin is prepared and a small incision is made over the end of the feeding tube. An appropriately sized rubber or plastic feeding tube (which can be connected to a feeding syringe) is passed via the incision into the esophagus and advanced caudally. The tube is passed via the crop and distal (thoracic) esophagus into the proximal proventriculus. A crop wall and skin suture is placed around the tube. Tape is placed on either side of the feeding tube where it exteriorizes from the skin incision; the tape is then sutured to the skin. The capped distal feeding tube is then enclosed in a bandage wrap around the neck or attached to the bird's back. Regular small meals (smaller than if feeding into the crop) are administered and care is taken to flush the tube clean after each use. Ingluviotomy tubes may remain in place for several weeks as required. A trained bird need not be restrained for medication, fluid, and food administration, which is especially advantageous in an imprint bird (Fig. 11-12).

Catheter Duodenostomy

This author can see no justification for choosing a catheter duodenostomy over pharyngostomy, esophagostomy, or ingluviostomy tube placement.

COELIOTOMY

The caudal thoracic and abdominal air sacs receive fresh air from the trachea. It is important to appreciate that a coeliotomy is impossible without opening the posterior air sacs, which has a profound effect on both the effectiveness of inhalant anesthesia and on intraoperative heat loss. Once a coeliotomy incision is made, openings around the surgery site may be packed off or plugged with abdominal organs. Alternatively, parenteral anesthetic agents may be used. During any coeliotomy procedure, the bird's head should be raised at 30 to 40 degrees to prevent any surgical irrigation fluid from entering the lung field.

Left Lateral Coeliotomy

A left lateral coeliotomy is the most useful approach and is used for access to the gonads, left kidney, oviduct, ureter, proventriculus, and ventriculus. The bird is placed in right lateral recumbency, the wings are reflected dorsally, and the left leg is restrained in a dorsocaudal direction. The skin web between the abdominal wall and the left leg is

incised to facilitate further dorsal abduction of the left leg. A skin incision is created from the sixth rib to the level of the pubic bone on the left abdominal wall (Fig. 11-13). The superficial medial femoral artery and vein will be visualized traversing dorsal to ventral across the lateral abdominal wall medial to the coxofemoral joint (Fig. 11-14). These vessels should be cauterized with the bipolar forceps before transection.

The musculature (external and internal abdominal oblique and transversus abdominis muscles) should be tented away from the coelomic contents and incised with sharp fine scissors while protecting the internal viscera. The incision is extended from pubis to the eighth rib. Bipolar forceps are placed around each rib, from the caudal aspect, such that the forceps close over the anterior border of the rib to cauterize the intercostal vessels (Fig. 11-15) before transecting the rib (with large scissors). The last two to three ribs (i.e., numbers, 7 and 8) are transected in turn. A small retractor (e.g., Heiss or Alm) is then inserted between the cut rib ends to enable full visualization of the abdominal cavity. The Lone Star retraction system is invaluable for such surgeries (Fig. 11-16, *A*). On completion of the intercoelomic surgery the incision is closed using 4-0 to 6-0 absorbable monofilament synthetic material in a continuous or interrupted pattern in two layers. The intercostal muscles are opposed and no attempt is made to rejoin the transected ribs.

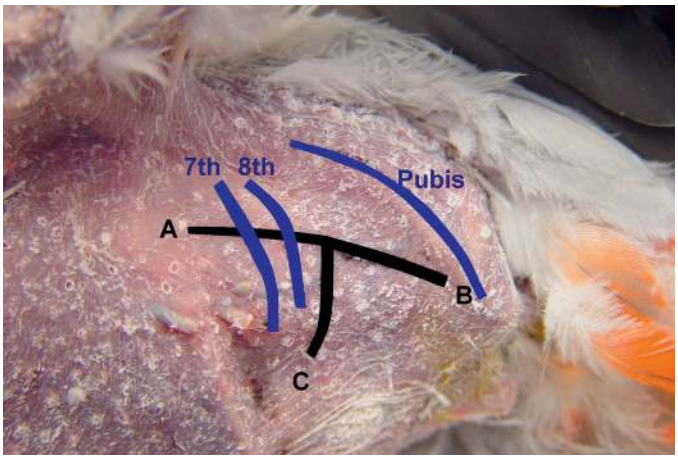


FIGURE 11-13 Landmarks for skin incision for left lateral coeliotomy (from sixth rib to pubis), with ventral flap if necessary to increase access.

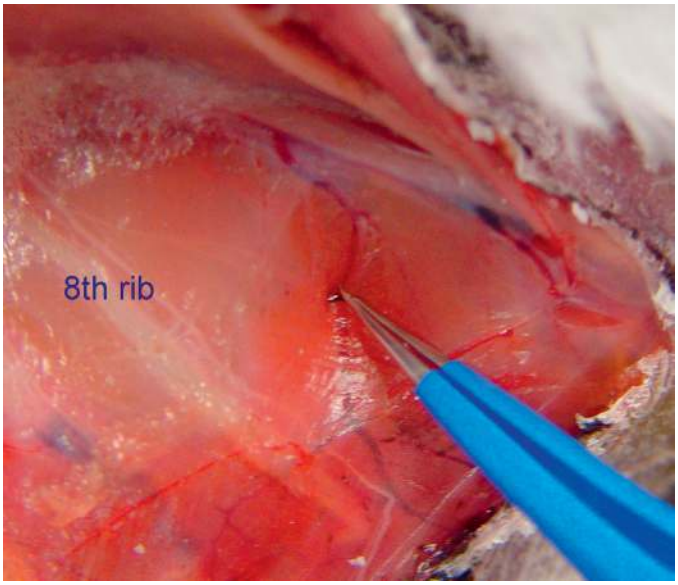


FIGURE 11-14 Medial femoral artery and vein to cauterize.

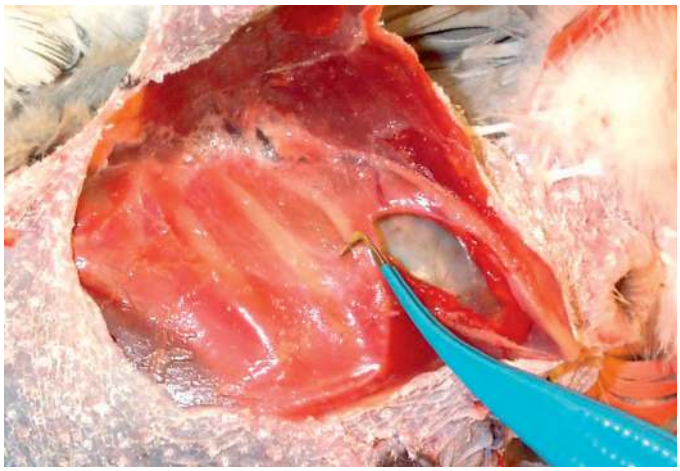


FIGURE 11-15 Bipolar radiosurgery-cauterized intercostal vessels.

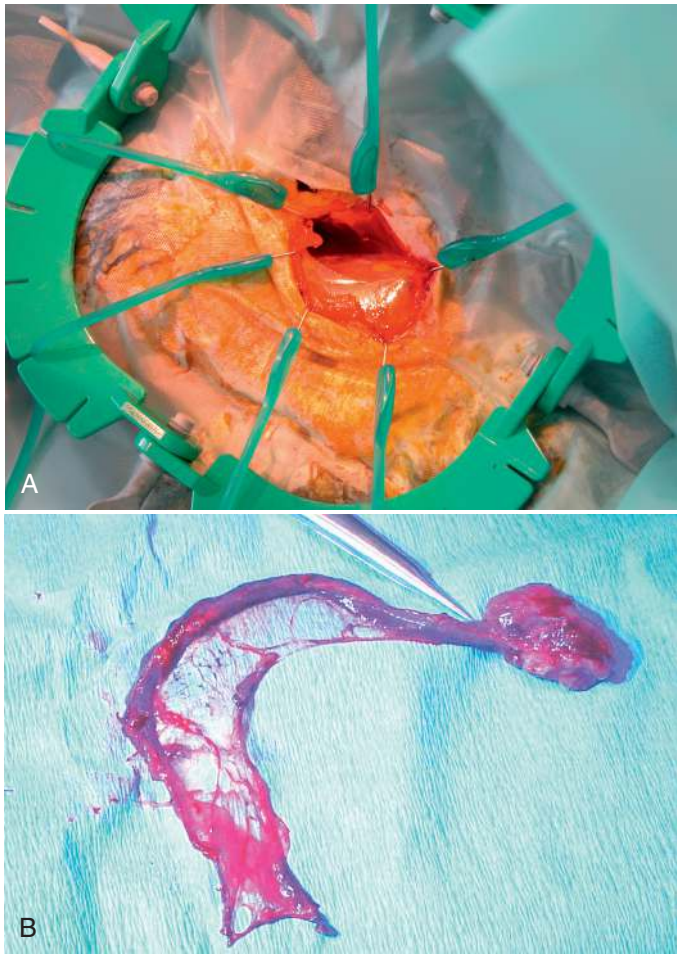


FIGURE 11-16 (A), Use of Lone Star retractor to maximize access. **(B),** Oviduct with intussusception after surgical removal.

Salpingohysterectomy

Removal of the avian ovary is challenging and typically dangerous (it is firmly attached to the dorsal abdominal wall, with a short ovarian artery, directly off the aorta); however, to prevent further egg laying one may instead remove all of the oviduct and uterus. This procedure may be indicated to prevent egg laying or to resolve oviductal pathology e.g., prolapse, neoplasia, or intussusception (Fig. 11-16, *B*). A review of ovariectomy techniques is discussed in Scott Echols (2002). Oviduct removal is not always effective at preventing further follicle release in ducks; in such cases, a GnRH implant is recommended following salpingohysterectomy, to prevent follicle release.

The first step is to recognize the oviduct. It will vary in size depending on breeding condition and will typically be situated dorsal to the intestine, ventral to the kidneys, and appear whiter compared with intestine, with a longitudinal pattern (Fig. 11-17). The oviduct is isolated and the ventral suspensory ligament (of the oviduct and uterus) is broken down with blunt dissection (Fig. 11-18). The oviduct is followed cranially to reach the infundibulum (Fig. 11-19). A significant blood vessel enters the infundibulum on the medial aspect from the ovary, and this should be clamped off with two hemoclips (Fig. 11-20) before transection. The dorsal suspensory ligament of the uterus should be identified extending from the dorsal abdominal wall to the uterus. In this ligament are a number of significant blood vessels that should be coagulated or clipped (Fig. 11-21). The uterus and oviduct are then exteriorized. As one moves dorsocaudally toward the dorsal abdominal wall, care should be taken in resecting the dorsal suspensory ligament to avoid resection of either ureter. The placement of a cotton bud in the cloaca will assist in delineating where the uterus should be clamped off (Fig. 11-22) immediately craniad to the cloaca. The latter is achieved by applying two clips to the uterus and transecting on the uterine side. Hemorrhage is controlled before closure of the abdominal muscle wall and the skin with a simple continuous suture pattern.

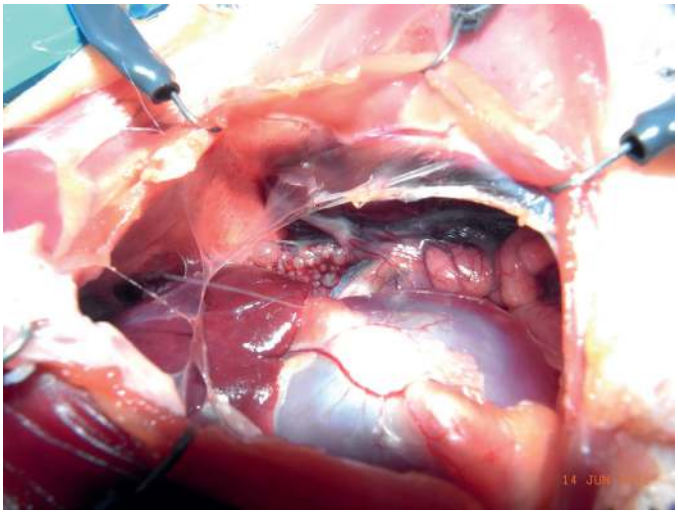


FIGURE 11-17 Identification of the oviduct.

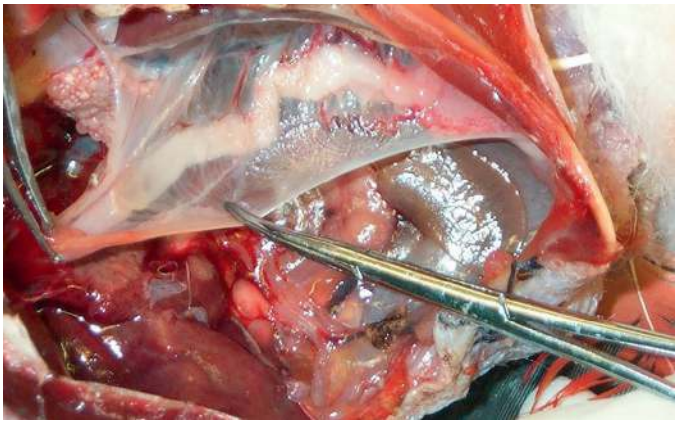


FIGURE 11-18 The ventral supporting ligament of the oviduct. It has minimal vasculature and is broken down by blunt dissection.

Caesarian Section

Caesarian section may be considered as an alternative when a hen is suffering from egg binding and either the egg or the hen are of high financial or conservational value, or she is suffering from egg binding that is nonresponsive to medical support or cloacal removal. Depending on the position of the egg a caudal left lateral or, more commonly, a ventral midline incision is made. The oviduct is incised directly over

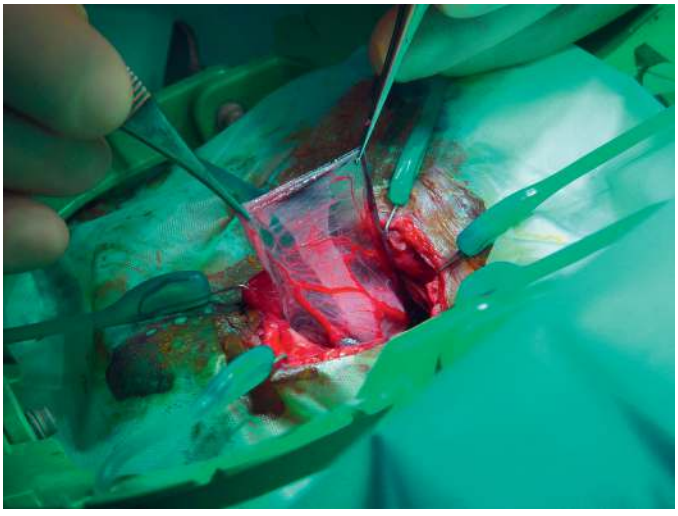


FIGURE 11-19 The oviduct is followed cranially to identify the infundibulum.

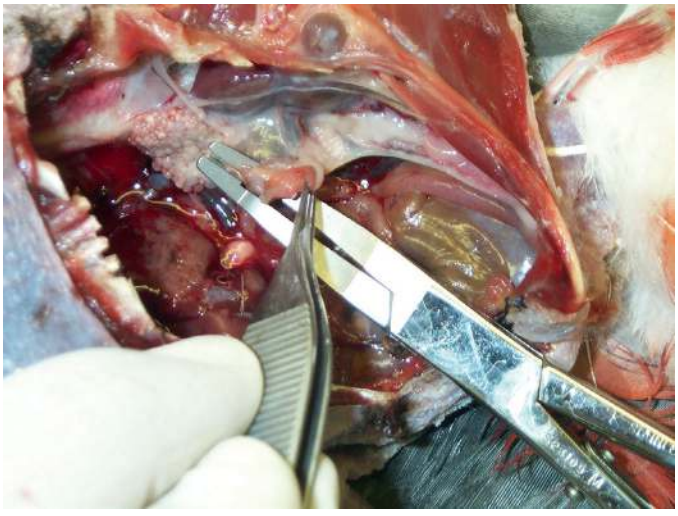


FIGURE 11-20 Hemoclip application of blood vessels supplying infundibulum.

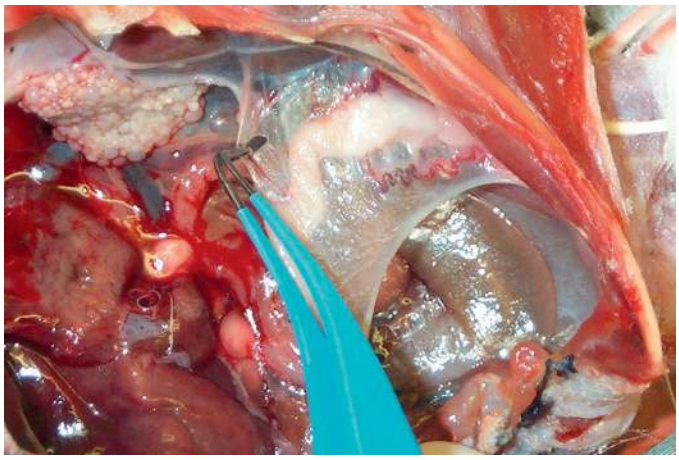


FIGURE 11-21 Vasculature in the dorsal oviductal suspensory ligament is cauterized.

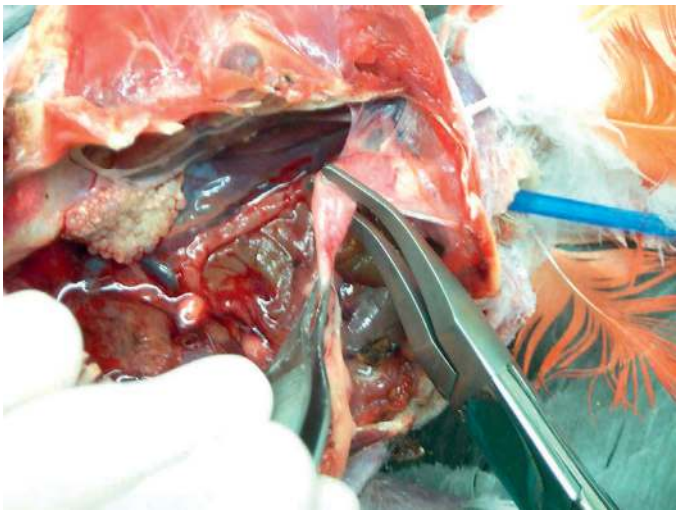


FIGURE 11-22 Hemoclip applied at junction of the oviduct and cloaca.

the egg, avoiding any prominent blood vessels. After egg removal, the oviduct is inspected and the cause of binding determined and rectified as necessary. If correction is not possible, salpingohysterectomy may be indicated at a later date but would be inappropriate at this time. The oviduct is closed with a single interrupted or continuous pattern using 4-0 or finer absorbable material.

Uterine Torsion

Uterine torsion egg binding is consequent to a range of etiologies. If the condition does not respond to medical support, particularly if there is a significantly distended coelom, then torsion of the oviduct should be considered as a differential diagnosis (Harcourt-Brown, 1996). In such cases the oviduct will have suffered a torsion (beyond which no eggs may pass). A number of eggs in a varying state of decay may be present in the proximal oviduct. For torsion to occur, there is typically a traumatic breach of the ventral or dorsal suspensory ligament through which the oviduct may have passed. Many such patients are in poor condition and represent poor surgical risk. Once optimally stabilized, a ventral midline surgical approach is used to access the oviduct. Typically initial surgical drainage of the oviduct is necessary, the torsion reduced, and the breach of the suspensory ligament repaired. A salpingohysterectomy may alternatively be performed.

Orchidectomy

Orchidectomy may also be performed by the left lateral coeliomic approach The testes (like the ovaries) are attached to the dorsal abdominal wall, adjacent to the aorta, connected by a short testicular artery. The left testicle is identified, the caudal pole is elevated, and a hemoclip is placed under the testicle. The testicle is then incised over the clip. Once achieved, a further clip is applied in a more craniad position, etc. If any testicular tissue is left, there is a possibility of regeneration. Access to the right testicle will be more difficult requiring blunt dissection through the air sac wall or via a fresh incision on the contralateral abdominal wall. A similar removal process may then be performed on this testicle.

Neutering

As described earlier, an ovariectomy is a very risky procedure whereas an orchidectomy is marginally less risky. Indications for neutering may be to prevent breeding (vasectomy is safer in a male bird and salpingohysterectomy is a preferred option in a female, although ova may still on occasion be released into the abdomen), but such a motivation is uncommon. Historically the technique has been most commonly applied to control persistent egg laying in females or aggression and hypersexuality in males. Currently the view is that by reducing the energy density of the diet (by converting seed- and nut-eating birds onto a pelleted and fresh fruit and vegetable diet), initially using GnRH products, and introducing behavioral modification training, surgery is rarely indicated. Alternatively laparoscopic neutering (ovary, testes, or vas deferens), radiosurgical or laser obliteration, is an effective technique in juvenile birds.

Endoscopic Testicular Biopsy

Infertility in psittacine birds should be thoroughly investigated (see Chapter 13). In situations where male infertility is suspected 3 mm endoscopic biopsies may be harvested from bird's testes outside of the normal breeding season and microbiologically and histologically examined.

Proventriculotomy for Access to Proventriculus or Ventriculus

Birds are able to mount a very fast (compared with mammals) inflammatory response and wall off noxious agents, often rendering them harmless. In diving ducks and other species that have a highly muscular ventriculus and are prone to ingesting peculiar and often sharp foreign bodies, one often finds items such as segments of glass, wood, and metal walled off within the coelomic cavity. These objects will have originated from the ventriculus.

Proventriculotomy is most commonly indicated for the removal of foreign objects that are not retrievable per os or ingluvies with rigid or flexible endoscopes. Proventricular biopsy is not recommended as the diagnostic method for PPDS because of the unacceptable risk of postoperative wound dehiscence and fatal coelomitis. Although this technique has been described, ventriculotomy is generally avoided because of the highly muscular walls (the postoperative physiologic muscular activity of pulling sutures through the muscle wall), the inability to form an inversion closure, and the increased vascularity compared with the proventriculus. Ventricular foreign bodies can be accessed via an incision in the isthmus between the proventriculus and the ventriculus.

Access is gained via the left lateral coeliotomy approach; sufficient exposure is necessary to visualize the suspensory membranes and to avoid the proventricular vessels along its greater curvature. The ventriculus (gizzard) is identified as a muscular organ with a white

tendinous lateral aspect. Blunt dissection is used to break down the ventricular suspensory attachments. Two (3-0) stay sutures are placed in the white tendinous part of the ventriculus, elevated, and approximated to the external abdominal wall. In some species exteriorization is possible (Fig. 11-23). Depending on the size of the patient it is advantageous to pack off the coelom about the ventriculus with saline-soaked gauze swabs to minimize the effect of any leakage. The triangular portion of liver, which covers the isthmus, is identified (Fig. 11-24). Using a sterile cotton bud the liver is elevated, revealing the optimum incision site into the isthmus (junction between the proventriculus and the ventriculus), to facilitate biopsy or access to the proventriculus or ventriculus for foreign body removal (Fig. 11-25). An initial stab incision is made that is extended with iris scissors. Suction must be available at the time of initial incision to aspirate enteric contents and avoid spillage.

An endoscope may be passed into the gut via the incision in both cranial and caudal directions to verify that all foreign objects have been removed. The incision is closed in two continuous layers (opposed then inverted) using 4-0 to 8-0 synthetic absorbable monofilament material, after which the liver is tacked in place over the proventricular incision site. The body of the proventriculus has a poor ability to hold sutures and tears readily as sutures are tightened. Care should be taken to place sutures a sufficient distance from the wound edge so that they do not tear through, but not so far that undue pressure has to be used to close the wound, as this will also lead to tearing. Suture placement in normal gut surgery includes the submucosa in view of its greater collagen content; however, the avian proventriculus has minimal collagen, so greater care is required. The placement of collagen patches over a traditional ventricular closure does not reduce the incidence of wound breakdown. As birds have no mesentery, enterotomy carries a higher risk of postoperative coelomitis. The liver plays the role of the mesentery in overlying the closed isthmal incision. The ventricular suspensory ligaments are not repaired. Closure is as described earlier. Care should be taken to minimize collateral damage during incision and repair of the isthmus. It has been demonstrated in turkeys that the entire neural network situated within the isthmus must remain intact for normal gastroduodenal motility to occur.

Neoplasia of the proventriculus and ventriculus is uncommonly reported in psittacine birds. In the author's experience they are most commonly presented in aged (greater than 30 years) Amazon parrots. Carcinoma of the proventriculus is more common than adenocarcinoma of the ventriculus. Clinical signs may include the passage of undigested seed and regurgitation. The bird may appear sick and weak, often suffering from secondary infections or other complications. Sizeable lesions may be visualized radiographically (optimized by per os catheter placement of barium in the proventriculus) and may be confirmed via endoscopic biopsy. The gross appearance of proventricular adenocarcinoma is often not obvious and only likely to be differentiated on histopathological examination. By the time of diagnosis, such cases do not normally lend themselves to surgical removal; however, such lesions may be amenable to chemotherapy (Fillipich, 2004).

FIGURE 11-23 Exteriorization of ventriculus by placement of stay sutures in ventricular fascia.

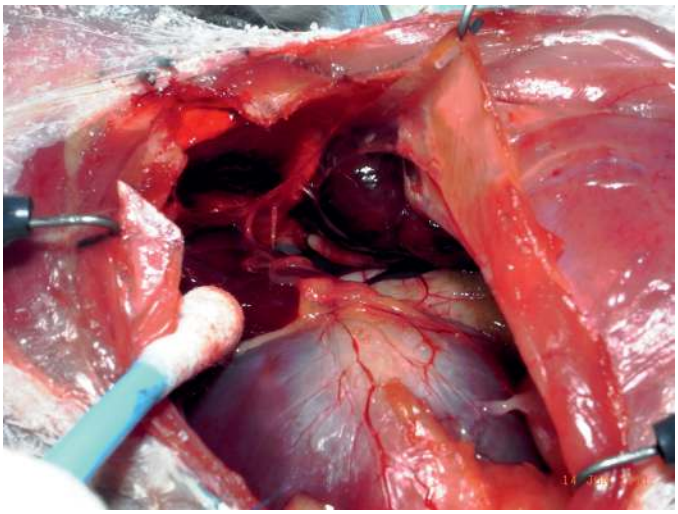


FIGURE 11-24 Identification of triangular liver lobe covering isthmus.

Yolk Sacculectomy

In neonate chicks, the presence of an infected or unretracted yolk sac necessitates surgical removal. Chicks that eat early after hatching and those affected by reduced gut motility are believed to have a higher incidence of unretracted yolk sacs. Yolk sac infections are often concurrent with umbilical infections, enteritis, or septicemia. Clinical signs

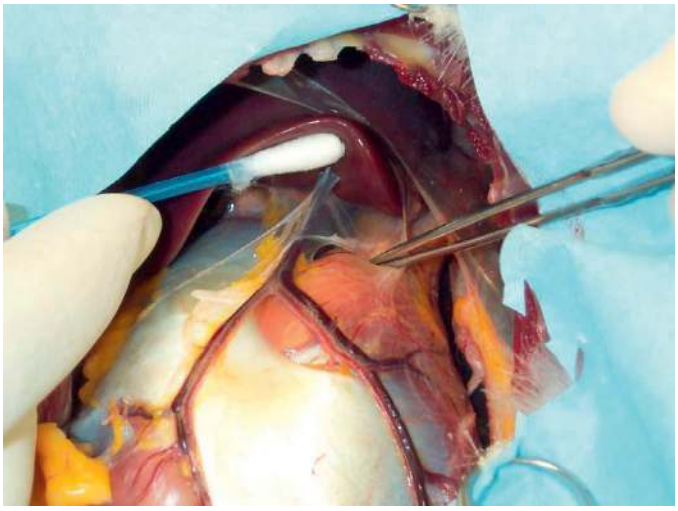


FIGURE 11-25 Elevation of the liver lobe to reveal the proventricular-ventricular isthmus showing incision site for access to ventricular lumen.

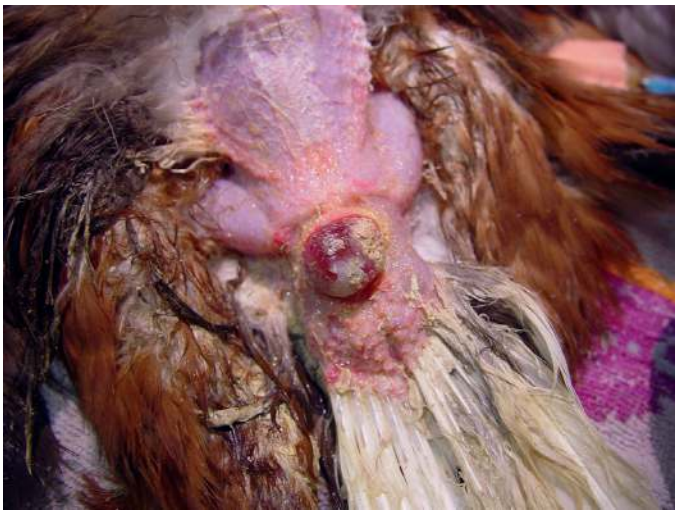


FIGURE 11-26 Per cloacal colon prolapse (*Parabuteo unicinctus*).

include anorexia, lethargy, constipation, diarrhea, weight loss, and abdominal distention. Noninvasive ultrasonographic diagnosis is readily achieved. By the time of diagnosis medical therapy alone is rarely efficacious. Following induction of anesthesia, the bird is placed in dorsal recumbency with the head elevated. A small incision is delicately created cranial to the umbilicus. This incision is extended around the umbilicus and the umbilical stump is excised. The yolk sac is exteriorized and the duct ligated. Care is taken to avoid rupture or spillage of the yolk sac contents. The abdominal incision is closed in two layers. Surgical survival rates in sick neonates are not high.

Enterotomy

Enterotomy is an infrequent procedure typically necessary following trauma to the gastrointestinal tract, iatrogenic surgical damage, intussusception, torsion, adhesions, enteroliths, or areas of necrosis. This procedure carries a guarded to grave prognosis. If the colon is prolapsed via the cloaca (Fig. 11-26), an intussusception must be present, typically originating at the level of the vestigial ceca, a short distance above the cloaca. Such cases require an immediate midline (with or without flap) coeliotomy and reduction of the intussusception, which may contain a length of devitalized gut. An enterectomy will be required to remove any devitalized gut. Intussusception has also been seen secondary to linear foreign bodies or following enteric infections. Midline flap incisions give optimal access. Microsurgical instrumentation and techniques are mandatory. Blood vessel appositional clamps (e.g., Acland clamps) are invaluable to atraumatically achieve intraoperative intestinal occlusion while maintaining the tissue sections in apposition during suture placement. These vascular clamps are designed to avoid tissue slippage while maintaining low pressure to avoid tissue damage. The clamps may be used individually or preferably attached to a bar or rectangle so that both ends of the tissue are adjacent to each other. When passing needles through fine tissue, it is important that the needle is encouraged to follow its natural curvature, otherwise an excessive needle hole is created. The arcing instrument action achieved by finger rolling of round-bodied instruments minimizes this problem.

Intestinal Anastomosis

Anastomosis of the intestine may be performed using an end-to-end technique using 6-0 to 10-0 material with a simple appositional method (Fig. 11-27); however, the most common postoperative

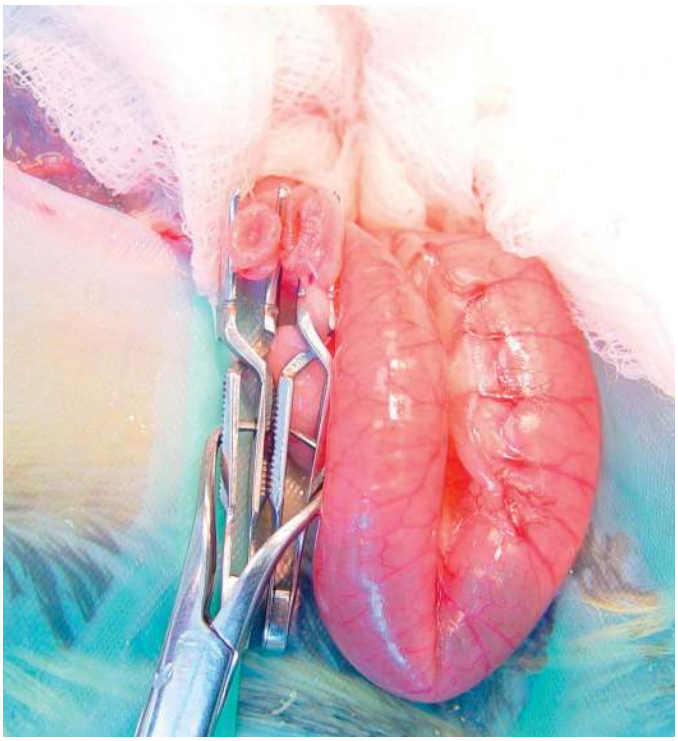


FIGURE 11-27 Appositional blood vessel clamps use to present gut ends for anastomosis.

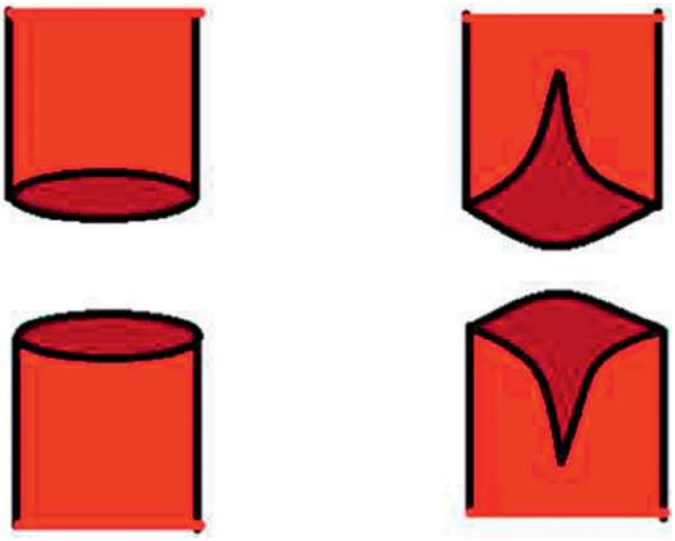


FIGURE 11-28 The gut wall incision to be joined is increased in diameter to maximize functional lumen size.

complication arises from a reduced functional lumen. To maximize the functional postoperative diameter, the length of the two ends are increased, as shown in Figure 11-28. If the site of the intussusception is the vestigial ceca (Fig. 11-29), and hence only a matter of centimeters cranial to the cloaca, then after the far lateral wall is anastomosed (Fig. 11-30) a section of appropriate diameter sterile tubing may be passed from the operative site caudally via the cloaca then readvanced cranially across the anastomosis site (Fig. 11-31). Once in place, the anastomosis repair is completed around the tube (maintaining a functional

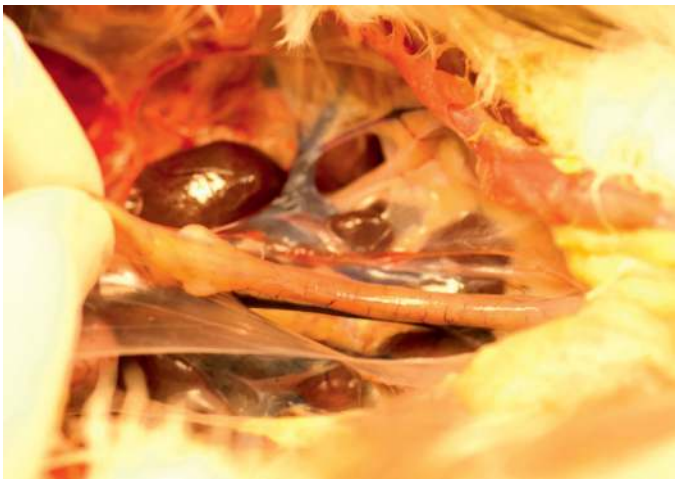


FIGURE 11-29 Vestigial ceca in an eagle. This is the most common site for the origin of a colonic intussusception.

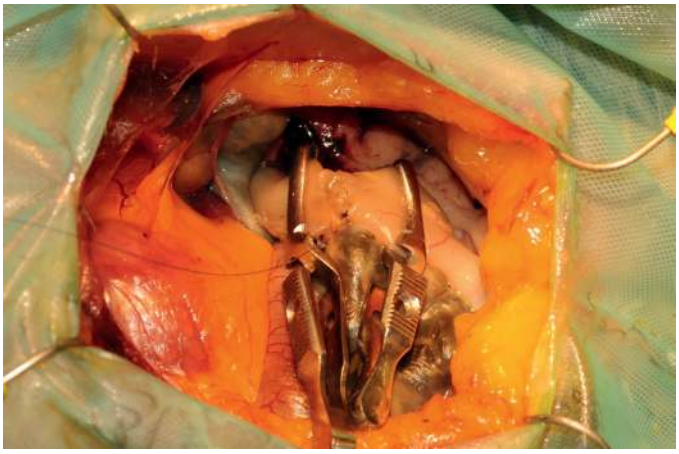


FIGURE 11-30 Anastomosis site with distal side repaired.

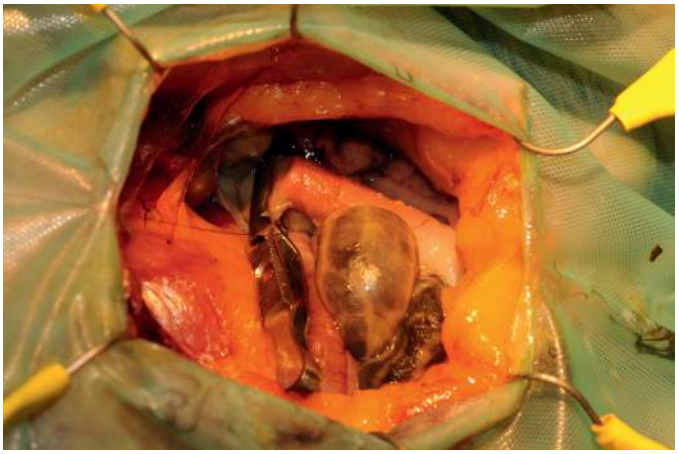


FIGURE 11-31 Anastomosis site with rubber drift in place. The anterior wall of the colon is not yet repaired.

lumen; Fig. 11-32), and when completed the tube is withdrawn per cloaca (Forbes, 2013).

If the gut is less than 2 mm in diameter then six to eight simple interrupted sutures are used (similar to a blood vessel anastomosis). If the gut is greater than 2 mm in diameter a continuous pattern may be used. A continuous pattern is advantageous as it reduces surgical time, yields improved apposition and reduces leak incidence, and there is reduced tissue irritation and improved endothelialization. Sutures are initially placed at 12 PM and 6 PM then placed in the caudal section of gut before placing sutures in the anterior aspect.

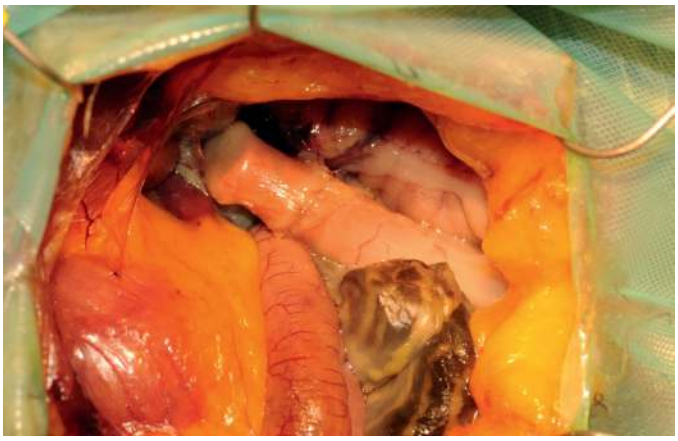


FIGURE 11-32 Anastomosis site repaired before removal of rubber drift per cloaca.

Ventral Midline Coeliotomy

This approach gives poor visibility of the dorsal and cranial coelom. However, it facilitates surgery of the small intestine, pancreatic biopsy, liver biopsy, or cloacopexy and is used in diffuse abdominal diseases such as peritonitis, egg binding, and cloacal prolapses. The bird is placed in dorsal recumbency, the midline prepared, and the legs abducted caudally. The skin of the abdominal wall is tented and an initial incision is made using scissors (care is taken to prevent iatrogenic visceral damage). The incision is extended with fine scissors. This incision can be extended dorsally at both ends or either end along the costal border cranially or to the pubis caudally to create a flap on one of both sides of the midline to increase access. This approach is particularly useful for access to the caudal uterus and cloaca.

Abdominal Hernia

Abdominal hernias are most common in obese female psittacines, especially cockatoos and budgerigars. They are often related to chronic hyperestrogenism (most common in "hand-reared owner-fixated birds"), resulting in increased oviductal size, ovarian follicular development, and often hepatomegaly from lipidosis or other coelomic space-occupying masses, creating excessive tension on the abdominal wall. High-energy diets lead to obesity and also serve as a drive to egg production (increasing liver size and follicular activity, respectively). Before any consideration of surgery, hormonal activity must be abated with GnRH, and the bird must be converted from a seed-based diet to a more balanced (pelleted) or fresh-food diet and exercise enforced (e.g., 30 minutes walking daily) to achieve a significant weight reduction.

Avian abdominal hernia is dissimilar to mammalian hernia. There is no specific hernia ring; instead there is a thinning and longitudinal splitting between the muscle fibers. Surgery to pull the sides of the deficit together will simply result in repeated adjacent splitting. It is recommended that any space-occupying organs that can be removed should be removed, particularly the oviduct, so salpingohysterectomy is performed concurrent with abdominal wall repair. The owner should be warned that this may not be effective, and placement of a

surgical mesh across the hernia site may be required. Such surgery is only contemplated if trauma and skin abrasion over the herniated mass are ongoing following salpingohysterectomy. An extensive ventral midline bilateral flap approach is used. Mesh is attached bilaterally to the pubis, to each eighth rib, and to the sternum. These meshes are generally well tolerated, although intense attention to sterility during surgery is mandatory. Herniation can occur secondary to abdominal lipoma, cystic structures, neoplasia, or other space-occupying masses.

CLOACAL CONDITIONS

Cloacal conditions are common in pet birds with varied etiologies such as cloacitis (caused by papilloma; Fig. 11-33), neoplasia, uroliths (Fig. 11-34), mycobacteriosis, parasites, neoplasia, cloacal prolapse associated with oviductal (Fig. 11-35) or urethral obstruction, cloacal fistula (Fig. 11-36), or other oviductal disease or behavioral (hypersexuality and lack of dominance, resulting is self-abuse and cloacal stretching; Fig. 11-37) abnormalities (Doneley, 2010; Forbes, 2013).

Organs Prolapsed Through the Cloaca

Apart from partial cloacal prolapses, prolapsed cloacal masses (papilloma, neoplasia, or mycobacterial granuloma) and total prolapses can occur where the colonic, urethral, and oviductal junctions may be everted (Fig. 11-38). Alternatively, the oviduct or colon may be prolapsed (see Figs. 11-26 and 11-35). Differentiation of the tissues

FIGURE 11-33 Cloacal papilloma.

FIGURE 11-34 Cloacal urolith.

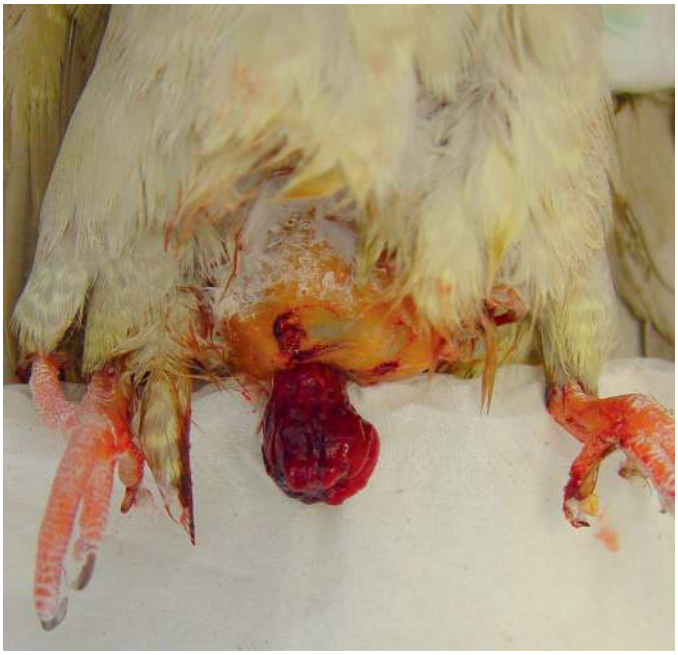


FIGURE 11-35 Cloacal oviduct prolapse.

FIGURE 11-36 Cloacal fistula.

FIGURE 11-37 Cloacal stretching.

involved is important and is achieved by assessing the size of the structures present (Forbes, 2013). Birds presented with such prolapses are suffering extreme shock. Fluid therapy, analgesia, and antiinflammatory therapy are all mandatory. If a colonic or uterine prolapse is present, this is inevitably secondary to an intussusception. Pushing the offending organ back through the cloacal opening and placing a purse string suture is not acceptable or effective. A coeliotomy, with reduction or removal of the intussuscepted material, is essential.

Cloacal Papilloma

Cloacal papillomas are particularly common in South American species, e.g., macaws and Amazons. All such birds should undergo a choanal and cloacal examination for papillomas as a routine part of any clinical examination. One or more cotton buds are passed into the cloaca then gently retracted to evert the cloacal lining. Birds with cloacal or choanal papilloma will go on to develop intestinal, pancreatic, or biliary adenocarcinoma (Schmidt *et al.*, 2003). Cloacal papillomas are caused by oncogenic herpes virus. Cloacal, colonic, or oviductal prolapses may resemble neoplasms, particularly if the prolapse tissue is necrotic. Histologic examination of cloacal tissues is advised.

Many treatment modalities have previously been suggested ranging from repeated alternate day application of silver nitrate, inclusion of 2% capsicum in the diet, autogenous (high antigen-loaded) vaccination, cryosurgery, radiosurgery, and YAG laser therapy. However, in light of the current knowledge regarding the etiology (oncogenic herpes virus infection), contagious nature, and long-term patient prognosis of these cases, excessive efforts to achieve a complete surgical resolution now seem futile.

Cloacolith

These are firm, rough-surfaced urate aggregates (see Fig. 11-34). They are uncommon and the pathogenesis is unclear. This author has experienced them most frequently in carnivorous birds, especially after extended periods of nesting or brooding behavior when feces have not been voided as frequently as normal. Birds present with repeated straining, often passing scant traces of blood. This condition is readily diagnosed on digital exploration of the cloaca. After the bird is anesthetized, the cloacolith may be fragmented with artery forceps and removed piecemeal. Analgesics and antibiosis should be administered.

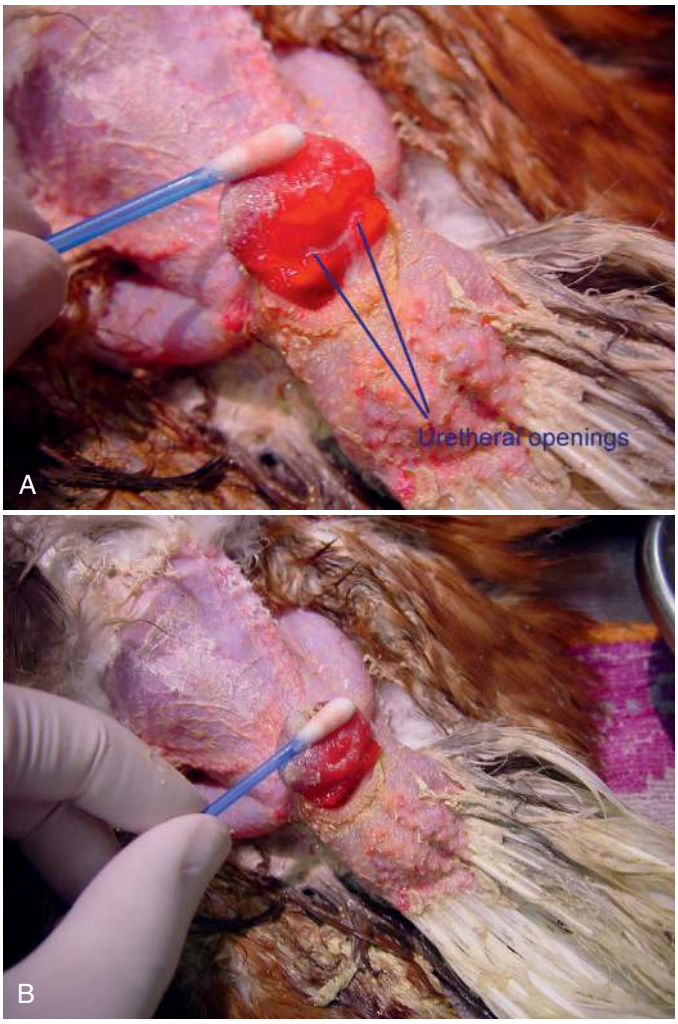


FIGURE 11-38 Cloacal prolapse showing **(A)** ureter openings and **(B)** colon opening in a Harris hawk (*Parabuteo unicinctus*).

Cloacopexy

Cloacal prolapse is the common indication for a cloacopexy, and is seen most often in hypersexual birds. Cockatoos are most commonly afflicted. Initial amelioration of activity may be achieved with GnRH therapy. Behavioral modification is important, particularly gaining dominance over the bird and reducing its psychological and nutritional drive toward sexual activity.

For cloacopexy a cotton bud is advanced into the cloaca, which is used to tent the cloaca within the abdomen, to confirm its position (Fig. 11-39). A horizontal incision is made over the most anterior portion being careful not to incise the thin-walled cloaca (Fig. 11-40). A fat pad is often present on the ventral aspect of the cloaca and should be removed. Where marked stretching and distention of the cloaca has occurred, the cloaca may also be attached bilaterally to the eighth rib. In such cases, a suture with a swaged on needle is passed through the skin, (from external to internal) just cranial to the eighth rib (the

FIGURE 11-39 Cotton bud passed per cloaca, showing most anterior position of cloaca and eighth ribs bilaterally.

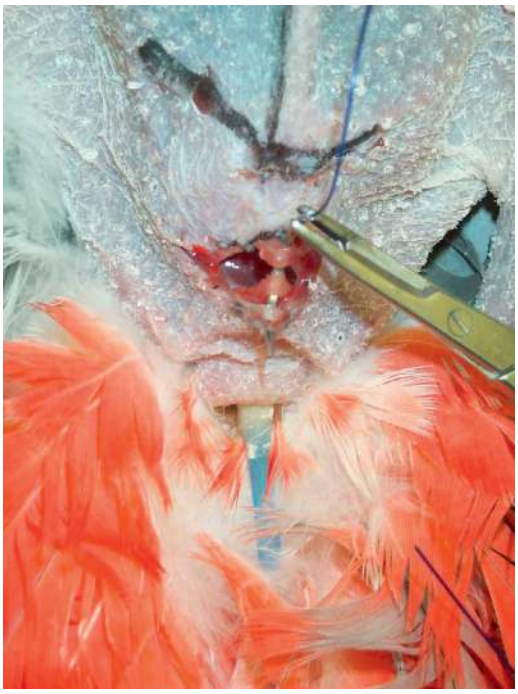


FIGURE 11-41 The midline incision is closed encompassing the cloacal wall within the closure.

FIGURE 11-40 The cloaca in an African grey parrot (*Psittacus erithacus*) is exteriorized and if a fat pad is found on the ventral aspect, this is carefully removed.

viscera is held away from the needle as it enters the coelom by a finger within the coelomic cavity). The needle is then exteriorized via the coelomic incision, turned around and returned via the incision into the coelom, it is then passed (internal to external) just caudal to the last rib. Once sutures have been placed bilaterally, then they are tied off against the free end (i.e. external to the skin), which had been left cranial to the eighth rib. (This should only be done if the stretched cloaca will reach and remain in contact with the medial aspect of the eighth rib such that an adhesion forms here.) As the midline incision is closed, two further sutures are placed through the cloacal wall and incorporated in the abdominal wall closure (Fig. 11-41). Although surgical recommendations exist for the correction of cloacal prolapses, recurrence is common, particularly if the hypersexual behavior is not addressed. Cloacoplasty is often performed simultaneously with cloacopexy.

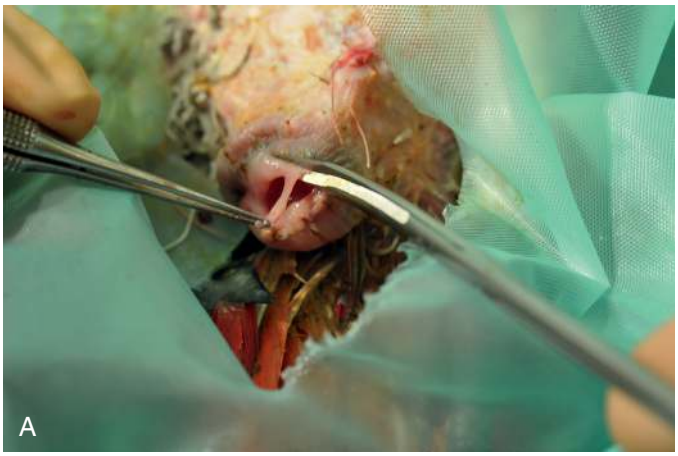
Cloacoplasty

Cloacoplasty (reducing the internal diameter of the cloacal aperture) is also recommended where there is atony and stretching of the vent sphincter. The internal mucosal margin of the vent is excised bilaterally (Fig. 11-42, *A*), up to 60% of the circumference (to provide a cut edge for healing). The edges are then sutured from side to side (Fig. 11-42, *B*) to reduce the diameter of the cloacal aperture. This condition most commonly occurs in hypersexual cockatoos that masturbate.

RESPIRATORY TRACT SURGERY

Tracheotomy

This procedure is most commonly indicated in the treatment of syringeal aspergilloma or retrieval of a foreign body from the trachea. The bird is placed in dorsal recumbency with the head toward the surgeon. The head is elevated at 45 degrees to the tail to facilitate interoperative visualization into the anterior thoracic cavity. A skin incision is made cranial to the thoracic inlet. The crop is identified, bluntly dissected, and displaced to the right (Fig. 11-43). If additional access is required the clavicle on one side is resected (Fig. 11-44). Optimal access is achieved with a Lone Star retractor. The interclavicular air sac is entered and the trachea elevated. The sternotrachealis (attached bilaterally to the ventral aspect of the trachea) is transected (Fig. 11-45). In most species it is impossible to completely exteriorize the syrinx. The tracheal incision should be as close to the syrinx (or foreign body) as possible to close the incision afterward. Tracheotomy is conducted, using a fine blade or hypodermic needle, cutting one half of the tracheal circumference between two adjacent tracheal cartilages (Fig. 11-46). Stay sutures are placed around two tracheal rings, on either side of the incision, and used to abduct the edges and maximize access into the trachea (Fig. 11-47). Aspergilloma or foreign material



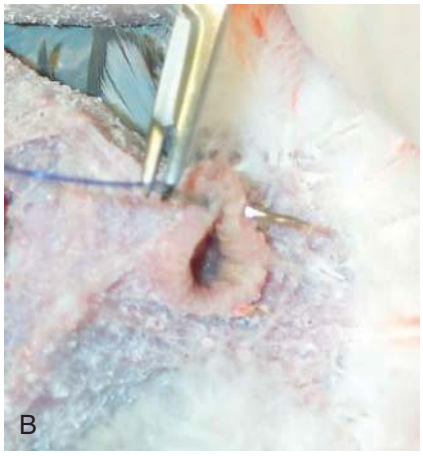


FIGURE 11-42 (A), Internal cloacal mucosa is removed bilaterally. **(B),** Sutures are placed bilaterally via the skin, muscle, and mucosa to effect the reduction in internal diameter.

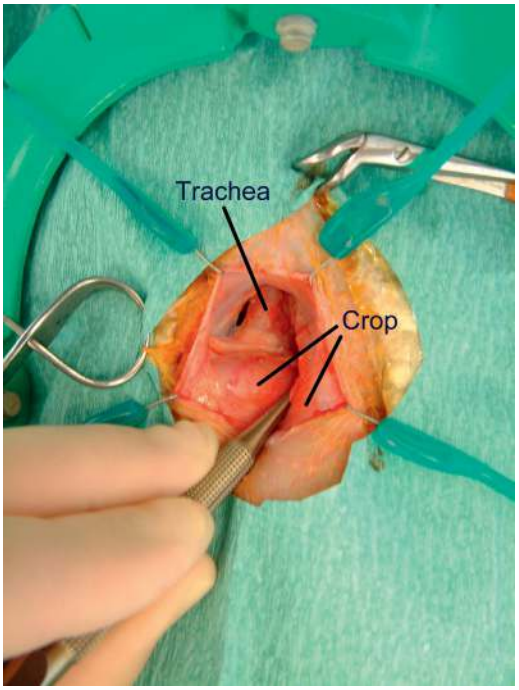


FIGURE 11-43 With the head elevated at 45 degrees, the skin is incised and crop reflected to the right.

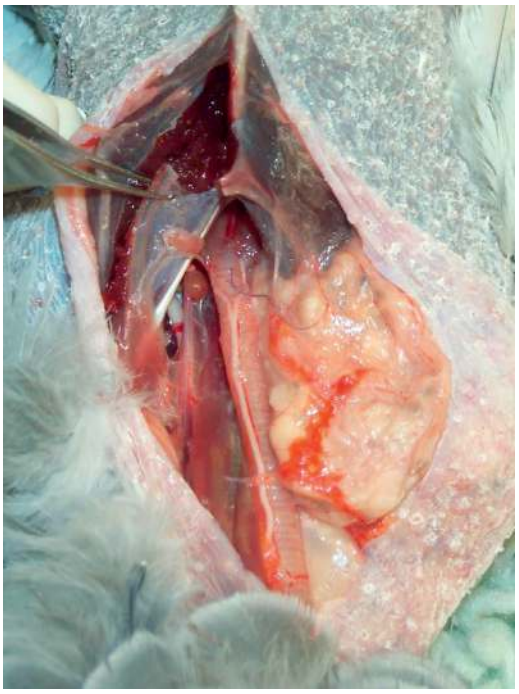


FIGURE 11-44 If additional access is required the clavicle is retracted on one side.

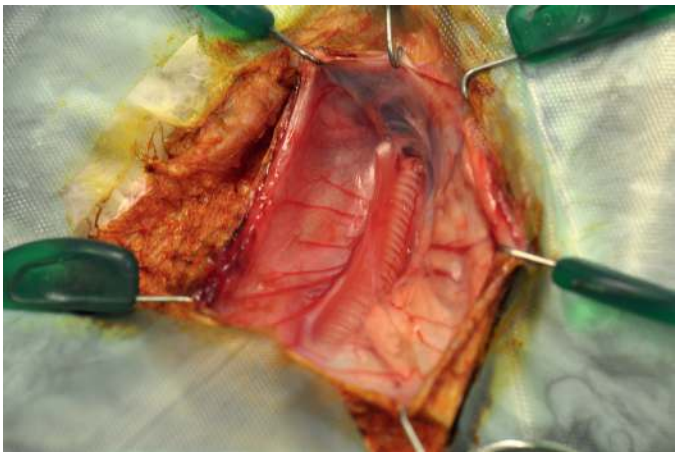


FIGURE 11-45 The sternotrachealis muscle is located on either side of the trachea and resected.

may be removed with Harris ring tip forceps, biopsy crocodile forceps, or by suction applied to an intravenous catheter (Fig. 11-48). The incision is repaired with single interrupted sutures (6-0 Maxon, two to three sutures only) placed to include two rings on either side of the incision (Fig. 11-49). On closure the two ends of the clavicle are apposed but not rejoined. The muscle is replaced and sutured into position. The crop is sutured back into place, to create an airtight repair around the thoracic inlet, using a continuous suture pattern and absorbable suture material. The skin is closed in a routine manner.

Trachectomy

In cases where severe tracheal stenosis has occurred following trauma (e.g., recent intubation; Fig. 11-50) or infection (e.g., a caseated aspergillomata), tracheal resection and removal of the affected tissue may be performed. Depending on the site of the lesion, in most individuals

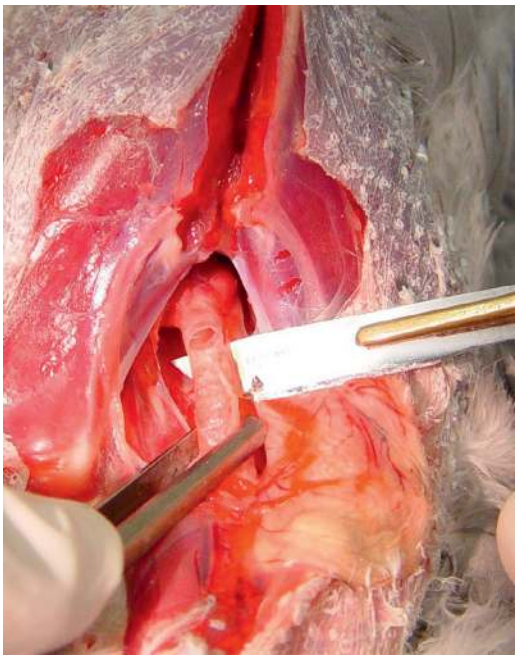


FIGURE 11-46 The tracheal incision is created with fine blade or hypodermic needle, as close to the syrinx as possible, but in a position that can be sutured closed.

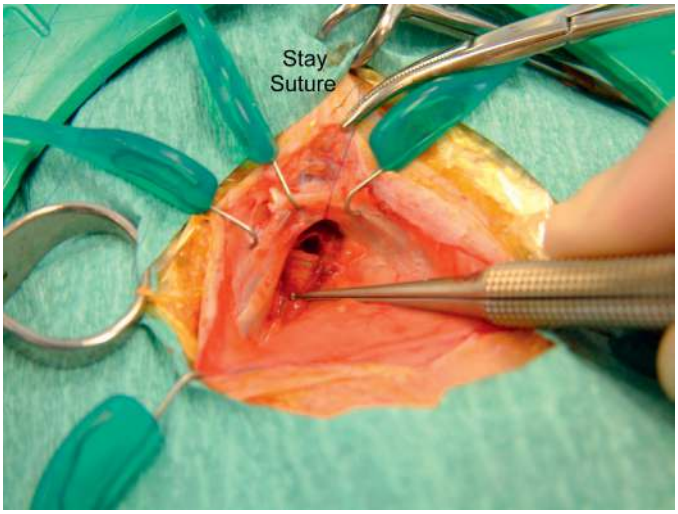


FIGURE 11-47 Stay sutures are placed across two tracheal rings on either side to abduct and maximize view of the lesion.

six tracheal rings can be removed and the trachea successfully rejoined. In such cases, close apposition of cartilages following surgery using a suture material that elicits minimal tissue reaction (e.g., PDS, Ethicon) is used to minimize the risk of intraluminal granuloma formation. Trauma to tracheal tissues during surgery must be minimized. It is preferable to place sutures in the trachea at the time of resection to facilitate apposition and anastomosis. Two to four sutures are used (depending on patient size) and are all preplaced before any are tied. The dorsal suture is then tightened first (Fig. 11-51).

BIOPSIES

Lung Biopsy

A lung biopsy may be collected endoscopically or surgically (which is typically safer). The bird is laid in lateral recumbency, the leg extended

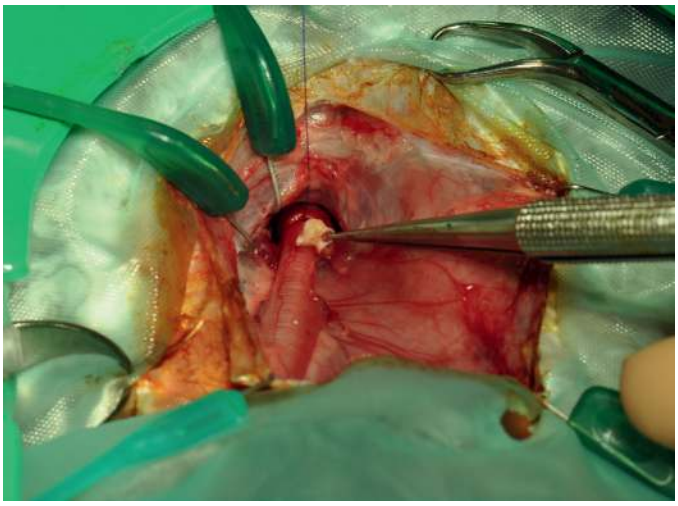


FIGURE 11-48 The lesion may be removed using fine forceps or a fine tip attached to suction.

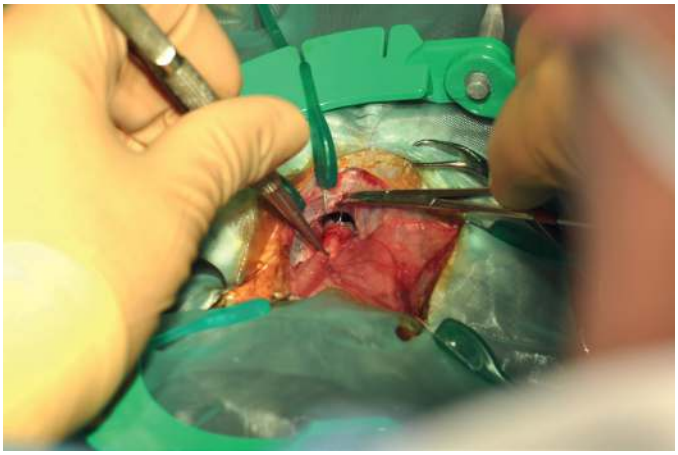


FIGURE 11-49 The tracheal incision is closed using minimal numbers of fine, absorbable nonirritant material (e.g., 6-0 PDS), placed across two rings on each side.

caudally and the wing abducted dorsally, and the fifth (i.e., fifth of eight) rib is located (typically at the caudal extremity of the scapula; Fig. 11-52). A skin incision is made over the rib from the scapula to the level of the uncinate process. The incision is continued down onto the rib. The lung tissue may be visualized on either side of the rib. A section of rib (0.5 cm) overlying the lung is removed and a biopsy is harvested using iris scissors from beneath the incision (Fig. 11-53). The skin alone is closed.

Liver Biopsy

If two consecutive bile acid levels are elevated in excess of 25% above the normal upper limit, liver biopsy is indicated. Hemochromatosis, amyloidosis, chronic active hepatitis, hepatic lipidosis, toxin insult (e.g., aflatoxicosis), and cirrhosis are the most common anticipated findings. With the patient in dorsal recumbency a 2- to 3-cm incision through skin and then abdominal musculature is created parallel to and 0.5 cm caudal to the caudal edge of the sternum, just to the right of the midline. The liver is often directly opposed to the ventral aspect of the sternum. The surface of the liver is visualized in an attempt to collect a representative biopsy. Two fine artery forceps are

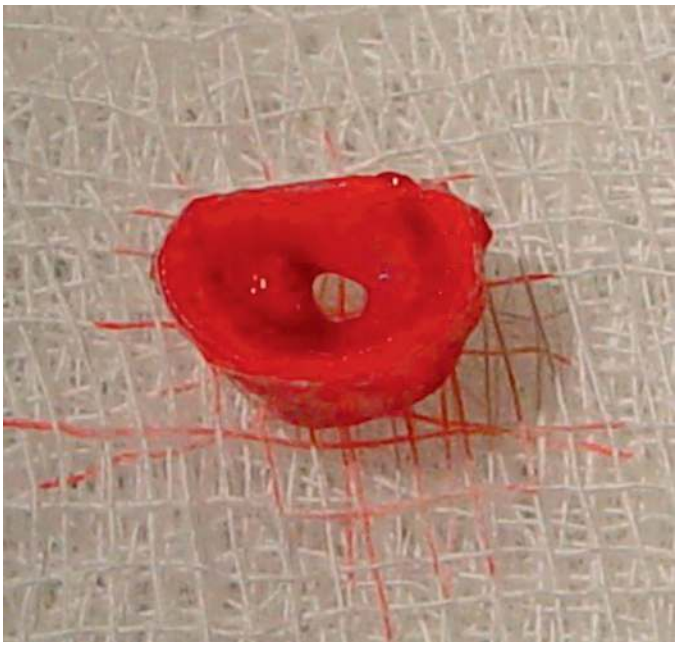


FIGURE 11-50 Tracheal stricture after successful removal.

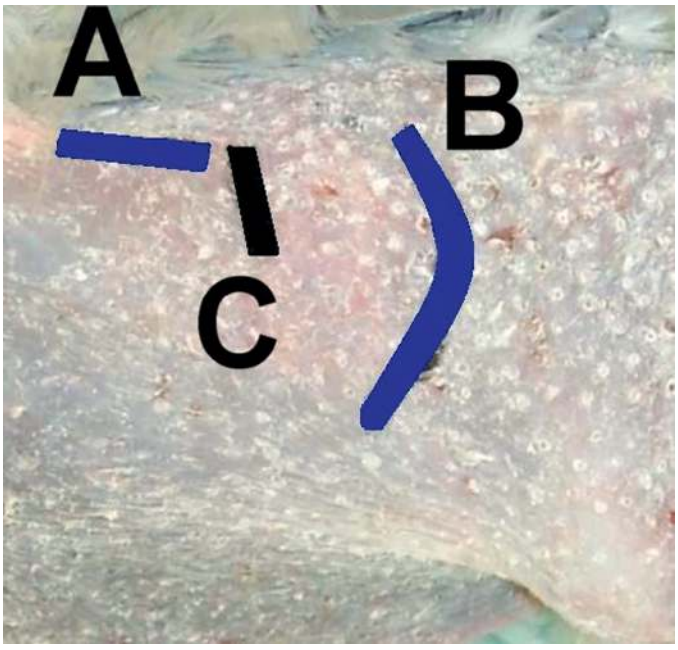


FIGURE 11-52 Lung biopsy incision site caudal to scapula *(A)*, and medial to fifth rib *(C)*, 8th rib shown *(B)*.

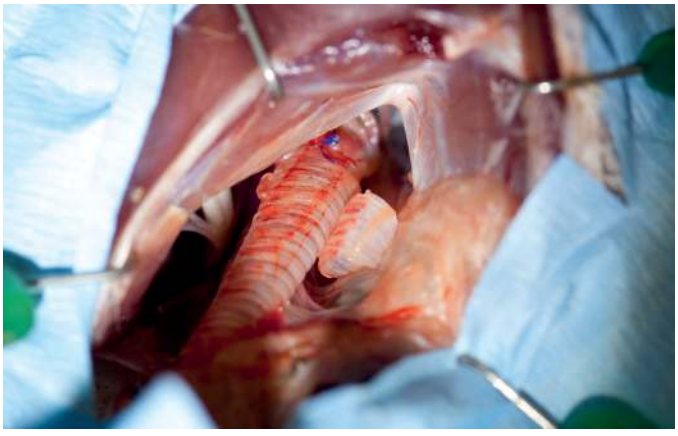


FIGURE 11-51 Completed trachectomy with length of trachea removed adjacent.

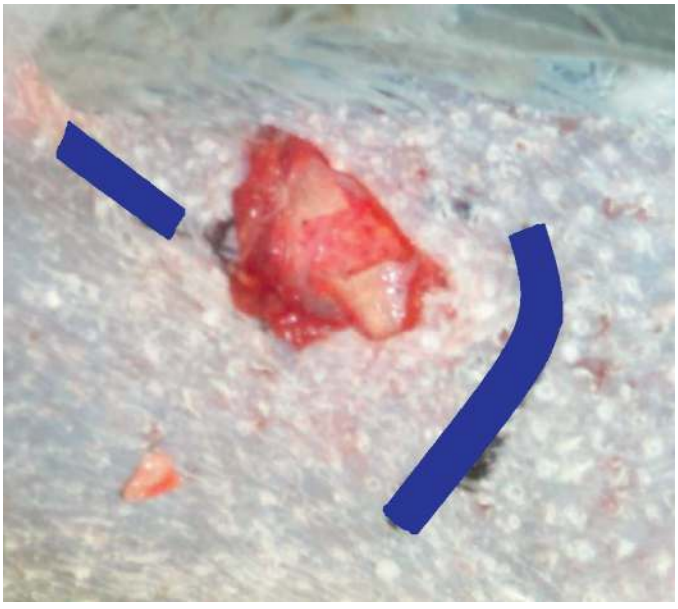


FIGURE 11-53 Lung biopsy.

triangulated to isolate a wedge of liver tissue (1 cm wide and 0.75 cm deep; Fig. 11-54). The segment of liver is removed and the forceps removed a minute later. Radiosurgery is not normally used to harvest biopsies, as cauterized tissue yields poor histopathological results.

Pancreatic Biopsy

A number of pancreatic diseases have been reported (Schmidt *et al.*, 2003; Doneley, 2010), but little research has been conducted into the clinical significance of amylase and lipase levels (Thrall *et al.*, 2012), although a fourfold increase in amylase level may be suggestive of pancreatic pathology. Clinical signs associated with avian pancreatitis include anorexia, abdominal discomfort (colic), weight loss, polyuria, polydipsia, abdominal distension, polyphagia, or pale bulky feces, although many cases are asymptomatic. The bird is anesthetized, intubated, and placed in dorsal recumbency. A small (1 to 2 cm) craniocaudal incision is made in the mid-abdominal region. Care is taken not to damage underlying viscera. The ascending and descending loops of the small intestine (within which the dorsal and ventral lobes of the pancreas are found) are readily located and exteriorized. If no lesions are grossly apparent in other areas of the pancreas, the distal most aspect of the organ is harvested. The distal pancreatic lobe should be gently elevated before biopsy collection to ensure that the arterial supply to the distal portion is not damaged during collection. The incision is closed in a routine manner.

Renal Biopsy

Renal biopsy is a frequently used technique in the diagnostic workup of kidney disease. The technique is simple and carries few risks if

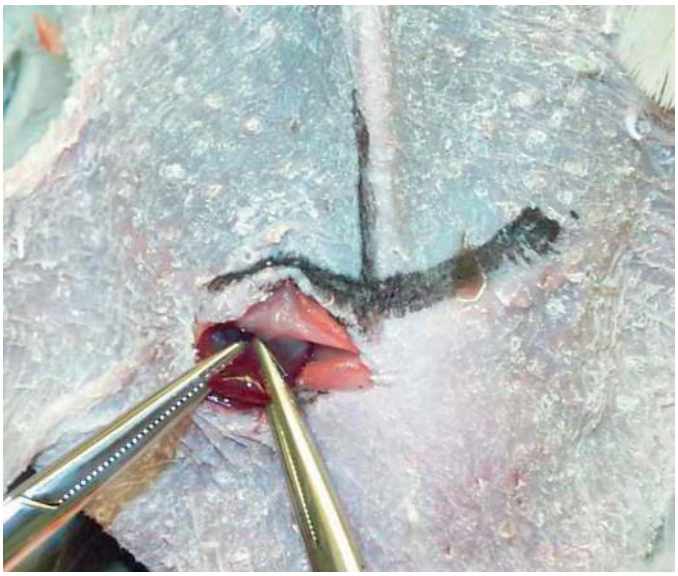


FIGURE 11-54 Liver biopsy, 1 cm left lateral of midline, 0.5 cm caudal to sternum. The right liver lobe is more caudal than the left and is easier to biopsy.

undertaken endoscopically. For further information on biopsies, see the section on Biopsies in Chapter 6.

DEVOICING BIRDS

This procedure is considered to be a mutilation by the Royal College of Veterinary Surgeons and quite rightly is illegal in the UK. Even in countries where it is not prohibited, it is a risky procedure with an uncertain short- and long-term outcome.

POSTSURGICAL CARE

Postsurgical care greatly effects the outcome of the procedure. Prevention of self-trauma; a rapid recovery; sufficient analgesia, fluid, thermal and nutritional support; and minimization of stress are vital.

REFERENCES

Antinoff N: It isn't always PDD: Three cases with proventricular enlargement. *Proceedings of the Annual Conference Association of Avian Veterinarians*, Denver, pp 35–37, 2001.

De Voe RS, Trogdon M, Flammer K: Diet modification and L-carnitine supplementation in lipomatous budgerigars. *Proceedings Association of Avian Veterinarians*, Denver, pp 161–163, 2003.

Doneley B: Disorders of the cloacal. In *Avian medicine and surgery in practice. Companion and aviary birds*, Boca Raton, FL, 2010, CRC Press.

Fillipich LJ: Tumor control in birds, *Semin Avian Exot Pet Med* 13(1):25–43, 2004.

Forbes NA: Review of 15 per cloaca prolapses in birds. *Proceedings of the ICARE Conference*, pp 119–121, 2013.

Forbes NA, Cooper JE, Higgins RJ: Neoplasms of Birds of Prey. In Lumeij JT, Remple JD, Redig PT, et al, editors: *Raptor biomedicine III, including bibliography of diseases of birds of prey*, Lake Worth, FL, 2000, Zoological Education Network.

Gregory C, Latimer K, Campagnoli RP, et al: Histologic evaluation of the crop for diagnosis of proventricular dilatation syndrome in psittacine birds, *J Vet Diagn Invest* 8(1):76–80, 1996.

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.

Harcourt-Brown NH: Tendon repair in the pelvic limb of birds of prey Part II. Surgical techniques. In *Raptor biomedicine III*. In Lumeij JT, Remple JD, Redig PT, et al, editors: *Raptor biomedicine III, including bibliography of diseases of birds of prey*, Lake Worth, FL, 2000, Zoological Education Network.

Huynh M, Sabater M, Brandão J, et al: Use of an esophagostomy tube as a method of nutritional management in raptors: a case series, *J Avian Med Surg* 28(1):24–30, 2014.

Monks D, Zsivanovits P, Cooper J, Forbes NA: Successful treatment of tracheal xanthogranulomatosis in a red-tailed hawk (*Buteo jamaicensis*) by tracheal resection and anastomosis, *J Avian Med Surg* 20(4):247–252, 2006.

Schmidt RE, Reavill DR, Phalen DN: *Pathology of pet and aviary birds*, Ames, IA, 2003, Iowa State Press.

Scott Echols M: Surgery of the avian reproductive tract, *Semin Avian Exot Pet Med* 11(4):177–195, 2002.

Scott Echols M: Wound care and management. *North America Veterinary Community Proceedings*, pp 1655–1657, 2008.

Thrall MA, Weiser G, Allison R, et al: *Veterinary hematology and clinical chemistry*, Hoboken, NJ, 2012, Wiley-Blackwell.

Van Sant F: The hazards of non-food item ingestion. *Proceedings of the Annual Conference Association of Avian Veterinarians*, pp 72–77, 2001.

12

Orthopedic Surgery

MANAGEMENT OF ORTHOPEDIC ISSUES IN BIRDS

Patrick T. Redig, Julia Ponder

The objectives in avian fracture management are to stabilize the fracture, maintain length and rotational alignment of the bone during healing, and promote rapid recovery. While retention of bone length and angular and rotational alignment of the limb is sought, exact anatomical reduction of bone fragments may incur undue soft tissue damage. Rapid recovery is enhanced by reduction of morbidity associated with fixator installation, protection of soft tissue, and overall patient management.

The desired characteristics of orthopedic appliances include rigidity, versatility, efficacy, and lightness of weight. There are several design and functional objectives that a fixation device must be able to meet:

- Stabilize the forces that apply tension, compression, torsion, shearing, and bending to the bone. With oblique and comminuted fractures, protection against collapse is also essential.
- Allow weight bearing and range of motion activity without damage to the limb, especially joints, or adjacent body parts.
- Promote load sharing to the extent the fracture will allow it. Oblique fractures have no inherent ability for load sharing; hence, the fixation must be strong enough to bear the entire load applied to the bone. Transverse fractures, on the other hand, have good load-sharing capacity, and efforts to achieve good anatomical reduction are beneficial. In some cases, it may be desirable to convert an oblique fracture to a transverse one and accept minor shortening of the limb, especially in fractures of the tibiotarsus. As healing progresses, the fixator may be partially dismantled by dynamic destabilization, shifting load sharing to the healing bone and increasing the healing rate.
- With good fixation and good overall vascular condition at a fracture site, healing can often be achieved in 18 to 25 days, which is well within the life span of a fixation device. However, some fractures require more time, and the integrity of the fixator must be maintained for the duration. Loosening of pins is not an inevitable consequence and can be avoided by proper placement, the use of positive-profile threaded pins, and solid overall construct design.

THE AVIAN SKELETON AND FRACTURE MANAGEMENT

The fixator is one half of the equation in fracture repair; the patient is the other. The avian skeleton is fundamentally and significantly different from the mammalian skeleton and presents unique challenges to fracture fixation. The bone cortices are thin and brittle but very strong. Their strength is derived from their monocoque (i.e., egg shell-like) anatomy. There is also less holding power for fixation hardware. It is essential that fixation pins obtain solid purchase in two cortices. There is a paucity of soft tissue over many of the long bones; thus, comminuted bone fragments may be displaced and are prone to lose their vascular supply. Additionally, the skin is very thin and bone fragments exteriorize easily. Bone so exposed is most often not viable and formation of a sequestrum in 2 to 3 weeks is highly likely if dead bone is not removed. There is a dearth of cancellous bone in the avian skeleton and established methods for bone grafting have not been proven clinically. Last, regarding pelvic limb fractures, given the bipedal locomotion of birds, a unilateral fracture puts tremendous strain on the contralateral leg that must be managed. Successful fracture management in birds requires not only application of good fixation but also the management of many of these other challenges that are unique to birds. Across a wide range of avian long bone fractures and many thousands of patients, a device known as the external skeletal fixator (ESF)-intramedullary (IM) pin tie-in, IM pinning bonded to an ESF, has proven to be effective in meeting the characteristics and challenges of avian skeletal fixation (see below; Redig, 2000).

MATERIALS

The materials and devices used in avian fracture repair are IM pins, both Steinmann pins and K-wires (Kirschner wires less than 1.6 mm [0.062 inch] in diameter) joined together with various configurations of ESFs using conventional bars and clamps, a tubular bar (FESSA) system, a polymer connecting bar (methacrylate or epoxy) system, or locking bone plates comprising an ESF bar. Bone plating has received scant attention in avian species for a variety of reasons—morbidity associated with placement, the need for follow-up surgery to remove plates in wild birds destined for release, and lack of demonstrated efficacy (Christen *et al.*, 2005)—however, research in their use continues (Gull *et al.*, 2012). Last, ESF devices are relatively inexpensive and have minimum equipment requirements for installation, which promotes their widespread use.

For ESF devices, partially threaded, positive-profile thread pins[a] designed specifically for birds and small exotics are preferred over unthreaded pins. These offer a greater holding power in the thin cortices. These pins also have a roughened surface on the end opposite the threading, which is designed to engage the matrix of acrylic bonding materials. Negative-profile K-wires or Steinman pins may be used as secondary anchor pins in a complex construct, but they should not be used in a high-stress application as they are likely to break at the junction of the threads with the pin. Special "center-threaded" pins with positive-profile threads for type II fixators are also available in larger sizes.

Resinous materials such as methacrylate and epoxies are one category of material used to connect the pins of an ESF. Among acrylics, dental acrylic[b] or horse-hoof repair products[c,d] are suitable. The acrylic can be molded over the pins after curing to a dough stage or loaded in a syringe during the liquid stage of curing and injected into a molding material such as a Penrose drain or plastic drinking straw.

The construct that has proven to be strong, versatile, and effective using these various materials is the ESF–IM pin tie-in fixator (TIF; also

called a hybrid fixator); it has yielded exceptional results in a variety of fractures involving most avian long bones (Redig, 2000). It consists of an IM pin that fills approximately 60% to 75% of the medullary cavity and at least two ESF pins placed at the proximal and distal ends of the affected bone (Fig. 12-1). The IM pin is bent at 90 degrees at its exit point and rotated into the same plane as the ESF pins. A piece of thin-walled latex tubing (e.g., Penrose drain) approximately the diameter of the bone being repaired is placed onto the pins as a mold. The mold is filled with acrylic injected with a syringe, which, after curing, binds all of the pins together. This technique was developed to provide longitudinal and rotational stability for humeral fractures without resorting to additional external coaptation such as a figure eight bandage in the postoperative period and has been extended to other long bones. It has been subjected to biomechanical testing, and

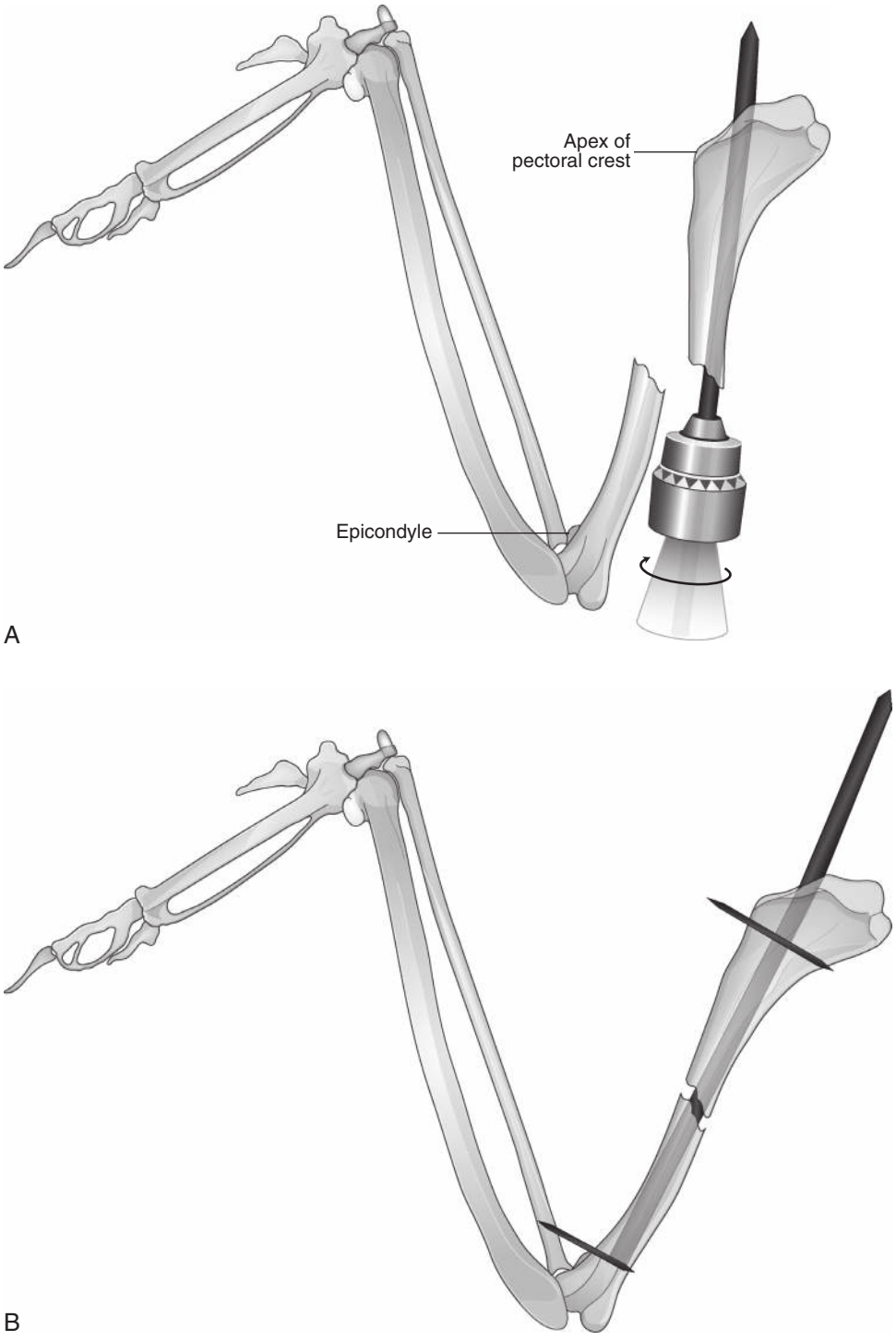


FIGURE 12-1 **(A),** Placement of an intramedullary (IM) pin by retrograde insertion into the proximal fragment of the humerus. **(B),** Fracture reduction and normograde driving of the IM pin into the distal humeral fragment and the placement of the external skeletal fixator (ESF) pins at proximal and distal locations.

Continued

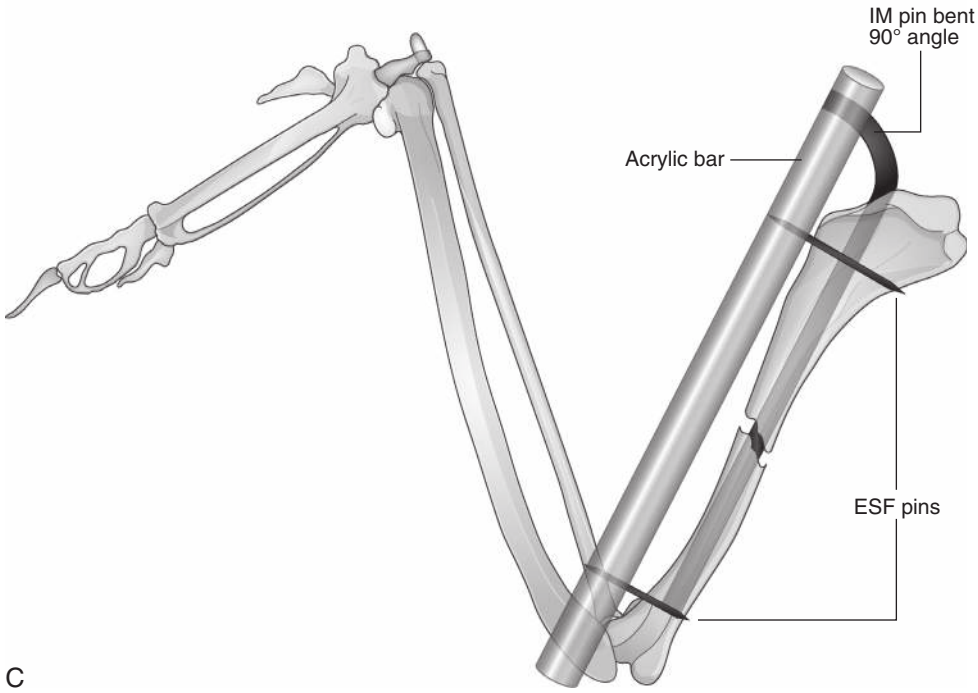


FIGURE 12-1, cont'd (C), Completed tie-in fixator (TIF). Note 90 degree bend in IM pin and inclusion of it along with both ESF pins into a cylindrical, acrylic connecting bar.

results indicate that in both basic and enhanced (additional ESF pins) conformations, it exceeds the strength requirements for stabilizing avian bones (Van Wettere, 2009a,b).

This section is organized according to the layout of the bones in the appendicular skeleton, commencing with the pectoral limb. Surgical approaches, fixation techniques, and preoperative and postoperative radiographs of actual cases are used to present a broad but detailed overview.

METHODS OF FIXATION FOR THE HUMERUS

General Considerations

The humerus can be divided into three zones for evaluation of fractures and selection of fixation devices. The *proximal zone* extends from the subcondylar region near the shoulder joint to the distal extension of the pectoral crest. The *diaphyseal zone* extends from the distal end of the pectoral crest to the apex of the distal diaphyseal curvature proximal to the condyles. The *distal zone* involves the curved subcondylar portion of the distal humerus.

Application of the Tie-In Fixator to Diaphyseal Fractures of the Humerus: the Archetypical Construct

Diaphyseal fractures of the humerus are repaired readily unless there is excessive comminution or extensive exteriorization of bone fragments. Most diaphyseal fractures are oblique, with the proximal fragment tending to project through the dorsal surface of the wing and the distal fragment either projecting through the skin on the ventral surface or pulled up against the radius and ulna because of the contraction of the carpal extensor muscles. Since there is no weight bearing on this limb, oblique fragments, despite their lack of load sharing, are readily held in stable configuration. The radial nerve crosses from caudal to cranial in the midshaft and must be preserved during the dorsal surgical approach. Manipulation of this nerve is a constant feature of managing diaphyseal fractures. The triceps tendon courses distally on the caudal aspect of the bone, wrapping around the distal condyle and attaching to the olecranon. The triceps muscle is very strong and the bending moment it applies to the humerus is a force that must be counteracted by the fixation. The general steps involved in the placement of the TIF on a mid-diaphyseal humeral fracture are illustrated in Figure 12-1 and demonstrated in the series of in-surgery images and radiographs in Figure 12-2. Postoperatively, it is essential that passive range of motion physical therapy and stretching exercises of the patagium are conducted to prevent patagial contraction. This should be instituted beginning no later than the third day after surgery. Most diaphyseal humeral fractures will heal in 21 to 26 days.

A Method of Fixation for Distal and Subcondylar Humeral Fractures: the Cross-Pin Tie-In Fixator

The cross-pin method of repairing distal fractures is a variation of the TIF; however, the placement of the K-wires is technically different from the placement of a conventional IM pin. Part of the stability achieved is from lateral pressure of the pins on the walls of the medullary cavity of the bone. The procedure is performed by driving two small K-wires (0.045 inch, typical in birds weighing 500 to 1200 g, 0.062 inch in 1200- to 2000-g patients) retrograde from the fracture site, exiting the condyle opposite the side of the marrow cavity from which they are introduced and at a shallow angle (15 to 20 degrees) to the long axis of the bone. They should be retrograded until the ends are flush with or just slightly beyond the edge of the fracture site (Fig. 12-3, *A*). The fracture is reduced and the pins are driven alternatingly into the proximal fragment, 1 to 2 cm at a time until well into the proximal fragment (Fig. 12-3, *B*). The most common mistake is to insert one pin too far in advance of the other. External fixation pins are inserted in the proximal and distal fragments as described above. An acrylic bar is

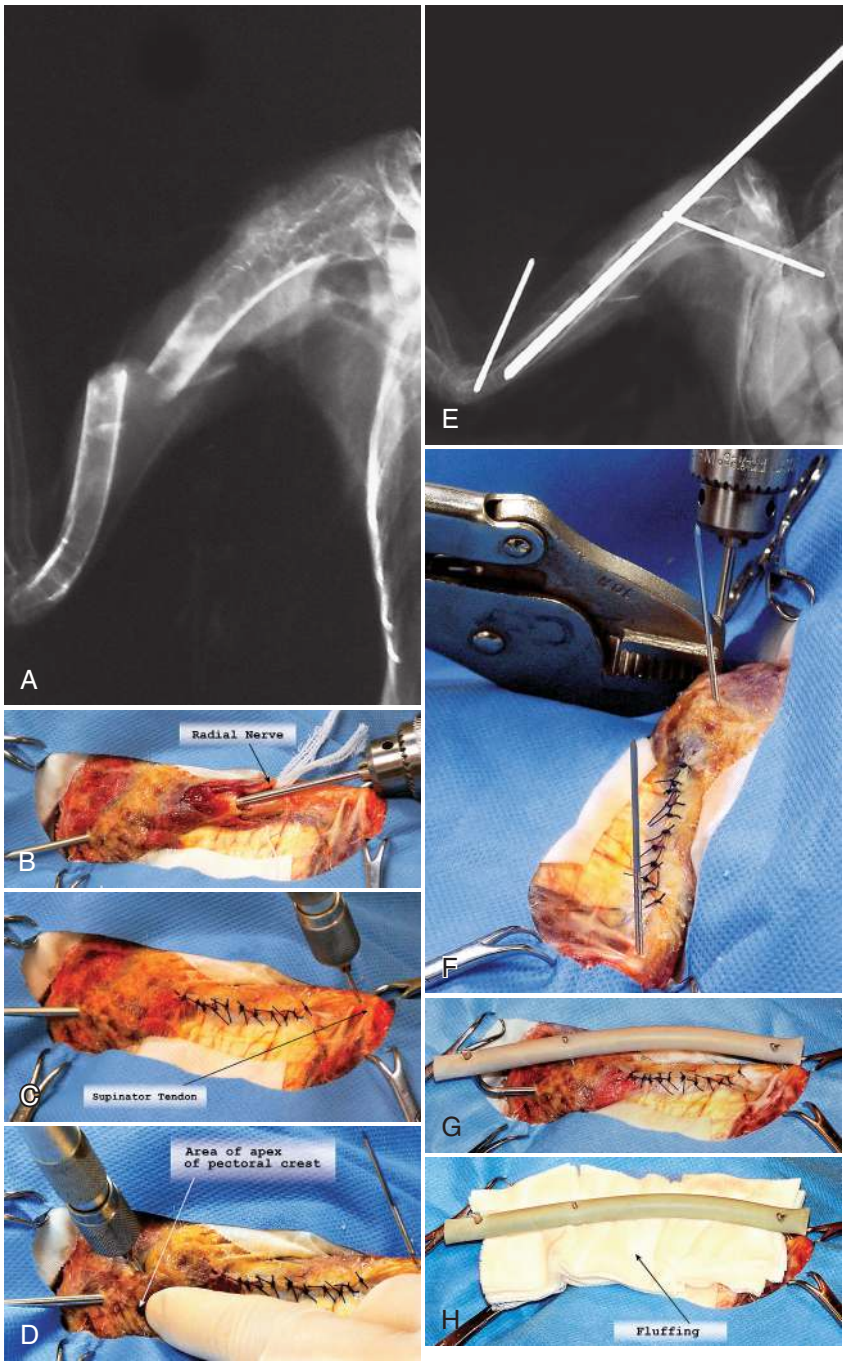


FIGURE 12-2 Fixation of a mid-diaphyseal humeral fracture using a tie-in fixator (TIF). **(A),** Preoperative radiograph. This case consisted of a closed, transverse midshaft diaphyseal fracture in a short-eared owl (*Asio otus*). **(B),** Introduction of the intramedullary (IM) pin. The pin was inserted in retrograde fashion into the proximal fragment at the fracture site. Note the retraction of the radial nerve with a gauze loop. **(C),** Placement of the external skeletal fixator (ESF) pins. Positive-profile threaded pins (Imex Veterinary[a] Inc., Longview, TX) were used. The first was placed in the diaphysis proximal to the condyles at the level of the epicondyle to which the tendon of origin of the supinator and common digital extensor attaches (see also Fig. 12-1, *B*). **(D),** The second ESF pin was placed in the diaphysis of the proximal humerus at a point adjacent to the apex of the curvature of the pectoral crest (see Fig. 12-1, *B*), a point that can be palpated for reference. To protect the soft tissues from damage by the pin threads, a tissue tunnel was created with a hemostat and the surrounding muscles were retracted. **(E),** Intraoperative image taken at the point where the IM pin has been placed in the proximal and distal fragments and the two 1.15-mm (0.045-inch) diameter pins. ESF acrylic interface pins have been installed. **(F),** Bending of the IM pin. To tie the IM pin to the ESF, the end of the IM pin was bent 90 degrees. It is imperative to stabilize the pin with locking pliers to prevent transfer of bending forces to the bone. **(G),** Fixator bar attachment. Application of the fixator bar is accomplished by forcing a piece of thin-walled rubber tubing (e.g., Penrose drain 10-mm [3/8-inch] diameter) over the pins. It is then filled with an acrylic horse-hoof repair material injected through the nozzle of an irrigating syringe. After the acrylic has cured, excess material is trimmed away. **(H),** "Fluffing" (sterile 2 × 2 gauze pads) was placed between the fixator and the skin to absorb exuded fluids and reduce postoperative swelling and movement of soft tissues. It was changed 18 to 24 hours postoperatively. Note use of acrylic for the fixator bar in this instance.

Continued

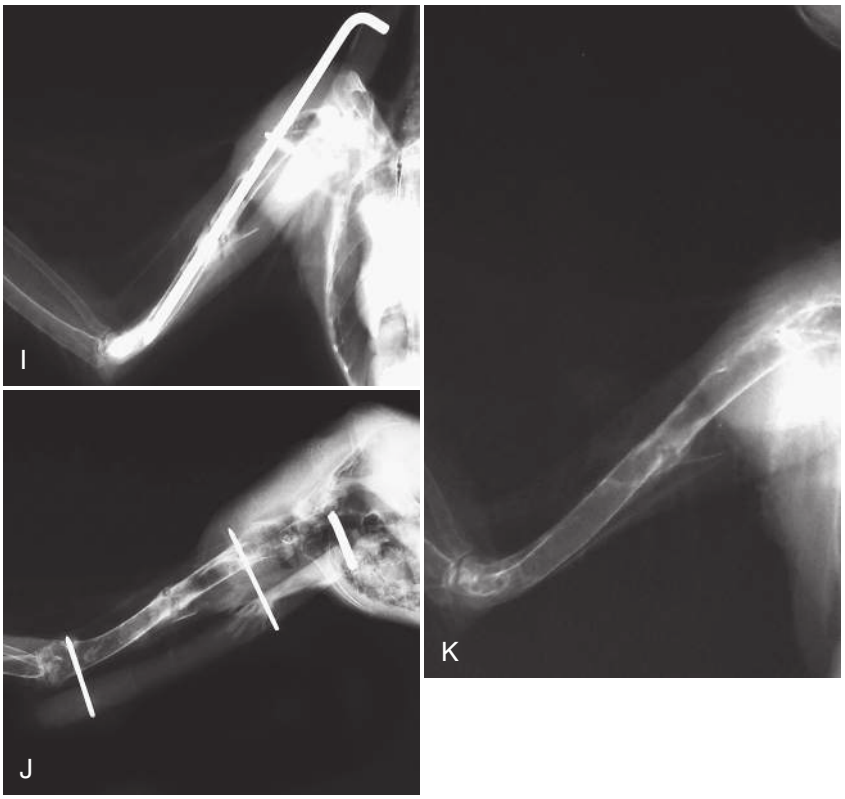


FIGURE 12-2, cont'd **(I),** Postoperative radiograph after completion of TIF. We chose to use only proximal and distal ESF pins in this case to reduce morbidity in the vicinity of the highly traumatized soft tissues adjacent to the fracture site. **(J),** Radiograph, 21 days postoperatively. The IM pin has been removed but the ESF pins and connecting bar were left in place for an additional week. **(K),** Radiograph after removal of fixator; healing was complete in 30 days.

installed to tie all of the pins together. Radiographs taken from a case illustrating this kind of fracture management are shown in Figure 12-3, *C, D*.

Note: In all instances of repair of pectoral limb bones it is essential to fold the wing against the body in the normal perching position during the curing process of the acrylic fixator bar or other affixing of the connector bar to achieve proper rotational alignment.

A Method for Fixation of Proximal Zone Humeral Fractures: Tension Band (Tie-In Fixator)

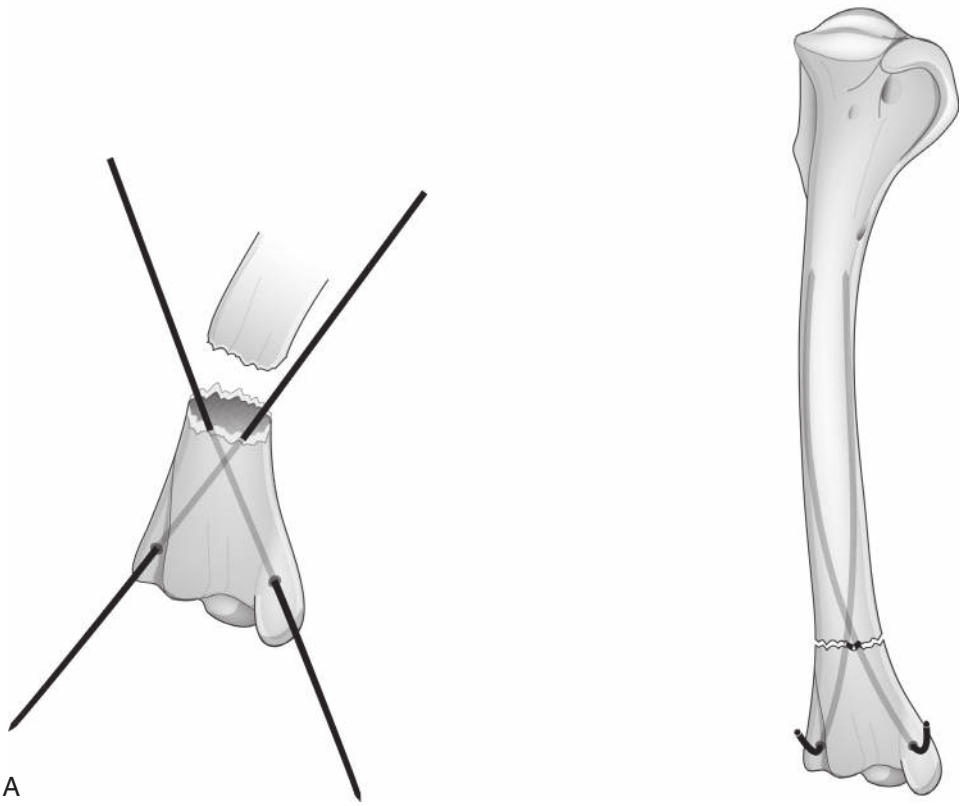
Fractures that occur in the proximal zone are most often transverse. Very proximal fractures, stabilized by the heavy pectoral muscle, may be managed with coaptive bandaging if the fragments are well aligned. All other humeral fractures will require fixation to heal. A complicating factor for fixation is the curvature of this segment of bone. With a short, curved proximal fragment, it is difficult to gain sufficient purchase on the proximal fragment with an IM pin that would be used in a conventional TIF and there is insufficient bone to accept an ESF pin proximal to the fracture site.

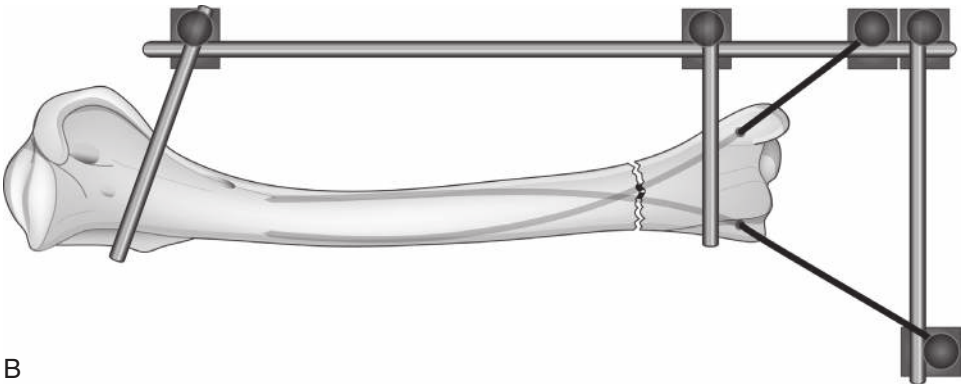
An alternative approach is to use a variation of the TIF using a tension band (Fig. 12-4). It involves exposure of the proximal humerus from the dorsal aspect (Fig. 12-2, *D*) and placement of two small-diameter K-wires in cross-pin fashion at the fracture site in the proximal fragment (0.045 inch, typical in a 1-kg patient). These pins are driven retrograde and exit on either side of the pectoral crest (Fig. 12-4, *E*). The fracture is reduced and the pins are driven into the distal fragment, resulting in pressure on the sides of the bone. A cerclage wire (the only place where cerclage wire will be recommended) is passed through a hole drilled transversely in the distal fragment about one bone diameter distal to the fracture site and another drilled transversely through the proximal fragment just caudal to the exit point of the K-wires (Fig. 12-4, *F*). Periosteal elevation of the deltoid muscle and the pectoral muscle from the humerus may be helpful in accomplishing this. The wire is threaded between the fragments in a figure eight pattern and tightened, completing a tension band. The desired result is alignment of the fragments so that the curvature of the proximal humerus is retained. The K-wires are left projecting at the head of the humerus for future retrieval. (In birds less than 300 g it is sufficient to simply place the K-wires.) The muscle is sutured back to the pectoral crest and skin is closed over the top. The wing is taped to the body postoperatively for 10 days. For additional support in larger birds, two ESF pins may be placed in the distal fragment and the K-wires protruding from the proximal fragment are bent at 90 degrees and a methacrylate bar is used to connect all elements together in a TIF. The wing should only need to be bandaged to the body for about 1 week when this type of support is provided.

METHODS OF FIXATION FOR DIAPHYSEAL FOREARM FRACTURES

General Considerations

There are many factors to consider when selecting a management approach for forearm fractures ranging from coaptation with a figure





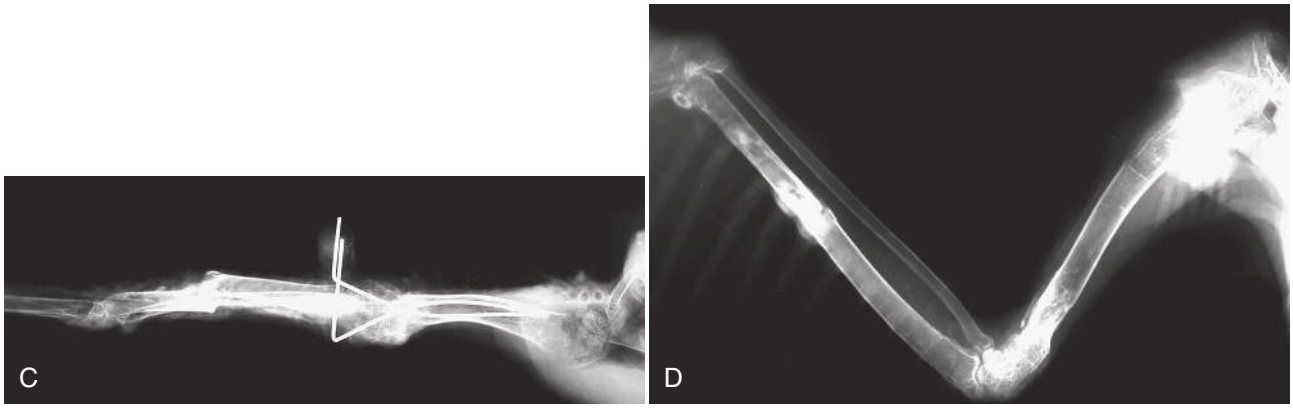
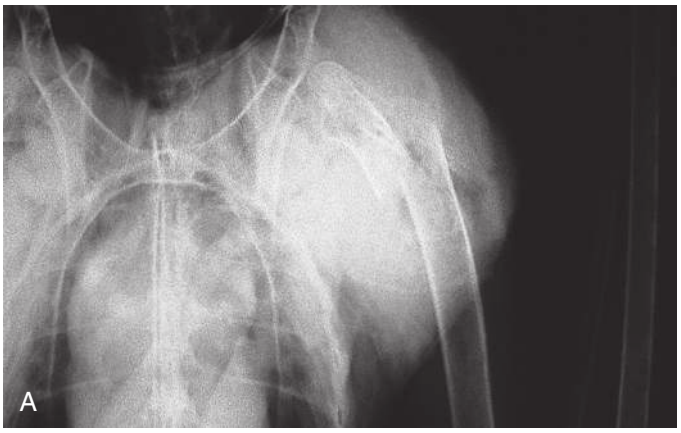


FIGURE 12-3 Cross-pin tie-in fixator (TIF) method for distal humeral and femoral fractures. **(A),** Placement of small-diameter pins in the fragments of a distal humeral fracture where there is otherwise insufficient bone to gain purchase on the distal fragment with a conventional intramedullary pin. **(B),** The cross-pin TIF as it would be applied to a distal humeral fracture. **(C),** Posterior–anterior view. In this postoperative radiograph of a great horned owl, the relationship of the cross-pins to the condyles and the fracture site and the way the pins were incorporated into a TIF are shown. **(D),** Ventrodorsal radiograph, 4 weeks postoperatively and after removal of the fixator; the completely healed fracture is shown.



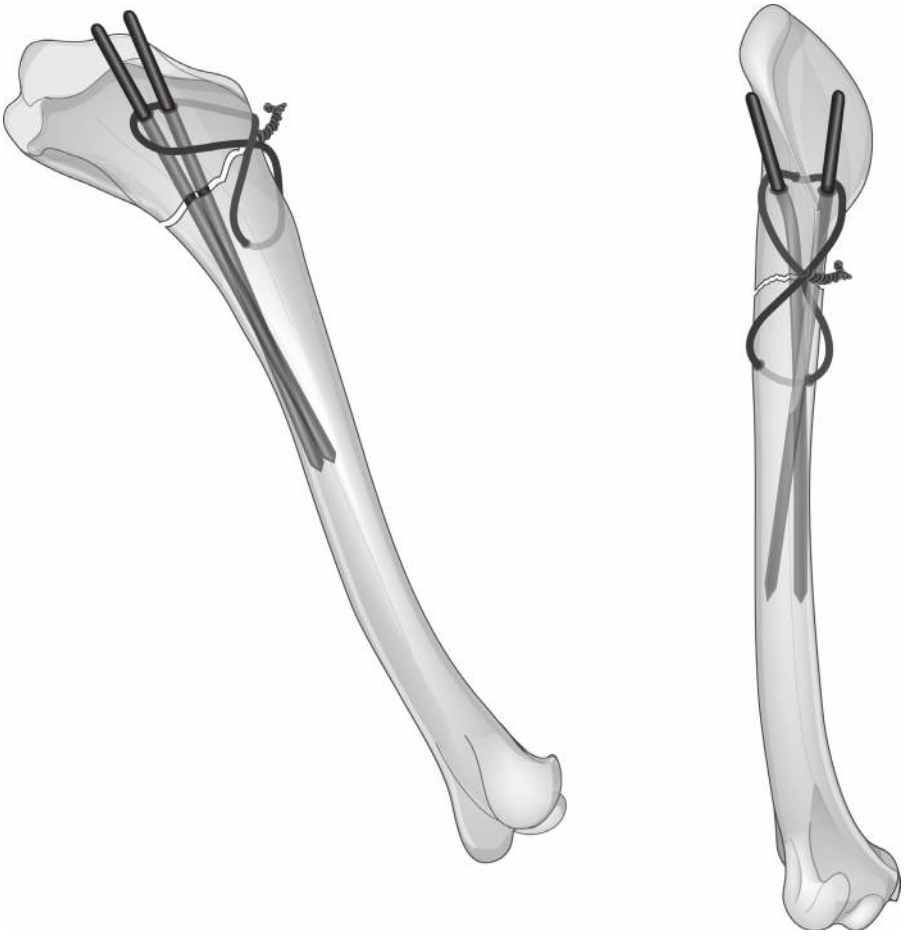


FIGURE 12-4 Treatment of a fracture of the proximal humerus. **(A),** Preoperative radiograph of a fracture in the proximal segment of the humerus of a bald eagle (*Haliaeetus leucocephalus*). Here the transverse fracture was accompanied by a partial avulsion of the pectoral crest. **(B),** Illustration of the tension band method of fixation for **(A)**. Following surgical exposure of the proximal fragment, two 1.6-mm (0.062-inch) diameter Kirschner wires (K-wires) were driven retrograde in cross-pin fashion from sites dorsal and ventral to the pectoral crest in the proximal fragment, the fracture was reduced, and the pins were driven normograde into the distal fragment. Transverse holes were drilled in the distal fragment one bone diameter distal to the fracture site and another through the blade of the pectoral crest. Cerclage wire was passed in figure eight fashion through the holes and behind the K-wires.

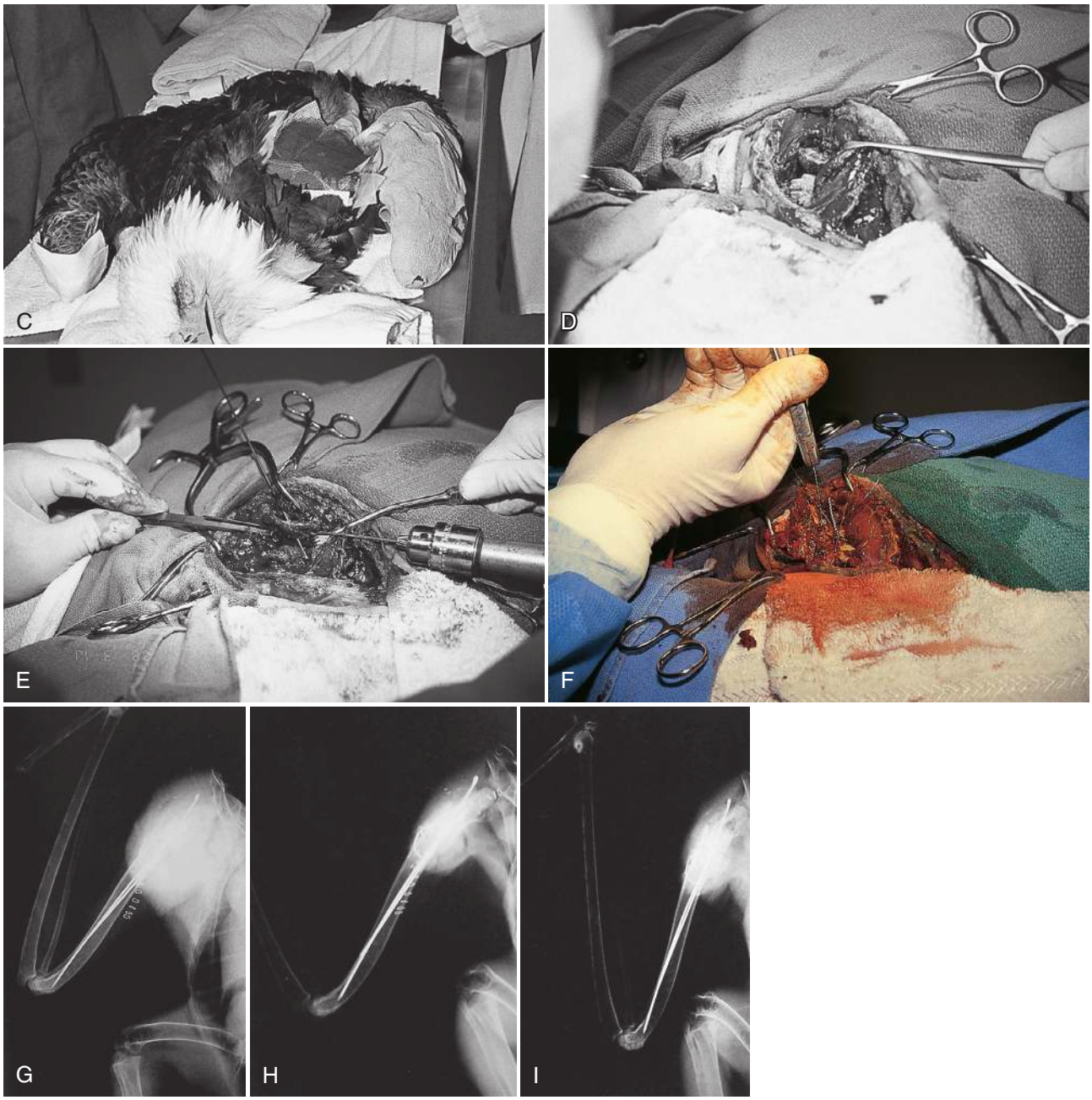


FIGURE 12-4, cont'd **(C),** Preparation of the operative site for a dorsal approach to the humerus. Feathers were plucked from a site craniad to the shoulder to just distal to the midshaft of the humerus. **(D),** Exposure of the fracture site. The belly of the deltoid muscle was split longitudinally and reflected, leaving its attachment to the diaphysis of the humerus intact while allowing exposure to the blade of the pectoral crest. Ventral to the crest, the major pectoral muscle was elevated. **(E),** Placement of K-wires. Two 1.6-mm (0.062-inch) K-wires were retrograded into the proximal fragment, crossing in the intramedullary space and exiting on either side of the pectoral crest. Following this, the fracture was reduced and the pins were driven into the distal fragment. **(F),** Placement and tightening of the cerclage wire in figure eight fashion. The large tightened wire is the figure eight cerclage. The smaller wire being tightened is one of several used to reattach an avulsed portion of the pectoral crest. **(G),** Postoperative radiograph. Normal anatomical realignment has been achieved, preserving the curvature of the proximal humerus. **(H),** Radiograph, 23 days postoperatively. Alignment and approximation of fragment satisfactory, and callus forming well. Passive range of motion physical therapy was started on postoperative day 2. **(I),** Radiograph, 31 days postoperatively. Healing was complete. One K-wire was removed, the other was not retrievable. The cerclage wire was a permanent implant.

eight bandage and taping to the body to full fixation. These considerations include whether one or both bones (radius and/or ulna) are fractured and the location of the fracture(s). Most forearm fractures may be repaired with the TIF applied to the ulna and, if necessary, stabilization of the radius with an IM pin to avoid synostosis. Nonsurgical methods, such as figure eight bandaging (Fig. 12-5), may be a good option in some cases. For instance, well-aligned, midshaft fractures of the ulna where the radius is intact and comminuted proximal ulna fractures (where fixation cannot be applied) may respond well to coaptation. Similarly, coaptation may be the only viable option for proximal radial fractures because of the shortness of the proximal fragment and difficulty in accessing the fracture site.

The most frequently occurring complication of forearm fractures is the formation of a bony bridge or synostosis between the two bones. It is a direct result of insufficient stabilization of the fractures. It occurs most frequently with unstabilized radial fractures or in cases where both bones are fractured. Because of the separation of the bones in the proximal region, synostoses are less likely to occur compared with distal fractures where the two bones are in closer proximity.

Important Aspects of Pin Placement for Fixators Applied to the Radius and Ulna

The method and location of the placement of IM pins in the radius and ulna is critical in minimizing joint morbidity. The radius can be repaired by retrograde placement of the IM pin with the pin exiting at the distal end. The anatomy of the carpal joint allows this without undue morbidity. The ulna, however, must be pinned in normograde fashion with the pin inserted in the proximal end just distal to the point of attachment of the triceps tendon (Fig. 12-6, *A*). Retrograde placement of the IM pin in the proximal ulnar fragment is contraindicated as the pin exits the ulna at the olecranon and will damage the joint, the triceps tendon, or both. In addition, a pin exiting at the olecranon will cause joint damage from movement associated with controlled physical therapy during the postoperative period. Specific recommendations for fixation of various types and locations of forearm fractures are detailed below.

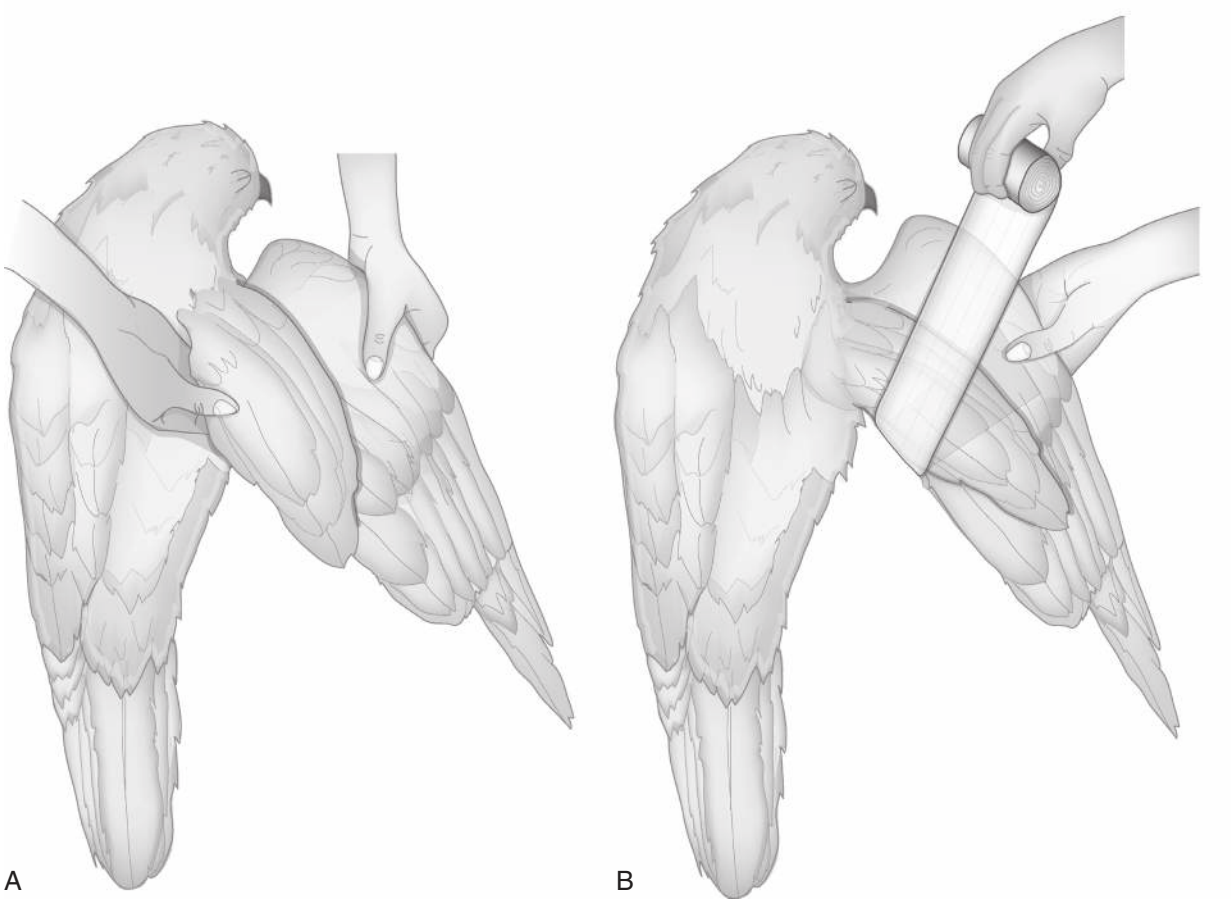


FIGURE 12-5 The figure eight bandage is most easily applied with the patient under anesthesia and in sternal recumbency. The affected wing is partially extended and the tertiary coverts are identified and included under the bandage **(A)**. Conforming gauze (e.g., Johnson & Johnson[e] Kling gauze) is used for the first layer of the bandage. The free end of the gauze is held in place with the fingers of one hand on the ventral side of the wing at the leading edge **(B)** and brought from underneath the wing behind the tertiary coverts and over the dorsal surface. Four rounds of gauze are applied in this fashion, keeping the gauze evenly distributed between the elbow and the axilla.

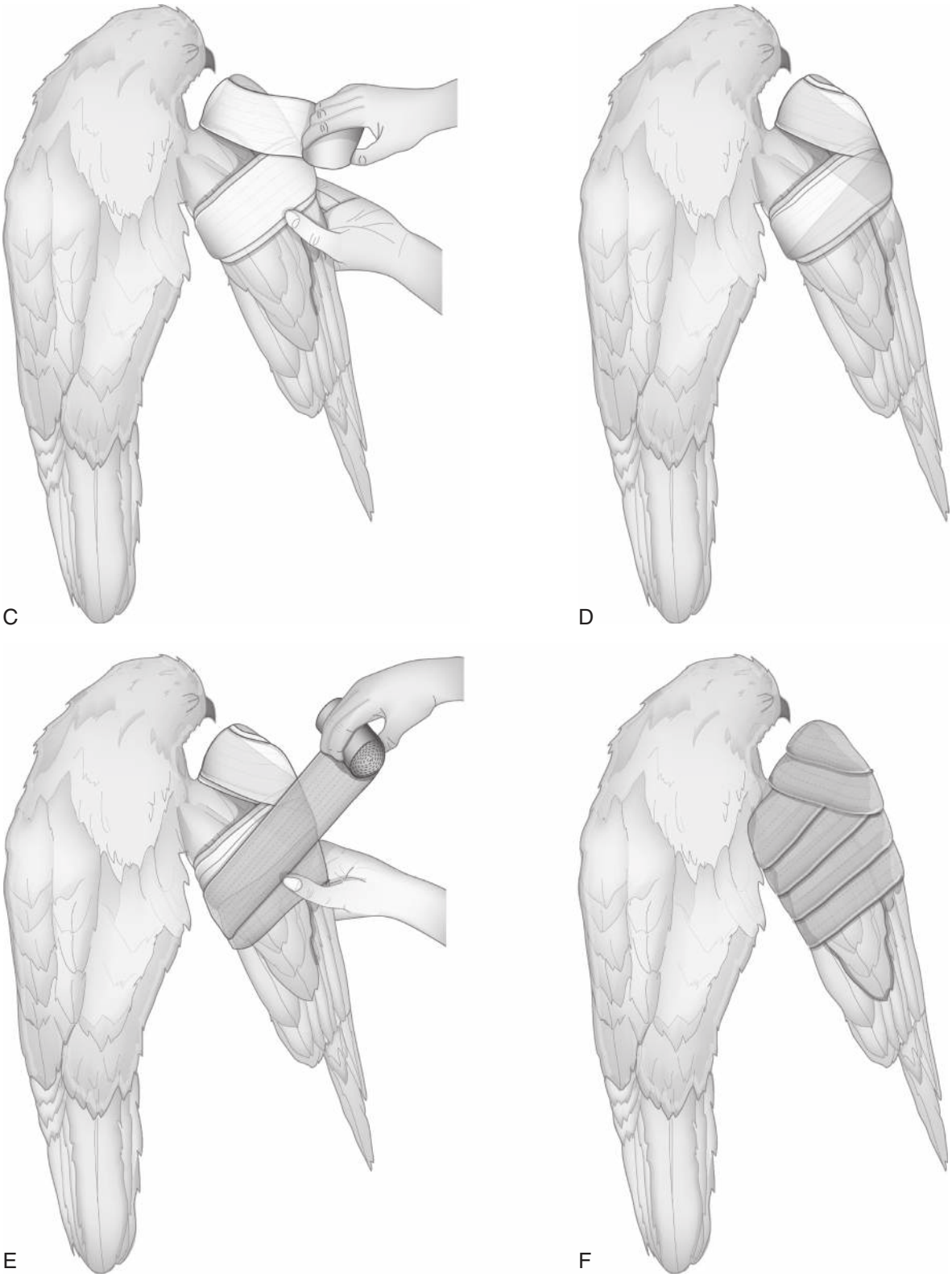
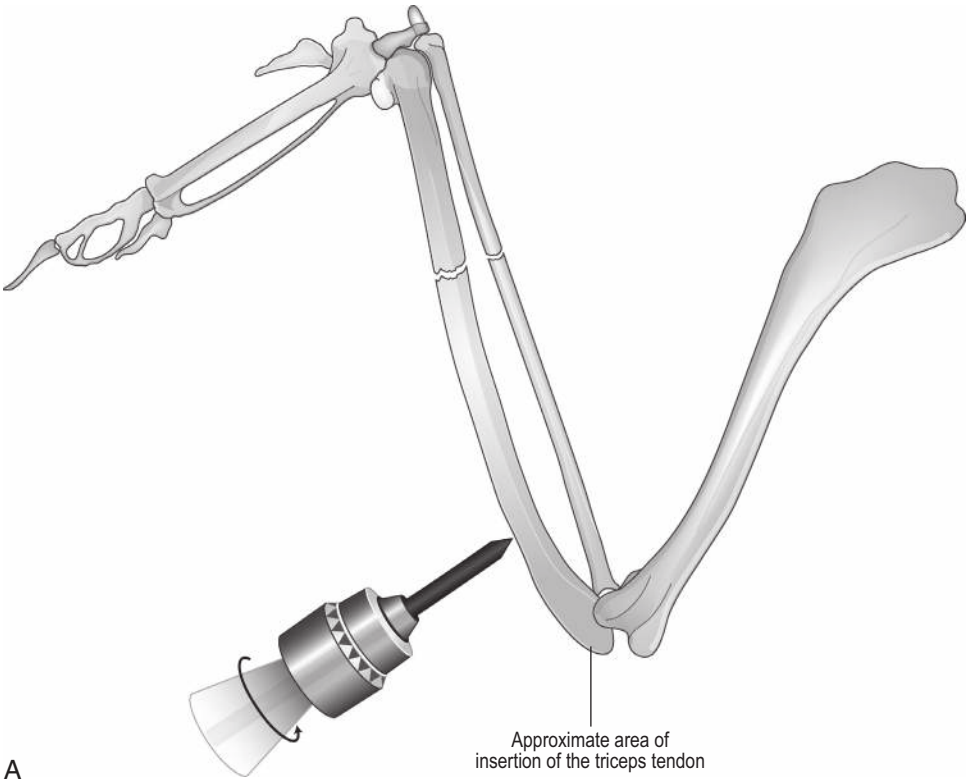
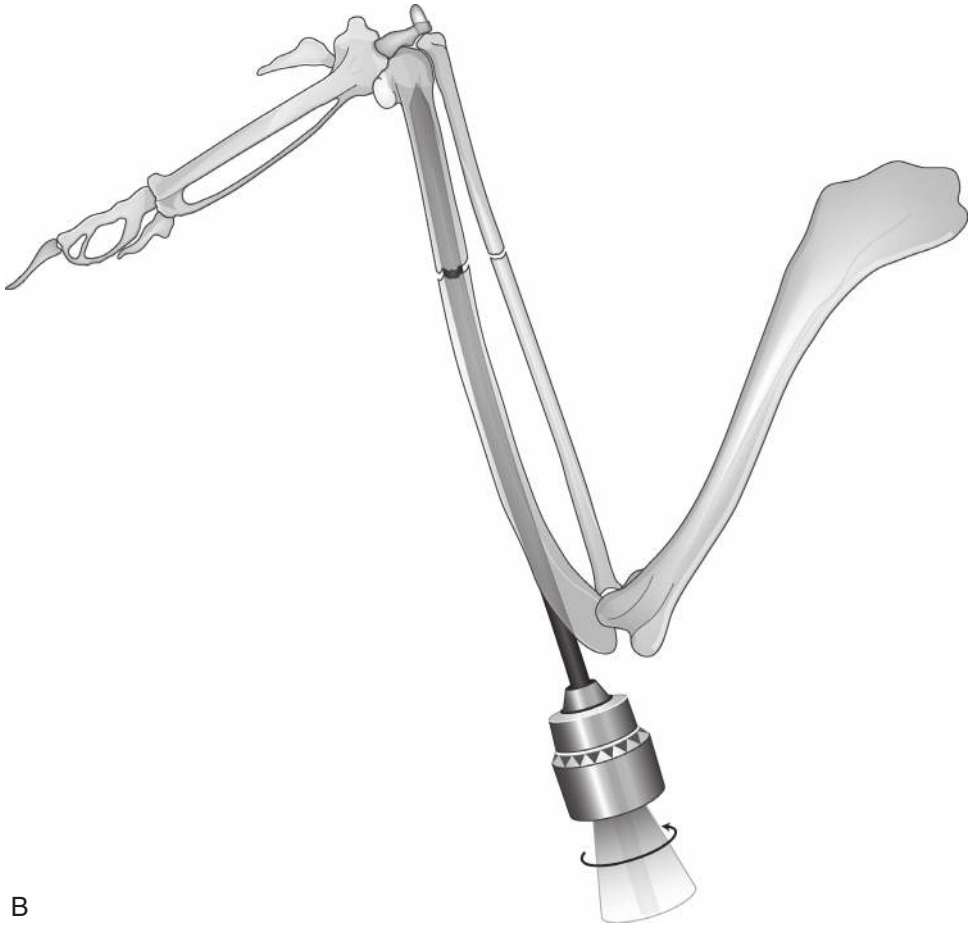


FIGURE 12-5, cont'd After this, the wrap is extended by bringing the gauze around the leading edge of the wing, anterior to the humerus **(C)**, and continuing in a figure eight until a bulky, but not tight, wrap has been applied **(D)**. An overwrap of nonadherent stretch bandage (e.g., 3M Vetrap[f]) is applied over the gauze. This should begin at the elbow like the conforming gauze wrap **(E)** and extend cranially, completing a figure eight as shown in **(F)**. When completed the bandage should stabilize the elbow and carpal joints without overflexing the carpus. The leading edge of the primary feathers should lie parallel to the secondary feathers.



A



B

FIGURE 12-6 Illustrations for normograde placement of an intramedullary (IM) pin in the ulna. The ulnar pin is introduced on the caudal aspect of the ulna between the feather follicles of the two most proximal secondary feathers and nearly perpendicular to the long axis of the bone **(A)**. An elongated hole is cut in the cortex of the bone and the angle of the pin with the bone reduced so that, as the pin penetrates the ulnar cortex, the pin is longitudinally aligned with the bone. The pin is now driven normograde through the reduced fracture and driven just short of the distal end of the bone **(B)**.

C

D

FIGURE 12-6, cont'd The distal external skeletal fixator (ESF) pin is inserted and the IM pin is driven forward gently to lodge against the ESF pin **(C)**. Conversion of the pin in the ulna to a tie-in fixator by the addition of the proximal ESF pin, bending the IM pin 90 degrees **(C)** and applying a connecting bar, yields the final results **(D)**.
Note: The circle around the bar indicates that the remainder of the acrylic bar was edited out for clarity.

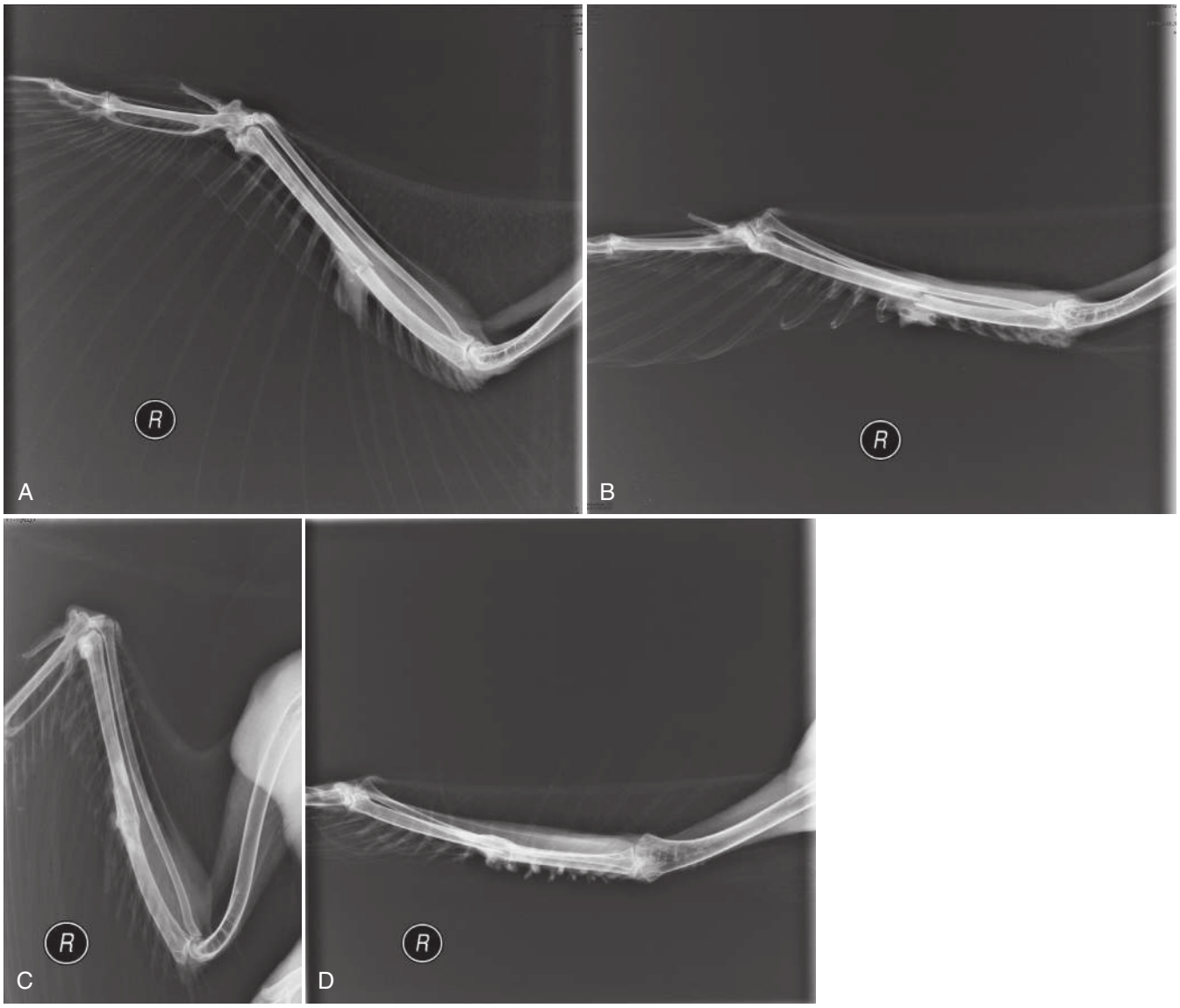


FIGURE 12-7 Radiographs of a great horned owl with a midshaft fracture of the ulna and an intact radius. Because of the acceptable alignment in both ventrodorsal (VD) and posterior–anterior (PA) planes **(A, B)**, this fracture was managed with a figure eight bandage and additional taping of the wing to the body for the first 2 weeks. The fracture healed satisfactorily in 4 weeks as can be seen in both the VD **(C)** and PA **(D)** views.

Specific Management Recommendations

Proximal Ulnar Fractures, Radius Intact or Fractured: Various Scenarios

Because of the short proximal fragments and the complexity of soft tissue elements around the elbow, fractures located in this region most often are best managed conservatively (i.e., bandaging only) or with a blend of low-impact surgery and coaptation. In some cases where the radius is intact, proximal to midshaft ulnar fractures respond well to conservative management. In all instances of radial fractures except those that are within two to three bone diameters of the proximal end, insertion of an IM pin is recommended. Similarly, for any fractures of the ulna occurring in the distal half, fixation is recommended.

1. Radius intact, ulna fractured (Fig. 12-7). When the ulnar fracture is stable (e.g., well-aligned transverse fractures) a suitable treatment option is simple coaptation with a figure eight bandage). Alignment of the ulnar fragments seen in both ventrodorsal and caudocranial radiographs is important when using this option.
2. Proximal, comminuted ulnar fracture with intact radius (Fig. 12-8). There are no viable surgical options in these cases. Careful management of the soft tissues and immobilizing the wing with a well-padded figure eight bandage has been effective. One attempt to manage such a fracture with a small plate failed because of necrosis of the traumatized soft tissue that was sutured over the plate.
3. Proximal ulnar fracture with radial fracture. If the radius is fractured and the ulnar fracture is too short to obtain purchase with fixation hardware, an IM pin in the radius with no fixation applied to the ulna (Fig. 12-9) has yielded satisfactory results; coaptation is required for 7 to 10 days postoperatively.

Note: Introduction of the IM pin into the radius can be accomplished by retrograde insertion at the fracture site or, with good technique, it may be drilled into the distal end of the radius and

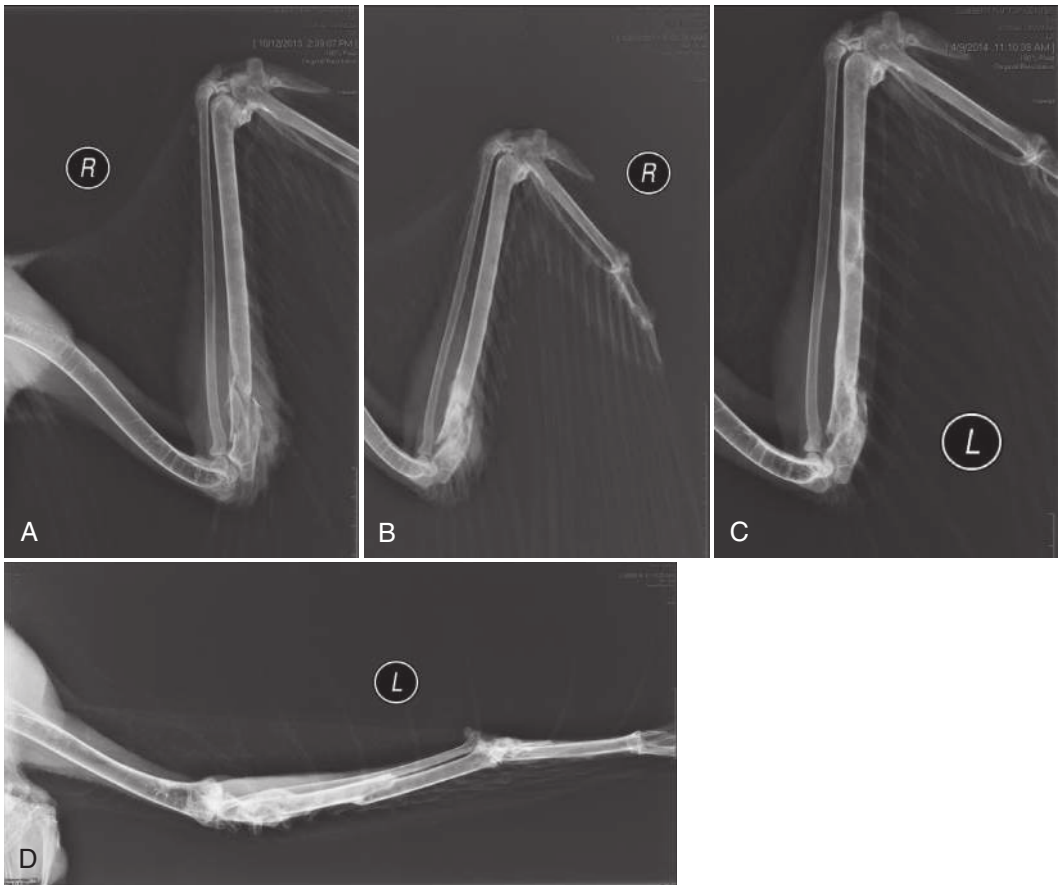


FIGURE 12-8 Proximal, comminuted ulnar fracture with intact radius.There are no viable surgical options in these cases. Careful management of the soft tissues and immobilizing the wing with a well-padded figure-of-eight bandage has been effective. Note in this case, the ulna fractured a second time for unknown reasons at a point distal to the original fracture during the healing process. Both fractures healed over a 6-week period. One attempt to manage such a fracture in another case with a small plate failed, owing to necrosis of the traumatized soft tissue that was sutured over the plate.

normograded into the proximal fragment after fracture reduction. The latter approach is preferred for proximal radial fractures. Where coaptation with a figure eight bandage or taping the wing to the body for 7 to 10 days is advantageous, physical therapy should be undertaken within the first week by temporarily removing the bandage while the patient is anesthetized to conduct the exercise.

Midshaft and Distal Ulnar Fractures, Radius Intact or Fractured: Various Scenarios

In fractures of one or both bones of the forearm beyond the midpoint there is an increasing imperative to apply fixation to minimize the risk of synostosis brought on by uncontrolled movement of the bone fragments during healing. In most cases an IM pin in the radius accompanied by a TIF on the ulna is the fixation of choice. There are other options, such as the use of a type I fixator on the ulna where there is extensive comminution, so the choice depends on the characteristics of the injury. Further, if flight is not a required outcome, a less robust means of stabilization may be indicated.

1. Displaced midshaft ulnar fracture with radius intact (Fig. 12-10). In contrast to Figure 12-7, these cases are more satisfactorily managed by stabilizing the ulna. Choices would be a TIF where there is little or no comminution and healthy soft tissues, or a type I fixator (Fig. 12-10) where there is comminution and soft tissue damage from a high-energy agent (e.g., a shotgun pellet). Note the use of three ESF pins on either side of the fracture to provide adequate stability.
2. Radius fracture with diaphyseal ulnar fracture. For fractures in the midregion of the forearm, pinning of the radius accompanied by either a type I ESF (Fig. 12-11), an IM pin (Fig. 12-12), or a TIF on the ulna (Fig. 12-13) provide acceptable options. Additional bandaging of the wing for 12 to 14 days postoperatively is needed with the first two options, while it is generally not required except to provide patient comfort when a TIF is used.

Note: A TIF (Fig. 12-13) is the best option in any ulnar fracture in the distal two thirds of the bone where return to flight is the desired outcome and fracture/soft tissue condition permits its application. Additional, small ESF pins may be placed along the diaphysis of the ulna in birds with very long forearms (eagles and cranes) for additional stability. Use of temporary fixation at time of admission: in Figure 12-13, *B*, note the use of a two-pin ESF with a conventional Kirschner–Ehmer bar and clamps as a temporary fixator. This can be applied quickly during the admission examination and will prevent bone ends from exteriorizing and improve circulation while waiting for the opportune time to apply a definitive fixation. Fracture repair may be

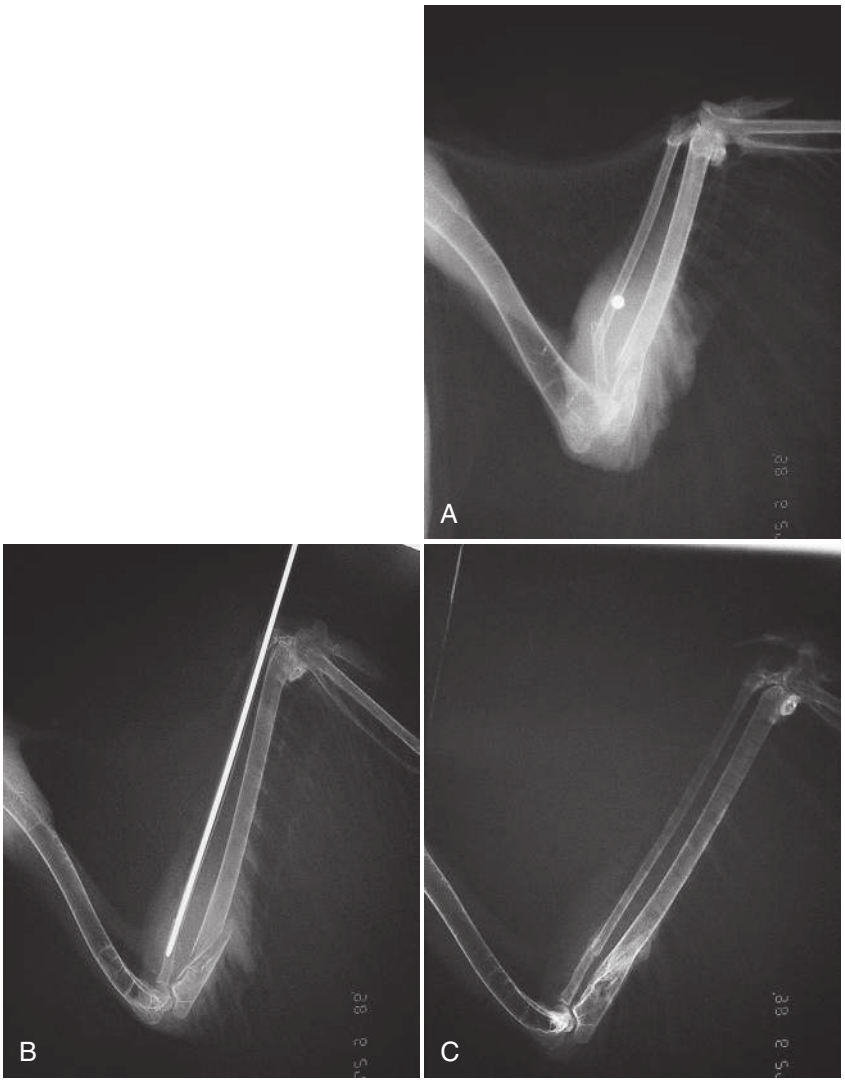


FIGURE 12-9 This red-tailed hawk (*Buteo jamaicensis*) was admitted with a high-energy, comminuted fracture of the proximal radius and ulna **(A)** that was repaired by retrograde placement of an intramedullary pin in the radius only **(B)** to preserve remaining soft tissue in the vicinity of the ulnar fracture. The wing was supported with a figure eight bandage and a satisfactory union was realized in 33 days **(C)**.

delayed this way for up to a week; the wing with a temporary fixator applied is taped to the body in the meantime.

Special Cases of the Forearm: Radius Fractured Proximally or Distally, Ulna Intact

Management options include (1) no fixation, and coaptation, which is recommended only for very proximal radial fractures and (2) an IM pin exiting at the distal end of the radius for diaphyseal and distal fractures, which may be installed either normograde or retrograde depending on the circumstances.

Proximal radial fractures are common in falcons and occasionally in other raptors, presumably from overhead wire strikes. They may or may not be accompanied by varying degrees of elbow luxation. In most cases the proximal fragment is too short for pinning (Fig. 12-14). Coaptation, applied for 3 to 4 weeks with intermittent physical therapy beginning after the second week, is most commonly used. If the ulna is luxated from the humerus, imbrication of the edges of the triceps tendon and the common digital extensor tendon will aid in stabilization of the joint (Ackerman and Redig, 1997). Undesired outcomes include arthritis at the elbow and nonunion.

Radial fractures in the distal three quarters of the bone are best managed by IM pinning (Fig. 12-15). The radius is a very mobile bone and even when the ulna is intact and the wing is stabilized with a figure eight bandage, the radius tends to move, hence, the requirement for immobilization. There is a high probability of the formation of a synostosis, a bony bridge between the two bones, especially with more distally located fractures, if the radius is not stabilized. Synostoses require surgical intervention for a bird to regain flight capability (Beaufrere *et al.*, 2012; Fig. 12-16).

METHODS OF FIXATION FOR THE MAJOR METACARPAL

General Considerations

Fractures of the major metacarpal bone are challenging to manage. The majority of metacarpal fractures are high-energy fractures. The energy

Text continued on p. 332

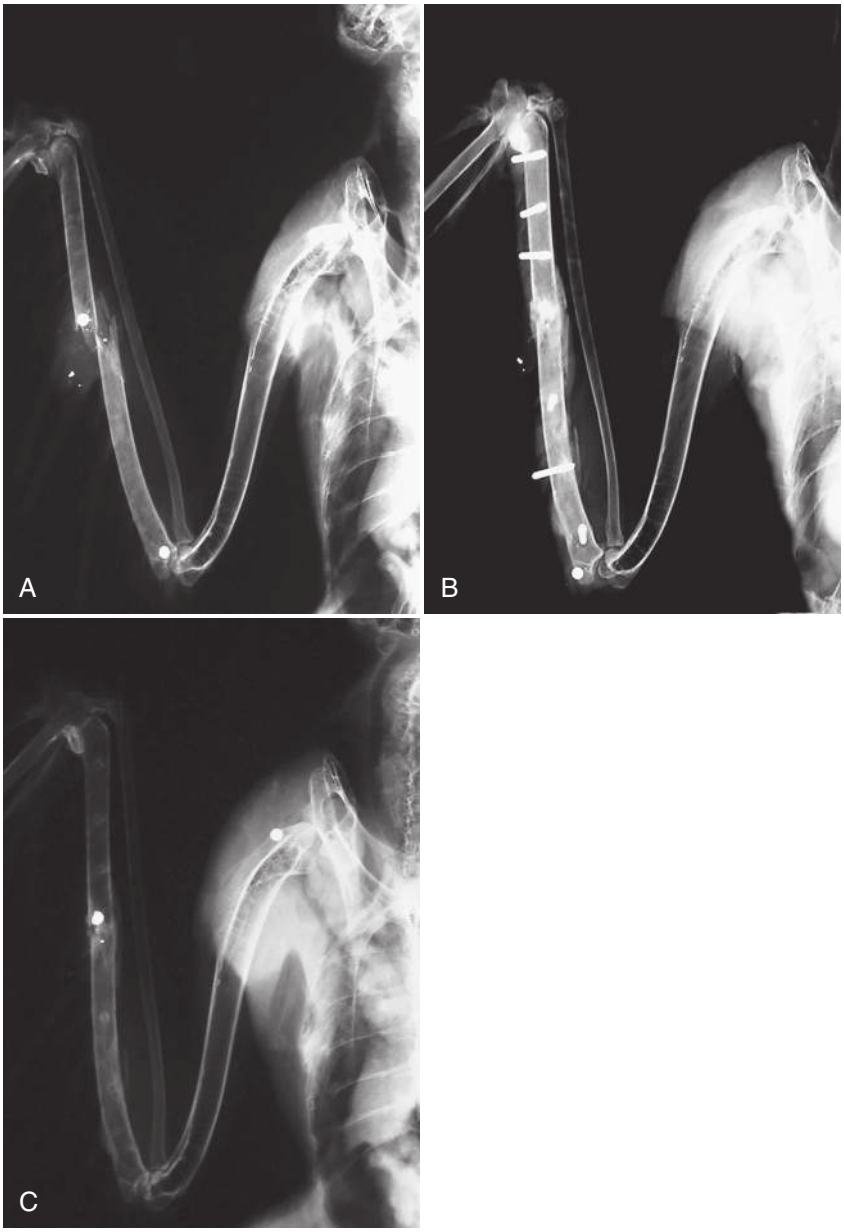


FIGURE 12-10 **(A),** This red-tailed hawk presented with a midshaft fracture of the ulna in which there was displacement of the fragments and a high risk of synostosis with the radius if reduction and stabilization were not undertaken. **(B),** Radiograph, 2 weeks postoperatively, showing the implantation of a type I external skeletal fixator. Because of the soft tissue damage present at the fracture site, this type of fixation was chosen instead of a tie-in fixator. To assure rigidity and longevity of the fixator, three positive-profile threaded pins were used, placed perpendicularly to the bone, on either side of the fracture. **(C),** Radiograph 5 weeks postoperatively and after removal of the fixator. Healing of the fracture with a minimum of external callus can be seen.

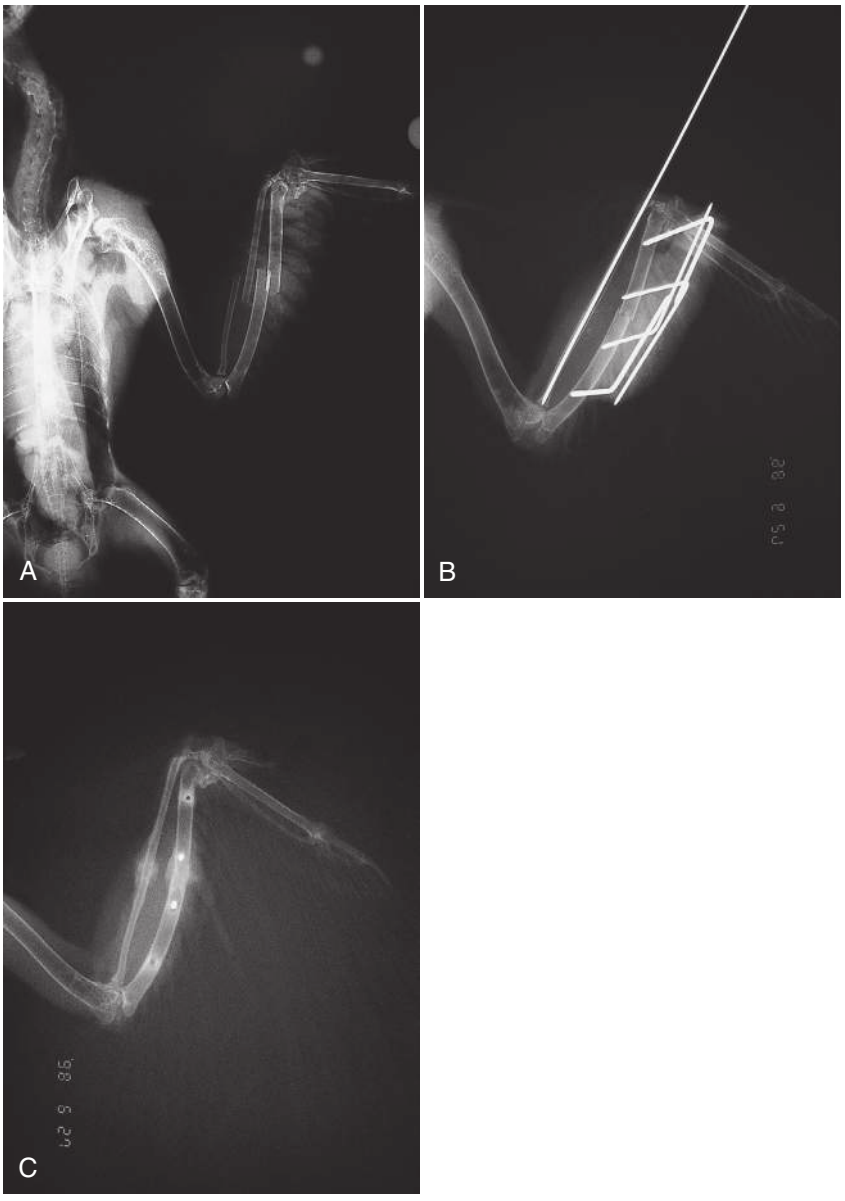


FIGURE 12-11 Radiographs of fixation with an external skeletal fixator (ESF) on the ulna and an intramedullary (IM) pin in the radius. This Cooper's hawk (*Accipiter cooperi*) was admitted with fractures of the radius and ulna **(A)**. An IM pin was inserted in the radius in retrograde fashion, exiting at the carpus. A type I ESF was applied to the ulna with thermoplastic tape connector bar **(B)**. Healing progressed normally and the fixation was removed in 28 days **(C)**. Application of a tie-in fixator to the ulna also would have been a suitable option.

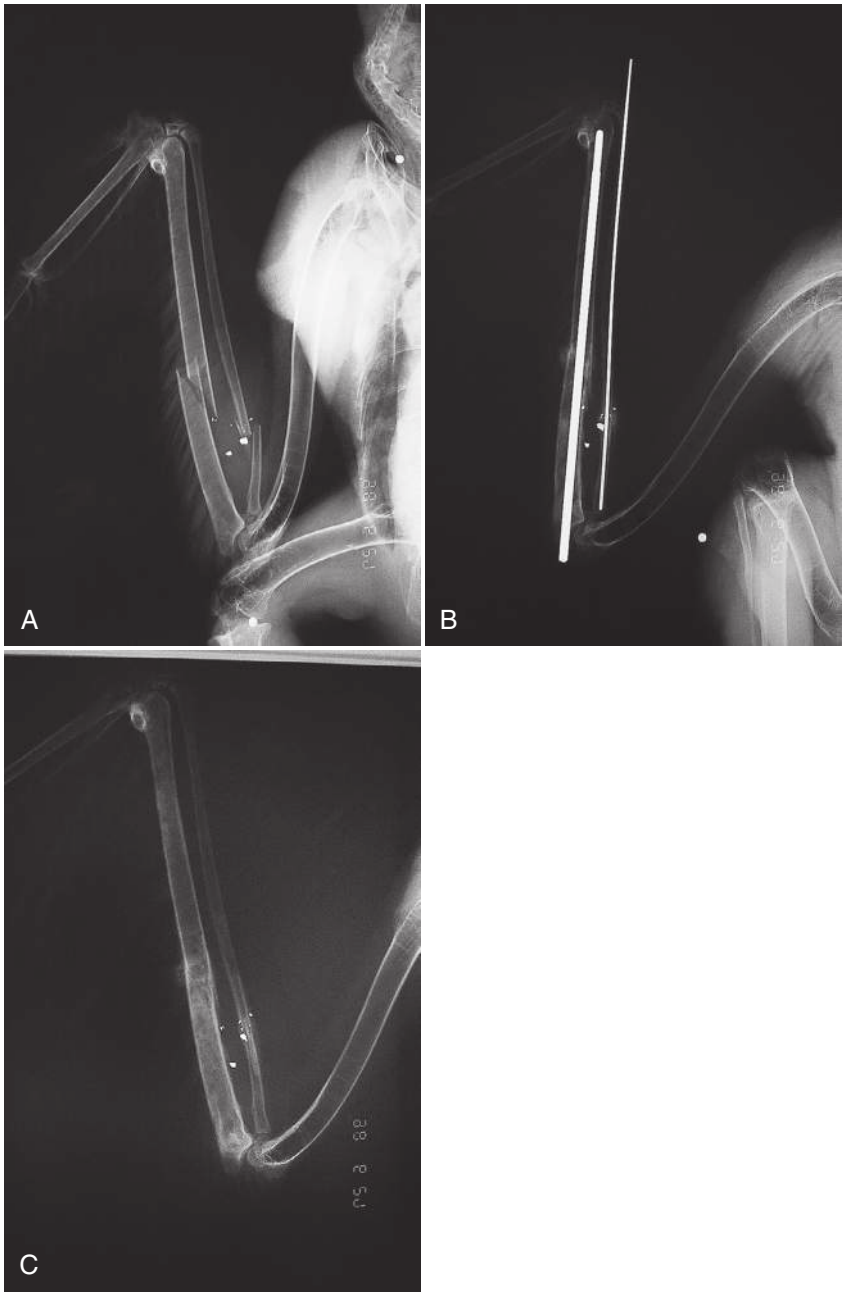


FIGURE 12-12 Where both bones are fractured, intramedullary (IM) pins, one each in the radius and the ulna, may yield satisfactory results. The radial pin is placed first by retrograding it in the distal fragment, exiting at the metacarpus. This golden eagle (*Aquila chrysaetos*) was admitted with a low-energy projectile injury to the radius and ulna **(A)** with minimal accompanying soft tissue injury. IM pins were inserted in the radius and ulna, the former in retrograde fashion exiting at the carpus, the latter in normograde fashion **(B)**. Healing of both fractures was complete in 70 days **(C)**. Management using this method required bandaging in a figure eight for 14 days postoperatively. A TIF would have been a suitable option for the ulnar fracture also.

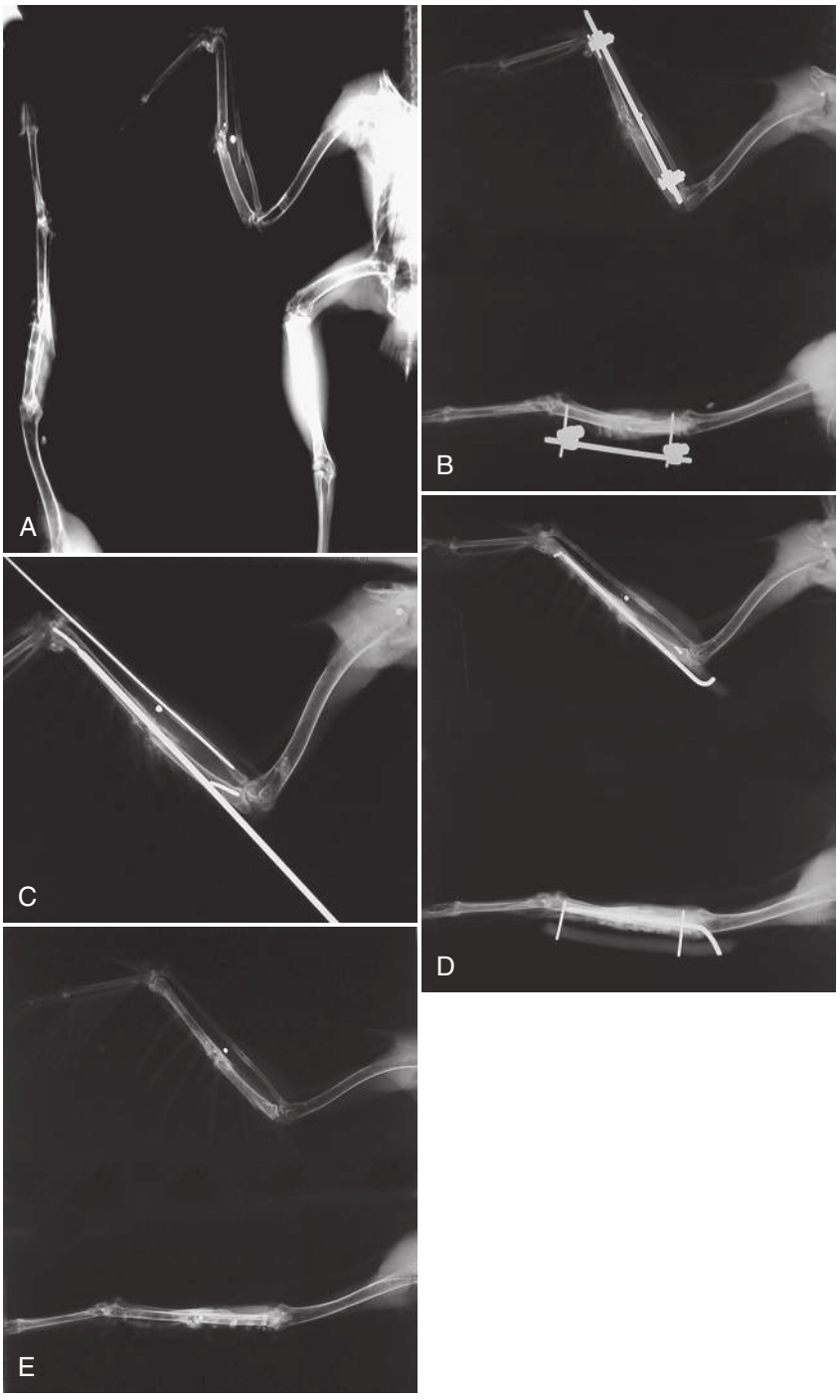


FIGURE 12-13 Use of a tie-in fixator for forearm fracture repair. **(A),** Ventrodorsal and craniocaudal views of midshaft fractures of the radius and the ulna in a red-tailed hawk caused by a projectile. The degree of displacement of the fragments can be seen readily in the posterior-anterior view. **(B),** Application of a two-pin temporary fixator. Installed quickly at the time of admission and accompanied by figure eight coaptation, this device is used to reestablish bone length and relieve stress from the soft tissues until full surgical fixation can be undertaken. **(C),** Intraoperative radiograph showing the placement of intramedullary pins in both the radius and the ulna and the external skeletal fixator pins at the proximal and distal ulna, nominally the same ones used in the temporary fixator. **(D),** Ventrodorsal and posterior-anterior views, 3 weeks postoperatively—callus formation can be seen clearly. **(E),** Ventrodorsal and craniocaudal views, 5 weeks postoperatively after fracture healing and removal of the fixation hardware.

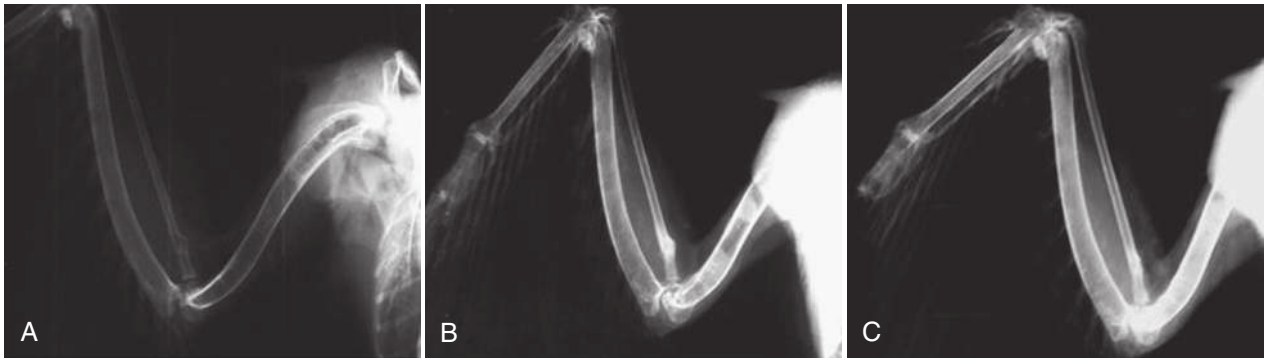


FIGURE 12-14 Proximal radial fractures (within two to three bone diameters of elbow). **(A),** Admission. **(B),** Three weeks. **(C),** Five weeks. This peregrine falcon was admitted with a low-energy, closed transverse fracture of the proximal radius. The ulna was intact and the elements of the elbow joint were luxated. In this location, the radius was protected and contained by soft tissue. This fracture was adequately managed with coaptation with a figure eight bandage; however, the outcome was accompanied by joint degeneration most likely arising from the original injury.

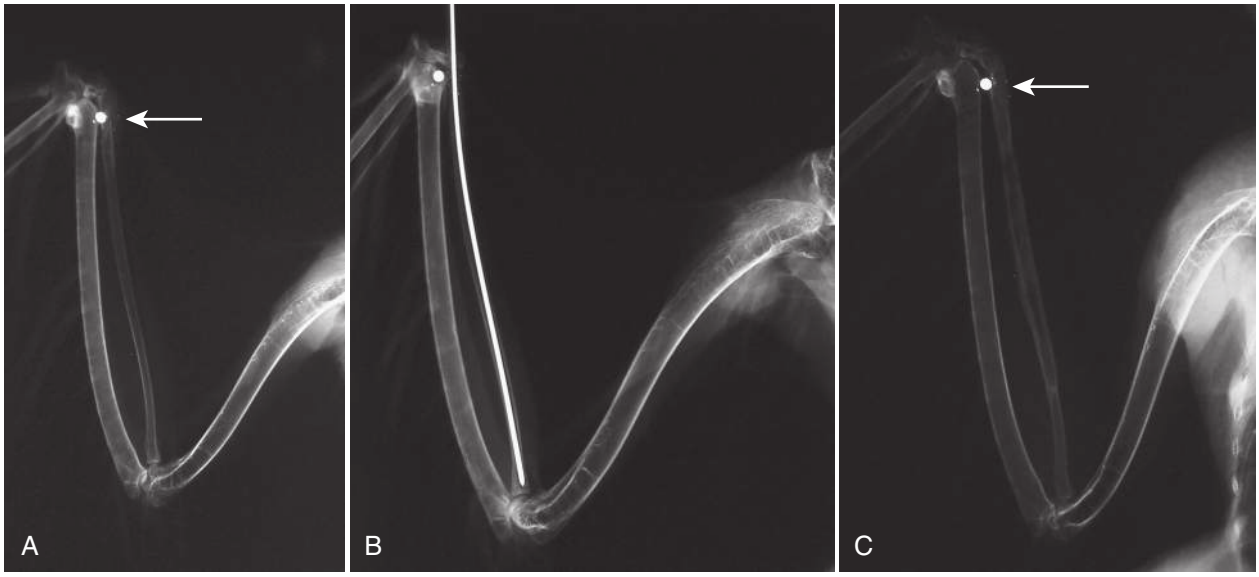


FIGURE 12-15 Distal radial fracture in a bald eagle (*Haliaeetus leucocephalus*) caused by a projectile. **(A),** The *arrow* indicates the site of the fracture. **(B),** Postoperative ventrodorsal view after an intramedullary pin has been placed. In this instance, the pin was introduced in the distal end of the radius at the metacarpus and driven normograde through the distal fragment and into the proximal fragment. **(C),** Five weeks post-operatively after the fracture had healed and the fixation removed. The site of the fracture is indicated by the *arrow*.

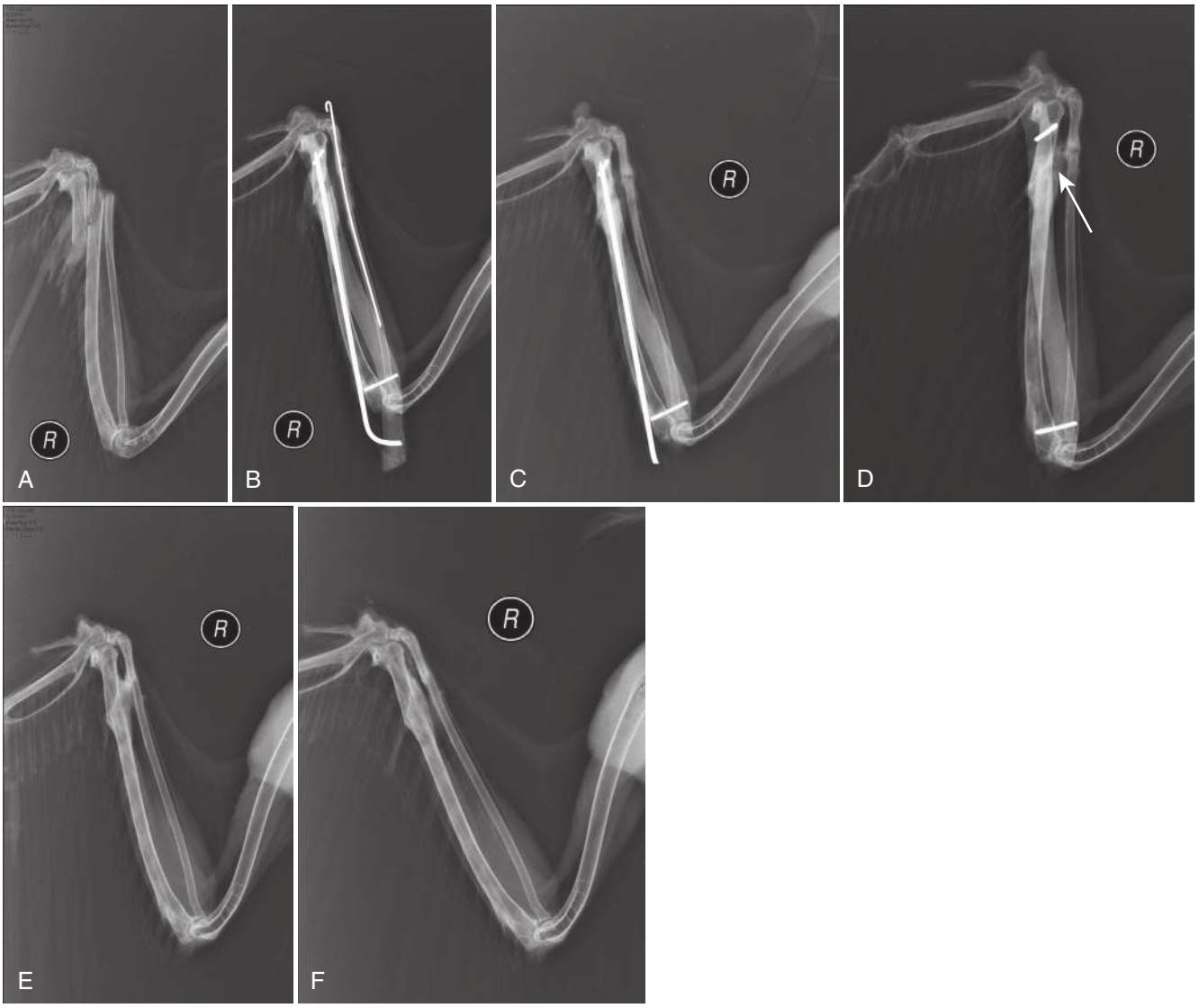


FIGURE 12-16 Radiographs of a great grey owl (*Strix nebulosa*) showing development and management of a synostosis. **(A),** Admission radiograph (ventrodorsal view). **(B),** Postoperative view of tie-in fixator applied to ulna with intramedullary (IM) pin in radius. **(C),** Three weeks postoperatively: IM pin removed from radius, callus forming on ulna, and incomplete union on radius with some evidence of instability. **(D),** Eight weeks postoperatively: IM pin removed from ulna, external skeletal fixator still in place, and development of synostosis is apparent (*arrow*). **(E),** Three months postoperatively: fixation removed, mature synostosis is present, and wing extension impaired. **(F),** After surgical separation of the bones to relieve the synostosis. Synostosis may have been avoided if the IM pin had remained in the radius until there was more complete healing at the fracture site.

of the fracturing agent—a fence wire, power line, or projectile—is concentrated over a very small area that has little soft tissue protection. The small amount of soft tissue present absorbs a good portion of that energy and is severely damaged in the process. Metacarpal fractures typically present as open and/or comminuted. The minor metacarpal bone is capable of providing internal support and load sharing if it is not fractured, improving the prognosis. However, the success rate with any type of management is lower than that seen with other long bone fractures, except the tarsometatarsus.

Treatment options range from coaptation using a reinforced splint (curved-edge splint) and figure eight bandage to type I ESFs. Metacarpal fractures are highly unstable and reestablishment of load sharing is not possible, hence, the fixator or coaptation device must bear the entire load during healing. The TIF has been less successful than other fixation modes.

Coaptation is suitable for the low-energy, transverse reducible fracture, especially if the minor metacarpal is intact. Many metacarpal fractures are very proximal providing no opportunity for purchase of fixation hardware, making coaptation the only choice. Because the wing must be bound in a splint for approximately 3 weeks, the potential for immobilization-related morbidity is substantial. Splints made from moldable material (e.g., SAM Splint or various thermoplastics molded into a "curved-edge splint") have been a satisfactory way to coaptively stabilize metacarpal fractures (Figs. 12-17 and 12-18).

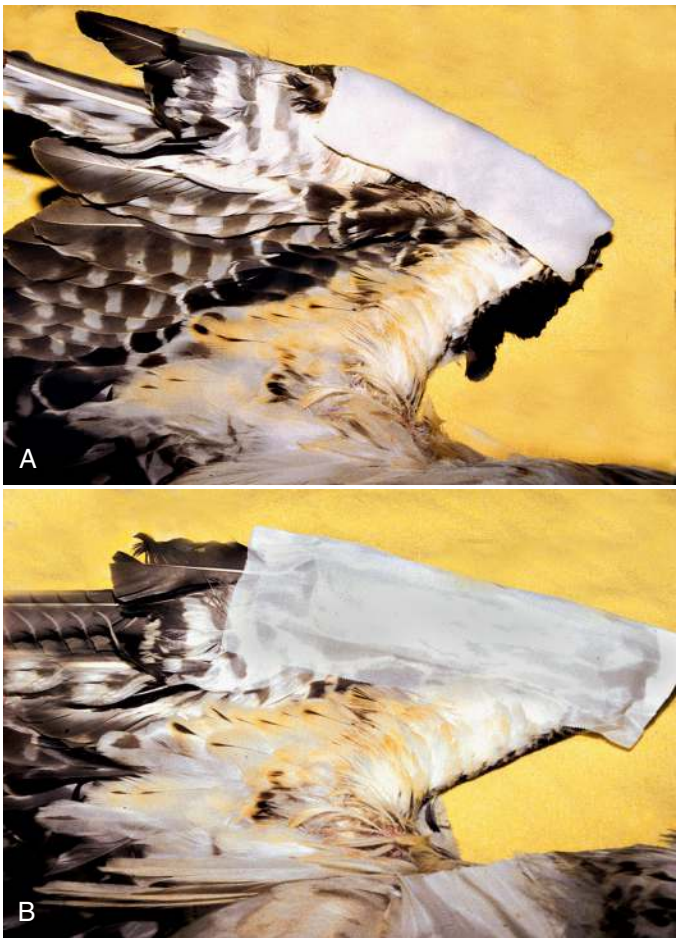


FIGURE 12-17 **(A),** Illustration of the curved-edge splint. **(B),** Curved-edge splint made from moldable material for an osprey (*Pandion haliaetus*). Veterinary thermoplastic[m] is a heat-activated casting tape. A strip of material was cut long enough to span the distance from the proximal end of the radioulnar-carpometacarpal joint to well beyond the joint with the second phalanx and wide enough to span the width of the metacarpal bone.

Trimming the long primaries short will unload distracting forces from the wing and may enhance healing. Feathers can be replaced later by imping.

Type I external fixation is another choice for simple metacarpal fractures or those with extensive soft tissue damage, comminution, or fragment displacement, because reduction, alignment, and stabilization can be accomplished with minimal manipulation of the soft tissue (Fig. 12-19). The use of an intraoperative jig made from KE components is useful in achieving bone alignment and holding it until an acrylic bar cures.

Note: Fixation stabilization will improve the rate of healing compared with simple splinting. One caveat—with a fixation device in place, the wing should be bandaged to the body because a sudden flapping of the wing may generate enough force to dislodge the fixator. The least desirable choice for the metacarpus is an IM pin: as the liability of morbidity associated with implanting the pin is incurred, however, stability of the limb is not achieved without further immobilization. Delaying application of fixation by 5 to 7 days postinjury to allow soft tissues some time to recover before insulting them again with hardware placement has led to improved healing success with metacarpal fractures. This is particularly true in falcons, where metacarpal fractures are most often accompanied by moderate to severe edema (Fig. 12-20). This must be reduced before repair of the fracture is attempted. This is accomplished by twice daily hot packing the wing for 5 to 10 minutes, application of dimethylsulfoxide over the area once or twice, and administering peripheral vasodilating drugs (e.g., isoxuprine[h]). In the meantime, the wing is kept bandaged in a figure eight and taped to the body. No other bone in the avian skeleton requires more attention to careful assessment and selection of a proper fixation device to maximize the healing potential of a fracture.

METHOD OF FIXATION FOR FRACTURES OF THE FEMUR

General Considerations

With abundant soft tissue protection afforded by heavy muscle, the femur responds favorably to most attempts at fixation. The approach to fixation resembles that of the humerus, and the ESF-IM pin tie-in works well. For insertion of the IM pin, the femur is approached from the lateral aspect. The bird is laid with the contralateral side down. The affected leg is abducted and the distal portion of the ipsilateral wing is placed underneath the leg, between the medial aspect of the leg and the body wall. The incision is made at approximately the 4 o'clock position on the femoral shaft as viewed from the distal end on, extending from the condyles at the distal end to the proximal end. Blunt dissection is used to separate the quadriceps femoris muscle from the ventral flexor muscle group. The femoral artery, vein, and nerve lie deep and ventral to the femur; they will be visualized but do not present a serious hazard during repair of the bone.

Specific Recommendation for Fractures of the Femur

Femoral fractures can be managed in a manner closely analogous to the humerus. The application of a TIF is illustrated diagrammatically in Figure 12-21. For diaphyseal fractures, the IM pin for the TIF is typically introduced at the fracture site and retrograded proximally exiting at the hip. The distal ESF pin is placed from lateral to medial through the condyles. The proximal ESF pin is placed from lateral to medial by palpating the dorsal rim of the acetabulum and selecting a point on the femur one-to-two bone diameters distad. A smaller pin than that used distally is selected because it must share the marrow cavity with the IM pin. As the medial side of the femur cannot be palpated, determination of proper drilling depth for the pin must be done by "feel." Characteristically, resistance to the rotation of the pin chuck can be felt when the trocar of the pin is drilling through bone cortex. Resistance is felt when passing through the first cortex, lack of resistance as the pin is threaded through the pneumatic medullary cavity, and increased resistance again when the trocar strikes the opposite cortex. Gentle downward pressure is applied to the pin while threading continues. Two to three full rotations of the chuck, after an increase in resistance, is sufficient to seat the pin in the opposite cortex. Back and forth movements of the pin chuck should now result in the entire bone fragment moving in concert. If gross movement of the bone is not detected, it means that only one cortex has been engaged. After placement of the ESF pins, the exteriorized portion of the IM pin is bent at 90 degrees and the elements are bonded together with an acrylic bar. Examples of femoral fracture management with a TIF are depicted in Figures 12-22 and 12-23.

Fractures of the proximal femur may be repaired using a tension band wiring system using two K-wires and cerclage wire (Harcourt-Brown, 1996). Distal fractures may be repaired using a cross-pin method similar to the distal humerus (see previous section). Again,

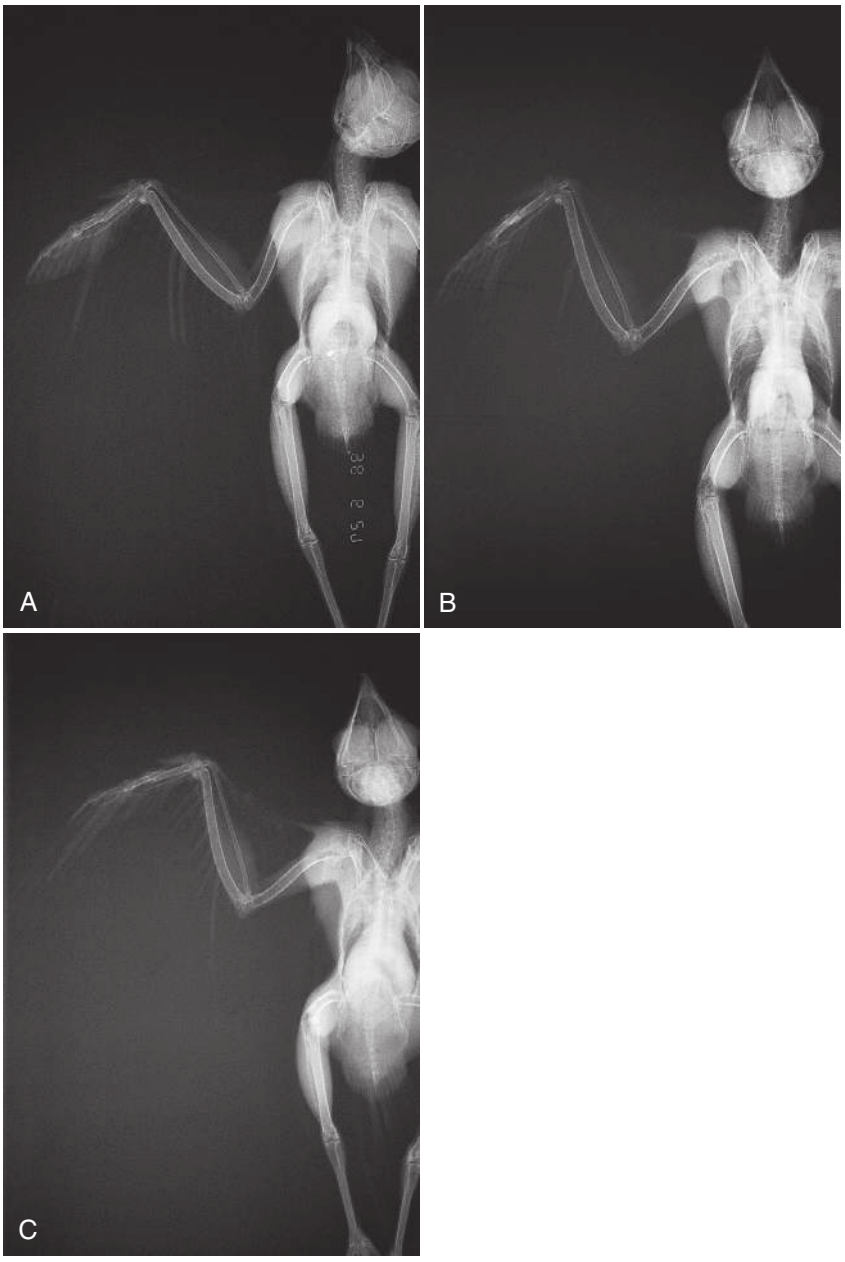


FIGURE 12-18 This American kestrel (*Falco sparverius*) was admitted with a closed, midshaft major metacarpal fracture accompanied by severe soft tissue contusion **(A)**. A curved-edge splint (made from a foam-clad aluminum splint material—SAM Splint) was applied and the fracture was healing slowly at 24 days **(B)**. Complete union was realized in 60 days **(C)**.

tying one or both of the cross-pins to an ESF will provide excellent stabilization.

METHODS OF FIXATION FOR FRACTURES OF THE TIBIOTARSUS

General Considerations

The tibiotarsus is a very straight bone with a narrow medullary cavity that tapers from proximal to distal. The proximal two thirds are well protected by soft tissue and the primary loads borne during normal use are compressive. Satisfactory orthopedic management of fractures of this limb mandates rotational alignment of the stifle and hock joints and lateral-to-medial alignment of the fragments; anterior to posterior alignment is less critical. To preserve integrity of the contralateral foot, immediate postoperative weight bearing is desirable, although injury to soft tissue often results in temporary impaired use even though the fixation is capable of load bearing. Fractures in the proximal one third are most often transverse, thus offering opportunities for load sharing. TIFs have proved very effective in managing fractures of this bone. Both ends of the tibiotarsus are protected by adjacent leg bones and associated joints; hence, penetration of the proximal and distal ends is a potential morbidity factor when the IM pin is inserted. Careful placement of the IM pin mitigates this potential problem. Type I or type II ESF fixation has proven to be less suitable, but may be a suitable choice in some instances.

Text continued on p. 339

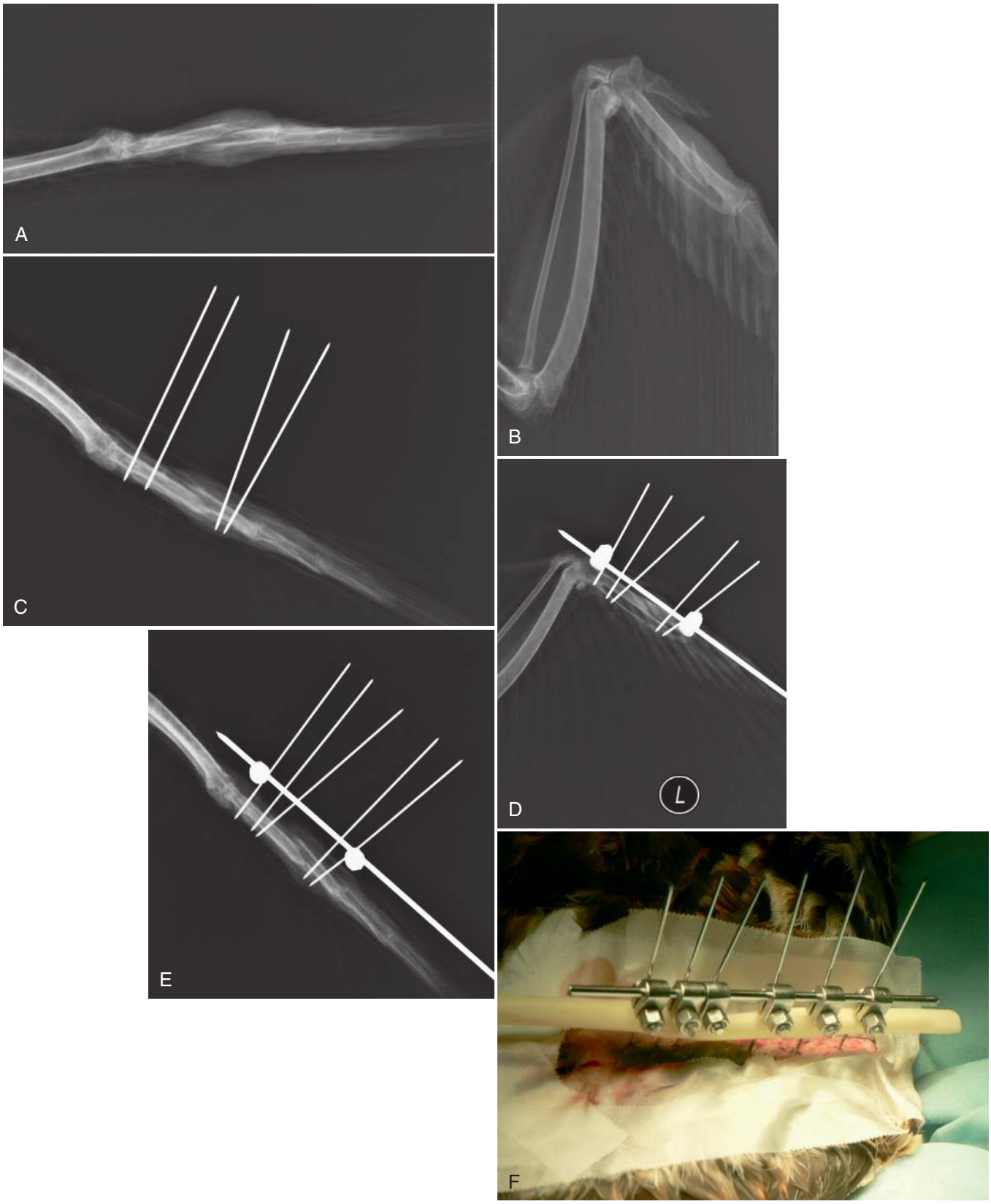


FIGURE 12-19 Application of a type I fixator to a proximal major metacarpal fracture in a peregrine falcon (*Falco peregrinus*). **(A),** Note the vertical displacement of the fragments seen in the posterior-anterior view. **(B),** Admission radiographs, posterior–anterior (PA) and ventrodorsal (VD) views. **(C),** Initial placement of threaded interface pins on either side of the fracture. Note decrease in swelling of soft tissue compared with admission radiographs; the ESF pins were used to manipulate the fragments into alignment that was verified radiographically before application of the acrylic bar. **(D-F),** Intraoperative radiograph in VD and PA projections showing use of a jig made from KE clamps and a stainless steel bar for alignment of fragments before installing the acrylic bar. Latex mold was placed over the pins before placement of jig.

Continued

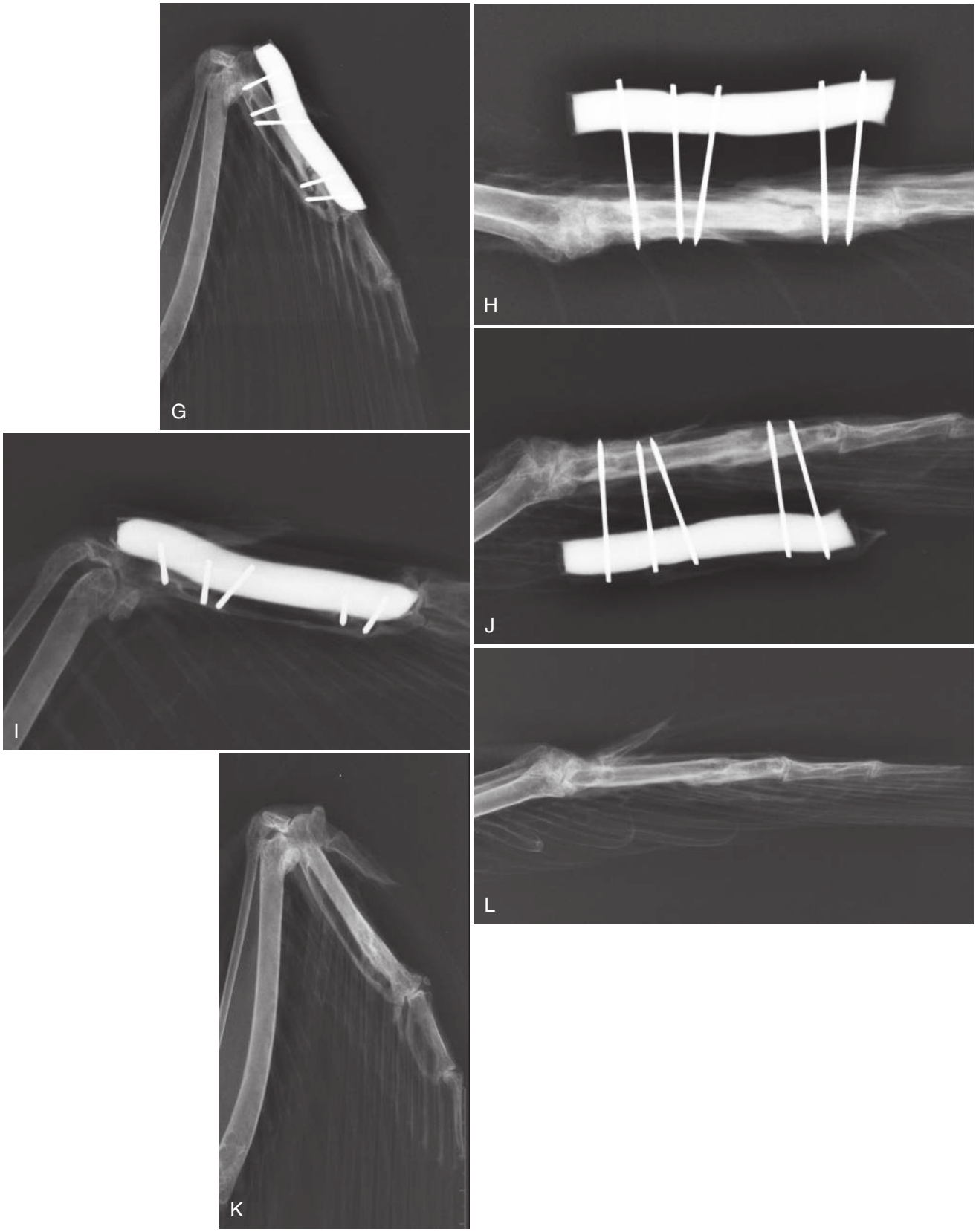


FIGURE 12-19, cont'd **(G, H),** Twelve days postoperative VD and PA views showing acrylic bar in place; note initial stages of callus formation. **(I, J),** Twenty-two days postoperative VD and PA views. Fracture is healed and remodeling is underway. Fixation was removed at this point. **(K, L),** Sixty-one days postoperative VD and PA images in this prerelease radiograph. Complete healing with satisfactory alignment is evident.

FIGURE 12-20 Wing-tip edema as is typically seen in falcons with metacarpal fractures.

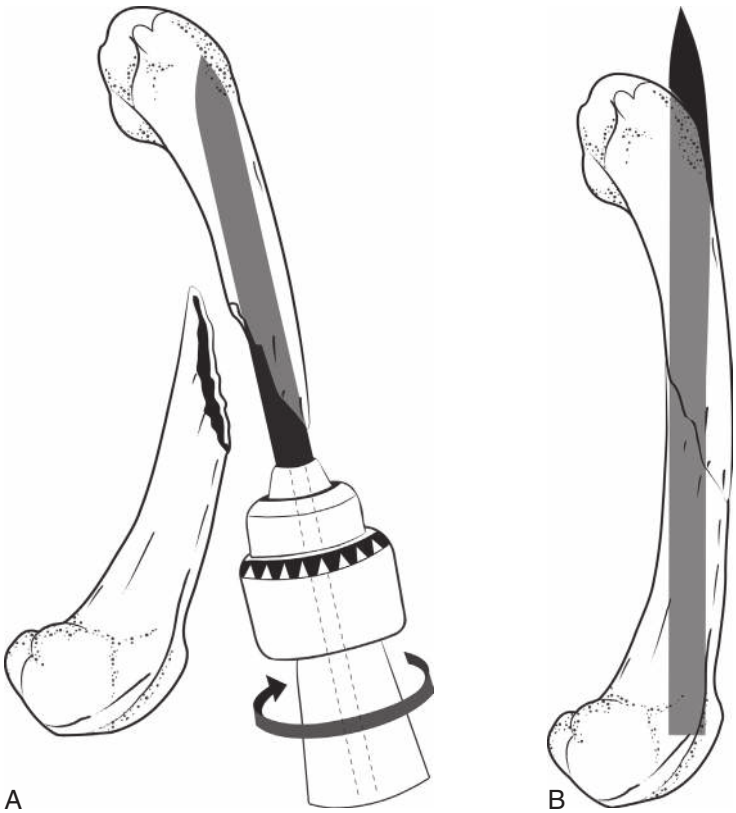


FIGURE 12-21 (A-D) Application of a Tie-in Fixator to a midshaft femoral fracture. **(A),** the femur is approached from the lateral aspect, separating the major muscle groups and exposing the diaphysis of the bone. An appropriately-sized IM pin is inserted in the proximal fragment and driven retrograde, exiting at the hip. **(B),** The fracture is reduced and the pin driven into the distal fragment. The surgical site is now closed.

Continued

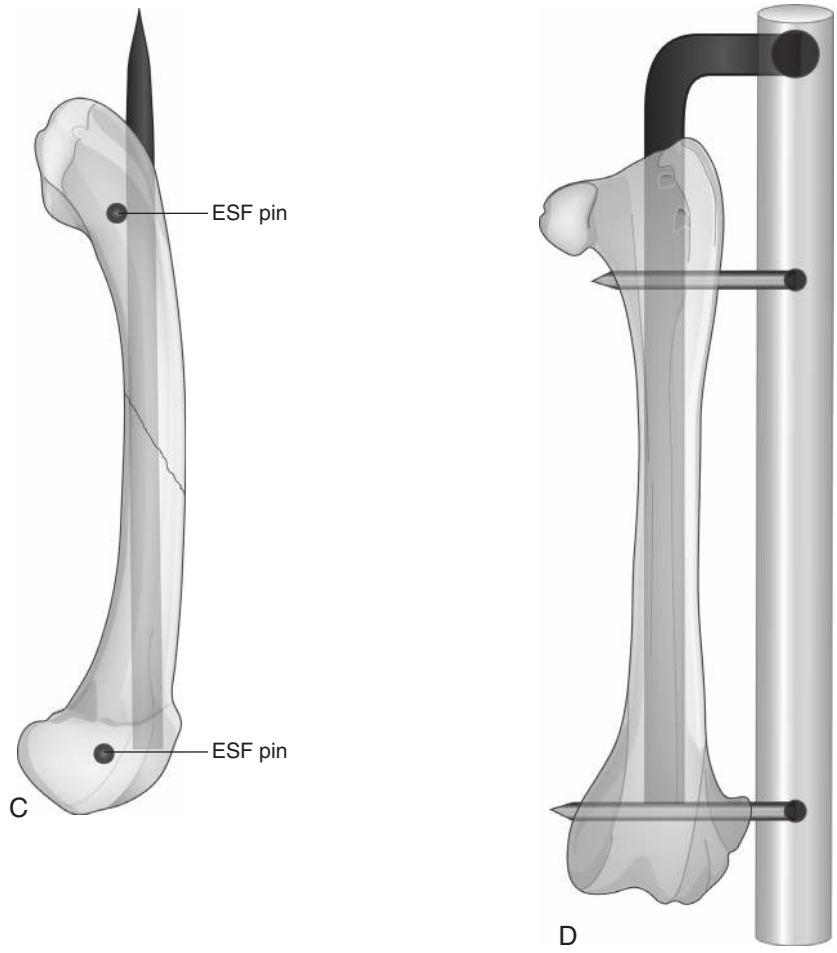


Figure 12-21, cont'd **(C),** positive-profile threaded ESF pins are placed at the distal end through the condyles and in the proximal fragment. **(D),** The IM pin is bent at 90° and rotated into the same plane as the ESF pins. An acrylic bar is applied as described above.

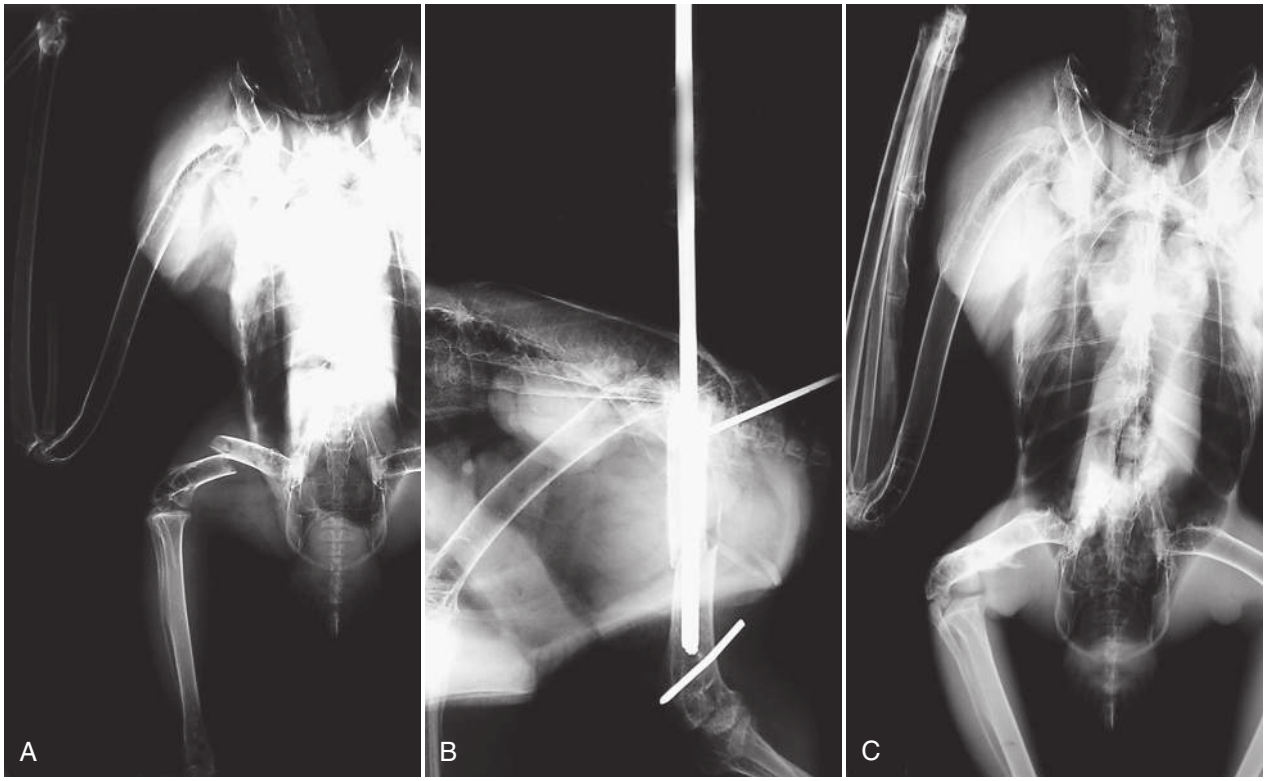


FIGURE 12-22 Bald eagle (*Haliaeetus leucocephalus*) with a midshaft femoral fracture. **(A),** Admission radiograph. **(B),** Intraoperative radiograph taken to check placement of intramedullary and external skeletal fixator pins. **(C),** Radiograph taken at 5 weeks postoperatively after fracture healing and removal of all hardware.

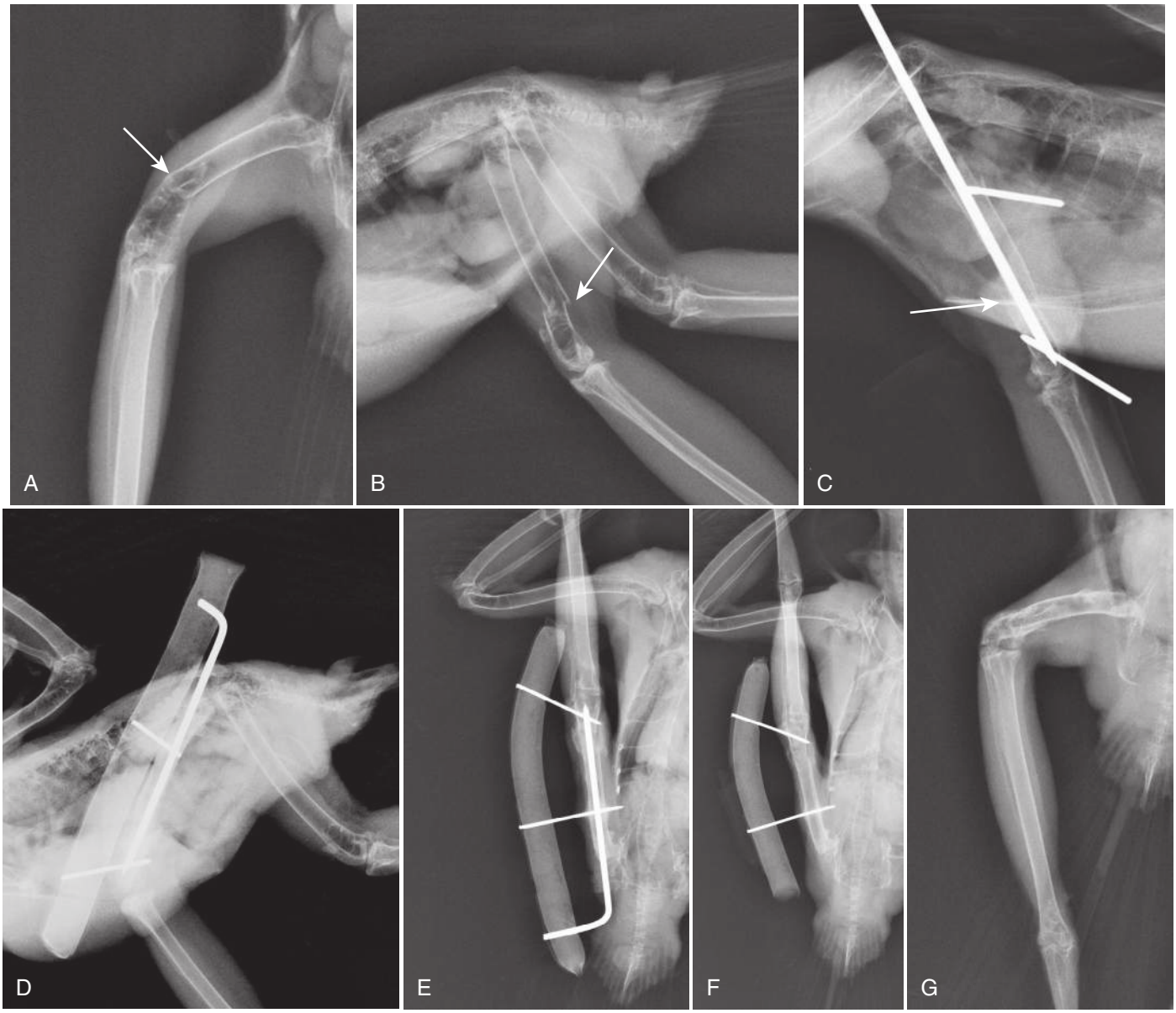


FIGURE 12-23 Admission radiographs, anterior–posterior **(A)** and lateral **(B)** of a Coopers hawk that presented with a transverse, closed distal diaphyseal fracture of the femur. *Arrows* indicate fracture site. **(C),** Intraoperative radiograph showing placement of external skeletal fixator (ESF) and intramedullary (IM) pins. *Arrow* indicates location of the fracture. **(D, E),** Radiographs, lateral and posterior–anterior (PA) taken 3 weeks postoperatively. Callus formation is evident, especially in the PA view. **(F),** PA radiograph taken 5 weeks postoperatively. Fracture is healed and the callus is undergoing remodeling. The IM pin was removed; ESF left in place for continued support. **(G),** Ventrodorsal radiograph taken 6 weeks postoperatively. The fracture is healed and remodeled. All fixation has been removed.

Fractures of the Tibiotarsus

Among raptors kept for falconry purposes, fractures of the tibiotarsus in the proximal one half arising from bating accidents are seen frequently (Harcourt-Brown, 1996). These are typically low-energy transverse fractures. Wild casualty birds most often have complicated and comminuted, high-energy fractures involving the tibiotarsus. Because of the large muscle masses, especially in the proximal region, tibiotarsal fractures are seldom open and prognosis is good. Two caveats exist for wild casualty birds: (1) nerve damage often accompanies their fractures of the tibiotarsus, leading to slow return to use of the lower limb, and (2) spinal injury often accompanies these fractures but may be hard to detect at admission because of the analgia in the broken limb. Failure to properly assess this condition may lead to an unnecessary and unproductive fixation procedure.

Historically, a type II ESF was advocated by many surgeons and yielded satisfactory results in some cases (Redig, 1986a; Hess, 1994; Harcourt-Brown, 1996; Bennett, 1997). More recently a variety of methods involving IM pins and compression plates (Sanchez-Migallon Guzman *et al.*, 2007), interlocking nails (Hollamby *et al.*, 2004), and TIFs (Redig, 2000, Bueno & Redig, 2015) have been reported to yield successful results. From a large number of cases involving raptors ranging in size from 120-g kestrels to 5-kg eagles, we have

found that an adaptation of the TIF produces exceptional results, again making it the method of choice in all but those cases involving severe comminution, in which a type II ESF may be a more appropriate choice, or for very distal fractures, where cross-pinning is recommended (Harcourt-Brown, 1996). Many of the potential complications ascribed to the combination of IM pin and ESF components asserted in some published articles, such as soft issue, joint compromise, lack of patient acceptance of external hardware, and bone infection (Hollamby *et al.*, 2004), have been entirely unrealized.

Another unique method for fixation was the reported use of a ring fixator to stabilize a tibiotarsal fracture and allow for lengthening of the fixator during the healing process to promote new bone formation (bone transport osteogenesis) and reestablish length where there was loss of bone (Johnston *et al.*, 2008).

Coaptation (e.g., tubular-type splints or Schroeder–Thomas variants) has a low rate of success in managing tibiotarsal fractures.

Specific Recommendations for Application of the TIF to the Tibiotarsus

When selecting the IM pin, use the marrow cavity diameter as measured at the distal end on a mediolateral view, because this bone tapers substantially in the distal portion (Harcourt-Brown, 1996); a pin that is approximately 60% of the bone diameter viewed on the lateral radiograph is suitable. Insertion of a pin that is too large results in pressure necrosis and loss of bone (Johnston *et al.*, 2008).

For application of the TIF to the tibiotarsus, the IM pin is introduced to the tibial table on the medial aspect of the femorotibial joint and passed normograde into the retropatellar fossa (Sanchez-Migallon Guzman *et al.*, 2007) and into the proximal fragment (Fig. 12-24). Initially the pin is placed through the skin at the medial edge of the patellar tendon parallel to the joint surface. The trocar of the pin is worked underneath the tendon. The pin is elevated and aligned with the long axis of the proximal tibiotarsal fragment, displacing the patellar tendon laterally, and advanced distally. The fracture is reduced and the pin is advanced into the distal fragment, stopping short of the area defined by the supratendinal ridge.

Threaded ESF pins are placed transversely both distally and proximally. The distal pin must be placed one-to-two bone diameters proximal to the hock joint to avoid injury to vessels and tendons at the end of the bone, and its insertion point should not be distal to the supratendinal ridge (Fig. 12-24, *D*). The proximal pin should be introduced on the craniolateral aspect just distal to the tibial plateau and craniad to the fibula. It should be directed caudomedially to avoid the neurovascular bundles on the medial side of the proximal tibiotarsus (Harcourt-Brown, 2000) and aligned with the distal pin when the leg is rotated into proper anatomical alignment. The IM pin is again bent at 90 degrees and directed laterally so that it can be joined to the ESF pins with an acrylic bar or conventional fixator clamps and a bar (Fig. 12-24, *D*).

Postoperatively, incomplete weight bearing on the affected leg for 3 to 5 days is expected because of transitory neuroparalysis arising either from the injury itself or the surgical procedure; "knuckling" may be seen occasionally. It is important to wrap the digits of the affected limb with protective materials (e.g., Vetrap) to prevent abrasion of the dorsal surfaces. Concurrently, the temporary asymmetric weight bearing predisposes bumblefoot formation in the contralateral foot, so it also should be bandaged. Examples of healing of tibiotarsal fractures are presented in Figures 12-25 and 12-26.

Note: Type I fixators are typically not strong enough to stabilize a tibiotarsal fracture. Conversion to a type II fixator improves the construct; however, the medial connector bar at the level of the proximal ESF pin will cause damage to the body wall because of the upward projection of the stifle when the bird is at rest. This may be reduced by angling the proximal ESF pin distally from lateral to medial. If a type II fixator is selected, results will be improved by the use of a center-threaded pin. Proximal fractures of the tibiotarsus in which the proximal fragment is too short to accept a TIF may be managed with a transarticular fracture as depicted in Figure 12-27. Distal tibiotarsal fractures (distal to the supratendinal ridge) are best managed with a type II transarticular fixator construct in which two-to-three interface pins are placed in the tibiotarsus, one through the condyles of the tibiotarsus and two in the tarsometatarsus. The hock joint is flexed in a normal perching position and a contour-following acrylic bar is attached.

METHODS OF STABILIZATION AND FIXATION FOR TARSOMETATARSAL FRACTURES

General Considerations

Like the metacarpus, the tarsometatarsus has a paucity of soft tissue coverage and therefore many of the same management problems. Anatomically, it is quite different because it has no medullary cavity in the proximal one third in hawks and owls, but in falcons a marrow cavity runs the full length (Harcourt-Brown, 2000). In cross section, it is a U-shaped bone formed embryonically from the fusion of elements of the metatarsal and tarsal bones. The flexor tendons run in a deep channel on the caudal aspect; veins are present on the lateral and medial aspects and arterial blood supply along with nerves and tendons are found on the cranial aspect. Also, the bone is protected by articular surfaces at both ends. These factors combine to render IM pinning a poor fixation choice. Additionally, when a bird is perching at rest, the bone is positioned at an angle to the perching surface so load bearing applies bending forces, as well as rotational forces to the bone.

Specific Recommendations for Fixation Choices

In birds weighing up to 150 g with closed and otherwise uncomplicated fractures, an effective means of stabilization has been coaptation in the form of a tape splint combined with taping the hock in flexion so the tarsometatarsus is splinted by the tibiotarsus (Figs. 12-28 and 12-29). A cast of a thermoplastic material can be used, particularly in a closed fracture in which no wound management procedures are required. Schroeder–Thomas splints are another alternative for birds weighing less than 1 to 1.5 kg (Redig, 1986b).

Fixator application in the form of a type II ESF is applicable in a wide variety of situations and is the method of choice in any situation where there is comminution or there are open wounds that require management (Fig. 12-30). As with the tibiotarsus, it is advantageous for at least two of the through-and-through pins to be center-faced pins. Care must be taken not to pass the pins through the flexor channel on the caudal aspect of the bone. Because of the bending loads applied to this bone, it is useful to place three pins on either side of the fracture site, if possible, rather than only two. In another variation, where there was a very short proximal fragment in an eagle, stabilization was achieved by placement of two ESF pins in the distal fragment—one in the proximal fragment and two in the distal tibiotarsus. These were bound together medially and laterally with an acrylic bar (type II configuration). In addition, another bar spanning the distance from the most proximal pin in the tibiotarsus to the most distal ESF pin in the tarsometatarsus was attached on the lateral side to fix the hock joint in place and provide additional stability to the construct (Fig. 12-31).

Another reported successful method of fixation of the tarsometatarsus was the use of a locking plate as an external fixator bar (Montgomery *et al.*, 2011). This is a unique application of a locking plate in a type I external fixator construct.

Text continued on p. 349

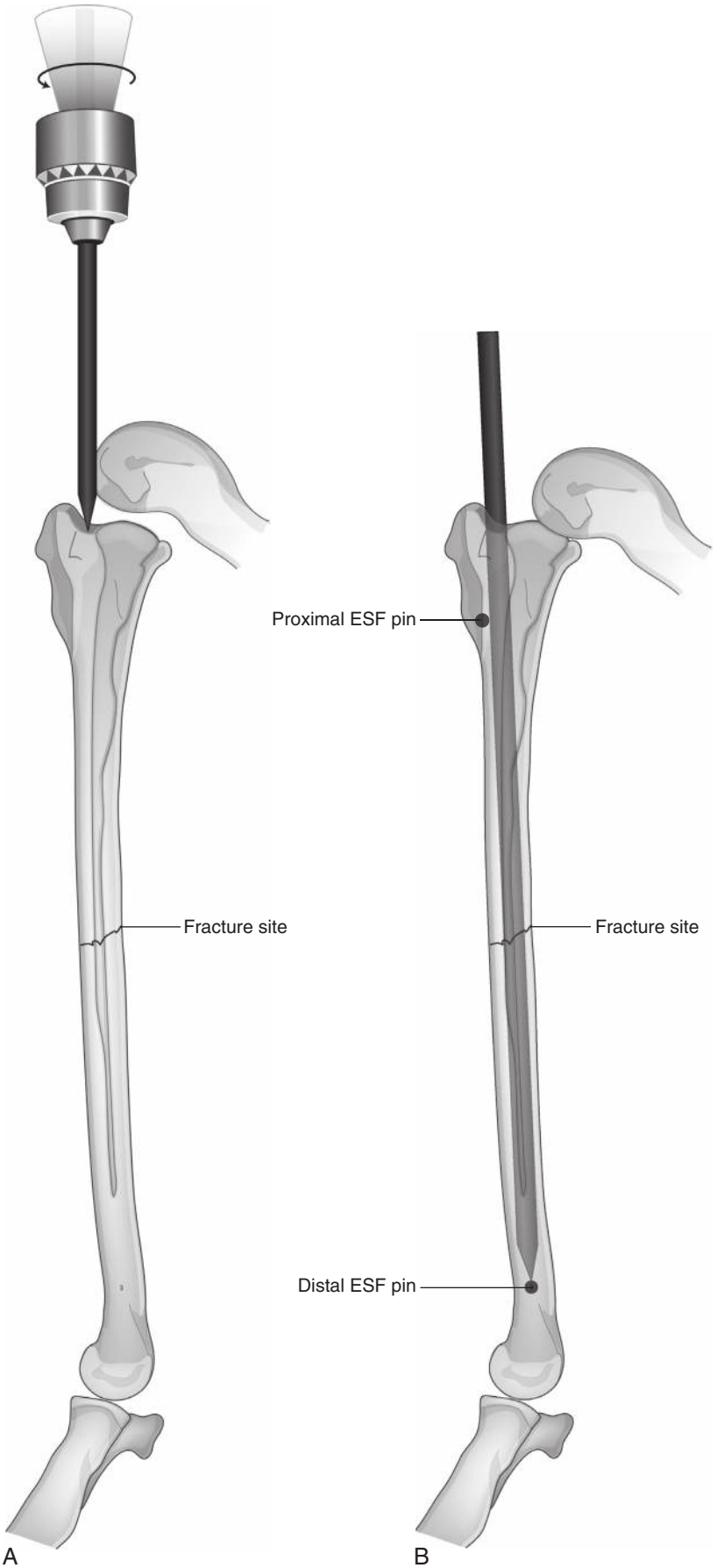
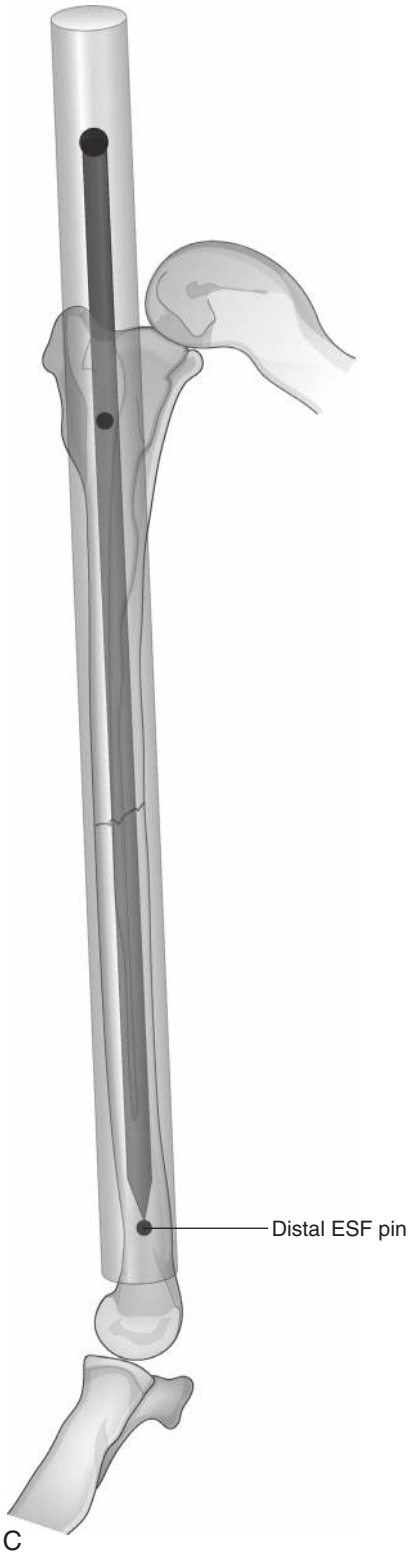


FIGURE 12-24 Application of a type I fixator to the tibiotarsus. **(A),** Lateral view of the introduction of an intramedullary (IM) pin into the tibiotarsus. See text for details of insertion of the pin. **(B),** Relative placement of the IM pin and the proximal and distal external skeletal fixator (ESF) pins. *Continued*



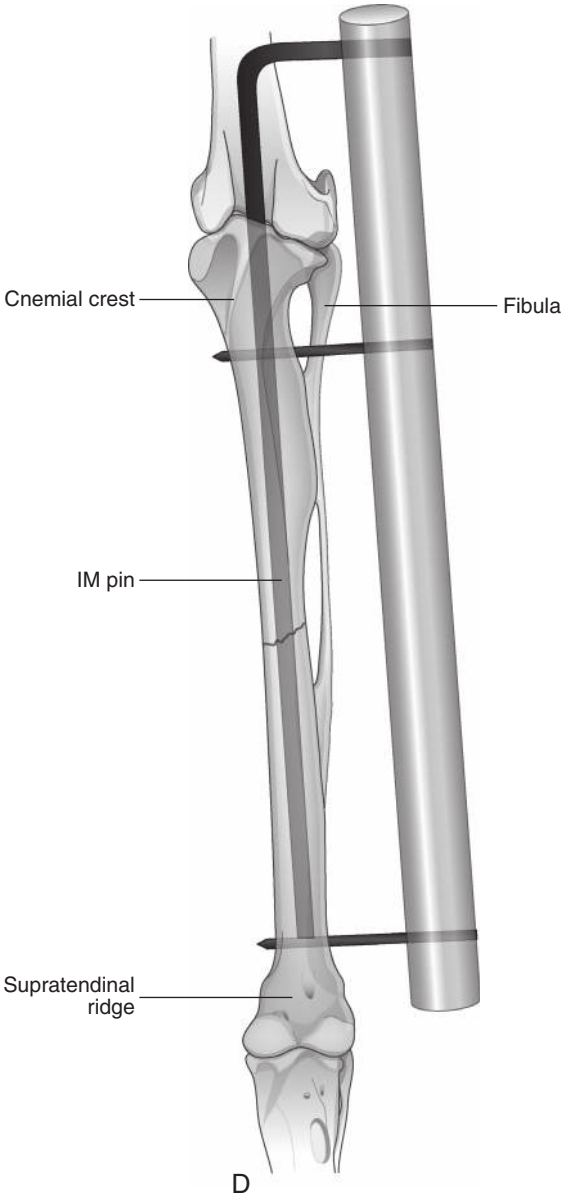


FIGURE 12-24, cont'd (C), The tie-in fixator (TIF) as viewed from the lateral side of the tibiotarsus. **(D),** Craniocaudal view of the TIF applied to the tibiotarsal bone. Note that the distal ESF pin is inserted proximal to the supratendinal ridge and that the connector bar is on the lateral side of the leg.

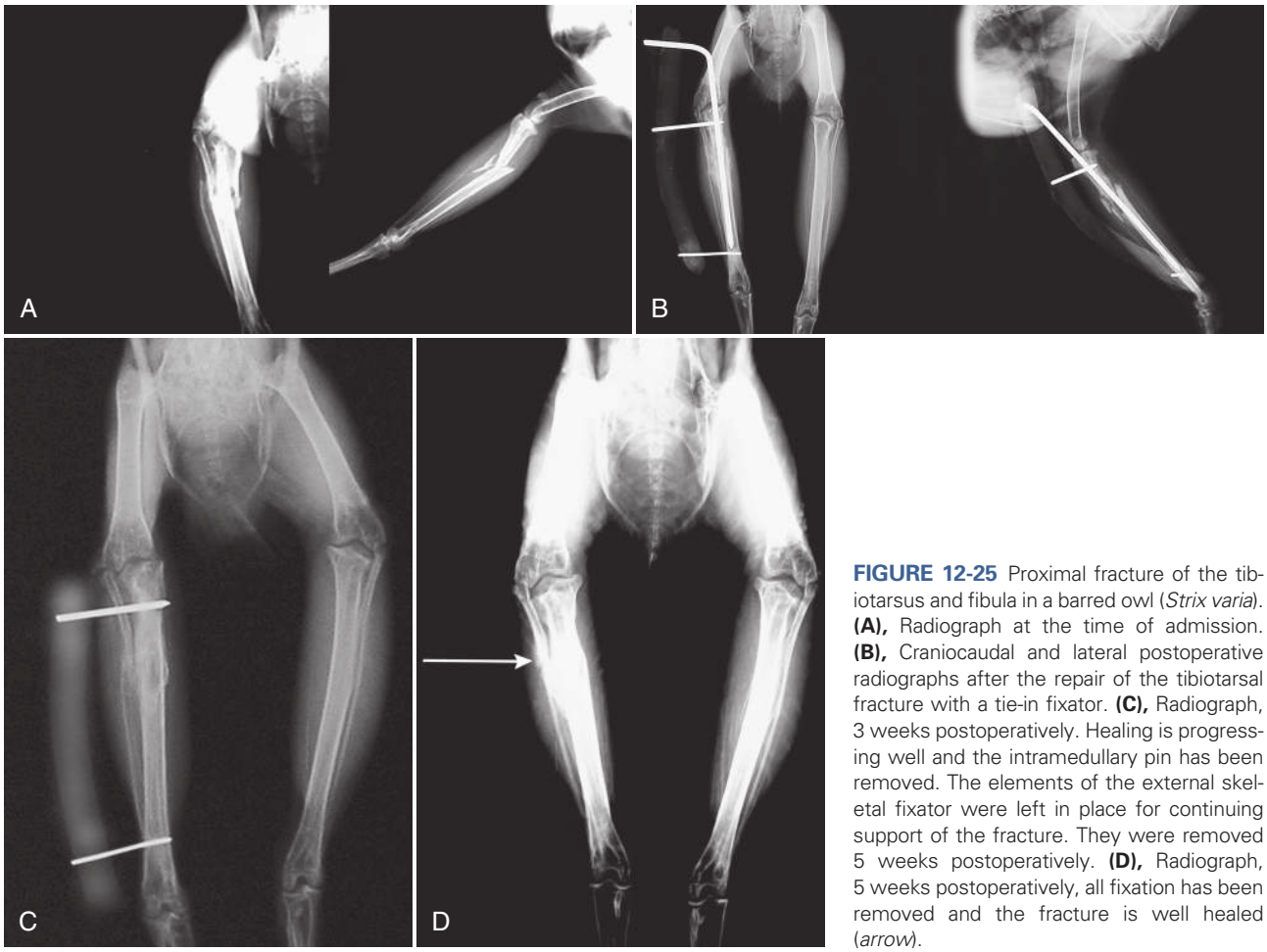


FIGURE 12-25 Proximal fracture of the tibiotarsus and fibula in a barred owl (*Strix varia*). **(A),** Radiograph at the time of admission. **(B),** Craniocaudal and lateral postoperative radiographs after the repair of the tibiotarsal fracture with a tie-in fixator. **(C),** Radiograph, 3 weeks postoperatively. Healing is progressing well and the intramedullary pin has been removed. The elements of the external skeletal fixator were left in place for continuing support of the fracture. They were removed 5 weeks postoperatively. **(D),** Radiograph, 5 weeks postoperatively, all fixation has been removed and the fracture is well healed (*arrow*).

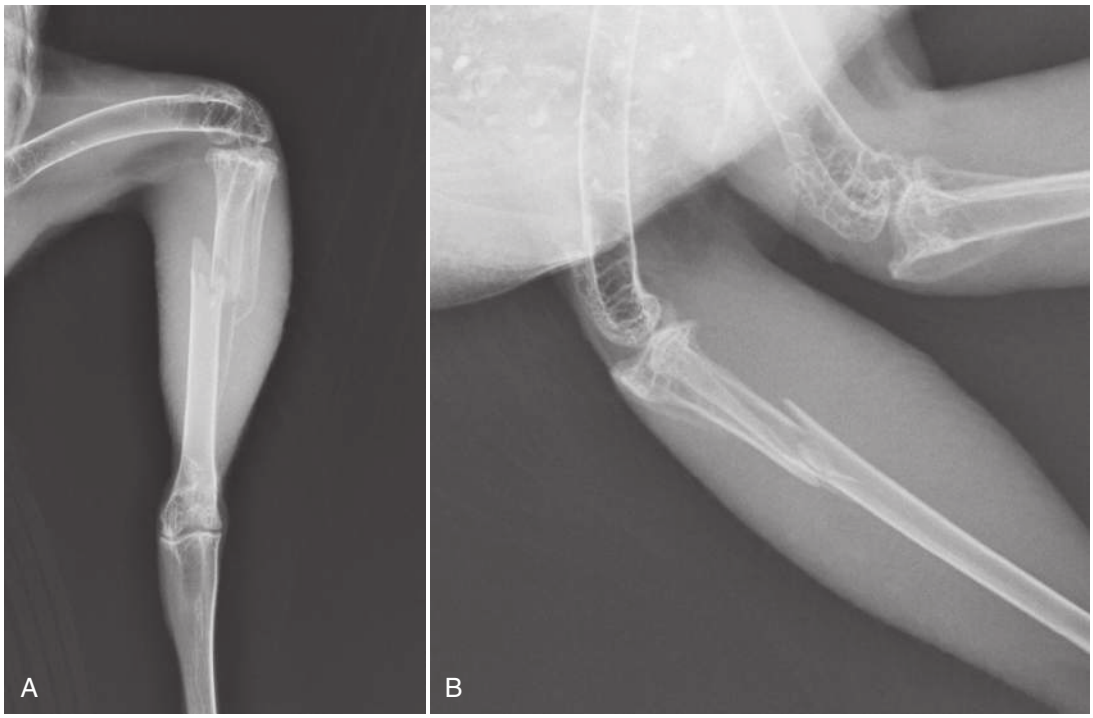
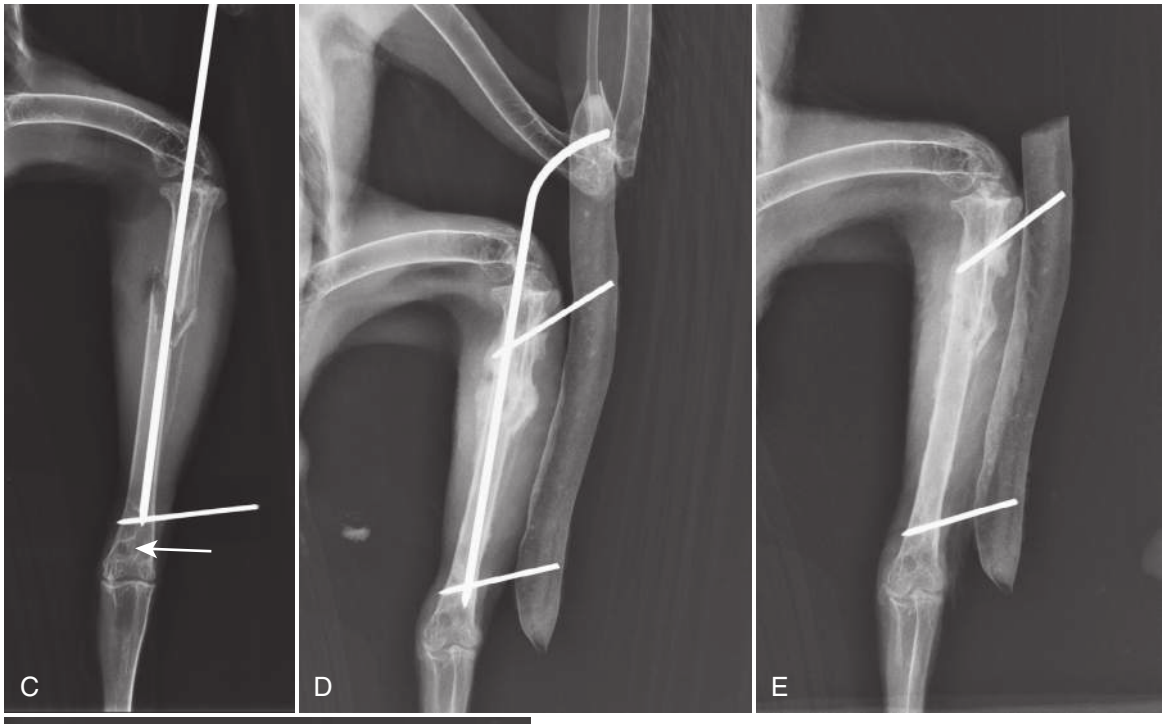


FIGURE 12-26 This red-tailed hawk (*Buteo jamaicensis*) was admitted with a closed, oblique proximal fracture of the tibiotarsus. Admission radiographs **(A, B),** ventrodorsal (VD) and lateral, respectively.

Continued



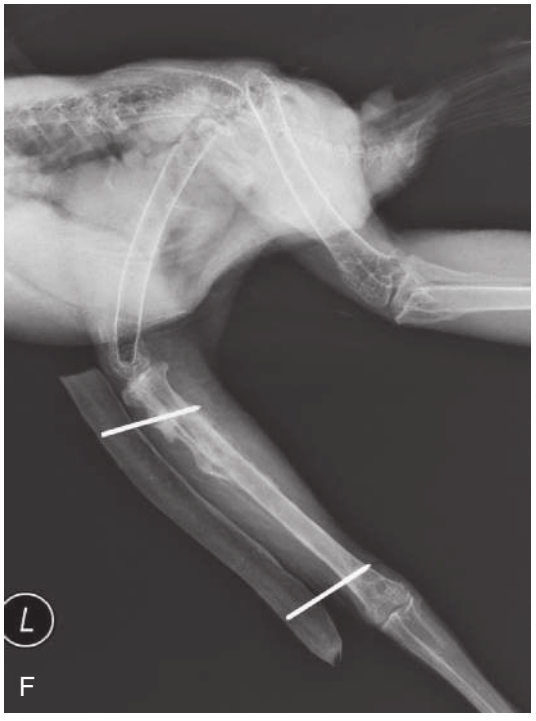


FIGURE 12-26, cont'd **(C),** Intraoperative radiograph (VD) for assessment of placement of the distal external skeletal fixator (ESF) pin and the intramedullary (IM) pin. The IM pin was placed first using a normograde approach from the medial aspect of the stifle joint. Note that the IM pin was driven to a point proximal to the supratendinal ridge (*arrow*). The ESF pin was placed from lateral to medial after the insertion of the IM pin. **(D),** Radiograph (anterior–posterior [AP]) taken at 2 weeks postoperatively shows completed fixator with acrylic bar on the lateral side of the leg. Callus formation is evident and the IM pin was removed. **(E, F),** Radiographs taken at 4 weeks postoperatively in AP **(E)** and lateral **(F)** positions showing healing and near complete remodeling of the fracture. The remaining fixation was removed at this point.

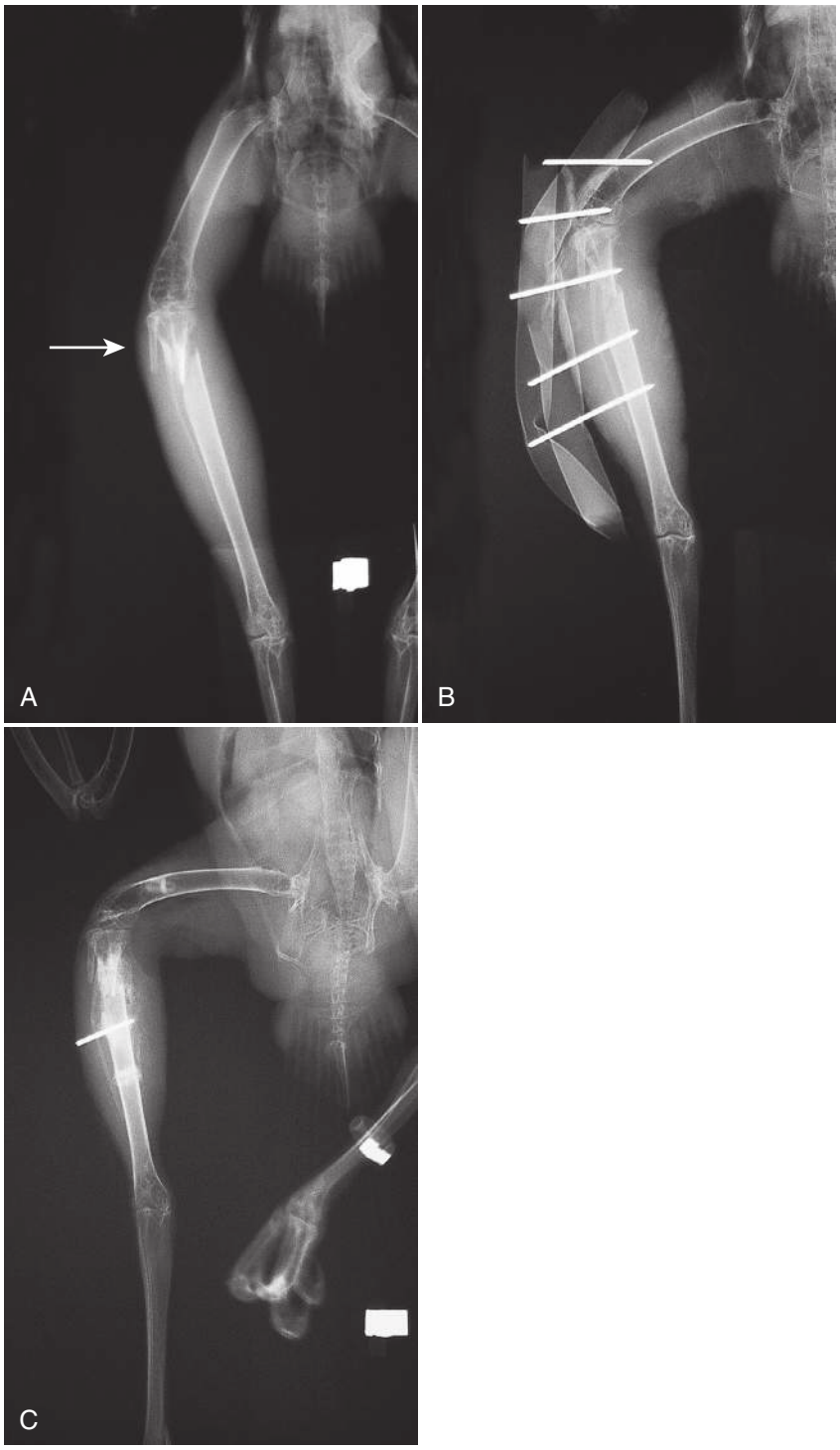


FIGURE 12-27 Method of transarticular fixation for proximal tibiotarsal fractures. **(A),** This Cooper's hawk (*Accipiter cooperi*) fractured its proximal tibiotarsus in a struggle with a cock pheasant. **(B),** The proximal fragment was too short to be stabilized adequately with a conventional type II fixator. Accordingly, a type I transarticular fixator was applied, consisting of a 0.045-inch diameter positive-profile acrylic interface threaded pins, with two pins distal to the fracture: one pin in the short proximal tibiotarsal fragment, and two in the femur. A piece of 3/8-inch (9.6-mm) diameter Penrose drain was pressed over the pins and the leg was positioned with the stifle joint flexed as in a normal perching configuration. The tubing was filled with horse-hoof repair acrylic. The fracture site was accessed from the medial side and the alignment of the bones after reduction was visually observed while the fixator bar was applied and the acrylic cured. Where the tubing became constricted following the acute angle at the stifle, some of the tubing was removed after the acrylic cured and the concave surface of the flexure was reinforced with additional acrylic material. **(C),** The fracture was slow to heal but union was achieved in 27 days. One pin had broken below the skin line and was left in place. Full function returned in 4 months.

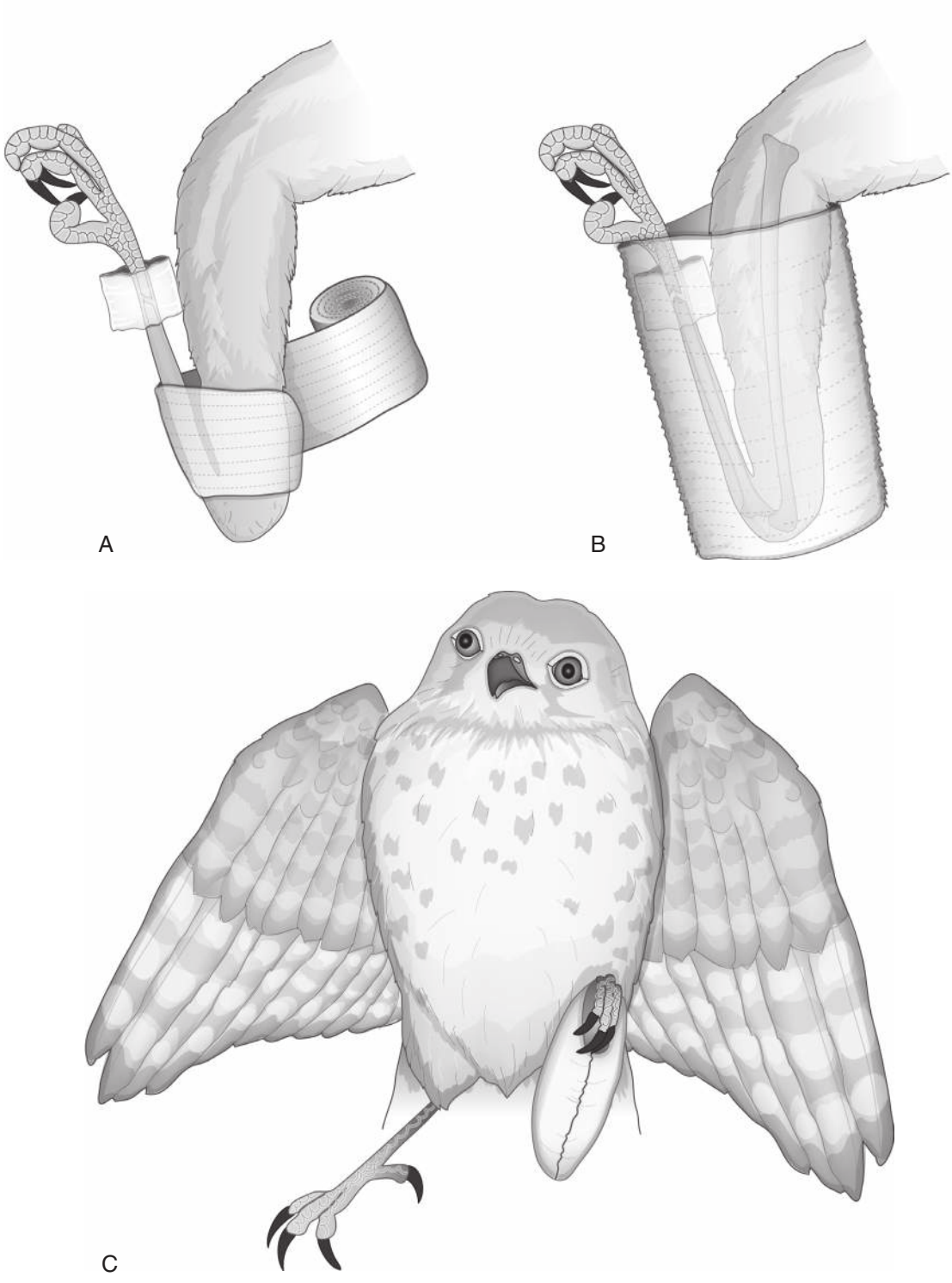


FIGURE 12-28 Tarsometatarsal fractures. **(A),** Coaptation by tape splint **(B, C)** combined with taping the hock in flexion.

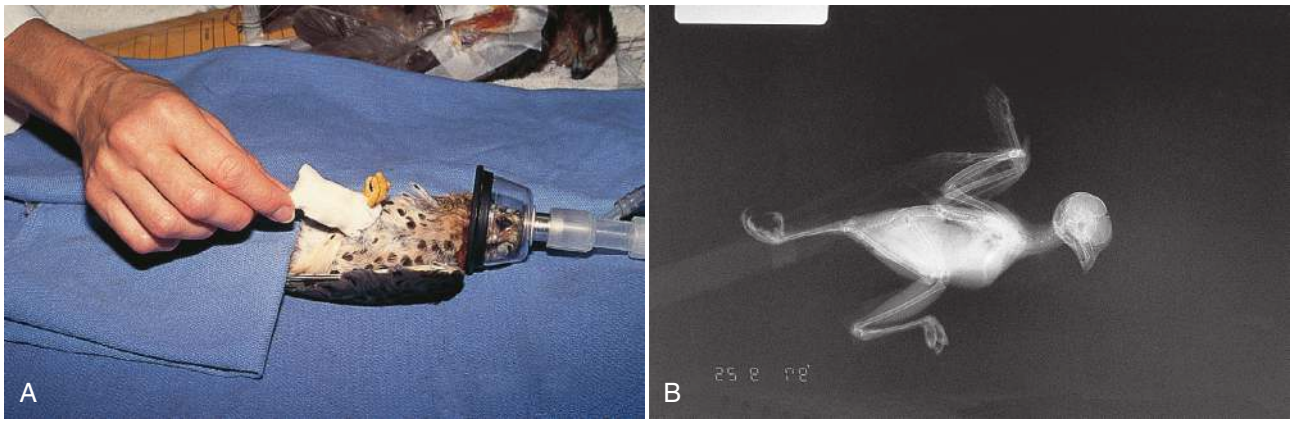


FIGURE 12-29 This American kestrel (*Falco sparverius*) presented with a closed, midshaft tarsometatarsal fracture. **(A),** The fracture was stabilized with an Altman tape splint and the leg was splinted to the tibiotarsus using conforming gauze and Vetrap applied directly to the leg. **(B),** The fracture healed uneventfully in 18 days.

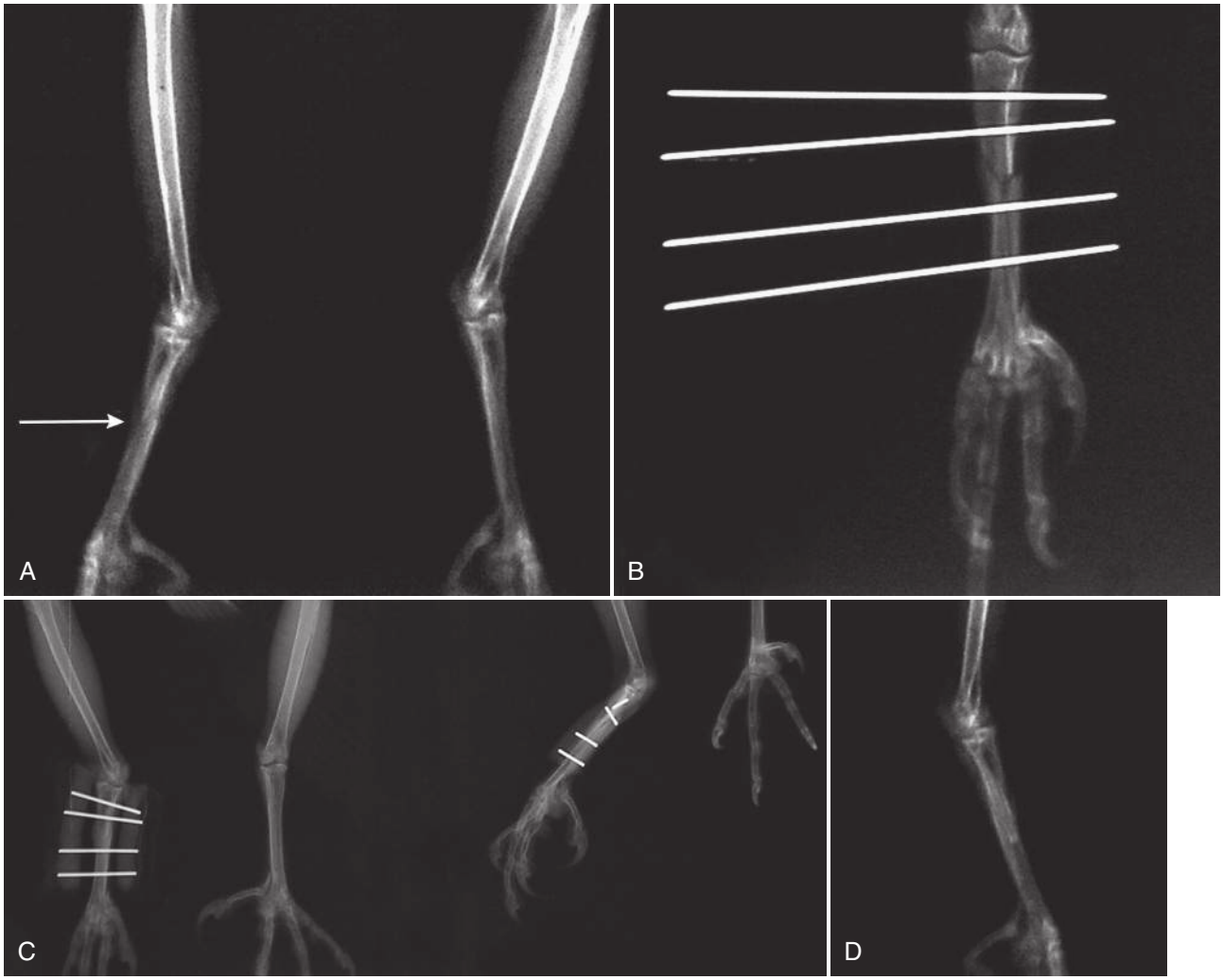


FIGURE 12-30 This prairie falcon (*Falco mexicanus*) was presented with an open transverse fracture of the tarsometatarsus. **(A** to **C),** To facilitate access to the wound for regular management, a type II fixator was applied. **(D),** Healing was complete in 35 days.

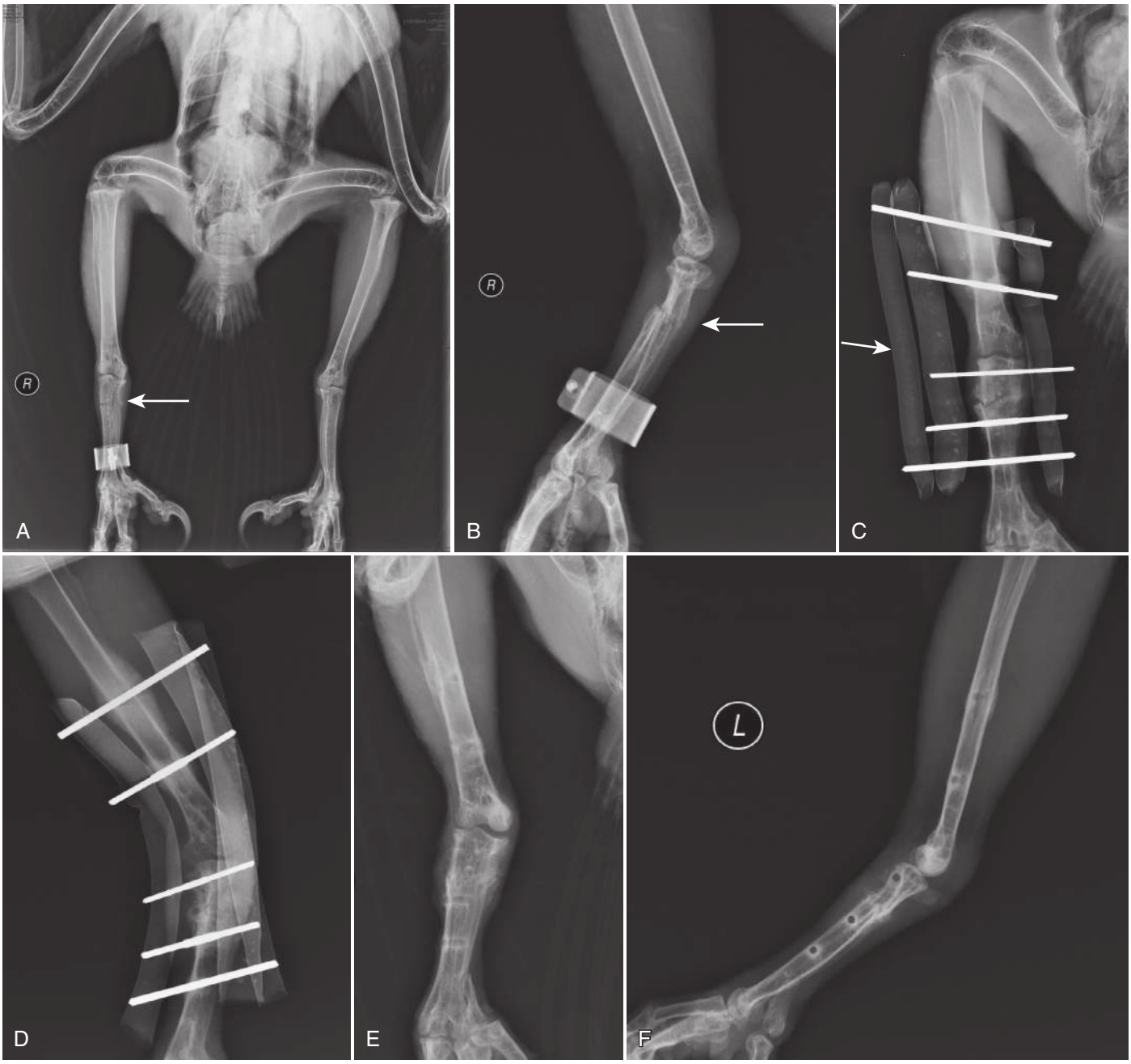


FIGURE 12-31 Tarsometatarsal repair in a bald eagle using a type II transarticular fixator. Radiographs in ventrodorsal (VD) **(A)** and mediolateral **(B)** projections of a bald eagle that presented with a closed, transverse proximal fracture (*arrows*) of the tarsometatarsal bone. A transarticular, reinforced type II acrylic external skeletal fixator (ESF) pin was chosen as the method of fixation. These radiographs taken at 3 weeks post-operatively in VD **(C)** and mediolateral **(D)** views. In the mediolateral view, the fixed joint angle can be seen, as well as the center-threaded pins used in three locations to anchor the fixator. In the VD view, the lateral most acrylic bar is spanning the fixator from the most proximad ESF pin to the most distal (*arrow*) to reinforce the construct. **(E, F),** Radiographs taken 8 weeks postoperatively show a fracture healed with the hardware removed. Note the anterior location of the pin holes on the tarsometatarsus. The eagle was over-wintered and released at 8 months postoperatively with a radio transmitter. At 2 months postrelease it had moved over 600 miles from the release site.

POSTOPERATIVE MANAGEMENT OF LONG BONE FRACTURE REPAIR PATIENTS

Postoperatively, patients are inspected within 24 hours of surgery most often under isoflurane anesthesia. In uncomplicated cases, bandaging material is removed and the surgical site is cleaned. A thin layer of a triple antibiotic ointment (e.g., neomycin, bacitracin, polymyxin B) is applied over the suture line and pin tracts and a light absorbent bandage is reapplied. Complicated open fractures require daily wound treatment until granulation is well under way. Bactericidal cephalosporin antibiotics are given perioperatively (e.g., cefotaxime [Claforan][i]) and patients are maintained on oral enrofloxacin (Baytril[j]) or clavulanated amoxicillin (Clavamox[k]) for up to 5 days postoperatively. In patients with open contaminated fractures and infected bones, clindamycin (Antirobe[l]) is used instead. Radiographs are taken 10 to 14 and 20 to 24 days postoperatively and at biweekly intervals thereafter, if needed. Up to 10 days postoperatively adjustments in alignment can be made. By 21 days, uncomplicated fractures will be well on their way to complete healing and partial dismantling (dynamic destabilization; Egger, 1993) of the fixator may take place. Some fractures will be healed already. If healing is not progressing well, evidence of sequestra will be seen radiographically at around 3 weeks. Sequestra should be surgically removed when the extent is clear and reassessment of the patient and the repair process should be made accordingly. In uncomplicated cases, all fixations should be removed by 6 weeks. Bone strength is usually substantial at this point, and the patient can be allowed full use of the limb.

PHYSICAL THERAPY FOR MANAGING WING FRACTURES

Physical therapy in the form of passive range of motion (PROM) exercise is started 1 to 2 days postoperatively for humeral fractures and at approximately 10 days for other wing fractures. The patient is typically anesthetized for this. PROM is done in 5 minute sessions twice weekly for the first 1 to 2 weeks, after which no further gains are likely to be achieved. Stretch and hold exercises are alternated with range of motion movements to the extent the limb will allow at any given time (Fig. 12-32). Care is taken not to overextend the limb during these exercises.

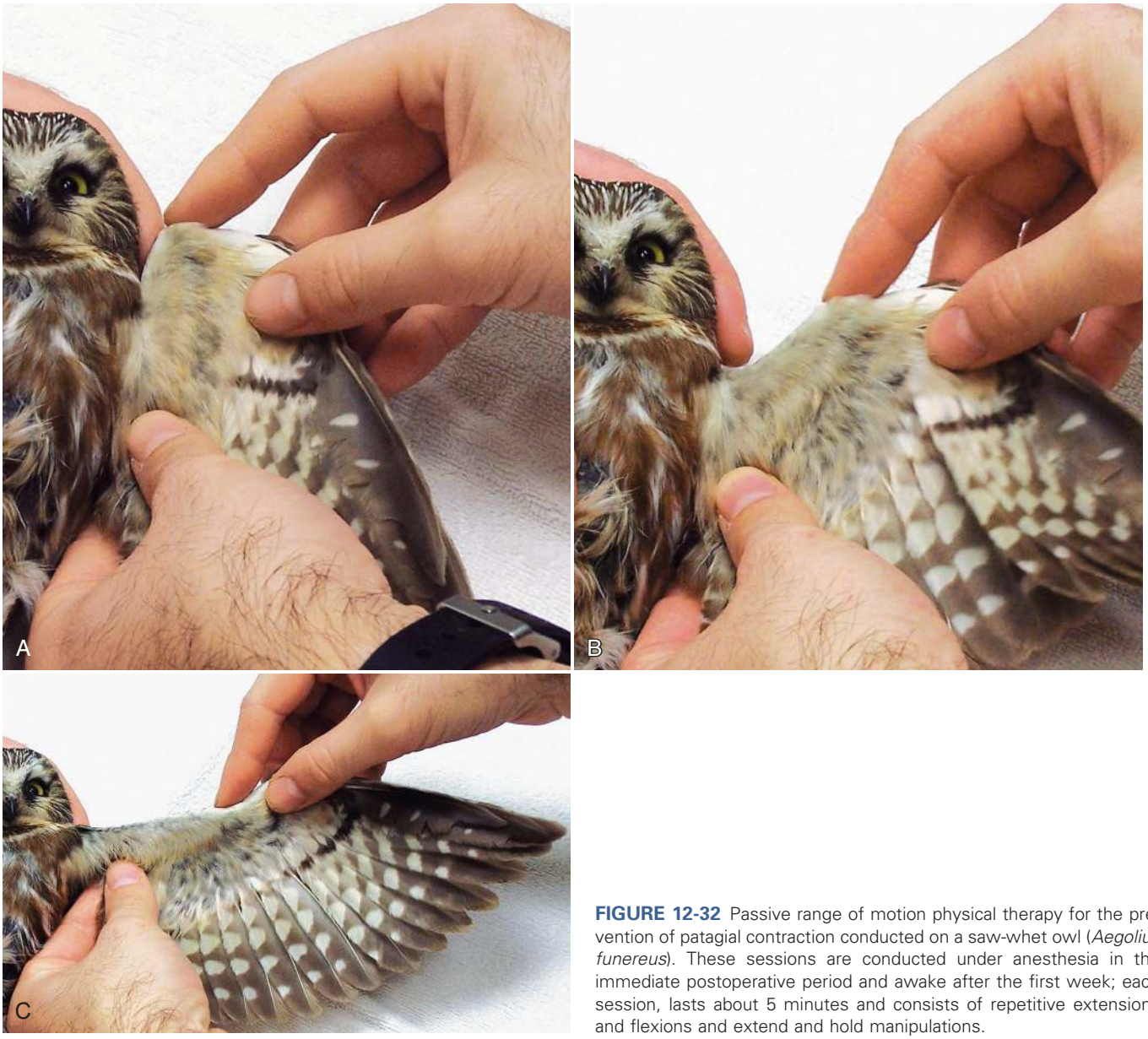


FIGURE 12-32 Passive range of motion physical therapy for the prevention of patagial contraction conducted on a saw-whet owl (*Aegolius funereus*). These sessions are conducted under anesthesia in the immediate postoperative period and awake after the first week; each session, lasts about 5 minutes and consists of repetitive extensions and flexions and extend and hold manipulations.

Management of the Patagium

The patagium is a web of elastic fibers that stretches with wing extension to form the leading edge of the wing and between the shoulder and the carpus. Severe contraction of the patagium often accompanies humeral fractures that have been coaptively supported with a figure eight bandage and/or not provided with range of motion physical therapy postoperatively. The bone may heal satisfactorily, but there can be severe restriction to wing extension after the fracture is healed (see Fig. 12-27). There are two ways of preventing this problem: (1) apply adequate fixation to the fracture, which allows full range of motion postoperatively without additional immobilization by bandaging (e.g., a TIF), and (2) institute passive physical therapy and patagial massage within the first postoperative week and maintain this throughout the healing period. This is performed under isoflurane anesthesia and consists of an approximately 5 minute session performed every 2 days. The exercises consist of range of motion movements and stretch and hold manipulations (Fig. 12-33). Particular attention needs to be paid to focal areas of thickening along the leading edge of the wing (ligamentum propatagialis). Kneading and stretching of this ligament will minimize or remove any thickening. By instituting this procedure beginning on the second postoperative day, contraction problems of the patagium and reduction in wing extension can be avoided.

Lacerations of the patagium may be managed either by suturing or by secondary intention healing. However, the elastic fibers of the patagium do not hold sutures and the area of the wound should be protected from movement during healing. A suitable method for providing this protection consists of using a manila cardboard (file folder) stent cut to a shape and size approximately 20% larger than the wound area. After wound dressing material has been applied to the wound, this cardboard is placed over the wound. Sutures are placed through the patagium and through the cardboard, working around the perimeter of the latter (Fig. 12-34). Because of the

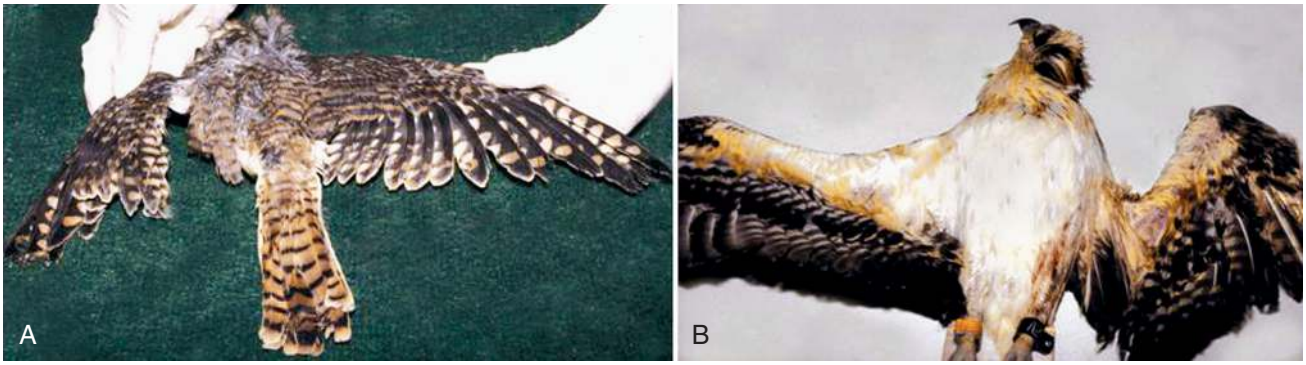


FIGURE 12-33 Severe patagial contraction can occur during the healing of a humeral fracture if a figure eight bandage is used to provide coaptive support (**[A];** American kestrel [*Falco sparverius*]) and no postoperative physical therapy is given (**[B];** osprey [*Pandion haliaetus*]).

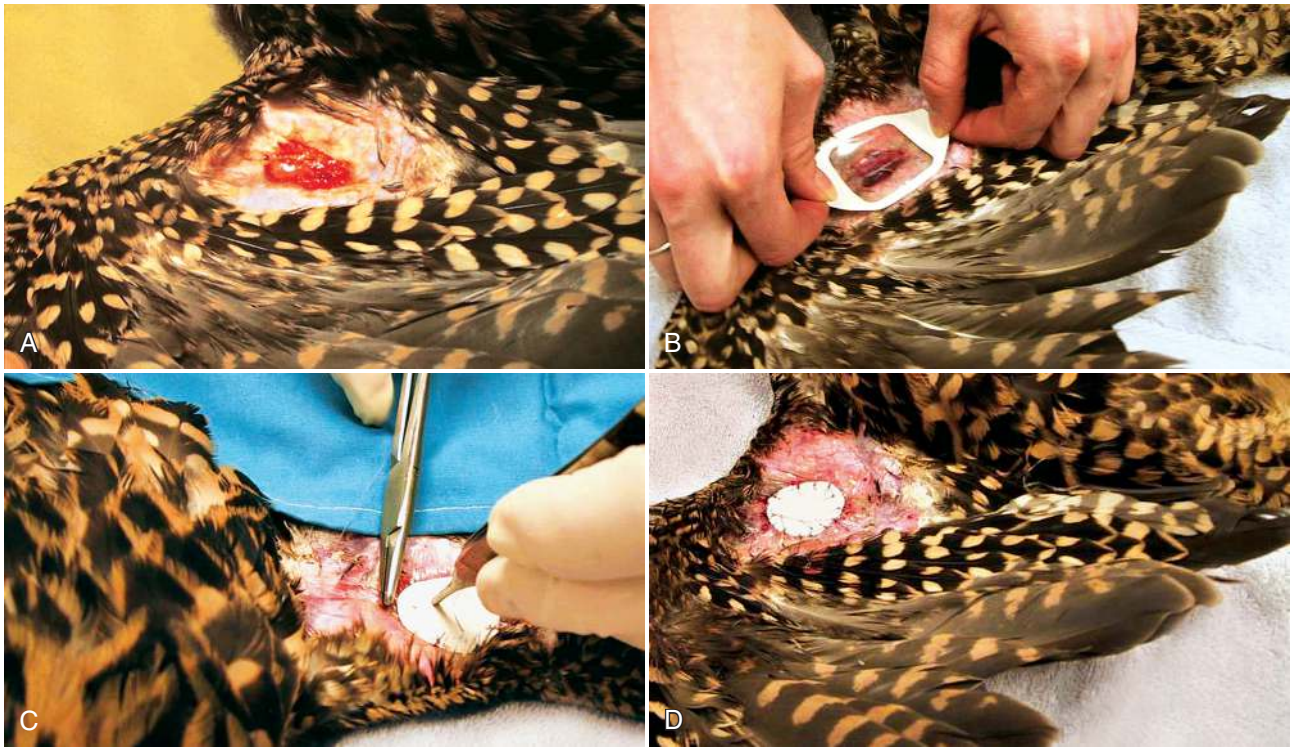


FIGURE 12-34 Management of a patagial wound using a cardboard stent. **(A),** Nonperforating wound on ventral side of patagium (hybrid falcon). **(B),** Application of wound dressing material over wound. **(C),** Suturing a cardboard stent over the treated and dressed wound. **(D),** Completed stent in place. The wing should be bound to the body and subjected to passive range of motion exercise approximately twice weekly.

loosening of the sutures in the patagium, this stent is replaced at weekly intervals or more often as needed, until the wound is healed (see Chapter 11).

REFERENCES

Ackerman J, Redig PT: Surgical repair of elbow luxations in raptors, *J Avian Med Surg* 11:247–254, 1997.

Beaufrere H, Ammersbach M, Nevarez J, et al: Successful treatment of a radioulnar synostosis in a Mississippi kite (*Ictinia mississippiensis*), *J Avian Med Surg* 26:94–100, 2012.

Bennett RA: Orthopedic surgery. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Bueno I, Redig PT, Rendahl AK: External skeletal fixator intramedullary pin tie-in for the repair of tibiotarsal fractures in raptors: 37 cases (1995-2011), *JAVMA* 247(10):1154–1160, 2015.

Christen C, Fischer I, von Rechenberg B, et al: Evaluation of a maxillofacial miniplate compact 1.0 for stabilization of the ulna in experimentally induced ulnar and radial fractures in pigeons (*Columba livia*), *J Avian Med Surg* 19(3):185–190, 2005.

Egger EL: External skeletal fixation—general principles. In Slatter DH, editor: *Textbook of small animal surgery*, ed 2, Philadelphia, PA, 1993, WB Saunders.

Gull JM, Saveraid TC, Szabo D, et al: Evaluation of three miniplate systems for fracture stabilization in pigeons (*Columba livia*), *J Avian Med Surg* 26(4):203–212, 2012.

Harcourt-Brown NH: Foot and leg problems. In Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *Manual of raptors, pigeons and waterfowl*, Ames, IA, 1996, Iowa State University Press.

Harcourt-Brown NH: Tendon repair in the pelvic limb of birds of prey. In Lumeij JT, Remple JD, Redig PT, et al, editors: *Raptor biomedicine III*, Lake Worth, FL, 2000, Zoological Education Network.

Hess R: Orthopedic techniques for the pelvic limb. In Fudge AM, Redig PT, editors: *Seminars in avian and exotic pet medicine*, Philadelphia, PA, 1994, WB Saunders.

Hollamby S, Dejardin LM, Sikarskie JG, et al: Tibiotarsal fracture repair in a bald eagle (*Haliaeetus leucocephalus*) using an interlocking nail, *J Zoo Wildl Med* 35(1):77–81, 2004.

Johnston MS, Thode IIIHP, Ehrhart NP: Bone transport osteogenesis for reconstruction of a bone defect in the tibiotarsus of a yellow-naped amazon parrot (*Amazona ochrocephala auropalliata*), *J Avian Med Surg* 22(1):47–56, 2008.

Montgomery RD, Crandall E, Bellah JR: Use of a locking compression plate as an external fixator for repair of a tarsometarsal fracture in a bald eagle (*Haliaeetus leucocephalus*), *J Avian Med Surg* 25:119–125, 2011.

Redig PT: Non-surgical management of avian fractures. In Harrison GJ, Harrison LR, editors: *Clinical avian medicine and surgery*, Philadelphia, PA, 1986a, WB Saunders.

Redig PT: Evaluation and non-surgical management of avian fractures. In Harrison GJ, Harrison LR, editors: *Clinical avian medicine and surgery*, Philadelphia, PA, 1986b, WB Saunders.

Redig PT: The use of a hybrid fixator (intramedullary pin-external skeletal fixator) for stabilization of long bone fractures in raptors—a review of 26 cases. In Lumeij JT, Remple JD, Redig PT, et al, editors: *Raptor biomedicine III*, Lake Worth, FL, 2000, Zoological Education Network.

Sanchez-Migallon Guzman D, Bubenik LJ, Lauer SK, et al: Repair of a coracoid luxation and tibiotarsal fracture in a bald eagle (*Haliaeetus leucocephalus*), *J Avian Med Surg* 21:188–195, 2007.

Van Wettere AJ, Wallace LJ, Redig PT, et al: Mechanical evaluation of various external skeletal fixator–intramedullary pin tie-in configurations using a tubular plastic bone model, *J Avian Med Surg* 23(4):263–276, 2009a.

Van Wettere AJ, Redig PT, Wallace LJ, et al: Mechanical evaluation of external skeletal fixator—intramedullary pin tie-in configurations applied to cadaveral humeri from red-tailed hawks (*Buteo jamaicensis*), *J Avian Med Surg* 23(4):277–285, 2009b.

LUXATIONS

FRACTURES AND LUXATIONS OF THE CORACOID BONE

The coracoid is a stout strong bone that connects the cranial edge of the sternum to the shoulder joint complex. It opposes the powerful contraction of the major pectoral muscle during the downstroke of the wing. It is fractured or luxated most often when the bird has a frontal collision with an object. The force required to break this bone must be very large, hence, a clinician should be looking for an associated injury such as fracture or luxation of other shoulder elements (scapula and furcular), internal organ damage (ruptured air sacs and displaced liver), or retinal detachment that may have a bearing on ultimate disposition of the case and the decision to proceed initially. Both lateral and ventrodorsal radiographs are necessary to detect and properly assess coracoid injury.

Surgical and conservative approaches to management of coracoids have been successful. Surgical options have included plating and the insertion of an intramedullary pin. Sanchez-Migallon Guzman *et al.* (2007) and Davidson *et al.* (2005) reported the use of a plate to repair luxation and fracture of the coracoid, respectively, in bald eagles leading to their release back to the wild. Unfortunately, the majority of birds seen with coracoid fractures are much smaller and the difficult access to this bone, requiring detachment and elevation of the major pectoral muscle from the keel, renders surgical management problematic. In a large number of small- to medium-sized raptors, intramedullary pinning was found to have only a 20% success rate (P.T. Redig, unpublished data) for full recovery. Conversely, evaluation of conservative management (cage rest) over a large number of raptors, including eagles, has shown that, despite the important structural role played by the coracoid, satisfactory recovery allowing flight capability that appears to be sufficiently strong for release to the wild has been realized (Scheelings, 2014) (Fig. 12-35). Occasional, but rare, problems with this method (Bennett, 1997) have been reported. Conservative management is recommended over surgical management for nearly all cases of coracoid fractures.

LUXATIONS OF THE ELBOW

Moderate success has been obtained in the surgical repair of caudodorsal luxations of the elbow (Ackerman and Redig, 1997). Such intervention must take place early in the postinjury period (i.e., at 2 to 3 days) to be successful. A curved incision is made over the lateral surface of the wing that includes the distal end of the humerus and the proximal portion of the antebrachium. The tendon of origin of the supinator muscle, if still intact, is transected to provide exposure of the joint. The end of the ulna is levered into place by inserting a flat periosteal elevator in between the proximal ulna and the dorsal humeral condyle and levering the ulna distally until it aligns with the humeral condyle. Application of traction to the distal ulna is helpful in this maneuver. The cut ends of the tendon of the supinator are sutured. A pseudocollateral ligament is made by suturing the edge of the triceps tendon to the common digital extensor tendon with the surgeon's preferred choice of suture material. After closure, the elbow is stabilized with a transarticular external skeletal fixator for 7 to 10 days. Physical therapy is instituted following removal of the fixator and the wing is kept immobilized in between periods of coaptation.

Minor luxations of the elbow can be managed more effectively compared with simple coaptation by incising the skin over the elbow and suturing the edge of the triceps tendon to the common digital

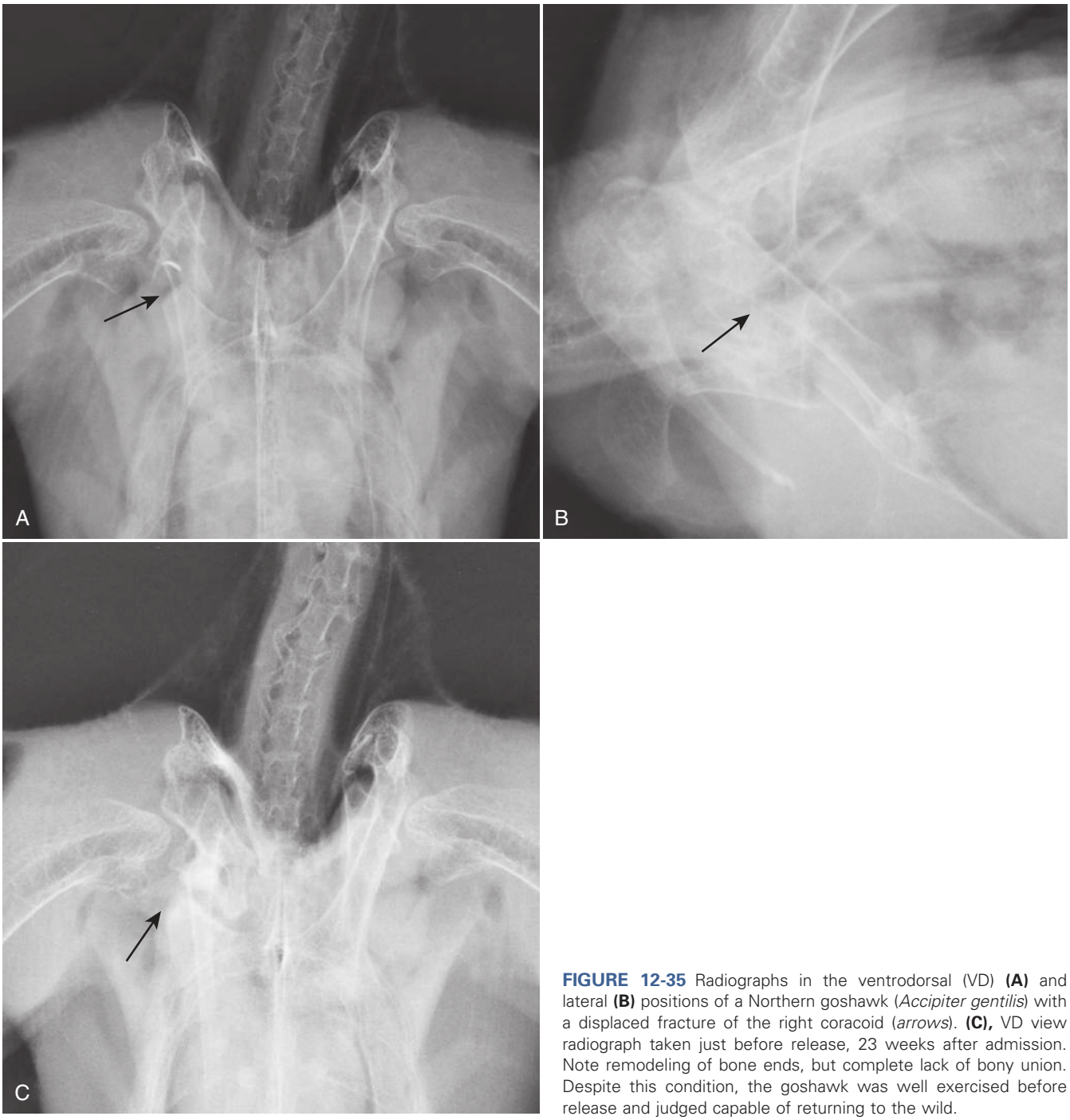


FIGURE 12-35 Radiographs in the ventrodorsal (VD) **(A)** and lateral **(B)** positions of a Northern goshawk (*Accipiter gentilis*) with a displaced fracture of the right coracoid (*arrows*). **(C),** VD view radiograph taken just before release, 23 weeks after admission. Note remodeling of bone ends, but complete lack of bony union. Despite this condition, the goshawk was well exercised before release and judged capable of returning to the wild.

extensor. This procedure is highly recommended on a preemptive basis for patients with proximal radial fractures (see Fig. 12-12), because these often have a degree of ulnar subluxation associated with them that is not apparent upon physical examination or by radiology.

LUXATIONS OF THE STIFLE

Stifle luxations may be repaired by a transarticular external skeletal fixator involving the implantation of threaded ESF pins in the femur and tibiotarsus. These are then connected by an acrylic bar molded to fit the contour of the stifle fixed in a partially flexed perching position (see Fig. 12-21). If the acrylic is molded within a latex tube (Penrose drain), the flexion at the stifle will create a constriction and a thinned area in the acrylic bar. This is remedied by placing a reinforcing dollop of acrylic material in the acute angle after removing the latex tubing from the surface of the fixator bar.

Another method of stifle stabilization developed and tested both experimentally and clinically by Villaverde *et al.* (2005) is the use of a nonabsorbable braided or monofilament suture material that is placed through holes drilled in the distal femur and proximal tibiotarsus and threaded between the two, passing caudal to the patellar tendon. It resembles the use of an extracapsular method used in

dogs where the suture material is anchored to the lateral fabella. Further clinical assessment is needed to establish its utility in birds. Luxations of the hock have reportedly been repaired in a similar manner (Zsivanovits, 2011).

Luxations of the shoulder elements and the hip are typically managed with cage rest and, in cases involving the shoulder, with coaptive bandaging of the wing to the body for a period of 10 to 14 days. Excision arthroplasty has been reported for management of femoral head and neck fractures (Burgdorf-Moisuk *et al.*, 2011).

ACKNOWLEDGMENTS

The author wishes to express sincere thanks to veterinary faculty at The Raptor Center, including Dr. Julia Ponder and Dr. Michelle Willete and residents Dr. David Howard, Dr. Elizabeth Stone, Dr. Janette Ackermann, Dr. Jalila Abu, Dr. Juli Ponder, Dr. Hugo Lopez, and Dr. Miguel Saggese, who have been instrumental in the long progression of the development and testing of these methods, along with the veterinary technical staff of TRC, including Lori Arent MS, Toni Guarnera CVT, and Jane Goggin, who expend countless hours and great skill and care in the postoperative management of orthopedic patients. Similar thanks go to Dr. Luis Cruz who was a coauthor on this chapter in the 2nd edition. Very special thanks go to Dr. Chikako Akaki, who produced the original illustrations for this text. Additionally, revisions of existing illustrations, along with new ones for the 2nd edition and reused herein, were provided by Giovanny Rojas, a professional advertising/graphic designer who volunteered his expertise. Portions of this material have been published previously in the *Proceedings of the Association of Avian Veterinarians* and will also be reproduced in an upcoming book on avian anatomy and orthopedics by P.T. Redig, S. Orosz, and S. Echols.

REFERENCES

Ackerman J, Redig PT: Surgical repair of elbow luxations in raptors, *J Avian Med Surg* 11:247–254, 1997.

Bennett RA: Orthopedic surgery. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Burgdorf-Moisuk A, Whittington JK, Bennett RA, et al: Successful management of simple fractures of the femoral neck with femoral head and neck excision arthroplasty in two free-living avian species, *J Avian Med Surg* 25:210–215, 2011.

Davidson, JE, Mitchell MA, Ramirez S: Plate fixation of a coracoid fracture in a bald eagle (*Haliaeetus leucocephalus*), *J Avian Med Surg* 19(4):303–312, 2005.

Sanchez-Migallon Guzman D, Bubenik LJ, Lauer SK, et al: Repair of a coracoid luxation and tibiotarsal fracture in a bald eagle (*Haliaeetus leucocephalus*), *J Avian Med Surg* 21:188–195, 2007.

Scheelings TF: Coracoid Fractures in Wild Birds: A Comparison of Surgical Repair Versus Conservative Treatment, *J Avian Med Surg* 28(4):304–308, 2014.

Villaverde S, Benito de la Víbora JB, González R, et al: Management of the femorotibial luxation by coaptation splinting, intramedullary pins, external skeletal fixator (ESF) and tension bands: Pilot study in domestic pigeons (*Columba livia domestica*). *Proceedings of the 8th European Association of Avian Veterinarians*, Arles, France, pp 196–202, 2005.

Zsivanovits P: Repair techniques in case of intertarsal joint luxation: two case reports. *Proceedings of 12th European Association of Avian Veterinarians*, Madrid, Spain, pp 58, 2011.

FURTHER READING

Bennett RA, Kuzma AB: Fracture management in birds, *J Zoo Wildl Med* 23:5–38, 1992.

Brown RE, Klemm RD: Surgical anatomy of the propatagium. *Proceedings of the Association of Avian Veterinarians*, pp 176–181, 1990.

Degernes LA, Roe SC, Abrams CF Jr: Holding power of different pin designs and pin insertion methods in avian cortical bone, *Vet Surg* 27:301–306, 1998.

Muller MG, Nafeez JM: *A New Approach for Tibiotarsal Fractures in Falcons with the FixEx Tubulaire Type F.E.S.S.A System*. www.falconhospital.com/Cms_Bin/Cms/doc_manager/fractures. Accessed August 20, 2014.

Piermattei DL, Flo GL: *Brinker, Piermattei and Flo's handbook of small animal orthopedics and fracture repair*, ed 3, Philadelphia, PA, 1997, WB Saunders.

Rochat MC, Hoover JP, Digesualdo CL: Repair of a tibiotarsal varus malunion in a bald eagle (*Haliaeetus leucocephalus*) with a type 1A hybrid external skeletal fixator, *J Avian Med Surg* 19:121–129, 2005.

Simpson GN: Wing problems. In Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *Manual of raptors, pigeons and waterfowl*, Cheltenham, UK, 1996, British Small Animal Association.

PRODUCT REFERENCE LIST

[a]Acrylic Half-pins, IMEX Veterinary Inc., IMEX® Veterinary, Inc. 1001 McKesson Drive Longview, Texas 75604 USA.

[b]Caulk Dental Acrylic, Dentsply International Inc., 211 West Philadelphia Street, York, PA 17405.

[c]Technovit, Jorgensen Laboratories, Inc., 1450 North Van Buren Ave., Loveland, CO 80538.

[d]Hoof Wall Restorative Material, Vettec Inc, 600 East Hueneme Road, Oxnard, CA 93033-8600.

[e]Kling Gauze, Johnson & Johnson Products, Inc., New Brunswick, NJ 08903.

[f]Vetrap, 3M Animal Care Products, St Paul, MN 55144-1000.

[g]SAM Splint, Moore Medical Corporation, PO Box 06050-4066, New Britain, CT 06050-2620.

[h]Isoxsuprine, Geneva Pharmaceuticals, Inc., 2655 West Midway Blvd, Broomfield, CO 80020.

[i]Claforan, Aventis Pharmaceuticals, 399 Interpace Parkway, Parsippany, NJ 07054.

[j]Baytril, Bayer Corporation, Pharmaceutical Division, 400 Morgan Lane, West Haven, CT 06516.

[k]Clavamox, Zoetis Inc., 100 Campus Drive, Florham Park, NJ 07932.

[l]Antirobe, Pharmacia & Upjohn, 100 South Highway 206, Peapack NJ 07977.

[m]Veterinary Thermoplastic Tape, Imex Veterinary Inc., Longview, TX.

EXTERNAL SPLINTING

Jean-Michel Hatt

Splints are a type of bandage applied to long bones and are common in avian medicine when a high degree of immobilization is required. Indications include immobilization of luxation of fractures (including greenstick fractures) and protection of suboptimal surgical fracture repair or joints.

In mammals the most frequently used splint is the Robert Jones bandage. In avian medicine the main splints are the Altman splint, the Spica splint, and the U-shaped splint (Metasplint type). Table 12-1 summarizes different methods of fracture splinting in relation to the affected bone. Splints are typically used in small birds, with a body weight below 200 g as the primary method for fracture repair. In larger birds surgical fixation is the preferred method for fracture repair. Any method of splint will only be successful for fracture treatment if the joints proximal and distal to the fracture are adequately immobilized.

The Altman splint (Fig. 12-36) is made from adhesive tape applied bilaterally over the lower limb. Depending on the size of the bird, several layers of tape have to be used and reinforcement of the edges

TABLE 12-1 Splint Techniques and Bandages for the Immobilization of Different Fracture Types

	Altman Type Tape Splint	U-Shaped Splint	Spica Splint	Comments
Coracoid/clavicle/ scapula				• Figure eight bandage including thorax • Cage rest alone 5-6 weeks • Surgery may be recommended in coracoid fractures, especially when fracture ends are dislocated
Humerus			X	• Figure eight bandage often a better choice, as it is easier and tolerated better by most birds • Bandage only for short-term stabilization, before surgery
Radius/ulna				• Figure eight bandage best choice for stabilization
Metacarpal bones		X		• This splint can also be used for protection of carpal bones, especially in wild birds of prey
Pelvis				• Cage rest for 5-6 weeks • Prognosis is good as long as there are no neurological deficits
Femur			X	• The femur should be immobilized in—a perching position, because this will automatically bring the fracture ends in physiological apposition
Tibiotarsus	X	X		• Immobilization in a perching position
Tarsometatarsus	X	X		• Immobilization in a perching position
Phalanges	X	X		• Immobilization with a "shoe" (e.g., made from cardboard), which is taped to the phalanges

In birds with a body weight greater than 200 g, splints are usually not recommended as the primary method of fracture treatment.

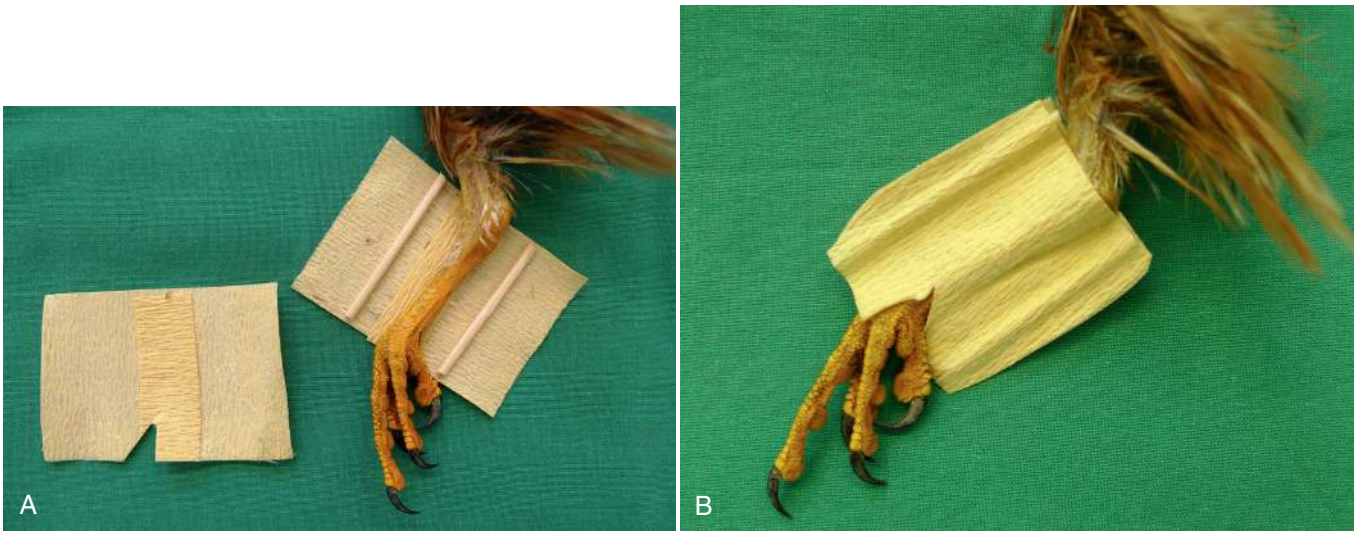


FIGURE 12-36 Altman splint in a kestrel (*Falco tinnunculus*) to immobilize a metatarsal bone. **(A),** The second tape has not yet been applied. **(B),** Additional strength is achieved by including wooden toothpicks in the splint.

is possible by inserting a toothpick or similar material. When applying the tape, the leg is immobilized in a perching position.

The spica splint is used to immobilize the humerus or the femur and the torso is included in the bandage. The U-shaped splint is typically applied to the metacarpus or the phalanges (Fig. 12-37). This method is also used to protect the carpal joints in birds of prey. This is particularly important in wild birds during rehabilitation because some species tend to be nervous and try to escape. Useful materials to make the U-shaped splint are veterinary thermoplastic tape and the SAM splint (i.e., a thin core of aluminium alloy sandwiched between two layers of closed cell foam; Fig. 12-37).

Compared with surgical fracture fixation, splints result in larger callus and take approximately twice as long to heal (i.e., 4 to 8 weeks). The prolonged immobilization of joints can result in loss of joint flexibility. Therefore after 2 weeks at the latest, it is recommended to change the splint under general anesthesia and implement physical therapy to the joint. If splinting is used in the presence of an open fracture, bandage changes need to be performed daily to allow for wound management and antimicrobial treatment.

When applying a splint pay special attention to the distal part of the limb for the occurrence of swelling or color changes, which could indicate impaired vascularization.

USE AND APPLICATION OF THE FESSA EXTERNAL SKELETAL FIXATOR FOR FRACTURE AND LUXATION REPAIR

Jean-Michel Hatt

External skeletal fixation of long bones is arguably the most frequently used method of fracture repair in birds. Several systems, commercially available and self-made, have been used over the years that differ in

FIGURE 12-37 **(A),** U-shaped splint in a sparrow hawk (*Accipiter nisus*) to immobilize a metacarpal fracture. **(B),** After application, but before applying tape on the splint.

the type of connecting bar. The Fixateur Externe du Service de Santé des Armées (FESSA) tubular skeletal fixator is a commercially available system that has proven especially useful in a wide range of skeletal problems in birds (Hatt *et al.*, 2007). A system of screws and holes are used to attach the pins in the tube (Fig. 12-38). The screws of the FESSA system have a hexagonal head filling to pass with an Allen key. The lack of clamps makes the system exceptionally light weight and allows the insertion of more transosseous pins over a small distance than with other commercial systems. Compared with self-made connecting bars made from polymethylmethacrylate, the difference in weight of the FESSA system was found to be clinically irrelevant (Hatt *et al.*, 2007).

In veterinary medicine the application of the FESSA system has been described in domestic mammals, such as small dogs, cats, and rabbits, by several authors (Chancrin *et al.*, 1990; Reichler *et al.*, 1997; Haas *et al.*, 2003). The connecting bar is made of stainless steel or titanium and different models are available with diameters of 6, 8, and 12 mm, for which pins of up to 2 and 2.5 mm, respectively, may be used. The length of the fixators varies from 30 to 200 mm. Pins may be placed perpendicular to the bone or, by exiting through the neighboring hole, at an angle of approximately 30 degrees (Fig. 12-38). In the 6-mm diameter connecting tube one screw is used to secure the pin unilaterally; in the larger systems the pin is secured by two screws bilaterally.

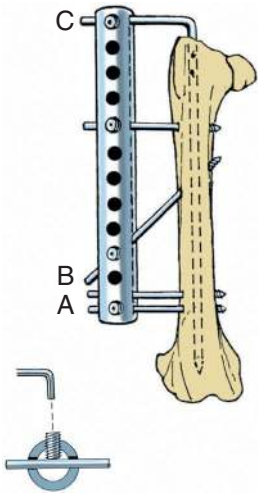


FIGURE 12-38 Schematic view of the FESSA tubular skeletal fixator on an avian femur. Fixation of pin perpendicular (*A*) or at a 30 degree angle (*B*). When used as a type I tie-in external fixation (*C*), the tie-in pin is also secured by the FESSA connecting bar. The insert is a transverse section of the tube showing the fixation of the pin with a screw.

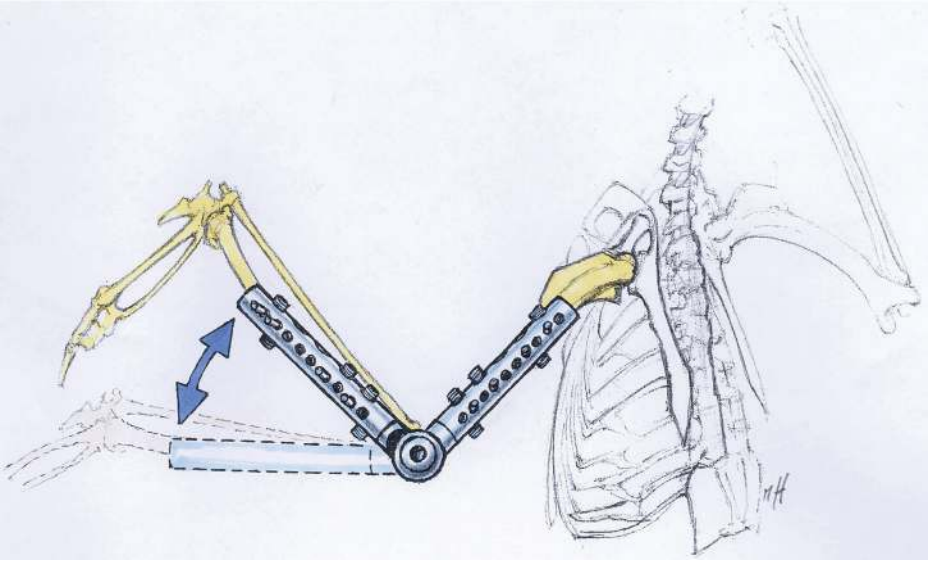


FIGURE 12-39 Schematic view of two FESSA connecting bars that have been linked with a hinge for the treatment of an elbow luxation. The hinge may be loosened temporarily to allow readjustment or physical therapy.

TREATMENT OF FRACTURES

The FESSA system has been used in a wide range of bird species and in birds with a body weight from just below 100 g to several kilograms. It was found to be well tolerated by birds, but psittacine birds sometimes play with the screws and might loosen them. The author suggests that screws should be secured with a drop of tissue glue or by covering the connecting bar with Vetrap. When choosing the connecting tube size the general guide is that its width should correspond to the bone width, and that it should cover at least 70% of the bone's length to reduce the risk of a proximal or distal refracture of the fixator. Two connecting bars can be linked, as shown in Fig. 12-39, to exactly fit the bone length. In curved bones an angular elongation is possible by using a hinge in the FESSA 6- and 8-mm diameter.

When using the FESSA type I or type II fixator the author first applies one pin distal most to the fracture and then secures the connecting bar on that pin (Hatt, 2008). Subsequently the holes in the FESSA fixator are used to guide the pins, which are placed using a low-speed drill or a mini hand chuck for more precise pin placement.

When a type I tie-in external fixation is used the intramedullary pin is inserted first and the exiting part is bent at a 90-degree angle. The connecting bar is applied first to the tie-in pin and the holes in the bar are used as a guide for subsequent pins. Postoperatively a modification of the fixation is easy by adding or removing pins or by loosening the screws and readjusting the connecting bar. The latter can also be used to increase strength of fixation by placing the connecting bar closer to the bone.

Like other external fixation systems the pins are left in place until fracture healing is evident, which typically is 2 to 4 weeks. With the FESSA system it is easy to induce a gradual dynamization of the fracture by selectively removing pins. This increases the loading of the healing fracture, which is intended to stimulate bone remodeling and healing. Following removal of the FESSA system, screws can be cleaned, sterilized, and reused.

TREATMENT OF LUXATIONS

The FESSA system can also be used as a hinged linear external skeletal fixator (HLESF) for the transarticular fixation of luxations (see Fig. 12-39). The hinges, which are provided to connect 6- and 8-mm FESSA tubes, are comprised by two half-spheres connected to each other with a screw. The interim round surface of the two spheres is roughened with a trailing edge or saw-like margin. The maximum angle between the two connecting bars is 180 degrees and the smallest angle is 40 degrees.

The hinge can be loosened to allow readjustments and joint dynamization as with passive range of motion during physical therapy sessions (see Fig. 12-39). Recently the use of the HLESF with the FESSA system has been described for the treatment of experimentally induced stifle luxation in pigeons (Azmanis *et al.*, 2014). Clinical healing was achieved within 6 weeks. Other applications for the HLESF could be elbow luxations, and in larger birds metacarpophalangeal and intertarsal joint luxations. When using the HLESF with physical therapy it is most important to ensure that the hinge is centered over the center of rotation of the joint.

ACKNOWLEDGMENT

The author thanks Matthias Haab from the Department of Horses, Vetsuisse Faculty University of Zurich, for his drawings (see Figs. 12-38 and 12-39).

REFERENCES

Azmanis PN, Voss K, Hatt JM: Evaluation of short-term outcomes of experimental stifle luxation in feral pigeons (*Columba livia domestica*) treated with a hinged transarticular external skeletal fixator and physical therapy, *Int J Appl Res Vet Med* 12:157–166, 2014.

Chancrin JL, Boubee T, Marguin M: Utilisation du fixateur externe du service de santé des armées (FESSA) en chirurgie orthopédique vétérinaire: à propos de 29 cas, *Prat Med Chir Anim* 25:217–223, 1990.

Haas B, Reichler IM, Montavon PM: Use of the tubular external fixator in the treatment of distal radial and ulnar fractures in small dogs and cats, *Vet Comp Orthoped Traumatol* 16:132–137, 2003.
Hatt JM: Hard tissue surgery. In Chitty J, Lierz M, editors: *Raptors, pigeons and passerine birds*, Gloucester, 2008, British Small Animal Veterinary Association.
Hatt JM, Christen C, Sandmeier P: The tubular external skeletal fixator (F.E.S.S.A.): clinical application in fracture repair in 28 birds, *Vet Rec* 160:188–194, 2007.
Reichler IM, von Werthern CJ, Montavon PM: Der tubuläre Fixateur externe (F.E.S.S.A.): klinische Anwendung zur Frakturversorgung bei 6 Zwerghunden und 20 Katzen, *Kleintierpraxis* 42:407–419, 1997.

USE AND APPLICATION OF PLATES FOR FRACTURE REPAIR

David Sanchez Migallon Guzman

Bone plates provide rigid stability and maintain anatomic reduction, allowing early return to function. Plates counteract bending and rotational moments and shear and compression forces. In birds, an advantage of bone plates is excellent patient tolerance because plates are completely internal and provide rigid fixation with less callus formation. Disadvantages of bone plating are high costs for instrumentation and implants, specialized training in placing the plates, prolonged anesthesia times, soft tissue disruption, and potential thermal conduction (Martin, 1994).

Plate fixation in birds had been historically discouraged because fractures are often comminuted, the cortices were considered too thin for adequate screw purchase, and the subcutaneous space is limited in the distal wings and legs (MacCoy, 1992). New equipment and techniques have allowed bone plating to become an integral part of avian orthopedics and a standard of practice when the resources and knowledge are available. Multiple examples of application of plate fixation exist in the literature in different avian species as small as the domestic pigeon (*Columba livia*) and as large as the emu (*Dromaius novaehollandiae*) and in bones such as the coracoid, humerus, ulna, femur, tibiotarsus, or tarsometatarsus (Bush *et al.*, 1976; Bauck *et al.*, 1987; Kuzma and Hunter 1989; Howard 1990; Hatt *et al.*, 2001; Davidson *et al.*, 2005; Sanchez-Migallon Guzman *et al.*, 2007; Rahal *et al.*, 2008; Montgomery *et al.*, 2011; Gull *et al.*, 2012).

Standard techniques for bone plating are used and additional recommendations exist for birds (Fig. 12-40). If a screw is stripped, a nut may be fashioned from a small piece of a polypropylene syringe case, drilled, and tapped to hold screw threads (Howard, 1990). Intramedullary (IM) polymethylmethacrylate has been used also to improve screw purchase in avian bone (Kuzma and Hunter, 1991). The combination of an IM pin and bone plate has been reported in birds to reduce internal plate stress, increasing fatigue life of the plate (Sanchez-Migallon Guzman *et al.*, 2007). Improper plate length and surface application may contribute to implant failure. Longer plates than similar canine and feline fractures might be required in birds (Howard, 1990; Gouvêa *et al.*, 2011). In a study in pigeons with tibiotarsal fractures, titanium miniplates with a different number of holes, with or without spacers, were compared. The longer plates tested with eight holes and four screws in each fragment healed faster and had a lower rate of bending compared with the shorter plates tested (Gouvêa *et al.*, 2011). The surface application of bones, usually the convex surface, might be difficult to access in some bones like the ulna. Further studies regarding plate length and tension surfaces in birds are needed.

Different types of plates are available to use in birds, including veterinary cut-to-length plates (VCPs), dynamic compression plates, limited contact dynamic compression plates, locking plates, and miniplates. Most can be bent to contour the bones, and some, like the VCP, can be cut to the appropriate length. VCPs also may be stacked to improve their strength. Most of the plates available in the market are made of stainless steel, but other materials like titanium are also available. In pigeons with ulnar fractures repaired with 6 or 11 hole 1.0-mm titanium miniplates were too weak and resulted in bending (Christen *et al.*, 2005; Gull *et al.*, 2012), while stainless steel with eight holes and 1.3-mm adaption plates with two screws in each fragment were strong enough (Gull *et al.*, 2012);. Screws with a small thread pitch are recommended, as they have stronger holding power. Studies evaluating the holding power of cortical screws in birds are needed.

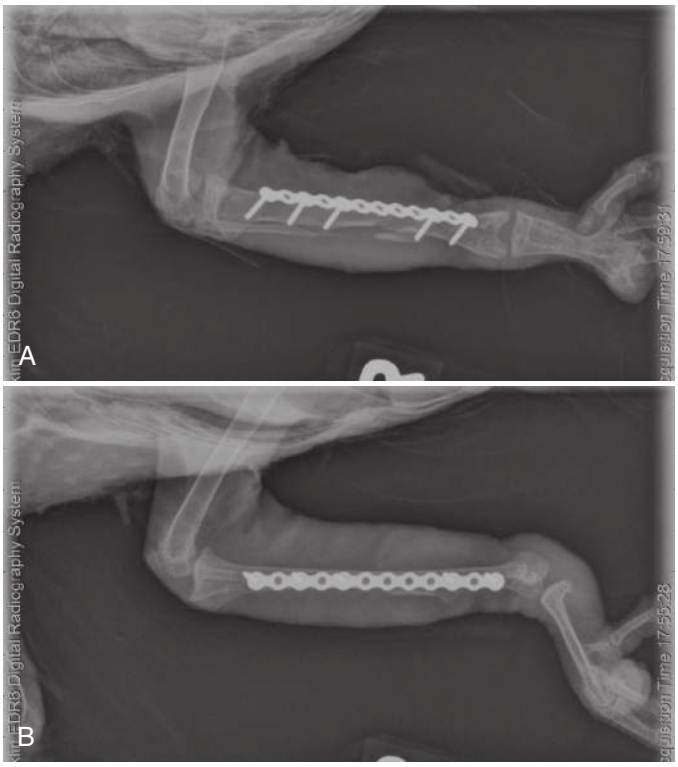


FIGURE 12-40 Adult blue and gold macaw with a comminuted oblique fracture of the mid-diaphysis of the right tibiotarsal bone with proximal, lateral, and cranial displacement of the distal fracture segment. A 12-hole straight 1.5-mm-locking plate was applied to the medial aspect of the tibiotarsus and 3 × 9 mm screws were used on the proximal fracture fragment and 2 × 9 mm screws were used on the distal fracture fragment.

External coaptation should be applied for 24 hours following bone plate application to help reduce soft tissue swelling. The patient should be confined to a small cage with only limited exercise. Physical therapy is encouraged during this time. Plate removal is not necessary unless it causes a problem. It appears that cold sensitivity, stress protection, and weight of the implants are not a major concern for most birds (Bennett, 1997).

REFERENCES

Bauck L, Fowler JD, Cribb P: Tibial osteotomy in an emu, *Comp Anim Pract* 1(5):26–30, 1987.
Bennett A: Orthopedic surgery. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.
Bush M, Hughes JL, Ensley PK, et al: Fracture repair in exotics using internal fixation, *J Am Anim Hosp Assoc* 12(6):746–753, 1976.

Christen CFI, Von Rechenberg B, Flückiger M, et al: Evaluation of a maxillofacial miniplate compact 1.0 for stabilization of the ulna in experimentally induced ulnar and radial fractures in pigeons (*Columbia livia*), *J Avian Med Surg* 19(3):185–190, 2005.

Davidson JR, Mitchell M, Ramirez S: Plate fixation of a coracoid fracture in a bald eagle (*Haliaeetus leucocephalus*), *J Avian Med Surg* 19(4):303–308, 2005.

Gouvêa AS, Alievi MM, Noriega V, et al: Microplacas de titânio em fraturas de tibiotarso em pombos domésticos, *Cienc Rural* 41(3):476–482, 2011.

Gull JM, Saveraid TC, Szabo D, et al: Evaluation of three miniplate systems for fracture stabilization in pigeons (*Columba livia*), *J Avian Med Surg* 26(4):203–212, 2012.

Hatt JM, Schläpfer NI, Von Werthern C: Use a maxillofacial mini-plate compact 1.0 for the repair of a distal fracture of the tibiotarsus in an African Grey parrot (*Psittacus erithacus*). A case report, *Tieraerztl Prax* 29(K):135–138, 2001.

Howard DJ: The use of bone plates in the repair of avian fractures, *J Am Vet Med Assoc* 26:613–622, 1990.

Kuzma AB, Hunter B: Osteotomy and derotation of the humerus in a turkey vulture using intramedullary polymethylmethacrylate and bone plate fixation, *Can Vet J* 30(11):900–901, 1989.

Kuzma AB, Hunter B: A new technique for avian fracture repair using intramedullary polymethylmethacrylate and bone plate fixation, *J Am Anim Hosp Assoc* 27:239–248, 1991.

MacCoy DM: Treatment of fractures in avian species, *Vet Clin North Am Small Anim Pract* 22(1):225–238, 1992.

Martin HRB: Orthopedic surgical techniques. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Montgomery RD, Crandall E, Bellah JR: Use of a locking compression plate as an external fixator for repair of a tarsometatarsal fracture in a bald eagle (*Haliaeetus leucocephalus*), *J Avian Med Surg* 25(2):119–125, 2011.

Rahal SC, Teixeira CR, Pereira-Junior OC, et al: Two surgical approaches to fracture malunion repair, *J Avian Med Surg* 22(4):323–330, 2008.

Sanchez-Migallon Guzman D, Bubenik LJ, Lauer SK, et al: Repair of a coracoid luxation and a tibiotarsal fracture in a bald eagle (*Haliaeetus leucocephalus*), *J Avian Med Surg* 21(3):188–195, 2007.

Systemic Diseases
Disorders of the Integument

DISORDERS OF THE FEATHERS, SKIN, AND ADNEXA

Andrés Montesinos

Dermatologic problems are among the most common conditions veterinarians treat in birds. Bird owners often readily detect avian skin or feather disorders because they are easy to see. Although these conditions are highly visible, avian dermatologic diseases represent a complex and often frustrating problem for veterinarians to diagnose and treat. The health of a bird's skin and feathers reflects all aspects of its clinical and environmental condition. Because of the multifactorial nature of many skin diseases, finding an etiologic diagnosis is often quite difficult.

The various conditions and factors that can affect feather structure, growth, condition, or color are listed in Table 13-1 (Figs. 13-1 to 13-4). Investigation of feather abnormalities should begin with a complete history, emphasizing diet, housing, and management. A complete physical examination should then be performed, even if the cause of the problem is obvious from the anamnesis and the bird's appearance. Appropriate diagnostic tests can then be considered that are based on initial findings and clinical suspicions. A minimum database should include a complete blood count (CBC), plasma biochemistry panel, and fecal parasite examination. Additional dermatologic tests may include feather pulp cytology and culture and feather follicle and skin biopsy of affected and unaffected zones. The therapeutic plan depends on the final or presumptive diagnosis. In all cases it is important to correct any dietary imbalances and make necessary improvements in housing, environment, and management.

In most birds, but especially in psittacines, feather loss is a result of feather picking by the bird or by another bird. Causes of feather loss

TABLE 13-1 Causes of Abnormal Feather Appearance

Feather Appearance	Possible Etiologies
Stress bars	Illness, stress, malnutrition
Broken or damaged feathers	Injury, brushing against badly designed cages, mineral deficiency, feather picking
Poor feather condition	Inability to preen, feather mites, lice, internal parasites, low humidity, inappropriate photoperiod, household aerosols, cigarette smoke, mycotoxins, systemic illness, malnutrition
Feather dystrophy	Psittacine circovirus infection, chronic polyomavirus infection, polyfolliculitis (lovebirds), feather-duster disease (budgerigars), pinching-off syndrome (some raptors species)
Color change	Malnutrition, liver disease, hypothyroidism, thyroxine therapy, early circovirus infection, chronic inflammation of the feather follicle

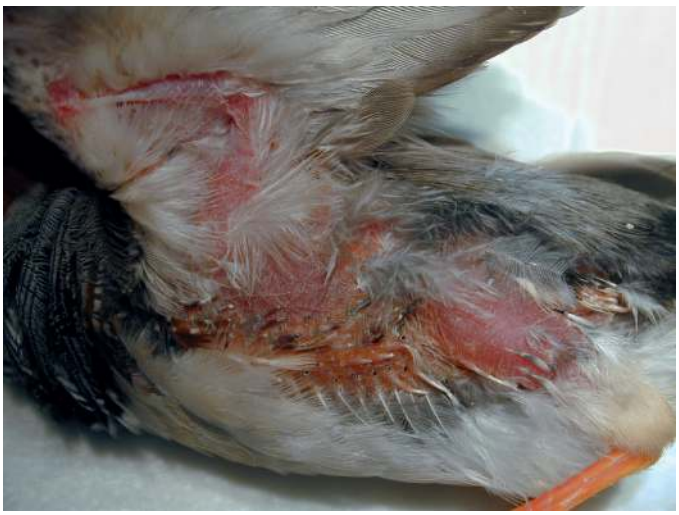


FIGURE 13-1 Feather loss in a zebra finch (*Poephila guttata*) from cagemate aggression.

FIGURE 13-2 Filamentous appearance of the feather in a budgerigar (*Melopsittacus undulatus*) suffering from polyomavirus infection.

FIGURE 13-3 European kestrel (*Falco tinnunculus*) affected by the "pinching-off" syndrome, which is characterized by generalized feather abnormality with increased cytokeratin formation at the base of the replacing feathers. Etiology is unknown.

TABLE 13-2 Causes of Feather Loss without Feather Picking

Normal Skin	Abnormal Skin
Normal apteria or normal molt (inexperienced bird owner)	Ectoparasites (e.g., *Cnemidocoptes*)
Excessive or irregular molt pattern induced by irregular photoperiod or malnutrition	Polyomavirus infection
Previous damage to feather follicles	Yeast dermatitis (lovebirds, canaries, and finches)
Manifestation of circovirus or polyomavirus infection	Dermatophytosis (gallinaceous birds)
Genetic condition (baldness in lutino cockatiels)	Abscesses, tumors, xanthomas
Obesity (budgerigars, Amazon parrots)	Pyoderma (bacterial dermatitis)
Systemic infection	Liver or kidney disease Hypothyroidism

FIGURE 13-4 Abnormal feather color and greasy appearance of a Patagonian conure (*Cyanoliseus patagonus*) treated with corticoids ointment over 3 weeks.

unassociated with feather picking are summarized in Table 13-2. Feather picking is undoubtedly the most frustrating dermatologic condition seen in psittacines and in other species such as ratites, ibises, and raptors. In many cases feather picking is multifactorial in etiology and has primary, predisposing, and perpetuating factors that can pose a diagnostic and therapeutic challenge (Gaskin *et al.*, 2014; Van Zeeland *et al.*, 2009). Table 13-3 summarizes the various causes of feather picking and self-trauma in captive birds (Figs. 13-5 to 13-11).

The general approach to treating feather picking is to determine whether there are any physical causes. Investigation begins with a thorough history and physical examination. An important first step is to establish that the feather loss is indeed caused by the bird picking or pulling its feathers. Feather loss unassociated with feather picking can occur on any part of the body, including the head, but the hallmark of feather picking by the bird is normal feathering on the head (i.e., the one area inaccessible to the beak). During the first contact with a

TABLE 13-3 Causes of Feather Picking

Skin Disease	Systemic Disease
Pox virus infection (lovebirds)	Malnutrition (deficiency of vitamin A, niacin, riboflavin, biotin, zinc, methionine, lysine, arginine, folic acid, vitamin E, or selenium)
Cutaneous neoplasms (self-trauma over affected area)	Giardiasis (especially cockatiels)
Bacterial dermatitis or folliculitis (*Staphylococcus* sp.)	Internal parasites (nematodes and cestodes)
Fungal dermatitis or folliculitis	Intestinal candidiasis
Poor feather condition (see Table 13-1)	*Chlamydia* infection
Inadequate bathing	Liver or kidney disease
Polyfolliculitis (lovebirds and budgerigars)	Generalized bacterial or fungal disease
Injury or skin irritation	Osteomuscular disease, especially if it causes pain
Hypersensibility (food and environmental)	Chronic heavy metal toxicity (zinc)
External parasites (mites and lice)	
Circovirus infection	
Other Causes:	
Normal preening or nesting behavior (observations by an inexperienced owner)	
Overpreening (e.g., young parrots raised in isolation from other adults birds)	
Inappropriate preening (e.g., young parrots raised with other psittacines species)	
Sexual frustration	
Psychological: boredom, frustration, anxiety or fear; environmental change; attention-seeking, jealousy, incompatible company, social isolation or change; separation anxiety, overcrowding, and others.	
Habitual behavior, despite the resolution of the trigger or physical cause	

FIGURE 13-5 Skin necrosis and skin automutilation in a yellow-fronted Amazon parrot (*Amazona ochrocephala*). In this case a hypersensitivity reaction of the skin was diagnosed by paired skin biopsies. This presentation is often described as "Amazon foot necrosis syndrome" in some avian medicine books.

FIGURE 13-6 Skin ulceration with pyoderma and polyfolliculitis in the chest region of a peach-fronted lovebird (*Agapornis roseicollis*). Hypersensitivity, circovirus, or polyomavirus, and skin infections should be considered as etiologic agents of this syndrome.

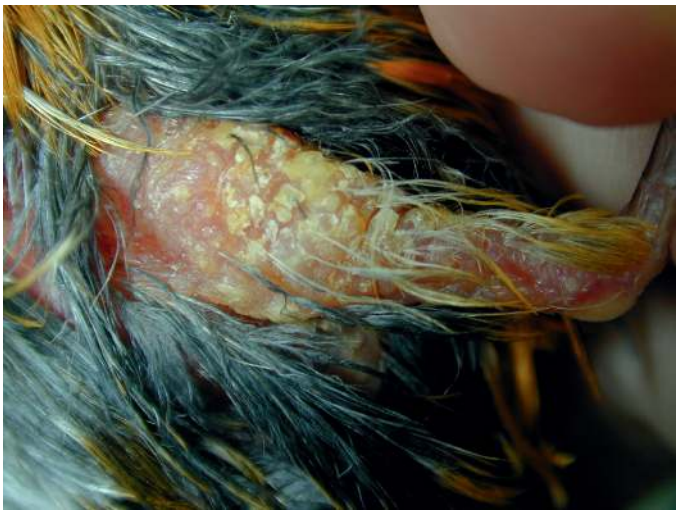


FIGURE 13-7 Malassezia dermatitis in a canary (*Serinus canaria*). This infection is very common in this species and is commonly secondary to indiscriminate antibiotic treatments or secondary to the use of medical ointment on the feathers.

FIGURE 13-8 African grey parrot (*Psittacus erithacus*) with chronic psittacine beak and feather disease.

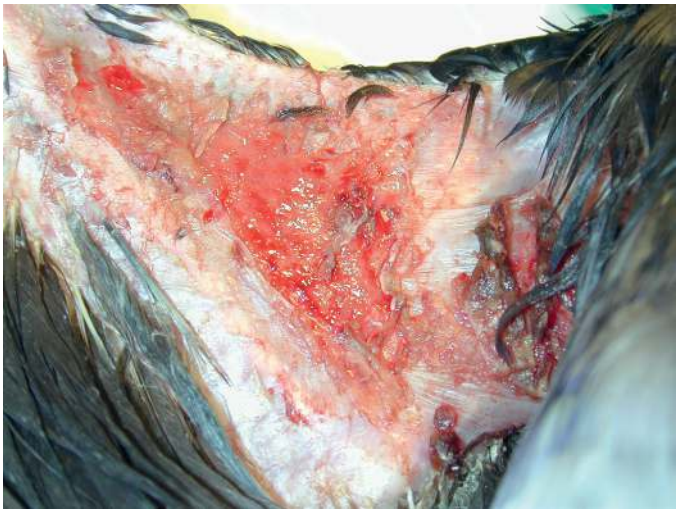


FIGURE 13-9 Ulcerative, exudative, and spontaneous bilateral skin lesions under both patagium membrane in an African grey parrot (*Psittacus erithacus*). This presentation has been associated with methicillin-resistant *Staphylococcus aureus* pyodermal infection, circovirus infection, and food hypersensitivity or contact allergens. Usually responds to antibiotic therapy and wound cleaning but could be very frustrating. Etiology is unknown.

FIGURE 13-10 Dorsal view of the head and neck of a saker (*Falco cherrug*) falcon affected by the so-called "feather duster" syndrome. This condition is characterized by severe feather abnormalities including change of feather coloration, deformed rachis resulting in curling of the feathers, loss of flight and tail feathers, and constriction of the terminal end of the calamus. (Courtesy Dr. Jaime Samour.)

FIGURE 13-11 Foot from the same falcon from Figure 13-10 showing changing talon coloration typical of "feather duster" syndrome observed in falcons. In addition, the beak of affected birds also tends to show changes in coloration. This medical condition is very rare in falcons and is probably caused by a circovirus, although this has never been positively diagnosed. (Courtesy Dr. Jaime Samour.)

FIGURE 13-12 Uropygial or preen gland carcinoma in a budgerigar (*Melopsittacus undulatus*). Confirmation of such diagnosis always requires histopathology analysis. (Courtesy Robert Doneley.)

feather-picking bird, conduct an in-depth discussion with the owner or the curator, a thorough physical examination, a feather examination (gross and microscopic), skin scraping (ectoparasites and cytologic examination), a feather pulp preparation, and a complete fecal examination (direct examination and flotation, with special attention to *Giardia* in susceptible species, such as cockatiels). If a medical cause is suspected, also consider performing a CBC, a plasma biochemistry panel, and probably *Chlamydia* or viral screens. If a diagnosis is not possible the first time, evaluate owner compliance with the initial recommendations (e.g., change of diet) and consider more diagnostic tests such as heavy metal testing (especially zinc), radiography, paired skin/follicle biopsies, or a thyroid-stimulating hormone stimulation test. If after these tests there is still no evidence of a medical problem, a high probability exists that the problem is purely behavioral (Clubb *et al.*, 2007).

UROPYGIAL OR PREEN GLAND

There are two major conditions that affect the uropygial gland. One is impaction, which is normally related to hypovitaminosis A. Gland function is assessed at physical examination by wiping a finger across the tip. An oily secretion should be present. If not, blockage is likely, which may lead to impaction and abscess. Gently squeezing the gland may resolve the impaction, but if it does not, then surgery is necessary. The other condition affecting this gland is neoplasia, which always requires surgical excision and histopathological analysis (Fig. 13-12).

FURTHER READING

Clubb SL, Cray C, Arheart KL, et al: Comparison of selected diagnostic parameters in African grey parrots (*Psittacus erithacus*) with normal plumage and those exhibiting feather damaging behavior, *J Avian Med Surg* 21(4):259–264, 2007.

Gaskins LA, Hungerford L: Non-medical factors associated with feather picking in psittacine birds, *J Avian Med Surg* 28(2):109–117, 2014.

Van Zeeland Y, Spruit B, Rodenburg T, et al: Feather damaging behavior in parrots: a review with considerations of comparative aspects, *Appl Anim Behav Sci* 121(2):75–95, 2009.

DISORDERS OF THE MUSCULOSKELETAL SYSTEM

METABOLIC BONE DISEASE

Kerri Morgan

Metabolic bone diseases (also known as osteodystrophies) occur as a result of nutritional or physiologic imbalances that interrupt the metabolism of calcium and/or phosphorus. This can result in disruption of bone growth, bone modeling, or remodeling (Thompson, 2007). Metabolic bone diseases are traditionally classified as fibrous osteodystrophy, rickets, osteomalacia, and osteoporosis (Thompson, 2007). Although these are distinct pathologic entities, they seldom occur in isolation within a single individual, and the cause of each can differ among species (Thompson, 2007). Fibrous osteodystrophy and rickets are the most clinically relevant metabolic bone diseases in companion avian medicine, and there is often confusion in classification and etiologies of these diseases.

Fibrous Osteodystrophy

Nutritional secondary hyperparathyroidism manifests as fibrous osteodystrophy in young, rapidly growing animals, and has been reported on a number of occasions in both wild and captive avian species (Tangredi and Krook, 1999; Toyoda *et al.*, 2004; Phalen *et al.*, 2005; Morgan *et al.*, 2011) (Fig. 13-13). In contrast, primary hyperparathyroidism, usually resulting from a functional parathyroid gland adenoma (Thompson, 2007), has not been reported in any avian species to the author's knowledge.

Etiology

Fibrous osteodystrophy results from a dietary imbalance of calcium and phosphorous and/or a deficiency in vitamin D (Tangredi and Krook, 1999; Thompson, 2007; Morgan *et al.*, 2011). Typically, a combination of these dietary imbalances induces hypocalcemia, stimulating a prolonged and excessive secretion of parathyroid hormone, resulting in excessive osteoclastic bone resorption and its replacement with fibrous tissue and poorly mineralized immature bone (Thompson, 2007) (Fig. 13-14).

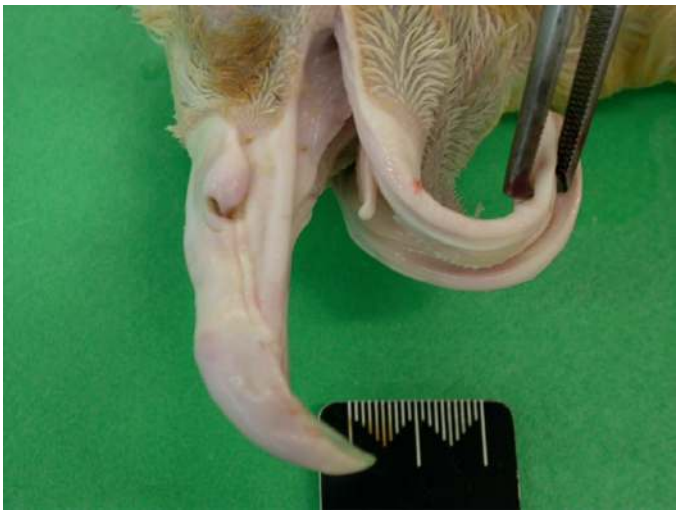


FIGURE 13-13 Fibrous osteodystrophy in a semiwild, hand-fed 24-day-old Northern Royal albatross (*Diomedea sanfordi*) chick resulting in pliable, rubbery bones. Parental abandonment resulted in in situ feeding of hoki fillets (*Macruronus novaezelandiae*), with a calcium : phosphorus ratio of 1 : 13, leading to nutritional secondary hyperparathyroidism. (From Morgan KJ, Alley MR, Gartrell BD, et al: *NZ Vet J* 59[5]:248–252.)

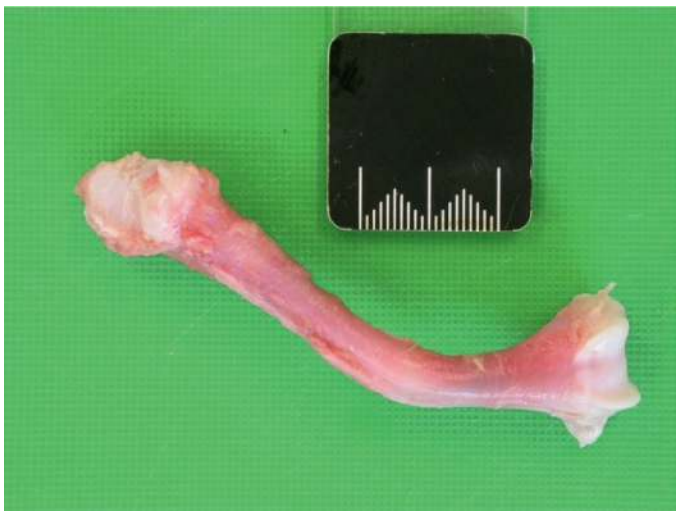


FIGURE 13-14 Folding fracture in a 24-day-old Northern Royal albatross (*Diomedea sanfordi*) chick as the result of fibrous osteodystrophy. (From Morgan KJ, Alley MR, Gartrell BD, et al: *NZ Vet J* 59[5]:248–252, 2011.)

Clinical Signs and Clinical Pathology

Clinical signs associated with fibrous osteodystrophy in birds include severe weakness, depression, anorexia, and leg and wing paralysis (Kumashiro and Amimoto, 1987; Toyoda *et al.*, 2004; Adkesson and Langan, 2007). Anemia and elevated alkaline phosphatase (ALP) has been reported in individuals with fibrous osteodystrophy (Toyoda *et al.*, 2004). Although hypocalcemia, with or without hypophosphatemia, has been reported to be associated with fibrous osteodystrophy in avian species (Long *et al.*, 1983; Toyoda *et al.*, 2004), total serum calcium is generally considered a poor indicator of physiologic calcium activity in sick animals, and it may also be influenced by sex, season, and state of reproduction (Adkesson and Langan, 2007). Instead, ionized calcium, the active metabolic form of calcium, is a more appropriate indicator of available calcium as it may be decreased even though total serum calcium is within normal limits (Hanley *et al.*, 2004; Adkesson and Langan, 2007). Although this value varies with species, normal values are usually between 1.0 and 1.3 mmol/L in healthy animals (Adkesson and Langan, 2007).

Gross Pathology

Depending on their specific history, birds affected by fibrous osteodystrophy may present emaciated and cachexic (Adkesson and Langan, 2007), or conversely they may be in good body condition (Tangredi and Krook, 1999; Morgan *et al.*, 2011). Gross bony changes associated with fibrous osteodystrophy are associated with the replacement of mineralized bone with fibrous connective tissue (Morgan *et al.*, 2011). Affected bones are reported to be rubbery and may be easily bent on application of digital pressure (Fig. 13-13; Adkesson and Langan, 2007; Morgan *et al.*, 2011). Pathologic fractures have been reported in long bones, mandibles, and ribs and folding fractures are commonly seen in the long bones (Long *et al.*, 1983; Tangredi and Krook, 1999; Morgan *et al.*, 2011). Curving deformities of long bones and ribs may be seen (Fig. 13-14; Phalen *et al.*, 2005; Adkesson and Langan, 2007; Morgan *et al.*, 2011). In some cases, gross enlargement of the parathyroid glands has been reported (Long *et al.*, 1983; Phalen *et al.*, 2005), although often no gross lesions are detected (Morgan *et al.*, 2011).

Radiography

Radiographic changes may include a generalized reduction in skeletal density; severe thinning of cortical bones; curving deformities of long bones; delayed and asymmetric pneumatization of the humeri; folding fractures of long bones; and pathologic fractures of ribs, vertebrae, skull, and long bones in various states of healing and remodeling (Figs. 13-15 and 13-16; Tangredi and Krook, 1999; Toyoda *et al.*, 2004; Phalen *et al.*, 2005; Morgan *et al.*, 2011).

Histopathology

Histopathologic changes associated with fibrous osteodystrophy include thin, porous cortical bone with abnormal bone remodeling (Morgan *et al.*, 2011). Affected trabeculae are thin and disorganized and are separated by fibrous connective tissue (Tangredi and Krook, 1999; Toyoda *et al.*, 2004; Phalen *et al.*, 2005; Adkesson and Langan, 2007; Morgan *et al.*, 2011). Affected osteoid seams are poorly mineralized and widened, with scalloped edges from an increase in osteoclastic activity (Fig. 13-17; Tangredi and Krook, 1999; Toyoda *et al.*, 2004; Phalen *et al.*, 2005; Morgan *et al.*, 2011). Osteoblastic activity may be increased (Toyoda *et al.*, 2004; Phalen *et al.*, 2005; Morgan *et al.*, 2011). The parathyroid glands of affected birds may show hypertrophy, hyperplasia, and cytoplasmic vacuolation of chief cells (Toyoda *et al.*, 2004; Phalen *et al.*, 2005; Morgan *et al.*, 2011).

Treatment and Prevention

Although there is little published literature outlining the specific treatment of fibrous osteodystrophy specifically in birds, treatment in other species relies upon correction of the underlying dietary imbalance (Tomsa *et al.*, 1999; Hanley *et al.*, 2004). Dietary calcium supplements and phosphate binders may be used in the early stages of treatment, and calcium carbonate supplementation is beneficial because it works both as a supplement and a binder (Hanley *et al.*, 2004). The use of parenteral calcium has been reported in individual birds with fibrous osteodystrophy (Kumashiro and Amimoto, 1987), and is indicated in animals with severe calcium deficiencies causing hypocalcemic flaccid paralysis or seizures (Hanley *et al.*, 2004). For animals with pathologic fractures, activity restriction, analgesia, and fracture stabilization is indicated (Tomsa *et al.*, 1999; Hanley *et al.*, 2004). External coaptation

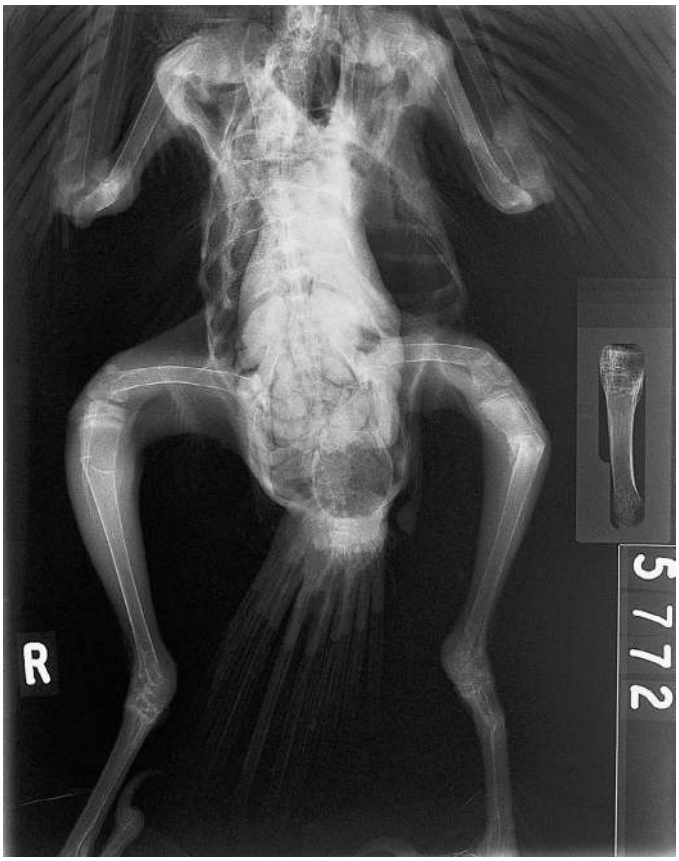


FIGURE 13-15 Radiograph of nestling raven (*Corvus corax*), approximately 3 weeks after hatching, with severe nutritional bone disease. This bird was reared in captivity. There are multiple skeletal distortions associated with pathologic fractures from very poor mineralization of bones. This is a result of calcium deficiency and develops rapidly in carnivorous birds fed on meat without bone.

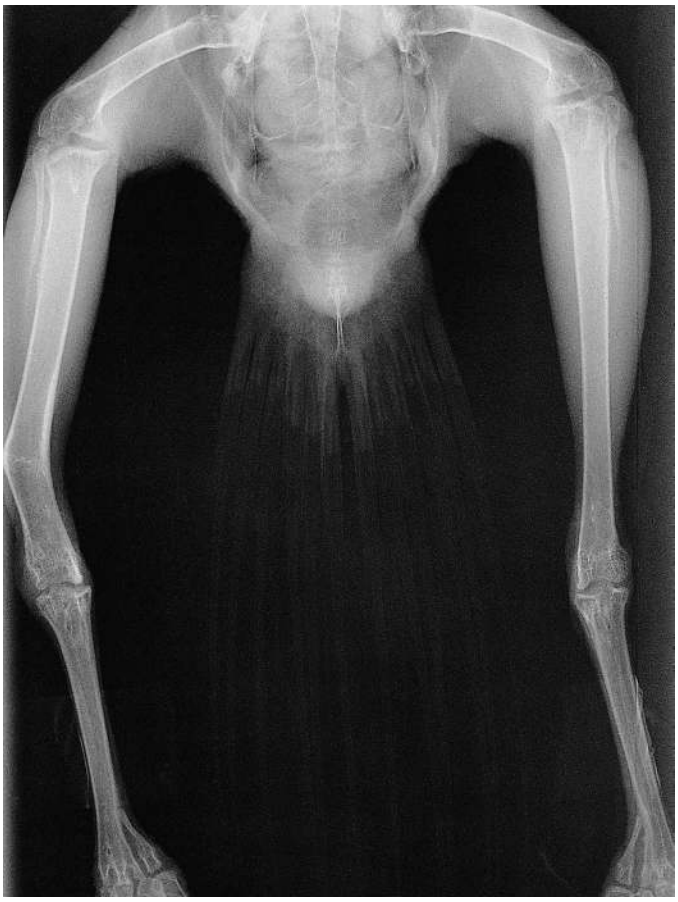


FIGURE 13-16 Radiograph of mature raven with deformity of the right tibiotarsus. This bird was reared in captivity. It is thought likely that this deformity arose through pathologic fracture during growth.

is usually indicated initially until bone quality has improved adequately to enable a more permanent repair if necessary (Hanley *et al.*, 2004). Appropriate analgesia should be instigated. In a single report of an osprey with fibrous osteodystrophy, clinical improvement was seen within 3 weeks of instigation of dietary correction and calcium supplementation, with almost complete recovery by 8 weeks (Kumashiro and Amimoto, 1987).

Fibrous osteodystrophy can be prevented with a diet containing an optimal calcium:phosphorus (Ca:P) ratio of 1.5:1 (Morgan *et al.*, 2011) and ensuring adequate dietary vitamin D and access to sunlight (Hanley *et al.*, 2004). Most commercial seed diets are highly deficient in calcium. For example, sunflower seeds have a Ca:P ratio of 1:7 and millet 1:6 (Mcwhirter, 1994). In some species of birds, individuals are able to self-regulate calcium intake, so free choice to calcium through cuttlebone or oyster shell is recommended (Donoghue and Stahl, 1997). However, expecting birds to *ad lib* calcium requirements does not work in all cases. For example, fibrous osteodystrophy has been diagnosed in wild kaka chicks (*Nestor meridionalis*) thought to be from their free-ranging, supplementary fed parents preferentially selecting and feeding corn to their chicks (unpublished observations). Corn has a high dietary imbalance of calcium and phosphorus with a Ca:P of 1:37 (Mcwhirter, 1994).

Rickets and Osteomalacia

Rickets and osteomalacia have a similar etiology and pathogenesis. It is only the age at which they develop that differentiates these disease entities: osteomalacia is a disease of adults and rickets is only ever seen in young growing animals (Thompson, 2007). Both diseases are characterized by defective bone formation, and rickets is also accompanied by abnormal endochondral ossification at growth plates (Thompson, 2007).

Etiology

Usually resulting from a deficiency in either vitamin D or phosphorus (Thompson, 2007), these diseases occur because of impaired mineralization of physeal and epiphyseal cartilage during endochondral ossification (rickets only) and of newly formed osteoid (rickets and osteomalacia; Dittmer and Thompson, 2011). There is doubt that calcium deficiency on its own can induce rickets or osteomalacia: this is more likely to lead to fibrous osteodystrophy or osteoporosis (Thompson, 2007). However, rickets caused by a deficiency in vitamin D often results in concurrent fibrous osteodystrophy from a reduction in intestinal absorption of calcium (Thompson, 2007).

Vitamin D exists in two forms: vitamin D_2 (ergocalciferol; mainly from plants and yeasts) and vitamin D_3 (cholecalciferol; Thompson, 2007). Although vitamin D_3 may be dietary in origin (mainly from fatty fish and cod liver oil), most of it is formed through photochemical conversion of 7-dehydrocholesterol in the skin by UVB (Thompson, 2007). In chickens, this occurs of feet and legs (Lewis and Gous, 2009; Ewbank *et al.*, 2013), and vitamin D_3 is 10 times more potent than vitamin D_2 (Thompson, 2007). Therefore, in birds, vitamin D deficiency may occur with inadequate access to sunlight.

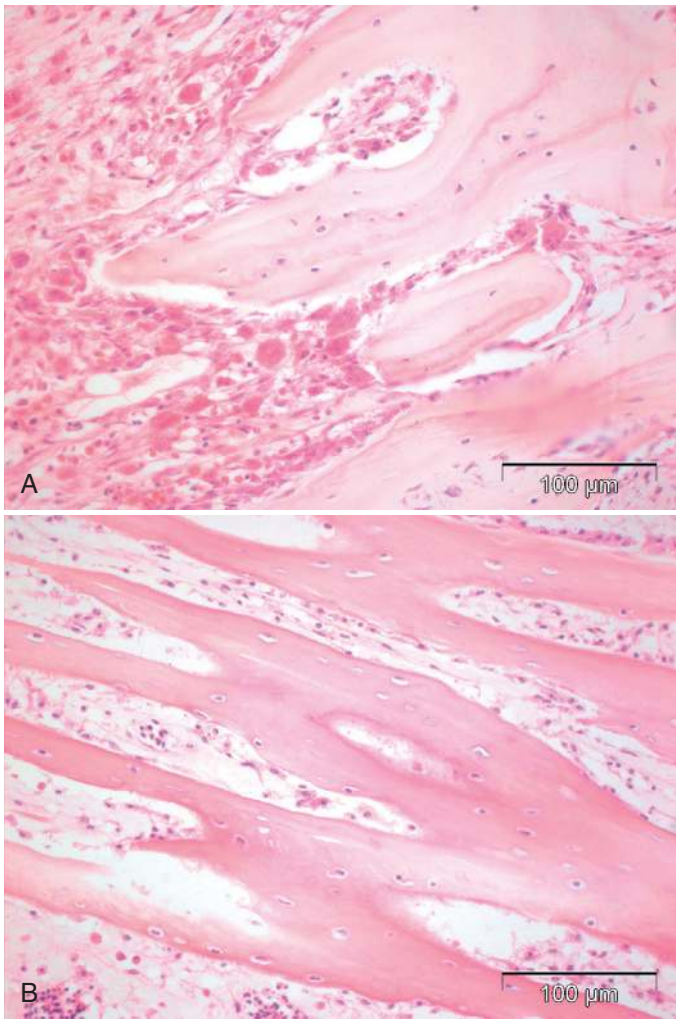


FIGURE 13-17 **(A),** Light photomicrograph of a section of tibiotarsus from a 24-day-old Northern Royal albatross (*Diomedea sanfordi*) chick with fibrous osteodystrophy, illustrating disorganized trabeculae and intertrabecular fibrous tissue together with numerous osteoclasts and osteoblasts (H&E scale bar = 200 µm). (From Morgan KJ, Alley MR, Gartrell BD, et al: *NZ Vet J* 59[5]:248–252, 2011.). **(B),** Light photomicrograph of a section of tibiotarsus from an unaffected 28-day-old Northern Royal albatross (*D. sanfordi*) chick, showing normal trabeculae (H&E scale bar = 200 µm). (Morgan KJ, Alley MR, Gartrell BD, et al: *NZ Vet J* 59[5]:248–252, 2011.)

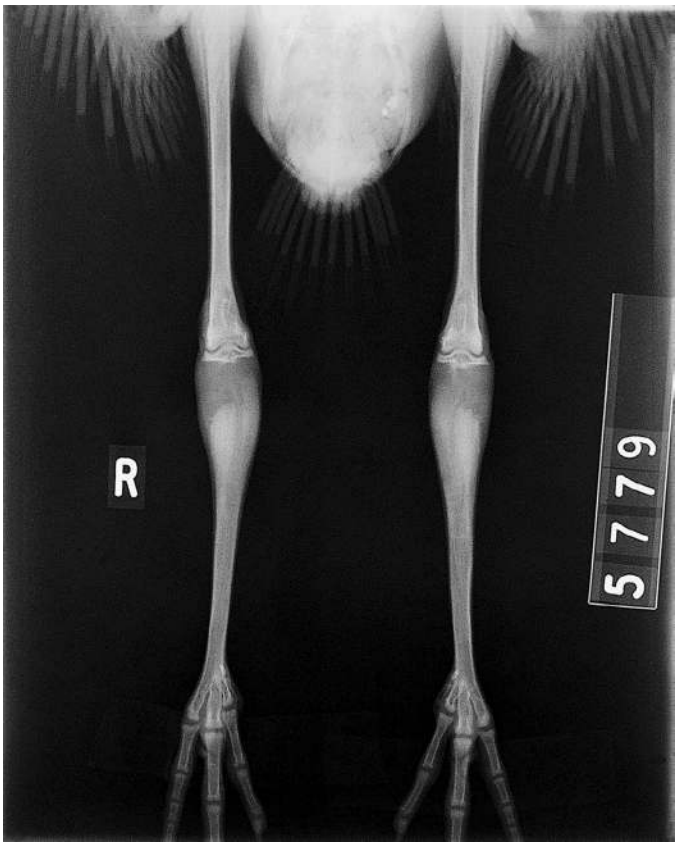


FIGURE 13-18 Radiograph of a demoiselle crane (*Anthropoides virgo*) chick with rickets. Bilateral physeal thickening of the proximal tarsometatarsi is seen as an area of radiolucency immediately beneath the radiopaque epiphysis.

Although well established as a cause of rickets and osteomalacia (Thompson, 2007), phosphorus deficiency is uncommon cause of these diseases, and is considered virtually impossible in carnivores because of the high levels of dietary phosphorus (Thompson, 2007).

Clinical Signs and Clinical Pathology

Rickets is seen in young, growing animals when skeletal growth is most rapid (Long *et al.*, 1983; Hedstrom *et al.*, 1986). Reported bony abnormalities from rickets are similar to lesions seen with fibrous osteodystrophy: affected birds may have pliable, twisted beaks; long bone deviations and folding fractures; tibiotarsal rotation; softening of bones; marked deviation of the vertebral column; and pelvic limb asymmetry (Long *et al.*, 1983; Nichols *et al.*, 1983; Ewbank *et al.*, 2013). Birds may present with an inability to support themselves (Nichols *et al.*, 1983) and may walk with a waddle or hopping motion from adduction of pelvic limbs, progressing to severe lameness (Hedstrom *et al.*, 1986). Birds with rickets may have suboptimal growth rates (Lewis and Gous, 2009) and severely affected birds may show systemic signs such as weakness, decreased mentation, lethargy, and dehydration (Hedstrom *et al.*, 1986; Ewbank *et al.*, 2013). An experimental study on vitamin D-deficient rickets showed an increase in plasma ALP, with normal calcium, and normal or elevated phosphorous (Hedstrom *et al.*, 1986). In this study, vitamin D_3 metabolites were also depleted (Hedstrom *et al.*, 1986). In rickets caused by phosphorous deficiency, hypophosphatemia may be seen, although a serum phosphorous concentration within normal limits does not necessarily rule out phosphorous-deficient rickets, and serum calcium concentrations are usually normal or increased (Thompson, 2007).

Radiography

Irregular thickening of the growth plates of long bones is the hallmark of rickets (Thompson, 2007), and is visible radiographically as an area of radiolucency beneath the more radiopaque epiphysis (Figs. 13-18 and 13-19; Long *et al.*, 1984a). There is usually a generalized osteopenia and poor corticomedullary definition of long bones (Nichols *et al.*, 1983; Long *et al.*, 1984b; Ewbank *et al.*, 2013). Other radiographic changes reported with rickets in avian species have included bowing deformities and folding fractures of the long bones (Nichols *et al.*, 1983; Long *et al.*, 1984b), vertebral scoliosis, and pelvic asymmetry (Ewbank *et al.*, 2013).

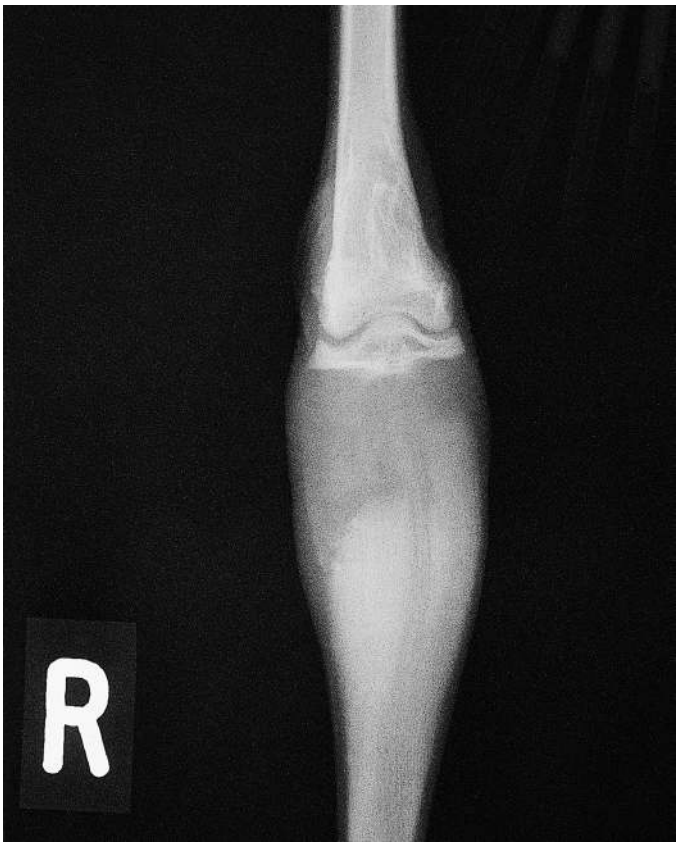


FIGURE 13-19 Radiograph of the tibiotarsal-tarsometatarsal joint of a demoiselle crane (*Anthropoides virgo*) chick with rickets. The growth plate is abnormally thick and irregular because of the presence of non-mineralized cartilage.

Gross Pathology

Gross pathologic changes associated with rickets are most prominent at the metaphyseal and epiphyseal regions of long bones and costochondral junctions, although there may be marked changes in severity between different bones in a single animal (Thompson, 2007). Physeal thickening is evident at one or more sites (Thompson, 2007). In experimental vitamin D-deficient rickets in broiler chickens, lengthening of the subepiphyseal growth plate band was grossly evident upon sagittal sectioning of affected bones (Long *et al.*, 1984b). Abnormal endochondral ossification beneath articular cartilage may also be seen, with irregular thickening of the articular cartilage, and there may be infolding and irregularity of the articular surface from collapse of weakened subchondral bone (Thompson, 2007).

The diaphysis of long bones affected by rickets and osteomalacia are shorter and thicker with a narrowed medullary cavity, and they are more susceptible to weight-bearing trauma (Thompson, 2007). Pathologic fractures of limbs, ribs, or vertebrae may be seen with rickets and advanced cases of osteomalacia (Thompson, 2007). As osteomalacia occurs in adult animals, growth plate involvement is not seen, although articular surface collapse may be observed (Thompson, 2007).

Histopathology

Rickets is characterized by the persistence of hypertrophic chondrocytes at the growth plates and beneath articular cartilage: both sites of endochondral ossification (Thompson, 2007). The underlying trabecular bone is often disrupted, and clumps of hypertrophic chondrocytes may be left behind in the metaphysis (Thompson, 2007). Trabeculae are thickened and irregular in shape and may contain microfractures and infractions because of trabeculae fragility (Thompson, 2007). Concurrent fibrous osteodystrophy lesions are often present if lesions are from vitamin D deficiency, as this leads to hypocalcemia (Thompson, 2007). Because osteomalacia only occurs in adult animals, growth plate involvement is not characteristic (Thompson, 2007).

Depending on the animal's age, cortical lesions occur from inadequate mineralization of osteoid during remodeling (osteomalacia) or growth (rickets). Trabeculae are reduced in number and size, and cortices are thin and porous (Thompson, 2007).

Treatment and Prevention

As with fibrous osteodystrophy, treatment relies upon correction of nutrient imbalances, and supportive care of the patient, including fracture management, as previously described. Some treatment success has been demonstrated with vitamin D-deficient rickets in birds and other species through parenteral administration of vitamin D with or without phosphorus, as well as ensuring access to ultraviolet (UV) light (Cousquer *et al.*, 2007; Stieger-Vanegas *et al.*, 2013). In other species, vitamin D-deficient rickets has been successfully treated with parenteral and oral calcium (MacKenzie *et al.*, 2011).

Rickets and osteomalacia are preventable with a balanced diet and adequate access to UV light. Vitamin D_3 can be provided by adding cod liver oil and cooked liver to the diet, and concentrations are reported to be adequate in seeds ripened in the sun (Donoghue and Stahl, 1997), although there is a suggestion that high-fat seed diets may interfere with calcium uptake from the intestinal tract (Mcwhirter, 1994). Piscivores should be fed whole fish to ensure intake of vitamin D through ingestion of viscera (Morgan *et al.*, 2011).

Osteoporosis

Osteoporosis is an imbalance between bone formation and resorption (in favor of resorption), and is characterized by a reduction in quantity of bone, without alteration in quality (Thompson, 2007). Osteoporosis is considered a normal process of aging, although there are a number of diseases that can result in accelerated bone loss or an increase in bone resorption (Thompson, 2007). These may include long-term disuse, dietary deficiencies of a specific nutrient (usually calcium, phosphorus, copper, or vitamin D), starvation, severe gastrointestinal parasitism (although likely to be secondary to malabsorption), corticosteroid induced, vitamin A toxicity, hyperthyroidism, and chronic metabolic acidosis (Thompson, 2007). Usually lameness is not observed clinically and is often first identified through the occurrence of a bone fracture without a history of excessive trauma (Thompson, 2007). Cage-layer osteoporosis is one of the most significant skeletal diseases in layer hens and is thought to be the result of periods of high egg production through mobilization of structural bone without the opportunity for regeneration. This process may be further exacerbated by dietary mineral deficiencies (Chunxiang *et al.*, 2010).

REFERENCES

Adkesson MJ, Langan JN: Metabolic bone disease in juvenile Humboldt penguins (*Spheniscus humboldti*): investigation of ionized calcium, parathyroid hormone, and vitamin D3 as diagnostic parameters, *J Zoo Wildl Med* 38(1):85–92, 2007.

Chunxiang L, Zheng J, Xinhan L: Biochemical mechanism of gallium on prevention of fatal cage-layer osteoporosis, *Biol Trace Elem Res* 134:195–202, 2010.

Cousquer GO, Dankoski EJ, Patterson-Kane JC: Metabolic bone disease in wild collared doves, *Vet Rec* 160:78–84, 2007.
Dittmer K, Thompson K: Vitamin D metabolism and rickets in domestic animals: a review, *Vet Pathol* 48(2):389–407, 2011.
Donoghue S, Stahl S: Clinical nutrition of companion birds, *J Avian Med Surg* 11(4):228–246, 1997.
Ewbank AC, Ruder MG, McRuer DL, et al: What is your diagnosis?, *J Avian Med Surg* 27(2):156–161, 2013.
Hanley CS, Wilson GH, Hernandez-Divers SJ: Secondary nutritional hyperparathyroidism associated with vitamin D deficiency in two domestic skunks (*Mephitis mephitis*), *Vet Rec* 155:233–237, 2004.
Hedstrom OR, Cheville NF, Horst RL: Pathology of vitamin D deficiency in growing turkeys, *Vet Pathol* 23:485–498, 1986.
Kumashiro H, Amimoto A: Successful treatment of a few months old osprey (*Pandion*) suspected of nutritional secondary hyperparathyroidism, *J Jpn Vet Med Assoc* 40(10):701–703, 1987.
Lewis PD, Gous RM: Responses of poultry to ultraviolet radiation, *Worlds Poult Sci J* 65(3):499–510, 2009.
Long P, Choi G, Rehmel R: Oxyphil cells in a red-tailed hawk (*Buteo jamaicensis*) with nutritional secondary hyperparathyroidism, *Avian Dis* 27(3):839–843, 1983.
Long PH, Lee SR, Rowland GN, et al: Experimental rickets in broilers: gross, microscopic and radiographic lesions. I. Phosphorus deficiency and calcium excess, *Avian Dis* 28(2):460–474, 1984a.
Long PH, Lee SR, Rowland GN, et al: Experimental rickets in broilers: gross, microscopic, and radiographic lesions. III. Vitamin D deficiency, *Avian Dis* 28(4):933–943, 1984b.
MacKenzie JM, Crawford J, Ghantous S: Successful therapy of vitamin D-dependent rickets in a kitten, *J Am Anim Hosp Assoc* 47(4):290–293, 2011.
Mcwhirter P: Malnutrition. In Ritchie B, Harrison G, Harrison L, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.
Morgan KJ, Alley MR, Gartrell BD, et al: Fibrous osteodystrophy in two Northern Royal albatross chicks (*Diomedea sanfordi*), *NZ Vet J* 59(5):248–252, 2011.
Nichols D, Montali RJ, Pickett C, et al: Rickets in double-crested cormorants (*Phalacrocorax auritus*), *J Zoo Anim Med* 14:115–124, 1983.
Phalen DN, Drew ML, Contreras C, et al: Naturally occurring secondary nutritional hyperparathyroidism in cattle egrets (*Bubulcus ibis*) from central Texas, *J Wildl Dis* 41(2):401–415, 2005.
Stieger-Vanegas SM, Garret R, McKenzie EC, et al: Vertebral fractures in two alpaca crias with rickets syndrome, *Aust Vet J* 91(10):437–440, 2013.
Tangredi BP, Krook LP: Nutritional secondary hyperparathyroidism in free-living fledgling American crows (*Corvus brachyrhynchos brachyrhynchos*), *J Zoo Wildl Med* 30(1):94–99, 1999.
Thompson K: Bones and joints. In Maxie MG, editor: *Jubb, Kennedy, and Palmer's pathology of domestic animals*, vol 2, ed 5, Edinburgh, UK, 2007, Elsevier.
Tomsa C, Glaus T, Hauser B, et al: Nutritional secondary hyperparathyroidism in six cats, *J Small Anim Prac* 40(11):533–539, 1999.
Toyoda T, Ochiai K, Komatsu M, et al: Nutritional secondary hyperparathyroidism and osteodystrophia fibrosa in a Hodgson's hawk-eagle (*Spizaetus nipalensis*), *Avian Pathol* 33(1):9–12, 2004.

DEVELOPMENTAL LIMB DISORDERS IN GROWING BIRDS

Brett Gartrell

In addition to the complex syndromes associated with metabolic bone disease, there are a variety of developmental conditions of the long bones and joints that can affect growing birds. The etiology of these conditions is often multifactorial, and in most cases is a combination of dietary imbalance (especially but not limited to manganese deficiency and high-protein diets), rapid growth, and poor substrates (Naldo *et al.*, 1998; Hahulski *et al.*, 1999). A genetic predisposition is often postulated but is difficult to prove. There is confusion in the terminology used to describe these conditions, and outside of domestic poultry research, information is sparse and usually restricted to case studies or series. There is a need for experimental study of these conditions in birds other than poultry, particularly regarding predisposing factors and treatment options.

These conditions are more frequently reported in long-limbed precocial birds with extended growing phases and include bustards (Naldo *et al.*, 1998; Stiévenart, 2008), cranes and storks (Wolf *et al.*, 2001), ratites (Wolf *et al.*, 1996; Hahulski *et al.*, 1999; Prier *et al.*, 2013), rails (McLelland *et al.*, 2011), seabirds (Drew and Kreeger, 1986; Pitman *et al.*, 2012), and waterfowl (Kreeger and Walser, 1984; Smith, 1997; Ferraz *et al.*, 2010). However, there are reports of these conditions in most bird species including short-limbed altricial species such as raptors (Zsivanovits *et al.*, 2006), cockatoos, and parrots (Bauck, 1995; Harcourt-Brown, 2004; Ferraz *et al.*, 2010).

Rotational Limb Deformities

The most commonly reported developmental disorders of the pelvic limb include rotational limb deformities of the tibiotarsus (Figs. 13-20 to 13-26) and medial luxation of the gastrocnemius tendon from the hock joint, which is often referred to as perosis (Fig. 13-27). These two conditions are often conflated, because severe rotation of the tibiotarsus can lead to perosis, but perosis can also be from other abnormalities such as shallow intercondylar areas of the distal tibiotarsus. Rotational deformities of the tibiotarsus can be either varus or valgus. Developmental disorders of the femur, tarsometatarsus, and phalanges (rolled toes) occur but are less commonly reported.

Rolled Toes

Rolled toe is the medial rotation of the phalanges of young growing birds. This condition is most often seen in bustards (Naldo *et al.*, 1998) and ratites (Stewart, 1994). It has been suggested that rolled toes develop secondary to perosis (Gewalt and Gewalt, 1966). Riboflavin deficiency and embryo malpositioning are factors that may cause this condition (Anderson, 1983). In ostriches, the incidence of rolled toes appears to be related to genetic abnormalities, incubation problems, and unsuitable substrates during the first week of life (Stewart, 1994).

FIGURE 13-20 Mild distortion of the left leg of a buff-crested bustard (*Eupodotis ruficrista*). This appears to be from rotation of the tibiotarsus rather than outward bending of the tarsometatarsus.

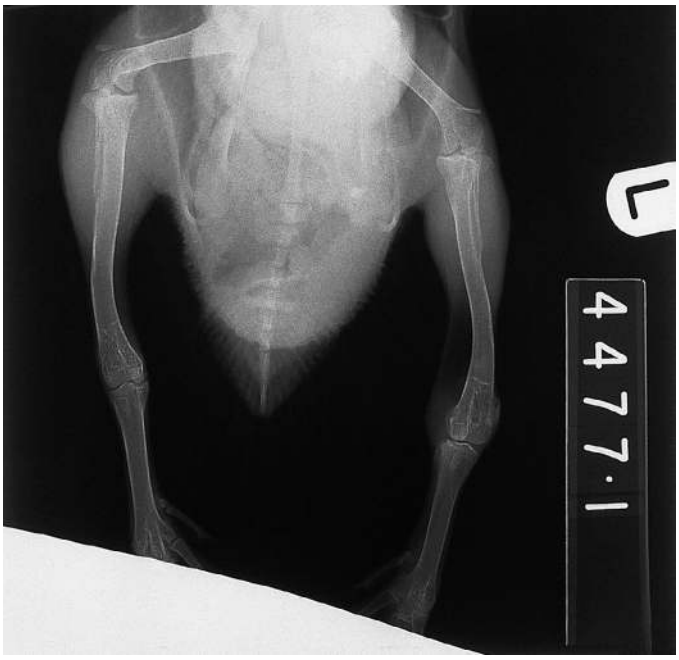


FIGURE 13-21 Inward deviation of the distal part of the tibiotarsus in a mallard (*Anas platyrhynchos*). This may have been associated with excessive rates of weight gain during the early stages of growth.

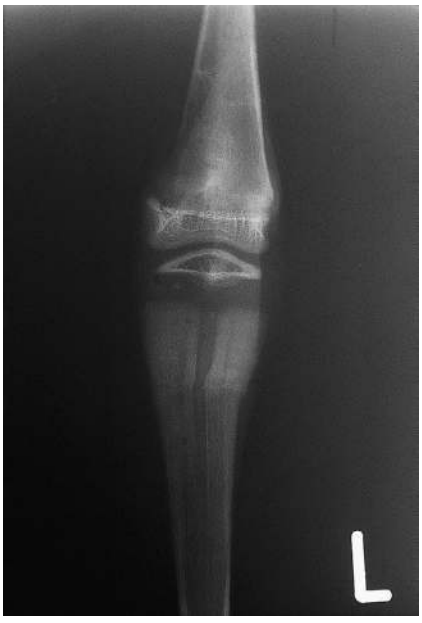


FIGURE 13-23 Intertarsal joint of a growing rhea (*Rhea americana*). A lesion is visible in the left side of the recently mineralized part of the proximal tarsometatarsus. This could have been caused by trauma or possibly by a metabolic disturbance to the growth plate a few days earlier. It has caused a slight bend in the proximal tarsometatarsus.

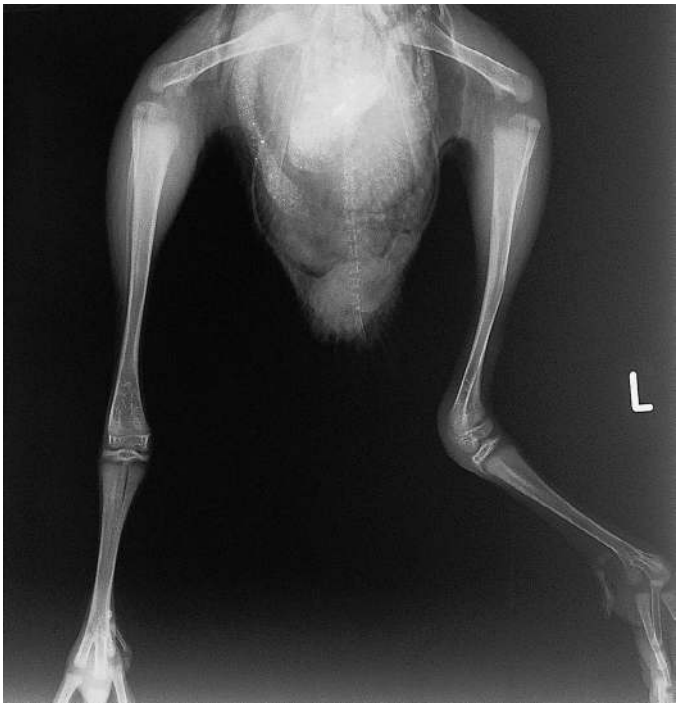


FIGURE 13-22 Severe (approximately 90°) rotation of the left tibiotarsus of a Hawaiian goose (*Branta sandvicensis*) during growth.

FIGURE 13-24 The tarsometatarsus bones of a young rhea, showing twisting and bending deformities.

Chondrodystrophy

Most pelvic limb deformities that arise in growing birds are thought to be from an underlying chondrodystrophy, which results in uneven growth of the long bones. This may be from trauma, vascular damage, or infection and has been experimentally produced by surgically damaging the periosteum (Rackard *et al.*, 2002). The relationship of the chondrodystrophies seen in other bird species to the well-described syndrome of tibial chondrodysplasia in poultry is uncertain. The tibial chondrodysplasia in poultry has a similar variety of proposed causative factors and some authors believe that multiple mechanisms for inducing this disorder can occur (Orth and Cook, 1994). Some conditions, such as splay leg deformity seen in very young birds, are thought to be primarily related to exposure to slippery flooring in the early stages of growth (Naldo *et al.*, 1998; Harcourt-Brown, 2004; Fig. 13-28).

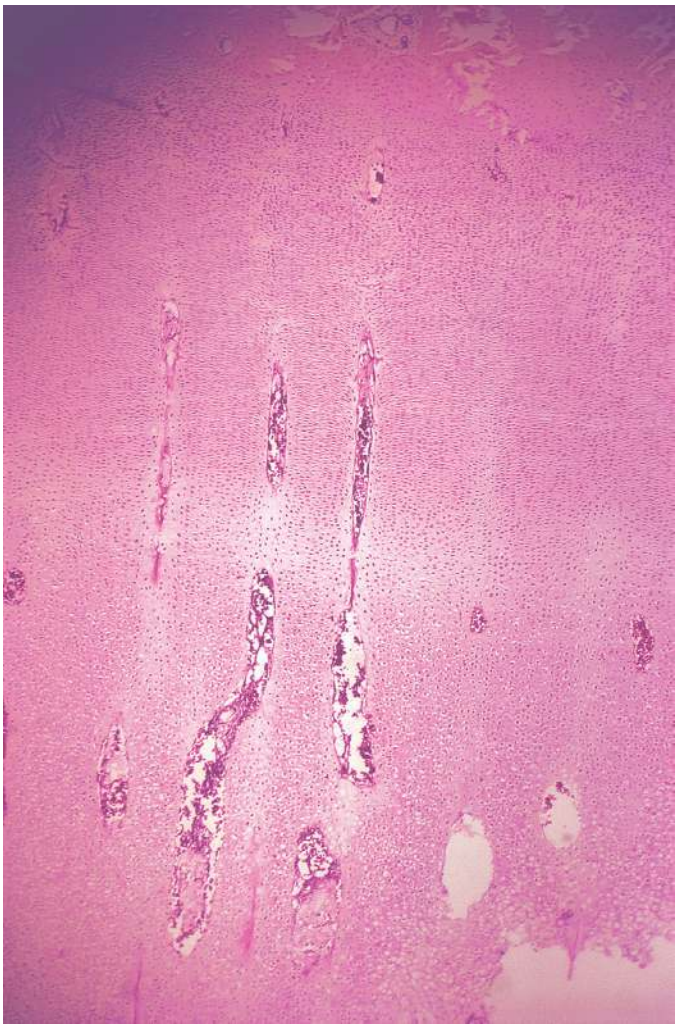


FIGURE 13-25 Section through the proximal tarsometatarsus growth plate of a growing rhea. This illustrates the depth of the proliferation (flat cell) zone, into which blood vessels penetrate deeply from the metaphyseal (distal) side.

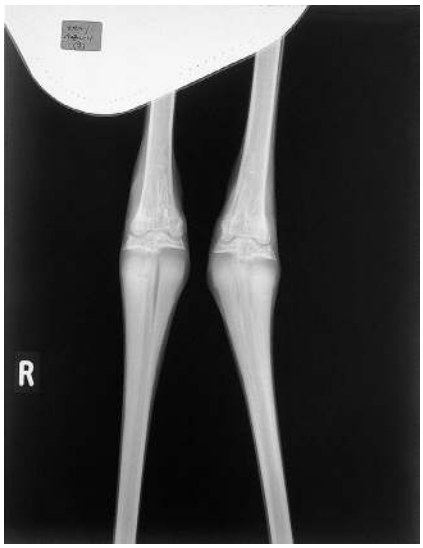


FIGURE 13-26 Lateral bending deformity of the proximal tarsometatarsus of a growing sarus crane (*Grus antigone*). The proximal end of the tarsometatarsus of this species grows rapidly and it seems likely, therefore, that this deformity was caused by a disturbance to the growth plate (trauma or metabolic), resulting in slowing of the lateral side of the plate a few days before this radiograph was taken.

FIGURE 13-27 Perosis in a kiwi chick (*Apteryx mantelli*). The *arrow* indicates the position of the lateral luxation of the gastrocnemius tendon. (Courtesy Massey University.)

Angel Wing

Reports of developmental disorders of the wings are mostly limited to descriptions of "angel wing," which is a valgus rotational deformity of the carpometacarpus resulting in a dorsolateral rotation of the primary wing feathers (Smith, 1997; Zsivanovits *et al.*, 2006; Fig. 13-29). The relatively low incidence of reporting of wing bone deformities suggests that the earlier load bearing of the pelvic limb plays a role in the developmental disorders of the hindlimb. A link between early load bearing and limb deformities has been postulated for parrots (Harcourt-Brown, 2004). The angel wing rotational deformity of the carpometacarpus is thought to be from high-protein diets leading to rapid primary wing feather maturation while the skeletal components of the wing are still immature (Zsivanovits *et al.*, 2006). The origin of the rotation is thought to be the weight of the growing feathers rather than a growth plate deformity as is seen in the pelvic limb. It can present either unilaterally or bilaterally.

Diagnosis

The diagnoses of these conditions are best based on recognizing an abnormality in wing carriage and stance or gait at a distance examination and by localizing the site of deformity using careful physical examination, radiographs, and if available, computed tomographic analysis. Early stages of these conditions can be difficult to recognize and most cases are only brought to veterinary attention when the developmental abnormality is advanced.

Distance examination of the bird is important in determining the natural resting position of the limb. More advanced locomotion studies are usually not necessary, but for the pelvic limbs, can include the use of sand flooring to examine footprint traces, force plate measurements, and video analysis of the gait. Many birds can be trained to use a treadmill safely, which allows detailed examination of gait characteristics such as stride length, placing, and crossing over at a variety of speeds (Fig. 13-30).

FIGURE 13-28 Bilateral splay leg deformity in a newly hatched shore plover (*Thinornis novaeseelandiae*). (Courtesy Massey University.)

FIGURE 13-29 Rotational deformity of the carpometacarpus in birds is known as angel wing. **(A),** Bilateral angel wing in a juvenile Northern goshawk (*Accipiter gentilis*). (Courtesy Petra Zsivanovits). **(B),** Unilateral angel wing in a juvenile white-faced heron (*Ardea novaehollandiae*). (Courtesy Massey University.)

Close physical examination of the bird should focus on looking for symmetry, limb alignment, joint mobility ranges, and stability and overall conformity of the limbs. Checking the alignment of the limbs at full flexion and extension often gives the best indication of both the origin and severity of the malformation. When dealing with an unfamiliar species, comparison of the affected limb to the contralateral limb is useful but can be problematic in bilateral conditions. Comparing the affected bird to a normal individual of the same age is ideal.

Radiographic evaluation of angular limb deformity is essential to examine the state of the growth plates, looking for evidence of asymmetry or pathology and to determine how much more limb growth remains. When limbs are pulled into full extension for standard radiographic positioning, the degree of rotational deformity is often masked. To document the resting limb position, it is recommended that additional radiographic images are taken with the joint above the deformity stabilized in a lateral or ventrodorsal projection and the lower limb placed in its resting position (Fig. 13-31). Radiographic examination of tibiotarsal rotation in ostriches was facilitated by noting the position of the tibiotarsal nutrient foramen (Hahulski *et al.*, 1999). A key recommendation is that veterinarians involved with captive rearing programs should build up a database of normal radiographic images of growing birds as both a comparison for affected birds and to establish guides to periods of growth plate closure, because these are not well documented for most species.

Computed tomographic imaging and three-dimensional acrylic modeling of the affected limbs are increasingly used by small animal surgeons to both characterize angular limb deformities and plan corrective surgery in small animals (Crosse and Worth, 2010; Kwan, *et al.*, 2014) and this modality should also be useful in birds. The authors were able to use this technique to characterize a bilateral femoral neck malformation in a juvenile kiwi that presented with a crossed limb stance suggestive of tibiotarsal rotation (Fig. 13-32). A recent study used computed tomography and densitometry to show sex-related differences in tibiotarsal maturation and periods of susceptibility to deformity in domestic geese (Charuta *et al.*, 2014).

Treatment

The treatment of developmental limb disorders in growing birds is complex. The method used for correction of the deformity must take into account the amount of limb growth remaining in the bird and the desired end result. Some techniques, such as tarsal joint arthrodesis (Meij *et al.*, 1996), may be useful for companion or aviary birds but should not be considered for free-ranging wildlife. Treatment options are best considered at two stages of limb development.

First, when significant limb growth remains conservative methods may be used to correct the malformation. The best chance of resolving rotational deformities of the carpometacarpus (angel wing) is by bandaging the wing into a normal position with a figure eight wing bandage. However, this is only successful if performed while the carpometacarpus is still mineralizing (Zsivanovits *et al.*, 2006). Similarly, for pelvic limb malformations a variety of conservative bandaging and strapping techniques have been used to apply tension to correct

FIGURE 13-30 Kiwi (*Apteryx mantelli*) walking on a treadmill for gait assessment. (Courtesy Massey University.)

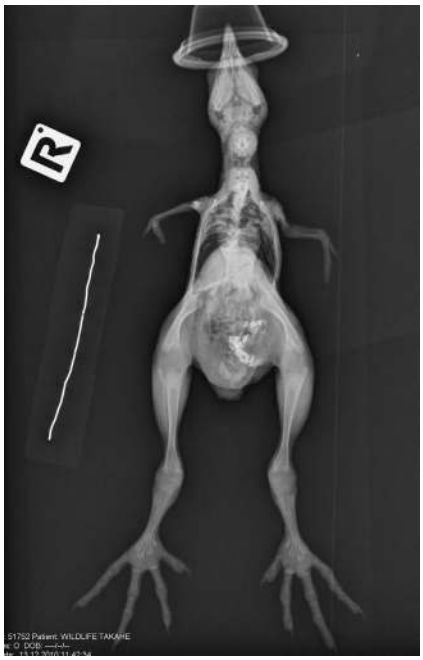


FIGURE 13-31 Ventrodorsal radiograph of a takahe chick with bilateral rotational limb deformities of tibiotarsus and tarsometatarsus. (Courtesy Massey University.)

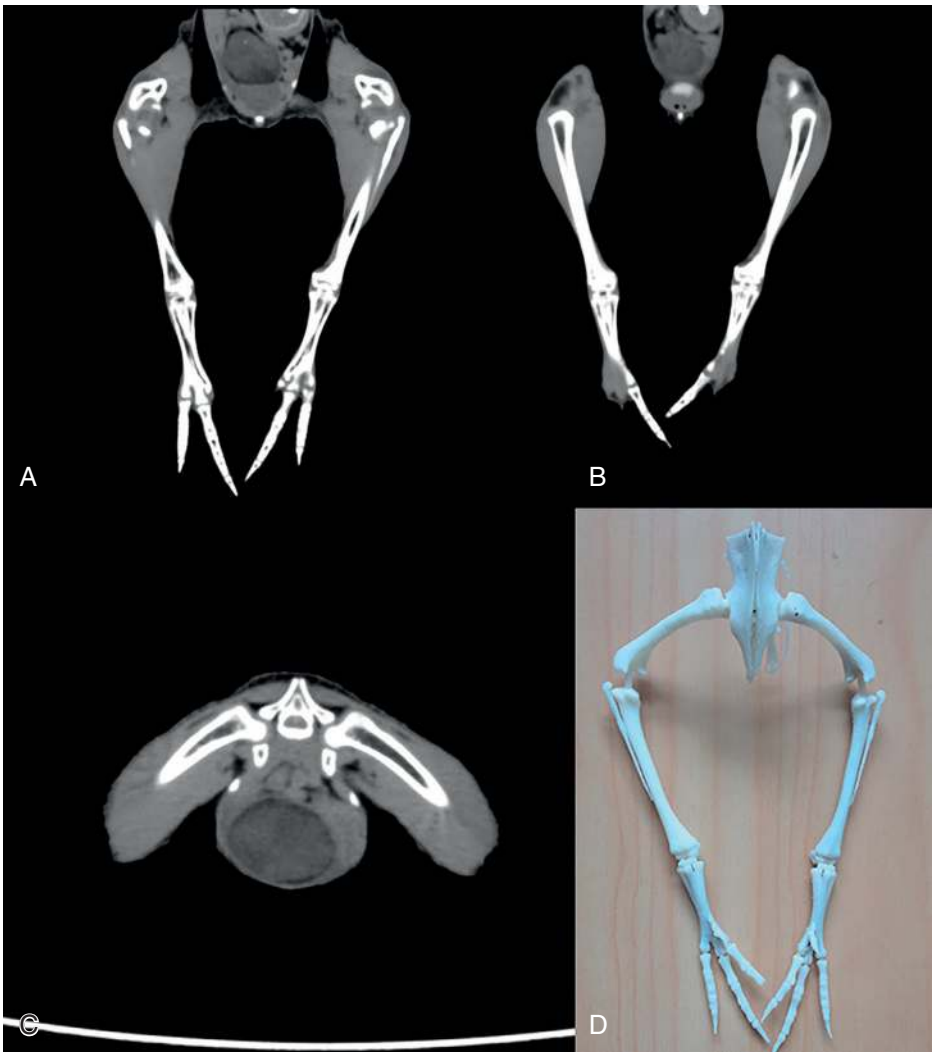


FIGURE 13-32 Computed tomographic imaging of a kiwi (*Apteryx mantelli*) that presented with a severe bilateral varus presentation of the hindlimbs, with slices showing longitudinal sections of the tarsometatarsi **(A)**, the tibiotarsi **(B)**, and the femurs **(C)**. **(D),** A 3D printed acrylic reconstruction of the synsacrum and pelvic limbs of the kiwi chick constructed from the computed tomographic imaging. (Courtesy Massey University.)

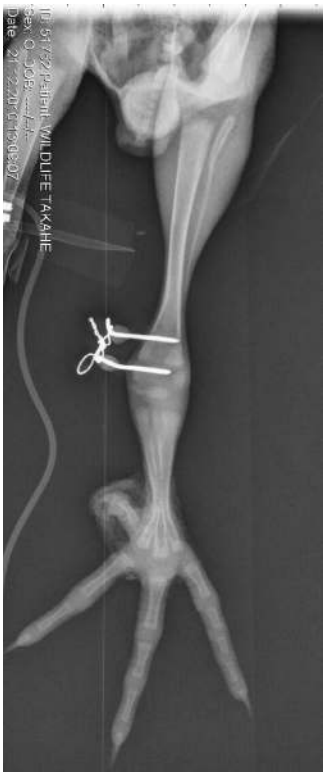


FIGURE 13-33 Ventrodorsal radiograph taken postoperatively after placement of a transphyseal bridging wire in a takahe (*Porphyrio hochstetteri*) chick with a rotational deformity of the tibiotarsus. (Courtesy Massey University.)

the malformed limb (Stiévenart, 2008). These techniques are best used for minor to moderate malformations. Complications from these techniques include failure to correct the deformity, ankylosis of joints, and traumatic fractures and joint injuries from restricted mobility in bandages.

When severe malformation is present and there is still significant limb growth remaining, the bird may have to have multiple corrective surgeries before the final position of the limb is attained. If corrective osteotomies are attempted while significant limb growth remains, there is a high complication rate from the interference of fixation devices with the growing limb. Surgical correction of pelvic limb deformities using periosteal stripping or tension band wiring across growth plates to achieve symmetric bone growth has been recommended to overcome these difficulties (Rackard *et al.*, 2002; Fig. 13-33), and this has been used successfully to correct angular limb deformities in flamingos (Zollinger *et al.*, 2005).

If limb growth is complete or the malformation is severe, then conservative methods to correct limb conformation will not work and corrective osteotomies are required. A variety of techniques extrapolated from human and small animal orthopedics has been used, but most are based on wedge osteotomies of the affected bone and stabilization with some kind of external fixator device (Meij *et al.*, 1996; Ferraz *et al.*, 2010; Fig. 13-34). Tarsal joint arthrodesis has been used to salvage some function in companion birds (Meij *et al.*, 1996). Planning of corrective osteotomies requires a sound knowledge of normal anatomy and conformation and good quality diagnostic imaging of the affected limbs. The complication rate after this type of surgery is high and includes failure to correct the deformity, reduced limb function, traumatic fractures of the limb around fixation devices, neuropathy, osteomyelitis, and tendon luxation.

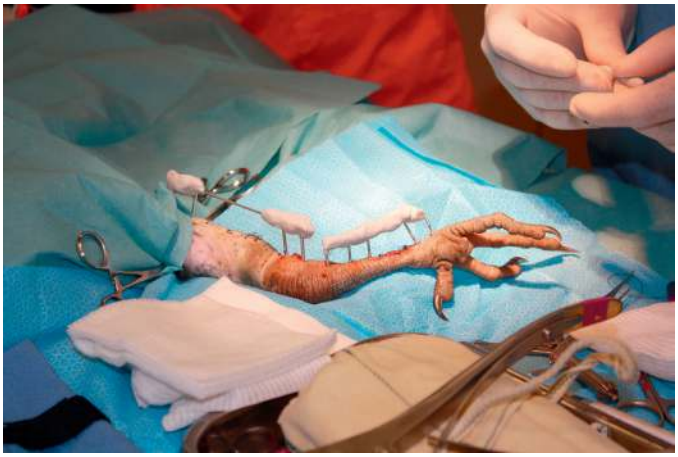


FIGURE 13-34 Type 1 external fixators on the tibiotarsus and tarsometatarsus of a takahe (*Porphyrio hochstetteri*) after wedge osteotomies of both bones for correction of rotational limb deformity. (Courtesy Massey University.)

Luxation of the gastrocnemius tendon, also known as perosis, has been treated using a variety of techniques including joint imbrication, suturing of the tendon, and deepening of the intercondylar space using a wedge technique (Wolfe, 1978; Naldo *et al.*, 1998). However, these techniques are likely to fail unless the underlying limb deformity is addressed.

Prevention

Prevention of this condition is a major concern of captive breeding facilities and revolves around correcting dietary deficiencies, improving flooring (especially relating to splay leg deformity), and keeping growth in optimal ranges. In New Zealand, the takahe (*Porphyrio hochstetteri*) recovery team dropped the incidence of rotational limb deformities in hand-reared takahe by adding potassium permanganate supplement to the diet and were able to eradicate it completely when they switched the management of chicks to parent rearing only (McLelland *et al.*, 2011).

REFERENCES

Anderson MP: Bone disease in neonatal and juvenile birds. *Proceedings of the American Association of Zoo Veterinarians*, pp 171–175, 1983.

Bauck L: Nutritional problems in pet birds, *Semin Avian Exot Pet Med* 4(1):3–8, 1995.

Charuta A, Tatara MR, Gruzewska A, et al: Morphological and densitometric research of the tibial bone in the post-natal development in domestic geese, *Anim Sci Papers Rep* 32(3):251–260, 2014.

Crosse KR, Worth AJ: Computer-assisted surgical correction of an antebrachial deformity in a dog, *Vet Comp Orthop Traumatol* 23(5):354–361, 2010.

Drew ML, Kreeger TJ: Skeletal abnormalities in wings of free-flying juvenile white pelicans (*Pelecanus erythrorhynchus*) in Minnesota, *J Wildl Dis* 22(3):447–449, 1986.

Ferraz VCM, Ferrigno CRA, Isaza R, et al: Treatment of tarsal joint deformities with hinged transarticular external fixators in three young birds, *Vet Comp Orthop Traumatol* 23(5):362–365, 2010.

Gewalt W, Gewalt I: Über Haltung und Zucht der Grosstrappe (*Otis tarda*), *Zool Garten NF* 32:265–322, 1966.

Hahulski G, Marcellin-Little DJ, Stoskopf MK: Morphologic evaluation of rotated tibiotarsal bones in immature ostriches (*Struthio camelus*), *J Avian Med Surg* 13(4):252–260, 1999.

Harcourt-Brown N: Development of the skeleton and feathers of dusky parrots (*Pionus fuscus*) in relation to their behaviour, *Vet Rec* 154(2):42–48, 2004.
Kreeger TJ, Walser MM: Carpometacarpal deformity in giant Canada geese (*Branta canadensis maxima* Delacour), *J Wildl Dis* 20(3):245–248, 1984.
Kwan TW, Marcellin-Little DJ, Harrysson OLA: Correction of biapical radial deformities by use of bi-level hinged circular external fixation and distraction osteogenesis in 13 dogs, *Vet Surg* 43(3):316–329, 2014.
McLelland JM, Gartrell BD, Roe WD: A retrospective study of post-mortem examination findings in takahe (*Porphyrio hochstetteri*), *NZ Vet J* 59(4):160–165, 2011.
Meij BP, Hazewinkel HAW, Westerhof I: Treatment of fractures and angular limb deformities of the tibiotarsus in birds by type II external skeletal fixation, *J Avian Med Surg* 10(3):153–162, 1996.
Naldo JL, Bailey TA, Samour JH: Musculoskeletal disorders in bustard pediatric medicine, *J Avian Med Surg* 12(2):82–90, 1998.
Orth MW, Cook ME: Avian tibial dyschondroplasia: a morphological and biochemical review of the growth plate lesion and its causes, *Vet Pathol* 31(4):403–404, 1994.
Pitman RL, Ballance LT, Bost CA: Incidence of wing deformities ("Angel Wing") among masked boobies at clipperton Island: life history consequences and insight into etiology, *Wilson J Ornithol* 124(3):597–602, 2012.
Prier EA, Gartrell BD, Potter MA, et al: Characterization of hatch-size and growth rates of captive and wild-reared brown kiwi (*Apteryx mantelli*) chicks, *Zoo Biol* 32(5):541–548, 2013.
Rackard SM, Carr AJ, Callanan JJ, et al: An avian model of limb deviation induced by periosteal surgery, *Res Vet Sci* 73(3):237–241, 2002.
Smith K: Angel wing in captive-reared waterfowl, *J Wildl Rehab* 20(4):3–5, 1997.
Stewart J: Ratites. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.
Stiévenart C: A novel, nonsurgical method for the treatment of tibiotarsal rotation in houbara bustard (*Chlamydotis macqueenii*) chicks, *J Avian Med Surg* 22(1):25–30, 2008.
Wolf P, Kummerfeld N, Mischok D: Disturbed health and growth of skeleton in emus und rheas due to faults in feeding and mineral supply, *Dtsch Tierarztl Wochenschr* 103(12):519–522, 1996.
Wolf P, Müller H, Kummerfeld N: Developmental disorders of the skeleton in black storks (*Ciconia nigra*), *Prakt Tierarzt* 82(4):265–269, 2001.
Wolfe DA: Surgical correction of perosis in a 3-week-old mallard duck, *Vet Med Small Anim Clin* 73(12):1567–1570, 1978.
Zollinger TJ, Backues KA, Burgos-Rodriguez AG: Correction of angular limb deformity in two subspecies of flamingo (*Phoenicopterus ruber*) utilizing a transphyseal bridging technique, *J Zoo Wildl Med* 36(4):689–697, 2005.
Zsivanovits P, Monks DJ, Forbes NA: Bilateral valgus deformity of the distal wings (angel wing) in a Northern goshawk (*Accipiter gentilis*), *J Avian Med Surg* 20(1):21–26, 2006.

DISORDERS OF THE DIGESTIVE SYSTEM

Peter Sandmeier

This section addresses diseases of the digestive system in birds. Although diseases affecting many different avian species are discussed here, there is an emphasis on those affecting psittacines, which represent the most common species seen in general practice. Common infectious diseases affecting the digestive system are addressed, but more detailed information can be found in Chapter 14.

BEAK

Anatomy and Physiology

The beak, or rhamphotheca, is a horny skin structure of keratinized epidermis covering the upper and lower jaws. The size and shape of the beak is a good indicator of the diet eaten by a particular species. Seed eaters have a strong wedge-shaped beak. Fruit eaters and insect eaters have a delicate pointy beak. The beaks of ducks and geese are wide and flat, with horny lamella along the edge, allowing them to filter the water for food. Carnivorous birds have a strong pointed curved beak (Sandmeier, 2016).

Symptoms

Most abnormalities of the beak can be readily observed by the owner and many owners will seek veterinary help before the bird has trouble eating (Box 13-1).

Pathology of the Beak Growth Plate

The most common causes of beak deformities are pathologies affecting the growth plate of the beak at the base of the cere. These include trauma, especially bites in psittacines, *Cnemidocoptes* mange in budgerigars and occasionally other psittacines species, chronic nasal discharge (Fig. 13-35, Raidal and Butler, 2001), or neoplasia.

Congenital Deformities

Congenital deformities commonly seen in juvenile psittacines include scissor beaks and prognathia inferior. Causes of these deformities are speculative, but include metabolic bone disease, hand-feeding technique, hereditary causes, or trauma to the growth plate of the beak. Congenital deformities can be corrected in nestling parrots that still have a soft beak (Fig. 13-36; Tully *et al.*, 2005, Harcourt-Brown, 2013). Correction in adult birds, however, is difficult. For more details see Chapter 8.

BOX 13-1 Causes of Beak Abnormalities

- Trauma to the beak or growth plate
- *Cnemidocoptes* mites (especially in budgerigars)
- Chronic rhinitis
- Congenital or acquired beak deformities such as scissor beak or prognathia inferior
- Neoplasia
- Viral infection (circovirus causing psittacine beak and feather disease)
- Malnutrition causing hyperkeratosis and bad horn quality
- Metabolic disorders, often because of liver disease

FIGURE 13-35 Chronic nasal discharge can destroy the beak growth plate below the nostril. In severe cases with bilateral pathology this can lead to an upper beak consisting of three separate parts.

FIGURE 13-36 Correction of scissor beak in a juvenile bird with a soft maxilla is possible.

FIGURE 13-37 Severe bite wounds of the maxilla can lead to the loss of the entire upper beak. Birds that survive the acute incident can do well on an adjusted diet.

Trauma

Acute beak trauma is normally from bite wounds in psittacines (Fig. 13-37) or trauma caused by mesh or during transport in long-beaked birds such as storks and ibises. Treatment includes debridement and disinfection and protection of the traumatized area using composite, which also gives the patient an occlusal surface that allows independent feeding. Fractures of long mandibles or maxillae, for example, in storks, can be stabilized using an external fixator such as FESSA (Fig. 13-38; Hatt *et al.*, 2007).

Malnutrition

Poor beak quality can be caused by malnutrition, which in psittacines is a cascade of deficiencies resulting in a dull, flaky beak (see Chapter 3).

Infections

Infections affecting the horny structure of the beak include psittacine beak and feather disease (caused by circovirus) and bacterial or fungal infections (Figs. 13-39 and 13-40) affecting the epidermis and dermis of the beak.

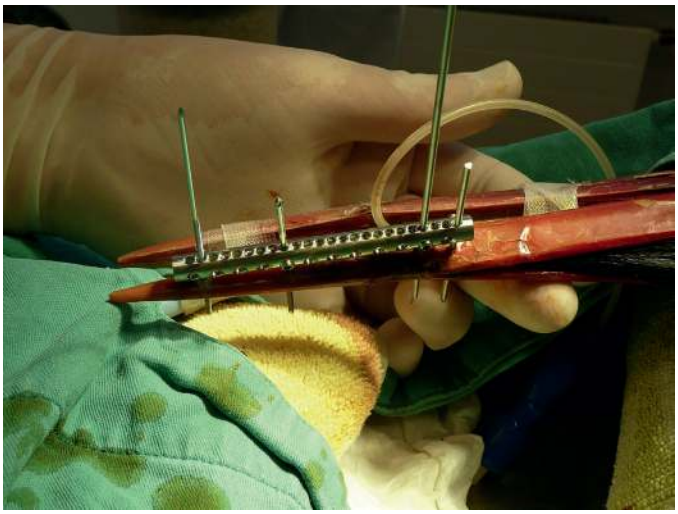


FIGURE 13-38 In storks and other long-billed avian species fractures of the beak can be treated using a FESSA type II external fixator.

FIGURE 13-39 Fungal infections of the horny structures of the beak require intensive and long-term treatment.

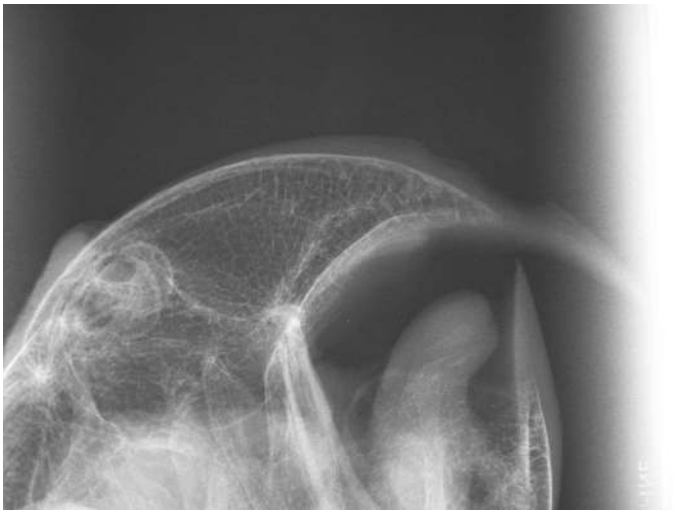


FIGURE 13-40 Radiographs can help assess involvement of the bony structures in chronic infections of the beak.

Metabolic Disease

Metabolic disease is a very common cause of poor beak quality and secondary deformation of the beak. The most common cause for this is chronic liver disease (see below, Liver). Many of these birds will also have overgrown nails and those suffering from hepatic lipidosis will be obese.

Diagnostic Workup and Treatment

A serious workup for a deformed beak or beak of bad quality would include an exact inspection of the beak and beak growth plate under isoflurane anesthesia, testing for circovirus and a chemistry panel including bile acids, and whole-body radiographs to assess for liver disease. Additional testing could include cultures of beak lesions and radiographs of the beak to assess the bony structure of the maxilla and mandible (see Fig. 13-40).

In most cases a deformed beak will need regular trimming under isoflurane anesthesia using a Dremel. Treatment of bacterial or fungal infections of the horny structures includes local trimming of the affected beak area and systemic treatment based on culture and sensitivity. Fungal infections affecting the dermis of the beak need to be treated with an antifungal, such as itraconazole, for at least 2 months.

ORAL CAVITY, TONGUE, AND SALIVARY GLANDS

Anatomy and Physiology

The oropharyngeal cavity is open to the nasal cavity through the choanal slit. During inspiration this slit is closed by the tongue. The infundibulum is caudal to the choana and at the opening of the eustachian tubes. The number of salivary glands in the oropharynx depends on the diet of the species and is higher in seed eaters than fish and meat eaters.

The size, form, and function of the tongue vary according to feeding and living habits of the bird. In many birds it is used to move food within the beak, and in others, such as hummingbirds or woodpeckers, it is used to collect food and can be obtruded. Nectar-eating species, such as lories or honey eaters, have a specialized brush-tipped tongue. In psittacine birds the tongue also has a tactile function. Compared with mammalian species, the avian tongue has few taste buds. (Sandmeier, 2016).

Symptoms

Symptoms include the inability to eat and swallow, salivation, open beak, dyspnea, and plaques within the oral cavity sometimes partially occluding the epiglottis (Box 13-2).

Metaplasia from Hypovitaminosis A

Metaplasia of salivary and lacrimal gland epithelium from hypovitaminosis A is common in larger psittacines (especially African grey parrots) that eat a seed-based diet. Accumulation of keratin and necrotic, cellular debris leads to whitish plaques within the oral cavity. The most common location is sublingual (Fig. 13-41); (which can often also be visualized as an external swelling at the base of the lower beak), but they can also be seen within the mucosa of the palate along the choanal slit. Secondary bacterial or fungal infections often lead to formation of granulomas that will either occlude the epiglottis or develop within the choanal slit. Treatment includes injectable vitamin A, surgical debridement of granulomas, and lancing and removal of mucosal plaques. Systemic treatment of secondary bacterial and fungal infections is based on culture and sensitivity testing. The prognosis is good in parrots that only suffer from metaplastic glands if surgically removed and a long-term diet improvement can be achieved, but it is often doubtful to poor in parrots with granulomas and large areas of inflamed mucosa within the oral cavity, choanal slit, and nasal cavity.

Infectious Stomatitis and Plaques within the Oropharynx

Causes of infectious stomatitis and plaque within the oropharynx (see Box 13-2) include avipoxvirus, bacterial infections (often Enterobacteriaceae, *Pseudomonas aeruginosa* [Samour, 2000]), trichomoniasis (especially in pigeons, budgerigars, and raptors fed on pigeons), candidiasis (Samour and Naldo, 2002), and infections from *Capillaria* spp. Other diseases observed within the oropharynx include neoplastic lesions and traumatic lesions.

Diagnostic Workup

After a precise inspection of the oropharyngeal cavity, swabs should be taken. If necessary, endoscopy under isoflurane anesthesia can be performed. Fresh swabs of oral plaques should be examined directly with a drop of saline solution and a coverslip looking for motile flagellates such as *Trichomonas* species. A Gram or Diff-Quick stain sample will help diagnose yeast such as *Candida* spp. and bacteria or *Capillaria* spp. (uncommon). Differentiation of bacteria is achieved by culture and sensitivity testing.

BOX 13-2 Causes of Oral Plaques

- Metaplasia as a result of hypovitaminosis A
- Avipoxvirus
- Bacterial stomatitis
- Trichomoniasis
- Candidiasis
- *Aspergillus* granulomas
- *Capillaria* spp.
- Neoplasia
- Trauma

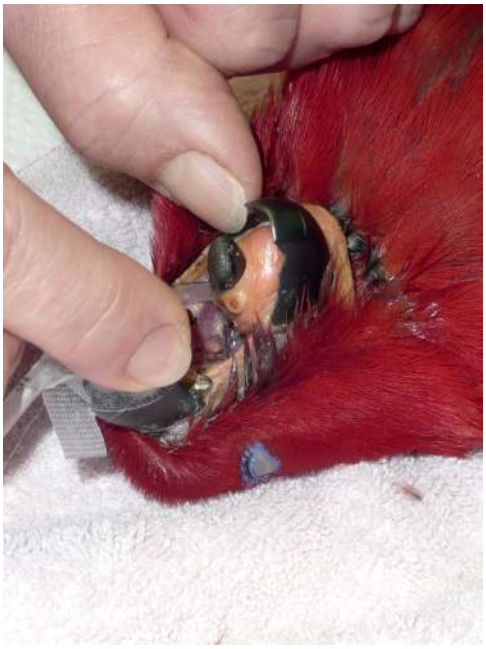


FIGURE 13-41 Metaplasia of salivary glands from hypovitaminosis A leads to accumulation of keratin and necrotic, cellular debris causing severe sublingual swelling. This can normally be seen as an external swelling at the base of the beak.

ESOPHAGUS AND CROP

Anatomy and Physiology

The esophagus runs down the right side of the neck adjacent to the trachea. The diameter and number of longitudinal folds is larger than in mammals, especially in species that eat large portions of food like birds of prey and fish eaters. The crop is a dilatation of the esophagus situated just proximal to the entrance to the pectoral cavity and directly under the skin. It is generally larger in granivorous and carnivorous birds; in many insectivorous birds no distinct crop can be observed. The crop serves as a storage chamber allowing a continuous supply of nutrition to the gastrointestinal tract. Although food in the crop is predigested by saturation of food with saliva and gastric fluids by retrograde esophageal movement, there is no actual production of digestive fluid or enzymes by the mucosa of the crop wall. Pigeons have the unique capability of producing crop milk. Under the influence of prolactin the epithelial cells of the crop wall are desquamated producing a milk-like secretion that is fed to the nestlings by both male and female parents (Sandmeier, 2016).

Symptoms

Symptoms of diseases affecting the crop and esophagus include regurgitation/vomiting (often leading to glued feathers on the head), but are sometimes only noticed because the bird is chewing on regurgitated crop content and has slightly pasted feathers along the aperture of the beak (Box 13-3). Other symptoms include anorexia and weight loss, sometimes diarrhea, and a visible or palpable swelling of the crop.

Ingluvitis and Esophagitis

Trichomonas infections are one of the main causes of crop and esophageal disease. These are especially common in budgerigars (Sandmeier, 2014), pigeons, and birds of prey (Samour and Naldo, 2003; see Chapter 14). Other causes include viral or bacterial infections, yeast infections caused by *Candida* spp. (Fig. 13-42) or *Macrorhabdus ornithogaster* (especially in budgerigars, see Chapter 14), and toxins or uremia.

Other Crop Diseases

Sour crop develops as a secondary problem of crop stasis. This symptom is especially common in hand-fed nestling birds. Common causes include inadequate hygiene with food preparation and feeding utensils, insufficient temperature within the brooder, and/or feeding formula that is too cold. As the hand-feeding formula remains in the crop it starts fermenting and a secondary growth of yeast and bacteria develop.

Crop burns are also regularly observed in juvenile hand-fed psittacines. Hand feeding formula warmed in the microwave and not well stirred can develop hot spots that scorch the mucosa of the crop, leading to a necrosis of the crop wall and the overlying skin. Once the scab that develops falls away the bird will lose hand-fed formula through this defect leading to dirty and pasted feathers at the base of the neck (Fig. 13-43). These crop burns need to be treated surgically.

Impaction of the crop is commonly caused by phytobezoars (Fig. 13-44). These develop in small psittacines, such as cockatiels and budgerigars, from ingestion of frayed sisal fibers that are nibbled off perches. In backyard poultry phytobezoars develop in birds that have access to nonmowed pastures and eat large amounts of long grass. Although phytobezoars can be removed through the oral cavity using foreign-body forceps, they are often too big and the risk of asphyxiation while pulling the bezoar through the oropharynx is considerable. Surgical removal through a crop incision is the method of choice.

Although uncommon, ingluvial calculi have been described (Arnall and Keymer, 1975). Pendulous crops can develop as a sequel to ingluvial inflammation and stasis. The most common cause for pendulous

BOX 13-3 Causes of Regurgitation/ Vomiting

- *Trichomonas gallinae* (especially in budgerigars)
- *Macrorhabdus ornithogaster* (especially in budgerigars)
- Bacterial or mycotic ingluvitis
- Toxic ingluvitis (plants, chemicals, and heavy metal)
- Foreign bodies
- Severe hepatic or renal disease
- Stress, trauma, obstructive lesions
- Neoplasia
- Partner feeding, courtship
- Physiologic cast regurgitation in birds of prey

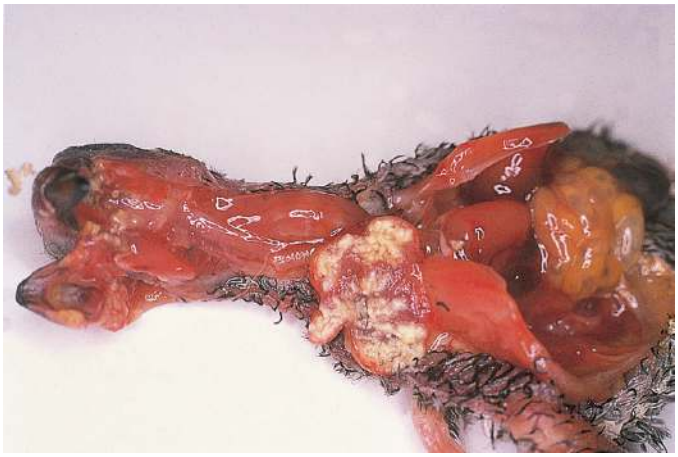


FIGURE 13-42 White necrotic plaques involving the mucosa of the crop and the buccal cavity caused by *Candida* spp.

FIGURE 13-43 Crop burns are commonly caused by hand feeding formula that is warmed in a microwave without stirring.

FIGURE 13-44 Phytobezoar removed from the crop of a cockatiel. Sisal fibers are nibbled off older sisal perches that have started fraying.

FIGURE 13-45 Undigested seeds in the feces are commonly seen in psittacines suffering from proventricular dilatation disease.

crop in female budgerigars is a weakness of the connective tissue following hormonal imbalances. Accompanying signs can include abdominal hernia, abdominal distention from cystic ovaries, and hyperostosis of the long bones seen on radiographs.

Neoplasia affecting the crop and esophagus can include leiomyosarcoma and squamous cell carcinoma, but neoplasia of the avian gastrointestinal system is not very common.

Diagnostic Workup and Treatment

The diagnostic workup of crop disease starts with the direct microscopic examination of a crop flush, especially looking for motile flagellates such as *Trichomonas* spp. Diff-Quick or Gram stains are needed to diagnose *Candida* spp. and bacteria. Culture and sensitivity testing should be considered. Foreign bodies as well as inflammation or neoplastic lesions of the crop wall can be observed during an endoscopic inspection of the crop. A pendulous crop can be diagnosed on radiographs when using barium sulfate as a contrast medium.

PROVENTRICULUS AND VENTRICULUS

Anatomy and Physiology

The avian stomach is divided into a proventriculus and ventriculus or gizzard. The proventriculus is a glandular stomach producing digestive enzymes similar to the stomach in dogs and cats. In seed-eating species such as psittacine birds, chickens, and pigeons the ventriculus is extremely muscular and the mucosal surface is covered by a koilin layer (cuticle). Together with the grit within the ventriculus a grinding of seeds is achieved. In piscivores and carnivores the ventriculus only has a thin muscular layer and no cuticle.

Food in the stomach is propelled between proventriculus and ventriculus in a series of cycles combining the mechanical and digestive functions of the stomach. In birds of prey and owls undigestible parts such as skin and bones are formed into a pellet within the ventriculus and then regurgitated (Sandmeier, 2016).

Proventricular Dilatation Disease in Psittacines

Proventricular dilatation disease, caused by avian bornavirus, is the most common cause of proventricular disease in psittacines. Classical signs include shedding undigested seeds in the feces (Fig. 13-45), weight loss, vomiting and/or diarrhea, ataxia, and tremor. In most diseased psittacines proventricular dilatation can be observed on radiographs (Figs. 13-46 and 13-47). For details and literature see Chapter 14.

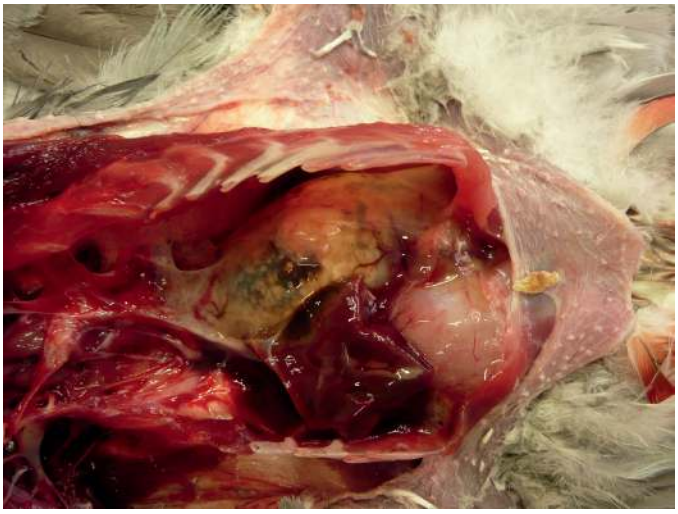


FIGURE 13-46 A dilated and thin-walled proventriculus observed at necropsy is suspicious for proventricular dilatation disease.

Macrorhabdus Ornithogaster

Macrorhabdus ornithogaster is the most common disease in budgerigars seen in daily practice. Still commonly called "Megabacteria", this yeast that colonizes the proventricular mucosa should be considered in every ill budgerigar, especially with symptoms of weight loss, diarrhea, soiled pericloacal feathers, or vomiting. For details and literature see Chapter 14.

Proventricular or Ventricular Dilatation

In addition to a bornavirus infection, other causes including bacterial proventriculitis, fungal proventriculitis, atony of the proventriculus

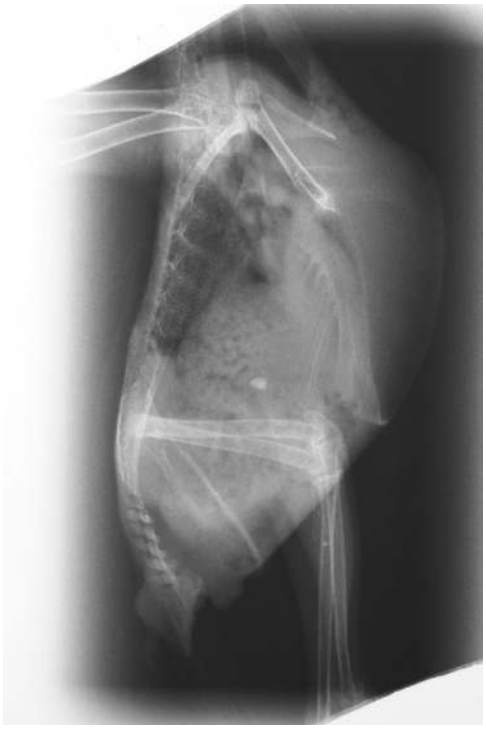


FIGURE 13-47 On radiographs proventricular dilatation disease typically presents with a dilated proventriculus filled with flocculent, radiodense food content.

BOX 13-4 Causes of Proventricular Dilatation on Radiographs

Proventricular dilatation disease in psittacines
Proventricular atony caused by heavy metal toxicity
Bacterial or mycotic proventriculitis
Proventricular foreign body
Neoplasia of the proventriculus

caused by lead poisoning, and neoplasia, foreign bodies, or impaction should be considered in psittacines with a dilated proventriculus demonstrated on radiographs (Box 13-4).

Impaction of the proventriculus and ventriculus is seen in falcons that ingest fine sand adhered to their food, in backyard chickens that ingest large amounts of long grass, and in juvenile ostriches that repeatedly ingest coarse and fibrous vegetable matter.

Stomach Worms

Various species of stomach worms have been described in different avian species. Because they all require an intermediate invertebrate host, worms are only found in free-living and recently captured birds or those kept in outdoor enclosures with soil and growing plants. The most common stomach worm seen in general practice is *Amidostomum anseris* in ducks and geese (Fig. 13-48). For more details see Chapter 14.

Diagnostic Workup

The proventriculus and ventriculus are difficult to access. Radiographic assessment of size, contour and content with or without contrast studies using barium sulfate is the most useful diagnostic tool. Experienced clinicians can endoscopically access the proventriculus of middle to larger sized birds for visual assessment or sampling of content and mucosa. Fecal examinations and examination of crop content can help give additional information on proventricular and ventricular diseases.

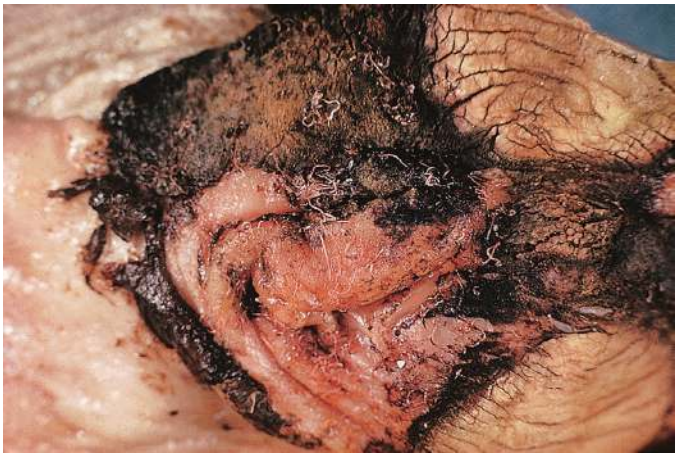


FIGURE 13-48 Gizzard worms (*Amidostomum anseris*) in a goose.

INTESTINES

Anatomy and Physiology

In general the avian intestine is shorter than in mammals, but similar to mammals, the intestine of seed-eating birds is longer than that of fish-eating or meat-eating birds. The duodenum is loop shaped, surrounds the pancreas, and receives the opening of the bile and pancreatic ducts. The jejunum and ileum are arranged in a series of coils and allow digestion under the influence of bile and pancreatic enzymes.

The colorectum is very short and opens into the coprodeum of the cloaca. Resorption of water out of urine and feces from the colorectum and cloaca is important for species living in arid regions.

Birds have paired ceca. In many species (e.g., psittacine birds, passerines, pigeons, and raptors) they are vestigial, whereas in others (e.g., chickens, waterfowl, and ratites), the ceca are very voluminous and contain lymphatic tissue. In these species cellulose is digested by the bacterial content within the ceca. Content of the ceca is voided separately as a dark brown glutinous mass (Sandmeier, 2016).

Viral Infections

Viral infections that also affect the intestinal tract and that need to be considered when dealing with birds with diarrhea include Newcastle disease (paramyxovirus type 1), Pacheco disease in psittacines (psittacine herpesvirus 1), anatid herpesvirus enteritis in ducks and geese, adenovirus in psittacines and pigeons, rotavirus in pheasants, etc. For more details on viral infections see Chapter 14.

Bacterial Infections

Common causes of bacterial enteritis in birds include many species of Enterobacteriaceae (Fig. 13-49), *Pseudomonas* and *Aeromonas* spp., and *Chlamydophila psittaci.* Clostridial necrotic enteritis is commonly seen in quails and other Galliformes, but can affect many other species. *Mycobacterium* spp. is most commonly isolated from the alimentary tract. Typical symptoms are often weight loss and death, rather than acute diarrhea. For more details on bacterial infections see Chapter 14.

Protozoal Infections

Coccidial infections are a common cause of diarrhea in many avian species, especially Galliformes, but also Columbiformes, Anseriformes,

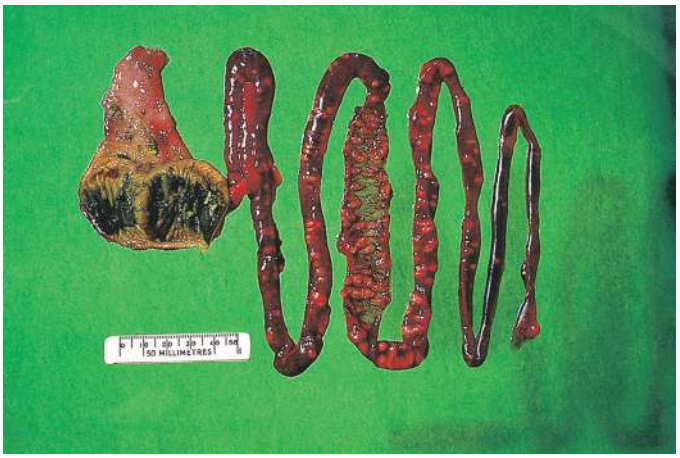


FIGURE 13-49 Multiple necrotic nodules throughout the severely inflamed intestine of a macaw caused by *Salmonella typhimurium*.

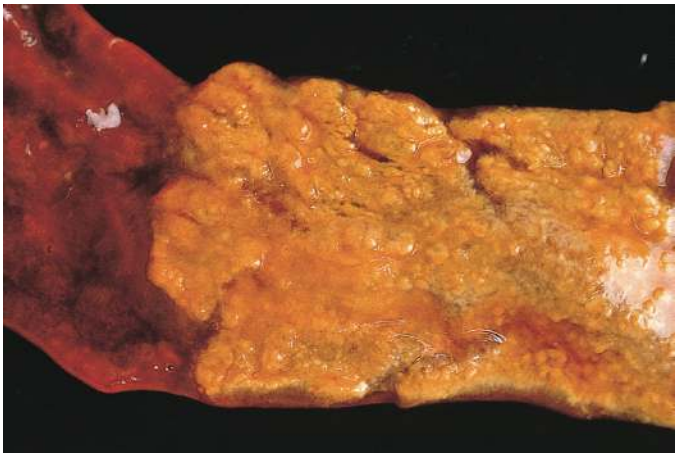


FIGURE 13-50 Severe diphtheric enteritis in a toucan caused by a heavy infestation of *Capillaria* spp.

FIGURE 13-51 Nodular typhlitis in a pheasant caused by *Heterakis* spp.

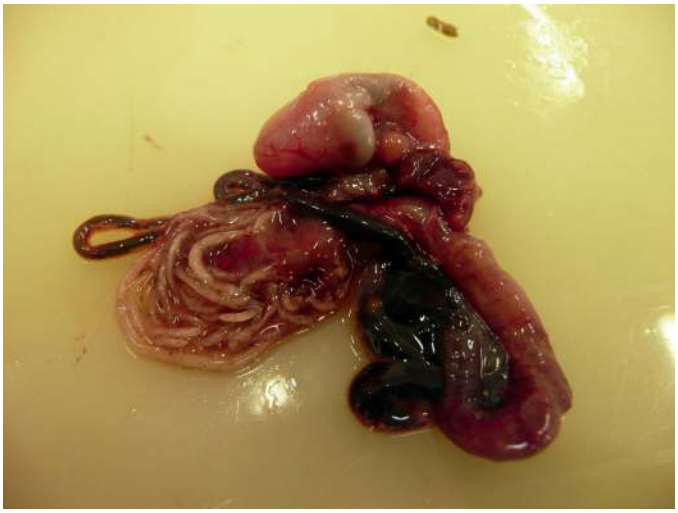


FIGURE 13-52 Cloacal papilloma with a cauliflower-like appearance in an Amazon parrot.

Passeriformes, and many others. Flagellate infections include giardiasis and trichomoniasis in many species, histomoniasis in pheasants and cochlosomiasis in finches and other passerines. For more details on protozoal infections see Chapter 14.

Nematode, Cestode, and Trematode Infections

Common nematode infections in pet birds include infections with ascarids and *Capillaria* spp. (Fig. 13-50). *Heterakis* spp. are common in the ceca of Galliformes and other species (Fig. 13-51). In psittacines ascarids can lead to sudden death from worm-induced ileus (Fig. 13-52) rather than from diarrhea.

Because tapeworms require an intermediate invertebrate host they are only found in free-living and recently captured birds or those kept in outdoor enclosures with soil and growing plants. Trematode infestations are uncommon in avian species. For more information on parasite diseases see Chapter 14.

Other Diseases of the Intestines

Other noninfectious causes of diarrhea include toxic enteritis caused by poisonous plants, heavy metal, and other toxins. Intestinal volvulus or invagination, intestinal foreign bodies, intestinal impaction, or intestinal neoplasia can all cause ileus symptoms or a rupture of the bowel leading to peritonitis.

Various extraintestinal diseases can also lead to diarrhea or symptoms that are often mistaken for diarrhea. These include all causes of polyuria, salpingitis, and other diseases of the oviduct, diseases of the liver, and diseases of the cloaca.

Diagnostic Workup and Treatment

Diagnostic workup of intestinal disease is based on the evaluation of the feces (see Fig. 13-45 and Figs. 13-53 to 13-59). This includes parasitological evaluation, Diff-Quick or Gram stains, chlamydia testing, bacterial cultures and sensitivity, and, if necessary, virus testing. Radiographic examination (with or without contrast studies using barium sulfate) of the coelomic cavity helps diagnose ileus symptoms, foreign bodies, and neoplastic lesions. Radiographs in combination with blood sampling are useful for eliminating extraintestinal causes of diarrhea.

In addition to treating the primary cause a symptomatic treatment of patients with diarrhea is also important. Treatment includes replacement of lost fluids and electrolytes, stopping additional losses, and supporting intestinal function. Fluids and electrolytes are replaced using intravenous, interosseous, or subcutaneous infusions in addition to oral supplementation using a diluted hand-feeding formula. Various injectable products that influence gut motility can be used, as well as oral products against diarrhea (see Appendix 6). An important aspect of supporting intestinal function is the use of prebiotics to support gut flora and probiotics that can help rebuild healthy gut flora.

FIGURE 13-53 "Popcorn" feces from a budgerigar caused by pancreatic insufficiency.

FIGURE 13-54 Bird owners commonly cannot differentiate between polyuria as shown here and diarrhea.

FIGURE 13-55 Biliverdinuria and hematuria in this sample that does not contain any feces.

FIGURE 13-56 Birds with liver disease will develop biliverdinuria.

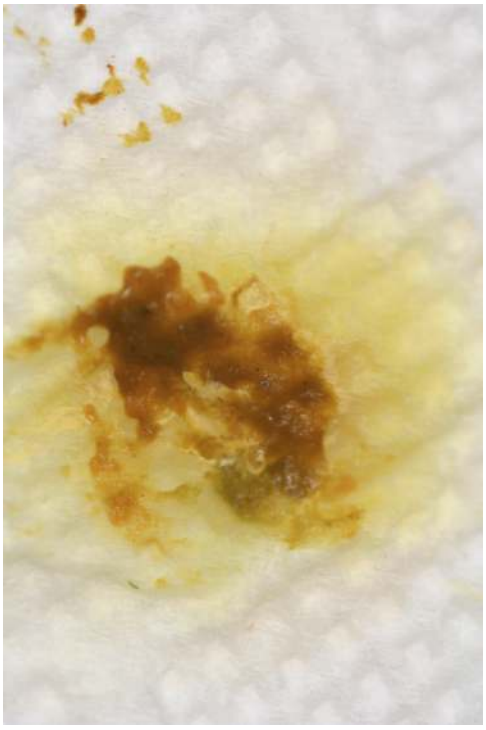


FIGURE 13-57 Diarrhea is characterized by loose feces and needs to be differentiated from polyuria.

CLOACA

Anatomy and Physiology

The cloaca consists of three compartments and collects the excretion of the digestive tract, the urinary tract, and the genital tract. The proximal coprodeum is the extension of the colorectum and is not distinctly delineated from this. There is, however, a distinct fold between the coprodeum and the subsequent urodeum. During defecation the coprourodeal fold is everted to the cloacal lips protecting the ureteral and reproductive openings from fecal contamination.

The urodeum contains the openings of the urinary and reproductive tracts. The ureters open dorsally and the spermatic ducts open ventrolaterally into the urodeum. In females the oviduct opens

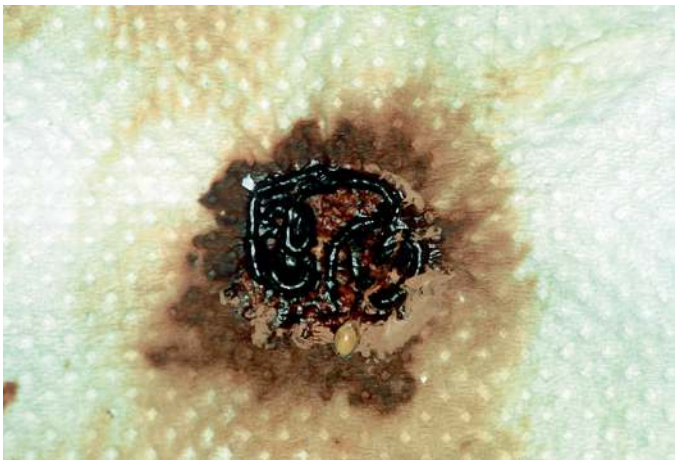


FIGURE 13-58 Hematochezia can easily be differentiated from hematuria.

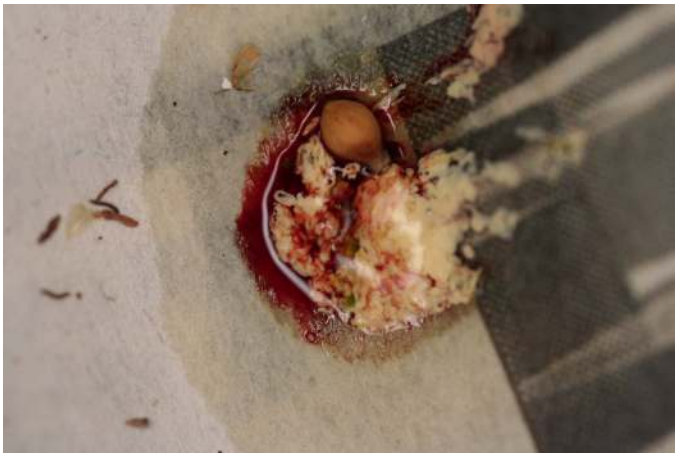


FIGURE 13-59 Hematuria can easily be differentiated from hematochezia.

ventrolaterally on the left side into the urodeum. The urodeum is once again separated from the onfollowing proctodeum by the uroproctodeal fold.

The proctodeum is the distal shortest section of the cloaca between the uroproctodeal fold and the vent. In juvenile birds it contains the dorsal opening into the cloacal bursa (see the lymphatic system). Although most avian species have no phallus, some such as ratites and waterfowl have an erectile phallus (Sandmeier, 2016).

Cloacitis

Cloacitis is often associated with simultaneous enteritis, salpingitis, or chronic renal disorders. Papillomas, neoplasia, cloacal calculi, or cloacal prolapses will also induce a cloacitis. Symptoms include a pasted vent and straining. Endoscopic evaluation of the cloaca is used to make a diagnosis.

Per Cloacal Prolapses

Cloacal prolapses need to be differentiated from prolapses of the rectum, of the oviduct, or of the phallus (Box 13-5 and Fig. 13-60). Common causes include egg binding, chronic hypersexual behavior in imprinted parrots (especially cockatoos), intracloacal masses, chronic diarrhea, or other intestinal irritations, chronic salpingitis, chronic cloacitis, and pressure from intracoelomic masses (Forbes, 2013). The

BOX 13-5 Causes of Per Cloacal Prolapses

- Egg binding
- Chronic hypersexual behavior in imprinted parrots (especially cockatoos)
- Cloacal papillomas
- Cloacal neoplasia
- Chronic cloacitis
- Chronic diarrhea
- Chronic salpingitis
- Prolapse of the phallus in waterfowl
- Intracoelomic masses

FIGURE 13-60 Cloacal prolapse as a sequel to egg binding in a budgerigar.

phallus can prolapse in male waterfowl and other species with a phallus. In individual cases the cause cannot always be determined. Apart from treating the primary cause, the prolapse needs to be kept moist and clean. Necrotic mucosa needs to be removed and the cloaca must be repositioned and fixed. This can be attained by performing a percutaneous cloacopexy or a purse string suture. Severe cloacal prolapses often can only be permanently repaired by performing a coeliotomy and bilateral surgical stitching of the proximal aspect of the cloaca to the caudal rips and by performing a cloacopexy by incorporating the ventral aspect of the cloaca into the surgical closure of the linea alba.

Cloacal Masses

Along with neoplasia in many species, large psittacines also commonly present with cloacal papillomas with a cauliflower-like appearance (see Fig. 13-52). For more information on these herpesvirus-induced papillomas see Chapter 14.

Diagnostic Workup

A thorough palpation and visual inspection of the cloaca, preferably performed under isoflurane anesthesia, is important. Endoscopic evaluation of the air-infiltrated cloaca, possibly including biopsies of the cloacal mucosa, can provide important additional information. Radiographic interpretation of the cloaca can often be facilitated by infiltrating barium sulfate through the vent into the cloaca. Samples obtained from the cloaca can be cultured and used for cytology or for virus PCRs. (Taylor and Murray, 2002).

PANCREAS

Anatomy and Physiology

The pancreas is located within the duodenal loop. It features a dorsal and a ventral lobe, as well as a small splenic lobe, which contains most of the endocrine tissue. The exocrine pancreas produces the same digestive hormones as in mammals (amylases, lipases, and proteases). The endocrine pancreas produces insulin, glucagon, and somatostatin. Current evidence indicates that in avian species glucagon is the dominant pancreatic hormone, whereas in mammals, this is insulin (Sandmeier, 2016).

Pancreatic Disease

An acute pancreatitis is associated with acute general illness without specific symptoms, but a chronic lesion will cause maldigestion, loss of weight, and general chronic illness. In budgerigars a chronic pancreatic insufficiency can lead to pale bulky feces that look like popcorn (see Fig. 13-53). In individual cases, an etiologic diagnosis often cannot be made. Known causes of pancreatitis include paramyxovirus and other viral infections. Zinc intoxication will cause atrophy and degeneration of the exocrine pancreatic tissue (Speer, 1998).

Diagnostic Workup and Treatment

Diagnosis of acute pancreatic disease in avian patients is difficult. Many birds will be systemically ill and other signs of disease will often predominate. Although not very specific, high amylase and lipase values could be suggestive of pancreatic disease. Signs of maldigestion such as popcorn feces or nondigested seeds in the feces could be an indication of chronic pancreatic disease. Supportive treatment can help an avian patient survive an acute pancreatitis. Exocrine pancreas function can be supported by supplementing affected patients with pancreas enzymes.

LIVER

Anatomy and Physiology

The avian liver has a large right lobe and a small left lobe. The bile duct from each lobe drains into the duodenum. The right bile duct contains a gallbladder, which is missing in many species such as Psittaciformes, pigeons, and some Passeriformes.

Because of the lack of biliverdin reductase, the end product of hemoglobin metabolism within the liver in avian species is biliverdin and not bilirubin. Therefore an icteric bird will develop biliverdinuria, which can be recognized by green coloring of the urates (Sandmeier, 2016).

Symptoms

Clinical symptoms of hepatic disease are often not very specific. Any fluffed-up and anorectic bird or birds with weight loss could be suffering from liver disease. Many will have diarrhea or poor fecal consistency, and those suffering from acute, severe hepatic disease will develop biliverdinuria (see Fig. 13-56). Chronic liver disease affecting metabolic processes will lead to changes in feather color (Fig. 13-61) and overgrown nails and beak. Most birds suffering from hepatic lipidosis will be obese. Severe hepatomegaly or ascites after liver disease (Fig. 13-62) can lead to abdominal distention, which may cause dyspnea. Signs of central nervous system disease can be caused by hepatic encephalopathy (Davies, 2000).

Hepatitis

Common causes of viral hepatitis include herpesvirus in many species (Pacheco disease in psittacines, inclusion body disease in birds of prey, pigeon herpesvirus, and Marek's disease in Galliformes), adenovirus in many species, goose virus hepatitis caused by parvovirus, and acute polyomavirus and circovirus in psittacines. For more details see Chapter 14.

FIGURE 13-61 Chronic liver disease affects metabolic processes leading to change in feather color, as seen in the Amazon on the left in comparison to its healthy partner.

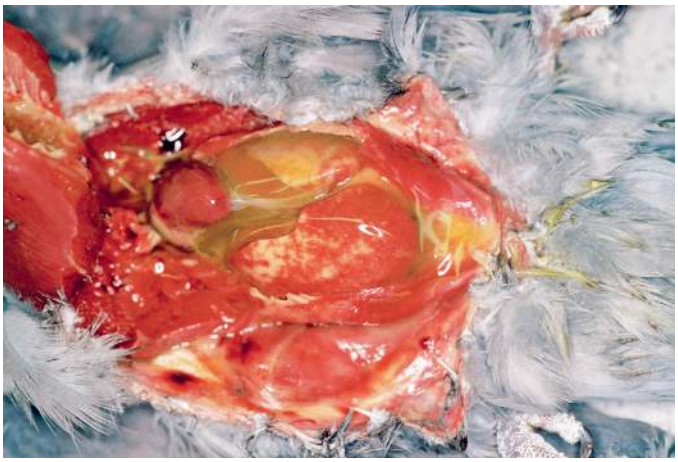


FIGURE 13-62 Severe liver disease leads to ascites.

All bacterial pathogens that cause enteritis can also cause hepatitis. Common species include all Enterobacteriaceae, *Mycobacteria* spp. (Fig. 13-63), *Yersinia pseudotuberculosis* (Fig. 13-64), and *Chlamydia psittaci*. For more details, see Chapter 14. Aspergillosis and other fungal infections do not commonly cause acute hepatitis, but mycotoxins will lead to chronic liver disease and cirrhosis. Parasites that commonly cause hepatitis include *Histomonas meleagridis*, causing black-head disease in pheasants and turkeys, and *Trichomonas gallinae* in budgerigars, which as a sequel to ingluvitis and enteritis, can invade the liver leading to acute hepatitis. For more details see Chapter 14.

Hepatic Lipidosis

Hepatic lipidosis is commonly seen in psittacine birds fed a high-fat, seed-based diet and lack of exercise (Fig. 13-65). Budgerigars, amazon parrots, and birds with ovarian disorders appear predisposed. Most affected birds will be obese, although chronic cases may have lost weight and only have small areas of residual fat deposits. Clinical signs can include change in feather color (loss of green pigment in amazons leading to black feathers [see Fig. 13-61]), overgrown toenails and beak, and anorexia and apathy in birds with liver failure. Hepatic lipidosis

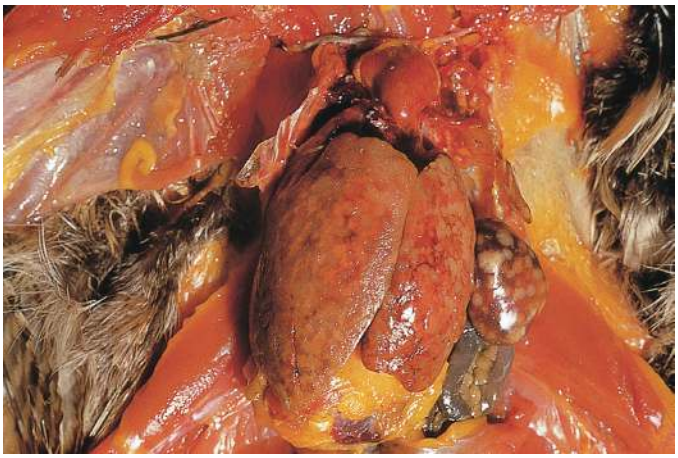


FIGURE 13-63 Hepatitis and splenitis in a pheasant caused by *Mycobacteria* spp.

does not normally cause biliverdinuria. Hepatomegaly and general obesity on radiographs (Fig. 13-66), hypercholesterolemia, high triglycerides, high enzymes such as aspartate aminotransferase (AST) and lactate dehydrogenase (LDH) (although not liver specific and often not even increased), and high bile acids are all findings commonly related to hepatic lipidosis. A definitive diagnosis is based on histopathology of an endoscopic or keyhole liver biopsy.

Iron Storage Disease

Iron storage disease is a common problem in many captive avian species, including mynahs and starlings (Sturnidae), birds of paradise (Paradisaeidae), bowerbirds (Ptilonorhynchidae), hornbills (Bucerotidae), toucans (Ramphastidae), lorikeets, and other psittacines (Psittacidae). This disease is defined as an accumulation of excessive amounts of iron in parenchymal cells with resultant cellular damage (Dorrestein and Mete, 2013). The etiology of iron storage disease in avian species remains unknown, but it is speculated that the

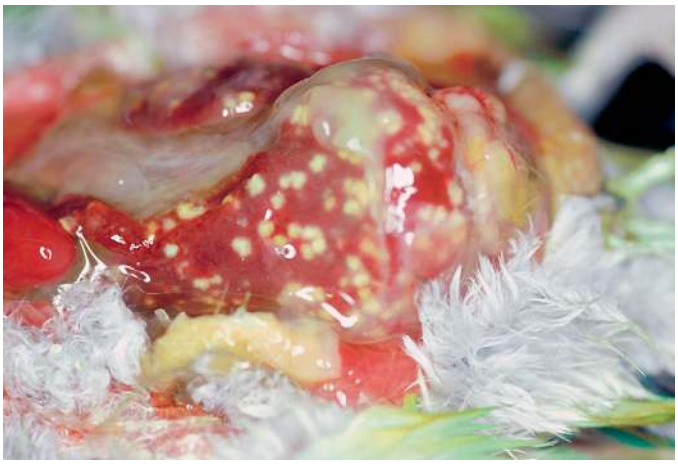


FIGURE 13-64 Granulomatous lesions in the liver of a canary caused by *Yersinia pseudotuberculosis*.

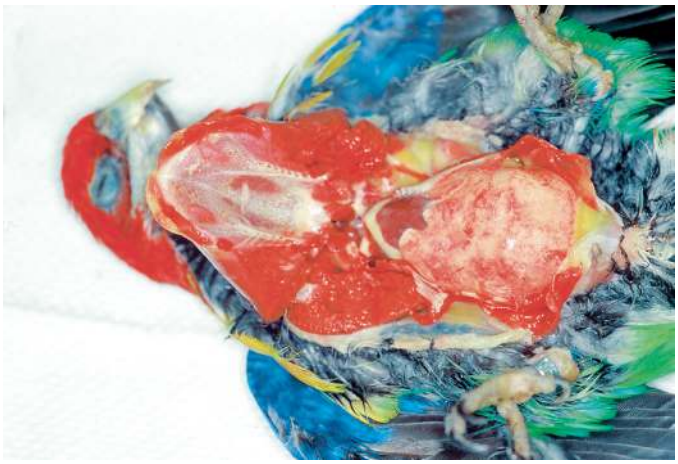


FIGURE 13-65 Hepatic lipidosis in an obese eastern rosella (*Platycercus eximius*).

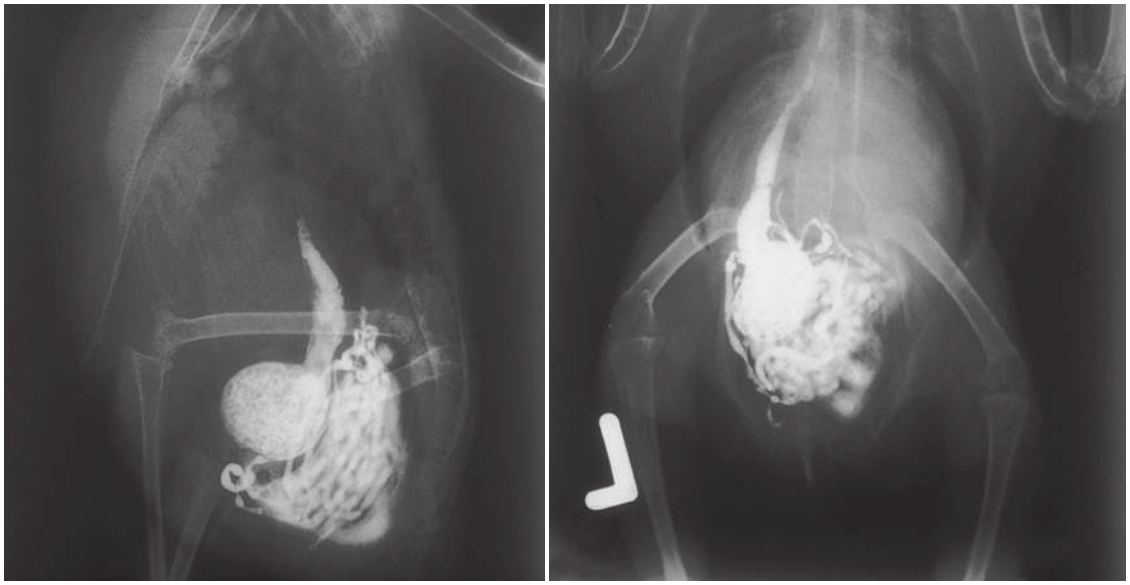


FIGURE 13-66 Severe hepatomegaly in an obese parrot suffering from hepatic lipidosis.

combination of an adaptation to a natural diet poor in iron and the supply of high-iron diets in captivity is the main cause. Early clinical signs are not very specific, often just general listlessness and loss of feather glossiness. In advanced cases, hepatomegaly and ascites causing dyspnea are common. A tentative diagnosis can be made based on the clinical signs, radiography and laboratory testing indicating liver disease, and the species of bird affected. Definitive diagnosis is based on a liver biopsy. Liver iron content can be calculated using quantitative image analysis, which computes the percentage of a Prussian blue-stained histologic slide that stains positive for iron, by chemical analysis, or by MRI (Sandmeier, 2012). Treatment includes dietary modification to minimalize iron intake by feeding a low-iron diet; reducing dietary vitamin C, such as citrus fruits, which increase the availability of iron in a diet; and by adding tannin-rich or phytate-rich ingredients, such as oak bark tea rather than normal drinking water, which reduce iron availability. Medical treatment is based on chelation therapy using the oral iron chelator deferiprone (Sandmeier, 2012). Treatment will only be successful if started during early stages of the disease, before severe liver disease develops, and if the patient is monitored long term.

Other Liver Diseases

Amyloidosis of the liver is generally a secondary condition following chronic infections such as bumblefoot, aspergillosis, or mycobacteriosis. Clinical signs are similar to those of all chronic liver diseases, including general sickness and hepatomegaly on radiographs and possibly increased enzymes and bile acids (Carnarius *et al.*, 2013). The diagnosis is made by endoscopic or keyhole liver biopsy. Treatment is based on general treatment of chronic liver disease, and it is important to address and correct the initial cause.

Toxic causes of liver disease include mycotoxins, plants, heavy metals, and pharmaceuticals (see Chapter 10). Chronic liver toxicity will lead to self-perpetuating liver fibrosis and lipid infiltration, often making a definitive diagnosis of the original toxic agent difficult (Hochleithner, 2006).

Neoplastic liver disease should be considered in all birds with hepatomegaly and chronic disease (Fig. 13-67). Diagnosis is based on a liver biopsy. In most cases treatment is not possible and the prognosis is bad. Common lesions include hepatocellular carcinoma, fibrosarcoma, biliary adenocarcinoma (often associated with cloacal papillomas and herpesvirus infections), malignant lymphoma, myeloblastic leukemia (especially in budgerigars), and Marek's and avian leukosis in poultry.

Blunt trauma to the coelomic cavity can cause a rupture of the liver leading to death from acute blood loss (Fig. 13-68).

Diagnostic Workup and Treatment

Diagnosis of liver disease is based on demonstrating hepatomegaly on radiographs and blood sampling, especially looking at bile acids, nonliver-specific enzymes such as aspartate aminotransferase (AST) and lactate dehydrogenase (LDH), albumin and globulin, cholesterol, and triglycerides, and hematologic changes indicative of inflammation or infection. Definitive diagnosis is based on liver biopsy, which can be performed as a keyhole procedure in the midline directly caudal to the sternum or by endoscopy.

Acute liver disease is treated by supportive care including infusion therapy, if possible etiologic treatment of infectious causes, and nutritional support by gavage feeding. Ascites can be relieved by puncturing and draining the coelomic cavity using a small gauge needle. Although chronic liver disease normally cannot be cured, the use of dietary modification and supportive therapy with products such as milk thistle, artichoke extracts, and ursodeoxycholic acid, or amino acids such as methionine can often help control the disease.

FIGURE 13-67 Myeloblastic leukemia causing severe hepatomegaly in a red-fronted parakeet (*Cyanoramphus novaezelandiae*).

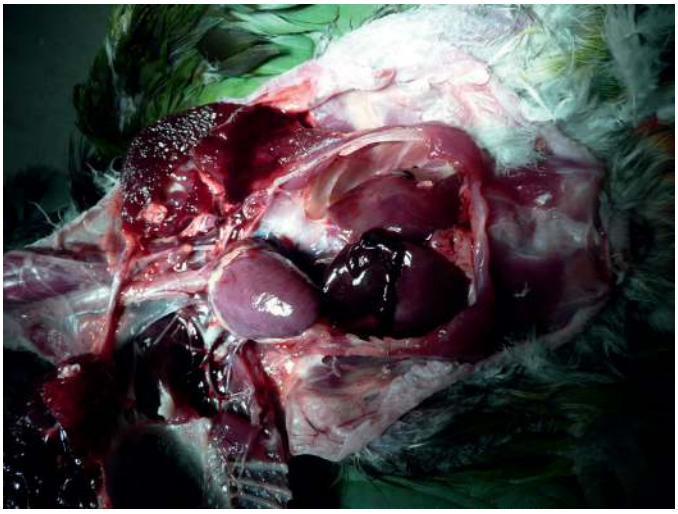


FIGURE 13-68 Blunt trauma can cause a tear of the liver and death from acute hemorrhage.

REFERENCES

Arnall L, Keymer IF: *Bird diseases: an introduction to the study of birds in health and disease*, Neptune City, NJ, 1975, TFH Publications.

Carnarius M, Hafez HM, Lierz M: New tools for the diagnosis of amyloidosis in falcons. *Proceedings of the 1st International Conference on Avian, Herpetological and Exotic Mammal Medicine,* Wiesbaden, Germany, p 367, 2013.

Davies RR: Avian liver disease: etiology and pathogenesis, *Semin Avian Exot Pet Med* 3:115–125, 2000.

Dorrestein GM, Mete A: Iron storage in birds. *Proceedings of the 1st International Conference on Avian, Herpetological and Exotic Mammal Medicine,* Wiesbaden, Germany, pp 289–304, 2013.

Forbes N: Review of fifteen per cloacal prolapses in raptors. *Proceedings of the 1st International Conference on Avian, Herpetological and Exotic Mammal Medicine,* Wiesbaden, Germany, pp 120–122, 2013.

Harcourt-Brown N: Beaks are modified skin: consequences for repair. *Proceedings of the 1st International Conference on Avian, Herpetological and Exotic Mammal Medicine,* Wiesbaden, Germany, pp 370–372, 2013.

Hatt J-M, Christen C, Sandmeier P: Clinical application of an external fixator in the repair of bone fractures in 28 birds, *Vet Rec* 160:188–194, 2007.
Hochleithner M, et al: Evaluating and treating the liver. In Harrison GJ, Lightfoot T, editors: *Clinical avian medicine*, Lake Worth, FL, 2006, Spix Publishing.
Raidal SR, Butler R: Chronic rhinosinusitis and rhamphothecal destruction in a Major Mitchell's cockatoo (*Cacatua leadbeateri*), *J Avian Med Surg* 15(2):121–125, 2001.
Samour JH: *Pseudomonas aeruginosa* stomatitis as a sequel to trichomoniasis in captive saker falcons (*Falco cherrug*), *J Avian Med Surg* 14:113–117, 2000.
Samour JH, Naldo JL: Diagnosis and therapeutic management of candidiasis in falcons in Saudi Arabia, *J Avian Med Surg* 16:129–132, 2002.
Samour JH, Naldo JL: Diagnosis and therapeutic management of trichomoniasis in falcons in Saudi Arabia, *J Avian Med Surg* 17:136–143, 2003.
Sandmeier P, et al: Use of deferiprone for the treatment of iron storage disease in 3 hornbills, *J Am Vet Med Assoc* 240:75–81, 2012.
Sandmeier P: Anatomy and physiology. In Chitty J, Monks D, editors: *Manual of avian practice*, Gloucester, UK, 2016, British Small Animal Veterinary Association. in publication.
Sandmeier P: Wellensittiche. In Fehr M, Sassenburg L, Zwart P, editors: *Krankheiten der Heimtiere*, Hannover, Germany, 2014, Schlütersche Verlag, in preparation.
Speer B: A clinical look at the avian pancreas in health and disease. *Proceedings of the Association of Avian Veterinarians,* St. Paul, MN, pp 57–64, 1998.
Taylor M, Murray MJ: The psittacine cloaca: a clinical review. *Proceedings of the Association of Avian Veterinarians,* Monterey, California, pp 265–269, 2002.
Tully TN, et al: Use of a dental composite to correct beak deviation in psittacines species. *Proceedings of the European Association of Avian Veterinarians,* Arles, France, pp 203–206, 2005.

FURTHER READING

Gelis S: Evaluating and treating the gastrointestinal system. In Harrison GJ, Lightfoot T, editors: *Clinical avian medicine*, Lake Worth, FL, 2006, Spix Publishing.
Lloyd C: Raptors: gastrointestinal disease. In Chitty J, Lierz M, editors: *BSAVA manual of raptors, pigeons and passerine birds*, Quedgeley, Gloucester, UK, 2008, British Small Animal Veterinary Association.
Lumeij JT: Gastroenterology. In Ritchie B, Harrison GF, Harrison L, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.
Pees M: Pigeons: gastrointestinal disease. In Chitty J, Lierz M, editors: *BSAVA manual of raptors, pigeons and passerine birds*, Quedgeley, Gloucester, UK, 2008, British Small Animal Veterinary Association.
Schmidt RE: Pathology of gastrointestinal diseases of psittacines birds, *Semin Avian Exot Pet Med* 2:75–82, 1999.

DISORDERS OF THE RESPIRATORY SYSTEM

Bárbara Arca-Ruibal

Respiratory problems in avian patients are common complaints from owners seeking veterinary advice. This section serves as a general overview for the multiple conditions that can affect the respiratory system of avian patients. For more detailed information the reader is advised to consult the related sections and references in this book.

RESPIRATORY SYSTEM ANATOMY AND HEALTH IMPLICATIONS

It is important for veterinarians seeing avian patients to be familiar with the anatomy and physiology of the avian respiratory system. This knowledge will help evaluate the site and nature of the respiratory distress and to formulate a diagnostic and treatment plan. For a comprehensive review of the avian respiratory system anatomy and physiology the reader is referred to more detailed texts (Tully and Harrison, 1994; Heard, 1997; Powell, 2000; O'Malley, 2005).

The components of the upper respiratory tract include the external nares, operculum, nasal concha, infraorbital sinus, choanal slit, and glottis. The trachea, syrinx, bronchi, lungs, parabronchi, and air sacs constitute the lower respiratory tract. Respiratory diseases in any area of the upper or lower respiratory tract can affect the adjacent areas of the respiratory system (Clippinger, 1997) and upper respiratory diseases can progress to lower respiratory diseases. Some of the anatomic and physiologic adaptations of the respiratory system in birds that can have clinical significance are discussed in the following list:

- The extensive and complex structure of the infraorbital sinus often makes upper respiratory infections difficult to treat and prevent (Tully, 1995). A common clinical manifestation of sinusitis is periorbital swelling from a collection of pus and debris in the infraorbital sinus. This accumulation is caused by the dorsal location of the communication of the sinus with the caudal and the middle nasal concha, because mucopurulent material from the sinus can only drain into the nasal cavity from these openings (Tully and Harrison, 1994; Orosz and Lichtenberger, 2011). The left and the right sinus communicate in some avian species, such as psittacines, insectivorous passerines, and Anseriformes, whereas they may not communicate in other species, such as noninsectivorous passerines (Heard, 1997; Campbell and Ellis, 2007). This anatomic difference among species should be considered when collecting representative samples by sinus aspiration.
- The avian trachea is larger and wider than the trachea in mammals, it lacks a tracheal ligament, and is composed of complete tracheal rings (Heard, 1997). These anatomic adaptations make birds more prone to tracheal obstructions from foreign bodies and more vulnerable to tracheal mucosal damage and subsequent tracheal stenosis (Doneley and Raidal, 2010). The tracheal lumen is narrowed at the level of the syrinx making this area a common site for lodging foreign bodies and tracheal granulomas (Orosz and Lichtenberger, 2011).
- Birds have higher oxygen demands than mammals and their lungs are 10 times more efficient at capturing oxygen (O'Malley, 2005). This is possible because, among other modifications, the blood-gas barrier in birds is much thinner than in mammals and the air capillaries of birds (the equivalent of the mammalian alveoli) are thinner (3 to 10 μm) and present in a larger number than their mammalian counterparts. For these reasons fluid can accumulate in an air capillary faster than in an alveolus with the clinical consequence that birds can deteriorate faster than mammals (Orosz and Lichtenberger, 2011). The rapid efficiency of gas exchange in birds also makes them more susceptible to inhaled toxic agents and infections (O'Malley, 2005).
- Air sac internal conditions (large, well oxygenated, warm, and poorly vascularized) replicate the ideal conditions for incubation of pathogens (Tully, 1995; Bailey, 2008). The caudal air sacs are more prone to disease because they receive ambient air directly without being filtered by the epithelial cells of lungs (Bailey, 2008) and possibly by the air layering that occurs in them (Tully and Harrison, 1994). The air sac connection with pneumatic bones allows introduction of infection into the lungs and air sacs via compound fractures of those bones, especially the humerus (O'Malley, 2005).

CLINICAL PRESENTATION AND INVESTIGATION OF RESPIRATORY DISEASES

A full clinical history should be taken and a visual inspection should be performed before starting any diagnostic tests to assess the condition of the patient. Clinical examination should be performed carefully as severely compromised patients can decompensate rapidly and may require stabilization before any further handling or tests are performed (Fig. 13-69).

Box 13-6 summarizes the most common clinical signs associated with upper and lower respiratory diseases in birds. Diagnostic techniques used to investigate respiratory diseases in birds are presented in Box 13-7. Techniques used in the collection of samples from the avian respiratory system are described in Box 13-8. It is recommended that all birds presented with respiratory signs should be tested for chlamydiosis.

INFECTIOUS CAUSATIVE AGENTS

A number of infectious causative agents can affect the avian upper and lower respiratory tract. Table 13-4 summarizes the most common infectious causes reported in birds.

Text continued on p. 391

FIGURE 13-69 Budgerigar (*Melopsittacus undulatus*) in an oxygen chamber. The bird was presented dyspneic and was placed in the oxygen chamber before starting the diagnostic workout. The bird was diagnosed with aspiration pneumonia. (Courtesy Dr. Andrés Montesinos.)

BOX 13-6 Common Clinical Signs Associated with Upper and Lower Respiratory Diseases in Birds

Disorders of the Upper Respiratory System

Rhinitis and Sinusitis

Open-mouth breathing
Plugged nares, discharge, sneezing
Choanal discharge and inflammation
Periorbital swelling, epiphora
Dyspnea, exercise intolerance
Head shaking, beak rubbing, and yawning
Conjunctivitis, sunken eyes
Neck stretching, inflamed cere

Disorders of the Lower Respiratory System

Tracheitis

Vocalization changes
Dilated or inflamed glottis
Open-mouth breathing
Coughing
Rasping or rattling inspiration and/or expiration
Tachypnea, dyspnea

Pneumonia and Airsacculitis

Change of voice, dyspnea, tail bobbing
Inspiratory/expiratory difficulty, coughing, stridor
Lethargy
Exercise intolerance
Tachypnea, dyspnea
Open-mouth breathing
Inappetence/vomiting
Weight loss

BOX 13-7 Diagnostic Techniques Used in the Investigation of Respiratory Tract Diseases in Birds

Cytology: Samples obtained directly from nares, choana, sinus, glottis, and trachea, and by nasal flush, sinus aspiration, tracheal washing, air sac lavage, and air sac biopsy
Microbiology: Samples obtained as for cytology and any solid lesions; Isolated agents may be primary or secondary etiology
Histopathology: Biopsy of lesions
Endoscopy: Examination of oropharynx, choanal slit, trachea, syrinx, air sacs, ostium, and lung and sample collection (Fig. 13-70)
Radiography: Rhinography and sinography if anatomic abnormality, mass, or foreign body is suspected in upper respiratory tract; tracheal abnormalities and lesions of the syrinx, lungs, or air sacs; another condition such as space-occupying lesions
CT scan and MRI: Examine anatomy and patency of infraorbital sinus and localize lesions in sinuses (Fig. 13-71, *A* and *B*)
Hematology and biochemistry: May indicate inflammatory or infectious responses and chronicity
Immunology: Avian influenza, PMV1, *Chlamydia*
Serology: *Chlamydia*, *Aspergillus*, PMV1 and Avian influenza
PCR: *Mycoplasma*, *Chlamydia*, PMV1, Avian influenza, other virus
Transillumination: Trachea, sinus, and rhinal cavity (Fig. 13-72)
Parasitology: Feces and oropharynx samples to check for parasites known to affect respiratory tract.
Exercise/Tolerance test: Respiratory rate in a healthy patient after exercise test should be normal after 2 minutes.
Auscultation: Sounds may not correlate with severity of the disease and can be normal with severe pathology; inspiratory sounds in URT disease and expiratory sounds in LRT disease; moist or harsh respiratory sounds are suggestive of bronchopulmonary disease and clicking or crackles indicate thickened air sacs.

ELISA, Enzyme-linked immunosorbent assay; LRT, lower respiratory tract; PCR, polymerase chain reaction; PMV-1, Paramyxovirus-type 1; URT, upper respiratory tract.

BOX 13-8 Sample Collection Techniques Used in the Investigation of Diseases of the Respiratory System in Birds

Upper Respiratory System

Choanal swabs: Samples should be taken from the rostral portion as the caudal portion is likely to be contaminated with oropharynx flora.

Nasal flush: Can be used to collect samples and therapeutically to clean debris and rhinoliths. A Luer lock syringe is pressed against the nares forming a seal and the fluid is infused. Drainage of the fluid should flow free from the choana into the mouth. Recommended volumes are 10 to 20 mL/kg.

Infraorbital sinus flush/aspiration: Useful for sample collection, flushing of sinuses, and medication. Advantage of minimal sample contamination. There are two approaches: (1) A needle is passed into the sinus at a perpendicular angle though the skin at a point midway between the imaginary line traced between the naris and the medial canthus of the eye and (2) enter the sinus from a rostral direction by inserting the needle just caudal to the commissure of the mouth; the needle is directed ventral to the zygomatic arch ending in the sinus under the eye (Campbell and Ellis, 2007).

Lower Respiratory System

Tracheal wash: With the patient anesthetized a sterile catheter is passed through the glottis into the trachea to the point just cranial to the syrinx and 1 to 2 mL/kg of sterile saline is introduced and immediately aspirated.

Air sac lavage: Three to 5 mL/kg of sterile saline can be injected through the body wall into the last intercostal space or through the biopsy channel of the endoscope and recovered by aspiration with a urinary catheter or similar device.

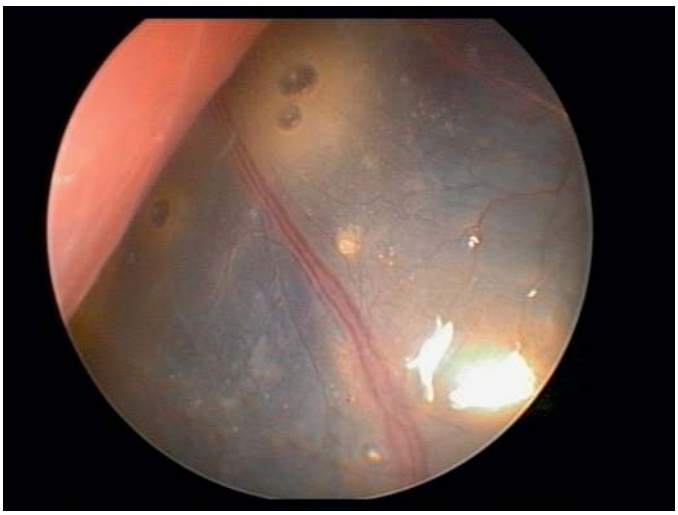


FIGURE 13-70 Abnormal presence of fluid in the caudal thoracic air sac. Samples can be taken aided by endoscopy for cytology and culture to identify the causative agent.

FIGURE 13-72 Transillumination of the trachea of a zebra finch (*Taeniopygia guttata*) showing respiratory mites infestation. (Courtesy Dr. Andrés Montesinos.)

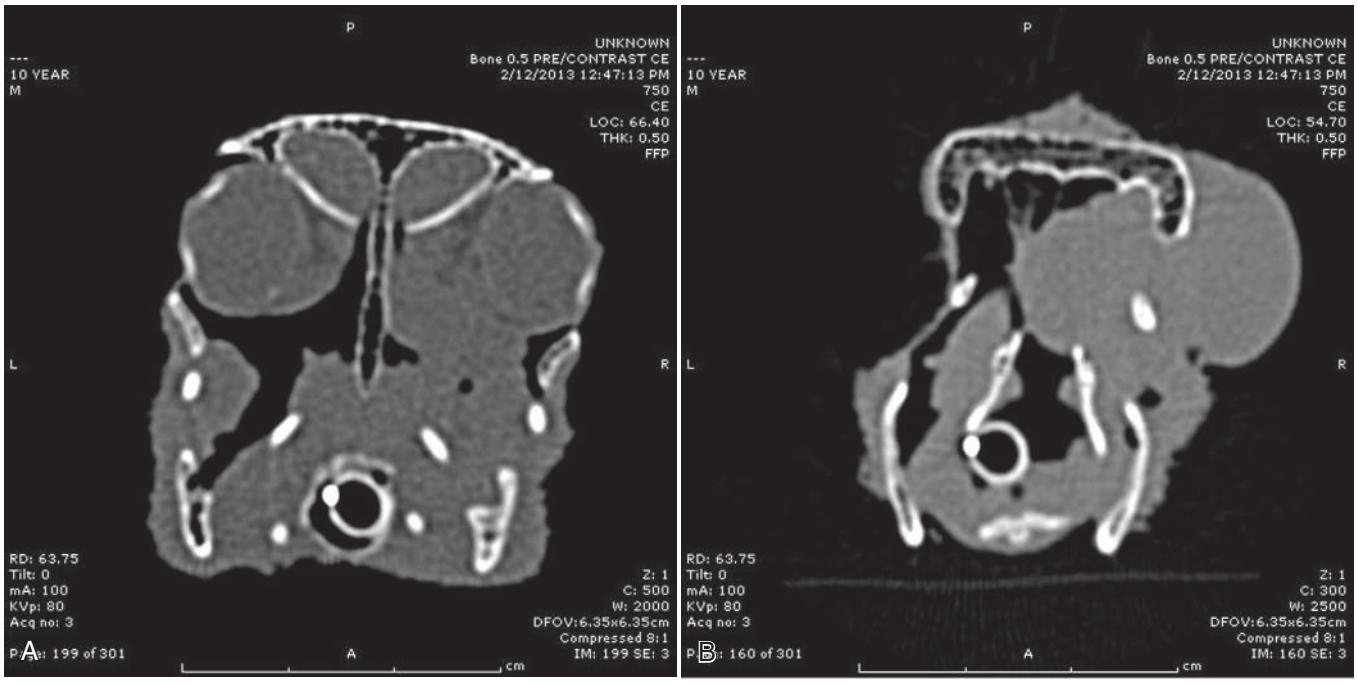


FIGURE 13-71 **(A)** and **(B),** Computed tomography scan images of a long-billed corella (*Cacatua tenuirostris*) showing right-sided sinusitis caused by *Cryptococcus* sp. (Courtesy Dr. Robert Doneley.)

TABLE 13-4 Infectious Causative Agents of Respiratory Diseases in Birds

Causative Agent			RSI	Clinical Signs/Presentation	Diagnosis	Treatment
Bacterial	*Mycoplasma* sp.	Especially *M. gallisepticum*; described in poultry, game birds, canaries, finches, psittacines, birds of prey, and greater flamingos	UR, LR	Clear nasal discharge, sneezing, conjunctivitis, rhinitis, infraorbital sinus swelling Lethargy, weight loss, decreased egg production, death	Culture difficult Serology, ELISA, PCR	Tylosin, tetracycline, or enrofloxacin
	Chlamydia psittaci	Latent infections activated during stress, highly contagious Zoonotic	UR, LR	Clear nasal or mucopurulent nasal or ocular discharge, sneezing, conjunctivitis, sinus distention Chronic unfitness, acute anorexia, diarrhea, dyspnea, lime green feces, sudden death	Culture difficult Cytology, serology, ELISA, PCR	Doxycycline is the drug of choice Immunity short-lived, birds are susceptible to reinfection after treatment
	Pseudomonas aeruginosa	Usually secondary invader Toxin producer	UR, LR	Anorexia, weight loss, unilateral or bilateral sinusitis, coughing, mucopurulent nasal and choanal discharge	Isolation and culture	Based on culture and sensitivity testing Resistant to many commonly used antibiotics
	Other bacteria	Gram-positive and gram-negative *E. coli*, *Pasteurella multocida*, *Klebsiella pneumoniae*, spirochetes, *Haemophilus*, etc.	UR, LR	Acute or chronic clear or mucopurulent nasal and ocular discharge, swelling in the periorbital area, swelling choanal slit region, tissue distortion of nares and beak, enophthalmia, excess mucus in the choana and trachea, "lockjaw syndrome" in cockatiels Malaise, weight loss, exercise intolerance	Cytology and culture	Based on culture and sensitivity testing
Fungal	*Aspergillus* sp.	Most common respiratory disease in birds, affects all avian species Predisposed by stress, poor management, malnutrition, and toxins	UR, LR	Rhinitis: no discharge or severe, chronic, mucopurulent discharge, severe granulomatous sinusitis/rhinitis Acute: dyspnea, cyanosis, lethargy, anorexia, polyuria, polydipsia, sudden death Chronic: decreased appetite and activity, exercise intolerance, weight loss, tachypnea, and dyspnea in advanced cases Tracheitis: obstructive airway disease by granuloma formation, change of vocalization, severe dyspnea, open-mouth breathing, gurgling respirations, cough, sudden death	Endoscopy (see Fig. 13-73), radiology, cytology, culture, serology, hematology, biochemistry	Antifungal therapy and supportive care
	Candida sp.	*C. albicans* most commonly isolated opportunistic pathogen, originates in oropharynx and spreads from the choana	UR, LR	Dyspnea, white plaques in the oral cavity and choanal Usually in conjunction with primary bacterial sinusitis	Cytology, endoscopy	Antifungal therapy
	Cryptococcus neoformans	Widespread in pigeons, reported in other birds Not common, predilection for upper respiratory tract Zoonotic	UR	Depression, dyspnea unresponsive to treatment Clear nasal discharge, swelling of sinus Flushing of gelatin masses from sinus	Cytology and histopathology, culture, necropsy	Antifungal therapy
	Histoplasma capsulatum	Reported in poultry and zoo birds Found in pigeon and poultry fecal material and enclosed aviaries and dirt Zoonotic	LR	Similar signs as cryptococcosis, with initial pneumonia progressing to disseminated necrotic granulomas	Culture and histopathology	

TABLE 13-4 Infectious Causative Agents of Respiratory Diseases in Birds—cont'd

Causative Agent			RSI	Clinical Signs/Presentation	Diagnosis	Treatment
Viral	Avipoxvirus	Family Poxviridae, DNA virus Affects most species of birds Respiratory signs if cutaneous or oral lesions interfere with the airflow through the nares or respiratory tract Septicemic form in canaries	UR, LR	Respiratory distress, dyspnea Mucoid rhinitis and tracheitis, bronchopneumonia	Bollinger bodies in biopsy and histopathology of lesions Electron microscopy, necropsy	Prevention of vector exposure, separation of affected birds, vaccination
	Paramyxovirus	Family Paramyxoviridae Newcastle disease virus RNA virus Affects most avian species, great variety in virulence	UR, LR	Respiratory signs from acute to mild or subclinical infection Usually associated with neurologic and gastrointestinal symptoms Acute upper respiratory infections with mucoid nasal discharge and necrotic foci at the sides of the choanal cleft	Serology, virus isolation, PCR	Vaccines available for some species Preventive measures and control
	Avian influenza	Family Orthomyxoviridae RNA virus Only Influenza A causes natural infections in birds	UR, LR	Mild respiratory signs to 100% mortality depending on the virulence of strain Purulent rhinitis, bleeding disorders, dyspnea, lethargy, discharge from nares and eyes, severe tracheitis, pneumonia, edema of the head, cyanosis of comb and wattles, anorexia, decreased egg production, diarrhea	ELISA, virus isolation, PCR	No treatment Prevention and control Vaccines available for some species
	Reovirus	Family Reoviridae RNA virus Reported in Galliformes, Anseriformes, psittacines, pigeons, and raptors	UR	Respiratory (coughing, nasal discharge, increased lung sounds), enteric signs and arthritis/tenosynovitis	ELISA, PCR, electron microscopy, virus isolation	Prevention and control Vaccines available for some species
	Pneumovirus	Family Paramyxoviridae, subfamily Pneumovirinae RNA virus "Swollen head syndrome" in chickens, severe rhinotracheitis in turkeys, mild infections in pheasants, guinea fowl, and ostriches	UR	Sneezing, swelling of infraorbital sinuses, conjunctivitis	ELISA, PCR	Prevention and control
	Herpesvirus	Family Herpesviridae DNA virus Infectious laryngotracheitis, Amazon tracheitis virus Affects adult and growing birds older than 8 weeks	LR	Acute, severe respiratory distress Bloody mucus from glottis, swelling infraorbital sinus Death by asphyxiation as result of epithelial reaction in trachea	ELISA, PCR, immunofluorescence, virus neutralization, immunodiffusion	Prevention and control measures
	Adenovirus	Family Adenoviridae DNA virus "Fowl adenovirus" lower respiratory infections in birds	LR	Tracheal rales, coughing, respiratory distress Other systems also affected	PCR, necropsy, virus isolation	
	Polyomavirus	Family Polyomaviridae DNA virus	LR	Chronic disease, emaciated, poor growth	Histopathology, PCR, serology	Prevention and control measures

Continued

TABLE 13-4 Infectious Causative Agents of Respiratory Diseases in Birds—cont'd

Causative Agent			RSI	Clinical Signs/Presentation	Diagnosis	Treatment
Parasitic	Mites	*Sternostoma tracheacolum* in canaries and Gouldian finches and other bird species (Fig. 13-74)	LR	Typical clicking sound during inspiration According to the severity of the infestation from inapparent to severe respiratory signs resulting in death and asphyxiation	Transillumination of the trachea, cytology, histopathology	Ivermectin orally or topically Recurrence is common
	Trichomonas sp.	Protozoal parasite usually affecting GI system Affects respiratory sinuses and trachea in raptors and Columbidae and produces necrotic masses in trachea in psittacines	UR	Sensory depression, ruffled feathers, anorexia, weight loss, regurgitation, vomiting Whitish caseous and necrotic masses in mucosa	Wet mount preparations	Dimetridazole, metronidazole, carnidazole, ronidazole
	Nematodes	*Syngamus trachea* (especially Galliformes and Anseriformes), *Serratospiculum* (common in diurnal birds of prey; Fig. 13-75), filaroides	LR	Some species have asymptomatic infections, others can cause severe dyspnea, frothing, and bloody mucus discharge from trachea and glottis	Parasitology, endoscopy	Benzimidazoles, ivermectin, physical removal
	Cryptosporidium sp.	Reported in more than 30 avian species Zoonotic	LR	Sinusitis, coughing, sneezing, dyspnea Severe and fatal tracheitis and pneumonia	Cytology, acid fast staining, direct immunofluorescence PCR, ELISA	
	Toxoplasma gondii	Reported in Columbiformes, Passeriformes, Psittaciformes, Strigiformes, Galliformes, Anseriformes, Sphenisciformes, and Pelecaniformes	LR	Dyspnea, anorexia, emaciation, conjunctivitis, neurologic signs, crusty exudates around eye, blindness, death	Serology, histopathology, PCR	
	Sarcocystis sp	Reported in more than 60 avian species	LR	Usually no antemortem clinical signs, severe respiratory distress	Histopathology	Trimethoprim and sulfadiazine

ELISA, Enzyme-linked immunosorbent assay; GI, gastrointestinal; LR, lower respiratory; PCR, polymerase chain reaction; RSI, respiratory system involved; UR, upper respiratory.

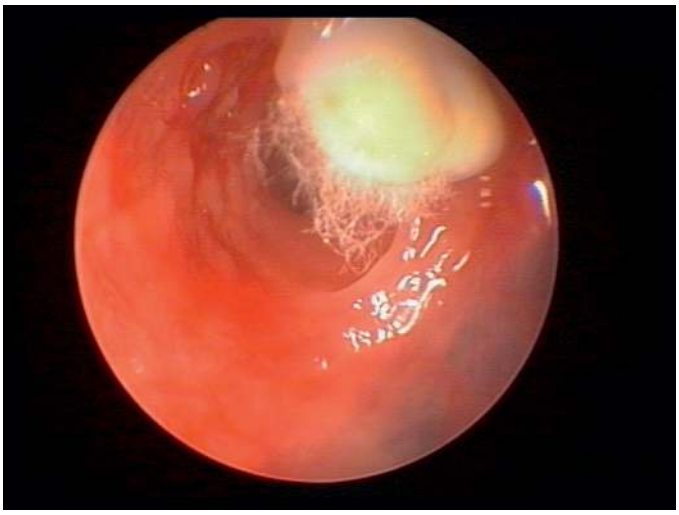


FIGURE 13-73 Endoscopy of the caudal thoracic air sac of a falcon. A fungal granuloma in the ostium can be observed. *Aspergillus niger* was cultured from a biopsy of the lesion.

FIGURE 13-74 Respiratory mites (*Sternostoma tracheolum*) in the airways of a Gouldian finch (*Erythrura gouldiae*) found during post-mortem examination. (Courtesy Dr. Andrés Montesinos.)

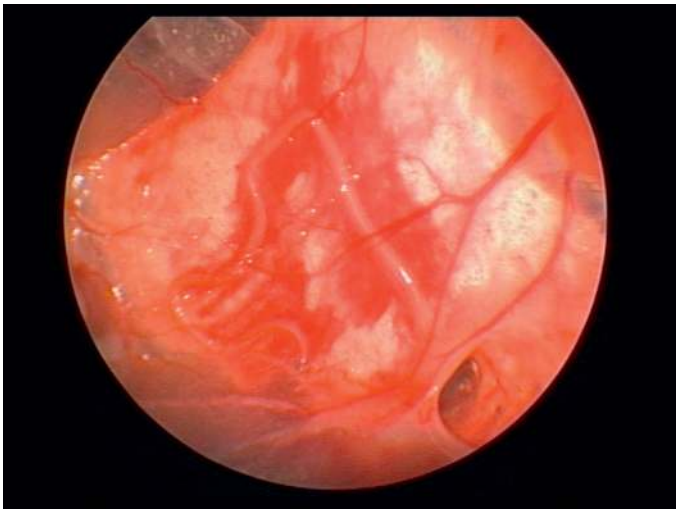


FIGURE 13-75 Endoscopy of the caudal thoracic air sac of a falcon. Lung hemorrhage caused by *Serratospiculum* sp. nematode can be observed. Even though some authors do not consider *Serratospiculum* pathogenic, severe infestations can cause lung damage and predispose birds to secondary bacterial and fungal infections.

FIGURE 13-76 Rhinolith in a 25-year-old African grey parrot (*Psittacus erithacus*). The diet of this bird was homemade food and peanuts. (Courtesy Dr. Andrés Montesinos.)

NONINFECTIOUS CAUSATIVE AGENTS

Vitamin A Deficiency

Vitamin A is necessary to maintain the normal epithelial health. Deficiency causes squamous metaplasia of the mucous membranes altering the function of the respiratory system, predisposing it to fungal and bacterial infections. In avian species, changes in the epithelium occur when vitamin A levels in the liver are lower than 50 IU/g (MacWhirter, 1994). Hypovitaminosis is caused by deficient diets or intestinal lesions that interfere with the conversion of carotenoids in vitamin A (Schmidt *et al.*, 2003). White caseous material (keratinized epithelium) accumulates in the sinus and predisposes the bird to secondary bacterial and fungal sinusitis and granuloma formation. Large accumulations can result in distortion of the nares and sinus. Hypovitaminosis A also may lead to a partial or complete tracheal obstruction as a result of the thickening and sloughing of part of the lining of the syrinx (MacWhirter, 1994). Initial treatment involves parenteral supplementation followed by diet modification and resolution of any primary causes. Care must be taken when given vitamin A supplements to birds because over supplementation can potentially be toxic.

Anatomic Disorders

Choanal atresia has been described in African grey parrots (*Psittacus erithacus*) and an umbrella cockatoo (*Cacatua alba*; Tully and Harrison, 1994). The presence of a permanent membrane or bony plate between the palate and choana or the absence of the choanal slit will prevent the normal drainage of nasal secretions into the oral cavity. In young birds clinical signs include chronic ocular or nasal discharge with possible distended infraorbital sinuses with clear secretions (Schmidt *et al.*, 2003). Older birds can present with a history of chronic mucopurulent nasal discharge unresponsive to antibiotics. Diagnosis can be confirmed by endoscopy and nasal sinus contrast studies. A corrective surgery technique has been described by Harris (1999).

Anatomic deformations of the upper respiratory tract resulting from primary diseases such as trichomoniasis or avipox can predispose birds to secondary recurrent respiratory problems and infections.

Foreign Bodies

Nasal plugs and rhinoliths can obstruct the nares and act as a nidus for infection (Fig. 13-76). They are produced by the accumulation of dust, debris, and desiccated nasal discharge. Rhinoliths can alter the normal shape of the nares and are associated with poor husbandry. Treatment involves removal by first moistening them and then cleaning using a small bone curette. Care must be taken not to mistake the normal operculum for foreign material. Nasal flushing after cleaning will help remove any remaining material (Morrisey, 1997; Bowles *et al.*, 2005; Bailey, 2008). Food particles, cage substrates, or other small items can be inhaled and lodged in the nares and choana causing rhinitis and sinusitis. Initially the nasal discharge will be clear, but the foreign body will act as a nidus for secondary infections and the discharge will become mucopurulent. Diagnosis can be confirmed by endoscopy and contrast radiography. Treatment involves the removal of the particles by nasal flushing and endoscopy.

Accidental aspiration of seeds or other materials can cause a partial or total tracheal obstruction with subsequent acute onset of respiratory signs. Commonly described foreign bodies are millet seeds in cockatiels (Schmidt *et al.*, 2003; Doneley, 2011), but more unusual foreign bodies have also been reported (Cannon, 2006). Inhaled foreign bodies may not completely occlude the tracheal lumen but can trigger an inflammatory response leading to tracheal stenosis and occlusion (Doneley and Raidal, 2010). Diagnosis is made by tracheal endoscopy and radiology. Treatment includes placement of an air sac cannula to stabilize the patient and removal of the foreign body. If tracheal stricture is present resection of the affected area and anastomosis is necessary.

Bronchial foreign bodies can cause local inflammatory response and lead to bronchopneumonia. Silicosis and pneumoconiosis have been described in birds (Clippinger, 1997).

Iatrogenic Tracheitis and Stenosis

Postintubation tracheal stenosis has been described in birds (de Matos *et al.*, 2006; Evans *et al.*, 2009, Jankowski *et al.*, 2010) and it is believed

to be more common than reported in the literature with a mortality up to 70% (Sykes *et al.*, 2013). It has been documented in Anseriformes, Accipitriformes, Galliformes Psittaciformes, Ciconiiformes, Columbiformes, Gruiformes, and Passeriformes. It is diagnosed by tracheoscopy and radiology. Initial treatment is done to stabilize the patient with oxygen supplementation and the placement of an air sac cannula, followed by surgical resection of the affected part and anastomosis. Successful treatment has been reported even after large segments of trachea were removed (Jankowski *et al.*, 2010).

Inhaled Toxic Agents and Irritants

Inhaled toxins not only can cause irritation and damage of the respiratory system but also can compromise the immune system (Lightfoot and Yeager, 2008). Nicotine from tobacco smoke can affect birds, especially those chronically exposed in smoking households. Birds exposed to second-hand smoke have been found to have levels of a nicotine metabolite (cotinine) similar to those reported in humans with clear evidence of clinical alteration and resultant disease from environmental tobacco smoke (Cray *et al.*, 2005). Clinical signs include coughing, sneezing, rhinitis, sinusitis, and conjunctivitis from continuous irritation of the respiratory tract; dermatitis; and feather-destructive behavior (Dumonceaux and Harrison, 1994). Secondary respiratory infections are common. Severe clinical signs will require supportive care, but long-term management will involve the removal of the source of smoke from the environment. Clinical signs can take from several weeks to months to resolve after placement in a smoke-free environment (Tully and Harrison, 1994).

Suspected sodium hypochlorite inhalation toxicosis has been reported in birds (Wilson *et al.*, 2001). The gases produced by undiluted sodium hypochlorite (5% chlorine bleach) caused pulmonary and severe tracheal lesions. Clinical signs, noticed 6 days after exposure, include respiratory distress, depression, poor appetite, and death. Death was considered to be the result of hypoxia secondary to blockage of the trachea or pulmonary congestion and, in some cases, sepsis secondary to invasion of bacteria through the altered tracheal mucosa. Respiratory distress and necrotizing tracheitis following involuntary inhalation of ivermectin diluted in propylene glycol has also been described (Schmidt *et al.*, 2003).

Polytetrafluoroethylene (PTEF), or Teflon toxicity, is one of the most common causes of airborne toxicity in pet birds (Lightfoot and Yeager, 2008). Teflon can be found in commonly used nonstick-coated household items (e.g., cookware and irons) and heat lamps. PTEF is safe to use if the recommended temperature limit is not reached, but PTEF toxic gasses and particulate materials are released when PTFE is overheated at temperatures higher than 280° C (530° F) by a process called pyrolysis (Wells *et al.*, 1982). The target organ of PTEF toxicity is the lung, and it causes severe pulmonary edema, necrosis, and hemorrhage. Even though sudden death is the most common presentation, other clinical signs such as somnolence, dyspnea, wheezing, incoordination, weakness, respiratory distress. and terminal convulsions can be seen depending on the degree of exposure (Dumonceaux and Harrison, 1994). Minimally exposed birds may respond to immediate transfer to fresh air and supportive care (oxygen therapy, nebulization, bronchodilators, analgesics, antiinflammatory drugs, diuretics, and antimicrobials to prevent secondary infections). However, prognosis of birds exposed to PTEF is generally poor and death usually occurs before treatment can be initiated (Dumonceaux and Harrison, 1994; Lightfoot and Yeager, 2008).

Smoke inhalation toxicity is caused by nonirritant (carbon monoxide, carbon dioxide, and hydrogen cyanide) and irritant gases (aldehydes, hydrogen chloride, and sulfur dioxide) and particulated matter released by combustion (LaBonde, 1995). Birds exposed to fumes from fires, malfunctioning furnaces and engine exhausts, burning food or cooking oil, self-cleaning ovens, or other sources of smoke can develop signs of toxicity (Stoltz *et al.*, 1992; Dumonceaux and Harrison, 1994; Verstappen and Dorrestein, 2005; Bailey, 2008; Redig and Arent, 2008). Smoke inhalation can cause similar signs of PTFE toxicity. Immediate clinical signs may not be apparent and can develop several hours or days after exposure (LaBonde, 1995; Verstappen and Dorrestein, 2005). Treatment includes transfer to a well-ventilated area and supportive care (oxygen therapy, nebulization, bronchodilators, analgesics, antiinflammatory drugs, diuretics, and antimicrobials to prevent secondary infections). It is not recommended to use corticosteroids in the treatment of gaseous toxicities as no benefits of their use have been proven and they can predispose to secondary respiratory infections (LaBonde, 1995; Verstappen and Dorrestein, 2005).

Miscellaneous airborne toxins such as air fresheners, hair products, nail polish, scented candles and plugs, aerosols, gasoline fumes, paints, mothballs, fumigants, cleaning products (Richardson, 2005), and plastic overheated in a microwave (Lightfoot and Yeager, 2008) can potentially cause irritation and damage of respiratory tract. Birds should be removed from the toxic environment and placed in a well-ventilated or oxygenated environment as soon as possible and given supportive care as in other airborne toxicities.

Aspiration Pneumonia

Aspiration of food and fluids in birds can occur iatrogenically with improper feeding or medication techniques or with the loss of normal protective mechanisms in very weak birds (Clippinger, 1997; Bailey, 2008). It most commonly happens in hand-reared psittacine birds and sick birds that are being tube fed. Regurgitation of gastrointestinal contents during recovery from anesthesia will also cause aspiration pneumonia (Tully, 1995). Presentation can be acute with severe respiratory distress or a chronic recurrent progressive pneumonia. Clinical signs can be caused by physical obstruction of the airways, physical damage to the respiratory epithelium, the inflammatory response elicited by the material and secondary bacterial and fungal infections (Clippinger, 1997). Presumptive diagnosis is usually made by the history of possible aspiration. Early radiographies can be normal but evidence of localized pulmonary lesions will be evident with time (Clippinger, 1997). Persistent leukocytosis can also be seen in chronic pneumonia cases. Affected birds should be hospitalized and given oxygen therapy and supportive care in the form of nebulization, fluid therapy, broad-spectrum antibiotics, and antifungals (Graham, 2004; Bailey, 2008). Prognosis is variable (Clippinger, 1997). It is advised that fluids and nutritional support to very weak birds be given by the intravenous or intraosseous route to prevent aspiration (Bailey, 2008).

Neoplasia

Neoplastic processes can affect the upper and lower respiratory tract in birds. Nasal/sinus carcinoma or adenocarcinoma in pet birds can become quite large, distorting the skull and affecting the brain in severe cases (Schmidt *et al.*, 2003). Clinical signs include acute onset of nasal discharge and dyspnea and chronic sinusitis nonresponsive to antibiotics (Noonan *et al.*, 2014). No treatment has been described.

Squamous cell carcinoma (SCC) can affect the nasal sinuses and oral cavity (Schmidt *et al.*, 2003; Reavill, 2004; Diaz-Figueroa *et al.*, 2006). SCCs are malignant, locally aggressive tumors usually associated with hemorrhage and necrosis of the surrounding tissues that tend to

reoccur. Dyspnea, recurrent infections, dysphagia, chronic stomatitis, and nasal discharge are common clinical signs. Metastasis is rare.

Malignant lymphoma can be presented as multicentric or solitary. Common presentation in the upper respiratory tract in Amazon parrots is a mass in the choana and in African grey parrots a periorbital or retrobulbar mass (Schmidt *et al.*, 2003). No evidence of retroviral association has been proven in pet birds.

Malignant melanoma in the nasal sinuses has been described in an African grey parrot as part of an infiltrative neoplasia involving the oral cavity and beak (André *et al.*, 1993).

Tracheal tumors are considered rare in birds (Schmidt *et al.*, 2003). Tracheal osteochondroma has been reported in a Fischer's lovebird (*Agapornis fischeri*) with a presentation of dyspnea and stridor from tracheal stenosis produced by the tumor (Weissengruber and Loupal, 1999). Diagnosis was made by endoscopy and histopathology.

Primary pulmonary tumors are rare in birds; however, metastatic tumors of various malignancies are commonly reported (Garner *et al.*, 2009). Clinical signs associated with tumors in the lower respiratory system are dyspnea, wheezing, and tail bobbing (Tully, 1995). Diagnosis is helped by endoscopy and biopsy and histology of the lung and air sacs for identification.

Primary pulmonary and air sac carcinomas are considered rare in pet birds (Schmidt *et al.*, 2003). Pulmonary carcinomas have been diagnosed in psittacines (André and Delverdier, 1999; Jones *et al.*, 2001; Baumgartner *et al.*, 2008; Fredholm *et al.*, 2012), great horned owls (*Bubo virginianus*; Rettenmund *et al.*, 2010), red-shouldered hawks (*Buteo lineatus*; Greenlee *et al.*, 2011), and red-legged partridges (*Alectoris rufa*; Zafra *et al.*, 2011). Air sac carcinomas are described in cockatoos (Marshall *et al.*, 2004, Raidal *et al.*, 2006) and in a Timneh African grey parrot (*Psittacus erithacus timneh*; Azmanis *et al.*, 2013). Carcinomas can extend through the air sacs into the humerus or vertebrae affecting their normal function. Even though antemortem diagnosis is not uncommon, no effective therapy has been described.

Unique pulmonary tumor in cockatiels, also referred to as undifferentiated pulmonary tumor, pulmonary bimorphic tumor, or pulmonary sarcoma, has been characterized by Garner *et al.*, (2009). Birds usually are presented with a history of respiratory difficulties and a solitary mass in the lung. The tumors are clearly malignant, aggressive, and locally invasive within the lung and can extend to air sacs. Tumors can invade adjacent vertebra and produce paralysis or cause death by tracheal compression after extension into the thoracic inlet. Diagnosis is usually made postmortem and no treatment has been described (Reavill 2004; Garner *et al.*, 2009).

Respiratory hamartoma was reported in a cockatiel (*Nymphicus hollandicus*). The bird was presented with a history of coelomic distention and diagnosis was achieved by histopathology. Complete surgical resection was achieved and there was no recurrence after 2 years (Rosenwax *et al.*, 2013).

Fibrosarcoma can occur in air sacs, syrinx, and lung (Schmidt *et al.*, 2003). They have been described as locally invasive tumors that rarely metastasize and with a high potential for recurrence giving them a guarded prognosis.

Metastatic adenocarcinoma and carcinoma, fibrosarcoma, melanoma, mesothelioma, and osteosarcoma have been also reported in the avian lung (Schmidt *et al.*, 2003).

Coelomic Cavity Disease

Ascites (caused by heart disease, hypoalbuminemia, liver disease, egg-related peritonitis), enlarged abdominal organs, or masses (tumor, abscess, egg) in the coelomic cavity can produce pronounced respiratory signs by compression of the air sacs. Usually there is a history of decreased appetite and not doing well before respiratory symptoms develop. Clinical signs include increased respiratory rate and effort that progresses into severe dyspnea and open-mouth breathing when handling. Coelomocentesis and cytology of fluids, ultrasound, endoscopy, and radiology are useful in the investigation of the etiology (Orosz and Lichtenberger, 2011).

TREATMENT OF RESPIRATORY DISEASES

Treatment plans should be tailored according to the etiologic agents and anatomic involvement. Antiparasitic, antibacterial, and antifungal pharmaceutics used in avian medicine are presented in Appendix 6 of this book. The use of antimicrobials should be based on culture and sensitivity of the causative agent and ancillary tests. Supportive care according to the needs of the patient can include fluid therapy, tube feeding, oxygen therapy (mask or oxygen cage), nebulization, bronchodilators, analgesics, and antiinflammatory drugs.

Nebulization is a useful technique to hydrate and deliver topical medications to the mucous membranes of the respiratory system. To reach the lower respiratory tract the nebulizing equipment must be able to produce particles less than 3 µm in diameter. More detailed information about nebulizing equipment and common nebulizing agents can be found in Appendix 6.

Nasal and infraorbital sinus flushes (Figs. 13-77 to 13-79) can be used to loosen and remove debris from the sinus as described in Box 13-8.

Surgical Procedures

Air sac cannulation is an emergency procedure required by severe dyspneic patients with partial or total lower airway obstruction. Placement technique can be found in Chapter 11.

Surgical debridement of the supraorbital region and the infraorbital sinuses is recommended when caseous masses develop. For the infraorbital sinus, the sinusotomy incision is made midway between the medial canthus of the eye and the nares as is done for sinus aspiration. The sinus is explored and cleared of necrotic debris. The incision is irrigated often and left to heal by second intention or closed using a simple continuous pattern with monofilament suture (Morrisey, 1997; Bowles *et al.*, 2005).

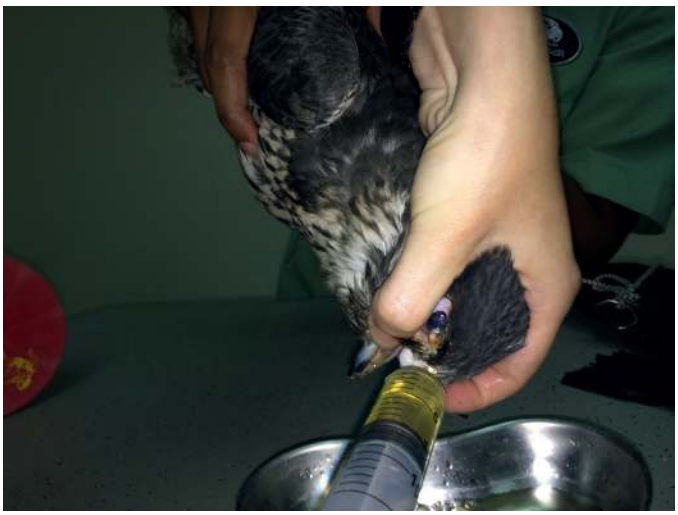


FIGURE 13-77 Nasal flushing in a peregrine falcon (*Falco peregrinus*).

FIGURE 13-78 Infraorbital sinus flush/aspiration in an orange-winged amazon (*Amazona amazonica*). This technique is useful to for collecting samples and dislodging exudate and foreign bodies from the sinus and to injecting contrast media for sinography. (Courtesy Dr. Andrés Montesinos.)

FIGURE 13-79 Pet chicken (*Gallus domesticus*) with sinusitis caused by *Mycoplasma gallisepticum*. The bird made a full recovery after sinus draining and appropriate antimicrobials. (Courtesy Dr. Andrés Montesinos.)

Sinus trephination will be required to gain access to areas of the sinus not accessible by sinus flushing. The site for trephination varies according to the species and anatomy should be studied before the surgery to avoid injury to the eyes. The detailed surgical technique has been described by Bowles *et al.*, (2005).

Tracheotomy is indicated in case of syringeal and tracheal obstructions or to retrieve tracheal foreign bodies. Tracheotomy is indicated in cases of severe tracheal stenosis. Both surgical techniques are described in Chapter 11.

REFERENCES

André JP, Delverdier M: Primary bronchial carcinoma with osseous metastasis in an African grey parrot (*Psittacus erithacus*), *J Avian Med Surg* 13:180–186, 1999.

André JP, Delverdier M, Cabanié P, et al: Malignant melanoma in an African grey parrot (*Psittacus erithacus erithacus*), *J Assoc Avian Vet* 7:83–85, 1993.

Azmanis P, Stenkat J, Hübel J, et al: A complicated, metastatic, humeral air sac cystadenocarcinoma in a timneh African grey parrot (*Psittacus erithacus timneh*), *J Avian Med Surg* 27:38–43, 2013.

Bailey TA: Respiratory diseases. In Chitty J, Lierz M, editors: *Manual of raptors, pigeons and waterfowl*, Gloucestershire, UK, 2008, British Small Animal Veterinary Association.

Baumgartner WA, Sanchez-Migallon Guzman D, Hollibush S, et al: Bronchogenic adenocarcinoma in a hyacinth macaw (*Anodorhynchus hyacinthinus*), *J Avian Med Surg* 22:218–225, 2008.

Bowles HL, Odberg E, Harrison GJ, et al: Surgical resolution of soft tissue disorders. In Harrison GJ, Lightfoot T, editors: *Clinical avian medicine*, Palm Beach, FL, 2005, Spix Publishing.

Campbell TW, Ellis CK: Comparative cytology. In *Avian and exotic animal hematology and cytology*, ed 3, Oxford, UK, 2007, Blackwell Publishing.

Cannon MJ: Unusual tracheal foreign body in an African grey parrot, *Vet Clin North Am Exot Anim Pract* 9:551–556, 2006.

Clippinger TL: Diseases of the lower respiratory tract in companion birds, *Semin Avian Exot Pet Med* 6:201–208, 1997.

Cray C, Roskos J, Zielezienski-Roberts K: Detection of cotinine, a nicotine metabolite, in the plasma of birds exposed to secondhand smoke, *J Avian Med Surg* 19:277–279, 2005.

de Matos RE, Morrisey JK, Steffey M: Postintubation tracheal stenosis in a blue and gold macaw (*Ara ararauna*) resolved with tracheal resection and anastomosis, *J Avian Med Surg* 20:167–174, 2006.

Diaz-Figueroa O, Tully TN Jr, Williams J, et al: Squamous cell carcinoma of the infraorbital sinus with fungal tracheitis and ingluvitis in an adult Solomon eclectus parrot (*Eclectus roratus solomonensis*), *J Avian Med Surg* 20:113–119, 2006.

Doneley B: *Avian medicine and surgery in practice: companion and aviary birds*, London, 2011, Manson Publishing.

Doneley RJT, Raidal SR: Tracheal resection and anastomosis in a red-tailed black cockatoo (*Calyptorhynchus banksii banksii*), *Aust Vet J* 88:451–454, 2010.

Dumonceaux G, Harrison GJ: Toxins. In Ritchie RW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and applications*, Lake Worth, FL, 1994, Wingers Publishing.

Evans A, Atkins A, Citino SB: Tracheal stenosis in a blue-billed currasow (*Crax alberti*), *J Zoo Wildl Med* 40:373–377, 2009.

Fredholm DV, Carpenter JW, Schumacher LL, et al: Pulmonary adenocarcinoma with osseous metastasis and secondary paresis in a blue and gold macaw (*Ara ararauna*), *J Zoo Wildl Med* 43:909–913, 2012.

Garner MM, Latimer KS, Mickley KA, et al: Histologic, immunohistochemical, and electron microscopic features of a unique pulmonary tumor in cockatiels (*Nymphicus hollandicus*): six cases, *Vet Pathol* 46:1100–1108, 2009.

Graham JE: Approach to the dyspneic avian patient, *Semin Avian Exot Pet Med* 13:154–159, 2004.

Greenlee JJ, Nieves MA, Myers RK: Bronchial carcinoma in a red-shouldered hawk (*Buteo lineatus*), *J Zoo Wildl Med* 42:153–155, 2011.

Harris D: Resolution of choanal atresia in African grey parrots, *Exot DVM* 1:13–17, 1999.

Heard DJ: Avian respiratory anatomy and physiology, *Semin Avian Exot Pet Med* 6:172–179, 1997.

Jankowski G, Nevarez JG, Beaufrere H, et al: Multiple tracheal resections and anastomoses in a blue and gold macaw (*Ara ararauna*), *J Avian Med Surg* 24:322–329, 2010.

Jones MP, Orosz SE, Richman LK, et al: Pulmonary carcinoma with metastases in a Moluccan cockatoo (*Cacatua moluccensis*), *J Avian Med Surg* 15:107–113, 2011.

LaBonde J: Toxicity in pet avian patients, *Semin Avian Exot Pet Med* 4:23–31, 1995.

Lightfoot TL, Yeager JM: Pet bird toxicity and related environmental concerns, *Vet Clin North Am Exot Anim Pract* 11:229–259, 2008.

MacWhirter P: Malnutrition. In Ritchie RW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and applications*, Lake Worth, FL, 1994, Wingers Publishing.
Marshall K, Daniel G, Patton C, et al: Humeral air sac mucinous adenocarcinoma in a salmon-crested cockatoo (*Cacatua moluccensis*), *J Avian Med Surg* 18:167–174, 2004.
Morrisey JK: Diseases of the upper respiratory tract of companion birds, *Semin Avian Exotic Pet Med* 6:195–200, 1997.
Noonan BP, de Matos R, Butler BP, et al: Nasal adenocarcinoma and secondary chronic sinusitis in a hyacinth macaw (*Anodorhynchus hyacinthinus*), *J Avian Med Surg* 28:143–150, 2014.
O'Malley B: *Clinical anatomy and physiology of exotic species*, London, 2005, WB Saunders.
Orosz SE, Lichtenberger M: Avian respiratory distress: etiology, diagnosis, and treatment, *Vet Clin North Am Exot Anim Pract* 14:241–255, 2011.
Powell FL: Respiration. In Whittow CG, editor: *Sturkie's avian physiology*, ed 5, San Diego, CA, 2000, Academic Press.
Raidal SR, Shearer PL, Butler R, et al: Airsac cystadenocarcinomas in cockatoos, *Aust Vet J* 84:213–216, 2006.
Reavill DR: Tumors of pet birds, *Vet Clin North Am Exot Anim Pract* 7:537–560, 2004.
Redig PT, Arent LR: Raptor toxicology, *Vet Clin North Am Exot Anim Pract* 11:261–282, 2008.
Rettenmund C, Sladky KK, Rodriguez D, et al: Pulmonary carcinoma in a great horned owl (*Bubo virginianus*), *J Zoo Wildl Med* 41:77–82, 2010.
Richardson J: Implications of toxic substances in clinical disorders. In Harrison GJ, Lightfoot T, editors: *Clinical avian medicine*, Palm Beach, FL, 2005, Spix Publishing.
Rosenwax AC, Gabor M, Reece RL: Respiratory hamartoma in a cockatiel (*Nymphicus hollandicus*), *Aust Vet J* 91:531–533, 2013.
Samour JH, Naldo J: Serratospiculiasis in captive falcons in the Middle East: a review, *J Avian Med Surg* 15:2–9, 2001.
Schmidt RE, Reavill DR, Phalen DN: Respiratory system. In Schmidt R, Reavill D, Phalen D, editors: *Pathology of pet and aviary birds*, Ames, IA, 2003, Iowa State Press.
Stoltz JH, Galey F, Johnson B: Sudden death in ten psittacine birds associated with the operation of a self-cleaning oven, *Vet Hum Toxicol* 34:420–421, 1992.
Sykes IV JM, Neiffer D, Terrell S, et al: Review of 23 cases of postintubation tracheal obstructions in birds, *J Zoo Wildl Med* 44:700–713, 2013.
Tully TN: Avian respiratory diseases: clinical overview, *J Avian Med Surg* 9:162–174, 1995.
Tully TN, Harrison GJ: Pneumonology. In Ritchie RW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and applications*, Lake Worth, FL, 1994, Wingers Publishing.
Verstappen FALM, Dorrestein GM: Aspergillosis in Amazon parrots after corticosteroid therapy for smoke-inhalation injury, *J Avian Med Surg* 19:138–141, 2005.
Weissengruber G, Loupal G: Osteochondroma of the tracheal wall in a Fischer's lovebird (*Agapornis fischeri*, Reichenow 1887), *Avian Dis* 43:155–159, 1999.
Wells RE, Slocombe RF, Trapp AL: Acute toxicosis of budgerigars (*Melopsittacus undulatus*) caused by pyrolysis products from heated polytetrafluoroethylene: clinical study, *Am J Vet Res* 43:1238–1242, 1982.
Wilson H, Brown CA, Greenacre CB, et al: Suspected sodium hypochlorite toxicosis in a group of psittacine birds, *J Avian Med Surg* 15:209–215, 2001.
Zafra R, Pérez J, Mozos E, et al: Concurrent pulmonary adenocarcinoma and silicate pneumoconiosis in a red-legged partridge (*Alectoris rufa*), *Avian Dis* 55:128–132, 2011.

FURTHER READING

Alexander DJ: A review of avian influenza in different bird species, *Vet Microbiol* 74:3–13, 2000.
Alexander DJ: Newcastle disease, *Br Poult Sci* 42:5–22, 2001.
Arca-Ruibal B, Wernery U, Zachariah R, et al: Assessment of a commercial sandwich ELISA in the diagnosis of aspergillosis in falcons, *Vet Rec* 158:442–444, 2006.
Beernaert LA, Pasmans F, Van Waeyenberghe L, et al: Aspergillus infections in birds: a review, *Avian Pathol* 39:325–331, 2010.
Cray C, Watson T, Rodriguez M, et al: Application of galactomannan analysis and protein electrophoresis in the diagnosis of aspergillosis in avian species, *J Zoo Wild Med* 40:64–70, 2009.
Dalhausen RD: Implications of mycoses in clinical disorders. In Harrison GJ, Lightfoot T, editors: *Clinical avian medicine*, Palm Beach, FL, 2005, Spix Publishing.
Dubey JP: A review of toxoplasmosis in wild birds, *Vet Parasitol* 106:121–153, 2002.
Filippich LJ: *Tumor control in birds, Semin Avian Exot Pet Med 13*:25–43, 2004.
González-Hein G, Fredes F, Hidalgo H: Diagnostic challenge, *J Exot Pet Med* 16:270–272, 2007.
Jones RC: Avian reovirus infections, *Rev Sci Tech* 19:614–625, 2000.
Lennox AM, Nemetz LP: Tracheostomy in the avian patient, *Semin Avian Exot Pet Med* 14:131–134, 2005.
Levisohn S, Kleven SH: Avian mycoplasmosis (*Mycoplasma gallisepticum*), *Rev Sci Tech* 19:425–442, 2000.
Malik R, Krockenberger MB, Cross G, et al: Avian cryptococcosis, *Med Mycol* 41:115–124, 2003.
Phalen DN: Implications of viruses in clinical disorders. In Harrison GJ, Lightfoot T, editors: *Clinical avian medicine*, Palm Beach, FL, 2005, Spix Publishing.
Reck C, Menin Á, Canever MF, et al: Rapid detection of *Mycoplasma synoviae* and avian reovirus in clinical samples of poultry using multiplex PCR, *Avian Dis* 57:220–224, 2013.
Sréter T, Varga I: Cryptosporidiosis in birds—a review, *Vet Parasitol* 87:261–279, 2000.
Velasco MC: Candidiasis and cryptococcosis in birds, *Semin Avian Exot Pet Med* 9:75–81, 2000.

DISORDERS OF THE CARDIOVASCULAR SYSTEM

Hugues Beaufrère, David Sanchez-Migallon Guzman

ANATOMY OF THE CARDIOVASCULAR SYSTEM

The avian heart has many similarities to the mammalian heart and also many differences, some of which may be of great clinical relevance. For instance, the right atrioventricular (AV) valve is muscular, lacks chordae tendineae, and has its own innervation. Other anatomic and physiologic peculiarities of clinical significance are the negative mean electrical axis (MEA), the higher heart rate and blood pressure, and the ascending aorta curving to the right instead of the left, as seen in mammals.

ETIOLOGY AND PATHOPHYSIOLOGY OF CARDIOVASCULAR DISORDERS

Cardiac disease in birds is an under-recognized problem. The different etiologies associated with cardiac disease include congenital, degenerative, nutritional, infectious, toxic, neoplastic, and idiopathic (Boxes 13-9 and 13-10). Cardiac disease may involve the pericardium, myocardium, endocardium, valves, impulse forming and conducting systems, and major blood vessels.

Pericardial diseases most frequently found in birds include pericarditis and hydropericardium. Myocardial diseases most frequently identified include dilated cardiomyopathy, myocarditis, and myocardial fibrosis, which can lead to myocardial failure and is primarily right sided. Endocardial diseases most frequently diagnosed include endocarditis, valvular insufficiency, and valvular stenosis. Vascular diseases, in particular atherosclerosis, are commonly reported in captive birds;

BOX 13-9 Noninfectious Cardiovascular Lesions Reported in Birds

Pericardium
Hydropericardium
Hemopericardium

Vasculature
Truncus arteriosus
Aortic hypoplasia
Aneurysm
Atherosclerosis

Myocardium
Dilated cardiomyopathy
Hypertrophic cardiomyopathy
Myocardial fibrosis
Atrial septal defect
Ventricular septal defect

Valvular
Endocardiosis
Fissures
Stenosis
Idiopathic degeneration

BOX 13-10 Infectious Cardiovascular Lesions and Etiologies Reported in Birds

Pericarditis/Epicarditis
Listeria monocytogenes
Riemerella anatipestifer (turkeys and ducks)
Chlamydia psittaci
Mycoplasma gallisepticum
Salmonella spp.
Escherichia coli
Mycobacterium spp.
Aspergillus spp.
Trichomonas gallinae (pigeons)
Reovirus

Endocarditis
Enterococcus spp.
Streptococcus spp.
Staphylococcus spp.
Pasteurella multocida
Erysipelothrix rhusiopathiae
Lactobacillus jensenii
E. coli
Reovirus

Intravascular/Intracardiac Parasites
Trichomonas gallinae (pigeons)
Splendidofilaria spp.
Chandlerella spp.
Cardiofilaria spp.
Paronchocerca spp.
Sarconema spp. (swans and geese)
Schistosomes (geese)

Myocarditis
Escherichia coli
Salmonella spp.
Listeria monocytogenes
Pasteurella multocida
Mycobacterium spp.
Aspergillus spp.
West Nile virus
Eastern equine encephalitis virus
Avian leucosis virus
Parvovirus (geese and Muscovy ducks)
Avian encephalomyelitis virus
Reovirus
Avian paramyxovirus 1
Avian influenza
Proventricular dilation disease (ABV)
Sarcocystis spp.
Leucocytozoon spp.
Toxoplasma gondii
Isospora serini (atoxoplasmosis, passerines)

Pericardial Effusion
Fowl adenovirus (serotype IV)
Reovirus
Polyomavirus

Cardiac Neoplasias
Marek's disease virus
Avian leukosis virus
Reticuloendotheliosis virus

less commonly reported are aneurysms and arterial rupture. Cardiovascular neoplasms are rare in birds.

Right is more common then left congestive heart failure in birds, which is suspected to be related to the particular anatomy of the right AV valve. In addition, the pathophysiology of avian heart failure has been well studied in the prevalent chicken ascites syndrome, but this disease is mainly related to production systems and rapid growth so not all findings may translate well to companion avian patients. Because right heart failure is more common in birds, signs of fluid retention from the systemic circulation usually prevail, such as ascites, hepatic congestion, pericardial effusion, jugular distention, and dyspnea from air sac compression. Pulmonary edema and congestion are seen in left heart failure; however, any cardiologic sign can be encountered in bilateral congestive heart failure. Pleural effusion is possible in birds but, if occurring, does not cause dyspnea.

Alterations of the electrocardiogram (ECG) are common but do not always correlate with clinical signs and are rarely primary disease processes in birds. Arrhythmias can be classified into excitability disturbances and conduction disturbances. Excitability disturbances have various causes and are common in dilated cardiac chambers and organic diseases. AV blocks are associated with disrupted conduction, and may be normally found in some avian species (e.g., racing pigeons) and may occur with some frequency during anesthetic events. However, a drop in blood pressure or clinical signs (e.g., syncope) associated with AV blocks are abnormal but have been documented in birds. Reported arrhythmias in birds are summarized in Table 13-5.

Atherosclerosis is an inflammatory and degenerative disease of the arterial wall characterized by the disorganization of the arterial intima from the accumulation of inflammatory cells, fat, cholesterol, calcium, cellular debris, and inflammatory cells and potentially leading to complications such as stenosis, ischemia, thrombosis, hemorrhage, and aneurysm. Atherosclerosis is probably an underlying lesion in the majority of noninfectious cardiovascular diseases diagnosed in pet birds and is undoubtedly the most common lesion of the cardiovascular system. The etiology and development of the atherosclerotic lesions can be broadly explained by the response-to-injury hypothesis. Although this widely accepted hypothesis has been constantly refined, it postulates that damage to the endothelium lining of the artery sets the stage for atherogenesis and is associated with endothelial dysfunction, inflammation, oxidative stress, and entrapment of oxidized lipoproteins in the arterial wall (Ross *et al.*, 1977; Cullen *et al.*, 2005; Falk, 2006; George and Lyon, 2010; Libby, 2012).

Histologic lesions have been well described in psittacines and appear similar to the human cardiovascular system found on necropsy in psittacine birds. Several risk factors have been suggested that may promote the development of atherosclerosis in psittacine birds and include age, gender, species, increased plasma total cholesterol and triglycerides, high-energy and high-fat diet, physical inactivity, and thyroid disease (Ross *et al.*, 1977; Bavelaar and Beynen, 2003, 2004; Garner and Raymond, 2003; Fricke *et al.*, 2009; Reavill and Dorrestein, 2010; Pilny *et al.*, 2012; Beaufrère *et al.*, 2013). Female sex and age have been definitely quantified and confirmed as important risk factors (Beaufrère *et al.*, 2013). In addition, African grey parrots (*Psittacus erithacus*), Amazon parrots (*Amazona* spp.), and cockatiels (*Nymphicus hollandicus*) are relatively susceptible to the disease whereas cockatoos (*Cacatua* spp.) and macaws (*Ara* spp.) are relatively resistant. Clinical signs are uncommon in psittacine atherosclerosis but, when present, consist of sudden death, congestive heart failure, dyspnea, neurologic signs, respiratory signs, exercise intolerance, and ataxia (Finlayson, 1965; Johnson *et al.*, 1992; Lumeij and Ritchie, 1994; Phalen *et al.*, 1996; Vink-Nooteboom *et al.*, 1998; Mans and Brown, 2007; Simone-Freilicher, 2007; St Leger, 2007; Shrubsole-Cockwill *et al.*, 2008; Fricke *et al.*, 2009; Sedacca *et al.*, 2009; Beaufrère *et al.*, 2011a; Beaufrère *et al.*, 2011b; Grosset *et al.*, 2012; Fig. 13-80).

TABLE 13-5 Selected Arrhythmias and Some Documented Causes in Birds

Arrhythmias	ECG Changes	Causes
Excitability Disturbances		
Respiratory sinus arrhythmia	Slowing of HR during expiration	Physiologic
Sinus bradycardia	Low HR. normal sinus rhythm	Vagal stimulation, atropine, anesthesia, hypokalemia, hyperkaliemia, vitamin E deficiency, vitamin B_1 deficiency, acetylcholinesterase inhibitors
Sinus tachycardia	High HR, normal sinus rhythm	Sympathetic, catecholamine stimulation
Atrial tachycardia	Series of fast atrial extrasystoles	Atrial distension, ectopic foci
Atrial fibrillation	No normal P waves, irregular SS intervals	Atrial enlargements, cardiac disease
VPC	Wide, bizarre QRS unrelated to P	Ectopic foci, hypokalemia, vitamin B_1 deficiency, vitamin E deficiency, PMV1, AI, myocardial infarction
Ventricular tachycardia	Series of VPCs	Similar causes as for VPCs
Ventricular fibrillation	Chaotic ventricular depolarization	Myocardial hypoxia, shock, severe disorders
Conduction Disturbances		
1st degree AV block	Long PR intervals	Anesthetics, increased vagal tone
2nd degree AV block	Long PR intervals, some P without QRS	Anesthetics, increased vagal tone, occasionally normal in pigeons, parrots, raptors
3rd degree AV block	Escape ventricular rhythm (slow and bizarre QRS), no consistent PR	Severe cardiomegaly
Bundle branch block	Short PR, bizarre and widened QRS	Lead, myopathy, myocarditis, uncommon in birds

From Sturkie, 1976; Odom *et al.*, 1991; Lumeij and Ritchie, 1994; Aguilar *et al.*, 1995; Martinez *et al.*, 1997; Kushner, 1999; Cote and Ettinger, 2005; Zandvliet, 2005; Rembert *et al.*, 2008; Van Zeeland *et al.*, 2010; Westerhof *et al.*, 2011.
AI, Avian influenza; AV, Atrioventricular; ECG, eletrocardiogram; HR, heart rate; PMV1, paramyxovirus type 1; VPC, ventricular premature contraction.

HISTORY AND CARDIOVASCULAR EXAMINATION

A complete history and thorough physical examination should be performed. Parrots are most often diagnosed with congestive heart failure and atherosclerosis whereas commercial poultry (e.g., broilers, turkeys) suffer more from cardiac diseases related to selection for production (Oglesbee and Oglesbee, 1998; Julian 2002, 2005; Krautwald-Junghanns *et al.*, 2004; Fricke *et al.*, 2009; Beaufrère *et al.*, 2013). Specific history of cardiac diseases may include dyspnea, exercise intolerance, falling off the perch, hindlimb ataxia, altered mentation, neurologic signs, syncope, collapse, and sudden death. Nonspecific signs of disease are also frequently present such as lethargy and anorexia. Coughing does not usually occur in birds from an enlarged heart because the aorta curves to the right and a cardiac enlargement does not cause bronchial compression (Rosenthal and Miller, 1997). Upon physical examination, findings often present with cardiovascular diseases include ascites, cyanosis or hypoperfusion (bluish or pale comb in chicken, bluish periorbital skins in some parrot species [e.g., African grey parrots, macaws] and increased ulnar vein refilling time), and increased dyspnea when restrained. Ascites in particular is frequently present in cases of congestive heart failure but is also common with other conditions. In case of significant insufficiency, a systolic murmur may be audible on cardiac auscultation. A complete cardiac examination is better performed under mild sedation, because the high avian heart rate usually decreases when the patient is anesthetized.

Diagnostic Tests

Blood pressure is higher in birds than in any other vertebrate. Direct arterial blood pressure is typically obtained by placing an arterial catheter either in the superficial ulnar artery in the proximal inner forearm or in the deep radial artery in the distal inner forearm (Fig. 13-81). The external carotid artery can also be used. Reference values have been

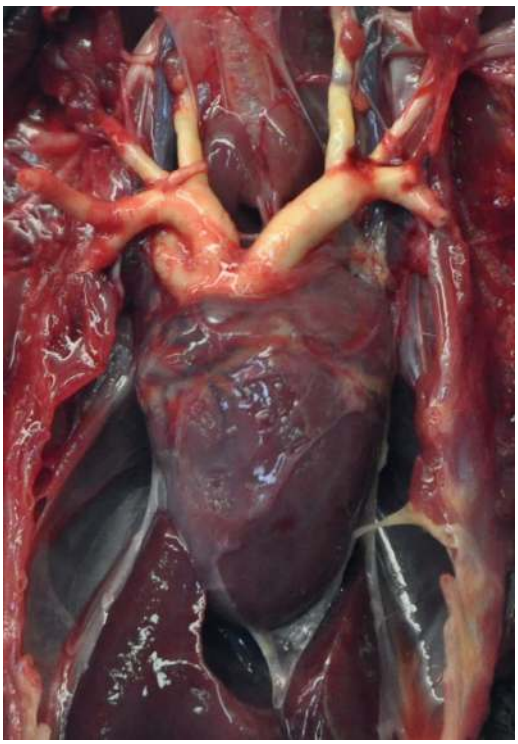


FIGURE 13-80 Adult, male African grey (*Psittacus erithacus*) parrot presented for emergency examination for extreme lethargy and collapse. Despite treatment, the bird died during hospitalization. On gross necropsy, the heart was enlarged and the great vessels were markedly distended with thick, gritty walls that when cut were partially occluded by white, luminal plaques. The lungs were diffusely dark pink. The histopathologic diagnosis was severe, chronic multifocal atherosclerosis with mural mineralization of the heart and great vessel, with moderate myocardial fibrosis and edema of the lungs.

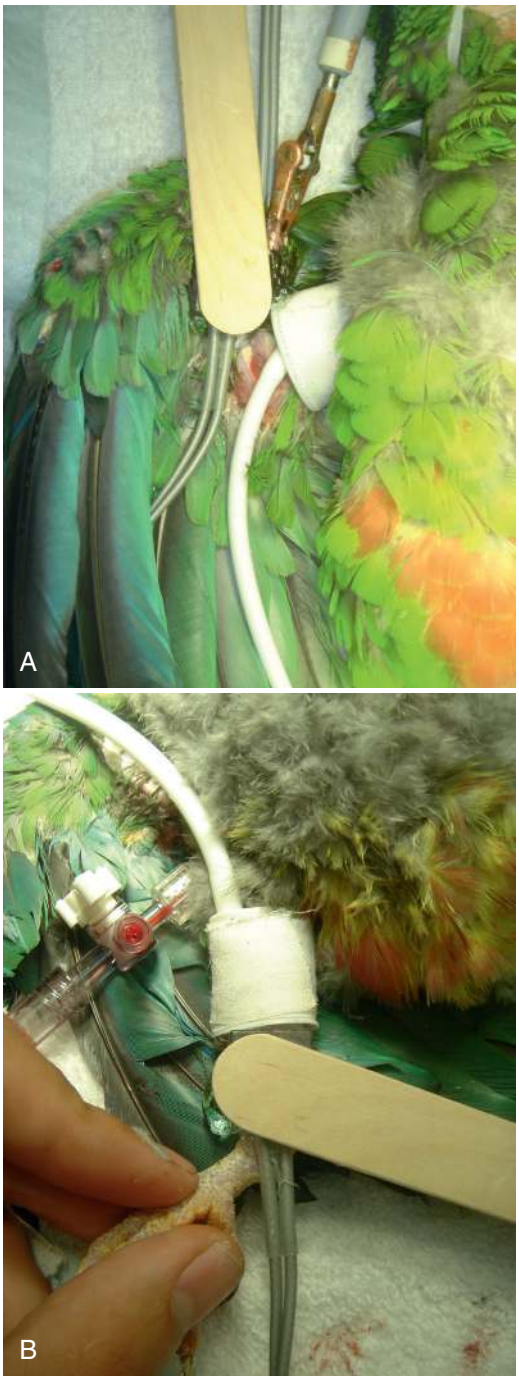


FIGURE 13-81 (A, B), The Doppler probe is placed with ultrasound gel to ensure conduction over the proximal or distal ulnar and distal tibiotarsal artery.

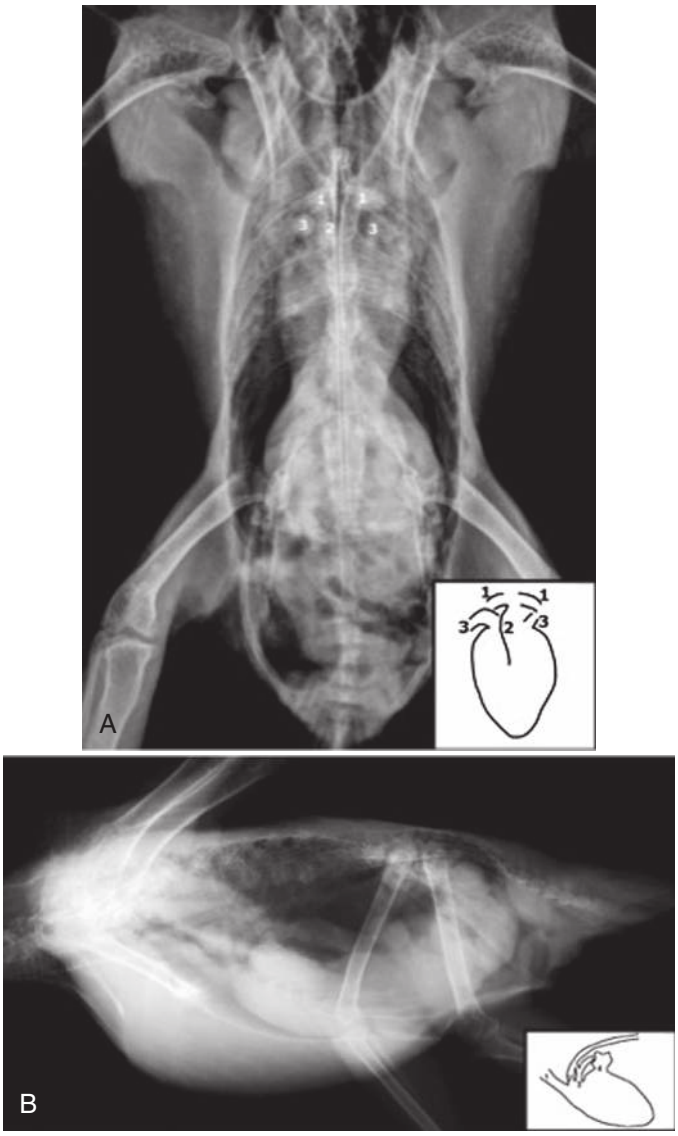


FIGURE 13-82 (A, B), Ventrodorsal radiographic view of a yellow-naped Amazon parrot (*Amazona auropalliata*). *1,* Brachiocephalic trunks; *2,* aorta; *3,* pulmonary arteries. Lateral radiographic view on right lateral recumbency. *1,* brachiocephalic trunks; *2,* aorta; *3,* pulmonary arteries; *4,* pulmonary veins.

published for several species of birds (Table 13-6). Indirect blood pressure can be obtained using a Doppler transducer and a sphygmomanometer usually placed on the wing or leg with a cuff measured at 30% to 40% of the limb circumference. However, it has been consistently demonstrated that values obtained with this method do not have good agreement with direct systolic blood pressure measurements and therefore may be of limited clinical value as a diagnostic tool (Acierno *et al.*, 2008; Zehnder *et al.*, 2009; Johnston *et al.*, 2011). In general, hypotension is defined as a systolic blood pressure lower than 90 mm Hg and a mean lower than 60 mm Hg (Lichtenberger and Ko, 2007). On the other hand, values for hypertension in birds have been poorly defined and are expected to be higher than in mammals because of their greater blood pressure. Systolic values over 200 mm Hg have been proposed as hypertensive (Lichtenberger and Ko, 2007).

Radiologic examination is of low sensitivity for cardiovascular diseases (Fig. 13-82), but severe cardiac enlargement and vascular mineralization may be detected (Fig. 13-83; Pees *et al.*, 2006c; Mans and Brown, 2007). Other changes that frequently accompany cardiovascular radiologic signs include loss of coelomic contrast and air sac space from ascites, hepatomegaly from hepatic congestion, and occasionally overinflation of the axillary diverticula of the interclavicular air sac. Several ratios have been determined but the most practical is the heart width to thoracic width ratio on the ventrodorsal view, because these

TABLE 13-6 Direct Arterial Blood Pressure in Selected Species of Birds

Species	SAP*	MAP*	DAP*	Reference
Amazon parrot (isoflurane, $n = 8$)	133 (88-177)	117 (76-158)	102 (58-146)	Schnellbacher *et al.*, 2012
Amazon parrot (isoflurane, $n = 16$)	163 (127-199)	155 (119-191)	148 (112-184)	Acierno *et al.*, 2008
Pigeon (isoflurane, $n = 15$)	93 (73-113)	82 (54-110)	72 (46-98)	Touzot-Jourde *et al.*, 2005
Red-tailed hawk (conscious, $n = 8$)	220 (119-331)	187 (104-271)	160 (70-2500	Hawkins *et al.*, 2003
Red-tailed hawk (sevoflurane, $n = 6$)	178 (124-232)	159 (109-209)	143 (95-191)	Zehnder *et al.*, 2009
Great horned owl (conscious, $n = 6$)	231.5 (157-306)	203 (146-260)	178 (128-228)	Hawkins *et al.*, 2003
Bald eagle (isoflurane, $n = 17$)	195 (165-225)	171 (142-200)	148 (120-176)	Joyner *et al.*, 2008
Bald eagle (sevoflurane, $n = 17$)	144 (116-172)	139 (111-167)	134.5 (106-163)	Joyner *et al.*, 2008
Chicken (anesthetized, $n = 40$)	141 (118-163)	136 (114-158)	131 (109-153)	Koch *et al.*, 1983
Turkey (conscious, $n = 20$)	302 (289-315)	253 (242-264)	204 (194-214)	Speckmann and Ringer, 1963
Pekin duck (anesthetized, $n = 72$)	165 (138-192)	143 (111-174)	121 (85-157)	Langille and Jones, 1975

Mean (mean ± 2 SD reference interval) mm Hg.
DAP, Distal aortic perfusion; MAP, mean arterial pressure; SAP, systolic arterial pressure.

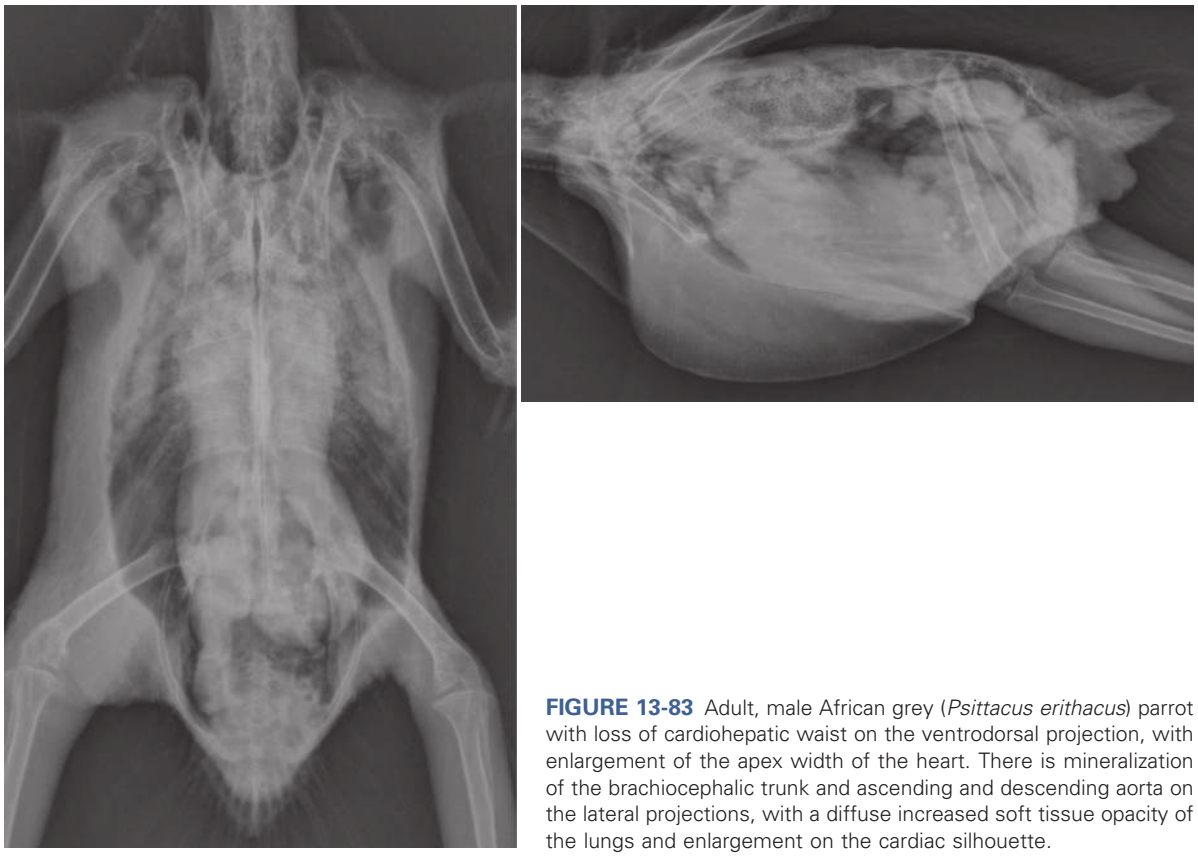


FIGURE 13-83 Adult, male African grey (*Psittacus erithacus*) parrot with loss of cardiohepatic waist on the ventrodorsal projection, with enlargement of the apex width of the heart. There is mineralization of the brachiocephalic trunk and ascending and descending aorta on the lateral projections, with a diffuse increased soft tissue opacity of the lungs and enlargement on the cardiac silhouette.

two measurements are highly correlated in birds (Fig. 13-84). In medium-sized psittacines, this ratio is 51% to 61% (Straub *et al.*, 2002; Pees *et al.*, 2006c). In Harris's hawks, this ratio was found to have similar values (Barbon *et al.*, 2010). This ratio may also vary depending on the respiratory phase with a variability as high as 10% (Lumeij *et al.*, 2011). In falcons, this ratio is considerably greater with an upper limit of 70% (Barbon *et al.*, 2010; Lumeij *et al.*, 2011). Regression-based reference intervals for cardiac radiographic sizes have been determined in peregrine falcons (*Falco peregrinus*), red-tailed hawks (*Buteo jamaicensis*), screech owls (*Otus asio*), and Canada geese (*Branta canadensis*; Hanley *et al.*, 1997; Lumeij *et al.*, 2011). With this approach, a predictive reference interval is calculated using established regression equations based on either the thoracic or the sternal width on ventrodorsal views and compared with the measured value of the patient (Table 13-7). The sternal width should be measured at the same level as the heart width, but the sternal landmarks may be obscured by an enlarged heart or fluids in diseased birds.

An ECG allows identification and characterization of arrhythmias, conduction disorders, and chamber sizes. The avian ECG is typically obtained in the frontal plane by placing two front electrodes on the propatagia and one (left) or two (earth on right) back electrodes on the knee webs using needle electrodes or flat clips (Fig. 13-85). Each lead evaluates the cardiac electrical activity on a different plane and a standard examination classically includes three bipolar leads (I, II, and III) and three augmented unipolar leads (aVR, aVL, and aVF). A proper ECG recording is easier to obtain on anesthetized birds as few will tolerate the procedure or movement and muscle tremors may impair the recordings (Oglesbee *et al.*, 2001). However, ECG tracings on conscious birds can still be obtained on pigeons, some raptors, and lethargic birds. Recordings need to be performed at 50 to 100 mm/s with 100 mm/s as optimal to better assess QRS complex morphology. Electrocardiographic measurements are typically performed on lead II tracings. The normal avian ECG is usually composed of P, S, T, and a small R wave (Fig. 13-86 and Table 13-8). The Q wave is usually missing and a Ta wave is present in certain birds. The QRS complexes are mainly the (Q)rS type on lead II, meaning the S wave is the most prominent. This contrasts with mammals where the QRS complexes are most often of the qRs type. Interpretation of the ECG follows the same rules as in mammals—it should be methodical and include determination of the heart rate, heart rhythm, MEA, and measurements (Lumeij and Ritchie, 1994). Contrary to mammals, the cardiac MEA is negative in birds with a prominent S wave that gives negative QRS

TABLE 13-7 Regression-Based Equations for Reference Heart Width (in Centimeters) in Selected Avian Species

Species	*N*	Regression Equation	R^2
Peregrine falcon	60	HW = 0.83 × SW + 0.37 ± 0.16	0.68
		HW = 0.41 × TW + 1.27 ± 0.18	0.33
Red-tailed hawk	50	HW = 0.42 × SW + 0.20 × TW + 3.42 ± 2.02	0.50
Screech owl	50	HW = 0.36 × SW + 0.13 × TW + 7.03 ± 1.40	0.36
Canada goose	50	HW = 0.27 × SW + 0.21× TW + 15.15 ± 5.00	0.27

From Hanley CS, Murray HG, Torrey S, et al: *J Avian Med Surg* 11(1):15–19, 1997 and Lumeij JT, Shaik MSA, Ali M: *J Am Vet Med Assoc* 238(11):1459–1463, 2011.
The higher the R^2, the better the precision of the reference limits. These equations are for the 95% confidence interval of the fitted value; the 95% confidence interval of the predictive value is slightly wider but predictive equations are not practical.
HW, Heart width; SW, sternal width; TW, thoracic width.

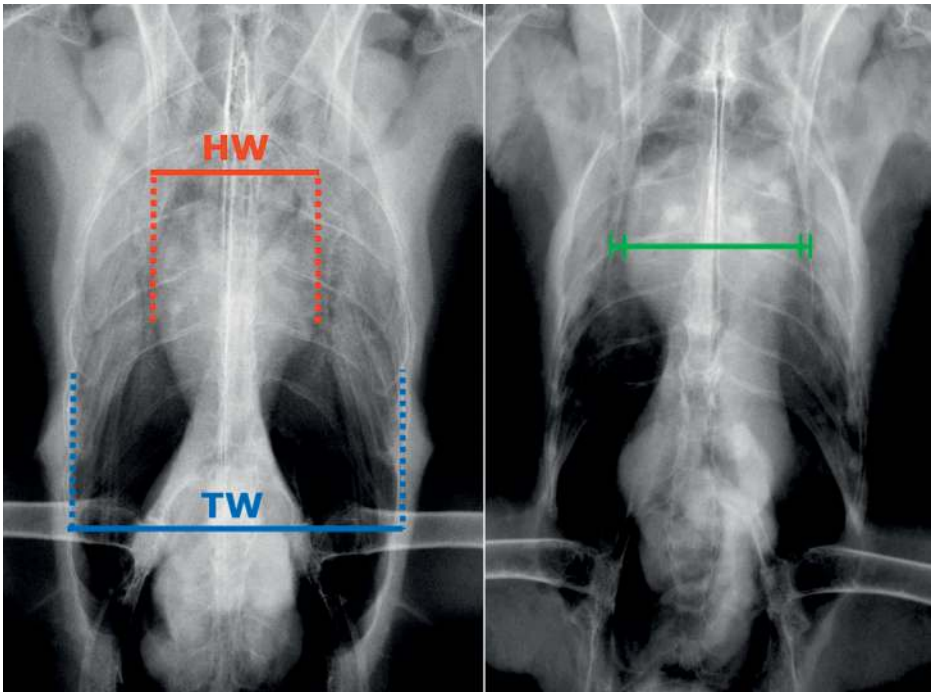


FIGURE 13-84 *Left,* Heart width and thoracic width landmarks in a macaw (*Ara* spp.). *Right,* heart width and sternal width landmarks in a peregrine falcon (*Falco peregrinus*).

TABLE 13-8 Electrocardiogram Measurement Reference Values for Lead II in Selected Avian Species

Species	Racing Pigeon	Amazon Parrot	Grey Parrot	Macaw	Cockatoo	Red-tailed Hawk	Bald Eagle	Pekin Duck	Chicken
N	60	37	45	41	31	11	20	50	72
Heart rate	160-300	340-600	340-600	255-555	259-575	80-220	50-160	200-360	180-340
P amplitude	0.4-0.6	0.25-0.60	0.25-0.55	0.03-0.47	0.13-0.53	–0.1-0.175	0.050-0.325		
P duration	0.015-0.020	0.008-0.017	0.012-0.018	0.009-0.021	0.009-0.025	0.020-0.035	0.030-0.060	0.015-0.035	0.035-0.043
PR interval	0.045-0.070	0.042-0.055	0.040-0.055	0.040-0.068	0.039-0.071	0.050-0.090	0.070-0.110	0.04-0.08	0.073-0.089
S amplitude	1.5-2.8	0.7-2.3	0.9-2.2	0.27-1.43	0.27-1.59	0.300-0.900	0.150-1.450	0.35-1.03	0.10-1.0
QRS duration	0.013-0.016	0.010-0.015	0.010-0.016	0.002-0.030	0.014-0.026	0.020-0.030	0.020-0.040	0.028-0.044	0.02-0.028
T amplitude	0.3-0.8	0.3-0.8	0.18-0.6	0.12-0.80	0.17-0.97	0.000-0.300	0.050-0.200	0.04-0.40	0.03-0.28
QT interval	0.060-0.075	0.050-0.095	0.048-0.080	0.053-0.109	0.065-0.125	0.080-0.165	0.110-0.165	0.08-0.12	
MEA	–83 to –99	–90 to –107	–79 to –103	–76 to –87	–73 to –89	–50 to –110	–30 to –150	–160-95	–91 to –120

From Sturkie, 1976; Lumeij and Stokhof, 1985; Nap *et al.*, 1992; Burtnick and Degernes, 1993; Cinar *et al.*, 1996; Oglesbee *et al.*, 2001.
Note: Amplitude in millivolts and duration in seconds. To obtain a 95% reference interval, all published results in the form of mean ± SD were reported as mean ± 2 SD and in the form of mean ± SEM were reported as mean ± 2 SEM $\sqrt{n}$, when only the range or a 95% reference interval was published, it was reported as is.
MEA, Mean electrical axis.

FIGURE 13-85 To obtain an electrocardiogram, four electrodes using needles (25 gauge) placed through the skin are attached in the following manner: one in each proximal propatagial area and one each in the cranial inguinal web.

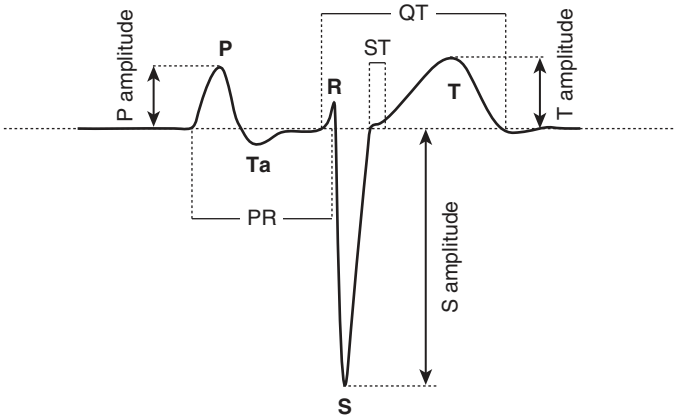


FIGURE 13-86 Normal avian electrocardiographic complex with depiction of the different measurement landmarks.

complexes on lead II. This is caused by subepicardial and endocardial transmission of the depolarization (Lumeij and Ritchie, 1994). However, some poultry birds have a positive MEA and QRS complexes such as broilers and Pekin ducks. Measurements that are usually taken include P amplitude and duration, PR interval, S amplitude, QRS duration, ST segment, T amplitude, QT interval, and MEA. The T wave is always positive in birds in lead II and a change in polarity indicates myocardial hypoxia (Lumeij and Ritchie, 1994). In high heart rates, generally over 300 to 500 bpm, P and T waves may be fused (atria depolarized before ventricles are completely repolarized) and the P wave not discernible (Sturkie, 1976; Zandvliet, 2005). This P on T phenomenon also seems to be a normal finding in Amazon and African grey parrots (Nap *et al.*, 1992; Zandvliet, 2005). ST segment elevation is common in healthy birds and does not indicate cardiac diseases as in mammals (Lumeij and Ritchie, 1994; Oglesbee *et al.*, 2001). The ST segment is often short or absent with the S wave merged with the T wave (ST slurring) (Lumeij and Ritchie, 1994; Zandvliet, 2005). With the electrophysiologic specificities of birds in mind, the interpretation of the avian ECG is similar to that of mammals. Reference intervals have been published for several species (Table 13-8). Anesthesia is suspected to affect the ECG measurements only in heart rate, QT interval, and the frequency of some arrhythmias (AV blocks; Nap *et al.*, 1992; Aguilar *et al.*, 1995; Joyner *et al.*, 2008).

Echocardiography allows identification of abnormalities in cardiac anatomy and function. However, cardiac ultrasound examination presents some major limitations in birds because of the location of the

FIGURE 13-87 Echocardiography using the ventromedial approach in a hyacinth macaw (*Anodorhynchus hyacinthinus*).

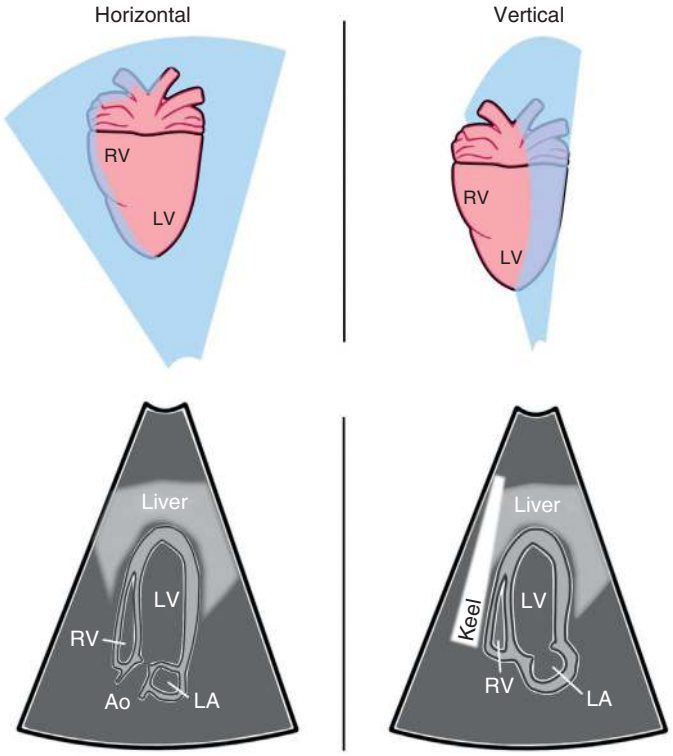


FIGURE 13-88 Echocardiographic horizontal four-chamber view and vertical two-chamber view in birds as classically obtained through the ventromedial approach.

heart in an indentation of the keel surrounded by air sacs. Therefore available acoustic windows and cardiac views are limited. Two standardized approaches have been described for transcoelomic examination: a ventromedial and a parasternal approach (Krautwald-Junghanns *et al.*, 1995; Pees *et al.*, 2004, 2006; Pees and Krautwald-Junghanns, 2005). A high probe frequency and frame rate are recommended, as well as a small transducer. The ventromedial approach consists of placing the probe caudal to the keel and imaging the heart cranially using the liver as an acoustic window to avoid air sacs laterally and the keel ventrally (Fig. 13-87). This is the most common approach in psittacine and raptorial birds. It can be performed on the bird awake, sedated, or anesthetized. Only two views can classically be obtained through this approach: the horizontal four-chamber view and the vertical two-chamber view by rotating the probe by 90 degrees (Fig. 13-88). All views are longitudinal (long axis) and cardiac transverse views (short axis) and M-mode echocardiography that has better temporal resolution cannot be performed in birds by the transcoelomic approach. This precludes the establishment of the same echocardiographic standards in birds as in small animal cardiology (Thomas *et al.*, 1993). Therefore the amount of information gathered from the transcoelomic echocardiographic examination is very limited in birds and all morphometric and functional measurements have to be performed on two-dimensional images (B-mode).

Measurements on several cardiac cycles should be averaged to obtain representative values. Reference intervals have been published for ventricular and atrial dimensions and fractional shortening [(diastole − systole)/diastole %] in several avian species (Table 13-9; Krautwald-Junghanns *et al.*, 1995; Pees *et al.*, 2004, 2006c). Transcoelomic echocardiography is also thought to underestimate the true fractional shortening of the highly efficient avian heart (Beaufrère *et al.*, 2012). Right ventricular measurements are also not routinely taken in mammals because of the complex three-dimensional configuration (Bélanger, 2005). Furthermore, recent evidence suggests that taking echocardiographic measurements may not be clinically useful considering the avian heart size, heart rate, current equipment resolution, and the fact that observers can add up to 30% variability (Beaufrère *et al.*, 2012). In dogs and cats, in most cases, a good impression of cardiac chamber size and function can be achieved without having made any quantitative measurements (Boon, 2011). Likewise, an adequate morphologic and functional assessment of the avian heart can be performed qualitatively during the echocardiographic examination. Nevertheless, pathologic changes seen in birds are usually severe when cardiac disease is present with drastic chamber dilation (most often the right heart), pericardial effusion, ascites, and poor contractility, which do not require measurements for confirmation (Fig. 13-89). If measurements are taken for follow-up, it is recommended that the same operator and equipment are used and changes in measurements should be greater than 20% to be considered genuine.

Color Doppler echocardiography can be used for detection of turbulence and reflux indicative of valvular insufficiency with right AV insufficiency most commonly imaged. Spectral Doppler echocardiography can be used to measure inflow and outflow velocities, and reference intervals have been published in a few species (Table 13-10; Straub, 2003; Pees *et al.*, 2004, 2006c; Straub *et al.*, 2004). Fortunately, echocardiographic examinations are easier and more rewarding in birds with cardiac disease because ascitic fluid, pericardial effusion, hepatomegaly, and cardiac enlargement greatly improve acoustic windows and facilitate the procedure. The parasternal approach consists of placing the probe laterally (typically on the right to avoid the ventriculus) behind the ribs and above the sternum and imaging the heart craniomedially (Krautwald-Junghanns *et al.*, 1995; Pees *et al.*, 2006c). This can be performed on pigeons, some raptors, and is the approach of choice in gallinaceous birds (especially younger chickens). In these birds, the limited caudal extension of the ribs and the larger fenestration of the keel allow a lateral approach to the heart. Typically,

TABLE 13-9 Echocardiographic Reference Intervals in Millimeters in Selected Avian Species Obtained in the Horizontal Four-Chamber View

Parameter	African Grey Parrots	Amazon Parrots	Cockatoos	Diurnal Raptors*	Pigeons (parasternal)
N	60	10	10	100	50
Left Ventricle					
Systole length	18.4-26	16.5-25.7	16.4-21.6	9.1-20.3	15.9-19.9
Systole width	4.8-8.8	4.3-9.1	3.0-9.8	4.1-8.5	4.4-6.0
Diastole length	20.2-27.8	17.7-26.5	16.7-23.1	11.0-21.8	17.3-22.9
Diastole width	6.6-10.6	6.4-10.4	5.3-11.3	5.3-10.1	6.2-8.6
FS (%)	13.8-31.4	14.4-31.2	11.6-39.6		
Right Ventricle					
Systole length	6.4-12.0	5.8-13.0	7.9-12.7	7.3-18.1	
Systole width	1.0-4.6	1.7-4.5	7.9-12.7	0.9-3.3	
Diastole length	7.7-15.3	7.7-12.9	6.7-15.9	8.9-18.9	8.3-11.5
Diastole width	2.6-7.0	2.6-7.8	2.5-4.5	0.9-4.1	3.0-5.0
FS (%)	17.0-64.6	26.7-41.5	12.7-53.9		
Aorta					
Systole diameter	2.8-4.4	2.0-4.0			
Diastole diameter	2.8-5.2	2.2-4.6		2.0-3.6	2.8-3.2

From Boskovic *et al.*, 1995; Pees *et al.*, 2004, 2006c.
Note: To obtain a 95% reference interval, all published results in the form of mean ± SD were reported as mean ± 2 SD and in the form of mean ± SEM were reported as mean ± 2 SEM √n, when only the range or a 95% reference interval was published, it was reported as is. Echocardiographic measurements may not be reliable and clinically useful.
*European diurnal raptors included common buzzard, European sparrowhawk, northern goshawk, and black kite.
FS, Fractional shortening.

TABLE 13-10 Spectral Doppler Echocardiographic Reference Intervals in Millimeters per Seconds in Selected Avian Species Obtained in the Horizontal Four-Chamber View

Species	*N*	Left Diastolic Inflow	Right Diastolic Inflow	Aortic Systolic Outflow
Amazon parrots		0.12-0.24	0.12-0.32	0.67-0.99
Cockatoos		0.02-0.62		0.40-1.16
African grey parrots		0.27-0.51		0.63-1.15
Macaws		0.40-0.68		0.55-1.07
Harris's hawks	10	0.13-0.25	0.15-0.27	0.75-1.43
Falcons	15	0.18-0.38	0.17-0.37	1.07-1.43
Common buzzard	10	0.16-0.28	0.13-0.25	1.04-1.68
Barn owls	10	0.14-0.26	0.10-0.34	0.84-1.32

From Straub, 2003; Straub *et al.*, 2004; Pees *et al.*, 2006c.
Note: To obtain a 95% reference interval, all published results in the form of mean ± SD were reported as mean ± 2 SD and in the form of mean ± SEM were reported as mean ± 2 SEM √n, when only the range or a 95% reference interval was published, it was reported as is. Parrots were anesthetized, and raptors were awake.

more imaging planes can be obtained and transverse views have been described in pigeons and chickens (Krautwald-Junghanns *et al.*, 1995; Martinez-Lemus *et al.*, 1998). A transesophageal echocardiographic protocol has been implemented in several species of birds in an attempt to alleviate the limitations associated with the transcoelomic approach. With this technique, a transesophageal ultrasonographic probe is inserted into the upper digestive system, with the bird under general anesthesia, and the heart is imaged from inside the proventriculus (Beaufrère *et al.*, 2010).

Fluoroscopic angiography visualizes the heart and vascular tree in real time. Under general anesthesia, the bird is initially positioned in left lateral recumbency on a fluoroscopy table (Fig. 13-90). A bolus of nonionic iodinated contrast agent (2 mL/kg IV; iohexol 240 mg/mL; Omnipaque, GE Healthcare Inc., Princeton, NJ) is injected at a rate of 1 to 2 mL/kg/s, through a catheter inserted into the basilic or medial metatarsal vein during video acquisition at a rate of 30 frames per second for the best resolution. The same bolus is repeated to obtain the ventrodorsal view with the bird placed in dorsal recumbency. The brachiocephalic trunks, aorta, pulmonary arteries, pulmonary veins, and caudal vena cava can be seen. The brachiocephalic trunks and aorta can be seen pulsating with the heartbeats. Marked lumen changes can be observed during the cardiac cycle. This procedure is easy and inexpensive and can be recorded for further analysis and measurements. For measurement, to account for different degrees of magnification, a calibrated marker should be kept in the field during fluoroscopic acquisition, but fluoroscopic angiography is likely more useful for qualitative assessment and investigation of aneurysms and stenosis.

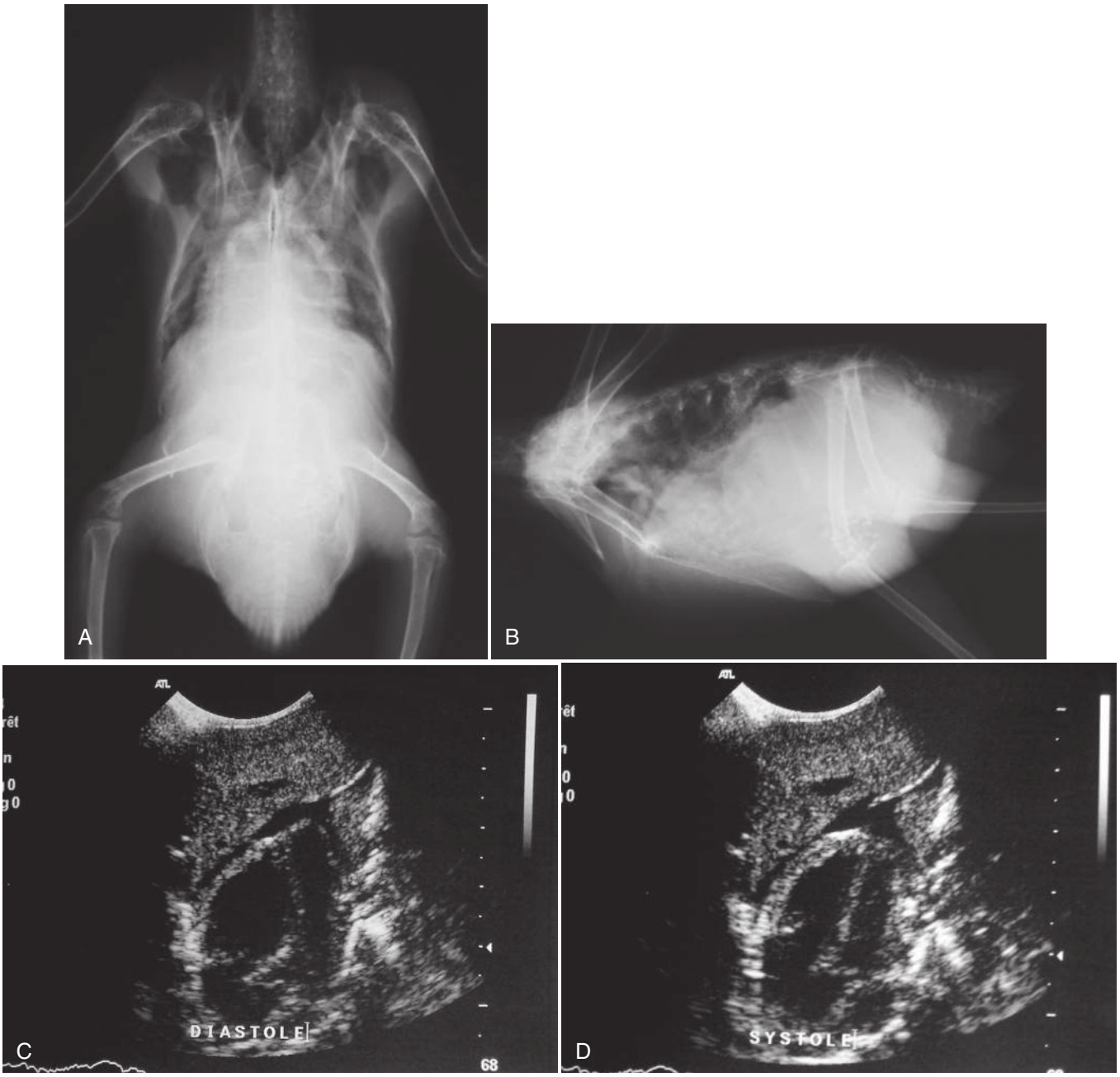


FIGURE 13-89 **(A, B),** Ventrodorsal and laterolateral radiographic views of an African grey (*Psittacus erithacus*) parrot with ascites. **(C, D),** Enlarged cardiac silhouette and echocardiographic images (systole and diastole) of the heart with enlarged chamber size and pericardial effusion of the same bird diagnosed with congestive heart failure.

TREATMENT

The treatment of cardiac disease first requires stabilization of the patient. Coelomocentesis and O_2 supplementation should be considered if severe ascites is present and the bird shows dyspnea. Endoscopically guided pericardiocentesis should be considered when severe pericardial effusion results in diastolic heart failure. The midline approach is recommended for pericardiocentesis so no fluid leaks into the air sacs. An endoscopic needle is used with its sheath and should only protrude by 1 to 2 mm to prevent puncturing the heart during the procedure. A permanent surgical window or partial pericardectomy can be performed if necessary (Fig. 13-91). Doses of selected cardiac therapeutic agents in birds are presented in Table 13-11.

Diuretics are indicated to reduce fluid overload, edema, and effusion. Furosemide is the most commonly used diuretic, and it should not be used alone long term because it further activates the renin-angiotensin-aldosterone system (Schroeder, 2010). Electrolytes and renal parameters (e.g., uric acid) should be monitored when using furosemide chronically and hypokalemia is a frequently reported side effect. Cardiac tamponade is a contraindication for the use of

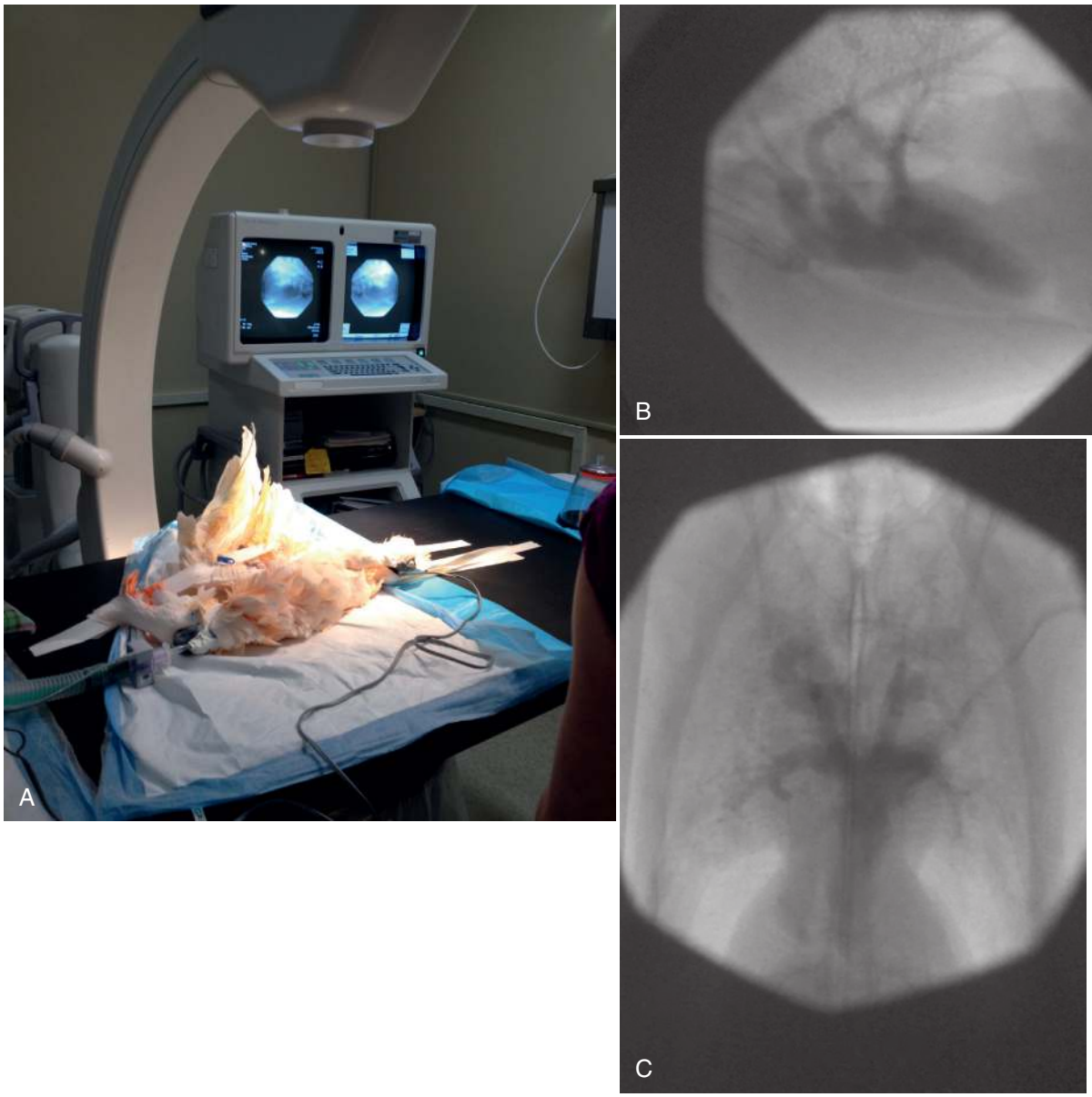


FIGURE 13-90 **(A),** Fluoroscopy in a Moluccan (*Cacatua moluccensis*) cockatoo with the bird positioned on the table in left lateral recumbency. **(B, C),** Left lateral and ventrodorsal views with contrast highlighting the left atrium and ventricle, brachiocephalic trunks, aorta, and pulmonary veins.

furosemide because it decreases cardiac preload, which is necessary in this disease to support ventricular filling and maintain cardiac output. Finally, it should be used with caution in patients with renal diseases.

Angiotensin-converting enzyme (ACE) inhibitors block the formation of angiotensin II. They promote venous and arterial vasodilation and limit aldosterone production. As a result, they decrease preload and afterload, with a risk of hypotension and hyperkaliemia (Bulmer, 2010). Enalapril is the most commonly used ACE-inhibitor in birds and has been reported to be safe and effective in companion psittacine birds. Positive inotropes are used to enhance cardiac contractility. Disadvantages include an increase in myocardial oxygen consumption and arrhythmias (except probably with pimobendan). They are contraindicated in hypertrophic cardiomyopathy and aortic and pulmonic stenosis. Digoxin, a digitalis glycoside, enhances contractility by directly inhibiting the Na/K ATPase pump, which results in intracellular calcium accumulation through the activation of the Na/Ca exchanger (Fuentes, 2010). Along with being a weak positive inotrope, it is also a negative chronotrope and positive lusitrope. Digoxin has been used in birds; however, its use is becoming more controversial because of the existence of new positive inotropes and because of its potential adverse effects, mostly gastrointestinal and proarrhythmic. Recommended therapeutic levels for digoxin are 0.8 to 1.2 ng/mL (Fuentes, 2010). Pimobendan is a positive inotrope and arterial vasodilator (inodilator) with its action from calcium sensitization of myofibrils and phosphodiesterase III inhibition. Pimobendan has been evaluated in psittacine birds, and it is recommended to compound the

TABLE 13-11 Dosages of Selected Cardiac Therapeutic Agents in Birds

Drug	Species	Dose	Basis	References
Diuretics				
Furosemide	Parrots, raptors	0.15-2 mg/kg PO, IM every 12-24 h	EU	Pees *et al.*, 2006c
	Chickens	5 mg/kg PO	PD	Esfandiary *et al.*, 2010
		2.5 mg/kg IM	PD	Esfandiary *et al.*, 2010
Spironolactone	Chickens	1 mg/kg PO	PD	Esfandiary *et al.*, 2010
	Parrots	1 mg/kg PO every 12 h	EU	Sedacca *et al.*, 2009
Positive Inotropes				
Digoxin	Budgerigars	0.02 mg/kg PO every 24 h	PK	Hamlin and Stalnaker, 1987
	Sparrows	0.02 mg/kg PO every 24 h	PK	Hamlin and Stalnaker, 1987
	Quaker parrots	0.05 mg/kg PO every 24 h	PK	Wilson *et al.*, 1989
Pimobendan	Amazon parrots	10 mg/kg PO every 12 h	PK	Sanchez-Migallon Guzman *et al.*, 2014
	Harris's hawk	0.25 mg/kg PO every 12 h	PK, EU	Sanchez-Migallon Guzman *et al,*. 2014
	Parrots	0.25 mg/kg PO every 12 h	EU	Sedacca *et al.*, 2009; Van Zeeland *et al.*, 2010
Dobutamine	Amazon parrots	5-15 μg/kg/min (CRI)	PD	Schnellbacher *et al.*, 2012
Dopamine	Amazon parrots	5-10 μg/kg/min (CRI)	PD	Schnellbacher *et al.*, 2012
Negative Inotropes				
Propranolol	Most species	0.2 mg/kg IM, 0.04 mg/kg IV	EU	
Atenolol	Most species	5-10 mg/kg PO every 12-24 h	EU	
Diltiazem	Most species	1-2 mg/kg PO every 8-24 h	EU	
Vasodilators				
Enalapril	Pigeons	1.25 mg/kg PO every 12-18 h	PK	Pees *et al.*, 2006a
	Amazons	1.25 mg/kg PO 12-18 h	PK	Pees *et al.*, 2006a
	Parrots	2.5-5mg/kg PO every 12 h	EU	Straub *et al.*, 2003; Pees *et al.*, 2006a
Benazepril	Parrots	0.5 mg/kg PO every 24 h	EU	Sedacca *et al.*, 2009
Parasympatholytics				
Atropine	Most species	0.01-0.02 mg/kg IM	EU	
Glycopyrrolate	Most species	0.01-0.02 mg/kg IM	EU	
Antiarrhythmics				
Lidocaine	Amazon parrot	2.5 mg/kg IV	PK	Da Cunha *et al.*, 2011
Mexiletine	Parrots	4-8 mg/kg PO every 12-24 h	EU	
Propantheline	Parrots	0.1-0.3 mg/kg PO every 8 h	EU	Van Zeeland *et al.*, 2010

CRI, Constant rate infusion; EU, empirical use; IM, intramuscular; IV, intravenous; PD, pharmacodynamics study; PK, pharmacokinetic study; PO, by mouth.

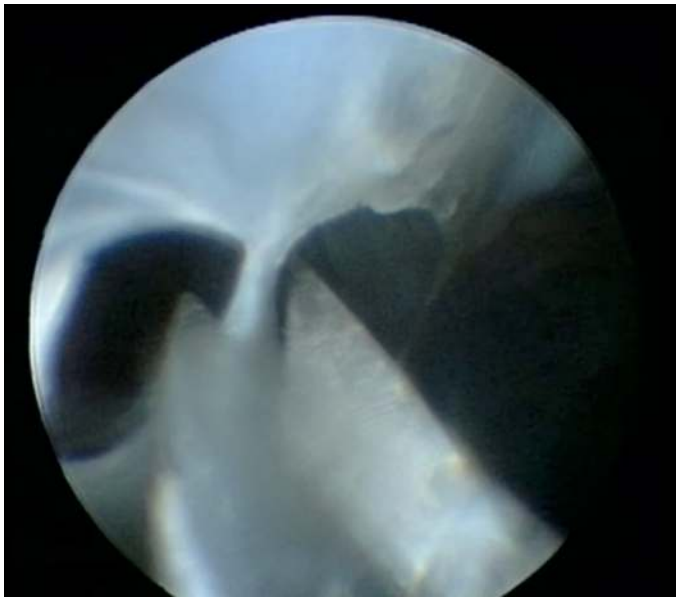


FIGURE 13-91 Pericardiotomy in a domestic chicken (*Gallus gallus domesticus*) with severe pericardial effusion using the ventromedial approach through the ventral hepatoperitoneal space. The opening into the pericardial spaced was used to collect a fluid sample to be submitted for analysis.

tablet to achieve a larger amount of the drug to be absorbed. Pimobendan pharmacokinetics is likely very different in other avian groups (e.g., raptors), and dosages might not be able to be extrapolated. Dobutamine is a potent positive inotrope that exerts its activity by selective β1-adrenergic activity. Because it is short lived, it is used as a constant rate infusion (CRI). Dobutamine CRI may be useful for correcting severe hypotension in psittacines caused by anesthesia maintained with 2.5% isoflurane or in refractory cases with severe systolic dysfunction and cardiogenic shock.

REFERENCES

Acierno MJ, da Cunha A, Smith J, et al: Agreement between direct and indirect blood pressure measurements obtained from anesthetized Hispaniolan Amazon parrots, *J Am Vet Med Assoc* 233(10):1587–1590, 2008.

Aguilar RF, Smith VE, Ogburn P, et al: Arrhythmias associated with isoflurane anesthesia in bald eagles (*Haliateeus leucocephalus*), *J Zoo Wildl Med* 26(4):508–516, 1995.

Barbon AR, Smith S, Forbes N: Radiographic evaluation of cardiac size in four falconiform species, *J Avian Med Surg* 24(3):222–226, 2010.

Bavelaar FJ, Beynen AC: Severity of atherosclerosis in parrots in relation to the intake of alpha-linolenic acid, *Avian Dis* 47(3):566–577, 2003.

Bavelaar FJ, Beynen AC: Atherosclerosis in parrots: a review, *Vet Q* 26(2):50–60, 2004.

Beaufrère H, Pariaut R, Nevarez JG, et al: Feasibility of transesophageal echocardiography in birds without cardiac disease, *J Am Vet Med Assoc* 236(5):540–547, 2010.

Beaufrère H, Holder KA, Bauer R, et al: Intermittent claudication-like syndrome secondary to atherosclerosis in a yellow-naped Amazon parrot (*Amazona ochrocephala auropalliata*), *J Avian Med Surg* 25(4):266–276, 2011a.

Beaufrère H, Nevarez J, Gaschen L, et al: Diagnosis of presumed acute ischemic stroke and associated seizure management in a Congo African grey parrot, *J Am Vet Med Assoc* 239(1):122–128, 2011b.

Beaufrère H, Pariaut R, Rodriguez D, et al: Comparison of transcoelomic, contrast, and transesophageal echocardiography in anesthetized red-tailed hawk (*Buteo Jamaicensis*), *Am J Vet Res* 73(10):1560–1568, 2012.

Beaufrère H, et al: Prevalence and risk factors in psittacine atherosclerosis: a multicenter case-control study, *J Am Vet Med Assoc* 242:1696–1704, 2013.

Bélanger MC: Echocardiography. In Ettinger SJ, Feldman EC, editors: *Textbook of veterinary internal medicine*, St Louis, MO, 2005, Elsevier Saunders.

Boon JA: Evaluation of size, function, and hemodynamics. In Boon JA, editor: *Veterinary echocardiography*, ed 2, Oxford, UK, 2011, Wiley-Blackwell.

Boskovic M, Krautwald-Junghanns ME, Failing K, et al: Moglichkeiten und grenzen echokardiographischer untersuchungen bei tag-und nachgreivogeln (Accipitriformes, Falconiformes, Strigiformes), *Tierarztl Prax* 27:334–341, 1995.

Bulmer BJ: Angiotensin converting enzyme inhibitors and vasodilators. In Ettinger SJ, Feldman EC, editors: *Textbook of veterinary internal medicine*, ed 7, St Louis, MO, 2010, Elsevier Saunders.

Burtnick NL, Degernes LA: Electrocardiography on fifty-nine anesthetized convalescing raptors. In Redig PT, Cooper JE, Remple JD, Hunter DB, editors: *Raptor biomedicine*, Minneapolis, MN, 1993, University of Minnesota Press.

Cinar A, Bagci C, Belge F, et al: The electrocardiogram of the Pekin duck, *Avian Dis* 40(4):919–923, 1996.

Cote E, Ettinger SJ: Electrocardiography and cardiac arrhythmias. In Ettinger SJ, Feldman EC, editors: *Textbook of veterinary internal medicine*, ed 6, St Louis, MO, 2005, Elsevier Saunders.

Cullen P, Rauterberg J, Lorkowski S: The pathogenesis of atherosclerosis. In vonEckardstein A, editor: *Atherosclerosis: diet and drugs*, Berlin, Germany, 2005, Springer.

Da Cunha AF, Stout R, Tully T, et al: Pharmacokinetics/Pharmacodynamics of bupivacaine and lidocaine in chickens. In *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, 2011, p 313.

Esfandiary A, Rajaian H, Asasi K, et al: Diuretic effects of several chemical and herbal compounds in adult laying hens, *Int J Poult Sci* 9(3):247–253, 2010.

Falk E: Pathogenesis of atherosclerosis, *J Am Coll Cardiol* 47(8):7–13, 2006.

Finlayson R: Spontaneous arterial disease in exotic animals, *J Zool* 147:239–343, 1965.

Fricke C, Schmidt V, Cramer K, et al: Characterization of atherosclerosis by histochemical and immunohistochemical methods in African grey parrots (*Psittacus erithacus*) and Amazon parrots (*Amazona* spp, *Avian Dis* 53:466–472, 2009.

Fuentes VL: Inotropes: inodilators. In Ettinger SJ, Feldman EC, editors: *Textbook of veterinary internal medicine*, ed 7, St Louis, MO, 2010, Saunders Elsevier.

Garner MM, Raymond JT: A retrospective study of atherosclerosis in birds. *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp 59–66, 2003.

George SJ, Lyon C: Pathogenesis of atherosclerosis. In George SJ, Johnson J, editors: *Atherosclerosis: molecular and cellular mechanisms*, Weinhein, Germany, 2010, Wiley-Vch.

Grosset C, Sanchez-Migallon Guzman D, Keating MK, et al: Central vestibular disease in a blue and gold macaw (*Ara ararauna*) with vasculopathy and cerebral infarction and hemorrhage. *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, p 257, 2012.

Hamlin RL, Stalnaker PS: Basis for use of digoxin in small birds, *J Vet Pharmacol Ther* 10(4):354–356, 1987.

Hanley CS, Murray HG, Torrey S, et al: Establishing cardiac measurement standards in three avian species, *J Avian Med Surg* 11(1):15–19, 1997.

Hawkins MG, Wright BD, Pascoe PJ, et al: Pharmacokinetics and anesthetic and cardiopulmonary effects of propofol in red-tailed hawks (*Buteo jamaicensis*) and great horned owls (*Bubo virginianus*), *Am J Vet Res* 64(6):677–683, 2003.

Johnson JH, Phalen DN, Kondik VH, et al: Atherosclerosis in psittacine birds. *Proceedings of the Annual Conference of the Association of Avian Veterinarians, Australian Committee*, pp 87–93, 1992.

Johnston MS, Davidowski LA, Rao S, et al: Precision of repeated, doppler-derived indirect blood pressure measurements in conscious psittacine birds, *J Avian Med Surg* 25(2):83–90, 2011.

Joyner PH, Jones MP, Ward D, et al: Induction and recovery characteristics and cardiopulmonary effects of sevoflurane and isoflurane in bald eagles, *Am J of Vet Res* 69(1):13–22, 2008.

Julian RJ: Cardiovascular disease. In Jordan F, Pattison M, Alexander D, Faragher T, editors: *Poultry diseases*, ed 5, London, 2002, WB Saunders.

Julian RJ: Production and growth related disorders and other metabolic diseases of poultry—a review, *Vet J* 169(3):350–369, 2005.

Koch JE, Buss G, Lobaugh B, et al: Blood pressure of chickens selected for leanness or obesity, *Poult Sci* 62(5):904–907, 1983.

Krautwald-Junghanns M-E, Schulz M, Hagner D, et al: Transcoelomic two-dimensional echocardiography in the avian patient, *J Avian Med Surg* 9:19–31, 1995.

Krautwald-Junghanns ME, et al: Research on the anatomy and pathology of the psittacine heart, *J Avian Med Surg* 18(1):2–11, 2004.

Kushner LI: ECG of the month. Atrioventricular block in a muscovy duck, *J Am Vet Med Assoc* 214(1):33–36, 1999.

Langille BL, Jones DR: Central cardiovascular dynamics of ducks, *Am J Physiol* 228(6):1856–1861, 1975.

Libby P: Inflammation in atherosclerosis, *Nature* 420(6917):868–874, 2012.

Lichtenberger M, Ko J: Critical care monitoring, *Vet Clin North Am Exot Anim Pract* 10(2):317–344, 2007.

Lumeij JT, Ritchie BW: Cardiology. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine,principles and applications*, Lake Worth, FL, 1994, Wingers Publishing.

Lumeij JT, Stokhof AA: Electrocardiogram of the racing pigeon (*Columba livia domestica*), *Res Vet Sci* 38(3):275–278, 1985.

Lumeij JT, Shaik MSA: Ali M,Radiographic reference limits for cardiac width in peregrine falcons (*Falco peregrinus*), *J Am Vet Med Assoc* 238(11):1459–1463, 2011.

Mans C, Brown CJ: Radiographic evidence of atherosclerosis of the descending aorta in a grey-cheeked parakeet (*Brotogeris pyrrhopterus*), *J Avian Med Surg* 21(1):56–62, 2007.

Martinez LA, Jeffrey JS, Odom TW: Electrocardiographic diagnosis of cardiomyopathies in Aves, *Poult Avian Biol Rev* 8(1):9–20, 1997.
Martinez-Lemus LA, Miller MW, Jeffrey JS, et al: Echocardiography evaluation of cardiac structure and function in broiler and leghorn chickens, *Poult Sci* 77(7):1045–1050, 1998.
Nap AM, Lumeij JT, Stokhof AA: Electrocardiogram of the African grey (*Psittacus erithacus*) and Amazon (*Amazona* spp.) parrot, *Avian Pathol* 21(1):45–53, 1992.
Odom TW, Hargis BM, Lopez CC, et al: Use of electrocardiographic analysis for investigation of ascites syndrome in broiler chickens, *Avian Dis* 35(4):738–744, 1991.
Oglesbee BL, Oglesbee MJ: Results of post-mortem examination of psittacine birds with cardiac disease, 26 Cases (1991-1995), *J Am Vet Med Assoc* 212(11):1737–1742, 1998.
Oglesbee BL, Hamlin RL, Klingaman H, et al: Electrocardiographic reference values for macaws (*Ara* species) and cockatoos (*Cacatua* Species), *J Avian Med Surg* 15(1):17–22, 2001.
Pees M, Straub J, Krautwald-Junghanns M-E: Echocardiographic examinations of 60 African grey parrots and 30 other psittacine birds, *Vet Rec* 155(3):73–76, 2004.
Pees M, Krautwald-Junghanns ME: Avian echocardiography, *Semin Avian Exot Pet Med* 14(1):14–21, 2005.
Pees M, Kuhrig K, Demiraij F, et al: Bioavailability and compatibility of enalapril in birds. *Proceedings of the Annual Association of Avian Veterinarians*, pp 7–11, 2006a.
Pees M, Krautwald-Junghanns M-E, Straub J: Evaluating and treating the cardiovascular system. In Harrison GJ, Lightfoot TLC, editors: *Clinical avian medicine*, Palm Beach, FL, 2006c, Spix Publishing.
Phalen DN, Hays HB, Filippich LJ, et al: Heart failure in a macaw with atherosclerosis of the aorta and brachiocephalic arteries, *J Am Vet Med Assoc* 209(8):1435–1440, 1996.
Pilny AA, et al: Evaluation of *Chlamydophila psittaci* infection and ther risk factors for atherosclerosis in pet psittacine birds, *J Am Vet Med Assoc* 240(12):1474–1480, 2012.
Reavill DR, Dorrestein GM: Pathology of aging psittacines, *Vet Clin North Am Exot Anim Pract* 13(1):135–150, 2010.
Rembert MS, Smith JA, Strickland KN, et al: Intermittent bradyarrhythmia in a Hispaniolan Amazon parrot (*Amazona ventralis*), *J Avian Med Surg* 22(1):31–40, 2008.
Rosenthal K, Miller M: Cardiac disease. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.
Ross R, Glomset J, Harker L: Response to injury and atherogenesis, *Am J Pathol* 86(3):675–684, 1977.
Sanchez-Migallon Guzman D, Beaufrère H, Kukanich B, et al: Pharmacokinetics of a single oral dose of pimobendan in Hispaniolan Amazon parrot (*Amazona ventralis*), *J Avian Med Surg* 28(2):95–101, 2014.
Schnellbacher RW, da Cunha AF, Beaufrère H, et al: Effects of dopamine and dobutamine on isoflurane-induced hypotension in Hispaniolan Amazon parrots (*Amazona ventralis*), *Am J Vet Res* 73(7):952–958, 2012.
Schroeder NA: Diuretics. In Ettinger SJ, Feldman EC, editors: *Textbook of veterinary internal medicine*, ed 7, St Louis, MO, 2010, Saunders Elsevier.
Sedacca CD, Campbell TW, Janice MB, et al: Chronic cor pulmonale secondary to pulmonary atherosclerosis in an African grey parrot, *J Am Vet Med Assoc* 234(8):1055–1059, 2009.
Shrubsole-Cockwill A, Wojnarowicz C, Parker D: Atherosclerosis and ischemic cardiomyopathy in a captive, adult red-tailed hawk (*Buteo jamaicensis*), *Avian Dis* 52(3):537–539, 2008.
Simone-Freilicher E: Use of isoxsuprine for treatment of clinical signs associated with presumptive atherosclerosis in a yellow-naped Amazon parrot (*Amazona ochrocephala auropalliata*), *J Avian Med Surg* 21(3):215–219, 2007.
Speckmann EW, Ringer RK: The cardiac output and carotid and tibial blood pressure of the turkey, *Can J Biochem Physiol* 41(11):2337–2341, 1963.
St Leger J: Avian atherosclerosis. In Fowler ME, Miller RE, editors: *Wild animal medicine: current therapy*, ed 6, Philadelphia, PA, 2007, WB Saunders.
Straub J, Pees M, Krautwald-Junghanns M-E: Measurement of the cardiac silhouette in psittacines, *J Am Vet Med Assoc* 221(1):76–79, 2002.
Straub J: Effect of handling-induced stress on the results of spectral doppler echocardiography in falcons, *Res Vet Sci* 74(2):119–122, 2003.
Straub J, Pees M, Enders F, et al: Pericardiocentesis and the use of enalapril in a Fischer's lovebird (*Agapornis fischeri*), *Vet Rec* 152:24–26, 2003.
Straub J, Forbes NA, Pees M, et al: Pulsed-wave doppler-derived velocity of diastolic ventricular inflow and systolic aortic outflow in raptors, *Vet Rec* 154(5):145–147, 2004.
Sturkie PD: Heart: contraction, conduction, and electrocardiography. In Sturkie PD, editor: *Avian physiology*, ed 3, New York, 1976, Springer Verlag.
Thomas WP, Gaber CE, Jacobs GJ, et al: Recommendations for standards in transthoracic two-dimensional echocardiography in the dog and cat. Echocardiography Committee of the Specialty of Cardiology, American College of Veterinary Internal Medicine, *J Vet Intern Med* 7(4):247–252, 1993.
Touzot-Jourde G, Hernandez-Divers SJ, Trim CM: Cardiopulmonary effects of controlled versus spontaneous ventilation in pigeons anesthetized for coelioscopy, *J Am Vet Med Assoc* 227(9):1424–1428, 2005.
Van Zeeland YRA, Schoemaker NJ, Lumeij JT: Syncopes associated with second degree atrioventricular block in a cockatoo. *Proceedings of the Annual Conferenceof the Association of Avian Veterinarians*, pp 345–346, 2010.
Vink-Nooteboom M, Schoemaker NJ, Kik MJL, et al: Clinical diagnosis of aneurysm of the right coronary artery in a white cockatoo (*Cacatua alba*), *J Small Anim Pract* 39(11):533–537, 1998.
Westerhof I, Van de Wal M, Lumeij JT: Electrocardiographic changes in a galah (*Eolophus roseicapilla*) with lead poisoning. *Proceedings of the European Association of Avian Veterinarians Conference*, pp 59–60, 2011.
Wilson RC, Zenoble RD, Horton CR, et al: Single dose digoxin pharmacokinetics in the quaker conure (*Myiopsitta monachus*), *J Zoo Wildl Med* 20(4):432–434, 1989.
Zandvliet MJ: Electrocardiography in psittacine birds and ferrets, *Semin Avian Exot Pet Med* 14(1):34–51, 2005.
Zehnder AM, Hawkins MG, Pascoe PJ, et al: Evaluation of indirect blood pressure monitoring in awake and anesthetized red-tailed hawks (*Buteo jamaicensis*), effects of cuff size, cuff placement, and monitoring equipment, *Vet Anaesthes Analges* 36(5):464–479, 2009.

DISORDERS OF THE ENDOCRINE SYSTEM

Miguel D. Saggese

The endocrine system in birds includes the thyroid, parathyroid, adrenal, and ultimobranchial glands; hypophysis; pancreas; and gonads. Although the pineal gland is an endocrine organ responsible for the regulation of the diurnal cycle, temperature, and circadian locomotor activity (Ellis, 1976), there appears to be little recorded data on pathologic disorders. However, there are at least two records of neoplasms in this organ: a pineoblastoma in a cockatiel (Wilson *et al.*, 1988) and a pinealoma in a dove (Reece, 1992).

The hormones secreted by the endocrine glands are released directly into the bloodstream, reaching all parts of the body. This way they are able to stimulate or inhibit the functions of a wide range of other organs. Even small concentrations of a hormone can have a profound effect on body processes, influencing or causing pathologic changes in tissue far removed from its source. This must be remembered when attempting a clinical diagnosis of endocrine disorders and when hormones are used in the prevention or treatment of certain diseases.

There are several outstanding contributions discussing the anatomy, physiology, and pathology of the endocrine system in birds (Kronberger, 1973; Arnall and Keymer, 1975; Ebert, 1978; Epple and Stetson, 1980; Shöne and Arnold, 1980; Petrak and Gilmore, 1982; Gabrisch and Zwart, 1984; King and McLelland, 1984; Follett and Goldsmith, 1985; Leach, 1992; Joyner, 1994; Latimer, 1994; Lumeij,

1994; Lothrop, 1996; Oglesbee *et al.*, 1997). Since the last edition of this book, new comprehensive reviews have considerably expanded our knowledge and understanding of the avian endocrine system (Schmidt *et al.* 2003; de Matos 2008a,b; Pilny 2008; Ritchie and Pilny 2008; Schmidt and Reavill 2008, 2014). However, most of our knowledge of the avian endocrine system comes from the domestic chicken (*Gallus gallus*), and reports on disorders of the endocrine glands in companion birds continue to be limited to pathologic findings on postmortem examination. Clinical signs, antemortem diagnoses, and treatment of these disorders still are rarely reported in the scientific literature. They usually consist of case reports or a series of cases. Thus limited advances have been made in our understanding of the companion bird endocrine system since the previous edition of this book.

The prevalence of pathologic findings of the endocrine glands of birds submitted for necropsy suggest that most, if not all, of these disorders still remain a diagnostic challenge in live birds. In this section we discuss the anatomy, physiology, and clinical aspects of endocrine disorders in companion birds. The effect of heavy metals, polybrominated diphenyl ethers, dioxins, and other chemical disruptors that are known to interfere with the normal functioning of the endocrine glands, causing deleterious effects on several avian body systems, especially in free-ranging birds, are not discussed here as they are beyond the scope of this volume. Several review articles have been published recently on this subject (Ottinger *et al.*, 2008, 2009; Kumar and Holt, 2014).

We strongly encourage clinicians to investigate underlying endocrine disorders behind some clinical syndromes observed in their avian patients and publish these results. Hormone testing and hormone stimulation tests have become more available as many universities and veterinary diagnostic laboratories now provide these services. Most of these tests still need to be validated for the wide array of companion birds practitioners work with on a daily basis, creating numerous opportunities for potential research collaborations.

THYROID GLANDS

Unlike mammals, birds have two thyroid glands. They are situated at the base of the neck on either side of the trachea near the syrinx and jugular veins and are pinkish red or yellowish in color. Their size varies according to the species, age, and seasonal changes. The thyroid glands influence the growth rate of the young and govern the metabolic rate of all age groups. They increase in size when demands are greatest, for example, during cold weather and egg laying. The normal thyroid weights are approximately 0.02% of the total bodyweight. In a budgerigar weighing 35 g, each thyroid weighs approximately 3 mg and measures about 2 mm in length and 1 mm in width (Blackmore and Cooper, 1982). The left thyroid is usually slightly larger than the right. The normal thyroids of small passerine birds, such as the canary (*Serinus canaria*), are rounded and pinhead in size. The size of the thyroid glands is also influenced by a number of factors, including sex, age, diet, environmental factors, and secretory activity (King and McLelland, 1984).

In response to thyroid stimulating hormone (TSH) released by the pituitary gland, the thyroids secrete 3,5,3′-triiodo L-thyronine (T_3) and 3,5,3′,5′- tetraiodo L-thyronine (thyroxine, T_4). These hormones can be produced only when there is sufficient iodine and tyrosine available to the body. The secretion of TSH, T_3, and T_4 is influenced by the season, photoperiod, gonadal cycle, and state of molting (Zenoble *et al.*, 1985). In contrast to that in mammals, the avian thyroid produces more T_4 than T_3 (Astier, 1980). For a useful and comprehensive review of the anatomy, physiology, and physiopathology of the avian thyroid gland the reader is referred to the work by Merryman and Buckles (1998a,b), Schmidt *et al.* (2003), Schmidt and Reavill (2008), and Ritchie and Pilny (2008). Common disorders of the thyroids are listed in Table 13-12.

TABLE 13-12 Disorders of the Thyroid Gland

Disorder	Species
Goiter	
Hypothyroidism (thyroid hyperplasia and dysplasia).	Budgerigars, cockatiels, canaries, pigeons;[1-3] white backed vulture, southern caracara[4]
Dietary iodine deficiency	
Hyperthyroidism (thyrotoxicosis).	
Excess dietary iodine or iodine supplementation	Not reported in nondomesticated species
Neoplasia	
Carcinoma	Budgerigar,[5] dwarf sulfur-crested cockatoo, European pochard[6]
Adenocarcinoma	Budgerigar, house sparrow,[7] Andean goose, Laysan teal,[4] bald eagle[8]
Cystadenocarcinoma	Saker falcon[9]
Follicular cystadenoma	Caracara[10]
Cystic fibroadenoma	Black-chested buzzard[11]
Adenoma	Budgerigar,[2,5,6] jackdaw,[6] canary[6]
Neoplasia (unspecified)	Budgerigar, cockatiel,[14] scarlet macaw[13]
Other Disorders	
Thyroiditis associated with septicemia	Budgerigar, various species,[2,14] red-vented cockatoo[15]
Amyloidosis	Various species[13]
Cyst formation	Budgerigar,[2] mandarin duck, Elliot's pheasant, Eleonora's falcon[12]
Atrophy and hypertrophy	Guillemots[16]
Hemiagenesis	Japanese quail[17]

[1]Blackmore DK: *Vet Rec* 75:1068–1072, 1965.
[2]Blackmore DK, Cooper JE: Diseases of the endocrine system. In Petrak ML, editor: *Diseases of cage and aviary birds*, ed 2, Philadelphia, PA, 1982, Lea & Febiger.
[3]Sasipreeyajan J, Newman JA: *Avian Dis* 32:169–172, 1988.
[4]Griner LA: *Pathology of Zoo Animals*, San Diego, CA, 1983, Zoological Society of San Diego.
[5]Beach JE: *Vet Rec* 74:10–14, 63–68, 134–140, 1962.
[6]Wadsworth PF, Jones DM, Pugsley SL: *J Zoo Anim Med* 16:73–80, 1985.
[7]Petrak ML, Gilmore CE: Neoplasms. In Petrak ML, editor: *Diseases of cage and aviary birds*, Philadelphia, PA, 1982, Lea & Febiger.
[8]Bates G, Tucker RL, Ford S, et al: *J Zoo Wildl Med* 30:439–442, 1999.
[9]Samour JH, Naldo JL, Wernery U, Kinne J: *Vet Rec* 149:277–278, 2001.
[10]Forbes NA, Cooper JE, Higgins RJ: Neoplasms of birds of prey. In Lumeij JT, Remple JD, Redig PT et al, editors: *Raptor biomedicine III*, Lake Worth, FL, 2000, Zoological Education Network.
[11]Hammmerton AE: *Proceedings of the Zoological Society of London*, 112:149, 1943.
[12]Wadsworth PF, Jones DM: *Avian Pathol* 8:279–284, 1979.
[13]Schlumberger HG: *Ohio J Sci* 55:23–43, 1955.
[14]Von Zipper J, Tamasohke CH: Pathologische Schilddrüsenbefunde bei Vögeln. In *Diseases of Zoo Animals, 14th International Symposium*, Wroclaw, Poland, 1972. Akademie Verlag, Berlin, Germany, pp 113–122.
[15]Richkind M, Gendron AP, Howard EB, et al: *Vet Med Small Anim Clin* 77:1548–1554, 1982.
[16]Jefferies DJ, Parslow JLF: *Environ Pollut* 10:293–311, 1976.
[17]Wight PAL: *J Comp Pathol* 96:235–236, 1986.

Diagnosis of thyroid diseases is frequently a necropsy and/or a histopathologic finding. Neoplasia of the thyroid gland is common in old birds, potentially causing dyspnea and/or dysphagia from stenosis of the trachea and esophagus, respectively, caused by the enlargement of this gland. Recently Shimonohara *et al.* (2013) reported a high incidence of tumors, including thyroid adenomas, in advanced age pigeons maintained in a research facility. However, another pathologic survey found thyroid hyperplasia in 30 birds (0.024%) from a total of 12,500 cases examined (Schmidt and Reavill, 2002), suggesting neoplasia of this gland is relatively uncommon.

Goiter, associated with hypothyroidism, is the most common clinical disorder of the thyroid gland in companion birds. History could expose the presence of iodine binding elements in the bird's diet or iodine deficiency. Physical examination can reveal changes in size of the thyroid gland. Any birds showing a detectable enlargement around the neck area should be investigated for a possible hyperplasia, neoplasia, inflammation, or hypertrophy of the thyroid gland. Fine-needle aspiration biopsy and cytology may help elucidate the source and etiology of this enlargement. A hyperplasic, neoplastic, and hypertrophied thyroid gland can be identified in dorsoventral and lateral radiographs. Ultrasound, magnetic resonance imaging (MRI), and/or computed axial tomography can identify less obvious enlargements of the thyroid, which occur in the initial phases of certain tumors or in goiter.

Nonspecific signs of hypothyroidism include growth retardation in young animals, molting problems and feather loss, changes in behavior, weight loss, lethargy, and weakness. A disorder of the thyroid should be considered in the differential diagnosis of these clinical presentations and syndromes. Reduced fertility can be a result of thyroid disorders, as the thyroid hormones have a significant role in the normal functioning of the reproductive tract, including ovarian development and development of the ovarian follicles.

Total iodine levels can be measured in biopsy samples of the thyroid (Loukopoulos *et al.*, 2015). Total and free thyroxine can be measured in birds by radioimmunoassay, and reference values have been reported for some species of parrots, pigeons, and other companion birds (Greenacre *et al.*, 2001; Brandao *et al.*, 2012). Comparison of the patient's thyroid hormone blood concentration with those of clinically healthy specimens of the same species or genus can overcome the lack of reference values and facilitate the clinical diagnosis of hypothyroidism. However, most ancillary diagnostic tests used for the diagnosis of thyroid disorders in domestic animals have not been validated for birds and, when used, they should be interpreted with caution. The use of a TSH stimulation test has been recommended in avian patients (Schmidt and Reavill, 2008), but the interpretation of these results deserves further investigation on its value for the diagnosis of hypothyroidism.

Medical management of hypothyroidism and goiter in companion birds can be achieved by empirical supplementation of thyroid hormones and iodine, respectively. Practitioners are encouraged to exhaust all of the available diagnostic modalities before implementing such empirical treatments in their patients and use them cautiously and with adequate close monitoring of their patient's response to therapy.

PARATHYROID GLANDS

These are two to four very small yellow organs situated close to the posterior pole of each thyroid. They play an important role in calcium metabolism. The parathyroids secrete parathyroid hormone (PTH) in response to a decrease in the concentration of plasma calcium. PTH increases the plasma calcium concentration by increasing tubular reabsorption of calcium by increasing bone resorption and calcium absorption from the gastrointestinal system. PTH is also involved in the synthesis of 1,25-dihydroxyvitamin D_3 and increasing the excretion of phosphorus through the renal system. Disorders of the parathyroids are listed in Table 13-13.

TABLE 13-13 Disorders of the Parathyroid Glands

Disorder	Species
Nutritional secondary hyperparathyroidism Dietary insufficiency of calcium, excess phosphorus, and combination of both leading to hypocalcemia	Psittacines[1], birds of prey[2]

[1]Altman RB: Disorders of the skeletal system. In Petrak ML, editor: *Diseases of cage and aviary birds*, ed 2, Philadelphia, PA, 1982, Lea & Febiger.
[2]Cooper JE: *Veterinary aspects of captive birds of prey*, ed 2, with 1985 supplement, Saul, Gloucestershire, UK, 1985, Standfast Press.

When egg laying commences, there is a dramatic increase in the demand of calcium for eggshell production. In the domestic fowl, the shell gland in the oviduct secretes approximately 5 g of calcium carbonate in 20 h. This necessitates the withdrawal of 2 g of calcium ions from the blood each day at a rate of 100 mg/h, and the calcium is resorbed from the bones. This procedure, however, cannot continue indefinitely because the bones would soften and fracture. Therefore extra calcium has to be absorbed from food. Because the turnover of calcium is high, birds have developed a special type of medullary bone in the marrow of the long bones. At times when requirements for eggshell calcium are low, calcium ions in the blood are transferred to the medullary bone. When calcium is required, PTH mobilizes this calcium, thus avoiding its withdrawal from the structural bones.

PTH also plays a role in the contraction of muscles, in blood clotting, and in the secretion of phosphates by the tubules of the kidneys. It is believed that the parathyroid is not under the control of any other endocrine organ, but is stimulated by the level of calcium in the blood. The level of minerals in the blood of healthy birds is constant, but in certain diseases it may fall markedly and result in softening of the bones (Epple and Stetson, 1980).

Most of the knowledge about this gland and its role in calcium metabolism comes from the chicken (de Matos, 2008a). Limited information exists for companion birds and disorders of this gland are rarely reported. Given the role of the parathyroid glands in calcium homeostasis, clinical signs associated with disorders of this gland are the result of abnormal calcium metabolism and its effects on the neuromuscular and osteoarticular systems and the female reproductive tract (de Matos, 2008a). Spontaneous fractures, seizures, muscle tremors, egg-binding, abnormal eggshells, radiographic evidence of decreased mineralization of bones, metastatic calcification, and changes in ionized calcium levels in blood are usually associated with abnormal calcium metabolism and increased PTH blood levels.

The most common disorder of this gland is parathyroid hyperplasia associated with nutritional secondary hyperparathyroidism (de Matos, 2008a). African grey (*Psittacus erithacus*) and other species of parrots are commonly affected as result of poor nutritional practices. Seizures associated with hypocalcemia are common in African grey parrots.

Levels of PTH can be elevated even with normal ionized calcium blood levels in birds with nutritional secondary hyperparathyroidism and as a result of diets deficient in calcium and vitamin D. Primary hyperparathyroidism has been reported and should be suspected in birds with very high ionized calcium blood levels and adequate calcium and phosphorus diet intakes. Unfortunately, determination of PTH levels is rarely pursued because these tests are not validated for birds and are not commercially available.

Other primary disorders of the parathyroid gland include neoplasia. Systemic inflammation and infections affecting this gland are rare. A detailed discussion of calcium homeostasis and disorders is presented elsewhere in this book, Chapter 3 and by de Matos (2008a).

ADRENAL GLANDS

These are paired structures situated near the anterior pole of each kidney, but in a few species, for example, rheas (Rheidae), bald eagles (*Haliaeetus leucocephalus*), and others, the glands are fused into a single organ. The adrenals vary considerably in shape and size in different species and are directly related to bodyweight. They are just visible to the naked eye in the smaller passerines as pink or creamy gray specks. In some species they have a yellowish tinge. Each gland is composed of two main sections as in mammals, but in birds the tissues are not demarcated into an outer cortex and inner medulla, as the two types are mixed.

The medullary or chromaffin tissue is composed of two types of cells. One type releases epinephrine and the second type releases norepinephrine. Epinephrine is involved in the process of glycogenolysis and norepinephrine is involved in gluconeogenesis. The cortical tissue is responsible for the secretion of corticosterone, aldosterone, and 18-hydroxycorticosterone. The adrenal glands in birds secrete more corticosterone than aldosterone. Cortisol is not a significant adrenal steroid produced in birds, in contrast to mammals. Moreover, corticosterone in birds has both glucocorticoid and mineralocorticoid activities. The level of corticosterone in the blood plasma is increased under certain stressful conditions such as excessive cold, deprivation of water, surgical procedures, excessive handling, or administration of certain drugs. It plays an important role in carbohydrate, fat, and electrolyte metabolism. In addition, in marine birds this hormone appears to increase the secretion of sodium chloride by the nasal glands. When the intake of salt is excessive, the level of this hormone in the blood is dramatically increased. Aldosterone has the opposite effect in salt metabolism, because it acts on the kidney tubules and increases sodium reabsorption from the filtrate passing through the glomeruli in the renal cortex. Corticosterone levels in blood also increase during oviposition and follow a diurnal rhythm. The adrenal cortex is influenced by the adrenocorticotropic hormone (ACTH) secreted by the anterior lobe of the pituitary gland (Epple and Stetson, 1980).

In free-ranging birds adrenal steroids, especially corticosterone, are usually measured in feathers, blood, urine, and/or feces as indicators of well-being and nutritional, toxicologic, and environmental stress (Möstl *et al.*, 2005; Lattin *et al.*, 2014). Rarely are these studies conducted in captive pet birds.

Disorders of the adrenals have been summarized by Schmidt *et al.* (2003) and more recently by de Matos (2008b). Common pathologies of this organ are listed in Table 13-14.

Avian hyperadrenocorticism has been rarely reported in the medical avian literature. Primary and secondary hyperadrenocorticism can occur in companion birds. Cornelissen and Verhofstad (1999) diagnosed hyperadrenocorticism in a greater than 30-year-old scarlet macaw (*Ara macao*). Postmortem examination revealed an adrenal adenocarcinoma as the etiology. Pituitary-dependent hyperadrenocorticism has been reported in a 13-year-old female Moluccan cockatoo (*Cacatua moluccensis*; Starkey *et al.*, 2008). The cockatoo exhibited feather loss, polyuria, polydipsia, and abdominal hernia.

Signs of hyperadrenocorticism include polyuria/polydipsia, weight changes, hyporexia or anorexia, feather loss, dermatitis, collapse, weakness, delayed wound healing, muscle atrophy, and hyperglycemia. In hyperadrenocorticism caused by a pituitary tumor, blindness and other neurologic signs can also be present as the pituitary grows and compresses surrounding structures. Chronic infectious diseases, such as mycobacteriosis or aspergillosis, may be the result of immunosuppression from the continuous release of endogenous steroids by the adrenal gland. Changes in the hemogram and clinical biochemistry are not pathognomonic. Enlarged adrenal glands (hypertrophy or neoplasia) may be detected in radiographs and other ancillary imaging techniques such as ultrasound , computed axial tomography, and MRI and observed during laparoscopic investigation. Unilateral enlargement is usually associated with a neoplastic gland and bilateral enlargement is more likely caused by a pituitary ACTH-secreting tumor. The combination of clinical signs, adrenomegaly, and an increase in the measurement of corticosterone blood levels strongly supports a diagnosis of hyperadrenocorticism in the live bird.

The use of ACTH stimulation and dexamethasone suppression tests have been reported in birds, although variable results and lack of validation for different species precludes their use at this time in avian practice and requires more research.

Pathologic reports of adrenal glands affected by degenerative, inflammatory, and neoplastic changes suggest that hypoadrenocorticism (Addison's disease) and other clinical forms of hypoadrenalism may occur in birds but probably remain underdiagnosed. Treatment of confirmed adrenal gland disorders have not yet been reported in companion birds.

TABLE 13-14 Disorders of the Adrenal Glands

Disorder	Species
Chronic recurrent disorders, associated in psittacines with such diseases as inclusion body hepatitis	Psittacines, especially cockatoos and African grey parrots
Chronic disorders associated with long-standing infections such as aspergillosis, and mycobacteriosis, or permanent damage caused by a variety of factors	
Neoplasia	
Adenomas, cortical hyperplasia	Budgerigar,[1,2] white-crowned parrot[3]
Carcinoma (unspecified, adrenal cortical cells)	Little owl,[2] pigeon[4]
Neoplasia of adrenal and ovary with metastases	Nicobar pigeon[3]
Adrenal cortical carcinoma	Long-crested eagle[5]
Adrenal adenocarcinoma	Scarlet macaw[6]
Adrenal carcinoma	Budgerigar[7]

[1]Beach JE: *Vet Rec* 74:10–14, 63–68, 134–140, 1962.
[2]Blackmore DK, Cooper JE: Diseases of the endocrine system. In Petrak ML, editor: *Diseases of cage and aviary birds*, ed 2, Philadelphia, PA, 1982, Lea & Febiger.
[3]Griner LA: *Pathology of Zoo Animals*, San Diego, CA, 1983, Zoological Society of San Diego.
[4]Gratzl E, Köhler H: *Spezielle Pathologie and Therapie der Geflügelkrankheiten*, Stuttgart, Germany, 1968, Ferdinand Enke Gratzl and Köhler, 1968.
[5]Halliwell WH, Graham DL: Neoplasms in birds of prey. In Fowler ME, editor: *Zoo and wild animal medicine*, Philadelphia, PA, 1978, WB Saunders.
[6]Cornelissen H, Verhofstad A: *J Avian Med Surg* 13:92–97, 1998.
[7]Latimer KS, Greenacre CB: *J Avian Med Surg* 9:141–143, 1995.
IBH, inclusion body hepatitis.

ULTIMOBRANCHIAL GLANDS

These are small, pink lentil-shaped glands situated at the entrance of the thoracic inlet, craniolaterally to the origin of the carotid arteries. They are about 2 to 3 mm in size in the adult domestic fowl. The ultimobranchial glands are integrated by the C cells, parathyroid nodules, vesicles, and lymphoid tissue (King and McLelland, 1984). The most important function of these glands is the secretion of calcitonin by the C cells. The exact role of calcitonin in birds is not fully understood but does not appear to have an evident role in calcium metabolism, as it does in mammals. Clinical manifestations of disorders of the ultimobranchial glands have not been reported in birds.

HYPOPHYSIS

The hypophysis or pituitary is a small gland linked to the ventral aspect of the diencephalic brainstem. This gland is integrated by the anterior lobe or adenohypophysis derived from the embryonic stomodeum and the posterior lobe or neurohypophysis derived from the diencephalon. The adenohypophysis is divided into the pars tuberalis and the pars distalis. Unlike mammals, the pars intermedia is not a separate entity, and the equivalent cells of this are integrated within the pars distalis. The neurohypophysis is in direct communication with the brain, and it is composed of the median eminence, the infundibulum, and the neural lobe (King and McLelland, 1984).

The adenohypophysis produces seven hormones that are mostly named after the ductless glands that each controls. ACTH maintains adrenal gland activity and controls the production of adrenal corticosteroids. TSH, as its name implies, controls the activity of the thyroid gland and the secretion of thyroid hormones. Follicle-stimulating hormone (FSH) stimulates the gonads in both sexes, as does luteinizing hormone (LH). Prolactin, or lactogenic hormone, suppresses FSH and appears to regulate the deposition of fat in birds before migration. It also causes broodiness and, in those species that have them, stimulates the production of brood patches. In pigeons this hormone also stimulates the production of "crop milk." The somatotropic hormones or growth hormones appear to regulate growth in immature birds. Finally, the function of the so-called melanocyte-stimulating hormone is not clearly understood.

The neurohypophysis receives and stores two hormones, arginine vasotocin and mesotocin. These are produced by the hypothalamic nuclei and transported by the hypothalamo-hypophyseal tract. Vasotocin is an antidiuretic hormone that differs slightly from the mammalian counterpart by one amino acid residue. Its main action in birds is to inhibit fluid loss from the kidneys by decreasing glomerular filtration. The other known actions of vasotocin include the control of water reabsorption and the stimulation of peristaltic movements of the oviduct during oviposition (Epple and Stetson, 1980).

Neoplasms of the pituitary gland are relatively common in companion birds, particularly in budgerigars and other parrots (Dezfoulian *et al.*, 2011; Langohr *et al.*, 2012, Table 13-15). As seen in mammals, clinical signs depend on the localization of the neoplasia (adenohypophysis/neurohypophysis), cell type (adenoma/carcinoma), growth rate, and if they are secreting or nonsecreting tumors. Nevertheless, most neoplasms of the pituitary gland are diagnosed during postmortem examination. In live birds, a sign of a tumor may not be present until the tumor grows enough to enlarge the size of the gland causing compression of surrounding structures. Visual deficit, exophthalmia, behavioral changes, altered mental status (depression), and neurologic signs (ataxia, seizures, head-tilt, and opisthotonus) alone or combined are clinical manifestations of pituitary tumors. Changes in body condition, anorexia, feather loss, reproductive disorders, and signs of hyperadrenocorticism can be observed in functional secreting tumors of this gland.

TABLE 13-15 Disorders of the Hypophysis Gland

Disorder	Species
Neoplasia and hyperplasia	Budgerigar,[1-6] cockatiel[7,8]
Adenoma	Yellow-naped Amazon parrot[9]

[1]Schlumberger HG: *Cancer Res* 14:237–245, 1954.
[2]Schlumberger HG: *Cancer Res* 17:823–832, 1957.
[3]Beach JE: *Vet Rec* 74:10–14, 63–68, 134–140, 1962.
[4]Blackmore DK, Cooper JE: Diseases of the endocrine system. In Petrak ML, editor: *Diseases of cage and aviary birds*, ed 2, Philadelphia, PA, 1982, Lea & Febiger.
[5]Petrak ML, Gilmore CE: Neoplasms. In Petrak ML, editor: *Diseases of cage and aviary birds*, Philadelphia, PA, 1982, Lea & Febiger.
[6]Bauck LA: Pituitary neoplastic disease in 9 budgerigars. *Proceedings of the Association of Avian Veterinarians*, Hawaii, pp 87–90, 1987.
[7]Curtis-Velasco M: *J Assoc Avian Vet* 6:21–22, 1992.
[8]Wheler C: *J Assoc Avian Vet* 6 (2):92, 1992.
[9]Romagnano A, Mashima TY, Barnes HJ, et al: *J Avian Med Surg* 9:263–270, 1995.

MRI and computed axial tomography examination of the brain and hypophysis should be included in the physical and neurologic investigation of patients presenting compatible signs with a pituitary neoplasm, especially when other causes of neurologic disorders and blindness haven been previously ruled out. Treatment of pituitary tumors and hyperplasia has not been reported in companion birds.

PANCREAS

The pancreas is situated at the first duodenal loop and is integrated in the majority of birds by the dorsal, ventral, and splenic lobes. This organ has both endocrine and exocrine functions but only the islets of Langerhans, which contain endocrine cells, are dealt with in this section.

In birds, in contrast to mammals, there are three types of islet. The dark islets contain alpha (α) and delta (δ) cells and the lighter islets contain beta (β) and δ cells. Mixed islets contain all three cell types. One type of a cell in the dark islets produces glucagon, which is a hormone that in birds plays a more important role than insulin in carbohydrate metabolism by raising the plasma glucose. It also plays an important role in fat metabolism. The β cells produce the well-known hormone insulin, but the levels of this hormone in the avian pancreas are considerably lower than in mammals. Insulin also plays a minor role in carbohydrate metabolism, but its main role is as an anabolic hormone. In mammals insulin takes sugars out of the circulating blood and stores them in the liver in the form of glycogen or distributes them to the skeletal muscles and other organs, where they are temporarily stored for emergencies. When insulin is absent, as in diabetes, this leads to high levels of glucose in the blood. In birds, however, the role of insulin is much less clear. On occasions it seems that its role is reversed, so that the polypeptide hormone glucagon functions similarly to insulin in mammals. In some species of birds, however, it seems possible that insulin may help prevent hyperglycemia. The δ cells secrete somatostatin, a hormone with a possible role in regulating the secretion of insulin and glucagon (Epple and Stetson, 1980). For a comprehensive review of the physiology and different disorders affecting the endocrine pancreas, the reader is referred to the work of Lothrop (1996), Speer (2001), and Pilny (2008).

Neoplastic, inflammatory, and degenerative disorders can affect the exotic pancreas, as well the pancreatic islets (Schmidt *et al.*, 2003; Schmidt and Reavill, 2014; Table 13-16). However, until recently clinical manifestations of endocrine pancreatic diseases such as diabetes mellitus were considered to be quite rare in birds. Pilny (2008) provided the most current summary of diabetes mellitus in birds in which its physiopathology is not well understood. Both avian insulin and glucagon play an important role in regulating carbohydrate metabolism. The latter apparently is the main hormone involved in maintaining normal blood glucose levels. Birds normally have much higher normal blood glucose levels than their mammalian counterparts, with values that would be considered harmful and consistent with diabetes in dogs and humans.

As birds normally have a high fasting blood glucose level and insulin appears to play a relatively minor role in controlling carbohydrate metabolism, the definition of the disease in birds compared with that in mammals needs some modification. A definition modified from Stogdale (1986) is suggested as follows: "A metabolic disorder in which the ability to oxidize carbohydrates is more or less completely lost, usually due to faulty pancreatic activity, especially of the islets of Langerhans, and to consequent disturbance of the normal insulin and glucagon mechanism."

It has been suggested that, compared with mammals, diabetes mellitus may be associated with hepatic disorders as well, implying a potential role for the liver in the control of blood glucose levels. Appleby (1984) observed enlargement and pale brown discoloration of the liver. Both he and Altman and Kirmayer (1976) found hepatic necrosis and evidence of lipidosis in diabetic birds. The latter workers also recorded hepatic fibrosis, and in their four cases in budgerigars could detect no pancreatic lesions, unlike Appleby (1984), who found evidence of degenerative changes in the islets. More recently, Gancz *et al.* (2007) reported an adult, male chestnut-fronted macaw (*Ara severa*) and an adult, female military macaw (*A. militaris*) with diabetes mellitus after extensive clinical diagnostic laboratory investigation. In addition, hemosiderosis in hepatic macrophages and hepatocytes was diagnosed through biopsies in the two birds. The authors highlighted the need to perform further clinical investigations to ascertain a possible relationship between diabetes mellitus and hemosiderosis in large psittacines.

Diabetes mellitus has been reported for several species in the avian medical literature (Pilny, 2008; Schmidt and Reavill, 2014). A combination of persistent hyperglycemia combined with glycosuria (urine is negative for glucose in normoglycemic birds), polyuria, and polydipsia are common clinical signs consistent with diabetes mellitus. Additional findings in these patients may include polyphagia and initial obesity later followed by weight loss, weakness, dehydration, and even emaciation in the more advanced cases. Normal blood glucose levels vary among different species, but they are consistently in the range of 160 to 400 mg/dL, and rarely more. Reference blood glucose levels for common avian species are provided elsewhere in this book. Repeated glycemic values greater than 600 to 1000 mg/dL accompanied with the previously discussed clinical signs are consistent with a diagnosis of diabetes mellitus.

Treatment of diabetes mellitus in birds is considered empirical, as the metabolism of carbohydrates such as glucose is not clearly understood, less the pharmacologic effects of the hormones involved in their control. Limited information comes from practitioners following the same recommendations and guidelines described for mammalian counterparts in the form of case reports. Thus insulin therapy has been applied in several occasions to decrease blood glucose levels in diabetic birds. Results may vary as control of glycemia does not only depend on insulin, but to a large extent it depends on glucagon and even on somatostatin. Pilny (2008) and Gancz *et al.* (2005, 2007) summarized the empirical use of different protocols aimed at medically treating diabetes mellitus in birds. Caution is recommended when these treatments are implemented by avian practitioners.

Insulin therapy should be implemented taking the same precautions used for dogs and cats. Ideally, birds should be hospitalized and closely monitored during the first days of treatment. Blood glucose

TABLE 13-16 Disorders of the Pancreas

Disorder	Species
Diabetes mellitus	Budgerigar,[1,2] cockatiel,[1] racing pigeon,[3] canary, toco toucan,[4] Amazon parrots, macaws, cockatoos,[5] red-tailed hawk,[6] African grey parrot,[7] yellow-collared macaw, black-capped lory, yellow-naped amazon, parakeets[8]
Neoplasia (unspecified)	Budgerigar[9,10]
Islet cell tumor	Parakeet[10]
Adenocarcinoma	Peaceful dove[11]
Pancreatic atrophy	Budgerigar,[12,13] blue and gold macaw,[14] peregrine falcon[15]
Acute pancreatic necrosis	Galah cockatoo[16]
Exocrine pancreatic insufficiency	Yellow-naped amazon,[17] racing pigeon[18]
Pancreatic adenocarcinoma	Yellow-naped Amazon[17]
Pancreatic hypoplasia	Eclectus parrot[19]

[1]Altman RB, Kirmayer AH: *J Am Anim Hosp Assoc* 12:531–537, 1976.
[2]Appleby EC, Keymer IF: More tumours in captive wild mammals and birds. A second brief report. In *Sonderdruck aus Verhandlungsbericht des XIII Internationalen Symposiums über die Erkrankungen der Zootiere*, Helsinki. Berlin, Germany, 1971, Academie-Verlag.
[3]Murphy J: Diabetes in toucans. *Proceedings of the Association of Avian Veterinarians*, pp 165–170, 1992.
[4]Douglass EM: *Mod Vet Pract* 62:293–295, 1981.
[5]Woerpel RW, Rosskopf WJ: *Vet Clin North Am* 14:249–286, 1984.
[6]Waller-Pendleton E, Rogers D, Epple A: *Avian Pathol* 22:631–635. 1993.
[7]Candeletta SC, Homer BL, Garner MM, et al: *J Assoc Avian Vet* 7:39–43, 1993.
[8]Lothrop CD Jr: Diseases of the endocrine system. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Philadelphia, PA, 1996, Williams & Wilkins.
[9]Schlumberger HG: *Cancer Res* 17:823–832, 1957.
[10]Ryan CP, Walder EJ, Howard EB: *J Am Anim Hosp Assoc* 18:139–142, 1982.
[11]Griner LA: *Pathology of Zoo Animals*, San Diego, CA, 1983, Zoological Society of San Diego.
[12]Beach JE: *Vet Rec* 74:10–14, 63–68, 134–140, 1962.
[13]Hasholt J: *Nord Vet Med* 24:458–461, 1972.
[14]Quesenberry KE, Liu SK: *J Am Vet Med Assoc* 189:1107–1108, 1986.
[15]Samour and Naldo, 2002.
[16]Pass DA, Wylie SL, Forshaw D: *Aust Vet J* 63:340–341, 1986.
[17]Ritchey JW: *Vet Pathol* 34:55–57, 1997.
[18]Amman O, Visschers MJM, Dorrestein GM, et al: Exocrine pancreatic insufficiency in a racing pigeon (*Columba livia domestica*). *Proceedings of the European Association of Avian Veterinarians*, Arles, France, pp 179–183, 2005.
[19]Ritzman TK: Pancreatic hypoplasia in an eclectus parrot (*Eclectus roratus polychloros*). *Proceedings of the Association of Avian Veterinarians*, Portland, Oregon, pp 83–87, 2000.

levels should be monitored frequently to avoid hypoglycemic crisis while bringing this value to its normal reference range.

Precautions should be taken when considering treating a diabetic patient, as no recommended guidelines are currently available for the management of this avian disease. Further scientific research is needed in this field, because probably more birds will be diagnosed with diabetes mellitus in the future.

Decrease in the amounts of carbohydrates offered in the diet may empirically help reduce blood glucose levels, which occurs in mammals, although given the diverse range of avian species and their differences in feeding habits, this could be challenging. Diets that include high levels of fiber, fruits, and vegetables and low levels of carbohydrates, when feasible, may have a beneficial role in the diabetic avian patient.

OVARY

In most bird species only the left ovary is functional. One exception is the brown kiwi (*Apteryx australis*), in which it has been established that both ovaries are fully functional. Although there have been many reports of fully developed left and right ovaries in Falconiformes, it seems very unlikely that both ovaries could be fully functional (King and McLelland, 1984). The ovary produces three hormones: estrogen, progesterone, and an androgen. Both the ovary and the testes are influenced by the same pituitary hormones. The FSH stimulates the development of the ovarian follicles and secretion of estrogens by the ovary. Estrogens are steroid hormones involved in the development of the secondary female sex characteristics, such as change in beak color in some species; development of the incubation or brood patch by interaction with prolactin; development and maintenance of female-type plumage; and development of the oviduct by interaction with the steroid progesterone, although very little appears to be known about its functions. In addition, estrogens are partly responsible for nest-building behavior and the mobilization of calcium in the body for the production of eggshells. The latter is performed in conjunction with hormone production by the parathyroid glands. Estrogens also stimulate the widening of the pelvic outlet to enable the eggs to be passed before egg laying. The ovary, like the testes, decreases in size in winter, when the hours of daylight are short, and then enlarges again when the lengthening days of spring stimulate the hypothalamus and pituitary gland. Apparently very little is known about the action of the androgens produced by the ovary. LH secreted by the pituitary induces actual ovulation or shedding of the ova. As the ova enlarge, they form grape-like clusters, rupture from their follicles, and drop into the funnel-shaped opening or infundibulum of the oviduct (Epple and Stetson, 1980).

In many species of birds, the ovary is pale to dark yellow, which is related to lipid deposits. However, some species have a "melanistic" ovary from the presence of melanocytes. In these species the immature ovary tends to vary from black through dark green and gray to pale green in color. As the ovary develops and increases in size during the breeding season, the color also changes.

Congenital, inflammatory (infectious and noninfectious), toxic, and neoplastic disorders have been reported for the avian ovary (Schmidt *et al.*, 2003; Table 13-17). The most common clinical manifestation of ovarian diseases is a reduced or absent ability to lay eggs or decreased hatchability and exaggerated function of the ovary (chronic egg laying). Neoplasms of the ovary are common in companion birds such as budgerigars and other psittacines. These and other common disorders of the avian female reproductive tract are discussed elsewhere in this book.

TABLE 13-17 Disorders of the Ovary

Disorder	Species
Neoplasia[1]	Budgerigar,[2-8,21] various psittacine and passerine species[2,5,9], barred-shouldered dove[3]
Granulosa cell tumor	Budgerigar,[2,4,5,8,10,11] sulfur-crested cockatoo,[10] Chinese painted quail,[11] red-legged honeycreeper,[11] pigeon,[12] military macaw[13]
Adenocarcinoma	Australian thicknee[14]
Diffuse papillary adenocarcinoma	Crimson rosella[11]
Adenoma	Pet birds[15] (unspecified)
Polyostotic hyperostosis	Budgerigars[5,16-18]
Sex reversal and other reproductive disorders of endocrine origin	Various species, poultry[17,19]
Ovarian cysts of possible endocrine origin	Canaries, budgerigars, pheasants, and cockatiels[20]

[1]MrzeL L, Pogacnik M, Josipovic D: *Vet Glasnik* 33:989–993, 1979.
[2]Petrak ML, Gilmore CE: Neoplasms. In Petrak ML, editor: *Diseases of cage and aviary birds*, Philadelphia, PA, 1982, Lea & Febiger.
[3]Keymer IF: *Avian Pathol* 9:405–419, 1980.
[4]Frost C: *Vet Rec* 73:621–626, 1961.
[5]Beach JE: *Vet Rec* 74:10–14, 63–68, 134–140, 1962.
[6]Campbell TW: Neoplasia. In Harrison GJ, Harrison LR, editors: *Clinical avian medicine and surgery*, Philadelphia, PA, 1986, WB Saunders.
[7]Effron M, Griner L, Benirschke K: *J Natl Cancer Inst* 59:185–198, 1977.
[8]Reece RL: *Avian Pathol* 21:3–32, 1992.
[9]Neumann U, Kummerfeld N: *Avian Pathol* 12:353, 1983.
[10]Ratcliffe HL: *Am J Cancer* 17:116–135, 1933.
[11]Griner LA: *Pathology of Zoo Animals*, San Diego, CA, 1983, Zoological Society of San Diego.
[12]Chalmers GA: *Avian Dis* 30:241–244, 1986.
[13]Stoica G, Russo E, Hoffman JR: *Lab Anim* 18(5):17–20, 1989.
[14]Appleby EC, Keymer IF: More tumours in captive wild mammals and birds. A second brief report. In *Sonderdruck aus Verhandlungsbericht des XIII Internationalen Symposiums über die Erkrankungen der Zootiere*, Helsinki. Berlin, Germany, 1971, Academie-Verlag.
[15]Schmidt RE: Neoplastic diseases. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.
[16]Schlumberger HG: *Am J Pathol* 35:1–23, 1959.
[17]Arnall L, Keymer IF: The endocrine system. In Arnall L, Keymer IF, editors: *Bird diseases: an introduction to the study of birds in health and disease*, Neptune City, NJ, 1975, TFH Publications.
[18]Stauber E, Papageorges M, Sande R, et al: *J Am Vet Med Assoc* 196:939–940, 1990.
[19]Blount WP: *Diseases of poultry*, London, 1947, Baillière Tindall & Cox.
[20]Speer BL: Diseases of the urogenital system. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.
[21]Campbell TW, Stuart LD: *Vet Med Small Anim Clin* 79:215–218, 1984.

TESTES

The testes are paired, cylinder-shaped gonads located in most species at the base of the anterior lobe of each kidney. As a general rule, in immature birds, the left testis tends to be larger than the right. Conversely, as birds mature, the right gonad becomes larger and heavier than the left. The morphometrics of the testis increase considerably

TABLE 13-18 Disorders of the Testes

Disorders	Species
Neoplasia	
Seminoma	Budgerigar[1-8]
Sertoli cell tumor	Budgerigars,[1-3,9-11] Japanese quail[12]
Interstitial cell tumor	Budgerigar[1,3]
Leiomyosarcoma	Budgerigar[1]
Hemangioma	Budgerigar[1]

[1]Petrak ML, Gilmore CE: Neoplasms. In Petrak ML, editor: *Diseases of cage and aviary birds*, Philadelphia, PA, 1982, Lea & Febiger.
[2]Arnall L: *Vet Rec* 70:120–128, 1958.
[3]Beach JE: *Vet Rec* 74:10–14, 63–68, 134–140, 1962.
[4]Rewell RE: *J Pathol Bacteriol* 60:155, 1948.
[5]Lombard LS, Witte EJ: *Cancer Res* 19:127–141, 1959.
[6]Blackmore DK: *Vet Rec* 75:1068–1072, 1965.
[7]Griner LA: *Pathology of Zoo Animals*, San Diego, CA, 1983, Zoological Society of San Diego.
[8]Turk RJ, Kim J, Gallina AM: *Avian Dis* 25:752–755, 1981.
[9]Frost C: *Vet Rec* 73:621–626, 1961.
[10]Effron M, Griner L, Benirschke K: *J Natl Cancer Inst* 59:185–198, 1977.
[11]Reece RL: *Avian Pathol* 21:3–32, 1992.
[12]Gorham SL, Ottinger MA: *Avian Dis* 30:337–339, 1986.

during the breeding season. The color of the testes in many species is yellow, as a result of lipid deposits, whereas in other species the color varies from pale green through gray and dark green to black, because of the presence of melanocytes. The color of the testes in these species changes according to sexual activity and size of the gonad.

The testes produce testosterone in a small group of glandular cells situated between the sperm-producing tubules. It passes directly into the bloodstream and reaches all parts of the body. Testosterone generates and maintains the secondary sexual characteristics, such as the typical male head and body shape of a particular species, its posture, voice, sexual behavior, plumage, and color. It is also responsible for the increase in size of the fleshy wattles and cere of some species (King and McLelland, 1984).

The sperm and testosterone output depends on the pituitary hormones FSH and LH. FSH stimulates the growth of the testes and development of sperm and LH stimulates the interstitial glandular cells between the seminiferous tubules to produce testosterone (Epple and Stetson, 1980).

Disorders of the testes are listed in Table 13-18. Budgerigars and other species of psittacines appear to be more susceptible to testicular neoplasia. Feminization syndromes as result of functional Sertoli cell tumors have been described in these species. These and other common disorders of the male reproductive tract are discussed elsewhere in this book.

ACKNOWLEDGMENTS

The author would like to acknowledge the contribution of Dr. Ian Keymer and Dr. Jaime Samour to this section in the previous edition of this book.

REFERENCES

Altman RB: Disorders of the skeletal system. In Petrak ML, editor: *Diseases of cage and aviary birds*, ed 2, Philadelphia, PA, 1982, Lea & Febiger.
Altman RB, Kirmayer AH: Diabetes mellitus in the avian species, *J Am Anim Hosp Assoc* 12:531–537, 1976.
Amman O, Visschers MJM, Dorrestein GM, et al: Exocrine pancreatic insufficiency in a racing pigeon (*Columba livia domestica*). *Proceedings of the European Association of Avian Veterinarians,* Arles, France, pp 179–183, 2005.
Appleby EC, Keymer IF: More tumours in captive wild mammals and birds. A second brief report. In *Sonderdruck aus Verhandlungsbericht des XIII Internationalen Symposiums über die Erkrankungen der Zootiere, Helsinki*, Berlin, Germany, 1971, Academie-Verlag.
Appleby RC: Diabetes mellitus in a budgerigar, *Vet Rec* 115:652–653, 1984.
Arnall L: Experiences with cage-birds, *Vet Rec* 70:120–128, 1958.
Arnall L, Keymer IF: The endocrine system. In Arnall L, Keymer IF, editors: *Bird diseases: an introduction to the study of birds in health and disease*, Neptune City, NJ, 1975, TFH Publications.
Astier H: Thyroid gland in birds. In Epple A, Stetson MH, editors: *Avian endocrinology*, New York, 1980, Academic Press.
Bates G, Tucker RL, Ford S, et al: Thyroid adenocarcinoma in a bald eagle (*Haliaeetus leucocephalus*), *J Zoo Wildl Med* 30:439–442, 1999.
Bauck LA: Pituitary neoplastic disease in 9 budgerigars. *Proceedings of the Association of Avian Veterinarians,* Hawaii, pp 87–90, 1987.
Beach JE: Diseases of budgerigars and other cage birds. A survey of post-mortem findings, *Vet Rec* 74:10–14, 63–68, 134–140, 1962.
Blackmore DK: The pathology and incidence of thyroid dysplasia in budgerigars (*Melopsittacus undulatus*), *Vet Rec* 75:1068–1072, 1965.
Blackmore DK, Cooper JE: Diseases of the endocrine system. In Petrak ML, editor: *Diseases of cage and aviary birds*, ed 2, Philadelphia, PA, 1982, Lea & Febiger.
Blount WP: *Diseases of poultry*, London, 1947, Baillière Tindall & Cox.
Brandão J, Manickam B, Blas-Machado U, et al: Productive thyroid follicular carcinoma in a wild barred owl (*Strix varia*), *J Vet Diagn Invest* 24:1145–1150, 2012.
Campbell TW: Neoplasia. In Harrison GJ, Harrison LR, editors: *Clinical avian medicine and surgery*, Philadelphia, PA, 1986, WB Saunders.
Campbell TW, Stuart LD: Ovarian neoplasia in the budgerigar (*Melopsittacus undulatus*), *Vet Med Small Anim Clin* 79:215–218, 1984.
Candeletta SC, Homer BL, Garner MM, et al: Diabetes mellitus associated with chronic lymphocytic pancreatitis in an African gray parrot (*Psittacus erithacus erithacus*), *J Assoc Avian Vet* 7:39–43, 1993.
Chalmers GA: Neoplasms in two racing pigeons, *Avian Dis* 30:241–244, 1986.
Cooper JE: *Veterinary aspects of captive birds of prey*, ed 2, with 1985 supplement, Saul, Gloucestershire, UK, 1985, Standfast Press.
Cornelissen H, Verhofstad A: Adrenal neoplasia in a scarlet macaw (*Ara macao*) with clinical signs of hyperadrenocorticism, *J Avian Med Surg* 13:92–97, 1998.
Curtis-Velasco M: Pituitary adenoma in a cockatiel (*Nymphicus hollandicus*), *J Assoc Avian Vet* 6:21–22, 1992.
de Matos R: Calcium metabolism in birds, *Vet Clin North Am Exot Pract* 11:59–82, 2008a.
de Matos R: Adrenal steroid metabolism in birds: anatomy, physiology, and clinical considerations, *Vet Clin North Am Exot Anim Pract* 17:35–57, 2008b.
Dezfoulian O, Abbasi M, Azarabad H, et al: Cerebral neuroblastoma and pituitary adenocarcinoma in two budgerigars (*Melopsittacus undulatus*), *Avian Dis* 55:704–708, 2011.
Douglass EM: Diabetes mellitus in a toucan, *Mod Vet Pract* 62:293–295, 1981.
Ebert U: *Vogelkrankheiten*, ed 2, Hannover, Germany, 1978, Verlag Schaper.
Effron M, Griner L, Benirschke K: Nature and rate of neoplasia found in captive wild mammals, birds, and reptiles at necropsy, *J Natl Cancer Inst* 59:185–198, 1977.
Ellis LC: Endocrine role of the pineal gland, *Am Zool* 16:100–101, 1976.
Epple A, Stetson MH: *Avian endocrinology*, New York, 1980, Academic Press.
Follett BK, Goldsmith AR: In Campbell B, Lack E, editors: *A dictionary of birds*, Carlton, Staffordshire, UK, 1985, T & AD Poyser.
Forbes NA, Cooper JE, Higgins RJ: Neoplasms of birds of prey. In Lumeij JT, Remple JD, Redig PT, et al, editors: *Raptor biomedicine III*, Lake Worth, FL, 2000, Zoological Education Network.
Frost C: Experiences with pet budgerigars, *Vet Rec* 73:621–626, 1961.

Gabrisch K, Zwart P: *Krankheiten der Heimtiere*, Hannover, Germany, 1984, Schlütersche.
Gancz AY, Wellehan JFX, Boutette J, et al: Diabetes mellitus in large psittacines: a possible relationship with excessive iron storage. *Proceedings of the Association of Avian Veterinarians,* Monterey, California, pp 267–269, 2005.
Gancz AY, Wellehan JF, Boutette J, et al: Diabetes mellitus concurrent with hepatic haemosiderosis in two macaws (*Ara severa*, *Ara militaris*), *Avian Pathol* 36:331–336, 2007.
Gorham SL, Ottinger MA: Sertoli cell tumors in Japanese quail, *Avian Dis* 30:337–339, 1986.
Gratzl E, Köhler H: *Spezielle Pathologie and Therapie der Geflügelkrankheiten*, Stuttgart, Germany, 1968, Ferdinand Enke.
Greenacre CB, Young DW, Behrend EN, et al: Validation of a novel high-sensitivity radioimmunoassay procedure for measurement of total thyroxine concentration in psittacine birds and snakes, *Am J Vet Res* 62:1750–1754, 2001.
Griner LA: *Pathology of Zoo Animals*, San Diego, CA, 1983, Zoological Society of San Diego.
Halliwell WH, Graham DL: Neoplasms in birds of prey. In Fowler ME, editor: *Zoo and wild animal medicine*, Philadelphia, PA, 1978, WB Saunders.
Hammmerton AE: *Proceedings of the Zoological Society of London,* 112:149, 1943.
Hasholt J: Atrophy of the pancreas in budgerigar, *Nord Vet Med* 24:458–461, 1972.
Jefferies DJ, Parslow JLF: Thyroid changes in PCB-dosed guillemots and their indications of one of the mechanisms of action of these materials, *Environ Pollut* 10:293–311, 1976.
Joyner KL: Theriogenology. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.
Keymer IF: Disorders of the avian female reproductive system, *Avian Pathol* 9:405–419, 1980.
King AS, McLelland J: *Birds: their structure and function*, London, 1984, Baillière Tindall.
Kronberger H: *Haltung von Vögeln. Krankheiten der Vogel*, Jena, Germany, 1973, VEB Gustav Fischer.
Kumar E, Holt WV: Impacts of endocrine disrupting chemicals on reproduction in wildlife, *Adv Exp Med Biol* 753:55–70, 2014.
Langohr IM, Garner MM, Kiupel M: Somatotroph pituitary tumors in budgerigars (*Melopsittacus undulatus*), *Vet Pathol* 49:503–507, 2012.
Latimer KS: Oncology. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.
Latimer KS, Greenacre CB: Adrenal carcinoma in a budgerigar (*Melopsittacus undulatus*), *J Avian Med Surg* 9:141–143, 1995.
Lattin CR, Ngai HM, Romero LM: Evaluating the stress response as a bioindicator of sub-lethal effects of crude oil exposure in wild house sparrows (*Passer domesticus*), *PLoS ONE* 9:e102016, 2014.
Leach MW: A survey of neoplasia in pet birds, *Semin Avian Exot Pet Med* 1(2):52–64, 1992.
Lombard LS, Witte EJ: Frequency and types of tumors in mammals and birds of the Philadelphia Zoological Gardens, *Cancer Res* 19:127–141, 1959.
Lothrop CD Jr: Diseases of the endocrine system. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Philadelphia, PA, 1996, Williams & Wilkins.
Loukopoulos P, Bautista AC, Puschner B, et al: An outbreak of thyroid hyperplasia (goiter) with high mortality in budgerigars (*Melopsittacus undulatus*), *J Vet Diagn Invest* 27:18–24, 2015.
Lumeij JT: Endocrinology. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Winger Publishing.
Merryman JI, Buckles EL: The avian thyroid gland. Part one: a review of the anatomy and physiology, *J Avian Med Surg* 12:234–237, 1998a.
Merryman JI, Buckles EL: The avian thyroid gland. Part two: a review of the function and pathophysiology, *J Avian Med Surg* 12:238–242, 1998b.
Möstl E, Rettenbacher S, Palme R: Measurement of corticosterone metabolites in birds' droppings: an analytical approach, *Ann NY Acad Sci* 1046:17–34, 2005.
Mrzel L, Pogacnik M, Josipovic D: Frequency and characteristics of tumors in cage birds, *Vet Glasnik* 33:989–993, 1979.
Murphy J: Diabetes in toucans. *Proceedings of the Association of Avian Veterinarians*, pp 165–170, 1992.
Neumann U, Kummerfeld N: Neoplasms in budgerigar (*Melopsittacus undulatus*): clinical, pathomorphological and serological findings with special consideration of kidney tumors, *Avian Pathol* 12:353, 1983.
Oglesbee BL, Orosz S, Dorrestein GM: The endocrine system. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.
Ottinger MA, Lavoie E, Thompson N, et al: Neuroendocrine and behavioral effects of embryonic exposure to endocrine disrupting chemicals in birds, *Brain Res Rev* 57:376–385, 2008.
Ottinger MA, Lavoie ET, Thompson NB, et al: Is the gonadotropin releasing hormone system vulnerable to endocrine disruption in birds?, *Gen Comp Endocrinol* 163:104–108, 2009.
Pass DA, Wylie SL, Forshaw D: Acute pancreatic necrosis of galah (*Cacatua roseicapilla*), *Aust Vet J* 63:340–341, 1986.
Petrak ML, Gilmore CE: Neoplasms. In Petrak ML, editor: *Diseases of cage and aviary birds*, Philadelphia, PA, 1982, Lea & Febiger.
Pilny AA: The avian pancreas in health and disease, *Vet Clin North Am Exot Anim Pract* 11:25–34, 2008.
Quesenberry KE, Liu SK: Pancreatic atrophy in a blue and gold macaw, *J Am Vet Med Assoc* 189:1107–1108, 1986.
Ratcliffe HL: Incidence and nature of tumors in captive wild mammals and birds, *Am J Cancer* 17:116–135, 1933.
Reece RL: Observations on naturally occurring neoplasms in birds in the State of Victoria, Australia, *Avian Pathol* 21:3–32, 1992.
Rewell RE: Seminoma of the testis in a collared dove (*Streptopelia risoria*), *J Pathol Bacteriol* 60:155, 1948.
Richkind M, Gendron AP, Howard EB, et al: Pseudomonas septicemia associated with autoimmune endocrinopathy in a red-vented cockatoo, *Vet Med Small Anim Clin* 77:1548–1554, 1982.
Ritchey JW: Exocrine pancreatic insufficiency in a yellow-napped Amazon (*Amazona ochrocephala*) with pancreatic adenocarcinoma, *Vet Pathol* 34:55–57, 1997.
Ritchie M, Pilny AA: The anatomy and physiology of the avian endocrine system, *Vet Clin North Am Exot Anim Pract* 11:1–14, 2008.
Ritzman TK: Pancreatic hypoplasia in an eclectus parrot (*Eclectus roratus polychloros*). *Proceedings of the Association of Avian Veterinarians,* Portland, Oregon, pp 83–87, 2000.
Romagnano A, Mashima TY, Barnes HJ, et al: Pituitary adenoma in an Amazon parrot, *J Avian Med Surg* 9:263–270, 1995.
Ryan CP, Walder EJ, Howard EB: Diabetes mellitus and islet cell carcinoma in a parakeet, *J Am Anim Hosp Assoc* 18:139–142, 1982.
Samour JH, Naldo JL: Pancreatic atrophy in a peregrine falcon (*Falco peregrinus*), *Vet Rec* 151:124–125, 2002.
Samour JH, Naldo JL, Wernery U, Kinne J: Thyroid cystadenocarcinoma in a saker falcon (*Falco cherrug*), *Vet Rec* 149:277–278, 2001.
Sasipreeyajan J, Newman JA: Goiter in a cockatiel (*Nymphicus hollandicus*), *Avian Dis* 32:169–172, 1988.
Schlumberger HG: Neoplasia in the parakeet. I. Spontaneous chromophobe pituitary tumors, *Cancer Res* 14:237–245, 1954.
Schlumberger HG: Spontaneous goiter and cancer of the thyroid in animals, *Ohio J Sci* 55:23–43, 1955.
Schlumberger HG: Tumors characteristic for certain animal species. A review, *Cancer Res* 17:823–832, 1957.
Schlumberger HG: Polyostotic hyperostosis in the female parakeet, *Am J Pathol* 35:1–23, 1959.
Schmidt RE: Neoplastic diseases. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.
Schmidt RE, Reavill DR: Thyroid hyperplasia in birds, *J Avian Med Surg* 16:111–114, 2002.
Schmidt RE, Reavill DR: The avian thyroid gland, *Vet Clin North Am Exot Anim Pract* 11:15–23, 2008.
Schmidt RE, Reavill DR: Lesions of the avian pancreas, *Vet Clin North Am Exot Anim Pract* 17:1–11, 2014.

Schmidt RE, Reavill DR, Phalen DN: *Pathology of pet and aviary birds*, Ames, IA, 2003, Iowa State Press.
Shimonohara N, Holland CH, Lin TL, et al: Naturally occurring neoplasms in pigeons in a research colony: a retrospective study, *Avian Dis* 57:133–139, 2013.
Shöne R, Arnold P: *Der Wellensittich. Heimtier und Patient*, Jena, Germany, 1980, VEB Gustav Fischer.
Speer BL: Diseases of the urogenital system. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.
Speer BL: A clinical look at the avian pancreas in health and disease. *Proceedings of the European Association of Avian Veterinarians,* Munich, pp 134–140, 2001.
Starkey SR, Morrisey KJ, Stewart JE, et al: Pituitary-dependent hyperadrenocorticism in a cockatoo, *J Am Vet Med Assoc* 232:394–398, 2008.
Stauber E, Papageorges M, Sande R, et al: Polyostotic hyperostosis associated with oviductal tumor in a cockatiel, *J Am Vet Med Assoc* 196:939–940, 1990.
Stogdale L: Definition of diabetes mellitus, *Cornell Vet* 76:156–174, 1986.
Stoica G, Russo E, Hoffman JR: Abdominal tumor in a military macaw (diagnosis: metastatic ovarian carcinoma), *Lab Anim* 18(5):17–20, 1989.
Turk RJ, Kim J, Gallina AM: Seminoma in a pigeon, *Avian Dis* 25:752–755, 1981.
Von Zipper J, Tamasohke CH: Pathologische Schilddrüsenbefunde bei Vögeln. In *Diseases of Zoo Animals, 14th International Symposium*, Wroclaw, Poland, 1972, Akademie Verlag, Berlin, Germany, pp 113–122.
Wadsworth PF, Jones DM: Some abnormalities of the thyroid gland in non-domesticated birds, *Avian Pathol* 8:279–284, 1979.
Wadsworth PF, Jones DM, Pugsley SL: A survey of mammalian and avian neoplasms at the Zoological Society of London, *J Zoo Anim Med* 16:73–80, 1985.
Waller-Pendleton E, Rogers D, Epple A: Diabetes mellitus in a red-tailed hawk (*Buteo jamaicensis*), *Avian Pathol* 22:631–635, 1993.
Wheler C: Pituitary tumors in cockatiels, *J Assoc Avian Vet* 6(2):92, 1992.
Wight PAL: Hemiagenesis of the thyroid gland in the quail (*Coturnix coturnix japonica*), *J Comp Pathol* 96:235–236, 1986.
Wilson RB, Holscher MA, Fullerton JR, et al: Pineoblastoma in a cockatiel, *Avian Dis* 32:591–593, 1988.
Woerpel RW, Rosskopf WJ: Clinical experience with avian laboratory diagnostics, *Vet Clin North Am* 14:249–286, 1984.
Zenoble RD, Kemppainen RJ, Young DW, et al: Endocrine responses of healthy parrots to ACTH and thyroid stimulating hormone, *J Am Vet Med Assoc* 187:1116–1118, 1985.

DISORDERS OF THE REPRODUCTIVE SYSTEM

Miguel D. Saggese

Disorders of the reproductive system are relatively common in birds. In individual pet birds, reproductive conditions are usually life-threatening situations (e.g., egg binding, egg yolk coelomitis, and prolapsed phallus) that require intensive medical and/or surgical approaches. In aviary birds, veterinarians may be asked to investigate infertility, low breeding success, and/or breeding failure, usually as a result of nutritional imbalance or deficiency, chronic infections, genetic and/or management problems. The captive breeding of birds for aviculture, commercial, research, and ex situ conservation reasons is an art and a science. The reader is encouraged to consult other resources for current recommendations and strategies regarding captive breeding of birds in aviaries, medical disorders, the investigation of infertility, and control of reproduction in captive birds (Speer, 1991, 1995, 1996; Schubot *et al.*, 1992; Romagnano, 1996, 2005; Styles, 1997, 2002; Millam, 1999; Jones, 2001; Forbes, 2002; Samour, 2010).

This section describes the most commonly observed disorders of the reproductive tract of companion and individual pet birds. The anatomy and physiology of the avian male and female have been extensively reviewed by Whittow (2000), Reece (2009), Johnson (2014), and Vizcarra *et al.* (2014).

REPRODUCTIVE DISORDERS OF THE MALE BIRD

In male birds, both testes are present, located cranially and slightly dorsal to the kidneys. In young birds and those that have not reached sexual maturity, the avian testes are usually very small, and rarely identified in radiographs. Seasonality, breeding strategies, latitude, social behavior and social dominance, nutrition, and taxonomy, are all factors that can influence the age birds achieve sexual maturity. For example, small passerines may reach sexual maturity before their first year, whereas other birds such as eagles, pelagic seabirds, and condors may require up to 5 to 7 years to become sexually mature. Size of the testes varies along the reproductive cycle and interpretation of enlargements of this organ in radiographs and other imaging techniques in sexually mature birds should be carefully evaluated to avoid confusion with pathologic causes of increased testicular size.

Only testes and efferent conducts (rete testis, epididymis, and ductus deferens) are conserved among different avian orders and families, and are similar to their mammalian counterparts, both anatomically and in function (Schmidt *et al.*, 2003) (Fig. 13-92). In most avian species the testes are a white or pale yellow color (Schmidt *et al.*, 2003). Pigmented testicles are present in some species of neotropical vultures, parrots, and some species of passerines and owls. Seasonal testicular

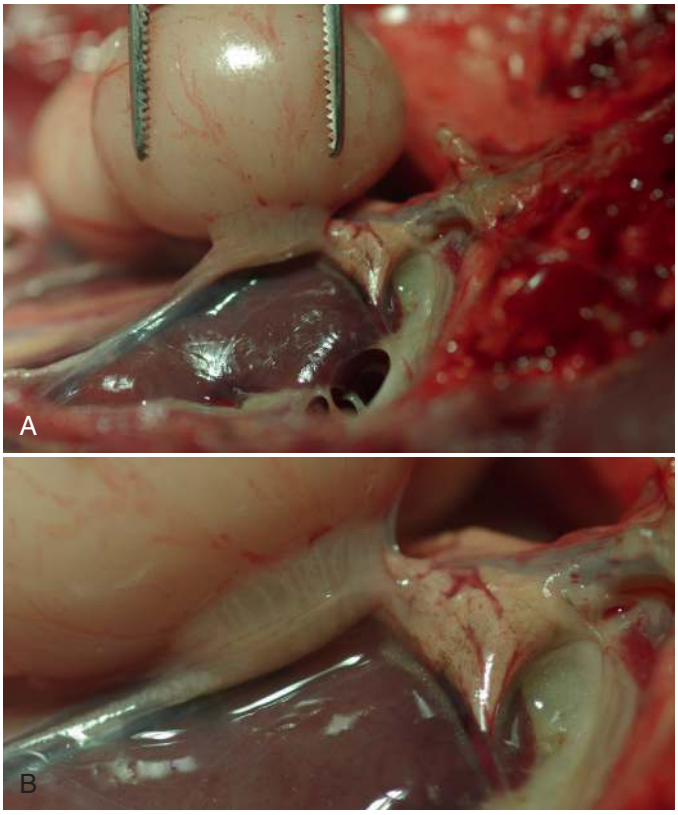


FIGURE 13-92 **(A** and **B),** Close-up view of the reproductive tract of a mature, adult male ring-neck (*Phasianus colchicus*) pheasant. Note the numerous efferent tubules originating from the rete testis, the epididymis, and the vas deferens. The close association between the head of the epididymis and the adrenal gland can be clearly observed. This anatomical interrelation has to be considered when performing orchiectomy in birds. (Courtesy Dr. Jaime Samour.)

melanization can also occur in some birds associated with high mtDNA substitution rates in testicular cells (Galván *et al.*, 2011). Other reproductive organs, such as the seminal glomus (a sperm-storing structure at the end of the efferent conducts), is characteristic of finches and other passerines and budgerigars (*Melopsittacus undulatus*; Samour *et al.*, 1986, 1988), whereas the phallus is only found in a small number of species.

The most common avian testicular disease is neoplasia of every type of cell found in the testis, including seminomas, Sertoli cell tumors, Leydig cell tumors, teratomas, and metastatic neoplasia (Reavill and Schmidt, 2003; Schmidt *et al.*, 2003). Clinical signs in avian patients with testicular neoplasia sometimes can be undetected if the tumor is not functional or, as result of its reduced size, unable to cause mechanical interference. In contrast, in severely enlarged tumors abdominal distention, dyspnea, and ipsilateral paresis and paralysis are common findings. Bilateral enlargement of the testes can be observed in normal birds as a result of increased gonad activity, whereas only one testis is typically affected with testicular neoplasia and other pathologic conditions. Other signs are rare.

Feminization syndrome is a common clinical sign in birds with functional Sertoli cell tumors (Razmyar *et al.*, 2005) and is commonly observed in budgerigars where color changes of the cere, from blue to pink or salmon coloration, are known to occur (Schmidt *et al.*, 2003), but is uncommon in other avian species. Radiographic and ultrasound investigation of the coelomic cavity allows identification of enlarged testes in most cases of neoplasia. Definitive diagnosis of testicular neoplasia is made by histopathological examination of an adequate biopsy sample obtained during exploratory coeliotomy or laparoscopic surgery of these masses (Hernandez-Divers *et al.*, 2007). Hematology, clinical biochemistry, and other ancillary diagnostic tests help rule out inflammatory infectious causes of enlargement, but are of limited value for the diagnosis of testicular neoplasia. Orchiectomy should be considered if biopsy results are compatible with nonmalignant neoplasia. Bilateral neoplasia of the testes is extremely rare.

Orchitis, the inflammation of the testicle, can be unilateral or bilateral. Unilateral orchitis is usually the result of localized trauma or the extension of another inflammatory process affecting the coelomic cavity, rarely caused by an ascending infection of the efferent ducts. More commonly orchitis (alone or combined with epididymitis) is a bilateral condition resulting from systemic infectious diseases. Several species of bacteria and viruses can be hematogenously disseminated and reach the testicles. Common causes of bacterial orchitis include *Salmonella* spp., *Escherichia coli*, *Mycobacterium* spp., and *Chlamydia psittaci.* Viruses such as infectious bronchitis virus, avian influenza virus, West Nile virus, and paramyxovirus type 1 are common potential causes of viral orchitis and epididymitis (Crosta *et al.*, 2002). Aspergillosis can also cause granulomatous orchitis. Rarely is orchitis diagnosed as a unique clinical condition; typically the gonads are affected in conjunction with other organs, as most of these microorganisms cause systemic infections.

Clinical signs in birds with acute orchitis are usually nonspecific and can be masked by general signs of disease associated with a systemic infection. Chronic orchitis is usually associated with infertility. Leukocytosis, characterized by moderate to severe heterophilia with or without monocytosis, is a common finding in localized and systemic bacterial and fungal infections whereas lymphocytosis is more characteristic of inflammatory responses caused by viral infections. Diagnostic imaging, such as radiographs and ultrasound, can be useful to assess testicular enlargement and the presence of cysts and/or testicular abscesses (Hofbauer and Krautwald-Junghanns, 1999). Laparoscopy is useful to confirm gross lesions of the organ and to obtain biopsy samples (Crosta *et al.*, 2002; Hänse *et al.*, 2013). Histopathological findings, culture, isolation, and/or PCR identification of the microorganism present are required for a definitive diagnosis of orchitis. Therapy for orchitis depends on the etiology.

The majority of avian species lack a phallus. It is a well conserved structure in Anseriformes, Craciformes, paleognaths, some Galliformes, and the kiwi (*Apteryx australis*). Exteriorization of the phallus is normal in excited birds previous to or during mating. Prolapse of this organ is a medical emergency and requires an immediate therapeutic response. Acute prolapse is characterized by the presence of an edematous phallus without signs of inflammation or necrosis. In this situation, the bird should be isolated from other birds, especially from the females, to reduce sexual stimulation. Prevention of additional self-mutilation is essential and this can be achieved by keeping the bird in darkness. Application of cold packs or submerging the bird in cold water may help reduce the swelling. Topical administration of granulated sucrose may help reduce the edema initially. Aggressive fluid therapy and administration of short-term steroids such as methyl prednisolone can also contribute to reducing inflammation and edema. Topical administration of antihemorrhoid ointment is recommended by some practitioners. Prognosis of this condition depends on a favorable initial response to therapy. If the phallus persists in being prolapsed after reintroduction in the cloaca, a tobacco pouch suture should be performed and kept in for several days. Chronic prolapse of the phallus is characterized by variable degrees of necrosis of the organ, inflammation, and usually infection. In most cases, partial or total amputation is recommended in addition to supportive therapy, analgesics, and antibiotic administration.

REPRODUCTIVE DISORDERS OF THE FEMALE

In the avian female, only the left ovary is present and functional. Exceptions are members of the order Falconiformes and the kiwi, which can have both ovaries and oviducts (Fig. 13-93). The size of the avian ovary is also variable, and sexual maturity also depends on multiple factors as it does for males. In sexually mature birds, the ovary can produce hundreds of follicles and oocytes, and as long as the condition for egg laying persists, birds are able to produce eggs for prolonged periods of time. In certain occasions it is advantageous to maximize reproduction, but it also can be a severe medical problem. After ovulation, the ovum enters the avian oviduct, which is divided

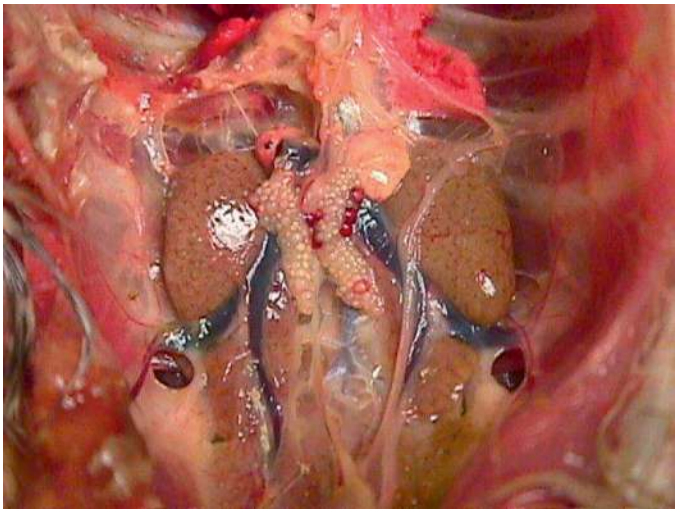


FIGURE 13-93 Left and right ovaries in a red-tailed hawk (*Buteo jamaicensis*).

in five sections: infundibulum, magnum, isthmus, shell gland, and vagina. Female birds have sperm-storage tubules or glands located in the distal oviduct where they can maintain the sperm cells alive for prolonged periods of time. The eggshell covers and protects the gamete and associate structures. It is composed of calcium bicarbonate and is secreted bythe shell gland section or uterus of the oviduct.

Of all the reproductive disorders that can affect the female avian patient, egg binding or dystocia (the unsuccessful passing of an egg through the reproductive tract) is one of the most commonly diagnosed problems by avian practitioners (Hudelson and Hudelson, 1994). This life-threatening condition usually has multiple causes, including mechanical obstructions (presence of a mass along the oviduct), excessively large eggs, failure of the smooth muscle to properly contract, salpingitis (inflammation of the oviduct), obesity, coelom-occupying masses, oviduct hypoplasia, hypocalcemia, abnormalities of the egg and eggshell, cloacoliths, excessive egg production, and multiple ovulations. In most cases, egg binding or dystocia is a sign of an overlooked underlying problem (Hudelson and Hudelson, 1994; Fig. 13-94). Once the initial problem is resolved, the cause(s) of egg binding should be pursued by the avian veterinarian to prevent relapses.

In most avian patients, dystocia is an emergency situation where the whole general condition of the bird is affected by the compression of the internal organs, air sac spaces, and vasculature by the retained egg and requires immediate intervention. The egg size/body size ratio and the length of the problem are directly proportional to the severity of egg-binding consequences. Smaller birds, such as passerines and small psittacines, are usually severely affected and they hold a more guarded prognosis compared with larger birds. Sudden death is not an uncommon outcome, especially in birds that inhabit large aviaries or those with limited care by the owners, as they cannot detect the problem early.

Birds with egg binding usually present with depression or stupor, closed eyes, fluffed feathers, tenesmus, dehydration, muscular fatigue, paresis, and dyspnea. Abdominal hernias are sometimes observed in passerines and small psittacines. Minimal manipulation of these patients is recommended, especially for birds that appear to be extremely debilitated and have moderate to severe dyspnea. Cage resting and oxygen therapy may contribute to an initial improvement of the bird's condition before a physical examination and ancillary diagnostic methods are pursued.

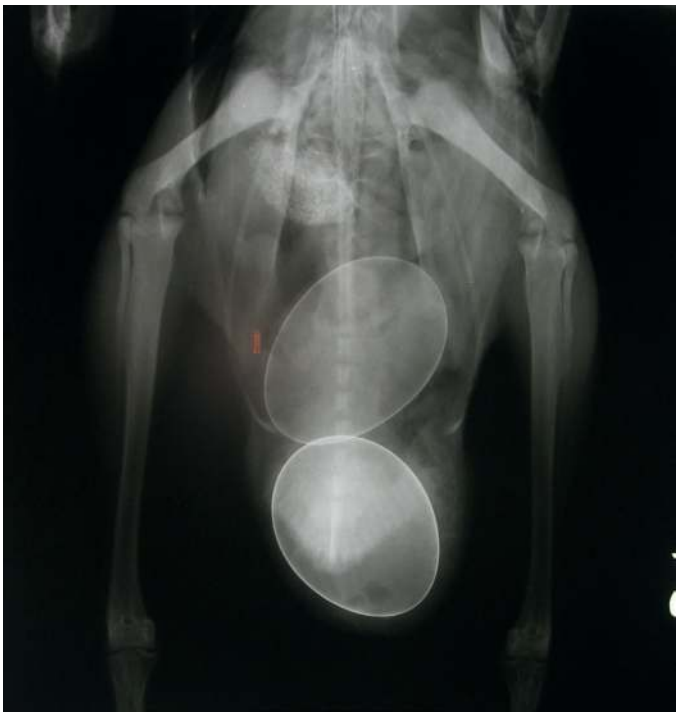


FIGURE 13-94 Ventrodorsal radiograph of a common goose (*Anser anser. domesticus*). Dystocia is seen as result of multiple egg laying and mal alignment of eggs in the oviduct. (Courtesy Dr. Jalila Abu.)

Diagnosis of egg binding relies on the observed clinical signs combined with the presence of unpassed egg(s) in the oviduct or cloaca. A differential diagnosis with other causes of dyspnea should be performed as soon as possible, and a presumptive diagnosis of a primary respiratory problem without investigating egg binding or the presence of abdominal masses could have serious consequences for the companion bird. A careful physical examination that includes gentle palpation of the coelomic cavity and exploration of the cloaca should reveal the presence of unpassed egg(s). Radiographs may be necessary to confirm a clinical diagnosis of egg binding, particularly in medium- and large-sized birds, but minimum handling of the patient is recommended in these cases and radiographs should be taken only when the bird's condition permits. Survey radiographs taken with the bird in standing position or perching, even inside their cages, have diagnostic value for egg binding, and there is no need for additional views and the stress it generates.

Medical treatment should be implemented as soon as the patient condition allows and as quick as possible, with the primary goal of stabilizing the patient and removing the bound egg. Fluids should be administered subcutaneously in the lateral flanks or interscapular area to avoid accidental pressure on the abdomen caused by the fluids if administered in the inguinal fold. Birds should be placed in a quiet, warm, and humid environment if their overt condition allows before any attempts to remove the egg are made. Most patients present several hours after the problem started, exhausted and very weak, and are poor candidates for extreme maneuvers. Owners should be informed of the severity of this condition and the potential risk of death from lifesaving measures done while instituting treatment and/or by the veterinarian's attempts to remove the egg.

When the egg is clearly visible in the cloaca, gentle manual expression after lubrication of the cloacal mucosa with a few drops of mineral oil could be all that is needed to promote egg expulsion in an already exhausted patient. Aspiration of egg content, breaking the eggshell, and careful removal of its fragments may be necessary when manual extraction is not possible, but only if the egg is located in the cloaca. In patients showing clinical signs of dystocia but with the egg still in the oviduct, it is presumed that uterine contractions are no longer present or they are not strong enough to mobilize the egg. In these cases, the use of prostaglandin E_2 (PGE_2) by careful topical application with a cotton swab may help stimulate oviduct contractions. Precautions need to be taken to avoid human contact with this product as it can be easily absorbed through the skin and mucosa. Oxytocin has been reported in the past for its use in egg binding, but it requires multiple doses, and its effectiveness is much less than PGE_2 and is no longer recommended.

Lack of response to medication or implosion of the egg is indication for surgical removal. A caesarian section is performed though a midline or left lateral coeliotomy incision to access the oviduct, remove the egg, and, when possible and indicated, eliminate the cause of dystocia. If the oviduct is nonviable, a salpingohysterectomy is indicated. Detailed surgical procedures for the reproductive tract are described elsewhere in this volume.

Prolapse of the oviduct is a potential consequence of egg binding. Necrosis of the prolapsed section is relatively common and requires surgical debridement. If the prolapse is rapidly diagnosed, reducing the

edema by topical or systemic medical treatment (as for males with prolapse of the phallus) may facilitate gentle reintroduction of the oviduct by using a blunt probe. Concurrent administration of PGE_2 to promote antiperistaltic movements is recommended. Lacerations of the oviduct are potential complications of prolapse and are difficult to diagnose, particularly in smaller species, but any bleeding not coming from the cloacal mucosa or gastrointestinal tract should prompt an investigation. Adhesions as result of oviduct lacerations may predispose the patient to further dystocia.

Egg-yolk coelomitis is caused by the presence of egg yolk in the coelomic cavity, which commonly occurs from ectopic ovulation and triggers an inflammatory response. Egg yolk coelomitis is usually, and at least initially, an aseptic inflammatory process, but it can become septic as result of contamination from concurrent infection of the oviduct, intestinal rupture, hematogenous infection, cloacal ascension of bacteria, and/or concurrent infection of other coelomic organs.

Clinical signs are usually nonspecific and unapparent at the beginning. In more advanced cases, common signs of egg-yolk coelomitis include enlarged abdomen, ascites, depression, dehydration, anorexia, dyspnea, and even sudden death. The hemogram may show anemia and leukocytosis from heterophilia (with toxic heterophils in septic egg-yolk coelomitis) and monocytosis in some cases. Clinical biochemistry results are not pathognomonic of this condition but may indicate a systemic chronic inflammatory response is ongoing. Diagnostic images reveal increased radiodensity in the coelomic cavity, which could be localized around the ovary or, more commonly, diffusely distributed. Laparoscopy is usually contraindicated in egg-yolk coelomitis because of the limited visualization of internal organs and the risk of rupture of the caudal thoracic air sacs, invasion of the lungs and caudal thoracic air sacs with the coelomic exudate, and subsequent death by asphyxia. Confirmation of a presumptive diagnosis of egg-yolk coelomitis can be achieved by fine-needle aspiration and analysis of the coelomic content, which will reveal an inflammatory cellular exudate. Culture and antibiogram of this exudate are always indicated to identify septic coelomitis and implement appropriate antibiotic therapy.

Birds respond poorly to the surgical treatment of this condition, which requires coeliotomy and aspiration of coelomic content. Broad-spectrum antibiotic therapy, or preferably as indicated by results of the antibiogram, accompanied by intensive supportive care, is indicated in infectious egg-yolk peritonitis. Prognosis in most cases of egg-yolk coelomitis is guarded. In those cases successfully managed, prevention of relapse by ovariectomy is mandatory.

Oophoritis/salpingitis refers to the inflammation of the ovary and oviduct, respectively. Most cases have an infectious origin and are difficult to diagnose in the companion pet bird as clinical signs including anorexia, fever, and depression are nonspecific and reproductive failure may not be evident. The hemogram will reveal leukocytosis, heterophilia, and/or monocytosis depending of the chronicity of the problem and the presence of bacteria, such as *Salmonella* spp. and *Mycobacterium* spp., usually associated with chronic granulomatous inflammation. Rarely the ovary and oviduct are the only organs affected by bacterial, fungal, or viral infections and most cases of oophoritis are secondary to salpingitis or generalized systemic infections. Diagnosis is made by a combination of clinical signs; radiographs; ultrasound; laparoscopic exploration of the coelomic cavity; biopsy; histopathology; and isolation of bacteria, virus, or fungi by microbiological culture or molecular diagnosis. When the ovary and/or oviduct are the only organs affected, treatment may consist of supportive therapy, antibiotics upon results of the antibiogram, and ovariohysterectomy.

Ovarian masses are usually neoplasia, abscesses, and cysts. Neoplastic or cystic ovaries are presumptively diagnosed by the presence of clinical signs compatible with ovarian dysfunction, anorexia, depression, and ascites. Paresis or paralyses are potential manifestations of ovarian tumors compressing the ipsilateral ischiatic nerve, but this is not pathognomonic of ovarian tumors as enlargements of other intracoelomic organs, particularly the kidneys, may cause similar signs. Diagnostic imaging, laparoscopic examination, biopsy, histopathology, and culture of abnormal ovarian tissue should contribute to the diagnosis of these neoplasia and cysts in the live bird. Abdominal exudates and transudates may mask the radiologic diagnosis of ovarian cysts and tumors. Ultrasound may provide better results if neoplasia or cysts are suspected (Hofbauer and Krautwald-Junghanns, 1999). Abscesses may be accompanied by changes in the hemogram and blood proteins, whereas these changes are less frequent in the cases of tumors and cysts.

Ovarian tumors can be the result of neoplastic changes in granulosa cells and any other cells (Reavill and Schmidt, 2003). Limited information exists on the medical treatment of ovarian tumors, but ovariectomy is indicated in most cases of nonmalignant ovarian neoplasia. The drainage of ascites and other coelomic collections associated with some ovarian tumors has recently been suggested as a long-term therapeutic approach and management for ascites forming neoplasia (Keller *et al.*, 2013). Therapeutic ovariectomy is sometimes limited by the size of the bird, the veterinarian avian surgical skills, and available equipment.

Chronic egg laying in parrots is the result of prolonged hormonal stimulation on the ovary (Mans and Taylor, 2008). This could be caused by an excessively long induced photoperiod, continuous presence of one or more males in the cage or aviary, removal of eggs, availability of permanent nesting sites, and overstimulation of an imprinted bird by the owner. Treatment includes elimination or reduction of these predisposing factors combined with the administration of leuprolide acetate, a gonadotropin-releasing hormone (GnRH) agonist, to further reduce serum gonadotropin levels (Bowles and Zantop, 2000; Mans and Taylor, 2008). Leuprolide acetate is a deposit, long-acting medication. It is a very expensive drug, requires reconstitution before use, and does not include preservatives for prolonged conservation. As a GnRH, leuprolide acetate inhibits synthesis and secretion of gonadotropins (luteinizing hormone and follicle-stimulating hormone). However, its uses in avian medicine is off-label and the response may vary among different avian species. Avian veterinarians are encouraged to consider these limitations when implementing a medical approach to chronic egg laying and also to report results of these treatments in different avian species. Other GnRH agonists, such as deslorelin acetate, are currently being investigated for their potential use in avian medicine (Petritz *et al.*, 2013). A combination of medical therapy and correction of contributing factors is recommended for optimum results.

REFERENCES

Bowles HL, Zantop DW: Management of chronic egg laying using leuprolide acetate. *Proceedings Annual Conference Association of Avian Veterinarians*, pp 105–108, 2000.

Crosta L, Gerlach H, Burkle M, et al: Endoscopic testicular biopsy technique in Psittaciformes, *J Avian Med Surg* 16:106–110, 2002.

Forbes NA: Captive raptor propagation, *Vet Clin North Am Exot Anim Pract* 5:649–676, 2002.

Galván A, Møller P, Erritzøe J: Testicular melanization has evolved in birds with high mtDNA mutation rates, *Int J Evol Biol* 25:988–998, 2011.

Hänse M, Krautwald-Junghanns ME, Reitemeier S, et al: Testicular biopsy in psittacine birds (Psittaciformes): comparative evaluation of testicular reproductive status by endoscopic, histologic, and cytologic examination, *J Avian Med Surg* 27:247–257, 2013.

Hernandez-Divers SJ, Stahl SJ, Wilson GH, et al: Endoscopic orchidectomy and salpingohysterectomy of pigeons (*Columba livia*): an avian model for minimally invasive endosurgery, *J Avian Med Surg* 21:22–37, 2007.

Hofbauer H, Krautwald-Junghanns ME: Transcutaneous ultrasonography of the avian urogenital tract, *Vet Radiol Ultrasound* 40:58–64, 1999.
Hudelson S, Hudelson P: Egg binding, hormonal control and therapeutic consideration, *Comp Cont Educ* 15:427–432, 1994.
Johnson AL: Reproduction in the female. In Scanes CG, editor: *Sturkie's avian physiology*, New York, 2014, Academic Press.
Jones RG: Endoscopy guided vasectomy in the immature Japanese quail (*Coturnix coturnix japonica*), Master of Sciences Thesis, University of Minnesota, 2001.
Keller KA, Beaufrère H, Brandão J, et al: Long-term management of ovarian neoplasia in two cockatiels (*Nymphicus hollandicus*), *J Avian Med Surg* 27:44–52, 2013.
Mans C, Taylor WM: Update on neuroendocrine regulation and medical intervention of reproduction in birds, *Vet Clin North Am Exot Anim Pract* 11:83–105, 2008.
Millam JR: Reproductive management of captive parrots, *Vet Clin North Am Exot Anim Pract* 2:93–110, 1999.
Petritz OA, Sanchez-Migallon Guzman D, Paul-Murphy J, et al: Evaluation of the efficacy and safety of single administration of 4.7 mg deslorelin acetate implants on egg production and plasma sex hormones of Japanese quail (*Coturnix coturnix japonica*), *Am J Vet Res* 74:316–323, 2013.
Razmyar J, Dezfoulian O, Shojadoost B, et al: Sertoli cell tumor in a pigeon, *J Avian Med Surg* 19:286–288, 2005.
Reavill D, Schmidt R: Tumors of the psittacine ovary and oviduct: 37 cases. *Proceedings Annual Conference Association of Avian Veterinarians*, pp 67–69, 2003.
Reece WO: *Functional anatomy and physiology of domestic animals*, Ames, IA, 2009, Wiley-Blackwell.
Romagnano A: Avian obstetrics, *Semin Avian Exot Pet Med* 5:180–188, 1996.
Romagnano A: Reproduction and pediatrics, *BSAVA manual of psittacine birds*, ed 2, New York, 2005, Harcourt-Brown.
Samour J: Vasectomy in birds: a review, *J Avian Med Surg* 24:169–173, 2010.
Samour J, Smith CA, Moore HDM, et al: Semen collection and spermatozoa characteristics in budgerigar (*Melopsittacus undulatus*), *Vet Rec* 118:397–399, 1986.
Samour J, Spratt DMJ, Holt WV, et al: Ultrastructure and secretory nature of the seminal glomus in budgerigars (*Melopsittacus undulatus*), *Res Vet Sci* 45:195–197, 1988.
Schmidt RE, Reavill DR, Phalen DN: *Pathology of pet and aviary birds*, ed 1, Ames, IA, 2003, Blackwell Publishing.
Schubot RM, Clubb SL, Clubb K: *Psittacine aviculture: perspectives, techniques, and research*, Loxahatchee, FL, 1992, Avicultural Breeding and Research Center.
Speer BL: A clinical approach to psittacine infertility. *Proceedings Annual Conference Association of Avian Veterinarians*, pp 173–187, 1991.
Speer BL: Clinical reproductive avian medicine. *Proceedings Annual Conference Association of Avian Veterinarians*, pp 23–33, 1995.
Speer BL: Diseases of the urogenital system. In Altman RB, Club SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, New York, 1996, Saunders.
Styles DK: Reproduction in the aviary and incubation procedures in psittacine and soft-billed birds. *Proceedings of Annual Conference Association of Avian Veterinarians*, pp 327–338, 1997.
Styles DK: Reproductive management of captive psittacine collections, *Vet Clin North Am Exot Anim Pract* 5:475–487, 2002.
Vizcarra J, Alan R, Kirby J: Reproduction in male bird. In Scanes CG, editor: *Sturkie's avian physiology*, New York, 2014, Academic Press.
Whittow GC: *Sturkie's avian physiology*, ed 5, San Diego, CA, 2000, Academic Press.

DISORDERS OF THE NERVOUS SYSTEM

David Sanchez-Migallon Guzman

ANATOMY OF THE NERVOUS SYSTEM

The avian nervous system has many similarities with the mammalian nervous system but also has many differences, some of which may be of great clinical relevance. For instance, the brain is lissencephalic, without convolutions, with an underdeveloped cerebral cortex and a well-developed corpus striatum, which is considered the center for association in birds. The cranial nerves of birds correspond to those of mammals, with some minor differences. The spinal cord in birds is as long as the neural canal without a cauda equina. Birds also have two spinal cord enlargements, cervical and lumbosacral, as well as a unique structure in the lumbosacral spinal cord between the dorsal columns called the glycogen body. The spinal cord has an internal vertebral plexus that runs the entire length of the vertebral column. The brachial plexus is formed by ventral branches of a variable number of spinal nerves. There are three nerve plexuses in the lumbosacral region: lumbar, ischiatic, and pudendal. These nerve roots lie embedded in the fovea of the synsacrum and are surrounded by the kidneys.

ETIOLOGY AND PATHOPHYSIOLOGY OF NEUROLOGIC DISORDERS

Neurologic disorders are common in birds and may affect the central nervous system (CNS), which includes the brain and spinal cord, and the peripheral nervous system (PNS). The PNS includes cranial nerves, spinal cord nerve roots, spinal nerves, peripheral nerve branches, and the neuromuscular junction. Seizures can result from primary or secondary disorders of the brain that cause spontaneous depolarization of cerebral neurons. Seizures have three components: aura, ictus, and the postictal phase. Ataxia results from disorders that interfere with the recognition or coordination of position changes involving the head, trunk, or limbs. It is divided into three categories: sensory, vestibular, and cerebellar. Paralysis and paresis result from disorders that cause motor deficits. Paresis may present with upper motor neuron clinical signs (loss of voluntary function, normal or increased spinal reflexes, and increased tone), which affect the cerebrum, brainstem, or spinal cord, or with lower motor neuron clinical signs (loss of voluntary function, loss of reflexes, and weakness and muscle atrophy), which affect peripheral nerves. Head tilt, circling, or nystagmus can be caused by central (brainstem) or peripheral (middle or inner ear) vestibular disease. Peripheral lesions cause head tilt toward the side of the lesion, and usually the bird falls or circles toward the side of the lesion. Central lesions may cause head tilt or circling in the opposite direction.

The different etiologies for avian neurologic disorders include vascular, inflammatory/infectious, traumatic, toxic, anomalous (congenital), metabolic, idiopathic, nutritional, neoplastic, and degenerative (using the mnemonic VITAMIND; Box 13-11). The neurologic disorders might involve the CNS and or the PNS.

Vascular

Neurologic signs as a result of ischemic and hemorrhagic cerebral infarctions have been reported in birds and in most instances secondary to atherosclerosis (Beaufrere *et al.*, 2011; Grosset *et al.*, 2014). The pathophysiology of a brain infarction is based on the principle that the brain relies on a constant supply of glucose and oxygen to maintain ionic pump function. When perfusion pressure falls to critical levels, ischemia results in infarction. With cerebral infarction, neurologic deficits develop abruptly and depend on the location of the vascular insult and can result in seizures, vestibular signs, motor deficits, cognition impairment, and monocular vision loss. Intravascular cartilaginous emboli in the spinal cord of turkeys has also been reported and attributed to handling in animals with preexisting cartilage abnormalities (Stedman *et al.*, 1998). In some of these cases the turkeys developed myelomalacia or spinal cord necrosis consistent with thrombosis and resultant ischemia, and others recovered after a period of paresis and ataxia.

BOX 13-11 Etiologic Classification Using the VITAMIND Mnemonic of Neurologic Diseases Reported in Birds

Vascular
Atherosclerosis
Nontraumatic cerebrovascular accidents
Fibrocartilaginous embolism
Lipid embolism

Inflammatory/Infectious
Viral
Avian bornavirus
West Nile virus
Avian paramyxovirus
Avian influenza virus
Togaviruses (eastern and western equine encephalitis)
Avian picornavirus
Polyomavirus
Gallid herpesvirus 2
Picornavirus
Adenovirus

Bacterial
Salmonella spp., *Pasteurella* spp., *Klebsiella* spp., *Pseudomonas* spp., *Streptococcus* spp., *Enterococcus* spp., *Chlamydia psittaci, Mycobacterium* spp. *Escherichia coli, Coxiella* spp., *Listeria* spp., *Staphylococcus* spp.

Parasitic
Baylisascaris procyonis
Toxoplasma gondii
Sarcocystis spp.
Schistosoma spp.
Chandlerella quiscali
Leucocytozoon spp.

Fungal
Aspergillus spp.
Cryptococcus spp.
Mucor spp.

Prions
Toxic/Trauma
Lead, zinc, copper, mercury
Organophosphates
Carbamates
Clostridium botulinum toxin
Cyanobacteria toxin
Domoic acid
Salt poisoning
Toxic plants
Ivermectin
Chloroquine
Tick toxicity
Mycotoxins

Traumatic Brain Injury with or without Skull Fractures
Spinal cord trauma with vertebral fractures, subluxations, luxations, and ruptured intervertebral disk
Brachial plexus avulsion

Anomalous
Hydrocephalus
Hemivertebrae
Lafora body neuropathy

Metabolic
Hypocalcemia
Hypoglycemia
Hepatic encephalopathy

Idiopathic
Idiopathic epilepsy

Neoplastic/Nutritional
Primary neoplasia of the nervous system, such as astrocytomas, glioblastomas, oligodendrogliomas, choroid plexus papillomas, neuroblastoma, ganglioneuromas, meningiomas, pituitary adenoma, and peripheral nerve sheath tumor
Metastatic neoplasia of the brain carcinoma, lymphosarcoma, hemangiosarcoma, and malignant melanoma
Pulmonary, renal, or gonadal neoplasia with compression of spinal cord and sciatic nerve
Thiamine (vitamin B_1) deficiency
Riboflavin (vitamin B_2 deficiency
Cobalamin (vitamin B_{12}) deficiency
Vitamin E deficiency

Degenerative
Lysosomal storage disease

Inflammatory

Inflammatory disease of the CNS can be either noninfectious or infectious. Noninfectious diseases that can cause inflammatory lesions include some toxins, autoimmune disease, and immune-mediated conditions, none of which have been well documented in birds (Schmidt *et al.*, 2003). Viruses, bacteria, fungi, protozoa, and metazoan parasites all can cause inflammatory disease of the CNS. Bacterial organisms, including *Salmonella* spp., *Pasteurella* spp., *Klebsiella* spp., *Pseudomonas* spp., *Streptococcus* spp., *Enterococcus* spp., *Escherichia coli*, *Coxiella* spp., *Listeria* spp., *Staphylococcus* spp., *Chlamydia psittaci*, and *Mycobacterium* spp. can cause meningitis, encephalitis, and/or myelitis. The infection of the brain and meninges can be from direct extension of an infection from the sinuses, nasal cavity, or inner ear or may be the result of a bacteremia or septicemia (Schmidt *et al.*, 2003). Bacterial granulomas may compress or invade the central or peripheral nervous system. Bacterial discospondylitis resulting in neurologic deficits caused by *Staphylococcus aureus* has been diagnosed in a penguin (Field *et al.*, 2012), and suspected to be caused by *Corynebacterium amycolatum* in another (Bergen and Gartrel, 2010). Fungal organisms like *Aspergillus* spp., *Cryptococcus* spp., and *Mucor* spp., may cause inflammation in the nervous system (Fig. 13-95). Fungal granulomas may compress or invade CNS or peripheral nerves and cause unilateral or bilateral paresis or paralysis (Greenacre, 1992). Parasites, such as *Baylisascaris procyonis*, *Toxoplasma gondii*, *Sarcocystis* spp., *Schistosoma* spp., *Chandlerella quiscali*, *and Leucocytozoon* spp., have been reported to cause neurologic disease in birds. Avian bornavirus, West Nile virus

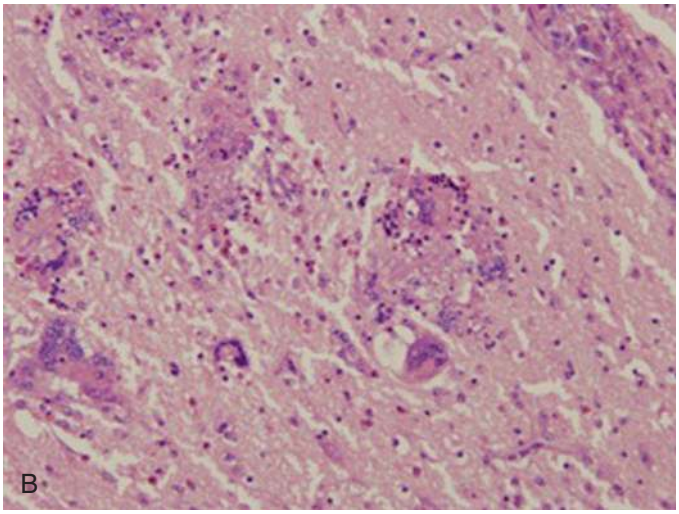


FIGURE 13-95 (A), Orange-winged Amazon parrot (*Amazona amazonica*) with fungal encephalitis. Note the abnormal posture of the left foot and unkempt plumage. **(B),** Histopathology (H&E) of the brain showing the inflammatory cells and fungal organisms.

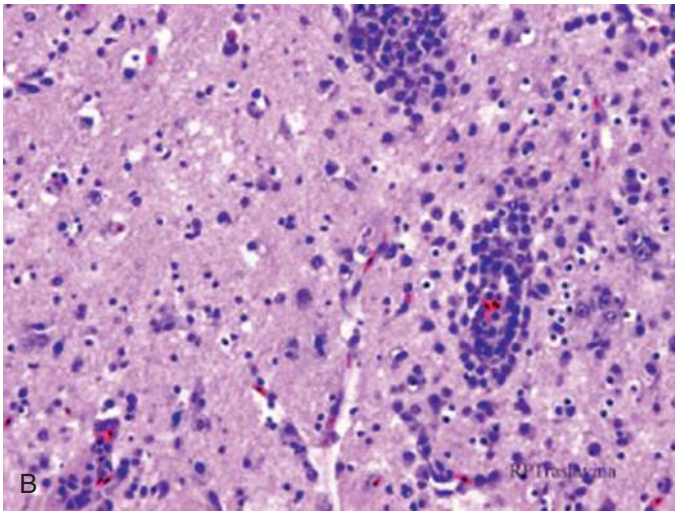


FIGURE 13-96 (A), White cockatoo (*Cacatua alba*) with encephalitis caused by West Nile virus. Note the abnormal posture of the head and wings that were accompanied by tremors. **(B),** Histopathology (H&E) of the brain showing the encephalitis with perivascular cuffing. (Courtesy Ryan Traslavina.)

(Fig. 13-96), avian paramyxovirus, avian influenza virus, togaviruses (eastern and western equine encephalitis), avian picornavirus, avian polyomavirus, gallid herpesvirus 2, and avian adenovirus are known to affect the nervous system of birds.

Toxic

Lead toxicity is one of the most common causes of toxicity in birds, especially in waterfowl, raptors, and companion birds. Lead toxicity causes encephalopathy as a result of diffuse perivascular edema, increases in cerebrospinal fluid, and necrosis of nerve cells. Peripheral neuropathy is caused by demyelination of nerves and blockage of the presynaptic transmission by competitive inhibition of calcium. The pathogenesis of lead toxicity in the nervous system has been studied in mallard ducks (Hunter and Wobeser, 1980). Zinc toxicity could result in neurologic signs, but lesions in the nervous system are rarely described. Organophosphates and carbamates are cholinesterase inhibitors that bind to and subsequently inactivate acetylcholinesterase, causing an accumulation of acetylcholine at the postsynaptic receptors. Organophosphate bonds are considered irreversible, and carbamate bonds are slowly reversible over several days. Two types of neuropathy and corresponding clinical signs have been described related to toxicosis with acetylcholinesterase inhibitors. Acutely, clinical signs are related to excessive stimulation of acetylcholine receptors. Signs include weakness, ataxia, wing twitching and muscle tremors, opisthotonus, seizures, and prolapse of the nictitans (Bennet, 1994). The second type of neuropathy is an organophosphate ester-induced neuropathy, which is not associated with an inhibition of acetylcholine. The onset of clinical signs is delayed (7 to 21 days after exposure) and is the result of a symmetric, distal primary axonal degeneration of the central and peripheral nervous systems, with secondary myelin degeneration (Bennet, 1994). *Clostridium botulinum* toxins (the most common is type C, but occasionally types A and E) are a frequent cause of neurologic disorders in aquatic birds (Smith *et al.*, 1983), and have been reported rarely in other groups of birds (Rocke and Bollinger, 2007). The toxins interfere with the release of acetylcholine at the motor endplate causing signs of peripheral neuropathy. 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. Mycotoxins, such as trichothecenes, have also been reported to cause neurologic signs in chickens (Wyatt *et al.*, 1973) and cranes (Roffe *et al.*, 1989), although primary histopathological lesions in the nervous system are not reported.

Algal biotoxins, such as saxitoxins, brevetoxins, domoic acid, and cyanotoxins, are neurotoxins that have been associated with mass mortalities of aquatic birds (Landsberg *et al.*, 2007). Saxitoxins block sodium channels, preventing signal transmission along nerves. Domoic acid is a neuroexcitatory amino acid analog of L-glutamate produced by diatoms and some algae. Domoic acid binds to glutamate receptors in the CNS, causing sustained membrane depolarization leading to neuronal excitotoxic death. Elevation of endogenous glutamate potentiates the process. Neurons in the limbic system, including the hippocampus, are most at risk.

Plants have also been reported to cause neurologic signs in birds. Crown vetch (*Coronilla varia*) was found to be poisonous to a budgerigar that ingested leaves from a plant next to its cage (Campbell, 2006). Toxicity is from nitroglycoside, a chemical that may affect the nervous system and can cause formation of methemoglobin. Some *Kalanchoe* species have also been documented to cause neurotoxicity in chickens in an experimental study. The chickens showed depression, ataxia, muscle tremors, seizures, paralysis, and death (Williams and Smith, 1984). Avian vacuolar myelinopathy affects bald eagles (*Haliaeetus leucocephalus*), American coots (*Fulica americana*), waterfowl, and other birds in the southeastern United States resulting in severe vacuolation of white matter of the brain. The cause of the disease is thought to be a naturally produced toxin produced by a cyanobacteria associated with aquatic macrophytes, most frequently hydrilla (*Hydrilla verticillata*; Wiley *et al.*, 2009). Tick paralysis is caused by neurotoxins of several species of hard and soft ticks, and results in a motor polyneuropathy characterized by a progressive, ascending flaccid motor paralysis (Luttrell *et al.*, 1996; Monks *et al.*, 2006). Clinical signs include ataxia, paresis, paralysis, areflexia, hypotonus, respiratory failure, and death if ticks are not removed. Iatrogenic toxicities have also been reported in birds, including numerous drugs like dimetridazole, chlorpyrifos, ivermectin, chloroquine, and others.

Trauma

Head trauma is common in wild and companion birds. Causes of head trauma in companion birds include flights into windows, ceiling fans, and mirrors and bites from other animals and other crush injuries, whereas in wild birds these include flights into cars, wind turbines, and windows; shooting; trapping; and predatory wounds. Traumatic brain injury can be primary, from direct tissue damage to the brain (e.g., lacerations, contusions, hemorrhage) with or without skull fractures, or secondary from compromised blood flow to the tissue that can lead to ischemia and metabolic derangements. Brain injuries described in birds flying into towers and windows are damage to the cerebellum, blood vessel rupture, herniation of the cerebellum and medulla through the foramen magnum, extensive subdural bleeding, and intracranial edema (Veltri and Klem, 2005). Spinal cord trauma can result from vertebral fractures (Stauber *et al.*, 2007; Fig. 13-97), subluxations (Fraga-Manteiga *et al.*, 2013), luxations, and ruptured intervertebral disks (Emerson, 1992). Spinal cord injury can also be primary, as direct damage to the tissue, or secondary caused by reduced tissue perfusion, microvascular damage, thrombus formation, and vasospasm, which lead to necrosis of the compromised tissue.

Peripheral nerve trauma also occurs in birds with different degrees of nerve injury. Bone fractures or penetrating wounds may be associated with peripheral nerve injuries, causing unilateral peripheral neuropathies. Brachial plexus avulsion from trauma occurs most commonly in wild birds with denervation of the affected wing, which results in lack of pain perception, paralysis, and muscle atrophy of the wing (Shell *et al.*, 1993). Trauma resulting in damage to the sympathetic innervation of the eye, which includes one upper motor neuron that originates in the hypothalamus and two lower motor neurons that originate in the thoracolumbar spinal cord and the cranial cervical ganglion, causes Horner syndrome in birds (Gancz *et al.*, 2005; Fig. 13-98).

FIGURE 13-97 (A), Barred owl (*Strix varia*) with a thoracic vertebral compression fracture secondary to trauma. Note the abnormal posture of the legs that were accompanied by hypermetria during movement. **(B),** Histopathology (H&E) of the fractured vertebral body with dorsal displacement and compression of the spinal cord.

Anomalous (Congenital)

Hydrocephalus has been reported in birds, and usually involves the lateral ventricles, which are grossly distended, and leads to compression of the overlying cortex (Schmidt *et al.*, 2003). While it appears that in some cases it might be a congenital lesion, it seems also to be reported in older birds, indicating that it could possibly be acquired

FIGURE 13-98 Swainson's hawk (*Buteo swainsoni*) with Horner's syndrome secondary to trauma. Note the raised feathers on the head and dropped upper eyelid on the right eye.

(Wack *et al.*, 1989; Fleming *et al.*, 2003; Johnston *et al.*, 2006; Keller *et al.*, 2011; Fig. 13-99). Hemivertebrae, a type of congenital vertebral anomaly resulting from a lack of formation of one half of a vertebral body, was suspected in a 10-week-old African black-footed penguin (*Spheniscus demersus*). Lafora body disease is presumed to be an inherited defect of carbohydrate metabolism that causes the formation of typical polysaccharide complexes called Lafora bodies. It has been reported in a cockatiel (*Nymphicus hollandicus*; Britt *et al.*, 1989). In chickens, an autosomal recessive mutation resulted in high susceptibility to seizures, and has been used for extensive research as a model for mammalian epilepsy (Crawford, 1970).

Metabolic

Disorders of sodium and osmolality produce CNS neuronal depression, with encephalopathy as the major clinical manifestation; these disorders can also provoke CNS neuronal irritability. Neurologic signs have been reported in a conure after eating salted mixed nuts (Lightfoot and Yeager, 2008), and in house sparrows (*Passer domesticus*) that ingest small numbers of road salt granules (Bollinger *et al.*, 2005). Conversely, hypocalcemia (McDonald, 1988) and hypomagnesemia (Kirchgessner *et al.*, 2012) cause mainly CNS neuronal irritability with seizures. In contrast, disorders of potassium rarely produce clinical signs in the CNS but may be associated with muscle weakness as the major clinical manifestation. Hypoglycemia is another metabolic cause of seizures, especially in young weanling birds, but also in birds with food deprivation and endocrine and liver disease. Hepatic encephalopathy has been reported in birds with compromised hepatic function like hepatic lipidosis (Fig. 13-100), hemochromatosis (Spalding *et al.*, 1986), hepatic neoplasia, and other hepatopathies (Bennet, 1994), while there are no reports documenting it with hepatic shunts. Nitrogenous waste products, including ammonia, accumulate in the blood crossing the blood-brain barrier and resulting in toxicity in the CNS.

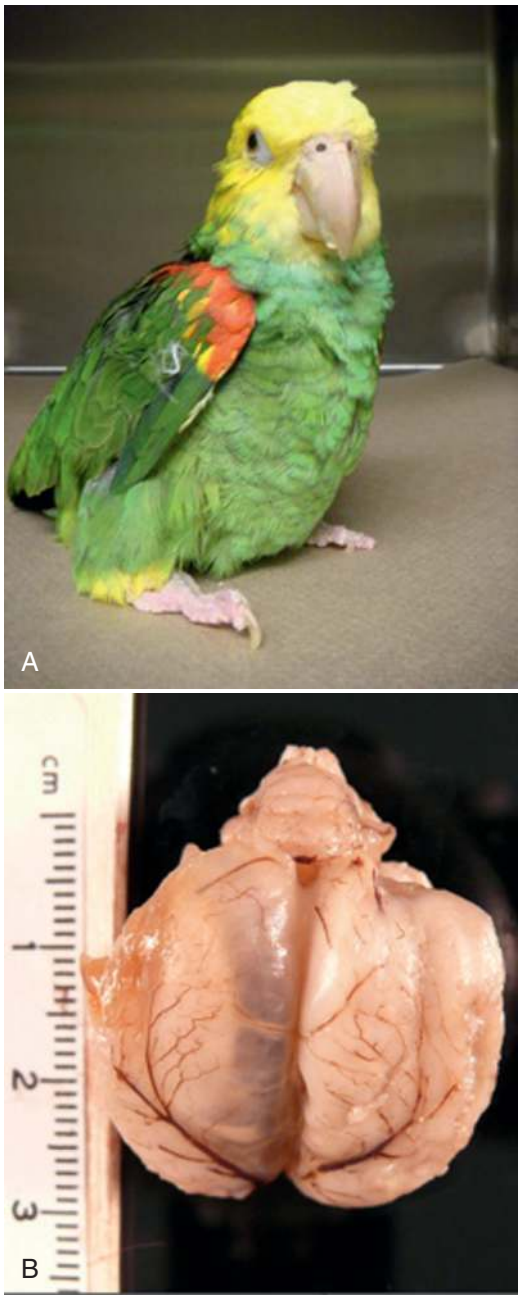


FIGURE 13-99 **(A),** Yellow-headed Amazon parrot (*Amazona oratrix*) with severe hydrocephalus. Note the wide stance that was accompanied by decreased vision in one eye. **(B),** Gross image of the brain shows thinning of the layers on the right cerebral hemisphere.

Idiopathic

Idiopathic epilepsy is a diagnosis of exclusion and may be applied when all other possible causes of seizure activity have been ruled out and the bird is normal between seizure events.

Nutritional

Hypovitaminosis B_1 (thiamine), B_2 (riboflavin), B_6 (pyridoxine), and B_{12} (cyanocobalamin) have been reported to cause neuropathies. Hypovitaminosis B_1 in birds has been studied experimentally in several species including pigeons (Swank, 1940), quails (Pruthi and Verma, 2000), and chickens (Olkowski and Classen, 1996). There are also cases

FIGURE 13-100 Blue-fronted Amazon parrot (*Amazon aestiva*) with suspected hepatic encephalopathy secondary to severe hepatic lipidosis. Note the wide stance balance that was accompanied by a depressed to obtunded mentation.

reported in the literature affecting raptors (Ward 1971; Carnarius *et al.*, 2008), and honey eaters (Holz and Phelan, 2000). Thiamine deficiency is also a concern in piscivorous birds fed fish with thiaminases. The lesions in the nervous system are variable depending on the species, age, and chronicity of the deficiency and could affect the CNS causing polioencephalomalacia and peripheral nerve myelin degeneration. Hypovitaminosis B_2 has been extensively studied in chickens (Cai *et al.*, 2009) and pigeons (Wada *et al.*, 1996) and leads to myelin degeneration of the peripheral nerves, resulting in clinical signs that include toes curled outward and leg and wing paralysis. Hypovitaminosis B_6 in chickens (Gries and Scott, 1972) has also been studied. The affected birds showed neurologic clinical signs that included ataxia, head tilt, and death without histopathological abnormalities in the nervous system. Vitamin E deficiency can cause encephalomalacia that results in tremors, ataxia, head tilt, cycling, and/or recumbency and it is mostly a disease of captive piscivorous birds (Dierenfeld, 1989), but has been reported in chickens (*Gallus gallus domesticus*; Hislop and Whittle, 1967), turkeys (*Meleagris gallopavo*; Jortner *et al.*, 1985), emus (*Dromaius novaehollandiae*; Aye *et al.*, 1998), raptors (Dierenfeld, 1989), and rarely in psittacines (Schmidt *et al.*, 2003).

Neoplastic

Primary neoplasia of the nervous system reported in birds include astrocytomas, glioblastomas, oligodendrogliomas, choroid plexus papillomas, neuroblastomas, ganglioneuromas, meningiomas, pituitary neoplasia, and peripheral nerve sheath tumor (Bennet, 1994; Schmidt *et al.*, 2003). Pituitary neoplasia has more frequently been reported in young to middle age budgerigars (Langohr *et al.*, 2012), but also in other species (Romagnano *et al.*, 1995). The majority of the pituitary tumors in the budgerigars were invasive and some metastasized. Clinical signs reported include ataxia, seizure-like activity, abnormal posture, blindness, head tilt, and circling. Peripheral nerve sheath tumor was reported in a golden eagle (*Aquila chrysaetos*) with

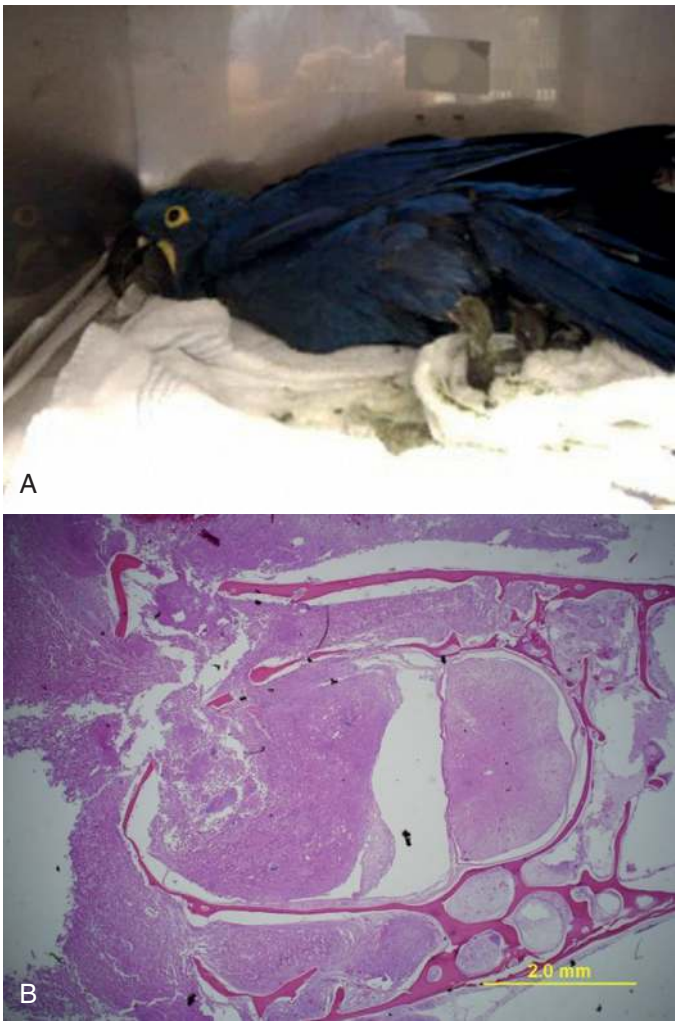


FIGURE 13-101 **(A & B),** Hyacinth macaw (*Anodorhynchus hyacinthinus*) with bronchogenic carcinoma invading the thoracic vertebral bodies and causing compression of the spinal cord. Note the position of the body in sternal recumbency accompanied by severe paralysis of the hindlimbs. **(B),** Gross image and histopathology of a transverse section of the vertebral bodies affected illustrating show invasion of the neoplasia and compression of the spinal cord.

tetraplegia (Wernick *et al.*, 2014). Metastatic neoplasia affecting the nervous system reported in birds including brain carcinoma, lymphosarcoma, hemangiosarcoma, and malignant melanoma have also been reported (Schmidt *et al.*, 2003). Pulmonary neoplasia in birds was reported to invade the vertebrae causing compression of the spinal cord and hindlimb paresis or paralysis (Baumgartner *et al.*, 2008; Fredholm *et al.*, 2012; Fig. 13-101). Renal neoplasia, most commonly carcinomas in budgerigars, will cause sacral nerve plexus compression and unilateral limb paresis or paralysis (Simova-Curd *et al.*, 2006; Fig. 13-102, magnification ×2).

Degenerative

Lysosomal storage disease, a wide group of disorders characterized by the accumulation of macromolecules in the lysosomes of various cells, was described in emus with severe, progressive and eventually fatal gangliosidosis (Bermudez *et al.*, 1995) and more recently in an African grey parrot (*Psittacus erithacus*). Cerebellar degeneration of unknown cause was seen in a turquoise parrot (*Neophema pulchella*). No gross change was noted, but histologically there was neuronophagia and

FIGURE 13-102 **(A),** Budgerigar (*Melopsittacus undulatus*) with paralysis of the right leg but still with deep pain perception secondary to renal adenocarcinoma. **(B),** Gross image showing the renal neoplasia that is compressing ischiatic plexus. (B, Courtesy Dr. DA Goldsmith.)

necrosis (Schmidt *et al.*, 2003). Mineralization is an occasional incidental finding in the brain, as is lipofuscin deposition in neurons. Lipofuscin is a common finding in the neural cell bodies of older parrots, and it is suspected that in most cases this pigment accumulation does not have a functional significance (Schmidt *et al.*, 2003).

HISTORY AND NEUROLOGIC EXAMINATION

In suspected neurologic disease, it is necessary to determine whether a neurologic lesion is causing the disease, then the location of the lesion and extent, the pathologic process, the prognosis, and finally the treatment. A complete history and physical examination should be performed. Species, age, and sex of the patient provide important clues to the diagnosis. Budgerigars (*Melopsittacus undulatus*) are more likely to develop renal neoplasia that can cause unilateral limb paresis by compressing the sciatic nerve. African grey parrots commonly present with seizures or ataxia associated with hypocalcemia. Female birds are more likely to develop vascular diseases (e.g., atherosclerosis) that could result in disorders of the neurologic system. Young birds are more likely to have congenital disorders and infectious disease. Older animals are likely to have vascular, neoplastic, or degenerative diseases. The onset, course, duration, and symmetry of the neurologic problem also provide important information. Acute presentations are more likely associated with vascular, infectious, traumatic, and toxic etiologies, and chronic presentations are more likely associated to metabolic, neoplastic, or degenerative causes. Husbandry may provide information regarding possible infectious, traumatic, toxic, trauma, metabolic, or nutritional etiologies. Birds housed outdoors in areas of risk are more likely to be exposed to infectious etiologies such as West Nile virus, *Sarcocystis*, or *Baylisascaris*.

Female cockatoos housed with males might be bitten in the neck by their mates resulting in cervical vertebrae fracture. Birds recently exposed to other birds may become ill as a result of infectious diseases like proventricular dilation disease. Psittacine birds that are in galvanized cages or with toys made of unknown metals are more likely to develop heavy metal toxicity. Birds with deficient or unbalanced diets in minerals, vitamins, and fatty acids are more likely to develop metabolic or nutritional diseases that affect the neurologic system. Specific history of neurologic disease may include a period of disorientation followed by loss of grip on the perch with subsequent tremoring and extension of the legs and wings or rigidity for varying length of time; limb weakness with loss or retention of some voluntary movement and deep pain; uncoordinated movements of the head or limbs; head tilt with or without falls, drifts, or circles toward one side; and impaired vision or hearing. Nonspecific signs of disease are also frequently present, such as lethargy and anorexia.

Physical examination may reveal findings like seizures, paresis, paralysis, ataxia, head tilt, nystagmus, intention tremor, dysmetria, and visual or hearing deficits that should be evaluated in further detail during the neurologic examination (Fig. 13-103). Nonspecific findings like low body condition, integumentary or musculoskeletal system abnormalities, and gastrointestinal or respiratory signs might add information regarding possible etiology. Birds with trauma may have blood in the oropharyngeal cavity, ears, or eyes or bruises or fractures. Horner syndrome in birds presents consistently with ptosis, and less often with miosis, and more rarely erection of the neck and head feathers, and is the result of loss of sympathetic innervation to the eye from a central or peripheral lesion (Gancz *et al.*, 2005). Birds with articular gout or severe arthritis can have abnormal gait mimicking neurologic disease. Psittacine birds with proventricular disease may be seen with CNS signs with or without gastrointestinal signs and low body condition. Vomiting, diarrhea, and/or hematuria might be seen in birds with heavy metal toxicity, and with common bacterial and viral infectious diseases. Respiratory signs may accompany neurologic signs in bacterial, viral, and fungal infectious diseases and respiratory toxicities.

The goal of the neurologic examination is to confirm a neurologic abnormality and to localize the abnormality within the nervous system. The neurologic examination includes observation, palpation, functional assessment of the cranial nerves, evaluation of proprioceptive deficits and spinal reflexes, and sensory evaluation (Box 13-12). The neurologic examination is covered in Chapter 5.

DIAGNOSTIC TESTING

Complete blood count, plasma chemistry panel, and survey radiography are the minimum tests required when diagnosing neurologic

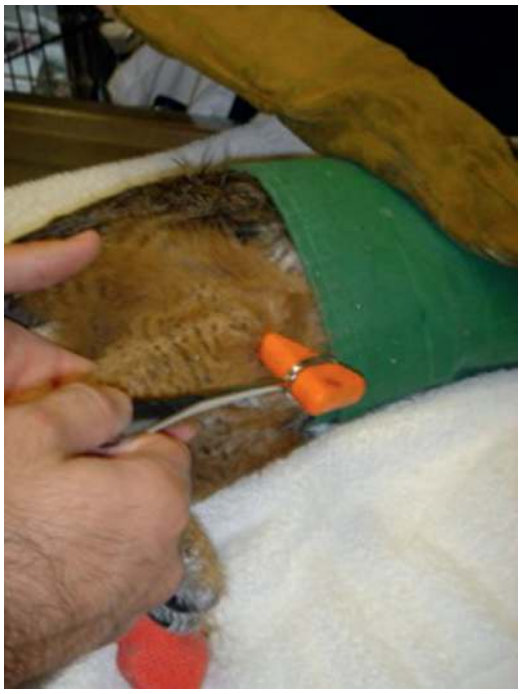


FIGURE 13-103 Great-horned owl (*Bubo virginianus*) with unilateral paralysis undergoing evaluation of the patellar reflex in the right limb during the neurological examination.

disorders. Ancillary neurodiagnostic tests include cerebrospinal fluid (CSF) analysis, myelography, computed tomography, MRI, scintigraphy, and electromyography. Additional tests to detect an infectious disease agent or antibodies and a toxin screen might be indicated based on the differential diagnoses.

Survey radiography should be used to evaluate the skull, axial skeleton, and vertebral column, particularly in cases of trauma. Sedation or general anesthesia facilitates exact positioning to yield the best image of the desired area. Orthogonal projections or three views of the skull should be obtained with the beam centered and collimated on the area of interest.

CSF analysis is most useful in characterizing infectious, neoplastic, or inflammatory disorders of the spinal cord or brain but is not performed routinely in birds. The subarachnoid space in birds is narrow, so that fluid collection from the cerebromedullary cistern is difficult. A proliferative network of blood vessels that participates in cerebrospinal production (choroid plexus) lies paramedially and bilaterally in the caudomedial aspect of the fourth ventricle. Large venous sinuses lie laterally on the interior surface of the occipital bones of the cranial cavity. The collection site must be approached at midline, or significant hemorrhage may occur. The atlanto-occipital joint is flexed at a 30-degree angle in a well-aligned, laterally recumbent patient and a 25 to 27 gauge needle with a translucent hub is placed at dorsal midline slightly caudal to the occipital protuberance, directed rostrally at a 45-degree angle to the horizontal axis of the head and advanced slowly through the skin at 1-mm intervals. The volume that can be collected ranges from 0.1 to 0.5 mL. This technique has been described in psittacine birds (Klappenbach, 1995). The author reported CSF of similar composition to mammals and mild deleterious effects in the animals.

Myelography is rarely used in clinical avian practice. Its limited use in birds is likely because of rare clinical indications, lack of familiarity with the technique, fear of iatrogenic injury to the spinal cord, and concern about potentially fatal complications of the procedure. The cerebellomedullary cistern is very small in many avian species and directly overlies a large venous plexus. It is considered by some authors that 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. This technique has been described in pigeons using the atlanto-occipital space for

BOX 13-12 Avian Neurologic Examination Form

Observation
Mentation: Bright/alert, obtunded, stuporous, comatose
Posture: Body is normal, recumbent, or opisthotonus and head has normal head tilt
Movement: Resting, normal, tremor, myoclonus seizures
Gait: Normal, paresis, ataxia, dysmetria, circling

Palpation
Head palpation
Spinal palpation
Limb palpation, LW, RW, LL, and RL

Cranial Nerves
I: Odor
II and V: Menace OS and OD
II and III: Pupillary light reflex OS and OD
III, IV, and VI: Strabismus OS and OD
III, IV, VI, and VIII: Oculocephalic reflex OS and OD
V: Palpebral reflex OS and OD
V: Beak tone
VI: Nictitans movement OS and OD
VIII: Head tilt L and R
  Nystagmus, horizontal vertical
  Positional, nonpositional
  Fast-phase L and R
IX, X, XI: Gag reflex
XII: Protrusion and retraction of the tongue

Postural Reactions
Conscious propioception L R
Hopping L R
Placing, optic, tactile R and L
Drop and flap R and L

Spinal Reflexes
Perineal
Patellar L and R
Leg withdrawal L and R
Wing withdrawal L and R

Sensory Evaluation
Feather retract

Assessment
1. Neurologically normal or abnormal
2. Neuroanatomical localization:
   a. Brain: Cerebrum, cerebellum, brainstem
   b. Spinal Cord: Cervical brachial plexus, thoracic lumbosacral plexus
   c. Peripheral nerve
   d. Neuromuscular
3. Differential diagnosis list
4. Diagnostic plan

LW, left wing; RW, right wing; LL, left leg; RL, right leg; OS, oculus sinister; OD, oculus dexter.

insertion of a 26-gauge 2.5-cm needle to deliver 0.2 mL iohexol over 1 minute, followed by elevation of the head for 5 minutes (Naeini *et al.*, 2006). Postmyelographic complications were not observed and the quality of the myelogram was considered acceptable. However, the cistern is relatively larger in pigeons than in some other avian species and accommodates a needle for injection of contrast medium. The fused vertebrae of the synsacrum, the glycogen body, and the absence of a cauda equina interfere with the typical mammalian technique of lumbosacral puncture for subarachnoid access necessitating a thoracolumbar approach. This technique has been evaluated in chickens using a 25-gauge 4-cm spinal needle at the site followed by administration of 0.8 to 1.2 mL/kg iohexol contrast medium into the subarachnoid space at 0.5 mL (Harr *et al.*, 1997). This technique showed some promise, but the authors were successful in only three of six animals, because they were unable to inject into the subarachnoid space or complications led to death in the other three cases.

Computed tomography (CT) imaging is best indicated to evaluate abnormalities in the skeletal structures and respiratory tract and has also been used in birds with neurologic disease because of large intracranial lesions or large spinal cord lesion; for other brain or spinal cord diseases additional techniques, such as MRI, may be necessary (van Zeeland and Schoemaker, 2011). Soft tissue structures are better visualized with contrast administration, especially where there is increased blood flow. A dose of 2.22 mg/kg of intravenous iodinated contrast material is recommended (Clippinger *et al.*, 2007). Examples of CNS disease diagnosed with the aid of CT imaging in birds include hydrocephalus, peripheral vestibular disease caused by otitis media (Delk *et al.*, 2014), spinal cord compression from vertebral invasion of bronchogenic carcinoma (Baumgartner *et al.*, 2008; Fig. 13-104), intervertebral disk rupture (Emerson, 1992), discospondylitis (Field *et al.*, 2012), and vertebral fracture. The limitations to the use of CT for imaging structures and organs are mainly dependent on the spatial resolution of the CT scanner and size of the patient.

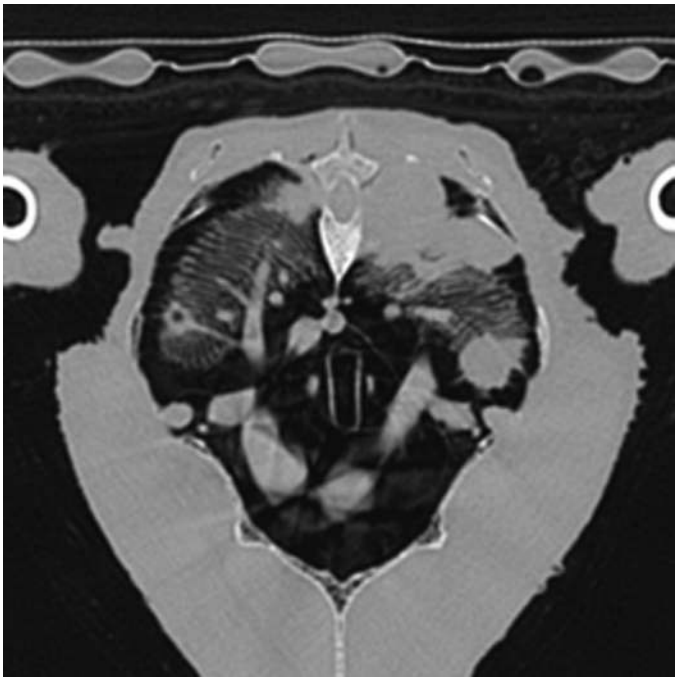


FIGURE 13-104 Transverse sagittal computed tomography image of the thorax of a hyacinth macaw (*Anodorhynchus hyacinthinus*) with bronchogenic carcinoma invading the thoracic vertebral bodies and causing compression of the spinal cord.

MRI is best indicated to evaluate soft tissue and has been found very useful for visualizing and evaluating various parts of the CNS, including the cerebral hemispheres, cerebellum, optic chiasm, brainstem, and spinal cord. Gadolinium, a paramagnetic intravenous contrast agent, can be used as a contrast medium for MRI studies (dose 0.25 mmol/kg). After administration of the compound via an intravenous catheter, the gadolinium is distributed throughout the body and changes the local magnetic field in tissues in which it is present in high concentrations (i.e., highly vascularized tissues), allowing delineation from the surrounding structures. Examples of diseases of the nervous system diagnosed with the aid of MRI in birds include ischemic infarct (Beaufrere *et al.*, 2011) and hemorrhagic infarct (Grosset *et al.*, 2014; Fig. 13-105), hydrocephalus (Fleming *et al.*, 2003; Keller *et al.*, 2011), peripheral vestibular disease caused by otitis media (Delk *et al.*, 2014), spinal cord trauma (Stauber *et al.*, 2007), peripheral nerve sheath neoplasia (Wernick *et al.*, 2014), spinal cord compression from hemivertebrae (Bradford *et al.*, 2008), and intervertebral disk rupture (Emerson, 1992).

Nuclear imaging or scintigraphy is a noninvasive method for evaluation of soft tissue and osseous structures associated with the nervous system. This technique involves intravenous administration of a small amount of a gamma-emitting radionuclide alone (e.g., Technetium-99m [99mTC]) that will allow it to accumulate in certain 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. This technique has been found useful for identifying spinal abnormalities in birds (Lung and Ackerman, 1993).

Electromyography (EMG), nerve conduction studies, and muscle biopsy are used to help differentiate transmission disorders, neuropathy, and myopathy in neuromuscular diseases. EMG is the study of the electrical activity of muscle by insertion of a recording electrode into the muscle. 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. Demonstrated electrical activity can be separated into three categories: insertional (associated with electrode movement), spontaneous (associated with resting muscle), and evoked (associated with electrical stimulation of nerves). EMG is useful in birds in the

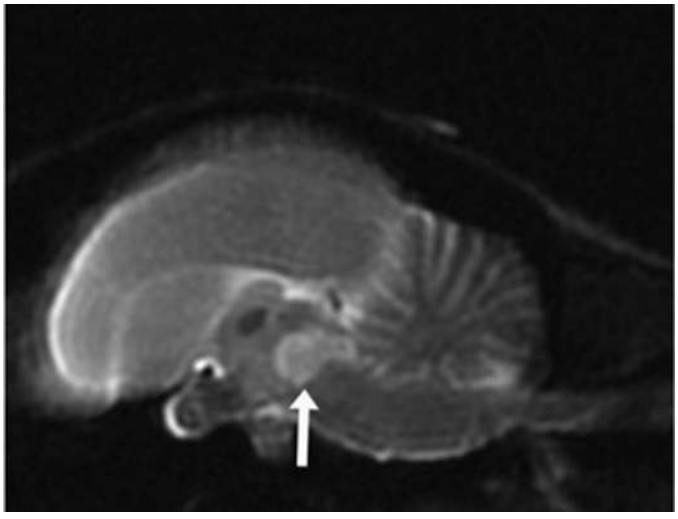


FIGURE 13-105 T2 sagittal magnetic resonance image of the brain of a blue and gold macaw (*Ara ararauna*) with a midbrain lesion corresponding to hemorrhage from a ruptured aneurysm.

diagnosis of traumatic brachial plexus injury (Shell *et al.*, 1993) and limb denervation (Holland and Jennings, 1997). Nerve conduction studies have become a simple and reliable test of peripheral nerve function (Table 13-19). The conduction velocity is measured between the two stimulus points on the nerve, 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. Reference ranges for the motor nerve conduction velocities have been established in barred owls and rheas (Clippinger *et al.*, 2000) and mallard ducks (Brenner *et al.*, 2008). Nerve conduction studies have been used in the diagnosis of peripheral demyelinating neuropathy secondary to lead toxicity (Platt *et al.*, 1999). Muscle biopsy is indicated to confirm, define, and possibly provide a cause for motor unit disease. In acute disease, a severely affected muscle is selected. With chronic disease, a muscle demonstrating only moderate changes is the best choice.

Treatment

Treatment of different disorders of the nervous system require addressing the primary cause targeting the therapy to a specific etiology while

TABLE 13-19 Cranial Nerves, Nerve Function, Applicable Tests to Evaluate Function, and Clinical Signs Associated with Dysfunction

Number and Name	Function	Test	Normal Response	Abnormal Response
I: Olfactory	Sensory: smell	Smelling alcohol	Aversion	No reaction
II: Optic	Sensory: vision	Menace response PLR	Blink or absent blink PLR present.	Absent blink PLR absent
III: Oculomotor	Motor: extrinsic ocular muscles and upper eyelid muscle Parasympathetic: intrinsic ocular muscle	Eyeball position and movement Menace response PLR	—	Ventrolateral deviation Drooped upper eyelid PLR absent with dilated pupil
IV: Trochlear	Motor extrinsic ocular muscle	Eye position, eye movement	Eye centered in palpebral fissure, eye moves in all directions	Dorsolateral deviation
V: Trigeminal Ophthalmic branch Maxillary branch Mandibular branch	Sensory: upper lid, forehead skin, nasal cavity, upper beak Sensory: both lids, hard palate, nasal cavity, lateral upper beak Motor: orbicularis, lower lid, (chewing) Sensory: lower beak skin, commissures	Response to touch Palpebral reflex Palpebral reflex Jaw palpation	Response to touch Palpebral reflex present Palpebral reflex present Closed mouth, good jaw tone	Lack of response Unable to blink Unable to blink Unable to close jaw
VI: Abducens	Motor: extrinsic ocular muscles, nictitans	Eye position, eye movement	Eye centered in palpebral fissure, nictitans moves	Medial deviation Nictitans immobility
VII: Facial	Motor: facial expression Sensory: taste Parasympathetic: most glands of the head	Taste alcohol	Facial symmetry Aversion	Facial asymmetry Poor taste Decreased secretions
VIII: Vestibulocochlear	Sensory: hearing, balance and coordination	Startle response	Startle reaction to handclap Oculocephalic reflex	Poor startle reaction Spontaneous nystagmus, prolonged or absent postrotatory nystagmus Head tilt, circling
IX: Glossopharyngeal	Sensory: taste and sensation in the tongue and trachea Motor: pharynx, larynx, crop and syrinx	Response to touch and taste of alcohol Gag reflex	Move tongue and aversion Swallowing	Poor taste and feel Poor gag reflex, dysphagia Voice change
X: Vagus	Sensory: larynx, pharynx, viscera Motor: larynx, pharynx, esophagus, crop Parasympathetic: glands, heart and viscera	Gag reflex	Swallowing	Poor gag reflex, dysphagia Voice change
XI: Spinal accessory	Motor: superficial neck muscles	Palpate for atrophy of muscles	Normal muscles and movement	Poor muscles or movement
XII: Hypoglossal	Motor: tongue, trachea, syrinx	Protrusion and retraction of tongue	Tongue protrudes symmetrically and retracts	Tongue deviates to the side, weak withdrawal

PLR, Pupillary light reflex.

TABLE 13-20 Dosages of Selected Drugs used in Birds

Drug	Species	Dose	Basis
Benzodiazepine			
Midazolam	Parrots, raptors	0.5-2 mg/kg IM, IV	EU
Diazepam	Parrots, raptors	0.5-2 mg/kg IV	EU
Antiepileptic			
Phenobarbital	African grey parrot	>20 mg/kg PO every 12 h	PK
		2-8 mg/kg IV	EU
KBr	Umbrella cockatoo	80 mg/kg PO every 24 h	EU
Levetiracetam	Hispaniolan Amazon parrot	5-10 mg/kg every 8-12 h	PK
Zonisamide	Hispaniolan Amazon parrot	20 mg/kg PO every 12 h	PK

EU, Empirical use; IM, intramuscular; IV, intravenous; PD, pharmacodynamics study; PK, pharmacokinetic study; PO, by mouth.

providing supportive care to recover from the primary lesions and preventing secondary damage.

Status epilepticus therapy is aimed at stopping the seizure activity and preventing the recurrence of seizures. Before starting antiepileptic therapy, a basic history from the owner must be collected with the signalment, previous history of seizures, current drug therapy, and history of trauma or exposure to toxins. Antiepileptic therapy should be started as soon as possible with benzodiazepines like midazolam (0.5 to 2 mg/kg IM or IV) or diazepam (0.5 to 1 mg/kg IV or IO). Benzodiazepines have a short duration of action needing frequent administration, and long-term usage leads to tolerance and decreased drug efficacy (Table 13-20). Additional diagnostic tests should be performed as soon as the patient is stable, and specific treatment for the underlying cause of the seizures should be initiated. Perches should be removed from the cage, soft bedding should be provided, and food and water should be placed in shallow bowls or spread on the cage floor if necessary. The maintenance antiepileptic drugs used for many years in avian medicine are phenobarbital and potassium bromide, but information in newer drugs like levetiracetam, zonisamide, and gabapentin have recently become available in birds. In epilepsies of unknown cause, antiepileptic treatment is introduced at the second convulsive epileptic seizure. An attempt at tapering off the antiepileptic drug can be done when the bird has been seizure free more than 2 years. The tapering of medication should be done slowly over several months before discontinuation of the treatment. If the epilepsy is structural, with an obvious demonstrated lesion, the epileptic focus rarely disappears spontaneously and the antiepileptic drugs are continued indefinitely.

Phenobarbital is a potent antiepileptic drug that is available in oral and parenteral forms. It is effective as an anticonvulsant in part because of its potentiating action on the inhibitory neurotransmitter gamma aminobutyric acid, which increases the seizure threshold and lowers the electrical activity of the seizure focus. The potential secondary hepatotoxicity necessitates close monitoring of liver parameters. Along with the common clinical side effects (sedation, polyuria, polydipsia, and polyphagia), its activation of the hepatic microsomal enzymes (p450 system) alters the metabolism of other antiepileptic drugs metabolized by the liver, such as levetiracetam and zonisamide. In some species phenobarbital might activate its own metabolism, necessitating multiple serum level measurements in the first 6 months of use and metabolic tolerance, forcing gradual increments of the dosage to maintain control, until higher end optimal therapeutic levels or hepatotoxicity are reached. Based on pharmacokinetic studies in African grey parrots, dosages higher than 20 mg/kg by mouth every 12 hours are needed in psittacine birds (Powers and Papich, 2011). Peak and trough serum levels should be checked 2 to 3 weeks after initiating therapy or changing the dose until significant therapeutic levels are reached (10 to 45 µg/mL) or seizures are controlled.

Potassium bromide can be used alone or in conjunction with phenobarbital when phenobarbital alone does not work to control the seizures adequately. The mechanism of action is through completion of the bromide ions with Cl^- transport, resulting in hyperpolarization. The side effects are similar to those of phenobarbital but without the potential for liver toxicity, and it does take a longer time for the body to eliminate the drug once it has been discontinued because of the long half-life. Based on clinical reports in psittacines, 80 mg/kg by mouth every 24 hours is recommended but pharmacokinetic studies are lacking (Heather, 2003). Peak and trough serum levels should be checked 2 to 3 weeks after initiating therapy or changing the dose until significant therapeutic levels are reached (1 to 3 mg/mL) or seizures are controlled, but it might take 2 to 3 months to reach a steady state. Levetiracetam is a newer antiepileptic drug available in oral and parenteral forms. The mechanism of action is not entirely understood, but it has been postulated to involve inhibition of excitatory neurotransmitter release by binding to the synaptic vesicle protein SV2A, modulating calcium-dependent exocytosis of neurotransmitters. It also suppresses the inhibitory effect of Zn^{2+} on gamma aminobutyric acid and glycine-gated currents. Based on pharmacokinetic studies in Hispaniolan Amazon parrots, 50 to 100 mg/kg by mouth every 8 to 12 hours is recommended in psittacines (Schnellbacher *et al.*, 2014). Zonisamide prevents seizure activity through several mechanisms of action including ion channel modulation, enhancement of neurotransmitters, and inhibition of carbonic anhydrase. Zonisamide is partially metabolized by the liver. Based on a study in Hispaniolan Amazon parrots, zonisamide administered at 20 mg/kg by mouth every 12 hours in psittacines is likely to achieve target plasma concentrations and be safe (Sanchez-Migallon, personal communication, April 2015). Peak and trough serum levels should be checked 1 week after initiating therapy or changing the dose until significant therapeutic levels are reached (10 to 40 µg/mL) or seizures are controlled. Other drugs like gabapentin and clonazepam have been used in clinical reports, but evidence regarding their efficacy is lacking (Beaufrere *et al.*, 2011). Long-term monitoring requires a detailed log of the number of seizures and duration, adjusting therapy with antiepileptic drugs as needed to control seizures.

Cerebrovascular accidents, such as hemorrhagic or ischemic strokes, require differentiation through MRI for specific medical treatment. Treatment of ischemic stroke includes thrombolytic agents, which are contraindicated in cases of hemorrhagic lesions. Surgical treatment for hemorrhagic stroke or vascular aneurysm might not be currently feasible in birds because of size limitations. Supportive treatment after a cerebral infarct or hemorrhage is aimed at maintaining adequate tissue perfusion and oxygenation and managing neurologic and nonneurologic complications.

Traumatic brain injury medical treatment is aimed at preventing or reducing the effects of secondary brain injury through maintaining adequate cerebral perfusion pressure, preventing or decreasing cerebral edema, and controlling intracranial pressure. The bird should be placed in a dark, quiet area with an environmental temperature of 23° C (73° F), because hyperthermia may contribute to greater posttraumatic brain damage. Elevation of the head (up to 30°) may decrease intracranial pressure by facilitating venous and cerebrospinal fluid drainage. The administration of oxygen in an oxygen cage is

recommended for moderate to severe cases. Slow administration over 15 minutes of mannitol (0.25 to 1 mg/kg IV) given every 4 to 6 hours as needed for up to three boluses should be considered, together with crystalloid fluids at 50% of the normal rate. Mannitol is contraindicated in hypovolemic patients. A clinical response to mannitol therapy may be seen within 5 to 10 minutes, and an improvement in mentation, posture, and pupillary signs indicates a positive response to mannitol therapy. If there is an insufficient response to the first dose of mannitol, an additional bolus can be given 40 to 60 minutes later. Lack of improvement with this therapy indicates that the brain damage will not be responsive to mannitol therapy and further therapy may not be warranted. The benefits that mannitol has on lowering intracranial pressure in the rest of the brain are likely to outweigh the concern that mannitol may result in worsening of intracranial hemorrhage. The side effects associated with repeated mannitol administration include hyperosmolarity, hypovolemia, and electrolyte disturbances (usually elevated sodium), therefore, adequate fluid therapy and monitoring are necessary. Hypertonic saline (7% to 7.8% NaCl) can be used instead of mannitol in hypovolemic or euvolemic patients to decrease intracranial pressure 3 to 5 ml/kg over 15 minutes, followed by crystalloid fluids at 50% of the normal rate. Hypertonic saline is contraindicated in hyponatremic patients. Slow administration of opioid drugs are recommended to treat pain and to avoid intracranial pressure elevation secondary to the sympathetic response to pain, but caution is warranted for patients unable to adequately ventilate. The use of nonsteroidal antiinflammatory drugs in cases of traumatic brain injury in birds is only recommended once the patient is stabilized. Corticosteroids are not recommended to treat traumatic brain injury.

For spinal cord trauma, like vertebral fractures, subluxations, and luxations, medical treatment is aimed at preventing further primary injury by careful handling and strict restriction of activity, and to prevent secondary injury to the spinal cord (Powers and Brofman, 2007). If needed, sedation or general anesthesia is recommended to facilitate handling and restrict activity. Intravenous fluid therapy is recommended to improve tissue perfusion and oxygenation. Opioid drugs are recommended to treat pain secondary to the trauma. Nonsteroidal antiinflammatories, gastrointestinal protectants, and nutritional support should be considered. Corticosteroids such as methylprednisolone sodium succinate have been used historically in the treatment of spinal cord injuries for their antiinflammatory and free radical scavenging properties. However, corticosteroids can cause severe side effects including hemorrhage, gastrointestinal ulceration, and immunosuppression in birds and are not currently recommended in most cases. Surgical stabilization is indicated for an unstable spinal fracture or subluxation with decompensating neurologic signs. Physical therapy consultation regarding an appropriate therapeutic treatment has the potential to expedite recovery and improve range of motion, muscle strength, proprioception, gait, and endurance, as well as correct movement dysfunction and decrease pain and inflammation. Physical therapy consists of examining and evaluating patient functional limitations, impairments, and disability, and may consist of active assisted range of motion; passive range of motion; active range of motion; stretching; and isometric, isotonic, and isokinetic strengthening; proprioceptive exercises; core stabilization; soft tissue massage; and other therapies. Any physical therapy routine should be tailored to the specific patient because all routines are not appropriate for all injury types or individuals, and treatment is adjusted based on patient response.

Bacterial CNS infections are treated with antibiotics with good CNS penetration including chloramphenicol, trimethoprim sulfa, metronidazole, fluoroquinolones, and third-generation cephalosporins. Fungal CNS infections are treated with antifungals with good CNS penetration like voriconazole, fluconazole, or amphotericin B. Parasitic CNS infection requires identification of the organism or empirical treatment with antiparasitic drugs against the presumptive organism. Medical treatment for discospondylitis consists of long-term antibiotic therapy against the causative organism or organisms cultured from the blood. Empirical therapy with amoxicillin with clavulanic acid or trimethoprim sulfa with a fluoroquinolone is recommended if there is no positive culture of the lesion or blood. Vestibular disease resulting from otitis media or otitis interna of bacterial etiology, long-term systemic antibiotics like third-generation cephalosporins and fluoroquinolones, and nonsteroidal antiinflammatory therapy should be considered. Surgery with removal of the affected tissue has also been reported to treat otitis media in a goose (Delk *et al.*, 2014).

REFERENCES

Aye PP, Morishita TY, Grimes S, et al: Encephalomalacia associated with vitamin E deficiency in commercially raised emus, *Avian Dis* 42(3):600–605, 1998.

Baumgartner WA, Sanchez-Migallon Guzman D, Hollibush S, et al: Bronchogenic adenocarcinoma in a hyacinth macaw (*Anodorhynchus hyacinthinus*), *J Avian Med Surg* 22(3):218–225, 2008.

Beaufrere H, Nevarez J, Gaschen L, et al: Diagnosis of presumed acute ischemic stroke and associated seizure management in a Congo African grey parrot, *J Am Vet Med Assoc* 239(1):122–128, 2011.

Bennet RA: Neurology. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine,principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Bergen DJ, Gartrell BD: Discospondylitis in a yellow-eyed penguin (*Megadyptes antipodes*), *J Avian Med Surg* 24(1):58–63, 2010.

Bermudez AJ, Johnson GC, Vanier MT, et al: Gangliosidosis in emus (*Dromaius novaehollandiae*), *Avian Dis* 39:293–303, 1995.

Bollinger TK, Mineau P, Wickstrom ML: Toxicity of sodium chloride to house sparrows (*Passer domesticus*), *J Wildl Dis* 41(2):363–370, 2005.

Bradford C, Bronson E, Kintner L, et al: Diagnosis and attempted surgical repair of hemivertebrae in an African penguin (*Spheniscus demersus*), *J Avian Med Surg* 22(4):331–335, 2008.

Brenner DJ, Larsen RS, Pascoe PJ, et al: Somatosensory evoked potentials and sensory nerve conduction velocities in the thoracic limb of mallard ducks (*Anas platyrhynchos*), *Am J Vet Res* 69(11):1476–1480, 2008.

Britt J, Paster M, Gonzales C: Lafora body neuropathy in a cockatiel, *Compan Anim Pract* 19:31–33, 1989.

Cai Z, Blumbergs PC, Finnie JW, et al: Selective vulnerability of peripheral nerves in avian riboflavin deficiency demyelinating polyneuropathy, *Vet Pathol* 46(1):88–96, 2009.

Campbell T: Crown vetch (*Coronilla varia*) poisoning in a budgerigar (*Melopsittacus undulatus*), *J Avian Med Surg* 20(2):97–100, 2006.

Carnarius M, Hafez HM, Henning A, et al: Clinical signs and diagnosis of thiamine deficiency in juvenile goshawks (*Accipiter gentilis*), *Vet Rec* 163(7):215–217, 2008.

Clippinger TL, Platt SR, Bennett RA, et al: Electrodiagnostic evaluation of peripheral nerve function in rheas and barred owls, *Am J Vet Res* 61(4):469–472, 2000.

Clippinger TL, Bennett RA, Platt SR: The avian neurologic examination and ancillary neurodiagnostic techniques,a review update, *Vet Clin North Am Exot Anim Pract* 10(3):803–836, 2007.

Crawford RD: Epileptiform seizures in domestic fowl, *J Hered* 61(5):185–188, 1970.

Delk KW, Mejia-Fava J, Jimenez DA, et al: Diagnostic imaging of peripheral vestibular disease in a Chinese goose (*Anser cygnoides*), *J Avian Med Surg* 28(1):31–37, 2014.

Dierenfeld E: Vitamin E deficiency in zoo reptiles, birds and ungulates, *J Zoo Wildl Med* 20(3):3–11, 1989.

Emerson CL, Eurell JAC, Brown MD, et al: Ruptured intervertebral disc in a juvenile king penguin (*Aptenodytes patagonica*), *J Zoo Wildl Med* 21(3):345–350, 1992.

Field CL, Beaufrere H, Wakamatsu N, et al: Discospondylitis caused by *Staphylococcus aureus* in an African black-footed penguin (*Spheniscus demersus*), *J Avian Med Surg* 26(4):232–238, 2012.
Fleming GJ, Lester NV, Stevenson R, et al: High field strength (4.7T) magnetic resonance imaging of hydrocephalus in an African Grey parrot (*Psittacus erithacus*), *Vet Radiol Ultrasound* 44(5):542–545, 2003.
Fraga-Manteiga E, Eatwell K, Smith S, et al: Traumatic atlanto-occipital subluxation and cranial cervical block vertebrae in a Golden Eagle (*Aquila chrysaetos*), *Vlaams Diergen Tijds* 82:211–215, 2013.
Fredholm DV, Carpenter JW, Schumacher LL, et al: Pulmonary adenocarcinoma with osseous metastasis and secondary paresis in a blue and gold macaw (*Ara ararauna*), *J Zoo Wildl Med* 43(4):909–913, 2012.
Gancz A, Malka S, Sandmeyer L, et al: Horner's Syndrome in a red-bellied Parrot (*Poicephalus rufiventris*), *J Avian Med Surg* 19(1):30–34, 2005.
Greenacre C: Leg paresis in a cockatoo caused by aspergillosis, *J Zoo Wildl Med* 23(1):122–126, 1992.
Gries C, Scott M: The pathology of pyridoxine deficiency in chicks, *J Nutr* 102(10):1259–1267, 1972.
Grosset C, Sanchez-Migallon Guzman D, Keating MK, et al: Central vestibular disease in a blue and gold macaw (*Ara ararauna*) with cerebral infarction and hemorrhage, *J Avian Med Surg* 28(2):132–142, 2014.
Harr KE, Kollias GV 2nd, Rendano V, et al: A myelographic technique for avian species, *Vet Radiol Ultrasound* 38(3):187–192, 1997.
Heather B: Management with potassium bromide of seizures of undetermined origin in an umbrella cockatoo, *Exot DVM* 5.4:7–8, 2003.
Hislop RI, Whittle TE: Nutritional encephalomalacia in the chicken, *World's Poult Sci J* 23(2):133–150, 1967.
Holland M, Jennings D: Use of electromyography in seven injured wild birds, *J Am Vet Med Assoc* 211(5):607–609, 1997.
Holz PH, Phelan JR: Thiamine deficiency in honeyeaters, *J Avian Med Surg* 16(1):21–25, 2000.
Hunter B, Wobeser G: Encephalopathy and peripheral neuropathy in lead-poisoned mallard ducks, *Avian Dis* 24(1):169–178, 1980.
Johnston HA, Lindstrom JG, Oglesbee M: Communicating hydrocephalus in a mature Goffin's cockatoo (*Cacatua goffini*), *J Avian Med Surg* 20(3):180–184, 2006.
Jortner BS, Meldrum JB, Domermuth CH, et al: Encephalomalacia associated with hypovitaminosis E in turkey poults, *Avian Dis* 29(2):488–498, 1985.
Keller KA, Sanchez-Migallon Guzman D, Muthuswamy A, et al: Hydrocephalus in a yellow-headed Amazon parrot (*Amazona ochrocephala oratrix*), *J Avian Med Surg* 25(3):216–224, 2011.
Kirchgessner MS, Tully TN Jr, Nevarez J, et al: Magnesium therapy in a hypocalcemic African grey parrot (*Psittacus erithacus*), *J Avian Med Surg* 26(1):17–21, 2012.
Klappenbach K: Cerebral spinal fluid analysis in psittacines. *Proceedings of the Association of Avian Veterianrians*, pp 39–42, 1995.
Landsberg J, Vargo G, Flewelling L, et al: Avian biotoxins. In Thomas NJ, Hunter DB, Atkinson CT, editors: *Infectious diseases of wild birds*, Ames, IA, 2007, Blackwell Publishing.
Langohr IM, Garner MM, Kiupel M: Somatotroph pituitary tumors in budgerigars (*Melopsittacus undulatus*), *Vet Pathol* 49(3):503–507, 2012.
Lightfoot TL, Yeager JM: Pet bird toxicity and related environmental concerns, *Vet Clin North Am Exot Anim Pract* 11(2):229–259, 2008.
Lung NP, Ackerman N: Scintigraphy as a tool in avian orthopedic diagnosis. *Proceedings of the American Association of Zoo Veterinarians*, p 45, 1993.
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.
McDonald LJ: Hypocalcemic seizures in an African grey parrot, *Can Vet J* 29(11):928–930, 1988.
Monks D, Fisher M, Forbes NA: *Ixodes frontalis* and avian tick-related syndrome in the United Kingdom, *J Small Anim Pract* 47(8):451–455, 2006.
Naeini AT, Dadras H, Naeini BA: Myelography in the pigeon (*Columba livia*), *J Avian Med Surg* 20(1):27–30, 2006.
Olkowski AA, Classen HL: The study of thiamine requirement in broiler chickens, *Int J Vitam Nutr Res* 66(4):332–341, 1996.
Platt SR, Helmick KE, Graham J, et al: Peripheral neuropathy in a turkey vulture with lead toxicosis, *J Am Vet Med Assoc* 214(8):1218–1220, 1200, 1999.
Powers L, Brofman P: Managing brain and spinal cord trauma in birds. *Proceedings of the Annual Conference of the Association of Avian Veterinarians*, pp 59–65, 2007.
Powers LV, Papich MG: Pharmacokinetics of orally administered phenobarbital in African grey parrots (*Psittacus erithacus erithacus*), *J Vet Pharmacol Ther* 34(6):615–617, 2011.
Pruthi AK, Verma PC: Sequential pathological changes in experimental thiamine deficiency in Japanese quail (*Coturnix coturnix japonica*), *Ind J Vet Pathol* 24(1):16–18, 2000.
Rocke T, Bollinger T: Avian botulism. In Thomas NJ, Hunter DB, Atkinson CT, editors: *Infectious diseases of wild birds*, Ames, IA, 2007, Blackwell Publishing.
Roffe TJ, Stroud RK, Windingstad RM: Suspected fusariomycotoxicosis in sandhill cranes (*Grus canadensis*). Clinical and pathological findings, *Avian Dis* 33:451–457, 1989.
Romagnano A, Mashima TY, Barnes HJ, et al: Pituitary adenoma in an Amazon parrot, *J Avian Med Surg* 263–270, 1995.
Schmidt R, Reavill D, Phalen D: Nervous system. In *Pathology of pet and aviary birds*, Ames, IA, 2003, Ames State Press, pp 165–176.
Schnellbacher RD, Beaufrère H, Arnold R, et al: Pharmacokinetics of levetriacetam in healthy Hispaniolan Amazon parrots (*Amazona ventralis*) following oral administration of a single dose, *J Avian Med Surg* 28(3):193–200, 2014.
Shell L, Richards M, Saunders G: Brachial plexus injury in two red-tailed hawks (*Buteo jamaicensis*), *J Wildl Dis* 29(1):177–179, 1993.
Simova-Curd S, Nitzl D, Mayer J, et al: Clinical approach to renal neoplasia in budgerigars (*Melopsittacus undulatus*), *J Small Anim Pract* 47(9):504–511, 2006.
Smith GR, Oliphant JC, Evans ED: Diagnosis of botulism in water birds, *Vet Rec* 112(19):457–458, 1983.
Spalding MG, Kollias GV, Mays MB, et al: Hepatic encephalopathy associated with hemochromatosis in a toco toucan, *J Am Vet Med Assoc* 189(9):1122–1123, 1986.
Stauber E, Holmes S, DeGhetto DL, et al: Magnetic resonance imaging is superior to radiography in evaluating spinal cord trauma in three bald eagles (*Haliaeetus leucocephalus*), *J Avian Med Surg* 21(3):196–200, 2007.
Stedman NL, Brown TP, Rowland GN: Intravascular cartilaginous emboli in the spinal cord of turkeys, *Avian Dis* 42(2):423–428, 1998.
Swank RL: Avian thiamine deficiency, a correlation of the pathology and clinical behavior, *J Exp Med* 71:683–702, 1940.
van Zeeland Y, Schoemaker NJ: Newest imaging techniques in avian medicine. *Proceedings of the European Association of Avian Veterinarians*, 2011.
Veltri CJ, Klem D: Comparison of fatal bird injuries from collisions with towers and windows, *J Field Ornithol* 76:127–133, 2005.
Wack RF, Lindstrom JG, Graham DL, et al: Internal hydrocephalus in an African grey parrot (*Psittacus erithacus timneh*), *J Assoc Avian Vet* 3(2):94–96, 1989.
Wada Y, Kondo H, Itakura C: Peripheral neuropathy of dietary riboflavin deficiency in racing pigeons, *J Vet Med Sci* 58(2):161–163, 1996.
Ward F: Thiamine deficiency in a peregrine falcon, *J Am Vet Med Assoc* 159(5):599–601, 1971.
Wernick MB, Dennler M, Beckmann K, et al: Peripheral nerve sheath tumor in a subadult golden eagle (*Aquila chrysaetos*), *J Avian Med Surg* 28(1):57–63, 2014.
Wiley FE, Twiner MJ, Leighfield TA, et al: An extract of *Hydrilla verticillata* and associated epiphytes induces avian vacuolar myelinopathy in laboratory mallards, *Environ Toxicol* 24(4):362–368, 2009.
Williams MC, Smith MC: Toxicity of *Kalanchoe* spp. to chicks, *Am J Vet Res* 45(3):543–546, 1984.
Wyatt RD, Colwell WM, Hamilton PB, et al: Neural disturbances in chickens caused by dietary T-2 toxin, *Appl Microbiol* 26(5):757–761, 1973.

Infectious Diseases

VIRAL DISEASES

Ulrich Wernery

Diseases	Family Subfamily	Genus	Pathology	Diagnosis
Infectious bursal disease (IBD; Gumboro disease)	Birnaviridae	*Avibirnavirus*	Ostriches, ducks, pheasants, turkey, chickens 3-6 weeks old: bursa severely enlarged, striped, edematous, morbidity 100%, mortality 30%, immunodeficient birds	Histopathology, RT-PCR, EM virus isolation on CAM of fertile eggs, Serology: AGID, FA
Lymphoid leukosis (avian leukosis)	Retroviridae	*Alpharetrovirus* (type C retroviruses)	Egg transmission, peak mortality in 6- to 9-month-old birds, avian leukosis sarcoma (Rous sarcoma) virus in chickens, pheasants, partridges, quail, fowl, turkeys, bustards, infiltration of lymphoid cells into many organs	Pathology, histopathology, PCR, ELISA Serology: VN, IFA, ELISA
Reticuloendotheliosis		Gammaretrovirus	Ducks, geese, pheasants, quail, turkeys, chickens: immunodepression, runting syndrome with abnormal feathering, atrophy of bursa and thymus, anemia, formation of neoplasias of lymphoreticular system	Gross and histopathology, RT-PCR Serology: ELISA, VN
Rotavirus Infection	Reoviridae (respiratory, enteric, orphan)	*Orthoreovirus*	Viral arthritis, 11 serotypes affecting Psittaciformes, pheasants, pigeons, raptors, geese, ducks, chickens, turkeys, and quail causing: arthritis, tenosynovitis, respiratory disease, hepatic necrosis, pericarditis, diarrhea, bursal atrophy, feather abnormalities, orthovirus was isolated from ostrich closely related to chicken orthovirus 138	RT-PCR, virus isolation in yolk sac of fertile eggs, fowl tissue culture Serology: AGID, but many birds are positive
		Rotavirus	Chickens and turkeys, enteritis and diarrhea in young birds, mortality 50%, also ducks, pigeons, lovebirds, pheasants	RT-PCR, Ag ELISA, EM feces, CPE in embryo liver cells
		Orbivirus	Transmitted by ticks, clinical signs of disease only described in cockatiels, budgerigars showing enlarged liver, spleen, and diarrhea	

Diseases	Family Subfamily	Genus	Pathology	Diagnosis
Adenovirus Infection	Adenoviridae	*Aviadenovirus I*	12 serologically distinct adenoviruses with the following pathology: quail bronchitis; inclusion body hepatitis of chickens; turkey viral hepatitis; disease in pigeons, budgerigars, ducks, geese, guinea fowl; splenomegaly of chickens; marble spleen disease of pheasants, inclusion body hepatitis, splenomegaly, enteritis in raptors including eagle owl caused by Aviadenovirus I, serotypes 1 and 4 and Siadenovirus	Pathology with intranuclear inclusion bodies PCR
Hemorrhagic enteritis of turkeys		*Siadenovirus (Adenovirus II)*	Acute onset with bloody droppings; mortality up to 10% with secondary intestinal hemorrhages, in turkeys over 4 weeks old, spleen enlarged, mottled, hemorrhages on tip of villi	Histopathology, PCR, AGID antigen detection Serology: AGID
Egg Drop Syndrome		Duck *Adenovirus A* (Atadenovirus)	Natural hosts are ducks and geese, introduced to the unnatural host, the chicken through Marek's disease vaccine? Egg production drops by up to 40%, soft-shell or shell-less eggs	PCR, FA, ELISA Serologic screening with HAI, VN when defective eggs
Infectious bronchitis	Coronaviridae	*Gammacoronavirus*	Chicken less than 6 weeks old: severe respiratory disease with secondary bacterial infection, Adults: drop in egg production, soft-shelled eggs, broiler glomerular nephritis	RT-PCR, FA from tracheal scrapings, virus injection into allantoic cavity of fertile eggs: dwarfing of embryos Serology: ELISA, AGID, VN
Blue comb (corona enteritis of turkeys)			Acute, highly contagious disease, up to 100% mortality in young poults, diarrhea, weight loss, cyanosis of head (blue comb)	Pathology, RT-PCR, virus isolation
Epidemic Tremor (avian encephalomyelitis) Viral hepatitis of turkey Duck virus hepatitis	Picornaviridae	*Tremovirus* Unassigned	Chicks, ducks, pheasants, turkeys, quail, most common in 7- to 10-day-old chicks, ataxia, tremor of head, mortality up to 50%, cockatoo enteritis, duck and turkey enteritis	Clinical signs, histopathology, RT-PCR, FA, virus isolation in yolk sac
		Avihepatovirus (Duck hepatitis A virus)	Highly contagious, mortality up to 95%, liver lesions covered by hemorrhagic foci, waterfowl are carriers	Pathology, RT-PCR, FA, virus isolation

Continued

Diseases	Family Subfamily	Genus	Pathology	Diagnosis
Chicken anemia virus infection	Circoviridae	*Gyrovirus*	Only chicken under 1 week of age without maternal antibodies, anemia, anorexia, hypoplasia of lymphoid organs, increased mortality rate	Clinical signs, gross and histopathology, PCR Serology: VN, IFA, ELISA
Beak and feather disease		*Circovirus*	Affects only psittacine species, debilitating, immunosuppressive disease of young psittacine birds, particularly cockatoos In the UAE African grey parrots show a prevalence of 58% followed by parakeets 44% and macaws 18%	PCR from EDTA blood and/or feathers
Papillomavirus infection	Papillomavirinae Polyomavirinae	*Papillomavirus* *Polyomavirus*	Benign skin tumors (papillomas), generalized infection, budgerigar fledging disease and other psittacines, French molt	Histopathology, immune histology Basophilic nuclear inclusions in liver and spleen, PCR
Goose parvovirus infection (goose viral hepatitis, Derzsy's disease)	Parvoviridae	*Dependovirus*	Highly contagious disease in young geese less than 4 weeks old and Muscovy ducklings, serofibrinous pericarditis, perihepatitis and excess fluid in abdominal cavity	Gross and histopathology, PCR, virus isolation in fertile goose eggs Serology: AGID, VN
West Nile virus infection Turkey meningoencephalitis virus	Flaviviridae	*Flavivirus* *Bengazavirus*	Mosquitoborne disease in turkeys in Israel and South Africa, progressive paresia and paralysis, wide spread Arbovirus infections including raptors with splenomegaly, myocarditis, meningoencephalitis, successful vaccination with equine vaccine, Bengazavirus causes lack of motor coordination in pheasants and partridges in Spain	PCR, virus isolation in embryonated eggs, tissue culture Serology: PCR, ELISA, VN
Rabies	Rhabdoviridae	*Lyssavirus*	No clinical changes associated with rabies have been observed in naturally infected birds	
Astrovirus Infection	Astroviridae	*Avastrovirus*	Fetal hepatitis in young ducklings, chickens, and turkeys, astrovirus produces enteritis	RT-PCR, EM, ELISA, virus isolation in embryonated eggs or chicken cell lines

Diseases	Family Subfamily	Genus	Pathology	Diagnosis
Togavirus Infection	Togaviridae	*Alphavirus* Eastern equine encephalitis virus Western equine encephalitis virus Species-specific encephalitis disease	Arthropod vectors (arboviruses), all avian species are considered susceptible: severe enteritis and neurologic signs Pheasants, emus, ostriches, chukars, English sparrows, chickens, turkeys, raptors(?): ruffled feathers, somnolence, depression, weakness, incoordination, torticollis, paresis, paralysis Avian viral serositis in macaws and ring-necked parakeets: enlarged, yellow liver, congested edematous lungs and fluid in abdomen	Virus isolation in cell culture, embryonated hens eggs, infant mice, PCR, IF Serology: ELISA IgM and IgG, VN, CFT
Borna disease Proventricular dilatation disease	Bornaviridae	*Bornavirus* (avian bornavirus)	It infects brains and nerves of horses but also "deadens" the nerve cells that control esophagus and proventriculum in birds	Clinical signs, histopathology, PCR, Panviral microarray

INFLUENZA

Etiology

The influenza virus belongs to the family Orthomyxoviridae, which is divided into three types (Fig. 14-1). The pleomorphic viruses are enveloped and measure 60 to 120 µm in diameter. The genome consists of single-stranded ribonucleic acid. Influenza A virus is the only type of veterinary significance. Influenza viruses possess two surface antigens that are important to identify and control. The most important is hemagglutinin (H), which is responsible for the virus's ability to agglutinate erythrocytes and to attach and penetrate host cells. The other surface antigen is neuraminidase (N), which is involved in the release of newly formed viral particles from host cells. Influenza A viruses are classified into 16 antigenically distinct HA (H1-H16) and 9 NA (N1-N9) subtypes. Few subtype combinations have been isolated from mammalian species, but all subtypes in most of the possible combinations have been found in birds (World Health Organization, 1980; Spielman *et al.*, 2004). Subtypes H5, H7, and H9 possess a high pandemic potential (Webster and Hulse, 2004) and highly pathogenic avian influenza (HPAI) isolates are restricted to subtypes H5 and N7. Aquatic birds, particularly ducks, are the reservoirs of influenza A virus, providing a source of novel subtypes that may one day infect mammals. The virus is often disseminated by migratory birds. It replicates in the intestinal tract of birds, which can result in fecal–oral transmission (Markey *et al.*, 2013). Detection of the virus of the H5 and H7 serotypes in avian species is notifiable to the World Organization for Animal Health (OIE).

Influenza viruses are relatively stable when outside the host, particularly in pond or lake water. In a cool environment, the virus remains infectious in feces for over a month. It can be destroyed in minutes by extremes in pH, heating to 56° C, exposure to sunlight, and by most detergents and disinfectants.

Distribution

The best known disease caused by an avian influenza virus (AIV) is fowl plague, which is now known as HPAI and is a listed disease by the OIE. Work with HPAI isolates should be performed in high-security laboratories. Fowl plague was first reported in 1878 and 1901, causing

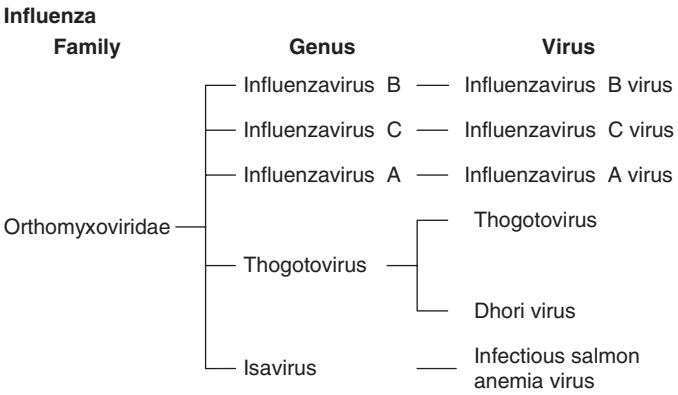


FIGURE 14-1 Classification of orthomyxoviruses.

severe losses in poultry, and in 1955 it was identified as AIV. From the 1970s onward, surveillance indicated the ubiquitous presence of AIV in waterfowl and the risk these birds pose to commercial chicken industries. In 1983 there was a large epidemic in the broiler industry of Pennsylvania, which cost $60 million to control, and in 1990 there was a severe outbreak in the Mexican broiler industry. In 1997 a highly pathogenic avian influenza virus (HPAIV) emerged in Hong Kong that killed approximately 150 million birds in Asia up to the beginning of 2005. The loss of their chickens left many farmers deep in debt, and the Asian poultry industry lost $15 billion by the end of 2004. The virus spread westward and reached Russia, where it killed 120,000 birds by mid-2005. In April 2005, this H5N1 strain also transferred to pigs and killed 147 of 418 tigers in Thailand in 2002 after eating infected raw chicken. The pandemic caused by the avian-origin HPAI H5N1 spread to Europe and Africa and remains endemic in Cambodia, Bangladesh, China, Egypt, and Vietnam. Wild birds played a potential role in disseminating the disease westward.

Since its detection in 1997, more than 17 reassortments have occurred that have claimed more than 70 human lives in Asia. An avian influenza outbreak caused by another HPAI virus, type H7N7, occurred in 2003 in the Netherlands. This virus was considerably less pathogenic

for humans than AH5N1 and killed only one person, but millions of chickens were destroyed to contain the disease. The latest global concern is a poultry-origin low-pathogenicity avian influenza (LPAI) H7N9 strain, which was recently detected in healthy pigeons. This strain has caused fatalities in humans in China.

Epizootiology

Severe acute respiratory syndrome (SARS) and Middle East respiratory syndrome (MERS) exploded onto the world's consciousness in March 2003 and 2012, respectively, and made us aware that zoonotic diseases will continue to increase because of closer interactions between people and animals. Influenza originates in aquatic birds and is carried by migratory ducks, geese, and herons, usually without harm. Not only are wild birds playing a significant role in spreading the disease, but trade through live markets and movement of domestic waterfowl are also very influential in the spread of the virus (Sinus *et al.*, 2005). It is estimated that twice a year 50 billion birds migrate around the globe, carrying viruses to all corners of the world. As the birds migrate, they can pass the viruses to domestic birds, such as chickens, via feces or during competitions for food, territory, and water (Garrett, 2005). The influenza virus does not undergo any significant change in these migratory birds (Osterholm, 2005), but when the virus is transmitted from wild to domesticated birds it undergoes changes that allow it to infect humans, pigs, and potentially other mammals. Once in the lung cells of a mammalian host, the virus can "reassort," or mix genes with influenza viruses that are already present. This process can lead to an entirely new viral strain capable of sustained human-to-human transmission.

Whether or not this particular H5N1 influenza strain will mutate into a human-to-human pandemic form is unclear, but scientific evidence points to the likelihood that such an event will take place, perhaps soon. H5N1 has now reached the Urals and migratory birds have transported it to Europe. Not only migratory birds may spread the virus to continents but also hunting falcons and their accompanying life prey birds, like houbara bustards may spread the disease to other countries when returning home (Naguib *et al.*, 2015). Influenza virus has not only been isolated from aquatic birds, which serve as a reservoir of the virus, but also from companions in aviaries and zoologic parks. It is transmitted through direct contact with feces and aerosol from infected birds, and contaminated water in overcrowded ponds or lakes is also considered to be an important source of the virus (Ritchie, 1995).

Scientists have observed that influenza virus can switch from the LPAI phenotype, which is common in wild birds and poultry, to HPAI phenotypes. This is achieved by the introduction of basic amino acid residues into the HAO cleavage site (Munster *et al.*, 2005). Because HPAI outbreaks in poultry originate with LPAI viruses present in waterfowl, influenza A virus surveillance in wild birds could function as an early warning system for HPAI outbreaks. Compulsory active surveillance of domestic bird flocks focusing on H5 and H7 AIV has been implemented in the European Union, and results from these serologic surveys clearly show that domestic ducks and geese have the highest seroprevalence for H5 and H7.

Minor antigenic and genetic diversity were observed among H genes of mallard influenza A isolates and those of HPAI viral strains. These new findings indicate that influenza A surveillance in wild birds provides an excellent opportunity for pandemic preparation, production of vaccines, and development of valid diagnostic tests.

Clinical Features

The virus may cause high mortality in some avian species and no clinical signs in others. Morbidity and mortality vary widely with the species of bird and strain of infecting virus; therefore, the OIE differentiates between HPAIV and low-pathogenicity avian influenza virus. HPAIV is an OIE list A disease, and both HPAI and LPAI isolates of subtypes H5 and H7 are notifiable to the OIE. When clinical changes are present, they may include mild to severe respiratory signs, anorexia, depression, decreased egg production, and diarrhea (Figs. 14-2 through 14-6). Highly virulent strains such as H5N1 damage endothelial cells, resulting in bleeding disorders, which are fatal to domestic poultry. In raptors H5N1 caused mainly encephalitis (van den Brand et al., 2015), and in other avian species like houbara bustards, patridges, sea gulls and quails, necrotizing pacreatitis was observed. Over the last 10 years 58 influenza viral strains were isolated from different bird species in the United Arab Emirates (Table 14-1).

Avians affected can show clinical signs and no disease. In two different bustard species, for example, the clinical signs were dyspnea, lethargy, discharge from eyes and nares, severe tracheitis, pneumonia, and pancreatitis (Wernery *et al.*, 2004). An H7N3 influenza A strain that was isolated from a healthy peregrine falcon (*Falco peregrinus*) induced severe disease in 6-week-old-chickens (see Fig. 14-5).

Diagnosis

Virus isolation is essential to establish the cause of an outbreak and to assess objectively the virulence of the causative virus. Virus is best isolated from cloacal swabs, but ground tissue specimens may also be inoculated into the allantoic cavity of 10- to 12-day-old embryonated chicken eggs and onto monolayers of cultures of chicken embryo fibroblasts. Fluid from the allantoic cavity and from cell cultures is subjected to hemagglutination and neuraminidase inhibition testing

FIGURE 14-2 Clear discharge from eyes and nostrils of a houbara bustard with influenza A infection. (Courtesy Dr. L. Molnar.)

FIGURE 14-3 Severe tracheitis with pus production in a houbara bustard with influenza A infection.

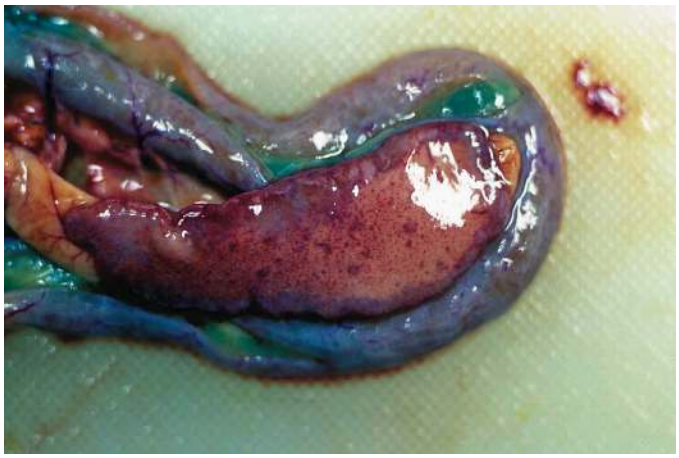


FIGURE 14-4 Pancreatitis in a houbara bustard caused by influenza A infection. H7N1 was identified from all these cases. (Courtesy Dr. J. Kinne.)

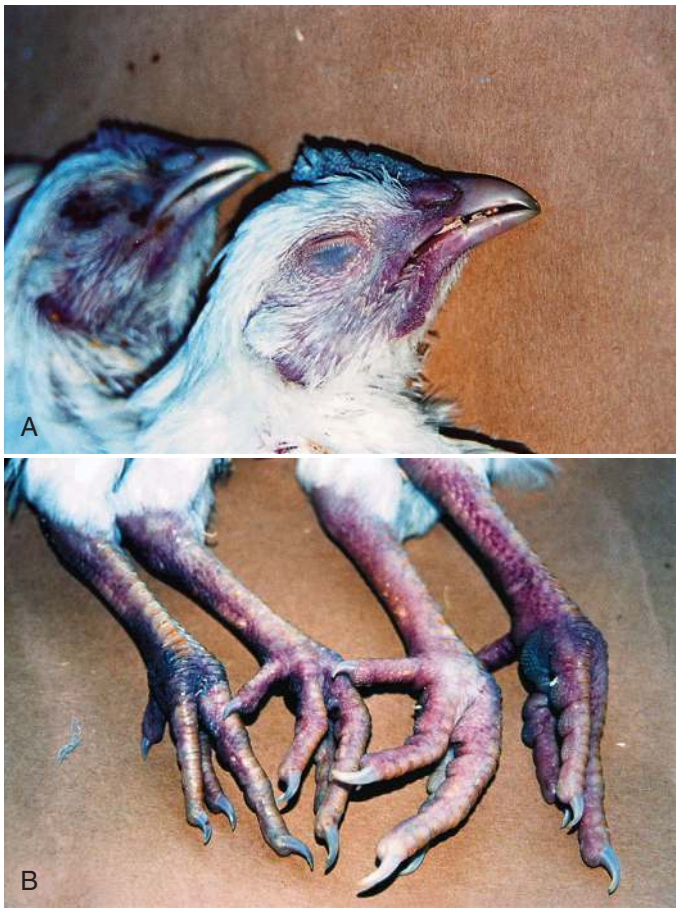


FIGURE 14-5 **(A, B),** Acute cyanosis of wattles, comb, and legs 3 days after infection of chickens with a falcon H7N3 strain. (Courtesy R. Manvell.)

FIGURE 14-6 Flu A and B enzyme-linked immunosorbent assay (ELISA) results showing positive controls (*C*) in both and a positive result for Flu A (*T*). Positive Flu A ELISA was from an oropharyngeal swab taken from a quail with H9N2 infection.

TABLE 14-1 Influenza Virus Strains Isolated from Different Avian Species over the Last 10 Years in the United Arab Emirates

Species	Avian Influenza Virus	Number of Isolates
Chicken	H9N2	8
Falcon	H5N1	4
	H7N3	5
	H9N2	1
	H7N1	1
Bustard	H9N2	10
	H7N1	7
	H1	1
	H5N1	1
	H10	1
Quail	H9N2	8
	H9N1	1
Stone curlew	H9N2	3
	H5N1	1
Plover	H9N2	1
Dove	H11	1
Pheasant	H9N2	2
Duck	H5N1	1
Sea gull	H5N1	1

using reference influenza A antisera. Reverse-transcription polymerase chain reaction (PCR) techniques and real-time PCR, which are rapid assays, have been developed for the detection and subtype identification of virus in clinical samples. Commercial antigen-detection immunoassays have also been used for fast diagnosis. These tests detect any influenza A virus and are generally based on monoclonal antibodies. They should be used for screening because they lack sensitivity (see Fig. 14-6). In several occasions the test was positive only after culture in embryonated eggs or in cells. Serologic testing for antibodies to influenza virus can be performed using different serologic methods like the agar gel immunodiffusion test, hemagglutination inhibition test, or competitive enzyme-linked immunosorbent assay.

Prevention and Control

Avian influenza is not eradicable and prevention and control are the only realistic goals. The Hong Kong outbreak of H5N1 was controlled by extensive culling together with well-managed surveillance and vaccination.

The following recommendations should be initiated:

- Monitor movement of poultry between farms and markets.
- Monitor birds in live markets and exports/imports.
- Improve biosecurity measures (e.g., prevent contact with wild aquatic birds).
- Separate land-based poultry, pigs, and aquatic avian species in farms and markets.
- Close live bird markets and keep all poultry indoors while HPAIV is circulating in the region.
- Conduct serologic and other epidemiologic studies in wild birds to determine whether HPAIV has become established in wild populations.
- Only allow controlled, effective vaccination in response to virulent outbreaks.

Attenuated subtype-specific vaccines are used in domestic fowl; however, these vaccines have limited application because of the speed of antigenic drift and reassortment.

REFERENCES

Garrett L: The next pandemic? *Foreign Affairs* 84(4):3–23, 2005.

Markey B, Leonard F, Archambault M, et al: *Clinical veterinary microbiology*, ed 2, London, 2013, Mosby Elsevier.

Munster VJ, Wallensten A, Baas C, et al: Mallards and highly pathogenic avian influenza ancestral viruses, northern Europe, *Emerg Infect Dis* 11:1545–1551, 2005.

Naguib MM, Kinne J, Chen H, et al: Emergence of reassorted highly pathogenic avian influenza H5N1 in Dubai, *J Gen Virol* in press, 2015.

Osterholm MT: Preparing for the next pandemic, *Foreign Affairs* 84(4):24–37, 2005.

Ritchie W: *Avian viruses*, Lake Worth, FL, 1995, Wingers Publishing.

Sinus LD, Domenech J, Benigno C, et al: Origin and evolution of highly pathogenic H5N1 avian influenza in Asia, *Vet Rec* 157:159–164, 2005.

Spielman D, Mauroo N, Kinoshita R, et al: Wild bird species and the ecology of virulent avian influenza. *Proceedings of the American Association of Zoo Veterinarians, American Association of Wildlife Veterinarians and Wildlife Disease Association*, pp 40–45, San Diego, CA, 2004.

van den Brand JMA, Krone O, Wolf PU, et al: Host-specific exposure and fatal neurologic disease in wild raptors from highly pathogenic avian influenza virus H5N1 during 2006 outbreak in Germany, *Vet Res* 46:24, 2015.

Webster RG, Hulse DJ: Microbial adaptation and change: avian influenza, *Rev Sci Tech* 23:453–465, 2004.

Wernery R, Wernery U, Kinne J, et al: *Colour atlas of falcon medicine*, Hannover, Germany, 2004, Schlütersch.

World Health Organization: A revision of the system of nomenclature for influenza viruses: a WHO memorandum, *Bull World Health Organ* 58:585–591, 1980.

NEWCASTLE DISEASE

Etiology

The family Paramyxoviridae contains some of the most important pathogens of domestic animals and humans (Fig. 14-7). The Newcastle Disease virus (NDV; Figs. 14-8 to 14-11), one of the most important diseases of poultry worldwide, belongs to the genus *paramyxovirus* and is recognized as serotype paramyxovirus 1 (PMV1). Nine avian paramyxovirus (APMV) serotypes have been differentiated so far (Box 14-1), and few strains have been isolated that could not be grouped (Telbis *et al.*, 1989). ND was first recognized in 1926 after large outbreaks in Java and Newcastle, UK. ND is a World Organization for Animal Health listed disease. APMV1 is divided into two classes based on genetic analysis. Strains of class II have been isolated from wild or domestic birds and include virulent and avirulent isolates, whereas class I strains are avirulent, and both have been found in wild birds (Briand *et al.*, 2014).

The virions of members of the family Paramyxoviridae are pleomorphic, roughly spherical enveloped particles with one large molecule of single-stranded RNA. Two glycoproteins from the surface projections possess both hemagglutinin and neuraminidase activities. NDV is relatively heat stable. It remains infectious in bone marrow and muscles of slaughtered chickens for at least 6 months at −20°C (−4°F) and up to 134 days at 1°C (33.8°F). It can survive for several years in dried mutes. Quaternary ammonium compounds, 1% to 2% Lysol, 0.1% cresol, and 2% formalin, are used for disinfection (Fenner *et al.*, 1987).

ND infects most avian species (Table 14-2), producing unapparent or mild disease in many and severe and lethal disease in others, indicating a great variety in its virulence. Differences in pathogenicity, efficacy of viral RNA replication, and humoral immunity even indicate different susceptibilities between one host species. Virulence is measured as a "neuropathic index" (NI) determined by intracerebral inoculation of day-old chicks. Lentogenic (NI 0.25: avirulent or mildly virulent), mesogenic (NI 0.6-1.8: intermediate virulence), and velogenic (NI 2.0: highly virulent) strains are differentiated. Paramyxovirus type 2 and 3 infections have been described in Passeriformes and parakeets with respiratory, enteric, and central nervous signs similar to those seen with PMV1.

Distribution

Three panzootics of ND occurred throughout the world between 1940 and 1948, between 1968 and 1972, and during the 1980s, primarily involving racing pigeons (Kaleta and Hettels-Resmann, 1992). However, a wide range of domestic and wild bird species are susceptible to PMV including chickens, turkeys, pigeons, ducks, geese, and pheasants. Changes in global climate and the speed of modern transport to any location of the globe may explain the recent finding of NDV and 33% seroprevalence in penguins in Antarctica (Thomazelli *et al.*, 2010).

Clinical Features

The clinical signs seen in birds infected with NDV vary widely and are dependent on many different factors (Alexander, 1995). One important factor is the variability of the virus' virulence. ND ranges from unapparent to severe to fatal. Although none of the clinical signs can be regarded as pathognomonic, certain clinical signs do appear to be associated with particular ND isolates. This has resulted in the grouping of NDV into five pathotypes based on the predominant signs in affected chickens (Alexander, 1995):

1. *Viscerotropic velogenic:* High mortality with swelling of tissues around the eyes, bloody diarrhea, and hemorrhagic lesions in proventriculus and intestines.

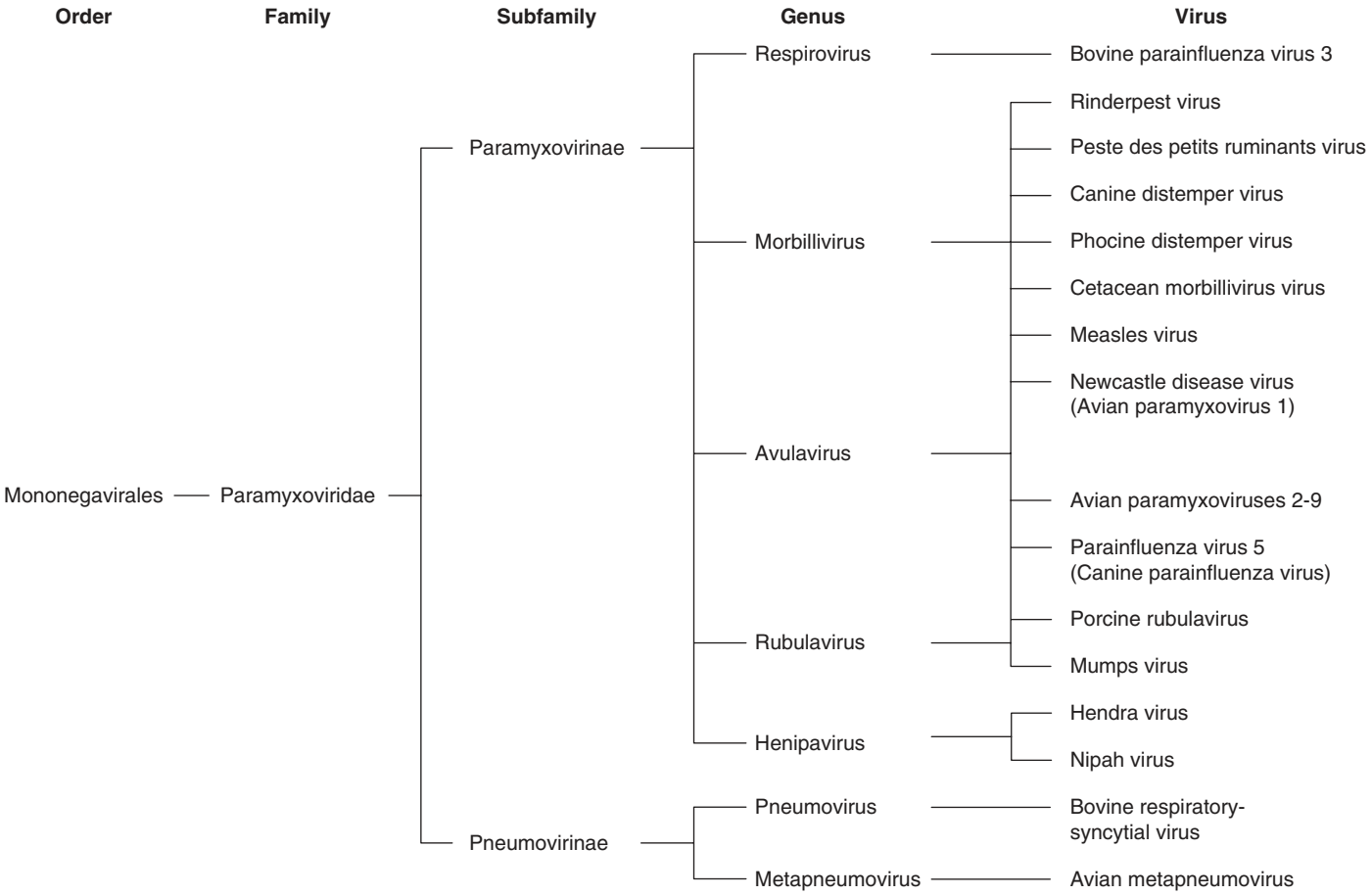


FIGURE 14-7 Classification of members of the family Paramyxoviridae of veterinary importance.

FIGURE 14-8 Newcastle disease in a domestic fowl. Note the typical petechial hemorrhages within the gizzard.

FIGURE 14-9 Domestic pigeon (*Columba livia*) affected with Newcastle disease, showing typical central nervous system signs of incoordination and torticollis. This is a seasonal disease affecting large flocks of pigeons in many parts of the world.

2. *Neurotropic velogenic:* High mortality followed by respiratory and nervous signs, characterized by paresis of limbs, wings, ataxia, torticollis, circling movements, and tremors.
3. *Mesogenic:* Respiratory signs, occasional nervous signs, head tics (falcon), and mortality is moderate to low.
4. *Respiratory lentogenic:* Mild or subclinical respiratory infection.
5. *Asymptomatic enteric:* Subclinical enteric infection.

These five groupings are by no means clear cut. In all bird species overlapping does occur. Acute and subacute disease associated with mesogenic and lentogenic virus strains are most common in developed countries with modern poultry industries (Fenner *et al.*, 1987).

Disease in chickens may consist of signs of depression, diarrhea, and nervous signs such as paralysis and torticollis. In layers, a drop in egg production to complete cessation of egg laying may follow the disease (Mayr, 1993). In pigeons, quail, and houbara bustards, Wernery *et al.* (1995) and Wernery and Manvell (2003) reported viscerotropic

FIGURE 14-10 Houbara bustard (*Chlamydotis undulata*) affected with Newcastle disease showing typical central nervous system signs of incoordination and torticollis. Large numbers of this species are affected with Newcastle disease when houbara bustards are kept in close proximity or are transported to Middle Eastern markets together with pigeons and domestic fowl.

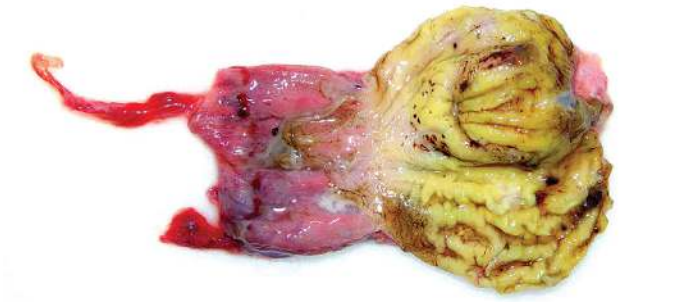


FIGURE 14-11 Newcastle disease in a saker falcon. Note the typical petechial hemorrhages within the proventriculus and ventriculus. The vomiting of semidigested blood is a common finding in falcons affected with Newcastle disease when such lesions are present in these organs. (Courtesy Dr. J. Samour.)

BOX 14-1 Avian Paramyxovirus: The Nine Classified Serotypes

- PMV1 Newcastle disease virus
- PMV2 Chicken/California/Yucaipa/56
- PMV3 Turkey/Wisconsin/68
- PMV4 Duck/Hong Kong/D3/75
- PMV5 Budgerigar/Japan/Kunitachi/75
- PMV6 Duck/Hong Kong/199/77
- PMV7 Dove/Tennessee/4/75
- PMV8 Goose/Delaware/1053/76
- PMV9 Duck/New York/22/78

From Ritchie BW: *Avian viruses, function and control*, Lake Worth, FL, 1995, Wingers Publishing.

and neurotropic pathotypes with diarrhea and nervous signs. Falcons suffering from PMV1 infections initially show gastrointestinal signs such as anorexia, vomiting, and paralytic ileus. Later in the course of the disease central nervous system signs develop. These signs are associated with ataxia, head tics, tremors, wing and leg paralysis and,

TABLE 14-2 Paramyxoviruses (788) Isolated from 14 Different Avian Species in the Middle East over a Period of 20 Years

Number	Avian Species	Number PMV1 Isolates
1	Falcon	247
2	Pigeon	192
3	Bustards*	187
4	Chicken	45
5	Quail	35
6	Dove	26
7	Partridge	21
8	Pheasant	9
9	Peacock	7
10	Stone curlew	6
11	Hawk	5
12	Sand grouse	4
13	Secretary bird	3
14	Ostrich	1
	Total	788

*Houbara and white-bellied bustard.

very rarely, torticollis (Wernery *et al.*, 1992). It is known that virulent strains may still replicate in vaccinated birds but the clinical signs will be greatly diminished in relation to the antibody level achieved (Alexander, 1995).

Pathologic Features

Lesions are highly variable, reflecting the variation in tropism and pathogenicity of NDV. As with clinical signs, no gross or microscopic changes are pathognomonic for any form of ND. Gross pathologic findings include hemorrhagic lesions throughout the intestines with typical petechiae in the proventriculus. Hemorrhagic changes and congestion are seen in the respiratory tract when respiratory signs are present. In birds showing neurologic signs before death, there is no evidence of lesions in the brain. In falcons with clinically reported gastrointestinal signs, the crop, proventriculus, ventriculus, and enteral tract are usually empty, apart from a considerable amount of bile, which stains parts of the duodenal and jejunal mucosa. In falcons with head tics and paralysis no gross lesions are detected.

Microscopic lesions have no diagnostic significance. In most tissues and organs where changes occur, hyperemia, necrosis, and edema are found. In the central nervous system nonpurulent encephalomyelitis may occur. In many birds mild to severe demyelinization in the cerebrum is observed.

Diagnosis

Because clinical signs and pathologic lesions are relatively nonspecific, diagnosis must be confirmed by virus isolation and to a lesser extent by serology. It is necessary to isolate NDV from infected birds and characterize the virus to exclude viruses of low virulence, which are ubiquitous in feral birds throughout the world, and live vaccines. The virus may be isolated from spleen, brain, or lung by allantoic inoculation of 10- to 12-day-old embryonated eggs or through infection of tissue culture chicken embryo fibroblasts. NDV is differentiated from other viruses by hemagglutination and hemagglutination inhibition

tests using polyclonal antisera. An assessment of the virulence of an NDV isolate is important and a number of in vivo tests are available like the intracerebral pathogenicity index (ICPI) and the intravenous pathogenicity index performed on specific pathogen-free chicken of different ages (Hitchner *et al.*, 1980). The mean death time, which is performed by inoculating the allantoic cavity of 9- to 10-day-old embryos, classifies isolates as velogenic (less than 60 hours to kill), mesogenic (60-90 hours to kill), and lentogenic (more than 90 hours to kill). Molecular techniques like real-time RT-PCRs have been used to detect NDV in clinical specimens such as oropharyngeal or fecal swabs.

Prevention and Control

Airborne infections are an increasingly important factor in veterinary centers, quarantine units, and breeding facilities. People who work in these centers are also susceptible to some airborne infections that can develop within them. Aspergillosis, mycoplasmosis, chlamydiosis, ND, and influenza are the common airborne infections, the source of which may be the birds themselves, the facility's staff, the facility itself, and built-in air-conditioning systems. A new mobile air-cleaning device/infection control unit is available that significantly reduces and even eliminates airborne pollution (Mattei *et al.*, 2002).

ND is a notifiable disease in most countries. Where the disease is enzootic, control can be achieved by proper hygiene combined with immunization. Live virus vaccines of naturally occurring lentogenic strains with an ICPI of less than 0.4 are commonly used. They are administered via drinking water, which must not contain chlorine or disinfectants, and via spraying. Vaccinated birds may shed the vaccine virus up to 15 days after vaccination.

Immunization of exotic birds with live vaccines via drinking water is not useful because of the poor serologic response. Inactivated vaccines administered subcutaneously are usually used for pigeons, houbara bustards, pheasants, quail, and falcons. It might be more effective to use vaccines from locally derived strains (Wernery *et al.*, 1995).

A killed vaccine, manufactured specifically against ND in falcons and containing strains from four different avian species, has been produced by the Central Veterinary Research Laboratory, Dubai, United Arab Emirates. The vaccine is widely used in falcons and has dramatically reduced PMV1 in hunting falcons.

Backyard poultry production makes a significant contribution to poverty alleviation and food security in Africa, Asia, Latin America, and the South Pacific. This is often threatened by severe outbreaks of ND and immunization of backyard poultry in rural and periurban areas is critical for maintaining healthy flocks to provide adequate nutrition and income for small families. A new ND vaccine can be directly administered in feed, drinking water, or reconstituted for ocular administration. The price of this new NDV-FDT vaccine is low, and the vaccine is stable at ambient temperatures (Lal *et al.*, 2014).

PNEUMOVIRUS INFECTIONS

Pneumovirus infections have been observed with severe rhinotracheitis in turkeys and decreased egg production and "swollen head syndrome" in chickens. Pheasants, guinea fowl, and ostriches exhibit mild disease. Infections in turkeys and chickens are associated with sneezing, swelling of infraorbital sinuses, and conjunctivitis. The disease resembles mycoplasmosis. For the diagnosis of this disease, a commercial avian pneumovirus antibody enzyme-linked immunosorbent assay (FlockChek APV Test Kit) is available from IDEXX Laboratories containing the three serotypes A, B, and C.

REFERENCES

Alexander DJ: Newcastle disease, *State Vet J* 5:21–24, 1995.

Briand F-X, Massin P, Jestin V: Characterisation of a type 1 avian paramyxovirus belonging to a divergent group, *Vet Microbiol* 168:25–33, 2014.

Fenner F, Bachmann PA, Gibbs EPJ, et al: *Veterinary virology*, New York, 1987, Academic Press.

Hitchner SB, Domermuth CH, Purchase HG, et al: *Isolation and identification of avian pathogens*, Philadelphia, 1980, American Association of Avian Pathologists.

Kaleta EF, Hettels-Resmann U: *Workshop on avian paramyxoviruses, Rauischholzhausen*, Germany, 1992, CEC, Giessen.

Lal M, Zhu C, McClurkan C, et al: Development of a low-dose fast-dissolving tablet formulation of Newcastle disease vaccine for low-cost backyard poultry immunization, *Vet Rec* 174(20):504, 2014.

Mattei D, Mordini N, Lonigro C, et al: MedicCleanAir® devices for air filtration, a low cost very effective method of managing ambiental *Aspergillus* spp. colonization, *Bone Marrow Transplant* 29(Suppl 2):5245, 2002.

Mayr A: *Rolle/Mayr medizinische mikrobiologie, infektions und seuchenlehre*, Stuttgart, 1993, Ferdinand Enke.

Telbis C, Neumann U, Siegmann D: Vorkommen von Paramyxoviren bei Wildvögeln: epizootiologische Aspekte, Eigenschaften *in vivo* und *in vitro*, *Zentralbl Veterinärmed B* 36:203–216, 1989.

Thomazelli LM, Aranjo J, Oliviera DB, et al: Newcastle disease virus in penguins from King George Island on the Antarctica region, *Vet Microbiol* 146:155–160, 2010.

Wernery U, Manvell R: Avian viral diseases in the United Arab Emirates (UAE). *Proceedings of the European Association of Avian Veterinarians*, Tenerife, pp 72–77, 2003.

Wernery U, Remple JD, Newmann U, et al: Avian Paramyxovirus serotype 1 (Newcastle disease virus)—infections in falcons, *J Vet Med* 39:153–158, 1992.

Wernery U, Alexander DJ, Neumann U, et al: Newcastle disease in captive falcons, *Middle East Falcon Research Group Specialist Workshop*, Abu Dhabi, pp 24–32, 1995.

FURTHER READING

Cornelissen H: Vaccination of over 200 bird species against Newcastle disease: methods and vaccination reactions. *Proceedings of the European Association of Avian Veterinarians*, Utrecht, pp 275–287, 1993.

Wernery R, Wernery U, Kinne J, et al: *Colour atlas of falcon medicine*, Hannover, Germany, 2004, Schlütersche.

HERPESVIRUS INFECTIONS

Etiology

More than 100 herpesviruses have been characterized and found in fish, amphibians, reptiles, humans, and in virtually every species of bird and mammal that has been investigated (Figs. 14-12 to 14-14). Their ubiquitous nature, evolutionary diversity, and involvement in many important medical and veterinary diseases make this group one of the most important global viruses. The classification of viruses within the family Herpesviridae is complex and not yet fully resolved. The family is divided into three subfamilies: Alphaherpesvirinae, Betaherpesvirinae, and Gammaherpesvirinae. All classified avian herpesviruses are within the genus *Mardivirus* and *Iltovirus* in the subfamily Alphaherpesvirinae (Table 14-3). Herpesviruses not assigned to a genus are listed in Table 14-4.

Classification into genera on the basis of the genome arrangement and serologic reactivity has just begun. The characteristic property of all herpesviruses is their lifelong persistence in the organism. They survive from generation to generation by establishing latent infections from which virus is periodically reactivated and shed.

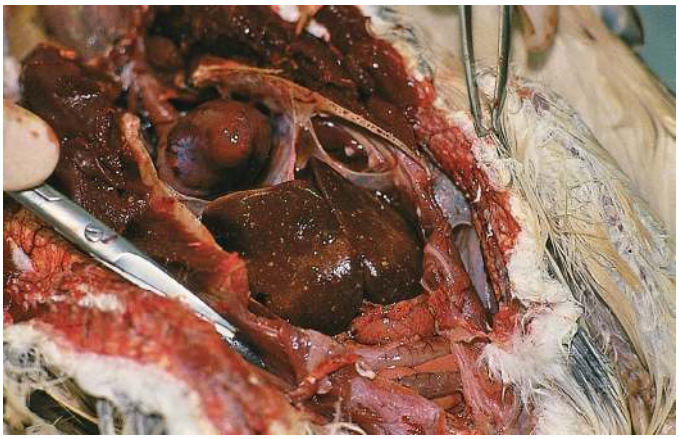


FIGURE 14-12 Herpesvirus infection in a gyrfalcon (*Falco rusticolus*). Note the multiple miliary necrotic lesions in the liver. An attenuated herpesvirus vaccine for falcons has been produced by CVRL, Dubai.

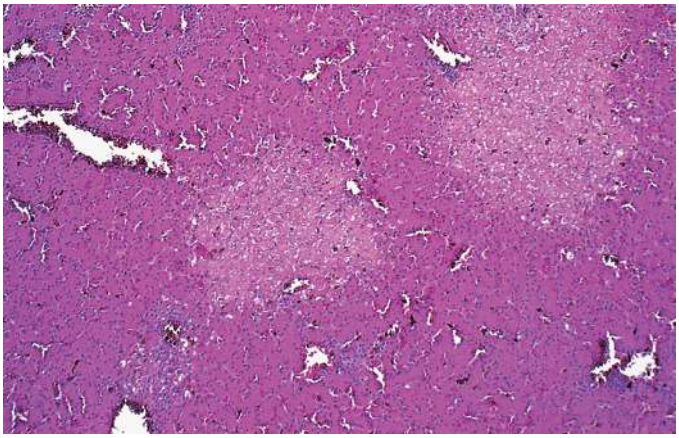


FIGURE 14-13 Multiple miliary necrotic lesions in the liver of the same gyrfalcon in Figure 14-12, H&E stain. (Courtesy Dr. J. Kinne.)

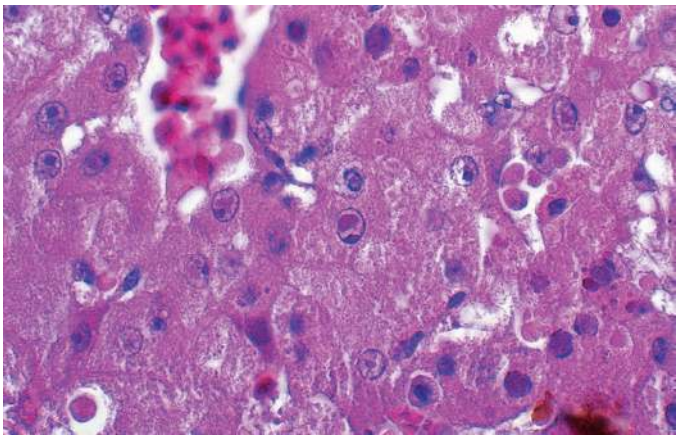


FIGURE 14-14 Herpesvirus inclusion body (center) in a liver section of the same gyrfalcon in Figure 14-12. H&E stain. (Courtesy Dr. J. Kinne.)

Many of the avian herpesviruses are not assigned to a genus. Internal papilloma disease in psittacines is also most probably caused by psittacid herpesvirus 1 (PsHV-1), and a new herpesvirus in African grey parrots (*Psittacus erithacus*) has been described and tentatively named psittacid herpesvirus 2 (PsHV-2). A new herpesvirus has been recently described in magnificent frigatebirds, which is named *Fregata magnificens* herpesvirus. Some of the herpesviruses have not even been listed by the International Committee on Taxonomy of Viruses (Box 14-2; Sandmeier, 2011).

BOX 14-2 Herpesvirus Described in the Literature but Not Listed by the International Committee on Taxonomy of Viruses

Psittacid herpesvirus PsHV-2
Parakeet herpesvirus
Amazon tracheitis herpesvirus
Toucan herpesvirus
Finch cytomegalovirus
Magnificent frigatebird herpesvirus

Herpesvirus characteristics, pathogenicity, and host specificity and the antigenic relationship among them are poorly understood (Mayr, 1993). Avian herpesviruses are only pathogenic to birds and vary widely in their virulence. The viruses are highly adapted to their natural hosts as a result of coevolution. A number of herpesviruses such as Marek's disease virus (gallid HV-2, GaHV-2) have been implicated in neoplastic transformation. The herpesvirus virion is enveloped by an icosahedral capsid consisting of 162 capsomers. Within the core of the nucleocapsid lies the genome, a single molecule of linear double-stranded DNA.

Herpesviruses are readily grown in cell cultures derived from their natural host species. *Alphaherpesvirinae* produce a rapid cytopathic effect whereas *Betaherpesvirinae* and *Gammaherpesvirinae* are slowly cytopathogenic in cell culture but produce similar intranuclear inclusion bodies. Intranuclear inclusion bodies are characteristic of herpesvirus infections and can usually be found in tissues from herpesvirus-infected birds and in appropriately fixed and stained cell cultures. Free herpesviruses are very sensitive to all disinfectants with virucidal properties and a temperature of 55°C (131°F) inactivates herpesviruses within seconds (Fenner *et al.*, 1987).

Avian herpesviruses in Table 14-3 cause hemorrhagic or neoplastic diseases and are of great importance to the commercial poultry industry. Many herpesviruses cause necrotic lesions, mainly in the liver and spleen, but some do cause hemorrhagic or neoplastic lesions, whereas others seem to be nonpathogenic (Table 14-5). Some of the important herpesvirus diseases are discussed separately and, for completeness, herpesvirus that do not cause disease are mentioned at the end of this chapter.

Marek's Disease

In 1907 the Hungarian physician–pathologist József Marek described paralysis associated with a polyneuritis affecting some domestic fowl kept in his backyard. For about 50 years Marek's disease was considered to be part of a large group of diseases referred to as the avian leukosis complex. The specific herpesvirus etiology of Marek's disease, however, was established in 1967.

Marek's disease is a contagious, lymphoproliferative disease of chickens caused by Marek's disease virus (MDV) and is prevalent wherever domestic poultry industry is found. It is a World Organization for Animal Health (OIE) listed avian disease occasionally detected in pheasants, turkeys, quail, and francolins (Mayr, 1993). The virus is slowly cytopathic and remains highly cell associated, so cell-free infectious virus is virtually impossible to obtain. MDV is an oncogenic herpesvirus, named GaHV-2. The virus is immunologically unique and is closely related to the herpesvirus of turkeys (HVT). Presently

TABLE 14-3 Avian Herpesviruses Listed by the International Committee on Taxonomy of Viruses

Family	Subfamily	Genus	Species	Acronym	Common Name
Herpesviridae	Alphaherpesvirinae	*Mardivirus*	Columbid herpesvirus 1	CoHV-1	Pigeon herpesvirus
	Betaherpesvirinae	*Iltovirus*	Gallid herpesvirus 2	GaHV-2	Marek's disease virus type 1
	(cytomegalovirus)		Gallid herpesvirus 3	GaHV-3	Marek's disease virus type 2
	Gammaherpesvirinae		Meleagrid herpesvirus 1	MeHV-1	Turkey herpesvirus
			Gallid herpesvirus 1	GaHV-1	Infectious laryngotracheitis
			Psittacid herpesvirus 1	PsHV-1	Pacheco disease virus

TABLE 14-4 Unassigned Viruses in the Subfamily Herpesvirinae

Disease	Virus
Duck plague enteritis	Anatid HV-1
Cormorant hepatitis	Phalacrocoracid HV-1
Inclusion body hepatitis of cranes	Gruid HV-1
Inclusion body hepatitis of storks	Ciconiid HV-1
Inclusion body hepatitis of falcons	Falconid HV-1
Inclusion body hepatitis of eagles	Accipitrid HV-1
Inclusion body hepatitis of owls	Strigid HV-1
Inclusion body hepatitis of quail	Perdicid HV-1
Penguin hepatosplenitis	Spheniscid HV-1

TABLE 14-5 Typical Lesions in Diseases Caused by Herpesviruses

Virus	Major Lesions
Pigeon herpesvirus	Upper respiratory disease, liver necrosis
Marek's disease virus	Lymphocyte infiltrations, neoplastic lesions
Turkey herpesvirus	Nonpathogenic
Infectious laryngotracheitis virus	Hemorrhagic tracheitis
Pacheco disease virus	Liver necrosis, internal papillomas (neoplastic lesions)
Eagle herpesvirus	Liver necrosis
Falcon and owl herpesvirus	Liver and spleen necrosis
Duck plague herpesvirus	Diphtheritic and hemorrhagic enteritis
Crane herpesvirus	Liver necrosis, diphtheritic and hemorrhagic enteritis
Stork herpesvirus	Liver necrosis
Quail herpesvirus	Liver necrosis
Cormorant herpesvirus	Nonpathogenic
Penguin herpesvirus	Hemorrhagic tracheitis
Psittacine herpesvirus 2	Papillomas (neoplastic lesions)
Toucan herpesvirus	Liver necrosis
Amazon tracheitis virus	Hemorrhagic tracheitis
Parakeet herpesvirus	Airsacculitis, tracheitis
Finch cytomegalovirus	Airsacculitis, conjunctivitis
Magnificent frigatebird herpesvirus	Nodular proliferative dermatitis (neoplastic lesions)

members of the genus Mardivirus can be divided immunologically into three serotypes:

- Serotype 1 (GaHV-2): Includes all pathogenic and nonpathogenic strains. It varies markedly in pathogenicity.
- Serotype 2 (GaHV-3): Contains avirulent and nononcogenic strains.
- Serotype 3 (meleagrid herpesvirus 1): Includes avirulent HVT (Markey *et al.*, 2013).

Marek's disease is a progressive disease with variable signs. Four overlapping syndromes are described (Fenner *et al.*, 1987):

- *Neurolymphomatosis* (classical Marek's disease) is an asymmetric paralysis of one or both legs or wings—one leg is held forward and the other backward.
- *Acute Marek's disease* occurs in explosive outbreaks in which a large proportion of birds in a flock shows depression followed after a few days by ataxia and paralysis; there are no localizing neurologic signs.
- *Ocular lymphomatosis* is recognized when the iris of one or both eyes is gray in color because of lymphoblastoid cell infiltration; there may be partial or total blindness.
- *Cutaneous Marek's disease* is readily recognized after plucking, when round nodular lesions up to 1 cm in diameter are observed, particularly at feather follicles.

Marek's disease is characterized by a mononuclear infiltrate within peripheral nerves and other tissues and organs. In the vast majority of cases a diagnosis can be made if the celiac, cranial, intercostal, mesenteric, brachial, sciatic, and greater splanchnic nerves are examined. In diseased birds, these nerves are up to three times their normal diameter.

Lymphomatous lesions, indistinguishable from those of avian leukosis, appear in the gonads, heart, proventriculus, and lungs but are seldom found in the bursa of Fabricius, which in cases of avian leukosis, is the site of most tumor development. Many apparently healthy birds are lifelong carriers and shedders of the virus. The virus is not transmitted *in ovo*. Marek's disease and avian leukosis (Figs. 14-15 and 14-16) are usually present in the same flock and both diseases also may occur in the same bird. The two diseases were long confused but can be differentiated by clinical and pathologic features (Table 14-6) and by specific viral and antibody tests. Congenital infection does not occur and chicks are protected by maternal antibodies for the first few weeks of life. They then become infected by inhaling virus dust. Epizootics of Marek's disease usually involve adult birds 2 to 5 months old and there is a high mortality rate of about 80%.

Diagnosis and Prevention

Diagnosis of MD should be based on a combination of clinical signs, gross and microscopic pathology, and laboratory testing including MDV isolation, PCR, or enzyme-linked immunosorbent assay (ELISA). Differentiation of Marek's disease from lymphoid leukosis

FIGURE 14-15 An unusual record of lymphoid leukosis in a female houbara bustard. Note the grossly enlarged liver. Until recently it was believed that lymphoid leukosis was confined to domestic poultry. During postmortem examinations, affected chickens usually display diffuse or nodular lymphoid growths involving mainly the liver, spleen, and bursa of Fabricius, but occasionally lesions also can be observed in the kidneys, gonads, and mesenterium.

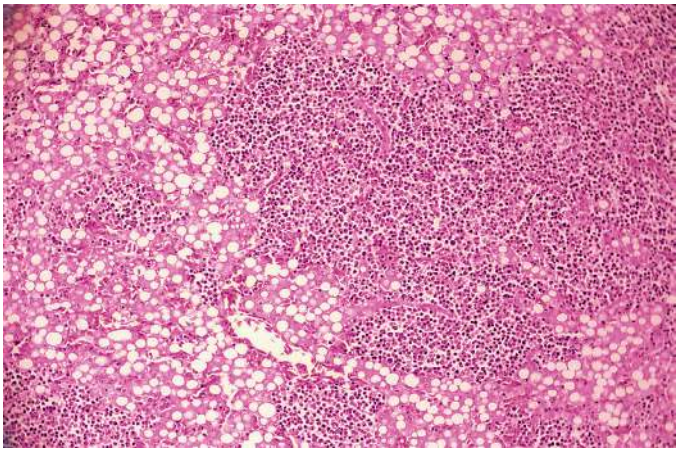


FIGURE 14-16 Histologic preparation of the liver from the same houbara bustard in Figure 14-15, showing diffuse lymphocytic infiltration. The neoplastic cell types are lymphoblasts and lymphocytes. (Courtesy Dr. J. Kinne.)

TABLE 14-6 Marek's Disease and Avian Leukosis: Clinical and Histologic Differentiation

Disease Parameter	Marek's Disease	Avian Leukosis
Etiology	Herpesvirus	Retrovirus
Signs	Frequently paralysis (wing and leg)	Nonspecific
Target cells	T-lymphocytes	Various hematopoietic cells
Age of onset of clinical signs	4 weeks	16 weeks
Gross Lesions		
Liver, spleen, kidney	+	+
Gonads, lung, heart	+	Rare
Nerve trunks	+	Rare
Iris	+	Rare
Skin	+	Rare
Bursa	Rare	+(nodular)
Histology		
Size of the affected lymphoblasts	Varied	Uniform
Intranuclear inclusion bodies	Yes	No

is important and is usually based on the age of birds affected, incidence of disease, and histopathological findings. Serum antibodies to GaHV-2 may be detected using agar gel immunodiffusion (AGID), ELISA, indirect immunofluorescence, and virus neutralization.

The current vaccination programs for Marek's disease have effectively decreased its natural incidence, but the eradication of Marek's disease from flocks of chickens is impossible. The vaccination prevents the development of the disease but not MDV infection. It is certain that there are no chicken flocks in the field that are completely free of MDV. Therefore isolation of MDV or antibody detection in such flocks is not a valid criterion for a diagnosis (Castro and Heuschele, 1992).

Marek's disease vaccines are injected *in ovo* at the 17th or 18th day of embryonation or subcutaneously at hatch. Avirulent strains of MDV are used as vaccines, but the antigenically related turkey Gammaherpesvirus is the preferred vaccine strain because it infects cells productively.

The production of chickens on the "all-in, all-out" principle would improve the efficacy of vaccination as a control measure, and a combination of three methods is used to control Marek's disease: vaccination, isolation and sanitation procedures, and breeding of resistant stock.

Duck Plague (Duck Viral Enteritis)

This Alphaherpesvirus infection was first recognized in 1923 in the Netherlands, where it was initially diagnosed as influenza; subsequently, it was recognized as a major disease in North America, China, India, and Europe. In addition to domestic ducks, free-living ducks, geese, swans, and other waterfowl are equally susceptible (Fenner *et al.*, 1987). Major epizootics occur worldwide, and migratory waterfowl may contribute to outbreaks within and among continents. Duck viral enteritis is an OIE-listed disease.

Virus strains vary in their virulence, although only a single antigenic type has been recognized. The virus grows on the chorioallantoic membrane of embryonated duck eggs and in duck embryo fibroblasts but poorly or not at all in chicken cells. As with members of this family, there is evidence that this virus can establish a latent infection.

Clinical signs of duck enteritis include depression, a drop in egg production, ruffled and dull feathers, ocular and nasal discharge, anorexia, labored breathing, watery diarrhea, extreme thirst, and ataxia followed by death. Morbidity and mortality vary from 5% to 100%. Lesions seen at necropsy are typical of vascular damage. Blood is present in the body cavities including gizzard and intestinal lumens. Petechial hemorrhages and focal necrosis are present in many tissues. Herpesvirus inclusions are most readily demonstrated in hepatocytes, intestinal epithelium, and lymphoid tissues, which can be used for diagnosis in combination with fluorescent antibody (FA) technique, virus isolation, and PCR.

Ingestion of contaminated water is thought to be the major mode of transmission, although the virus may also be transmitted by contact.

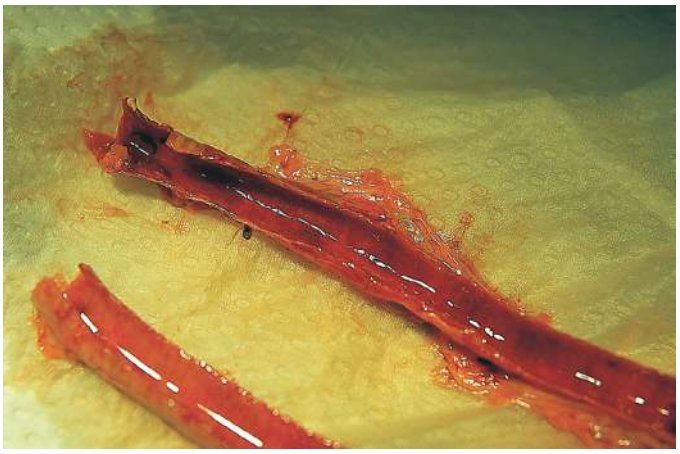


FIGURE 14-17 Diffuse hemorrhage in the trachea of a peacock (*Pavo cristatus*) with infectious laryngotracheitis.

TABLE 14-7 Pacheco Disease Virus (PsHV-1)

Serotype 1 = Genotypes 1 and 4 Serotype 2 = Genotype 2 Serotype 3 = Genotype 3	
Serotype 4 and 5	Only 1 isolate each
Genotypes 1, 2, and 3	Highly pathogenic for Amazon parrots
Genotype 4	Highly pathogenic for Amazon parrots in Europe but not in the United States Main cause of Pacheco disease in macaws and conures
Genotypes 2, 3, and 4	Pathogenic for African parrots
Genotype 1	Never isolated from African grey parrots

Chicken or duck embryo attenuated strains are used for immunization in combination with hygiene measures.

Avian Infectious Laryngotracheitis

This viral disease of domestic fowl is an OIE-listed disease that was first observed in the United States in 1926. It occurs among chickens worldwide, and it is rarely recognized as a cause of disease in other avian species (Fig. 14-17). It is caused by the gallid herpesvirus 1 (GaHV-1). Strains of the virus vary considerably in virulence but are antigenically homogenous. Infectious laryngotracheitis is an acute, highly contagious respiratory disease of domestic fowl characterized by distressed breathing with loud gasping, coughing, and expectoration of bloody mucus. However, signs and lesions can vary from peracute to mild. Birds of all ages are susceptible but disease is most common in those aged 4 to 18 months. In peracute infectious laryngotracheitis mortality can exceed 50% of the flock, and in acute infectious laryngotracheitis mortality is reduced to 10% to 15%, although morbidity is as high as in the peracute form. Chronic or mild infectious laryngotracheitis forms are of low morbidity and mortality (2% to 5%). The disease is always accompanied by lowered egg production. In infectious laryngotracheitis, there is severe laryngotracheitis characterized by necrosis, hemorrhage, ulceration, and the formation of diphtheritic membranes. Bloody mucoid pseudomembranes (casts) along the trachea lead to death through asphyxiation. The extensive diphtheritic membrane formation and death from asphyxia prompted the designation "fowl diphtheria." Infections with infectious laryngotracheitis virus are transmitted by direct or indirect contact but are not transmitted within eggs from infected chickens. The virus is shed through conjunctival excretions, tracheal mucus, and feces. The virus can become latent in the trigeminal ganglia of infected birds. Carrier birds shed the infectious laryngotracheitis virus intermittently throughout their lives.

Diagnosis and Control

Clinical and necropsy findings are characteristic for infectious laryngotracheitis. FA staining of smears and tissues and isolation of the virus from tracheal mucus, either by inoculation on the chorioallantoic membrane of embryonated eggs or cell cultures, confirm the diagnosis. Giemsa staining for infectious laryngotracheitis intranuclear inclusion bodies in virus-infected syncytial cells of the tracheal epithelium is another method of diagnosing infectious laryngotracheitis. Immunofluorescence tests for viral antigens using tracheal scrapings, AGID with tracheal exudates, ELISA assays detecting viral antigen, and PCRs are also important diagnostic test methods.

For control, site quarantine and hygiene measures should be the first approach. Immunization with attenuated live-virus vaccine via instillation of eye drops, spray, or drinking water protects birds against clinical disease but does not protect against infection with virulent virus or the development of a latent carrier status for either the virulent or the vaccine viruses. Despite a proper vaccination program it is to be expected that virulent virus persists in flocks and that some losses caused by infectious laryngotracheitis, either alone or in combination with other pathogens, will continue. There have been recent promising studies in the efficiency of a genetically engineered vaccine.

Pacheco Disease

Pacheco disease was first detected in 1929 in psittacine birds from a zoologic park in Brazil and confirmed as an avian herpesvirus in the United States. The disease is characterized by depression, biliverdinuria, and diarrhea and in some birds neurologic signs also were observed. The disease causes high mortality and is highly contagious. When necropsied there are often no gross pathologic alterations, but on histopathology necrotizing lesions and hemorrhages are observed in liver and other organs. So far four genotypes and five serotypes are known (Table 14-7). It seems that papillomas often found in psittacines are also caused by PsHV-1.

Clinically healthy but persistently infected birds can be identified by herpes PCR testing of choanal and cloacal swabs and blood.

Raptor Herpesvirus Causing Hepatosplenitis

Three distinct herpesviruses causing hepatosplenitis have been isolated from raptors:

- Inclusion body hepatitis of falcons: falconid HV-1 (FaHV-1).
- Inclusion body hepatitis of owls: strigid HV-1 (SHV-1).
- Inclusion body hepatitis of eagles: accipitrid HV-1 (AHV-1).

Raptor herpesviruses cause a fatal disease in birds of prey characterized by multifocal necrosis of the liver and spleen (Wheler, 1993). Clinical signs are nonspecific and range from sudden death to severe depression, anorexia, regurgitation, and weakness followed by death (Wheler, 1993; Remple, 1995). Very typical of the disease is the appearance of lime green urates in the feces. Microscopic lesions in liver and spleen reveal necrotic foci without any inflammatory response.

It has now been proven through sequence comparison that FaHV-1 and SHV-1 are identical to pigeon herpesvirus 1 (Gailbreath and Oaks,

FIGURE 14-18 A common kestrel (*Falco tinnunculus*) with FHV-1 infection. The kestrel was very weak and anorectic and excreted green mutes caused by the destruction of the liver parenchyma by the virus. The source of this infection is mainly pigeons that are fed to hunting falcons.

2008) confirming the general belief that feeding infected pigeons, which are often herpesvirus carriers without showing clinical signs, may infect falcons or owls. A herpes not serologically related to falcon, owl, or pigeon herpesvirus was isolated from a bald eagle and from eagles in Spain. A lesser spotted eagle was subcutaneously infected with a pathogenic herpesvirus that had killed falcons. The eagle did not show any clinical signs and it did not seroconvert. It seems that eagles may be refractory to FaHV-1 (Wernery and Kinne, 2004).

Diagnosis of this disease is confirmed by typical necropsy findings and by virus isolation from liver and spleen in chicken embryo fibroblasts followed by serologic identification. A cytopathic effect develops after 3 to 4 days that is characterized by foci of round refractile cells in the monolayer with subsequent formation of syncytia.

Herpesvirus infections in falcons are a constant threat to falconry (Fig. 14-18). The majority of herpesvirus infections occur in gyrfalcons and hybrids of this species, and it seems that this species has a greater susceptibility to the virus than other falcon species. Preliminary investigations have shown that falcons do not contract the infection orally but most probably ocularly and/or nasally (Wernery and Kinne, 2004).

No commercial vaccine is available but scientists at the Central Veterinary Research Laboratory, Dubai, have developed an attenuated herpesvirus vaccine, the efficacy and safety of which have been proved in different vaccination trials (Remple, 1995; Wernery and Manvell, 2003; Wernery *et al.*, 2001, 2003).

Avian Herpesvirus Infections Causing No Disease

These viruses include herpesviruses of cormorants, turkeys, and penguins. The phalacrocoracid HV-1 was isolated in 1951 from a single nesting cormorant on Lake Victoria, Australia. It is unrelated serologically to any of the other avian herpesviruses. No clinical, gross, or microscopic lesions have been associated with natural infections with turkey herpesvirus, which is serologically related to MDV. In two adult black-footed penguins that suffered from loss of condition and respiratory distress with microscopic lesions resembling infectious laryngotracheitis, inclusion bodies were detected in syncytial cells of the sinuses, trachea, and bronchi (Ritchie, 1995). A herpesvirus has been recently isolated from the liver of a houbara bustard, but when the virus was subcutaneously injected into the same species no sickness was detected.

REFERENCES

Castro AE, Heuschele WP: *Veterinary diagnostic virology*, St Louis, MO, 1992, Mosby Year Book.

Fenner F, Bachmann PA, Gibbs EPJ, et al: *Veterinary virology*, New York, 1987, Academic Press.

Gailbreath KL, Oaks L: Herpesviral inclusion body disease in owls and falcons is caused by the pigeon herpesvirus (Columbid herpesvirus 1), *J Wildl Dis* 44(2):427–433, 2008.

Markey B, Leonard F, Archambault M, et al: *Clinical veterinary microbiology*, St Louis, MO, 2013, Mosby Elsevier.

Mayr A: *Rolle/Mayr medizinische mikrobiologie, infektions und seuchenlehre*, Stuttgart, 1993, Ferdinand Enke.

Remple JD: Falcon herpesvirus and immune consideration for the production of a vaccine. *Presented at the Middle East Falcon Research Group Specialist Workshop*, Abu Dhabi, 1995.

Ritchie BW: *Avian viruses, function and control*, Lake Worth, FL, 1995, Wingers Publishing.

Sandmeier P: Avian herpesviruses: an update and literature review. *11th European AAV conference, 1st Scientific ECZM Meeting, Madrid Conference Proceedings*, 2011.

Wernery U, Kinne J: How do falcons contract a herpesvirus infection? Preliminary findings, *Falco* 23:16–17, 2004.

Wernery U, Manvell R: Avian viral diseases in the United Arab Emirates (UAE). *Proceedings of the European Association of Avian Veterinarians*, Tenerife, pp 72–78, 2003.

Wernery U, Joseph S, Kinne J: An attenuated herpesvirus may protect gyr hybrids from fatal inclusion body hepatitis. A preliminary report, *J Vet Med B Infect Dis Vet Public Health* 48(10):727–732, 2001.

Wernery U, Sanchez AL, Joseph S: Falconid response to the attenuated herpesvirus vaccine DuFaHe. *Proceedings of the European Association of Avian Veterinarians*, Tenerife, pp 72–78, 2003.

Wheler CL: Herpesvirus disease in raptors. A review of the literature. In Redig PT, Cooper JE, Remple JD, et al, editors: *Raptor biomedicine*, Minneapolis, 1993, University of Minnesota Press.

AVIAN POX

Etiology

The *poxviruses* are a large family of complex viruses infecting many species of vertebrates, as well as arthropods (Fig. 14-19), and members of the three genera (Orthopoxvirus, Yatapoxvirus, and Parapoxvirus) have zoonotic potential. However, infections of humans are generally associated with localized lesions rather than life-threatening illness, although severe diseases have occurred in immunocompromised patients. The most famous Orthopoxvirus infection is the cowpox, which has been exploited for the pivotal role of vaccination strategies and hence the global eradication of smallpox.

Although the genus Avipoxvirus (Figs. 14-20 to 14-30) is divided into 10 defined species (Box 14-3), many avian pox isolates are not clearly classified, and their status within the genus is unclear or not known. It is, for example, not yet clear whether the turkeypox virus and eventually also the pigeonpox virus are only variants or serotypes of the fowlpox virus (Mayr, 1993), which is the prototype of the avian poxviruses. Fowlpox is a term that describes a pox disease, particularly in chickens and turkeys. It is a World Organization for Animal Health-listed disease.

The poxviruses are the largest and the most complex of all viruses. Avian poxviruses are only distantly related at the antigenic level to other poxvirus genera (Binns and Smith, 1992). Fowlpox, pigeonpox, and turkeypox viruses are closely related and not strictly host specific. Under natural conditions they produce a disease in avians only, and their virions are larger than those of other poxviruses. The virions are typically brick shaped with dimensions of about 330 × 280×200 nm.

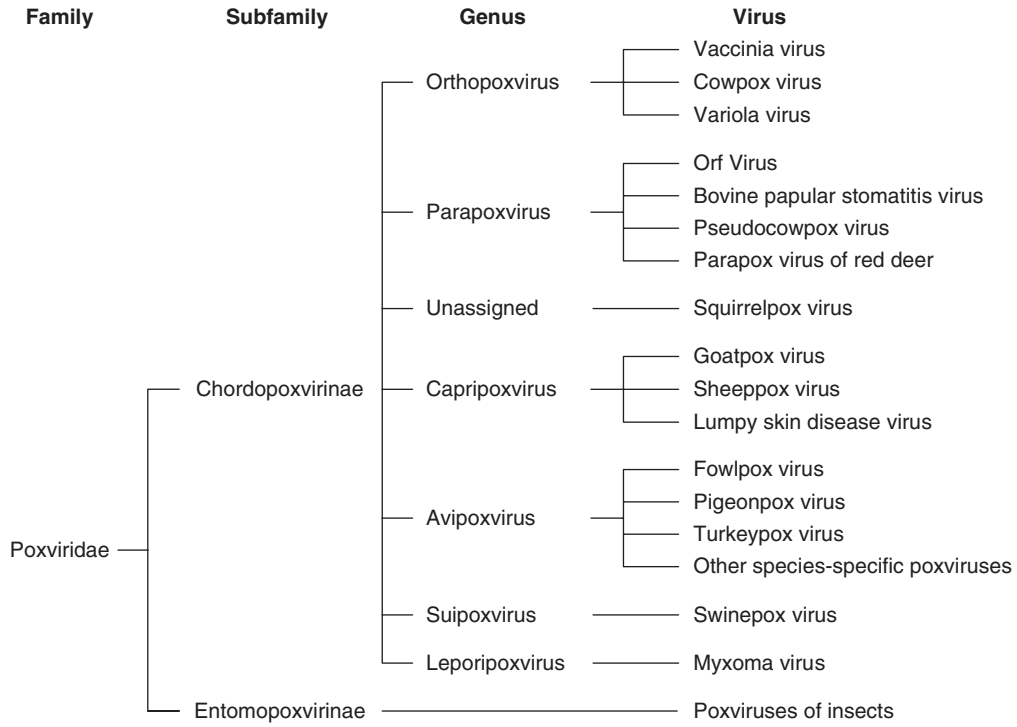


FIGURE 14-19 Classification of the poxviruses of veterinary importance.

FIGURE 14-20 A dried scab on the anterior aspect of the hock joint in a stone curlew produced by avian poxvirus.

FIGURE 14-21 Typical pox lesions on the cere of a stone curlew.

The genome consists of a linear, nonsegmented, and covalently closed, double-stranded DNA, which encodes for about 150 to 300 different proteins. Avian poxviruses are antigenically and immunologically distinguishable from each other to an extent, but various degrees of cross-relationships exist. The genetic profiles of fowlpox, pigeonpox, and juncopox viruses appear similar, but the genetic profiles of quailpox, canarypox, and mynapox viruses are fairly different from that of fowlpox.

Poxviruses are resistant to ambient temperatures and may survive many years in dried scabs. Orthopoxviruses and most avipoxviruses are ether resistant, but parapoxvirus, capripoxviruses, and leporipoxviruses are ether sensitive (Fenner *et al.*, 1987). Fowlpox virus is inactivated by 1% caustic potash. It withstands 1% phenol and 1:1000 formalin for 9 days.

Distribution

Avian pox is a common viral disease of domestic and free-living birds that occurs worldwide. The disease has been reported in more than 70 species of free-living birds representing 20 families (Castro and Heuschele, 1992).

Clinical Features

Avian pox is a relatively slow-spreading viral disease characterized by the development of two major forms and one minor form. The cutaneous form occurs through bites of arthropods that serve as vectors for the transmission of the virus. The diphtheric form is caused by inhalation or ingestion of the virus. It is associated with higher mortality and more severe lesions. A third, pneumonia-like form has been observed in canaries. Unfortunately, despite the complete sequencing of the

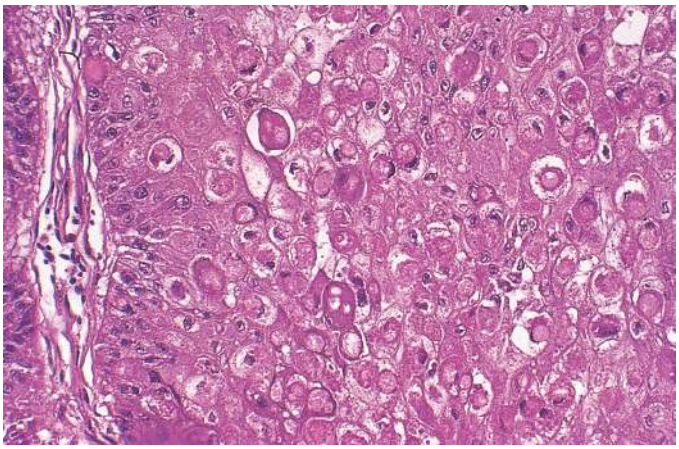


FIGURE 14-22 Bollinger intracytoplasmic bodies in a histologic preparation of the stratum spinosum of the skin in a peregrine falcon (*Falco peregrinus*). The presence of Bollinger bodies at microscopic examination is characteristic of avian pox infection. H&E stain. (Courtesy Dr. J. Kinne.)

FIGURE 14-23 A houbara bustard with a large scab on the lower eyelid. The lesions were produced by avian poxvirus. (Courtesy Dr. J. Samour.)

FIGURE 14-24 Postmortem examination 1 week after the onset of the disease. Note the "cauliflower" appearance of the lesions on the third eyelid and conjunctiva. (Courtesy Dr. J. Samour.)

FIGURE 14-25 Typical pox scabs on the feet of a peregrine falcon (*Falco peregrinus*). The most dangerous scabs on the feet are those developing around the last phalanx. Severe scabbing on this area may result in the loss of the last phalanx as a result of distal necrosis or self-mutilation. (Courtesy Dr. J. Samour.)

FIGURE 14-26 Large pox lesion on the cere of a saker falcon (*Falco cherrug*). (Courtesy Dr. J. Samour.)

FIGURE 14-27 Pox lesions on the lower eyelid and cere of an ostrich chick.

FIGURE 14-28 Early proliferative pox lesions around the cere of an immature domestic pigeon.

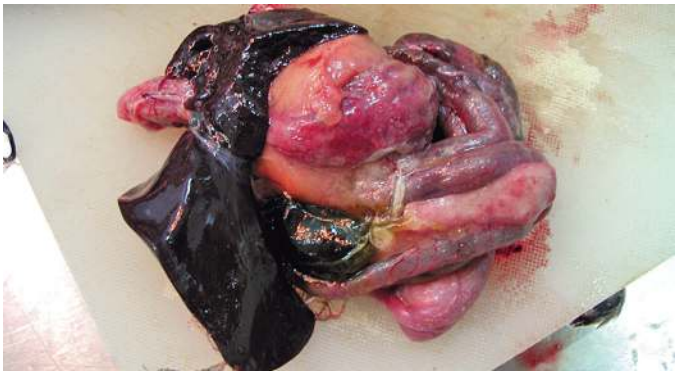


FIGURE 14-29 Pancreatitis and hepatomegaly in a houbara bustard with systemic pox.

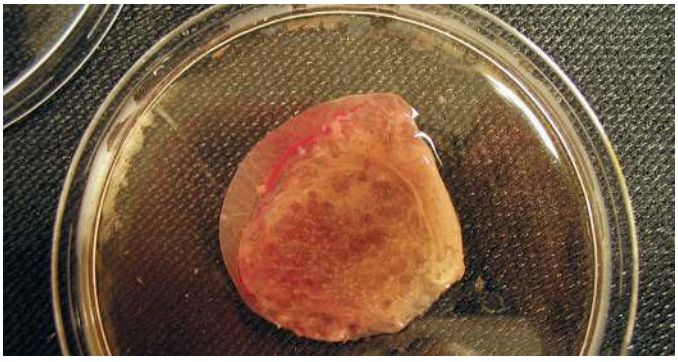


FIGURE 14-30 A pock lesion caused by the systemic poxvirus on the chorioallantoic membrane of an 11-day-old chicken embryo.

canarypox and fowlpox genomes little is known about the phylogenetic relationship between the two sequenced poxviruses and the other avipoxviruses and how this may relate to pathogenicity, epidemiology, and host range.

The cutaneous or dry pox form is characterized by the appearance of nodular scabs on various parts of unfeathered skin. The lesions vary in size and appearance. Removal of scabs leaves a hemorrhagic moist surface. When the crusts are dry they drop off, leaving scars. Mechanical transmission of the avian poxvirus by arthropods, especially mosquitoes, provides a mechanism for transfer of the virus between varieties of different bird species. The cutaneous form, which is most common, probably results from infection by biting arthropods or by direct contact with infected birds or their fomites. Poxviruses cannot pass intact skin and must enter the body through abrasions or cuts.

In the diphtheritic or wet pox form, the lesions occur on the mucous membranes of the mouth, nares, pharynx, larynx, esophagus, or trachea. This form is probably caused by aerosol infection. Tracheal lesions can cause difficulties in breathing, and the signs can resemble those of infectious laryngotracheitis in chickens and vitamin A deficiency. The diphtheric form often turns into a septicemic or systemic form, as recently reported in waterfowl (Anonymous, 2004) and houbara bustards (Kinne *et al.*, 2007).

During the septicemic form of the disease, only general clinical signs such as somnolence, cyanosis, and fatigue are observed in infected birds (canaries) without any cutaneous lesions. In these cases the virus may be found in the lungs. Areas of necrosis in the myocardium are also described (Mayr, 1993).

Pathologic Features

The virus multiplies at the site of entry resulting in formation of primary lesions. Pox lesions in the skin follow a typical developmental sequence. They commence as erythematous macules and become papular and then vesicular. The vesicles develop into pustules with a depressed center and raised, often erythematous edges. These lesions are the so-called pocks. The pustules rupture and a crust forms on the surface, and dry crusts fall off and leave residual scars. Histologically, pox lesions start as epidermal cytoplasmic swelling, vacuolation, ballooning degeneration, and production of intraepithelial vesicles affecting the cells of the outer stratum spinosum. Additional dermal lesions include edema and vascular dilatation and, at a later date, perivascular mononuclear and neutrophilic cell infiltration. Mucosal lesions are briefly vesicular and develop into ulcers rather than pustules.

During outbreaks of wet and systemic pox in houbara bustards and waterfowl, multiple discrete, pale yellow/cream-colored, raised necrotic lesions were irregularly distributed across the oropharyngeal mucosa, which can be easily confused with trichomoniasis. Mucoid rhinitis and tracheitis were also observed. Additionally, the birds had a focal bronchopneumonia with miliary necroses; a swollen, hemorrhagic and enlarged pancreas; and hepatomegaly. It is believed that inhalation or ingestion of virus-contaminated material may cause the diphtheritic

BOX 14-3 Avian Poxviruses

Species in the Genus Avipoxvirus (APV):

- Canarypox virus (CNPV)
- Fowlpox virus (FWPV)
- Juncopox virus (JNPV)
- Mynapox virus (MYPV)
- Pigeonpox virus (PGPV)
- Psittacine pox virus (PSPV)
- Quailpox virus (QUPV)
- Sparrowpox virus (SPPV)
- Starlingpox virus (SLPV)
- Turkeypox virus (TKPV)

Tentative Species in the Genus APV:

- Peacockpox virus (PKPV)
- Penguinpox virus (PEPV)
- Falconpox virus (FAPV)
- Houbara bustardpox virus (?)
- Stone curlewpox virus (?)

form of avian pox. Malnutrition, stress, or concurrent disease may contribute to outbreaks.

Diagnosis

Although pox lesions are easy to recognize, laboratory tests should be performed to confirm the diagnosis. Because of the large size and distinctive structure of poxvirus virions, electron microscopic examination of scab material or other lesions is the preferred method of laboratory diagnosis. Avian poxviruses produce pocks on the chorioallantoic membrane of embryonated hen's eggs, and viruses grow productively, but also abortively, in avian cell lines (Samour *et al.*, 1996). The virus multiplies in the cytoplasm of infected cells with the formation of inclusion bodies (Bollinger bodies) or elementary bodies (Borrel bodies), which can be stained with different staining methods. The specificity of the viral inclusions can be determined by fluorescent antibody and immunoperoxidase methods. Suitable serologic tests for the detection of antibodies include passive hemagglutination, agar gel immunodiffusion test, and enzyme-linked immunosorbent assays.

Prevention and Control

The vaccinia virus has become famous as a vector for expressing heterologous genes into its genome and using it for the production of recombinant vaccines. This is a relatively new approach with a potentially wide application in veterinary medicine. Avian poxviruses are not yet being used in this new method.

For prophylactic immunization against avian pox, live attenuated vaccines are commercially available. Chickenpox, pigeonpox, and turkeypox virus strains are used to protect many different avian species with varying degrees of success. Pigeonpox vaccines are less immunogenic and their immunity does not last very long. Pigeonpox vaccines and turkeypox vaccines are used in falcons with varying degrees of success. An attenuated falconpox vaccine has been successfully used in the Middle East for several years (Kaaden *et al.*, 1995, Wernery and Manvell, 2003). A canarypox vaccine has also been established for the vaccination of pet birds and an attenuated houbara bustard pox vaccine protects against dry and wet pox (Wernery *et al.*, 2007).

Vaccination procedures are often used in combination with insecticide spraying to reduce the number of arthropods. The live attenuated vaccines are administered either subcutaneously (wing web method) or intramuscularly (canarypox).

REFERENCES

Anonymous: Fowlpox, *Vet Rec* 155:728, 2004.

Binns MM, Smith GL: *Recombinant poxviruses*, Boca Raton, FL, 1992, CRC Press.

Castro AE, Heuschele WP: *Veterinary diagnostic virology*, St Louis, MO, 1992, Mosby Year Book.

Fenner F, Bachmann PA, Gibbs EPJ, et al: *Veterinary virology*, New York, 1987, Academic Press.

Kaaden OR, Riddle KE, Wernery U: Falcon pox in the Middle East. *Middle East Falcon Research Group Specialist Workshop*, Abu Dhabi, 1995.

Kinne J, Oliveros S, Joseph S, et al: Severe outbreak of pox in captive houbara bustards (*Chlamydotis undulata undulata*) in Morocco. *Proceedings of the European Association of Avian Veterinarians*, Zürich, pp 78–85, 2007.

Mayr A: *Rolle/Mayr medizinische mikrobiologie, infektions und seuchenlehre*, Stuttgart, Germany, 1993, Ferdinand Enke.

Samour J, Kaaden OR, Wernery U, et al: An epornitic of avian pox in houbara bustards (*Chlamydotis undulata*), *Zentralbl Veterinarmed B* 43:287–292, 1996.

Wernery U, Manvell R: Avian viral diseases in the United Arab Emirates (UAE). *Proceedings of the European Association of Avian Veterinarians*, Tenerife, pp 72–78, 2003.

Wernery U, Basker VJ, Joseph S, et al: An attenuated vaccine protects houbara bustards from systemic pox. *Proceedings of the European Association of Avian Veterinarians*, Zürich, pp 86–93, 2007.

BACTERIAL DISEASES

Peernel Zwart

CHLAMYDIOSIS

Definition

Chlamydiosis is an infectious disease caused by a gram-negative obligate intracellular organism (Figs. 14-31 to 14-33 and Table 14-8).

Etiologic Agent

The etiologic agent is *Chlamydia psittaci.* The species is divided in serotypes (genotypes).

Distribution

The infection occurs worldwide.

Species Susceptible

Psittacines: Genotype A (considered the major genotype for human psittacosis)

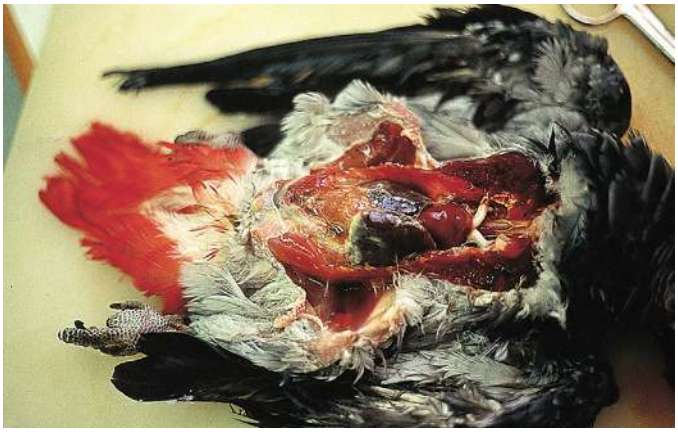


FIGURE 14-31 Chlamydiosis in an African grey parrot (*Psittacus erithacus*). Typical postmortem findings include peritonitis and serositis, as observed in this photograph. (Courtesy U. Wernery.)

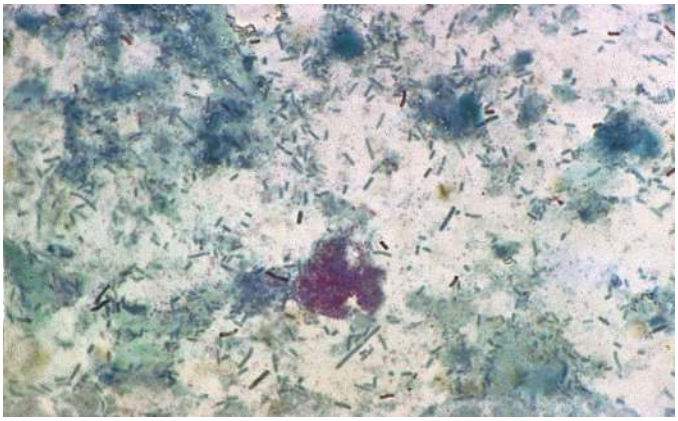


FIGURE 14-32 Intestinal contents contain larger aggregates of *Chlamydia* sp. Mealy amazon (*Amazona farinosa*). Stamp, ×1000.

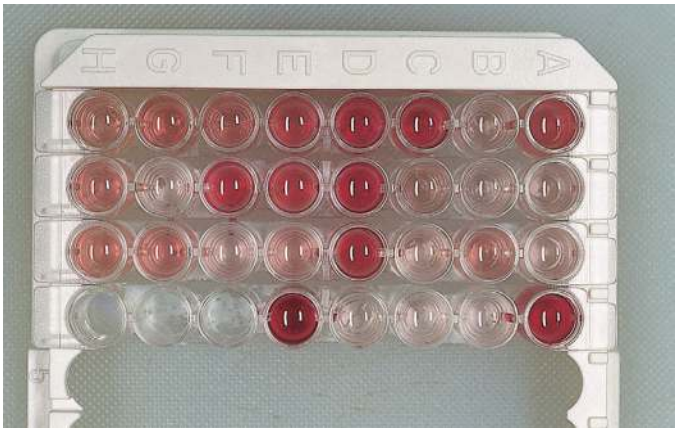


FIGURE 14-33 Enzyme-linked immunosorbent assay test for *Chlamydia* sp. antigen detection (Ideia EIA test, Oxoid Ltd, UK) widely used in birds. Results from a group of birds tested. The positive reactions are seen as red-magenta and negative reactions as clear. Pale pink reactions should be read with the aid of a colorimeter or the patient should be retested. (Courtesy U. Wernery.)

TABLE 14-8 Chlamydiosis: Clinical Signs, Postmortem Changes, and Differential Diagnosis

Clinical Sign	Postmortem Changes	Differential Diagnosis
Conjunctivitis	Swelling of eyelids	Pox, irritation, vitamin A deficiency
Keratitis	Hyperemia; exudation leads to adhesion of eyelids	
Nasal exudation	Hyperemia, exudation	Hyperemia, exudation
Respiratory distress	Pneumonia, airsacculitis	Aspergillosis
Fluffy feathers		General infections
Inappetence	Gastroenteritis	General infections
Diarrhea (green-gray)	Enteritis, swollen liver and spleen	Parasitic infections, intestinal mycosis, enteritis
Polydipsia (pigeon)		Enteritis

Feral pigeons: Genotype B (considered endemic in nonpsittacine birds)
Ducks: Genotype C
Poultry and turkeys: Genotype D
Various birds: Genotypes E and F (both are rare)

Transmission

Respiratory infections are especially important in the spread of the disease because nasal secretions may be rich in organisms, and feces also may contain large numbers of organisms. Other infective materials include tears, ocular exudate, and crop food or crop milk. Direct infection from bird to bird is accomplished through crop milk in pigeons. Indirect infection occurs via inhalation of droplets of nasal secretion or dried fecal particles. The new host is infected via the epithelia of either the respiratory or the digestive tract. When birds are caught in an aviary, whirling dust mixed with dried feces and feather particles is inhaled deeply both by birds and humans. Additional stress can evoke a manifest disease in latent carriers.

Diagnosis

Impression smears of organs (spleen, lung, liver, and intestinal contents) and lesions, stained with Stamp, give a first impression. Cultivation methods are laborious, time-consuming, and replaced by serologic techniques. Serologic tests are available. An enzyme-linked immunosorbent assay (ELISA) test is fairly sensitive, although it only indicates previous contact with the agent and false-negatives are possible. The prevalent method is the outer membrane protein A (ompA) genotype-specific real-time polymerase chain reaction.

Materials to be sent (before treatment with antimicrobial agents) for testing include:

1. Whole blood submitted in a heparin ethylenediaminetetraacetic acid containing tube (0.2 cc minimum).
2. Cloacal swab (collected on cotton-tipped wooden sticks and shipped in a specific transport medium)
3. Feces sample submitted in a sterile container

Treatment/Prevention

The therapeutic regimen should be meticulously and conscientiously followed to reach a favorable result.

- Enrofloxacin 10 to 15 mg/kg bodyweight.
- Doxycycline 75 mg/kg intramuscularly (IM) in the breast musculature, 9 injections at 5-day intervals.
- Chlortetracycline (CTC) 10 mg/kg orally daily over 45 days. CTC can also he dosed orally with specific medicated food or as a self-made prescription of a boiled mixture of seeds, rice, and water (2:2:3), to which 5% CTC is added.
- Stress must be minimized in the infected bird or flock. Breeding must be stopped. Any concurrent disease must be treated. If medicated pellets are to be used, the bird should be gradually converted to a pelleted diet. Supportive therapy, especially with multivitamins, is indicated.
- Prevention in the clinic or laboratory with 1:1000 dilution of quaternary ammonium compounds (alkyldimethylbenzylammonium chloride, e.g., Roccal or Zephiran) is effective, as well as 70% isopropyl alcohol, 1% Lysol, 1:100 dilution of household bleach (2 1/2 tablespoons per gallon), or chlorophenols.

AVIAN TUBERCULOSIS

Definition

Generalized chronic disseminated, granulomatous infectious disease caused by acid-fast bacteria (Figs. 14-34 to 14-41 and Table 14-9).

Etiologic Agent

The etiologic agent is *Mycobacterium avium* complex; i.e., *M. avium*, *M genavense*, *M. intracellulare*, and other strains. In some regions, *M. genavense* outnumbers the cases of *M. avium* (Tell *et al.* 2001).Various serotypes of *M. avium* occur. Geographic differences in the distribution of serotypes exist. In psittacines and wood pigeons (*Columba palumbus*) a specific enteric infection occurs that resembles paratuberculosis. On the basis of DNA analysis the "wood pigeon" mycobacteria are classified as *M. avium* subsp. *columbae* (Saxegaard and Baess, 1988).

Distribution

The infection occurs worldwide.

Species Susceptible

In principle all birds are susceptible to *M. avium/intracellulare* and *M. genavense*; however, the disease is rare in budgerigars and psittacines. Both species reveal a characteristic ingestion infection. The lesions are

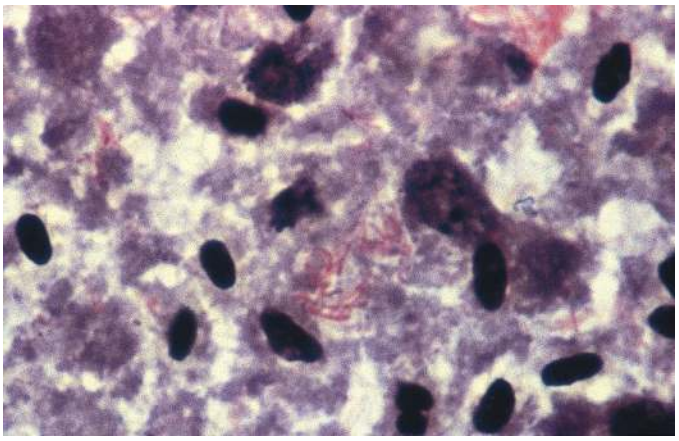


FIGURE 14-34 Acid-fast bacilli (Ziehl–Neelsen stain) in the feces of a gyrfalcon (*Falco rusticolus*), showing pink-stained rods of bacteria characteristic of *Mycobacterium* sp. ×1000. (Courtesy U. Wernery.)

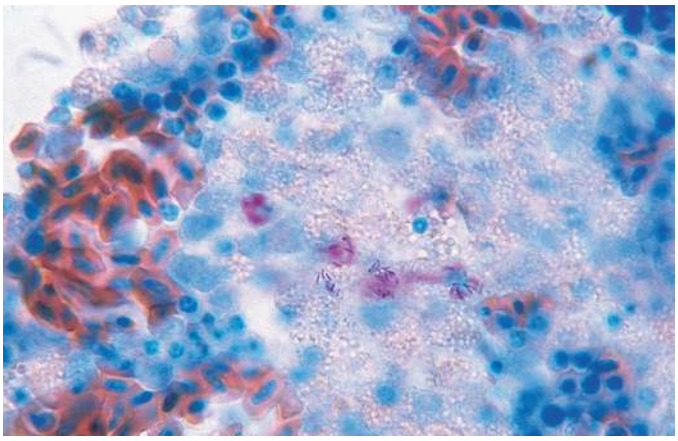


FIGURE 14-35 Bone marrow with acid-fast organisms located in macrophages of a green-cheeked amazon parrot (*Amazona viridigenalis*). Ziehl–Neelsen stain, ×1000.

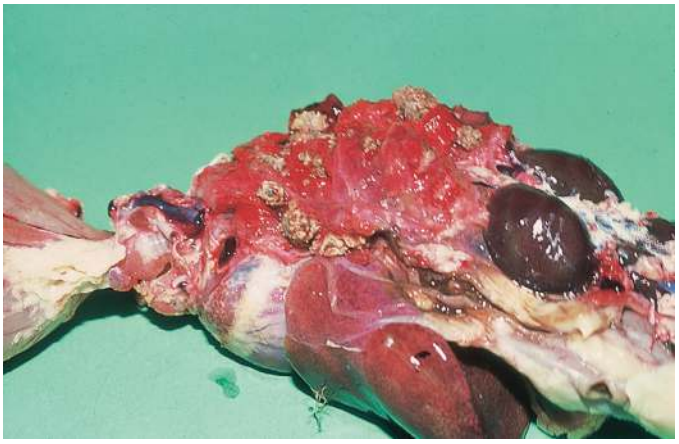


FIGURE 14-36 Lung dotted with tubercles, most probably caused by secondary hematogenous spread in this goshawk (*Accipiter gentilis*).

FIGURE 14-37 Tubercles in the liver, lungs, and air sacs of this ring-necked pheasant (*Phasianus colchicus*).

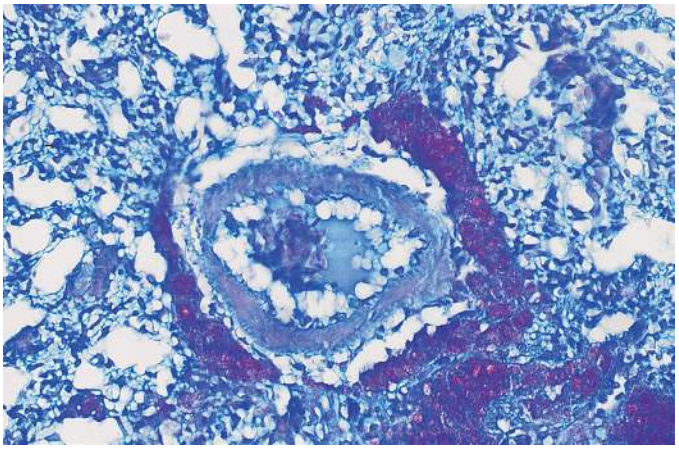


FIGURE 14-38 Localization of *Mycobacterium avium/intracellulare* in a lymph vessel in lung tissue surrounding an artery in this canary (*Serinus canaria*). Ziehl–Neelsen stain, ×250.

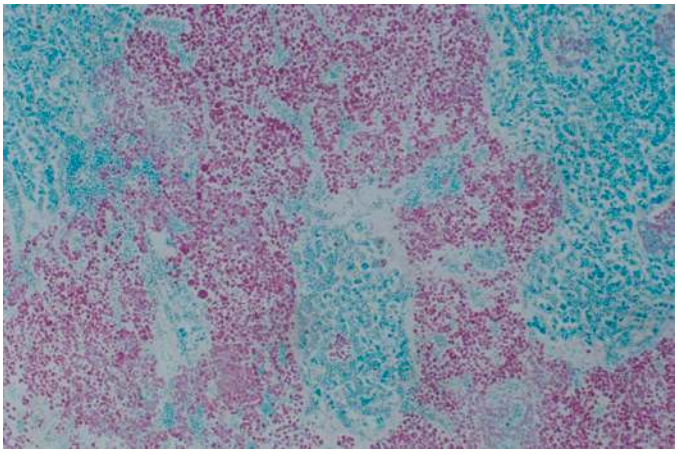


FIGURE 14-39 Spleen showing large nonencapsulated fields of macrophages loaded with *Mycobacterium avium/intracellulare* in a blue-fronted parrot (*Amazona aestiva*). Ziehl–Neelsen stain, ×250.

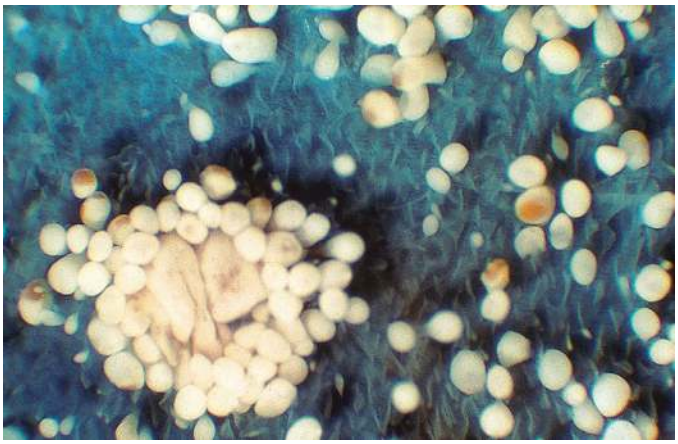


FIGURE 14-40 Small intestine. Enlargement of villi caused by accumulations of acid-fast mycobacteria (*Mycobacterium avium* subsp. *columbae*) in macrophages in a wood pigeon (*Columba palumbus*).

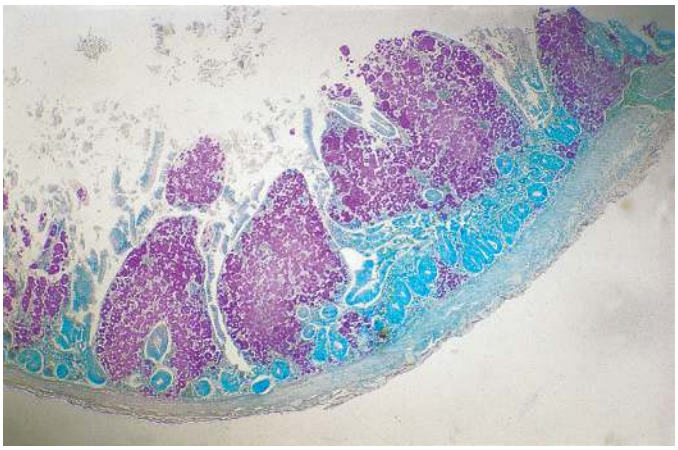


FIGURE 14-41 Small intestine, revealing enlargement of villi caused by massive accumulation of acid-fast mycobacteria (*Mycobacterium avium* subsp. *columbae*) in macrophages in an orange-winged amazon parrot (*Amazona amazonica*). Ziehl–Neelsen stain, ×250

primarily located in the intestinal tract, liver, and spleen, to be distributed to all organs at a later stage. In canaries and, in particular, Gouldian finches the lesions are located especially in the lungs. Woodpeckers showing an enlarged abdomen revealed an even distribution of acid-fast organisms accumulated in large numbers in macrophages. *M. avium* subsp. *columbae* is found in wood pigeons and psittacines located primarily in the intestinal villi.

Transmission

Transmission is mainly by oral uptake, especially in open tuberculosis (shedding via feces). Infection by aspiration is extremely rare in birds. It occurs especially in ducks when a fountain sprouts mud loaded with the bacteria. Infection is by direct contact, (i.e., conjunctivitis in ducks searching for food in the bacilli-loaded floor of a pond).

Diagnosis

Radiologic examination (Psittaciformes) reveals enlarged liver, spleen, and/or small intestine. Diagnosis is made by endoscopic examination of parenchymatous organs with biopsy and examination for acid-fast organisms. Fecal examination is performed for acid-fast organisms

TABLE 14-9 Avian Tuberculosis: Clinical Signs, Postmortem Changes, and Differential Diagnosis

Clinical Sign	Postmortem Changes	Differential Diagnosis
Chronic emaciation		Coligranulomas, yersiniosis, listeriosis, mycosis
Ruffled feathers, slow molting	Tubercles in organs	Chronic pathology of parenchymatous organs
Subcutaneous granulomata	Tubercles	Coligranulomas
Diarrhea	Tuberculous ulcerating granulomata Enlargement of intestinal villi (Psittaciformes, pigeon)	*Macrorhabdus ornithogaster* infection Candidiasis
Dyspnea (Passeriformes)	Larger areas of consolidation	Aspergillosis, chronic interstitial pneumonia
Paralysis	Tubercles in bone marrow of tibia and femur (Psittaciformes), arthritis Adenocarcinoma	Salmonellosis, renal adenocarcinoma (budgerigar), tumor of testicle (budgerigar)

(episodes of nonshedding may occur). Examination of blood reveals leukocytosis and heterophilia. Blood test with ELISA is especially suitable for flock diagnosis. At postmortem impression, smears of lesions stained with Hemacolor, Giemsa, or a comparable stain may reveal macrophages with rod-shaped blank spaces in the protoplasm, representing the unstained mycobacteria.

Treatment/Prevention

In view of the zoonotic capacities of the various mycobacterium species involved, therapy is not applied. Prevention is long-duration quarantine (3 to 5 months) for birds to be introduced into a colony.

PSEUDOTUBERCULOSIS (YERSINIOSIS)

Definition

Generalized disseminated infectious disease caused by gram-negative bacteria (Figs. 14-42 and 14-43 and Table 14-10).

Etiologic Agent

The etiologic agent is *Yersinia pseudotuberculosis* (serotypes 1 and 2).

Distribution

The infection occurs worldwide.

Species Susceptible

A wide variety of Passeriformes are susceptible, in particular aracaris and toucans. The disease is rare in budgerigars, Psittaciformes, and pigeons.

Transmission

Transmission is by fecal contact and by food contaminated by feces from small rodents.

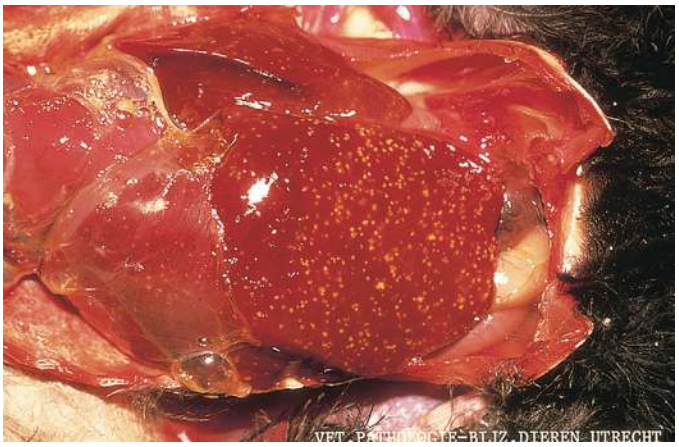


FIGURE 14-42 Liver with miliary foci caused by enormous spread of *Yersinia pseudotuberculosis* bacteria in an ornate umbrella bird (*Cephalopterus ornatus*).

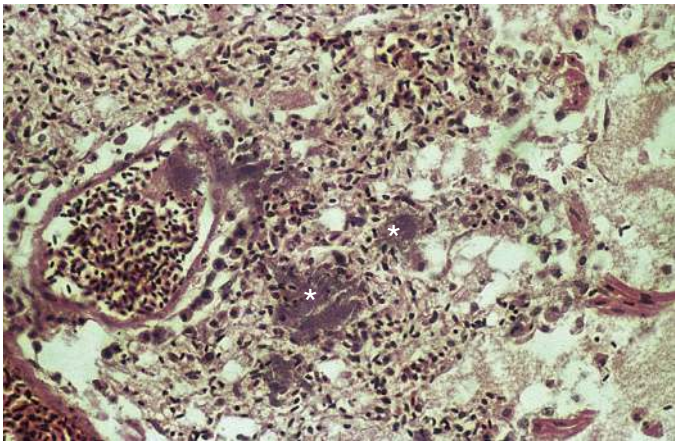


FIGURE 14-43 Lung with accumulations of *Yersinia pseudotuberculosis* bacteria in pulmonary blood–capillaries and hyperemia in a crimson rosella (*Platycercus elegans*). H&E, ×250.

Treatment/Prevention

Treatment includes antibiotics (bactericidal, e.g., ampicillin 1000-2000 mg or amoxicillin 200-400 mg per liter of water) over a period of 14 days and eventually longer and third-generation cephalosporins and fluoroquinolones. In canaries, enrofloxacin offers promising results (Haesebrouck *et al.*, 1995). Prevention includes good hygiene. Prevent contact with free-living birds. Stores for seeds used as food should be rodent proof. Mice, offered as food to larger birds, should be laboratory bred. Larger birds (toucans) can be vaccinated with a killed vaccine (Zwart *et al.*, 1981).

SALMONELLOSIS

Definition

Salmonellosis (Figs. 14-44 to 14-47 and Table 14-11) is a contagious infection caused by a gram-negative coccobacillus.

Etiologic Agent

The etiologic agent is *Salmonella* spp. and in general is *S. enterica* serovar Typhimurium (Porwollik *et al.* 2004). In racing pigeons, it is the serotype Typhimurium var. Copenhagen (which lacks the antigen 05).

TABLE 14-10 Pseudotuberculosis: Clinical Signs, Postmortem Changes, and Differential Diagnosis (Concerns Especially Passeriformes)

Clinical Sign	Postmortem Changes	Differential Diagnosis
High mortality (acute cases)		Inhalation of PTFE fumes
Apathy	Foci in liver and spleen	Salmonellosis, tuberculosis
Anorexia	Inflammation of the ceca (lymphoid)	Salmonellosis, tuberculosis
Respiratory distress	Foci in lungs (in toucans peracute catarrhal pneumonia)	Salmonellosis, colibacillosis, atoxoplasmosis, mycosis (aspergillosis)

PFTE, Polytetrafluoroethylene (Teflon).

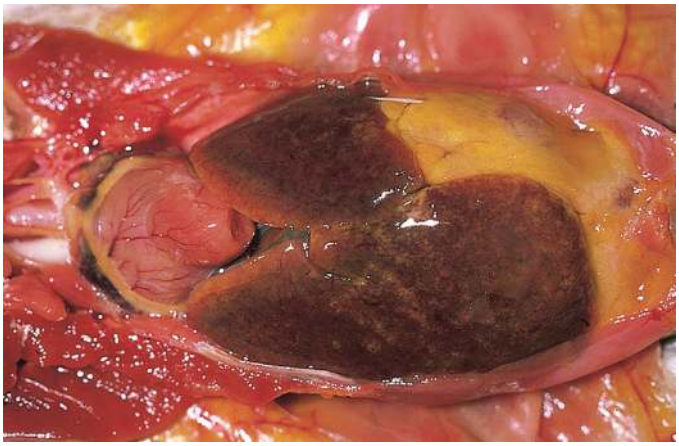


FIGURE 14-44 Salmonellosis with inflammation of the liver in a turtle dove (*Streptopelia turtur*). (Courtesy Dr. G.M. Dorrestein.)

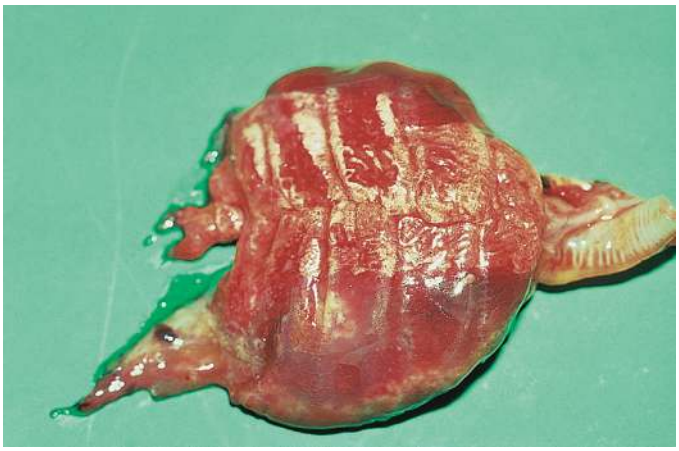


FIGURE 14-45 Catarrhal pneumonia caused by infection with the serotype Typhimurium var. Copenhagen in a pigeon.

Distribution

Salmonellosis occurs worldwide, especially in outdoor aviaries. Newly arrived individuals can introduce the disease.

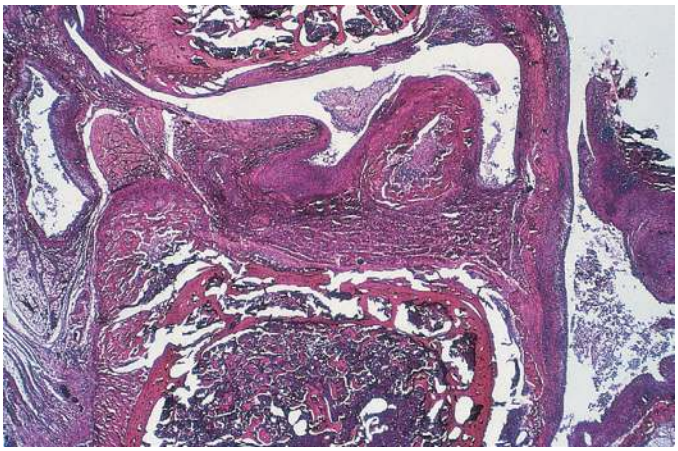


FIGURE 14-46 Arthritis and osteomyelitis in adjacent bone, caused by the serotype Typhimurium var. Copenhagen in a pigeon. H&E, ×10.

FIGURE 14-47 Abscessation of the brain caused by *Salmonella enterica* serovar Typhimurium infection in a canary.

Species Susceptible

Susceptible species include racing pigeons and Passeriformes with occasional infections in Psittaciformes (including the budgerigar). Young pigeons and weakened birds in general are susceptible. Deficient feeding (old seeds and rancid vitamin preparations on an oil base); overcrowding; and a cold, humid climate are predisposing factors.

Transmission

The infection is most often transmitted by oral uptake, either during crop feeding or via contaminated foods, or by birds roosting in the aviary. Aerogenic transmission is known. Birds may become infected by carrier birds and occasionally by humans.

Diagnosis

Diagnosis is made from clinical signs (especially arthritis [pigeon]), postmortem findings, and bacteriologic examination (use enrichment media).

Treatment/Prevention

Clinically ill animals are separated and eventually euthanized (Passeriformes, budgerigars, and pigeons). According to the resistance test, antibiotics are used over a period of 14 to 21 days. The result of the therapy is checked by culture between 3 and 6 weeks after treatment is finished. Occasionally therapy must be repeated. Hygiene is essential, especially in caged and aviary birds (Passeriformes, budgerigars, and pigeons).

To prevent spread from other infected flocks, owners should be warned not to participate in bird exhibitions during the season because of the risk of carriers.

TABLE 14-11 Salmonellosis: Clinical Signs, Postmortem Changes, and Differential Diagnosis

Clinical Sign	Postmortem Changes	Differential Diagnosis
General distress	Septicemia, hepatitis with inflammatory foci	Yersiniosis, colibacillosis, psittacosis, adenovirus, paramyxovirus
Polydipsia, polyuria, diarrhea	Enteritis	*Candida* mycosis, helminths, coccidia, hexamites, cochlosomiasis
Respiratory distress	Pneumonia	Aspergillosis
Conjunctivitis	Inflammation	Toxoplasmosis
Panophthalmia	Inflammation	Toxoplasmosis
Arthritis	Inflammation	
Central nervous system symptoms	Brain abscess	Paramyxovirus, deltamethrin intoxication

ESCHERICHIA COLI INFECTIONS

Definition

Colibacillosis (Figs. 14-48 and 14-49 and Table 14-12) is a contagious infectious disease caused by a gram-negative bacterium.

Etiologic Agent

The etiologic agent is *E. coli* (various serotypes), eventually in association with other Enterobacteriaceae or *Candida* spp. infections (Prattis *et al.*, 1990).

Distribution

The infection occurs worldwide.

Species Susceptible

Almost all species of bird at a given time may suffer from colibacillosis.

Transmission

The infection is transmitted by oral uptake of *E. coli* from the environment and excreters. Poor hygiene, overcrowding, stress factors, nutritional deficiencies, and concomitant infections are important factors in outbreaks.

Diagnosis

Diagnosis is made postmortem and with bacteriologic examination. If possible serotyping of the *E. coli* is done. In seed-eating Passeriformes, budgerigars, and other Psittaciformes, a fecal smear stained with Hemacolor will reveal large numbers of rod-shaped bacteria. In contrast, healthy birds in the groups previously mentioned that have little contact with feces harbor only minimal numbers of bacteria in the intestinal tract.

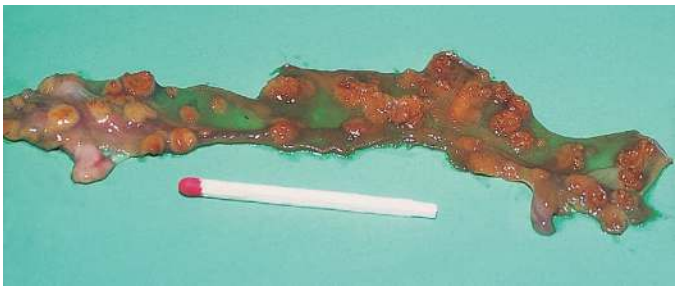


FIGURE 14-48 Multiple granulomata along the intestinal tract, caused by infection with *Escherichia coli* in an African grey parrot (*Psittacus erithacus*).

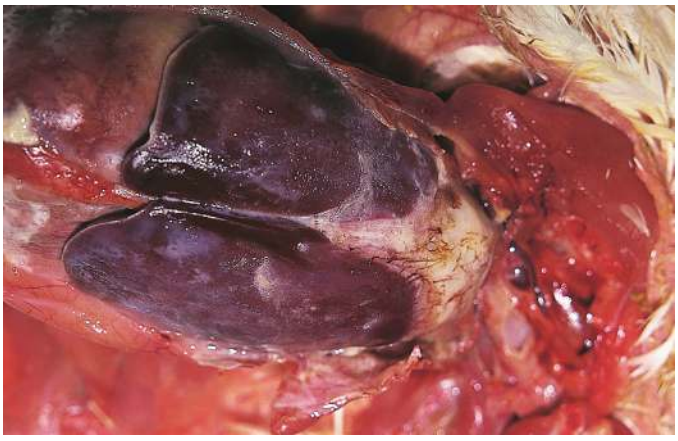


FIGURE 14-49 Colibacillosis in a domestic chicken (*Gallus domesticus*). (Courtesy Dr. U. Wernery.)

PASTEURELLOSIS

Definition

Pasteurellosis (Table 14-13) is a contagious infectious disease caused by a gram-negative bacterium.

Etiologic Agent

The etiologic agent is *Pasteurella multocida* (several serotypes) and occasionally *P. gallinarum.*

Distribution

The infection occurs worldwide.

Species Susceptible

The disease occurs with low frequency in canaries and budgerigars and is very rare in pigeons. Weakened young and old individuals develop the disease.

Transmission

The disease is transmitted by bites of cats and rats. Contact with contaminated rodent feces is also considered in the transmission of the disease.

Diagnosis

Diagnosis is made with clinical signs, bite wounds, bacteriologic examination of secretions, and postmortem examination. Blood smear reveals large numbers of bipolar bacteria.

TABLE 14-12 *Escherichia Coli:* Clinical Signs, Postmortem Changes, and Differential Diagnosis

Clinical Sign	Postmortem Changes	Differential Diagnosis
Adult Birds		
Enzootic		*Salmonella*
General malaise, emaciation		Aeromonads, pseudomonads
Apathy, conjunctivitis, rhinitis		Staphylococci
Diarrhea (Psittaciformes, pigeon)	Enteritis	Capillariidosis (pigeon)
Dyspnea	Pneumonia, airsacculitis	Aspergillosis
Swollen joints	Arthritis	Salmonellosis
Central nervous system symptoms		Paramyxovirus infection (pigeon)
Nestlings		
Sudden death		
Poor growth		*Cochlosoma* (Passeriformes)
Diarrhea		
Omphalitis (pigeon)		
Distended abdomen	Retained yolk sac	
Wet skin		
Dirty, humid nest		

TABLE 14-13 Pasteurellosis: Clinical Signs, Postmortem Changes, and Differential Diagnosis

Clinical Sign	Postmortem Changes	Differential Diagnosis
General malaise	Enlarged liver and spleen	Infections leading to septicemia (enteric), streptococci (*Streptococcus bovis*), *Escherichia coli* infection, and others
Respiratory distress	Edema of the lungs	Hyperthermia, aspergillosis
Nasal exudate	Rhinitis	Chlamydiosis
Conjunctivitis		Chlamydiosis
Overfilling of nasal sinuses		
Anorexia, diarrhea	Petechiae in intestinal wall	Coccidiosis, capillariidosis

Treatment/Prevention

Treatment includes wound care. Antibiotics are dosed according to sensitivity test. If such a test is not available, then doxycycline (75 to 100 mg/kg IM every second day over a period of 6 to 8 days) may prove to be effective. Prevention is by hygiene and cat- and rat-proof construction of aviaries.

OTHER BACTERIAL DISEASES

Definition

Different species of bacteria may cause small epidemics or disease among the birds under consideration (Figs. 14-50 to 14-52 and Table 14-14).

Etiologic Agents

- *Erysipelothrix rhusiopathiae*
- *Listeria monocytogenes*
- *Streptococcus* spp. and *Staphylococcus* spp.; in pigeons, especially *Streptococcus bovis* (Devriese et al., 1990)
- *Helicobacter jejuni*
- *Klebsiella* spp.
- *Pseudomonas/Aeromonas* spp.

Distribution

These infections are widespread.

Species Susceptible to Previously Listed Etiologic Agents

- *E. rhusiopathiae* is very rare in Passeriformes, budgerigars, Psittaciformes, and pigeons.
- Canaries, pigeons, and parrots are susceptible to *L. monocytogenes.*
- Streptococci and staphylococci are frequent in Passeriformes, budgerigars (especially birds in poor condition), and Psittaciformes, and enteric infections with *S. bovis* are especially seen in pigeons..
- *H. jejuni* is frequent in ornamental finches, and much less in canaries. Rare in budgerigars, Psittaciformes, and pigeons.
- *Klebsiella* spp. occur occasionally in canaries; frequently in ornamental finches and other Passeriformes; and very rare in budgerigars, Psittaciformes, and pigeons.
- *Pseudomonas/Aeromonas* spp. occur occasionally in Passeriformes and in immunosuppressed (antibiotic treatment) budgerigars, but are rare in pigeons.

Transmission

- *E. rhusiopathiae* is transmitted orally by contact with contaminated food or water; trauma will occasionally provide an entry.
- *L. monocytogenes* is believed to be transmitted by contact with rats.
- Streptococci and staphylococci enter via lesions of the mucosa in the oral cavity, digestive tract, respiratory tract, and/or conjunctiva.
- *H. jejuni* is likely to be transmitted by oral contact. Birds with diminished resistance are especially susceptible. This is generally caused by poor-quality food (lack of animal protein, vitamins, and minerals) or poor management (overcrowding, humidity, and cold environment).
- *Klebsiella* spp. invade after oral contact.
- *Pseudomonas/Aeromonas* spp. originate from a humid environment (water) containing some protein. Oral infection predominates. Severe respiratory infections are known after spraying water containing *Pseudomonas* spp.

Diagnosis

- *E. rhusiopathiae:* Bacteriologic examination
- *L. monocytogenes:* Bacteriologic examination
- Streptococci and staphylococci: Bacteriologic examination
- *H. jejuni:* Bacteriologic examination (special medium); fecal smear stained with Hemacolor reveals numerous undulated bacteria
- *Klebsiella* spp.: Bacteriologic examination
- *Pseudomonas/Aeromonas* spp.: Bacteriologic examination

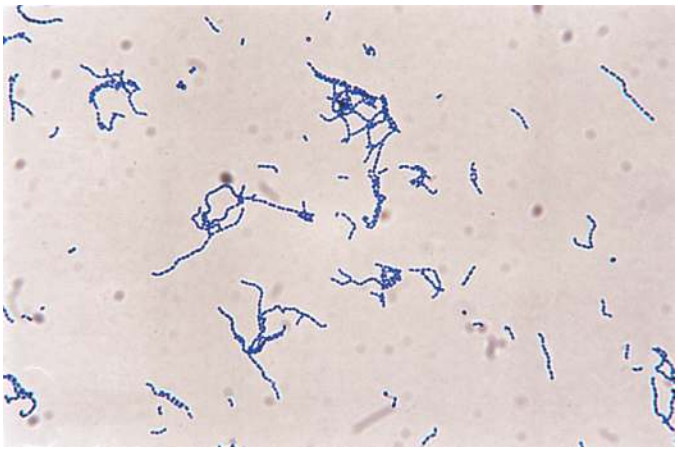


FIGURE 14-50 Microscopic appearance of *Streptococcus* sp. in Gram stain showing gram-positive, chain-forming cocci, ×1000. (Courtesy C. Silvanose.)

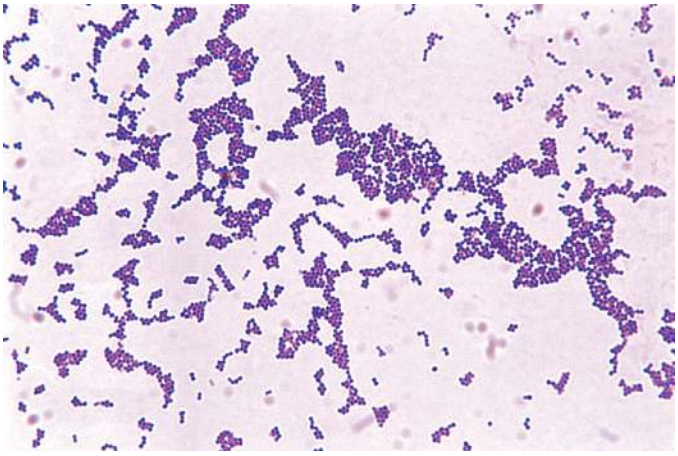


FIGURE 14-51 Microscopic appearance of *Staphylococcus* sp. in Gram stain showing gram-positive cluster-forming cocci, ×1000. (Courtesy C. Silvanose.)

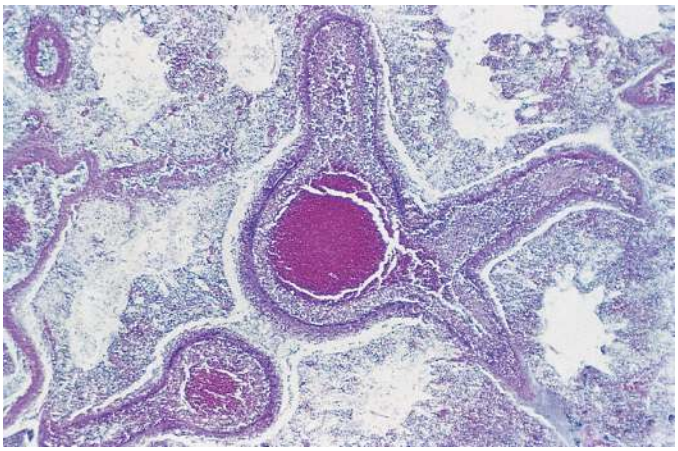


FIGURE 14-52 Lung with diffuse pneumonia and accumulations of *Pseudomonas aeruginosa* in lymph vessels situated around the blood vessels, a consequence of nebulizing water containing the microorganism in a canary. H&E, ×400.

TABLE 14-14 Other Bacterial Diseases: Clinical Signs, Postmortem Changes, and Differential Diagnosis

Clinical Sign	Postmortem Changes	Differential Diagnosis
Erysipelothrix rhusiopathiae		
Sudden death, acute illness	Septicemia, intoxication	Bacterial infections
Listeria monocytogenes		
Sudden death	Septicemia, intoxication	Bacterial infections
Streptococci and Staphylococci		
Abscessation, omphalitis	Septicemia, abscess	*Escherichia coli* infections, other respiratory distress such as tracheitis (Passeriformes) and pneumonia (budgerigar), bacterial infections
Bumblefoot	Swollen foot, abscessation (Psittaciformes)	Intoxication
Diarrhea	Enteritis (pigeon)	
***Helicobacter jejuni* (Passeriformes)**		
General malaise, whitish feces	Voluminous intestinal convolute, undigested starch in small intestine	Subchronic cochlosomosis
***Klebsiella* spp. (Budgerigar)**		
Respiratory distress	Pneumonia	Other bacterial infections
Central nervous system symptoms	Meningoencephalitis, hepatitis, nephritis	Adenovirus infection
***Pseudomonas* and *Aeromonas* spp.**		
Diarrhea, dehydration	Enteritis	Other bacterial infections
Pneumonia (Passeriformes)	Airsacculitis, pneumonia	

Treatment/Prevention

- *E. rhusiopathiae:* Ampicillin and amoxicillin orally via food and water
- *L. monocytogenes:* Ampicillin and amoxicillin orally via food and water
- Streptococci and staphylococci: Ampicillin and amoxicillin orally via food and water
- *H. jejuni:* Antibiotics and improvement of general condition (food and management)
- *Klebsiella* spp.: Antibiotics under the guidance of sensitivity tests
- *Pseudomonas/Aeromonas:* Antibiotics under the guidance of sensitivity tests.

REFERENCES

Devriese LA, Uyttebroek E, Gevaert D, et al: *Streptococcus bovis* infections in pigeons, *Avian Pathol* 19:429–434, 1990.

Haesebrouck F, Vanrompay M, de Herdt P, et al: Effect of antimicrobial treatment on the course of an experimental *Yersinia pseudotuberculosis* infection in canaries, *Avian Pathol* 24:273–283, 1995.

Porwollik S, et al: Characterization of *Salmonella enterica* Subspecies I. Genovars by use of microarrays, *J Bacteriol* 186:5883–5898, 2004.

Prattis SM, Cioffee Q, Reinhard G, et al: A retrospective study of disease and mortality in zebra finches, *Lab Anim Sci* 40:402–405, 1990.

Saxegaard F, Baess I: Relationship between *Mycobacterium avium*, *Mycobacterium paratuberculosis* and "wood pigeon mycobacteria." Determinations by DNA-DNA hybridization, *APMIS* 96:37–42, 1988.

Tell LA, Woods L, Cromie RL: Mycobacteriosis in birds, *Rev Sci Tech* 20:180–203, 2001.

Zwart P, Wiesner H, Göltenboth R: *Erfahrungen mit dem Einsatz einer Pseudotuberkulose-Totvakzine bei Vögeln, Verhandlungsbericht des XIII Internationalen Symposiums über die Erkrankungen der Zootiere* (vol 23), 1981, Institut für Zoo- und Wildtierforschung.

FUNGAL DISEASES

ASPERGILLOSIS

Patrick Redig

General Description

This fungal disease of the respiratory system (Figs. 14-53 to 14-74) is the most commonly occurring disease among wild birds held in captivity and is an occasional, but always possible, problem in companion birds. Rarely, it occurs among free-living birds that have become otherwise debilitated through injury or inanition. Although it may occur in individuals of virtually any species, there are clearly species predilections. Among North American raptors, goshawks (*Accipiter gentilis*), gyr falcons (*Falco rusticolus*), immature red-tailed hawks (*Buteo jamaicensis*), golden eagles (*Aquila chrysaetos*), and snowy owls (*Nyctea scandiaca*) are more likely to develop the disease. There is a tendency for raptors originating from arctic or subarctic climates to be more susceptible. A very useful and comprehensive review of aspergillosis in birds was published by Beernaert *et al.* (2010).

Several species of *Aspergillus* (*A. nigricans*, *A. terreus*, *A. nidulans*, *A. glaucus*, and *A. flavus*) may be involved in any of the common forms of this disease. In most clinical presentations among captive companion and falconry birds, *A. fumigatus* is by far the most commonly encountered organism. However, the occurrence of other species can lead to some of the variations seen in pathogenesis and response to treatment. Additionally, available serologic tests are highly specific for *A. fumigatus* and do not necessarily detect antibodies formed in response to one of the other species.

Text continued on p. 468

FIGURE 14-53 In one actual case of acute aspergillosis, a gyrfalcon (*Falco rusticolus*) was housed in a chamber adjacent to another in which a pointer dog was kept, separated by a short divider. A few days after straw was spread on the floor for the dog, the falcon succumbed to acute aspergillosis.

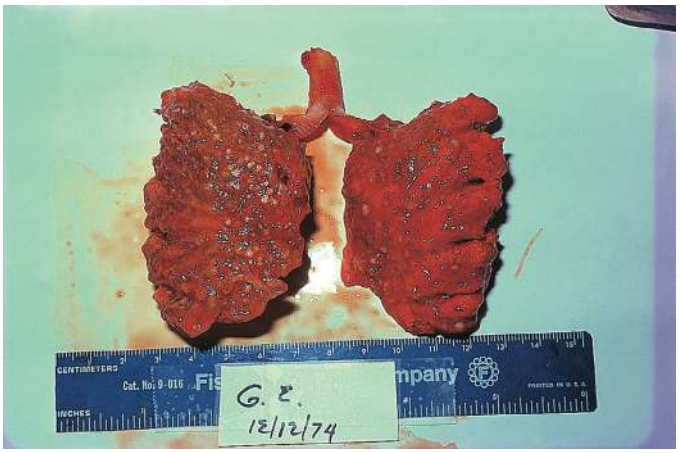


FIGURE 14-54 This pair of lungs was removed from a juvenile golden eagle (*Aquila chrysaetos*) that was admitted in severe respiratory distress and showing signs of an encounter with a porcupine. It was speculated that, in attempting to subdue the quilled prey item, the eagle inhaled a large number of spores that were present in leaf litter or other ground detritus. (Courtesy The Raptor Center, University of Minnesota.)

FIGURE 14-55 These lungs were removed from a gyrfalcon that was housed in a mews adjacent to a hayfield. The hay had been cut, but was rained on before being baled. After a week, the hay was turned with a hay rake and baled. A few days later the gyrfalcon succumbed acutely to aspergillosis. The lungs were hard and studded with thousands of miliary granulomas from which *Aspergillus fumigatus* was cultured. (Courtesy The Raptor Center, University of Minnesota.)

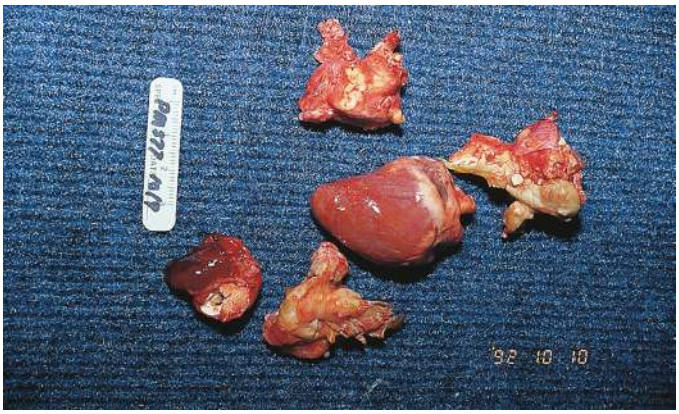


FIGURE 14-56 Chronic aspergillosis lesions removed from the air sacs of a gyrfalcon and displayed as they appeared in situ. The large mass anterior to the heart was found in the interclavicular air sac, a site that is more often affected in gyrfalcons than in other raptors. (Courtesy The Raptor Center, University of Minnesota.)

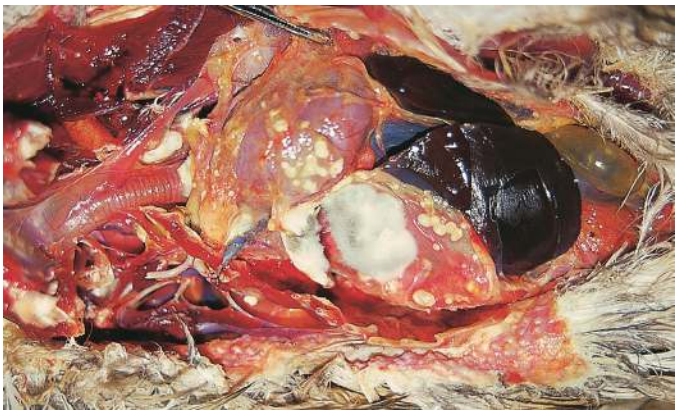


FIGURE 14-57 Lesions of aspergillosis may occur in just about any location throughout the body, including the pericardial sac, as shown here. This lesion occurred in a gyrfalcon. (Courtesy Dr. J. Samour.)

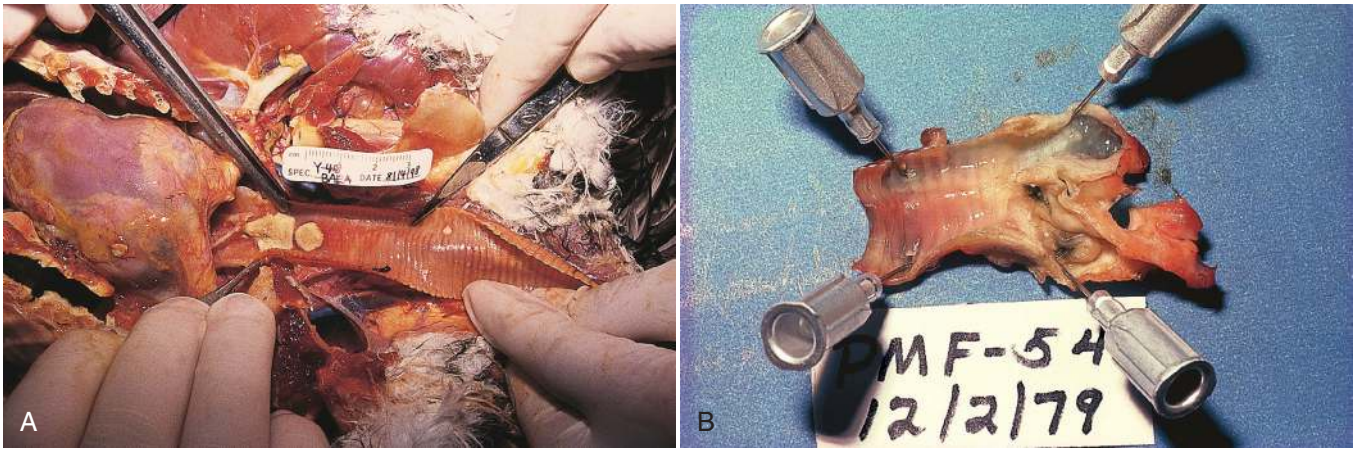


FIGURE 14-58 Lesions may occur in the main lumen of the trachea **(A)** or, more commonly, in the narrowed air passages of the syrinx **(B)**. Such cases exhibit rapid onset of progressively severe dyspnea but little debilitation. (Courtesy The Raptor Center, University of Minnesota.)

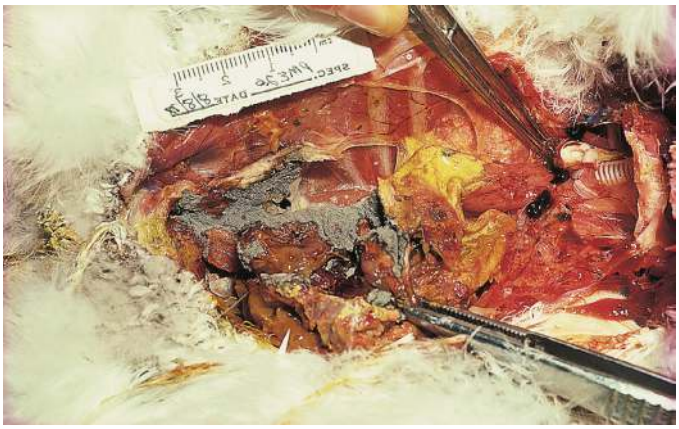


FIGURE 14-59 Free-growing mold (*Aspergillus fumigatus*) in the air sacs of a juvenile red-tailed hawk (*Buteo jamaicensis*). This case may represent gross immunologic failure. (Courtesy The Raptor Center, University of Minnesota.)

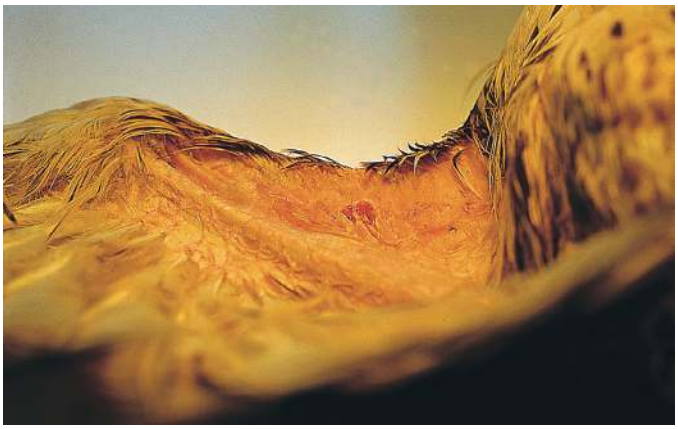


FIGURE 14-60 Cutaneous aspergillosis affecting the propatagium of the wing of a goshawk (*Accipiter gentilis*). The exudative lesion was secondary to an extensive lacerating wound, which had previously been treated with a hydroactive vapor seal dressing. The lesion eventually responded to daily application of enilconazole (dilution 10:1). Marked improvement occurred after 7 days and complete resolution within 12 d. It was necessary to debride the lesion extensively before each application. (Courtesy N.A. Forbes.)

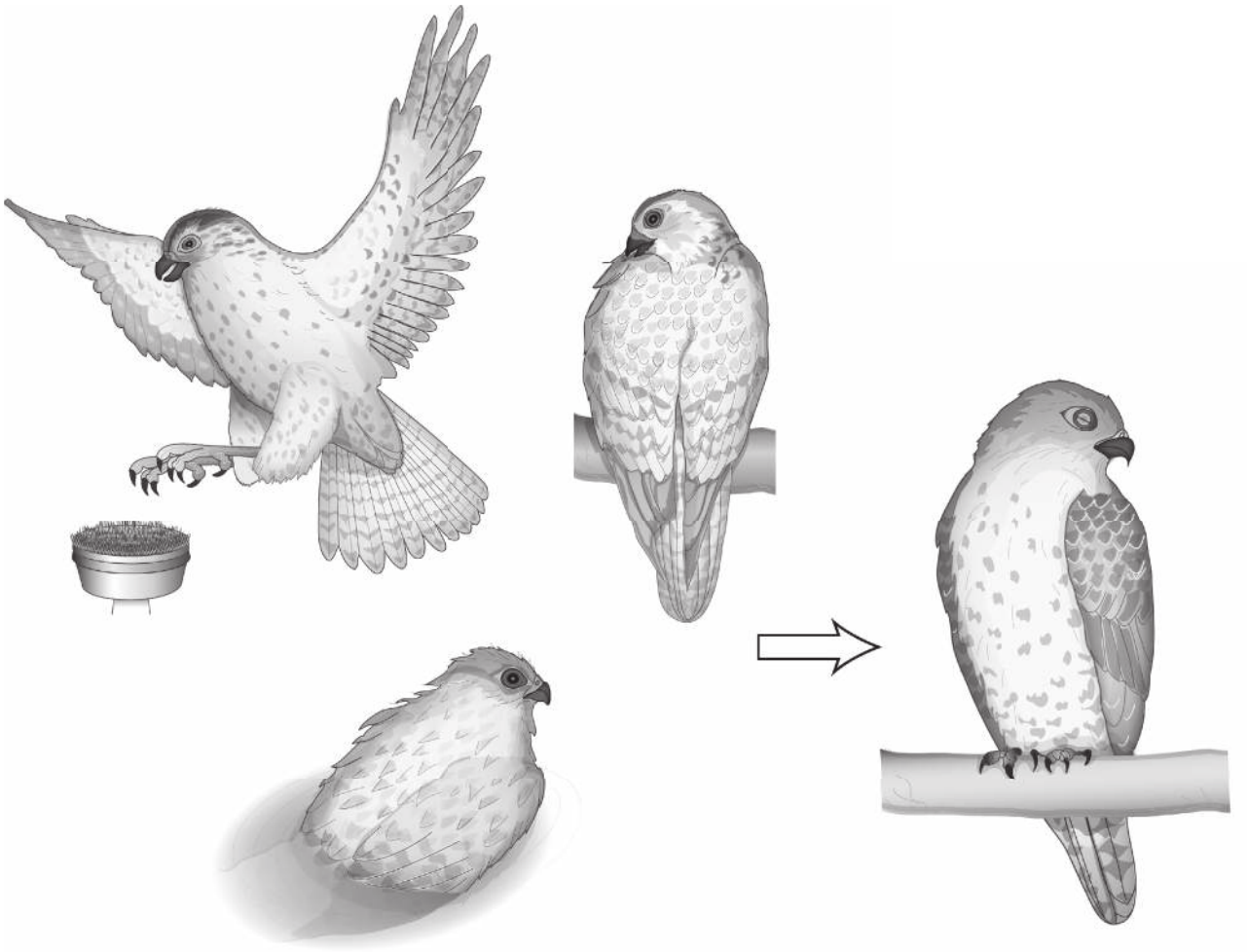


FIGURE 14-61 Subtle changes in behavior such as a decrease in preening activity, failure to bathe, or otherwise engage in routine activities are sometimes the first signs of the development of aspergillosis.

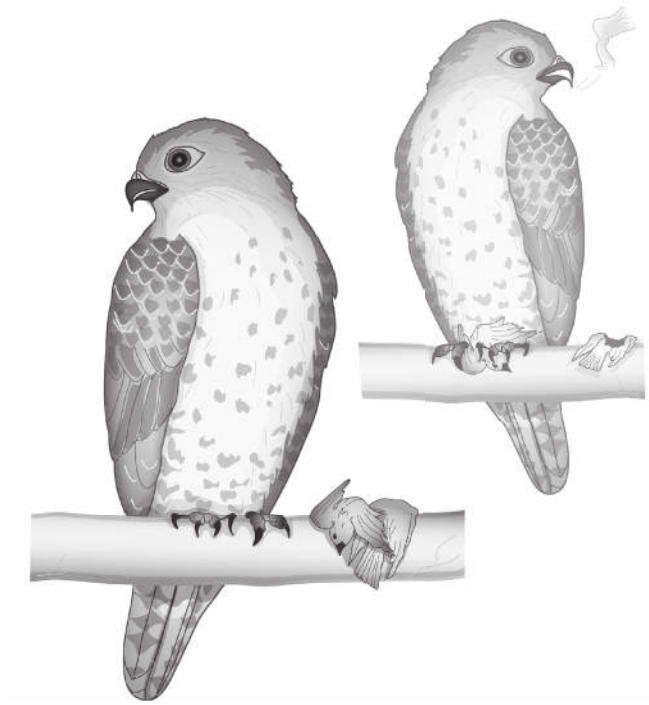


FIGURE 14-62 Another early sign of developing aspergillosis is partial anorexia. Affected birds may vigorously grasp a food item, pluck a few feathers from it, then sit with it clenched in their talons. If offered small pieces of meat by hand, they will take them in their beak and fling them away. Such behavior also may reflect gastrointestinal disease of other etiologies.

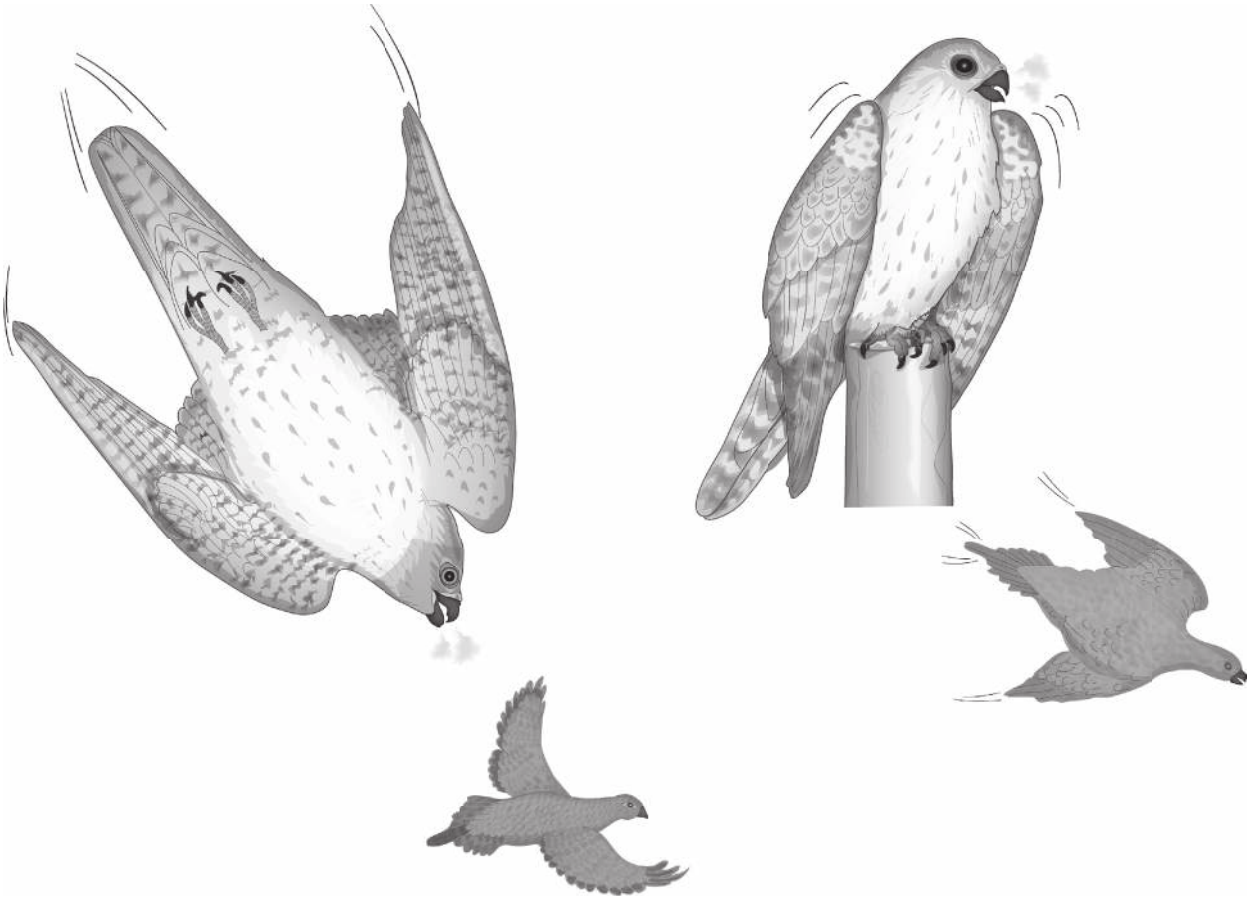


FIGURE 14-63 Loss of stamina or ability to pursue quarry may be an early sign of developing aspergillosis. The affected bird may mount to a regular pitch and begin its stoop when quarry is flushed, but quickly give up the chase and alight on the ground or an elevated perch, clearly out of breath.

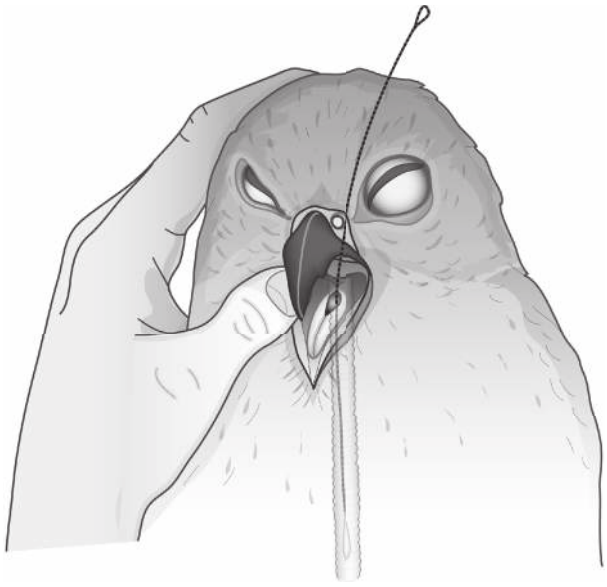


FIGURE 14-64 Tracheal culturing is conducted by passing a small nasopharyngeal swab deep into the trachea. This may be done with or without anesthesia. The material recovered on the swab is transferred immediately to Sabouraud dextrose agar and cultured at 37° C (98.6° F). Growth is typically apparent in 48 hours, but may require up to a week. Alternatively saline, at the rate of 3 cc/kg, may be flushed into the trachea, recovered by aspiration, and cultured in similar fashion. Cytologic examination may also be conducted on recovered material.

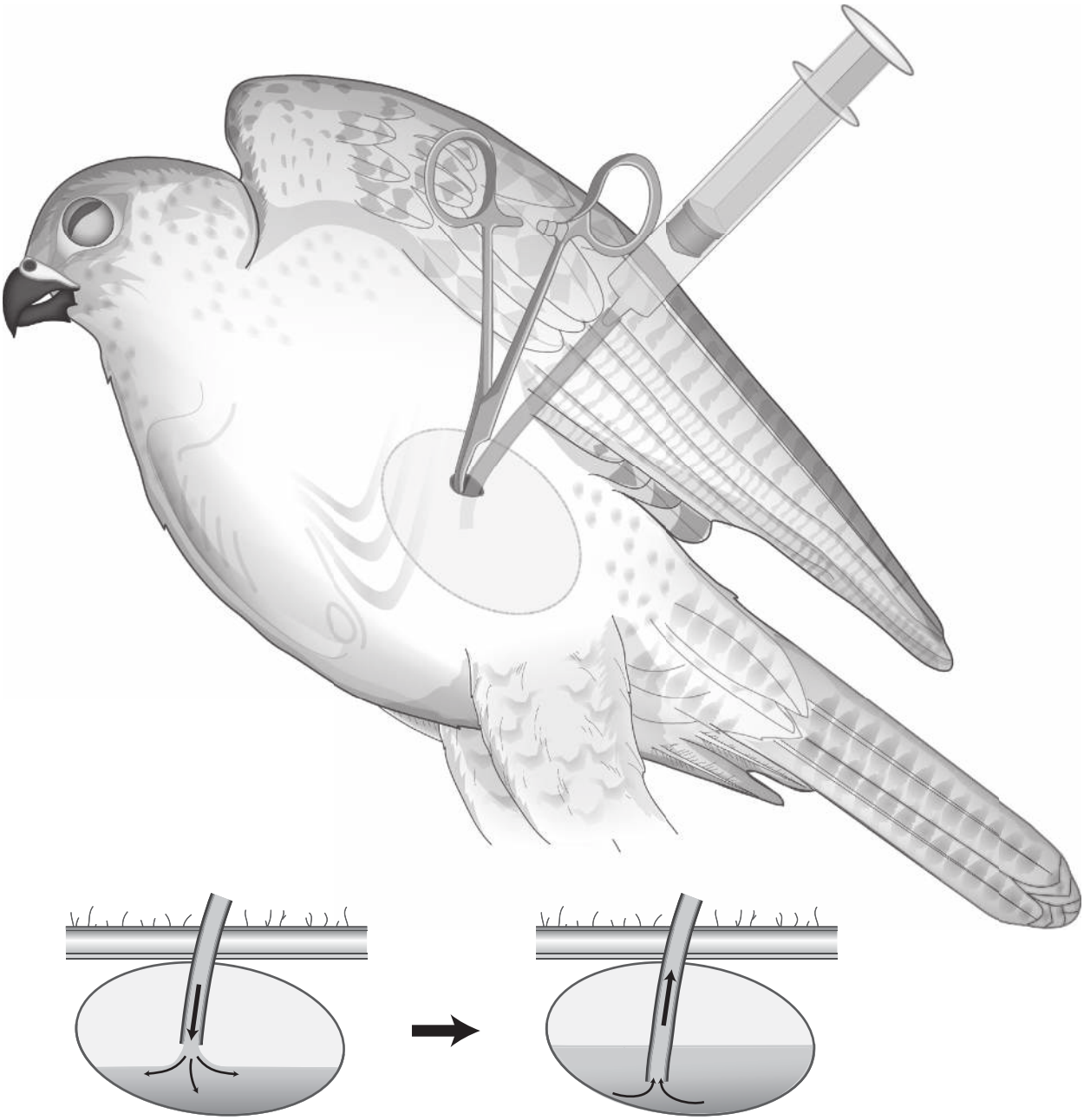


FIGURE 14-65 Air sacs may be cultured by irrigation and recovery of saline: 3 to 5 mL/kg may be injected through the body wall into the last intercostal space and recovered by aspiration with a urinary catheter or similar device. The recovered material is cultured and/or examined cytologically.

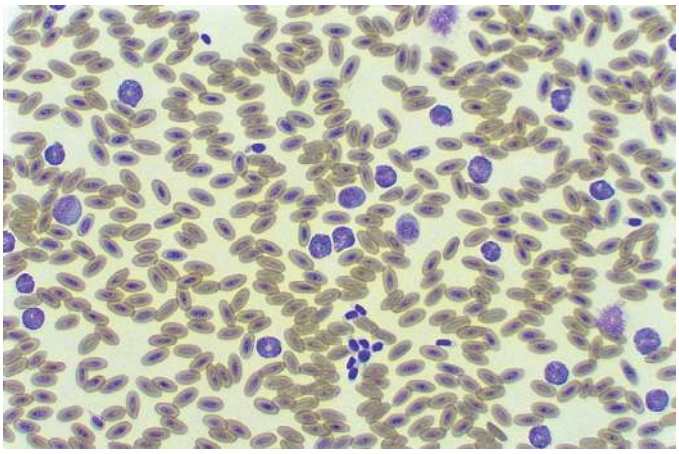


FIGURE 14-66 Elevated white cell counts characterized by heterophilia and monocytosis are characteristically seen with aspergillosis. Heterophils often exhibit toxic signs such as degranulation. Gyrfalcons have a less brisk response in the white cell compartment compared with other raptors, often exhibiting total white cell counts in the range of 12 to 15,000 cells/mm^3 and other raptors exhibit counts of 30,000 to 100,000 cells/mm^3, ×500. (Courtesy Dr. B. Aird.)

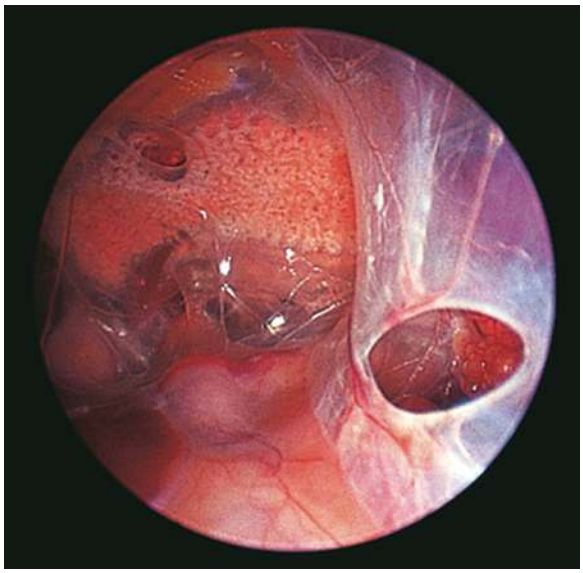


FIGURE 14-67 Endoscopic view of normal anatomy as seen at the level of the last intercostal space in a red-tailed hawk (*Buteo jamaicensis*). Note the clear, nonvascularized membranous nature of the air sac wall. The hole in the air sac was created with the endoscope to gain visual access to the anterior thoracic air sac). This effectively creates a communication between the two normally separate anterior and posterior portions of the avian respiratory system and should be avoided in a hunting falcon. (Courtesy Dr. M. Taylor.)

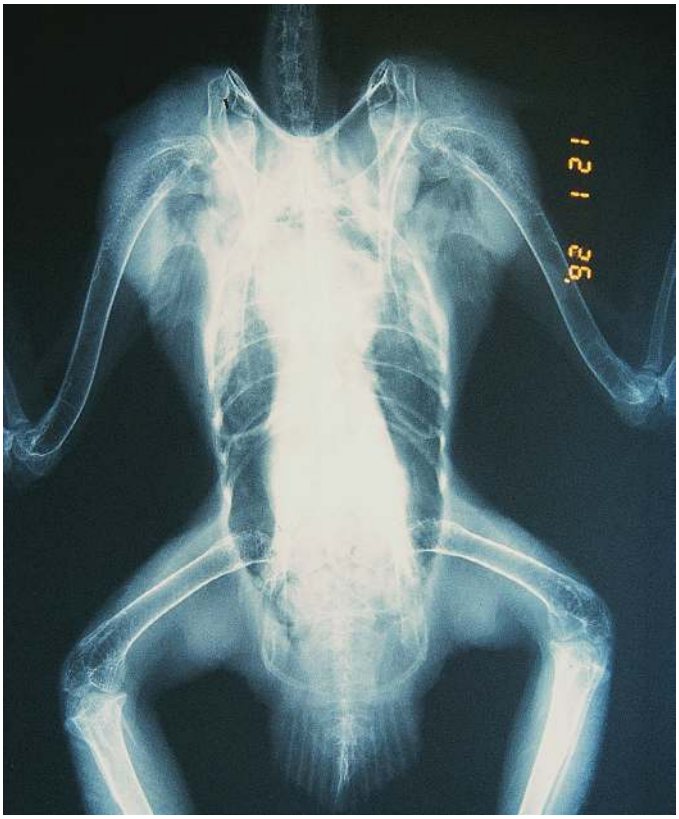


FIGURE 14-68 Radiograph of a gyrfalcon with well-developed chronic aspergillosis. There is evidence of radiographically dense material anterior and lateral to the heart and in the lower abdomen on either side of the gastrointestinal mass. (Courtesy The Raptor Center, University of Minnesota.)

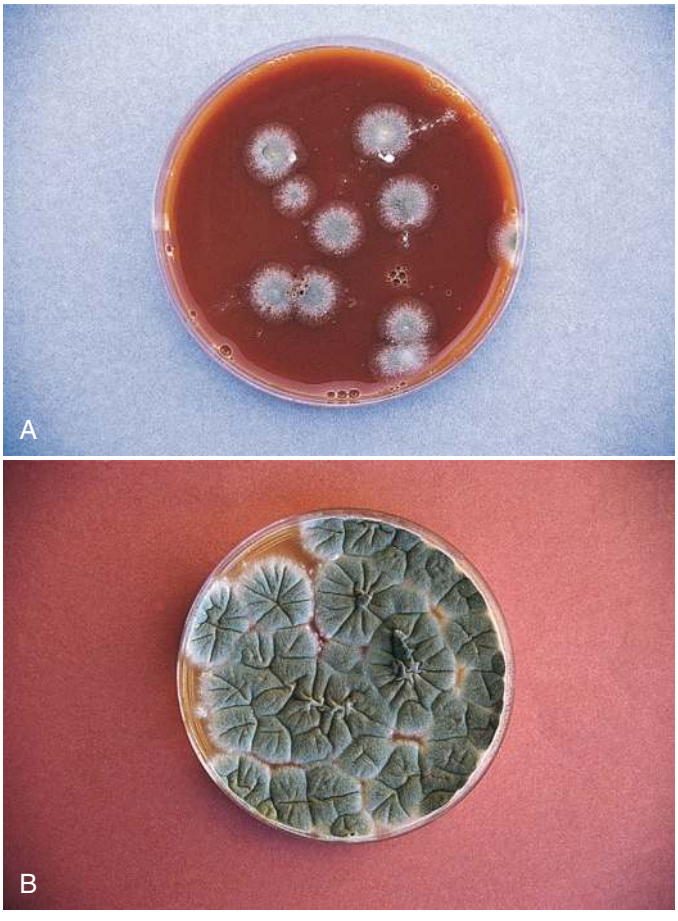


FIGURE 14-69 Cultural appearance of *Aspergillus fumigatus* on **(A)** blood agar and **(B)** Sabouraud dextrose agar after 4 days of incubation at 37°C (98.6°F). The colonies are a blue-green color with a powdery surface after 3 to 5 days of incubation. (Courtesy C. Silvanose.)

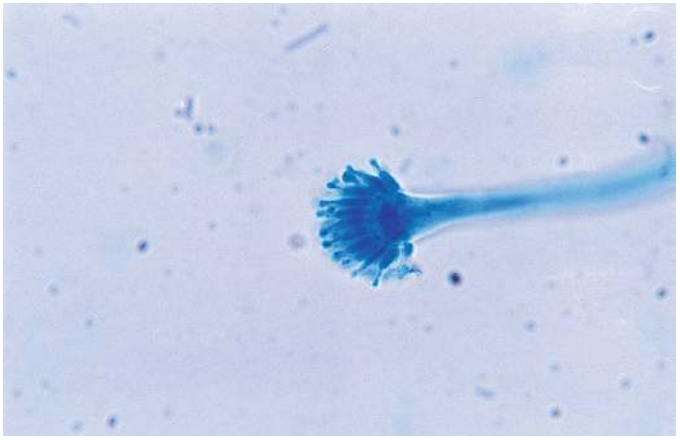


FIGURE 14-70 Microscopic appearance of *Aspergillus fumigatus* in lactophenol aniline blue stain showing a conidiophore, vesicle head, and one row of close-packed sterigmata crowned by conidiospores, ×400. (Courtesy C. Silvanose.)

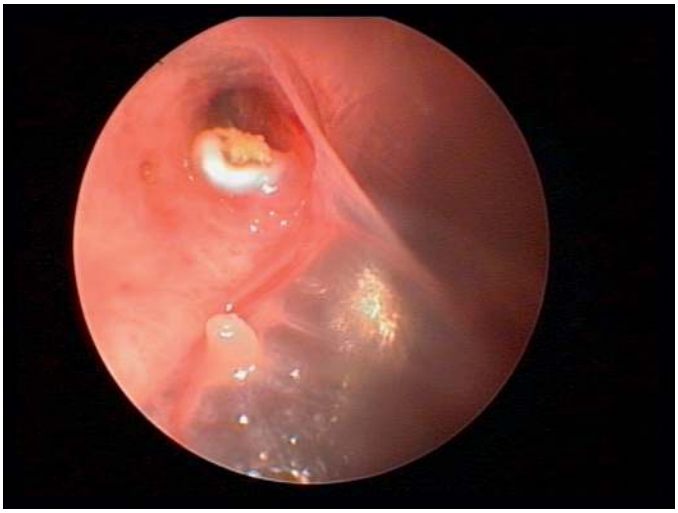


FIGURE 14-71 Endoscopic view of a plaque at the entrance of an ostium caused by *Aspergillus fumigatus*. In early stages of growth the surface of the plaque has a fuzzy white appearance but it will later turn blue-green. Spores borne on these plaques can cause development of secondary sites of infection within the respiratory system. Finding such growths in the coelomic cavity and/or air sac system confirms a diagnosis of clinical aspergillosis. (Courtesy Barbara Barca-Ruibal.)

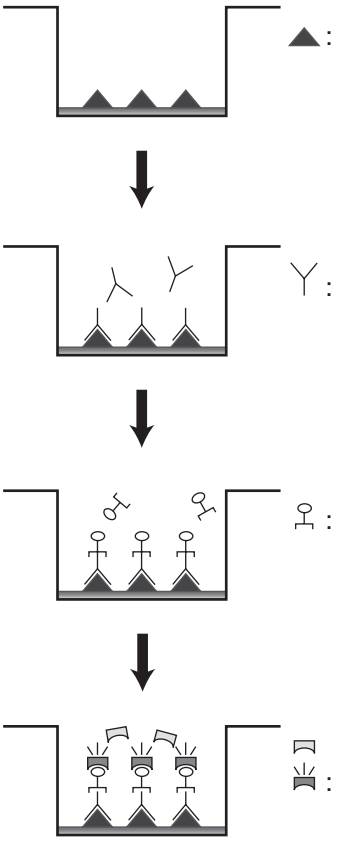


FIGURE 14-72 The indirect enzyme-linked immunosorbent assay is a useful aid for detecting antibodies to *Aspergillus fumigatus*. The enzyme-labeled second antibody (conjugate) detects antibodies bound to antigen, which is attached to a solid phase such as the well of a microtiter plate. A commercially available goat antiturkey conjugate adequately recognized immunoglobulins from *Buteo* species of raptors; however, specific conjugates must be made for use in falcons, accipiters, owls, or psittacines. (Courtesy The Raptor Center, University of Minnesota.)

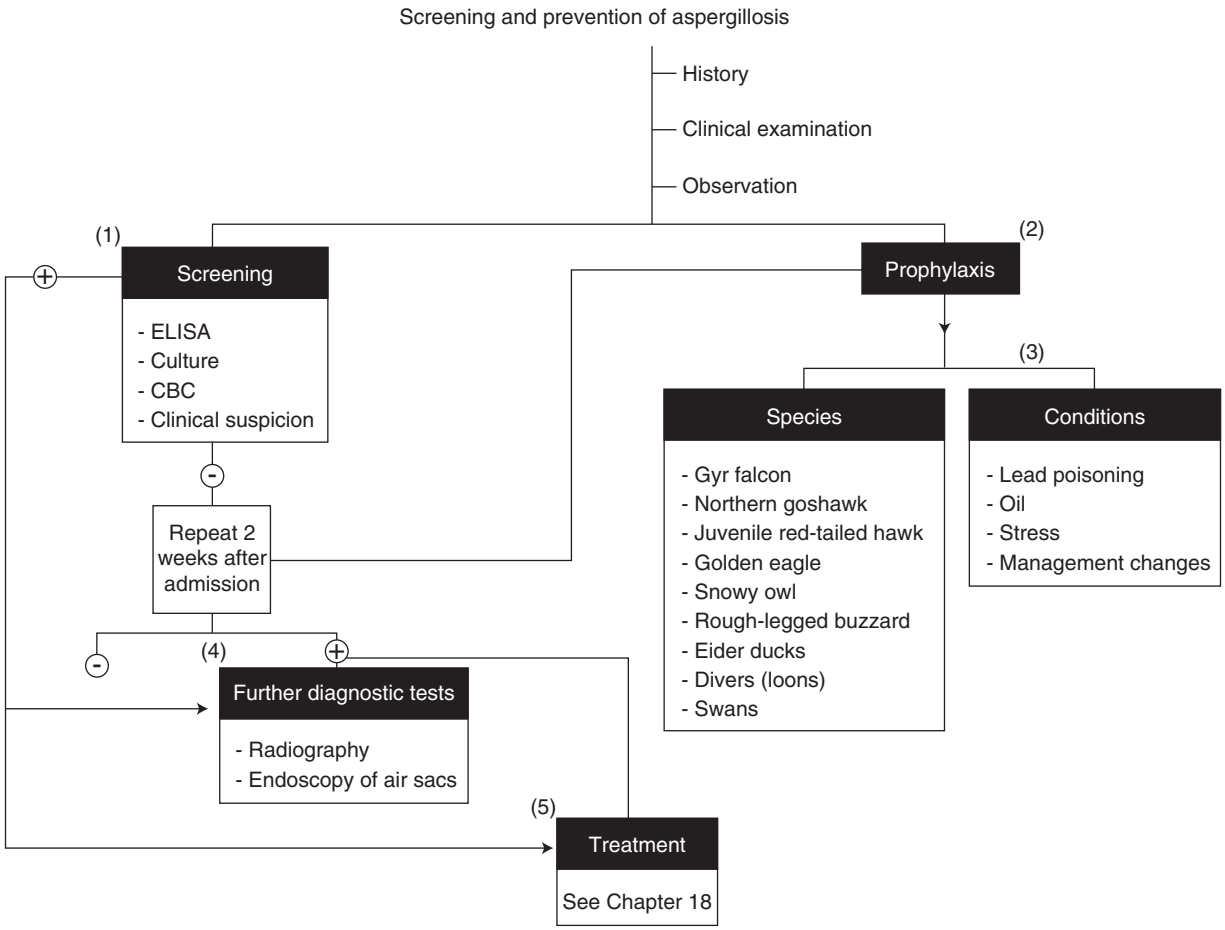


FIGURE 14-73 This flow chart is a depiction of the overall approach to patient assessment, prophylactic treatment, and management of clinical cases of aspergillosis. Prophylaxis and early detection of subclinical cases are essential for favorable outcomes. Once overt clinical signs of weight loss, dyspnea, and general debilitation are present, the prognosis is poor. CBC, Complete blood count; ELISA, enzyme-linked immunosorbent assay. (Modified with permission from Redig PT: Avian emergencies. In Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *Manual of raptors, pigeons and waterfowl*, Cheltenham, UK, 1996, British Small Animal Veterinary Association.)

FIGURE 14-74 An aspergillosis granuloma recovered from the syrinx of a golden eagle. Where the lesion is localized, surgical removal through a tracheostomy may yield immediate relief of dyspnea. Presence of the granuloma was detected endoscopically. (Courtesy The Raptor Center, University of Minnesota.)

The route of infection is inhalation, rendering the respiratory system the main target organ. Direct deposition of spores in the air on exposed vulnerable surfaces may also occur. Among parrots, and occasionally raptors, the upper respiratory system is often affected (sinuses, larynx, and syrinx). The most serious disease occurs with infection of the lower respiratory system (lungs and air sacs). Spores in the lower respiratory system can migrate widely throughout the coelomic cavity by the ramifying and interconnecting air sac system that reaches into every part of the body. Consequently, lesions may be found in the pericardium, on the kidneys, in the mesenteries, and among the vertebral bodies of the spinal column.

Although the respiratory system provides an ideal environment in which the thermophilic, oxyphilic *Aspergillus* fungus can grow, the organism is an opportunist and may colonize on superficial damaged epithelial and mucosal tissues. Mycotic keratitis and intraocular infections caused by *A. fumigatus* have been reported, and the author, on several occasions, found *A. fumigatus* colonizing skin wounds covered by a bandage.

Factors Implicated in Causality

Aspergillosis occurs typically as a result of inhalation of the ubiquitously available spores. Multiple infections in a single facility imply

common exposure rather than bird-to-bird spread. Infection can be overwhelming and acute, such as when a bird is exposed to a point source of heavy spore contamination, or it can occur as a result of low-level ambient exposure coupled with compromised immune function in the host. Some factors implicated as causal in the development of aspergillosis include:

- Recent capture
- Change of ownership
- Poor ventilation
- Neonatal and geriatric conditions
- Birds subjected to multiple doses of corticosteroids, especially dexamethasone
- Exposure to respiratory irritants such as cigarette smoke or ammonia
- Lead poisoning.

However, aspergillosis is also seen to occur, but with much lower frequency, in apparently well-adjusted trained birds for unapparent or undetermined reasons.

Respiratory System Forms of the Disease

Acute, chronic, and localized forms of the disease are recognized. An acute form occurs when a bird is exposed to a large number of spores from a point source. Hundreds of miliary foci of inflammation develop, mostly in the lung. This form is known as "brooder pneumonia" when it affects neonatal poultry, and it arises as a result of environmental contamination in the hatcher or brooder. In adult birds, it occurs from exposure to clouds of spores in poorly kept food or bedding, or other environmental sources. Moldy silage, leaf piles, bales of straw or shavings, and eucalyptus bark have been implicated as sources. In one situation, it was felt that the source of the spores was a mown field of alfalfa, adjacent to and downwind of the weathering yard in which a gyr falcon was housed, which had been rained on before it was finally raked and baled. The affected falcon became ill within a few days of the harvesting activity and died acutely with lungs studded with miliary granulomas. Other similar occurrences have been encountered when breeding chambers of falcons were cleaned without the birds being removed from the chamber.

Other forms of aspergillosis are more chronic and include focal lesions in the lungs and air sacs, pericardium, trachea, or syrinx, and occasionally the brain or anterior chamber of the eye. In all chronic forms, host immunosuppression is implicated in the pathogenesis. Localized forms involve granuloma formation in the syrinx or the sinuses.

Aspergillosis in gyr falcons is a serious problem for falconers. Historically in falconry, it is well established that the gyr falcon is highly impacted by this disease. With the large-scale production of gyr falcons and gyr hybrids through captive breeding and used in falconry, the number of encounters of birds with this disease have increased markedly in the last two decades. The disease most often becomes apparent at about the time the birds are becoming hard-penned, at approximately 10 weeks of age. Postmortem examination reveals extensive involvement of the air sacs and lungs, suggesting that the process has been ongoing for several weeks. The premise of immunosuppression or exposure to large numbers of spores from a point source does not appear to apply, as affected birds are typically raised in carefully monitored and managed facilities, provided with an abundance of nutritional food, and raised without overt evidence of stress. It is possible that the juvenile gyr falcon is constitutionally unable to defend itself against ambient exposure to *Aspergillus* spores. Other possibilities may include immunosuppression by exposure to high ambient temperatures occurring in temperate zones (where the majority of propagation occurs) in late July and early August when the greatest number of cases are seen, or exposure to some immunosuppressive, clinically unapparent viral agent. Another possibility is that the captive-raised gyr falcon is affected by unrecognized psychological stresses arising from a mismatch between the normal development it would experience in the wild in an Arctic environment and containment, relocation, or other artifacts of rearing in captivity. Research into these issues is necessary to reduce the incidence of this disease in young gyr falcons.

Diagnosis and Management

The management and diagnosis of aspergillosis is challenging because of the following:

- Variability and subtleness of the signs of the disease at onset
- Advanced state of the disease when clearly recognizable signs become apparent; immunologically suppressed birds may elicit a poor antibody response, rendering antibody-based diagnostic tests less useful
- The role played by immunosuppression in the pathogenesis: the host often puts up little defense and gives little help in the treatment, and in many instances the apparent causes of the immunosuppression appear to be very small and relatively innocuous events, such as a change of management of the bird

Clinical Appearance

The disease is typically seated in the respiratory system, although lesions may occur in other parts of the body. However, the early clinical signs of the chronic forms do not necessarily, and most often do not, result in expression of respiratory signs. Rather the early signs are subtle, nonspecific, and include:

- Change in behavior, especially a reduction in overall or expected levels of activity
- Change in voice
- Food flicking or anorexia
- Slight loss of stamina or willingness to chase quarry
- Apparent respiratory signs or weight loss—at this point the disease is extensively developed

Diagnosis

In General

The battery of tools to be used in establishing a diagnosis include:

- Clinical suspicion (signs, species, sex, time of year, and present and recent circumstances)
- Antibody detection with a high-level sensitivity test such as an enzyme-linked immunosorbent assay (ELISA)[a] or an antigen capture test[b]
- Tracheal culture and/or washes
- Air sac washes
- Hematology analysis characterized by heterophilic leukocytosis, toxic heterophils, and varying degrees of monocytosis; protein electrophoresis may exhibit a characteristic increase in β and γ globulins strongly associated with aspergillosis or other granulomatous disease
- Endoscopy is the single most useful tool in establishing a diagnosis of aspergillosis in clinically suspected cases
- Radiology is generally of limited value as a diagnostic tool during stages of development at which treatment is a reasonable possibility, but may yield useful information

Organisms recovered from swabs or washes are cultured on Sabouraud dextrose agar. Identity can be readily confirmed from wet mounts prepared with lactophenol aniline blue stain or other methylene blue–based stain.

Specifically

Clinical suspicion coupled with positive tracheal culture (taken from deep within the trachea with a nasopharyngeal swab[c]) and an elevated

white cell count (25,000 to 100,000 cells/mm^3+) is taken as circumstantial evidence of occurrence of aspergillosis and the basis on which to commence treatment. Endoscopy is invaluable because it allows examination of the trachea and the air sacs for lesions referable to aspergillosis. Whereas well-developed granulomas or lesions sprouting fungal hyphae leave little doubt, in many early cases only vascularized air sacs are seen. These should be regarded as strong evidence of an inflammatory response by the air sacs.

High-sensitivity serologic tests specifically for aspergillosis have limited global availability; however, where applied they have been very useful for assessing a patient's status regarding aspergillosis. The indirect ELISA measures the presence of antibody. A positive result indicates active infection, long-term exposure, or an elevated antibody level resulting from a previous infection. A negative result indicates no antibodies, either as a result of lack of disease or the inability to produce the antibodies. At The Raptor Center, University of Minnesota, three categories of response are used:

- *Below cut-off*, which implies no detectable antibodies, and is a category in which false-negatives have been encountered only in circumstances where the patient, in addition to having aspergillosis, had another debilitating condition such as tuberculosis or lead poisoning.
- *Midrange* (gray zone), which implies exposure and low-level antibody production caused by (1) ongoing exposure with no clinical disease, (2) low-level or early stage disease development, or (3) poor immune response to severe disease.
- *High-range*, which is associated with vigorous immune response and may bode well for recovery. An affected bird often yields a midrange response early in the disease that increases into the high range during the second to fourth week of treatment. Failure to show increasing optical density readings during treatment may imply lack of antibody response and be indicative of a guarded prognosis.

ELISA is a good screening test and, when used in conjunction with other parameters, aids the clinician in establishing a diagnosis. Its availability is presently limited and it is species or group specific, depending on immunoprotein recognition by antibodies produced in goats or rabbits. As such, a specific custom-made conjugate-labeled antibody is needed for psittacines, falcons, and accipiters, whereas immunoglobulins of other species of diurnal raptor cross-react well with a commercially available goat or rabbit antiturkey conjugate. A conjugate antibody has not been made for owls, and the other conjugates do not have sufficient cross-reactivity with owl serum to render them effective in antibody detection with this test.

The need for species-specific conjugates may be circumvented by the development of antigen capture tests, which detect molecular elements of the *Aspergillus* organism that may be present in the bloodstream. While the extent to which antigen may be present from nondisease-causing exposure compared with active infection is not yet clear, these tests nevertheless represent another potentially valuable means of assessing a patient's status regarding this disease. There is one polymerase chain reaction–based test available in the United States, which is useful when used on aspirates/washes taken from the trachea or air sacs or on serum (Dalhausen R, personal communication).

Treatment

The treatment options for aspergillosis are limited. Drugs used have included 5-fluorocytosine (5FC),[d] itraconazole,[e] fluconazole,[f] clotrimazole,[g] enilconazole,[h] voriconazole,[i] terbinafine HCl[j], and amphotericin B.[k] The last two are fungicidal. Amphotericin B is the gold standard against which other antifungal agents are compared in vitro. Amphotericin B, along with 5FC, itraconazole, and clotrimazole, and particularly the last two in combination, is efficacious in treating known cases of aspergillosis; fluconazole appears to be ineffective. Enilconazole and ketoconazole have also been used with a modicum of success by individual clinicians. Itraconazole is the most widely used antifungal agent at present, but work done in 2004 with voriconazole indicates that this recently available compound may have greater clinical utility than other forms of treatment (Di Somma *et al.*, 2004). This drug was found to be effective in treating clinical cases of aspergillosis in falcons when dosed at 12.5 mg/kg (twice a day for 3 to 5 days in heavily infected birds and then once a day for 3 to 6 weeks depending on severity), although further work has suggested that multiple doses may need to be given to maintain inhibitory concentrations throughout the day (Scope *et al.*, 2005). Much more research is needed, first to establish a means of assessing the antifungal sensitivity of recovered strains of *Aspergillus* spp. at the beginning and during courses of clinical treatment, and second to assess the efficacy of different drugs and treatment protocols in a laboratory-controlled infection model.

A representative treatment regimen for aspergillosis consists of amphotericin B administered intratracheally (1.5 mg/kg in a 1 cc volume of sterile water, twice a day) and intravenously (1 mg/kg given by bolus injections every 8 to 12 hours) for the first 3 to 4 days. Itraconazole is administered at 5 to 15 mg/kg twice a day, orally, for the first 5 days, then once daily thereafter for the duration of treatment, usually 3 to 4 months. Amphotericin B is replaced by nebulized clotrimazole (5% to 10% solution in polyethylene glycol with 5% dimethylsulfoxide [DMSO], obtained from a compounding pharmacy). Nebulization schedules vary widely; however, we typically provide two 1-hour sessions per day separated by a 12-hour interval (Redig, 1996). Most recently, voriconazole has come to the forefront as a clearly improved agent for treating aspergillosis (Di Somma *et al.*, 2004, Scope *et al.*, 2005). Dosed at 12.5 mg/kg by mouth twice a day for 4 days, followed by once daily, the drug is best absorbed with little or no food in the gastrointestinal tract. A liquid form is also available that can be concurrently nebulized. Because of the varying sensitivities of various strains of *A. fumigatus* to these agents, and uncertainties regarding pharmacokinetics in various species, combination therapies are recommended. Nebulization therapy should be undertaken with the realization that it will deliver material only to those areas of the respiratory system where there is airflow, and that its utility therefore may be limited.

Aggressive treatment of severe aspergillosis may be undertaken by a method of endoscopic laser ablation of granulomas and direct installation of antifungal agents into accessible granulomas. Methods for more expansive access to the thoracic cavity and respiratory system for such ablative procedures have been described (Hernandez-Divers, 2002).

From a prognostic and treatment perspective, aspergillosis can be categorized into four levels:

- *Class I:* A patient in this category may express vague signs of illness such as reduction in appetite, slight weight loss, decreased activity such as not flapping its wings as vigorously as usual when bathing, or loss of stamina. In the absence of endoscopic confirmation, or where endoscopic results are inconclusive, if two other indicators are positive (e.g., elevated white cell count and vague signs of illness, especially in a high incidence species), initiate treatment with a 2- to 3-week course of itraconazole (5 to 10 mg/kg twice daily for 5 days, followed by once daily at the same dose oral) or voriconazole (12.5 mg/kg once or twice daily, oral). Some individual birds may exhibit anorexia and vomiting after a few days at the higher dosages of itraconazole. Monitor white cell picture, tracheal culture, and patient condition. Typically, the prognosis is excellent.

- *Class II:* A patient in this category has discrete clinical signs referable to aspergillosis (respiratory difficulty), positive tracheal culture, and endoscopic confirmation of lesions or at least vascularized air sacs. Full-scale treatment should be undertaken as described in Class I. Prognosis is fair to good.
- *Class III:* In this category the patient presents with severe clinical signs (dyspnea, anorexia, vomiting, and notable weight loss), radiographically visible lesions, endoscopically visible lesions and, often, a low antibody response. In this case aggressive treatment is aided by surgical debulking of masses in air sacs with exploratory surgery. Prognosis is poor in goshawks and red-tailed hawks and guarded in large falcons.
- *Class IV:* This category is syringeal aspergillosis as detected by endoscopic evaluation of the trachea. In some, but not all, cases the lesion can be removed through an incision in the lower trachea, proximal to the syrinx. An air sac cannula is very helpful for administering anesthesia during such surgery. Following removal, the patient is treated with intratracheal amphotericin B for 5 days and itraconazole or voriconazole by mouth for about 3 weeks. The patient parameters of white cell count and distribution and its antibody status (ELISA) are monitored. If there is no further involvement of the respiratory system, the prognosis is excellent.

Depending on the site of the lesions and the severity of the disease, there are many aspects to treatment. The total treatment program typically extends over 3 months, but if recovery is likely, an initial favorable response to treatment is seen within 7 to 10 days.

Prevention and Prophylaxis

Clearly aspergillosis is to be prevented. Protection from exposure to aerosols that may contain spores is paramount. Prophylactic treatment with 5FC or itraconazole/voriconazole is recommended for newly captured or newly admitted birds of species that have an established track record of susceptibility, especially gyr falcons. The recommended protocol is to administer an antifungal (itraconazole or voriconazole) for 3 weeks. Gyr falcons will tolerate itraconazole only up to a dose of 8 mg/kg. The course of treatment may be extended if clinical indications warrant. This approach also should extend to individuals of highly susceptible species that are undergoing a change of management (e.g., transfer to new owner or new enclosure), regardless of age or other circumstance. Treatment for 1 week before the move and 2 weeks after is recommended. Domestically reared gyr falcons and gyr hybrids should be provided with this prophylactic regimen from a period beginning at 45 days of age through 75 to 90 days of age. If extreme heat conditions prevail during the months of August and September in any given locale, young gyr falcons should be provided with extended prophylactic treatment during this time. The tendency for these prophylactic regimens to induce drug-resistant strains of *Aspergillus* is unknown.

Aspergillosis is the most challenging medical problem affecting avian species. There are many tools available for diagnosis and treatment but no formulaic protocols that will guarantee success. Each case must be evaluated on its own merits and it is up to the clinician to select the proper tools and apply them effectively to achieve success. Prevention and prophylaxis are vital.

PRODUCT REFERENCE LIST

[a]Aspergillus ELISA testing. The Raptor Center, 1920 Fitch Avenue, St. Paul, MN 55108.

[b]Antigen capture testing for *Aspergillus* sp. University of Miami, School of Medicine, 1600 NW 10th Ave #1140, Miami, FL 33136.

[c]Nasopharyngeal calcium alginate tipped applicators. Hardwood Products, Guilford, ME 04443-0149.

[d]Ancobon. Valeant Pharmaceuticals International, Inc., U.S. Headquarters, 400 Somerset Corporate Blvd., Bridgewater, NJ 08807, USA.

[e]Sporanox. Janssen Pharmaceutica, Piscataway, NJ 08854.

[f]Diflucan. Pfizer Inc., New York, NY 10017.

[g]Lotrimin. Schering-Plough Healthcare Products Inc., Memphis, TN 38112 (1% clotrimazole product—obtain 5% clotrimazole in propylene glycol with 5% DMSO from compounding pharmacy).

[h]Imaverol. Janssen Pharmaceutica, Piscataway, NJ 08854.

[i]Vfend. Pfizer Island Pharmaceuticals, Ringaskiddy, Ireland.

[j]Lamasil. Novartis International AG, Basel, Switzerland.

[k]Fungizone. ER Squibb & Sons, Princeton, NJ 08540.

REFERENCES

Beernaert LA, Pasmans F, Van Waeyenberghe L, et al: *Aspergillus* infections in birds: a review, *Avian Pathol* 39(5):325–331, 2010.

Di Somma A, Bailey TA, Silvanose C, et al: The use of voriconazole for the treatment of aspergillosis in falcons. *Proceedings of SIVEA*, Rome, Italy, 2004.

Hernandez-Divers S: Endosurgical debridement and diode laser ablation of lung and air sac granulomas in psittacine birds, *J Avian Med Surg* 16:138–145, 2002.

Redig PT: Avian emergencies. In Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *Manual of raptors, pigeons and waterfowl*, Cheltenham, UK, 1996, British Small Animal Veterinary Association.

Scope A, Burhenne J, Haefeli W, et al: Pharmacokinetics and pharmacodynamics of the new antifungal agent voriconazole in birds. *Proceedings of the Association of Avian Veterinarians*, Arles, pp 217–221, 2005.

FURTHER READING

Abrams GA, Paul-Murphy J, Ramer JC, et al: Aspergillosis blepharitis and dermatitis in a peregrine falcon-gyrfalcon hybrid (*Falco peregrinus* × *Falco rusticolus*), *J Avian Med Surg* 15:114–120, 2001.

Aguilar RF, Redig PT: Diagnosis and treatment of avian aspergillosis. In Bonagura JD, editor: *Current veterinary therapy XI*, Philadelphia, PA, 1995, WB Saunders.

Arca-Ruibal B, Wernery U, Zachariah R, et al: Assessment of a commercial sandwich ELISA in the diagnosis of aspergillosis in falcons, *Vet Rec* 158:442–444, 2006.

Bauck L: Mycoses. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Beernaert LA, Baert K, Marin P, et al: Designing voriconazole treatment for racing pigeons: balancing between hepatic enzymes auto induction and toxicity, *Med Mycol* 47:276–285, 2009.

Beernaert LA, Pasmans F, Baert K, et al: Designing a treatment protocol with voriconazole to eliminate *Aspergillus fumigatus* from experimentally inoculated pigeons, *Vet Microbiol* 139:393–397, 2009.

Beernaert LA, Pasmans F, Van Waeyenberghe L, et al: Avian *Aspergillus fumigatus* strain resistant to both itraconazole and voriconazole, *Antimicrob Agents Chemother* 53:2199–2201, 2009.

Bennett RA: Approach to the thoracic cavity of birds, *Exot DVM* 1(3):55–58, 1999.

Cray C, Reavill D, Romagnano A, et al: Galactomannan assay and plasma protein electrophoresis findings in psittacine birds with aspergillosis, *J Avian Med Surg* 23:125–135, 2009.

Cray C, Watson T, Rodriguez M, et al: Application of galactomannan analysis and protein electrophoresis in the diagnosis of aspergillosis in avian species, *J Zoo Wildl Med* 40:64–70, 2009.

Espinel-Ingroff A: *In vitro* activity of the new triazole voriconazole (UK-109,496) against opportunistic filamentous dimorphic fungi and common and emerging yeast pathogens, *J Clin Microbiol* 36:198–202, 1998.

Jones M, Orosz SE: The diagnosis of aspergillosis in birds, *Semin Avian Exot Pet Med* 9:52–58, 2000.
Joseph V: Aspergillosis in raptors, *Semin Avian Exot Pet Med* 9:66–74, 2000.
Langhofer B: Emerging antifungals and the use of voriconazole with amphotericin to treat *Aspergillus*. *Proceedings of the Association of Avian Veterinarians*, New Orleans, pp 21–24, 2004.
Orosz SE: Overview of aspergillosis: pathogenesis and treatment options, *Semin Avian Exot Pet Med* 9(2):59–65, 2000.
Orosz SE, Frazier DL: Antifungal agents: a review of their pharmacology and therapeutic indications, *J Avian Med Surg* 9:8–18, 1997.
Pacetti SA: Caspofungin acetate for treatment of invasive fungal infections, *Ann Pharmacother* 37:90–98, 2003.
Redig PT, Post GS, Concannon TM: Development of an ELISA test for the diagnosis of aspergillosis in avian species. *Proceedings of the Association of Avian Veterinarians*, Miami, pp 165–178, 1986.
Silvanose CD, Bailey TA, Di Somma A: Susceptibility of fungi isolated from the respiratory tract of falcons to amphotericin B, itraconazole, and voriconazole, *Vet Rec* 159:282–284, 2006.
Yamakami YA, Hashimoto IT, Nasu M: PCR detection of DNA specific for *Aspergillus* species in serum of patients with invasive aspergillosis, *J Clin Microbiol* 34:2464–2468, 1996.

CANDIDIASIS

Christudas Silvanose

Candidiasis (Figs. 14-75 to 14-77) is one of the most common fungal diseases in birds. It is usually confined to the upper alimentary tract but can also invade the nasal cavity and sinuses. This disease is caused by fungi of the genus *Candida*. *Candida albicans* is the most common pathogenic species isolated from clinical specimens. However, *C. krusei* and *C. tropicalis* also have been reported from clinical cases. Candidiasis has been reported in several avian species including falcons, pigeons, parrots, pheasants, chickens, and turkeys. This disease is usually associated with malnutrition, inhibition of normal bacterial flora caused by prolonged use of broad-spectrum antibiotics, and poor husbandry, but mainly by repeated over-retention of food in the crop for a period of time (Samour and Naldo, 2002).

Clinical Signs and Lesions

Clinical signs are nonspecific and varied. Infected fowl chicks show inadequate and retarded growth, are apathetic, and their feathers are ruffled. In young turkeys, the symptoms include listlessness and diminished appetite, but especially noticeable is the way in which the head drops backward between the shoulder blades and the sunken breast. The eyes sink deep into their sockets and the head of the bird has a disheveled appearance. *Candida* spp. infection in birds is usually manifested by the presence of lesions in the oropharynx and crop, characterized by white circular ulcers with raised surface scabs causing thickening of the mucosa. In most cases, pseudomembrane and inflammatory changes, such as exfoliation of epithelial cells, are common. In certain chronic cases, beak rot, tongue rot, and enteritis are also observed. If the infection invades the intestinal mucosa, a malabsorption syndrome may develop. Affected falcons commonly showed amorphous diphtheritic membranes, from white-gray to gray-green in color, affecting the crop. On endoscopic examination the mucosal membrane of affected areas has a typical "Turkish towel" appearance (Fig. 14-78). On external examination, through palpation, the affected areas appear thicker to the touch (Samour and Naldo, 2002). Clinical signs include reduced appetite, anorexia, regurgitation, shredding and flicking of the food, and subsequent progressive weight loss.

Laboratory Diagnosis

Direct phase-contrast microscopic examination of sample suspension in normal saline is a quick and easy method of detecting the presence

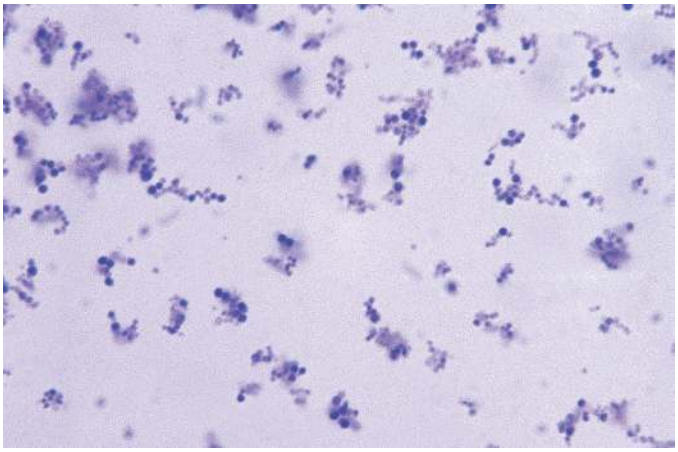


FIGURE 14-76 Microscopic appearance of *Candida albicans* in methylene blue stain, illustrating budding yeast cells, ×200.

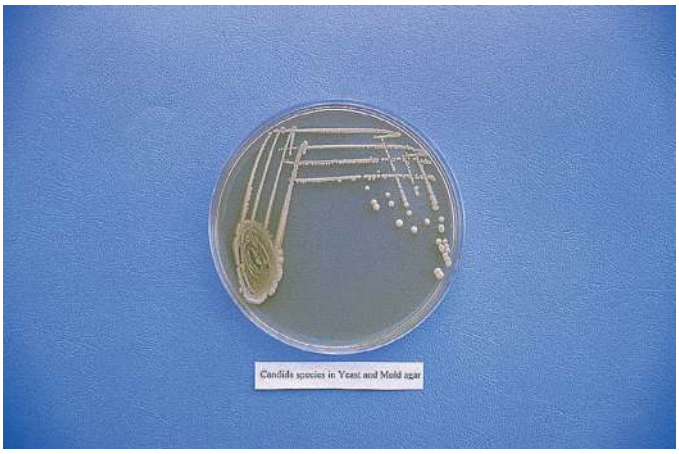


FIGURE 14-75 *Candida albicans* on yeast and mold agar. The colonies are white in color and 2 to 3 mm in size after 48 hours of incubation at 37°C (98.6°F).

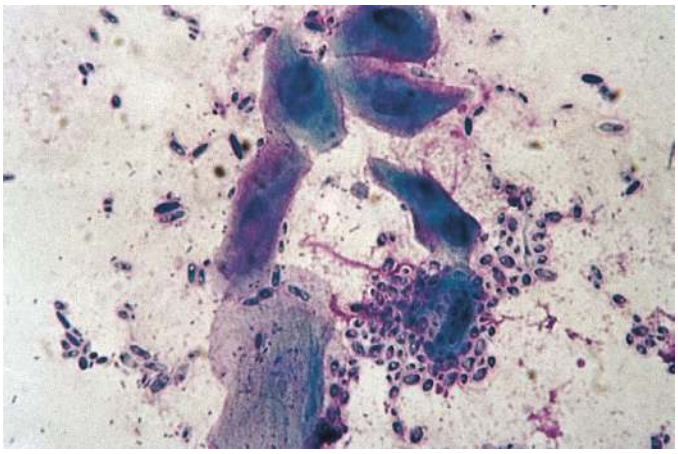


FIGURE 14-77 Oropharyngeal smear from a houbara bustard (*Chlamydotis undulata*) with candidiasis. The smear shows exfoliated epithelial cells and oval-elongated *Candida albicans*, ×1000.

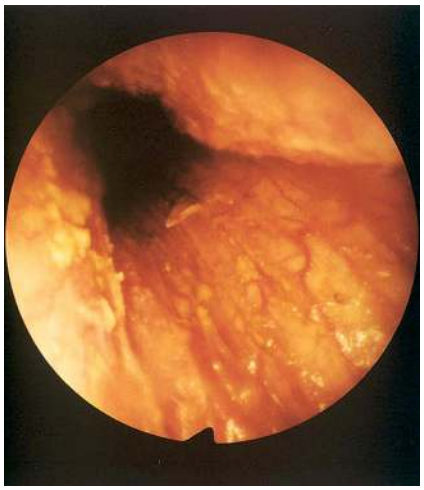


FIGURE 14-78 Early *Candida albicans* infection in a saker falcon (*Falco cherrug*) presented with a history of regurgitation and reduced appetite. On endoscopy examination the mucosal membrane of affected areas had a typical "Turkish towel" appearance. (Courtesy Dr. J. Samour.)

of *Candida* spp. The sample can also be examined by direct light microscopy with the help of lactophenol aniline blue stain. *C. albicans* is an oval, budding yeast that produces pseudohyphae, both in tissue and exudates and in broth culture. Eosin and methylene blue stain (Neat stain and Rapi-Diff) is also used for the detection of exfoliated epithelial cells and *Candida* spp. in the smear. In Gram-stained smears, *Candida* spp. appear as a gram-positive, oval budding yeast measuring 2 to 3× 4 to 6 µm, and elongated budding cells resembling hyphae called pseudohyphae. Culture or serologic studies are necessary for the species identification.

Culture studies are the most widely used diagnostic method for detecting *Candida* spp. These fungi grow well in yeast mold agar (Oxoid, UK) after 48 to 72 hours of incubation at 37°C (98.6°F). Media used for the primary isolation and identification of *Candida* spp. include *Candida* BCG agar, corn meal peptone yeast agar, and Sabouraud agar; corn meal agar, LIU Newton agar, rice infusion-oxgall-Tween 80 agar, and rice-Tween agar are used to detect the production of chlamydospores of *C. albicans.* BiGGY agar (Nickerson Medium) is used for the selective isolation, differentiation and presumptive identification of *C. albicans* and *C. tropicalis.*

The identification of *Candida* spp. is based on morphologic appearance by microscopic examination, cultural characteristics on primary and selective media, and biochemical reactions, which include assimilation and fermentation of carbohydrates. Vitek 2 YST ID card, MUAG Candi test, and Flow Uni-Yeast-Tek wheel are commercially available kits used for the identification of yeast from clinical specimens by assimilation and fermentation reactions. Germ tube test is used for the detection of pseudohyphae production of *C. albicans.* Serologic testing by agglutination reaction with specific antisera is also used for the diagnosis of *Candida* spp.

Treatment

The treatment for candidiasis in the upper digestive tract of birds includes the use of nystatin 200,000 to 300 000 U/kg by mouth every 12 hours for 7 to 10 days (Bauck, 1994; Boydell and Forbes, 1996; Oglesbee, 1997; Redig and Ackermann, 2000), ketoconazole 10 to 30 mg/kg by mouth every 12 hours for 7 days (Bauck, 1994; Boydell and Forbes, 1996; Oglesbee, 1997), itraconazole 5 to 10 mg/kg by mouth every 12 hours for 7 to 21 days (Bauck, 1994; Boydell and Forbes, 1996), and fluconazole 2 to 5 mg/kg by mouth every 24 hours for 7 days (Bauck, 1994; Oglesbee, 1997). The latter is postulated as the most effective antifungal agent for the treatment of tissue-based yeast infections in birds (Flammer, 1993). Nystatin suspension, 2 to 5 mL (100,000 IU/mL)/kg (weight of the bird) has also been used, applied directly to the mucosal membranes of the mouth of raptors (P.T. Redig, personal communication). The use of a miconazole gel preparation (Daktarin oral gel, Janssen-Cilag Ltd., UK) was successfully used in the treatment of upper digestive tract candidiasis infections in falcons (Samour and Naldo, 2002). This pharmacologic miconazole presentation may prove useful for similar infections in other avian species.

REFERENCES

Bauck L: Mycoses. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Boydell IP, Forbes NA: Diseases of the head (including the eyes). In Beynon PH, Forbes NA, Harcourt-Brown N, editors: *Manual of raptors, pigeons and waterfowl*, Quedgeley, Gloucester, UK, 1996, British Small Animal Veterinary Association.

Flammer K: An overview of antifungal therapy in birds. *Proceedings of the Association of Avian Veterinarians*, Nashville, TN, pp 1–4, 1993.

Oglesbee BL: Mycotic diseases. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian med surg*, Philadelphia, 1997, WB Saunders.

Redig PT, Ackermann J: Raptors. In Tully TN, Lawton MPC, Dorrestein GM, editors: *Avian medicine*, Oxford, UK, 2000, Butterworth-Heinemann.

Samour JH, Naldo JL: Diagnosis and therapeutic management of candidiasis in falcons in Saudi Arabia, *J Avian Med Surg* 16:129–132, 2002.

FURTHER READING

Biberstein EL: Candida. In Biberstein EL, Zee YC, editors: *Veterinary microbiology*, Oxford, UK, 1990, Blackwell Science.

McCluggage DM: Zoonotic disorders. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, MD, 1996, Williams & Wilkins.

Quinn PJ, Carter ME, Markey B, et al: *The pathogenic yeasts. Clinical veterinary microbiology*, London, 1994, Wolfe Publishing.

Richard JL, Beneke ESA: Mycoses and mycotoxicoses. In Purchase HG, Arp LH, Domermuth CH, Pearson JE, editors: *A laboratory manual for the isolation and identification of avian pathogens*, Athens, GA, 1989, American Association of Avian Pathologists.

Van Cutsem J, Rochette F: *Poultry: candidiasis: mycoses in domestic animals*, Beerse, Belgium, 1991, Janssen Research Foundation.

MACRORHABDUS ORNITHOGASTER

David Phalen

History and Description of *Macrorhabdus Ornithogaster*

Macrorhabdus ornithogaster is an anamorphic ascomycetes yeast that has only been found to grow at the junction of the proventriculus and ventriculus in birds (Tomaszewski *et al.*, 2003). It was first recognized in the early 1980s in the United States in budgerigars and was thought to be a yeast (Hargreaves, 1981). Concurrent investigations in the Netherlands described it in canaries and incorrectly concluded it was a bacterium, giving it the name Megabacterium, which continues to be used improperly (van Herck *et al.*, 1984). A subsequent study claimed

to be able to isolate the organism from budgerigar stomachs using traditional bacterial isolation methods; however, they did not characterize their isolate sufficiently and subsequently it was shown that the isolate that was described in this study was a bacterium and not *M. ornithogaster* (Phalen, 2013).

The true nature of *M. ornithogaster* was only recently conclusively demonstrated. Studies in Australia demonstrated that it was not sensitive to antibiotics, but it was sensitive to amphotericin, suggesting that it was in fact a fungus (Filippich and Perry, 1993). It was shown to stain for chitin, a protein that is only produced by eukaryote organisms, proving that it was not a bacterium. Investigators were then able to purify the organism and sequence portions of the DNA that code for ribosomal RNA. Comparing this sequence to other known yeast it was then shown that *M. ornithogaster* was not only a novel species of yeast, but in fact was the only known representative of an entirely new genus of yeasts (Tomaszewski *et al.*, 2003).

M. ornithogaster can infect many species of birds (Phalen, 2013). There is convincing evidence that it can cause disease in its host, but it is also clear that many birds live with this organism without obvious signs. The only effective treatments for *M. ornithogaster* are a few antifungal drugs, and these drugs do not always lend themselves to large-scale flock treatment. Because *M. ornithogaster* was thought to be a bacterium (Megabacteria) for more than 20 years many assumptions about this organism's biology have subsequently proven to be untrue. Continued referencing of some of these flawed studies and anecdotal reports often creates confusion for veterinarians and bird owners alike (Phalen, 2013).

Host Range

The reported host range of *M. ornithogaster* includes a wide range of psittacine birds, passerine birds, poultry, and other species. It has a worldwide distribution and is found in both wild and captive birds (Martins *et al.*, 2006; Phalen, 2013).

The species of psittacine birds most commonly infected with *M. ornithogaster* are budgerigars (*Melopsittacus undulatus*), lovebirds (*Agapornis* sp.), and to lesser extent cockatiels (*Nymphicus hollandicus*). Infection has also been reported to be common in parrotlets (*Forpus* sp.). In wild Australian birds, the organism is commonly found in recently fledged galahs (*Eolophus roseicapilla*) and corellas (*Cacatua* sp.) with chronic diarrheal disease and weight loss. These birds have other intestinal parasites and at least some have concurrent infections with the psittacine beak and feather disease virus. The full host range of *M. ornithogaster* in psittacine birds is unknown and infection should be considered in any species of psittacine bird presenting with gastrointestinal signs (Phalen, 2013).

Passerine species infected with *M. ornithogaster* include pet canaries (*Serinus canaria*), zebra finches (*Taeniopygia guttata*), and Gouldian finches (*Erythrura gouldiae*). It has also been found in a range of wild European finches and the siskin (*Carduelis spinus*), and in feral European goldfinches (*C. carduelis*) and wild caught feral European goldfinches and green finches (*C. chloris*) captured for the pet trade in Australia (Phalen, 2013).

M. ornithogaster infections have now been reported in chickens (*Gallus gallus*) in Europe, North and South America, and Australia. Other gallinaceous birds reported to be infected with *M. ornithogaster* include the gray partridge (*Perdix perdix*), the Japanese quail (*Coturnix japonica*), domestic turkey (*Meleagris gallopavo*), chukar partridge (*Alectoris chukar*), and guinea fowl (genus and species not reported). Infection has also been reported in ducks, geese, and ibises, although no supporting evidence on how the diagnosis was made in ibises was provided. Recently *M. ornithogaster* has been reported in captive raised greater rheas (*Rhea americana*). Morphologically, these organisms are consistent with those that have been reported in other species; however, they still remain to be characterized by molecular techniques (Martins *et al.*, 2006; Phalen, 2013).

There are two reports of an organism resembling *M. ornithogaster* infecting the upper respiratory tract of a dog and a cat. These organisms were never described, and because *M. ornithogaster* is microaerophilic, its growth on respiratory epithelium does not seem plausible. Recent infection attempts in mice provide additional evidence that *M. ornithogaster* cannot grow in mammals (Hanafusa *et al.*, 2013).

Isolation attempts from stomach contents of greater rheas using growth conditions inconsistent with the metabolic requirements of *M. ornithogaster* have resulted in the isolation of a small motile organism, which the investigators suggest is *M. ornithogaster.* This uncharacterized organism has been shown to colonize the stomach of mice. Because this organism grows in conditions incompatible for *M. ornithogaster* growth, because it has morphologic characteristics never seen in *M. ornithogaster* either in vivo or in vitro, and because it has never been characterized genetically, it is the author's opinion that identifying this organism as *M. ornithogaster* is premature and is likely to be incorrect (Hanafusa *et al.*, 2007).

Clinical Manifestations

The signs of *M. ornithogaster* in birds include vomiting, regurgitation, diarrhea, and chronic weight loss (Fig. 14-79). It has been seen in young and adult birds. Disease in budgerigars is most common in middle-aged birds. An acute hemorrhagic disease has been reported in parrotlets. Weight loss, anorexia, melena, and anemia are commonly seen in cockatiels and occasionally in other species that have gastric ulceration secondary to *M. ornithogaster* infection. Canaries and other finches with *M. ornithogaster* infections are often found dead with no premonitory signs, but are generally emaciated, suggesting that they had been ill for at least a few days before death (Phalen, 2013).

Diagnosis in the Live Bird

Detection of *M. ornithogaster* infection in the live bird is most often done by microscopic examination of the feces. Feces made into a slurry with water or saline can be scanned for *M. ornithogaster* using 40×

FIGURE 14-79 Budgerigar (*Melopsittacus undulatus*) with *Macrorhabdus* infection and a history of vomiting. The pink stain on the feathers is from an antibiotic that had been vomited after the owner had attempted home treatment.

magnification. Alternately, fecal smears can be stained with a quick stain or Gram stain. A rapid way of concentrating *M. ornithogaster* and separating it from other solid matter in the feces is to homogenize a dropping with approximately 20 times its volume of physiologic saline in a small tube, let it sit for 10 seconds, and then examine a small drop of the suspension collected from the meniscus. Because *M. ornithogaster* take longer to settle than most other material in the feces, it is more easily seen in wet preparations after this treatment (Phalen, 2013). A PCR assay to detect *M. ornithogaster* in the feces is also available in North America (Veterinary Molecular Diagnostics, Milford, OH).

M. ornithogaster are long, slender, straight stiff rods with rounded ends when found in feces (Figs. 14-80 and 14-81). In some circumstances the long rods may bend slightly in a gentle curve. Y-shaped organisms can be seen (see Fig. 14-81), but these are extremely rare. Viewed directly in a wet mount, small oblong refractile structures (nuclei) found at regular intervals are readily seen. The nuclei stain with Giemsa. *M. ornithogaster* range in length from 20 to 80 µm and are consistently 2 to 3 µm across. They often stain poorly with quick and Gram stains and instead of staining uniformly they only pick up small droplets of the stain. When they do stain well, they are gram positive and stain dark blue with quick stains (Fig. 14-82). Unlike bacteria and other yeasts, the contents of the cell stain but not the cell wall. It is the author's impression that they do not stick well to glass slides unless the slide has been heat fixed. It is also the author's impression that heat fixing makes them more likely to stain uniformly.

Birds infected with *M. ornithogaster* may shed the organism in low numbers, in large numbers, or not at all. It has been the author's experience that the majority of birds exhibiting disease as the result of *M. ornithogaster* infection shed large numbers of organisms. However, this may not always be the case, and the absence of *M. ornithogaster* in the feces does not completely rule out infection (Phalen, 2013).

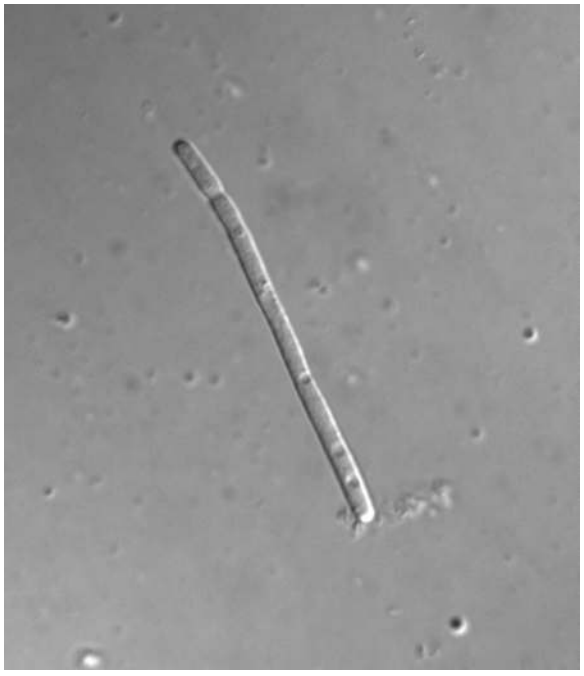


FIGURE 14-80 Unstained *Macrorhabdus ornithogaster*. Original magnification ×100. (From Phalen DN: Update: Diagnosis and management of *Macrorhabdus ornithogaster* (Formally Megabacteria), *Vet Clin North Am Exot Anim Pract* 17(2):203–210, 2014.)

There can be other things in the feces that resemble *M. ornithogaster* (Fig. 14-83). An unknown structure commonly seen by the author in the droppings of many birds is approximately the size of *Macrorhabdus*, but has a straight and not rounded terminal end that appears to be the result of the structure breaking off of something larger. *M. ornithogaster* always has rounded ends. Filamentous gram-positive bacteria can also approach the size of *M. ornithogaster.* These bacteria, however, are often segmented, are thinner than *M. ornithogaster*, and generally curve back and forth and thus are readily distinguished from *M. ornithogaster.*

Postmortem Diagnosis

M. ornithogaster infection is readily made at postmortem examination. A saline preparation of a scraping of junction (isthmus) of the proventriculus and the ventriculus demonstrates the organisms, which are generally abundant. *M. ornithogaster* is also readily demonstrated in

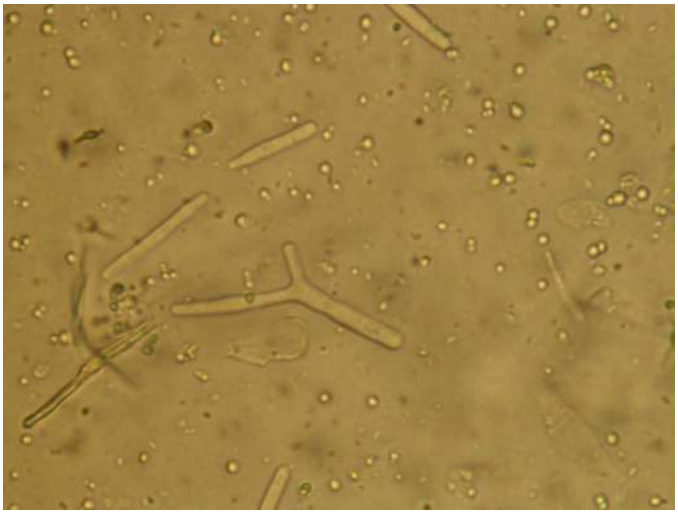


FIGURE 14-81 Unstained wet mount of *Macrorhabdus ornithogaster* showing typical rod-shaped organisms and an unusual Y-shaped organism. Original magnification ×100.

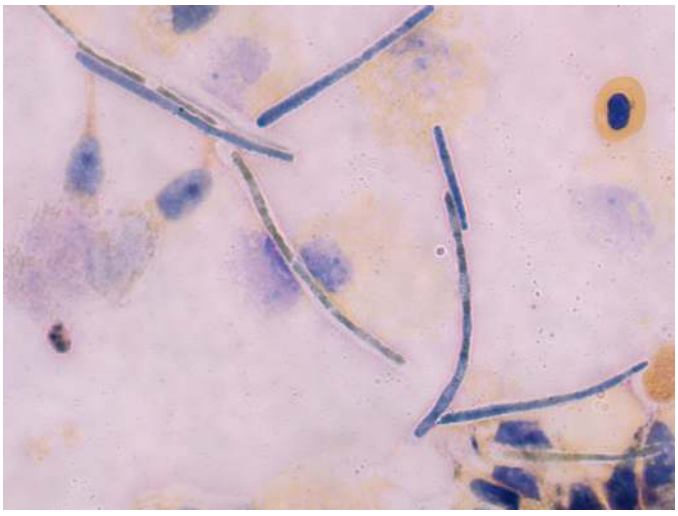


FIGURE 14-82 *Macrorhabdus ornithogaster* stained with Gram stain. Original magnification ×100. (From Phalen DN: Update: Diagnosis and management of *Macrorhabdus ornithogaster* (Formally Megabacteria), *Vet Clin North Am Exot Anim Pract* 17(2):203–210, 2014.)

FIGURE 14-83 Long filamentous bacteria in a wet mount from a budgerigar's feces that could be mistaken for *Macrorhabdus ornithogaster*.

H&E stained sections of the isthmus. They are eosinophilic and are found forming the characteristic log-jam patterns on the surface of and between the mucosal glands. Because they are a fungus, they stain with silver stains and the periodic acid-Schiff stain (Tomaszewski *et al.*, 2003).

Showing that the *M. ornithogaster* infection contributed to the cause of the bird's death, however, requires more proof than just finding the organism. Budgerigars and passerines with disease caused by *M. ornithogaster* will grossly have a thickened mucosa of the proventriculus and there will be increased mucus in the lumen. Some birds may have one or more bleeding ulcers of the proventriculus. In birds with clinical signs caused by *M. ornithogaster* infection, growth extends beyond the isthmus into the proventriculus and the koilin of ventriculus and may disrupt the structure of the koilin. A lymphoplasmacytic inflammation is common in birds with heavy *M. ornithogaster* growth, but is less likely in birds with minimal superficial colonization of the organism (Phalen, 2013).

Growth in Vitro

M. ornithogaster is readily grown *in vitro* given the correct substrate and conditions. It must be provided with a microaerophilic environment and grown in a medium with a pH between 3 and 4. Traditional cell culture media containing up to 20% fetal bovine serum and 1% to 5% glucose or sucrose have been shown to support its growth. Its optimal growth temperature is 42°C. Addition of antibiotics to the growth media is recommended to prevent the overgrowth of bacteria. It can be cultured from isthmus scrapings or from feces (Hanafusa *et al.*, 2007).

Treatment

There are few treatment trials that have been done in birds with *M. ornithogaster* infection (Phalen, 2013). In many of these trials, the measure of successful treatment was the cessation of *M. ornithogaster* shedding in the feces, as opposed to the less common trial where treated birds were killed and the stomach examined directly (Filippich and Perry, 1993; Kheirandish and Salehi, 2011; Phalen, 2013). While it is likely that the cessation of shedding may be the result of a cure, it is also possible that some of these treated birds may have remained infected at low levels.

Amphotericin B is used widely to treat *M. ornithogaster* and appears to be effective and safe when administered orally by gavage and in some circumstances in water. Various doses have been recommended. The author has used 100 mg/kg twice a day for 14 days with direct oral administration, but has been gradually reducing the amount and is now using 25 mg/kg twice a day for 14 days with apparent success. Success of treatment has been judged by the rapid cessation of *M. ornithogaster* shedding and resolution of signs. Amphotericin B can be purchased as a powder (Gallipot, St. Paul, MN) and compounded into a formula that can be given orally. The 2.5% water-soluble powder from Vetafarm (Wagga Wagga, New South Wales, Australia) that has been used extensively in the past is no longer available. There is one report of *M. ornithogaster* resistant to amphotericin B. How widespread the resistance may be is not known (Phalen, 2013).

The ability of nystatin to kill *M. ornithogaster* may vary from strain to strain. In vitro trials by Bradley *et al.* (2005), showed that *M. ornithogaster* was sensitive to nystatin at concentrations of 0.1 U/mL. In one clinical trial the authors also saw a cessation of *M. ornithogaster* shedding after treatment with nystatin. In a recent study a flock of budgerigars was treated with nystatin at 3,500,000 IU/L of drinking water for 2 days then 2,000,000 IU/L for 28 days (Kheirandish and Salehi, 2011). Some birds in this study were euthanized after the end of treatment and were found free of infection. Resistance to nystatin by some strains of *M. ornithogaster* is likely based on clinical trials done by others.

Research done by Bradley *et al.* (2005) showed that cultured *M. ornithogaster* in vitro are highly sensitive to sodium and potassium benzoate and sodium sorbate. Treatment attempts with trials of sodium benzoate in drinking water in live birds have been performed by the author and others. The author's experiences have not been uniformly successful, and in many cases, shedding and clinical signs have not resolved (Phalen, 2013). The reason for the failure of treatment in these instances is not known, but adequate consumption of the treated water may be to blame. In another trial where a flock of breeding budgerigars was treated, *M. ornithogaster* shedding stopped but some of the treated birds died. The cause of the deaths was not determined but could have been the result of sodium toxicity. Water consumption in the treated budgerigars was very high because they were feeding young and it was the middle of the summer and daytime temperatures were very high (Phalen, 2013). Trials of potassium benzoate have not been performed, but it may be safer than sodium benzoate because it is more difficult to get potassium toxicity from ingested potassium than it is to get sodium toxicity from ingested sodium. The use of any of these chemicals requires additional research before they can be recommended for routine use. There are many potential sources of sodium and potassium benzoate. The product used by the author is purchased as a 99% pure product (Sigma-Aldrich, St. Louis, MO).

Fluconazole has been used to effectively treat *M. ornithogaster* in experimentally infected chickens at a dosage of 100 mg/kg. In trials in budgerigars, this dosage was found to be toxic and a lower dosage was not effective. Gentian violet was found to prevent *M. ornithogaster* growth in vitro. Gentian violet at moderate concentrations, however, was found to be toxic to budgerigars (D. Phalen, unpublished information).

Conclusion

M. ornithogaster is found in many species of birds around the world. It can be a significant cause of both morbidity and mortality. Detecting the infection in the live bird requires the direct observation of the organism in the feces or its detection by PCR; however, these assays are not sufficiently sensitive that a negative result rules out infection. Diagnosis is readily made at postmortem by examination of scrapings of the isthmus and histopathology of the proventriculus

and ventriculus. The only consistently proven treatment for infected birds is direct oral administration of amphotericin B, although nystatin and sodium benzoate may also be effective under some circumstances.

ACKNOWLEDGMENT

This section is a modified version of Phalen DN: Update: diagnosis and management of *Macrorhabdus ornithogaster* (formally Megabacteria), *Vet Clin North Am Exot Anim Pract* 17 (2):203-210, 2013.

REFERENCES

Bradley A, Yasuka H, Phalen DN: *Macrorhabdus ornithogaster*: inhibitory drugs, oxygen toxicity and culturing from feces. USDA sponsored summer research program, Athens, GA, July 2005.

Filippich LJ, Perry RA: Drug trials against Megabacteria in budgerigars (*Melopsittacus undulatus*), *Aust Vet Pract* 23:184–189, 1993.

Hanafusa Y, Bradley A, Tomaszewski EK, et al: Growth and metabolic characterization of *Macrorhabdus ornithogaster*, *J Vet Diagn Invest* 19:256–265, 2007.

Hanafusa Y, Costa E, Phalen DN: Infection trials in mice suggest that *Macrorhabdus ornithogaster* is not capable of growth in mammals, *Med Mycol* 51:669–672, 2013.

Hargreaves RC: A fungus commonly found in the proventriculus of small pet birds. *30th Western Poultry Disease Conference and 15th Poultry Health Symposium*, Davis, CA, University of California at Davis, pp 75–76, 1981.

Kheirandish R, Salehi M: Megabacteriosis in budgerigars: diagnosis and treatment, *Comp Clin Pathol* 20:501–505, 2011.

Martins NRS, Horta AC, Siqueira AM, et al: *Macrorhabdus ornithogaster* in ostrich, rhea, canary, zebra finch, free range chicken, turkey, guinea-fowl, columbine pigeon, toucan, chukar partridge and experimental infection in chicken, Japanese quail and mice, *Arq Bras Med Vet Zootec* 58:291–298, 2006.

Phalen DN: Update: Diagnosis and management of *Macrorhabdus ornithogaster* (formally Megabacteria), *Vet Clin North Am Exot Anim Pract* 17(2):203–210, 2013.

Tomaszewski EK, Logan KE, Kurtzman CP, et al: Phylogenetic analysis indicates the "megabacterium" of birds is a novel anamorphic ascomycetous yeast, *Macrorhabdus ornithogaster*, gen. nov., sp. nov, *Int J Syst Evol Microbiol* 3:1201–1205, 2003.

van Herck H, Duijser T, Zwart P, et al: A bacterial proventriculitis of canaries, *Avian Pathol* 13:561–572, 1984.

FAVUS OR RINGWORM INFECTION

Jaime Samour

Favus or ringworm infection is a dermatophytosis seldom reported in birds (Figs. 14-82 to 14-87). The infection is caused by invasion of the keratinized layers of the skin and appendages by dermatophytes or ringworm fungi (Keymer, 1982). Pathogenic dermatophytes appear to have a worldwide distribution.

Host Range

The infection occurs occasionally in free-living passerines, and is reported mostly in the UK (Blackmore and Keymer, 1969). In captivity, the disease is relatively uncommon. It is mostly seen in Psittaciformes, Columbiformes, and Passeriformes. Other orders of birds in which the infection has been recorded include Struthioniformes, Anseriformes, Falconiformes, and Galliformes.

FIGURE 14-84 Microscopic appearance of *Trichophyton verrucosum* in lactophenol aniline blue stain. The main characteristics of this fungus are septate hyphae and half-empty chlamydospores, ×400. (*Courtesy* C. Silvanose.) This image is for the purpose of differentiation with *T. mentagrophytes*.

FIGURE 14-85 Microscopic appearance of *Trichophyton mentagrophytes* in lactophenol aniline blue stain. The hyphae of this fungus are spiral or tangled in shape, × 400. (Courtesy C. Silvanose.)

FIGURE 14-86 Favus-like lesions affecting the head and legs of a free-living European blackbird (*Turdus merula*). Dermatitis of this type appears to be multifactorial in origin and may be associated with fungal infections such as *Trichophyton* and *Cladosporium* spp., as well as mite infestations (Keymer IF: Mycoses. In Petrak ML, editor: *Diseases of cage and aviary birds*, ed 2, Philadelphia, 1982, Lea & Febiger) and bacteria (e.g., *Staphylococcus* spp. and *Escherichia coli*). Frequently it is difficult to isolate and identify the fungi involved.

FIGURE 14-87 A black Orpington (*Gallus gallus domesticus*) hen presented to a veterinarian for examination with a history of intense pruritus and widespread feather loss worsening in the past month. On histopathology of biopsies obtained from the face and neck, multifocal aggregates of pink-stained round conidia were observed. Based on the morphologic characteristics, the fungal bodies were determined to be most likely *Microsporum* sp. (Courtesy Robert Delaney.)

Cause

According to Perry (1987) there are more than 20 species of pathogenic dermatophytic fungi. The most commonly isolated microorganism is *Microsporum gallinae*; however, *M. gypseum* and *Trichophyton simii* and *T. megninii* have also been recorded (Baron and Doneley, 2014). Less frequently, other fungi (e.g., *Aspergillus*, *Candida*, *Cladosporium*, *Helminthosporium*, *Mucor*, *Malassezia* [also called *Pityrosporum*], and *Paecilomyces* spp.) may be involved.

Clinical Signs

Most commonly affected are the unfeathered areas of the skin of the head and, when present, also the comb and wattles. Less frequently, lesions affect other parts of the body such as the neck, the legs, and the leading edge of the wings. However, Baker (1996) recorded unilateral feather loss of the body caused by a *Trichophyton* sp. in a budgerigar (*Melopsittacus undulatus*). In most cases partial or complete alopecia is present and the skin becomes thickened and corrugated, grayish white or yellowish in color, and sometimes with the formation of encrustations and scabs. Crusty exudates may occur around the feather follicles. Sometimes the skin lesions are rough and porous in appearance. Usually there is little evidence of pruritus. Tudor (1983) isolated a *Paecilomyces* sp. and *Mucor circinelloides* from the feather shafts of pigeons (*Columba livia*) and various psittacines, which he regarded as the cause of pruritus and feather pulling. Some mycotic infections were associated with bacteria.

In a recent report, a 16-month-old black Orpington hen (*Gallus gallus domesticus*) was taken to a veterinarian with clinical signs of intense pruritus and progressively worse feather loss for a month. At examination, the head showed considerable feather loss and white crusting on the unfeathered areas of the skin including typical scutula around the base of the feather follicles. These are saucer-like crustings that are characteristic of favus infections. The aural cavity was inflamed and contained a large amount of caseous exudates. The rest of the body, including the neck, underneath the wings, and on the legs, also showed considerable feather loss. Histopathology of biopsies obtained from the head and neck revealed follicles with multifocal suppurative folliculitis with intralesional fungal organisms, follicular keratosis, multifocal epithelial cell degeneration, and severe chronic lymphoplasmacytic and histiocytic perifolliculitis. On PAS staining, multifocal aggregates of variable-sized, pink-stained round conidia 0.5 to 1 um in diameter were observed. Based on the morphologic characteristics, the fungal bodies observed in the sample were most likely *Microsporum* sp. The hen was treated with grisovin 125 mg once a day for a period of 3 months, making significant improvement (Baron and Doneley, 2014).

Hine *et al.* (1990) recorded candidiasis of the uropygial gland of chinstrap penguins (*Pygoscelis antarctica*). Forbes (personal communication, 1997) also encountered *Malassezia* sp. (a yeast-like fungus) associated with feather loss and greasy skin in Harris's hawks (*Parabuteo unicinctus*) and found *Candida* and *Aspergillus* spp. to be relatively common secondary infective agents in chronic bumblefoot, especially when the foot remains bandaged for long periods with infrequent dressing changes. Sartory (1942) recorded *Aspergillus fumigatoides* var. *roseus* n. sp. as pathogenic to the feathers of pigeons (*C. livia*). Krautwald-Junghanns (1990) described favus in parrots (*Amazona* spp.) associated with *Aspergillus* spp., namely *A. fumigatus* and *A. niger.* She also described pruritus caused by *Mucor* spp. in African grey parrots (*Psittacus erithacus*), small parakeets, and other species. She said that in these infections the fungi may spread to the respiratory system and cause death. According to Perry (1987) *Helminthosporium* spp. infection of the skin and feathers in psittacines can cause extensive feather loss.

Pathology

Lesions are usually superficial and confined to the epidermis, dermis, and base of the feathers. Hyperkeratosis is associated with infiltration of the keratinized layers of the skin and feather follicles by fungal hyphae; acanthosis, acantholysis, and hydropic degeneration of cells in the stratum spinosum may also occur with infiltration of the underlying dermis by mononuclear cells (Droual *et al.*, 1991).

Diagnosis

The disease can be suspected from the macroscopic appearance of the lesions. The presence of septate hyphae on microscopic examination of skin scrapings, softened and cleared by crushing and soaking in aqueous potassium hydroxide (10% to 20% KOH w/v) for about 30 minutes, confirms the diagnosis when associated with histopathological lesions. Frequently, the fungi prove difficult to isolate and identify, even when using special culture media. Some species of normally saprophytic fungi may be found in association with the lesions, making it difficult to determine their significance. It is believed that sometimes mycotic infections may be secondary to immunosuppressive disorders. Perry (1994) stated that dermatophytes of birds do not fluoresce under ultraviolet light.

Differential Diagnosis

The disease is most likely to be confused with skin lesions caused by epidermoptic, cnemidocoptic, or *Neocheyletiella* mites. Malnutrition, especially hypovitaminosis A, affecting the health of the skin, may be a predisposing cause in some cases. Perry (1987) described "pseudofavus" as a nonspecific disorder of unknown etiology affecting birds in late summer and early autumn. It closely resembles favus but microscopic examination reveals no evidence of fungal involvement.

Control and Treatment

A good standard of hygiene and avoidance of malnutrition are essential prophylactic measures. Tudor (1983) recommended STA (salicylic acid 3 g, tannic acid 3 g, and ethyl alcohol qs 100 mL) or copper sulfate (1:2000 dilution) applied to affected areas of the skin as safe and effective for fungal infections. However, since then, several other fungicidal agents have been developed. For example, Broadbent (1994) reported the successful treatment of *T. mentagrophytes* infection of an ostrich (*Struthio camelus*) using three treatments with natamycin (Mycophyt, Mycofarm) at 4-day intervals followed by "three soakings" with an enilconazole emulsion (Imaverol, Janssen Pharmaceuticals) at 3-day intervals. The natamycin only reduced the scabbing, but subsequent use of enilconazole produced total resolution. Forbes (personal communication, 1997) described treatment of cutaneous aspergillosis in a goshawk (*Accipiter gentilis*; Fig. 14-11). Hine *et al.* (1990) satisfactorily treated candidiasis of the uropygial gland of penguins by feeding them fish containing itraconazole (Sporanox, Janssen Pharmaceuticals) at a dosage rate of 10 mg/kg/day for 20 days. For the treatment of favus caused by *M. gallinae*, Bradley *et al.* (1995) recommended miconazole nitrate 2% (Micatin, Advanced Care Products). It should be remembered that oil/lipid-based products for topical application are contraindicated in birds because they mat the plumage and lead to excessive preening.

ACKNOWLEDGMENTS

The author would like to acknowledge the contribution of Dr. Ian Keymer to this section in the previous edition of this book; I would like to profusely thank Robert Doneley for his unstinting assistance with the preparation of this section.

REFERENCES

Baker JR: Survey of feather diseases of exhibition budgerigars in the United Kingdom, *Vet Rec* 139:590–594, 1996.

Baron H, Doneley B: Favus in a backyard chicken (*Gallus gallus domesticus*). *Proceedings of the Annual Conference of the Association of Avian Veterinarians Australasian Committee and the Unusual Pets and Avian Veterinarians Special Interest Group* (Australian Veterinary Association), Cairns, pp 4–7, 2014.

Blackmore DK, Keymer IF: Cutaneous diseases of wild birds in Britain, *Br Birds* 62:316–331, 1969.

Bradley FA, Bickford AA, Walker RL: Efficacy of miconazole nitrate against favus in oriental breed chickens, *Avian Dis* 39:900–901, 1995.

Broadbent RS: Favus in ostrich, *Vet Rec* 135:536, 1994.

Droual R, Bickford AA, Walker RL, et al: Favus in a backyard flock of game chickens, *Avian Dis* 35:625–630, 1991.

Hine S, Sharkey P, Friday RB: Itraconazole treatment of pulmonary, ocular and uropygeal (*sic*) aspergillosis and candidiasis in birds—data from five clinical cases and controls. *Proceedings of the American Association of Zoo Veterinarians*, South Padre Island, TX, pp 322–327, 1990.

Keymer IF: Mycoses. In Petrak ML, editor: *Diseases of cage and aviary birds*, ed 2, Philadelphia, 1982, Lea & Febiger.

Krautwald-Junghanns ME: Befiederungsstörungen bei Ziervöegeln, *Prakt Tierarzt* 71:5–14, 1990.

Perry RA: Avian dermatology. In Burr EW, editor: *Companion bird medicine*, Ames, IA, 1987, Iowa State University Press.

Perry RA: A diagnostic approach to avian self-mutilation syndromes. In Cross GM, editor: *Proceedings of the Australian association of the avian veterinarians*, 1994, Currumbin, pp 159–178.

Sartory A: *Aspergillus fumisalordes* var. *roseus* n. sp., pathogenic to pigeon feathers, *C R Acad Sci Gen* 214:565, 1942.

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.

FURTHER READING

Bradley FA, Bickford AA, Walker RL: Diagnosis of favus (avian dermatophytosis) in Oriental breed chickens, *Avian Dis* 37:1147–1150, 1993.

Fonseca E, Mendoza L: Favus in a fighting cock caused by *Microsporum gallinae*, *Avian Dis* 28:737–741, 1984.

Perry RA, Gill J, Cross GM: Disorders of the avian integument. In Rosskopf WJ Jr, Woerpel RW, editors: *Veterinary clinics of North America, small animal pet avian medicine*, Philadelphia, 1991, WB Saunders.

Reavill D: Fungal diseases. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, MD, 1996, Williams & Wilkins.

Riedel BB: Favus and its treatment with a quaternary ammonium compound, *Poult Sci* 29:741–742, 1950.

Saif YM, Fadly AM: *Diseases of poultry*, Ames IA, 2008, Blackwell Publishing.

PARASITES

Rolf Schuster, Oliver Krone

Parasites are living creatures that live permanently or temporarily in or on other living organisms (hosts) for the purpose of nutrition, development, and reproduction. In addition, they also benefit from transportation (phoresy) and distribution. In contrast to commensalists, mutualists, and symbionts, the parasite harms its host. Parasites are a heterogenic group of organisms consisting of protozoans, helminths, and arthropods. Depending on their location parasites can be divided into ectoparasites, living on the surface of the host (on the skin and in feathers) and endoparasites, living in the host's organs, blood, or tissues. Most avian arthropods are ectoparasites whereas protozoans and helminths are endoparasites. Temporary parasites have only short contact with the host, mainly for feeding (mosquitoes, fleas, and ticks), and steady parasites spend their whole life or a considerable span of the life cycle on or in the host (protozoa, helminths, and lice). A division between periodic and permanent parasites is useful for ectoparasites. The life cycle of parasite species may include free-living stages. These species are called periodic parasites (fleas, mosquitoes, chigger mites, and myiasis flies). Contrary to this, species where all stages have a parasitic lifestyle are called permanent parasites (ticks, most of the mites, lice, and bugs). Host-specific parasites can be grouped into stenoxenous (with a narrow host range: *Eimeria* spp., lice, feather mites, louse flies) and euryxenous species (with a broad host spectrum: *Isospora lacacei*, bed bugs). Life cycles can be simple, including only one host (homoxenous parasites) or complex by involving one or more intermediate hosts before reaching the definitive host, where maturation and sexual development occurs (heteroxenous parasites). Usually parasites are considered as pests, but parasites with complex life-cycles are sensitive to environmental changes and can therefore be indicators for the integrity of habitats.

PROTOZOA

Protozoans are eukaryotic single-cell organisms possessing a real cell nucleus. Parasitic protozoa of major importance for birds are united into three classes: Trichomonadea (*Trichomonas* and *Histomonas*), Coccidea (*Eimeria*, *Caryospora*, *Isospora*, *Sarcocystis*, and *Toxoplasma*), and Haematozoa (*Haemoproteus*, *Leucocytozoon*, and *Plasmodium*).

Trichomonadea

Trichomonas gallinae is a parasitic flagellate that inhabits the upper digestive and respiratory tracts primarily of columbiform and psittaciform birds, raptors, and captive bustards and causes canker or roup (in pigeons) and frounce (in raptors). The parasite is pear-shaped to round

and measures 10 to 20 µm. It has four anterior flagella and an axostyle (recurrent flagellum)-directed posterior. The undulating membrane does not reach the posterior end of the trophozoite. *T. gallinae* causes clinical illness mainly in young birds, while adult birds may be latently infected without showing clinical symptoms. The life cycle of *T. gallinae* is direct from one host to the next without resistant cyst stages. Pigeon nestlings become infected via feeding on "pigeon milk" produced in the crop of the adults and raptors become infected when feeding on infected prey. *T. gallinae* can reach other birds (turkeys, chickens and passerines) via contaminated drinking water when infected pigeons and other wild birds have access to the same water source. The parasites enter the water from the mouth of the infected host.

Closely related *Tetratrichomonas gallinarum*, *Tritrichomonas eberthi,* and *Chilomastix gallinarum* are dwellers of the ceca of chickens, turkeys, and related birds. *Tetratrichomonas anatis*, *T. anseris*, and *Cochlosoma anatis* inhabit the caeca of water fowl. These flagellates are considered apathogenic and have a worldwide distribution. Other intestinal flagellates are *Spironucleus columbae* in pigeons, *S. meleagridis* in turkeys and chickens.*Histomonas meleagridis* is the pathogen that causes the infectious typhlohepatitis or blackhead disease in galliform birds (mainly turkeys, but also pheasants, partridges, and chickens). The morphology of the parasite depends on the location and the stage of the disease. It is round or amoeboid with clear ectoplasm and granular endoplasm from 4 to 30 µm. A single flagellum may be present when trophozoites are in the cecal lumen. Parasites in lesions of the cecal wall or in mucosal tissue of the liver lack flagellum. Shed with feces of infected birds, trophozoites of *H. meleagridis* do not survive because the parasite does not form cysts. *Heterakis gallinarum*, a cecal nematode of galliform birds, plays a role in the transmission of *H. meleagridis.*

Coccidea

Eimeriidae

Together with the haematozoans (Haemosporida and Piroplasmida) coccideans belong to the phylum Apicomplexa—a large group of obligate, intracellular parasitic protists that possess a unique organelle, called an apicoplast, and an apical complex involved in penetrating a host cell.

Birds are hosts of three Coccidea families: Eimeriidae, Cryptosporidiidae, and Sarcocystidae. Representatives of the genera *Eimeria*, *Isospora*, *Tyzzeria*, and *Caryospora* are typical pathogens belonging to the family Eimeriidae. They are host-specific parasites of epithelial cells, mainly of the intestine. The life cycle of *Eimeria*, *Isospora*, and *Tyzzeria* is direct and does not require intermediate hosts but is characterized by an alteration of asexual and sexual multiplying generations. In life cycles of *Caryospora* species intermediate hosts may be involved. *Caryospora* sporozoites invade host tissues but stay dormant. In a database on avian Eimeriidae compiled by Duszynski *et al.* (1998), 224 *Eimeria* and 401 *Isospora* species were mentioned. Most of the *Eimeria* species ($n = 68$) were described in galliform birds, while passeriform birds had the greatest variety of *Isospora* species ($n = 347$). The same source listed 21 avian *Caryospora* species; most of them were described in birds of prey and owls. Of the 11 known species of the genus *Tyzzeria*, six occur in birds. The status of two *Wenyonella* species described in ducks in India is uncertain. Despite the large number of above mentioned pathogens coccideans in free-ranging birds usually are not of clinical importance. The main morphologic differences in oocysts between the above mentioned genera can be seen after sporulation:

1. *Eimeria:* Four sporocysts with two sporozoites in each (quadrosporocystic, diplosporozoit)
2. *Isospora:* Two sporocysts with four sporozoites in each (diplosporocystic, tetrasporozoic)
3. *Caryospora:* One sporocyst with eight sporozoites (monosporocystic, octosporozoit)
4. *Tyzzeria:* No sporocyst, eight sporozoites in oocyst (asporocystic, octosporozoit)
5. *Wenyonella:* Four sporocysts with four sporozoites in each (quadrosporocystic, quadrosporozoit)

According to Yabsley (2008), 197 known avian *Eimeria* species parasitize 162 avian hosts. Most of the known species occur in galliform followed by anseriform, charadriiform, and gruiform birds. Ten bird orders do not contain any *Eimeria* host. The homoxenous life cycle of *Eimeria* coccidia has been intensively studied in species infecting commercial poultry and consists of endogenous schizogony followed by gamogony (Fig. 14-88) and exogenous sporulation resulting in the formation of sporulated oocysts (Fig. 14-89). Most of the avian coccideans complete their life cycle in the alimentary tract and only a few cases of an extra intestinal location of development stages are known (Table 14-15).

Isospora species occur in a number of free-living songbirds; in cage birds, especially in estrildid finches (zebra finches, strawberry finches, and red-cheeked cordon bleu finch); and in canaries. Compared with the genus *Eimeria*, avian *Isospora* species are less host specific. Thus *I. lacacei* has been described in more than 100 passerine birds and intrafamiliar host specificity is suggested for the *Isospora* genus. Most of the descriptions of avian *Isospora* species were based on the morphology of their oocysts and because oocysts were excreted with host feces, it was assumed that these species have strictly intestinal cycles similar to those of *Eimeria* spp. Extraintestinal development stages found in macrophages in canaries and in the viscera of sparrows with concurrent *Isospora* infection were allocated to new genera, *Atoxoplasma* and *Lankesterella*, respectively. However, it was shown experimentally that these parasites could not be transmitted by blood transfusion but only by ingestion of *Isospora* oocysts. Using the example of canary *Isospora*, it was shown experimentally that the development of some species (*I. canaria*) is restricted to the intestinal epithelium, while sporozoites of others (*I. serini*) penetrate into macrophages within the intestinal mucosa and are transported to various organs where further multiplication takes place. Because the above mentioned extraintestinal forms were developmental stages of *Isospora* and did not belong to other parasites, the recognition of *Atoxoplasma* and *Lankesterella* could no

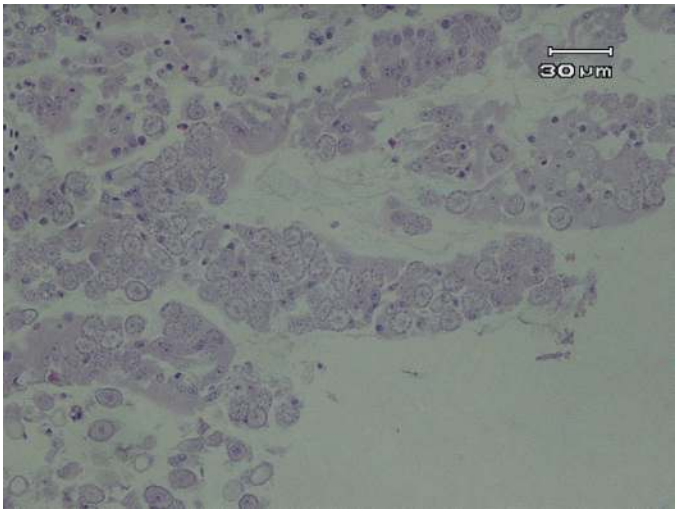


FIGURE 14-88 Histologic section of a partridge intestine showing a large number of macrogamonts of *Eimeria alectoreae.* (Courtesy Dr. J. Kinne, CVRL, Dubai.)

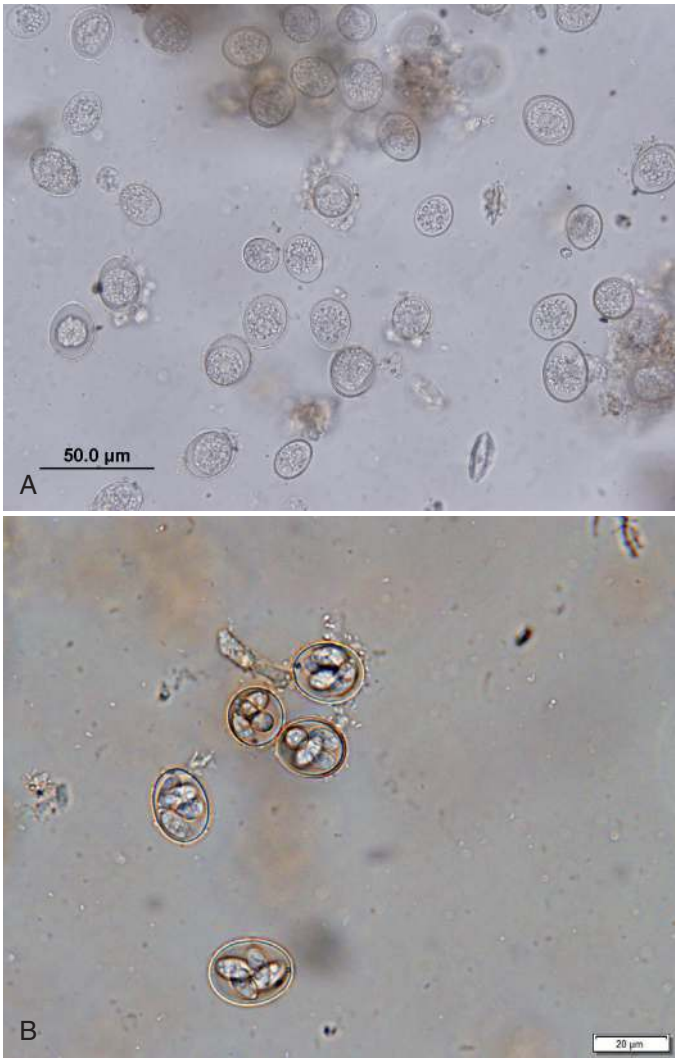


FIGURE 14-89 Unsporulated *Eimeria* oocysts in a fresh chicken (*Gallus domesticus*) fecal sample **(A)** and *Eimeria maxima* sporulated **(B)**. The sporulated oocyst contains four sporocysts with two sporozoites in each.

TABLE 14-15 *Eimeria* Species in Selected Avian Hosts

Host Species	*Eimeria* Species	Clinical Form of Coccidiosis
Chicken	*E. acervulina, E. brunetti, E. hagani, E. maxima, E. mivati, E. mitis, E. necatrix, E. praecox, E. tenella*	Intestinal
Turkey	*E. adenoeides, E. dispersa, E. gallopavonis, E. innocua, E. meleagridis, E. meleagrimitis, E. subrotunda*	Intestinal
Quail	*E. bateri, E. coturnicus, E. dispersa, E. tsunodai, E. virginianus*	Intestinal
Partridges	*E.* alectoreae, *E. caucasica E. kofoidi, E. legionensis, E. padulensis, E. procera*	Intestinal
Pheasants	*E. colchici, E. duodenalis* and *E. phasiani*	Intestinal
Guinea fowl	*E. gorakhpuri, E. grenieri, E. numidae*	Intestinal
Peafowl	*E. arabica, E. kharjensis, E. mandali, E. mayurai, E. muticus, E. patnaiki, E. pavonina, E. pavonis, E. pavoaegyptica, E. riyadhae*	Intestinal
Pigeons/doves	*E. labbeana, E. columbarum*	Intestinal
Ducks	*E. danailovi, E. mulardi E. boschadis*	Intestinal Renal
Geese	*E. anseris, E. nocens, E. kotlani E. truncata*	Intestinal Renal
Cranes	*E. reichenowi, E. gruis*	Disseminated visceral

longer be justified and they are now treated as junior synonyms. Thus independent of the course of the life cycle all coccideans with diplosporocystic, tetrasporozoite oocysts and a single polar Stieda body were assigned to the genus *Isospora* (Fig. 14-90).

Coccidia of the genus *Caryospora* are phylogenetically old parasites with most of the species occurring in reptiles. Among the 15 *Caryospora* species identified in raptors and owls, six are found exclusively in the Falconidae family (Table 14-16). Their oocysts are large and spherical to ovoid without a micropyle. Their sporocysts lack Stieda bodies and enclose eight stubby sporozoites and a residuum (Fig. 14-91). *Caryospora* species have a facultative diheteroxenic (predator–prey) life cycle where facultative intermediate hosts (mainly rodents) become infected by ingesting sporulated oocysts. It is assumed that sporozoites invade extraintestinal tissues of the intermediate host, stay dormant as hypnozoites, and do not undergo multiplication. Feeding experiments using *C. kutzeri* revealed a slightly shorter prepatent period compared with direct transmission. Along with raptors and owls, *Caryospora* species were detected in gulls (*C. argentati* and *C. undata*), in the European robin (*C. jiroveci*), and in the Cuban blackbird (*C. gloriae*). Another undescribed species was recently found

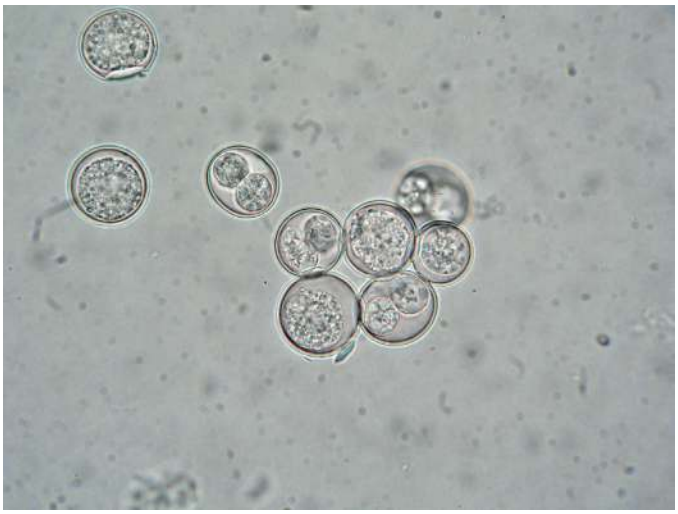


FIGURE 14-90 *Isospora rothschildi* oocysts in a fecal sample of a Bali mynah (*Leucopsar rothschildi*). The oocyst contains two sporocysts after sporulation. (Courtesy N. Pantchev, IDEXX Vet Labor, Ludwigsburg.)

TABLE 14-16 Morphologic Characterization of *Caryospora* Oocysts in Falcons

	Oocyst		Sporocyst			
Species	**Shape**	**Size (µm)**	**Shape**	**Size (µm)**	**Locality**	**Host**
C. boeri	Subspherical	36.6 × 33.4	Ovoid	27.8 × 19.6	Europe	*Falco tinnunculus*
C. falconis	Spherical	29.5-36.5	Spherical	21.0-23.0	Europe	*F. peregrinus, F. subbuteo, F. tinnunculus*
C. kutzeri	Subspherical	38.7 × 34.1	Ovoid	24.6 × 21.0	Europe	*F. biarmicus, F. cherrug, F. jugger, F. mexicanus, F. peregrinus, F. rusticolus, F. subbuteo, F. tinnunculus*
C. megafalconis	Subspherical or ovoid	43.6 × 35.8	Spherical	23.8	Europe	*F. cherrug, F. rusticolus, F. tinnunculus*
C. neofalconis	Subspherical	27.0 × 23.8	Ovoid	18.8 × 14.8	Europe	*F. biarmicus, F. mexicanus, F. peregrinus, F. subbuteo, F. tinnunculus*
C. biarmicusis	Ovoid	40.2 × 34.7	Spherical	20.1	Arabia	*F. biarmicus*
C. cherrughi	Ovoid	32.1 × 29.3	Ellipsoid	24.1 × 19.6	Arabia	*F. cherrug*

FIGURE 14-91 Sporulated *Caryospora megafalconis* oocyst in a fecal sample from a gyr falcon (*Falco rusticolus*). The oocyst contains a single sporocyst with eight sporozoites and a sporocysts rest body.

TABLE 14-17 *Cryptosporidium* Species Isolated from Birds

	Oocyst Size			
Species	**Length (µm)**	**Width (µm)**	**Location**	**Natural Hosts**
C. baileyi	6.0-7.5	4.8-5.7	Conjunctiva, nasopharynx, bronchi, air sacs, small and large intestines, Bursa fabricii	Gulls, chickens, cormorants, cranes, weaver birds, turkeys, ducks, geese, quail, bulbuls, ostriches, parakeets, parrots, falcons
C. meleagridis	4.5-6.0	4.2-5.3	Small and large intestines, bursa of Fabricius	Turkeys, parrots, chickens, cockatiels, partridges
C. galli	8.0-8.5	6.2-6.4	Proventriculus	Chickens, finches, capercaillie, parrots, flamingos, cardinals, hornbills

in farmed African houbara bustards (R. K. Schuster, unpublished information).

Cryptosporidiidae

Cryptosporidiidae are small intracellular but extraplasmatic euryxenous parasites that were recently recognized as significant pathogens of humans and of a large number of other vertebrate hosts. Three avian species, *Cryptosporidium baileyi*, *C. meleagridis*, and *C. galli* (Table 14-17) are recognized and at least 10 other avian genotypes of other *Cryptosporidium* species have been identified in more than 30 bird species. *C. baileyi* is the most frequent avian species but *C. meleagridis* is able to infect humans, as well as causing up to 10% of cases of human cryptosporidiosis. Two other species, *C. anserinum* from geese and *C. tyzzeri*, were not adequately described and are considered *nomina nuda*. Avian cryptosporidiosis manifests in respiratory disease, enteritis, and renal disease. The route of infection is orally with a life cycle similar to Eimeriidae including merogony followed by gamogony. A certain proportion of the oocysts is not excreted and causes autoinfections. *C. baileyi* can enter the host when oocysts are placed on the conjunctiva. *Cryptosporidium* oocysts are small and contain four naked sporozoites. They can be diagnosed in carbol fuchsin-stained fecal smears or mucosal scrapings from respiratory organs (Fig. 14-92). Molecular tools are used to confirm the exact species affiliation.

Sarcocystidae

The coccidia of the genera *Sarcocystis* and *Frenkelia* belong to the class Sporozoea (subclass: Coccidia). These protozoan parasites live in the mucosal layers of the intestine, where they sexually reproduce resulting in diplosporocystic tetrasporozoic oocysts. Oocysts occur in feces sporulated and are surrounded by a thin wall that often ruptures releasing sporocysts (Fig. 14-93). The sporocysts excreted by the feces of the definitive host must be ingested by an intermediate host (rodents and birds). Within the intermediate host the parasite multiplies asexually several times before cysts are built in the muscle (*Sarcocystis*) or brain (*Frenkelia*). The life cycle of the parasite is completed when a cyst in the mouse/bird is ingested by the raptor. Infections with *Sarcocystis*/*Frenkelia* spp. are seldom pathogenic. Nestlings may develop clinical symptoms such as diarrhea, feces with blood, and emaciation. Odening (1998) listed seven *Sarcocystis* spp. for the Falconiformes and

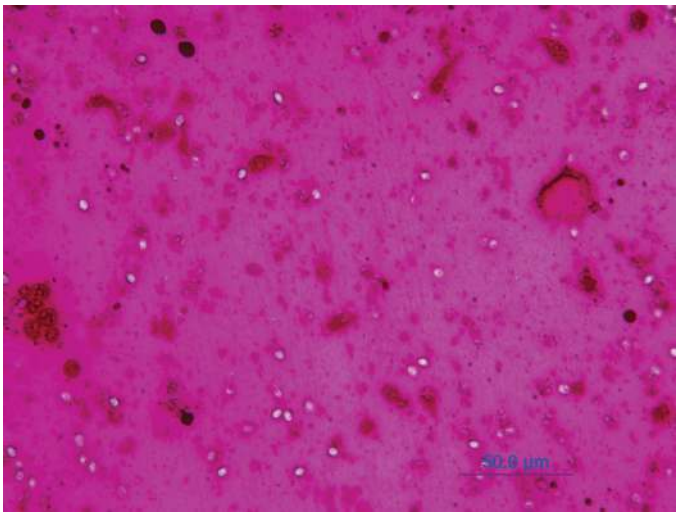


FIGURE 14-92 Fast-acid stain of nasal discharge of a falcon with *Cryptosporidium baileyi* infection. Oocysts in a thin smear remained unstained at a red background.

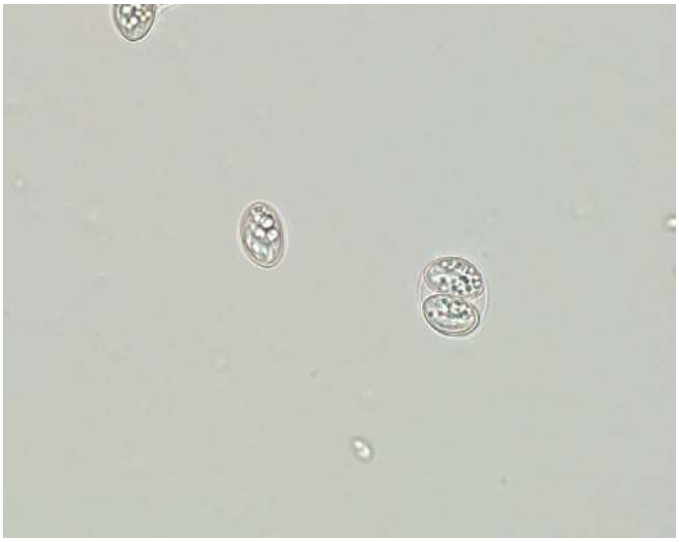


FIGURE 14-93 *Sarcocystis calchasi* from northern goshawk. Oocyst contains two sporocysts (*right*) and liberated sporocyst (*left*). (Courtesy P. Olias, Washington University, St. Louis, MO.)

four for the Strigiformes. He also declared the genus *Frenkelia* to be a synonym of *Sarcocystis* not only because of their same morphology but also because of their developmental features.

HELMINTHS

Helminths are worm-like parasites. Avian helminths are represented in three phyla of the Animal Kingdom: Platyhelmintha with digenetic trematodes and cestodes, Nematoda, and Acanthocephala. All of these helminths are endoparasites colonizing various organs and tissues. Regarding their ontogeny, helminths can be divided into biohelminths that have an indirect life cycle and require intermediate hosts and geohelminths that have a direct life cycle. Leeches belonging to the phylum Annelida also can parasitize on birds, mainly as ectoparasites.

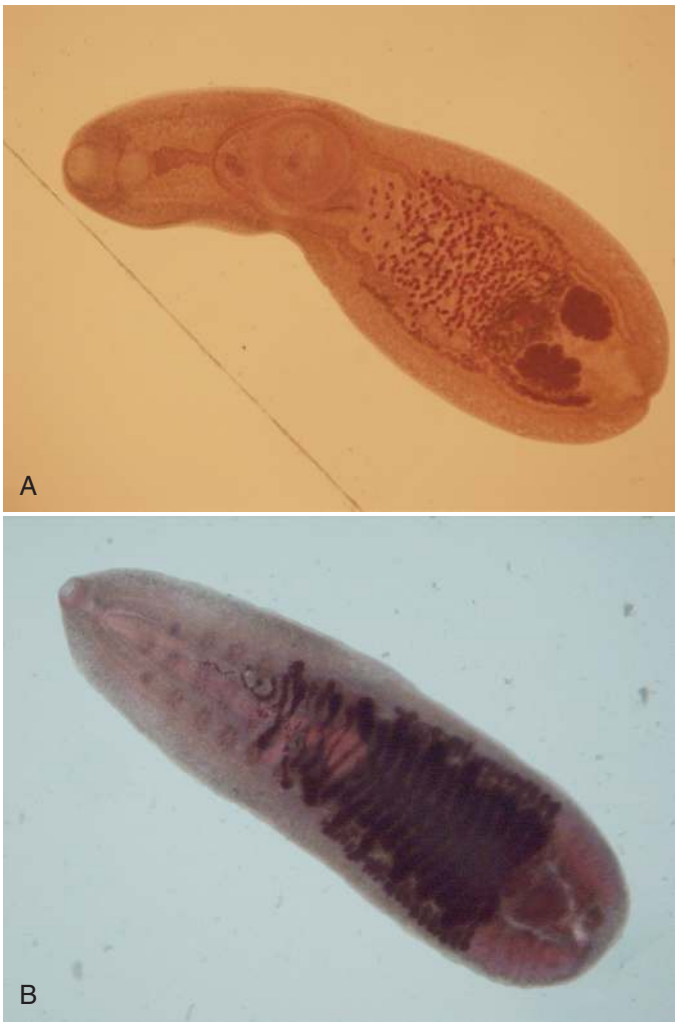


FIGURE 14-94 *Pittacium* sp. from greater flamingo (*Phoenicopterus roseus*) **(A)**. Oral sucker and pharynx are situated at the anterior end, followed by an esophagus. Two blind-ending intestinal branches commence before ventral sucker and cease at the posterior end. Paired testes and ovary at posterior end, uterus filled with eggs between gonads and ventral sucker, and genital opening posterior to intestinal bifurcation. *Notocotylus triserialis* from duck **(B)**. Ventral sucker is absent. It is replaced by three rows of "ventral glands."

Trematoda

With more than 2500 genera in more than 140 families, the subclass Digenea (digenetic trematodes) is a large group of obligate endoparasites of vertebrates known as flukes. Their body is bilaterally flattened and contains a primitive alimentary tract with blind-ended intestinal branches, primitive protonephridial excretory and nervous systems, and reproductive organs (Fig. 14-94). With the exception of representatives of the family Schistosomatidae (Fig. 14-95), all avian trematodes are hermaphrodites. Most of the species possess an oral sucker and an acetabulum functioning as attachment organs. The size of avian fluke species varies between less than 1 mm (*Centrocestus armatus* in cormorants) and several centimeters (*Cathemasia hians* in storks). The majority of flukes inhabit the intestinal tract, but some have adapted to other organs/cavities. All Schistosomatidae species, e.g., occur in the circulatory system (Fig. 14-96). *Collyriclum faba* is found in pairs in subcutaneous cysts located around the vent of songbirds in Eurasia and America.

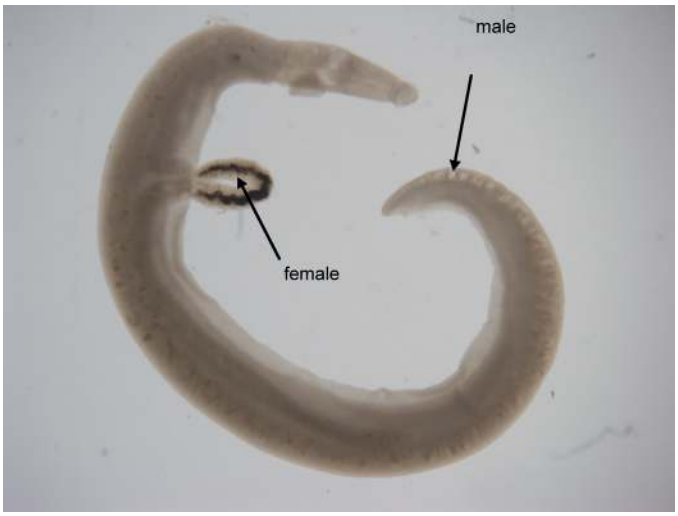


FIGURE 14-95 *Austrobilharzia* sp. found in mesenterial veins of a herring gull (*Larus argentatus*). These schistosomatid flukes live in pairs. The slender female is situated in the gynecophoric groove of the male.

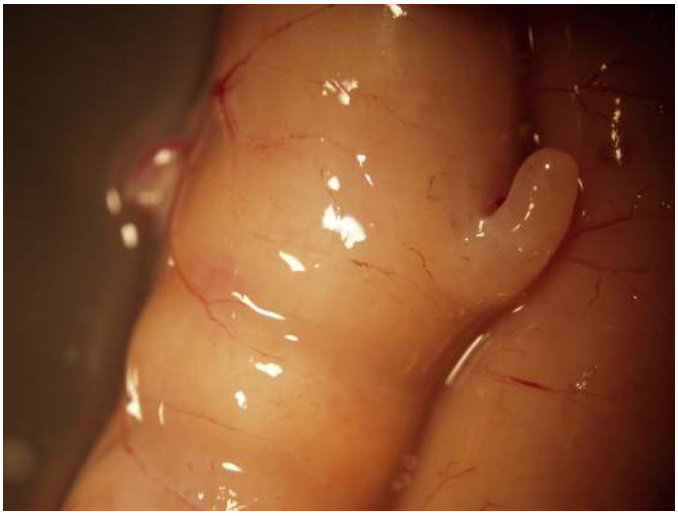


FIGURE 14-96 *Gigantobilharzia melanoidis* inhabits small venous blood vessels of the intestine. Female worms can be recognized by a brown pigment in their intestinal tubes.

Flukes have a diheteroxenic or tetraheteroxenic (mostly aquatic or semiaquatic) life cycle where mollusks are the compulsory first intermediate hosts (Table 14-18). Polyheteroxenic cycles are rare in avian species. However, some species (i.e., *Strigea falconispalumbi*) have developed the most complex life cycles among the parasites with raptors as definitive hosts (Krone 2007). Dicrocoeliidae, Brachylaimidae, and Leucochloridiidae species have land snails as intermediate hosts. Life cycle stages consist of eggs, miracidia, sporocysts, rediae, cercariae, metacercariae, and marita (adult worm). Birds become infected by ingesting infective metacercariae attached to substrates (water plants, snail shells, etc.; Fig. 14-97) or are located in tissues or organs of second intermediate hosts (Fig. 14-98) or by transcutaneous penetration of schistosomatid furcocercariae (Fig. 14-99).

Adult flukes inhabit those sites of the body with a connection to the outer world so they can pass their eggs into the environment. The

FIGURE 14-97 *Notocotylus triserialis* metacercariae on the shell of a snail.

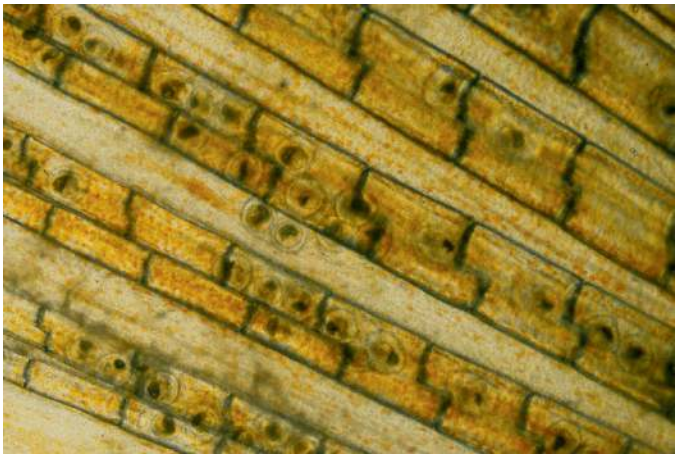


FIGURE 14-98 *Metorchis xanthosomus* metacercariae in the caudal fin of an id.

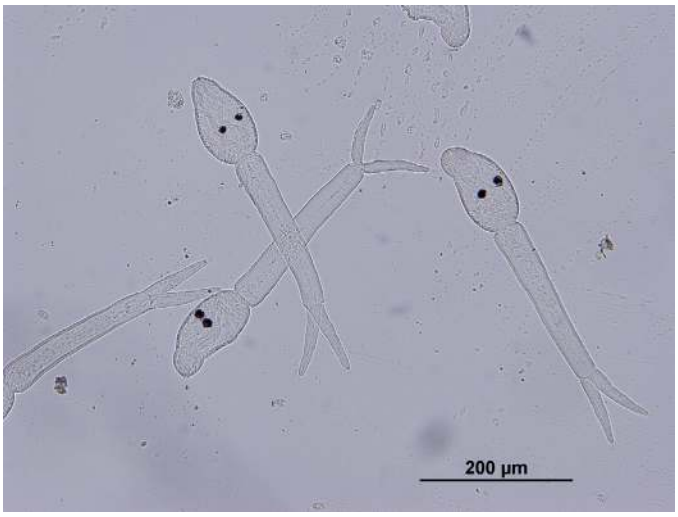
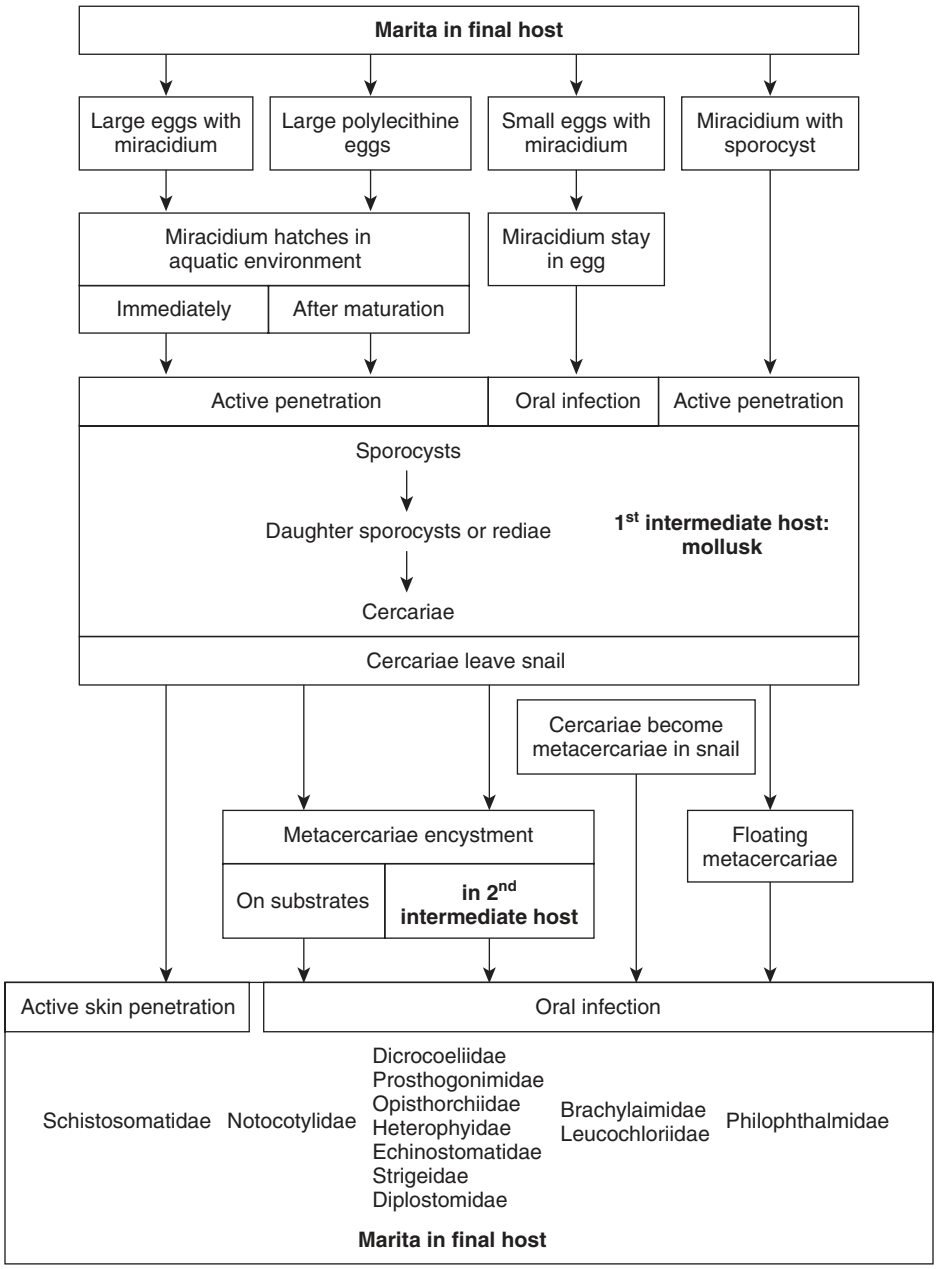


FIGURE 14-99 *Gigantobilharzia* furcocercariae. Certain types of cercariae are equipped with eye spots, which enable them to ascend to the surface of the water body in search of a final host.

TABLE 14-18 Basic Life Cycles of Trematodes



majority of avian trematodes dwell in the digestive system. These are representatives of the families Brachylaimidae, Leucochloridiidae, Leucocloridiomorphidae, Clinostomidae, Echinostomatidae, Echinochasmidae, Parorchiidae, Psilostomidae, Notocotylidae, Plagiorchiidae, Eumegacetidae, Microphallidae, Pleurogenidae, Heterophyidae, Galactosomatidae, Strigeidae, Bolbocephalidae, Diplostomidae, and Cyathocotylidae. Most of the species inhabit the small intestines and ceca, but specialized trematodes can be found in the mouth cavity (*Clinostomum* spp. in herons), in the esophagus (*Cathemasia hians* in storks), in the rectum (*Pittacium* spp.), in the bursa of fabricii (*Prosthogonimus*), and even on the rim of the cloaca (species of genera *Cloacitrema*, *Pygorchis*, and *Stomylotrema*). The *Trichobilharzia* spp. live in the nares of waterfowl and can cause the disease "swimmers itch" in humans Other location sites are bile ducts and the gallbladder (Opisthorchiidae, Dicrocoeliidae, and Gymnophallidae), eyelids (Philophthalmidae), respiratory system (Cyclocoelidae and Orchipedidae), excretory system (Renicolidae and Eucotylidae), or oviduct (Prosthogonimidae). *Collyriclum faba* is located in subcutaneous tissue cysts around the cloacal opening. Bird schistosomes inhabit venous blood vessels mainly of the gut. Their eggs must break through to reach the intestinal lumen. *Strigea falconispalumbi*, an intestinal fluke of raptors, can use birds as paratenic hosts. In this case metacercariae are located in connective tissues under the skin or between muscles (Fig. 14-100).

Cestoda

With representatives in more than 240 genera in two orders, birds have the most diverse cestode fauna (Table 14-19). The order Pseudophyllidea has only one family (Diphyllobothtriidae) with avian hosts.

Diphyllobothrium dendriticum, *Digramma interrupta*, *Schistocephalus solidus*, and *Ligula intestinalis* occur in the intestines of fish-eating birds (gulls, grebes, and cormorants). Within the order Cyclophyllidea, six cestode families consist exclusively of avian-specific species, but only two cestode families contain genera species that can be found in both birds and mammals. All Amabiliidae occur in grebes with the exception of the genus *Amabilia*, which is found in flamingos. Representatives of the Progynotaeniidae parasitize charadriiform birds and flamingos, whereas tapeworms of families Acoleidae and Dioecocestidae are found only in charadriiform birds. Within the family Anoplocephalidae avian, species are restricted to the subfamily Anoplocephalinae. Most of these species were found in psittacid birds. A species of veterinary importance within this subfamily, *Aporina delafondi*, is a parasite of pigeons. The majority of Paruterinidae species occur in passeriform birds. Species of the genera *Ascometra* and *Octopetalum* are specific parasites of gruiform and galliform birds. Several species of the genus *Cladotaenia* infect falconiform birds, whereas *Paruterina* species parasitize owls. The family Davaineidae contains a number of veterinary important species within the genera *Davainea*, *Raillietina*, *Cotugnia*, and *Skrjabinia*. Species of the genera *Otiditaenia* and *Idiogenes* can be found in bustards and *Houttuynia struthionis* can be a serious obstacle in ostrich farming. Metadilepididae species use passeriform and caprimulgiform birds as final hosts. The widest range of diversity of avian-specific species parasitize in two families: Dilepididae and Hymenolepididae. Most of the species here occur in a wide variety of wild birds but *Choanotaenia infundibulum*, *Amoebotaenia cuneata*, *Echinolepis carioca*, *Hymenolepis cantaniana*, *Microsomacanthus setigera*, and *Wardium farciminosum* are found in landfowl; *Diorchis stefanski*, *Microsomacanthus compressa*, *M. collaris*, *Sobolevicanthus gracilis*, *Wardium aequabilis*, and *Fimbriaria fasciolaris* are parasites of waterfowl. *Burhinotaenia delachauxi* is a frequently occurring tapeworm in stone curlews (*Burhinus oedicnemus*). The genus *Hispaniolepis* with *H. falsata* and *H. fedtschenkoi* occur in bustards and galliform birds, respectively.

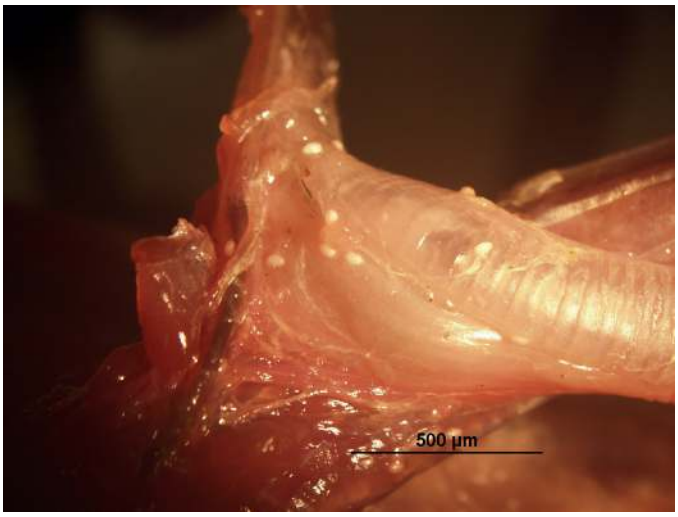


FIGURE 14-100 *Strigea falconispalumbi* metacercariae in subcutaneous tissues of a greenshank (*Tringa nebularia*).

Cestodes are gut-dwelling platyhelminths with a white to yellowish-colored body divided into scolex, neck, and a segmented strobila (Fig. 14-101). Their size varies from several millimeters (*Davainea proglottina*) up to 50 cm (*Otiditaenia conoides*). The scolex, the holdfast organ of tapeworms, has four suckers and most of the species are equipped with an armed rostellum (Fig. 14-102). Very few species lack an armed rostellum. The rostellum in many hymenolepid and dilepidid species is retractile into a rostellar sheet. Size and shape of rostellar hooks can be used for determination. In some cestodes (*Raillietina*

TABLE 14-19 Cestode Families of the Orders Pseudophyllidea and Cyclophyllidea Occurring in Birds, Mammals, Reptiles, and Amphibians.

		Number of Cestode Genera Found in Host Classes				
Order	**Family**	**In Birds (Only)**	**In Birds and Mammals**	**In Mammals (Only)**	**In Reptiles (Only)**	**In Amphibians (Only)**
Pseudophyllidea	Diphyllobothriidae	3	1	8	3	1
Cyclophyllidea	Anoplocephalidae	11	0	37	3	0
	Catenotaeniidae	0	0	6	0	0
	Nematotaeniidae	0	0	0	3	2
	Progynotaeniidae	6	0	0	0	0
	Acoleidae	2	0	0	0	0
	Dioecocestidae	4	0	0	0	0
	Amabiliidae	6	0	0	0	0
	Davaineidae	21	8	0	0	0
	Dilepididae	95	1	8	0	0
	Dipyliniidae	0	0	3	0	0
	Paruterinidae	23	0	0	0	2
	Metadilepididae	8	0	0	0	0
	Hymenolepididae	51	1	31	0	0
	Mesocestoididae*	0	0	1	0	0
	Taeniidae	0	0	2	0	0

*Birds, rodents, reptiles, and amphibians serve as intermediate hosts for *Mesocestoides* spp.

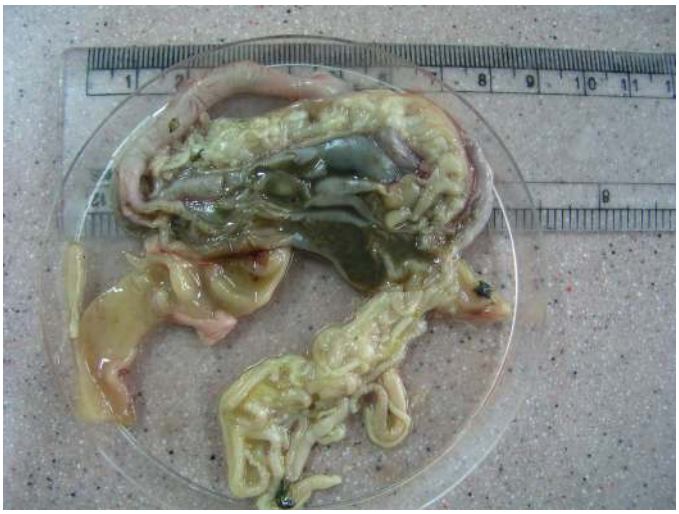


FIGURE 14-101 Intestinal constipation of the small intestine of a houbara bustard (*Chlamydotis undulata*) by *Otiditaenia conoides*.

FIGURE 14-103 *Raillietina* sp. from pigeon. The rostellum of the scolex is armed with a large number of hammer-shaped minute hooklets. Also, the four suckers bear hooklets.

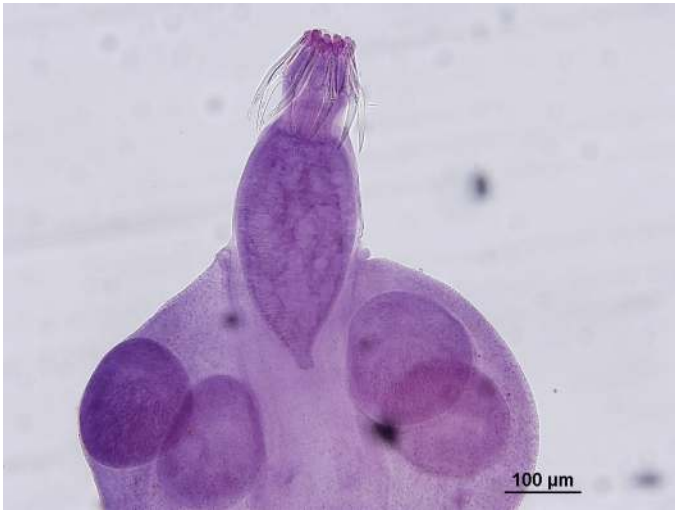


FIGURE 14-102 *Flamingolepis liguloides* from greater flamingo (*Phoenicopterus roseus*). The scolex consists of four suckers and a retractile armed rostellum.

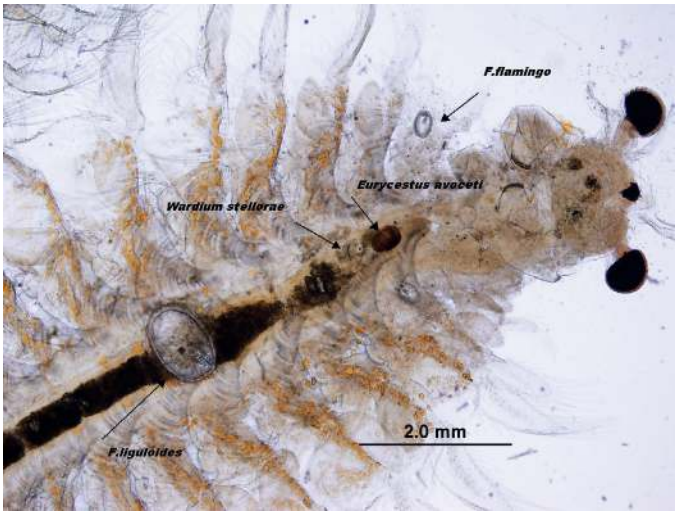


FIGURE 14-104 Cysticercoids of different cestode species in the body of a brine shrimp.

spp.) suckers also can be armed (Fig. 14-103). Pseudophyllid tapeworms have sucking groves (bothria) instead of suckers. The strobila consists of segments (proglottids).

The life cycle of diphyllobothriid cestodes is triheteroxenic. Larvae (coracidia) that emerge from eggs are ingested by aquatic copepods that serve as first intermediate hosts. A variety of freshwater fish serve as second intermediate hosts for diphyllobothriids. Cyclophyllid avian cestodes have a diheteroxenic life cycle with arthropods (crustaceans, beetles, ants, grasshoppers, and flies) and other avertebrates (annelids and molluscs) are involved as intermediate hosts. Larval stages found in the body cavity of the intermediate hosts are different types of cysticercoids (Fig. 14-104). Paruterinid cestodes of the genera *Cladotaenia* and *Paruterina*, cestodes of raptors and owls, use rodents as intermediate hosts. Their larval stage called cladothyridium (Fig. 14-105) is located in the liver of intermediate hosts. Both cysticercoids and cladothyrids already have the scolex of the adult cestode.

Birds mainly act as final hosts for tapeworms, but few cases of cestode larvae (tetrathyridia of *Mesocestoides* spp.) discovered in the abdominal cavity of chickens, turkeys, or in cutaneous cysts in a starling have been published in the literature.

Nematoda

Birds are hosts to a large variety of nematodes located mainly in the alimentary and respiratory tracts. Some spirurids use birds as paratenic hosts. The length of avian nematodes varies from several millimeters up to 35 cm (*Serratospiculum* sp.; Fig. 14-106). Most of the species have a cylindrical form, tapering at either end, and are round at the cross section. Male and female nematodes have a similar appearance, except the genera *Tetrameres* and *Microtetrameres* where females are nearly spherical (Fig. 14-107). Depending on species, the cuticle forms various structures, such as cervical and caudal papillae, alae, cordons, and a copulatory bursa (Fig. 14-108) in male bursate nematodes. The digestive system of nematodes is tubular with a mouth opening that may be surrounded by lips (Fig. 14-109). In some species the mouth opens into a buccal cavity. The esophagus in many of the nematodes is divided into a muscular and glandular part. It is followed by the

FIGURE 14-105 Cladothyridium, the larval stage of *Cladotaenia* spp., isolated from a liver of a common vole. The invaginated scolex is armed with minute hooks.

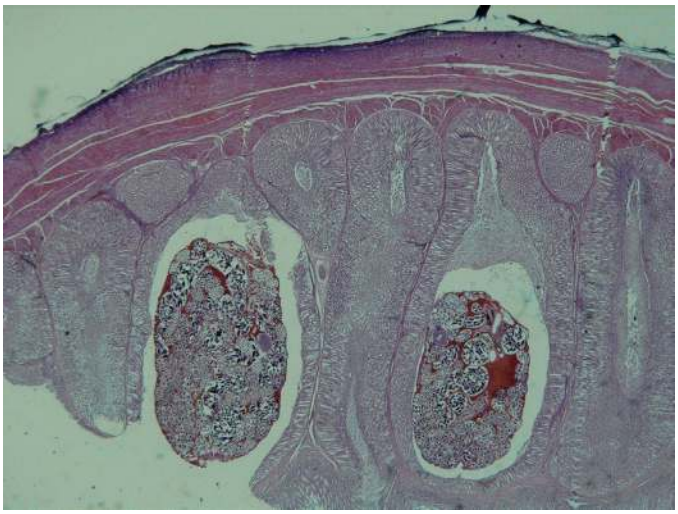


FIGURE 14-107 *Tetrameres* sp. in the stomach of a greater flamingo (*Phoenicopterus roseus*). Histologic section. (Courtesy Dr. J. Kinne, CVRL, Dubai.)

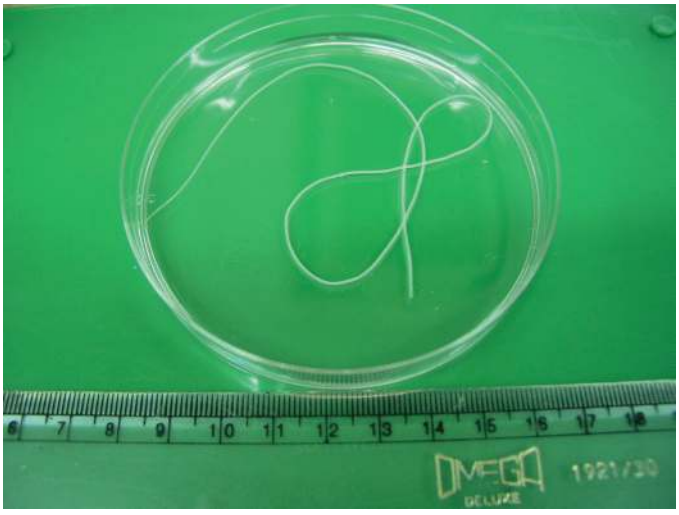


FIGURE 14-106 Female *Serratospiculum* sp. removed from air sac of a Peregrine falcon (*Falco peregrinus*).

FIGURE 14-108 *Trichostrongylus tenuis* from pheasant, posterior end of a male. The copulatory bursa is formed by rips connected by a membrane. The slightly curved spicules are of the same length and the gubernaculum is spindle-shaped.

intestine that opens at the posterior end. In ascarids, both esophagus and intestine at their junction may have blind-ending appendices. The esophagus of hairworms has a capillary form surrounded by a single column of cells known as stichosome. Nematodes are unisexual parasites. The female reproductive system comprises an ovary, oviduct, and uterus which may be paired. The male organs consist of a single testes and a vas deferens terminating in an ejaculatory duct into the cloaca. Spicules and gubernaculums are chitinous accessory male organs used during copulation.

The basic life cycle of nematodes includes eggs and four larval stages. The majority of avian nematode species (*Contracaecum*, *Porrocaecum*, *Eustrongylides*, *Trichinella*, *Histriche*s, and all spirurids) have an indirect life cycle (biohelminths) that involves a variety of avertebrates (mainly arthropods) as intermediate hosts. The rest are soil-transmitted geohelminths with a direct life cycle (*Trichostrongylus*, *Amidostomum*, *Epomidiostomum*, *Heterakis*, *Ascaridia*, and *Subulura*). In some nematodes with a primarily direct life cycle (*Syngamus* and *Cyathostoma*) paratenic hosts (earth worms) may enhance the chance of contact of the parasite and its avian host. Filaroid nematodes are vectorborne parasites and are transmitted by blood-sucking arthropods.

Avian nematodes are mainly found in the digestive tract (Fig. 14-110). Some of them also inhabit the respiratory system (Fig. 14-111 and Table 14-20). Apart from digestive and respiratory systems adult filarioid nematodes in birds (16 genera) can be found in subcutaneous and connective tissues, in the body cavity, or in heart and blood vessels. These are *Pelecitus fulicaeatrae* in tendons of the legs of coots, *Eulimdana* spp. in the neck of waders and gulls, or *Sarconema eurycerca* in the heart of swans. Their microfilariae circulate in the circulatory system or inhabit the skin of the host (Fig. 14-112). Larval nematodes, such as *Physocephalus* spp., *Spirocerca lupi*, or *Paraspiralatus sakeri* encyst in the wall of the alimentary tract or under the skin and between

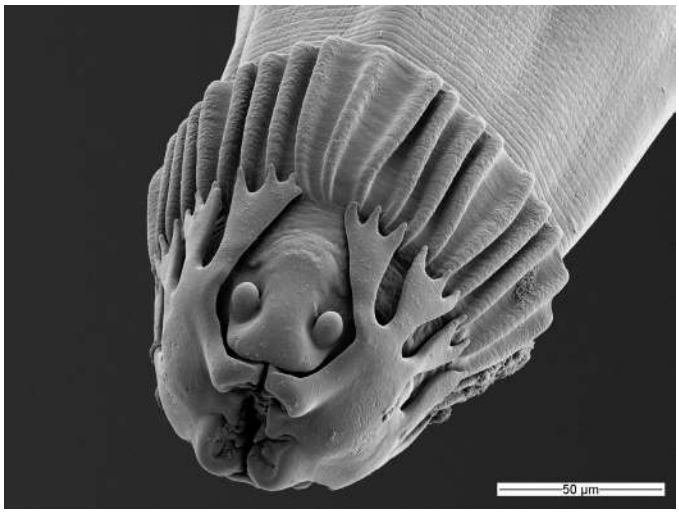


FIGURE 14-109 *Histiocephalus laticaudatus* from houbara bustard (*Chlamydotis undulata*), anterior end. The posterior border of pseudolabia is armed with denticulate plates and the cervical cuticule is inflated with longitudinal striations. There are two striking submedian papillae on ventral and dorsal sites, SEM. (Courtesy D. Viertel and G. Wibbelt, IZW Berlin.)

FIGURE 14-110 Massive ascarid (*Porrocaecum angusticolle*) infection caused a constipation of the small intestine of a Gyr falcon (*Falco rusticolus*).

TABLE 14-20 Location of Selected Adult Avian Nematodes

Location	Nematode	Main Hosts
Esophagus/ proventriculus	*Capillaria contorta, Tetrameres fissispina, Dispharynx nasuta*	Phasianidae
	Cosmocephalus obvelatus, Sexanocara skrjabini	Fish-eating birds
	Echinuria uncinata, Hystriches tricolor, Eustrongylides papillosus	Anatidae
	Histiocephalus laticaudatus	Bustards
Gizzard	*Amidostomum anseri, Epomidiostomum uncinatum*	Anatidae
	Cheilospirura hamulosa	Phasianidae
Small intestines	*Ascaridia galli*	Chicken
	Porrocaecum spp.	Raptors, herons, cranes
	Contracaecum spp.	Fish-eating birds
	Capillaria caudinflata, C. bursata, C. obsignata	Phasianidae
Large intestines	*Heterakis gallinarum, Subulura brumpti*	Phasianidae
Trachea/bronchi	*Syngamus trachea*	Phasianidae , songbirds
	Cyathostoma bronchiale	Anatidae
Air sacs	*Serratospiculum* spp., *Serratospiculoides* spp.	Falconidae

FIGURE 14-111 The gapeworm, *Syngamus trachea*, is a parasite located in the upper respiratory tract. The shorter male is in copula with the female forming a Y or gape-like structure.

muscles (Fig. 14-113). *Trichinella pseudospiralis* in its larval stage is another extraintestinal parasite that might infect birds of prey. Its larvae are located in muscle fibers (Fig. 14-114).

Acanthocephala

Acanthocephalans, also known as thorny-headed or spiny-headed worms, belong to a relatively small group of obligatory endoparasites in the alimentary tract of vertebrates. The length of avian species varies from several millimeters to greater than 10 cm. At adult stages acanthocephalans are gut dwellers. The fixatory organ (proboscis) is deeply inserted into the intestinal wall (Fig. 14-115). Acanthocephalans are diecious with a somewhat hidden sexual dimorphism. Their life cycle is indirect. Eggs excreted with the host's feces contain a larval stage (acanthor) embedded in membranes. Obligate or facultative coprophagic crustaceans and insects serve as intermediate hosts. The acanthor leaves the egg membranes in the gut of the intermediate host and migrates into the body cavity. Here it forms a second larva (acanthella) that later encysts to become a cystacanth. Many acanthocephalan species are known to infect paratenic hosts, and only after being ingested by a suitable final host the larva develop into the adult stage. Of an estimated 1100 representatives worldwide, more than 100 acanthocephalan species were found in birds in Europe and Asia alone. *Filicollis anatis*, *Polymorphus minutus*, and *P. magnus* are frequent

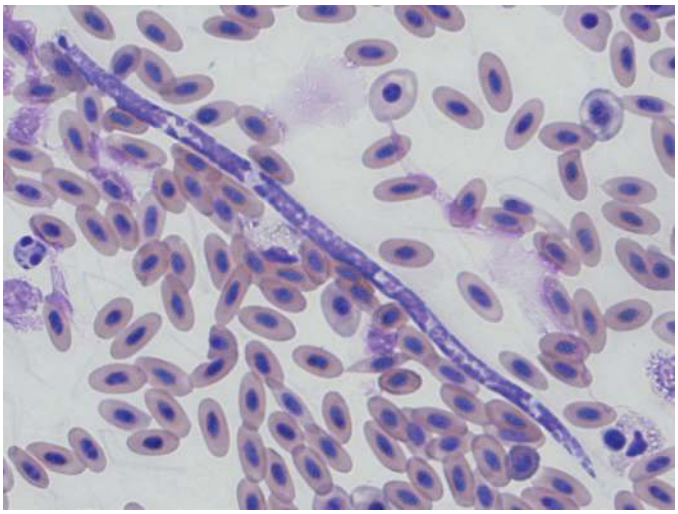


FIGURE 14-112 Microfilaria of *Francofilaria basiri* in a stained blood smear of a partridge.

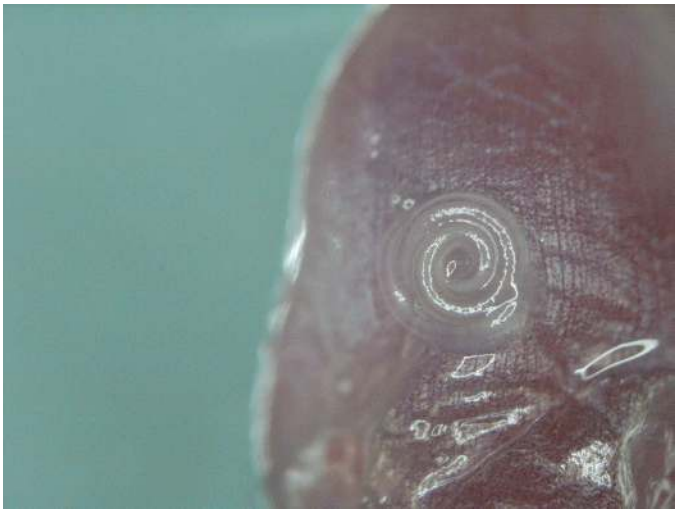


FIGURE 14-113 *Paraspiralatus* larva on the surface of pectoral muscles of a houbara bustard (*Chlamydotis undulata*).

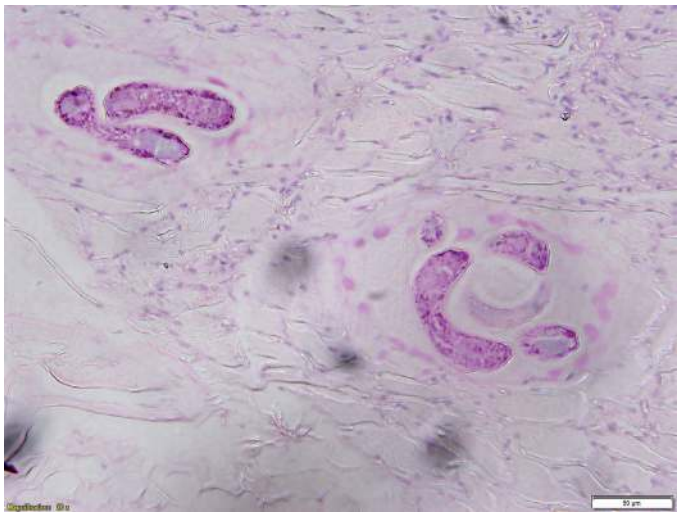


FIGURE 14-114 *Trichinella pseudospiralis* in the muscles of a European goshawk (*Accipiter gentilis*). Histologic section. (Courtesy Dr. J. Kinne, CVRL Dubai.)

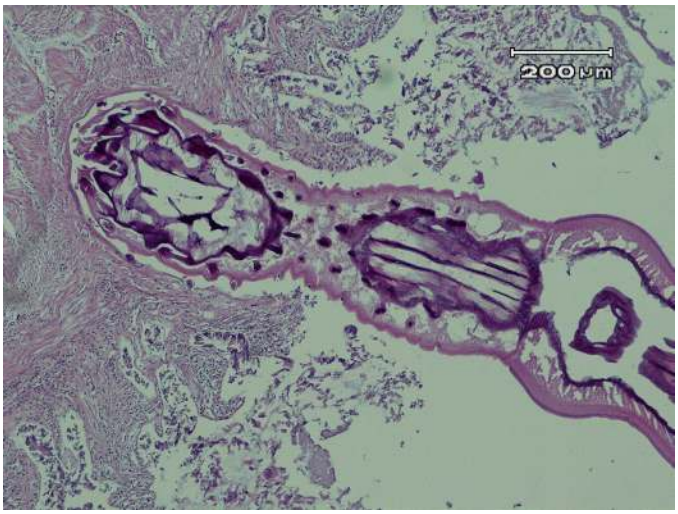


FIGURE 14-115 Proboscis of *Sphaerirostris embae* deeply embedded in the intestinal mucosa of a houbara bustard (*Chlamydotis undulata*). Histologic section. (Courtesy Dr. J. Kinne, CVRL, Dubai.)

FIGURE 14-116 Massive *Sphaerirostris embae* infection in the small intestine of a houbara bustard (*Chlamydotis undulata*).

parasites of ducks, *Sphaerirostris embae* (Fig. 14-116) and *Empodius taeniatus* (Fig. 14-117) occur in bustards, and a number of *Centrorhynchus* species parasitize birds of prey and owls.

Leeches

Leeches are bloodsucking parasites and predators in freshwater, marine, and moist terrestrial ecosystems. Leeches are hermaphrodites. Their segmented body consists of head segments containing the anterior brain and the sucker, midbody segments with ganglia and reproductive organs and posterior segments that are fused together posterior to the sucker. Of the 12 known families, four (Glossiphoniidae, Ornithobdellidae, Hirudinidae, and Haemadipsidae) with 33 species have been found to parasitize birds. Nasal leeches of the genus *Theromyzon* are specialized to feed on waterbirds. Their preferred feeding sites are nasal cavities, trachea, and beneath the nictitating membrane of the eyes.

FIGURE 14-117 Constipation of the small intestine of a houbara bustard (*Chlamydotis undulata*) by *Empodius taeniatus*. This relatively large acanthocephalan species shows a pseudosegmentation of its body and can be confused with tapeworms.

FIGURE 14-118 *Hyalomma* sp. larva found on a marsh harrier (*Circus aeruginosus*).

ARTHROPODS

The phylum Arthropoda consists of two major groups that contain parasites of birds. These are the arachnids with the subclass Acari (ticks and mites) and Mandibulata with the class of insects. Most of the arthropods are ectoparasites. However, some mites or at least their development stages (nest mites, air sac mites, burrowing mites) and larvae of myiasis flies are well adapted to endoparasitic lifestyle. The systematic position of pentastomids is unclear as they are situated somewhere between helminths and arthropods.

Acari

The class Arachnida unites the Acari (ticks and mites) with representatives of 13 other subclasses of joint-legged invertebrates (spiders, cave spiders, solifuges, scorpions, pseudoscorpions, etc.). Except larval stages, which are hexapod, acari have four pairs of legs and the cephalothorax and abdomen are fused together. Mouthparts consist of paired pedipalps and chelicerae. In addition, ticks possess an unpaired hypostome. The supraorder Anactinotrichida (formerly: Parasitiformes) consists of the two orders Metastigmata (ticks) and Mesostigmata (parasitiform mites), while the supraorder Actinotrichida (formerly known as Acariformes) combines the two orders of prostigmatic (formerly: Trombidiformes) and astigmatic mites (formerly: Sarcoptiformes).

Ticks

Ticks belong to the suborder Metastigmata (Ixodida) with about 800 known species. Hard ticks (Ixodidae) are of minor direct disease importance for birds because only larvae and nymphs of three host species attack birds, and adults are mainly found on mammals (Fig. 14-118). Nevertheless, birds, especially migrating species, play a role in the life cycle and distribution of ticks of the genera *Ixodes*, *Amblyomma*, and *Hyalomma* and may be of importance in the epidemiology of tickborne diseases.

Of the more than 50 described species of the genus *Argas*, three species are of major importance for birds. *A. reflexus* (Fig. 14-119) and *A. polonicus* can be found mainly on pigeons, whereas *A. persicus* is more often associated with chickens. In the absence of preferred hosts, these ticks will infest other birds and can affect humans as well. Female *Argas* spp. are up to 11 mm in length and up to 8 mm wide; males are smaller. The egg-shaped body is dorsoventrally flattened with defined margins. The surface cuticle has a leather like and unsclerotized structure. The capitulum with mouthparts can be seen only from the ventral side. Like all argasids, *Argas* species are nocturnal. They shelter in cracks and cleaves and they leave only for blood meals. Soft ticks have a multihost life cycle including adults, eggs, and one larval and up to four nymphal stages. The whole life cycle depends on environmental conditions and the availability of hosts and lasts between 3 and 6 months and up to 3 years. In the absence of hosts, adult soft ticks can fast for several years. Apart from blood feeding, these parasites are vectors and can transmit *Borrelia anserina* and *Aegyptianella pullorum.* In the absence of preferred hosts, avian soft ticks also attack humans and become pests, especially in cities with feral pigeon problems.

Mesostigmatic Mites

Representatives of this group of mites are bloodsucking parasites with relatively long legs. Avian species belong to the families Dermanyssidae, Macronyssidae, and Rhinonyssidae. The life cycle includes adults, eggs, and hexapode larval and two octopode nymphal stages. Three species of these mites are avian ectoparasites. The red mite or poultry mite, *Dermanyssus gallinae*, is a stationary periodical, nocturnally active, euryxenous pest that shelters at daytime in hideaways (cracks and cleaves in poultry houses, below perches, bird nests, etc.) and attack their host in the dark to suck blood. In the absence of hosts these mites can survive up to 5 months. The northern fowl mite, *Ornithonyssus sylviarum* of the family Macronyssidae is a serious pest of poultry and wild birds in northern Europe and America, while the tropical fowl mite, *O. bursa*, is widely distributed in warmer climates (Fig. 14-120). Both *Ornithonyssus* spp. are stationary permanent parasites that can survive only 3 weeks when separated from their hosts. In the absence of avian hosts *D. gallinae* and *Ornithonyssus* spp. can attack mammals and even humans. Another mesostigmatic mite, *Sternostoma tracheacolum*, of the family Rhinonyssidae, is a bloodsucking mite of the respiratory passages of canaries and finches. It has been also found in a wide range of wild and domestic birds.

Prostigmatic Mites

Prostigmatic mites have a relatively big body and a well-developed gnathosoma (capitulum) with mouthparts (chelicerae) constructed to grab or to pierce and pedipalps suitable to feel or to grab. Representatives of the families Cheyletidae, Harpyrhynchidae, Syringophilidae,

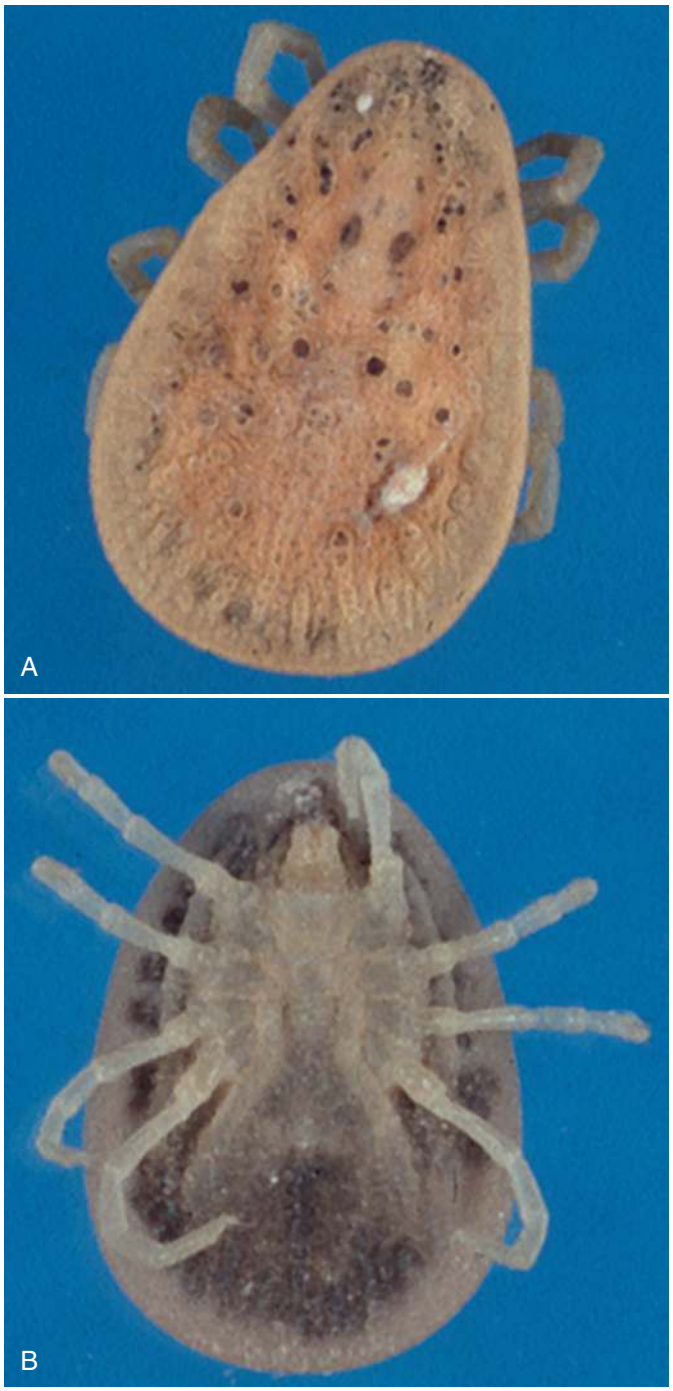


FIGURE 14-119 The pigeon tick, *Argas reflexus*. **(A),** Dorsal view. **(B),** Ventral view. (From Kleine-Tebbe J, Heinatz A, Grazer I: Bites of the European pigeon tick (argas reflexus): Risk of IgE-mediatec sensitizations and anaphylactic reactions, *J Allergy Clin Immunol* 117(1): 190–195, 2006.)

and Trombiculidae are avian parasites. Of the large family Cheyletidae only a few species are of parasitologic interest because most of them are free-living predators. More than 25 species of the genus *Ornithocheyletia* have been described mainly in wild birds. *O. hallae* parasitizes pigeons and doves. This mite pierces the surface layers of the skin and causes itching reactions. Many Cheyletidae species are predators. Members of the genera *Cheletosoma*, *Cheletoides*, and *Cheletopsis* can also be found on birds where they prey on quill mites.

FIGURE 14-120 The tropical fowl mite, *Ornithonyssus bursa*.

Several species of the genus *Harpyrhynchus* parasitize birds of different orders. These mites are less than 500 µm in length and live in colonies in the follicles at the basis of feathers and are transmitted by contact. Female mites drill into the intact skin and lay eggs. *H. nidulans* is one of these species found in pigeons. Mites cause inflammation and formation of bean-sized inflammatory nodules that contain hundreds of adult mites, larvae, and eggs, resulting in feathers falling off.

Quill mites of the family Syringophilidae invade the calamus, the lower part of the quill. More than 100 species have been described worldwide. *Syringophilus bipectinatus* and *Peristerophila columba* are found in chickens and pigeons, respectively. These slender mites seem to be apathogenic. At molt, mites leave the quill and enter freshly developing feathers.

Chigger or harvest mites of the family Trombiculidae have a worldwide distribution and consist of approximately 1600 species. In these mites, only the hexapod larvae live a parasitic lifestyle (Fig. 14-121). Protonymphs and tritonymphs are aphagous resting stages while deutonymphs and adults are predators of soil avertebrates. Representative of the genus *Neotrombicula*, *N. autumnalis*, *N. carpathica*, and *N. inopinata* are the best investigated species. In birds, the yellow to orange-red colored egg-shaped larvae (up to 500 µm long) can be found between feathers of the belly and upper legs and also in the upper respiratory tract. Chigger mites are also important for their role as vectors for *Orientia tsutsugamushi*, *Coxiella burnetii*, and *Anaplasma phagocytophilum* (formerly *Ehrlichia phagocytophilum*).

Astigmatic Mites

The order Astigmata is characterized by missing spiracles. Gaseous exchange takes place over the whole surface of the mite. These mites are relatively small, and avian representatives can be found in the families Hypoderidae, Analgidae, Dermoglyphidae, Epidermoptidae, Pterolichidae, Knemidocoptidae, and Laminosioptidae.

Nest mites of the family Hypoderidae spend a part of their life cycle as subcutaneous parasites (hypopi) in pigeons and wild birds (Fig. 14-122). Deutonymphs of *Hypodectes propus* can enter subcutaneous

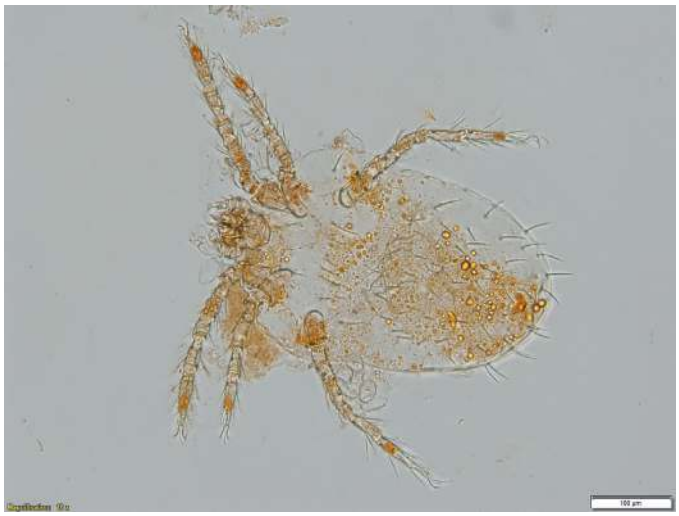


FIGURE 14-121 Chigger mite, a larval stage of the family Trombiculidae.

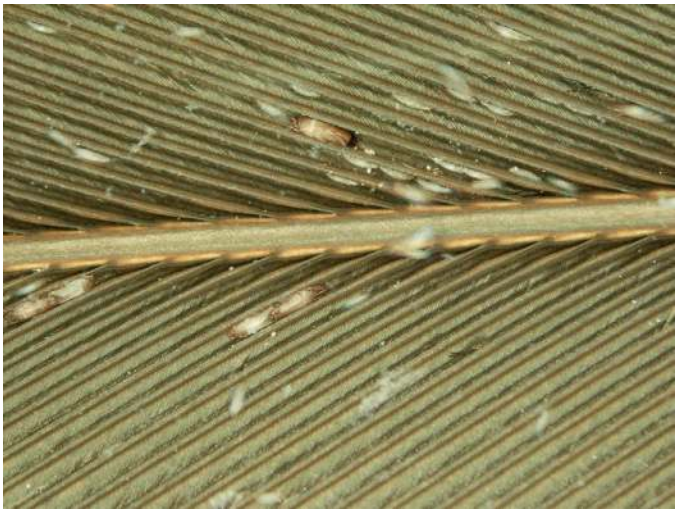


FIGURE 14-123 *Ptiloxenoides phoenicopteri* and their eggs on a wing feather of a lesser flamingo (*Phoenicopterus minor*).

FIGURE 14-122 Deutonymphs of *Hypodectes propus* under in the subcutis of a pigeon (*Columba livia*).

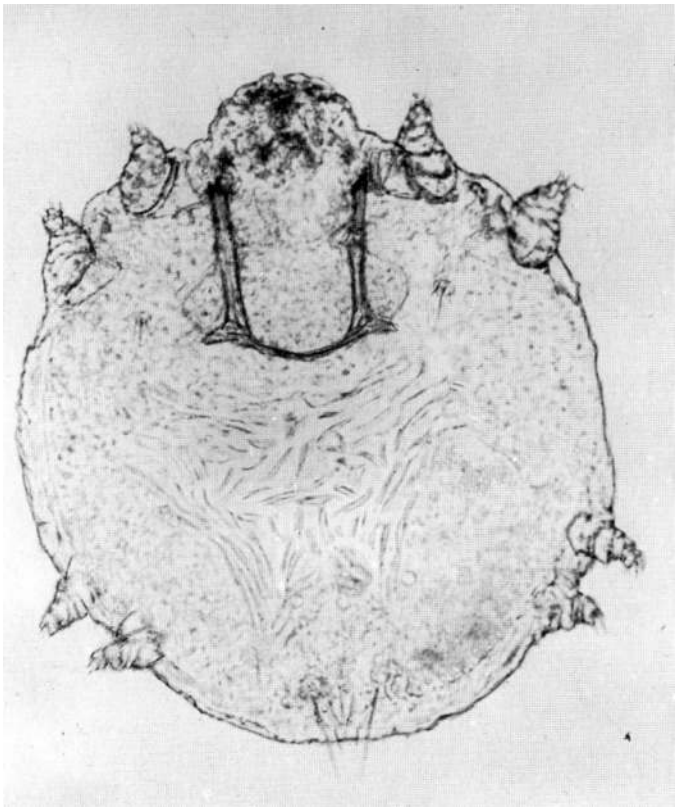


FIGURE 14-124 *Knemidocoptes mutans*, the mite that causes scaly leg disease in chickens. (Courtesy H. Mehlhorn, University of Dusseldorf.)

adipose tissues and leave the host after growing up. The process of entering and leaving the host's skin causes restlessness, dermatitis, and may lead to abnormal molt and loss of feathers. Adult mites are free living.

Feather mites of the family Analgoidea (Fig. 14-123) is a diverse group of strongly stenoxenous parasites counting more than 2000 different species in more than 20 families. Members of Falculiferidae and Analgidae dwell on the surface of feathers, Dermoglyphidae species also inhabit feather quills and Epidermoptidae species live on the skin (Table 14-21). Most of these mites have a size of around 500 µm, but deutonymphs of *Falculifer rostratus* can reach a length of 1.3 mm. The life cycle of feather mites consists of oviparous adults one larval and two nymphal stages. Birds become infected with teleonymphs by contact. In the past feather mites were considered to be apathogenic, but under conditions of high host concentrations, they can cause skin and feather alterations.

Burrowing mites of the family Knemidocoptidae seen in birds (Fig. 14-124) are closely related to Sarcoptidae species in mammals. Seven species of these are stationary permanent parasites known to parasitize on birds (Table 14-22). Depending on the site of alterations they are called scaly face mites, scaly leg mites, or depluming mites. These mites are relatively small (males are up to 170 µm and females are up to 480 µm) with a circular to oval body and short stubby extremities bearing long unsegmented pedicles with stalked pulvilli and claws in male and female specimens, respectively. Fertilized females

TABLE 14-21 Location of Selected Feather Mites of Fowl and Ornamental Birds

Family	Genus	Species	Host	Predilection Site
Analgidae	*Megninia*	*M. cubitalis*	Chicken	Between rami and on upper quill of wing feathers and tectrices of back and breast
		M. columbae	Pigeon	
		M. ginglymura	Turkey	
	Leptosphyra	*L. velata*	Duck	
Dermoglyphidae	*Dermoglyphus*	*D. elongatus*	Chicken	On feathers and in quills of wing feathers
		D. minor	Chicken	
		D. passerinus	Canary birds	
Falculiferidae	*Falculifer*	*F. rostratus*	Pigeon	
Epidermoptidae	*Epidermoptes*	*E. bilobatus*	Chicken	On skin of head, neck, breast
	Rivoltasia	*R. bifurcata*	Chicken	
Pterolichidae	*Pterolichus*	*P. obtusus*	Chicken	
		P. columbae	Pigeon	
	Sideroferus	*S. lunula*	Budgerigar	
Xolalgidae	*Dubininia*	*D. melopsittaci*	Psittacids	On feathers

TABLE 14-22 Avian Burrowing Mites (Knemidocoptidae)

Species	Hosts	Location of Alterations
Knemidocoptes mutans	Chicken, pheasants	Lower unfeathered legs, between toes
K. pilae	Budgerigars and other psittacids	Around beak and eyes, lower legs and cloaca
K. prolificus (Syn. *K. laevis*)	Geese	Around beak and eyes, lower legs and cloaca
K. jamaicensis	Finches, canaries, other passerine birds	Skin and joints
Procnemidocoptes jansseni	Lovebirds	Around beak and eyes, lower legs
Neocnemidocoptes gallinae	Chicken, pheasants	Skin at the base of feathers on rump and back
N. laevis (Syn. *Mesoknemidocoptes laevis*)	Pigeon	Skin at the base of feathers on back and head

bore hollows into skin folds or into the plain epidermis resulting in a comb like meshwork. Mites are transmitted by contact. The life cycle of the viviparous parasites consisting of adults and one hexapod larval and two octopod nymphal stages is completed in 20 and 26 days for males and females, respectively. Under favorable conditions they can survive isolated from their hosts for up to 2 weeks.

Mites of the Family Cytodidae are parasites of the respiratory tract of birds, rodents, and bats. The air sac mite, *Cytodites nudus*, parasitizes the airways, lungs, and air sacs of galliform birds and has been found in canaries. The males of these oval-shaped mites are up to 575 µm long and females reach a length of up to 650 µm. The smooth cuticle bears delicate striations and is covered with sparse short hairs and extremities end with unsegmented pedicles with stalked pulvilli.

The fowl cyst mite, *Laminosioptes cysticola* (Laminosioptidae), is an internal parasite that invades subcutaneous tissues of the neck, breast, flanks, and abdomen of galliform birds and pigeons. The cylindrically shaped male and female mites are relatively small and reach a length of up to 230 and 260 µm, respectively. The smooth and nearly hairless body bears only few long bristles and the legs are short. The first and second pair of legs bear claws and the third and fourth pair end with short unsegmented pedicles with stalked pulvilli. These mites may enter superficial muscle layers and get encapsulated by connective host tissues and become calcified forming yellowish white nodules up to 1 mm.

Insecta

Uniting more than one million species, insects are by far the most diverse class in the Animal Kingdom and it is estimated that more than 370,000 insects have a parasitic lifestyle. Entire orders among insects consist only of parasitic species (Anoplura, Mallophaga and Siphonaptera) and other orders, like Diptera, contain bloodsucking representatives that harm not only by hematophagia but play an important role as vectors. Parasitic insect groups of importance for birds are bugs, biting lice, mosquitoes, blackflies, biting midges, myiasis-causing flies, louse flies, fleas as true parasites and beetles as pests. Beetles are also important intermediate hosts for *Serratospiculum* spp. (Samour and Naldo, 2001) and related nematodes. Insects differ from ticks and mites by the presence of three pairs of legs and the clear division of the body into caput, thorax and abdomen. Their mouthparts consist of paired mandibles, maxillae, labrium and unpaired labrum and hypopharynx. Depending on lifestyle and food uptake they can be modified as biting–licking (biting lice), licking–sucking (housefly), or piercing–sucking (sucking lice, bugs, and fleas). With very few exceptions insects are oviparous. Most insects have a holometabolic development that consists of egg, larva, pupa, and imago. Lice and bugs and some other groups undergo a hemimetabolic development (incomplete metamorphosis), where larval stages are similar to adults and a pupal stage is missing. Temporarily periodic bloodsucking insects differ in their main activity. Diurnal species are active during the day while nocturnal species are active during the night. Crepuscular insects prefer the twilight as the main activity period.

Hemiptera

The order Hemiptera (true bugs) with more than 80,000 species is widespread throughout tropical and temperate regions. Veterinary

FIGURE 14-125 *Cimex lectularius*, male (dorsal view) and female (ventral view).

FIGURE 14-126 *Colpocephalum falconi*, a representative of the Amblycera group. Amblycera are characterized by the presence of two pairs of eyes and antennae in antennal grooves. Mouthparts are situated at the anterior part of the caput.

important species are restricted to the families Cimicidae and Reduviidae. Reduviid bugs, especially from domestic and peridomestic environments, are not host specific and may get their blood meal from a large variety of hosts, including birds. In contrast to reduviid bugs, most of the cimicid bugs or bed bugs are associated with avian hosts and some of them (*Cimex lectularius*, *C. hemipterus*, *C. columbarius*, *Haematosiphon inodorus*, and *Ornithocoris toledoi*) are economically important pests of poultry. Cimicid bugs (Fig. 14-125) are wingless, temporarily permanent, hematophagous, euryxenous, and nocturnal active parasites. The size of the dorsoventrally flattened insects ranges from 3 to 6 mm. They puncture cutaneous blood vessels of the host with their mandibular stylets. After precedented blood meals female bed bugs lay clutches of three to five eggs (a total of 200) in cracks and crevices of houses and outbuildings, dovecots, nest boxes, etc. First-stage larvae hatch after a 2- to 3-week incubation period and the whole development includes five larval stages completed in 6 weeks. The entire life span lasts up to 18 months. Bed bugs can fast for more than 12 months.

Mallophaga

Mallophaga (biting or chewing lice) is a group of obligatory ectoparasites mainly of birds and to a lesser extent (only 12%) of mammals. The large diversity of avian mallophaga of more than 3800 different species in 253 genera can be explained by the strict stenoxeny of most species and their presence in nearly all bird species. Up to 20 different lice species can be found on a single bird species. Depending on species, size varies between 0.5 and 14 mm. The body of the wingless insects is dorsoventrally compressed and clearly divided into caput, thorax, and abdomen. Mallophages are divided into two morphologically distinct suborders, Amblycera and Ischnocera, containing 30% and 70% of all avian species, respectively. Examples for avian amblyceran genera are *Menopon*, *Eomenacanthus*, *Menacanthus*, *Hohorstiella*, *Bonomiella*, *Trinoton*, *Colpocephalum*, and *Neocolpocephalum* (Fig. 14-126). Representatives of this group feed by chewing soft areas of the skin causing an area of localized bleeding from where they drink. Ischnoceran genera, like *Goniocotes*, *Chelopistes*, *Stenocrotaphus*, *Campanulotes*, *Coloceras*, *Lipeurus*, *Cuclotogaster*, *Anatoecus*, *Columbicola* and *Anaticola*, parasitize domestic birds (Fig. 14-127). Ischnocera species are restricted to feathers and feed on feathers and dead skin dandruff.

FIGURE 14-127 *Columbicola columbae* is an Ischnocera species. The mouthparts are located in the center of the ventral caput.

Mallophages are stationary permanent parasites with a paurometabolic development. The whole life cycle includes eggs (Fig. 14-128), three larval instars, and the adult stage on the host. Larvae hatch after a 4- to 10-day incubation of eggs and each larval stage is completed within 3 to 12 days. Adults live up to 1 month and females lay one egg per day after fertilization. Transmission of biting lice is mainly by contact horizontally and vertically, but it is also known that Ischnoceran lice can be transmitted via phoresis on hippoboscid flies.

Nematocera

The suborder Nematocera consists of four important families of bloodsucking insects: mosquitoes (Culicidae), blackflies (Simuliidae), biting midges (Ceratopogonidae), and sandflies (Phlebotominae) as one subfamily of moth flies (Psychodidae). Females of all these holometabolic insects are temporarily periodic parasites and their role as avian parasites is underestimated (Table 14-23).

TABLE 14-23 Ornithophilic Nematocera Species: Their Breeding Sites and Vector Role

Family	Ornithophilic Species	Bloodsucking Activity	Breeding Sites	Vector Role in Relation to Birds
Culicidae	*Culex pipiens, Anopheles plumbeus, Aedes vexans*	Nocturnal	Marshes, swamps, puddles, water tanks, leaf axils, pitcher plants, or tree holes	Bird malaria, Togavirus and Flavivirus
Simuliidae	*Simulium annulus, S. canonicolum, S. meridionale*	Diurnal (early morning and evening shortly after sunset)	Fast-running streams with clear water with submerged or emergent water plants	*Leucocytozoon* spp., *Splendidofilaria fallisensis*
Ceratopogonidae	*Culicoides arbicola, C. beckae, C. edeni, C. festivipennis, C. kibunensis, C. knowltoni, C. minutissimus, C. stellifer*	Crepuscular (at twilight hours at dusk and dawn)	Habitats ranging from moist compost or leave litter to mud at the margins of ponds and lakes or floating weed	*Haemoproteus* spp., *Leucocytozoon caulleryi*
Phlebotominae	*Phlebotomus papatasi, P. sergenti, Lutzomyia longipalpis*	Nocturnal	Terrestrial breeding sites: animal burrows, shelters, caves, damp leaf litter	None

The mentioned Phlebotominae species are not really ornithophilic but are known to suck blood on avian hosts.

FIGURE 14-128 Louse nits on a wing feather barb of an Egyptian vulture (*Neophron percnopterus*).

Myiasis-Causing Diptera

Myiasis is the infestation of healthy or necrotic tissues of the live body with dipteran larvae. Obligate myiasis is caused by larvae of fly species that require a living host as part of their life cycle. Facultative myiasis maggots normally develop in decaying organic matter and occasionally infest necrotic wounds. Avian myiasis is mainly diagnosed in nestlings of nest-dwelling birds (Passeriformes, Falconiformes, Strigiformes, Coraciiformes, and Columbiformes) and handicapped birds. It is caused by species of Calliphoridae (Protocalliphora), Muscidae (*Philornis*, *Passeromyia*, and *Mydaea*), and Neottiophilidae (*Neottiophilium* and *Actinoptera*). *Protocalliphora* species with more than 30 representatives can be found in Europe, North America, northern Africa, and temperate Asia. *Philornis* is reported from South America and *Passeromyia* and *Mydaea* from Asia and Australia. Nestskipper flies of the genus *Neottiophilium* are found in the Palearctic. Other calliphorids associated with avian myiasis include *Calliphora* and *Lucilia* species in Europe. Also *Wohlfahrtia magnifica* in the Old World and *W. opaca* in the New World can cause myiasis in birds. Females of calliphorid and muscid species are attracted by odor and lay eggs whereas sarcophagid flesh flies deposit their larvae on the body of their hosts. The parasitic stage lasts up to 1 week and larvae leave the host to pupate.

Hippoboscidae

Louse flies (family: Hippoboscidae) are a relatively small group of dorsoventrally flattened, dark-colored, obligate hematophagous viviparid flies of 2 to 10 mm in length with piercing–sucking mouthparts. Their larval development takes place in the uterus and the female gives birth to fully developed larvae ready to pupate. The family has three subfamilies consisting of approximately 213 different species. While all species of the subfamily Lipopteninae parasitize mammals, most of the Ornithomyinae species parasitize birds. Ten of the 16 genera of this subfamily have species associated with birds. The best known are *Crataerina hirundinis*, parasites of swallows and martins; *C. acutipennis*, *C. melbae*, *C. pacifica*, and *C. pallida*, parasites of swifts; and the pigeon louse fly, *Pseudolynchia canariensis* (Fig. 14-129). The life cycle of stenoxenous *Crataerina* species of migrating birds appears to be temperature mediated and associated with the presence of hosts at nesting sites. On return from winter habitats swifts are louse fly free and become infected with freshly hatched parasites. At the beginning of the season, transmission seems to be horizontal, and during the breeding season transmission is undoubtedly vertical from adults to nestlings. Hippoboscids have a low fecundity and produce only 12 to 15 larvae deposited in the nest. Most of the pupae are produced during the nesting period. Pupae remain in diapause until the following spring. *C. pallida* takes blood meals of 20 to 40 mg every 5 days. They feed on the lower rump of nestlings. On adults they are often found feeding on the belly and neck. *P. canariensis* was found on a wide range of avian hosts (13 families in eight orders) but columbiform birds are the main hosts. The main distribution of pigeon louse flies is in tropical, subtropical, and temperate areas with mild winters. In contrast to *C. pallida*, its pupae die at low temperatures. The longevity of pigeon louse fly imagines was determined experimentally and lasts 17 days

FIGURE 14-129 The pigeon louse fly, *Pseudolynchia canariensis*.

FIGURE 14-130 *Ceratophyllus gallinae* (female) is one of the most common fleas on domesticated and a variety of wild birds. *Ceratophyllus* species have a comb on the first thoracic segment (pronotal ctenidium). (Courtesy R. Schmäschke, University of Leipzig.)

when fed at 24-hour intervals. During this time, a female produces an average of nine pupae. *P. canariensis* can transmit *Haemoproteus columbae*. The infection with this hemoparasite is tolerated by adult pigeons but may be fatal in nestlings. *H. turtur* is another species transmitted by the vector. It is very host specific for turtle doves. Mallophages and a number of avian-associated mites are found to be phorets on *P. canariensis*. The subfamily Hippoboscinae contains one avian species, *Struthiobosca struthionis*, a parasite of ostriches (*Struthio camelus*) in East and South Africa.

Siphonaptera

The order Siphopnaptera consists of approximately 2500 described species in 239 genera and 19 families. Fleas are small, wingless, and obligate hematophagous parasites of warm-blooded vertebrates. Their size ranges from 1.6 to 9 mm. The compact, laterally compressed body bears caudal-directed spines and bristles in a species-specific pattern and most species are equipped with combs of spines. Males are usually smaller than females and sexes differ in the structure of antennae. In veterinary important species, the third pair of legs is stronger than the others and is well adapted for jumping. Both sexes are hematophagous with piercing–sucking mouthparts. Fleas have a holometabolic development. Female flies deposit eggs on substrates in the environment of the host. Three worm-like instars have chewing mouthparts and feed on organic substrates (hair, dandruff, and dead insects). Feces of the adult fleas that contain undigested dry blood may be a requirement for the development of some species. Larvae pupate on the ground covering themselves in a silk cocoon to which, with dust and other fine particles, they adhere. After 1 to 2 weeks fleas are ready to hatch stimulated by vibration that signals the presence of a suitable host. The vast majority of fleas have mammals as hosts and only 6% of the species prefer avian hosts. Only five families (Ceratophyllidae, Leptopsyllidae, Pulicidae, Pygiopsyllidae, and Rhopalopsyllidae) contain avian-related species. Fourteen species of the genus *Ceratophyllus* (Fig. 14-130) were detected in bird nests in Europe. Of these, *C. gallinae*, *C. tribulis*, and *C. garei* were found to have a broad host spectrum including 20 to 60 birds. *C. sciurorum* has rodents (dormice, tree squirrels, and yellow-necked and wood mice) as main hosts but also has been detected in nests of more than 30 bird species. The moorhen fly, *Dasypsyllus gallinulae*, which originally was distributed in South America, was also detected in Europe and is considered low host specific. Species with a narrow host range were *C. fringillae*, *C. styx*, *C. hirundinis*, *C. borealis*, *C. rossittensis*, *C. rusticus*, *C. vagabundus*, *C. columbae*, and *Ornithophaga mikulini* of the family Leptopsyllidae. *Echidnophaga gallinacea* (Fig. 14-131), known as the hen flea, belongs to the family Pulicidae and has a cosmopolitan distribution. It is one of the 21 species of sticktight fleas more commonly seen on mammals. The host spectrum of hen fleas is not restricted to avian hosts; it can also be found on carnivores, rodents, horses, and even humans. Contrary to the above-mentioned species, females of *E. gallinacea* are stationary periodic parasites (Fig. 14-132). Fluke-armed ledges on their maxilla allow sticktight fleas to be attached to the host as long as 3 weeks.

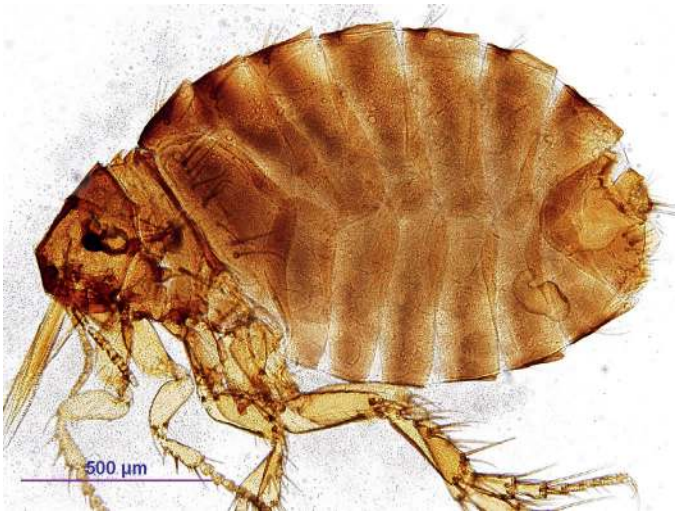


FIGURE 14-131 *Echidnophaga gallinacea* (female). The hen flea is combless and has long piercing mouthparts armed with minute barbs.

PENTASTOMIDA

Pentastomida is a group of about 100 species of parasites of the respiratory system. Their systematic affiliation between helminths and arthropods is unsolved. Because of the resemblance of the genus *Linguatula*

FIGURE 14-132 *Echidnophaga gallinacea* around the eye of a houbara bustard (*Chlamydotis undulata*).

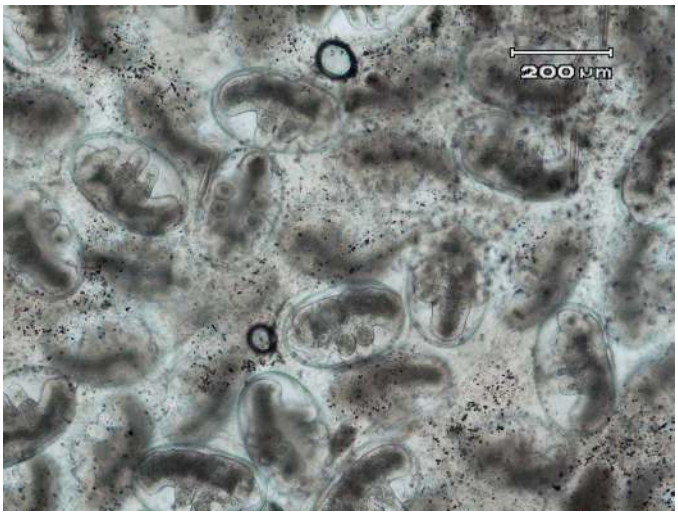


FIGURE 14-133 *Reighardia* eggs containing hexapod larval stages in tracheal smear of a herring gull (*Larus argentatus*).

to a tongue, these parasites are called tongue worms. Most species occur in reptiles and only two species of the genus *Reighardia*, *R. sternae and R. lomviae*, are known to utilize marine birds (gulls and alcids) as an avian-definitive host (Fig. 14-133). In contrast to all other species with an indirect life cycle, the genus *Reighardia* has a direct bird-to-bird transmission. In a relatively recent publication mature *Raillietiella trachea* were described in vultures in Punjab, Pakistan (Riley *et al.*, 2013).

REFERENCES

Duszynski DW, Upton SJ, Lee Couch L: The coccidia of the world: http://biology.unm.edu/biology/coccidia/table.html, 1998.

Odening K: The present state of species-systematics in Sarcocystis Lankester, 1882 (Protista, Sporozoa, Coccidia), *Syst Parasitol* 41:209–233, 1998.

Riley J, Oaks JL, Gilbert M: *Raillietiella trachea* n. sp., a pentastomid from the trachea of an oriental white-backed vulture Gyps bengalensis taken in Pakistan, with speculation about its life cycle, *Syst Parasitol* 56:155–161, 2013.

Samour JH, Naldo J: Serratospiculiasis in captive falcons in the Middle East: a review, *J Avian Med Surg* 15(1):2–9, 2001.

Yabsley MJ: Eimeria. In Atkinson CT, Thomas NJ, Hunter DB, editors: *Parasitic diseases of wild birds*, Ames, IA, 2008, Wiley-Blackwell.

FURTHER READING

Anderson RC: *Nematode parasites of vertebrates: their development and transmission*, Eastbourne, UK, 2006, CPI Antony Rowe.

Atkinson CT, Thomas NJ, Hunter DB: *Parasitic diseases of wild birds*, Ames, IA, 2008, Wiley-Blackwell.

Barus V, Sergeeva TP, Sonin MD, et al: *Helminths of fish-eating birds of the Palaearctic Region. 1. Nematoda*, Prague, 1978, Publishing House of the Czechoslovak Academy of Sciences.

Beck W, Pantchev N: *Praktische Parasitologie bei Heimtieren*, ed 2, Hannover, Germany, 2013, Schlütersche Verlagsgesellschaft.

Bray RA, Gibson DI, Jones A: *Keys to the trematode* (vol 3), London, UK, 2002, CABI Publishing and The Natural History Museum.

Chochlova IG: *Acanthocephala of land vertebrates of the USSR fauna*, (in Russian), Moscow, 1985, Nauka.

Gibson DI, Jones A, Bray RA: *Keys to the trematode* (vol 1), London, 2002, CABI Publishing and The Natural History Museum.

Hiepe T, Jungmann R: Veterinärmedizinische Protozoologie. In Hiepe T, editor: *Lehrbuch der Parasitologie*, Jena, Germany, 1983, Gustav Fischer Verlag.

Hiepe T, Ribbeck R: Veterinärmedizinische Arachno-Entomologie. In Hiepe T, editor: *Lehrbuch der Parasitologie*, Jena, Germany, 1982, Gustav Fischer Verlag.

Hiepe T, Lucius R, Gottstein B: *Allgemeine parasitologie*, Stuttgart, 2006, Parey.

Hiepe T, Nickel S, Buchwalder R: Veterinärmedizinische Helminthologie. In Hiepe T, editor: *Lehrbuch der parasitologie*, Jena, Germany, 1985, Gustav Fischer Verlag.

Ioff IG, Mikulin MA, Skalon OI: *Key to the fleas of Central Asia and Kazakhstan*, (in Russian), Moscow, 1965, Medicina.

Jones A, Bray RA, Gibson DI: *Keys to the trematode* (vol 2), London, 2002, CABI Publishing and The Natural History Museum.

Khalil LF, Jones A, Bray RA: *Keys to the cestode parasites of vertebrates*, Eastbourne, UK, 2006, CPI Antony Rowe.

Kornjushin VV: *Fauna of Ukrain. 33: Davainoidea, Biuterinoidea, Paruterinoidea*, (in Russian), Kiev, 1989, Naukova Dumka.

Krone O: Endoparasites. In Bird DM, Bildstein KL, editors: *Raptor—research and management techniques*, Surrey, 2007, Hancock House Publishers.

Lane RP, Crosskey RW: *Medical insects and arachnids*, London, 1995, Chapman Hall.

Lehane MJ: *Biology of blood-sucking insects*, London, 1991, Harper Collins Academic.

Lindsey DS, Blagburn BL: Cryptosporidium. In Atkinson CT, Thomas NJ, Hunter DB, editors: *Parasitic diseases of wild birds*, Ames, IA, 2008, Wiley-Blackwell.

Pellerdy L: *Coccidia and coccidiosis*, Budapest, 1965, Akademiai Kiado.

Schnieder T: *Veterinärmedizinische parasitologie*, ed 6, Stuttgart, 2006, Parey.

Schöne R, Schmäschke R, Sachse M: Federmilben: http://www.federmilben.de/en/home.html, 2007.

Sonin MD: *Key to the Trematodes of fish-eating birds of the Palaearctic (Brachylaimidae, Clinostomidae, Cyclocoelidae, Fasciolidae, Notocotylidae, Plagiorchidae, Schistosomatidae)*, (in Russian), Moscow, 1985, Nauka.

Sonin MD: *Key to the Trematodes of fish-eating birds of the Palaearctic (Opisthorchiidae, Renicolidae, Strigeidae)*, (in Russian), Moscow, 1986, Nauka.

Sood ML: *Nematode parasites of birds from South Asia*, Lucknow, India, 2006, International Book Distribution.

Sreter T, Varga I: Cryptosporidiosis in birds—a review, *Vet Parasitol* 87:261–279, 2000.

Taylor MA, Coop RL, Wal RL: *Vet parasitol*, ed 3, Oxford, UK, 2007, Blackwell Publishing.

Weidner H, Sellenscho U: *Vorratsschädlinge und hausungeziefer*, ed 6, Heidelberg, 2003, Spektrum Akademischer Verla.

HEMOPARASITES

Michael A. Peirce

Blood parasites occur in nearly all species of birds, but those living in extreme climatic conditions and in areas where vectors are absent are

usually found to be infected only when transferred to other locations such as exhibits in zoologic collections or captive breeding programs or as pets. These birds are frequently totally susceptible to infection by a wide range of parasites, many of which may be pathogenic (Peirce, 1989).

The extent of mortality in wild populations caused by hemoparasites is difficult to gauge. Unless there is a noticeable die-off in a specific population where a veterinary diagnosis confirms the involvement of hematozoa either as sole infection or as a component of concomitant infection with other disease agents, most sick or dead birds rapidly become prey to predators and scavengers. Thus nearly 90% of all records of mortality and pathogenicity caused by avian hematozoa have been described from domestic species (chickens, turkeys, ducks, and geese) and only 5% from free-living birds (Bennett *et al.*, 1993b).

With the exception of microfilariae (the immature stages of filarial worms) and *Aegyptianella*, most blood parasites are protozoa. Diagnosis is dependent on Giemsa-stained thin smears of peripheral blood, but for the more common genera, molecular techniques are being used more frequently (Perkins, 2014). Although most postmortem blood smears are of little value taxonomically, they are still valuable in identifying parasites, at least to the generic level, and in correlating endogenous stages that may be found on histopathology examination.

PLASMODIUM SPECIES

The genus *Plasmodium* (Figs. 14-133 to 14-139) is an apicomplexan parasite closely related to *Haemoproteus* and *Leucocytozoon*. Bennett *et al* (1993a) recorded approximately 34 species of *Plasmodium* from birds, but this number has nearly doubled based on the application of molecular techniques (Perkins, 2014). These species can be grouped into five subgenera (Table 14-24) according to specific morphologic characteristics such as size and shape of gametocytes and schizonts. More recently an additional subgenus, *Bennettinia*, has been established to accommodate the single species *P. juxtanucleare* (Perkins, 2014).

With the one known exception of *Plasmodioides*, which is transmitted by *Anopheles* spp., all other subgenera and species are transmitted by culicine mosquitoes. When an infected vector bites a new host, parasite sporozoites are passed into the blood, where they eventually reach the liver and undergo development into preerythrocytic schizonts. These produce merozoites, which enter the erythrocytes and develop into macrogametocytes (female) or microgametocytes (male) or segmenters (asexual schizonts). All stages contain melanin pigment granules. The merozoites produced from asexual schizonts repeat the erythrocytic cycle. The intraerythrocytic merogony cycles continue indefinitely unless the host's immune system or death intervenes. Thus there is a potential for persistence of infection with frequent relapses. Second-generation and subsequent generations of exoerythrocytic schizonts may occur in tissues other than the liver. In early infections, only trophozoites and schizonts may occur in erythrocytes, so specific diagnosis is difficult. Likewise, in established infections only gametocytes may be present and if these are of a species with an elongated form they can easily be confused with those of *Haemoproteus*. In these situations molecular techniques may provide a more definitive diagnosis.

Some avian species of *Plasmodium* are known to occur only in the type host from which the parasite was originally described. Other *Plasmodium* spp. are found in a wide range of hosts and families and often the morphology is markedly different. This frequently makes a definitive diagnosis to species level difficult unless tissue stages and vectors are also known.

TABLE 14-24 Key to the Avian Subgenera of *Plasmodium*

1a	Parasites lacking pigment; gametocytes and schizonts large; mature parasites displace host cell nucleus; present only in circulating leukocytes	*Plasmodioides*
1b	Parasites with pigment	2
2a	Gametocytes round or nearly so; mature parasites typically displace host cell nucleus toward pole	*Haemamoeba*
2b	Gametocytes elongate; mature forms do not displace host cell nucleus toward pole	3
3a	Schizonts present in circulating erythrocyte precursors, not in mature erythrocytes	*Huffia*
3b	Schizonts in mature erythrocytes, not in erythrocyte precursors	4
4a	Erythrocytic schizonts generally larger than erythrocyte nucleus and contain noticeable amount of cytoplasm	*Giovannolaia*
4b	Erythrocytic schizonts smaller than erythrocyte nucleus; without noticeable cytoplasm	*Novyella*

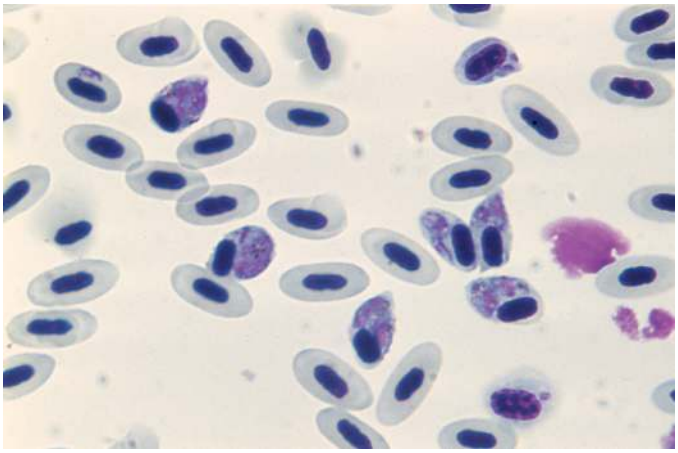


FIGURE 14-134 *Plasmodium* (*Haemamoeba*) *relictum* from a house sparrow (*Passer domesticus*).

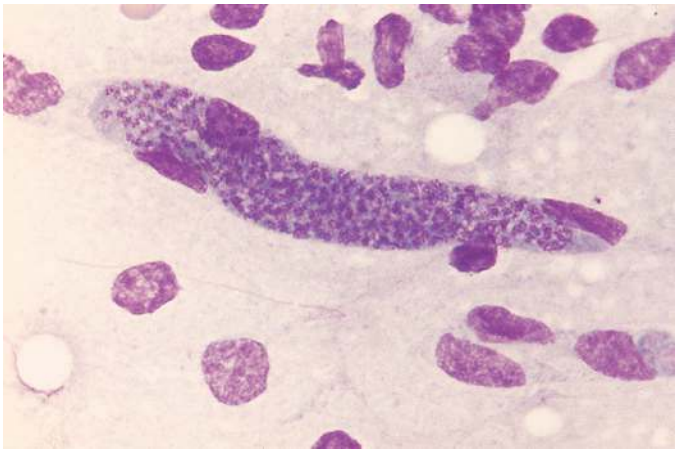


FIGURE 14-135 *Plasmodium* (*Haemamoeba*) *gallinaceum* schizont in a brain smear from a chicken (*Gallus domesticus*).

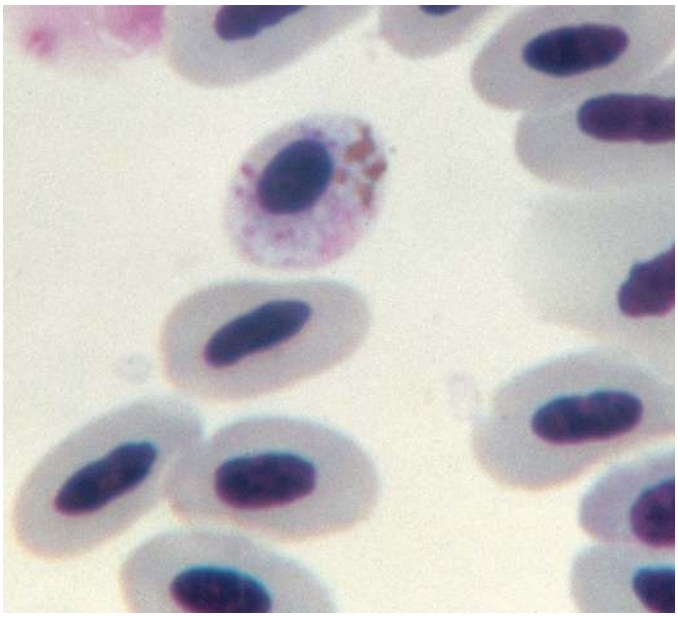


FIGURE 14-136 *Plasmodium* (*Giovannolaia*) *circumflexum* microgametocyte.

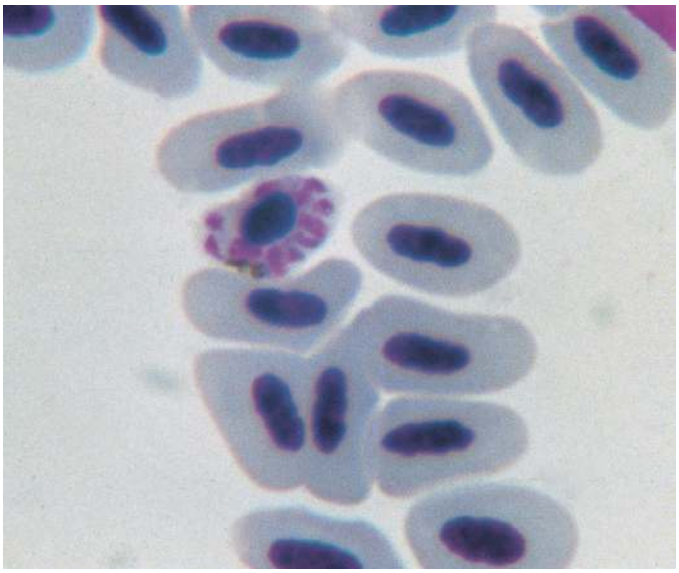


FIGURE 14-137 *Plasmodium* (*Giovannolaia*) *circumflexum* schizont.

Specific clinical signs of *Plasmodium* infection are lacking, but listlessness, lethargy, and anemia may be indicators of the disease. Generally *Plasmodium* spp. are more pathogenic in domestic birds: *P. gallinaceum* in chickens (frequent involvement of brain schizonts), *P. durae* in turkeys, and *P. circumflexum* in ducks and geese. The species that occurs most in free-living birds is *P. relictum*, which has been recorded in over 380 hosts from 74 families. This parasite and *P. elongatum* are frequent causes of mortality in penguins in zoological collections.

P. vaughani has been recorded from 270 hosts and is particularly common in passerine species, and *P. circumflexum* occurs in approximately 140 avian hosts. Other species of *Plasmodium* are less common and are considered to be of little clinical significance, with the possible exception of *P. juxtanucleare* in chickens.

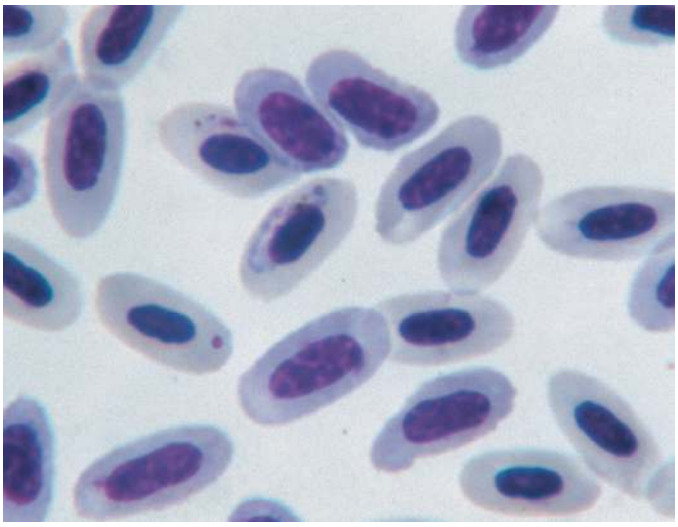


FIGURE 14-138 *Plasmodium* (*Novyella*) *rouxi* microgametocyte.

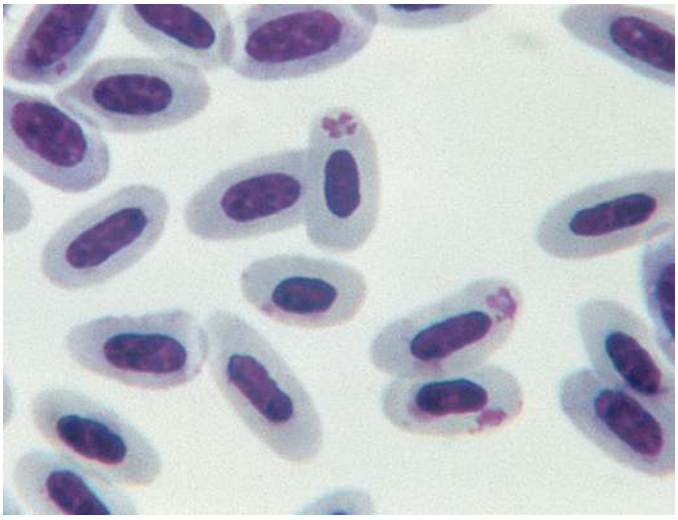


FIGURE 14-139 *Plasmodium* (*Novyella*) *rouxi* schizont.

Mixed infections with two species of *Plasmodium* are not uncommon, complicating diagnosis even further. The application of molecular techniques can often separate these species where morphology alone is insufficient. Xenodiagnosis, by blood inoculation into laboratory birds such as canaries, is possible for many *Plasmodium* spp. Color illustrations of the more common species can be found in Garnham (1966).

HAEMOPROTEUS SPECIES

Species of *Haemoproteus* (Figs. 14-140 to 14-145) differ from those of *Plasmodium* in that the erythrocytic stages produce only gametocytes. Macrogametocytes and microgametocytes can be differentiated in Giemsa-stained thin blood smears. Generally, the nucleus of macrogametocytes is more compact and the cytoplasm denser. Melanin pigment granules tend to be evenly distributed in macrogametocytes but more clustered in polar positions in microgametocytes. The pigment granules are similar to those of *Plasmodium* but are frequently larger. There are approximately 169 species of *Haemoproteus* (Peirce, 2005 and unpublished observations), the majority of which are host specific to

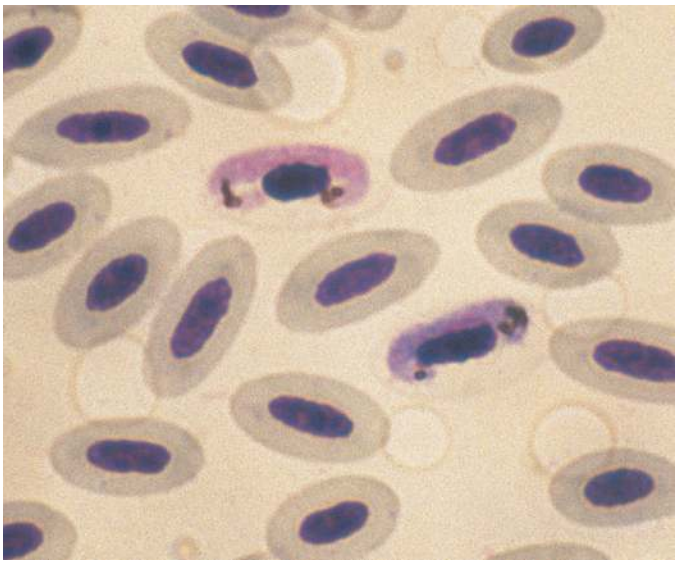


FIGURE 14-140 *Haemoproteus psittaci* macrogametocyte and microgametocyte (microhalteridial haemoproteid) from African grey parrot (*Psittacus erithacus*).

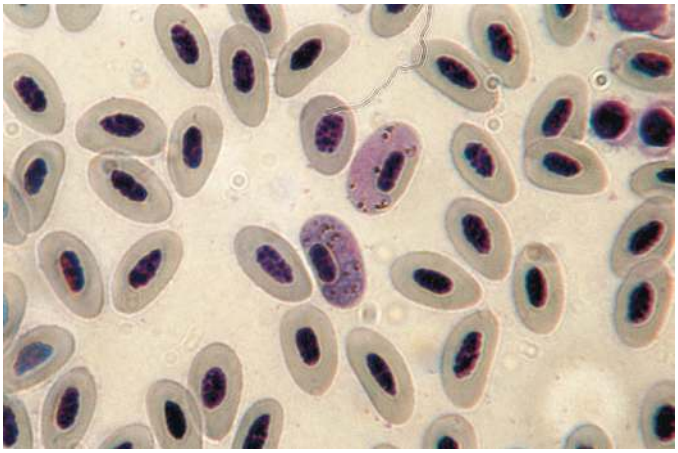


FIGURE 14-141 *Haemoproteus syrnii* macrogametocyte and microgametocyte (halteridial haemoproteid). Parasite of Strigidae.

the family level and can be divided into five distinct morphologic forms (Bennett and Peirce, 1988): microhalteridial, halteridial, circumnuclear, rhabdosomal, and discosomal. Halteridial species are most common and account for the largest group (65%). The vectors are known for only 12 species, and 10 of these are transmitted by *Culicoides* spp. Only two are transmitted by hippoboscids. Schizonts of *Haemoproteus* occur in a wide range of organs but are usually most frequent in the lung and liver tissue. Few species of *Haemoproteus* are known to be pathogenic; *H. meleagridis* in turkeys, *H. nettionis* in ducks and geese, and *H. columbae* in pigeons and doves are the general exceptions. No definitive clinical signs of haemoproteid infection are applicable, but the general observations pertaining to *Plasmodium* infections also can be considered to apply to *Haemoproteus.* A clinically sick emerald-spotted wood dove in Zambia with mixed infection with *H. columbae* and *Leucocytozoon marchouxi* was 25% under average weight (Peirce 1984).

Mixed infections with *Haemoproteus* and *Plasmodium* are common and multiple invasion of erythrocytes can occur. Differential diagnosis

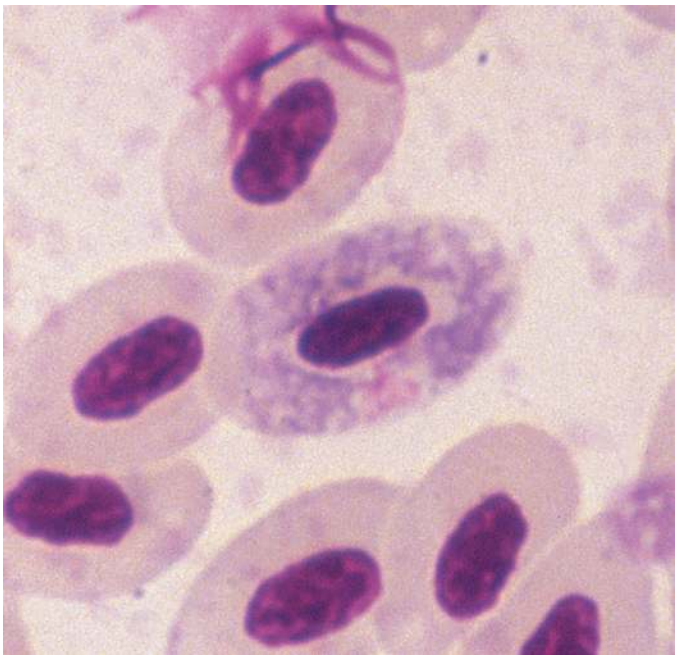


FIGURE 14-142 *Haemoproteus handai* macrogametocyte (circumnuclear haemoproteid). Parasite of psittacines.

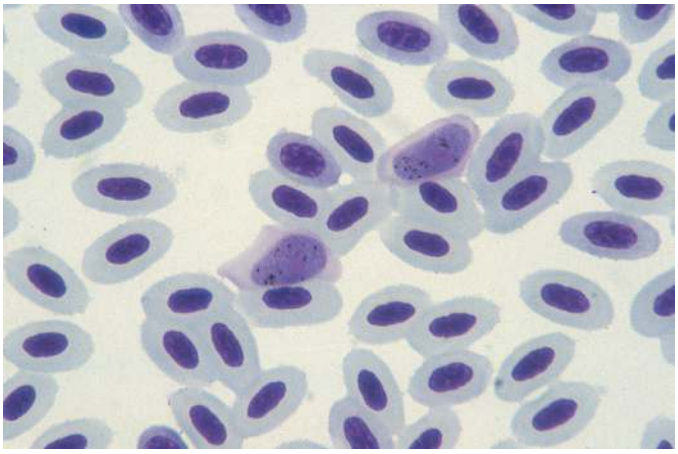


FIGURE 14-143 *Haemoproteus enucleator.* Two macrogametocytes (rhabdosomal haemoproteid), from a pygmy kingfisher (*Ispidina picta*).

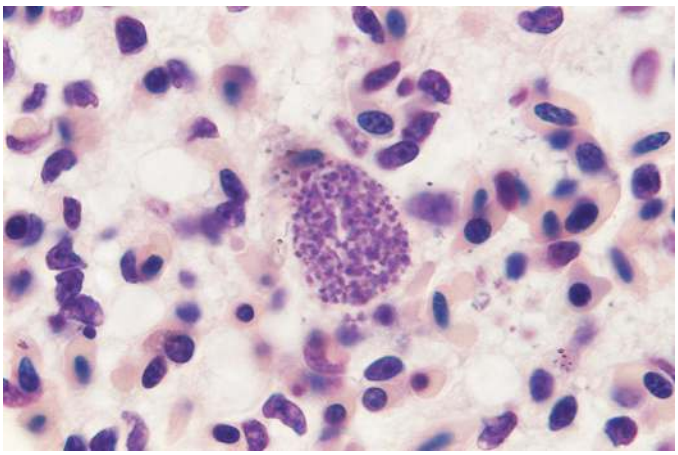


FIGURE 14-144 Postmortem section of lung from a crowned crane (*Balearica pavonina gibbericeps*) showing schizont of the microhalteridial haemoproteid *Haemoproteus balearicae.*

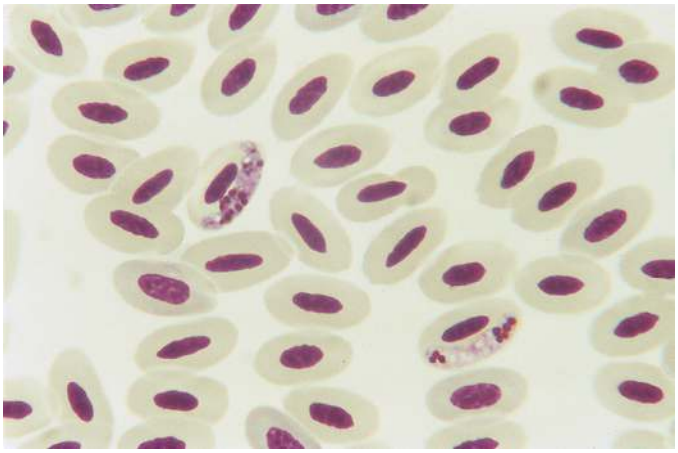


FIGURE 14-145 Immature macrogametocyte and microgametocyte of *Haemoproteus columbae* showing distinctive large, purple volutin granules in a Cape turtle dove (*Streptopelia capicola*).

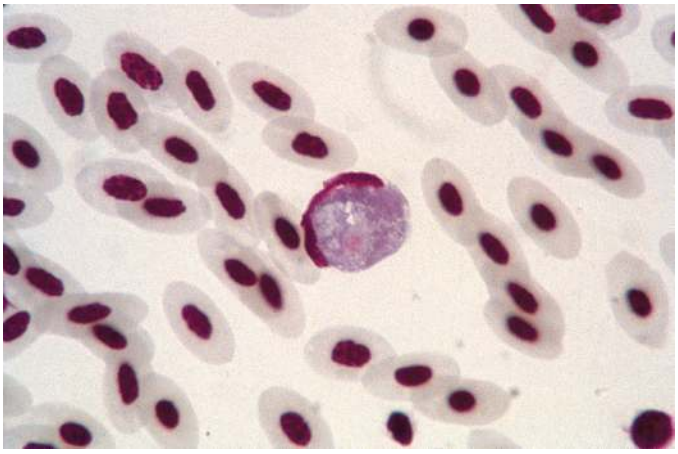


FIGURE 14-146 *Leucocytozoon neavei* macrogametocyte (round morph) from yellow-necked spurfowl (*Francolinus leucoscepus*).

is required but may not always be possible when parasitemias are low. Some species of *Haemoproteus* (e.g., *H. enucleator*) may completely expel the host cell (erythrocyte) nucleus, similar to some *Plasmodium* spp. (e.g., *P. relictum*). Some species of *Haemoproteus* contain reddish-purple volutin granules, which can often be quite large. The exact significance of these granules is not clear but they may represent strain differences. They have often been mistaken for schizonts of *Plasmodium*, from which differential diagnosis is required.

LEUCOCYTOZOON SPECIES

Species of *Leucocytozoon* (Figs. 14-146 to 14-150) have a life cycle similar to that of *Haemoproteus.* In some species, the gametocytes develop in erythrocytes, in others in monocytes and lymphocytes. Although the macrogametocytes and microgametocytes of *Leucocytozoon* can be differentiated on similar characteristics from those of *Plasmodium* and *Haemoproteus*, they lack the presence of melanin pigment seen in the other two genera, although small reddish granules are often present.

As the gametocytes develop they cause considerable distortion of the host cell. Two distinct morphologic forms occur: round and elongated (fusiform). In some species, only one type of morph occurs, but

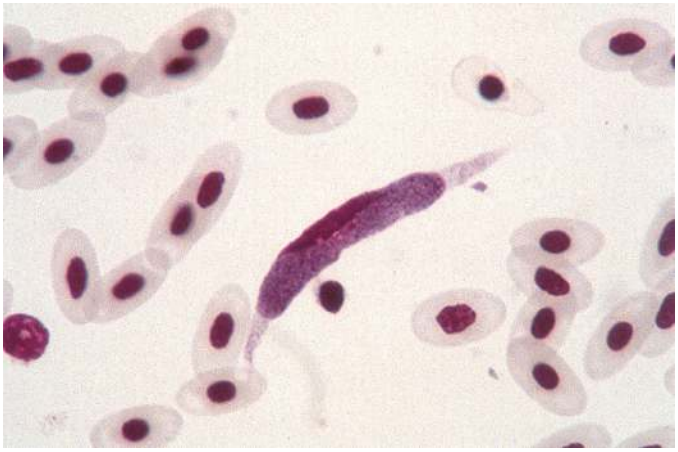


FIGURE 14-147 *Leucocytozoon neavei* macrogametocyte (fusiform morph) from yellow-necked spurfowl.

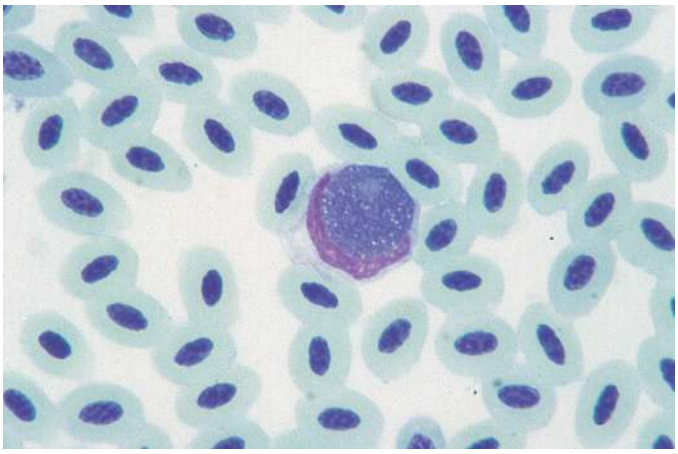


FIGURE 14-148 *Leucocytozoon marchouxi* macrogametocyte from pink pigeon (*Columba mayeri*).

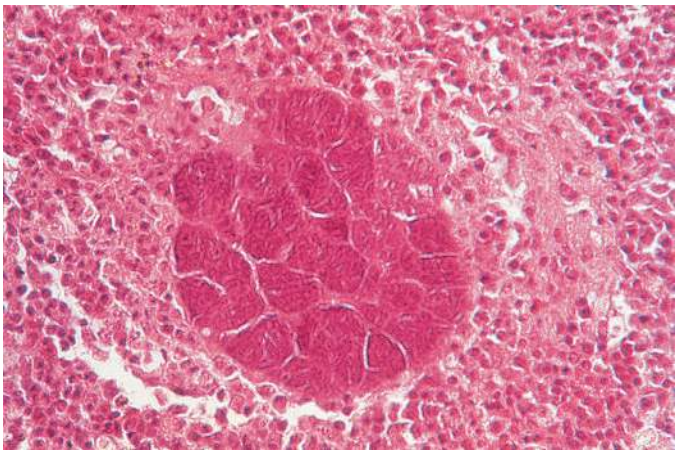


FIGURE 14-149 *Leucocytozoon marchouxi* megaloschizont in spleen of pink pigeon.

in others both may occur. In species with both morphs, the round ones arise from the first-generation hepatic schizonts and the fusiform morphs from second-generation megaloschizonts (Fallis *et al.*, 1974). The precise significance of megaloschizonts is not known, because they also occur in species with only round morphs.

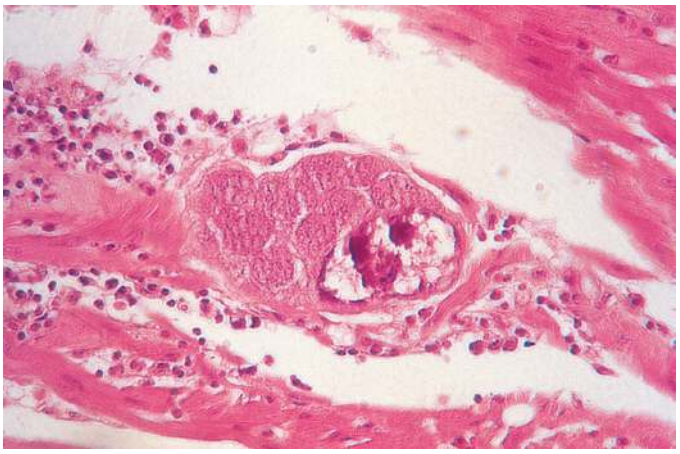


FIGURE 14-150 *Leucocytozoon marchouxi* megaloschizont in heart muscle of pink pigeon.

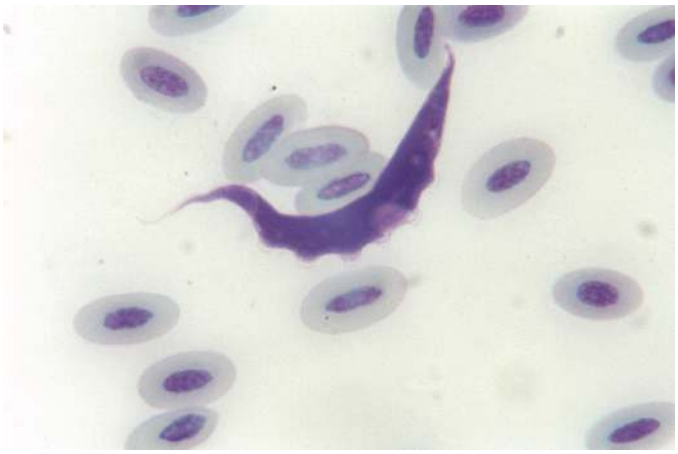


FIGURE 14-151 *Trypanosoma corvi.*

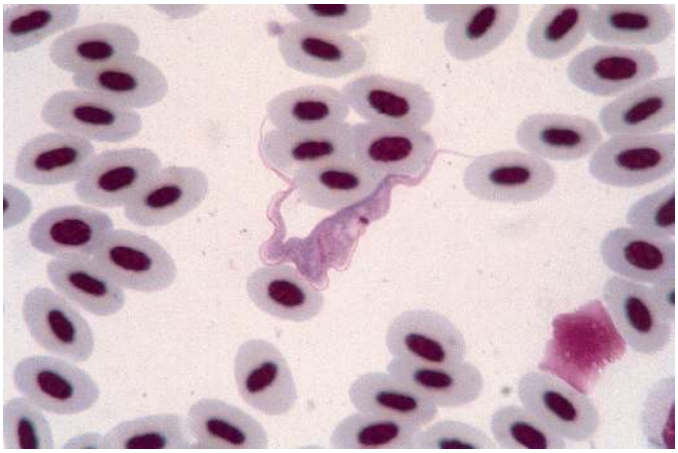


FIGURE 14-152 *Trypanosoma bouffardi.*

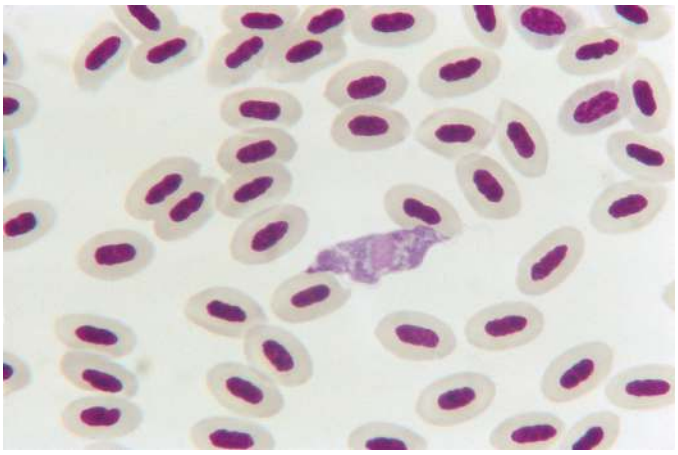


FIGURE 14-153 *Trypanosoma everetti.*

There are approximately 86 species of *Leucocytozoon* (Peirce, 2005 and unpublished observations), most of which are host specific at least to the family level and, with the exception of *L.* (*Akiba*) *caulleryi*, which is transmitted by *Culicoides*, the vectors are all simuliids. Pathogenicity caused by *Leucocytozoon* is generally more common than with *Haemoproteus.* High levels of mortality have been recorded with *L.* (*Akiba*) *caulleryi* in chickens, *L. smithi* in turkeys, and *L. simondi* in both domestic and free-living ducks and geese. Until recently, there was little evidence of pathogenicity with *L. marchouxi* in pigeons and doves (Peirce, 1984), but recent studies have shown this species to be pathogenic in the endangered pink pigeon (*Columba mayeri*) in Mauritius, with recorded mortality in squabs. It has also been shown to be a species producing megaloschizonts, although only round morphs occur. There is also some evidence that *L. danilewskyi* may be pathogenic in certain species of owls.

The endogenous stages (schizonts) occur in nearly all organs and muscle, causing severe damage and necrosis (Peirce *et al.*, 2004). It is the degree of tissue damage that causes mortality rather than the gametocyte parasitemia. Very high levels of parasitemia have been observed in some hosts without any obvious clinical signs of disease. Outward clinical signs of the disease are similar to those of *Plasmodium* together with weight loss, as birds become too sick to feed.

Mixed infections with *Plasmodium* and/or *Haemoproteus* are common and differential diagnosis is required. It is often in cases of concomitant infection that the usually benign single infections may become pathogenic.

TRYPANOSOMA SPECIES

Trypanosomes (Figs. 14-151 to 14-153) are very pleomorphic and of the approximately 98 species described from birds probably no more than 8 to 10 are valid. They are transmitted by a variety of vectors, including mosquitoes, hippoboscids, simuliids, and mites. Very high parasitemias are not uncommon, but there is no evidence to indicate that any species is pathogenic. There is very little evidence of host specificity. Detection by examination of Giemsa-stained thin blood smears will only illustrate trypanosomes when present in the host at reasonable levels. A more reliable method is to use the microhematocrit tube and prepare a smear from the buffy coat formed after centrifuging. Trypanosomes are frequently observed in mixed infections with other hematozoa.

HEPATOZOON SPECIES

There are 17 species of *Hepatozoon* (Figs. 14-154 and 14-155) described from birds (Bennett *et al.*, 1992; Peirce, 2005). The parasites normally invade monocytes but occasionally lymphocytes may be targeted. The full life cycle is unknown for any of the avian species but an argasid tick and a flea have been shown as probable vectors for *H. atticorae* of

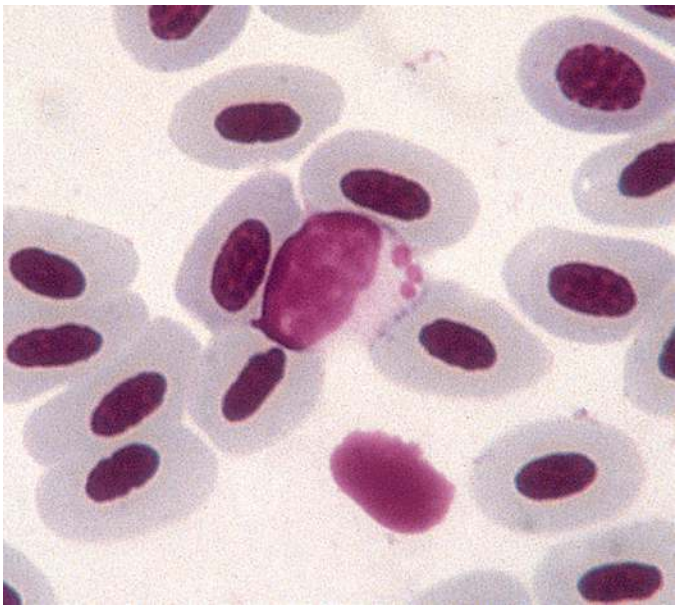


FIGURE 14-154 *Hepatozoon estrildus* (early stage) from blue waxbill (*Uraeginthus angolensis*).

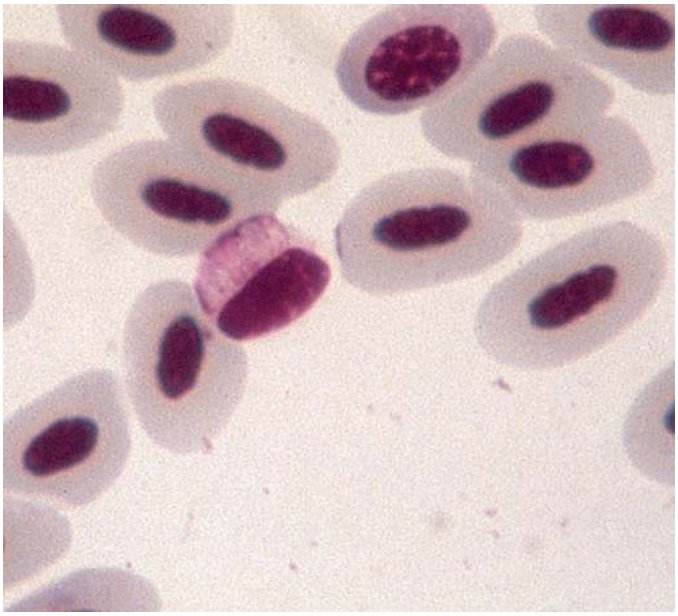


FIGURE 14-155 *Hepatozoon estrildus* mature parasite from blue waxbill (*Uraeginthus angolensis*).

swallows. Ixodid ticks and mites and other arthropods may also be involved in transmission. In general none of the avian species are considered to be pathogenic with the exception of *H. kiwii.* The genus probably has a far more common distribution than current records suggest, but is most likely overlooked in screening blood smears. Known hosts range from the tropics to Antarctica.

BABESIA SPECIES

There are approximately 16 species described of the avian piroplasm *Babesia* (Fig. 14-156). The group was reviewed by Peirce (2000). The parasites invade erythrocytes, where the trophozoites multiply by

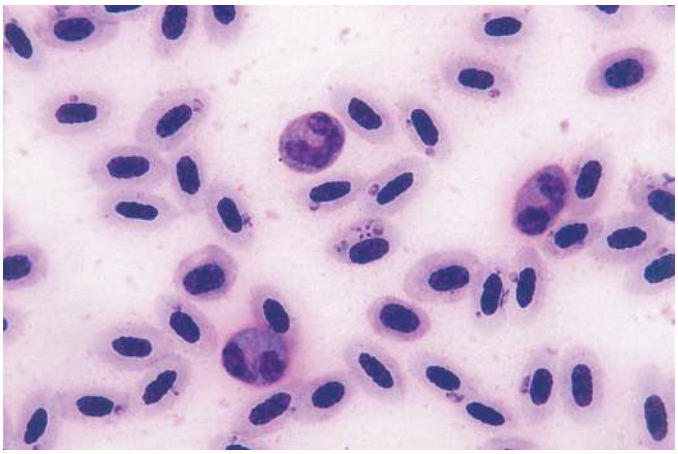


FIGURE 14-156 *Babesia shortii* from kestrel (*Falco tinnunculus*).

binary fission forming pairs or by schizogony forming tetrads. Until recently only *B. shortti* occurring in Falconiformes was thought to be pathogenic (Samour and Peirce, 1996), but *B. kiwiensis* from *Apterygidae* also is pathogenic in kiwi chicks (Peirce *et al.*, 2003). The disease in birds follows a similar pattern to that in mammals, where multiple invasion of erythrocytes leads to destruction of cells, anemia, jaundice, and death.

None of the vectors of avian *Babesia* spp. are known but they are assumed to be ixodid ticks, although an argasid tick of the genus *Ornithodoros* has been suggested as a possible vector of *B. shortti* in prairie falcons.

Removal of ticks from birds and prevention of reinfestation is required to control *Babesia* infections. Differential diagnosis between *Babesia* and the trophozoite stages of *Plasmodium* and early gametocytes of *Haemoproteus* is required. The tetrads of *Babesia* are morphologically similar to the small schizonts of some *Plasmodium* spp. (e.g., *P. rouxi*). The main differential characteristics of *Babesia* are the absence of melanin pigment granules and the distinctive white vacuole.

The prevalence of *Babesia* in birds is probably greater than records would suggest. The small number of records is no doubt from misdiagnosis of *Plasmodium* or *Haemoproteus.* The occurrence of mixed infections should also be considered. Experience with *B. shortti* infections in falcons indicates that an early diagnosis is required if appropriate chemotherapy is to be successful.

ATOXOPLASMA SPECIES

The life cycle and taxonomic position of *Atoxoplasma* parasites (Fig. 14-157) has been the subject of much discussion and confusion. It is now generally accepted that the genus is a member of the Eimeriidae family and is closely related to *Isospora.* Separation of the two genera requires molecular techniques. Extraintestinal stages result from the ingestion of *Isospora*-like oocysts, where the parasites invade mononuclear leukocytes. The parasites are particularly common in passerine birds and about 19 species have been described.

High parasitemias are common in nestlings and young birds but are rarely pathogenic, because most infections are subclinical. Recent studies, however, have shown that, with some species, mortality can occur in adult birds, particularly when kept in captivity (McGill *et al.*, 2010). Postmortem Giemsa-stained impression smears of liver and spleen (Fig. 14-158) frequently reveal large numbers of parasites. There are a few reports attributed to *Atoxoplasma* sp. where specific clinical signs have been described. The parasite has been implicated as a cause

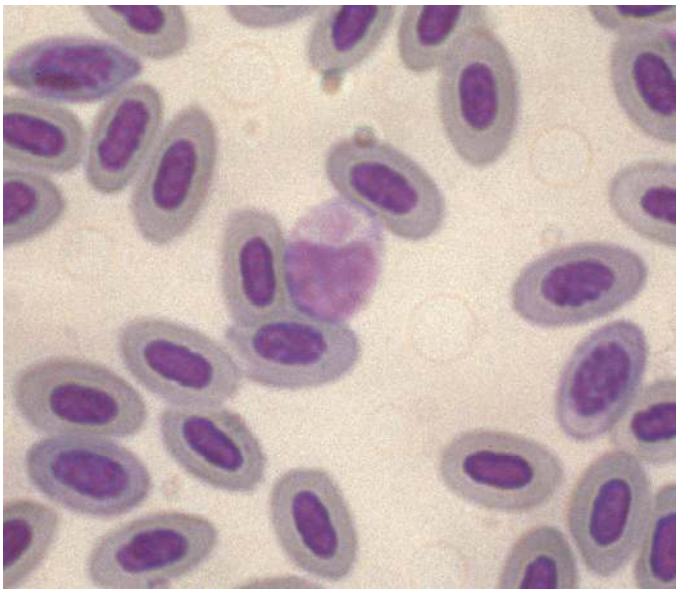


FIGURE 14-157 *Atoxoplasma* from willow warbler (*Phylloscopus trochilus*).

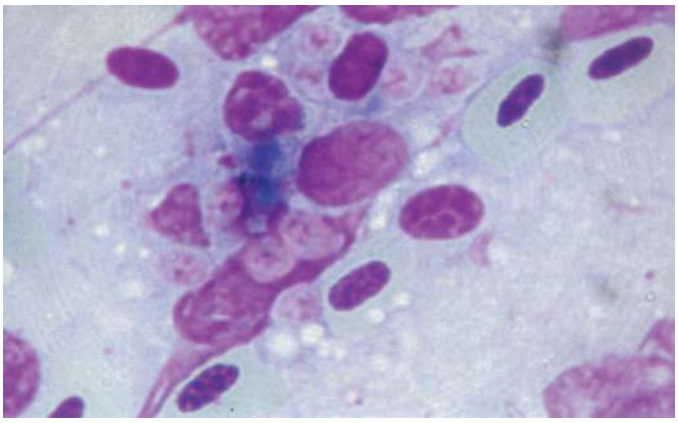


FIGURE 14-158 *Atoxoplasma* in spleen impression smear from superb starling (*Spreo superbus*).

of "going light" in greenfinches (Cooper *et al.*, 1989) and as a cause of inappetence and "fluffed-up feathering" in 4- to 8-week-old bullfinches. Histopathology of one bird with enlarged liver and spleen revealed various pathologic changes including encapsulated granulomata with necrotic areas and general hepatic congestion (McNamee *et al.*, 1995). Preventive measures recommended are good aviary hygiene, especially frequent replacement of drinking water and bathing bowls to prevent ingestion of sporulated oocysts.

AEGYPTIANELLA SPECIES

Parasites of the genus *Aegyptianella* (Fig. 14-159) are rickettsias with close affinities to *Anaplasma*, *Eperythrozoon*, etc., all of which are grouped in the family Anaplasmataceae. In domestic poultry *A. pullorum* is pathogenic. The intraerythrocytic parasites cause severe anemia, jaundice, and frequently death. The vector of *A. pullorum* is the argasid tick *Argas persicus* and the control of this tick is essential in preventing exposure of chickens to the disease. Parasites resembling *A. pullorum* have been recorded from turkeys, ducks, geese, and other hosts, including psittacines from South America and East Africa. Many

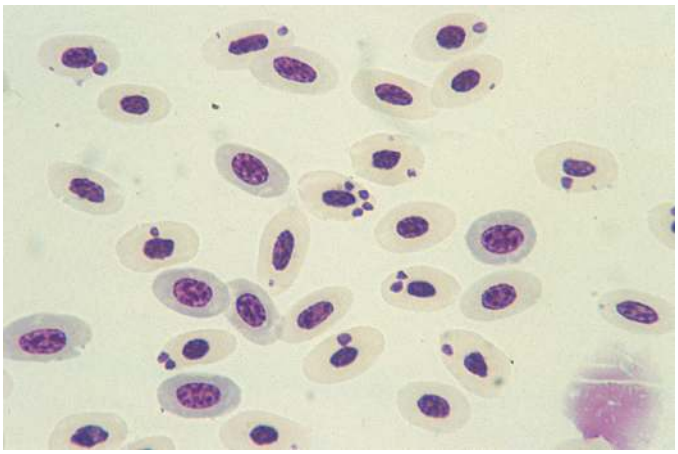


FIGURE 14-159 *Aegyptianella pullorum.*

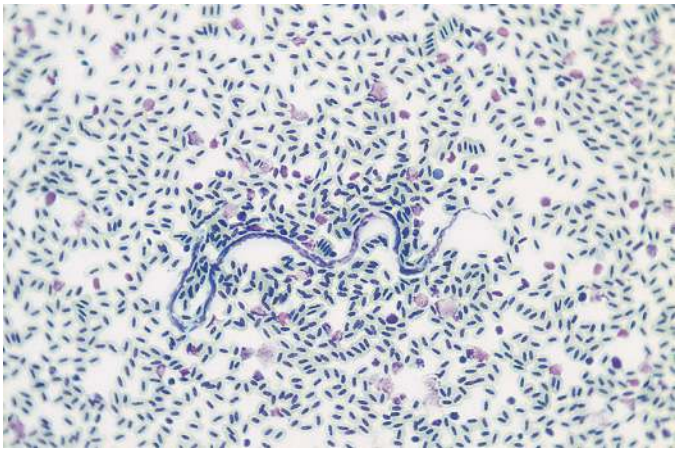


FIGURE 14-160 Microfilaria from Senegal parakeet (*Psittacula krameri*).

of these records are probably of closely related species. *Aegyptianella botuliformis* has been described from guinea fowl in South Africa (Huchzermeyer *et al.*, 1992) and more recently *A. minutus* from passerine birds in Malaysia (Peirce, 1999).

Differential diagnosis is required to separate *Aegyptianella* from early trophozoites and gametocytes of *Plasmodium* and *Haemoproteus*, respectively. The absence of melanin pigment is usually indicative of *Aegyptianella* and in higher parasitemias the morphology is more varied.

MICROFILARIAE

Microfilariae are the larval stages of filarial worms, of which there are many genera and species occurring in birds. The morphology varies considerably from short and stumpy to long and thin (Fig. 14-160). The larvae usually demonstrate a circadian rhythm coinciding with vector activity. During this time large numbers may be present in peripheral blood. The majority of species appear to be benign but, where concomitant infection with other parasites or diseases occurs, large numbers of larvae may cause some degree of morbidity.

DISCUSSION

The accurate diagnosis of blood parasites requires good quality, Giemsa-stained, thin blood smears. Smears prepared postmortem are useful in indicating the presence of parasites, even if this is restricted

to the generic level, only because of the morphologic distortion that rapidly occurs following death. If a bird has been dead for a few hours, it is still possible to produce slides by opening up the heart (usually the last area to clot) and scraping the blood clot with the edge of a microscope slide, which will release sufficient blood cells to make a reasonable thin smear. Without the back-up of blood smears it is often difficult to correlate tissue stages of parasites present on histopathology examination. The use of molecular techniques is becoming more frequent as an adjunct to morphologic diagnosis.

Prevention and control of many blood parasites is difficult and in some cases is almost impossible. Parasites such as *Babesia*, *Aegyptianella*, and probably *Hepatozoon* can be controlled to some degree by eliminating tick vectors by use of safe acaricides. However, those parasites transmitted by flying insects, especially hemosporidia, are more difficult to control unless the birds are maintained in fly-proof cages or aviaries, which is both costly and inconvenient. An added problem is that this approach renders birds reared under such conditions as totally susceptible to infection if released into the wild as part of a captive breeding or rehabilitation program.

No specific recommendations for chemotherapy of infections have been included, for several reasons. First, there are few, if any, drugs licensed for use in exotic avian hosts. Many birds can react severely to certain drugs and the treatment often causes more problems than the disease itself. A misdiagnosis can result in the administration of a drug that has no effect on the disease present. In cases where drug therapy is deemed necessary, appropriate chemotherapy and dose rates are at the discretion of the veterinarian responsible.

As seen from available evidence, the more pathogenic parasites generally occur in domestic species, and treatment of whole flocks is usually undertaken with established prophylactics for specific disease entities. In birds exposed to infections not encountered in their normal host range, the resulting disease picture can be totally different. For example, penguins may die of *Plasmodium* infection without the detectable presence of any erythrocytic stages, because the damage is caused by exoerythrocytic schizonts in the tissues.

The majority of hemoparasites occurring in birds within their normal host range are usually benign and it is not exceptional to find infections with six or more different parasites in a single host without any signs of ill effect. Many parasites cause seasonal relapses, often associated with the onset of breeding in the avian host and an increase in vector availability. Such relapses in free-living birds do not appear to cause any significant problems. When such birds are suffering with other concomitant disease agents or are under stress, normally benign parasites may become pathogenic. These factors are particularly important in monitoring the disease status of endangered species as part of a captive breeding program and in pet or collection birds not bred in captivity.

The spectrum of blood parasites to which both domestic and free-living birds can be exposed is wide and varied. Traditionally, textbooks of avian medicine have concentrated on the postmortem histopathology changes often associated with specific parasitic diseases and their prevention and treatment. The emphasis in this section has been to present pointers to differential diagnosis of the peripheral blood forms to aid in the correct identification and subsequent treatment when necessary.

REFERENCES

Bennett GF, Peirce MA: Morphological form in the avian haemoproteidae and annotated checklist of the genus *Haemoproteus* Kruse 1890, *J Nat Hist* 22:1683–1696, 1988.

Bennett GF, Bishop MA, Peirce MA: Checklist of avian species of *Plasmodium* Marchiafava and Celli, 1885 (Apicomplexa) and their distribution by avian family and Wallacean life zones, *Syst Parasitol* 26:171–179, 1993a.

Bennett GF, Earle RA, Peirce MA: New species of avian *Hepatozoon* (Apicomplexa: Hemogregarinidae) and a re-description of *Hepatozoon neophrontis* (Todd and Wolbach, 1912) Wenyon, 1926, *Syst Parasitol* 23:183–193, 1992.

Bennett GF, Peirce MA, Ashford RW: Avian haematozoa: mortality and pathogenicity, *J Nat Hist* 27:993–1001, 1993b.

Cooper JE, Gschmeissner S, Greenwood AG: Atoxoplasma in greenfinches (*Carduelis chloris*) as a possible cause of "going light.", *Vet Rec* 124:343–344, 1989.

Fallis AM, Desser SS, Khan RA: On species of Leucocytozoon, *Adv Parasitol* 12:1–67, 1974.

Garnham PCC: *Malaria parasites and other haemosporidia*, Oxford, UK, 1966, Blackwell Science.

Huchzermeyer FW, Horak IG, Putterill JF, et al: Description of *Aegyptianella botuliformis* n. sp. (Rickettsiales: Anaplasmataceae) from the helmeted guineafowl, *Numida meleagris*, *Onderstepoort J Vet Res* 59:97–101, 1992.

McGill I, Feltrer Y, Jeffs C, et al: Isosporoid coccidiosis in translocated cirl buntings (*Emberiza cirlus*), *Vet Rec* 167:656–660, 2010.

McNamee P, Pennycott T, McConnell S: Clinical and pathological changes associated with *Atoxoplasma* in a captive bullfinch (*Pyrrhula pyrrhula*), *Vet Rec* 136:221–222, 1995.

Peirce MA: Weights of birds from Balmoral, Zambia, *Bull Br Ornithol Club* 104:84–85, 1984.

Peirce MA: The significance of avian haematozoa in conservation strategies. In Cooper JE, editor: *Disease and threatened birds*, 1989, ICBP Technical Publication.

Peirce MA: A new species of *Aegyptianella* from south-east Asia, *Vet Rec* 145:288, 1999.

Peirce MA: A taxonomic review of avian piroplasms of the genus *Babesia* Starcovici, 1893 (Apicomplexa: Piroplasmorida: Babesiidae), *J Nat Hist* 34:317–332, 2000.

Peirce MA: A checklist of the valid avian species of *Babesia* (Apicomplexa: Piroplasmorida), *Haemoproteus, Leucocytozoon* (Apicomplexa: Haemosporida), and *Hepatozoon* (Apicomplexa: Haemogregarinidae), *J Nat Hist* 39:3621–3632, 2005.

Peirce MA, Jakob-Hoff RM, Twentyman C: New species of haematozoa from Apterygidae in New Zealand, *J Nat Hist* 37:1797–1804, 2003.

Peirce MA, Lederer R, Adlard RD: Pathology associated with endogenous development of haematozoa in birds from southeast Queensland, *Avian Pathol* 33:445–450, 2004.

Perkins SL: Malaria's many mates: past, present, and future of the systematics of the order Haemosporida, *J Parasitol* 100:11–25, 2014.

Samour JH, Peirce MA: *Babesia shortti* infection in a saker falcon, *Vet Rec* 139:167–168, 1996.

PARASITIC DISEASES

Peter Coutteel, Peter Wencel

COCCIDIAL DISEASES

Definition

Protozoal parasitic diseases are caused in birds by coccidial parasites mostly belonging to the genera: *Eimeria*, *Isospora*, *Atoxoplasma*, *Caryospora*, *Sarcocystis*, *Frenkelia*, *Toxoplasma*, *Cryptosporidium*, and *Tyzzeria*, all belonging to the phylum Apicomplexa.

Synonyms

It is important to distinguish the terms coccidiosis and coccidiasis. The former describes clinical disease, and the latter refers to the infection without any discernible signs of a disease.

Etiologic Agent

Coccidial diseases, based on tissue tropism of developmental stages in the avian hosts, can be broadly divided into two groups (Table 14-25):

TABLE 14-25 Coccidial Diseases: Life Cycle, Avian Host, Sporulated Oocyst Morphology, Oocyst Shedding, Sporogony, Transmission, Affected Organs, and Diagnostic Findings

Genus	Life Cycle	Avian Host	Sporulated Oocyst Morphology	Oocyst Shedding	Sporogony	Transmission	Affected Organs	Diagnostic Findings
Atoxoplasma	Direct	Definitive	Two sporocysts, four sporozoites in each	In feces	Exogenous	Orally via sporulated oocysts	Small intestine, lungs, liver, spleen, heart, brain	Merozoites and meronts in mononuclear cells in blood and in affected organs, preferably together with gamonts and oocysts in small intestine
Caryospora	Direct or indirect	Definitive	One sporocyst with eight sporozoites	In feces	Exogenous	Orally via sporulated oocyst or caryocysts in prey	Small intestine	Oocysts and/or developmental stages in the small intestine mucosa
Cryptosporidium	Direct	Definitive	Four sporozoites, naked in oocyst	In feces, urine, respiratory secretions	Endogenous	Orally via sporulated oocysts, or autoinfection via thin-walled oocysts	GIT (from proventriculus to cloaca), respiratory tract, urinary tract	Very small oocysts, and/or developmental stages attached to apical surfaces of the epithelial cells (location according to species) in GIT, conjunctivae, respiratory tract, kidneys, ureters, bursa
Eimeria	Direct	Definitive	Four sporocysts, two sporozoites in each	In feces, urine	Mostly exogenous	Orally via sporulated oocysts	Duodenum, jejunum, ileum, ceca, kidneys, liver (single report)	Oocysts, and/or developmental stages in GIT (location and type of the lesions are often species specific) mucosa
Disseminated visceral coccidiosis	Direct	Definitive (cranes)	Four sporocysts, two sporozoites in each	In feces, respiratory secretions	Exogenous	Orally via sporulated oocysts	GIT, liver, spleen, lungs, heart, kidney, bursa, thymus, skin	Meronts in extraintestinal nodules, meronts and/or oocysts in lungs and GIT, merozoites free and in phagocytic cells in blood and in affected organs
Isospora	Direct	Definitive	Two sporocysts, four sporozoites in each	In feces	Exogenous	Orally via sporulated oocysts	Small intestine	Oocysts and/or developmental stages in the small intestine mucosa
Sarcocystis	Indirect	Definitive and/or intermediate	Two sporocysts, four sporozoites in each; oocyst wall is very fragile, thus intact oocysts are rarely seen	In feces, only in definitive hosts	Endogenous	Intermediate hosts: orally via sporulated oocysts Definitive hosts: orally via sarcocysts in prey	Lung, liver, spleen, kidney, brain, skeletal muscles, and other organs	Intermediate hosts : meronts in endothelial cells in various organs, sarcocysts in skeletal muscles Definitive hosts: sporulated oocysts or free sporocysts in feces and/or developmental stages in the small intestinal mucosa
Toxoplasma	Indirect	Intermediate	None	No oocysts are shed by birds	None	Orally via sporulated oocysts (cats are the source of oocysts) or tachyzoites/bradyzoites in prey	Lungs, liver, spleen, kidney, brain, eyes, heart, and other organs	Tachyzoites (free or inside mononuclear cells) and/or bradyzoites in tissue cysts

GIT, Gastrointestinal tract.

FIGURE 14-161 Swollen abdomen, intestinal coccidiosis, goldfinch (*Carduelis carduelis*).

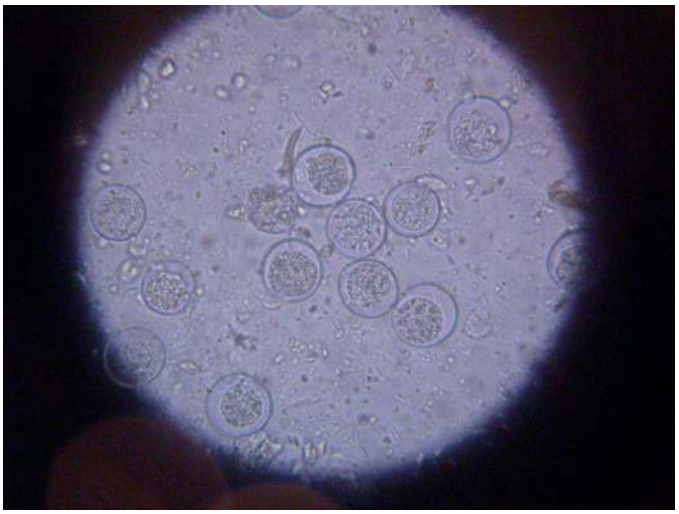


FIGURE 14-162 Unsporulated oocysts in fecal sample, goldfinch (*Carduelis carduelis*), ×400.

- The first group includes coccidia in which the entire life cycle is confined to the intestine i.e., the majority of *Eimeria*, *Isospora*, and *Caryospora.*
- The second group contains species in which part of the life cycle (all or part of merogony) is located in various extraintestinal locations (some *Eimeria* and *Sarcocystis* species, *Toxoplasma*, and all *Atoxoplasma*; Fig. 14-161). Cryptosporidia may reproduce in epithelial cells lining gastrointestinal (GIT), urogenital, and/or respiratory tract (location depends on the parasite species).

Sporulated coccidian oocysts differ in their size and morphology (Fig. 14-162). The detailed measurements, number of sporocysts and sporozoites per oocyst, together with their structural features have been used for decades for species determination. This is, however, seldom used in clinical practice. Currently with the advent of molecular analysis methods, many coccidia species can be specifically identified using polymerase chain reaction.

Although coccidia are widespread among birds worldwide, the best-studied avian coccidia belong to *Eimeria* spp., which cause disease in poultry, generating very high financial losses to the industry each year (Figs. 14-163 and 14-164). Studies on poultry coccidiosis provide a wealth of information about the biology, pathology, immunity, and treatment of avian coccidiosis in general. Current methods of coccidiosis prevention in poultry includes use of highly advanced techniques such as subunit or *in ovo* vaccination with live attenuated vaccine against several economically important species of *Eimeria.*

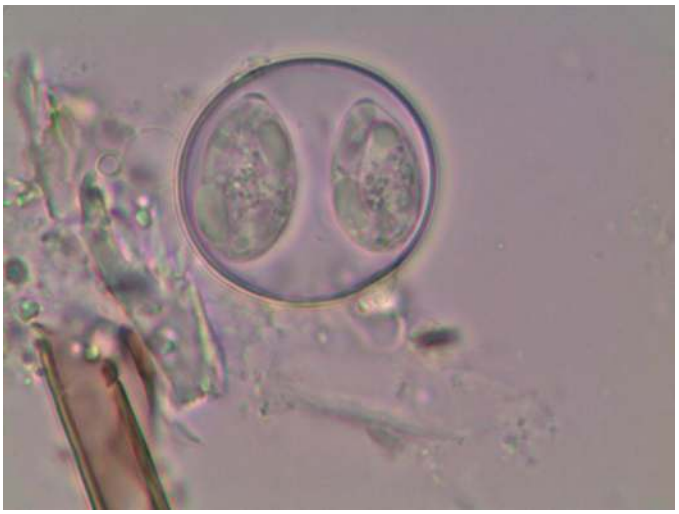


FIGURE 14-163 Sporulated *Isospora* spp. oocyst, showing two sporocysts, canary (*Serinus canaria*), ×1000.

Life Cycle

The life cycle of typical intestinal monoxenous coccidia (i.e., *Eimeria*) can be divided into three steps: sporogony, schizogony, and gametogony. The average life cycle of intestinal coccidia takes about 7 days to complete and in contrast to other pathogens (bacteria, viruses), it has a self-limiting nature. There is no known cross-immunity between different species of coccidia, i.e., infection with one species of *Eimeria* does not protect against infection and diseases caused by another *Eimeria* spp.

Distribution

Distribution is worldwide.

Susceptible Species

- These include 196 species of *Eimeria* in approximately 9464 avian species belonging to 27 orders (Struthioniformes, Casuariiformes, Rheiformes, Tinamiformes, Sphenisciformes, Gaviiformes, Podicipediformes, Procellariiformes, Pelecaniformes, Ciconiiformes, Phoenicopteriformes, Anseriformes, Falconiformes, Galliformes, Gruiformes, Charadriiformes, Columbiformes, Psittaciformes, Cuculiformes, Strigiformes, Caprimulgiformes, Apodiformes, Coliformes, Trogoniformes, Coraciiformes, Piciformes, and Passeriformes).
- 140 enteric *Isospora* species in various avian hosts.
- 150 species of *Caryospora* exist worldwide, mostly in raptors.
- Toxoplasma has been reported in orders Anseriformes, Falconiformes, Galliformes, Gruiformes, Charadriiformes, Columbiformes, Psittaciformes, Strigiformes, and Passeriformes.
- 12 species of *Sarcocystis* may use birds as definitive hosts (mostly Falconiformes and Strigiformes), 22 species as an intermediate hosts (Ciconiiformes, Anseriformes, Falconiformes, Galliformes, Gruiformes, Columbiformes, Psittaciformes, Coliformes, Piciformes, and Passeriformes), and at least two as both definitive and intermediate hosts.

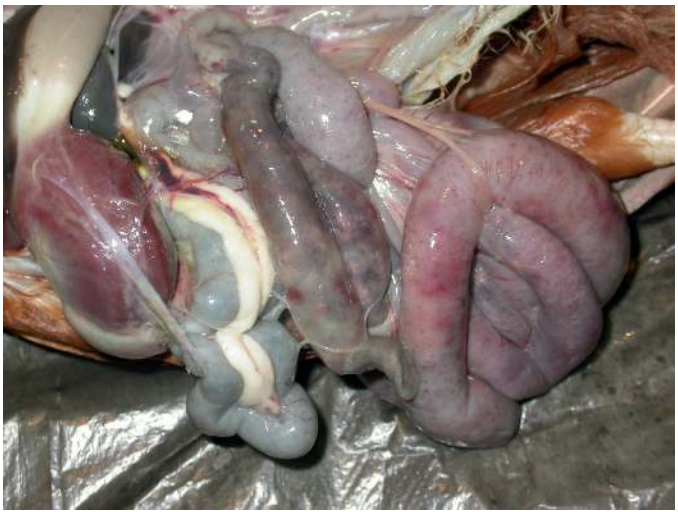


FIGURE 14-164 *Eimeria necatrix,* so called "salt and pepper" lesions in the small intestine of the backyard chicken (*Gallus domesticus*). Black and white spots (plaques and petechiae) are associated with schizogony (second-generation schizonts). In this species, no oocysts but characteristic clusters of large schizonts will be found in the microscopic preparation of the mucosa scrapings from the small intestine.

Clinical Symptoms

Coccidiosis is mostly the disease of young and immature birds in captivity, and is rather strictly host specific, i.e., coccidiosis in pigeons will not spread on fowl and reverse. The most commonly observed clinical signs typical for the specific diseases are listed below.

Atoxoplasmosis

See relevant section.

Cryptosporidiosis

- Nonbloody diarrhea
- Weakness
- Dyspnea
- Swelling of facial region
- Conjunctivitis
- Deaths
- Shedding of sporulated oocysts (very small, approximately 7 to 10 µm, resembling yeast cells)
- Clinical signs can be missing or only transient

Intestinal and Renal Coccidia (*Eimeria, Isospora,* and *Caryospora*)

- Loss of appetite, loss of weight, loss of coordination
- Diarrhea (often with blood)
- Enlarged abdomen (dilatation of intestines)
- Ruffled feathers
- Shedding of nonsporulated oocysts (size and shape variable from small, i.e., *E. mitis*, approximately 15 × 14 µm, to large, *E. maxima*, approximately 30 × 20µm)
- Deaths

Disseminated Coccidiosis in Cranes (*Eimeria reichenowi* and *E. gruis*)

- Emaciation
- Diarrhea
- Oral granulomas
- Shedding of nonsporulated oocysts (*E. reichenowi*, round, measuring approximately 17.8 × 15.3 µm, *E. gruis*, pyriform, measuring approximately 18 × 11.4 µm).
- Deaths

Sarcocystosis

- Sudden deaths (especially in Old World psittacines)
- Weakness
- Dyspnea
- Neurologic signs (in pigeons they may resemble infection with pigeon paramyxovirus type 1)
- Muscular signs
- Blood in trachea and oral cavity
- There may be no clinical symptoms of infection preceding death
- Only definitive hosts shed sporulated oocysts (intact oocysts are rarely seen, because of their fragility; the main findings are small, yeast cell-sized sporozoites lying free in feces).
- Birds may be infected asymptomatically

Toxoplasmosis

- Emaciation
- Diarrhea
- Dyspnea
- Ruffled feathers
- Blindness and neurologic signs (especially in canaries)
- No oocysts are shed
- Birds may be infected asymptomatically
- There may be no clinical symptoms of infection before death

Pathologic Findings

Atoxoplasmosis

Atoxoplasmosis is found in passerine birds mostly canaries, Old World finches, starlings, and mynahs. See relevant section.

Cryptosporidiosis

Cryptosporidiosis is found in a wide variety of avian hosts.

Respiratory form:

- Excessive amount of mucus in trachea and nasal cavity
- Swollen head
- Air sacculitis
- Pneumonia
- Conjunctivitis
- Lesions in lower respiratory tract are often complicated with fungal infections

Intestinal form:

- Distention of the small intestine or ceca with gas or mucus
- Excessive mucus, hemorrhages, and/or distention of the proventriculus walls
- Inflammation of the bursa of Fabricius

Renal form:

- Enlargement of kidneys
- Pale discoloration of kidneys

Intestinal Coccidiosis

Pathologic changes vary according to coccidia species and pathogenicity of the strain, intensity of infection, age, and immune status of the host including previous infections with the same species of coccidia. Subclinical infections with mild or no readily visible lesions may be responsible for the poor quality of young birds. Coccidia species differ in the location of their reproduction sites (along the GIT—from duodenum to cloaca, and across the intestine wall—from submucosa through the base of the crypt to the villus tip), which influences the

location and type of lesions. Gross lesions in affected small intestine and/or ceca may include:

- Dilatation
- Petechiation and hemorrhage
- Discoloration
- Formation of species-specific macroscopic lesions visible on mucosal or serosal surfaces of the small intestine and/or ceca (i.e., transversely arranged gross white plaques in duodenal mucosa—*Eimeria acervulina* or enlargement and distention of ceca with clotted blood in *Eimeria tenella*, in chickens). Very often there are mixed species infections with two or more coccidia species producing lesions in different locations along the GIT.
- Excessive production of mucus, crust lesions, pseudomembranes, and cores (especially in ceca).
- All abovementioned lesions may be complicated with other pathogens such as bacteria (especially anaerobic), yeasts, and protozoa.
- Gross lesions in birds from clinical cases may be obvious, subtle, or virtually absent.

Renal Coccidiosis

Renal coccidiosis is seen in Sphenisciformes, Procellariiformes, Pelecaniformes, Anseriformes, and Apterygiformes. Depending on intensity of the infection, enlargement and discoloration of the kidneys with or without pale streaking and mottling and nodulation are seen.

Disseminated Coccidiosis in Cranes

Disseminated coccidiosis is seen in wild and captive cranes, but not reported in *Balearica* spp. cranes.

- Orange-white granulomas in various organs: trachea, esophagus, GIT, liver, heart, kidney, spleen, thymus, bursa, and others
- Pneumonia
- Congestion of lungs
- Enteritis
- Splenomegaly and hepatomegaly

Sarcocystis

Sarcocystis has wide variety of avian hosts (Fig. 14-165). Intermediate hosts are especially pathogenic for Old World psittacines.

- Lung congestion and edema
- Hemorrhage
- Enlargement of kidneys, liver, and spleen
- Development of sarcocysts in skeletal muscles ("rice breast disease")

Toxoplasmosis

Toxoplasmosis has a wide variety of avian hosts (Figs. 14-166 and 14-167).

- Pneumonia
- Enlargement of liver and spleen often with pale foci throughout the parenchyma
- Pericarditis and myocarditis
- Ocular lesions (especially in canaries)
- Enteritis, and in acute cases necrosis of the intestine and death
- Encephalitis

Diagnosis

Diagnosis of coccidiosis relies on observation of clinical signs and finding oocysts and/or developmental stages together with gross and/or microscopic lesions. In avian intestinal coccidial diseases the diagnostic tools include:

- Fecal examination for the presence of oocysts (direct or concentrated). Especially in passerines, feces for examination should be pooled from evening and night because of increased oocyst

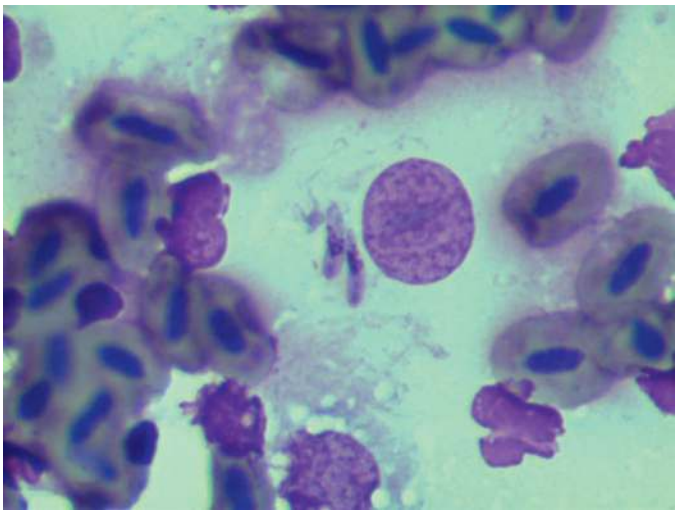


FIGURE 14-165 *Sarcocystis* sp. Free merozoites from the lung impression smear from a white cockatoo (*Cacatua alba*), ×Diff-Quick 1000.

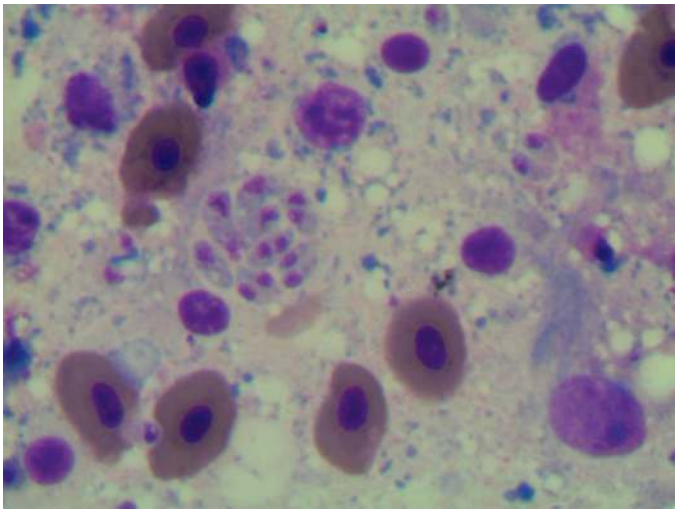


FIGURE 14-166 *Toxoplasma gondii* tachyzoites, lung impression smear, peacock (*Pavo indicus*), Diff-Quick, ×1000.

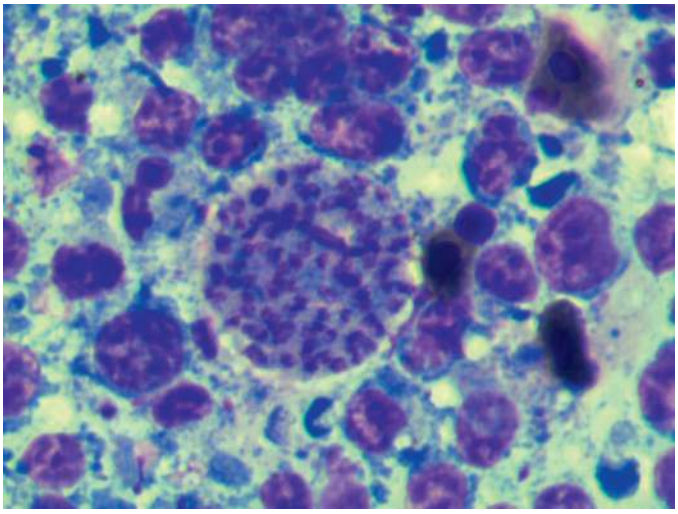


FIGURE 14-167 *Toxoplasma gondii* tissue cyst, lung impression smear, peacock (*Pavo indicus*), Diff-Quick ×1000.

shedding at roosting time, which favors transmission of the parasite between birds.
- Direct examination of intestinal mucosa scrapings for the presence of developmental stages (schizonts, merozoites, and oocysts).
- Cytology, histopathology, immunohistochemistry, and molecular methods.

Standard identification of coccidia species requires sporulation of the oocysts (sporulation time is an important feature), and detailed analysis of their morphologic characters such as size and shape of oocyst and sporocysts (length and width), oocyst color, number of sporocysts and sporozoites, and their detailed morphologic characters (presence or absence of Stieda body, residuum, micropyle cap, polar bodies, etc.). Today it is often replaced by the use of molecular methods. Other coccidial diseases in which the development forms of the parasite are located in various visceral organs and cryptosporidia often require cytology, histopathology, immunohistochemistry, or other methods (serologic and/or molecular) for diagnosis.

Transmission

Infectious forms of avian coccidia are ingested orally.

Treatment

Treatment of coccidial diseases often requires combining the medical treatment, revision of the management system, and thorough disinfection. Coccidia tend to thrive in young and immunologically naive individuals kept in overcrowded and wet conditions with high infective pressure from the environment. Taking care to provide the birds with a dry and clean environment (i.e., use of the grid floor) where they cannot contact large numbers of infective oocysts substantially lowers the risk of developing the clinical coccidiosis and other stress- and density-related diseases.

Substances used to combat coccidiosis can be divided into two major groups: coccidiocidals and coccidiostats, which kill and suppress the development of parasite, respectively.

The commonly used drugs include:
- Sulfonamides and trimethoprim potentiated sulfonamides: They have a coccidiostatic effect by inhibiting the synthesis of folic acid.
- Triazines i.e., toltrazuril: This realizes a coccidiocidal effect by disruption of nuclear division and functioning of mitochondria.
- Amprolium (coccidiostat): A thiamine antagonist used alone or in combination with folate antagonists, i.e., ethopabate.
- Coccidiostats less commonly used outside poultry:
  - Synthetic coccidiostats, i.e., robenidine, decoquinate, and nicarbazin
  - Ionophore coccidiostats (altering levels and transport of cations Na^+, K^+, Ca^{2+}, and Mg^{2+} in and out of the parasite cells), i.e., salinomycin, monensin, narasin, maduramycin, semduramicin, and lasalocid.

Coccidia differ in their productivity from low to very high. Ingestion of one sporulated oocyst may result in the production of several thousand up to several millions of next-generation oocysts. This clearly shows the necessity of appropriate disinfection during and after the outbreak of the disease to reduce the infective pressure from the environment where under appropriate conditions oocysts can survive several months. There are very few toxic substances used for killing oocysts in the environment and these include ammonia water or formaldehyde fumes, and wet flaming (flaming previously wetted surfaces) is probably still the method of choice as it gives the best results.

Prevention

- Sound hygiene practices including maintaining clean, dry environment with proper ventilation.
- Avoiding overcrowding. All birds should have adequate room, feel safe, and get adequate rest at night.
- Allowing birds to develop immunity by early infection with a low number of coccidians.
- Regular health checks with fecal examination.
- Strategic pharmacologic treatment during critical moments (change of feed, periods of stress, i.e., weaning, molting, placing in a new environment), in especially susceptible species, and in problematic collections with previous history of coccidiosis.
- Use of coccidiostats and/or vaccines in appropriate species.

ATOXOPLASMOSIS

Definition

Atoxoplasmosis is a protozoal disease of certain passerine birds caused by a coccidian parasite belonging to the genus *Isospora.* Atoxoplasma is now considered a junior synonym, but it will be used in this section to ease comprehension and for the benefit of the reader.

Synonyms

Synonyms include visceral coccidiosis, thick liver disease, big liver disease, black spot disease, and going light. The last two names are often used as synonyms of circovirosis and infection with *Macrorhabdus ornithogaster*, respectively, leading to much confusion among fanciers and breeders.

Etiologic Agent

- Coccidian parasite *Atoxoplasma* is an etiologic agent.
- Atoxoplasms possess a simple life cycle in which part of the asexual (two schizogonies) and entire sexual phase (gametogony and oocyst production) takes place in the small intestine, while the majority of asexual reproduction (five schizogonies) occurs in the mononuclear cells in various extraintestinal locations, i.e., lungs, liver, spleen, and brain.
- Ingested infective oocysts release sporozoites that penetrate the intestinal mucosa and leave the intestine by means of mononuclear cells, in which they undergo few rounds of asexual reproduction outside the intestine. Merozoites finally migrate back to the intestine to complete their asexual development and go through the sexual phase, which will terminate in the production of unsporulated oocysts. These are shed in feces in relatively low numbers (100 to 200 every 24 hours, in contrast to intestinal species of coccidia), and sporulate in the environment.

Distribution

Distribution is worldwide, except Antarctica.

Susceptible Species

Susceptible species include passerines, especially in the families Fringillidae and Sturnidae.

Canaries, European finches, starlings, and mynahs most commonly present with clinical atoxoplasmosis. It is believed that atoxoplasms possess a high level of host specificity (i.e., *Isospora serini* infecting only canaries). According to some publications, intraleukocytic stages (merozoites) of *Atoxoplasma* spp. have been found in at least 58 bird families.

Clinical Symptoms

- Weight loss
- Respiratory distress with or without respiratory sounds (rales, clicking sounds, and whistling)
- Hepatomegaly, visible through the abdominal wall (Fig. 14-168)

FIGURE 14-168 Enlarged liver visible through the abdominal wall in young canary (*Serinus canaria*).

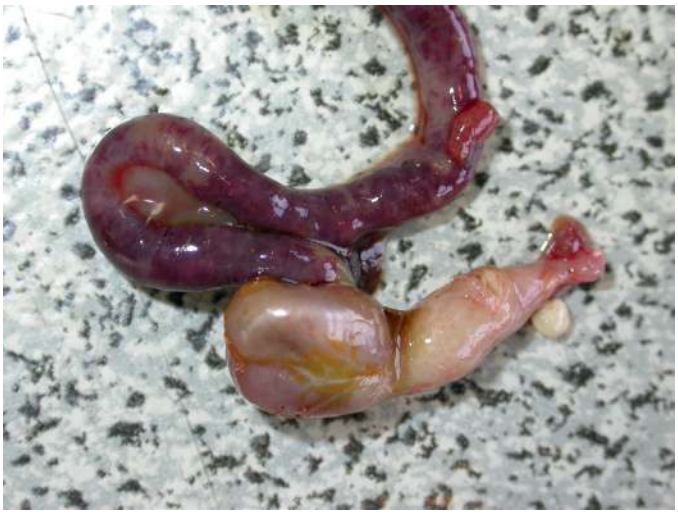


FIGURE 14-170 Dilated duodenum with hemorrhages.

FIGURE 14-169 Hepatomegaly and splenomegaly in bullfinch (*Pyrrhula pyrrhula*).

- Central nervous signs (up to 20% of affected birds): loss of balance, seizures, circling, nystagmus
- Enteritis (enlarged abdomen with dilated intestines, often red colored)
- Diarrhea
- Mortality (up to 80%), especially in young birds during stressful periods i.e., weaning and molting, sometimes acute deaths without premonitory signs.
- Many susceptible hosts necropsied for other reasons often show a few *Atoxoplasma* spp. merozoites in leukocytes mostly from lung imprints, which suggest that there is a high rate of asymptomatic infections and latency in healthy-looking birds from all age groups.

Pathologic Findings

- The majority of necropsied birds from atoxoplasmosis cases show hepatomegaly, splenomegaly, and enteritis. The liver may be enlarged to various extent, sometimes with necrotic foci throughout the parenchyma, and the spleen may be severely enlarged (Fig. 14-169).
- The intestines, mostly duodenum and proximal jejunum, are often dilated with thickened walls and marked hemorrhages visible on the serosal surface (Fig. 14-170).
- Lungs and bronchi are grossly unaffected even in the presence of clinical respiratory signs.
- Skeletal muscles and myocardium may be also affected.

Diagnosis

- A tentative diagnosis can be made on the basis of anamnesis; however, definite diagnosis can only be made by finding *Atoxoplasma* merozoites in the cytoplasm of leukocytes from blood or internal organs, which requires the use of cytology or histopathology.
- In air-dried, Romanowsky-stained (Diff-Quik or Hemacolor) cytologic preparations of lung, liver, and spleen impressions or buffy coat smears of atoxoplasm, merozoites are fairly large (often exceeding the size of red blood cell nuclei) inclusions found in the cytoplasm of mononuclear cells. These inclusions are whitish colored, ovoid structures with a diffuse pink or purple center, and often displace the nucleus of a host cell or mold it into a crescent shape. Merozoites may occur single or in clusters (meronts), contrasting well with the blue cytoplasm of their host cells (Fig. 14-171).
- Finding unsporulated coccidian oocysts in fresh feces is not diagnostic for *Atoxoplasma* spp., even in the presence of suggestive clinical signs. Also examination of oocyst morphology for identification of parasite genus or species is impractical in clinical conditions.
- There are available molecular methods for detection of *Atoxoplasma* spp. in clinical samples.

Transmission

Transmission is through the fecal–oral route. To become infected, susceptible hosts need to ingest infective sporulated oocysts.

Treatment

Different anticoccidials have been used in the treatment of atoxoplasmosis with varying results. Intraleukocytic stages are generally considered nonresponsive to treatment; however, long use of sulfonamides (5 days a week for several months) and/or toltrazuril (2 days a week for several months) suppress oocyst shedding and aid in control of the disease at least by lowering the infective pressure from the environment.

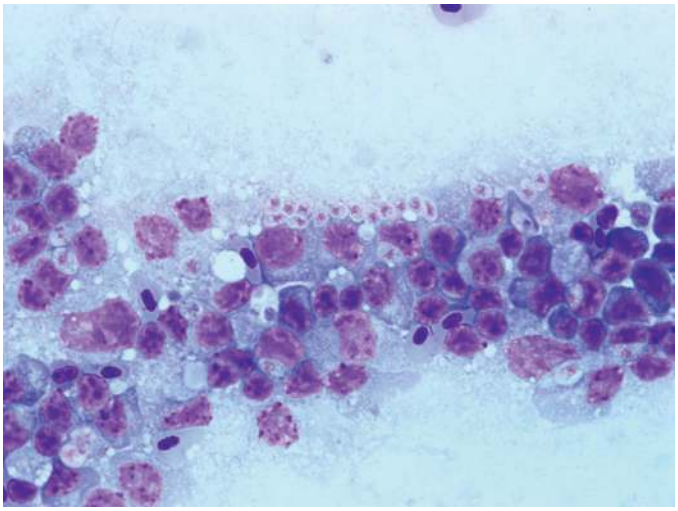


FIGURE 14-171 Atoxoplasma in spleen in canary (*Serinus canaria*).

FIGURE 14-172 Trichomonas sinusitis in color canary (*Serinus canaria*).

Prevention

Mild infections can stimulate the immune system and prevent systemic atoxoplasmosis. See also coccidiosis.

TRICHOMONOSIS

Definition

Trichomonosis is caused by the parasitic flagellate *Trichomonas gallinae.*

Synonyms

Synonyms include canker, roup, and frounce (in raptors). The term trichomonosis refers to a clinical form of the disease, whereas trichomoniasis depicts a carrier stage in which no signs of the disease can be recognized.

Etiologic Agent

Trichomonas gallinae are small, 5 to 20 µm size, oval to pea-shaped flagellates with four free, anteriorly placed flagella, axostyle protruding from the posterior end, and an undulating membrane. Trichomonads have a direct life cycle and reproduce by binary fission. Described parasite forms include motile trophozoite and nonmotile pseudocyst stages. Trichomonads observed under the light microscope in freshly processed clinical samples are rapidly moving (circling in in a jerky manner with no clear direction, not escaping the microscope field), translucent small flagellates that may appear single or in clusters. Under higher magnification one can often observe wave like movements of an undulating membrane. Different strains of *T. gallinae* differ in their virulence from apathogenic to very virulent.

Distribution

Distribution is all over the world except Antarctica, Greenland, and the northern parts of North America, Europe, and Asia.

Susceptible Species

Susceptible species include birds in orders: Columbiformes, Falconiformes, Psittaciformes, Passeriformes, Galliformes, Anseriformes, and Gruiformes. Color canaries, suffering from hypervitaminosis A, often caused by massive administration of coloration, are extremely sensitive (Fig. 14-172).

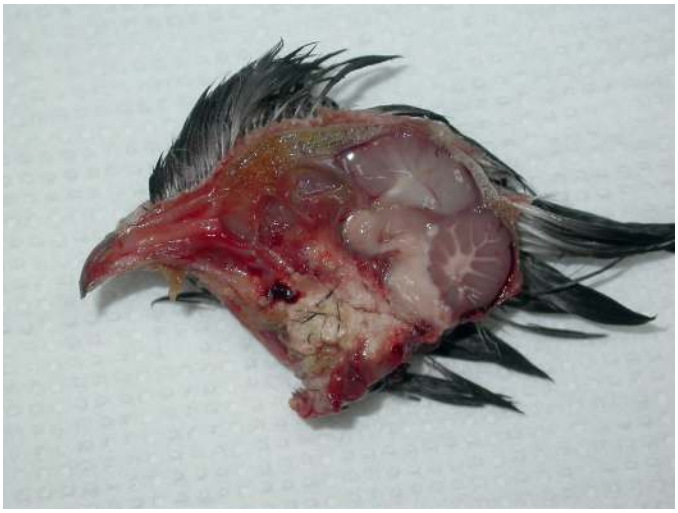


FIGURE 14-173 Trichomonas stomatitis, domestic pigeon (*Columba livia*).

Clinical Symptoms

- Clinical symptoms vary widely and may include unspecific signs such as weight loss, listlessness, and ruffled and matted feathers.
- Signs suggestive of trichomonosis, especially in susceptible hosts, may include deaths and poor growth of nestlings; stasis; delayed emptying time and foul-smelling crop contents; excessive salivation; problems with swallowing; regurgitation; diarrhea; palpable or visible caseous lesions in the oral cavity, bursa of Fabricius, and umbilicus; swelling of sinuses and facial region; respiratory rales; dyspnea; conjunctivitis; and death of severely affected individuals.

Pathologic Findings

- These vary depending on the virulence of the trichomonad strain and susceptibility of the host and may range from mild to severe catarrhal inflammation of the oral cavity, throat, and the crop mucosa to ulcerative and caseous lesions of various extent in the upper digestive tract (mouth, throat, crop, and thoracic esophageal-proventriculus junction; Fig. 14-173), upper respiratory tract, and

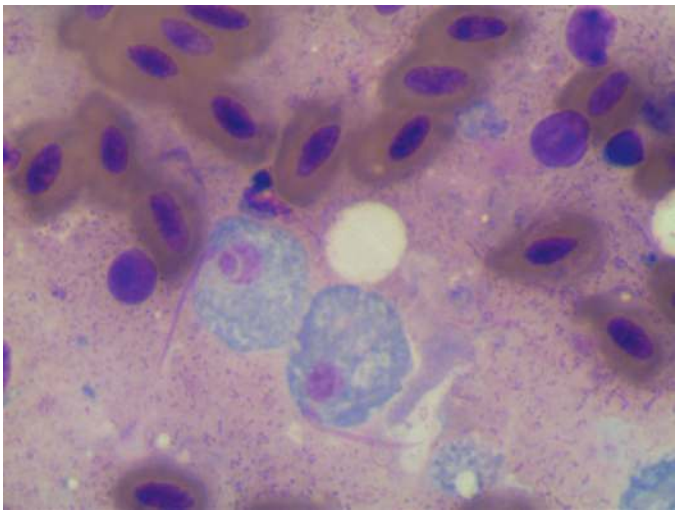


FIGURE 14-174 Trichomonads, lung impression smear, Diff-Quick, ×1000, in a domestic pigeon (*Columba livia*).

head (sinuses, skull, skin of the neck, and trachea). In very virulent strains the liver, lungs (Fig. 14-174), pericardium, air sacs, and pancreas are affected.
- Especially in young pigeons, white cheesy masses can be found in the umbilicus and bursa of Fabricius.
- Trichomonosis can mimic various diseases of which the most important in differential diagnosis are candidiasis, capillariidosis, pox, herpesvirus infections in pigeons and raptors, and vitamin A deficiency. All listed differentials are often complicated by trichomonads alone or together with bacteria and yeasts.

Diagnosis
- Finding characteristic motile trophozoites in fresh wet preparations of crop contents and swabs taken from the oral cavity, conjunctivae, and cloaca, observed under light microscopy (100× or 400× magnification).
- When direct microscopic examination of fresh samples cannot be performed or when fresh sample lacks motile trophozoites, diagnostic methods may include cytology, histopathology, molecular diagnosis, and culturing of the organism.

Transmission
Trichomonads may be transmitted directly via crop milk and saliva through billing and courtship feeding and via drinking water, moist grain (able to survive several days), and feces (especially in "wet nests").

Treatment
Various drugs from the nitroimidazole family, ronidazole, and metronidazole are most commonly used. Advanced lesions often need to be treated surgically together with appropriate chemotherapy.

Prevention
- Identification and treatment of carrier birds, cleaning and regular disinfection of drinkers and drinking water; wild birds should have no access to water, feed, and birds in the collection.
- In case of raptors, prevention is through feeding deep-frozen, cleaned, or parasite-free pigeons (trichomonads are able to survive 48 hours in pigeon carcass).
- In susceptible species and in problematic collections regular health checks and cleanups with appropriate medication may help control the disease.

ASCARIDIOSIS

Definition
Ascaridiosis is caused by parasitic nematodes of the genus *Ascaridia.*

Synonyms
Synonyms include ascaridiosis and ascarosis.

Etiologic Agent
Ascaridia spp. (approximately 40 species have been reported in birds) are one of the most common nematodes in birds. They are large (16 to 120 mm), thick yellowish-white worms parasitizing the small intestine and sometimes the ceca of susceptible hosts. Females are often larger than males. Ascarids have a simple, direct life cycle. Nonembryonated, elliptical thick-walled single-cell eggs with fine granular brownish contents and colorless shell (approximately 80 × 50 µm) are shed in feces and embryonate in the environment with appropriate humidity and temperature in 2 to 3 weeks. Infective eggs hatch in the proventriculus or duodenum of the susceptible host releasing second-stage larvae that will invade small intestine mucosa where they molt to L3. Depending on the species and host, larvae either return to the intestinal lumen where they molt and mature into adult worms or they may arrest their development at certain stage (i.e., L3) to become the predominant form of the parasite and become its reservoir throughout the life of the host. Ascarids may use transport or paratenic hosts, such as earthworms or grasshoppers, but they do not develop or accumulate in them.

Distribution
Distribution is in all continents, except Antarctica.

Susceptible Species
Ascarids have been reported in 139 host species in orders Struthioniformes, Tinamiformes, Ciconiiformes, Anseriformes, Falconiformes, Galliformes, Gruiformes, Columbiformes, Psittaciformes, Cuculiformes, Strigiformes, Caprimulgiformes, and Passeriformes. Ascarid species vary widely in their host specificity and distribution from cosmopolitan to restricted to certain geographic region and host species. Young birds are more susceptible to infection than adults, which develop some resistance and immunity.

Clinical Symptoms
- Suggestive for *Ascaridia* is weight loss, emaciation, matted feathers, slimy green feces, and vomiting.
- Nonspecific symptoms include weakness, anemia, diarrhea, loss of appetite, abdominal pain, growth retardation, soiled vent, lameness and leg paralysis, and sudden deaths.

Pathologic Findings
- Ascarids inhabit the small intestine, in particular the duodenal loop, but can be found in the esophagus, crop, gizzard, body cavity, liver, bile and pancreatic ducts, oviduct, and egg.
- Adult nematodes and their larvae irritate the intestinal mucosa, which may result in edema, hyperemia, hemorrhage, destruction of villi, dilatation of crypts, and nodulation. Heavy infections may cause intestine occlusion, intussusception, or perforation (Fig. 14-175).
- Ectopic migration of larvae through intestinal wall, liver, lung, and mesenteries may provoke intensive leukocyte infiltration around larvae and elicit granulomatous lesions (Figs. 14-176 and 14-177).

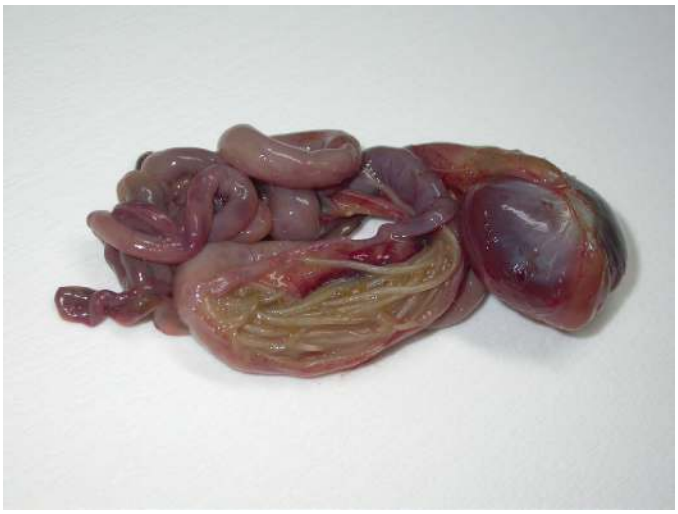


FIGURE 14-175 Ascarids in the small intestine in a pigeon (*Columba livia*).

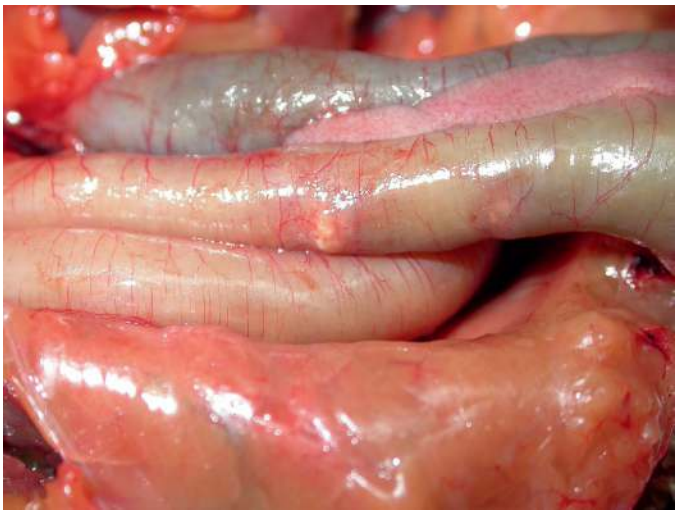


FIGURE 14-176 Intestinal granulomatous lesions. Ascaridiosis in a domestic pigeon (*Columba livia*).

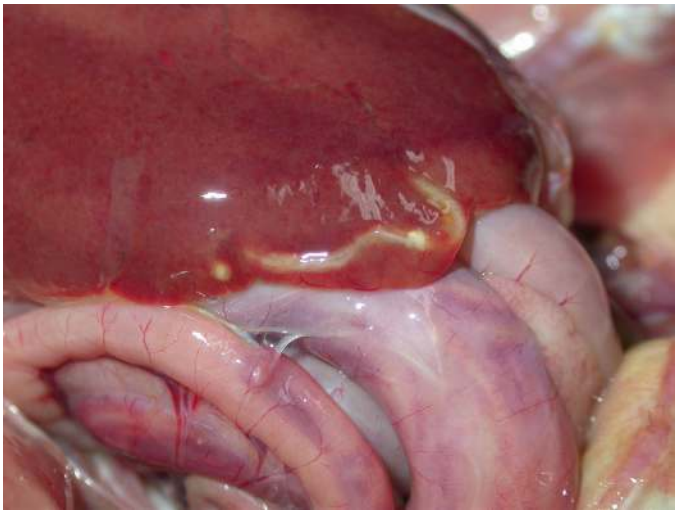


FIGURE 14-177 Adult worm in liver parenchyma in a pigeon (*Columba livia*).

Diagnosis

Diagnosis includes finding and identifying adult worms and/or their eggs.

Transmission

Direct ingestion of embryonated eggs, larvae, or transport/paratenic hosts carrying infective eggs.

Treatment

Benzimidazole anthelmintics, levamisole, ivermectin, moxidectin, pyrantel pamoate, and others, followed by vitamin A supplementation and thorough sanitation.

Prevention

Infection requires ingestion of embryonated eggs or larvae; therefore, successful treatment should combine expelling the adult worms from the intestine and destruction of eggs in the environment or disrupting the cycle by prevention of birds contacting infective eggs by use of grid floors or providing a dry and clean environment. Ascarid eggs are resistant to many commonly used disinfectants; steam cleaning or flaming are the most effective disinfecting methods.

CAPILLARIIDOSIS

Definition

Capillariidosis is caused by nematodes belonging to the genera *Baruscapillaria*, *Capillaria*, *Echinocoleus*, *Eucoleus*, *Ornithocapillaria*, *Pterothominx*, and *Tridentocapillaria*, collectively named as Capillarids.

Synonyms

Synonyms include capillariidosis, capillariosis, cropworm, hairworm, threadworm, and wireworm.

Etiologic Agent

Avian capillarids are a heterogeneous group of nematodes belonging to seven genera, all in the family Trichuridae, subfamily Capillarinae. Their taxonomy is complicated and often leads to confusion, with single species often listed under various names, synonyms, or arranged into a complex of species under one name. Capillarids are small, 8 to 60 mm long, and thin (less than 100 µm wide) nematodes burying in the mucosa of the GIT. They are almost impossible to see with the naked eye (thus the name hairworm). Freshly passed, unembryonated eggs are characteristically double operculated with distinctive, big plugs at both ends (Fig. 14-178). Ova may be symmetric or asymmetric (long axis through one plug not meeting opposite plug). They, are single cell with fine granular contents, various patterns of shell and coloration (colorless, golden brown, and bile green), measuring on average 55 × 25 µm. Egg morphology is not sufficient for identification of genus or species of the parasite.

Different species inhabit various segments of the avian GIT, from the oral cavity to the distal small intestine and less often the ceca. Mixed infections are not rare, with one host infected with several species parasitizing at different locations. Capillarids may have direct or indirect life cycles.

- In species with a direct life cycle (homoxenous), unembryonated eggs voided with feces embryonate under favorable conditions in the environment (*Eucoleus contortus*: 24 to 40 days, *Baruscapillaria obsignata*: 3 to 13 days), and after reaching L1 stage they become infective to the susceptible avian hosts. Ingested infective eggs hatch in the GIT of the bird and liberated larvae undergo further

FIGURE 14-178 Eggs inside the adult capillaria worm in a domestic pigeon (*Columba livia*).

FIGURE 14-179 Intestinal loop filled with capillaria in a chaffinch (*Fringilla coelebs*).

development in the gastrointestinal mucosa (four molts) until maturation. On average, the prepatent period in capillarids lasts 3 to 4 weeks. Paratenic hosts, mostly oligochetes may be used by certain homoxenous capillarids.

- In capillarids with an indirect life cycle (heteroxenous) i.e., *Pterothominx caudinflata*, *P. bursata*, *Eucoleus annulatus*, *E. dispar*, obligate intermediate hosts like oligochetes (one species *P. philippinensis* uses fish) are required by the parasite to become infective. The degree and speed of development inside the intermediate hosts varies between capillarid species. In some, larvae are infective already after hatching in the earthworm intestine; in others they require further development.

Depending on the capillarid species, birds become infected by ingestion of infective eggs, free or carried by paratenic hosts (direct life cycle) or obligate intermediate hosts (complex life cycle) carrying infective eggs or larvae. In some capillarids, rodents are suspected to serve as transport hosts (*Baruscapillaria falconis* and *Capillaria tenuissima*). Among avian capillarids one species *P. philippinensis* is zoonotic.

Distribution

Distribution is worldwide.

Susceptible Species

Capillarids have been reported in orders Struthioniformes, Tinamiformes, Gaviiformes, Podicipediformes, Pelecaniformes, Ciconiiformes, Anseriformes, Falconiformes, Galliformes, Gruiformes, Charadriiformes, Columbiformes, Psittaciformes, Strigiformes, Caprimulgiformes, Apodiformes, Piciformes, and Passeriformes. Some capillarid species such as *Eucoleus contortus* are known to infect a wide variety of avian hosts from at least nine orders, whereas some species infect only a single host, i.e., *Pterothominx moraveci* is found only in Port Lincoln Parrots (*Barnardius zonarius*).

Clinical Symptoms

Clinical symptoms vary according to the intensity of infection, parasite species, and susceptibility of the host. Some affected birds may show no or minimal clinical signs. Nonspecific clinical signs include poor performance, weight loss, anemia, diarrhea, ruffled feathers, and central nervous system signs. Capillariosis often presents in three forms: oropharyngeal, esophageal, and intestinal.

- Signs suggestive of oropharyngeal capillariosis include mucoid-stringy deposits, yellowish-white plaques, granulomas, and abscesses in the pharynx, tongue, and rictus.
- Esophageal form includes palpable masses in the esophagus and crop, problems with swallowing and contractility of the crop.
- Very sticky, scant, and dark green feces, sometimes containing blood, are suggestive for the intestinal form of capillariosis. Heavy infections result in poor digestion and absorption.

Pathologic Findings

Capillarids inhabit various parts of the avian GIT. *Eucoleus annulatus* and *E. contortus* live in the mucosa of oral cavity and crop, *Baruscapillaria obsignata*, *Pterothominx caudinflata*, *P. bursata* in small intestine (Fig. 14-179), and *Capillaria anatis* and *Baruscapillaria obsignata* in the ceca.

They burrow tunnels in the mucosa and sometimes submucosa, where gravid females lay eggs, which are shed into the gut lumen together with sloughed epithelium. Upper gastrointestinal capillarids are considered more pathogenic than intestinal-dwelling species. They may cause inflammation, exudation, ulceration, hyperkeratosis, and thickening of the mucosa from the oral cavity to the proventriculus, dilatation of the crop, and squamous metaplasia of mucous gland in the esophagus. Secondary bacterial or fungal colonization are common and lead to complicated lesions in the form of fibrinonecrotic plaques, diphtheritic membranes, and large caseous masses, sometimes involving underlying bones. In severe cases parasites may penetrate into the muscularis. Intestinal species cause thickening, erosions, and ulceration of intestinal mucosa. Severity of lesions varies according to parasite species and intensity of infection. Lesions may be more severe in aberrant hosts.

Diagnosis

Diagnosis is made after finding and identifying adult worms and/or their eggs.

Transmission

Transmission occurs in monoxenic species by direct ingestion of embryonated eggs or paratenic hosts carrying infective eggs. In

heteroxenic species it occurs by ingestion of an intermediate host carrying infective stages of the parasite. In several species considered heteroxenic, direct life cycle with paratenic hosts are suspected to exist (*Eucoleus dispar*).

Treatment

See Syngamosis.

Prevention

See Ascaridiosis.

SYNGAMOSIS

Definition

Syngamosis is caused by the parasitic nematode *Syngamus* spp.

Synonyms

Synonyms include syngamiasis, gapeworm, gapes, redworm, tracheal worm, and forked worm.

Etiologic Agent

Syngamus spp. are strongylid nematodes infecting the respiratory tract of susceptible avian hosts. Adult *S. trachea* are bright red worms locked in copula in a characteristic Y shape (Fig. 14-180). Females are much larger than males measuring 5 to 40 mm and 2 to 6 mm in length, respectively. Small males are permanently affixed to the tracheal mucosa, whereas females attached permanently to males but not to trachea are able to attach and detach to feed at different locations around the anchoring site of their male. Adult females lay eggs into the tracheal lumen, which are then coughed up, swallowed, and passed in feces to embryonate in the environment.

Eggs are bipolar, ellipsoidal, and double operculated. They are 90 × 50 µm in size, with a smooth thin shell containing eight cell morula, when passed freshly. Optimal temperature for development of *S. trachea* eggs is 29°C. Under these conditions it takes only 7 days for the larva to develop inside the egg into the infective third stage. The development of eggs does not take place in temperatures at or below 16°C, and at 17°C the eggs will become infective in 42 days. Infective larvae (L3) may hatch spontaneously and live free in wet soil for some time.

FIGURE 14-180 *Syngamus* spp. showing the characteristic Y-shape. The small male is permanently attached to the trachea, in a gray crowned crane (*Balearica regulorum*).

Birds may contract an infection in different ways:

- Directly by ingesting embryonated infective eggs and free-living infective larvae
- Indirectly by eating paratenic host containing encysted or free larvae

When ingested by a suitable host, the infective eggs hatch in the small intestine and penetrate its wall. The majority of larvae are transported to the lungs, most likely via the portal blood, but some also migrate directly through the tissues. The larvae reach the lungs as early as 4 hours after infection. Worms can be detected in the lungs in 7 days, in the trachea at 11 days, and fertile females can be found at 14 days post infection. The life cycle is simple, direct, or indirect.

Distribution

Syngamus spp. has been reported in Europe, Asia, and North America, but its range may be worldwide.

Susceptible Species

Susceptible species are primarily birds that feed on the ground, most commonly Galliformes and Passeriformes. Prevalence varies greatly by host species and age. Adult birds have generally less intensive infections, and in some occasions are able to expel the worms. Young birds of susceptible species may virtually perish.

Clinical Symptoms

- Suggestive symptoms for syngamosis in highly susceptible hosts (i.e., gamebirds, peacocks, corvids) include gasping for air, coughing, head shaking, stretching neck, open mouth breathing, and producing grunting sounds.
- Nonspecific signs include weakness, weight loss, and emaciation often together with respiratory distress. Heavily affected birds eventually die of suffocation.

Pathologic Findings

- Worms attach to the mucosal lining of the trachea and bronchi causing their inflammation through mechanical damage, irritation, and possibly parasite secretions. Male worms are attached permanently to the mucosa or tracheal ring cartilage, which in certain species (especially turkeys and pheasants) may cause nodulation on the internal and external surface of the trachea.
- In seriously affected birds, worms can block the tracheal lumen causing suffocation (Fig. 14-181).
- Pneumonia in young birds is the result of host response to migration of larvae through lungs (Fig. 14-182).
- Adult gapeworms feed on blood, but the overall loss of blood caused by their parasitism is minimal.

Diagnosis

- Finding and identifying adult worms in the trachea and bronchi, endoscopically or during necropsy
- By transillumination of the trachea (Fig. 14-183)
- Clinical symptoms and finding characteristic eggs in feces and/or in the mouth cavity

Transmission

Direct ingestion of infective eggs, free-living infective larvae, or transport/paratenic hosts carrying encysted or free larvae.

Treatment

Gapeworm infections can be treated by various anthelmintics with fenbendazole, flubendazole, and ivermectin are most commonly used. During and after treatment birds can show severe respiratory symptoms because the tracheal worms have to be coughed out.

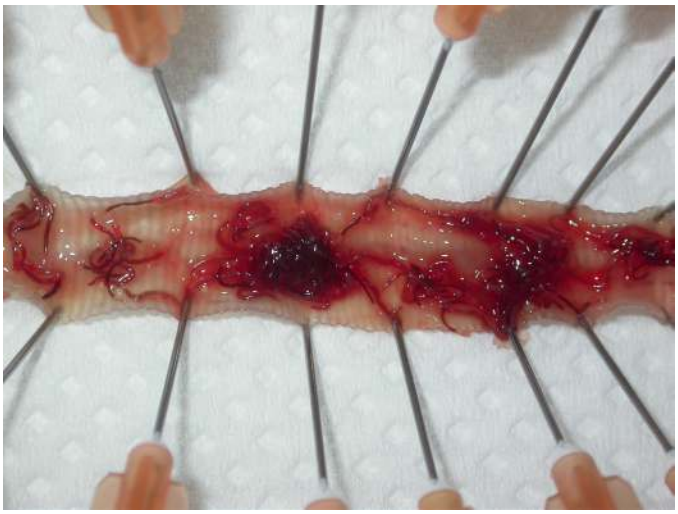


FIGURE 14-181 Mass of gapeworms in trachea of 3-week-old peacock (*Pavo cristatus*).

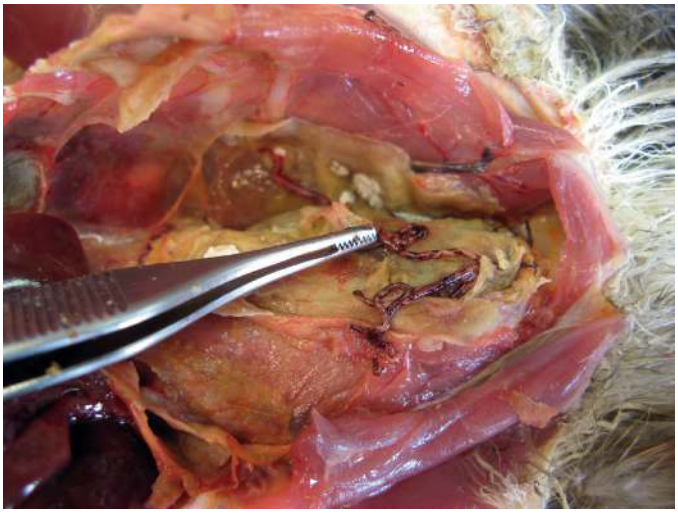


FIGURE 14-182 Migration of syngamus in abdominal air sac, Harris's hawk (*Parabuteo unicinctus*).

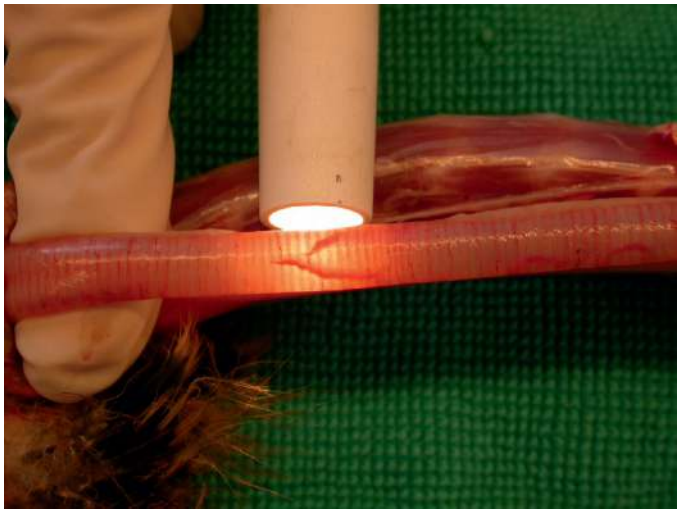


FIGURE 14-183 Transillumination of the trachea in a gray-crowned crane (*Balearica regulorum*).

Prevention

Larvae encysted in the earthworms may remain infective for 4.5 years; therefore control of infections must rely on reduction of infected paratenic hosts in the bird's environment by destroying earthworms, snails, and other transport hosts that transfer the parasite or by alternating the rearing pens. Gapeworms are most dangerous to young birds, therefore, susceptible species in problematic areas should be regularly dewormed in 2- to 4-week intervals, especially during hot months.

NORTHERN AND RED MITES

Definition

Infestation of birds with the northern mite *Ornithonyssus sylviarum* or the red mite *Dermanyssus gallinae.*

Synonyms

For *O. sylviarum* they include northern mite, white mite, and northern fowl mite. For *D. gallinae*: they include red mite, chicken mite, poultry mite, and roost mite.

Etiologic Agent

- Northern mite (*O. sylviarum*, Family: Macronyssidae) is a common ectoparasite of cage birds and poultry. Northern mites are dark colored (red to black), flattened eight-legged (nymphs and adults) parasitic arthropods, approximately 0.7 to 1 mm in length and 0.4 mm in width. They spend the entire life cycle on their avian hosts (c.f., red mite). Five developmental stages have been described: egg, larva, protonymph, deuteronymph, and adult. Only protonymphs and adults are hematophagous. The life cycle (from egg to egg-laying female) can be completed in 5 to 7 days under favorable conditions (18 to 20°C; northern mites prefer lower temperatures than red mites). Adult females lay two to five eggs at the base of feathers (especially in the vent or back) about 2 days after feeding on blood. Birds are the only suitable hosts for northern mites, but parasites can bite and parasitize (but not reproduce) on other animals and humans. Depending on the source, northern mites are reported to stay alive in the environment, outside their host, for several days up to 2 months.
- Red mites (*D. gallinae*, Family: Dermanyssidae) are small (1 mm length, 0.4 mm width), whitish-gray colored mites that turn red when engorged with blood. The main difference in clinical importance between northern and red mites is that the latter enter birds only for feeding during the night, while the rest of their life cycle takes place outside the host (it is considered a temporary parasite). The white (0.4 × 0.25 mm) eggs are laid in hiding places including all kinds of nooks and crannies in aviaries and cages, nests, and nesting material but also dry manure and litter. Depending on the temperature and humidity (red mites prefer temperatures of 25°C or more and high humidity) eggs hatch in 2 to 3 days.

Developmental stages are the same as in the northern mite, and the life cycle may be completed in 7 to 10 days. Protonymphs, deuteronymphs, and adults are hematophagous. The mites feed for about 60 minutes every 2 to 4 days. The majority are active and feed after dark, but some may remain on the host during the daytime. Red mites enter birds from perches but they may also drop from the ceiling. They are attracted to birds because of their high temperature and skin lipids.

Distribution

Distribution is worldwide.

Susceptible Species

All species of birds are susceptible.

Clinical Symptoms

In adults:

- Nervousness (during the night, red mite; during the whole day, northern mite)
- Itchiness
- Loss of feathers
- Frequent scratching, excessive preening
- Tiredness
- Decreased egg production
- Hens abandoning nests
- Anemia
- Dyspnea
- Death

In nestlings:

- Late embryo mortality and problems with hatching (through hens leaving their nests)
- Unthriftiness
- Apathy
- Anemia
- Death

Pathologic Findings

- Birds affected with bloodsucking mites often show anemia (a high number of red mites can drink up to 6% of blood volume a day; data from chickens) and poor body condition.
- Apart from the presence of adult mites and/or their eggs on the plumage of living or dead birds (Figs. 14-184 and 14-185), sometimes it is possible to find pinprick size bites on the skin (especially under the wings and on the bare skin of nestlings).
- Mites and their eggs can sometimes be found during postmortem examination in the crop or in the intestine contents, but also during standard microscopic fecal examination.
- Bloodsucking mites are successfully transmitting several infectious diseases (several arboviruses, fowlpox, *Salmonella enteritidis*, *Pasteurella multocida*, *Coxiella burnetii*, and *Borrelia anserina*).

FIGURE 14-184 Northern mites on the body and escaping from a dead canary (*Serinus canaria*).

Diagnosis

- Northern mite: Includes finding dark colored mites, mite debris (shed skins, dried blood, and mite feces), and masses of eggs (attached to the base of feathers, especially on vent and rump) on live or dead birds. In passerine birds the neck apterium is a particularly useful site to examine for the presence of northern mites. In poultry, the best site is the vent area. Sometimes eggs can be found on the base of the feathers (Fig. 14-186). The tape strip method can be very useful to detect the parasites on a living bird (Fig. 14-187).
- Red mite: Includes finding red-colored mites (red when gorged with blood, white when fastened) during the nighttime on birds and during daytime in hiding places. Red mites are often found accidentally by finding smudges of blood on the underside of perches or furnishing from accidentally crushed parasites when exposed during cleaning (especially detaching perches, feeders, etc.). When facing anemia, respiratory distress, and deaths, especially in nestlings, even without visible presence of parasites on birds, red mites should always be considered as possible culprits.
- Mites (northern and red), their excrements, and dried blood can be found in the nesting material and under the nests. Nestlings may

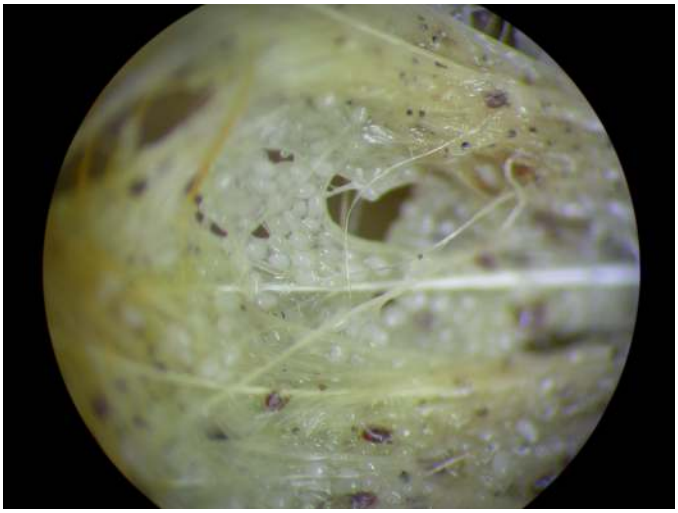


FIGURE 14-185 Northern mite eggs and debris from canary (*Serinus canaria*). Magnification ×10.

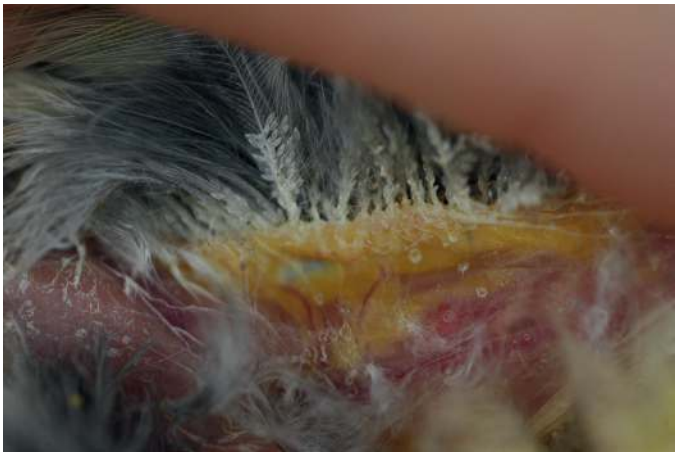


FIGURE 14-186 Northern mite eggs attached to the feather bases of a canary (*Serinus canaria*).

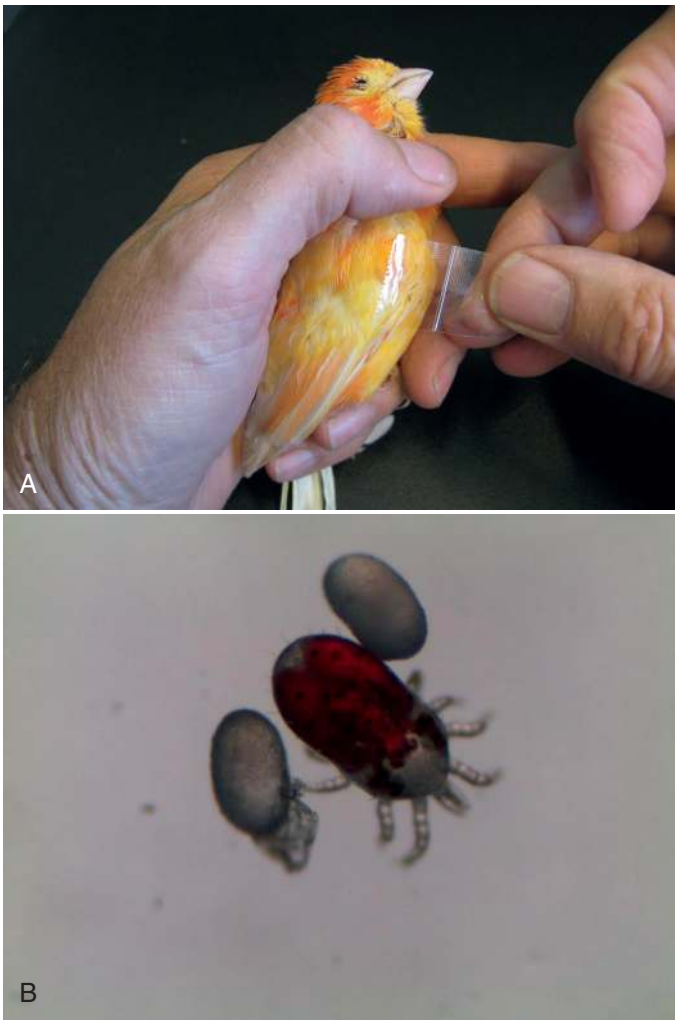


FIGURE 14-187 **(A),** Tape strip method for collection. **(B),** Red mite and eggs under microscope.

show pinprick bites with dried blood, especially under the wings. Some breeders cover the cages with white cloth at night and examine it in the morning for the presence of hidden red mites.

Transmission

Transmission is direct, from bird to bird, by active movement of mites between cages and closely situated aviaries, through fomites but also people, rodents, and wild birds. During heavy infestation the mites can be found everywhere in the aviary (on birds and in their surroundings) and will readily crawl on humans, causing considerable itching. There may be literally an explosion of mites, when in a few days their number can rise into tens of thousands. With red mites this takes place in hot weather conditions (when their eggs hatch in 2 to 3 days). With northern mites predisposing factors include stressful conditions, with a large number of birds grouped in a small area, such as during shows or exhibitions.

Treatment

It is important to acknowledge that the entire life cycle of the northern mite takes place on the host, whether red mites reproduce in the environment, and enter their hosts only for feeding at night (they are photophobic). Northern mites are reported to be able to survive up to 2 months outside their hosts, whereas red mites can survive up to 8 months in their hiding places. Most of the mites will die fast if they are devoid of hiding places that protect them from direct sunlight and in conditions with low humidity (less than 35%).

Treatment of mite infestation should always include both birds and their environment, should be performed scrupulously (it may be necessary to get rid of all furnishings), and often repeatedly to kill larvae and nymphs from recently hatched eggs (which are not affected by chemicals).

Drugs used for treating the birds can be delivered parenterally (intramuscularly and subcutaneously), as a spot-on, dusting, or spraying. A force bath can be added to the drinking or bath water. Drugs include amitraz, avermectins (doramectin, ivermectin, Moxidectin), carbaryl, coumaphos, malathion, phoxim, pyrethroids spinosad, or tetrachlorvinphos. For detailed information on dosages and route of administration of specific substances please consult the formulary in Appendix 6.

Mites can develop resistance against used chemicals. Sulfur, diatomaceous earth, and synthetic amorphous silicas have been used for treatment of mite infestations with good results without development of resistance (different mode of action—causing desiccation of mites through destroying a waxy cuticle).

Prevention

- Prophylactic use of acaricides in the environment and on birds, especially before the breeding season
- Inspection and prophylactic treatment of new birds entering the collection
- Control of mite vectors (infected birds, wild birds, rodents, material, etc.)
- Regular examination of birds for the presence of mites.

FURTHER READING

Altman RB, Clubb SL, Dorrestein GM, et al: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.

Arends JJ: External parasites and poultry pests. In Calnek BW, editor: *Diseases of poultry*, ed 10, London, 1997, Mosby-Wolfe.

Atkinson CT, Nancy J, Thomas NJ, et al: *Parasitic diseases of wild birds*, Hoboken, NJ, 2008, Wiley-Blackwell.

Canon JM: *A guide to basic health and disease in birds: their management, care and well-being: their management, care and breeding*, ed 2, Burleigh, Australia, 2002, ABK Publications.

Chitty J, Lierz M: *BSAVA Manual of raptors, pigeons and passerine birds*, Gloucester, UK, 2008, British Small Animal Veterinary Association.

Coles BH: *Essentials of avian medicine and surgery*, Oxford, UK, 2007, Blackwell Publishing.

Cooper J, Cooper M: *Captive birds in health and disease*, Fordingbridge, Hampshire, 2003, Hancock House Publishers, World Pheasant Association.

Cooper JE: *Birds of prey: health and disease*, ed 3, Oxford, UK, 2002, Blackwell Science.

Doneley R: *Avian medicine and surgery in practice: companion and aviary birds*, Boca Raton, FL, 2010, CRC Press.

Greve JH: Parasites of the skin. In Rosskopf WJ Jr, Woerpel RW, editors: *Diseases of cage and aviary birds*, ed 3, Baltimore, MD, 1996, Williams & Wilkins.

Griener EC, Ritchie BW: Parasites. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.

Harcourt-Brown N, Chitty J: *BSAVA manual of psittacine birds*, ed 2, Gloucester, UK, 2005, British Small Animal Veterinary Association.

Harrison GJ, Lightfoot TL: *Clinical avian medicine*, Palm Beach, FL, 2006, Spix Publishing.

Mitchell M, Tully TN: *Manual of exotic pet practice*, Philadelphia, PA, 2008, Saunders.
Olsen GH, Orosz SE: *Manual of avian medicine*, St Louis, MO, 2000, Mosby.
Orosz S: Avian dermatology. In Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders.
Patisson M, MacMullin PF, Bradbury JM, et al: *Poultry diseases*, ed 6, Philadelphia, PA, 2007, Saunders.
Randall CJ: *Diseases and disorders of the domestic fowl and turkey*, ed 2, London, 1997, Mosby-Wolfe.
Saif YM, Barnes HJ, Glisson JR, et al: *Diseases of poultry*, Hoboken, NJ, 2008, Wiley-Blackwell.
Stroud R: *Diseases of canaries*, Worcestershire, UK, 2006, Home Farm Books.
Tudor DC: *Pigeon health and disease*, Ames, IA, 1991, Iowa State University Press.
Tully TN, Dorrestein G, Jones A: *Handbook of avian medicine*, ed 2, Philadelphia, PA, 2009, Saunders.

15

Reproduction

HOUSING AND HOUSING REQUIREMENTS

Sven Hammer, Christiana Hebel

What is the ideal place for breeding birds in captivity? This section summarizes the housing requirements for breeding birds in captivity. Breeding setups will vary depending on the species, their individual needs, and the breeding center itself. Ideal conditions should always mimic the natural environment. It is important to distinguish between facilities that breed for hobby or commercial purposes and zoologic facilities. However, there are some aspects that need to be considered in any breeding facility. For any breeding program, it is ideal to have a breeding aviary with a separate quarantine station for new arrivals, a nursery for hand-rearing and, ideally, isolation facilities for sick birds. For collections with visitors, it might be best to breed sensitive species off show to avoid disturbance. It might be preferable also to keep surplus birds for exhibition purposes, or to house them at mixed-species aviaries.

GENERAL CONSIDERATIONS

a) Species-specific environmental parameters: temperature (weather protection, climate-controlled heating or air conditioning), humidity, substrate (floor cleaning), vegetation, and perches (different diameters for different species, natural material; some species might need rocks) (Figs. 15-1 and 15-2).
b) Safety, security, and hygiene: predators, diseases, pest control should be addressed on a regular basis; clean water (for drinking and bathing); fresh, clean food (keep in mind the destructive behavior of parrots).
c) Aviary size: As a general rule, the larger the aviary, the better it is for the natural behavior of the bird, but it might also be more difficult to clean and to keep control of the birds. A separate capture area is very important, especially in flock breeding species to reduce stress and disturbance. These capture areas should have visual barriers from the rest of the aviary. Even with pairs, it is very helpful to have a separating compartment that can be used for pairing up or for individual treatment without disturbing the breeding pair partner. Minimum guidelines are often given by national authorities or international breeding programs.
d) Aviary design: Adapted to the natural conditions of the species e.g., a long-distance flying/open-field species need long-shaped aviary with high landing places/perches, whereas rain forest species might need thick bushes and hiding places. Other features to take into consideration when planning breeding aviaries include visual barriers to avoid disturbance, wires of cages (galvanized wires can lead to heavy metal intoxication), perches (diameter, material, location [horizontal-vertical]), and other sitting areas (use of species-specific natural material), bathing facilities, and placement of food bowls so as to avoid urates and fecal contamination. All maintenance and civil engineering access should be from outside of the breeding facility to avoid any disturbance of the bird, even in emergencies such as an electrical breakdown.
e) Nest boxes: Should be built of species-specific material like rock or wood or even (palm) trunks. The shape, location, and size should be adapted to the typical natural nest places particular to the species, and the boxes should be easily accessible to allow frequent control. Nesting material needs to be provided so that the bird is stimulated to build its own nest. Nest boxes (not next to the door) may be placed in such a way as to allow access from the service area/corridor (easier check for nest boxes). The nest box material is essential (falcons, gravel; parrots, plywood), as is the box style for certain species. Always offer several nest places/boxes to give the birds a choice.

Video surveillance is ideal to avoid disturbance (Fig. 15-3). The best method is to place a surveillance camera inside and outside the nest box to observe behavior and to keep a daily record (Figs. 15-4 to 15-7).

Reproductive failure might be the result of inappropriate enclosure design, lack of suitable nesting sites, unsuitable nest positioning, lack of nest building materials, or other necessary environmental requirements (Table 15-1). Partial construction of appropriate nest sites and provision of suitable materials may be vital to initiate breeding. In some species, the key to successful egg laying is having just the right size access hole into the nest box.

BREEDING PAIRS AND FLOCK MANAGEMENT

Sven Hammer, Christiana Hebel

For any successful avian breeding program, a deep knowledge of the biology and natural environmental conditions of the species is essential. To visit and study the natural habitat and to exchange knowledge with field biologists on species that have never bred in captivity is of particular importance. It is also vital to keep a genetically diverse population and to avoid inbreeding. Ideally, parents of the offspring should be known; therefore, detailed record keeping is necessary. The keeping and breeding of endangered species should be an integral part of an international breeding program under a studbook management to preserve genetic diversity.

Closely related species or subspecies should not be housed together, as it might end in hybridization, which should be avoided. In captivity, most birds are not allowed to select their partners, but in some species (cranes), it might be beneficial for producing offspring. Forced pairing can result in decreased fertility, intolerance, or even aggression up to death of one partner. Imprinted birds are not suitable for natural breeding but can be used for artificial insemination.

Breeding birds need to be in the correct group structures (pair, flock), group size, and sex ratio to support natural breeding. Care has

FIGURE 15-1 These climate-controlled breeding aviaries are provided with indoor and outdoor areas.

FIGURE 15-2 Natural trees or palm trunks are ideal nests for certain species in captivity, such as some owl species, hornbills, and woodpeckers.

to be taken when breeding different species in the same aviaries, because this might result in disturbance or stress and decreased breeding success.

Some bird species nest in colonies (in nature, up to thousands of birds); these species often need a minimum flock size if successful breeding is targeted (penguins, flamingos, budgerigars). Mirrors might be helpful to stimulate even small numbers of a colony to breed. Other species are monogamous and rejoin every year, or pair for life (geese, ducks, and falcons) (Fig. 15-8). Especially monogamous species are best bred in separate breeding aviaries to minimize conflicts.

Record keeping and daily control are essential tools, but it must be kept in mind that often a minor disturbance can stop breeding.

FIGURE 15-3 Video camera equipment allows the operator to observe the birds at a distance without disturbance and to take notes on courtship behavior, copulation, egg laying, and the rearing of young.

FIGURE 15-4 Hoopoe (*Upupa epops*) with young inside a tree trunk nest. A video camera was installed on the top of the nest for observation.

Cameras installed in the aviary or nest can be an excellent supportive tool for avoiding direct disturbance while keeping an especially close eye on sensitive species (falcons, parrots).

The main target of the veterinarian regarding breeding management is to maintain a healthy population. Therefore, preventive medicine, regular health checks, and avoiding disease transmission through quarantine measures and/or isolation are the most important factors. The individual bird still plays an important role, especially when it is genetically valuable or is used for artificial insemination. Nutritional variations are essential before the breeding season starts.

It is ideal to keep a breeding group/program as isolated as possible and to allow new birds access only after passing an intensive quarantine and health check survey. A yearly health check out of the breeding season is a good tool and highly recommended. This should be adapted to the needs, problems, and risks that a breeding group/facility/species is facing and should be updated on a yearly basis. The health check should include screenings for diseases (psittacine beak and feather

FIGURE 15-5 Video control desk in a modern falcon breeding facility in the Middle East. Two sets of cameras are installed in each breeding chamber. One is focused on the nest; the second, a pan-tilt-zoom camera, allows the operator in the control room to focus and zoom in on different areas of the chamber with the aid of a joystick. Such a system allows video recording, reviewing at different speeds, and retrieving footage of particular interest at any given date for examination. This particular system is also Internet-based, permitting the operator to observe and monitor breeding activity from any locality in the world. (Courtesy Dr. Jaime Samour.)

TABLE 15-1 Advantage and Disadvantage of the Aviary Location

Location	Advantage	Disadvantage	Comments
Indoors	Controlled light intensity, photoperiod, temperature and humidity, easier pest control	Vitamin D deficiency, air quality control, filters needed, high operational costs	Below standard might increase disease risk
Outdoors	Natural light intensity and photoperiod, natural temperature and humidity, fresh air	Disturbance (public), pest control might be more difficult, ectoparasites as disease vectors	Weather protection (wind, sun, rain, snow) needs to be provided
Combination	See above Main problem: environmental difference from outdoors to indoors, because this increases the risk for infections, especially if high humidity is only outdoors. It is not natural to have significant environmental changes just by passing through a door!		Might be an option if all-year-round outdoor housing cannot be provided due to extreme climate (e.g., winter in Europe, summer in the Middle East)
Free-Ranging	Environmental enrichment, encourages natural behavior, more exercise	Predators might be a risk, spread of disease, difficult control of the birds	Wing clipping or pinioning (in some countries prohibited by law, higher threat by predators)

FIGURE 15-6 An unusual observation, made through a closed-circuit video system, showing a male gyr (*Falco rusticolus*) falcon feeding the hen at the early stages of courtship during the breeding season. The photograph was obtained directly from the monitor in the control room. (Courtesy Dr. Jaime Samour.)

FIGURE 15-7 A pair of gyr (*Falco rusticolus*) falcons copulating, as observed through the monitor in the control room. The pan-tilt-zoom camera zoomed in on the pair for the purpose of obtaining the photograph. The features of the computer program displayed on the screen allow image enhancement and video recording and retrieving. (Courtesy Dr. Jaime Samour.)

disease, polyomavirus, bornavirus, and *Chlamydia psittaci*), a reproductive evaluation (endoscopy of gonads and offspring history), a nutrition evaluation, and preventive medicine measures such as vaccination and parasite control (Fig. 15-9). Ideally an individual health rating system for each bird (e.g., by number score: 1 = very good … 5 = poor) should be established in combination with an individual rating for the group (e.g., color score: red = low priority and high risk for other individuals, green = priority group and no risk for other birds in the breeding program).

Reproductive failure can be the result of a variety of factors: inappropriate management (stress), environmental conditions (climate, aviary design, light management), incorrect diet and/or nutritional deficiency, incorrect age and social structure, wrong pairing, hormonal problems, imprinting, handicap, underlying disease, or infertility (Fig. 15-10).

FIGURE 15-8 A pair of gyr (*Falco rusticolus*) falcons housed in a breeding facility displaying typical monogamous pair bonding. This behavior is accentuated during the courtship period within the breeding season. (Courtesy Dr Jaime Samour.)

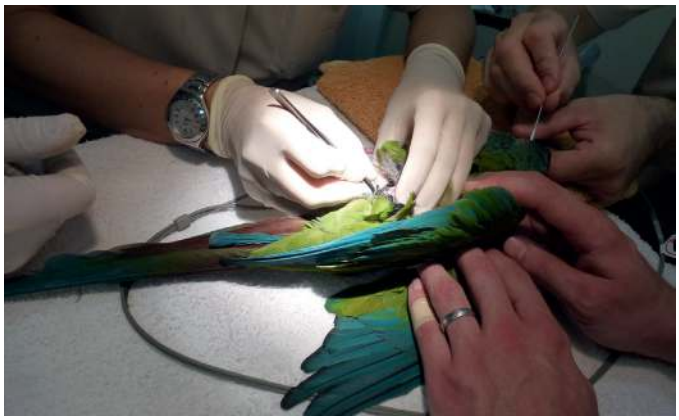


FIGURE 15-9 Health check on a blue headed macaw (*Primolius couloni*). Preparations for endoscopy examination for evaluation of the gonads and for sample collection for health screening.

FIGURE 15-10 Successful breeding in marabou stork (*Leptoptilos crumeniferus*) using the correct sex ratio and social structure and adequate environmental conditions.

SEMEN COLLECTION

Jaime Samour

Burrows and Quinn (1935) first introduced the world to a method for semen collection in avian species by using the domestic fowl (*Gallus domesticus*). Since then, semen collection and artificial insemination have become well-established procedures, not only in the poultry industry, but also within captive breeding programs for both common and endangered nondomesticated species throughout the world.

The sharp decline in certain raptor populations in North America prompted the establishment of programs to breed these species in captivity. The best-known examples of artificial insemination in the propagation of endangered species in captivity include the breeding program established by the Peregrine Fund in the early 1970s as a result of the precarious status of the peregrine falcon in the USA; others include those developed by the International Crane Foundation and the Patuxent Wildlife Research Center in the USA to breed endangered crane species in captivity. The techniques for semen collection and artificial insemination developed by falconers in the 1970s are currently used to breed hybrid falcons on a commercial basis.

Semen samples are usually collected from birds using the cooperative, the massage, and the electrical stimulation methods.

COOPERATIVE METHOD

The cooperative massage was developed primarily by falconers to obtain semen samples from different birds of prey. The technique involves the use of males imprinted onto their handlers. These are birds that are hand-reared in captivity without the presence of conspecifics. It is usually required that the handler interact constantly with the bird by simulating its different calls, presenting food, and mimicking its displays. Later, at the onset of the breeding season, the interaction is intensified and directed to solicit copulation. This is achieved by simulating the courtship ritual, which consists of a complex series of displays and vocalizations. Once the necessary level of conditioning has been achieved, the bird is enticed to copulate on a special hat worn by the handler (Boyd and Schwartz, 1983). The semen is subsequently collected from a rubber ring fitted around the hat or from small wells simulating a honeycomb pattern embedded in custom-made rubber hats (Fig. 15-11).

Conversely, semen samples have been collected from other avian species using a cooperative collection method involving dummy females. For instance, a freeze-dried female fitted with an artificial

FIGURE 15-11 Peregrine falcon (*Falco peregrinus*) "copulating" on a purpose-made rubber hat with numerous wells, where semen is deposited and can be collected. (Courtesy Adrian and Francisco Gonzalez.)

FIGURE 15-12 Male zebra finch (*Taeniopygia guttata*) "copulating" with a freeze-dried dummy female. The dummy is fitted with an artificial cloaca, where the semen is subsequently collected. (Courtesy of Professor T.R. Birkhead.)

FIGURE 15-13 Semen collection from a gyrfalcon (*Falco rusticolus*) male using the massage method. The falcon has been wrapped with a kitchen towel and positioned on a soft towel on the lap of a sitting handler, who holds the bird's legs. The operator massages the lower back and abdomen to collect the semen sample.

cloaca has been successfully used to collect semen samples from individual male zebra finches (*Taeniopygia guttata*) (Pellatt and Birkhead, 1994) (Fig. 15-12). In addition, a slightly different method has been successfully used to collect semen samples from houbara bustards (*Chlamydotis undulata*). During the breeding season, tame hand-reared males housed individually are presented with a female dummy composed simply of a flat wooden platform with a hinged neck covered by a preserved houbara bustard skin, including the head. The dummy is presented to a sexually active displaying male, who soon begins the courtship display and copulates with the dummy. The semen is collected by the attending operator in a Petri dish placed underneath the male (Saint Jalme, *et al.*, 1994). Dummies and teaser females also have been used successfully for semen collection in ostriches (*Struthio camelus*) (Rybnik, *et al.*, 2007). The authors emphasized the need for individual male ostriches to learn how to mount a dummy. The success of using teaser females to collect semen samples depends largely on the crouching behavior of the teaser female, the temperament of male ostriches, and the acceptance of the semen collector by the male ostrich (Rybnik, *et al.*, 2007).

MASSAGE METHOD

The massage method was originally designed for the collection of semen samples from the domestic fowl (Burrows and Quinn, 1935), but slightly modified versions have been applied successfully in other breeding programs. In birds of prey, the bird is usually wrapped with a soft towel and placed on its chest on a padded table or is held upside down while the legs are firmly held by an operator (Fig. 15-13). Semen collection begins with a series of strokes applied to the lower back and abdomen carried out for approximately 1 or 2 minutes. Subsequently, the strokes are directed laterally to the cloaca until the handler exposes the papillae of the ductus deferentis. At this point, milking strokes are applied until the semen appears at the tip of the papillae (Weaver, 1983). This is where the method gets its other commonly used name, "stripping." This procedure is repeated several times until enough semen has been collected (Fig. 15-14). For semen collection in American kestrels (*Falco sparverius*), birds are not held as previously described. In this species, probably due to their small size, male kestrels are restrained by a gloved hand and the body rested on a block, leaving the wings free to flap as would be normal for males during natural copulation (Bird and Laguë, 1977). Conversely, semen samples are commonly obtained from other species such as cranes while restrained manually by an operator and maintained in a standing position. Semen samples are easily obtained from some passerine species by gently pressing the "promontory," a small paired protuberance located on either side of the cloaca (Fig. 15-15). These structures correspond to the seminal glomera, a paired structure formed by convolutions of the terminal portion of the ductus deferens, located on either side of the proctodeum (Wolfson, 1952). Semen samples can be collected from budgerigars (*Melopsittacus undulatus*) using a similar method (Samour, *et al.*, 1986), because this species also possesses seminal glomera (Samour, *et al.*, 1988) (Fig. 15-16).

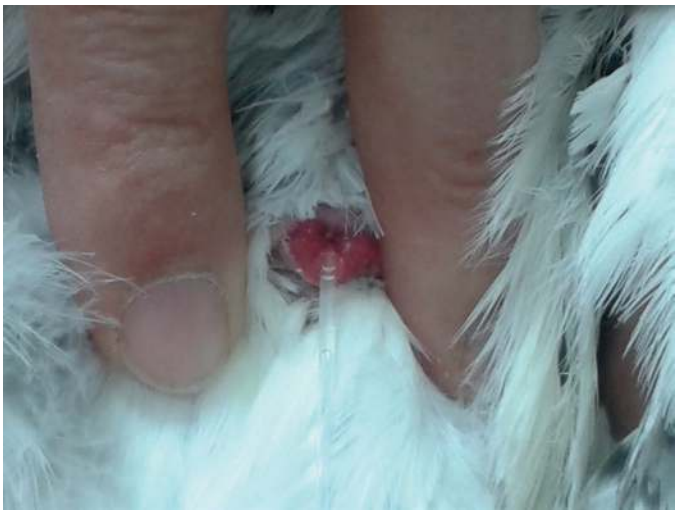


FIGURE 15-14 Semen sample collected from the falcon mentioned above. Samples are collected into capillary tubes or cannulae directly from the cloaca. Contamination with urates and/or feces should be avoided at all times.

Modifications to the massage semen collecting method to accommodate differences in the size and nature of a species have recently been described for the following: blue rock pigeon (*Columba livia*) (Sontakke, *et al.*, 2004); piping guan (*Pipile cumanensis cumanensis*)

FIGURE 15-15 Semen collection from a house sparrow (*Passer domesticus*) using the massage method. This species, in common with other Passeriformes and the budgerigar (*Melopsittacus undulatus*), possesses seminal glomera. Semen can be obtained from this and similar species by pressing gently on both sides of the cloaca during the nuptial phase of the gonadal cycle. Note the normal pasty appearance and the brown coloration of the semen.

FIGURE 15-16 Semen sample collection from a budgerigar (*Melopsittacus undulatus*). The bird has been placed in a rubber mask commonly used for anesthesia in small-animal practice. This is used to ease the handling and semen collection procedures. The sample is collected using microcapillary tubes. Semen is simply expressed out by pressing gently on either side of the cloaca.

(DeMatteo, *et al.,* 2004); white-backed vulture (*Gyps bengalensis*) (Umapathy, *et al.,* 2005); various psittacine species (Stelzer, *et al.,* 2005); blue-fronted parrots (*Amazona aestiva aestiva*) (Della Volpe, *et al.,* 2011), rockhopper penguin (*Eudyptes chrysocome*) (Waldoch, *et al.,* 2007; Waldoch, *et al.,* 2012) and cockatiels (*Nymphicus hollandicus*) (Neumann, *et al.,* 2013).

A novel technique for collecting semen samples from quail (*Coturnix japonica*) was recently described using a teaser female. The technique involves exposing males to a female but preventing copulation. When males are at the peak of excitement, they are removed from the cage, and semen is expressed out by applying gentle pressure to both sides of the cloaca (Chelmońska, *et al.,* 2008).

The use of oxytocin before semen collection to enhance semen volume, spermatozoa concentration, motility, and percentage of live spermatozoa was recently described in ostriches (Suttiyotin, *et al.,* 2012). Significant increments in all parameters studied were observed after the injection of 5IU of oxytocin via the phallic vein. The use of oxytocin to enhance seminal characteristics before collection is a well-established procedure in domesticated animals such as bulls (Palmer, *et al.,* 2004), dogs (Traas and Kustriz, 2004), and rams (Bozkurt, *et al.,* 2007).

ELECTRICAL STIMULATION METHOD

The first study on collecting semen from avian species using electrical stimulation seems to have been reported in waterfowl by Serebrovskii and Sokolovskaja (1934). In this study, the positive pole of the stimulator was attached to the skin over the synsacral area of ducks while the negative pole was immersed in water. The duck's bill was also immersed in water. Strong electrical shocks of 80 volts each of 3 to 4 seconds' duration with 1- to 2-second intervals were applied to collect semen samples. Semen samples were also collected from drakes by connecting the positive pole to a needle attached to the skin of the synsacral region while the negative pole was inserted into the cloaca. Thirty volts was applied for 3 seconds and repeated three to five times at intervals of 5 seconds (Watanabe, 1957). More refined methods continued developing over the years. For instance, semen samples were obtained from Peking ducks (*Anas platyrhynchos*) and Muscovy ducks (*Cairina moschata*) by using a single probe for the first time. This was inserted into the cloaca, and 25 to 30 volts was applied for 6-second periods, then repeated after 5-second intervals. The authors concluded that semen samples collected by electrical stimulation were of better quality than those obtained by massage (Csuka, *et al.,* 1977).

Similar techniques for semen collection using electrical stimulation have been subsequently used over the years with single probes fitted with 2 or 3 ring electrodes or 2 or 3 longitudinal strip electrodes mounted onto plastic or polypropylene probes. This has been used with domestic fowl (Kono and Hiura, 1983) and waterfowl, including mallard ducks, Muscovy ducks, and Hawaiian geese (*Branta sandvicensis*) (Samour, *et al.,* 1985). Semen samples were collected from the above-mentioned waterfowl species under anesthesia. More recently, semen samples were collected from Siamese domestic cockerels (*Gallus gallus*) (Kanatiyanont, *et al.,* 2012) and different species of psittacines (Lierz, *et al.,* 2013) using electrical stimulation. In both studies, plastic probes fitted with parallel longitudinal strip electrodes were used (Fig. 15-17). Cockerels undergoing semen collection using electric stimulation were subjected to anesthesia using isoflurane throughout the procedure (Kanatiyanont, *et al.,* 2012), whereas anesthesia was not used in the work with psittacine birds (Lierz, *et al.,* 2013). A feature in this study with psittacines was the incorporation of wells on both sides of the probe for the placement of microcapillary tubes for direct flow of semen into the tubes (Lierz, *et al.,* 2013). Although this technique seems to be practical for the collection of semen, it could also allow the collection of urates and fecal material, making the sample unsuitable for insemination. This type of probe has been superseded by a relatively new design incorporating a wide handle compressed laterally to ease the handling procedure and a short, round steel stem containing one pole and a single steel ball at the end with the second pole. The ball at the end of the probe ensures that no mechanical damage to the cloacal tissue occurs during placement (see Fig. 15-17). The probe is then connected to the electroejaculation unit, which consists mainly of a potentiometer, an analog voltmeter to display the voltage intensity, and a foot pedal (Fig. 15-18), which allows the operator to use both hands to hold the probe in place and to collect the sample (Fig. 15-19) while the second operator concentrates on handling the bird (Daniel Neumann, personal communication). It has been postulated that the

FIGURE 15-17 A new recently designed probe integrates a wide handle compressed laterally to ease handling; a short, round stem containing one pole; and one steel ball at the end containing the second pole. The ball prevents mechanical damage to the delicate tissues of the cloaca during placement. (Courtesy Parrots Reproduction Consulting.)

FIGURE 15-18 The main electroejaculation unit consists mainly of a potentiometer, a voltmeter with an analog display of the voltage intensity, and an operating foot pedal to provide hands-free operation of the unit during the semen collection procedure. (Courtesy Parrot Reproduction Consulting.)

best way to collect semen samples using electroejaculation from birds is to introduce the probe into the cloaca, carry out the electrical stimulation as indicated for the species, remove the probe, and then to provide an abdominal massage similar to that described above and to collect the semen sample. This method is very similar to that used in the first published study on the use of electroejaculation in psittacine birds (Harrison and Wasmund, 1983); the technique is similar also to the method currently used successfully in psittacine birds (Daniel Neumann, personal communication).

FIGURE 15-19 Semen collection from an African grey parrot (*Psittacus erithacus*) using the new probe design and a hands-free operation electroejaculation unit. (Courtesy Parrot Reproduction Consulting.)

In a previous study with waterfowl, birds were wrapped in a soft towel, and a single probe fitted with two bronze rings acting as electrodes was inserted into the cloaca. The probe was connected to a potentiometer and a voltmeter with a range of 0 to 30 volts. Stimulation started with pulses of 5 volts, each lasting 3 seconds at 5-second intervals. The pulse was given five to eight times, after which the voltage was increased in 5-volt stages up to 30 volts (Samour, *et al.*, 1985).

Acknowledgments: I am most grateful to Daniel Neumann from Parrot Reproduction Consulting for unselfishly sharing the photographic material on the new electroejaculation system and for providing invaluable information on its use.

REFERENCES

Boyd LL, Schwartz CM: Training imprinted semen donors. In Weaver JD, Cade TJ, editors: *Falcon propagation: a manual of captive breeding*, Boise, Idaho, 1983, The Peregrine Fund Inc.

Bozkurt T, Turk G, Gur S: Effects of exogenous oxytocin on serologic and seminal steroids and semen characteristics in rams, *Turk J Vet Anim Sci* 31:303–309, 2007.

Burrows WH, Quinn JP: A method of obtaining spermatozoa from the domestic fowl, *Poultry Sci* 14(251):253–254, 1935.

Chelmońska B, Jerysz A, Łukaszewicz E, et al: Semen collection from Japanese Quail (Coturnix japonica) using a teaser female, *Turk J Vet Anim Sci* 32:19–24, 2008.

Csuka J, Stasko J, Spronc A, et al: Odber semena od kacerov metodou elektroegakulacie, *Hydinarstvo, Ivanka pri Dunaji* 16:99–108, 1977.

Della Volpe A, Schmidt V, Krautwald-Junghanns ME: Attempted semen collection using the massage technique in blue fronted amazon parrots (*Amazona aestiva aestiva*), *J Avian Med Surg* 25(1):1–7, 2011.

DeMatteo KE, Karagiannis KL, Asa CS, et al: Semen collection and artificial insemination in the common piping guan (*Pipile cumanensis cumanensis*): potential applications for Cracidae (Aves: Galliformes), *J Avian Med Surg* 35(4):447–458, 2004.

Harrison GJ, Wasmund D: *Preliminary studies of electroejaculation to facilitate manual semen collection in psittacines. Proceedings of the Annual Conference of the Association of Avian Veterinarians,* 1983, pp 207–213.

Kanatiyanont N, Kornkaewrat K, Suthanmapinunt P, et al: Effect of semen collection techniques on semen quality and sperm motility parameters in Siamese fighting cocks (*Gallus gallus*), *Thai J Vet Med* 42(4):439–445, 2012.

Kono K, Hiura Y: Semen collection by rectal electroejaculation of the domestic fowl, *Japanese Poult Sci* 4(20):267–270, 1983.

Lierz M, Reinschmidt M, Müller H, et al: A novel method for semen collection and artificial insemination in large parrots (Psittaciformes), *Scientific Reports* 3:2066, 2013.

Neumann D, Kaleta EF, Lierz M: Semen collection and artificial insemination in cockatiels (*Nymphicus hollandicus*)—a potential model for psittacines, *Tierarztl Prax Ausg K Kleintiere Heimtiere* 41(2):101–105, 2013.
Palmer CW, Amundson SD, Brito LFC, et al: Use of oxytocin and cloprostenol to facilitate semen collection by electroejaculation or transrectal massage in bulls, *Anim Reprod Sci* 80:213–223, 2004.
Pellatt EJ, Birkhead TR: Ejaculate size in zebra finches (*Taeniopygia guttata*) and a method for obtaining ejaculates from passerine birds, *Ibis* 136:97–106, 1994.
Rybnik PK, Horbanczuk JO, Naranowicz H, et al: Semen collection in the ostrich (*Struthio camelus*) using a dummy or a teaser female, *Brit Poultry Sci* 48(5):635–643, 2007.
Saint Jalme M, Gaucher P, Paillat P: Artificial insemination in houbara bustards (*Chlamydotis undulata*); influence of the number of spermatozoa and insemination frequency on fertility and ability to hatch, *J Reprod Fertil* 100:93–103, 1994.
Samour J, Smith CA, Moore HDM, et al: Semen collection and spermatozoa characteristics in budgerigar (*Melopsittacus undulatus*), *Vet Rec* 118:397–399, 1986.
Samour J, Spratt DMJ, Holt WV, et al: Ultrastructure and secretory nature of the seminal glomus in budgerigars (*Melopsittacus undulatus*), *Res Vet Sci* 45:195–197, 1988.
Samour J, Spratt DMJ, Hutton RE, et al: Studies on semen collection in waterfowl by electrical stimulation, *Brit Vet J* 141(3):265–268, 1985.
Serebrovskii AS, Sokolovskaja II: Electro-ejaculation in birds, *Probl Zivotnovodsva* 5:57–59, 1934.
Sontakke SD, Umapathy G, Sivaram V, et al: Semen characteristics, cryopreservation and successful artificial insemination in the blue rock pigeon (*Columba livia*), *Theriogenology* 62:139–153, 2004.
Stelzer G, Crosta L, Bürkle M, et al: Attempted semen collection using the massage technique and semen analysis in various psittacine species, *J Avian Med Surg* 19(1):7–13, 2005.
Suttiyotin P, Khumpim P, Kimsakulvech S, et al: Improvement of semen quantity and quality in ostriches by oxytocin injection before semen collection, *Suranare J Sci Technol* 19(1):9–14, 2012.
Traas AM, Kustriz MV: Effect of administrating oxytocin or prostaglandin F2 Alpha on characteristics of the canine ejaculate, *Canadian Vet J* 45:999–1002, 2004.
Umapathy G, Sontakke S, Reddy A, et al: Semen characteristics of the captive Indian white-backed vulture (*Gyps bengalensis*), *Biol Reprod* 73:1039–1045, 2005.
Waldoch J, Root T, Dubach JM, et al: Semen characteristics and artificial insemination in rockhopper penguins (*Eudyptes chrysocome chrysocome*), *Zoo Biol* 31:166–180, 2012.
Waldoch J, Root T, Ramer J, et al: Semen collection and characterization in rockhopper penguins (*Eudyptes chrysocome chrysocome*), *J Zoo Wildlife Med* 38:13–17, 2007.
Watanabe M: An improved technique of the artificial insemination of ducks, *J Fac Fish Anim Husbandry*, Hiroshima University 1:363–370, 1957.
Weaver JD: Artificial insemination. In Weaver JD, Cade TJ, editors: *Falcon propagation: a manual of captive breeding*, Boise, Idaho, 1983, The Peregrine Fund Inc.
Wolfson A: The cloacal protuberance—a means for determining breeding condition in live male passerine birds, *Bull Chic Acad Sci* 10:1–23, 1952.

SEMEN QUALITY ASSESSMENT

Jaime Samour

The study of the different aspects of the basic reproductive physiology of an individual species is essential for the implementation of artificial insemination programs. The analysis of semen samples forms part of the assessment of the reproductive potential of males and should be carried out on all individuals selected to participate in artificial breeding programs as semen donors. The most important assays to be carried out in samples include semen volume, color, and density. Additional analyses may be required for the preparation of suitable diluents for artificial insemination. These should also include pH, osmolarity, Na^+, Cl^-, K^+, and glucose and fructose concentration.

VOLUME

Semen volume in birds can be estimated by collecting samples using graduated glass microcapillary tubes (Stelzer, *et al.,* 2005) or by using calibrated positive displacement pipettes (Umapathy *et al.,* 2005).

Semen volume obtained from avian species ranges from 300 µL in the rockhopper penguin (*Eudyptes chrysocome chrysocome*) (Waldoch, *et al.,* 2012); 1.09 ± 0.13 mL in ostriches (*Struthio camelus*) (Rybnik, *et al.,* 2007); 0.37 ± 0.26 mL in the Indian white-backed vulture (Umapathy, *et al.,* 2005); 0.1 to 2.0 µL in various psittacine species (Stelzer, *et al.,* 2005); 10.5 ± 2.6 µL in the domestic pigeon (*Columba livia*) (Sontakke, *et al.,* 2004); 23.8 µL in the piping guan (*Pipile cumanensis cumanensis*) (DeMatteo, *et al.,* 2004); 0.08 ± 0.05 mL in the North African houbara bustard (*Chlamydotis undulata undulata*) and 0.07 ± 0.05 mL in the Asian houbara bustard (*Chlamydotis undulata macqueenii*) (Saint Jalme, *et al.,* 1994); 3.5 to 13 µL in the budgerigar (*Melopsittacus undulatus*) (Samour, *et al.,* 1986a); 0.3 ± 0.1 mL in the Muscovy duck (*Cairina moschata*); 0.15 ± 0.05 mL in the mallard duck (*Anas platyrhynchos*); and 0.15 ± 0.05 mL in the Hawaiian geese (*Branta sandvicensis*) (Samour, *et al.,* 1985). Other studies reported semen volumes ranging from 4 ± 1 to 20 ± 2 µL in the American kestrel (*Falco sparverius*) (Bird and Laguë, 1977); 1.02 ± 0.30 to 2.78 ± 1.17 µL in the Quaker parrot (Anderson, *et al.,* 2002); and 35.6 ± 12.1 µL in the Magellanic penguin (*Spheniscus magellanicus*) (O'Brien, *et al.,* 1999). In the peregrine falcon (*Falco peregrinus*), the volume of semen samples collected was estimated at 95.4 ± 51.7 µL (mean ± standard deviation) (Parks, *et al.,* 1986).

COLOR

The color of semen samples is directly related to the concentration of spermatozoa and the volume of seminal plasma contained in the sample. In the American kestrel, the color of semen samples has been classified into different numeric categories ranging from 0 = opaque, 1 = clear, 2 = very pale amber, 3 = pale amber, 4 = amber, and 5 = deep amber (Bird and Laguë, 1977). Conversely, the color of semen samples from Muscovy and mallard ducks and the Hawaiian geese has been denoted as milky-cream to milky (Samour, *et al.,* 1985), and in piping guans from cloudy-white to yellow (DeMatteo, *et al.,* 2004). Estimating the color of semen samples can only be determined reliably with the use of a color scale, because this parameter is subject to operator prejudice.

DENSITY

Density is a parameter that refers to the appearance and consistency of semen samples. For instance, semen samples from passerine are highly dense and comprise a rather packed mass of spermatozoa with very little to no seminal plasma (Wolfson, 1960), whereas semen samples from most avian species are more watery in consistency. In a recent observation by the author, semen samples collected from common house sparrows (*Passer domesticus*) were pale to medium dark-brown in coloration, with a volume ranging from 0.5 to 1.5 µL and typically showing a consistency similar to paste. Semen samples from piping guans have been described as watery to thick in consistency (DeMatteo, *et al.,* 2004). Nevertheless, this parameter is nowadays very seldom used to evaluate semen samples from birds, because it is difficult to quantify and subject to operator judgment.

ANCILLARY SEMEN ANALYSES

The pH, osmolarity, Na, Cl, K, and glucose/fructose are basic biochemical components of the seminal plasma and should form part of our knowledge of the basic reproductive biology in any species. Our understanding of these parameters is vital for the selection or the production of diluents suitable for artificial insemination using either freshly collected or frozen-thawed semen samples.

pH

The pH in semen samples can be estimated in whole semen samples, but estimating it in seminal plasma is preferable. This can be obtained by centrifugation of the samples at 15000 *g* for 2 minutes (Samour, *et al.,* 1986b). The pH of the samples is estimated by using pH indicator strips or a dedicated pH benchtop analyzer. In the budgerigar (*Melopsittacus undulatus*), the pH of seminal plasma was estimated at 8.20 ± 0.05 (mean ± standard error of mean) (Samour, *et al.,* 1986b). More recently, pH value from semen samples from the blue-naped parrot (*Tanygnathus lucionensis*) was estimated at 9.5; for the golden-capped conure (*Aratinga auricapilla*), it was estimated at 8.9 (Stelzer, *et al.,* 2005). The pH in uncontaminated semen samples collected from captive Indian white-backed vulture (*Gyps bengalensis*) was estimated at 7.1 ± 0.21 (mean ± standard deviation) (Umapathy, *et al.,* 2005), whereas pH of semen samples originated from Bonelli's eagle (*Hieraaetus fasciatus*) was 6.86, and the imperial eagle's (*Aquila adalberti*) was 6.8 (Blanco, *et al.,* 2002). The pH of semen samples from different species of cranes has been determined. This includes 7.5 ± 0.5 for the greater sandhill crane (*Grus canadensis tabida*) 8.0 ± 0.1 for the Florida sandhill crane (*G. c. pratensis),* 8.0 ± 0.1 for the Mississippi sandhill crane (*G. c. pulla*) and 8.0 ± 0.1 for the whooping crane (*G. americana*) (Gee, *et al.,* 1985). The pH of semen samples from domestic pigeons (*Columba livia*) was estimated at 6.8 ± 0.2 (mean ± standard deviation) (Sontakke, *et al.,* 2004), whereas the pH in semen samples collected from the seaside sparrow (*Ammodramus maritimus*) ranged from 6.0 to 6.4 (Gee and Sexton, 1983).

The temperature in the seminal glomera of passeriformes is 4° to 5°C lower than the body temperature (Wolfson, 1954). Similarly, budgerigars possess seminal glomera (Samour, *et al.,* 1988a), and spermatozoa is presumably stored under the same conditions. Lower temperatures could increase metabolic activity of the spermatozoa and subsequent production of CO_2. Furthermore, at the time of copulation, a loss of CO_2 may occur at the time of semen transfer to the cloaca of the hen. This may be due to the fact that budgerigars do not possess relatively long papillae of the vasa deferentia to afford a more direct transfer of the semen into the oviduct. Instead, it is assumed that semen is exposed to the cloacal environment, where diffusion of CO_2 could take place, as well as to contaminants from cloacal fluids. Both factors may contribute to the alkalinity of the semen in budgerigars. This may be similar in the blue-naped parrot and the golden-capped conure, because semen samples collected from both species have shown a degree of alkalinity (Stelzer, *et al.,* 2005). This theory has not been fully investigated to date.

The determination of the pH in semen samples is vital in the selection or preparation of diluents, because studies in the budgerigar have shown that the spermatozoa had greater motility in media of pH range 7.8 to 8.2 and remained inactive in more acidic media (Samour, *et al.,* 1986b).

Osmolarity

Osmolarity can be measured in whole semen samples, but it is always preferable to carry out this assay using seminal plasma. Osmolarity is commonly measured using a vapor pressure osmometer due to the small sample size required for the analysis. The osmolarity of semen samples in the seaside sparrow was estimated at 334 mOs/kg (Gee and Sexton, 1983), in the greater sandhill crane at 296 ± 32 mOs/kg, in the Florida sandhill crane at 302 ± 12 mOs/kg, in the Mississippi sandhill crane at 311 ± 17 mOs/kg, and in the whooping crane at 270 ± 36 mOs/kg (Gee, *et al.,* 1985). In addition, osmolarity of semen samples in Aleutian Canada geese was 270 ± 40 mOs/kg (Gee and Sexton, 1990), in the Magellanic penguin was 415.3 ± 6.4 mOs/kg (O'Brien, *et al.,* 1999), and in the domestic pigeon was 340 ± 1.2 mOs/kg (Sontakke, *et al.,* 2004). The osmolarity of the seminal plasma in the budgerigar was estimated at 329.9 mOs/kg (Samour, *et al.*, 1986b).

Na^+, Cl^-, K^+

Sodium, K^+, and Cl^- have been estimated in seminal samples from the budgerigar (Samour, *et al.,* 1986b). In this study Na^+ and K^+ were estimated using a FLM3 flame photometer, whereas Cl^- was estimated using a chloride titrator (Samour, *et al.,* 1986b).

In the seminal plasma of the budgerigar, the concentration of Na^+ was 158.6 ± 8.4 mEq/L, the Cl^- was 109.2 ± 0.18 mEq/L, and the K^+ was 16.39 ± 6.24 mEq/L (Samour, *et al.,* 1986b). In the budgerigar the Na^+ value of 158.6 ± 8.4 was very similar to the value of 150 mEq/L in the blood plasma (Samour, *et al.,* 1986b). Similar relationships have been reported in domestic fowl (Lake, *et al.,* 1958). The Na^+ and K^+ values in the budgerigar were within the range of values reported for the domestic fowl. However, the K^+ value in seminal plasma of 16.39 mEq/L in the budgerigar was higher than 2.9 mEq/L in blood plasma (Samour, *et al.,* 1986b). The Cl^- value of 109.2 mEq/L in the seminal plasma of the budgerigar was slightly lower than the value of 121 mEq/L in the blood plasma (Samour *et al.,* 1986b), but it was similar to the value reported in the blood plasma of the domestic fowl (Lake and El Jack, 1964).

Glucose/Fructose

Glucose and fructose were calculated in seminal plasma samples from budgerigars. These were estimated using an ultraviolet spectrophotometer at a wavelength of 340 nm using commercially available kits. The glucose concentration in the seminal plasma of budgerigars was estimated at 4.25 ± 0.96 mmol/L (mean ± standard deviation), which was significantly lower than the 21.6 mmol/L in the blood plasma. The higher content of glucose in blood plasma in relation to that in the seminal plasma of budgerigars is similar to the data reported for the domestic fowl (Lake, *et al.,* 1958).

The fructose concentration in the seminal plasma of the budgerigar was estimated at 0.59 ± 0.29 mmol/L (Samour, *et al.,* 1986b). Domestic fowl spermatozoa are able to convert glucose to fructose (Lorenz, 1958). Therefore the fructose content in the seminal plasma of the budgerigar may be the product of the metabolic activity of the spermatozoa and the subsequent conversion of glucose to fructose.

REFERENCES

Anderson SJ, Bird DM, Hagen MD: Semen characteristics of the Quaker parakeet (*Myiopsitta monachus*), *Zoo Biol* 21:507–513, 2002.

Bird DM, Laguë PC: Semen production of the American kestrel, *Can J Zoolog* 55:1351–1358, 1977.

Blanco JM, Gee GF, Wildt DE, et al: Producing progeny from endangered birds of prey: treatment of urine-contaminated semen and a novel intramagnal insemination approach, *J Zoo Wildlife Med* 33(1):1–7, 2002.

DeMatteo KE, Karagiannis KL, Asa CS, et al: Semen collection and artificial insemination in the common piping guan (*Pipile cumanensis cumanensis*): potential applications for Cracidae (Aves: Galliformes), *J Avian Med Surg* 35(4):447–458, 2004.

Gee GF, Sexton TJ, et al: Seaside sparrow sperm preservation. In Quay TL, editor: *The seaside sparrow, its biology and management*, 1983, North Carolina Biology Survey, pp 163–165.
Gee GF, Sexton TJ: Cryopreservation of semen from the Aleutian Canada goose, *Zoo Biol* 9:361–371, 1990.
Gee GF, Bakst MR, Sexton TJ: Cryogenic preservation of semen from the greater sandhill crane, *J Wildlife Manage* 49(2):480–484, 1985.
Lake PE, El Jack MH: *Further observations on the chemical composition of the seminal plasma of the domestic cock. Proceedings of the 5th International Congress of Animal Reproduction and Artificial Insemination,* Trento, Sec II, 1964, pp 359–363.
Lake PE, Butler EJ, McCallum JW, et al: A chemical analysis of the seminal and blood plasma of the cock, *Q J Exp Physiol* 43:309–313, 1958.
Lorenz FW: Carbohydrate metabolism of cock spermatozoa, *Nature* 182:397–398, 1958.
O'Brien JK, Oehler DA, Malowski P, et al: Semen collection, characterization and cryopreservation in a Magellanic penguin (*Spheniscus magellanicus*), *Zoo Biol* 18:199–214, 1999.
Parks JE, Heck WR, Hardaswick V: Cryopreservation of peregrine falcon semen and post-dialysis to remove glycerol, *J Raptor Res* 20(1):16–20, 1986.
Rybnik PK, Horbanczuk JO, Naranowicz H, et al: Semen collection in the ostrich (*Struthio camelus*) using a dummy or a teaser female, *Brit Poultry Sci* 48(5):635–643, 2007.
Saint Jalme M, Gaucher P, Paillat P: Artificial insemination in houbara bustards (*Chlamydotis undulata*); influence of the number of spermatozoa and insemination frequency on fertility and ability to hatch, *J Reprod Fertil* 100:93–103, 1994.
Samour J, Baggott GK, Williams G, et al: Seminal plasma composition in budgerigars (*Melopsittacus undulatus*), *Comp Biochem Physiol* 84a(4):735–738, 1986b.
Samour J, Smith CA, Moore HDM, et al: Semen collection and spermatozoa characteristics in budgerigar (*Melopsittacus undulatus*), *Vet Rec* 118:397–399, 1986a.
Samour J, Spratt DMJ, Holt WV, et al: Ultrastructure and secretory nature of the seminal glomus in budgerigars (*Melopsittacus undulatus*), *Res Vet Sci* 45:195–197, 1988a.
Samour J, Spratt DMJ, Hutton RE, et al: Studies on semen collection in waterfowl by electrical stimulation, *Brit Vet J* 141(3):265–268, 1985.
Sontakke SD, Umapathy G, Sivaram V, et al: Semen characteristics, cryopreservation and successful artificial insemination in the blue rock pigeon (*Columba livia*), *Theriogenology* 62:139–153, 2004.
Stelzer G, Crosta L, Bürkle M, et al: Attempted semen collection using the massage technique and semen analysis in various psittacine species, *J Avian Med Surg* 19(1):7–13, 2005.
Umapathy G, Sontakke S, Reddy A, et al: Semen characteristics of the captive Indian white-backed vulture (*Gyps bengalensis*), *Biol Reprod* 73:1039–1045, 2005.
Waldoch J, Root T, Dubach JM, et al: Semen characteristics and artificial insemination in rockhopper penguins (*Eudyptes chrysocome chrysocome*), *Zoo Biol* 31:166–180, 2012.
Wolfson A: Sperm storage at lower than body temperature outside the body cavity in some Passerine birds, *Science* 120:68–71, 1954.
Wolfson A: The ejaculate and the nature of coition in some passerine birds, *Ibis* 102:124–125, 1960.

SPERMATOZOA QUALITY ASSESSMENT

Jaime Samour

SPERMATOZOA CONCENTRATION

Spermatozoa concentration, commonly known as sperm count, measures the number of spermatozoa contained in semen samples. The count can be obtained manually using a cell counting chamber or electronically using an automated analytical system.

MANUAL

There are several types of cell counting chambers commonly used to determine sperm concentration in semen samples. The most frequently used is the hemocytometer or Neubauer chamber (Fig. 15-20). The original hemocytometer was invented by Louis-Charles Malassez (1842-1909), a French anatomist and histologist who originally designed it to count blood cells. This hemocytometer was developed further by Professor Otto Neubauer (1874-1957), a German biochemist and physician, so the hemocytometer was designated the Improved Neubauer counting chamber. The Neubauer chamber consists of a thick crystal slide measuring 30 mm wide by 70 mm long and 4 mm thick. This slide is provided with a central vertical depression containing two counting grids. The chamber is formed by covering the counting area with a dedicated thick coverslip. It is a common practice among laboratory technicians to breathe over the counting chamber before applying the coverslip to ensure the formation of Newton rings, which indicate adequate attachment on both contact areas. The sample must be diluted to a concentration that allows adequate counting.

The Neubauer chamber was used to estimate spermatozoa concentration in semen samples from budgerigars (Samour *et al.,* 1986a). In this study, semen samples were diluted 1:800 with formal citrate solution containing 2.9 g of trisodium citrate dihydrate and 0.1 mL of formaldehyde 40% solution in 100 mL deionized water (Dott and Foster, 1975). In the Magellanic penguin, semen samples were diluted, 1 µL of semen in 9 µL of 4% saline solution (O'Brien *et al.,* 1999). In peregrine falcons, spermatozoa concentration was estimated by diluting semen samples 1:3 with a cryopreservation medium (Lake and Stewart, 1978) and then diluted 1:1 with a 4% glutaraldehyde solution (Parks *et al.,* 1986). To allow adequate counting, the dilution factor in any avian species depends entirely on the number of spermatozoa in the semen sample. The counting chamber is filled up by capillary action using either a microcapillary tube or a pipette. It is always recommended to line a Petri dish with a filter paper and to wet it using a small amount of distilled water. Break off the wooden handle of a bacteriology swab and position two pieces, approximately 6 cm long, on either side of the dish. Rest the loaded chamber on the sticks and store within the Petri dish to avoid dehydration of the sample; then

FIGURE 15-20 The Improved Neubauer Counting Chamber has been widely used over the years to determine the spermatozoa concentration in semen samples. (Courtesy Dr. Melodiya Magno.)

wait for about 5 minutes before proceeding with the counting. Spermatozoa concentration is determined by counting the number of spermatozoa contained in 25 groups of 16 small squares at the four corners and in squares in the central area of the chamber. The concentration is calculated using the following formula:

$$\text{Spermatozoa concentration} = \frac{\text{Number of cells} \times 10{,}000}{\text{Number of squares} \times \text{dilution}}$$

Another very popular type of counting chamber is the Makler. At 10 microns deep, this is the shallowest of all counting chambers. The semen sample is immersed in a tube containing hot water at 50° to 60°C to immobilize the spermatozoa. A drop of the undiluted semen sample is placed in the chamber. Counting is performed as previously described for the Neubauer chamber or hemocytometer.

Other less commonly used counting chambers include the Fuchs-Rosenthal, Petroff-Hausser, Burker-Turk, and Thomas counting chambers.

The spermatozoa concentration in American kestrels (*Falco sparverius*) was estimated from 8 ± 2 to 53 ± 7 × 10^3/mm^3 (mean ± standard error of mean) (Bird and Laguë, 1977), from 9.5 to 11.3 × 10^9/mL in the budgerigar (Samour, *et al.,* 1986a), from 47.4 × 10^6/mL in the peregrine falcon (Parks, *et al.,* 1986), from 2.69 ± 0.01 (mean ± standard error of mean) in the helmeted guinea fowl (*Numida meleagris*) (Nwakalor, *et al.,* 1988), from 7.8 ± 0.7 to 58.7 ± 24.9 (mean ± standard error of mean) in the Hispaniola parrot (*Amazona ventralis*) (Brock, 1991), from 369 ± 436 × 10^6/mL (mean ± standard deviation) in the houbara bustard (Saint Jalme, *et al.,* 1994), and from 608.4 ± 101.2 × 10^6/mL (mean ± standard error of mean) in the Magellanic penguin. More recently, sperm concentration in the Quaker parrot was estimated from 74.8 ± 40.4 to 579.3 ± 87.6 × 10^6/mL (mean ± standard deviation) (Anderson, *et al.,* 2002), 382. 6 × 10^6/mL (in the piping guan (DeMatteo, *et al.,* 2004), 0.5 to 14 × 10^9/mL in the domestic pigeon (Sontakke, *et al.,* 2004), 58.4 ± 33.2 × 10^6/mL (mean ± standard error of mean) in the Indian white-backed vulture (Umapathy, *et al.,* 2005), 0.61 to 2.86 × 10^6/μL in the golden-capped parakeet (*Aratinga auricapilla*), from 9.69 to 13.74 × 10^6/μL in the blue-naped parrot (*Tanygnathus lucionensis*) (Stelzer, *et al.,* 2005), 4.21 ± 0.27 × 10^9/mL in the ostrich (Rybnik, *et al.,* 2007), and 47.09 × 10^6/mL in rockhopper penguins (Waldoch, *et al.,* 2012).

ELECTRONIC

Spermatozoa concentration using electronic systems can be carried out using a dedicated cell counter (e.g., NucleoCounter SP-100, ChemoMetec A/S, Denmark) or dedicated computer-assisted semen analysis (CASA) system (e.g., IVOS II Clinical, Hamilton Thorne Bioscience, USA) (Fig. 15-21).

MOTILITY

Spermatozoa motility is one characteristic commonly estimated in semen samples. Traditionally this parameter has been estimated by diluting a small aliquot of the semen sample with saline solution or semen extender and placing it on a prewarmed slide or in a counting chamber. The slide or counting chamber is examined under light microscopy. The operator then estimates the percent of motile spermatozoa and takes notes on the nature of their movements (Fig. 15-22). The motility of the spermatozoa is typically classified as (1) no motility, (2) less than 50% of the spermatozoa showing activity, (3) more than 50% of the spermatozoa motile but the majority of individual tracks only circular and local, (4) more than 80% of the

FIGURE 15-21 The IVOS II computer-assisted semen analysis (CASA) system enables the operator to calculate spermatozoa concentration and measure several parameters related to spermatozoa motility, including average path velocity, progressive velocity, curvilinear velocity, beat cross frequency, straightness of track, amplitude of lateral head displacement, and linearity of track of the spermatozoa. (Courtesy Hamilton Thorne, Inc.)

FIGURE 15-22 A more standard and less sophisticated system to evaluate semen samples. Such systems are commonly used in avian breeding centers to assess the quality of the spermatozoa before artificial insemination is carried out using freshly collected semen samples. The system is integrated with a warm plate, on which glassware can be preheated and samples can be temporarily placed before examination. The microscope is provided with a heated stage and a video camera connected to a monitor for observation of the type and quality of the movement of the spermatozoa.

spermatozoa motile and individual movements spread over several counting squares (Saint Jalme, *et al.,* 1994). A slightly modified scale is as follows: 1 = no motility, 2 = less than 50% motile, 3 = more than 50% motile but movement not progressive, 4 = more than 50% motile and movement progressive, and 5 = more than 80% motile and progressive movement (Anderson *et al.,* 2002). Another assessment of spermatozoa motility using a scale from 1 to 5 is as follows: 1 = slight side-to-side, no forward progress; 2 = rapid side-to-side, no forward progress; 3 = rapid side-to-side, forward progress in spurts; 4 = slow, steady, forward progress; and 5 = rapid, steady, forward progress (DeMatteo, *et al.,* 2004). This type of assessment is subject to operator partiality and is therefore not reliable.

Early semiautomatic analytical systems widely used for determining motility were designed to assess the swimming speed and linearity of the spermatozoa. These early systems relied on a computer and a graphic tablet linked to a video camera. The computer-camera interface allowed the projection of real-time images, along with a graphic display, onto the monitor. Motile spermatozoa were projected onto the monitor together with a cursor, the position of which was controlled by the operator by means of the graphic tablet. The swimming speed and linearity measurements of individual spermatozoa were made by tracking the chosen spermatozoa with the cursor. To avoid any bias, a randomly positioned box was displayed in the monitor as a guide to sampling (Samour, *et al.,* 1988b).

More sophisticated analytical systems have been introduced into the market and have been successfully used to determine spermatozoa motility in avian semen samples. These systems, classified as computer-aided sperm analyzers, are capable of measuring several parameters, including the following: average path velocity, progressive velocity, curvilinear velocity, beat cross frequency, straightness of track, amplitude of lateral head displacement, and linearity of track of the spermatozoa. Such systems have been successful in determining the above-mentioned parameters in spermatozoa of the Indian white-backed vulture (Umapathy, *et al.,* 2005), the domestic pigeon (Sontakke, *et al.,* 2004), domestic fowl (Kanatiyanont, *et al.,* 2012), and budgerigar (Gloria, *et al.,* 2014).

SPERMATOZOA MORPHOLOGY

Morphologic characteristics of spermatozoa have been observed in semen samples from domesticated pigeons (Sontakke, *et al.,* 2004) and the Indian white-backed vulture (Umapathy, *et al.,* 2005), by diluting 2 µL of semen in 100 µL of 0.5% glutaraldehyde solution. Smears were then made, and spermatozoa were observed under 400× light microscopy. Conversely, 2 µL of Magellanic semen samples was diluted with 8 µL of 8% glutaraldehyde solution. Then smears were made, and the morphology of the spermatozoa was observed under oil immersion (O'Brien, *et al.,* 1999) (Figs. 15-23 and 15-24).

PERCENTAGE OF LIVE AND DEAD SPERMATOZOA

This assessment is commonly carried out using commercially available supravital eosin-nigrosin stains. The original stain was developed in the 1950s primarily to assess the number of dead and live spermatozoa contained in semen samples of humans (Williams and Pollak, 1950) and domesticated farm animals (Swanson and Bearden, 1951). The principle is that dead spermatozoa such as those with a damaged cell membrane will uptake the eosin, staining the head of the spermatozoa red. The nigrosin component of the stain is used as a counterstain to ease visualization of the unstained live spermatozoa. This stain is also widely used to assess the morphologic characteristics of the spermatozoa. Preparations are made by depositing one drop of the supravital eosin-nigrosin stain on a prewarmed slide. A drop of undiluted semen of similar size is then placed beside the stain and mixed gently with the tip of the pipette. Smears are then made using the slide-to-slide technique, drying the slide by air or over a warm plate. Under 400× magnification, a total number of 100 spermatozoa are counted, and the number of dead (stained) or live (unstained) spermatozoa are counted. A 2% solution of eosin has also been used to

FIGURE 15-23 The two types of avian spermatozoa are those from passerine and those from nonpasserine species. A typical spermatozoon from a domestic pigeon (*Columba livia*), a nonpasserine species, is characterized by a long cylindrical head and a long tail.

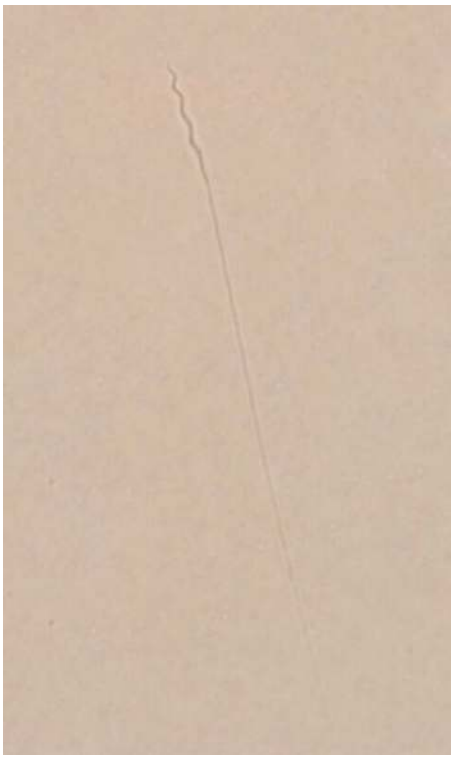


FIGURE 15-24 A typical spermatozoon from a domestic sparrow (*Passer domesticus*), a passerine species, is characterized by a long, spiral-shaped head and a long tail.

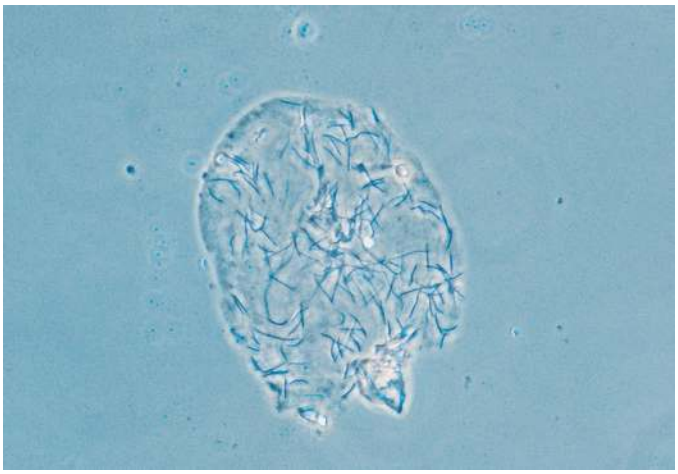


FIGURE 15-25 Zona pellucida-free hamster ova spermatozoa penetration assay for the assessment of the fertilizing capacity of budgerigar (*Melopsittacus undulatus)* spermatozoa after a freezing and thawing trial. Note the numerous spermatozoa bound to the surface of the slightly disrupted ovum.

assess the number of dead spermatozoa in avian semen samples (Stelzer, *et al.,* 2005).

ZONA-FREE HAMSTER OOCYTE PENETRATION ASSAY

The zona-free hamster oocyte penetration assay was developed primarily to assess the fertilizing capacity of spermatozoa originated from patients undergoing fertility diagnostic testing in human in vitro fertilization clinics (Yanagimachi, 1984). The interaction between ova and spermatozoa in Metazoa is dictated by unique cell membrane properties that, in general terms, would only allow homologous gamete fertilization (Bedford, 1981). Hamster oocytes show an almost unique capability of allowing spermatozoa from most other mammalian species to fuse with their oolemma, to enter the vitellus, and to undergo chromatin decondensation. However, the *zona pellucida* often plays a major role in excluding foreign spermatozoa. However, when this is removed using trypsin, for instance, heterologous spermatozoa may be able to fuse with the oolemma (Yanagimachi, 1984).

The zona-free hamster oocyte penetration assay has been carried out successfully in avian species such as the budgerigar (Samour, *et al.,* 1986b) (Fig. 15-25) and in domesticated mammalian species such as the bull (Kumar, *et al.,* 2013). The technique is commonly carried out by preparing the hamster ova using standard methodology (Rogers, *et al.,* 1979). Diluted semen samples are then placed under silicone oil within a Petri dish and put in an incubator at 37° C in air. Ten zona-free hamster ova were placed in each drop of diluted spermatozoa with a final concentration of 0.79×10^9/mL and incubated for a minimum of 12 hours. The ova are subsequently retrieved and examined under light microscopy. This technique made it possible to identify the presence of the nucleus, midpiece, and tail of budgerigar spermatozoa in the vitellus of hamster ova (Samour, *et al.,* 1986b).

The poultry industry has used a similar functional assay to determine spermatozoa penetration of the perivitelline layer of the hen's ovum as assessed on oviposited eggs (Bramwell, *et al.,* 1995).

REFERENCES

Anderson SJ, Bird DM, Hagen MD: Semen characteristics of the Quaker parakeet (*Myiopsitta monachus*), *Zoo Biol* 21:507–513, 2002.

Bedford JM: Why mammalian gametes don't mix?, *Nature* 291:286–288, 1981.

Bird DM, Laguë PC: Semen production of the American kestrel, *Can J Zoolog* 55:1351–1358, 1977.

Bramwell RK, Marks HL, Howarth B: Quantitative determination of spermatozoa penetration of the perivitelline layer of the hen's ovum as assessed on oviposited eggs, *Poultry Sci* 74:1875–1883, 1995.

Brock MK: Semen collection and artificial insemination in the Hispaniolan parrot (*Amazona ventralis*), *J Zoo Wildlife Med* 22(1):107–114, 1991.

DeMatteo KE, Karagiannis KL, Asa CS, et al: Semen collection and artificial insemination in the common piping guan (*Pipile cumanensis cumanensis*): potential applications for Cracidae (Aves: Galliformes), *J Avian Med Surg* 35(4):447–458, 2004.

Dott HM, Foster GC: Preservation of different staining of spermatozoa by formal citrate, *J Reprod Fertil* 45:57–60, 1975.

Gloria A, Contri A, Carluccio A, et al: The breeding management affects fresh and cryopreserved semen characteristics in *Melopsittacus undulatus*, *Anim Reprod Sci* 144(1-2):48–53, 2014.

Kanatiyanont N, Kornkaewrat K, Supthanmapinunt P, et al: Effect of semen collection techniques on semen quality and sperm motility parameters in Siamese fighting cocks (*Gallus gallus*), *Thai J Vet Med* 42(4):439–445, 2012.

Kumar S, Khan S, Sivakumar A, et al: Zona-free hamster oocyte penetration test: a reliable in vitro bioassay to assess bull fertility, *Adv Anim Vet Sci* 1(4):102–106, 2013.

Lake PE, Stewart JM: Preservation of fowl semen in liquid nitrogen—an improved method, *Brit Poultry Sci* 19:187–194, 1978.

Nwakalor LN, Okeke GC, Njoku DC: Semen characteristics of the guinea fowl (*Numida meleagris meleagris*), *Theriogenology* 29(3):545–554, 1988.

O'Brien JK, Oehler DA, Malowski P, et al: Semen collection, characterization and cryopreservation in a Magellanic penguin (*Spheniscus magellanicus*), *Zoo Biol* 18:199–214, 1999.

Parks JE, Heck WR, Hardaswick V: Cryopreservation of peregrine falcon semen and post-dialysis to remove glycerol, *J Raptor Res* 20(1):16–20, 1986.

Rogers BJ, Campen HV, Ueno M, et al: Analysis of human spermatozoa fertilizing ability using zona-free ova, *Fertil Steril* 32:664–670, 1979.

Rybnik PK, Horbanczuk JO, Naranowicz H, et al: Semen collection in the ostrich (*Struthio camelus*) using a dummy or a teaser female, *Brit Poultry Sci* 48(5):635–643, 2007.

Saint Jalme M, Gaucher P, Paillat P: Artificial insemination in houbara bustards (*Chlamydotis undulata*); influence of the number of spermatozoa and insemination frequency on fertility and ability to hatch, *J Reprod Fertil* 100:93–103, 1994.

Samour J, Moore HD, Smith CA: Avian spermatozoa penetrate zona-free hamster oocytes *in vitro*, *J Exp Zool* 239:295–298, 1986b.

Samour J, Smith CA, Moore HDM, et al: Semen collection and spermatozoa characteristics in budgerigar (*Melopsittacus undulatus*), *Vet Rec* 118:397–399, 1986a.

Samour JH, Markham JA, Moore HDM: Semen cryopreservation and artificial insemination in the budgerigar (*Melopsittacus undulatus*), *J Zool*, London 216:169–176, 1988b.

Sontakke SD, Umapathy G, Sivaram V, et al: Semen characteristics, cryopreservation and successful artificial insemination in the blue rock pigeon (*Columba livia*), *Theriogenology* 62:139–153, 2004.

Stelzer G, Crosta L, Bürkle M, et al: Attempted semen collection using the massage technique and semen analysis in various psittacine species, *J Avian Med Surg* 19(1):7–13, 2005.

Swanson EW, Bearden HJ: An eosin-nigrosin stain for differentiating live and dead bovine spermatozoa, *J Anim Sci* 10(4):981–987, 1951.

Umapathy G, Sontakke S, Reddy A, et al: Semen characteristics of the captive Indian white-backed vulture (*Gyps bengalensis*), *Biol Reprod* 73:1039–1045, 2005.

Waldoch J, Root T, Dubach JM, et al: Semen characteristics and artificial insemination in rockhopper penguins (*Eudyptes chrysocome chrysocome*), *Zoo Biol* 31:166–180, 2012.

Williams WW, Pollak OJ: Study of sperm vitality with the aid of Eosin-Nigrosin stain, *Fertil Steril* 1:178–181, 1950.

Yanagimachi R: Zona-free hamster eggs: Their use in assessing fertilizing capacity and examining chromosomes of human spermatozoa, *Gamete Res* 10:187–232, 1984.

TESTICULAR BIOPSY

Lorenzo Crosta, Petra Schnitzer, Linda Timossi

Infertility and poor breeding performance are commonly encountered in avicultural collections. As a general rule, the causes of avian infertility are divided into two groups: medical and nonmedical. The most common medical causes of avian infertility include lameness or foot problems, ophthalmic problems, malnutrition, diseases of the reproductive tract, systemic diseases, cloacal abnormalities, endocrine diseases, and some toxicoses. Among the nonmedical causes of infertility, we may consider immaturity and/or sexual inexperience, wrong perches (too loose or wrong material, size, or substrate), wrong nest (size, shape, and location), aviary disturbances, lack of social stimulation, homosexual pairings, and heavy cloacal feathering (this especially applies to some pigeon, canary, and budgerigar breeds).

For the aforementioned reasons, when approaching infertility problems in birds, a complete evaluation of the management procedures and laboratory testing are of primary importance. When diagnostic tests and management reviews are unable to determine a diagnosis for the cause of infertility, semen collection and testicular biopsy may be indicated in male birds. This normally follows a general coelioscopy, to rule out:

1) Other possible, nondiagnosed causes of the infertility
2) The feasibility of the surgery (e.g., testicles may be hidden, covered by a lesion, apparently not present).

TECHNIQUE

Endoscopic testicular biopsy is performed under general anesthesia using isoflurane (Fig. 15-26). The anesthetic concentration depends on bird species and also on the individual patient, but generally patients are maintained between 2% and 3% isoflurane. As a rule, patients are treated with meloxicam (1 to 2 mg/kg IM) 20 to 30 minutes before surgery. Due to the surgery's relatively short duration, birds are not intubated, and the procedure is performed using a face mask. Anesthesia is generally monitored by a Doppler monitor, whose probe is located on one of the the wing veins (*V brachiales, V. basilica, V. ulnaris, V. ulnaris superficialis*).

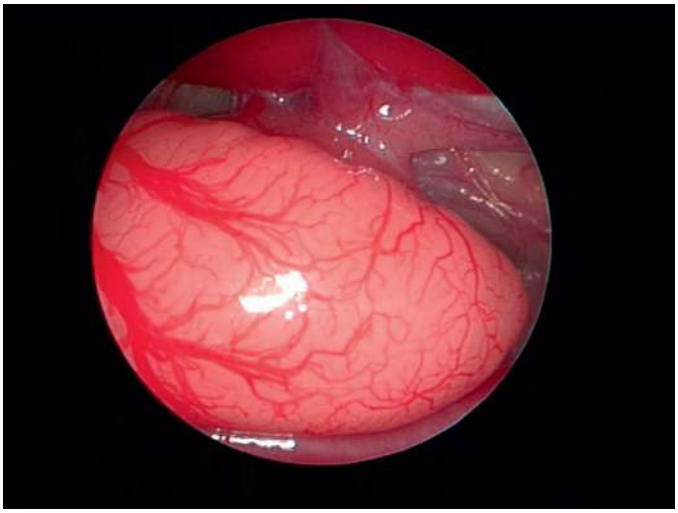


FIGURE 15-26 Endoscopy view of the testis of a mature male domestic pigeon (*Columba livia*) in the nuptial phase of the gonadal cycle. Note the large size of the gonad and the extensive vascularization. Great care must be exercised when collecting a testicular biopsy to avoid trauma to any large blood vessel.

Unless there are specific indications for an alternative entry site (for example, hip problems or old leg fractures), the entry site is caudal to the leg. Both left and right lateral approaches are used to inspect both sides of the coelom.

A small cutaneous incision is made where the caudal thigh muscle (semimembranous muscle, *M. flexor cruris medialis*) crosses the bird's last ribs. Furthermore, a delicate digital palpation of the area allows the surgeon to locate a small depression where the muscle crosses the rib. Then the abdominal muscles are bluntly dissected with an Adson, or a small, curved mosquito forceps, and the endoscope is inserted into the abdominal coelom, and the testicle is visualized.

In most cases, the preliminary inspection is performed with a 2.7 "short" rigid endoscope (length 12 cm), with an offset of 30 degrees. In the experience of the authors, this tool is perfect for a quick and easy observation. If the decision to proceed with the biopsy is made, the operator can switch to a longer optic (18 cm, 30 degrees offset). All the instrumentation is from Karl Storz GmbH, Tuttlingen, Germany. This endoscope is inserted into its operating sheath; the biopsy forceps is already inserted into the working channel, with its handling tip hanging loosely out of the posterior end of the sheath.

1) Once the testicle is located and the operator is ready to proceed, at least two factors must be considered.
2) The testes are extraperitoneal organs, and they are covered and suspended by two layers: the mesorchium and the peritoneum.
3) The avian testicle is a very small and delicate organ, and using too much force to pull the forceps may damage the testicle, if not pull it away entirely.

The first problem is easily overcome by tearing the peritoneum and mesorchium directly with a gentle bite of the biopsy forceps. This will create a small window and expose the testicular surface.

The second problem is solved by pushing the endoscope and its sheath smoothly against the testicle before pulling the biopsy forceps. This will keep the organ in place and reduce the risk of major damage.

Summarizing the technique:

1) Make a general coelioscopy.
2) If everything looks OK, insert endoscope, sheath, and biopsy forceps.
3) Open a smooth window in the testicle ligaments over the place you want to take the biopsy from.
4) Proceed with the forceps and gently bite the organ.
5) Do not pull the forceps, but instead drive the endoscope against the testicle and push it smoothly.
6) Take the biopsy.

After the biopsy has been taken, put the testicular tissue into the appropriate media (generally 10% buffered formalin) in a very small vial (for example, an Eppendorf tube). Before waking up the bird, check inside the coelom for any sign of hemorrhage or any other possible lesions.

CONCLUSION

In the past few years, several birds have undergone testicular biopsy, and many of them have been re-examined after a period of time. The information has been valuable and, coupled with histologic results, allows the authors to conclude the chapter with some new insights. Unlike semen analysis that may offer some value for male birds but that may not provide an etiologic diagnosis, a testicular biopsy may provide an opportunity for a histologic, and eventually etiologic, diagnosis. Further, although semen collection is performed during the breeding season, biopsy examination of male birds should be performed at the end of it, because a biopsy taken during the culmination phase of breeding, when testicles reach their maximum size, can lead to partial leakage of tissue into the coelom.

Testicular biopsy is a relatively easy technique and can give very interesting results. As with any other surgery, and considering the small size of the patient and the biopsied organs, serious damage can occur to the testis. Although we have not observed any major lesions at follow-up, we have seen scars and minor deformation of the testicle after the biopsy. Luckily, the cases that were biopsied again did not highlight any histologic alteration; in our perspective, this means that scarring will not alter testicular function. However, it is imperative that the technique be performed only by experienced avian endoscopists.

Also the histopathological results of such small samples (generally 3 to 5 French) are highly dependent on the experience of the pathologist. Having access to a trained avian pathologist is mandatory if we want our diagnosis to be reliable.

The two previous points are especially important when biopsies are performed in specimens that belong to endangered species, where a wrong technique or pathologic interpretation of the biopsy may lead to catastrophic results for an entire recovery program.

Finally, even if results may be extremely interesting concerning the diagnosis and any possible infertility, sadly, they do not always meet the expectations of the bird's owner.

FURTHER READING

Baumel, et al: *Handbook of avian anatomy: acta anatomica avium*, ed 2, n° 23, Cambridge, Massachusetts, 1993, Publication of the Nuttall Ornithological Club.

Crosta L, Gerlach H, Bürkle M, et al: Endoscopic testicular biopsy technique in psittaciformes, *J Avian Med Surg* 16(2):106–110, 2002.

Crosta L, Gerlach H, Bürkle M, et al: Physiology, diagnosis and diseases of the avian reproductive tract, *Vet Clin North Am Exot Anim Pract* 6:57–83, 2003.

Fox N: Handling and assessing semen quality. In *Understanding the bird of prey*, Blaine, Wash., 1995, Hancock House Publishers, pp 84–87.

Gartrell BD: Assessment of the reproductive state in male swift parrots (*Lathamus discolor*) by testicular aspiration and cytology, *J Avian Med Surg* 16(3):211–217, 2002.

Gee GF, Mirande CM: Crane artificial insemination. In Ellis DH, Gee GF, Mirande CM, editors: *Cranes: their biology, husbandry and conservation*, Blaine, Wash., 1996, Hancock House Publishers, pp 205–217.

Haense M, Schmidt V, Schneider S, et al: *Testicular biopsies in Psittacine birds—comparison between histology and cytology. Proceedings of the 9th European AAV Conference and 7th Scientific ECAMS Meeting,* 2007, pp 350–356.

King AS, McLelland J: Coelomic cavities. In King AS, McLelland J, editors: *Birds, their structure and function*, Eastburne, England, UK, 1984, Baillière Tindall, pp 79–83.

König HE, Bragulla H, Liebich HG: Männliche Geschlechtsorgane (*Organa genitalia masculina*). In König HE, Liebich HG, editors: *Anatomie und propädeutik des geflügels*, Stuttgart, Germany, 2001, Schattauer, pp 125–132.

König HE, Feder F, Liebich HG: Verdauungsapparat (*Apparatus digestorius*). In König HE, Liebich HG, editors: *Anatomie und propädeutik des geflügels*, Stuttgart, Germany, 2001, Schattauer, pp 81–104.

Lierz M, Hafez HM: *Endoscopy guided multiple entry surgery in birds. Proceedings of the 8th European AAV Conference and 6th Scientific ECAMS Meeting,* 2005, pp 184–189.

Lierz M, Reinschmidt M, Müller H, et al: A novel method for semen collection and artificial insemination in large parrots (Psittaciformes), *Sci Rep* 3:2066, 2013.

Nickel R, Schummer A, Seiferle E: Leherbuch der Anatomie der Haustiere—Band V—2. *Auflage—anatomie der vögel*, Berlin, 1992, Paul Parey Verlag, pp 283–330.

Rupley A: Infertility. In *Manual of avian practice*, Philadelphia, 1997, WB Saunders, pp 481–482.

Samour J: *Vasectomy in birds. Proceedings of the 10th European AAV Conference and 8th Scientific ECAMS Meeting,* 2009, p 15.

Samour JH: The reproductive biology of the budgerigar (*Melopsittacus undulatus*): semen preservation techniques and artificial insemination procedures, *J Avian Med Surg* 16(1):39–49, 2002.

Speer BL: *A clinical approach to psittacine infertility. Proceedings of the Annual Conference of the Association of Avian Veterinarians,* 1991, pp 173–187.

Speer BL, et al: Diseases of the urogenital system. In Altman R, Clubb S, Dorrestein G, editors: *Avian medicine and surgery*, Philadelphia, 1997, WB Saunders, pp 639–643.

Stelzer G, Crosta L, Bürkle M, et al: Attempted semen collection using the massage technique and semen analysis in various psittacine species, *J Avian Med Surg* 19(1):7–13, 2005.

Taylor M: Biopsy techniques. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine and surgery: principles and application*, Lake Worth, Fla., 1994, Wingers Publishing Inc., pp 347–354.

Taylor M: Endoscopy in birds. In Murray MJ, Shildger B, Taylor M, editors: *Endoscopy in birds, reptiles, amphibians and fish*, Tuttlingen, Germany, 1998, Endo-Press, p 9.

ARTIFICIAL INSEMINATION

Sven Hammer, Christiana Hebel

Artificial insemination (AI) is an alternative reproductive method to natural copulation in which donated or collected semen of a male bird is introduced artificially into the oviduct or cloaca of a female bird to achieve fertilization. Originally developed for poultry in the 1930s, it is now used successfully in many captive breeding programs, in species conservation and reproductive research, and in hybridization in different species. It should be the last choice in captive breeding of free-living species due to the stress and disturbance induced by handling the birds during the breeding season. A full examination of the donated or collected semen is recommended before insemination. Semen should be evaluated for color, consistency, total volume, pH value, motility, spermatozoa concentration, and ratio of live to dead spermatozoa. If hatching is unsuccessful, the eggs should be evaluated for early embryonic death and infertility.

There are two methods for artificial insemination:

a) Forced AI, by grabbing the bird, turning it into ventrodorsal recumbency. and inserting the semen into the oviduct (Figs. 15-27 and 15-28). This can be very stressful for the bird.

b) Voluntary AI, (in general) in imprinted birds (raptors, mainly falcons and goshawks), by waiting for the female to bend forward and presenting herself for copulation. In this way no restraint is required.

FIGURE 15-27 A budgerigar (*Melopsittacus undulatus*) hen has been restrained and placed upside down using a rubber funnel. Gentle pressure is exerted on the abdomen until the cloaca is slightly everted and the opening of the oviduct is visualized. (Courtesy Dr. Jaime Samour.)

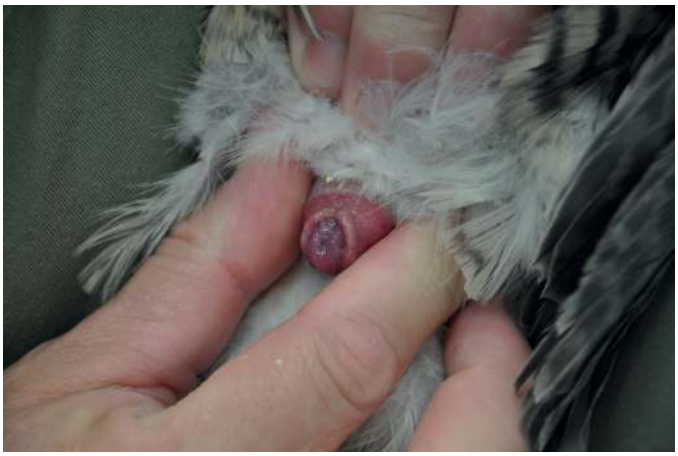


FIGURE 15-28 Forced artificial insemination in a falcon. The falcon has been placed on a handler's lap while the operator applies gentle pressure on the abdominal wall until the opening of the oviduct can be observed. (Courtesy Tobias Schultheis.)

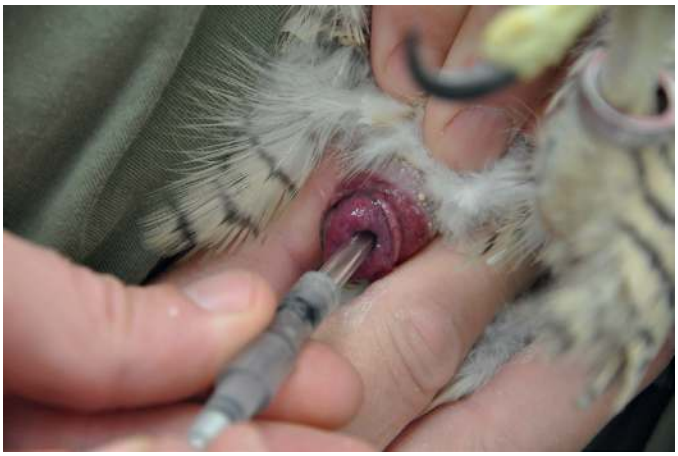


FIGURE 15-30 A short cannula, connected to a 1-mL syringe using a sleeve, is inserted into the oviduct to deposit the semen sample. Forced artificial insemination has the disadvantage of being stressful for the hen. (Courtesy Tobias Schultheis.)

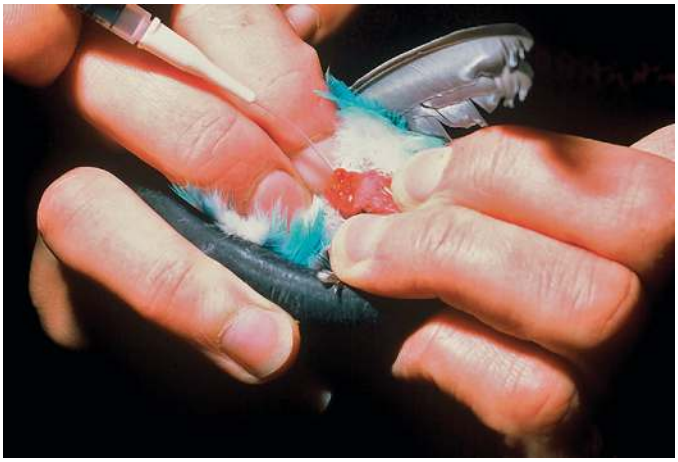


FIGURE 15-29 A lacrimal cannula is carefully inserted into the oviduct to a depth of 8 to 10 mm, and the semen is gently expelled using a 1 mL tuberculin syringe previously loaded with the semen sample. Budgerigars have been bred using both freshly collected and frozen-thawed semen. (Courtesy Dr. Jaime Samour.)

FIGURE 15-31 A small speculum is used to visualize the opening of the oviduct in a Spix's (*Cyanopsitta spixii*) macaw. This highly endangered species has benefited from artificial insemination.

The timing and frequency are fundamental for successful insemination. The best moment for insemination depends on the species, but it should always be conducted immediately after oviposition. During the breeding season, most females need to be stimulated by a male partner, even though in some species a female mate can stimulate ovulation. Successful AI depends on the insemination method, semen volume, spermatozoa concentration, spermatozoa motility, percentage of normal spermatozoa, and frequency of insemination. Limitations of this technique are based on semen quality (contamination with urates or fecal material, low spermatozoa concentration, and/or spermatozoa abnormalities), incorrect semen handling, and inconsistent semen collection.

TECHNIQUE

Collected semen is introduced carefully using a catheter, minitube, capillary tube, or lacrimal cannula into the oviduct of a laying female after careful manual manipulation of the cloaca. As most birds only have a single reproductive ovary, the oviduct opening is located at the left side of the cloaca. The opening is visualized by manual eversion pressuring the outside of the cloaca or by using a small speculum to visualize the opening of the oviduct. Semen can also be deposited into the cloaca, which is easier and quicker, but might compromise fertility. The catheter or tube is introduced gently into the oviduct (depth depends on species), and the undiluted or diluted semen is expelled gently through the catheter, capillary tube, or cannula (Figs. 15-29 to 15-31).

Semen volumes vary depending on species (2 to 5 µL in passerines, 50 to 150 µL in falcons to 400 to 1000 µL in ostriches). Storage of recently collected semen samples invariably results in loss of

spermatozoa quality; therefore, it is best to use freshly collected semen samples for insemination. Frozen-thawed semen samples have also been successfully used in a selected group of avian species (see section on semen cryopreservation in this chapter).

FURTHER READING

Brock M: Semen collection and artificial insemination in the Hispaniolan parrot (*Amazona ventralis*), *J Zoo Wildlife Med* 22(1):107–114, 1991.

Samour J, Smith CA, Moore HDM, et al: Semen collection and spermatozoa characteristics in budgerigars (*Melopsittacus undulatus*), *Vet Rec* 118:397–399, 1986.

Stelzer G, Crosta L, Bürkle M, et al: Attempted semen collection using the massage technique and semen analysis in various psittacine species, *J Avian Med Surg* 19:7–13, 2005.

Umapathy G, Sontakke S, Reddy A, et al: Semen characteristics of the captive Indian white-backed vulture (*Gyps bengalensis*), *Biol Reprod* 73:1039–1045, 2005.

Waldoch J, Root T, Dubach JM, et al: Semen characteristics and artificial insemination in rockhopper penguins (*Eudyptes chrysocome chrysocome*), *Zoo Biol* 31(2):166–180, 2012.

SEMEN CRYOPRESERVATION

Jaime Samour

Artificial insemination using freshly collected semen has been used successfully to solve some of the most common problems associated with captive breeding programs (Boyd, *et al.,* 1977). However, circumstances exist in which the use of freshly collected semen does not fulfill all the requirements of a comprehensive breeding program. The cryopreservation of spermatozoa can enhance captive breeding by allowing samples to be stored for future use. In addition, the storage of semen from founder members of a population can be used to maintain genetic diversity and equalize their genetic contribution to the gene pool after several generations (Ballou, 1984).

The first measure to preserve semen from avian species was carried out in the domestic fowl in 1948. Interestingly, the cryoprotectant action of glycerol was discovered accidentally when Meyer's histologic albumen preparation, containing 10% glycerol, was unintentionally used to dilute fowl spermatozoa. Since then, great advances have been made to preserve fowl semen (Lake and Stewart, 1978). In contrast, studies undertaken to develop techniques for cryopreserving semen from nondomesticated avian species have been both sporadic and lacking in continuity. Nevertheless, 11 different avian species have been bred over the years by artificial insemination using frozen-thawed spermatozoa (Table 15-2).

SEMEN DILUTION

After collection, semen samples have been diluted using a commercially available extender such as the Beltsville diluent (Sexton, 1977) in the greater sandhill crane (Gee, *et al.,* 1985), Aleutian Canada geese (Gee and Sexton, 1990), Magellanic penguin (O'Brien, *et al.,* 1999), and golden eagle (Knowles-Brown and Wishart, 2001), or by using Lake's diluent (Lake and Stewart, 1978) in the American kestrel (Brock, *et al.,* 1984), peregrine falcon (Parks, *et al.,* 1986), and houbara bustard (Hartley, *et al.,* 1999), or by using Biggers, Whitten and Whittingham medium (Biggers, *et al.,* 1971) in the budgerigar (Samour, *et al.,* 1988) and peregrine falcon (Samour, 1988), or by using Tyrode medium supplemented with albumin, lactate, and pyruvate (TALP) (Umapathy, *et al.,* 2005; Sontakke, *et al.,* 2004) in the domestic pigeon (Sontakke, *et al.,* 2004), Indian white-backed vulture (Umapathy, *et al.,* 2005) and wild-caught griffon vulture (*Gyps fulvus*) (Madeddu, *et al.,* 2009), or dimethylformamide in guinea fowl (*Numida meleagris*) (Seigneurin, *et al.,* 2013). The different diluents used contained either glycerol, dimethylsulphoxide (DMSO), dimethylacetamide (DMA), polyethylene glycol (PEG), or dimethylformamide as cryoprotectant agents. These agents protect the spermatozoa from severe damage due to the freezing process.

TABLE 15-2 Nondomesticated Avian Species Successfully Bred to Date Using Frozen-Thawed Spermatozoa

Species	Scientific name	Reference
American kestrel	*Falco sparverius*	Brock, *et al.,* 1984 Gee, *et al.,* 1993
Greater sandhill crane	*Grus canadensis tabida*	Gee, *et al.,* 1985
Peregrine falcon	*Falco peregrinus*	Parks, *et al.,* 1986
Budgerigar	*Melopsittacus undulatus*	Samour, *et al.,* 1988
Aleutian Canada goose	*Branta canadensis leucopareia*	Gee and Sexton, 1990
Impeyan pheasant	*Lophophorus impeyanus*	Durrant and Burch, 1991
Silver pheasant	*Lophura nycthemera*	Rose, 1996
Edwards pheasant	*Lophura edwards*	
Houbara bustard	*Chlamydotis undulata*	Hartley, *et al.,* 1999
Golden eagle	*Aquila chrysaetos*	Knowles-Brown and Wishart, 2001
Domestic pigeon	*Columba livia*	Sontakke, *et al.,* 2004

FIGURE 15-32 Programmable cell freezer used successfully for the cryopreservation of semen samples from budgerigar (*Melopsittacus undulatus*). A pump mounted on the top of the liquid nitrogen storage flask propels liquid nitrogen vapor into the loaded freezing chamber according to a designated program.

SEMEN CRYOPRESERVATION

The diluted semen is then placed into plastic or glass vials or plastic tubing or straws. After dilution, semen samples are cooled and then frozen using usually a programmable cell freezer (Fig. 15-32). Some of the most common programs used consist of cooling the samples at 8°C/min to +5°C within the cell freezer; then the semen samples are frozen at 6°C/min from +5° to −70°C and then to −196°C (Samour, *et al.,* 1988; Samour, 1988), 6°C/min to −196°C (Brock, *et al.,* 1984), 1°C/min from +5° to −20°C, 50°C/min from −20° to −80°C, then

FIGURE 15-33 Glycerol is known to inhibit fertility of spermatozoa when used in concentrations over 1%. Therefore glycerol must be removed from the samples before insemination. In a previous study with budgerigars (*Melopsittacus undulatus*), the glycerol used as a cryoprotectant agent was removed from the sample using disc dialysis.

rapidly immersed in liquid nitrogen (Gee, *et al.,* 1985; Samour, 1988; Gee and Sexton, 1990), 6°C/min to −180°C (Parks, *et al.,* 1986). Conversely, semen samples have been cryopreserved by placing the diluted samples on dry ice for 5 minutes and then plunging the samples into liquid nitrogen (O'Brien, *et al.,* 1999). In other studies for fast freezing, samples were cooled from 24° to 4°C at 3°C/min, then plunged into liquid nitrogen. For slow freezing, samples were cooled from 24° to 4°C at 1°C/min subsequently to −80°C at 8°C/min and then plunged into liquid nitrogen (Sontakke, *et al.,* 2004; Umapathy, *et al.,* 2005).

Another popular method for freezing semen is the formation of pellets by dropping the diluted samples directly into liquid nitrogen (Hartley, *et al.,* 1999). The pellets are subsequently transferred to vials for storage. Thawing is usually carried out by immersing frozen samples in a water or alcohol bath at +5°C.

Semen from the golden eagle (Knowles-Brown and Wishart, 2001) was successfully frozen using a commercially available freezing chamber known as "Mr. Frosty" (Nalgene). Samples were cooled to +5°C in small cryovials, then mixed with Lake's diluent containing 18% DMA. Samples were placed in the freezing chamber and then in a domestic freezer for 1.5 to 2 hr. After this period the samples were immersed in liquid nitrogen. Thawing was carried out in a water bath at 37°C before insemination. An interesting account evaluating the effect of breeding management on fresh and cryopreserved spermatozoa in the budgerigar was recently published (Gloria, *et al.,* 2014).

Glycerol is known to inhibit fertility of spermatozoa when used in concentrations over 1%. Therefore glycerol has to be removed from the samples before insemination (Fig. 15-33). In the studies that have used glycerol as a cryoprotectant agent, it has been removed using disc (Samour, *et al.,* 1988) or tube (Parks, *et al.,* 1986) dialysis. Other studies have used DMSO, DMA, PEG, or TALP as cryoprotectant agents; as a general rule, these do not require removal before insemination.

REFERENCES

Ballou JD: Strategies for maintaining genetic diversity in captive populations through reproductive technology, *Zoo Biol* 3:331–333, 1984.

Biggers JD, Whitten WK, Whittingham DG: The culture of mouse embryos *in vitro*. In Daniel JC Jr, editor: *Mammalian embryology*, Freeman, San Francisco, 1971, pp 86–116.

Boyd LL, Boyd NS, Dobler FC: Reproduction of prairie falcons by artificial insemination, *J Wildlife Manage* 41(1):266–271, 1977.

Brock MK, Bird DM, Ansah GA: Cryogenic preservation of spermatozoa of the American kestrel, *Int Zoo Yearb* 23:67–91, 1984.

Durrant BS, Burch C: Successful artificial insemination of cryopreserved pheasant semen, *J Androl* 12:56, 1991.

Gee GF, Sexton TJ: Cryopreservation of semen from the Aleutian Canada goose, *Zoo Biol* 9:361–371, 1990.

Gee GF, Bakst MR, Sexton TJ: Cryogenic preservation of semen from the greater sandhill crane, *J Wildlife Manage* 49:480–484, 1985.

Gee GF, Morrell CA, Franson JC, et al: Cryopreservation of American kestrel semen with dimethylsulfoxide, *J Raptor Res* 27:21–25, 1993.

Gloria A, Contri A, Carluccio A, et al: The breeding management affects fresh and cryopreserved semen characteristics in *Melopsittacus undulatus*, *Anim Reprod Sci* 144(1–2):48–53, 2014.

Hartley PS, Dawson B, Lindsay C, et al: Cryopreservation of houbara semen: a pilot study, *Zoo Biol* 18:147–152, 1999.

Knowles-Brown A, Wishart GJ: Progeny from cryopreserved golden eagle spermatozoa, *Avian Poult Biol Rev* 12:201–202, 2001.

Lake PE, Stewart JM: Preservation of fowl semen in liquid nitrogen—an improved method, *Brit Poultry Sci* 19:187–194, 1978.

Madeddu M, Berlinguer F, Ledda M, et al: Ejaculate collection efficiency and post-thaw semen quality in wild-caught griffon vultures from the Sardinian population, *Reprod Biol Endocrin* 7:1–7, 2009.

O'Brien JK, Oehler DA, Malowski P, et al: Semen collection, characterization and cryopreservation in a Magellanic penguin (*Spheniscus magellanicus*), *Zoo Biol* 18:199–214, 1999.

Parks JE, Heck WR, Hardaswick V: Cryopreservation of peregrine falcon and postthaw dialysis to remove glycerol, *J Raptor Res* 20:15–20, 1986.

Rose KA: *Evaluation, liquid storage and cryopreservation of silver pheasant (Lophura nycthemera) and Edwards pheasant (Lophura edwards) semen.* DVSc thesis, University of Guelph, 1996.

Samour JH: *Semen cryopreservation and artificial insemination in birds of prey. Proceedings of the 5th World Conference on Breeding Endangered Species in Captivity,* Cincinnati, 1988, Ohio, pp 271–277.

Samour JH, Markham JA, Moore HDM: Semen cryopreservation and artificial insemination in budgerigars (*Melopsittacus undulatus*), *J Zool* 216:169–176, 1988.

Seigneurin F, Grasseau L, Chapuis H, et al: An efficient method of guinea fowl sperm cryopreservation, *Poultry Sci* 92(11):2988–2996, 2013.

Sexton TJ: A new poultry extender. Part 1. Effect of extension on the fertility of chicken semen, *Poultry Sci* 56:1443–1446, 1977.

Sontakke SD, Umapathy G, Sivaram V, et al: Semen characteristics, cryopreservation and successful artificial insemination in the blue rock pigeon (*Columba livia*), *Theriogenology* 62:139–153, 2004.

Umapathy G, Sontakke S, Reddy A, et al: Semen characteristics of the captive Indian white-backed vulture (*Gyps bengalensis*), *Biol Reprod* 73:1039–1045, 2005.

FURTHER READING

Wishart GJ: The cryopreservation of germplasm in domestic and non-domestic birds. In Watson PF, Holt WV, editors: *Cryobanking the genetic resource: wildlife conservation for the future?*, London, 2001, Taylor and Francis, pp 179–200.

INCUBATION

Lorenzo Crosta, Alessandro Melillo, Petra Schnitzer

NATURAL INCUBATION

Oviparity is the most common reproductive strategy in nature. Invertebrates, fish, amphibians and reptiles, and birds produce eggs in which embryos develop. Unlike other zoologic groups, however, birds do not abandon their eggs but actively create around their eggs the correct conditions for the growth of the embryo. This process is defined as incubation and corresponds to the gestation of mammals. The embryos are exposed to additional risks outside the body of the

female, but, apart from the obvious advantage of not burdening the female with pregnancy, makes possible the cooperation of both sexes in the process.

The incubation, with the exception of the Australian megapodes or Australian brushturkey (*Alectura lathami*), which build a type of incubator composed of a large heap of plant material, the fermentation of which produces the necessary heat for the development of the embryo, is conducted through an adult individual who sits on the eggs, heating and moving them. This action is defined as brooding. The brooding duty, depending on the species, is a responsibility of only the cock (e.g., Ratites), only the hen (Galliformes, Anseriformes, several songbirds), or both members of a pair (almost all Psittaciformes, and Columbiformes). In the so-called parasitic species, the adults sitting on the eggs are not the biological parents but pairs of other species.

Certain species begin to brood as soon as the first egg is laid to protect it from environmental factors. This includes specimens from the Falconiformes, Psittaciformes, Strigiformes, herons, and pelicans, for example. The consequence of this is the hatching of chicks of different ages and hence different strengths. In birds of prey in particular, this leads to older and more vigorous chicks competing for food brought by the parents; the older chicks starve or even kill and eat their younger siblings (common in eagles). However, if something unexpected happens to the eggs or the older chicks, the younger will act as a reserve and prevent the total failure of the brood. In other species such as parrots or pigeons, the ability to produce crop milk of different densities, along with more sophisticated parental care, usually allow the growth of all the chicks.

Galliformes, Anseriformes, and most Passeriformes begin to brood after laying the last egg so all the chicks hatch at the same time. This is particularly useful for the species with precocial offspring, because all the chicks can follow the parents at the same time.

Brooding is mainly controlled by prolactin. Brooding birds change behavior, become more shy and stealthy or, conversely, fiercely defensive of the nesting site. Also the view of the brood triggers the behavior and, in general, the birds show a strong attraction for their eggs and egg-shaped objects, although not all in the same way. Penguins, on one hand, the eggs and, in the absence of them, may also incubate stones, whereas songbirds are usually more bonded to the nest and remain indifferent to the eggs out of the nest itself.

Brooding does not consist simply of "sitting on eggs" but is an active process that transfers heat to the eggs through the "incubation patches." These areas of thickened and much vascularized featherless skin are placed in contact with the eggs. In addition, eggs are also constantly turned over to allow the uniform distribution of heat and gas exchange through the pores of the shell. Down feathers fall from apterii before the first egg is laid; then capillary vascularization of the dermis increases, causing local edema. This important change, regressing after incubation, is controlled by estradiol (fall feathers) and prolactin (modifications of vascularization).

Incubation patches have different disposition depending on the species. There is only one, median (corresponding to the central apterium) in Columbiformes, Falconiformes, and many songbirds; two in Alciformes; and three in Galliformes and gulls. Other groups, however, do not have incubation patches. Ducks and geese, for example, actively pull feathers from their chest to place the skin in contact with the eggs. Gannets wrap the eggs with their richly vascularized webbed feet; the emperor penguin (*Aptenodytes forsteri*) places its only egg on the feet and covers it with a special skin fold. The temperature at the level of the incubation patch is close to 40°C (104°F) and induces a temperature of 34°C (93.2°F) on the egg. Of course, this temperature varies between the part of the egg in contact with the body and the part that rests in the nest, as well as between the eggs occupying a central or peripheral position. To allow uniform heat distribution, the brooding bird turns its eggs periodically, with the frequency depending on the species; the position also varies. Birds that nest outdoors also need to protect the eggs from overheating by the sun; they use their wings as an umbrella and adjust the humidity by bringing green leaves to the nest, bathing the plumage before returning to the nest, or regurgitating water directly onto the eggs. Some species cover the nest with leaves when they leave, either for camouflage or to decrease the fluctuation of the physical parameters. Species that nest in cavities can rely on the benefits of a more stable microenvironment.

Eggs can, however, withstand a drop in temperature, especially at the beginning of incubation. This is necessary to allow the parent to get away in case of danger or to feed. In species where both members of the couple share the brooding duties, eggs are rarely or never left alone. If only one sex is available to sit on them, the eggs have to endure periodic temperature changes that are directly proportional to various species' resistance to fasting. The hummingbird leaves the nest every few minutes, whereas the albatross can brood for weeks before being replaced by the partner; the emperor penguins can brood up to 2 months. Birds are aware of the environmental temperature and do not leave the nest easily during cold weather.

The division of incubation duties between the members of a pair is also interesting. It seems that sharing the job of brooding is a primitive subject that has since evolved to one sex specializing. Some species take turns at intervals of hours or days, often with characteristic displays. Among Columbiformes, the male broods during the day and the female at night, whereas the opposite occurs in ostriches (*Struthio camelus*). In songbirds generally, the female has a predominant role, and the male feeds her and guards the nest; with great variability of species, however, he also participates in the incubation. In other groups, Galliformes for example, incubation is up to the female only, who fasts for the whole period or leaves the nest to feed. In other species, only the male broods, as with the rhea, the emperor penguin, the kiwi (*Apterygidae*), the jacanas (*Jacanidae*), and other polyandric species. The red-legged partridge (*Alectoris rufa*) shows a quite peculiar behavior, as the female lays two consecutive broods, with the male in charge of one and herself of the second.

The incubation period varies from 12 days for many small passerines to 65 days for the emperor penguin, up to 80 days for kiwis and some great albatross (*Diomedeidae*).

Fundamental to the success of the reproductive process in birds is the nest. This is the place where the eggs are laid and incubated and where altricial chicks dwell until full development. The nests of birds in nature can be very simple. Most Columbiformes pile up a few twigs; various waders create a hole in the gravel; and some tropical terns do nothing, only balancing their single egg on a branch. Wonderfully complex structures include those of the penduline tit (*Remiz pendulinus*) and African weavers (*Amblyospiza albifron*).

Most Psittaciformes nest in cavities, usually in tree trunks, but also in clay walls, termite mounds, or cactus drums. Exceptions are monk parakeets (*Myiopsitta monachus*), which build complex spherical nests on branches, or hanging from trees or artificial structures, where they breed in a colony.

In captive breeding conditions, providing a nesting site appropriate and acceptable to the species and individuals is often the key to success. Geese, for example (*Anser* and *Branta* spp.), and several ducks (*Anas* spp.) build a rudimentary nest of grasses under a shelter (barrels, doghouses, or simple wooden planks) or even in grass and bushes. Many other ducks, such as wood and mandarin ducks (*Aix* spp.), require real nest boxes raised half a meter from the ground. In Anseriformes, incubation is reserved exclusively for females, but males of different species remain near the nest to protect it.

The Psittaciformes generally are cavity nesters and in captivity typically use different types of nest boxes. As with most of the cavity nesters, the parrots lay white eggs, two to four in number. Small domestic species like lovebirds or budgerigars can lay even more conspicuous clutches. Large species in particular can be very destructive toward wooden nests, and for this reason many breeders use metal containers or wooden boxes with metal frames. This solution is not ideal, because gnawing the nest box is a sign of adaptation, of the couple's desire to change the environment to make it suitable for breeding. For many species, *Pionus, Cacatua* and *Eclectus* spp., enlarging and modifying the entry of the nest is critical to achieving the reproductive condition. This can be achieved by fixing slats of wood or bark to partially occlude the entrance to the nest, allowing the birds to meet this need without excessively damaging the box. More appropriate is providing nests in many different shapes, sizes, locations, and materials and allowing the pair to choose a favorite. The main styles of nest boxes are the following: vertical, horizontal, and L-shaped, both vertical and horizontal. Vertical nests should have an internal ladder made from wire mesh under the entrance hole to prevent birds from jumping inside and breaking eggs. A possible solution to this problem is to hang the nests diagonally, but those in vertical L shapes are more effective. The horizontal nests are often preferred by the species of small size. Few species, including the budgerigar, lay on the bare wood; many others require some kind of padding. This can be transported from the outside, as, for example, lovebirds do with willow bark, dry grass, palm leaves, and other plant frayed, or the birds themselves can produce padding by gnawing the inside walls of the nest. To avoid damage to the nest box, soft wood bark can be screwed to the inner walls for the birds to crumble. The bottom of the nest can also be covered with wood chips, crumbled bark, or coconut fiber.

Some species of Psittaciformes have special needs. The palm cockatoo (*Probosciger aterrimus*) nests in old hollow trees open at the top and will rarely be recognized as adequate normal nesting boxes. There are testimonials of pairs of this species nesting in bins or barrels, but in general they require natural nests made from hewn logs. This species also lays a single egg and builds a nest of branches within the cavity. The kea (*Nestor notabilis)* in nature nest in cavities of rocks and usually prefer to lay on the ground rather than in normal nest boxes. A simple compromise can be just to put a nest box about 3 feet to the side on the ground and cover it with stones, or to build the structure with cement blocks. A fundamental element of the kea nest is a tunnel for access, which may be a concrete or wooden pipe of about 20 inches in diameter and 50 to 100 cm in length. The tunneling allows the female to prevent the male from getting into the nest. Another species with special needs is the red-headed lovebird (*Agapornis pullarius*), which in nature nests in termite mounds. To induce nesting in this species, nest boxes should be filled with sheets of cork, in which the female digs a tunnel to the nest chamber.

Columbiformes build very rudimentary nests; they are content to accumulate a few twigs over an intersection of branches, or in a basket. Domestic pigeons, which nest in cavities, take advantage of almost any hollow structure, such as holes in the wall, and ledges. Exotic doves and pigeons, which generally nest in bushes, collect twigs on intersections of the branches or in baskets. Several species are slow and reluctant to start, but once the right conditions are found and provided, they can reproduce well. Male and female share brooding, usually the male during the day and the female during the night; in general, the brooding period is rather short at around 2 weeks. Incubation begins immediately after the first egg is laid, so the youngsters hatch at different ages.

The most varied habits of incubation are found in the large Passerine group. In most commonly bred finches, such as the canary (*Serinus canaria*), the female builds a nest in the shape of a cup using plant material, and she stuffs it with animal hair and other soft material. The eggs of finches are colored in gray and pale blue streaked with red-brown. Only the hen incubates while the male feeds her. The *Estrildidae,* such as the Australian finches and the small African waxbills, instead build nests in cavities. The adaptation to the dark nest in these species is revealed by the white color of the eggs and the luminescent growths around and inside the beak of the chicks, useful to drive the cue for the parents. The crows together build bulky nests in trees, but then the female only will incubate the eggs, which are speckled on a green or brown background. Titmice and nuthatches (*Paridae* and *Sittidae*) are cavity nesters but build an elaborate and padded nest. Among *Timaliidae,* the Pekin robin *Leiothrix lutea* is quite commonly bred. In this species the male is the main manufacturer of the nest, made mostly of dried herbs and stuffed with herbs and animal hair, and suspended from a horizontal branch. In captivity they accept baskets or net hammocks but are not interested in nest boxes of any kind. The eggs are pale greenish or bluish with some brown spots on the obtuse pole. The female incubates during the night, and during the day the two sexes take turns frequently. Among *Sturnidae* is the hill mynah (*Gracula religiosa),* which is very much appreciated for its ability to mimic. These birds will use either a vertical or horizontal box and build a messy nest in it, taking leaves and dry grasses and any other materials. Eggs are pale blue with dark spots, and incubation, carried out by both pair members, lasts 14 days. The precious Bali mynah (*Leucopsar rothschildi*) are bred in a similar manner.

A special topic concerning avian reproductive strategies is reproductive parasitism. Some species rely on pairs of different species to incubate their eggs and raise their young or, more often, instead of their offsprings. Parasitism appears in the following orders: Cuculiformes (*Cuculidae*), Piciformes (*Indicatoridae*), Passeriformes (*Icteridae, Ploceidae*), and the little-known Anseriformes (*Anatidae*). The latter case involves a single South American species, the black-headed duck (*Heteronetta atricapilla*), which lays eggs in the nests of other ducks, especially the rosy-billed pochard (*Netta peposaca*), but also those of swans, coots, gulls, ibis, and other species. The ducklings, independent a few hours after hatching, move away from the nest of the host and live by themselves. Among *Cuculidae,* European species are highly specialized in parasitism, whereas almost all American species reproduce normally. After mating, the female of the common cuckoo (*Cuculus canorus*) starts searching for nests of songbirds under construction. The sight of one of them triggers the breeding cycle. A single female can produce a dozen eggs in an interval of 48 hours and always deposits one egg to parasitize a nest, taking advantage of the hours in which the nest is left unattended. The egg, brought in her beak, can be placed directly in the nest or on the ground to parasitize. The female does not lose interest in her eggs but controls their incubation, moving them to other nests if the chosen one was abandoned; this is not rare if the cuckoo is seen by the chosen pair. If the trick is not discovered, the parasite egg is incubated with the others, but it develops more rapidly and will hatch about a day before the others. A few hours after hatching, the cuckoo chick shows an intense cutaneous reflex as a result of any contact with its back, so everything that comes in contact with its back is raised and expelled from the nest. This may include eggs and chicks. In other species, this does not occur, but the chicks of the host species are still condemned to starvation given the rapid development of the parasite that monopolizes the nest. Small passerines are extremely diligent in feeding the little giant parasite, probably because of the superstimulus given by the giant red beak. Extremely interesting is the fact that females tend to lay eggs in the nests of

species that have grown and produced eggs very similar to their own. There is, therefore, the definition of distinct populations that parasitize different species, thus being able to coexist on the same territory. African indicators are Piciformes that parasitize other woodpeckers, but also starlings, bee-eaters and *Upupa.* The chicks have a sharp beak and, by the second day of life, use it to kill the adoptive siblings and remain alone in the nest. Among *Ploceidae,* the African widows (*Vidua* spp.) have developed a parasitic, but less bloody, behavior toward several species of waxbills. Widows have many similarities to the species that parasitize, even incorporating part of their song into their own. In this group the eggs are always white, but the interspecific mimicry is perfectly expressed by the luminescent wattles in the chick's oral cavity, which perfectly imitate the chosen waxbill's pattern. This is essential because the small widows are bred together to foster brothers and the widows requires full consistency of the buccal pattern.

The captive version of reproductive parasitism is the use of foster parents to support reproduction in species not yet well adapted to captivity. The best known example, which is linked to the case of widows, is that of the Society finch as a nanny for many species of Australian, Asian, and even African finches and waxbills. The Society finch is not a true species but a hybrid selected from several species of the genus Lonchura. This hybrid was responsible for the loss of conditioning to a precise buccal pattern, so the Society finches willingly feed chicks of different species, even mixed up in the same nest. There are some exceptions for certain African species whose behavior cue differs significantly. Also well known is the use of domestic canaries or the Mexican finch (*Carpodacus mexicanus*), appropriately trained to brood eggs and raise chicks of other finches. Similarly, small poultry breeds, usually silky or bantams, are used to brood and raise pheasant chicks. The use of foster parents improves the production of "difficult" species; however, the risk of inadvertently selecting strains deficient in reproductive instinct, as happened with many Galliformes, and the transmission of parasites and diseases between foster parents and fostered chicks, as with the *Cochlosoma,* which was widespread in the population of Gouldian finches, infected by the Society finches, should not be underestimated.

ARTIFICIAL INCUBATION

Current information on avian welfare and breeding seems to indicate that leaving the parent birds to incubate eggs and raise the chicks naturally may be beneficial to the mental wellness of young birds. This does not mean that hand-reared chicks cannot become good pets, but parent-reared birds can make very good pets as well. The concept is that parent-reared parrots, for instance, are mentally more balanced even when relating to human beings. Furthermore, parent-reared birds are more likely to become good replacements for aging breeding birds. Whatever the organization of a breeding facility may be, if the eggs are incubated artificially, the chicks will have to be hand-reared.

Good management of artificial incubation (AI) and hand-rearing techniques may guarantee an increment in the number of chicks, both for the pet bird market and for restocking the collection. However without long-term planning, the breeding stock could become easily exploited, with a shorter production life and long-term stress that will expose the breeding birds to disease. Importantly, if the breeder wants to use AI well and hopes to achieve good results, this involves a significant commitment at several levels:

- Financial (incubators, furniture, setting up one or more special rooms).
- Time for studying.
- Time for record keeping.
- Time for hand-rearing (artificially incubated chicks will have to be hand-reared).

A good plan for AI must be designed to meet the needs of the owner, but obviously the needs of the birds are of primary importance. There are several other factors to take into account:

- What is the main role of the owner?
- What kind of experience with artificial incubation and hand-rearing does he/she have?
- How much time does he/she have to spend in both activities?
- Number of breeding pairs in the facilities?
- Species in stock?
- Are the breeding birds experienced in rearing chicks?
- Is there any specific disease that might lead to different breeding techniques in the breeding facility? (e.g., diseases that are not transmitted vertically and make AI a good option).

Finally, although there is not a "gold standard" for a good incubation plan, AI has specific rules, and if the breeder does not follow the rules, results could be disappointing.

Among the most common reasons to incubate artificially avian eggs, we may list:

- Need to increase the production of eggs/chicks
- Very high biological value of a given species
- Very high financial value of a given species
- Ability/inability of a given pair to rear chicks successfully (e.g., they break the eggs, do not take care of chicks)
- Experience of a pair: Sometimes a young pair in a valuable species is not yet able to incubate and rear chicks; this may lead the owner to collect the first clutch to guarantee at least some chicks.

EGG FORMATION AND STRUCTURE

Knowledge of the structure of an egg is necessary for a successful incubation. It may seem pleonastic, but the egg must contain all the needed nutrients to support the good growth of the embryo. Adequate nutrition of the breeding birds, but especially of the female, is of primary importance; thus, the saying, "*The egg is what the hen eats!*"

The egg is composed of five fundamental parts:

1. Egg yolk: It is formed by the ovary, and its formation is independent of fecundation. It is constituted of the white yolk, yellow yolk, and germinal disc. These are surrounded by the vitelline membrane.
2. Egg white: It surrounds the egg yolk and is constituted of three layers of different thicknesses. Forty percent to 50% of the egg white is formed over the yolk in the magnum, and the rest is formed in the isthmus of the oviduct.
3. Chalazae: These are the yolk ligaments, and their function is to maintain the light germinal disc in an upright position. They run along the egg white, and are spiral shaped and strictly bonded to the vitelline membrane. They are formed in the isthmus and uterus of the oviduct.
4. Shell membrane: It is composed of a double layer, the inner and the outer shell membranes, adhering to the eggshell. Near the blunt egg pole, the two layers of the shell membrane separate to form the air cell. This will form only after the egg has been laid.
5. Eggshell: It is formed in the uterus of the oviduct. The eggshell is provided with pores that allow the exchange of air and elimination of excess gas. In some situations, they can allow the entrance of pathogens. The calcium needed for the formation of the eggshell comes from two sources: diet and bones. Mobilization of skeletal calcium starts 2 weeks before egg laying. In this period, birds need a good calcium supplementation in their diet.

PREREQUISITES FOR ARTIFICIAL INCUBATION OF EGGS

The most important prerequisite for eggs incubation is ***fertility.*** This can be assessed by observing the blood vessels within the egg, which can be seen very early after egg laying. Also, a good egg is not misshapen, has a smooth and even surface, and is not cracked.

It is better not to incubate broken or cracked eggs; however, if one decides to do so, the egg must be repaired. For the purpose, glue or candle wax can be used, but they tend to seal several eggshell pores around the crack and may limit egg respiration. For these reasons, altered eggs should be considered potentially infected; thus, it is better to incubate them in a separate incubator.

RECORD KEEPING

Developing a good record-keeping method will help the breeder evaluate the previous breeding history of the birds and make the right decisions about incubation procedures. To keep records well, some data will be written on the eggshell, and other information will be recorded on a PC. The best tool for writing on the eggshell is a blunt, soft pencil. There are several advantages in using a pencil:

- It cannot release any kind of toxic substance inside the egg
- It will not be washed away by most disinfectants.

Information that should be recorded on the eggshell includes:

- Date of laying
- Species
- ID number of the pair, or female, or aviary.

Somewhere else (generally a spreadsheet on the computer) we shall record:

- Egg weight (and size, if possible)
- Any abnormality (e.g., cracks, strange shape)
- Date when incubation actually starts.

STORING EGGS

It is better to allow the eggs to "rest" for a few days (2 to 5) before starting incubation, as this seems to increase hatchability of fertile eggs (at least in larger psittacines). Recently laid eggs are harvested from the nest and allowed to cool down a bit. If they are dirty, wait until they are dry and gently clean them with a soft brush.

Do not wash the eggs with water or dip them unless you are really sure of what you are doing!

Then the eggs are placed horizontally in a clean container and laid on a very soft substrate (i.e., millet seeds, very dry sand, specific trays for egg storing). Eggs can be stored at 13° to 16°C (55° to 60°F) for up to 5 days. It is still debated whether it is better to leave them as they are or to turn them, but most breeders do not turn them during storing.

INCUBATORS

Numerous commercial incubators are available in different brands, models, and prices (Fig. 15-34, *A-D*). Independent of the chosen model, it is important that the incubator is stable, able to monitor the inner temperature, and able to maintain it. For this reason, it is better to keep the incubators in an air-conditioned room with constant temperature and humidity.

Even if the chosen incubator has its own in built-in thermometer, it is always advisable to place also a precision thermometer capable of measuring temperature variations between 0.1° and 0.5°C.

Most incubators have a circulating air system. This means that the air is forced by a fan to move all around the incubator. In this way the temperature is more uniform within the box. However, new ideas and concepts are constantly developing on AI, some of which seem very promising.

A new concept in AI incubation is that Mother Nature should be imitated as much as possible. If we look at incubation under natural conditions, eggs are not incubated at a uniform temperature. Most hens develop what is normally referred to as the "brood patch" on the abdomen. When the hen is sitting on eggs, there is always a thermal gradient between the lower and upper parts of the egg under the hen. When the environmental temperature varies, the egg temperature will vary, too. This will cause a contraction and expansion of the egg content (that being a gel, will increase or decrease its volume, according to temperature). These contractions and expansions should promote and improve breathing and metabolism of the egg and embryo.

The new concept is to design incubators with a broad heater on the ceiling of the unit (imitating a brood patch); the eggs are kept on trays at the bottom of the box. In this way, the normal physiologic temperature fluctuations are respected, and the eggs should perform better. It is still too early to have a final opinion on the incubators built this way and on whether they perform better than "standard" incubators. However, the general feeling is that they do well. There are currently large professional breeding centers that are using such incubators.

Other parameters to evaluate when selecting an incubator are the following:

- Inner (usable) size
- Type of trays and their versatility
- Easiness for cleaning and maintenance
- Easiness for using
- Incubator mass (increasing the mass of the incubator, the stability of the temperature and humidity)
- Availability of technical assistance.

There are several reasons to have more than one incubator on hand. Bird keepers incubating eggs from different species should have several. If this is not possible, the breeder should have at least two incubators, plus a good hatcher, for the following reasons:

- There will be eggs of different sizes
- Eggs with different numbers of pores per cm^2
- Altered eggs, or eggs that produce foam or explode during incubation
- Broken and repaired eggs (risky eggs)
- Eggs with different needs in term of relative humidity (RH)
- Different numbers of turnings per day (smaller eggs need a higher number of turnings per day)
- One of the incubators may break down.

Please note:

- At least 1 month before the start of the breeding season, it is always advisable to run the machines to verify that everything is working properly.
- At least 2 weeks before egg laying starts, incubators must be started (they need time to settle properly).

Those two easy steps will avoid bad surprises when one really needs the equipment to work.

Regular maintenance of incubators includes a complete check of the different pieces of the units, such as the wet bulb, thermometer, hygrometer, timers, and the turning mechanism that moves the trays. Furthermore, the incubators should be cleaned and disinfected using nontoxic bactericidal products approved for eggs and incubators. At the end of the season, all the incubators (and brooders) must be cleaned carefully and fogged with a suitable disinfectant.

FIGURE 15-34 (A-D), The images show four types of incubators commonly used for eggs of different avian species. In some of these units, the eggs are placed horizontally over rollers, while in others, the eggs are placed vertically within holders. The incubator in **(B)** is a "contact" incubator, so called because during incubation the eggs are covered periodically by a plastic membrane simulating the effect of the brood patch of the hen. Many experienced breeders like moving around the eggs of some species using different incubators. Commonly, breeders start the incubation period by setting the eggs in an incubator and placing them horizontally for the first quarter. Later, they transfer the eggs to another incubator, placing them in the vertical position until pipping.

INCUBATION

AI can be extremely satisfying for the breeder, especially because he/she will have complete control of the eggs, and the hatching and development of the chicks will depend on his/her skill, precision, and perseverance. It must be remembered that different bird species lay eggs with different needs and incubation periods. This may range from 18 days in the budgerigar (*Melopsittacus undulatus*), to 30 to 31 days in the kea (*Nestor notabilis*), to 35 days in the gyrfalcon (*Falco rusticolus*), to 42 days in the ostrich (*Struthio camelus*).

Temperature

Heat is the major player for embryonic development. As a general rule, the higher the heat, the faster the development, and the lower the heat, the slower the development. From this perspective, it appears obvious that embryonic development depends mainly on the right temperature. Wrong! Too high temperatures will shorten incubation time, resulting in an incomplete absorption of the yolk, and temperatures that are too low, eventually kill the embryo.

Temperature can be monitored directly using a thermometer. Most bird breeders prefer to use two devices:

- A standard thermometer to be kept inside the incubator.
- An electronic thermometer, whose probe is inserted into the incubator as needed.

This latter tool is also used to check the function of the standard thermometer.

Recommended incubation temperatures for most avian species range between 36.9° and 37.5°C. The best result in parrots and some birds of prey is normally achieved with 37.2 ± 0.1°C (99°F).

Humidity

Humidity regulates the transpiration of the egg (and of the embryo) and helps with hatching time.

As a general rule, humidity is monitored using RH and expressed as a percentage. There are several ways to regulate RH and reach or maintain it. Among the most common, it is possible to:

- Introduce into the incubator a small container with distilled water (most incubators have a built-in container for the purpose).

- Reduce the airflow inside the incubator. This latter method will alter the ratio between air and water; however, in the experience of the authors, it is not advised, because it reduces the O_2 concentration on the eggs and slows down the removal of CO_2 produced by the eggs' metabolism.

Another technique used by some aviculturists is to introduce chicken eggs to maintain the appropriate level of humidity in the incubator. This technique works well, and it is maybe more physiologic than adding water to the cup; however, we should be extremely careful with the origin of the eggs, as they might carry disease.

Whatever the method, a wrong (especially too high) RH is one of the most common causes of embryonic death. Most aviculturists recommend an RH below 40% in the first half of the incubation and below 45% during the second half. In the end, it is not very important what RH is kept: The real goal is the highest possible percent of hatched eggs. So the right humidity is the one that works in a specific incubator in a given place, and the easiest way to verify whether the RH is right or wrong is to monitor the weight loss of the eggs during incubation.

- From the day an egg is laid until internal pipping, the egg should lose about 15% of its initial weight.
- Monitoring the egg weight (*and so the weight loss*) during incubation is the easiest (almost the only) way to understand whether we are doing it right.
- It is possible to divide the incubation time in thirds and weigh the eggs four times (day 0, 1, 1st, 2nd, and 3rd period). Some eggs have to be weighed more often to ensure the desired weight loss.

If weight loss differs too much from what is expected, RH should be adjusted or the eggs moved to an incubator with a different RH. The most common problem encountered during incubation of certain eggs is that the egg is not losing enough weight. The most common way to accelerate weight loss in such eggs is by drilling one or two small holes (1.5 to 2 mm) above the air chamber. This should be carried out during the second part of the incubation period. Some breeders favor the use of fine sandpaper in an area of the egg, presumably to open up the pores to allow more weight loss. The above-mentioned procedures should be carried out by an experienced operator only.

EGG POSITIONING AND TURNING

Turning is another crucial factor for good embryonic development. If the eggs are turned less than needed, or they are not turned at all, the embryo can adhere to the eggshell from inside. In that case, we will observe a malpositioned chick.

There are basically two ways to turn the eggs:

1. Rotation
   a. Rotating incubators have trays with rollers of different size
   b. Eggs are positioned horizontally
   c. When the rollers move, the eggs will turn over them.
2. Tilting
   a. Eggs are positioned vertically with the air chamber up (*not good for psittacines*)
   b. Tilting trays lean 45 degrees and have 90-degree movement.

In some cheap or older incubators, the trays have to be rotated manually, but the results are often discouraging. Turning eggs manually implies remembering to do it regularly. If one forgets, hatchability will decrease rapidly.

However, even when using the most up-to-date incubators, most aviculturists do turn the eggs 90 degrees once or more times every day "just in case." This is not a bad habit, but one should remember that turning eggs manually (with your bare hands) implies some risks.

- Eggs may be contaminated by dirty hands (wash hands or wear gloves)
- Eggs may be damaged by rough moves (be smooth)
- The embryo might always be placed on the same side (make a sign on the eggs).

CANDLING

Candling is the only way to evaluate egg fertility and to estimate the age of the embryo (Fig. 15-35). "Classical candling" is done in a darkroom: The egg is brought near a light source to transilluminate the shell (Fig. 15-36). Some typical features of the egg will tell the experienced operator whether the egg is fertile and the approximate age of the embryo. Fertility can be assessed after 4 to 7 days of incubation (*but real experts can do it before*), depending on the species, when ramified blood vessels are clearly visible.

- The air chamber becomes bigger while the embryo develops
- The embryo will not be visible until the end of the second trimester, when some movements may be seen

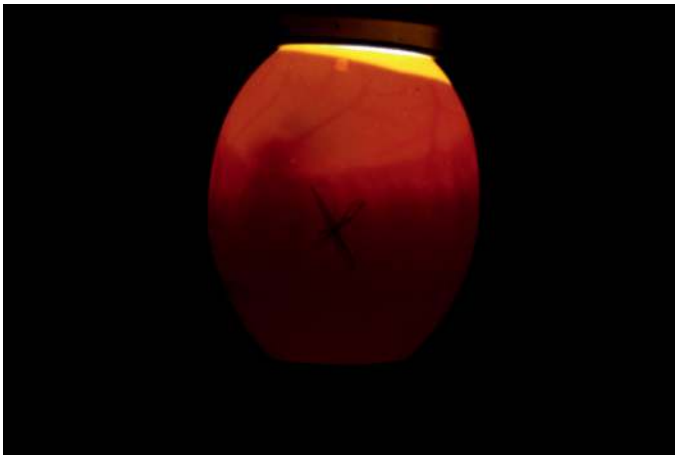


FIGURE 15-35 Candling is an effective way to assess the fertility of eggs during the incubation period. Eggs are commonly candled using a strong "cold" electrical light source e.g., a light-emitting diode (LED) lamp. Note the early blood vessel formation clearly visible across the egg.

FIGURE 15-36 Ostrich (*Struthio camelus*) eggs are difficult to candle due to the thickness of the eggshell, but it is possible within a darkroom with a very strong light source. The light in the room in the image was turned on for the benefit of the photograph. Note the dedicated egg trays for the ostrich eggs.

FIGURE 15-37 Electronic egg cardiac monitors (Buddy) are used to assess the heartbeat of embryos from the first quarter of the incubation period. These monitors are used effectively to monitor the embryo's development during incubation and to assess the embryo's health status during the hatching process.

FIGURE 15-38 Depending on their size, eggs should be placed in a cup or glass container at the first sign of external pipping and transferred to hatchers. Note the small hole made by the hatching chick just below the air chamber line.

- Activity of the embryo increases when pipping time approaches, and this is normally 2 to 3 days before hatching
- When the embryo dies early, we will note a dark band inside the egg.

Electronic Candling

In the past decade, digital or electronic monitors have been available on the market. Most of these devices work using infrared transmitters and sensors. They will amplify the cardiovascular signal of an embryo in the egg, allowing the breeder to detect the heartbeat of the embryo just a few days after incubation has started (e.g., Buddy, Avian Biotech International, Tallahassee, Fla., USA) (Fig. 15-37). Although these devices work well and are actually able to give good information, they should not be considered a total replacement for the standard candling, but instead be used as a device complementary to the classical technique.

HATCH AND ASSISTED HATCH

The hatching process starts about 48 to 72 hours before the expected hatch day. This is the right moment to place the eggs in the hatcher and, much better, in a separate unit. Good record keeping and knowledge of incubation length will help to understand when the process really starts.

Most aviculturists recommend setting the hatcher at a temperature slightly lower than in the incubator (37.0° to 37.1°C = 98.6° to 98.8°F) and at a humidity slightly higher (more than 50% relative). Whatever the settings in the hatcher, the eggs will be placed horizontally and will not be turned. It is highly recommended to place each egg in a single container (a plastic cup or similar) (Fig. 15-38).

Chicks should start breaking the eggshell 24 to 36 hours after breaking the air chamber membranes or internal pipping. From that time on, the chick will breathe fresh air from outside the egg and will start vocalizing. If 36 hours after the internal pipping there are no cracks in the eggshell, then a hole will be made in the shell, over the air chamber, so that the chick can breathe. In the majority of cases, this is enough for a successful, natural hatch (Fig. 15-39).

The time interval from pipping to hatching varies between species. On average, an interval of 24 to 72 hours is considered normal. During this time, the chick turns to break the shell and come out of the egg.

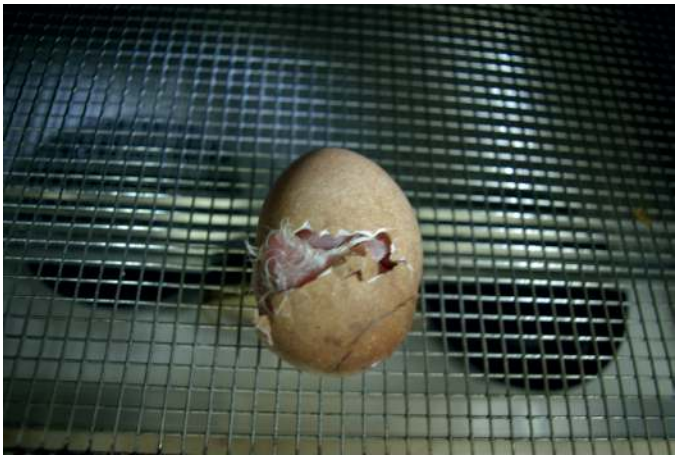


FIGURE 15-39 The chick in the photograph has started "cutting out" the eggshell. This process involves repeated pocking of the eggshell by the egg tooth on the dorsal aspect of the beak and the constant twisting of the chick's body during the hatching process.

If after this time there is no sign of movement, or if the chick is calling loudly (*or worse is not calling at all*), it is better to act (assisted hatch). Use the electronic heart monitor Buddy to assess cardiac function.

How to Proceed for Assisted Hatch

1. Maintain the egg in the same position of incubation.
2. Using a sterilized needle or fine drill bit, drill a hole over the air chamber (*blunt egg point*).
3. Using small tweezers, take away small parts of eggshell until the hole is big enough to allow for good visualization of the chick (likely wrapped in the membranes).
4. Use a cotton applicator, or a small paintbrush soaked with distilled water or sterile saline solution, to moisten the membranes.
5. If there is a net of small blood vessels, the chick is not ready to hatch. *In this case, place it back in the hatcher and WAIT!*
6. If blood vessels are not visible, it is better to break the membranes using a soaked applicator and to try to locate the bill of the chick.

7. Repeat the same action after 1 hour, widening the hole on the membranes and maintaining them moisturized.
8. Maintain the chick moist, especially around the bill.
9. Stimulate the chick to move and to free itself from the membranes with a gentle pressure of the applicator. *At this point, vocalization is a good sign!*
10. Always check the area around the umbilicus before freeing the chick completely.
11. When the chick is out of the eggshell, it is necessary to ligate and cut the umbilical cord.
12. Disinfect the navel and leave the chick in the hatcher for few hours to dry.

FURTHER READING

Anderson Brown AF, Robbins GES: *New incubation book*, Surrey, Canada, 2002, Hancock House Publishers Ltd.

Chitty J, Lierz M: *BSAVA manual of raptors, pigeons and passerine birds*, Gloucester, UK, 2008, Quedgeley.

Olsen GH, Orosz SE: *Manual of avian medicine*, St. Louis, 2000, Mosby.

REARING

Tom Bailey, Melodiya Nyela Magno

REARING METHODS AND BEHAVIORAL DEVELOPMENT

There are three methods of rearing birds in captivity: a) parent-rearing, b) hand-rearing, and c) mixed-rearing methods. The latter is basically a combination of the two previous methods.

The rearing methods of the chick influence the future purpose of the adult bird. Hand-rearing of many species creates imprinted birds (Figs. 15-40 to 15-44). Some species, such as falcons, are deliberately hand-reared so that they become imprinted on humans. These hand-reared birds will grow up as adults imprinted on humans and can be used in artificial insemination programs. Many of the hybrid falcons, such as gyr peregrine falcons (*Falco rusticolus—Falco peregrinus*), used in falconry today are produced by using semen obtained from imprinted male falcons to inseminate imprinted female falcons of another species, in this case a gyrfalcon. However, if a falcon is bred to become a natural breeder in the future, it is better if it is parent-reared in an aviary setting that is similar to the aviary the falcon will be introduced into for breeding in the future. Hand-reared psittacines are considered to become better, more tractable pets. Growth and

FIGURE 15-40 Brooders are essential to maintaining the necessary environmental conditions for hand-rearing chicks in the first stage of development. The Brinsea TLC 4 parrot brooder is a reliable unit that can be used for a wide variety of species. In tropical and arid countries, it is advisable to place these units over short legs within shallow dishes (e.g., Petri dishes) containing mineral or vegetable oil to prevent ants from climbing into the units and hurting and even killing chicks. (Courtesy Dr. Jaime Samour.)

FIGURE 15-41 The image shows a setup for second-stage rearing. It consists of open plastic boxes with heating pads placed under one half of the box, providing heating from below, and a lamp suspended from above to provide a hot spot. Lamps can be fitted with porcelain bulbs to provide heat without constant lighting into the box. It is important to use a spot check thermometer to constantly monitor the temperatures at different sites of the rearing box. Gradient temperature should be provided so chicks can move away from the heat source when desired. (Courtesy Dr. Jaime Samour.)

FIGURE 15-42 A newly hatched gyr (*Falco rusticolus*) chick as seen inside the hatcher. Under artificial incubation conditions, eggs should be placed within a hatcher immediately after external pipping with a humidity between 90% and 100% to avoid desiccation of the egg membrane covering the emerging chick. (Courtesy Dr. Jaime Samour.)

FIGURE 15-43 Crowned (*Goura cristata*) pigeon squab. Note the sticks at the bottom of the hand-rearing container. These should be replaced for washing and disinfection on a daily basis. Simulating natural nesting conditions helps in the development of chicks. This species is one of the largest pigeons in the world.

FIGURE 15-44 A stone curlew (*Burhinus oedicnemus*) chick within a brooder receiving an electrolyte solution via a 1-mL syringe. It is vital for chicks of this and another similar precocial species to have gradual access to larger areas for exercising the developing leg bones. The floor of such pens should be fitted with an antislippery surface such as rubber matting to avoid leg splaying. (Courtesy Dr. Jaime Samour.)

FIGURE 15-45 Large psittacine chicks are spoon fed in the latest stages of the hand-rearing process. Beak and oropharyngeal hygiene after each feed is important to avoid infections such as candidiasis and enteritis.

FIGURE 15-46 Juvenile blue and gold (*Ara ararauna*) macaws in a communal large cage. It is important for juvenile psittacines to have access to perches of various diameters in the early stages of their development. Placing young birds together reinforces the social skills needed to integrate into a flock or to select future breeding mates.

developmental abnormalities, especially metabolic bone disease, is a common medical condition in hand-reared chicks (see Disorders of the musculoskeletal system, Chapter 13).

Parent-reared psittacines may also acquire species-specific behavioral traits that will be lacking in hand-reared chicks (Figs. 15-45 to 15-48). Parent-reared chicks are considered more appropriate for reintroduction programs. Parent-reared birds have advantages if the parents provide adequate care to the chicks. However, leaving nestlings with parents has disadvantages, because parents do not always provide optimum care and may traumatize, fail to feed, or abandon chicks. Disease, cold, and competition with siblings may lead to problems with younger chicks. The environment of the nesting area is important, and in any investigation of disease in parent-reared neonatal chicks, close attention should be paid to hygiene and infestation by ectoparasites.

It a common practice when breeding some species in captivity to hand-rear chicks up to a certain age and then to introduce them to the biological or foster parents to continue the rearing process. This would represent the mixed rearing method. In most falcon breeding facilities around the world, for instance, chicks are hatched and reared until they

FIGURE 15-47 A group of gyr (*Falco rusticolus*) and gyrfalcon hybrid chicks begging for food. Rearing chicks of roughly the same age in a group setting helps create the right social environment to prepare for adulthood. (Courtesy Charles Schwartz.)

FIGURE 15-48 Ostrich (*Struthio camelus*) chicks tend to huddle together in the early stage of their development for warmth and security. Note that the chicks are housed in a suspended coop. Suspended lamps are placed over one or two corners of the coop to provide heat. Importantly, housing such inquisitive chicks under this system avoids direct contact with feces/urates and foreign objects easily picked up from solid floors. (Courtesy Dr. Jaime Samour.)

are able to hold their heads up. This is usually around 8 to 10 days. Close rings are commonly placed at this stage. Then chicks are introduced to the biological or foster parents. This method improves the survival rate of chicks significantly.

FURTHER READING

Canon JM: *A guide to basic health and disease in birds: their management, care and well-being*, ed 2, Australia, 2002, ABK Publications.

Chitty J, Lierz M: *BSAVA manual of raptors, pigeons and passerine birds*, Gloucester, UK, 2008, Quedgeley.

Gage LJ, Duerr RS: *Hand rearing birds*, Hoboken, N.J., 2008, Wiley-Blackwell.

NEONATOLOGY

Tom Bailey, Melodiya Nyela Magno

It is challenging to provide a comprehensive account of avian neonatology, because significant differences exist between the medical conditions that altricial and precocial species are susceptible to. In addition, great variations are found in the husbandry and rearing methods for the different avian groups. For more detailed information on species-specific medical issues in neonates and pediatric husbandry, the reader is advised to consult authoritative publications on raptors (Jones, 2008), bustards (Bailey, 2008), psittacines (Flammer and Clubb, 1994; Scott and Stoodley, 1996; Wilson *et al.*, 2006), ostriches (Huchzermeyer, 1998; Doneley, 2006), and on avian species in general (Gage and Duerr, 2008).

PREVENTIVE MEDICINE AND HUSBANDRY

Personnel

Nursery personnel should be familiar with neonatal anatomy and physiology, because critical decisions have to be made on whether a situation requires medical intervention. There are occasions wherein the best intervention is not intervening at all. For instance, certain species have pronounced pipping muscles after hatching, which can be mistaken to be edema by inexperienced personnel (Fig. 15-49).

Hygiene is of primary significance when handling neonates. Personnel should be encouraged to wear protective clothing and equipment, including rubber boots, laboratory coats, or overalls, and to wear face masks and gloves (Fig. 15-50).

Accidents leading to morbidity and mortality can be prevented if personnel pay attention to details and follow established protocols. For example, shifting an egg that has pipped externally from the incubator to the hatcher by following the correct timing can prevent accidents such as leg fractures resulting from long-legged hatchlings becoming compressed between rotating incubator rollers. Brooder doors left incompletely closed after hand feeding by careless personnel can result in neonates falling out of the brooders (Fig. 15-51). In addition, this results in suboptimal brooder temperature and hypothermia, with a reduced crop and gut-emptying rate. Additionally, personnel could provide toys with loose parts that could be accidentally ingested by psittacine neonates during play, leading to foreign body gastrointestinal obstruction.

Daily weight monitoring is an important task of personnel in a nursery, because body weight fluctuation is an early indication of health changes. For instance, underweight psittacine chicks might be underfed, or the formula might have excessive water and fewer solids. Please note that on the first 2 to 5 days posthatching, depending on the species, there is a physiologic weight loss as the yolk resorbs in the abdomen. This should not be confused with weight loss due to sickness or insufficient food.

Care and Attention of the Neonate

Naval care by applying iodine for the first 3 days posthatching and complementing with change of bedding material (i.e., paper towel) 4 to 5 times a day can prevent the occurrence of omphalitis (please see later). Frequent changing of nest bedding will contribute to an acceptably clean air space, especially in enclosed environments such as brooders.

Oropharyngeal and beak hygiene can significantly prevent occurrence of candidiasis. Fruit eaters with large beaks such as hornbills and some psittacines (e.g., hyacinth macaw), whose beaks form pockets, are prone to the deposition of baby formula and can benefit from gentle removal of food plaques after feeding (Fig. 15-52).

FIGURE 15-49 Recently hatched gyr (*Falco rusticolus*) falcon chick. Note the pronounced piping muscles on the dorsal aspect of the neck. The size of these muscles is normal at the time of hatching, and it should not be confused with edema. (Courtesy Dr. Jaime Samour.)

FIGURE 15-50 Nursery personnel should be encouraged to wear protective clothing when handling neonates. The photograph shows a sun conure (*Aratinga solstitialis*) chick after feeding.

Foot hygiene and care cannot be underestimated. Minor digit lacerations acquired by contact with certain abrasive nests or bedding materials, and/or hardened food or feces in the grooves of the digits, can lead to constricted toe syndrome (Fig. 15-53).

The size of the esophageal opening varies between the different species. For tiny neonates (i.e., lories, lorikeets, and conures) weighing approximately 5 grams at hatching, baby formula must be injected into the oropharynx very gradually to prevent aspiration into the trachea. In addition, personnel must be aware of the neonate's anatomy, such as whether the species has a crop. For species without a crop, the firmness of the abdomen can be checked to assess if the chick is full.

FIGURE 15-51 Brooders with large chicks must be kept under constant supervision to avoid accidents. The blue and gold macaw (*Ara ararauna*) chick in the image jumped out of the coop and nearly drowned in the dishes of water placed close by to increase the humidity in the brooder.

FIGURE 15-52 Hygiene is of vital importance in the nursery. Fruit eater chicks with large beaks such as this green winged macaw (*Ara chloropterus*) neonate benefit from adequate cleaning after each feed to avoid bacterial and fungal infections.

Knowledge of neonatal psychology and behavior is important when deciding on the appropriate housing requirements. The crowding of chicks commonly indicates a suboptimal environmental temperature. However, chukar partridge (*Alectoris chukar*) chicks, for instance, are inclined to overcrowd in corners, and this can result in mass

FIGURE 15-53 The constricted toe syndrome is a relatively common medical condition in neonates and usually occurs due to entanglement with nesting strands and/or hardened feces or feeding material.

FIGURE 15-54 Chukar partridge (*Alectoris chukar*) chicks have the tendency to crowd in corners of the coop, resulting in significant mortality due to asphyxiation and overheating. Using round enclosures during the first stage of development helps minimize mortalities due to crowding. Note that the chicks still huddle together, but they could move around freely if overheated. (Courtesy Dr. Jaime Samour.)

asphyxiation. Provision of enclosures without corners (e.g., round enclosures) may be helpful (Fig. 15-54). Hyperventilation in a neonate indicates hyperthermia or overfeeding, causing severe compression of air sacs by a distended proventriculus. It is important to monitor the temperature of respective age groups. With both altricial and some precocial species (e.g., ibises, flamingos), neonates can appear limp after hatching. Intervention is not needed, because the neonate is only exhausted from the pipping process. As a general rule, there is no need to feed immediately, because the hatchling will obtain energy from the unabsorbed yolk in its abdomen. Feeding can start only after the chick defecates its first stool (meconium), indicating that normal gut reflex and peristalsis are present. However, there are exceptions in some precocial species (see later). Some neonates are prone to ingesting objects and nest substrate or flooring out of curiosity, and/or ingesting large food items, leading to obstructions and impactions. Personnel should exercise extreme care and inspect containers and enclosures to avoid such accidents.

The Food

Food safety and hygiene are crucial to avian neonates. Food labels (stickers or tags in fruits) should be removed before food preparation to prevent intestinal foreign body obstruction. It is important to note that food items that are safe for one species can be unsafe for others. For example, ordinary chicken pellets can cause hemochromatosis (iron storage disease) in some species such as ramphastidae (e.g., toucans) if dietary levels exceed their iron requirement of 150 mg/kg a day (Cornelissen and Ritchie, 1994). Utensils used to prepare meat, fish, or mice (including pinkies) must not be used for cutting vegetables and fruits, to prevent bacterial cross-contamination. For effective cleaning of utensils, oil and biofilm that protect microbes from disinfectants must be removed by using products for deep cleaning such as F919 (Health and Hygiene, South Africa). Washing fruits and vegetables to remove soil contaminants can prevent the occurrence of bacterial enteritis and toxicities after ingestion of trace agricultural chemicals.

Sensory evaluation of food items, such as inspecting pellets for molds and smelling the baby food for rancidity, can eliminate the occurrence of aflatoxicosis and fatty acid deficiencies. In addition to fibrous plant material or seeds, growing neonates should be offered grit material (a dish of coarse sand) to assist in breaking down the hard food material into digestible products. It is important to recognize the danger of improper thawing methods. Thawing fish, meat, and whole-animal diets, inclusive of mice and pinkies, at room temperature can significantly increase microbial contamination. Thawing meat or fish by soaking can result in pale meat, depleted of iron and water-soluble vitamins. The presence of thiaminase in fish diets, which can lead to hypovitaminosis B1 in neonatal piscivores, should not be overlooked. Cleanliness and sanitation of the cold storage facility and equipment are essential. Refrigerators and freezers for food storage must not be used to contain unnecessary items such as carcasses or eggs for postmortem. Sharing hand-feeding utensils between neonates should be avoided at all times.

Food presentation and the manner of feeding baby formula should be considered. Baby formula with substandard temperature (< 40° C) can result in slow crop emptying. Heating of food using microwave ovens should be carried out with extreme caution (see later). For smaller psittacine species (e.g., lories, lorikeets, and conures), mixing the formula to achieve a completely homogenous mix can prevent slow crop emptying, impaction, and indigestion. In addition, aspiration can be prevented in these neonates through gradual pressing of the plunger of the feeding syringe when the esophageal opening is naturally small (Fig. 15-55). Most importantly, the hand feeder must synchronize with the gaping reflex, ensuring gradual injection of food into the esophageal opening to prevent aspiration. It is wise not to feed a neonate if gaping reflex is absent, which usually indicates absence of hunger. Other factors that can suppress the gaping reflex include fear, stress from noise, and disturbance. For highly diluted diets (e.g., egg-based liquid diets), the formula should be delivered slowly at the side of the beak. It is also recommended to hold the head of the neonate above the level of its shoulder until the baby formula moves down into the crop or below the level in the thoracic level in species without crop. When delivering the food, it is advisable to remove air pockets and bubbles from the syringe to prevent aerophagia. Overzealous provision of food to a gaping neonate can be life-threatening.

Chitin is indigestible to hand-reared passerine neonates, and excessive amounts of insect diet given in the very early stages of development can lead to gastrointestinal impaction and even death. When feeding insectivorous neonates (e.g., starlings, mynahs), early larval stages of insects (e.g., white mealworms) with minimal chitin material should be fed during the early stages of hand-rearing (Fig. 15-56). For

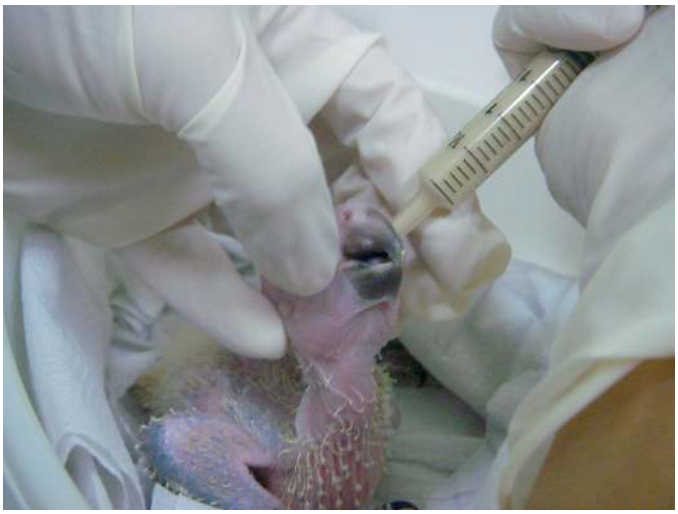


FIGURE 15-55 Aspiration pneumonia in small neonates can be prevented through the careful and gradual pressing of the plunger of the feeding syringe.

FIGURE 15-56 American robin fledglings. Note the numerous mealworms (*Tenebrio molitor*) and crickets (*Acheta domesticus*) in the diet. The bodies of these invertebrates have high chitin content, but their provision at this stage of development is considered safe. (Courtesy Susan Birch.)

further information on diets and feeding strategies for neonates, the reader is referred to Chapter 3.

The Immediate Environment

Thermoregulation using external heat sources such as brooder lamps or brooder equipment can be more of a problem than a solution when positioned too near or too far from the neonates, and/or in the event of equipment failure. Lamps kept too close to neonates can result in hyperthermia and death. Faulty brooder temperature and humidity

FIGURE 15-57 Quail (*Coturnix japonica*) chicks housed in a suspended coop. The sidewall is made of laminated plywood, and flooring is made of PVC-coated 5 × 5-mm welded mesh. Water troughs and feeders are available on both sides of the coop. Lighting and heat are provided by two suspended 200-W bulb lamps. Note that the corners of the coop have been removed by installing concave-shaped laminated plywood. (Courtesy Dr. Jaime Samour.)

displays can give false information; thus checking and calibrating systems before use are highly advised. This can also be achieved by placing a separate thermometer and hygrometer inside the brooder to provide a second reading. Data loggers are very useful tools to monitor temperatures in brooders. If light and heat intensity are correct, the neonate will be seen sleeping well. For precocial species, gradient temperature of at least 8° to 12°C from the main heat source is important (Fig. 15-57). This will allow neonates to freely move away from the heat source to achieve comfort and adequate thermoregulation. With precocial aquatic neonates (e.g., ducks, flamingos), if bathing dishes in an indoor setup are provided, it is advisable to install a heat lamp at one area of the enclosure or room. This will aid in the drying process of the skin and feathers and prevent hypothermia, which could prove fatal to neonates younger than 2 weeks old. Another good husbandry practice is to place a heating pad under the bathing dish to continuously provide warm water to precocial aquatic neonates. This is important in countries experiencing cold weather during the spring. A photoperiod of 24-hour light using conventional fluorescent bulbs does not allow precocial neonates to rest. To address the issue of "light overdose," an alternative practice is the use of porcelain bulbs emitting heat in the absence of light. Ideally, rearing rooms should be provided with a separate light source during the day that turns off automatically in late afternoon or early evening with the aid of a timer switch according to a previously designated photoperiod.

Air space management is equally as important as maintaining sanitized surfaces. Ammonia buildup commonly occurs when fecal waste from piscivorous neonates and raptors is not removed regularly from closed environments, such as brooders or small rooms. Respiratory distress and depression can be observed in neonates after inhaling excessive ammonia. Frequent changing of nest substrate and providing optimum negative pressure ventilation systems are two methods to manage this issue. The incubator air space can be kept sanitized by adding disinfectant in the water container and by removing unviable eggs before contents decompose.

The type of floor and nest substrates can affect the health of the neonate. Loose threads from towels can become entangled in the feet or rings of neonates and may cause constrictions. Synthetic soft mats

can be pinched off, or synthetic fibers can be ingested by neonates with instinctive pecking behavior (e.g., flamingos, ostriches), resulting in foreign body intestinal obstruction and impactions. In addition, a flooring that is too smooth and deficient in traction can result in development of angular and rotation leg deformities. In the case of nest-reared neonates (e.g., psittacines), it is a common practice to change nest substrate annually to prevent predisposing neonates to respiratory disorders.

Pest infestations such as ants attracted to traces of fruit diets on beddings of neonates can inflict painful bites (Fig. 15-58). This can be detrimental to small neonates after triggering neurogenic or anaphylactic shock. Rats can kill or injure nest-reared neonates in outdoor environments. Rat infestation in the food preparation area can also contribute to occurrence of salmonellosis and other bacterial enteritis. A good pest control program should be designed and implemented in and around incubation, hatching, and rearing units.

The simplicity of these recommendations cannot be underestimated, and implementing them can significantly reduce neonatal morbidity and mortality in hatching and rearing units.

PEDIATRIC HISTORY EVALUATION

It is important to note that the health of chicks within a rearing unit depends on many factors, including the health of the parents and breeding history, the condition of any siblings, and any problems encountered during incubation and hatching. The pediatric diet, preparation, and the amount and frequency of feeding are also part of the history. The rearing environment must be examined, including the substrate, cleaning and disinfection routines, and the temperature and humidity of brooders. The behavior of the chick, its feeding response, and the color, consistency, and volume of its feces, urine, and urates must also be assessed.

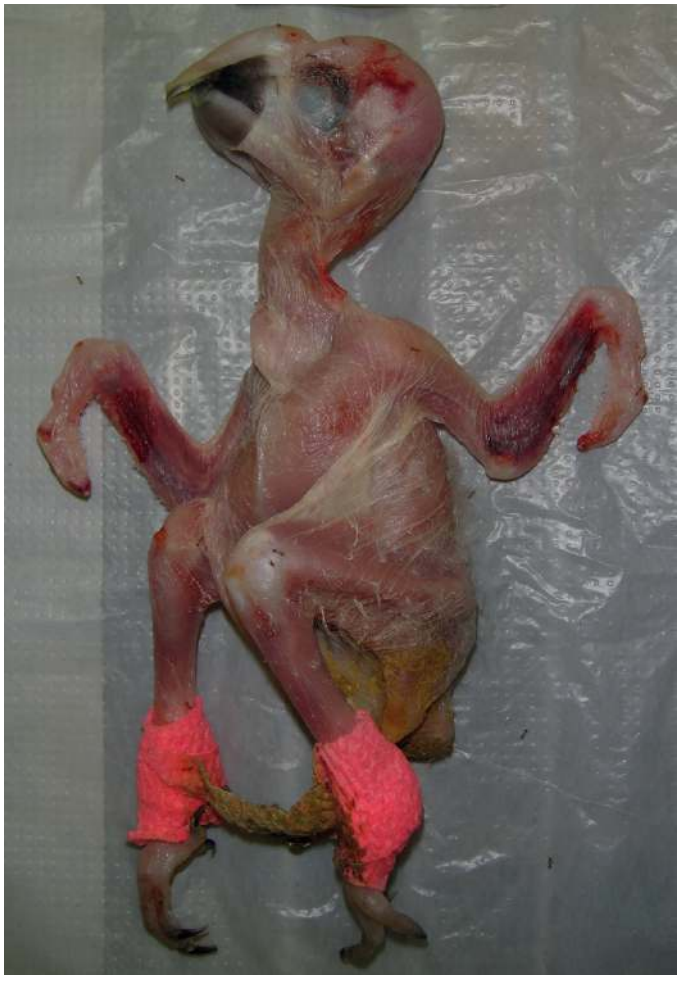


FIGURE 15-58 Scarlet macaw (*Ara macao*) neonate killed by ants within the brooder. Note the numerous hematomas sustained while trying in vain to avoid the painful ant bites.

POSTHATCHING CARE TO ASSISTED HATCHING CHICKS

Any chick that has had to be assisted during the hatching process needs extra attention. Assisted hatch chicks are susceptible to posthatching conditions due to the combination of exhaustion and an incompletely absorbed yolk sac (Fig. 15-59). Despite intensive and early antibacterial therapy, many of these chicks succumb to yolk sac infections. Some malpositioned chicks appear to be "physically exhausted" by the time intervention has provided a breathing hole or freed them. These chicks have a tendency to fade and die over the first 24 to 72 hours after hatching.

Once a diagnosis of malposition is made in a chick that has failed to internally pip, immediate but careful ovotomy over the site of the bill is recommended to provide a breathing hole for the chick. Assessment of membrane vascularity should determine the speed of further assistance. The use of radiosurgery to cut the membrane in which blood vessels had not regressed has been described (Olsen and Duvall, 1994) and may be applicable in some circumstances. Chicks that are assisted but that are still strong at the end of the process tend to be more viable compared with chicks that are "tired" because of delayed intervention. The success of assisted hatching is mainly down to timing, but unfortunately this is easier to determine retrospectively than early in the morning or late in the evening, when many of these cases tend to occur. It is important to provide antibacterial therapy to chicks from assisted hatches. It is well-known that the survival rate of chicks that hatch with larger umbilical protuberances is lower (Joyner, 1993).

FIGURE 15-59 Greater flamingo (*Phoenicopterus roseus*) chick recently hatched. The chick is weak and exhausted after the pipping and hatching process. This is normal, and nursery personnel should not intervene or immediately provide therapeutic support.

All chicks with assisted hatches should be given the following:

- Application of 1% iodine solution to the umbilici of chicks promptly.
- Administration of a broad-spectrum antibiotic for 72 hours and subcutaneous (SC) fluids or per os (PO) electrolytes for 24 to 48 hours, because newly hatched chicks are prone to dehydration.
- Supplementation of the rearing diet with probiotics (e.g., Avipro, Vetark, Winchester, UK) from 0 to 14 days.

RINGING

Captive bred birds should be close rung. This is normally done within the first 5 days after hatching for psittacines and at about 2 weeks of age in raptors. When ringing, a little Vaseline makes the task of slipping the ring over the toes easier.

ANESTHESIA OF CHICKS

The only anesthetic that can be recommended for chicks is isoflurane administered by a face mask. Maintaining body temperature is essential, especially in poorly feathered neonates. The use of heating pads and/or lamps and the use of soft towels to wrap neonates during handling cannot be overemphasized when handling in temperate countries or in air-conditioned rooms.

YOLK SAC DISORDERS

Normal Yolk Sac Resorption

The yolk of the egg supplies nutrition to the developing embryo and newly hatched chick, and antibodies for its passive protection. Before hatching, the yolk sac is withdrawn into the abdominal cavity, and the chick's navel closes over it. The speed of resorption is further affected by temperature, stress, and subclinical infections. Noy *et al.* (1996) demonstrated in poultry that providing hatchlings with drinking water early in the posthatch period improves the utilization of the yolk and facilitates motility of the digestive tract. Studies in domestic chickens (*Gallus gallus*) and turkeys (*Meleagris gallopavo*) have shown that access to feed immediately after hatching enhances the development of the intestine during the immediate posthatching period (Uni, 2005).

Unretracted Yolk Sac

Any chicks with an unretracted yolk sac and open umbilicus should be placed in a clean environment and the umbilicus swabbed with povidine iodine or chlorhexidine. If the yolk sac does not become internalized, it is susceptible to trauma and infection. Surgery to mechanically internalize some or all of the yolk sac and to carefully ligate and remove the rest may be necessary. Survival of chicks with partially unretracted yolk sacs is higher than survival of those with total unretraction. Amputation of the yolk sac may be necessary. A single sterile ligature is placed around the stalk, and the yolk sac is amputated distal to the ligature. Fluid therapy and administration of antibiotics are both important.

YOLK SAC INFECTION

Pathogenesis of Yolk Sac Infection

Omphalitis, yolk sac infection, and retention of the yolk sac are different aspects of the same condition and are common causes of mortality in chicks. The infection of the yolk sac can take place via different routes: through the shell, the chorioallantoic membrane (CAM), the albumen, and through the umbilicus at hatching. In species such as ratites (Huchzermeyer, 1998), bacteria penetrating the shell during incubation can remain localized under the CAM by the antibacterial action of the albumen. When, before hatching, the yolk sac is drawn into the abdominal cavity, any bacteria on and in the CAM can travel along the navel duct and penetrate the yolk sac. Ratite chicks that do not drink because they are kept at too low a temperature, or that become dehydrated because they are kept at too high a temperature, will satisfy their water requirements from the yolk sac, which becomes inspissated and cannot be resorbed further (Huchzermeyer, 1998).

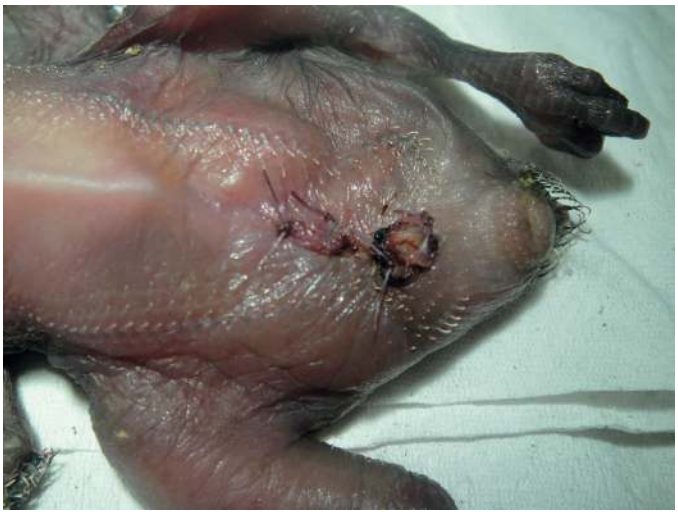


FIGURE 15-60 Victoria crowned (*Goura cristata*) pigeon with retained yolk sac immediately after surgery. The survival rate of such neonates varies considerably between species and individuals.

Diagnostic Features of Yolk Sac Infection

If the retained yolk sac is large enough, it can be palpated. In ratites, ultrasound has been used to visualize yolk sacs and to monitor the normal regression of yolk sacs in healthy chicks (Blue-McLendon and Homco, 1995). If the infection has taken place via the navel around hatch, it is possible to find an inflamed area on the yolk sac wall around the navel duct and around the umbilical region. In cases of an infection of intestinal origin, inflammation around the umbilicus and yolk sac is absent. A common finding at postmortem examination are green-colored yolk sac contents, and although this can sometimes be associated with an infected and autolyzing yolk sac, this can also occur in noninfected yolk sacs. The green discoloration occurs when bile pigments enter the yolk sac through the vitello-intestinal duct. In ratites, this is believed to take place because of the abnormal movement of intestinal contents if the intestine is empty, if the chick has not been fed, or if it is not eating (Huchzermeyer, 1998).

Treatment of Yolk Sac Infection and Retention

The yolk sac is inaccessible to the antibodies, which if given time, the chick might be able to produce against the bacteria; it is also inaccessible to antibiotics given to the chick. Thus the condition cannot be remedied in individual chicks by antibiotic therapy. Surgery to remove the retained yolk sac has been described (Bennett and Harrison, 1994), but success rates vary (Fig. 15-60). The problem is that the condition is generally very acute, with chicks often presenting profoundly and acutely ill and generally not in a suitable state for surgery (Box 15-1).

MUSCULOSKELETAL DISORDERS

Nutritional osteodystrophy, rickets, and osteomalacia are important neonatal conditions and have been covered in Chapter 13.

BOX 15-1 Prevention of Yolk Sac Infection

- Egg and incubator hygiene
- To prevent the entry of bacteria via the umbilicus, strict incubator hygiene is necessary, as has been discussed
- Fumigation/disinfection of eggs after collection/before setting
- Disinfection/fumigation of the incubator and hatcher
- Removal of infertile eggs and eggs with dead embryos
- Disinfection of all surfaces that come into contact with the hatchling's umbilicus during the first few days after hatching
- Nest hygiene—nest sites should be covered with clean sand at the start of the season, and surface sand should be regularly changed at intervals throughout the season
- Reduce environmental dust in incubation room—environmental dust that is drawn with the air into the incubator is also an important source of contamination
- Treatment of the navel—at hatch, a wound disinfectant should be sprayed or gently applied using cotton buds on the umbilical region of every chick

FIGURE 15-61 Barn owl (*Tyto alba*) neonate with lateral deviation of the maxilla, or scissors beak. Physical therapy and some trimming are indicated. This condition is most commonly seen in psittacine birds.

Splayed Legs

Splayed legs is a common condition in which one or both legs deviate laterally. If left untreated, this condition can progress to valgus deformities. Neonatal altricial species such as parrots or raptors can be packed in bowls with nonslippery substances or padding that allow chicks to sit with legs underneath them.

For precocial species such as bustards or game birds, splayed legs are easily treated at an early stage by hobbling the legs together with adhesive conforming bandage (VetRap, 3M Animal Care Products, USA), leaving enough freedom of movement to allow the chick to walk. For prevention of splayed legs in precocial species, it is important to provide a warm, nonslip surface and to watch the newly hatched chicks closely so that if problems do occur, legs can be hobbled promptly. Several factors, including incubation conditions and the condition of the floor of the rearing unit, are thought to contribute to the development of this condition in bustards.

Twisted or Rolled Toes

Rolled toe or medial rotation of the phalanges is a common finding in precocial chicks such as bustards and ratites. Although treatments using splints and bandages have been suggested for ratites, because of their size, it is often hard to apply bandages to the smaller species of birds; however, when it is mild, this condition usually corrects itself as the chick develops. Exercise on a thick, sandy substrate improves this condition very well.

Constricted Toes

Constricted toe syndrome is reported in eclectus parrots (*Eclectus roratus*), macaws, and African grey parrots (*Psittacus erithacus*). The condition consists of a constricting ring around a toe. This leads to dry gangrene and the loss of the portion of toe distal to the constriction. The etiology is uncertain, but increasing the humidity in the brooder appears to reduce the incidence. The circumferential constrictions should be debrided and incisions made laterally and medially through the band. The toe needs to be soaked daily in dilute povidine iodine solution and kept bandaged.

Beak Malformations

In parrots the three most common beak malformations are lateral deviations of the maxilla (scissor beak), prognathism (pug beak), and mandibular compression deformities. After 2 weeks of age, while the beak is still pliable, physical therapy and trimming are indicated (Fig. 15-61). After calcification, frequent trimming, acrylic implants, or extensions may be needed to correct the malformation. Corrective techniques are described by Martin and Ritchie (1994).

Slipped Wing

The term "slipped wing" is used to describe a condition in growing chicks where the increased weight of the rapidly developing primary feathers causes an overextension of the carpal joint and the outer wing starts to droop. If left untreated, the wing twists outward at the carpal joint, resulting in a permanent deformity. Slipped wing is also known as "angel wing" or "dropped wing." Preventive measures should be taken as soon as the chick's wings look as if they are going to droop. Micropore tape or common masking tape is used to tape the wingtip to the upper wing for support for 2 to 4 days, with the tape changed every couple of days as the chick grows. Although high growth rates and high-energy/high-protein diets are commonly blamed in some species like bustards, nondietary factors may also be involved. Taping the affected primaries in a natural position at the first sign of the outward turning will permanently correct the deformity.

GASTROINTESTINAL TRACT DISORDERS

Impactions

Clinical Description

Mechanical gastrointestinal tract conditions are not uncommon causes of death and morbidity in chicks, particularly ratites and bustards. Usually grit or bedding is the item that causes impaction. Impactions are usually caused by chicks ingesting foreign matter, which accumulates in the proventriculus and blocks the entrance to the ventriculus. With the entrance to the ventriculus blocked, no food can pass through, the ventriculus stops contracting, and the bird will die of starvation. Some ingested foreign bodies will also perforate through the proventriculus or ventriculus, causing problems with peritonitis and septicemia (Fig. 15-62).

Clinical Signs and Diagnosis

Impacted chicks show symptoms of starvation and gastric stasis, lower growth rate, and weight loss. Affected birds become anorexic, dull and

FIGURE 15-62 Intraoperative procedure to remove a large piece of wood ingested by a pink backed pelican (*Pelecanus rufescens*) neonate in a nursery. The wood blocked the exit of the proventriculus, arresting the movement of food into the ventriculus. The piece of wood was removed successfully by gastrostomy.

FIGURE 15-63 Cloacal prolapse in a neonate chick. This medical condition is very often related to nutritional deficiencies, diarrhea, impaction, and tenesmus.

may vomit. Septicemia is often a sequel to impactions, particularly if there is perforation of the intestinal tract, and in these cases the chick usually deteriorates rapidly. Palpation of the abdomen allows the distended proventriculus and/or ventriculus to be felt. Radiography is useful in screening for radiopaque foreign bodies.

Treatment and Prevention

Impacted material can be removed by a ventriculotomy operation in robust chicks of large species such as ratites and bustards. Gastric lavage is recommended in ratite chicks, followed by supportive therapy (fluids, warmth, and gastric stimulants such as metoclopramide or cisapride). Surgery carries a poor prognosis in young, delicate chicks from smaller species. Parenteral rehydration and the use of metoclopramide and liquid paraffin are recommended for these cases.

CLOACAL PROLAPSE

In young birds this condition is often associated with severe diarrhea, impaction, nutritional deficiencies, and tenesmus (Fig. 15-63). Cloacal prolapse occurs sporadically in individual bustard chicks. In ratites cloacal prolapse is associated with cryptosporidiosis and histomoniasis (Huchzermeyer, 1998). Purse string sutures are usually satisfactory as long as the primary cause, such as diarrhea, is dealt with. Cloacopexy is an alternative technique to purse-string sutures, and the technique is described in Chapter 11.

CROP STASIS

Crop stasis is common in neonatal psittacines. Primary causes include crop infection, foreign bodies, burns, dehydration of food in the crop, and food that is too hot or too cold. Secondary causes include distal gut stasis due to ileus, bacterial or fungal infection, polyomavirus, foreign bodies, and hypothermia. Management of crop stasis comprises crop culture, cytology, cloacal culture, and, in psittacines, the placement of crop bras. Medical therapy includes fluids, crop flushing, removal of foreign bodies, and antibiotics or antifungal therapy as indicated by culture results.

CROP BURNS

The temperature of the food is a critical factor to promote the correct feeding response in psittacines. Microwaves should be used very cautiously to heat food, because hot spots are created in the food that can cause burns to the crop. Crop burns occur secondarily to feeding excessively hot food. The burn must fistulate through before surgery is indicated. During this time, the area must be kept clean, and the bird should be given antibiotics and antifungals based on crop culture results. At surgery, the area around the fistula is debrided, the edges of the skin separated and freshened, and the wound closed in two layers, crop followed by skin. For further information on surgical repair of the crop, the reader is advised to see Chapter 11.

STARGAZING

Thiamine (vitamin B_1) deficiency can cause seizures, incoordination, and opisthotonus in raptor chicks. Signs resolve after administration of vitamin B complex.

INFECTIOUS DISEASES

Enteritis

Enteritis is an important cause of mortality in chicks and is influenced by many factors, including intestinal flora, nutrition, environmental factors, and pathogens.

Intestinal Flora

Birds hatch from the egg with a completely sterile digestive tract, and microbes are quickly picked up within the nest or incubator. The source of the microflora depends on the hygienic conditions of the environment and the presence or absence of the parents (Klasing, 1998). In captive breeding projects, chicks are incubated and hatched from sterilized eggs, incubators, and hatchers, and after hatching they may be hand-reared in a heavily disinfected environment. Under these circumstances, it is hard for chicks to acquire a normal intestinal microflora, and they are at risk of becoming colonized by

inappropriate bacteria. The normal microflora comprises useful bacteria that protect the intestine from infection with pathogenic bacteria by occupying the available attachment sites (competitive exclusion) and by creating an environment (pH and metabolites) in which pathogens cannot survive.

PROBIOTICS

Formulated probiotics can be used to provide some protection to chicks, but ideally complete protection can only come from providing a complete "species appropriate" intestinal flora. The chicks of many avian species have been observed eating the feces of their parents (Klasing, 1998). Coprophagy is considered by many authors to help seed the digestive tract with beneficial microflora from the established flora of the parents' posterior digestive tract (Klasing, 1998; Jeffrey, 1999). The feces of rabbits or goats have been used successfully in ostriches (Huchzermeyer, 1998). Anecdotally and clinically, commercial probiotic preparations appear to be beneficial, but efficacy studies are needed to demonstrate that these agents do good, rather than just causing "no harm."

ANTIBIOTICS

We are all guilty, aviculturists and veterinarians alike, of tending to combat any emerging disease of chicks with the liberal use of broad-spectrum antibiotics. Very often, instead of their desired effect, inappropriate use of antibiotics can suppress the normal intestinal flora and immune system of the chick.

BIOSECURITY AND SOURCES OF INFECTION

Prevention of infection by following strict hygiene is important. Biosecurity should be designed to keep pathogens out of avicultural rearing units. Pathogenic bacteria, including *Salmonella,* can be transmitted by flies, reptiles, rats, and mice that may be attracted to rearing facilities. The failure to establish a normal intestinal flora, or its imbalance by inappropriate use of antibiotics, can make the chick vulnerable to infection by pathogens causing enteritis, such as *Salmonella* and other enterobacteria. It should also be remembered that some precocial species are coprophagic, and any chicks with enteritis should be isolated because transmission within a group can occur rapidly.

ENVIRONMENTAL FACTORS

Environmental factors include:

- Heat: Chicks that have been exposed to excessive heat can develop dehydration, impaction, alimentary stasis, and superinfection.
- Cold: Similarly a lowered body temperature, most often caused by the overnight failure of heat lamps or brooder thermostats, can result in the reduction of the activity of the immune and digestive system.
- Stress: Stress has a negative effect on the functioning of the immune system. Common stress factors encountered by chicks reared in groups include death of pen mates so that chicks are reared as singles rather than in a group, and cold, heat, overcrowding and transfers to the next stage of rearing facilities.

FUNGAL GASTRITIS

Fungal infections of the digestive tract of bustards and other avian species are caused by fungi from the environment or by normal inhabitants of the intestinal tract that, under certain conditions, become invasive and pathogenic. Infections with *Candida* spp. can cause stomatitis, proventriculitis, and ventriculitis. Candidiasis in bustard chicks is a side effect of prolonged antibiotic therapy. Other fungi (*Mucor* spp., *Aspergillosis* spp., *Rhizopus* spp.) have been associated with outbreaks of gastric mycosis in ratites (Perelman and Kuttin, 1992).

AVIPOX VIRUS

The commonest manifestation of avipox virus is papules and erosions around the unfeathered skin of the eyes, beak/skin margin, and legs. A wet or diphtheritic form is also seen, and this form causes necrotic yellow plaques in the oropharyngeal cavity and respiratory tract. Avipox virus in adult birds is reviewed in Chapter 14.

POLYOMAVIRUS (BUDGERIGAR FLEDGLING DISEASE)

This disease is seen in a wide range of psittacines. The feather abnormalities seen in budgerigars (*Melopsittacus undulatus*) are less common in other species. Baby birds usually die after showing signs of depression, crop slowdown, diarrhea, subcutaneous hemorrhages, dyspnea, paralysis, or tremors. Some birds show signs of feather dystrophy.

PSITTACINE BEAK AND FEATHER DISEASE

This has been discussed in Chapter 14 (Fig. 15-64).

CHLAMYDIOSIS

A chlamydiosis outbreak in 3-week-old houbara bustard (*Chlamydotis undulata macqueenii*) chicks living in outdoor pens was reported at the National Wildlife Research Center, Taif, Kingdom of Saudi Arabia. The birds presented with severe clinical signs including blepharoconjunctivitis, tracheobronchitis and pneumonia, as well as poor-quality feathers. Successful treatment comprised enrofloxacin for a 2-week period (Gerlach, 1994), as well as mucolytic drugs. Chlamydiosis in adult birds is reviewed in Chapter 14.

FIGURE 15-64 Two young cockatoos severely affected by the psittacine beak and feather disease. This is a viral disease transmitted by a Circovirus. Note the characteristic absence of feathers and the severely distorted beak on one cockatoo. (Courtesy Robert Doneley.)

CONGENITAL PROBLEMS

These are not uncommon, and unfortunately issues caused by inbreeding are likely to become increasingly frequent in the future. It is becoming more difficult or impossible in Europe because of import restrictions for breeders to legally acquire wild-caught unrelated breeding stock. Given that few aviculturists have good record systems and that many unscrupulous breeders sell related birds as unrelated, it is inevitable that the limited captive pool of birds used for breeding will become more inbred with each passing generation. Splay legs, scoliosis, lordosis, opisthotonus, cerebellar defects, joint deviations, and stunted birds are examples of congenital and growth problems seen in psittacines and raptors. It is likely that genetic testing will become an important tool for diagnosing genetic and inbreeding issues in the future.

REFERENCES

Bailey TA: *Diseases of and medical management of houbara bustards and other otididae*, Abu Dhabi, 2008, Environmental Agency Abu Dhabi.

Bennett A, Harrison G: Soft tissue surgery. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, Fla., 1994, Wingers Publishing, pp 1096–1136.

Blue-McLendon A, Homco LD: *Ultrasound determination of yolk sac size in ostrich chicks. Proceedings of the Association of Avian Veterinarians*, 1995, pp 311–312.

Cornelissen H, Ritchie BW: Ramphastidae. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, Fla., 1994, Wingers Publishing, pp 1276–1283.

Doneley B: Management of captive ratites. In Harrison GJ, Lightfoot TL, editors: *Clinical avian medicine*, Palm Beach, Fla., 2006, Spix Publishing, pp 957–990.

Flammer K, Clubb S: Neonatology. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, Fla., 1994, Wingers Publishing, pp 805–841.

Gage LJ, Duerr RS: *Hand rearing birds*, Hoboken, N.J., 2008, Wiley-Blackwell.

Gerlach H: Chlamydia. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, Florida, 1994, Wingers Publishing Inc, pp 984–996.

Huchzermeyer FW: *Diseases of ostriches and other ratites*, Onderstepoort Veterinary Institute, Republic of South Africa, 1998, Agriculture Research Council.

Jeffrey JS: *Use of competitive exclusion products for poultry*. Poultry Fact Sheet 30. Cooperative Extension, University of California, USA. http://animalscience.ucdavis.edu/Avian/pfs30.htm, 1999.

Jones M: Raptors, faeces and behavioural development and disorders. In Chitty J, Lierz M, editors: *BSAVA manual of raptors, pigeons and passerine birds*, Gloucester, UK, 2008, Quedgeley, pp 250–259.

Joyner KL: Psittacine incubation and pediatrics. In Fowler ME, editor: *Zoo and wild animal medicine current therapy 3*, Philadelphia, 1993, WB Saunders, pp 247–260.

Klasing KC: *Comparative avian nutrition*, New York, 1998, CAB International, p 64.

Martin H, Ritchie B: Orthopedic surgical techniques. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, Fla., 1994, Wingers Publishing, pp 1137–1169.

Noy Y, Uni Z, Sklan D: Routes of yolk utilisation in the newly-hatched chick, *Brit Poultry Sci* 37:987–996, 1996.

Olsen GH, Duvall F: *Commonly encountered hatching problems. Proceedings of the American Association of Avian Veterinarians*, 1994, pp 379–384.

Perelman B, Kuttin ES: Aspergillosis in ostriches, *Avian Pathol* 21:159–163, 1992.

Scott P, Stoodley J: Neonatal husbandry problems. In Beynon PH, Forbes NA, Lawton MPC, editors: *Manual of psittacine birds*, Cheltenham, Gloucestershire, UK, 1996, British Small Animal Veterinary Association, pp 205–210.

Uni Z: Factors affecting intestinal function in the pre- and post-hatch period, *Avian Poult Biol Rev* 16(1):73–74, 2005.

Wilson L, Linden PG, Lightfoot TL: Early psittacine behavior and development. In Harrison GJ, Lightfoot TL, editors: *Clinical avian medicine*, Palm Beach, Fla., 2006, Spix Publishing, pp 60–72.

CONSERVATION OF AVIAN GENETIC RESOURCES BY PRIMORDIAL GERM CELL MEDIATED GERMLINE CHIMERA TECHNOLOGY

Chunhai Liu, Vijaya Baskar

Birds play very important roles in maintaining the healthy ecosystem of nature, and also in human life. They provide food sources in the form of meat and eggs. Birds also have been used to deliver messages and are a constant source of pleasure and entertainment. There are over 10,000 bird species in nature. The population of birds has dropped drastically recently, and some are threatened or facing extinction as a consequence of human behavior such as hunting, destruction of natural habitat, and misuse of pesticides. In the latest assessment of the world's bird species in 2014, 1373 species were considered threatened with extinction, and an additional 959 species are considered near threatened (Bird Life International, 2014). Hence a total of 2332 species, nearly one-fifth of the world's birds, are treated as global conservation priorities.

In the long run, preservation and restoration of their natural habitat is the best solution. However, captive breeding could help to protect and enlarge the existing populations. Conservation of genetic resources plays an important role in maintaining species biodiversity, in promoting sustainability, and in reintroducing conserved genetic information into the gene pool through captive breeding programs. In these programs, birds are kept in controlled artificial environments with favorable health care and nutrition supply. Female birds are kept under controlled photoperiod and temperature, and are reproduced through artificial insemination. Fertile eggs are hatched in incubators. Captive breeding programs have already achieved great success in conserving some birds, such as the crested ibis (*Nipponia nippon*), houbara bustard (*Chlamydotis undulata*), peregrine falcon (*Falco peregrinus*), and others.

Germ plasma banking could be achieved through cryopreservation of various forms of germ cell, gametes, and early embryos. The most common approach to conserving germ plasma of the male bird is semen cryopreservation. Polge (1951) first cryopreserved fowl spermatozoa. Thereafter, improvement of freezing protocols has been studied in terms of various cryoprotectants, freezing-thawing procedures, and storage. Semen of many domestic poultry and wild birds has been frozen, such as that of vultures (Madeddu, *et al.*, 2009), houbara bustards (Hartley *et al.*, 1999), cranes (Blanco *et al.*, 2012), and pigeons (Sontakke *et al.*, 2004). To conserve the germ plasma from premature individual birds, or some wild birds in which the semen quality and the recovery of frozen semen are extremely poor, the alternative method is to cryopreserve the testicular tissue. The recovered testicular tissue can be ectopically transplanted and complete spermatogenesis in the body of the castrated recipient birds (Song and Silversides, 2007). In female birds, it is unlikely to cryopreserve mature ova and early embryos. Alternatively, the ovarian tissue comprising primary oocytes before yolk deposition could be frozen down. The functionality of frozen ovarian tissue can be recovered successfully after thawing. In mammals, cryopreserved ovarian tissue can be recovered via in vitro maturation and in vitro fertilization. Since vitellogenesis and deposition in avian folliculogenesis, the mature ova could not be generated in vitro. Therefore cryopreserved ovarian tissue has to be transplanted back into the normal anatomic site of recipient birds (Liu *et al.*, 2010).

The fundamental aim of conservation is to keep the natural species continuity, sustainability, and genetic biodiversity. In general, this could be achieved through conserving a vehicle cell that possesses the entire genetic information of a bird, and that can be reintroduced into the natural gene pool. Germ cell is the only candidate that is given the mission to pass genetic information into subsequent generations. Therefore cryopreservation and manipulation of germ cells have superior advantages for gametes or embryos, especially in birds for which in vitro production of ova and embryos is not possible. In recent years, technology on manipulation of avian germ cells, primordial germ cells (PGCs) in particular, has developed rapidly. In chickens, PGCs can be collected, enriched, propagated in vitro, cryopreserved, and transplanted into a host embryo (Naito *et al.,* 1994a). These cells further complete the gametogenesis in the gonad of host birds, known as germline chimera. The conserved species could be eventually restored through these chimeric birds by natural breeding. In addition, germ cell mediated germline chimera technology also provides a platform for genetic modification of birds to improve their economic performance and their disease resistance.

PRIMORDIAL GERM CELL BIOLOGY

Primordial germ cells originate at the very early stage of embryogenesis and are the precursors of germ cell lineage, which pass the genetic information throughout generations.

The Origin of Avian Primordial Germ Cells

It is believed that PGCs form by two mechanisms, epigenesis and preformation. In epigenesis, PGCs specify from a group of pluripotent cells, which are induced by signaling from other surrounding somatic cells (e.g., *Mus musculus*). In preformation, PGCs specify from somatic cells via germ plasm, which is the determinant that gives rise to the germ cell lineage. Germ plasma has been found in the egg cell cytoplasm of some model organisms (e.g., *Drosophila melanogaster*). It consists of RNAs and proteins that are synthesized maternally from surrounding nursing cells and transported into immature oocytes during oogenesis (Extavour and Akam, 2003). Recently VASA protein was identified in many model organisms with possible function to bind target mRNAs involving in germline determination. Tsunekawa *et al.* (2000) identified chicken VASA homolog (*Cvh*) gene and found its germline-specific expression. CVH-expressing cells were found from the first cleavage of chicken embryos and exclusively in PGCs later. These findings suggested that the determination of chicken (or avian) germline could be in the mode of preformation, as lower vertebrates.

Migration of Avian Primordial Germ Cells

PGCs are migratory cells. They are located in the peripheral or extraembryonic area at the early stage of embryogenesis, and they migrate into the primitive genital ridges with embryonic development. Here, we illustrate the migration pattern of chicken PGCs as a model for avian PGCs. The fertilization of chicken ova occurs 15 minutes after ovulation. The zygote initiates the first division and continues embryogenesis. By the time of oviposition, the embryo has already developed into a structure containing about 60,000 cells, known as blastoderm at stage X (Eyal-Giladi and Kochav, 1976). With cytologic and immunohistochemical staining, the chicken PGCs are localized at the central zone of the area pellucida of the ventral surface of the epiblast. Then, these cells are gradually translocated to the developing hypoblast, which further carries PGCs anteriorly to an extraembryonic site called germinal crescent at stage 4 (Hamburger and Hamilton, 1951). Subsequently, the PGCs incorporate into the forming blood islands of vascular system on the yolk sac at stage 10 to 12 (Hamburger and Hamilton, 1951). With the formation of blood vessels, PGCs enter the blood's circulation (Ginsburg, 1994).

In the vascular system, PGCs migrate passively with the bloodstream to the vicinity of the genital ridges. These cells start leaving the vascular system and actively migrate toward the genital ridges through the tissues. By stage 17 (Hamburger and Hamilton, 1951), the majority of PGCs reach and settle in the genital ridges. Unlike mammalian PGCs, which migrate all the way through tissues before colonizing in the embryonic gonad, avian PGCs migrate passively with blood circulation before settling in the primitive genital ridges (Fig. 15-65). The mechanism of migration is conserved among different avian species. Therefore the transplanted PGCs are able to migrate into the gonad of another species. This unique migration route and conserved mechanism make it more accessible to collect and transplant PGCs between embryos.

Identification of Avian Primordial Germ Cells

Primordial germ cells from various sources can be identified either through their morphologic characteristics, or using cytologic and immunohistochemical staining techniques. PGCs are approximately 15 to 20 μm in diameter, larger than the somatic cells, rich in cytoplasmic granules. The nucleus is 8 to 12 μm positioned eccentrically with a pronounced membrane. Although PGCs of different avian species show slight differences in size, shape, or pattern of cytoplasmic granules, they are still distinguishable by morphologic characteristics from surrounding somatic cells. Chicken PGCs were characterized from the blood circulation by Swift in 1914, which were found rich glycogen in cytoplasm, and positively stained by Periodic Acid Schiff (PAS) reaction (Fujimoto *et al.,* 1976; Fig. 15-66, *B*). The cytoplasmic glycogen of PGCs reduces gradually after colonizing in the gonad; therefore, the PAS stain intensity of chicken PGCs varies from different stages. PGCs of some species are found negatively stained by PAS e.g., quail PGCs. Expression of alkaline phosphatase, which has been used to identify mouse PGCs, has also been found in chicken PGCs (Merchant-Larius *et al.,* 1985; Li *et al.,* 2010).

In addition, specific epitopes on the cell surface or in the cytoplasm are also applied to characterize avian PGCs through immunohistochemical staining techniques. Antibody against stage-specific embryonic antigen-1 (SSEA-1) expressing on murine primordial germ cells also showed cross reaction with chicken PGCs (Fig. 15-66, *C*) and turkey PGCs, but not with quail and duck PGCs. Epithelial membrane antigen-1 (EMA-1), and an ovomucin-like protein (OLP) were expressed on the surface of chicken PGCs (Fig. 15-66, *D*). The VASA/DDX homologue protein being expressed in the cytoplasm of germ lineage of many organisms was also found in avian PGCs (e.g., chicken and quail, Fig. 15-66, *F*). In addition, antibodies QH1, QCR-1, and QB2 were used for identification of quail PGCs (Table 15-3, Fig. 15-67, *B*).

Collection of Avian Primordial Germ Cells

Based on the migration route toward primitive genital ridges, avian PGCs are usually collected from the following sites: central area pellucida of blastoderm (stage X), germinal crescent area, blood circulation, and embryonic gonad. In practice, circulating PGCs can be collected by withdrawing a small amount of blood from the embryo at stage 14. When collecting from other sites, PGCs are dissected from the embryo along with accessory tissues. The number of PGCs varies among species and stages. In chickens, there are about 30 PGCs at the area pellucida of blastoderm. The number of PGCs increases gradually and reaches from 100 to 150 in the germinal crescent area, about 200 in the blood circulation. Postmigratory PGCs immediately start mitotic proliferation and increase rapidly to about 1500 cells in the gonad of chicken embryo at stage 28.

Isolation of Avian Primordial Germ Cells

Freshly collected PGCs are mixed with other somatic cells and released from tissue through mechanical or enzymatic dissociation. Density gradient centrifugation (Ficoll, Nycodenz) (Yasuda *et al.*, 1992; Zhao and Kuwana, 2003), immunomagnetic separation (Ono and Machida, 1999), and fluorescence-based flow cytometry (Mozdziak *et al.*, 2005) have been applied to enrich PGCs. Density gradient centrifugation methods are based on the density and dimension difference between PGCs and somatic cells (see Fig. 15-66, *A*). Immunomagnetic separation and fluorescence-based flow cytometry can be used to isolate larger numbers of cells, and applicable antibodies exclusively binding

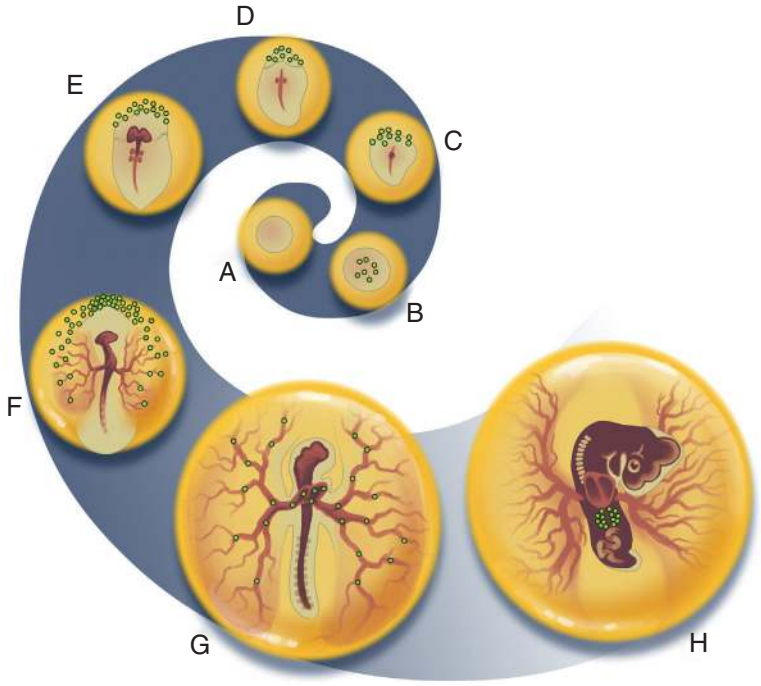


FIGURE 15-65 Migration of avian primordial germ cells. **(A),** Primordial germ cells (PGCs) located in the central zone of area pellucida of blastoderm (stage X). **(B),** Developing hypoblast carries PGCs anteriorly to the extraembryonic site. **(C),** PGCs in the germinal crescent area at stage 4, and **(D),** stage 8. **(E),** PGCs incorporate into the forming blood islands of vascular system at stage 10 to 12. **(F),** PGCs migrate with the blood circulation and to the vicinity of genital ridges. **(G),** PGCs colonized in the genital ridges. (Modified from Nieuwkoop and Sutasurya, 1979.)

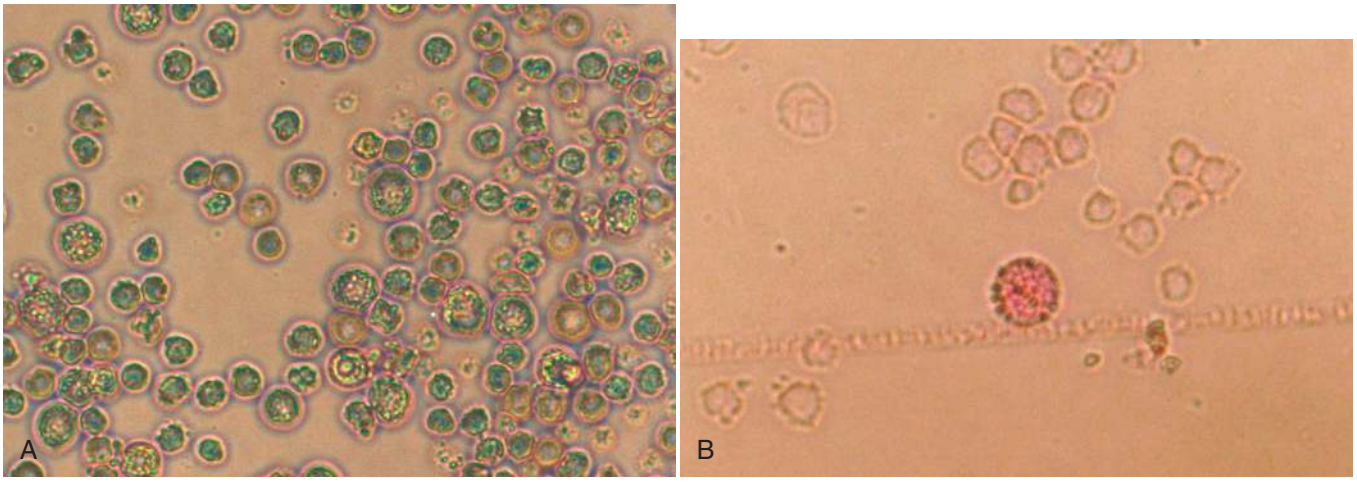


FIGURE 15-66 Identification of chicken (*Gallus gallus domesticus*) (PGCs). **(A),** Isolated chicken circulating PGCs by Ficoll density gradient centrifugation. **(B),** Periodic acid Schiff reaction (PAS) positively stained chicken circulating PGCs.

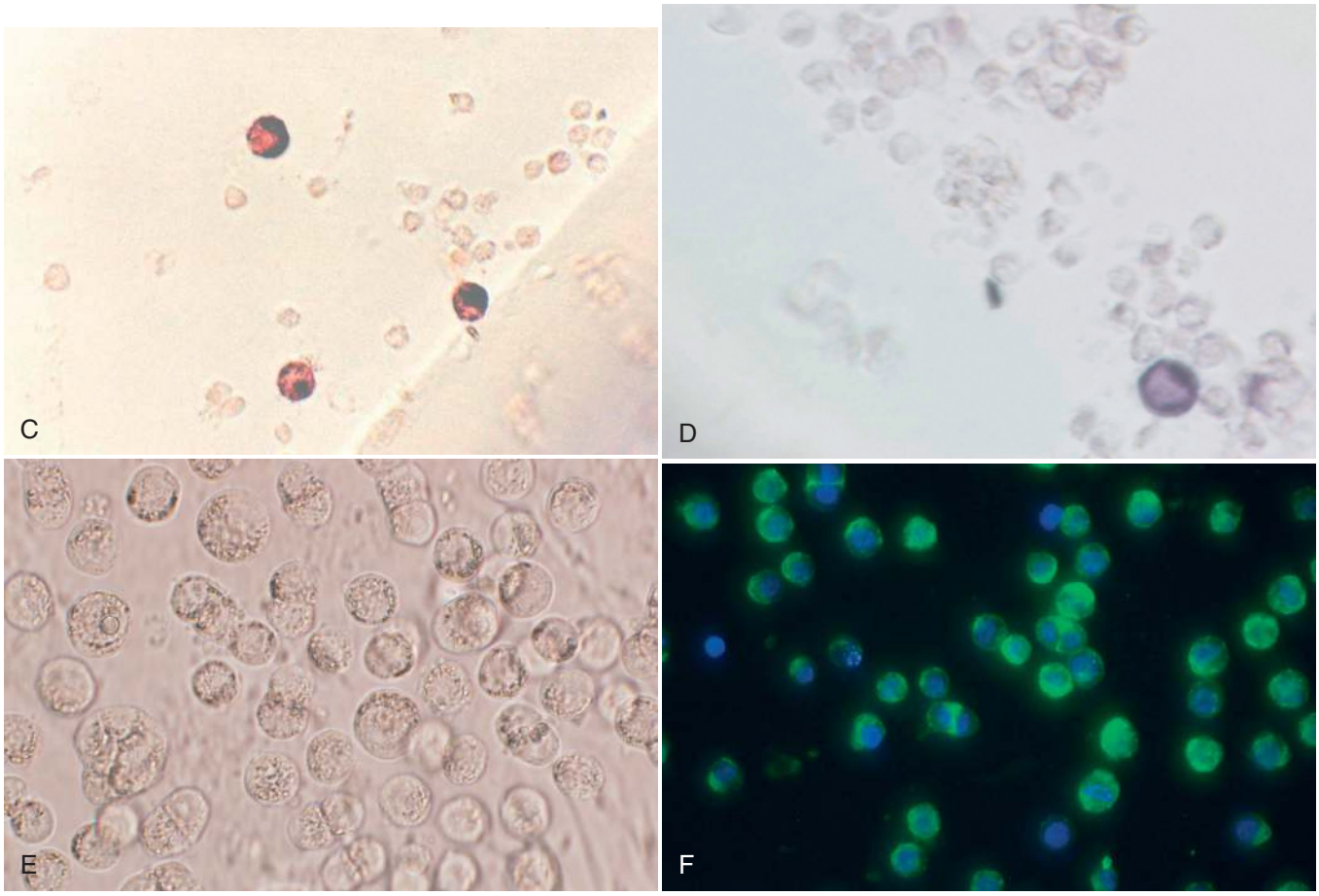


FIGURE 15-66, cont'd **(C),** Immunochemistry staining of chicken gonadal PGCs with anti-SSEA-1 (MC480); **(D),** With anti-EMA-1. **(E),** Chicken PGCs in culture. **(F),** Immunofluorescent staining of cultured chicken PGCs with anti-CVH (chicken VASA homolog protein).

TABLE 15-3 Antibody List for Identification of Avian PGCs

Species	Antibodies	References
Chicken PGCs	anti-SSEA-1, anti-SSEA-3, anti-SSEA-4, anti-integrin α6, anti-integrin β1, EMA-1, 2C9, anti-OLP, anti-VASA, anti-DAZL	Jung *et al.*, 2005; Karagenç *et al.*,1996; Halfter *et al.*,1996; Rengaraj *et al.*, 2010
Quail PGCs	QH1, QCR-1, QB2, Lectin, anti-VASA	Ono and Machida, 1999; Tsunekawa *et al.*, 2000; Armengol *et al.*, 2007
Turkey PGCs	anti-SSEA-1, anti-OLP	D'Costa and Petitte, 1999; Wade *et al.*, 2014
Pheasant PGCs	QCR-1	Kim *et al.*, 2005

PGCs are required. Most recently, a novel method was developed to purify chicken PGCs from circulation embryonic blood through lysis of red blood cells in an ammonium chloride-potassium buffer (Yamamoto *et al.*, 2007). Application of this lysis method for isolation of PGCs from other sources or species has not been reported.

Cryopreservation of Avian Primordial Germ Cells

Cryopreservation of avian PGCs has been attempted. Chicken PGCs from blood circulation and gonadal tissue were frozen by slow rate method in straw or plastic cryovial with DMSO or ethylene glycol as a cryoprotectant. The survival rate of gonadal PGCs ranged between 35.6% and 77.4% (Moore *et al.*, 2006) and 94.2% for circulation PGCs (Naito *et al.*, 1994b). The functionality of cryopreserved PGCs was confirmed through generation of functional spermatozoa or ova in germline chimeric chicken. The majority of work was published using chicken PGCs as a model. Cryopreservation of PGCs from wild birds for the purpose of genetic conservation has not been reported.

In Vitro Culture of Avian Primordial Germ Cells

Culture of PGCs has been a long-pursued dream as the potential target cell for genetic modification. The pioneer culture work was conducted with mouse PGCs. Initially, mouse PGCs could only proliferate for a limited time period in culture. With the addition of a group of cytokines (FGF_2, LIF, SCF), Matsui *et al.* (1992) found that mouse PGCs can proliferate indefinitely and dedifferentiate into pluripotent cells, known as embryonic germ cells. Karagenç and Petitte (2000) cultured chicken blastoderm cells on STO feeder and confirmed that soluble factors that secreted from the STO feeder cells facilitated the development of chick PGCs. Most recently, van de Lavoir *et al.* (2006) cultured

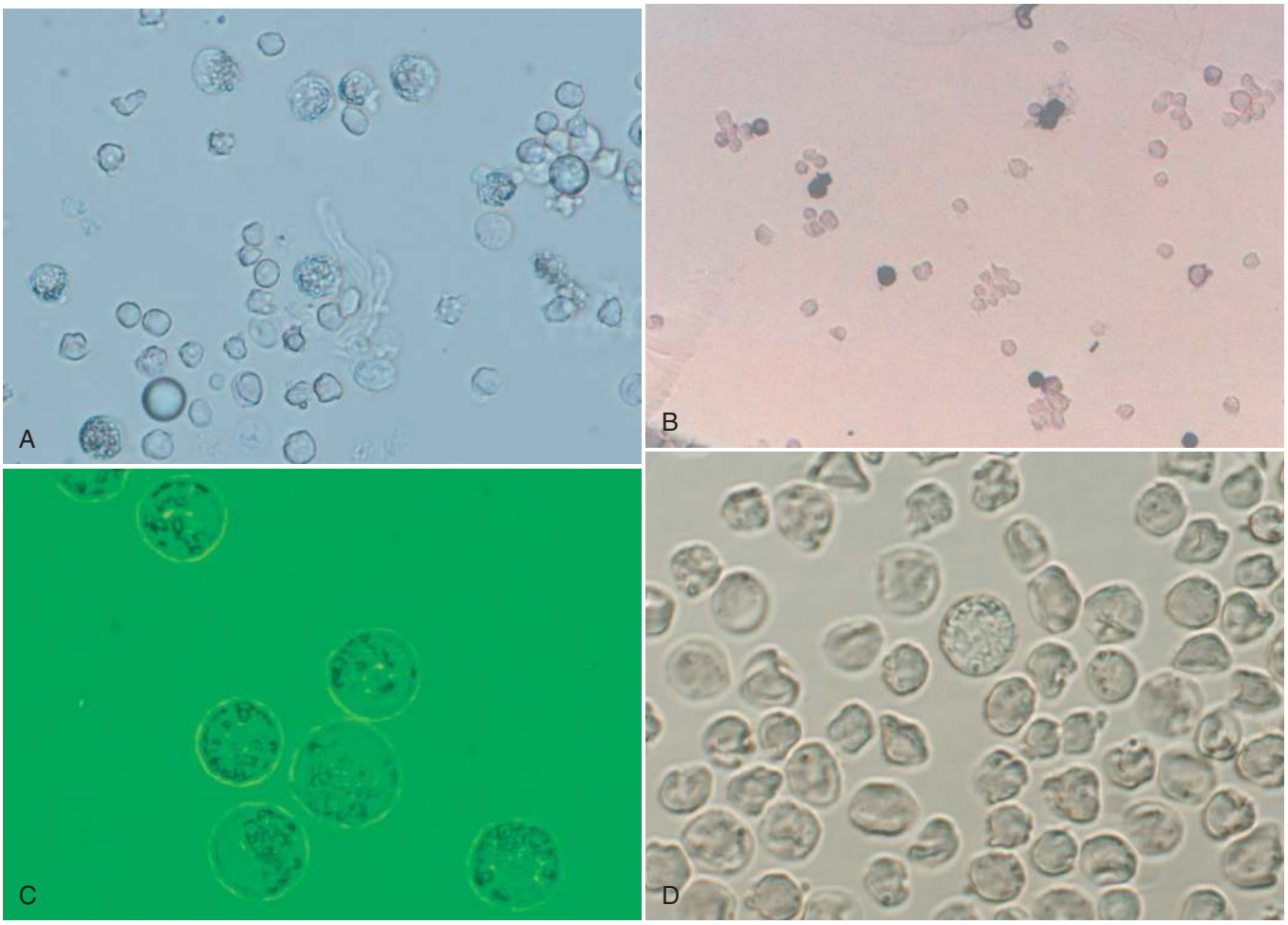


FIGURE 15-67 Quail (*Coturnix japonica*), duck (*Anas platyrhynchos*), and houbara bustard (*Chlamydotis undulata undulata*) primordial germ cells. **(A),** Isolated quail circulating PGCs by Ficoll density gradient centrifugation. **(B),** QB2 antibody stained quail gonadal PGCs. **(C),** Isolated duck circulating PGCs. **(D),** Houbara bustard circulating PGCs in blood.

chicken PGCs from embryonic blood on irradiated STO fibroblast feeder layers in KnockOut DMEM with preconditioned medium on BRL cells, 7.5% FBS, 2.5% chicken serum, 2 mM glutamax, 1× nonessential amino acids, 1 mM pyruvate, 0.1 mM β-mercapto-ethanol, 6 ng/mL SCF, and 4 ng/mL FGF. Chicken PGCs successfully proliferated into millions and still maintained the capability of germline transmission (see Fig. 15-67, *A* to *D*). This achievement has profoundly encouraged the research field of avian PGC culture. A few research groups confirmed van de Lavoir's result and further optimized the culture condition (Choi *et al.,* 2010; Macdonald *et al.,* 2010). Chicken PGCs from germinal crescent, embryonic gonad were also successfully cultured in the same system. Long-term culture of PGCs of other avian species has not been reported.

PRODUCTION OF GERMLINE CHIMERA

To produce avian germline chimera, the donor PGCs are introduced into the vascular circulation of the recipient embryo at an early stage. The transplanted PGCs can migrate and colonize the primitive embryonic gonad, where these cells resume the germline development and gametogenesis process.

Preparation of Recipient Embryos

The recipient eggs are usually incubated to stage 16, when the endogenous PGCs are passively migrating with the bloodstream. The incubation time required for the avian embryo varies among species (e.g., 56 to 60 hours for chicken embryo, 68 to 70 hours for duck embryos) and is also affected by other factors, such as the freshness of the egg and incubation temperature. There are two common ways to expose the embryo for injection: windowing on the original shell or using surrogate shell system (Liu *et al.,* 2012b). A small piece of shell is removed by a drill or forceps from the sharp end of the egg. Thus the recipient embryo will float up for injection. In the surrogate shell system, the recipient egg is cracked carefully. The entire egg content is transferred to another opened shell, which is prepared from a bigger-size egg, e.g., double yolk chicken egg. The surrogate shell system is applied to culture the recipient embryo; the original shell is difficult to candle because of strong eggshell pigmentation, or because the egg white is dense and viscous.

Transplantation of Donor Primordial Germ Cells

Donor cells can be freshly circulating PGCs, gonadal PGCs, or cultured PGCs. The number of PGCs for injection are a couple of hundred for fresh PGCs and up to two thousand for cultured PGCs.

FIGURE 15-68 Transplantation of primordial germ cells into dorsal aorta, or peripheral blood vessel of chicken embryo.

The PGCs are injected into the blood circulation with a fine glass pipette needle (sharp tip with an angle of 20 degrees and a diameter of 30 microns) by hand or through a micromanipulator under stereomicroscope. Usually, 1 to 3 μL of medium could be injected into each recipient embryo. The optimal site for injection is the dorsal aorta or a peripheral blood vessel (Fig. 15-68). To prevent high pressure after injection, a small amount of blood can be removed in advance.

After injection, the windowed eggs are sealed with Parafilm and incubated with the blunt end up for hatching. The growth and development of injected embryos are monitored through candling. For eggshells with strong pigmentation, the mortality can be checked by monitoring the heartbeat with infrared light equipment or with an embryonic heart monitor (e.g., Buddy Digital Egg Monitor, Avian Biotech International, Florida, USA). In the surrogate shell system, the open shell is closed with cling wrap and then fixed with glue, a rubber band, or a specially designed fixation ring. Injected embryos in the surrogate shell are incubated under normal conditions, but with a reduced rocking angle of 15 degrees. Great care is required during the last 2 to 3 days before hatching, when the embryos start to breathe. For example, holes must be made in the cling wrap to bring fresh air in, and cling wrap must be removed before hatching. The surrogate shell system has advantage that embryonic development can be easily observed through the transparent cling wrap.

IDENTIFICATION OF AVIAN GERMLINE CHIMERA AND PROGENY TEST

The putative germline chimeras are raised to sexual maturity under the same conditions as normal recipient birds. To confirm the gametogenesis of donor PGCs in the gonad of recipient birds, a progeny test is conducted by artificial insemination. A distinguishable marker that helps to trace the origin of resulting chicks is very important for identification of the donor-derived offspring. A species-specific genetic marker, introduced transgenic marker (e.g., green fluorescent protein gene) (Fig. 15-69, *A* and *B*), or plumage phenotype is commonly used (Fig. 15-69, *C* and *D*).

In male chimeric birds, semen is collected and analyzed by detecting the presence of specific genetic markers of donor species. Subsequently, the positive males are considered to be germline chimera and selected for progeny tests. Because there is no applicable method to identify the origin of ova from the female chimera, test crossing with the semen samples of donor species and analyzing the resulting chicks through phenotype or molecular tools is the only way to screen female germline chimera. Thus the donor PGCs eventually complete the gametogenesis process within the testis or ovary of recipient chimeric birds, also termed as a surrogate father or mother. The donor species or breeds could be restored through crossing the male and female germline chimeras.

APPLICATION OF GERMLINE CHIMERA TECHNOLOGY IN CONSERVING GENETICALLY ENDANGERED AVIAN SPECIES

During the last decades, avian primordial germ cell-mediated germline chimera technology has made great progress in PGC collection, characterization, culture, cryopreservation, and germline chimera production. It provides a strategy view of how to apply these technologies to the conservation of avian genetic resources in the future. Currently, most techniques are developed using chicken or other poultry as an experimental model. There are still many challenges involved in expanding these approaches to wild and endangered avian species in practice.

Using either freshly collected PGCs or cultured PGCs, the transmission rate of individual intraspecies chimeric chicken was reported as high as over 90% in some cases. However, considerable variation was observed among individual birds. Irradiation (gamma ray, x-ray) and drug administration (Busulfan) have been reported to delete endogenous PGCs from the recipient embryo before introduced exogenous cells. These methods could significantly improve the average germline transmission rate, but they cause higher mortality in the recipient embryo. To eliminate the competition of endogenous PGCs, a specialized germ-cell-free recipient line could be created using advanced genetic modification technology.

The majority of threatened or endangered wild birds are seasonal breeders with a small clutch size. It is impractical to collect PGCs from the egg and injected them into the embryo of the same species. Thus interspecies germline chimeras were produced through transferring PGCs of wild birds into domestic poultry. Transplanted chicken PGCs could differentiate into functional spermatozoa in the testis of chimeric quail, guinea fowl, and duck (van de Lavoir *et al.*, 2012; Liu *et al.*, 2012a; Fig. 15-70). Pheasant offspring were obtained from the chimeric chicken rooster (Kang *et al.*, 2008). Wernery *et al.* (2010) first reported a houbara bustard (*Chlamydotis undulata undulata*) offspring generated from a chimeric chicken (Fig. 15-71). However, the germline transmission rate of these chimeric roosters is still too low for practical application, and to date no success has been reported from the female chimeras. When transferring PGCs into the recipient embryos between distant phylogenetic species (e.g., crossing biological families or orders), in most cases, donor PGCs could migrate and colonize into the early gonad of the recipient species. The number of donor PGC-derived germ cells was drastically reduced during the first month after hatching. The unknown mechanism of interspecific barrier could exist during the process of xenogenic gametogenesis in the aspects of mitotic proliferation, differentiation, meiosis, and immune rejection.

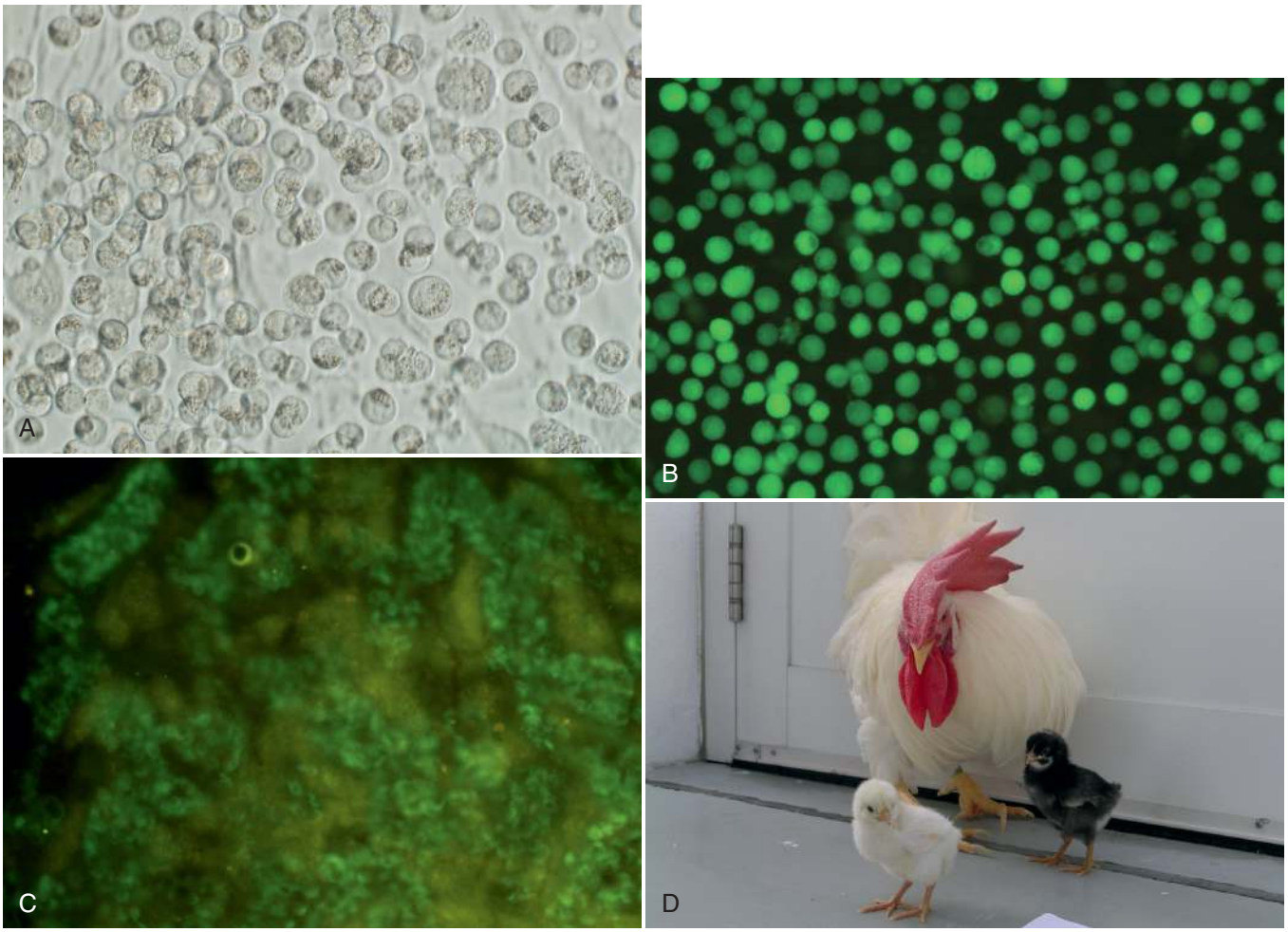


FIGURE 15-69 Germline transmission of chimeric chicken. **(A),** Barred Rock chicken PGCs in culture. **(B),** Chicken PGCs expressing green fluorescent protein in culture. **(C),** Exogenous Barred Rock chicken PGC derived germ cells (green) in the seminiferous tubules of a 4-week-old recipient white Leghorn chicken. **(D),** White Leghorn chimeric rooster producing Barred Rock chicken sperm (the chimeric rooster, exogenous PGCs derived Barred Rock chick, and endogenous germ cells derived white offspring).

FIGURE 15-70 A chicken offspring derived from chimeric male duck (the duck: surrogate father producing white Leghorn chicken semen; the Barred Rock hen: mother of the resulting white chick).

Therefore more basic research needs to be done to determine these unknown factors and to create functionally compatible recipient birds.

The expansion of chicken PGCs in a defined culture system proved the possibility of culturing avian PGCs into a germline-competent cell line. However, the established chicken PGC culture system seems not to work for other species. To develop a culture system for PGCs from a particular species, it is crucial to investigate the intrinsic properties of PGCs and the external factors that affect PGC survival and proliferation. Moreover, it was still unknown whether long-term cultured PGCs could maintain genetic and epigenetic stability. In recent years, induced pluripotent stem cells (IPS) have been created by reprogramming various types of mammalian somatic cells with Yamanaka factors (*Oct3/4, Sox2, Klf4, c-Myc*). These IPS cells could further differentiate into PGCs and give rise to functional gametes (Yang *et al.,* 2012; Imamura *et al.,* 2014). Interestingly, avian IPS-like cells were also obtained through reprogramming chicken and quail fibroblasts using human pluripotency related genes (Rosselló *et al.,* 2013; Lu *et al.,* 2012). If avian IPS could further differentiate into PGCs, these induced PGCs could reintroduce their genetic information into the gene pool through germline chimera technology. It could make current

FIGURE 15-71 A houbara bustard chick obtained from chimeric rooster. **(A),** Chimeric rooster that produces houbara bustard sperm, its houbara bustard offspring, and the mother houbara bustard. **(B),** Obtained houbara bustard chick.

technology independent of precious eggs. Therefore cryopreservation of somatic cells could be an option in the future for conservation of genetic resources from highly endangered avian species.

REFERENCES

Armengol C, Carretero A, Nacher V, et al: Carbohydrate characterization of quail primordial germ cells during migration and gonadal differentiation, *J Anat* 210:98–111, 2007.

Bird Life International: *One in eight of all bird species is threatened with global extinction*. www.birdlife.org/datazone/sowb/casestudy/106, 2014.

Blanco JM, Long JA, Gee G, et al: Comparative cryopreservation of avian spermatozoa: effects of freezing and thawing rates on turkey and sand hill crane sperm cryosurvival, *Anim Reprod Sci* 131:1–8, 2012.

Choi JW, Kim S, Kim TM, et al: Basic fibroblast growth factor activates MEK/ERK cell signaling pathway and stimulates the proliferation of chicken primordial germ cells, *PLoS ONE* 5:e12968, 2010.

D'Costa S, Petitte JN: Characterization of stage-specific embryonic antigen-1 (SSEA-1) expression during early development of the turkey embryo, *Int J Dev Biol* 43:349–356, 1999.

Extavour CG, Akam M: Mechanisms of germ cell specification across the metazoans: epigenesis and preformation, *Development* 130:5869–5884, 2003.

Eyal-Giladi H, Kochav S: From cleavage to primitive streak formation: a complementary normal table and a new look at the first stages of the development of the chick. I. General morphology, *Dev Biol* 49:321–327, 1976.

Fujimoto T, Ukeshima A, Kiyofuji R: The origin, migration and morphology of the primordial germ cells in the chick embryo, *Anat Rec* 185:139–145, 1976.

Ginsburg M: Primordial germ cell formation in birds, *Ciba Found Symp, Review* 182:52–61, 1994.

Halfter W, Schurer B, Hasselhorn HM, et al: An ovomucin-like protein on the surface of migrating primordial germ cells of the chick and rat, *Development* 122:915–923, 1996.

Hamburger V, Hamilton HL: A series of normal stages in the development of the chick embryo, *J Morphol* 88(1):49–92, 1951.

Hartley PS, Dawson B, Lindsay C, et al: Cryopreservation of houbara semen: a pilot study, *Zoo Biol* 18(2):147–152, 1999.

Imamura M, Hikabe O, Lin ZY, et al: Generation of germ cells in vitro in the era of induced pluripotent stem cells, *Mol Reprod Dev* 81:2–19, 2014.

Jung JG, Kim DK, Park TS, et al: Development of novel markers for the characterization of chicken primordial germ cells, *Stem Cell* 23:689–698, 2005.

Kang SJ, Choi JW, Kim SY, et al: Reproduction of wild birds via interspecies germ cell transplantation, *Biol Reprod* 79:931–937, 2008.

Karagenç L, Cinnamon Y, Ginsburg M, et al: Origin of primordial germ cells in the prestreak chick embryo, *Dev Genet* 19:290–301, 1996.

Karagenç L, Petitte JN: Soluble factors and the emergence of chick primordial germ cells in vitro, *Poultry Sci* 79:80–85, 2000.

Kim JN, Lee YM, Park TS, et al: Detection and characterization of primordial germ cells in pheasant *(Phasianus colchicus)* embryos, *Theriogenology* 63:1038–1049, 2005.

Li BC, Tian ZQ, Sun M, et al: Directional differentiation of chicken primordial germ cells into adipocytes, neuron-like cells, and osteoblasts, *Mol Reprod Dev* 77:795–801, 2010.

Liu C, Khazanehdari KA, Baskar V, et al: Production of chicken progeny (*Gallus gallus domesticus)* from interspecies germline chimeric duck (*Anas domesticus*) by primordial germ cell transfer, *Biol Reprod* 86:1–8, 2012a.

Liu C, Zu J, Baskar V, et al: Culture of chicken embryo in inter specific surrogate egg white, *Poultry Sci* 91:2866–2871, 2012b.

Liu J, Song Y, Cheng KM, et al: Production of donor-derived offspring from cryopreserved ovarian tissue in Japanese quail *(Coturnix japonica)*, *Biol Reprod* 83:15–19, 2010.

Lu Y, West FD, Jordan BJ, et al: Avian-induced pluripotent stem cells derived using human reprogramming factors, *Stem Cells Dev* 21:394–403, 2012.

Macdonald J, Glover JD, Taylor L, et al: Characterization and germ line transmission of cultured avian primordial germ cells, *PLoS ONE* 5:e15518, 2010.

Madeddu M, Berlinguer F, Ledda M, et al: Ejaculate collection efficiency and post-thaw semen quality in wild-caught Griffon vultures from the Sardinian population, *Reprod Biol Endocrin* 7:18, 2009.

Matsui Y, Zsebo K, Hogan BL: Derivation of pluripotential embryonic stem cells from murine primordial germ cells in culture, *Cell* 70:841–847, 1992.

Merchant-Larios H, Mendlovic F, Alvarez-Buylla A: Characterization of alkaline phosphatase from primordial germ cells and ontogenesis of this enzyme in the mouse, *Differentiation* 29:145–151, 1985.

Moore DT, Purdy PH, Blackburn HD: A method for cryopreserving chicken primordial germ cells, *Poultry Sci* 85:1784–1790, 2006.

Mozdziak PE, Angerman-Stewart J, Rushton B, et al: Isolation of chicken primordial germ cells using fluorescence-activated cell sorting, *Poultry Sci* 84:594–600, 2005.

Naito M, Tajima A, Tagami T, et al: Preservation of chick primordial germ cells in liquid nitrogen and subsequent production of viable offspring, *J Reprod Fertil* 102:321–325, 1994b.

Naito M, Tajima A, Yasuda Y, et al: Production of germline chimeric chickens with high transmission rate of donor-derived gametes produced by transfer of primordial germ cells, *Mol Reprod Dev* 39:153–161, 1994a.

Nieuwkoop PD, Sutasurya LA: *Primordial germ cells in the chordates: embryogenesis and phylogenesis*, Cambridge, UK, 1979, Cambridge University Press.

Ono T, Machida Y: Immunomagnetic purification of viable primordial germ cells of Japanese quail *(Coturnix japonica)*, *Comp Biochem Physiol* 122:255–259, 1999.

Polge C: Functional survival of fowl spermatozoa after freezing at −79 degrees C, *Nature* 167:949–950, 1951.

Rengaraj D, Zheng YH, Kang KS, et al: Conserved expression pattern of chicken DAZL in primordial germ cells and germ-line cells, *Theriogenology* 74:765–776, 2010.

Rosselló RA, Chen CC, Dai R, et al: Mammalian genes induce partially reprogrammed pluripotent stem cells on non-mammalian vertebrate and invertebrate species, *eLife* 2:e00036, 2013.

Song Y, Silversides FG: Production of offspring from cryopreserved chicken testicular tissue, *Poultry Sci* 86:1390–1396, 2007.

Sontakke SD, Umapathy G, Sivaram V, et al: Semen characteristics, cryopreservation, and successful artificial insemination in the blue rock pigeon (*Columba livia*), *Theriogenology* 62:139–153, 2004.

Swift CH: Origin and early history of the primordial germ cells in the chick, *Am J Anat* 15:483–516, 1914.

Tsunekawa N, Naito M, Sakai Y, et al: Isolation of chicken vasa homolog gene and tracing the origin of primordial germ cells, *Development* 127:2741–2750, 2000.

van de Lavoir MC, Collarini EJ, Leighton PA, et al: Inter specific germ line transmission of cultured primordial germ cells, *PLoS ONE* 7:e35664, 2012.

van de Lavoir MC, Diamond JH, Leighton PA, et al: Germ line transmission of genetically modified primordial germ cells, *Nature* 441:766–779, 2006.

Wade AJ, French NA, Ireland GW: The potential for archiving and reconstituting valuable strains of turkey (*Meleagris gallopavo*) using primordial germ cells, *Poultry Sci* 93:799–809, 2014.

Wernery U, Liu C, Baskar V, et al: Primordial germ cell-mediated chimera technology produces viable pure-line houbara bustard offspring: potential for repopulating an endangered species, *PLoS ONE* 5:e15824, 2010.

Yamamoto Y, Usui F, Nakamura Y, et al: A novel method to isolate primordial germ cells and its use for the generation of germline chimeras in chicken, *Biol Reprod* 77:115–119, 2007.

Yang S, Bo J, Hu H, et al: Derivation of male germ cells from induced pluripotent stem cells in vitro and in reconstituted seminiferous tubules, *Cell Proliferat* 45:91–100, 2012.

Yasuda Y, Tajima A, Fujimoto T, et al: A method to obtain avian germ-line chimaeras using isolated primordial germ cells, *J Reprod Fertil* 96:521–528, 1992.

Zhao DF, Kuwana T: Purification of avian circulating primordial germ cells by Nycodenz density gradient centrifugation, *Brit Poultry Sci* 44:30–35, 2003.

16

Postmortem Examination

Jörg Kinne

CARCASS EXAMINATION

A comprehensive necropsy is time-consuming, expensive, and may not always be necessary to deal with every detail as outlined below in the following sections (Keymer, 2008). However, if there is any chance of legal proceedings or the bird is a rare species, then it is essential to perform as detailed an examination as possible and keep full records. After the necropsy is finished, all remains of the carcass should be suitably preserved in case further examination of tissue proves necessary.

Carcasses destined for laboratory examination should, without delay, be thoroughly chilled in a refrigerator before packing and dispatch. Soaking bird carcasses in cold detergent at this stage is not recommended because it can damage the skin and hamper examination. Ectoparasites may also be lost and the skin will be unsuitable for taxonomic studies or taxidermy.

Carcasses should never be deep frozen unless the examinations are to be entirely of a toxicologic nature or there is likely to be a greater than one week delay before necropsy. When the carcasses thaw, artifacts are produced, thus hindering histopathological examination. The gross appearance of the organs is also altered by the exudation of fluid.

Packing material should be lightweight, water-proof, and have good insulating properties. A full history should be provided and enclosed in the parcel in a sealed plastic bag separate from the carcass, keeping it free from contamination by body fluids. The history should state the name and address of the sender/owner and date of death and/or method of euthanasia. The name of the species (including the scientific name) should also be given, as well as the age, sex, and reference number. It is also important to describe any clinical signs noted before death and details of treatment, if any.

It is essential that local postage and customs regulations are strictly adhered to and that there is no risk of any leakage from the parcel. When endangered species are involved, special Convention on International Trade in Endangered Species of Wild Fauna and Flora legislation may be applicable. Fresh tissues must not be dispatched to certain countries without a special license. Immediately beneath the first layer of packing there should be a clear message indicating the nature of the contents. If there is any risk that the carcass may contain a zoonotic infection, such as psittacosis, this should be clearly stated. This is very important, because the person who opens the parcel may not be a veterinarian or somebody who appreciates the potential risk of contracting a disease.

Carcasses not destined for another laboratory should be examined immediately and kept as cool as possible, preferably by storing them temporarily in a refrigerator at 4° C (39.2° F). Most carcasses will keep satisfactorily at this temperature for at least 2 days. If carcasses are too large to go into a standard refrigerator, they can be placed in a deep freezer for a short time, taking care not to actually freeze them. A moderately autolyzed carcass is usually preferable to one that has been deep frozen (Keymer, 2008).

Preparations for Necropsy

1. Read the history if there is one. Ascertain whether the bird died naturally or was euthanized. If euthanasia was performed, it is frequently not recorded in the history. When injections of pentobarbital sodium have been given, sometimes whitish deposits on the surfaces of some internal organs are seen. These artifacts can easily be confused with genuine lesions of disease, especially visceral gout.
2. Assemble equipment, including sterile instruments of an appropriate size for the carcass being examined. For very small specimens, ophthalmic instruments may be most suitable. Assign a necropsy reference number to the specimen and record the date and time the necropsy was performed in the laboratory daybook.
3. While performing stage 2, think about the history, clinical signs, management, and environment. If there is any reason to suspect a zoonotic infection, prepare to examine the carcass in a safety cabinet, if this is available. If the carcass is too large or no cabinet is available, then in addition to wearing standard protective clothing, including disposable surgical gloves, it is essential to wear a facemask and eye shields.
4. If the carcass is deep frozen, allow it to thaw out for a few hours before attempting examination. At room temperature this may take 48 hours for a large bird.
5. Before commencing the necropsy, check the Supplementary Diagnostic Procedures section at the end of this section to be fully prepared.
6. When performing a necropsy it is extremely important to *record all lesions as they are found* on the necropsy forms (Tables 16-1 and 16-2). Findings should be recorded by dictation to an assistant or by using a tape recorder. Do not wait until the necropsy has been completed before recording the findings. By this time the appearance of some of the lesions will have been forgotten (especially if interruptions have occurred) or lesions will have been destroyed either by accident or during subsequent dissection. Do not forget to take pictures as part of the documentation; they might be helpful for legal proceedings or publications. If for some reason a body system (e.g., the central nervous system) or some organs have not been examined (damage or decomposition), be sure to record this fact (see NE [not examined] on the Necropsy Sheet; Table 16-1).

External Examination

Skin and Appendages

Carcasses placed in a plastic bag should be first weighted and then ectoparasites collected inside the bag. Record whether the carcass

TABLE 16-1 Necropsy Sheet

Necropsy sheet _____ **Clinical reference number** _____

Pathology reference number _____

Name of species: _____ Scientific name: _____

Age: _____ Sex: _____

Identity number: _____ Pet name: _____

Owner's name: _____

Address: _____

Contact No: _____ E-mail: _____

Veterinarian's name: _____

Address: _____

Contact No: _____ E-mail: _____

Date of death: _____ Date of necropsy: _____

Postmortem Findings*

Body weight _____ (g) **Physical condition**: Normal ☐ Fat ☐ Thin ☐ Emaciated ☐ Other ☐

State of carcass: Fresh ☐ Refrigerated ☐ Deep frozen ☐ Decomposed ☐ Incomplete ☐

Systems

1. Skin/appendages	NLD ☐ NE ☐	8. Liver	NLD ☐ NE ☐
2. Skeletal	NLD ☐ NE ☐	9. Digestive	NLD ☐ NE ☐
3. Sensory	NLD ☐ NE ☐	10. Lymphoreticular	NLD ☐ NE ☐
4. Muscular	NLD ☐ NE ☐	11. Urinary	NLD ☐ NE ☐
5. Respiratory	NLD ☐ NE ☐	12. Reproductive	NLD ☐ NE ☐
6. Cardiovascular	NLD ☐ NE ☐	13. Nervous	NLD ☐ NE ☐
7. Endocrine	NLD ☐ NE ☐	14. Others:	

Postmortem findings/lesions

_____

_____

_____

_____

_____

_____

_____

_____

_____

_____

_____

_____

_____ (continue over)

Diagnosis: provisional ☐ final ☐

_____

_____

_____

_____

*Tick boxes and describe in the space provided. Under "Systems," if no lesions of skin/appendages, etc., are detected, tick the "NLD" box. If a system is not examined tick "NE." When lesions are found use the number given for the system and describe in the space beneath "Postmortem findings/lesions," continuing on back of sheet if necessary.

TABLE 16-2 Necropsy Sheet—cont'd

Necropsy sheet

Clinical reference number _____

Pathology reference number _____

Specimens Taken			Identification/Results
☐ Parasitology:	Helminths	☐	
	Protozoa	☐	
☐ Microbiology:	Bacteria	☐	
	Fungi	☐	
	Viral/other	☐	
☐ Hematology:	Blood smear ☐ Blood sample (EDTA) ☐ Blood sample (Heparin) ☐		
☐ Tissue impression smears:	Spleen	☐	
	Bone marrow	☐	
	Liver	☐	

☐ Histology

Skin	☐	Tongue	☐	Bursa of Fabricius	☐
Bone	☐	Esophagus	☐	Thymus	☐
Eye	☐	Crop	☐	Kidney	☐
Skeletal muscle	☐	Stomach	☐	Testis/ovary	☐
Lung/air sac	☐	Proventriculus	☐	Oviduct	☐
Heart	☐	Gizzard	☐	Cerebrum	☐
Anterior/posterior aorta	☐	Duodenum	☐	Cerebellum	☐
Thyroid	☐	Pancreas	☐	Spinal cord	☐
Parathyroid	☐	Small intestine	☐	Peripheral nerve	☐
Adrenal	☐	Cecum	☐	Other: _____	
Pituitary	☐	Large intestine	☐	Other: _____	
Liver	☐	Spleen	☐	Other: _____	

☐ Biochemistry _____

☐ Toxicology _____

☐ Electron microscopy _____

☐ Radiology _____

☐ Photography _____

*Tick boxes as applicable and complete details in spaces provided

_____

Veterinary surgeon

is wet or dry, fresh, chilled, or frozen and describe any skin lesions. Radiograph the entire carcass to screen for skeletal lesions such as minor fractures, which are not immediately obvious, and nutritional bone disease and foreign bodies such as lead shot in the gizzard or gunshot wounds (Fig. 16-1).

Rinse some water over the carcass to ease plucking the feathers and to score the body condition (Fig. 16-2). If the specimen is a rare species, measure wing, beak, and tail length (Bibby, 1985), or consult an ornithologist for instructions. Closely examine the carcass. If it is very small use a magnifying lamp or magnifying spectacles or a dissecting microscope. At this and every subsequent stage of the examination be prepared to take photographs. Look for any evidence of trauma, search for ectoparasites and collect samples, examine the cloaca, and look for lesions on feet and skin by parting feathers. Examine the plumage: normal molting must be differentiated from molting caused by pathologic causes.

Examine the uropygial or preen gland by parting the feathers over the last vertebra situated at the base of the tail. It is absent in Struthioniformes, Rheiformes, Casuariiformes, and bustards (Otididae). The gland is either absent or very small in some members of the Caprimulgiformes, Columbiformes, Psittaciformes, and Piciformes. It is especially well developed in most aquatic species, such as Sphenisciformes, Podicipediformes, and gulls (Laridae).

Search for brood or incubation patches. These develop only during egg laying and are present throughout incubation. In some species they occur in both sexes, in others they are confined to females, in others to males, and in some species they are absent. They are situated on the skin of the breast and appear as thickened, highly vascular areas of alopecia involving the dermis. There may be a single median patch or lateral patches. They must be differentiated from pathologic lesions.

It is also necessary to closely examine the oral cavity and the beak for lesions such as deformities. A horny layer, known as the

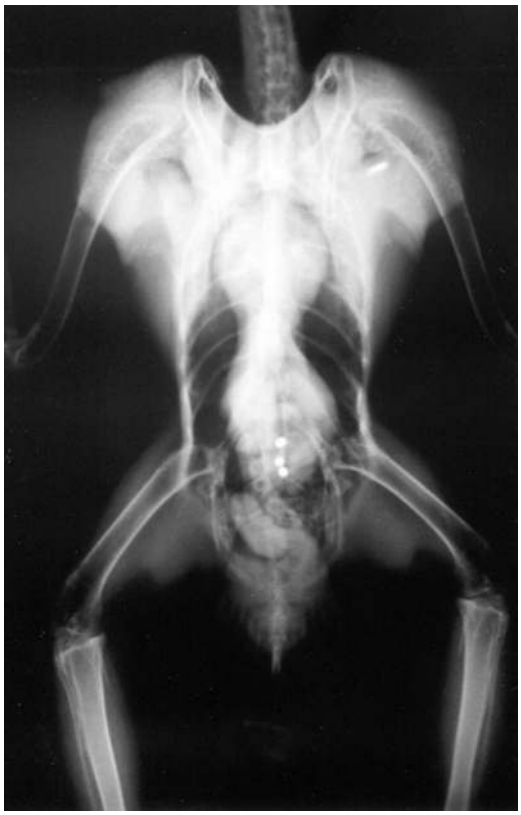


FIGURE 16-1 Radiograph of the entire carcass to detect skeletal lesions such as minor fractures, nutritional bone disease, and foreign bodies such as lead shot. Note three lead shots in the gizzard and the microchip near left wing.

FIGURE 16-2 Score the body condition: in this case poor condition. Note the prominent sternum.

rhamphotheca, covers the beak. This is a keratinized thickening of the stratified corneum of the epidermis. Beaks vary considerably in shape depending on feeding habits and diet. It is therefore necessary to be familiar with the normal appearance of the species being examined (see Arnall and Keymer, 1975; King and McLelland, 1984 for illustrations). In gannets (*Morus* spp.), for example, the external nares are

FIGURE 16-3 Ectoparasites (like biting lice) of large avian carcasses (like a rhea) can be detected by close examination.

closed and not visible at the normal site for most species (i.e., at the base of the upper mandible). These birds breathe through the commissures of the mouth.

Look for artificial methods of identity like subcutaneous identification microchips (see Fig. 16-1), leg rings (bands), tattoos, wing tags, or rubber stamping of wing feathers. Record any identification numbers on the necropsy record sheet.

If any skin lesions suggestive of dermatophytosis (ringworm) are found, then examine (the whole bird if possible) in a darkened room under a Wood's lamp, parting the feathers at frequent intervals and looking for the presence of fluorescing infected skin or feathers. However, many avian dermatophytes may not fluoresce. Collect specimens for microscopy and culture. If any ectoparasites are found (Fig. 16-3), collect all of them or alternatively a large representative sample. If they cannot be analyzed immediately transfer to a bottle of 70% ethyl alcohol that can be firmly sealed. Label the specimen immediately using a waterproof pen or pencil with the relevant reference number, both on a piece of card placed in the bottle and on the outside of the container and not on the lid, because this can be accidentally transferred to another container.

It may be necessary to remove the skin for forensic issues or taxidermy. However, this is best performed by an expert taxidermist. Examine the subcutaneous tissues carefully for any signs of trauma or other lesions.

Skeletal System

Screen for skeletal lesions and foreign bodies using standard radiographic techniques (see Fig. 16-1). If foreign bodies are located, they should be collected as they become available, labeled, and put in a safe place. If there is any possibility of legal proceedings, this should be done in the presence of a witness. Open the major limb joints and examine the articular surfaces. Note the appearance of the synovial fluid and take swabs for bacteriologic examination if considered necessary.

Sensory System

In birds the openings of the external auditory meatuses are not immediately obvious, because they are covered by feathers (the ear coverts). Therefore they need to be raised before examination. A meatus is

FIGURE 16-4 The eyes should be examined by deflecting the eyelids for evidence of macroscopic lesions and by indirect ophthalmoscopy (if mydriasis is present and the carcass is very fresh) for microscopic internal lesions.

FIGURE 16-5 The skin is carefully removed by blunt dissection using the hands, after the initial incision has been made using a scalpel. Removal of skin and subcutaneous tissues exposes the ventral surface of the pectoral muscles. (Courtesy Dr. I.F. Keymer.)

situated caudal to and slightly below each eye. In owls, unlike other species, the positions of the auditory meatuses are not quite symmetric and the openings are relatively large.

Examine the external nares. In some marine species these are absent. Examine the eyes initially by deflecting the eyelids (Fig. 16-4) for evidence of macroscopic lesions or parasites, ideally followed by indirect ophthalmoscopy (if mydriasis is present and the carcass is very fresh) for microscopic internal lesions. Remove the eyes and examine the orbits. If required for histopathological examination, fix the eyes without delay.

Muscular System

Initial examination of the muscles is performed soon after the carcass has been partly (Figs. 16-5 and 16-6) or fully skinned. At this stage muscles can be incised and examined for lesions, including parasites (Fig. 16-7). The final examination can be completed at the end of the necropsy.

Internal Examination

At this stage the carcass can be positioned for examination of the internal organs. Small carcasses should be placed on a board or postmortem room table and fixed securely in position with the ventral surface uppermost. They may either be nailed on a board through the feet and wings or secured with strings or ropes to cleats on the postmortem room table as appropriate. This is facilitated by dislocating the hip joints using both hands to grip the upper part of each leg and deflecting the legs dorsally to the body (i.e., downward toward the surface of the table). The carcass will then lie flat. For small birds the wings can be fixed in position by inserting pins between the ulna and radius at the distal end of these bones or through the carpal joint, and in larger birds this is done by using nails. If the skin is not required for taxonomic studies or taxidermy, dip the carcass in disinfectant solution and pluck the feathers from the neck, breast, and abdomen. This lessens the dispersal of feathers and feather "dust" (powder down) into the atmosphere and helps prevent contamination of the internal organs. Large carcasses such as ostriches may require no method of stabilization and stay in position on most surfaces.

FIGURE 16-6 Deflect the skin by blunt dissection to expose the subcutaneous tissues of the neck, pectoral muscles, rib cage, and abdominal and leg muscles to expose the body before full dissection. Note the well-defined crop on the right side of the neck. (Courtesy Dr. J. Samour.)

FIGURE 16-7 At this stage subcutaneous tissue can be examined for lesions including parasites (larvae of *Paraspiralatus sakeri* in subcutaneous tissue of a Houbara bustard).

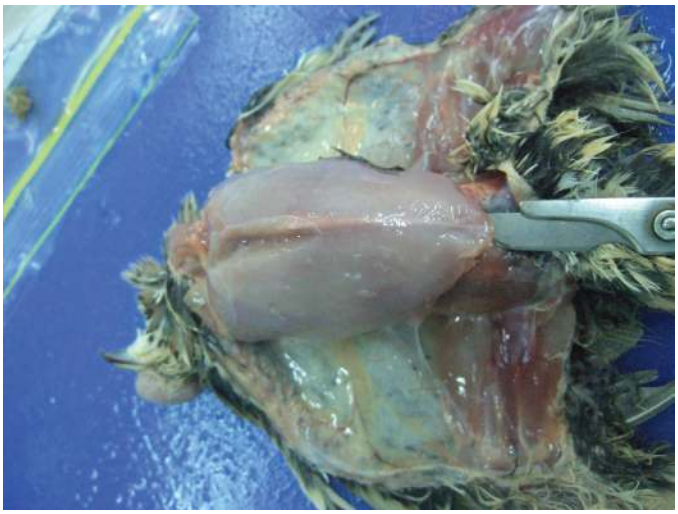


FIGURE 16-8 Make a small incision in the abdominal wall near the sternum using fine scissors to expose the abdominal viscera. Enlarge it toward to the cloaca and open the abdomen.

Respiratory and Cardiovascular Systems

If the skull is not needed, expose and examine the nasal passages, sinuses (including the infraorbital sinus), and internal nares using bone forceps. Do not expose the brain (see Nervous Section, below).

Open the carcass in the midline by starting the incision at the base of the throat extending it caudally to the region of the cloaca. Deflect the skin by blunt dissection to expose the subcutaneous tissues of the neck, pectoral muscles, rib cage, and abdominal and leg muscles (see Figs. 16-5 and 16-6). Make a small incision in the abdominal wall near the sternum using fine scissors to expose the abdominal viscera (Fig. 16-8). Enlarge it toward the cloaca and open the abdomen. This exposes the internal organs intact, including the liver, which should be carefully removed, partly by blunt dissection, for late examination. Unless the intact skeleton is required, expose the thoracic cavity by cutting through the rib cage and the coracoid and clavicle bones on both sides. Then deflect to one side the sternum with the pectoral muscles attached (Fig. 16-9).

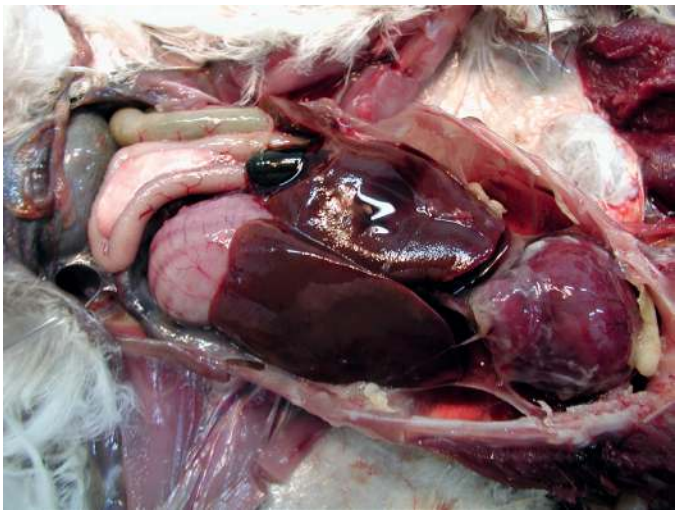


FIGURE 16-9 The sternum with the attached pectoral muscles has been removed and the abdominal muscles have been dissected to allow viewing of all major organs. Parts of the lung, the heart, the liver overlapping part of the gizzard, the duodenal loop surrounding the pancreas, and a small part of the lower intestinal tract are visible. The cranial air sac system is seen here between liver and heart with some fibrin at the edge.

Birds have well-developed air sacs, which are extensions of the lungs. They are transparent and sometimes difficult to detect when normal. The cranial air sacs should be examined before removing the internal organs (Fig. 16-9), otherwise they are difficult to identify again. Figs. 16-10 and 16-11 illustrate exposure of the spleen and the ventral surface of the neck by removal of the sternum. A true diaphragm is absent in birds. However, a pulmonary fold is present, situated ventral to the lungs, separating the pulmonary area of the coelom from the peritoneal area.

The next procedure is to remove the alimentary tract, from esophagus to cloaca. If the intact skeleton is required, this involves withdrawing the esophagus and trachea caudally through the thoracic cavity. Before this can be done, the subcutaneous tissues of the neck must be excised, and the trachea and esophagus exposed and separated from their attachments (Fig. 16-12), taking care to identify and collect the thymus and thyroids (Fig. 16-13).

In most species the trachea terminates in a swollen structure known as the syrinx, formed by the lowest rings of the trachea fused into a cylindrical tympanum. However, there are other variations and sometimes the organ can be a very large and complex structure. In male ducks (smaller species of Anatidae), for example, a cartilaginous enlargement (bulla) surrounds the syrinx. In most species the trachea is a simple straight tube; however, in some it is an elongated coiled structure that in some species emerges from the sternum and lies between the skin and the pectoral muscles, for example, in curassows (large species of Cracidae and Galliformes; King and McLelland, 1984).

Next, with fine scissors cut the great vessels (except the descending aorta), which overlie the ventral aspect of the esophagus (Fig. 16-14) a short distance from their cardiac origins. The esophagus is then severed immediately posterior to the pharynx so that the alimentary tract from pharynx to cloaca can be removed in one piece. This is examined later (see Digestive System, below). The heart and great vessels can now be removed, with the descending aorta carefully dissected at least as far as it becomes the terminal aorta and divides into the iliac arteries. The descending aorta and great vessels, which leave and enter the heart, can then be opened using fine scissors and the

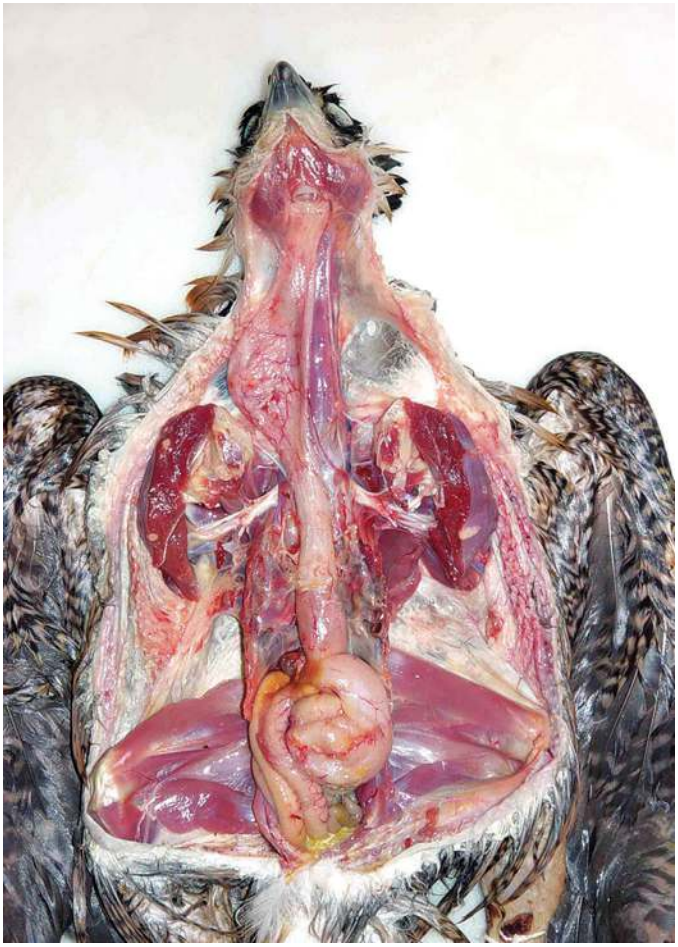


FIGURE 16-10 The heart and the liver have been removed to expose the organs underneath. The oval-shaped spleen is situated next to the proventriculus and gizzard. Note the ventriculus and intestinal tract stained with bile because of the proximity of the gallbladder. (Courtesy Dr. J. Samour.)

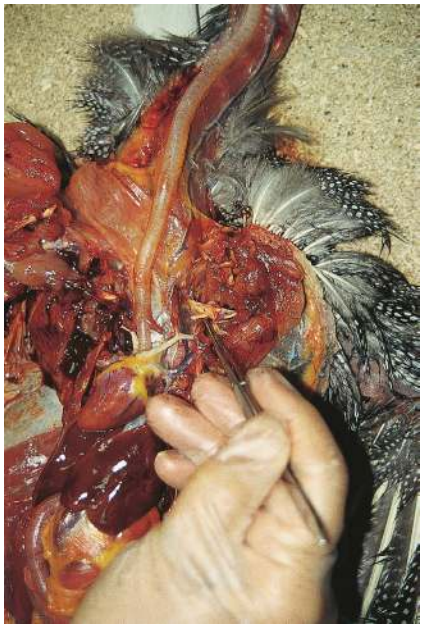


FIGURE 16-11 Ventral surface of neck exposed by removal of sternum, showing left brachial plexus (above tip of forceps), trachea, empty crop on the right side of the bird, heart, and liver. (Courtesy Dr. I.F. Keymer.)

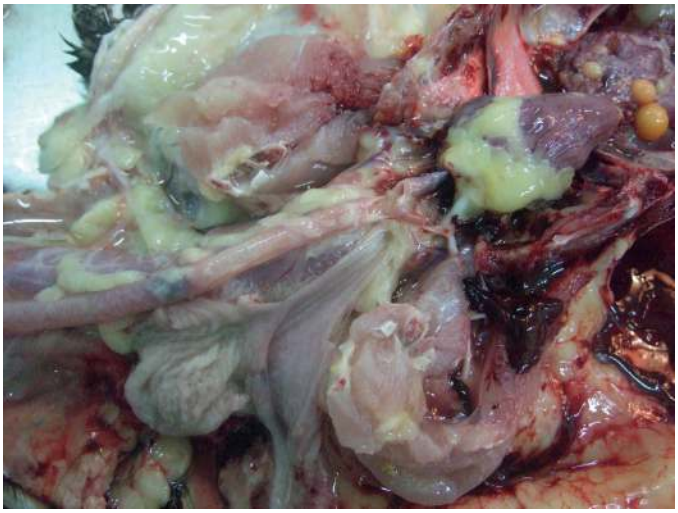


FIGURE 16-12 The trachea has been exposed; the crop and the caudal esophagus are opened.

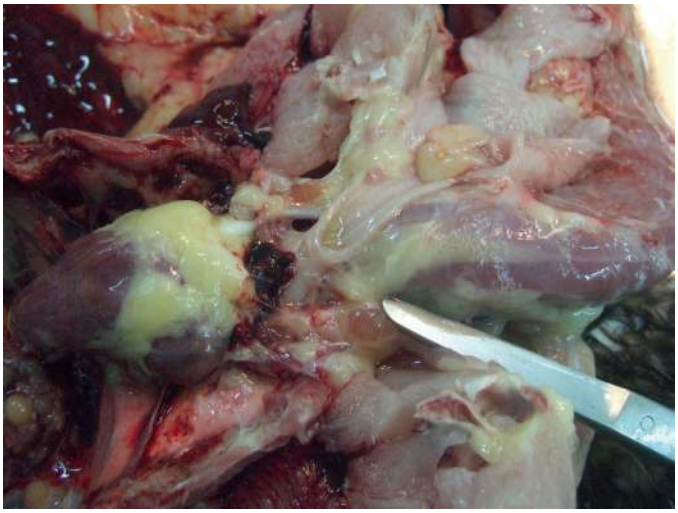


FIGURE 16-13 The thyroids are paired, ovoid structures at the base of the neck on each side of the trachea in close association with the jugular veins.

intima examined for lesions such as arteriosclerosis (Fig. 16-15). Examine the heart, both internally and externally, and the cardiac blood vessels. If the carcass is fresh, make blood smears from the heart blood and peripheral blood vessels to examine blood cells and look for hematozoa (Wernery *at al.*, 2004).

In birds, the lungs are closely attached to the wall of the thoracic cavity (Fig. 16-16) by fibrous strands, greatly reducing the extent of the visceral and parietal pleura. Examine the lungs (remove by blunt dissection) and the caudal thoracic air sacs (see Fig. 16-16). In addition to lesions of septicemia, these organs are frequently the site of mycosis and sometimes mycoplasmosis.

Endocrine System (Lymphoreticular System)

The thyroids are paired, dark-red ovoid structures at the base of the neck on each side of the trachea in close association with the jugular veins. In Figure 16-16, the right thyroid is visible immediately to the right of the trachea and anterior to the left carotid artery. The parathyroids are often difficult to find but can usually be detected

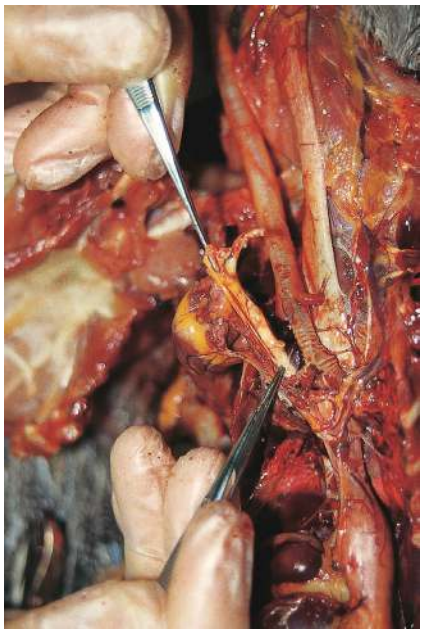


FIGURE 16-14 The great vessels (except the descending aorta) have been cut. The two bronchi are then severed anterior to each lung to expose the thoracic esophagus. (Courtesy Dr. I.F. Keymer.)

FIGURE 16-15 Opened heart and aorta showing intima of proximal descending aorta and the right brachiocephalic artery. The kidney and testes are also visible. The right testis is cream colored and normal in appearance. The left testis is unusually hypertrophied in comparison. (Courtesy Dr. I.F. Keymer.)

associated with the thyroid by using a magnifying glass or on histologic examination. They are small yellowish structures just caudal to the thyroids. Enlargement may occur with metabolic bone disease.

The thymus glands are bilateral, pale pinkish multilobular structures situated on each side of the neck close to the jugular vein. It may be necessary to take suspected glandular tissue for histopathological examination to make a definite identification because in small species these glands can be difficult to recognize. They involute as the bird reaches maturity but in some species may reenlarge after the breeding season. Under some circumstances, the thyroids and thymus may need to be weighed and/or measured.

In most birds, the adrenal glands are yellowish and paired, situated at the anterior end of each kidney (see Fig. 16-16). The pituitary gland

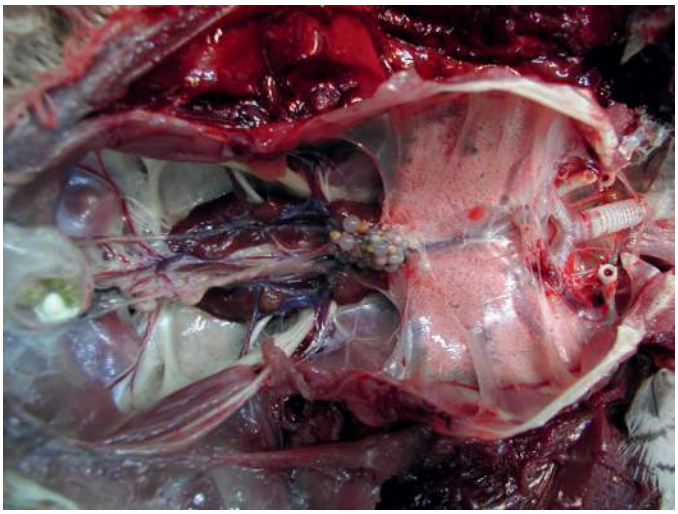


FIGURE 16-16 Ventral aspect of body with viscera removed except lungs, kidneys, and gonads. The caudal thoracic air sacs are visible between lungs and kidneys.

is situated on the ventral surface of the brain so it is necessary to remove the brain to examine it.

Liver

Examine the surface of the liver and weigh and measure the organ. If psittacosis is suspected, make "touch" impression smears of the intact surface for *Chlamydia psittaci* elementary bodies (Wernery *et al.*, 2004). Examine by slicing and make impression smears of the cut surface to examine for *Plasmodium* parasites or for *Chlamydia*, if indicated. Air dry touch impression smears and place others in a suitable fixative.

A gallbladder is present in many species, but is absent in most Columbidae, many psittacines, and the ostrich. Examine the gallbladder and check for patency of the bile duct and contents of the bladder. If gallstones are found, these should be collected for analysis and stored by refrigeration or deep frozen.

Digestive System

The buccal cavity and pharynx can be exposed by using scissors to cut through the commissures of the mouth on one side and bone forceps to cut the hyoid apparatus on both sides, enabling the head to be deflected. Do not overlook examination of the mouth and buccal cavity because these are common sites for lesions caused by a variety of agents (Fig. 16-17). Tongues vary in shape, size, and mobility in different species, so it is important to be aware of their normal appearance and that of the entire alimentary tract before proceeding with the dissection (King and McLelland, 1984; McLelland, 1991).

The esophagus leads directly into the glandular stomach, known as the proventriculus (Figs. 16-18 and 16-19). However, in some species a diverticulum of the esophagus occurs to form a crop. This is large in gallinaceous species, psittacines, and many seed-eating passerines. In pigeons (Columbiformes) the crop is divided into two large, lateral glandular sacs that secrete crop "milk" for feeding the young. The crop wall is highly vascular in pigeons, especially when producing this secretion. In psittacines it stretches transversely across the base of the neck. The crop is also fairly well developed in some birds of prey (Figs. 16-6 and 16-11). However, in some of these species and some passerines and in Anseriformes, it is less well developed and is seen as a spindle-shaped swelling of the esophagus near the thoracic inlet. In

FIGURE 16-17 Mouth and buccal cavity are common sites for lesions, like trichomoniasis.

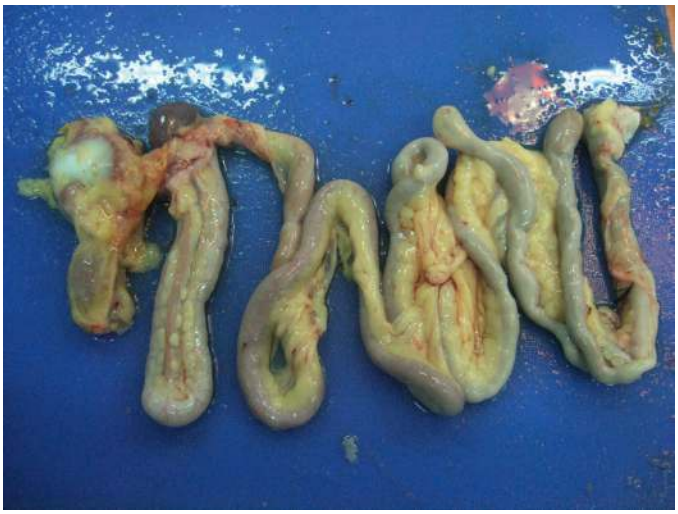


FIGURE 16-18 Gastrointestinal tract with proventriculus, muscular gizzard, duodenum forming loop round the pancreas, small intestine, two well-developed ceca, and large intestine (colorectum). Note the spleen between stomach and duodenum.

predominantly insectivorous or frugivorous passerines, the crop is also fusiform in shape. It is absent in some birds, such as penguins (Sphenisciformes), gulls (Laridae), and Caprimulgiformes. The males of some breeds of domestic pigeon (*Columba livia*), the ostrich, sage grouse (*Centrocercus urophasianus*), and great bustard (*Otis tarda*) have inflatable esophageal diverticula that act as resonating chambers when displaying.

The proventriculus precedes the gizzard or ventriculus. In nonseed-eating birds the gizzard is thin walled with little muscular tissue, and the junction with the proventriculus is difficult to detect externally; in such cases the two portions are frequently referred to as the "stomach." The degree of glandular and muscular tissue is related to the nature of the diet. The orifices of the gastric glands are easily visible to the naked eye where they open into the lumen. A well-developed sphincter separates the organ from the entrance to the gizzard. In predominantly seed-eating birds the gizzard has a thick muscular wall and is lined by

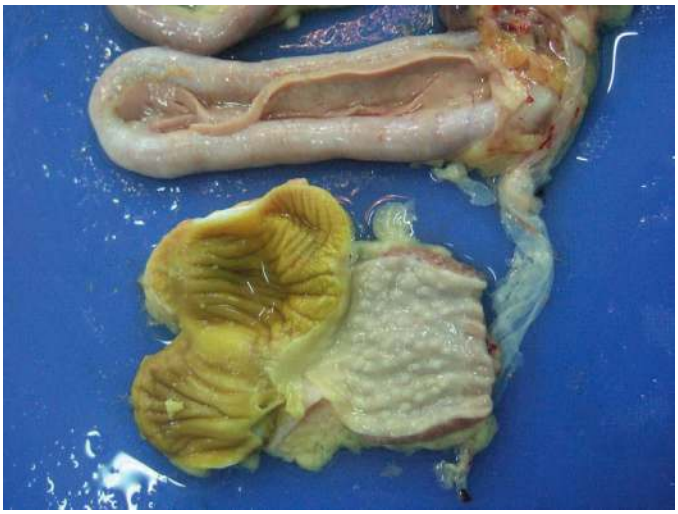


FIGURE 16-19 The opened proventriculus or glandular stomach (right) showing the mucosa and the muscular gizzard (left) showing the hardened cuticle of the koilin layer. This type of "stomach" is typical of a graminivorous bird. Note the U-shaped loop of the duodenum (top) enclosing the pancreas.

a hardened cuticle (the koilin layer; see Fig. 16-19). In the Sphenisciformes the distensible stomach extends caudally into the lower abdomen almost as far as the cloaca. However, in fruit eaters such as lories (many species of the subfamily Psittacinae of the family Psittacidae, e.g., *Domicella* spp.) it is lightly muscled and flaccid.

The intestinal tract is divided into small and large intestines (see Fig. 16-18), although these areas are not always immediately obvious. The duodenum is a U-shaped loop of the gut with a proximal descending part and a distal ascending part enclosing the pancreas (see Fig. 16-19). The duodenum then merges into the jejunum. The small intestine is typically divided into the duodenum, jejunum, and ileum. In nectar-feeding species, such as humming birds (Trochilidae), the small intestine is unusually short. In most species of bird, the various parts of the small intestine are not well differentiated and merge imperceptibly.

The ceca, when present, are regarded as part of the large intestine and mark the junction between the small and large intestines (see Fig. 16-18). Ceca are absent in some species, including some members of the Coraciiformes, Piciformes, and Psittaciformes such as the budgerigar (*Melopsittacus undulatus*). Most birds have two ceca, but herons (Ardeidae) and Gaviiformes have only one. In some species, e.g., Columbiformes, the ceca are vestigial and in some other species, such as small passerines, they may be difficult to detect with the naked eye. The largest ceca are seen in the Tetraoninae (grouse and capercaillies). In owls (Strigiformes), the distal extremities of the ceca are expanded, forming saccular structures. In the ostrich the ceca are long and sacculated.

The large intestine in birds (unlike many other vertebrates) is short and probably homologous to the mammalian rectum. It is sometimes referred to as the colorectum. Together with the genital and urinary tracts it empties into the cloaca. In psittacines, and many other families of bird, there is no clear distinction between colon and rectum, and this part of the gut is represented by a short colorectum. Its diameter is larger than the preceding small intestine. The cloaca is a chamber into which the terminal parts of both the digestive and urogenital systems open. The cloaca opens to the outside at the vent. The chamber is divided internally by two mucosal folds, which form three compartments: the coprodeum, urodeum, and proctodeum.

The alimentary tract (already removed) should be unraveled and examined systematically, but before opening the crop, proventriculus, and gizzard, suitable containers should be available to hold the contents, which may be required for toxicologic examination or for identification of any food that has been eaten. Open and examine one area of the tract at a time.

Examine serosal surfaces and look for adhesions. Note the contents and appearance of the mucosa throughout the gastrointestinal tract (GIT). Examine beneath the koilin layer of the gizzard. Take scrapings on a microscope slide from various areas; make wet preparations (preferably on a warmed slide to activate motile protozoa); and examine microscopically for *Macrorhabdus ornithogaster*, *Trichomonas* spp., *Giardia* spp. and *Hexamita* spp., coccidia, etc., and helminths. Collect parasites and/or prepare smears. Leave a small area of the tract intact for histopathological and other examinations. Never submit portions of gut that have been scraped with a scalpel for histologic, mycologic, or virologic examination because this damages tissues and causes contamination. Use only opened segments of the GIT for histology; otherwise there will be poor fixation of the mucosal surface. Examine the pancreas and do not delay fixation for histology.

Lymphoreticular System

The spleen is situated between the proventriculus and the gizzard on the right side (see Fig. 16-11). The shape varies considerably. It is elongated and sausage shaped in Passeriformes and Columbiformes, almost spherical in Galliformes and Psittaciformes, and roughly triangular in Anseriformes. When examining the spleen, it is important to remember that enlargement is not necessarily pathologic and can be physiologic. It may be necessary to make impression smears of the spleen (see Liver, above).

Using bone forceps, examine the bone marrow (Fig. 16-20), remembering that this may be replaced by extensions of the air sac system in some of the larger limb bones. Bone marrow can be collected for histologic examination by removing the bone marrow and wrapping it in muslin to form a "sausage" before fixation. Impression smears of the marrow are especially useful but must be made with care to avoid rupturing the cells. This is best performed by aspirating the marrow using a syringe and hypodermic needle. Bone marrow specimens must be collected as soon after death as possible, because the cells rapidly undergo degeneration.

Most birds do not have lymph nodes, but they are present in Anseriformes. In these birds one pair is situated near the thyroids and the other pair near the kidneys, and they are spindle shaped. The bursa of Fabricius, situated in the dorsal wall of the cloaca, is a lymphatic organ. It is most easily seen in the young because it involutes with age.

Urinary System

In birds, the kidneys are lobular and situated in bony depressions of the synsacrum. They extend from the posterior edges of the lungs to the end of the synsacrum. The urinary bladder is absent in all birds.

The appearance and especially the color of the kidneys should be noted. A brownish color, with tubules prominently distended with whitish urates indicates nephrosis. The kidneys can be removed by cutting through the external iliac and ischiatic blood vessels on the lateral surfaces and carefully lifting them out of the dorsal wall of the synsacrum using a scalpel (Fig. 16-21). This exposes the lumbosacral plexus (see Nervous System, below).

Reproductive System

Females. In most vertebrates the ovaries are paired internal organs, but in birds only the left ovary is usually present (Fig. 16-6). The exceptions are many Falconiformes of the families Cathartidae, Accipitridae, and Falconidae and the brown kiwi (*Apteryx australis*).

Record the appearance of the ovary and look for follicles. If a fully formed egg is found in the oviduct of an uncommon bird, it should be collected intact, in case it is needed for special studies. If there is any possibility of salmonellosis, then the ova should be taken for bacteriologic examination because some *Salmonella* spp. can be transmitted by egg. Completely open the oviduct and look for impactions with egg material and other lesions.

Males. The testes are paired, as in mammals, but are internal and situated just anterior to the kidneys. They are usually ovoid and variable in color from whitish gray (Fig. 16-22) to black. The single penile organ (lacking a urethra) is vestigial in most birds, except ratites and Anseriformes.

Nervous System

The nervous system, especially the autonomic system, is the most difficult to examine. When dysfunction of the autonomic nervous system (dysautonomia) is suspected, the autonomic ganglia need to be located

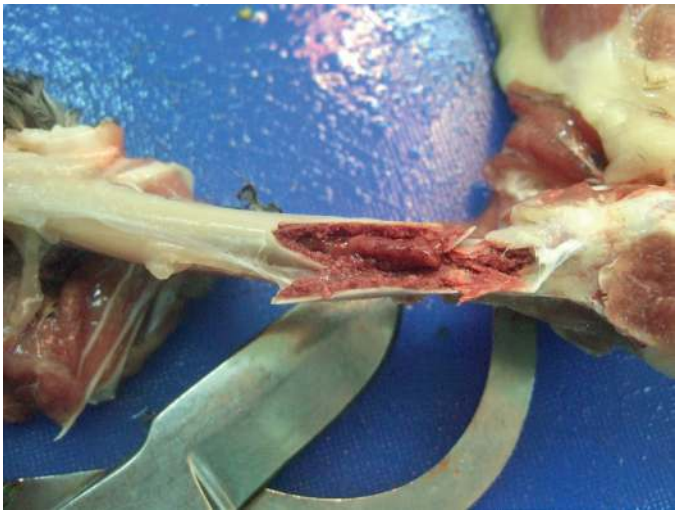


FIGURE 16-20 Using bone forceps, the right femur has been cracked open to expose the bone marrow.

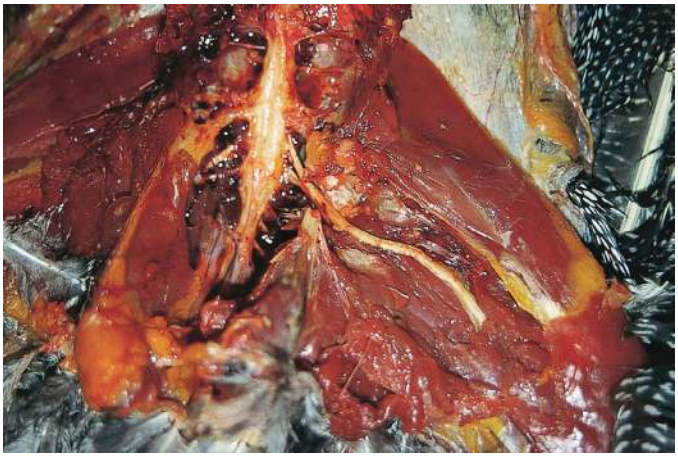


FIGURE 16-21 The kidneys have been removed from the synsacrum to expose the lumbosacral plexus on each side. Using bone forceps to cut through the ilium on the left side, the ischiatic nerve has been exposed. This is the largest nerve supplying the leg. (Courtesy Dr. I.F. Keymer.)

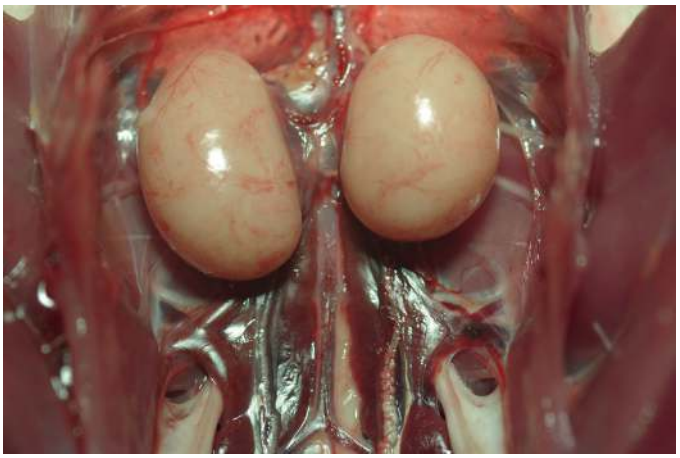


FIGURE 16-22 The testes are paired and situated just anterior to the kidneys. They are usually ovoid and variable in color, whitish gray or black.

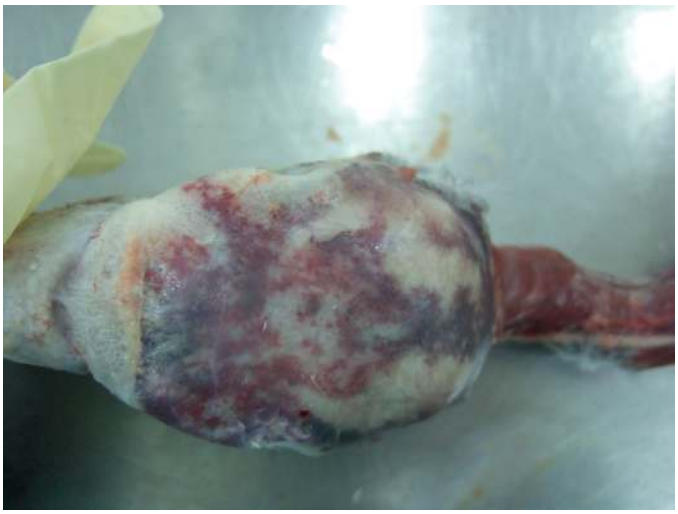


FIGURE 16-23 The head of a small bird with hemorrhages in the bony substance of the skull. These are usually agonal and of no pathologic significance, unless they are associated with contusions and/or hemorrhages intracranially and/or in the overlying skin of the head.

and examined histologically. This disorder occurs in proventricular dilatation disease of psittacines.

The peripheral nerves (especially the brachial and lumbosacral plexuses) can be examined at various stages of the necropsy (Fig. 16-21). The brachial plexus is very obvious in the brachial region, but to see the lumbosacral plexus, it is necessary to remove the kidneys. Nerves taken for histology should be pressed on a piece of card and fixed in formol-saline. Pressing flat on a card ensures that the nerve remains straight during fixation, preventing curling.

Frequently when skinning the head of a small bird in preparation for removal of the brain, hemorrhages can be observed in the bony substance of the skull (Fig. 16-23). These are usually agonal and of no pathologic significance, unless they are associated with contusions and/or hemorrhages intracranially and/or in the overlying skin of the head. These hemorrhages are also believed to occur in birds following severe terminal activity, such as after fright.

Brains of large species can be exposed by sawing off the top of the cranium using an oscillating or hand saw, so that it can be replaced should the skull be needed for taxidermy. To remove the brain, the skull is held upside down (Fig. 16-24) and the brain is gently detached out of the cranium with a scalpel handle, using gravity.

For the purposes of bacteriologic or virologic examinations, the brain might be split into smaller parts. Half of the brain should always be sent for histology because different diseases need to be looked for at different central nervous system locations.

Brains should always be examined with extra care, because handling and autolysis can cause artifacts that make histologic examination difficult. Preferably large brains should be partly sliced transversely to facilitate penetration of the fixative. It is preferable to keep the brain intact and renew the fixative every 12 hours for 3 to 4 days. Alternatively, a sagittal section of the skinned head can be made and fixed intact. The halved brain is removed after fixation. Use bone forceps and/or scalpel to remove the spinal cord. Place it on a piece of card before fixation. After removing the brain, the internal ear is exposed and inflammation might be observed (Fig. 16-25).

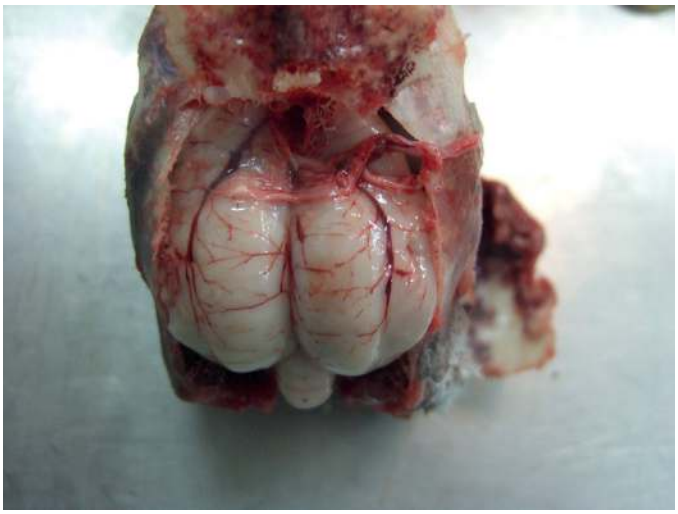


FIGURE 16-24 To remove the brain, the opened skull is held upside down and the brain is gently detached out of the cranium with a scalpel handle, using gravity.

Termination

Reread the history and check that nothing has been forgotten. Retain all carcass remains and refrigerate for 72 hours in case some of the remains are needed. With rare species or if legal procedures are expected, retain the carcass in a deep freezer and suitably preserve the remains of all organs. It also may be prudent to have a witness present to confirm that the necropsy findings have been accurately recorded and that specimens, which might be needed for forensic purposes, are correctly labeled and sealed. When deep freezing tissues, it is especially important to tie labels very securely to all carcass remains and containers so they do not become detached and to use waterproof markers. Even so, labeling often becomes illegible in deep freezers. Color-coded labels are helpful. Enter details and record a provisional or final diagnosis on the necropsy sheet (see Table 16-1) and elsewhere, for instance, in the practice or laboratory daybook.

Cleanse and disinfect instruments and area, including external surfaces of specimen containers. When tissues have to be transported to a laboratory for histopathological examination, the overall weight can be reduced by removing fixed tissues from the containers and sending them in formalin-soaked cloth gauze, carefully labeled and sealed in plastic bags. However, if this is done, care must be taken to ensure that

FIGURE 16-25 After brain removal, the internal ear is exposed and inflammation might be observed.

the tissues are not crushed when packing and that they cannot be damaged during transit.

Supplementary Diagnostic Procedures

Histopathologic Examination

Routinely collect at least the liver, lung, kidney, and any tissue showing macroscopic lesions. Do not use rat-tooth forceps for this purpose, because they damage the tissues. Always submit a representative portion of the lesion to include apparently normal tissue from the periphery and also obviously diseased tissue in the center. Do not use tissues that are discolored or autolyzed (e.g., as the result of being in contact with the intestinal tract or exposed for an excessive amount of time to the atmosphere, resulting in desiccation).

Some tissues, such as the alimentary tract, pancreas, bone marrow, brain, and kidney, decompose more quickly than heart and skeletal muscle. Fix tissues in 10% buffered formol-saline, using at least six times (preferably 10 to 20 times) the volume of the fluid to the volume of the specimens. Ideally tissues should not be more than 5 mm thick.

When specimens are large in diameter and if several are placed in one container, it is important to increase the volume of fixative proportionately and to replace with fresh fixative twice a week. Fixation time varies with different tissues; for example, those containing fibrous tissue take longest, but it can be hastened by slightly warming the formol-saline fixative. With some tissues (brain) it may be necessary for them to remain in the fixative for as long as 2 weeks before they can be processed for histologic examination.

Label specimen containers (not the lids) immediately after introducing tissues and keep different areas of the gut in separate containers. For urgent cases, tissues can be cut frozen using a cryostat, but the results are usually less satisfactory than when fixed with formalin.

Bacteriologic Examination

Routinely take swabs of contents of at least two areas of the intestinal tract. If the bird died naturally or was euthanized because it was sick, routinely culture heart blood, liver, and any organ showing macroscopic lesions. If tissues are not to be pooled, then use separate sterile instruments for each tissue collected. Place uncontaminated tissues in sterile Petri dishes (or use sterile swabs), label, and submit for subsequent examination in case they are needed (see Virologic Examination, following).

Mycologic Examination

Take for culture and other examinations any tissue suspected of being infected and label the container (see Bacteriologic Examination, previously).

Toxicologic Examination

Routinely take liver, kidneys, skeletal muscle, brain, content of crop and proventriculus and, if present, body fat. Label containers with a waterproof marker and place in deep freezer at –20° C (–4° F) until required.

Virologic Examination

Remove tissues as applicable and obtain instructions from the virologist who will examine them. Attempt sterile precautions, although under many circumstances some contamination of tissues is almost inevitable. If tissues are not to be pooled, then use separate sterile instruments for each tissue collected. If examination is likely to be delayed, tissues should be frozen at below –60° C (–76° F) and transported packed in dry ice or liquid nitrogen. When taking tissues for electron microscopy they should be cut in very small pieces not exceeding 1 mm^2 and fixed immediately in freshly prepared cold glutaraldehyde or similar fixative. If an ultracryomicrotome is available for "cryosectioning," tissues can be stored until required at –70° C (–94° F) instead of being fixed.

Parasitologic Examinations

In addition to collecting parasites as described previously and examining the intestinal tract, also tie off and label representative areas of the gut for subsequent examination, and refrigerate.

Polymerase Chain Reaction

This relatively recent test for identification of bacteria and viruses was believed to be more suited for use in research than for routine diagnostic procedures (Grainger and Madden, 1993). However, the last few years have seen significant advances in the development and application of polymerase chain reaction (PCR) techniques in avian medicine. PCR diagnostic techniques today are widely used by practitioners in the diagnosis of infectious diseases, including chlamydiosis (Rosenthal, 2001), psittacine beak and feather disease virus (Greenacre, 2002), polyomavirus and herpesvirus infections (Styles *et al.*, 2004), and avian bornavirus (Payne *et al.*, 2012; Hoppes *et al.*, 2013), as well as in the diagnosis of *Aspergillus fumigatus* in avian samples (Dahlhausen *et al.*, 2004).

ACKNOWLEDGMENTS

I am grateful to Dr. J. Samour and Dr. I.F. Keymer for providing photographs.

REFERENCES

Arnall L, Keymer IF: Sampling and necropsy. In Arnall L, Keymer IF, editors: *Bird diseases: an introduction to the study of birds in health and disease*, Neptune City, NJ, 1975, TFH Publications.

Bibby CJ: Measurement. In Campbell B, Lack E, editors: *A dictionary of birds*, British Ornithologists Union, Carlton, Staffordshire, UK, 1985, T & AD Poyser.

Dahlhausen B, Abbott R, Van Overloop P: Rapid detection of pathogenic *Aspergillus* species in avian samples by real-time PCR assay: a preliminary report. *Proceedings of the Association of Avian Veterinarians*, New Orleans, 2004, pp 37-40.

Grainger J, Madden D: The polymerase chain reaction: turning needles into haystacks, *Biologist* 40:197–200, 1993.
Greenacre CB: My case work-up for 3 common presentations. *Proceedings of the Association of Avian Veterinarians*, Monterey, CA, 2002, pp 222-241.
Hoppes SM, Tizard I, Shivaprasad HL: Avian bornavirus and proventricular dilatation disease: diagnostics, pathology, prevalence, and control, *Vet Clin North Am Exot Anim Pract* 16(2):339–355, 2013.
Keymer IF: Post-mortem examination, with anatomical notes. In Samour J, editor: *Avian medicine*, Philadelphia, PA, 2008, Mosby-Elsevier.
King AS, McLelland J: *Birds: their structure and function*, London, 1984, Baillière Tindall.
McLelland J: *A color atlas of avian anatomy*, Philadelphia, PA, 1991, WB Saunders.
Payne SL, Delnatte P, Guo J, et al: Birds and bornaviruses, *Anim Health Res Rev* 13(2):145–156, 2012.
Rosenthal K: Interpreting clinical pathological results in avian patients. *Proceedings of the Association of Avian Veterinarians*, Orlando, FL, 2001, pp 281-287.
Styles DK, Tomaszewski EK, Phalen DN: Psittacid herpesvirus associated with internal papillomatous disease in psittacine birds. *Proceedings of the Association of Avian Veterinarians*, New Orleans, 2004, pp 79-81.
Wernery U, Wernery R, Kinne J, et al: *Colour atlas of falcon medicine*, Hannover, Germany, 2004, Schlütersche.

FURTHER READING

Aughey E, Frye FL: *Comparative veterinary histopathology with clinical correlates*, The Veterinary Press, London, 2001, Manson Publishing.
Dorrestein GM: Clinical pathology and post-mortem examination. In Chitty J, Lierz M, editors: *BSAVA manual of raptors, pigeons and passerine birds*, Quedgeley, Gloucester, UK, 2008, British Small Animal Veterinary Association.
Evans HE: Anatomy of the budgerigar. In Petrak ML, editor: *Diseases of cage and aviary birds*, ed 2, Philadelphia, PA, 1982, Lea & Febiger.
Fowler ME: *Zoo and wild animal medicine*, ed 2, Philadelphia, PA, 1986, WB Saunders.
Fowler ME: Comparative clinical anatomy of ratites, *J Zoo Wildl Med* 22:204–277, 1991.
Fowler ME: Clinical anatomy of ratites. In Fowler ME, editor: *Zoo and wild animal medicine, current therapy 3*, Philadelphia, PA, 1993, WB Saunders.
Latimer KS, Rakich PM: Necropsy examination. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing.
Lowenstine LJ: Necropsy procedures. In Harrison GJ, Harrison LR, editors: *Clinical avian medicine and surgery*, Philadelphia, PA, 1986, WB Saunders.
Majó N, Dolz R: *Atlas of avian necropsy: macroscopic diagnosis sampling*, Zaragoza, Spain, 2012, Servet.
Schuster RK, Wibbelt G, Kinne J: On the life cycle and morphology of development stages of *Paraspiralatus sakeri* Gibbons et al., 2004 (Nematoda: Spiroidea, Spirocercidae), a heteroxenic stomach parasite of falcons, *Parasitol Res* 113(6):2047–2051, 2014.
Simpson VR: Post-mortem examination. In Beynon PH, Forbes NA, Lawton MPC, editors: *Manual of psittacine birds*, Cheltenham, UK, 1996, British Small Animal Veterinary Association.
Veit HP: Mistakes to avoid when submitting tissue for histologic evaluation, *Vet Med Small Anim Clin* 76:1143–1149, 1981.
Waine JC: Post-mortem examination technique. In Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *Manual of raptors, pigeons and waterfowl*, Cheltenham, UK, 1996, British Small Animal Veterinary Association.

EGG POSTMORTEM EXAMINATION

Jaime Samour

A comprehensive postmortem examination should be performed routinely in all eggs placed for artificial incubation after determining their viability through candling (e.g., PowerLux Egg Candler; Stromberg's Chicks and Game Birds, Pine River, MN) or by using an embryonic heart monitoring device (e.g., Buddy; Avian Biotech International, Tallahassee, FL) that the egg is infertile or the embryo is no longer thriving.

Embryonic death may occur during four distinct periods of incubation:

1. *First quarter*. Embryonic death tends to occur during the first week of incubation mainly from improper and rough handling, extended storage period and under incorrect temperature, adverse genetic traits, and aging parents.
2. *Second quarter*. Death of embryos in the middle of the incubation period may be attributed to nutrient deficiency within the egg, incorrect turning of the eggs, and inappropriate temperature and humidity within the incubator. The age of the parents is also believed to be an important factor for the arrest of embryonic development.
3. *Third quarter*. Embryonic death at the end of the incubation period is commonly caused by incorrect turning of the eggs, inappropriate temperature and humidity within the incubator, malposition of the developing embryo, infections caused by bacteria or fungi, and aging parents.
4. *Fourth quarter*. Fully developed chicks fail to hatch at the end of the incubation period mainly because of cervical edema linked to inadequate egg weight loss during incubation arresting the ability of the chick to move freely and to cutting out the shell during hatching. Malpositioning is also an important cause of hatching failure.

EGG EXAMINATION PROCEDURE

Eggs submitted for postmortem examination should be sent in a transparent plastic bag together with a complete egg postmortem examination form and egg record form containing all the relevant information including the identification (ID) of the egg, species, ID of the parents, date of laying, egg washing and or cleaning details (if any), storage time (if any) and temperature, date of setting, type of incubator (e.g., forced air), egg weight at laying, days of incubation, incubation temperature and humidity, and egg turning program.

- The eggs should be weighed, measured, and examined for cracks and shell abnormalities. A note should be made on the shape, color, and coloration patterns (if any; Fig. 16-26).
- The egg shell around the air cell should be thoroughly disinfected using ideally a suitable alcohol-based disinfectant product (e.g., surgical spirit; Fig. 16-27).
- An opening above the air cell is first made with a 16-gauge sterile hypodermic needle and the opening gradually enlarged using conventional round drill bits or triangular drill burr to make a small (3 to 5 mm) opening. Then, using a sharp pair of scissors, a small window (1 × 1 cm) is made to visualize the appearance and integrity of the air cell membrane to assess if internal piping had occurred.
- After a sterile procedure, the operator then removes part of the air cell membrane to expose the content and/or the developing embryo.
- Microbiology swabs are then collected for bacteriology and mycology cultures (Fig. 16-28).
- The egg shell is then gently cut on its circumference to expose the content (Figs. 16-29 and 16-30). If a developing embryo or fully developed chick is present, careful observations should be made on the size and its condition and the presence of the egg tooth, and positioning of the head in relation to other anatomic structures should be determined, in particular, to the right wing. Every effort

FIGURE 16-26 The eggs should be weighed, measured, and examined for cracks and shell abnormalities. A note should be made on the shape, color, and coloration patterns, if any. This egg is from an ostrich (*Struthio camelus*).

FIGURE 16-27 The egg shell around the air cell should be thoroughly disinfected ideally using a suitable alcohol-based disinfectant product, e.g., surgical spirit. This is an egg from a gyr falcon (*Falco rusticolus*).

FIGURE 16-28 After making a small opening above the air cell and opening up the membrane, microbiology swabs can be collected for bacteriology and mycology cultures.

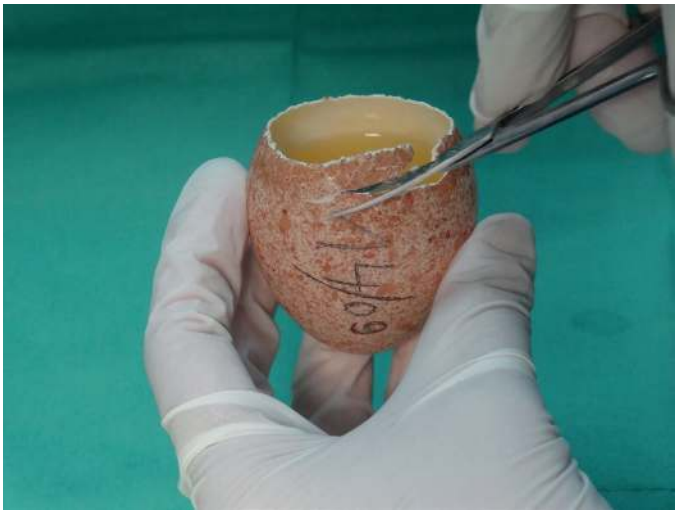


FIGURE 16-29 The egg shell is gently cut on its circumference to expose the content.

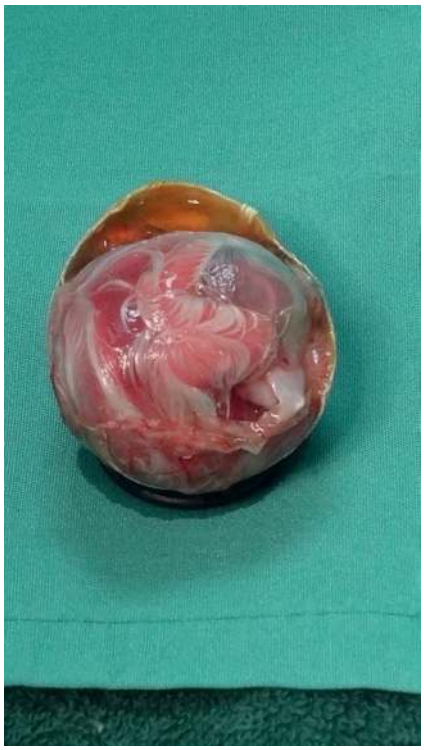


FIGURE 16-30 The egg's shell has been cut in its circumference to expose and determine the position of the embryo.

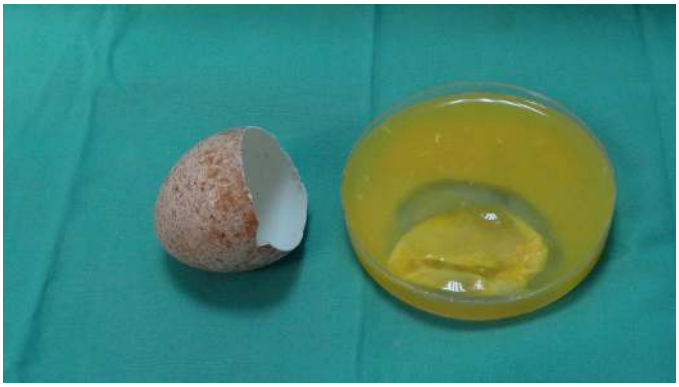


FIGURE 16-31 The content of the egg is then gently poured over a sterile Petri dish to examine further the content and to obtain additional samples if required.

should be made to classify the position of the chick following the standard postural classification method used in birds.

- The content of the egg is then gently poured over a sterile Petri dish to further examine the content and to obtain additional samples if required (Figs. 16-31 and 16-32). A note should be made if the yolk sac is still present outside the coelomic cavity of the chick and its size and appearance (Fig. 16-33).
- Full postmortem examination on fully developed chicks can be performed using standard postmortem techniques commonly used for birds and the necessary samples collected for further analyses.

ACKNOWLEDGMENTS

The author would like to thank Dr. Melodiya Magno for supplying all the images for this section.

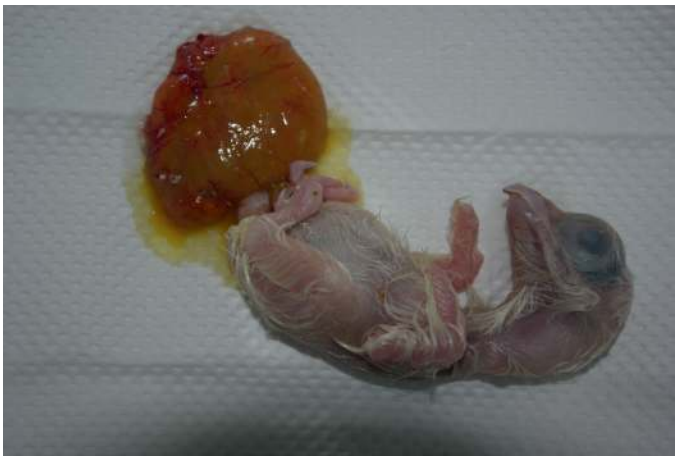


FIGURE 16-32 The embryo in this egg failed to hatch and probably died before taking up the normal hatching position. Please note the extensive yolk sac still present.

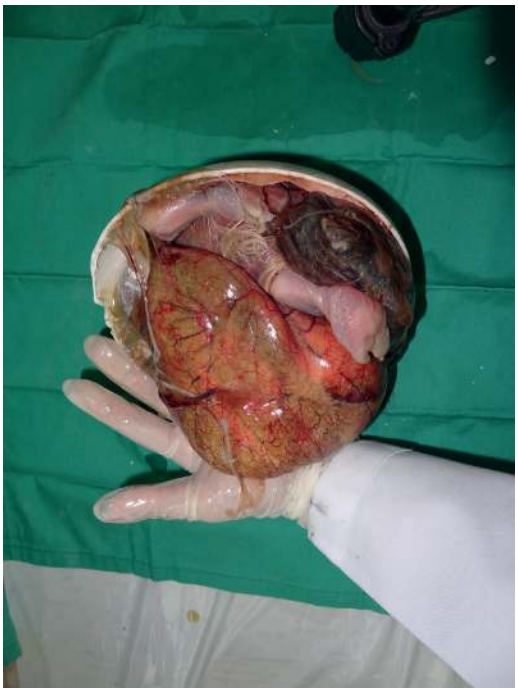


FIGURE 16-33 A note should be made if the yolk sac is still present outside the coelomic cavity of the chick and its size and appearance.

FURTHER READING

Anderson Brown AF, Robbins GES: *The new incubation book, Surrey*, BC, Canada, 2002, Hancock House Publishers.

Lierz M: Raptors: reproductive disease, incubation and artificial insemination. In Chitty J, Lierz M, editors: *BVAVA manual of raptors, pigeons and passerine birds*, Quedgeley, Gloucester, UK, 2008, British Small Animal Veterinary Association.

Olsen GH: Embryologic considerations. In Olson GH, Orosz SE, editors: *Manual of avian medicine*, St. Louis, MO, 2000, Mosby.

17

Forensic Investigations in Avian Medicine

John E. Cooper, Margaret E. Cooper

The Oxford Dictionary defines *forensic* as "relating to, used in or connected with a court of law," and the title of this chapter refers to any veterinary activities linking birds with the law. The primary emphasis is on legal issues such as the origin and parentage of captive birds; cause, mechanism, and manner of death; and assessment of health and welfare. The role of birds in causing ill health or death, for instance, by inflicting wounds on humans, is arguably also an area of avian forensic medicine but is not covered here.

Over the past 35 years there has been an exponential increase in the number of legal cases involving animals, including birds (Cooper and Cooper, 2007, 2013), in Western Europe and North America. As a corollary to this, the demand for expert veterinary opinion in court has greatly expanded.

Fields in which veterinary advice relating to birds may be sought include (1) determination of the cause and time of death, (2) identification and interpretation of apparently normal and patently pathological avian tissue, (3) unraveling circumstances relating to the origin and history of live and dead birds and their derivatives, and (4) assessment of welfare and whether a bird may be suffering.

Relatively little information specific to the forensic examination of birds has been published, with the exception of one or two early papers in the British literature (Cooper and Cooper, 1986, 1991). Other texts from North America (Stroud, 1995; Stroud and Adrian, 1996; Wobeser, 1996) focused attention on the need for specialized veterinary forensic laboratories and standard investigative techniques for wildlife. Cooper and Cooper (2007, 2013) discussed the application of forensic techniques to both domesticated and wild animals.

Here guidelines are given to assist veterinarians who may be asked to participate in avian forensic work.

GUIDELINES

General

1. Accept a legal case only if you feel competent to deal with it and are willing, if necessary, to appear in court. Bear in mind the possible costs, in terms of time, money, and reputation, of being involved in the legislative process. Laboratory and other tests are expensive. Confirm in advance that fees are available to cover such work.
2. Before providing an opinion or performing any investigation, ascertain what is required (this may be very different from routine diagnosis and/or treatment). Be prepared to consult colleagues, especially those with specialized knowledge of avian or forensic matters. Warn them beforehand that you are dealing with a legal case.
3. Maintain meticulous records of everything you do. Also use a tape recorder if working alone. Take photographs throughout the work. Regardless of whether records are handwritten, typed, or computerized, retain all raw data, even if some observations seem to be irrelevant at the time. Record data in unambiguous, scientific terms, but be prepared to translate this into lay terms if the case goes to court.
4. Ensure that material that might possibly need to be produced in court is not discarded. If in doubt, retain everything. Carcasses and tissues can be frozen or fixed for subsequent examination.
5. Remember that possession of a bird that has been illegally captured, illegally killed, or illegally imported may be an offense, even though you are a veterinarian. Clarify the situation and, if appropriate, apply to the relevant body for a license or written approval.

Live Birds

1. Observation of the bird and its environment should precede restraint, handling, or clinical examination. Note the bird's behavior, including commenting on how docile, tame, or habituated it appears to be; whether it might be imprinted; whether it is easily frightened by noises or other stimuli; and whether it responds normally to routine stimuli. Familiarize yourself with the natural history and biology of the species that is the subject of the investigation (Fig. 17-1). Consider involving a colleague, birdkeeper, or ornithologist that may have more knowledge of such aspects than you.
2. Perform as full an investigation as possible. In addition to standard clinical examination, record weight (mass) and standard measurements, give a condition score, and report specifically on the following: plumage (including evidence of molt or pinioning), beak, claws, soles of feet, presence/absence of leg rings (bands), jesses, telemetry equipment or other attachments, and presence of tattoo(s) or colored dyes. Pay special attention to clinical signs or lesions that may be relevant to a history of alleged neglect, cruelty, or persecution (Fig. 17-2). Throughout the clinical examination, practice strict hygiene, following a proper risk assessment (Cooper and Cooper, 2013).
3. Take photographs throughout the examination, even if only simple photographic equipment is available, especially if there are lesions that may change in appearance if there is any delay (Fig. 17-3).
4. Routinely take the following samples for laboratory tests: fresh droppings (feces and urates), blood smears, blood for hematology/clinical chemistry, dropped feathers, pellets/castings (where available), and ectoparasites. Mark all specimens carefully and fully on a label on the container, not on the lid.
5. Depending upon the circumstances be prepared to perform supporting investigations, e.g., radiography and ultrasonography, emesis, and lavage. Imaging may reveal significant lesions that cannot be easily detected on palpation (Figs. 17-4 and 17-5).

FIGURE 17-1 This injured owl has pink eyelids that look bruised; however, they are a normal feature of this species (Verreaux's eagle owl [*Bubo lacteus*]). Knowledge of normal biology is vital in forensic work.

FIGURE 17-3 The foot and leg of a common buzzard (*Buteo buteo*), with injuries suggestive of being caught in a trap. This photograph was taken immediately to have a contemporaneous image for legal purposes. Delay in recording details of such cases can result in changes to the lesions, which may prejudice the evidence.

FIGURE 17-2 A lesser flamingo (*Phoenicopterus minor*) requires competent handling and careful, methodical clinical examination. This individual was found with one leg missing and the other shows joint damage.

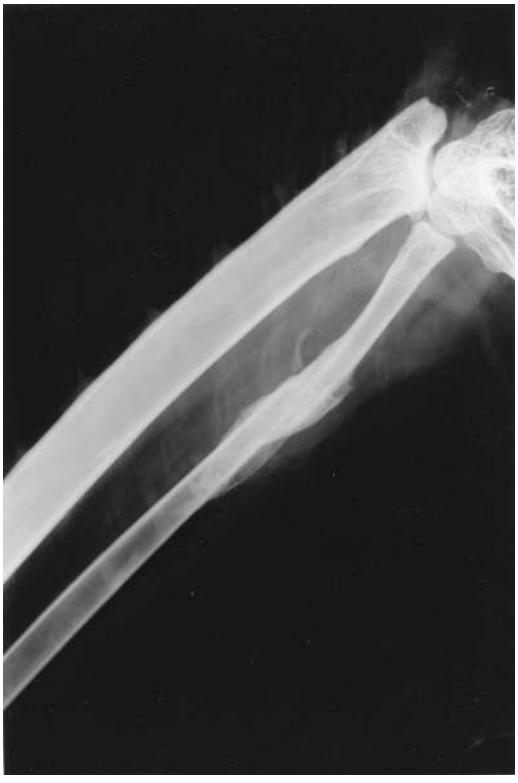


FIGURE 17-4 A routine plain radiograph of the (apparently normal) wing of a golden eagle (*Aquila chrysaetos*), submitted for forensic clinical examination, reveals a healed fracture of the radius and associated soft tissue swelling.

Dead Birds

1. Consider sending a dead bird to a specialist (avian) pathologist for postmortem examination, rather than necropsying the specimen yourself, especially if particular, nonroutine investigations are likely to be needed.
2. If you decide to deal with the case yourself, try to ensure that the carcass arrives in a fit state for examination; that is, as soon as possible after death; chilled, not frozen or fixed; properly packed, labeled, and dispatched in accordance with the relevant postal regulations. Useful data on these aspects were provided by Cooper (2002).
3. As a general rule, plan to perform the postmortem examination as soon as possible after receipt of the specimen. However, it is sometimes wise or necessary in forensic work to delay the examination for 24 to 48 hours (Cooper and Cooper, 2007; during which the specimen should be kept chilled at 4°C [39.2°F]), pending receipt of further information or instructions.
4. Initially perform a full external examination, paying particular attention to the molt, the condition of the plumage, and any evidence of injuries. The latter may range from palpable fractures to barely detectable, but potentially significant, feather damage (Fig. 17-6).

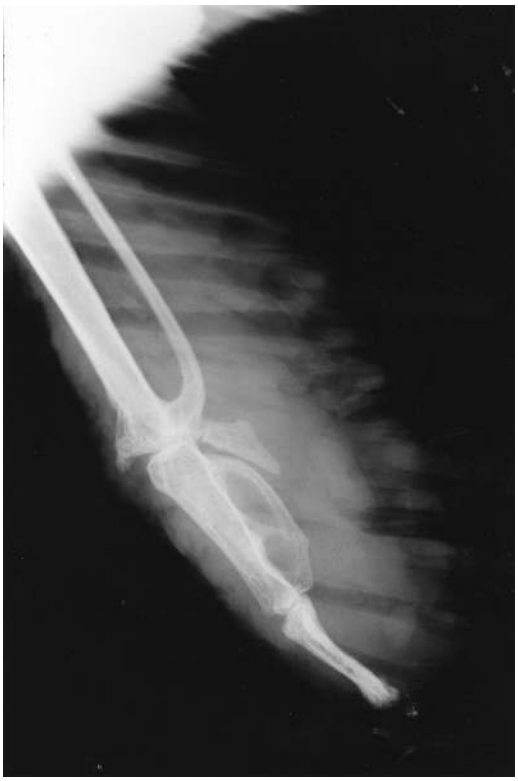


FIGURE 17-5 The degree and extent of soft tissue injury—in this case, fibrosis—may help in determining the age and pathogenesis of an injury. In this radiograph there is also evidence of osseous change in the wing tip.

FIGURE 17-6 Microscopic examination of apparently minor lesions on feathers may yield important forensic information. This scanning electron micrograph shows clubbing of barbs and barbules, which is a feature of ballistic (gunshot) damage.

5. Follow the same rules as with a live bird: perform standard morphometrics (mass and measurements), record body condition, etc. Wetting the plumage with an alcohol-based preparation will make it easier to find tattoos and superficial lesions. Skinning may detect subcutaneous bruising. Radiography (whole body, ventrodorsal, and lateral) should be performed routinely in legal cases; it will help locate shot, foreign bodies, skeletal injuries, and other abnormalities. Throughout the necropsy, practice strict hygiene, following a proper risk assessment (Cooper and Cooper, 2013).
6. A full (standard) internal examination is essential but, unlike dealing with "nonlegal" cases, this should not be confined to the detection of pathological lesions. Other findings may be equally relevant in court. The carcass should be skinned and body organs either weighed or retained for subsequent weighing. Everything should be kept that may be needed for toxicological or other analyses, including stomach and intestinal contents. Strict hygienic precautions should be followed.
7. The selection of laboratory samples from postmortem cases depends on personal preference: the author routinely takes wet preparations of intestinal contents for parasitological investigation (and examination of food remains) and pieces of lung, liver, and kidney for cytological and histopathological study. Samples can be examined in the practice if facilities and expertise are available or sent to a specialized laboratory (see the following section).
8. After gross examination and removal of samples for laboratory investigation, the whole carcass and the remaining tissues should be wrapped hygienically and labeled externally and deep frozen in a standard, dedicated freezer. They can be removed and thawed for further investigation or if they need to be produced in court. A note should be made on the postmortem examination records as to where the carcass and derivatives have been stored.

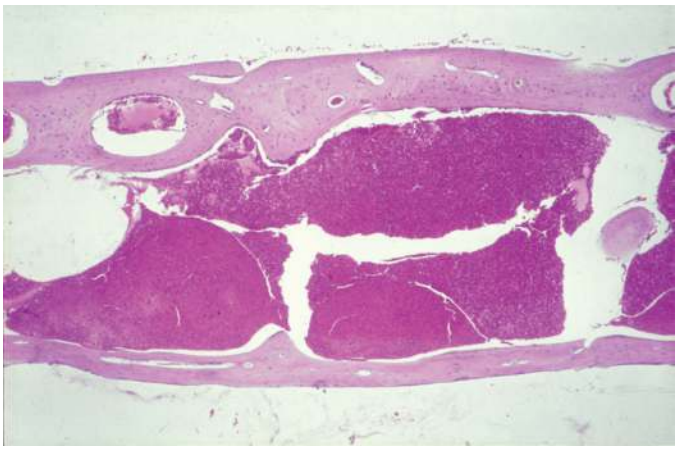


FIGURE 17-7 Histological examination of a goshawk (*Accipiter gentilis*), which appeared at necropsy to have damage to the skull. The section shows that the "bruising" is in fact agonal "intraosseous hemorrhage."

Samples for Laboratory Examination

1. Such samples may be obtained from live birds, from dead birds, and from the bird's habitat (if free living) or environment (if captive).
2. Unless the practicing veterinarian has appropriate facilities and expertise he should refer forensic samples to a specialized laboratory. If he *does* embark on in-house laboratory investigations, these should usually only involve standard simple tests, such as direct examination of feces. Use in the veterinary practice of a hand lens or dissecting microscope may detect parasites or lesions that warrant more detailed investigation in an appropriate laboratory (see Fig. 17-6). Some readily visible "lesions" may prove not to be pathological (Fig. 17-7). Throughout any such laboratory investigations, practice strict hygiene, following a proper risk assessment (Cooper and Cooper, 2013).

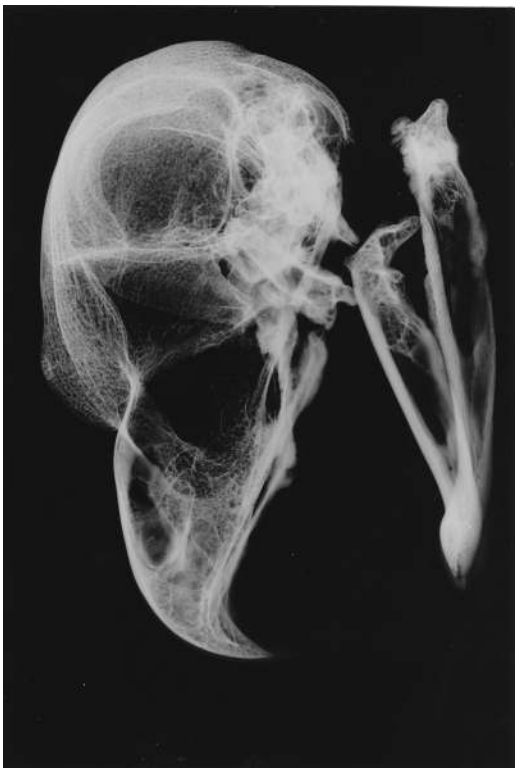


FIGURE 17-8 Radiographic examination of the skull of an owl reveals exostoses, attributable to an earlier *Trichomonas* sp. infection, that were not detected during clinical examination or necropsy. Imaging often helps detect such lesions and provides a record that can be produced, if necessary, as evidence.

3. All samples examined must be recorded in detail and every effort made to avoid mislaying or transposing material or results. Specialist opinions, for example, on the identification of parasites or the interpretation of histopathological sections, should be sought at an early stage. Similarly if special facilities are required, such as chemical analysis, arrangements must be made promptly and the laboratory warned in advance that their findings may be produced and discussed in court.
4. Comprehensive investigation of some samples may require further time, long after completion of the initial clinical or postmortem examination. For example, the ova, larvae, or pupae of insects found on the bird or in its environment are likely to need days or weeks before they metamorphose. Skeletal preparations may provide vital information that was missed during clinical or postmortem examination (Fig. 17-8).
5. The remains of all samples should, whenever feasible, be stored frozen or fixed for future reference. Slides and similar diagnostic material should be retained for as long as feasible. Photographs and photomicrographs of findings are very important and before storage must be properly labeled and cataloged because they may need to be produced in court.

DISCUSSION

The guidelines above are intended primarily to assist avian practitioners. They may need to be modified to suit individual circumstances. The most important messages arising from this chapter are that the practicing veterinarian who embarks on forensic work must have a methodical and consistent approach. His Achilles heel is how he performs, not his opinion. The veterinarian must also be prepared to present and to defend his findings in court. As an expert witness he has a duty to the court and not to support the prosecution's or defense's case. Such an impartial approach will require candor and openness, including a willingness to concede points and admit ignorance or omissions. Such transparency is not easy, especially if the veterinarian has been called as a witness by a deeply committed and powerful lobby, such as an influential animal welfare or conservation body.

The word *forensic* is derived from the Latin *forum* and is a reminder that forensic matters are liable to be discussed openly and debated at length in court. This approach is very different from routine avian veterinary practice where client confidentiality is still paramount and where the aim is usually to promote the health and welfare of the bird, not to contribute to legal wrangling.

Nevertheless, involvement in court cases is an important responsibility for the veterinary profession and the avian practitioner can, if properly prepared, make a significant contribution.

ACKNOWLEDGMENTS

We are grateful to Stephen Cooke, MRCVS, for reading and commenting on this document and to Pauline Muirhead for typing the first draft.

REFERENCES

Cooper JE: *Birds of prey: health & disease*, Oxford, UK, 2002, Blackwell.

Cooper JE, Cooper ME: Is this eagle legal? A veterinary approach to litigation involving birds. *Proceedings of the Forensic Zoology Discussion Group*, Zoo-technology, London, pp 27–30, 1986.

Cooper JE, Cooper ME: Legal cases involving birds: the role of the veterinary surgeon, *Vet Rec* 129:505–507, 1991.

Cooper JE, Cooper ME: *Introduction to veterinary and comparative forensic medicine*, Oxford, UK, 2007, Blackwell.

Cooper JE, Cooper ME: *Wildlife forensic investigation: principles and practice*, Boca Raton, FL, 2013, Taylor & Francis/CRC Press.

Stroud RK: Wildlife forensics: a new and challenging role for the comparative pathologist, *Comp Pathol Bull* XXVII:1–2, 1995.

Stroud RK, Adrian WJ: Forensic investigational techniques for wildlife law enforcement investigations. In Fairbrother A, Locke LN, Hoff GL, editors: *Non-infectious diseases of wildlife*, Iowa, IA, 1996, Iowa State University Press.

Wobeser G: Forensic (medico-legal) necropsy of wildlife, *J Wildl Dis* 32:240–249, 1996.

1 APPENDIX

Hematology Reference Values Table for Selected Avian Species

Merle M. Apo

Hematology Values for Selected Gruiformes

Species	Kori Bustard* *n* = 28	Houbara Bustard† *n* = 14	Heuglin's Bustard‡ *n* = 5	Black Bustard‡ *n* = 4	Buff-Crested Bustard§ *n* = 14	White-Bellied Bustard‡ *n* = 3
Scientific Name	***Ardeotis kori***	***Chlamydotis undulata***	***Neotis heuglinii***	***Eupodotis afra***	***Eupodotis ruficrista***	***Eupodotis senegalensis***
RBC, $\times 10^{12}$/L	2.30 ± 0.06 (1.74-2.95)	2.53 ± 0.09 (1.95-3.15)	2.18 ± 0.05 (2.05-2.35)	2.68 ± 0.18 (2.12-2.9)	2.89 ± 0.19 (2.00-4.27)	2.31 ± 0.08 (2.16-2.47)
Hb, g/dL	14.10 ± 0.16 (11.9-15.9)	14.72 ± 0.14 (13.7-15.7)	12.48 ± 0.37 (11.1-13.3)	14.62 ± 0.67 (13.6-16.6)	17.62 ± 0.52 (15.50-22.20)	15.23 ± 0.17 (14.9-15.5)
Hct, L/L	0.47 ± 0.05 (0.395-0.525)	0.47 ± 0.08 (0.42-0.51)	0.43 ± 0.005 (0.42-0.45)	0.44 ± 0.01 (0.42-0.49)	0.47 ± 0.01 (0.40-0.50)	0.47 ± 0.01 (0.45-0.50)
MCV, fL	208.5 ± 5.1 (161.9-275.4)	189.7 ± 8.85 (146.3-259.1)	198.14 ± 3.85 (185.1-206.3)	170.17 ± 14.04 (147.1-207.5)	172.85 ± 9.36 (105.39-220.00)	205.7 ± 8.3 (190.2-218.6)
MCH, pg	62.4 ± 1.6 (48.0-84.6)	58.9 ± 2.44 (46.6-74.3)	57.3 ± 2.57 (49.3-64.8)	55.32 ± 4.26 (47.1-65.5)	65.21 ± 4.46 (40.71-97.50)	66.03 ± 3.06 (60.3-70.8)
MCHC, g/dL	30.0 ± 0.4 (29.7-34.9)	31.16 ± 0.54 (26.1-34.1)	28.88 ± 1.15 (24.6-31.6)	32.52 ± 0.46 (31.5-33.5)	37.63 ± 1.18 (31.31-47.56)	32.1 ± 1.0 (30.6-34)
WBC, $\times 10^9$/L	7.29 ± 0.42 (3.05-12.85)	5.81 ± 0.29 (4.25-7.6)	4.24 ± 0.3 (3.41-5.2)	7.85 ± 2.22 (3.81-13.8)	5.66 ± 0.38 (4.00-9.80)	6.26 ± 0.7 (5.2-7.6)
Heterophils, $\times 10^9$/L	3.98 ± 0.32 (0.95-9.25)	3.64 ± 0.24 (1.99-4.82)	1.55 ± 0.17 (1.34-2.25)	2.92 ± 0.53 (1.52-4.21)	3.32 ± 0.32 (1.44-5.88)	2.73 ± 0.75 (1.92-4.25)
Lymphocytes, $\times 10^9$/L	2.21 ± 0.24 (0.41-5.45)	1.84 ± 0.15 (0.97-3.24)	1.91 ± 0.22 (1.33-2.44)	3.66 ± 1.56 (1.48-8.14)	1.11 ± 0.20 (0.31-3.03)	2.51 ± 0.17 (2.18-2.73)
Monocytes, $\times 10^9$/L	0.60 ± 0.07 (0.0-1.57)	0.15 ± 0.03 (0.0-0.42)	0.41 ± 0.1 (0.07-0.72)	0.68 ± 0.23 (0.42-1.38)	0.42 ± 0.10 (0.04-1.30)	0.45 ± 0.14 (0.22-0.72)
Eosinophils, $\times 10^9$/L	0.35 ± 0.05 (0.0-1.15)	0.07 ± 0.01 (0.0-0.23)	0.12 ± 0.02 (0.04-0.208)	0.17 ± 0.08 (0.07-0.41)	0.24 ± 0.04 (0.00-0.62)	0.24 ± 0.09 (0.06-0.36)
Basophils, $\times 10^9$/L	0.20 ± 0.03 (0.0-0.80)	0.07 ± 0.02 (0.0-0.26)	0.13 ± 0.089 (0.0-0.46)	0.4 ± 0.09 (0.26-0.69)	0.44 ± 0.08 (0.10-1.23)	0.31 ± 0.13 (0.07-0.54)
Thrombocytes, $\times 10^9$/L	5.5 ± 0.7 (1.49-18.0)	6.82 ± 0.59 (2.76-9.88)	5.11 ± 0.45 (3.85-6.57)	10.48 ± 2.99 (4.68-18.49)	8.81 ± 1.04 (4.00-15.00)	5.99 ± 2.9 (3.06-11.8)
Fibrinogen, g/L	2.42 ± 0.10 (1.42-4.5)	1.87 ± 0.26 (0.8-4.8)	1.71 ± 0.16 (1.12-2.11)	1.38 ± 0.19 (0.8-1.63)	1.7 ± 0.23 (0.66-4.31)	2.0 ± 0.15 (1.7-2.19)

*Howlett JC, Samour JH, D'Aloia M-A, et al: Normal haematology of captive adult kori bustards (*Ardeotis kori*), *Comparative Haematology International* 5:102–105, 1995.

†Samour JH, Howlett JC, Hart MG, et al: Normal haematology of the houbara bustard (Chlamydotis undulata macqueenii), *Comparative Haematology International* 4:198–202, 1994.

‡D'Aloia M-A, Samour JH, Bailey TA, et al: Normal haematology of the white-bellied (*Eupodotis senegalensis*), little black (*Eupodotis afra*), and Heuglin's (*Neotis heuglinii*) bustards, *Comparative Haematology International* 6:6–49, 1996.

§D'Aloia M-A, Howlett JC, Samour JH, et al: Normal haematology and age-related findings in the buff-crested bustard (*Eupodotis ruficrista*), *Comparative Haematology International* 5:10–12, 1995.

Mean ± standard error of mean (minimum-maximum).

Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Selected Gruiformes

Species	Manchurian Crane, *n* = 11	Sarus Crane, *n* = 9	Stanley Crane, *n* = 8	Crowned Crane, *n* = 11-22	Demoiselle Crane, *n* = 11
Scientific Name	***Grus japonensis***	***Grus antigone***	***Anthropoides paradisea***	***Balearica regulorum***	***Anthropoides virgo***
RBC, $\times 10^{12}$/L	2.2 ± 0.3 (1.9-2.7)	2.2 (2.0-2.5)	2.4 (2.2-2.7)	2.8 ± 0.3 (2.4-3.1)	2.7 ± 0.2 (2.3-3.0)
Hb, g/dL	14.3 ± 1.3 (12.6-16.8)	15.5 (13.8-17.7)	15.0 (13.4-16.8)	15.6 ± 1.9 (11.9-18.8)	14.9 ± 1.0 (13.1-16.2)
Hct, L/L	0.42 ± 0.04 (0.38-0.50)	0.45 (0.42-0.49)	0.42 (0.39-0.46)	0.47 ± 0.03 (0.44-0.52)	0.43 ± 1.1 (0.39-0.47)
MCV, fL	191 ± 9 (180-204)	203 (196-209)	174 (158-190)	171 ± 7 (156-182)	162 ± 7 (154-172)
MCH, pg	64.8 ± 4.5 (56.0-68.8)	69.6 (60.0-77.0)	61.9 (57.3-65.1)	64.3 ± 3.0 (59.8-70.2)	55.6 ± 2.4 (51.5-60.0)
MCHC, g/dL	34.3 ± 1.5 (32.7-37.2)	34.5 (30.6-37.0)	35.5 (33.1-39.5)	36.2 ± 2.3 (34.5-39.2)	34.3 ± 1.1 (32.6-36.2)
WBC, $\times 10^9$/L	9.5 ± 1.9 (5.7-11.6)	9.4 (3.5-12.2)	9.1 (2.9-16.9)	11.1 ± 2.8 (6.3-15.6)	5.3 ± 1.7 (2.9-8.6)
Heterophils, $\times 10^9$/L	6.7 ± 1.6 (4.5-9.3)	6.5 (1.4-9.5)	4.5 (1.0-10.0)	8.2 ± 3.0 (4.1-13.3)	3.8 ± 1.4 (1.7-6.6)
Lymphocytes, $\times 10^9$/L	1.9 ± 0.8 (0.5-2.9)	2.1 (1.2-3.0)	2.5 (1.1-4.2)	1.6 ± 0.6 (0.6-2.7)	0.8 ± 0.4 (0.4-1.5)
Monocytes, $\times 10^9$/L	0	(0.0-0.6)	(0.0-0.8)	(0.0-0.3)	(0.0-0.4)
Eosinophils, $\times 10^9$/L	(0.0-1.2)	(0.1-0.7)	(0.4-2.1)	(0.0-1.3)	(0.0-0.9)
Basophils, $\times 10^9$/L	(0.0-0.9)	(0.0-0.9)	(0.0-0.5)	(0.1-0.8)	(0.0-0.3)
Thrombocytes, $\times 10^9$/L	13 ± 2 (11-15)	15 (11-21)	18 (11-27)	(5-18)	12 ± 10 (4-32)
Fibrinogen, g/L	2.7 ± 0.4 (2.3-3.6)	2.7 (1.6-5.0)	3.5 (2.7-4.5)	-	2.5 ± 0.8 (1.4-3.7)

Source: Hawkey CM, Samour JH: The value of clinical hematology in exotic birds. In Jacobson ER, Kollias GV Jr, editors: *Exotic animals: contemporary issues in small animal practice*, New York, NY, 1988, Churchill Livingstone, pp 109–141.
Mean ± standard error of mean (minimum-maximum).
Hb, Hemoglobin; Hct, hematocrit; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Selected Falconiformes

Species	Turkey Vulture, *n* = 10	Egyptian Vulture, *n* = 4	Buzzard, *n* = 6	Golden Eagle, *n* = 4	Caracara, *n* = 9	Secretary Bird, *n* = 4
Scientific Name	***Cathartes aura***	***Neophron percnopterus***	***Buteo buteo***	***Aquila chrysaetos***	***Polyborus plancus***	***Sagittarius serpentarius***
RBC, $\times 10^{12}$/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)
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)
Hct, L/L	0.54 (0.51-0.58)	0.43 (0.37-0.46)	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)	34.0 (32.3-35.9)	35.2 (34.0-36.0)	36.9 (35.3-37.6)
WBC, $\times 10^9$/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, $\times 10^9$/L	11.8 (6.7-19.8)	4.0 (1.2-5.5)	5.5 (2.3-8.8)	10.4 (9.5-12.7)	4.2 (0.6-5.9)	5.3 (3.0-9.0)
Lymphocytes, $\times 10^9$/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, $\times 10^9$/L	(0.0-0.4)	(0.0-0.4)	0	0	(0.0-0.6)	(0.0-0.4)
Eosinophils, $\times 10^9$/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, $\times 10^9$/L	(0.0-2.3)	0	(0.0-0.6)	(0.0-0.2)	(0.0-0.3)	(0.0-0.4)
Thrombocytes, $\times 10^9$/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)

Source: Hawkey CM, Samour JH: The value of clinical hematology in exotic birds. In Jacobson ER, Kollias GV Jr, editors: *Exotic animals: contemporary Issues in small animal practice*, New York, NY, 1988, Churchill Livingstone, pp 109–141.
Mean ± standard error of mean (minimum-maximum).
*Range.
Hb, Hemoglobin; Hct, hematocrit; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Selected Falconiformes—cont'd

Species	Harris Hawk, *n* = 53	Ferruginous Hawk, *n* = 18	Red tailed Hawk, *n* = 15	Northern Goshawk, *n* = 43	Tawny Eagle, *n* = 29
Scientific Name	***Parabuteo unicinctus***	***Buteo regalis***	***Buteo jamaicensis***	***Accipiter gentilis***	***Aquila rapax***
RBC, $\times 10^{12}$/L	2.13-2.76*	2.41-3.59	2.2-3.35	2.6-3.48	2.32-2.83
Hb, g/dL	10.1-16.7	10.7-16.6	12.3-17.5	12.1-17.7	10.8-17.5
Hct, L/L	0.32-0.44	0.37-0.48	0.35-0.53	0.43-0.53	0.37-0.47
MCV, fL	147-163	150-178	157-168	141-156	163-188
MCH, pg	45.4-51.1	46-57.4	43-50.4	44.5-51.6	54-62
MCHC, g/dL	30.1-33.0	297-345	312-350	305-343	296-360
WBC, $\times 10^9$/L	4.8-10	4.5-6.8	3.4-7.5	4-11	5-9.5
Heterophils, $\times 10^9$/L	2.3-6.71	1.89-3.76	1.9-3.5	3.5-6.97	3.58-6.45
Lymphocytes, $\times 10^9$/L	0.6-2.36	0.78-1.74	1.3-1.1	1.38-1.93	0.51-2.72
Monocytes, $\times 10^9$/L	0.2-1.49	0.24-1.5	0.12-1.2	0-0.1	0.2-1.07
Eosinophils, $\times 10^9$/L	0-0.75	0.3-0.7	0.1-0.9	0-0.65	0.3-2.1
Basophils, $\times 10^9$/L	0-1.55	0.15-0.6	0-0.5	0-0.35	0-0.4
Thrombocytes, $\times 10^9$/L	10-59	8-47	4-33	15-35	19-25
Fibrinogen, g/L	<4.3	<3.5	<3	<3.5	<3.5

Source: Jennings IB: Haematology. In Beynon PH, Forbes NA, Harcourt-Brown NH: *Manual of raptors, pigeons and waterfowl*, Cheltenham, UK, 1996, British Small Animal Veterinary Association, pp 68–78.

*Range.

Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Selected Falconiformes—cont'd

Species	Saker Falcon,* *n* = 25	Peregrine Falcon,† *n* = 48	Gyrfalcons	Gyrfalcon,‡ *n* = 187	Lanner Falcon,§ *n* = 42	Laggar Falcon,§ *n* = 13	Merlin,§ *n* = 33
Scientific Name	***Falco cherrug***	***Falco peregrinus***	**Wild Caught**	***Falco rusticolus***	***Falco biarmicus***	***Falco jugger***	***Falco columbarius***
RBC, $\times 10^{12}$/L	2.65 ± 0.08 (2.05-3.90)	3.49 ± 0.21 (2.76-4.05)	3.91 ± 0.14** (3.1-5.12)	3.23 ± 0.28	2.63-3.98	2.65-3.63	2.85-4.1
Hb, g/dL	15.93 ± 0.38 (13.3-21.2)	14.82 ± 1.32 (11.6-19.1)	18.85 ± 0.23 (16.0-21.2)	15.00 ± 1.33	12.2-17.1	12.8-16.3	13.2-17.9
Hct, L/L	0.47 ± 0.59 (0.42-0.53)	0.40 ± 0.38 (0.26-0.58)	51.36 ± 0.90 (44-59)	0.45 ± 0.04	0.37-0.53	0.39-0.51	0.39-0.51
MCV, fL	183.16 ± 3.84 (135.8-219.5)	117.51 ± 7.70 (100.8-176.0)	135.83 ± 3.59 (106.18-162.36)	139.32 ± 5.44	127-150	123-145	105-130
MCH, pg	60.74 ± 1.42 (50.62-78.94)	—	49.44 ± 1.32 (39.17-59.67)	45.78 ± 1.84	42.3-48.8	38-47.7	36-45.9
MCHC, g/dL	33.28 ± 0.63 (28.33-40)	—	36.41 ± 0.16 (35.47-37.84)	—	317-353	312-350	340-360
WBC, $\times 10^9$/L	5.7 ± 0.31 (2.8-8.4)	12.56 ± 3.06 (7.6-21.2)	7.3 + 0.38 (4.2-10.8)	8.71 ± 3.80	3.5-11	5-9	4-9.5
Heterophils, $\times 10^9$/L	4.14 ± 0.24 (2.18-5.96)	4.52 ± 1.2 (1.38-7.53)	4.67 ± 0.34 (2.31-8.85)	58.53 ± 12.90%	1.65-8.8	3.5-6.57	3.2-4.03
Lymphocytes, $\times 10^9$/L	1.33 ± 0.09 (0.52-2.29)	5.52 ± 1.36 (1.75-7.53)	1.43 ± 0.10 (0.48-2.36)	37.54 ± 12.98%	1.1-5.13	1.7-4	1.2-1.56
Monocytes, $\times 10^9$/L	0.21 ± 0.03 (0.04-0.64)	0.25 ± 0.03 (0.12-0.62)	0.42 ± 0.05 (0.03-0.9)	3.72 ± 2.50%	0-0.9	0-0.85	0-0.5
Eosinophils, $\times 10^9$/L	0	2.3 ± 0.9 (1-4.77)	0.27 ± 0.04 (0.0-0.68)	0.20%	0-0.2	0-0.2	0-0.15
Basophils, $\times 10^9$/L	0.08 ± 0.01 (0-0.32)	—	0.05 + 0.02 (0.0-0.29)	0.0	0-0.45	0.17-0.83	0-0.15
Thrombocytes, $\times 10^9$/L	0.41 ± 0.03 (0.17-0.76)	2.97 ± 1.2 (1.25-7.15)	22.57 ± 1.04 (12.67-29. 93)	—	5-40	12-35	—
Fibrinogen, g/L	2.82 ± 0.14 (1.78-4.7)	—	3.61 ± 0.21 (1.72-5.63)	—	<4	<4	<4

Source: Samour J, John SK, Naldo JL: Normal haematology values in gyrfalcons (*Falco rusticolus*) in Saudi Arabia, *The Veterinary Record* 157:844, 2005.

*Samour JH, D'Aloia M-A, Howlett JC: Normal haematology of captive saker falcons (*Falco cherrug*), *Comparative Haematology International* 6:50–52, 1996.

†Dötlinger HS, Bird DM: Haematological parameters in captive peregrine falcons (*Falco peregrinus*). In: *Falco Newsletter 4*, United Arab Emirates, 1995, Middle East Falcon Research Group, National Avian Research Centre.

‡Mean ± SD: Wernery et al: Colour Atlas of Falcon Medicine, Hannover, 2004, Schlütersche, pp 12–36.

§Range: Jennings IB: Haematology. In: Beynon PH, Forbes NA, Harcourt-Brown NH, editors: *Manual of Raptors, Pigeons and Waterfowl*, Cheltenham, UK, 1996, British Small Animal Veterinary Association, pp 68–78

**Mean ± standard error of mean (maximum-minimum).

Hb, Hemoglobin; Hct, hematocrit; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Reference Hematologic Values (Mean ± SD) in Nestling (<105 Days Old, *n* = 21), Immature (3-6 yr Old, *n* = 14), Adult (>6 yr Old, *n* = 12), and Free-Living (Immature and Adult Pooled together, *n* = 26) Bearded Vultures

Parameter	Nestling	*n*	Immature	*n*	Adult	*n*	Free-living	*n*
Red blood cells ($\times10^{12}$/L)*	1.56 ± 0.30 (1.09-1.89)	12	2.99 ± 0.34 (2.45-3.65)	14	2.91 + 0.25 (2.34-3.26)	12	2.95 ± 3 (2.34-3.65)	26
White blood cells ($\times10^{9}$/L)*	6.90 ± 3.19 (3.20-15.80)	12	10.55 ± 2.19 (7.14-14.99)	14	9.72 + 2.18 (7.06-15.42)	12	10.17 ± 2.18 (7.06-15.42)	26
PCV (%)*	32.08 ± 5.0 (24-38.7)	12	47.21 ± 2.90 (43.5-51.8)	14	46.98 ± 4.02 (41-54.2)	12	47.1 ± 3.39 (41-54.2)	26
Hemoglobin (g/dL)*	8.54 ± 2.0 (6.3-12.2)	12	16.54 +1.15 (14.4-18.2)	14	16.41 ± 1.61 (14.4-18.6)	12	16.48 ± 1.36 (14.4-18.6)	26
Total plasma solids (g/dL)	4.26 + 0.54 (3.60-5.50)	12	4.30 ± 0.59 (3.50-5.8)	14	4.68 ± 0.43 (3.90-5.30)	12	4.47 ± 0.55 (3.5-5.8)	25
Fibrinogen (g/dL)*	0.45 ± 0.16 (0.22-0.68)	12	0.89 ± 0.19 (0.45-1.2)	14	0.75 ± 0.20 (0.50-1.10)	12	824.81 ± 204.0 (450-1,200)	26
Mean cell volume (fL)*	206.75 ± 16.3 (175.7-229.2)	12	159.35 + 16.77 (139.9-190.9)	14	162.06 ± 12.35 (137.6-180.9)	12	160.60 ± 14.67 (137.6-190.9)	26
Mean cell hemoglobin (pg)	55.26 ± 10.9 (33.9-72.3)	12	55.93 ± 7.25 (46.2-71.7)	14	56.69 ± 5.91 (48.5-64.8)	12	56.28 ± 6.55 (46.2-71.7)	26
Mean cell hemoglobin concentration (g/L)*	20.67 ± 1.6 (17.6-22.9)	12	35.08 ± 2.29 (32.1-38.4)	14	35.0 ± 2.82 (29.2-40.6)	12	35.04 ± 2.49 (29.2-40.6)	26
Heterophils ($\times10^{9}$/L)*	5.04 ± 0.26 (2.50-10.74)	12	7.50 ± 1.54 (5.28-10.49)	14	6.77 ± 1.91 (4.94-12.08)	12	7.16 ± 1.73 (4.94-12.08)	26
Lymphocytes ($\times10^{9}$/L) *	1.81 ± 1.01 (0.512-4.11)	12	2.52 ± 0.93 (1.13-4.50)	14	2.29 ± 0.54 (1.62-3.35)	12	2.41 ± 0.77 (1.13-4.5)	26
Eosinophils ($\times10^{9}$/L)	0.37 ± 0.25 (0-0.85)	12	0.25 ± 0.22 (0-0.60)	14	0.30 ± 0.27 (0-0.93)	12	0.28 ± 2.39 (0-0.93)	26
Monocytes ($\times10^{9}$/L)	0.12 ± 0.25 (0-0.86)	12	0.19 ± 0.20 (0-0.68)	14	0.37 ± 0.38 (0-1.11)	12	0.27 ± 1.39 (0-1.11)	26
Basophils ($\times10^{9}$/L)	0.04 ± 0.09 (0-0.39)	12	0.06 ± 0.19 (0-0.57)	14	0.05 ± 0.12 (0-0.39)	12	0.06 ± 0.14 (0-0.57)	26

Source: Hernandez M, Margalida A: Hematology and blood chemistry reference values and age-related changes in wild bearded vultures, *J Wildl Dis* 46(2):390–400, 2010.
Ranges in parentheses.
*Values significantly different between nestlings and immatures and adults (analysis of variance, $P < 0.05$) and between nestlings and free-living bearded vultures (*t*-test, $P < 0.05$).
PCV, Packed cell volume.

Reference Hematology Intervals for the Captive White-Backed Vultures (*n* = 21)

Parameter	Mean	SD	Min	Max	Reference Interval
Hb (g/L)	196.60	16.83	142.00	241.00	162.94-230.25
RBC ($\times10^{12}$/L)	2.55	0.22	2.01	3.03	2.11-2.98
HCT (L/L)	0.50	0.04	0.38	0.60	0.42-0.58
MCV (fL)	196.70	5.55	182.00	211.00	185.60-207.81
MCHC (g/dL)	39.30	1.53	35.40	42.00	36.25-42.35
WBC ($\times10^{9}$/L)	16.71	1.63	4.00	34.00	13.45-19.97
Het (mat) ($\times10^{9}$/L)	13.70	6.11	3.04	27.60	1.48-25.93
Het (immat; band) ($\times10^{9}$/L)	0.00	0.00	0.00	0.00	0.00-0.00
Lymph ($\times10^{9}$/L)	1.02	1.91	0.00	12.55	0.00-4.84
Mono ($\times10^{9}$/L)	1.57	1.04	0.10	5.78	0.00-3.65
Eos ($\times10^{9}$/L)	0.75	0.71	0.00	3.68	0.00-2.16
Bas ($\times10^{9}$/L)	0.00	0.00	0.00	0.00	0.00-0.00
PCV (%)	44.30	4.79	31.00	53.00	34.72-53.88

Source: Naidoo V, Diekmann M, Wolters K, Swan GE: Establishment of selected baseline blood chemistry and hematologic parameters in captive wild-caught African white-backed vultures (*Gyps africanus*), *J Wildl Dis* 44(3):649–654, 2008.
Bas, basophils; Hb, Hemoglobin; Hct, hematocrit; MCV, mean corpuscular volume; MCHC, mean corpuscular hemoglobin concentration; Het (mat), mature heterophils; Het (immat), immature heterophils; Lymph, lymphocytes; Mono, monocytes; Eos, eosinophils; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Selected Pelecaniformes

Species	American White Pelican *n* = 10	Brown Pelican *n* = 5
Scientific Name	***Pelecanus erythrorhynchos***	***Pelecanus occidentalis***
RBC, $\times 10^{12}$/L	2.3 ± 0.3 (1.9-2.7)	2.7 (2.6-2.8)
Hb, g/dL	13.0 ± 1.8 (9.8-16.6)	14.5 (14.3-14.8)
Hct, L/L	0.39 ± 0.04 (0.33-0.45)	0.46 (0.43-0.49)
MCV, fL	166 ± 9 (152-182)	168 (166-173)
MCH, pg	52.3 ± 2.9 (46.0-59.3)	53.4 (51.2-56.8)
MCHC, g/dL	32.6 ± 2.0 (28.4-35.2)	31.7 (30.4-32.9)
WBC, $\times 10^9$/L	9.5 ± 3.4 (5.0-15.0)	11.9 (6.6-19.4)
Heterophils, $\times 10^9$/L	7.2 ± 3.0 (4.2-9.3)	6.7 (4.0-9.5)
Lymphocytes, $\times 10^9$/L	3.4 ± 0.7 (2.7-4.5)	4.0 (2.5-7.0)
Monocytes, $\times 10^9$/L	(0.0-0.2)	(0.0-0.20)
Eosinophils, $\times 10^9$/L	(0.0-0.3)	(0.0-0.2)
Basophils, $\times 10^9$/L	(0.1-1.6)	(0.0-0.2)
Thrombocytes, $\times 10^9$/L	29 ± 6 (21-38)	(17-38)
Fibrinogen, g/L	0.9 ± 0.4 (0.3-1.5)	2.9 (2.6-3.1)

Source: Hawkey CM, Samour JH: The value of clinical hematology in exotic birds. In Jacobson ER, Kollias GV Jr, editors: *Exotic animals: contemporary issues in small animal practice*, London, 1988, Churchill Livingstone, pp 109–141.
Mean ± standard error of mean (minimum-maximum).
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Selected Sphenisciformes

Species	Gentoo Penguin, *n* = 9-20	King Penguin, *n* = 6-7	Rockhopper Penguin, *n* = 11-17	Black-footed Penguin, *n* = 12-42	Humboldt Penguin, *n* = 10-24
Scientific Name	***Pygoscelis papua***	***Aptenodytes patagonica***	***Eudyptes crestatus***	***Spheniscus demersus***	***Spheniscus humboldti***
RBC, $\times 10^{12}$/L	1.61 ± 0.14 (1.39-1.76)	1.58 ± 0.30 (1.18-2.02)	1.94 ± 0.20 (1.69-2.30)	1.74 ± 0.20 (1.32-2.12)	2.05 ± 0.31 (1.46-2.46)
Hb, g/dL	15.7 ± 1.9 (12.7-19.2)	16.9 ± 4.2 (10.2-22.0)	17.9 ± 1.1 (16.1-19.2)	16.8 ± 1.6 (13.4-19.5)	17.6 ± 2.5 (14.5-21.3)
Hct, L/L	0.45 ± 0.04 (0.37-0.51)	0.45 ± 0.08 (0.32-0.55)	0.46 ± 0.03 (0.41-0.50)	0.44 ± 0.04 (0.36-0.51)	0.48 ± 0.04 (0.41-0.54)
MCV, fL	258 ± 31 (215-301)	288 ± 18 (270-301)	234 ± 18 (210-267)	254 ± 11 (232-273)	228 ± 17 (194-267)
MCH, pg	95.0 ± 8.8 (81.4-110.5)	108.0 ± 12.5 (86.4-120.9)	91.7 ± 7.7 (80.3-104.0)	95.1 ± 4.5 (87.2-104.3)	85.3 ± 5.2 (76.0-94.3)
MCHC, g/dL	37.7 ± 1.7 (34.8-40.0)	37.5 ± 3.8 (31.9-41.5)	39.1 ± 0.8 (37.5-40.0)	37.8 ± 1.4 (35.4-40.0)	38.0 ± 1.7 (35.0-40.6)
WBC, $\times 10^9$/L	8.2 ± 4.1 (3.2-16.1)	4.3 ± 1.4 (2.8-6.7)	4.7 ± 1.4 (3.0-7.7)	9.3 ± 3.5 (3.5-16.3)	15.9 ± 5.1 (5.6-25.8)
Heterophils, $\times 10^9$/L	3.9 ± 1.3 (2.2-6.0)	2.3 ± 1.0 (1.4-3.9)	3.6 ± 1.1 (1.5-5.2)	8.1 ± 2.3 (5.0-12.3)	11.9 ± 4.5 (4.1-17.9)
Lymphocytes, $\times 10^9$/L	2.2 ± 1.0 (0.6-4.8)	2.0 ± 0.6 (1.3-2.8)	1.6 ± 0.8 (0.3-2.5)	3.1 ± 1.4 (0.8-5.2)	2.7 ± 1.8 (1.0-5.0)
Monocytes, $\times 10^9$/L	0	0	<0.1 (0.0-0.1)	0	<0.5 (0.0-0.5)
Eosinophils, $\times 10^9$/L	0	0	<0.3 (0.0-0.3)	<0.1 (0.0-0.2)	<0.2 (0.0-0.02)
Basophils, $\times 10^9$/L	0	0	<0.1 (0.0-0.1)	<0.1 (0.0-0.3)	<0.5 (0.0-0.5)
Thrombocytes, $\times 10^9$/L	—	—	7.3 ± 4.2 (4-15)	11 ± 5 (5-19)	9.5 ± 4.9 (7-20)
Fibrinogen, g/L	3.2 ± 0.8 (2.1-4.2)	2.4	2.9 ± 0.4 (2.2-2.7)	2.9 ± 0.4 (2.2-3.7)	3.5 ± 0.9 (2.0-5.3)

Source: Hawkey CM, Samour JH: The value of clinical hematology in exotic birds. In Jacobson ER, Kollias GV Jr, editors: *Exotic animals: contemporary issues in small animal practice*, New York, 1988, Churchill Livingstone, pp 109–141.
Mean ± standard error of mean (minimum-maximum).
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Selected Ciconiiformes

Species	Rosy Flamingo, *n* = 36	Chilean Flamingo, *n* = 24	Lesser Flamingo, *n* = 10	Greater Flamingo, *n* = 9
Scientific Name	***Phoenicopterus Ruber ruber***	***Phoenicopterus chilensis***	***Phoeniconaias minor***	***Phoenicopterus rube***
RBC, $\times 10^{12}$/L	2.6 ± 0.2 (2.3-3.0)	2.7 ± 0.2 (2.4-3.0)	2.7 ± 0.1 (2.3-2.9)	2.6 (2.3-2.8)
Hb, g/dL	17.1 ± 1.4 (13.8-19.1)	16.2 ± 1.1 (14.1-18.1)	16.8 ± 1.6 (15.2-19.5)	17.3 (15.9-19.6)
Hct, L/L	0.47 ± 0.04 (0.40-0.54)	0.46 ± 0.03 (0.41-0.51)	0.51 ± 0.03 (0.46-0.54)	0.50 (0.47-0.57)
MCV, fL	184 ± 7 (168-196)	171 ± 6 (161-183)	188 ± 6 (179-195)	193 (170-207)
MCH, pg	66.4 ± 4.0 (59.0-73.5)	60.6 ± 2.1 (57.3-64.8)	62.0 ± 5.4 (55.4-70.4)	66.2 (57.6-70.0)
MCHC, g/dL	35.9 ± 2.3 (31.7-39.8)	35.6 ± 0.9 (33.3-37.9)	33.0 ± 2.5 (30.8-37.5)	34.4 (33.5-35.2)
WBC, $\times 10^9$/L	5.1 ± 1.7 (2.4-8.7)	4.9 ± 2.5 (1.6-9.0)	61 ± 2.0 (3.8-8.5)	2.4 (0.9-3.4)
Heterophils, $\times 10^9$/L	2.4 ± 0.9 (1.0-4.4)	2.4 ± 1.7 (0.4-4.8)	4.6 ± 1.8 (1.7-6.9)	1.2 (0.2-3.0)
Lymphocytes, $\times 10^9$/L	1.6 ± 0.6 (0.7-3.0)	1.8 ± 0.6 (0.8-2.7)	1.2 ± 0.6 (0.5-2.4)	0.9 (0.4-1.6)
Monocytes, $\times 10^9$/L	(0.0-0.5)	0	(0.0-0.4)	(0.0-0.2)
Eosinophils, $\times 10^9$/L	(0.0-0.6)	(0.0-0.7)	0	(0.0-0.4)
Basophils, $\times 10^9$/L		(0.0-0.4)	(0.0-0.3)	(0.0-0.4)
Thrombocytes, $\times 10^9$/L	14 ± 6 (4-29)	15 ± 6 (6-33)	16 ± 8 (3-23)	4 (2-7)
Fibrinogen, g/L	2.7 ± 0.6 (1.7-3.7)	2.3 ± 0.6 (1.3-3.6)	2.3 ± 0.4 (1.4-2.9)	2.6 (1.5-3.3)

Source: Hawkey CM, Samour JH: The value of clinical hematology in exotic birds. In Jacobson ER, Kollias GV Jr, editors: *Exotic animals: contemporary issues in small animal practice*, New York, 1988, Churchill Livingstone, pp 109–141.
Mean ± standard error of mean (minimum-maximum).
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Selected Ciconiiformes—cont'd

Species	Night Heron *n* = 22	Marabou Stork *n* = 8	White Stork *n* = 16	Maguari Stork *n* = 5	Australian White Ibis *n* = 8	Scarlet Ibis *n* = 15
Scientific Name	***Nycticorax nycticorax***	***Leptoptilos crumeniferus***	***Ciconia ciconia***	***Ciconia maguari***	***Threskiornis M. molucca***	***Eudocimus ruber***
RBC, ×1012/L	2.6 ± 0.2 (2.3-2.9)	2.3 ± 0.1 (2.2-2.5)	2.4 ± 0.1 (2.1-2.7)	2.3 (2.2-2.7)	3.0 ± 0.2 (2.6-3.3)	3.2 ± 0.3 (2.6-3.8)
HB, g/dL	15.1 ± 1.4 (12.9-17.8)	15.8 ± 0.9 (14.8-17.1)	15.9 ± 1.1 (14.4-17.7)	16.1 (13.8-17.8)	18.2 ± 1.8 (14.6-19.5)	15.3 ± 1.0 (13.3-17.1)
Hct, L/L	0.45 ± 0.03 (0.40-0.50)	0.47 ± 0.04 (0.42-0.52)	0.45 ± 0.03 (0.41-0.48)	0.46 (0.42-0.50)	0.48 ± 0.04 (0.39-0.51)	0.49 ± 0.05 (0.41-0.53)
MCV, fL	178 ± 8 (162-194)	202 ± 12 (175-212)	189 ± 6 (172-195)	195 (186-210)	159 ± 7 (150-170)	153 ± 7 (142-164)
MCH, pg	59.5 ± 3.4 (53.2-64.0)	67.3 ± 3.4 (62.1-70.9)	67.2 ± 2.9 (60.2-69.9)	69.0 (61.3-75.7)	60.5 ± 3.6 (55.5-67.2)	48.8 ± 2.9 (45.2-53.4)
MCHC, g/dL	33.6 ± 1.7 (30.3-35.8)	33.5 ± 1.8 (31.1-35.9)	35.3 ± 1.5 (31.0-36.9)	35.3 (32.9-36.2)	38.1 ± 1.1 (36.8-39.4)	31.5 ± 1.3 (29.2-33.7)
WBC, $\times 10^9$/L	9.9 ± 2.8 (5.8-15.2)	19.5 ± 4.1 (14.4-23.3)	10.8 ± 3.1 (7.0-14.3)	9.8 (7.2-15.5)	6.7 ± 2.8 (2.1-10.0)	7.1 ± 3.2 (2.6-12.6)
Heterophils, $\times 10^9$/L	7.1 ± 2.4 (3.7-11.5)	12.7 ± 4.1 (7.6-18.7)	9.2 ± 3.2 (5.1-14.9)	5.9 (2.0-11.5)	5.7 ± 2.6 (1.4-8.8)	4.5 ± 2.5 (1.6-8.5)
Lymphocytes, $\times 10^9$/L	2.5 ± 0.9 (1.4-4.2)	4.1 ± 0.9 (2.5-5.3)	0.8 ± 0.4 (0.2-1.6)	2.6 (1.4-3.3)	1.0 ± 0.3 (0.5-1.5)	2.0 ± 0.7 (0.8-3.0)
Monocytes, $\times 10^9$/L	(0.0-0.9)	(0.0-2.3)	(0.0-0.3)	(0.0-0.7)	0	0
Eosinophils, $\times 10^9$/L	(0.0-1.1)	(0.2-4.1)	(0.0-0.7)	(0.7-2.2)	(0.0-0.3)	(0.0-0.8)
Basophils, $\times 10^9$/L	0	(0.0-0.7)	(0.0-0.5)	(0.0-0.8)	(0.0-0.3)	(0.0-0.7)
Thrombocytes, $\times 10^9$/L	16 ± 4 (8-5)	16 ± 3 (12-19)	19 ± 8 (8-32)	10 (8-11)	33 ± 12 (18-48)	22 ± 8 (11-35)
Fibrinogen, g/L	1.8 ± 0.6 (1.1-3.1)	3.2 ± 0.6 (2.6-4.4)	2.3 ± 0.4 (1.7-3.2)	1.7 (1.3-2.1)	2.3 ± 0.3 (1.9-2.7)	2.6 ± 0.6 (1.9-3.7)

Source: Hawkey CM, Samour JH: The value of clinical hematology in exotic birds. In Jacobson ER, Kollias GV Jr, editors: *Exotic animals: contemporary issues in small animal practice*, New York, 1988, Churchill Livingstone, pp 109–141.
Mean ± standard error of mean (minimum-maximum).
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Selected Strigiformes

Species	African Eagle Owl *n* = 6	European Eagle *n* = 14	Spectacled Owl *n* = 4	Tawny Owl *n* = 14	Boobook Owl *n* = 5	Barn Owl *n* = 10
Scientific Name	***Bubo africanus***	***Bubo bubo***	***Pulsatrix perspicillata***	***Strix aluco***	***Ninox novaeseelandiae***	***Tyto alba***
RBC, $\times 10^{12}$/L	2.4 (2.1-2.8)	1.9 ± 0.2 (1.4-2.3)	1.6 (1.4-1.8)	2.5 ± 0.2 (2.0-2.9)	2.5 (2.4-2.9)	2.7 ± 0.3 (2.2-3.0)
Hb, g/dL	15.8 (13.9-19.9)	14.2 ± 1.5 (11.7-16.8)	14.2 (12.4-16.3)	14.6 ± 1.1 (12.9-16.4)	15.1 (14.4-15.9)	14.2 ± 1.5 (12.7-16.4)
Hct, L/L	0.45 (0.41-0.53)	0.39 ± 0.04 (0.31-0.45)	0.42 (0.37-0.45)	0.40 ± 0.03 (0.36-0.47)	0.42 (0.40-0.45)	0.46 ± 0.03 (0.42-0.51)
MCV, fL	189 (171-214)	207 ± 17 (178-239)	261 (245-267)	158 ± 9 (147-177)	172 (165-175)	176 ± 22 (145-216)
MCH, pg	66.4 (58.9-76.2)	75.1 ± 8.1 (67.1-87.1)	87.8 (86.1-89.1)	56.8 ± 4.8 (49.8-66.6)	61.5 (60.8-61.6)	51.1 ± 5.7 (44.9-60.7)
MCHC, g/dL	35.1 (33.9-36.6)	36.3 ± 2.0 (33.8-38.4)	33.7 (32.3-36.2)	36.3 ± 0.9 (34.9-38.0)	36.0 (34.8-37.3)	31.8 ± 2.2 (28.9-34.9)
WBC, $\times 10^9$/L	6.2 (4.7-8.0)	10.8 ± 4.0 (5.3-18.6)	9.6 (6.9-11.1)	6.7 ± 3.3 (2.4-11.8)	6.4 (3.7-11.2)	16.6 ± 4.2 (11.5-22.3)
Heterophils, $\times 10^9$/L	3.0 (1.3-5.2)	6.9 ± 3.2 (2.6-11.8)	4.9 (2.8-7.6)	3.4 ± 2.0 (1.1-7.2)	4.6 (2.3-9.1)	8.9 ± 3.0 (5.2-12.5)
Lymphocytes, $\times 10^9$/L	2.3 (1.9-3.2)	3.8 ± 1.9 (1.9-6.7)	4.3 (2.7-7.3)	3.3 ± 1.4 (0.9-5.1)	1.4 (0.9-1.7)	5.0 ± 1.7 (2.5-7.5)
Monocytes, $\times 10^9$/L	0	0	0	(0.0-0.3)	(0.0-0.5)	(0.0-1.0)
Eosinophils, $\times 10^9$/L	(0.0-1.0)	(0.0-1.6)	(0.0-0.6)	(0.0-1.9)	(0.0-0.5)	(0.0-2.5)
Basophils, $\times 10^9$/L	(0.0-0.6)	(0.0-0.6)	(0.0-0.4)	(0.0-0.9)	(0.0-0.2)	(0.0-0.9)
Thrombocytes, $\times 10^9$/L	22 (14-29)	15 ± 3 (9-17)	18	17 ± 5 (10-24)	-	33 ± 15 (14-58)
Fibrinogen, g/L	5.2 (3.6-7.7)	3.3 ± 0.9 (1.4-5.0)	7.0 (6.4-8.8)	3.6 ± 0.7 (2.6-5.3)	2.8 (1.6-3.8)	2.7 ± 0.5 (1.9-3.3)

Source: Hawkey CM, Samour JH: The value of clinical hematology in exotic birds. In Jacobson ER, Kollias GV Jr, editors: *Exotic animals: contemporary issues in small animal practice*, New York, 1988, Churchill Livingstone, pp 109–141.
Mean ± standard error of mean (minimum-maximum).
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Selected Psittaciformes

Species	Bare-eyed Cockatoo n = 11	Greater Sulfur-Crested Cockatoo n = 10-15	Roseate Cockatoo n = 24-31	African Gray Parrot n = 11	Amazon Green Parrot n = 15	Kea n = 8
Scientific Name	***Cacatua sanguinea***	***Cacatua galerita***	***Eolophus roseicapillus***	***Psittacus erithacus***	***Amazona* sp.**	***Nestor notabilis***
RBC, $\times 10^{12}$/L	2.9 ± 0.3 (2.5-3.4)	2.7 ± 0.2 (2.4-3.0)	3.6 ± 0.2 (3.1-3.9)	3.3 ± 0.2 (3.0-3.6)	2.9 ± 0.3 (2.6-3.5)	2.6 ± 0.3 (2.3-3.1)
Hb, g/dL	17.0 ± 1.2 (15.4-19.0)	15.7 ± 1.0 (13.8-17.1)	16.7 ± 1.3 (14.0-18.8)	15.5 ± 1.0 (14.2-17.0)	15.5 ± 1.3 (13.8-17.9)	13.4 ± 2.2 (10.6-16.9)
Hct, L/L	0.53 ± 0.04 (0.47-0.60)	0.45 ± 0.03 (0.41-0.49)	0.54 ± 0.03 (0.49-0.60)	0.48 ± 0.03 (0.43-0.51)	0.51 ± 0.03 (0.44-0.56)	0.40 ± 0.04 (0.34-0.46)
MCV, fL	188 ± 11 (181-200)	165 ± 9 (145-187)	149 ± 8 (136-164)	145 ± 6 (137-155)	173 ± 11 (156-194)	154 ± 17 (137-186)
MCH, pg	60.5 ± 2.4 (56.6-63.1)	57.6 ± 2.1 (53.8-60.6)	45.9 ± 2.9 (43.5-51.3)	47.2 ± 3.0 (41.9-52.8)	52.6 ± 5.0 (44.7-58.6)	51.2 ± 9.0 (41.6-68.1)
MCHC, g/dL	32.1 ± 2.4 (28.7-36.9)	34.9 ± 1.3 (33.3-37.6)	31.0 ± 1.7 (27.5-33.9)	32.5 ± 2.0 (28.9-34.0)	31.6 ± 2.8 (28.9-35.8)	33.2 ± 2.6 (30.4-37.0)
WBC, $\times 10^9$/L	7.3 ± 2.8 (4.2-11.8)	6.4 ± 2.9 (1.4-10.7)	6.3 ± 3.1 (1.6-11.9)	7.0 ± 2.3 (3.3-10.3)	4.6 ± 1.4 (2.3-6.5)	16.0 ± 3.8 (12.1-22.6)
Heterophils, $\times 10^9$/L	5.2 ± 2.7 (2.8-10.6)	3.7 ± 1.7 (1.0-6.6)	4.6 ± 2.5 (0.6-9.2)	4.9 ± 1.7 (1.8-7.3)	2.9 ± 0.7 (1.6-3.8)	13.8 ± 3.5 (9.4-20.1)
Lymphocytes, $\times 10^9$/L	1.5 ± 0.9 (0.5-3.9)	1.9 ± 0.8 (1.0-3.6)	1.2 ± 0.5 (0.5-2.0)	1.4 ± 0.4 (0.7-2.1)	1.7 ± 0.8 (0.6-2.8)	1.9 ± 0.6 (1.1-2.7)
Monocytes, $\times 10^9$/L	(0.0-0.5)	(0.0-0.2)	(0.0-0.1)	(0.0-0.3)	(0.0-0.1)	0
Eosinophils, $\times 10^9$/L	(0.0-0.7)	(0.0-0.2)	(0.0-0.2)	0	(0.0-0.1)	(0.0-0.5)
Basophils, $\times 10^9$/L	(0.0-0.8)	(0.0-0.9)	(0.0-0.8)	(0.0-0.8)	(0.0-0.2)	(0.0-0.6)
Thrombocytes, $\times 10^9$/L	12 ± 7 (5-24)	13 ± 7 (7-24)		22 ± 9 (11-42)	32 ± 12 (10-67)	16 ± 5 (11-24)
Fibrinogen, g/L	2.0 ± 0.4 (1.5-2.8)	1.4 ± 0.3 (0.9-2.0)	1.8 ± 0.7 (0.8-3.5)	2.2 ± 0.5 (1.5-2.8)	2.2 ± 0.5 (1.4-3.0)	1.5 ± 0.2 (1.1-1.8)

Source: Hawkey CM, Samour JH: The value of clinical hematology in exotic birds. In Jacobson ER, Kollias GV Jr, editors: *Exotic animals: contemporary issues in small animal practice*, New York, USA, 1988, Churchill Livingstone, pp 109–141.
Mean ± standard error of mean (minimum-maximum).
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Selected Psittaciformes—cont'd

Species	White Cockatoo *n* = 5	Black Cockatoo *n* = 6	Palm Cockatoo *n* = 12	Golden Conure *n* = 7	Patagonian Conure *n* = 6
Scientific Name	***Cacatua alba***	***Calyptorhynchus funereus***	***Probosciger aterrimus***	***Aratinga guarouba***	***Cyanoliseus patagonus***
RBC, $\times 10^{12}$/L	2.98 ± 0.19 (2.75-3.20)	2.60 ± 0.10 (2.42-2.72)	2.62 ± 0.53 (1.96-3.59)	3.85 ± 0.19 (3.61-4.03)	3.54 ± 0.32 (3.16-4.09)
Hb, g/dL	15.7 ± 1.9 (13.9-18.4)	14.5 ± 1.6 (12.0-17.0)	14.6 ± 1.3 (12.8-16.7)	18.8 ± 1.0 (17.6-20.4)	15.0 ± 0.8 (14.3-16.2)
PCV, %	44.8 ± 4.4 (37.0-48.0)	43.0 ± 2.5 (40.0-46.0)	44.0 ± 3.6 (36.3-47.3)	52.3 ± 1.3 (50.0-54.0)	47.6 ± 3.1 (45.0-52.0)
MCV, fL	150.7 ± 14.3 (132.1-171.0)	165.6 ± 11.5 (154.2-183.6)	174.3 ± 32.9 (130.9-235.0)	136.1 ± 6.1 (125.9-143.7)	134.6 ± 7.1 (127.2-145.6)
MCH, pg	52.4 ± 10.2 (43.4-67.1)	55.8 ± 5.7 (48.5-62.7)	57.4 ± 8.7 (46.1-74.1)	49.0 ± 3.1 (45.1-54.4)	42.5 ± 2.4 (39.7-46.8)
MCHC, g/dL	33.5 ± 4.0 (30.0-39.2)	33.6 ± 1.9 (31.5-37.0)	33.1 ± 1.8 (31.1-35.5)	36.0 ± 2.5 (33.9-40.7)	31.6 ± 0.5 (30.9-32.3)
WBC, $\times 10^9$/L	6.7 ± 7.5 (1.3-18.7)	10.7 ± 6.5 (93.7-22.1)	6.5 ± 5.4 (1.4-17.60	6.5 ± 1.6 (4.2-8.0)	5.8 ± 2.1 (2.5-8.7)
Heterophils, %	45.1 ± 28.5 (17.6-83.0)	32.4 ± 20.9 (6.6-61.2)	53.9 ± 16.8 (23.6-75.0)	38.3 ± 8.7 (22.2-48.5)	40.7 ± 13.7 (23.5-62.7)
Lymphocytes, %	52.7 ± 27.5 (15.0-80.3)	63.1 ± 22.1 (32.7-89.5)	42.5 ± 15.3 (24.0-69.4)	57.7 ± 6.8 (48.6-68.7)	54.3-10.7 (34.7-65.8)
Monocytes, %	1.8 ± 1.6 (0.0-3.7)	4.1 ± 1.5 (2.7-6.7)	3.0 ± 2.2 (1.0-7.0)	2.5 ± 0.6 (1.4-3.1)	0.9 ± 1.1 (0.0-2.5)
Eosinophils, %	0.2 ± 0.4 (0.0-1.0)	0.0	0.5 ± 0.6 (0.0-1.4)	0.5 ± 0.8 (0.0-2.0)	0.2 ± 0.4 (0.0-1.1)
Basophils, %	0.2 ± 0.4 (0.0-1.0)	0.4 ± 0.9 (0.0-2.0)	0.2 ± 0.5 (0.0-1.4)	0.0	0.0

Source: modified from: Polo FJ, Peinado VI, Viscor G, Palomeque J: Hematologic and plasma chemistry in captive psittacine birds, *Avian Diseases* 42:523–535, 1998.
Mean ± SD (minimum-maximum).
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Selected Psittaciformes—cont'd

Species	Yellow-Naped Macaw *n* = 7	Red-Fronted Macaw *n* = 7	Severe Macaw *n* = 4	Scarlet Macaw *n* = 14	Green Winged Macaw *n* = 18	Military Macaw *n* = 21	Hyacinthine Macaw *n* = 16	Blue and Yellow Macaw, *n* = 35
Scientific Name	***Ara auricollis***	***Ara rubro genys***	***Ara severa***	***Ara macao***	***Ara chloroptera***	***Ara militaris***	***Anodorhynchus hyacinthinus***	***Ara ararauna***
RBC, $\times 10^{12}$/L	3.5 (2.9-3.9)	3.3 (2.6-3.5)	3.3 (3.1-3.6)	3.07 ± 0.43 (2.29-3.67)	3.20 ± 0.38 (2.65-4.05)	3.44 ± 0.59 (2.72-5.16)	3.19 ± 0.44 (2.45-4.260)	3.24 ± 0.50 (2.11-4.10)
Hb, g/dL	17.4 (15.0-19.2)	16.4 (14.4-19.6)	16.7 (16.0-17.1)	16.4 ± 2.2 (13.1-19.90)	13.6 ± 2.8 (9.6-18.7)	15.6 ± 2.3 (11.1-19.6)	15.8 ± 0.9 (14.3-18.0)	14.4 ± 1.2 (11.7-17.0)
Hct, L/L	0.50 (0.43-0.53)	0.46 (0.41-0.51)	0.49 (0.45-0.53)	47.4 ± 4.4 (40.0-54.0)	46.1 ± 3.8 (39.0-54.0)	47.1 ± 4.3 (37.0-54.5)	46.9 ± 2.5 (43.0-51.0)	44.6 ± 4.6 (31.5-51.8)
MCV, fL	144 (133-150)	142 (136-158)	149 (145-156)	151.6 ± 10.7 (135.3-168.5)	145.0 ± 13.9 (116.1-176.5)	137.0 ± 16.0 (105.6-172.8)	149.0 ± 17.8 (119.1-180.6)	141.0 ± 21.4 (102.4-199.1)
MCH, pg	50.6 (48.0-53.8)	50.8 (40.6-59.6)	51.4 (47.9-55.2)	52.1 ± 3.8 (44.3-58.5)	42.2 ± 8.1 (31.3-61.1)	44.9 ± 4.7 (36.5-51.8)	50.3 ± 6.6 (38.7-62.8)	46.4 ± 6.5 (34.6-61.9)
MCHC, g/dL	35.0 (32.9-36.9)	35.3 (29.3-38.4)	34.5 (32.1-35.6)	34.4 ± 2.2 (29.7-37.3)	29.6 ± 4.6 (21.9-34.9)	33.5 ± 2.5 (33.9-40.7)	33.8 ± 1.4 (32.3-36.8)	32.7 ± 3.4 (28.1-43.5)
WBC, $\times 10^{9}$/L	10.4 (5.0-16.9)	5.9 (3.0-8.1)	7.8 (4.2-10.2)	9.8 ± 4.5 (4.7-22.0)	16.9 ± 8.9 (3.8-30.0)	9.5 ± 4.5 (13.7–18.0)	9.7 ± 5.8 (1.5-19.2)	16.6 ± 9.0 (1.7-36.0)
Heterophils, %	8.2 (4.0-15.2)	4.1 (1.8-6.2)	5.2 (3.0-6.7)	39.9 ± 13.0 (26.0-67.0)	32.2 ± 13.4 (14.0-62.0)	41.5 ± 15.4 (12.0-62.5)	75.9 ± 12.1 (52.0-89.0)	37.2 ± 18.3 (12.8-60.0)
Lymphocytes, %	2.2 (0.9-4.8)	1.7 (1.0-2.6)	2.2 (0.5-3.6)	55.1 ± 11.4 (36.0-68.2)	34.0 ± 13.8 (35.0-84.2)	55.3 ± 14.5 (43.3-80.0)	30.3 ± 22.2 (10.0-77.3)	60.0 ± 17.6 (35.5-84.4)
Monocytes, %		0	(0.0-0.3)	3.4 ± 2.4 (0.0-8.1)	2.1 ± 2.2 (0.0-8.3)	2.4 ± 2.4 (0.0-8.0)	0.5 ± 0.6 (0.0-1.5)	1.3 ± 0.8 (0.0-2.0)
Eosinophils, %	0	0	(0.0-0.5)	1.2 ± 1.4 (0.0-4.0)	0.5 ± 0.9 (0.0-3.0)	0.3 ± 0.7 (0.0-2.1)	1.1 ± 1.4 (0.0-4.0)	0.7 ± 0.8 (0.0-2.0)
Basophils, %	(0.0-0.4)	(0.0-0.2)	(0.0-0.1)	0.4 ± 0.7 (0.0-2.0)	0.3 ± 0.5 (0.0-1.7)	0.2 ± 0.4 (0.0-1.2)	0.0	0.3 ± 0.6 (0.0-1.6)

Source: modified from: Polo FJ, Peinado VI, Viscor G, Palomeque J: Hematologic and plasma chemistry in captive psittacine birds, *Avian Diseases* 42:523–535, 1998.
Mean ± SD (minimum-maximum).
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Selected Psittaciformes—cont'd

Species	Blue-Fronted Amazon *n* = 7	Orange Winged Amazon *n* = 5	Yellow Amazon *n* = 8	Cuban Amazon *n* = 7	Festive Amazon *n* = 6	Red Lory *n* = 18
Scientific Name	***Amazona aestiva***	***Amazona amazonica***	***Amazona ochrocephala***	***Amazona leucocephala***	***Amazona festiva***	***Eos bornea***
RBC, $\times 10^{12}$/L	2.92 ± 0.55 (2.11-3.53)	3.08 ± 0.20 (2.81-3.32)	2.93 ± 0.16 (2.11-3.53)	3.30 ± 0.16 (3.09-3.49)	3.47 ± 0.29 (3.10-3.80)	3.17 ± 0.49 (2.62-4.72)
HB, g/dL	17.3 ± 0.9 (16.0-18.4)	16.3 ± 0.8 (15.5-17.5)	15.3 ± 1.7 (12.1-17.4)	16.7 ± 0.9 (15.2-17.7)	16.7 ± 0.5 (16.1-17.4)	16.0 ± 1.4 (14.2-18.7)
PCV, %	50.6 ± 6.2 (43.5-58.0)	48.4 ± 2.2 (46.0-51.0)	46.5 ± 4.4 (38.0-51.0)	49.6 ± 3.5 (44.0-54.0)	50.4 ± 2.6 (47.0-53.0)	48.8 ± 3.2 (44.0-54.0)
MCV, fL	180.0 ± 19.7 (163.0-208.9)	157.6 ± 5.9 (150.6–165.8)	159.0 ± 12.4 (135.4-175.4)	150.4 ± 7.3 (141.9–162.4)	145.8 ± 11.8 (134.8-163.7)	155.9 ± 13.7 (111.2-171.6)
MCH, pg	62.2 ± 9.9 (52.0-76.2)	53.1 ± 3.4 (48.3-57.3)	52.1 ± 4.9 (42.9-56.9)	50.8 ± 3.6 (44.8-55.2)	48.3 ± 4.5 (43.9-53.7)	51.7 ± 3.9 (39.6-55.7)
MCHC, g/dL	34.3 ± 2.6 (31.7-37.8)	33.7 ± 1.5 (32.1-36.0)	32.5 ± 1.0 (31.0-34.1)	33.8 ± 2.2 (31.4-37.2)	33.2 ± 1.2 (31.5-34.5)	32.9 ± 1.0 (31.2-35.6)
WBC, $\times 10^9$/L	6.5 ± 2.4 (4.7-11.0)	6.1 ± 3.8 (91.2-10.1)	4.2 ± 1.9 (2.2-7.7)	8.3 ± 7.7 (1.9-24.7)	4.1 ± 1.8 (2.2-7.0)	3.3 ± 2.2 (0.8-9.0)
Heterophils, %	30.7 ± 15.0 (12.4-46.6)	36.2 ± 7.2 (21.9-40.7)	30.6 ± 12.8 (12.3-51.9)	24.3 ± 3.3 (19.0-27.6)	25.1 ± 4.7 (21.9-32.2)	55.2 ± 17.4 (25.6-79.2)
Lymphocytes, %	67.0 ± 14.2 (52.4-83.5)	63.4 ± 7.0 (55.8-73.2)	67.2 ± 10.9 (48.1-80.0)	77.3 ± 1.3 (71.4-75.0)	71.0 ± 4.3 (65.5-75.8)	41.7 ± 16.6 (18.7-70.1)
Monocytes, %	1.7 ± 0.9 (1.0-3.1)	3.5 ± 1.5 (2.0-5.0)	1.9 ± 2.7 (0.0-7.7)	1.5 ± 2.1 (0.0-5.0)	2.4 ± 1.8 (0.0-4.2)	1.4 ± 1.2 (0.0-4.5)
Eosinophils, %	0.3 ± 0.5 (0.0-1.0)	1.0 ± 2.2 (0.0-5.0)	0.2 ± 0.4 (0.0-1.2)	1.5 ± 2.1 (0.0-5.0)	1.3 ± 0.9 (0.0-2.3)	1.5 ± 1.5 (0.0-4.6)
Basophils, %	0.2 ± 0.5 (0.0-1.0)	0.3 ± 0.7 (0.0-1.7)	0.2 ± 0.4 (0.0-1.2)	0.3 ± 0.4 (0.0-1.0)	0.0	0.2 ± 0.4 (0.0-1.2)

Source: Modified from: Polo FJ, Peinado VI, Viscor G, Palomeque J: Hematologic and plasma chemistry in captive psittacine birds, *Avian Diseases* 42:523–535, 1998.
Mean ± SD (minimum-maximum).
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; RBC, red blood cell; WBC, white blood cell.

Hematologic Reference Values in Captive Adult, Clinically Normal Indian Peafowl (n = 69)

Parameter	P2.5-P97.5*	Mean	SD	Range
Red blood cells ($\times 10^{12}$/L [$\times 10^6$/μL])	1.88-2.82	2.25	0.28	1.79-3.51
Hemoglobin (g/dL)	11.97-17.18	13.87	1.26	11.60-17.90
Packed cell volume (%)	33.23-44.78	38.13	2.95	32.0-46.0
Mean cell volume (fL)	144.44-195.19	170.46	13.58	131.0-196.8
Mean cell hemoglobin (pg)	51.39-71.83	61.89	5.16	50.90-73.70
Mean cell hemoglobin concentration (g/dL)	34.26-38.76	36.32	1.05	34.10-38.90
White blood cells ($\times 10^9$/L [$\times 10^3$/μL])	4.77-18.31	9.52	3.54	3.80-18.90
Heterophils ($\times 10^9$/L [$\times 10^3$/μL])	1.32-9.08	4.52	2.29	1.01-13.20
Lymphocytes ($\times 10^9$/L [$\times 10^3$/μL])	1.57-9.36	4.68	1.94	1.27-10.20
Monocytes ($\times 10^9$/L [$\times 10^3$/ (μL])	0.00-0.05	0.08	0.13	0.00-0.50
Eosinophils ($\times 10^9$/L [×103/μL])	0.00-0.52	0.16	0.16	0.00-0.74
Basophils ($\times 10^9$/L [×103/μL])	0.00-0.37	0.07	0.09	0.00-0.40
Thrombocytes ($\times 10^9$/L [×103/μL])	16.37-56.30	31.60	10.73	15.4-69.20
Fibrinogen (g/L)	1.00-3.89	2.20	0.70	1.00-4.00

Source: Samour J, Naldo J, Rahman H, Sakkir M: Hematologic and plasma biochemical reference values in Indian Peafowl (Pavo cristatus), *J Avian Med Surg* 24(2):99–106, 2010.
*Recommended reference values.
SD, standard deviation.

Hematology Values for Endangered Columbiformes

Species	Nicobar Pigeon $n = 16$	Pheasant Pigeon $n = 3$	Common Crowned Pigeon $n = 9$	Victoria Crowned Pigeon $n = 6$	Scheepmaker's Crowned Pigeon $n = 1$
Scientific Name	***Caloenas nicobarica***	***Otidiphaps nobilis***	***Goura cristata***	***Goura victoria***	***Goura scheepmakeri***
PCV, %	50.7 ± 2.6 (45-56)	41.7 ± 9.0 (33-51)	34.3 ± 3.3 (30.0-39.5)	37.6 ± 3.8 (33.8-42)	42.0
Hb, g/dL	17.0 ± 1.5 (12.7-19.7)	13.9 ± 2.8 (11.4-17.0)	10.8 ± 1.5 (8.2-12.7)	12.3 ± 1.6 (10.6-14.7)	12.7
RBC, 10^6/mL	3.40 ± 0.47 (2.67-4.33)	2.6 ± 0.4 (2.26-3.0)	2.23 ± 0.35 (1.80-2.80)	2.31 ± 0.23 (1.95-2.60)	2.30
MCV, fL	149.8 ± 16.5 (127.6-168.5)	158.9 ± 12.1 (146-170)	158.7 ± 14.7 (142.9-175.0)	166.9 ± 18.2 (134.6-178.3)	182.6
MCH, pg	50.0 ± 5.5 (41.3-57.6)	53.3 ± 3.1 (50.4-56.6)	50.8 ± 5.2 (44.2-57.3)	55.0 ± 7.3 (42.5-61.3)	55
MCHC, g/dL	33.5 ± 1.8 (28.3-36.1)	33.6 ± 0.8 (33.1-34.5)	31.9 ± 4.3 (27.9-38.0)	32.6 ± 1.5 (31.1-35.0)	30.1
WBC, 10^3/mL	4.23 ± 1.92 (2.0-8.25)	6.91 ± 2.43 (4.12-8.62)	17.71 ± 4.87 (11.75-25.12)	10.85 ± 6.54 (5.13-20.75)	19.38
Heterophils, %	52.2 ± 15.7 (42-71)	54.1 ± 11.4 (46-67)	66.0 ± 9.0 (55-78)	52.8 ± 12.0 (40-70)	56
Lymphocytes, %	37.4 ± 9.9 (27-51)	42.5 ± 13.6 (27-51)	30.3 ± 8.4 (18-40)	44.6 ± 12.1 (27-58)	37
Monocytes, %	2.1 ± 1.6 (1-5)	1.6 ± 0.8 (0-2)	1.0 ± 0.5 (0-2)	0.7 ± 0.8 (0-2)	2
Eosinophils, %	2.7 ± 2.3 (1-9)	0.6 ± 1.1 (0-2)	2.4 ± 1.4 (1-5)	2.5 ± 1.1 (1-4)	3
Basophils, %	0	1.2 ± 1.2 (0-2.5)	0.25 ± 0.5 (0-1)	0.3 ± 0.5 (0-1)	2

Source: Peinado VI, Polo FJ, Celdran JF, et al: Hematology and plasma chemistry in endangered pigeons, *J Zoo Wildl Med* 23:65–71, 1992.
Mean ± SD (minimum-maximum).
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; PCV, packed cell volume; RBC, red blood cell; WBC, white blood cell.

Inter-Species Comparison of Mean Percentages in Several Hematologic Parameters in Crested and Common Coots

Blood Parameters	Crested Coot	Common Coot*	*p* Value†	1–β‡
Haematocrit (%)	37.5 ± 3.13 (29), 8.34%	41.04 ± 5.8 (21), 14.13%	0.018	0.88
Lymphocytes (%)	78.51± 9.88 (29), 12.58%	67.85 ± 11.54 (20), 17%	0.000	0.96
Heterophils (%)	18.06 ± 7.66 (29), 42.41%	26.7 ± 10.41 (20), 38.98%	0.002	0.91
Eosinophils (%)	0.79 ± 1.2 (29), 155.69%	1.95 ± 2.62 (20), 134.35%	0.028	0.59
Basophils (%)	0.75 ± 1.55 (29), 206.66%	0.55 ± 1.23 (20), 223.63%	0.588	0.08
Monocytes (%)	2.20 ± 3.77 (29), 171.36%	2.2 ± 3.56 (20), 161.81%	0.975	0.05

Source: Rubio MD, Ildefonso N, Aguera RJ, et al: Plasma biochemistry and hematology of crested coots (*Fulica cristata*) and common coots (*Fulica atra*) from Spain, *Comp Clin Path* 23:385–391, 2014.
*Values shown are mean + standard deviation (sample size), coefficient of variation.
†*P* value of the ANOVA was obtained after conducting 10,000 Monte Carlo simulations with the original data set (see, Manly, 1997). Significant values are shown in bold type.
‡1–β power of the test (probability of rejecting the null hypothesis when it is in fact false (Zar, 1999).
ANOVA, analysis of variance.

Hematologic Values of Captive Horned Guans (Oreophasis Derbianus) from Three Different Facilities

Analyte	*n*	Mean or Median*	RI
WBC (1000/μL)	27	11	7-15
RBC (million/μL)	23	2.1	1.5-2.6
Hematocrit (%)	27	35	23-46
MCV (fL)	23	166	149-184
Heterophils/μL	27	6286	3,126-9,445
Lymphocytes/μL	27	4150	2,425-5,876
Heterophils/lymphocytes (total)	29	1.5	0.3-2.6
Monocytes/μL	24	339	0-941
Eosinophils/μL	25	461	0-969
Heterophils (%)	29	57*	16-65
Lymphocytes (%)	29	37*	28-79
Monocytes (%)	28	3.0*	0.0-29
Eosinophils (%)	29	3.2	0.0-7.6

Source: Cornejo J, Richardson D, Perez J: Hematologic and plasma biochemical reference values of the horned guan, *Oreophasis derbianus*, *J Zoo Wildl Med* 45(1):15–22, 2014.
RI indicates the reference interval and "*n*" the number of individuals sampled
*Values are $p < 0.01$ (Mann-Whitney *U*-test).

Hematologic Test Results from Clinically Normal Elegant-Crested Tinamou (n = 19)

Analyte	Mean ± SD (SEM)	Reference Interval	Median
White blood cells ($\times 10^3/\mu L$)	14.00 ± 3.15 (0.87)	8.90-21.20	13.90
Heterophils			
Absolute ($\times 10^3/\mu L$)	3.94 ± 2.34 (0.65)	1.39-8.64	2.98
Relative (%)	27.38 ± 13.65 (3.79)	13.00-55.00	23.00
Lymphocytes			
Absolute ($\times 10^3/\mu L$)	8.95 ± 2.43 (0.67)	5.18-13.11	8.31
Relative (%)	65.00 ± 14.58 (4.04)	33.00-84.00	69.00
Monocytes			
Absolute ($\times 10^3/\mu L$)	0.64 ± 0.50 (0.14)	0.00-1.48	0.50
Relative (%)	14.38 ± 3.52 (0.98)	0.00-12.00	3.00
Basophils			
Absolute ($\times 10^3/\mu L$)	0.32 ± 0.28 (0.08)	0.00-0.94	0.30
Relative (%)	2.23 ± 1.79 (0.50)	0.00-6.00	2.00
Eosinophils			
Absolute ($\times 10^3/\mu L$)	0.15 ± 0.23 (0.06)	0.00-0.64	0.00
Relative (%)	1.00 ± 1.41 (0.39)	0.00-4.00	1.00
Heterophil:lymphocyte	0.51 ± 0.44 (0.12)	0.15-1.67	0.32
Hematocrit (%)	33.77 ± 3.68 (1.02)	29.00-41.00	32.00

Source: Black P, Macek M, Tieber A, Weber M: Reference values for hematology, plasma biochemical analysis, plasma protein electrophoresis, and *Aspergillus* serology in elegant-crested tinamou (*Eudromia elegans*), *J Avian Med Surg* 27(1):1–6, 2013.
SD, standard deviation; SEM, standard error of the mean.

Hematology Values for Selected Ratites

Species	Ostrich	Cassowary
Scientific Name	***Struthio camelus***	***Casuarius* spp.**
RBC, $\times 10^6$/mL	1.7 (0.4)	2.1 (0.3)
HB, g/dL	12.2 (2)	14.5 (0.5)
PCV	32.0 (3.0)	50.8 (3.7)
MCV, fL	174 (42.0)	245.0 (41.0)
MCH, pg	61.0 (16.0)	70.0 (11.5)
MCHC, g/dL	33.0 (5.0)	28.5 (1.6)
WBC, 10^3/mL	5.5 (1.9)	18.0 (4.5)
Heterophils, %	62.6 (7.6)	77.7 (25.8)
Lymphocytes, %	34.1 (7.0)	19.7 (10.4)
Monocytes, %	2.8 (1.3)	2.4 (2.4)
Eosinophils, %	0.3 (0.5)	—
Basophils, %	0.2 (0.5)	—

Source: Stewart JS: Husbandry, medical and surgical management of ratites, part 2, *Proceedings of the Association of Zoo Veterinarians*, Greensboro, 1989, pp 119–122.
Mean (SD).
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; PCV, packed cell volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Juvenile Eclectus Parrots (Eclectus Roratus)

Parameter	30 Day	90 Day	All
RBC, $\times 10^6$/mL	1.95 (0.28)	3.22 (0.51)	2.69 (0.67)
HB, g/dL	8.83 (1.15)	15.42 (2.38)	12.46 (3.01)
HCT, %	33.7 (4.4)	53.8 (3.0)	43.8 (8.4)
MCV, fL	174 (25)	169 (27)	166 (26)
MCH, pg	43.9	49.1 (9.9)	45.5 (10.7)
MCHC, g/dL	26.1 (2.5)	28.7 (4.1)	27.7 (5.0)
WBC, #/mL	18,500 (6,900)	10,900 (3,700)	13,700 (6,300)
WBC, #/# (est.)/mL	17,000 (6,000)	10,500 (4,000)	13,500 (6,000)
Bands, %	0.2 (1.1)	0.4 (0.9)	0.5 (1.5)
Heterophils, %	62.8 (7.7)	52.1 (10.2)	53.9 (11.4)
Lymphocytes, %	30.4 (6.3)	40.8 (10.4)	39.5 (11.5)
Monocytes, %	5.5 (3.0)	5.2 (2.7)	5.0 (2.7)
Eosinophils, %	0.0 (0.0)	0.1 (0.4)	0.1 (0.3)
Basophils, %	1.2 (1.0)	1.5 (1.0)	1.1 (1.0)
Bands, #/mL	34 (188)	48 (111)	70 (221)
Heterophils, #/mL	11,800 (5,400)	5,900 (,2800)	7,700 (4,800)
Lymphocytes, #/mL	5,500 (2,100)	4,200 (1,200)	5,100 (2,000)
Monocytes, #/mL	930 (520)	532 (331)	639 (428)
Eosinophils, #/mL	0	9 (43)	8 (44)
Basophils, #/mL	209 (199)	175 (158)	152 (169)
Heterophils : lymphocytes (ratio)	2.2 (0.8)	1.4 (0.6)	1.6 (0.8)
PP (refrac), g/dL	2.8 (0.6)	3.9 (0.6)	3.5 (0.8)

Source: Clubb SL, Schubot RM, Joyner K, et al: Hematologic and serum chemistry reference intervals in juvenile eclectus parrots, *JAAV* 4:218–225, 1990.
Mean (SD).
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; PCV, packed cell volume; RBC, red blood cell; WBC, white blood cell.

Hematology Values for Juvenile Cockatoos (*Cacatua* spp.)

Parameter	30 Day	90 Day	All
RBC, $\times 10^6$/mL	196 (0.22)*	2.84 (0.49)*	2.53 (0.63)
Hb, g/dL	8.12 (0.83)*	14.04 (1.23)*	11.43 (2.90)
HCT, %	30.1 (2.8)*	47.6 (4.1)*	39.7 (9.0)
MCV, fL	155 (17)*	172 (28)*	160 (23)
MCH, pg	38.9 (11.7)*	49.0 (12.9)*	43.8 (10.8)
MCHC, g/dL	24.6 (7.9)*	28.5 (6.2)*	27.2 (6.1)
WBC, #/mL	13,700 (7,400)*	10,000 (2,800)*	12,900 (6,300)
WBC, #/# (est.)/mL	13,200 (6,700)*	10,400 (2,800)*	13,100 (5,900)
Bands, %	1.3 (2.3)*	1.3 (2.3)*	1.3 (2.3)
Heterophils, %	54.8 (9.7)*	49.0 (8.1)*	50.8 (11.7)
Lymphocytes, %	36.4 (8.1)*	43.6 (8.4)*	41.2 (11.9)
Monocytes, %	6.9 (3.4)*	4.9 (3.4)*	5.8 (3.4)
Eosinophils, %	0 (0)	0 (0.2)	0 (0.0)
Basophils, %	0.6 (0.9)*	1.2 (1.1)*	0.9 (1.1)
Bands, #/mL	150 (275)*	130 (290)*	160 (325)
Heterophils, #/mL	7,800 (5,000)*	4,400 (2,200)*	6,500 (4,500)
Lymphocytes, #/mL	4,900 (2,600)*	3,900 (2,000)*	4,900 (2,500)
Monocytes, #/mL	880 (530)*	440 (450)*	690 (525)
Eosinophils, #/mL	0 (0)	0 (0)	0 (0)
Basophils, #/mL	67 (130)*	115 (130)*	100 (140)
Heterophil lymphocyte (ratio)	1.6 (0.6)*	1.2 (0.4)*	1.4 (0.8)
PP (refrac), g/dL	2.3 (0.5)*	4.0 (0.8)*	3.2 (0.9)

Source: Clubb SL, Schubot RM, Joyner K, et al: Hematologic and serum biochemical reference intervals in juvenile cockatoos, *JAAV* 5: 16–26, 1991.
*Mean (SD).
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; PCV, packed cell volume; RBC, red blood cell; SD, standard deviation; WBC, white blood cell.

Hematology Values for Juvenile Umbrella Cockatoos (*Cacatua alba*)

Parameter	30 Day	90 Day	All
RBC, ×106/ml	1.98 (0.51)[n]	2.75 (0.49)[n]	2.54
HB, g/dL	7.9 (1.64)[n]	14 (0.92)[n]	11.6
HCT, %	29.5 (5.65)[n]	46.9 (2.92)[n]	39.3
MCV, fL	151 (25.6)[n]	175 (28.5)[n]	158.0
MCH, pg	35.3 (10.03)[n]	51.9 (8.57)[n]	43.6
MCHC, g/dL	21.8 (10.5)[n]	29.9 (1.1)[n]	27.0
WBC, #/mL	20,311 (5,717)[n]	10,238 (3,368)[n]	16 567.0
WBC, #/# (est.)/mL	19,190 (5127)[s]	10,500 (3184)[n]	16 412.0
Bands, %	1 (2.57)[n]	1.93 (2.76)[n]	1.31
Heterophils, %	58.4 (11.4)[s]	50 (9.7)[n]	54.1
Lymphocytes, %	34.4 (11.5)[n]	41.2 (9.9)[n]	38.1
Monocytes, %	5.77 (3.1)	5.29 (3.27)[n]	5.35
Eosinophils, %	0 (0.14)	0.07 (0.27)[n]	0.02
Basophils, %	0.45 (1.05)[n]	1.43 (0.94)[n]	1.03
Bands, #/mL	185 (331)[n]	192 (368)	202.0
Heterophils, #/mL	12,041 (4,993)[s]	4,465 (2,595)[n]	8,917.0
Lymphocytes, #/mL	6893 (2581)[s]	3663 (2076)[n]	5695.0
Monocytes, #/mL	118 (624)[s]	492 (529)[n]	843.0
Eosinophils, #/mL	0 (0)[n]	0 (0)[n]	0.00011
Basophils, #/mL	83 (181)[n]	137 (135)[n]	143.0
Heterophils: lymphocytes (ratio)	1.83 (1.05)[s]	1.33 (0.54)[n]	1.64
PP (refrac), g/dL	2.69 (0.71)	4.26 (0.55)[n]	3.56

Source: Clubb SL, Schubot RM, Joyner K, et al: Hematologic and serum biochemical reference intervals in juvenile cockatoos, *JAAV* 5:16–26, 1991.
Mean (SD).
s, mean is statistically different ($p < 0.05$) from the same parameter in all juvenile cockatoos; *n*, mean is not statistically different ($p > 0.05$) from the same parameter in all juvenile cockatoos.
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; PCV, packed cell volume; RBC, red blood cell; SD, standard deviation; WBC, white blood cell.

Hematology Values for Juvenile Macaws (*Ara* sp.)

Parameter	30 Day	90 Day	All
RBC, $\times 10^6$/mL	1.9 (0.3)*	3.7 (0.5)*	2.9 (0.8)
HB, g/dL	7.7 (0.9)*	15.4 (1.0)*	12.3 (3.3)
HCT, %	30.9 (3.3)*	49.5 (2.5) *	41.7 (8.4)
MCV, fL	165.5 (25.4)*	137 (19.2)*	149 (24.7)
MCH, pg	41.7 (6.1)*	42.8 (5.8)*	42.3 (6.2)
MCHC, g/dL	25.1 (1.9)*	31.1 (1.3)*	28.7 (2.9)
WBC, #/mL	19,300 (8,300)*	17,700 (4,900)*	19,200 (6,900)
WBC, #/# (est.)/mL	17,700 (5,100)*	18,300 (4,500)*	18,600 (5,880)
Bands, %	0.8 (1.6)*	0.3 (1.2)*	0.6 (1.7)
Heterophils, %	58.9 (11.1)*	53.9 (9.4)*	55.3 (1.0)
Lymphocytes, %	33.8 (9.7)*	41.6 (9.6)*	39.0 (10)
Monocytes, %	5.9 (3.3)*	3.6 (2.0)*	4.4 (2.9)
Eosinophils, %	0 (0)*	0.1 (0.2)*	0 (0.2)
Basophils, %	0.7 (0.9)*	0.6 (1.2)*	0.5 (1.0)
BAND, #/mL	134 (344)*	59 (230)*	110 (313)
Heterophils, #/mL	10,200 (7,600)*	9,400 (4,000)*	10,100 (5,800)
Lymphocytes, #/mL	5,500 (3,100)*	7,000 (2,500)*	6,800 (3,200)
Monocytes, #/mL	910 (643)*	627 (418)*	750 (545)
Eosinophils, #/mL	0 (0.0)*	9.3 (51)*	4.6 (35)
Basophils, #/mL	115 (190) *	75 (165)*	91 (175)
Heterophil : lymphocyte (ratio)	2.0 (1.0)*	1.4 (0.6)*	1.6 (0.8)
PP (refrac), g/dL	1.8 (0.40)*	3.5 (0.4)*	2.9 (0.8)

Source: Clubb SL, Schubot RM, Joyner K, et al: Hematologic and serum biochemical reference intervals in juvenile macaws (*Ara* sp), *JAAV* 5:154–162, 1991.
Mean (SD).
*Values for parameters are statistically different ($p < 0.05$) when letters are different.
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; PCV, packed cell volume; RBC, red blood cell; SD, standard deviation; WBC, white blood cell.

Hematology Values for Juvenile Blue and Gold Macaws (*Ara ararauna*)

Parameter	30 Day	90 Day	All
RBC, $\times 10^6$/mL	1.9 (0.3)*,n	3.5 (0.4)‡,n	2.7 (0.7)
HB, g/dL	7.9 (0.9)*,n	15 (0.9)‡,s	11 (2.9)
HCT, %	30 (2.7)*,n	48 (2.0)‡,s	40 (7.7)
MCV, fL	163 (27)*,n	137 (14)†,n	149 (122)
MCH, pg	43 (7.1) *,n	41 (3.7)*,n	38 (13)
MCHC, g/dL	26 (1.6)*,n	31 (1.4)‡,n	25 (9.5)
WBC, #/mL	19,200 (5,600)*,n	16,600 (4,300)†,n	18,928 (5,561)
WBC, #/# (est.)/mL	18,300 (5,600)	16,800 (4,300)	18,300 (5,600)
Bands, %	0.36 (1.3)*,n	0 (0)*,n	0.12 (0.7)
Heterophils, %	57 (11.6)*,n	48 (11)*,n	52 (10)
Lymphocytes, %	37 (10)*,n	47 (11)‡,n	42 (10)
Monocytes, %	5.3 (2.9)*,n	3.8 (2.2)*,n	4.3 (2.7)
Eosinophils, %	0 (0)*,n	0 (0)*,n	0 (0)
Basophils, %	0.9 (1.1)*,n	1.1 (1.7)*,n	0.9 (1.3)
Band, #/mL	0.36 (1.3)*,n	0 (0)*,n	0.12 (0.7)
Heterophils, #/mL	11,000 (4,600)*,n	8,100 (3,000)*,n	10,000 (3,800)
Lymphocytes, #/mL	7,000 (2,600)*,n	7,700 (2,600)†,n	8,000 (3,100)
Monocytes, #/mL	949 (498)*,n	639 (421)*,n	756 (446)
Eosinophils, #/mL	0 (0)*,n	0 (0)*,n	0 (0)
Basophils, #/mL	194 (245)*,n	156 (256)*,n	154 (229)
Heterophils : lymphocytes (ratio)	1.75 (0.85)*,n	1.19 (0.77)*,n	1.38 (0.69)
PP (refrac), g/dL	1.87 (0.2)*,n	3.62 (0.5)‡,n	2.86 (0.8)

Source: Clubb SL, Schubot RM, Joyner K, et al: Hematologic and serum biochemical reference intervals in juvenile macaws (*Ara* sp), *JAAV* 5:154–162, 1991.
Mean (SD).
*,†,‡: Values for parameters are statistically different ($p < 0.05$) when letters are different; s, mean is statistically different ($p < 0.05$) from the same parameter in all juvenile macaws; n, mean is not statistically different ($p > 0.05$) from the same parameter in all juvenile macaws.
Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; MCH, mean corpuscular hemoglobin; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; PCV, packed cell volume; RBC, red blood cell; SD, standard deviation; WBC, white blood cell.

Age-Related Hematological Changes in Captive Kori Bustards (*Ardeotis kori*)

	MONTHS									
Parameter	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**15**
RBC, $\times 10^{12}$/L	1.28 ± 0.06 (1.04-1.61)	1.57 ± 0.06 (1.22-2.01)	1.76 ± 0.08 (1.31-2.4)	2.06 ± 0.08 (1.39-2.63)	2.07 ± 0.08 (1.79-2.59)	2.14 ± 0.07 (1.84-2.46)	2.12 ± 0.07 (1.79-2.61)	2.0 ± 0.1 (1.72-2.6)	2.1 ± 0.04 (1.91-2.23)	2.08 ± 0.06 (1.81-2.47)
Hb, g/dL	7.5 ± 0.2 (6.8-8.3)	9.7 ± 0.3 (7.4-10.9)	10.9 ± 0.3 (9.6-13.1)	12.1 ± 0.3 (10.3-14.0)	12.1 ± 0.4 (9.1-14.0)	11.4 ± 0.4 (9.9-13.2)	11.7 ± 0.3 (10.5-13.1)	12.5 ± 0.4 (10.9-14.3)	12.8 ± 0.4 (10.9-14.2)	14.2 ± 0.4 (12.1-16.1)
Hct, L/L	0.23 ± 0.7 (0.19-0.26)	0.30 ± 0.9 (0.24-0.34)	0.35 ± 0.7 (0.30-0.41)	0.37 ± 0.7 (0.32-0.41)	0.39 ± 0.9 (0.33-0.44)	0.38 ± 0.9 (0.34-0.42)	0.39 ± 0.9 (0.35-0.45)	0.39 ± 1.0 (0.36-0.45)	0.39 ± 0.9 (0.36-0.43)	0.47 ± 0.9 (0.41-0.51)
MCV, fL	178.4 ± 17.9 (152.2-219.6)	195.2 ± 6.6 (159.2-225.4)	204.3 ± 8.1 (168.8-262.8)	185.2 ± 7.9 (144.0-241.0)	194.2 ± 7.8 (152.5-225.4)	179.9 ± 5.0 (160.6-210.1)	188.2 ± 5.6 (165.1-220.7)	200.5 ± 7.4 (138.5-219.3)	186.5 ± 3.6 (168.2-200.5)	226.5 ± 7.3 (195.5-273.5)
MCH, pg	59.3 ± 3.4 (42.2-77.6)	62.0 ± 1.5 (52.2-69.0)	63.5 ± 2.4 (49.2-79.4)	59.6 ± 2.3 (45.9-74.1)	59.5 ± 3.1 (42.1-73.7)	53.7 ± 2.1 (40.2-66.5)	55.8 ± 2.1 (43.7-66.5)	64.4 ± 2.5 (45.4-74.6)	60.5 ± 1.3 (57.1-68.6)	68.7 ± 2.4 (58.2-84.0)
MCHC, g/dL	33.2 ± 1.0 (27.8-37.2)	32.0 ± 0.9 (28.2-40.4)	31.2 ± 0.8 (27.5-38.1)	32.3 ± 0.5 (29.2-35.3)	30.5 ± 0.7 (24.3-33.0)	29.8 ± 0.7 (25.1-32.4)	29.6 ± 0.6 (25.6-32.3)	32.2 ± 0.6 (29.5-36.3)	32.5 ± 0.7 (29.8-35.5)	30.3 ± 0.5 (27.6-33.2)
WBC, $\times 10^9$/L	8.78 ± 0.45 (6.65-10.85)	10.2 ± 0.6 (6.1-14.75)	10.7 ± 0.7 (7.05-16.2)	12.7 ± 0.7 (8-18.8)	12.5 ± 0.4 (10.35-15.15)	13.5 ± 0.7 (9.8-16.1)	15.6 ± 0.7 (9.2-18.5)	13.5 ± 0.9 (9.25-17.05)	14.5 ± 0.5 (12.8-16.9)	14.10 ± 0.61 (11.25-17.8)
Heterophils, $\times 10^9$/L	5.42 ± 0.38 (3.72-6.94)	4.28 ± 0.30 (2.62-6.34)	5.14 ± 0.65 (2.58-11.8)	6.2 ± 0.6 (2.9-10.2)	5.1 ± 0.5 (1.5-7.5)	5.1 ± 0.9 (1.9-11.4)	6.34 ± 0.58 (2.76-8.83)	5.41 ± 0.58 (2.59-8.35)	6.27 ± 0.76 (4.08-10.8)	5.84 ± 0.61 (2.70-10.63)
Lymphocytes, $\times 10^9$/L	2.63 ± 0.26 (1.37-3.70)	4.64 ± 0.33 (2.50-6.97)	4.19 ± 0.27 (3.08-6.35)	5.1 ± 0.4 (2.9-7.8)	5.49 ± 0.45 (3.6-9.05)	6.7 ± 0.6 (3.0-10.5)	7.32 ± 0.59 (4.5-11.84)	6.38 ± 0.52 (3.8-9.29)	6.49 ± 0.59 (2.85-8.45)	6.49 ± 0.43 (4.37-9.43)
Monocytes, $\times 10^9$/L	0.24 ± 0.06 (0.08-0.63)	0.66 ± 0.15 (0.24-1.92)	0.80 ± 0.12 (0.00-1.58)	0.81 ± 0.08 (0.35-1.50)	1.13 ± 0.16 (0.49-2.03)	0.93 ± 0.14 (0.14-1.64)	1.12 ± 0.13 (0.45-1.78)	1.18 ± 0.13 (0.63-1.70)	0.75 ± 0.17 (0.13-1.41)	1.13 ± 0.07 (0.80-1.53)
Eosinophils, $\times 10^9$/L	0.28 ± 0.08 (0.0-0.65)	0.32 ± 0.04 (0.1-0.5)	0.38 ± 0.08 (0.07-1.03)	0.35 ± 0.05 (0.0-0.78)	0.42 ± 0.08 (0.0-1.06)	0.36 ± 0.07 (0.14-0.76)	0.48 ± 0.08 (0.0-0.89)	0.21 ± 0.06 (0.0-0.63)	0.53 ± 0.09 (0.13-0.92)	0.35 ± 0.05 (0.13-0.68)
Basophils, $\times 10^{99}$/L	0.21 ± 0.06 (0.0-0.47)	0.12 ± 0.04 (0.0-0.43)	0.04 ± 0.01 (0.0-0.13)	0.11 ± 0.04 (0.0-0.48)	0.05 ± 0.01 (0.0-0.16)	0.23 ± 0.57 (0.0-0.57)	0.08 ± 0.01 (0.0-0.14)	0.03 ± 0.01 (0.0-0.08)	0.08 ± 0.01 (0.02-0.14)	0.29 ± 0.06 (0.0-0.69)
Thrombocytes, $\times 10^9$/L	7.03 ± 1.79 (3.1-15.0)	8.1 ± 0.8 (4.07-15.6)	6.7 ± 0.4 (4.6-9.2)	7.9 ± 0.7 (3.7-15.0)	7.35 ± 0.61 (3.7-10.8)	9.98 ± 1.09 (4.9-17)	11.61 ± 1.0 (5.5-16.7)	10.6 ± 0.9 (6.3-15)	7.0 ± 1.0 (4.8-9.5)	6.52 ± 0.54 (4.05-10.2)
Fibrinogen, g/L	1.76 ± 0.18 (1.1-2.6)	2.0 ± 0.1 (1.2-2.8)	2.25 ± 0.2 (1.6-3.8)	2.57 ± 0.3 (1.6-4.0)	2.58 ± 0.49 (1.6-4.8)	2.83 ± 0.22 (1.7-4.0)	3.0 ± 0.2 (2.0-4.7)	2.7 ± 0.3 (1.1-5.4)	2.65 ± 0.27 (2.0-3.3)	3.0 ± 0.3 (1.3-5.3)

Source: Howlett JC, Samour JH, Bailey TA, Naldo JL: Age-related haematology changes in captive-reared kori bustards *(Ardeotis kori)*, *Comp Haematol Int* 8:26–30, 1998.
Mean ± standard error of mean (minimum-maximum).
Hb, hemoglobin; Hct, hematocrit; MCH, mean cell hemoglobin; MCHC, mean cell hemoglobin concentration; MCV, mean cell volume; PCV, packed cell volume; RBC, red blood cells; WBC, white blood cells.

Age-Related Hematology Values in Captive Masai Ostrich (*Struthio camelus massaicus*; *n* = 24)

		MONTHS											
Variable	**P**	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**	**11**	**12**
RBC, $\times 10^{12}$/L	<0.001	1.8 ± 0.04* (1.4-2.2)†	1.8 ± 0.04 (1.6-2.3)	1.6 ± 0.02 (1.4-1.8)	1.7 ± 0.04 (1.3-2.2)	1.8 ± 0.03 (1.4-2.0)	1.8 ± 0.03 (1.5-2.1)	1.9 ± 0.04 (1.6-2.5)	1.9 ± 0.03 (1.7-2.3)	2.1 ± 0.04 (1.7-2.5)	1.9 ± 0.03 (1.7-2.2)	2.1 ± 0.03 (1.7-2.6)	2.1 ± 0.04 (1.7-2.6)
Hb, g/dL	<0.001	12.7 ± 0.1 (11.1-13.8)	11.9 ± 0.2 (10.3-14.4)	10.7 ± 0.1 (9.7-11.9)	11.5 ± 0.2 (10.3-13.6)	11.3 ± 0.1 (10.1-12.9)	11.9 ± 0.1 (10.6-13.4)	13.3 ± 0.2 (12.0-14.8)	14.1 ± 0.1 (12.1-15.5)	14.9 ± 0.2 (13.4-16.3)	15.2 ± 0.1 (14.1-16.2)	15.5 ± 0.2 (13.9-17.5)	15.3 ± 0.2 (13.7-17.2)
PCV, %	<0.001	35.9 ± 0.5 (30.6-40.8)	34.1 ± 0.4 (30.5-38.9)	31.9 ± 0.3 (29.0-34.9)	33.4 ± 0.4 (29.1-37.0)	34.2 ± 0.4 (31.1-37.9)	35.1± 0.4 (32.0-39.4)	38.4 ± 0.7 (26.0-42.9)	40.9 ± 0.4 (38.0-43.9)	43.6 ± 0.5 (39.1-47.4)	44.4 ± 0.4 (40.5-47.9)	45.1 ± 0.5 (40.1-50.7)	44.6 ± 2.4 (40.1-49.9)
MCV, fL	<0.001	197.1 ± 3.2 (161.6-225.1)	185.5±2.9 (153.6-210.4)	194.0 ± 2.2 (178.3-209.9)	196.8 ± 3.9 (166.3-241.9)	192.3 ± 3.3 (171.71-231.9)	198.9 ± 3.2 (169.5-226.4)	194.9 ± 4.2 (143.9-228.6)	213.7 ± 3.08 (179.8-237.4)	203.6 ± 4.9 (125.3-232.8)	222.8 ± 2.9 (201.4-261.2)	219.0 ± 2.8 (196.1-247.8)	212.5 ± 3.2 (186.8-241.8)
MCH, pg	<0.001	70.1±1.3 (55.3-81.9)	64.6 ± 0.9 (56.8-74.7)	65.2 ± 0.7 (58.6-71.6)	67.4 ± 1.2 (59.2-84.5)	63.1 ± 0.9 (55.9-74.3)	67.9 ± 1.2 (58.8-80.0)	67.4 ± 1.3 (52.5-77.3)	74.6 ± 1.1 (62.4-83.4)	71.1 ± 1.2 (60.9-81.3)	76.32 ± 1.04 (69.31-90.69)	75.1 ± 0.9 (67.4-86.2)	72.9 ± 1.1 (63.6-83.8)
MCHC, g/dL	<0.001	35.4 ± 0.3 (32.9-37.9)	34.9.4 ± 0.5 (31.6-39.9)	33.6 ± 0.3 (31.8-37.2)	34.3 ± 0.3 (31.9-37.9)	32.8 ± 0.2 (30.6-35.2)	34.1 ± 0.3 (31.6-36.8)	34.8 ± 0.7 (32.3-48.5)	34.9 ± 0.2 (33.5-36.6)	34.3 ± 0.1 (32.7-35.6)	34.2 ± 0.2 (32.2-35.9)	34.3 ± 0.2 (32.7-35.5)	34.3 ± 0.2 (32.6-36.0)
WBC, $\times 10^9$/L	<0.001	11.9 ± 0.7 (5.4-18.9)	10.9 ± 0.8 (5.5-19.6)	11.3 ± 1.1 (3.8-28.1)	11,8 ± 0.9 (4.9-20.8)	14.3 ± 0.9 (6.1-22.1)	16.7 ± 0.8 (9.2-27.5)	23.9 ± 1.2 (17.1-38.6)	17.6 ± 0.9 (9.6-28.5)	24.2 ± 1.2 (13.1-34.7)	24.6 ± 1.5 (14.4-42.2)	24.5 ± 1.3 (17.3-39.2)	23.0 ± 1.1 (12.55-32.9)
Het, $\times 10^9$/L	<0.001	9.0 ± 0.6 (3.9-15.5)	8.7 ± 0.7 (3.1-16.5)	8.7 ± 0.9 (3.2-21.4)	9.4 ± 0.7 (3.8-16.6)	11.0 ± 0.8 (4.5-19.2)	12.9 ± 0.8 (6.5-23.5)	18.9 ± 1.2 (12.2-33.9)	14.1 ± 0.1 (12.1-15.5)	19.4 ± 1.2 (8.9-30.8)	18.5 ± 1.4 (9.7-36.5)	18.7 ± 1.3 (11.1-32.5)	17.4 ± 0.9 (8.5-26.0)
Lymph, $\times 10^9$/L	<0.001	2.7 ± 0.1 (1.3-4.6)	2.03 ± 0.1 (0.9-3.4)	2.2 ± 0.2 (0.6-5.1)	1.9 ± 0.2 (0.7-3.8)	3.1 ± 0.2 (1.2-5.3)	3.2 ± 0.2 (1.6-5.0)	4.6 ± 0.3 (2.5-7.1)	4.02 ± 0.2 (2.3-5.6)	4.4 ± 0.2 (2.4-6.6)	5.3 ± 0.2 (3.8-8.0)	4.9 ± 0.3 (2.4-7.9)	4.2 ± 0.2 (2.7-6.3)
Mon, $\times 10^9$/L	<0.001	0.02 ± 0.01 (0.0-0.2)	0.04 ± 0.01 (0.0-0.2)	0.04 ± 0.01 (0.0-0.2)	0.1 ± 0.03 (0.0-0.8)	0.04 ± 0.01 (0.0-0.2)	0.1 ± 0.04 (0.0-0.6)	0.3 ± 0.05 (0.0-1.1)	0.3 ± 0.03 (0.09-0.7)	0.2 ± 0.04 (0.0-0.7)	0.3 ± 0.04 (0.00-0.9)	1.15 ± 0.2 (0.2-2.9)	0.7 ± 0.09 (0.01-1.5)
Eos, $\times 10^9$/L	0.505	0.01 ± 0.0 (0.0-0.2)	0.0 ± 0.0 (0.0-0.07)	0.02 ± 0.01 (0.0-0.3)	0.09 ± 0.02 (0.0-0.3)	0.05 ± 0.01 (0.0-0.2)	0.2 ± 0.02 (0.0-0.4)	0.2 ± 0.05 (0.0-0.8)	0.2 ± 0.03 (0.0-0.7)	0.02 ± 0.04 (0.0-0.8)	0.2 ± 0.03 (0.00-0.5)	0.45 ± 0.06 (0.2-1.6)	0.37 ± 0.06 (0.0-1.02)
Bas, $\times 10^9$/L	0.327	0.2 ± 0.02 (0.0-0.4)	0.1 ± 0.02 (0.0-0.4)	0.2 ± 0.07 (0.0-1.4)	0.2 ± 0.04 (0.0-0.7)	0.09 ± 0.02 (0.0-0.4)	0.2 ± (0.040.0-0.8)	0.1 ± 0.03 (0.0-0.5)	0.07 ± 0.02 (0.0-0.4)	0.05 ± 0.02 (0.0-0.4)	0.3 ± 0.05 (0.00-0.8)	0.3 ± 0.03 (0.2-0.8)	0.3 ± 0.1 (0.0-1.0)
Throm, $\times 10^9$/L	<0.001	17.5 ± 1.0 (8.9-26.6)	15.7 ± 1.1 (7.6-25.5)	14.4 ± 1.1 (6.0-25.6)	13.9 ± 0.7 (6.9-20.3)	15.1 ± 0.7 (7.6-22.5)	17.7 ± 1.0 (10.3-26.7)	27.2 ± 1.4 (19.3-42.7)	22.3 ± 1.0 (11.7-32.9)	26.7 ± 1.7 (16.4-43.9)	26.8 ± 1.1 (18.0-36.0)	23.1 ± 1.3 (12.8-36.4)	24.6 ± 1.5 (12.8-38.0)

Source: Samour J, Naldo J, Libanan N, et al: Age-related hematology and plasma chemistry changes in captive Masai ostriches (*Struthio camelus massaicus*) *Comp Clin Path* 20:659–667, 2011.

*Mean ± standard error of the mean.

†Inner limits of the percentiles $P_{2.5}$-$P_{97.5}$ with a probability of 95% confidence interval.

Bas, basophils; Eos, eosinophils; Hb, Hemoglobin; Hct, hematocrit; Het, heterophils; Lymph, lymphocytes; MCHC, mean corpuscular hemoglobin concentration; MCV, mean corpuscular volume; Mono, monocytes; RBC, red blood cell; Throm, thrombocytes; WBC, white blood cell.

APPENDIX 2

Blood Chemistry Reference Values for Selected Avian Species

Merle M. Apo, Tom Bailey

Blood Chemistry Values for Endangered Columbiformes

Species	Nicobar Pigeon *n* = 16	Pheasant Pigeon *n* = 3	Common Crowned Pigeon *n* = 9	Victoria Crowned Pigeon *n* = 6	Scheepmaker's Crowned Pigeon *n* = 1
Scientific Name	***Caloenas nicobarica***	***Otidiphaps nobilis***	***Goura cristata***	***Goura victoria***	***Goura scheepmakeri***
Glucose (mmol/L)	16.04 ± 1.9 (12.8-19.8)	19.6 ± 2.5 (17.0-22.1)	12.8 ± 0.92 (11.1-13.8)	14.2 ± 2.32 (11.6-17.7)	19.4
Urea (mmol/L)	0.97 ± 0.22 (0.66-1.38)	0.85 ± 0.08 (0.73-0.94)	1.41 ± 0.37 (0.87-2.09)	1.66 ± 0.38 (1.14-2.13)	1.63
Uric acid (mmol/L)	611.4 ± 158.8 (390.1-830.3)	779.1 ± 273 (465-967.7)	405.0 ± 274 (133.2-951)	412 ± 177 (215-717)	305.1
Creatinine (mmol/dL)	0.36 ± 0.15 (0.22-0.65)	0.38 ± 0.08 (0.30-0.45)	0.79 ± 0.15 (0.64-0.94)	0.77 ± 0.10 (0.66-0.90)	0.68
Total protein (g/L)	32.6 ± 4.1 (26.3-37.7)	32.7 ± 7.5 (28.4-41.4)	44.6 ± 3.7 (38.7-49.3)	35.8 ± 4.0 (30.1-40.3)	40.5
Prealbumin (g/L)	3.33 ± 0.46 (2.58-4.11)	2.86 ± 0.32 (2.50-3.10)	12.25 ± 2.34 (9.95-16.45)	9.33 ± 4.69 (3.03-13.92)	12.80
Albumin (g/L)	45.50 ± 6.05 (34.34-58.47)	47.20 ± 0.57 (46.80-47.60)	48.69 ± 2.82 (45.01-53.73)	56.10 ± 4.18 (51.33-62.17)	50.43
Globulin (g/L)	4.57 ± 1.78 (1.22-7.93)	8.70 ± 6.79 (3.90-13.50)	6.46 ± 1.43 (3.58-7.70)	6.06 ± 3.18 (3.58-7.70)	9.29 ± 3.18
α-globulin (g/L)	4.57 ± 1.78 (1.22-7.93)	8.70 ± 6.79 (3.90-13.50)	6.46 ± 1.43 (3.58-7.70)	6.06 ± 3.18 (3.04-8.28)	9.29
β-globulin (g/L)	10.93 ± 4.08 (4.79-21.48)	17.20 ± 14.9 (5.90-34.10)	20.29 ± 3.07 (17.81-25.62)	15.31 ± 3.20 (11.59-19.28)	16.63
γ-globulin (g/L)	5.71 ± 2.67 (1.54-9.20)	14.70 ± 9.81 (5.20-24.80)	12.31 ± 2.15 (8.68-15.01)	11.97 ± 3.40 (8.28-16.80)	10.85
Albumin/globulin ratio	3.80 ± 1.40 (2.41-7.23)	2.08 ± 1.53 (1.00-3.16)	1.57 ± 0.16 (1.36-1.80)	1.94 ± 0.25 (1.56-2.20)	1.72
ALT (IU/L)	9.72 ± 6.56 (2.38-19.4)	12.19 ± 4.91 (8.72-15.66)	7.16 ± 1.47 (6.06-10.31)	6.43 ± 2.42 (3.24-8.83)	6.78
AST (IU/L)	123.0 ± 82.6 (40.1-265.3)	65.6 ± 9.51 (58.9-72.35)	54.08 ± 8.42 (41.16-64.34)	42.80 ± 11.92 (23.5-54.2)	39.07
LDH (IU/L)	584.0 ± 496.3 (172.9-1808)	101.3 ± 72.2 (50.2-152.4)	257.6 ± 131.4 (134.6-518.5)	219.5 ± 94.3 (126.8-344.1)	ND
AP (IU/L)	225.2 ± 195.0 (22.6-684.9)	490.5 ± 195.0 (267.1-626.0)	44.3 ± 22.6 (27.9-79.3)	22.4 ± 11.9 (11.3-41.5)	15.37
CPK (IU/L)	251.3 ± 168.3 (43.3-510.4)	233.7 ± 213.1 (106.8-479.7)	117.2 ± 51.9 (65.8-220.5)	341.6 ± 235.2 (146.7-750.0)	63.3
γ-GT (IU/L)	2.21 ± 1.69 (0.5-4.5)	ND	0.30 ± 0.79 (0.00-2.08)	0.42 ± 0.41 (0.00-0.96)	2.88
Magnesium (mmol/L)	1.08 ± 0.20 (0.93-1.22)	1.20 ± 0.11 (1.12-1.27)	0.83 ± 0.16 (0.67-1.10)	0.94 ± 0.18 (0.74-1.17)	ND
Phosphorus (mmol/L)	13.6 ± 0.18 (13.5-13.7)	13.6 ± 9.5 (6.91-20.4)	6.89 ± 1.05 (5.94-8.46)	5.64 ± 1.71 (4.13-8.04)	ND
Calcium (mmol/L)	2.61 ± 0.55 (2.22-3.0)	4.04 ± 0.24 (3.87-4.22)	2.22 ± 0.11 (2.1-2.35)	2.42 ± 0.19 (2.25-2.7)	ND
Chloride (mmol/L)	100.5 ± 11.9 (86.2-126.8)	130.3 ± 12.6 (121.4-139.2)	101.1 ± 3.32 (96.4-106.8)	104.9 ± 4.34 (100.5-109.0)	100.0
Cholesterol (mmol/L)	11.08 ± 2.79 (7.65-15.16)	10.2 ± 4.44 (6.01-14.88)	6.79 ± 1.53 (5.37-9.46)	6.28 ± 3.50 (4.47-12.44)	6.98
Triglycerides (mmol/L)	2.02 ± 0.62 (0.01-3.16)	8.45 ± 3.54 (4.36-10.5)	1.72 ± 1.26 (0.49-4.41)	1.60 ± 2.22 (0.42-5.58)	0.74
Osmolality (mOsmol/kg)	313.0 ± 18.7 (295.0-346.0)	311.3 ± 10.0 (301.0-321.0)	316.6 ± 8.5 (289.0-310.0)	315.4 ± 8.5 (305.0-328.0)	323.0

Source: Modified from: Peinado VI, Polo FJ, Celdran JF, et al: Hematology and plasma chemistry in endangered pigeons, *J Zoo Wildlife Med* 23:65–71, 1992.

Mean ± SD (minimum-maximum).

ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CPK, creatine phosphokinase; GT, glutamyl transferase; IU, International Units; LDH, lactic dehydrogenase; ND, not determined; SD, standard deviation.

Plasma or Serum Chemistry Values for Racing Pigeons

Species	Racing Pigeon	
Scientific Name	***Columba livia***	***n***
Sodium (mmol/L)	145 ± 0.3	68
Potassium (mmol/L)	4.4 ± 0.06	52
Calcium (mmol/L)	2.3 ± 0.03	52
Magnesium (mmol/L)	1.3 ± 0.03	50
Inorganic phosphorus (mmol/L)	0.83 ± 0.04	53
Chloride (mmol/L)	107 ± 0.5	55
Plasma iron (mmol/L)	23 ± 2.1	50
Iron-binding capacity (mmol/L)	37.5 ± 0.8	50
Osmolality (mOsmol/kg)	306 ± 0.9	55
Glucose (mmol/L)	16.6 ± 0.2	96
Creatinine (mmol/L)	28 ± 0.6	52
Uric acid (mmol/L)	375 ± 30	50
CK (EC2.6.3.2; IU/L)	203 ± 13.5	50
AP (EC3.1.31.; IU/L)	367 ± 25	65
LD (EC1.1.1.27; IU/L)	57 ± 3.2	50
ASAT (EC2.6.1.1; IU/L)	58.6 ± 2.5	50
ALAT (EC2.6.1.2; (IU/L)	25 ± 1.6	50
Total serum protein (g/L)	27 ± 0.4	93
Prealbumin (g/L)	2.8 ± 0.13	58
Albumin (g/L)	17.5 ± 0.36	58
α-globulin (g/L)	2.29 ± 0.08	58
β-globulin (g/L)	4.3 ± 0.14	58
γ-globulin (g/L)	1.9 ± 0.1	58

Source: Modified from: Lumeij JT, deBruijne JJ: Blood chemistry reference values in racing pigeons (*Columba livia domestica*), *Avian Pathol* 14:401–408, 1985.
Mean ± standard error of mean.
ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; GT, glutamyl transferase; IU, International Units; LDH, lactic dehydrogenase.

Biochemistry Values for Mourning Doves (*Zenaida macroura*)

Blood Chemistry Values	Male	*n*	Female	*n*
Glucose (mmol/L)	26.53 ± 0.47 (19.48-36.80)	94	25.91 ± 0.47 (18.87-39.85)	87
Sodium (mmol/L)	144.0 ± 033 (137.0-154.0)	97	143.43 ± 0.41 (125.0-156.0)	87
Potassium (mmol/L)	7.78 ± 0.19 (4.0-14.9)	96	7.81 ± 0.18 (4.5-12.0)	87
Chloride (mmol/L)	113.64 ± 0.33 (107.0-121.0)	94	112.6 ± 0.42 (96.0-123.0)	88
Total protein (g/L)	26.2 ± 0.4 (18-39)	97	26.6 ± 0.5 (17.0-39.0)	87
Albumin (g/L)	12.1 ± 0.2 (9-17)	97	12.2 ± 0.2 (10-17)	87
Globulin (g/L)	14.2 ± 0.3 (9-26)	97	14.4 ± 0.3 (07-26)	87
Calcium (mmol/L)	2.36 ± 0.02 (1.8- ± 2.87)	97	2.43 ± 0.02 (1.6-3)	87
Phosphorus (mmol/L)	1.22 + 0.03 (0.61-2.39)	97	1.25 ± 0.03 (0.67-2.61)	87
Cholesterol (mmol/L)	5.79 ± 0.14 (3.05-13.75)	94	5.97 ± 0.19 (2.1-12.12)	87
Magnesium (mmol/L)	1.17 ± 0.01 (0.86-1.59)	97	1.17 ± 0.01 (0.86-1.51)	87
Uric acid (mmol/L)	429.44 ± 13.68 (178.44-844.61)	96	429.44 ± 18.43 (154.64-1017)	87
AST (IU/L)	252.60 ± 1137 (94-709)	97	270.07 ± 11.10 (143-659)	86
GGT (IU/L)	11.16 ± 0.40 (9-37)	94	10.87 ± 0.31 (9-27)	86
LDH (IU/L)	905.35 ± 36.38 (312-1822)	95	1175 ± 108.11 (320-8528)	87

Source: Modified from: Schulz JH, et al: Blood plasma chemistries from wild mourning doves held in captivity, *J Wildl Dis* 36:541–545, 2000.
Mean ± standard error of the mean (range).
AST, Aspartate aminotransferase; CK, creatine kinase; GGT, γ-glutamyl transferase; LDH, lactic dehydrogenase.

Plasma Chemistry Values for the Kori and Houbara Bustard

Species	Kori Bustard*		Houbara Bustard†	
Scientific Name	***Ardeotis kori***	***n***	***Chlamydotis undulata***	***n***
Glucose (mmol/L)	13.2 ± 0.46 (9.21-1.98)	24	16.0 ± 0.38 (12.3-22.4)	38
Uric acid (mmol/L)	469 ± 29.7 (208-850.5)	26	585 ± 24.9 (344-904)	36
Creatinine (mmol/L)	57 ± 4 (20-110)	24	34 ± 2 (10-70)	35
Total bilirubin (mmol/L)	11.9 ± 0.51 (5.13-2.22)	25	8.55 ± 0.34 (1.71-13.6)	35
Total protein (g/L)	29.6 ± 1.6 (20.0-52.0)	25	35.8 ± 0.8 (26.0-50.0)	37
Albumin (g/L)	15.9 ± 0.8 (12.0-31.0)	25	14.3 ± 0.3 (11.0-19.0)	38
Globulin (g/L)	13.0 ± 0.8 (8.0-24.0)	22	21.2 ± 0.6 (13.0-34.0)	36
Albumin/globulin ratio	1.20 ± 0.47 (0.70-2.10)	23	0.70 ± 0.02 (0.50-1.10)	36
GGT (IU/L)	13.25 ± 0.47 (12.0-14.0)	4	372.91 ± 13.29 (200.0-508.0)	37
ALT (IU/L)	16.17 ± 2.24 (4.0-52.0)	23	36.03 ± 2.40 (8.0-76.0)	31
ALP (IU/L)	—		137.28 ± 13.33 (24.0-333.0)	38
AST (IU/L)	226.50 ± 10.80 (200.0-251.0)	4	372.91 ± 13.29 (200.0-508)	37
LDH (IU/L)	3862.50 ± 307.0 (2637.0-4689.0)	6	—	
CK (IU/L)	135.60 ± 20.90 (47.0-510.0)	24	—	
Ammonia (mmol/L)	465.90 ± 47.40 (172.0-932.0)	21	309.93 ± 14.28 (174.0-486.0)	32
Carbon dioxide (mmol/L)	27.47 ± 4.41 (10.0-94)	19	26.24 ± 0.80 (19.0-38.0)	37
Magnesium (mmol/L)	0.35 ± 0.02 (0.12-0.78)	24	1.07 ± 0.03 (0.65-1.72)	38
Phosphorus (mmol/L)	1.32 ± 0.08 (0.83-2.36)	26	1.30 ± 0.10 (0.41-3.32)	38
Calcium (mmol/L)	3.11 ± 0.20 (1.52-5.27)	24	2.39 ± 0.08 (0.87-3.02)	38
Potassium (mmol/L)	2.94 ± 0.19 (1.80-6.10)	25	3.89 ± 0.14 (1.80-5.50)	35
Sodium (mmol/L)	154.48 ± 1.42 (145.0-174.0)	25	151.23 ± 2.30 (112.0-179.0)	38
Chloride (mmol/L)	403.69 ± 3.43 (381.5-444.5)	23	401.66 ± 4.9 (343.0-469.0)	34
Cholesterol (mmol/L)	3.11 ± 0.17 (1.70-4.99)	26	5.46 ± 0.21 (3.62-8.14)	33
Triglycerides (mmol/L)	1.21 ± 0.09 (0.68-2.54)	25	2.74 ± 0.21 (1.09-4.48)	24
VLDL (mg/dL)	21.18 ± 1.44 (12.0-37.0)	22	—	

Source: Modified from: D'Aloia M-A, Samour JH, Bailey TA, et al: Normal blood chemistry of the kori bustard (*Ardeotis kori*), *Avian Pathol* 25:161–165, 1996.

†*Source*: Modified from: D'Aloia M-A, Samour JH, Howlett JC, et al: Normal blood chemistry of the houbara bustard (*Chlamydotis undulata*), *Avian Pathology* 25:167–173, 1996.

Mean ± standard error of mean (minimum-maximum).

ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; GGT, γ-glutamyl transferase; IU, International Units; LDH, lactic dehydrogenase; VLDL, very-low-density lipoprotein.

Blood Chemistry Values for Captive American Flamingos (*Phoenicopterus ruber*)

Assay	Value	*n*
Alkaline phosphatase (IU/L)	166.35 ± 128.78* (18.26-737.55)	17
Aspartate	273.07 ± 103.39 (70.42-475.72)	27
Aminotransferase (IU/L)		
Calcium (mmol/L)	3.34 ± 1.07 (1.23-5.44)	24
Carbon dioxide total (mmol/L)	16.77 ± 3.72 (9.47-24.07)	13
Creatinine kinase (IU/L)	1058.40 ± 1096.12* (157.15-3521.44)	27
Uric acid (mmol/L)	768.4 ± 278.9 (221.8-1314.5)	28
Total protein (g/L)	40.6 ± 4.5 (31.8-49.4)	27
Glucose (mmol/L)	10.97 ± 2.56 (5.95-16.0)	26
Potassium (mmol/L)	2.84 ± 0.50 (1.86-3.83)	18
Chloride (mmol/L)	116.92 ± 3.25 (110.55-123.30)	13
Sodium (mmol/L)	12.92 ± 4.69 (3.73-22.10)	18

Source: Modified from: Merritt EL, Fritz CL, Ramsay EC: Hematologic and serum biochemical values in captive American flamingos (*Phoenicopterus ruber ruber*), *J Avian Med Surg* 10:163–167, 1996.

Mean ± SD (95% reference range).

Data obtained from 16 males, 8 females, and 1 bird of unknown sex. Samples from 5 birds were obtained twice and not all samples were evaluated for every test.

*Intervals obtained by calculation with logarithmic transformation of data.

Blood Chemistry Values for Selected Falconiformes

Species	Saker Falcon*,† (*n* = 30, 38)	Peregrine Falcon†,‡ (*n* = 55, 14)	Lanner Falcon† (*n* = 26)	Gyr Falcon§ (*n* = 53)	Merlin† (*n* = 39)
Scientific Name	***Falco cherrug***	***Falco peregrinus***	***Falco biarmicus***	***Falco rusticolus***	***Falco columbarius***
Glucose (mmol/L)	12-14	11-16	11-15	20.4 (1.7)	9-12 (1.7)
Cholesterol (mmol/L)	4.5-8.6	3.9-10.5	3-8.8	5.44 (1.03)	3-7.8 (1.03)
Triglycerides (mmol/L)	0.79-1.25	—	—	—	1.02 (0.27)
GGT (IU/L), 37°C	0.8-5.9	0-7	—	—	—
AST (GOT; IU/L) 37°C	45-95	50-105	30-118	149 (110)	50-125
ALT (GPT; IU/L) 37°C	36-55	15-51	—	135 (125)	
ALP (IU/L)	285-450	97-350	180-510	—	54-310
CK (IU/L)	355-651	357-850	350-650	—	521-807
LDH (IU/L)	551-765	625-1210	434-897	1917 (879)	329-630
Uric acid (mmol/L)	320-785	326-675	318-709	370 (170)	174-800
Urea (mmol/L)	0.5-2.6	0.9-2.8	1.3-2.7	3.6 (2.2)	—
Creatinine (mmol/L)	23-75	41-91	37-75	38 (14)	16-50
Bile acids (mmol/L)	20-90	20-118	—	—	—
Bilirubin (mmol/L)	146.2-470.7	1336.3	—	78.6(29.07)	—
Total protein (g/L)	270-360	250-400	330-420	250 (87)	275-390
Albumin (g/L)	90-123	83-125	96-160	118 (17)	86-161
Globulin (g/L)	180-280	160-280	212-288	0.47-0.58	172-250
A:G ratio	0.45-0.57	0.4-0.55	0.44-0.57	0.47-0.58	0.47-0.58
Amylase (IU/L)	—	—	—	86 (116)	—
Potassium (mmol/L)	0.8-2.3	0.9-1.7	1-2.1	3.1 (0.9)	1-1.8
Chloride (mmol/L)	114-125	117-127	—	124 (8)	—
Sodium (mmol/L)	154-161	153-164	152-164	154 (12)	155-170
Calcium (mmol/L)	—	—	—	2.30 (0.27)	—
Phosphorus (mmol/L)	—	—	—	1.52 (0.40)	—

*Source: Modified from: Samour JH, D' Aloia M-A: Blood chemistry of the saker falcon (*Falco cherrug*), *Avian Pathol* 25:175–178, 1996.
†Source: Modified from: Jennings IB: Haematology. In Beynon PH, Forbes NA, Harcourt-Brown NH editors: *Manual of raptors, pigeons and waterfowl*, Cheltenham, UK, 1996, British Small Animal Veterinary Association, pp 68–78.
‡Altman RB, et al: *Appendix 1, Avian Medicine and surgery*, Philadelphia, PA, 1997, WB Saunders, pp 1004–1024.
§Lierz M: Plasma chemistry reference values for gyr falcons (*Falco rusticolus*). *Veterinary Record* 153:182–183, 2003.
ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; GGT, γ-glutamyl transferase; IU, International Units; LDH, lactic dehydrogenase.

Blood Chemistry Values for the Eurasian Buzzard (*Buteo buteo*)

Assay	*n* = 20	Assay	*n* = 20
Albumin (g/L)	145	Amylase (IU/L)	616.93
Total protein (g/L)	38.4	Uric acid (mmol/L)	218.0
Glucose (mmol/L)	18.7	Creatinine (mmol/L)	13.2
AST (U/L)	330.9	Urea (mmol/L)	2.12
ALT (U/L)	40.6	Calcium (mmol/L)	2.16
LDH (U/L)	2008.4	Phosphorus (mmol/L)	0.71
CK (U/L)	1604.1	Magnesium (mmol/L)	0.99
GGT (U/L)	0.3	Prealbumin (g/L)	3.06
ALP (U/L)	89.8	Albumin (g/L)	14.65
Total bilirubin (mmol/L)	0.51	α-globulin (g/L)	4.89
Cholesterol (mmol/L)	4.97	β- Globulin (g/L)	5.78
Triglycerides (mmol/L)	1.31	γ- Globulin (g/L)	13.08
Lipase (IU/L)	26.37		

Source: Modified from: Gelli D, Ferrari V, Franceschini F, et al: Serum biochemical and electrophoretic patterns in the Eurasian buzzard (*Buteo buteo*)—reference values, *Proceedings of the European Association of Avian Veterinarians*, Arles, 2005, pp 166–170.
ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; GGT, γ-glutamyl transferase; IU, International Units; LDH, lactic dehydrogenase; VLDL, very-low-density lipoprotein.

Blood Chemistry Values for Selected Falconiformes

Species	Bald Eagle	Peregrine Falcon	Red-Tailed Hawk	Great-Horned Owl
Scientific Name	***Haliaeetus leucocephalus***	***Falco peregrinus***	***Buteo jamaicensis***	***Bubo virginianus***
Acetyl cholinesterase delta pH (units/hr)	0.16 (0.06)	—	—	—
Alanine aminotransferase (IU/L)	25 (13)	62 (56)	31 (5)	39 (14)
Albumin (g/L)	10.9 (1.8)	9.6 (1.3)	13.4 (4.1)	12.7 (3.5)
Alkaline phosphatase (IU/L)	57 (12)	99 (44)	53 (18)	31 (7)
Amylase (IU/L)	1158 (376)	—	—	—
Aspartate aminotransferase (IU/L)	218 (63)	78 (31)	303 (22)	287 (65)
Bilirubin, total (mmol/L)	5.30 (1.36)	78.1 (34.8)	2.73 (1.36)	1.19 (1.02)
Blood urea nitrogen (mmol/L)	2.21 (1.76)	2.32 (0.09)	3.33 (0.33)	3.57 (2.09)
Calcium (mmol/L)	2.48 (0.11)	2.23 (0.11)	—	2.54
Chloride (mmol/L)	120 (3)	114.38 (43.36)	125 (3)	122
Creatine kinase (IU/L)	383 (300)	783 (503)	1124 (251)	977 (407)
Creatinine (mmol/L)	61.88 (22.9)	45.08 (19.4)	—	—
Glucose (mmol/L)	16.76 (1.38)	20.3 (1.60)	19.7 (0.88)	19.7
Osmolality (mmol/kg)	319 (6)	—	—	—
Phosphorus (mmol/L)	0.97 (0.16)	1.08 (0.22)	1.01 (0.16)	1.40
Potassium (mmol/L)	3.0 (0)	2.04 (0.81)	2.42 (0.73)	2.8
Protein, total (g/L; biuret)	35.1 (7.5)	26.3 (4.8)	41.7 (6.9)	43.3
Sodium (mmol/L)	156 (4)	143 (54)	157 (1)	156
Uric acid (mmol/L)	301.5 (198.0)	267.6 (252.1)	644 (303.3)	814.8 (642.0)

Source: Modified from: Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders, Co., Information from Professor Patrick Redig, Raptor Center, University of Minnesota, Minneapolis.

Blood Chemistry Values for Selected Falconiformes

Species	Golden Eagle*	Bald Eagle*	Tawny Eagle† (*n* = 29)	Red-Tailed Hawk*,‡	Harris Hawk*
Scientific Name	***Aquila chrysaetos***	***Haliaeetus leucocephalus***	***Aquila rapax***	***Buteo jamaicensis***	***Parabuteo unicinctus***
Glucose (mmol/L)	13.8-22.6	15.8-22.2	10.19-14.4	17.3-22.8 16.9-22.2	16.2-21.6
Cholesterol (mmol/L)	—	3.87-6.25	7.90-10.70	2.58-3.87	2.58-3.87
Triglycerides (mmol/L)	—	—	—	—	1.69-3.16
GGT (IU/L), 37°C	—	—	1-2.7	—	—
GOT (IU/L), 37°C	95-210	153-370	124-226	113-180 136-307	—
GPT (IU/L), 37°C	—	—	—	—	—
Uric acid (mmol/L)	261-713.7	329.5-285.5	412-575	446.1-1058.7 481.7-999.2	523.4-1278.8
Creatinine (mmol/L)	0.5-1.2	0.4-1	0.3-0.59	0.5-1.2	0.7-1.5
Bilirubin (mmol/L)	5.13-8.55	3.42-8.55	—	8.55-10.26	8.55-20.5
Total protein (g/L)	25-39	30-41	29-41	33-45 4.8	39-52
Potassium (mmol/L)	—	—	1.5-3.1	2.6-4.3	—

*Source: Modified from: Ivins GK, Weddle GD, Haliwell WH: Hematology and serum chemistries in birds of prey. In Fowler ME, editor: *Zoo and wildlife medicine*, edn 2, Philadelphia, 1986, WB Saunders, pp 286–290.
†Modified from: Jennings IB: Haematology. In Beynon PH, Forbes NA, Harcourt-Brown NH: *Manual of Raptors, Pigeons and Waterfowl*, Cheltenham, 1996, British Small Animal Veterinary Association, pp 68–78.
‡Modified from: Kollias GV, Mc Leish J: Effects of ketamine hydrochloride in red-tailed hawks (*Buteo jamaicensis*), biochemical and hematology, *Comp Biochem Physiol* 60:211, 1978.
Mean ± SD (minimum-maximum)
GGT, γ-glutamyl transferase; GOT, glutamic oxaloacetic transaminase; GPT, glutamic-pyruvic transaminase; IU, International Units.

Reference Intervals for Selected Blood Chemistry Parameters in Wild (*n* = 14) and Captive (*n* = 25) African White-Backed Vultures

Parameter	Mean	SD	Min	Max	Reference Interval
UA (mM/L)*	0.65	0.03	0.22	1.195	0.58-0.724
U (mM/L)*	3.21	1.01	1.10	4.7	1.18-5.23
U:UA*	5.47	1.39	2.20	12.7	1.392.69
Alb (g/L)†,‡	11.69	1.06	10.50	13.6	10.37-13.18
Globulin (g/L)‡	21.78	1.14	16	29	16.76-28.30
ALT (U/L)†,‡	35.51	1.99	6.00	95.00	9.233-144.45
AST (U/L)‡	1,620.92	2.24	241	5,120	238-6160
CK (U/L)†,‡	287.47	137.00	136.00	631.00	112.85-596.15

*Samples represent arithmetic mean and SD.
†Results obtained from the captive birds.
‡Results represent geometric mean and SD.
UA, uric acid; U, urea; U:UA, urea:uric acid ratio; Alb, albumin; ALT, alanine aminotransferase; AST, aspartate aminotransferase; CK, creatinine kinase.

Parameters from Wild Birds for which Normality could not be Established

Parameter*	Mean	SD	Min	Max	LCI	UCI
Ca2+ (mM/L)	1.71	0.77	0.77	2.87	1.49	1.93
K+ (mM/L)	1.41	0.61	0.66	3.12	1.16	1.67
Na+ (mM/L)	153.70	12.17	108	176	148.59	158.87
CK (U/L)	16,910	14,988	415	56,080	10,856	22,964
Alb (g/L)	12.79	0.88	11	15	12.41	13.10
TP (g/L)	34.96	3.75	27	43	33.30	36.54

Source: Naidoo V, Diekmann M, Wolters K, et al: Establishment of selected baseline blood chemistry and hematologic parameters in captive wild-caught African white-backed vultures (*Gyps africanus*), *J Wildlife Dis* 44(3):649–654, 2008.
*Alb, Albumin; CK, creatinine kinase; LCI, 95% lower confidence interval; TP, total proteins; UCI, 95% upper confidence interval.

Blood Chemistry Values for Selected Psittaciformes

Species	Cockatiel *n* = 364	Conure *n* = 85	Grand Eclectus parrot *n* = 9	Philippine Blue-Naped Parrot *n* = 7	Lovebird *n* = 78	Blue-Headed Parrot *n* = 16	Gray-Cheeked Parakeet *n* = 32
Scientific Name	***Nymphicus hollandicus***	***Aratinga* spp.**	***Eclectus roratus riedeli***	***Tanygnathus lucionensis***	***Agapornis* spp.**	***Pionus menstruus***	***Brotogeris pyrrhopterus***
Total protein (g/L)	22-50	25-45	30-50	30-50	22-51	26-50	25-45
Glucose (mmol/L)	13.8-24.9	13.8-19.4	9.99-19.9	10.5-19.4	11.1-22.2	9.99-16.6	11.1-19.4
Calcium (mmol/L)	2.12-3.25	2.0-3.75	2.25-4.0	2.5-4.0	2.25-3.75	2.5-3.75	—
SGOT (IU/L)	100-350	125-350	130-350	130-350	100-350	150-350	150-400
LDH (IU/L)	125-450	125-420	100-200	130-425	100-350	200-550	150-450
Creatinine (mmol/L)	8.84-35.3	8.84-44.2	8.84-35.3	8.84-35.3	8.84-35.3	8.84-26.5	8.84-35.4
Uric acid (mmol/L)	208.1-654	148.7-624.5	178.4-594.8	237-594.8	178.0-654.0	237.9-713.7	237.9-713.0
Potassium (mmol/L)	2.5-4.5	3.4-5.0	—	—	2.5-3.5	3.0-4.5	—
Sodium (mmol/L)	132-150	134-148	—	—	137-150	130-150	—
Thyroxine (mmol/L)	9.0-30.8	3.21-11.5	6.43-12.8	3.86-12.87	2.57-24.4	2.57-14.1	2.57-30.8

Source: Modified from: Woerpel RW, Rosskopf WJ: Clinical experience with avian laboratory diagnostics, *Vet Clin North Am* 14:254, 1984.
Source: Modified from: Woerpel RW, Rosskopf WJ, Monahan-Brennan M: Clinical pathology and laboratory diagnostic tools. In Burr EW, editor: *Companion bird medicine*, Ames, IA, 1987, Iowa State University Press, pp 180–196.
(Minimum-maximum).
ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; IU, International Units; LDH, lactic dehydrogenase; SGOT, serum glutamic oxaloacetic transaminase.

Blood Chemistry Values for Selected Psittaciformes—cont'd

Species	Budgerigar	African Gray Parrot	Amazon Parrot	Cockatoo
Scientific Name	***Melopsittacus undulatus***	***Psittacus erithacus***	***Amazona* spp.**	***Cacatua* spp.**
Total protein (g/L)	20-30	26-49	33-53	28-43
Ca (mmol/L)	1.6-2.8	1.75-2.3	1.87-2.42	1.9-2.22
P (mmol/L)	0.9-1.9	1.0-5.2	0.8-3.4	1.0-3.6
Uric acid (mmol/L)	178.4-511.5	184.3-416.3	77.3-333	208.1-553.1
Creatinine (mmol/L)	8.84-35.3	8.84-35.3	8.84-35.3	8.84-35.3
AST (IU/L)	55-154	28-200	35-200	32-180
ALT (IU/L)	5-20	2-21	4-13	5-12
LDH (IU/L)	154-271	105-420	65-420	130-353
CK (IU/L)	54-252	71-408	64-322	27-253
ALP (IU/L)	54-326	24-94	93-311	32-171
Amylase (IU/L)	187-582	211-519	106-524	—
Glucose (mmol/L)	14.5-22.1	12.4-17.0	12.2-16.6	11.6-17.6
Cholesterol (mmol/L)	4.44-7.39	5.61-8.53	4.68-8.01	—
Triglycerides (mmol/L)	1.23-3.05	0.57-1.58	0.66-2.25	—
K (mmol/L)	2.2-3.7	2.2-3.5	2.1-3.3	—
Na (mmol/L)	139-159	146-167	127-158	—
Cl (mmol/L)	95-144	110-128	97-127	—

Source: Modified from: Hochleithner M: References values for selected psittacine species using a dry chemistry system, *J Assoc Avian Veterinarians* 3:207–209, 1989.
Kodak Ektachem-25°C.
ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; IU, International Units; LDH, lactic dehydrogenase.

Blood Chemistry Values for Selected Psittaciformes—cont'd

Species	Hyacinthine Macaw	Blue and Yellow Macaw	Green-Winged Macaw	Scarlet Macaw	Military Macaw
Scientific Name	***Anodorhynchus hyacinthinus* n = 13**	***Ara ararauna* n = 30**	**Ara chloroptera n = 16**	***Ara macao* n = 12**	***Ara militaris* n = 16**
Glucose (mmol/L)	14.5 ± 0.9 (13.2-15.9)	15.1 ± 2.1 (10.1-19.0)	16.0 ± 1.7 (13.2-18.9)	13.8 ± 0.9 (12.0-15.2)	15.4 ± 1.7 (12.0-18.3)
Urea (mmol/L)	1.3 ± 0.4 (0.6-22.2)	1.3 ± 0.5 (0.4-2.1)	1.3 ± 0.6 (0.5-2.2)	1.9 ± 0.7 (1.2-3.9)	1.2 ± 0.7 (0.2-2.5)
Uric acid (mmol/L)	347 ± 136 (127-641)	296 ± 147 (113-629)	401 ± 216 (151-829)	277 ± 161 (143-583)	466 ± 210 (144-842)
Cholesterol (mmol/L)	3.1 ± 0.6 (2.2-4.3)	4.2 ± 0.9 (3.1-6.7)	4.2 ± 0.9 (2.1-5.3)	4.3 ± 1.1 (2.3-6.4)	4.2 ± 1.3 (1.7-6.6)
Triglycerides (mmol/L)	0.8 ± 0.3 (0.4-1.3)	1.2 ± 0.7 (0.4-2.5)	1.3 ± 0.5 (0.7-2.1)	1.0 ± 0.3 (0.5-1.6)	1.0 ± 0.5 (0.3-1.8)
Creatinine (mmol/L)	45.7 ± 8.8 (33.7-62.8)	49.1 ± 15.1 (26.1-76.8)	62.5 ± 14.3 (33.5-78.6)	49.3 ± 9.6 (31.6-61.0)	47.4 ± 8.9 (24.2-60.3)
	***n* = 12**	***n* = 29**	***n* = 15**	***n* = 7**	***n* = 14**
LDH (IU/L)	160 ± 90.7 (59.7-309)	140 ± 81.3 (61.7-349)	226 ± 91.6 (113-422)	368 ± 176 (132-610)	307 ± 101 (121-485)
AST (IU/L)	56.7 ± 12.8 (38.4-90.6)	56.2 ± 19.1 (33.0-105)	68.1 ± 24.1 (44.0-139)	49.0 ± 11.2 (34.2-65.6)	97.0 ± 65.5 (37.3-228)
ALT (IU/L)	7.5 ± 3.4 (2.5-14.4)	8.1 ± +3.3 (3.5-15.7)	6.4 ± 3.5 (1.7-13.2)	8.8 ± 5.4 (1.3-16.8)	13.2 ± 7.0 (2.6-23.7)
CPK (IU/L)	142 ± 128 (16.7-351)	131 ± 109 (35.4-428)	86.9 ± 45.4 (25.1-151)	64.0 ± 41.9 (20.3-132)	136 ± 167 (10.1-430)
ALP (IU/L)	45.6 ± 21.9 (12.7-82.9)	194 ± 81.9 (79.0-357)	132 ± 30.0 (86.1-177)	80.9 ± 23.1 (51.7-99.9)	75.9 ± 44.0 (27.6-156)
γ-GT (IU/L)	—	3.7 ± 5.8 (0.0-18.5)	0.5 ± 0.7 (0.0-2.0)	0.4 ± 0.5 (0.0-1.1)	3.0 ± 3.8 (0.0-11.8)
	***n* = 7**	***n* = 17**	***n* = 8**	***n* = 14**	***n* = 11**
Osmolality (mOsmol/kg)	298 ± 17.6 (263-319)	319 ± 6.2 (309-328)	315 ± 14.2 (251-339)	331 ± 24.0 (281-366)	29.6 ± 8.2 (17.2-44.4)
Sodium (mmol/L)	151 ± 20.7 (135-191)	150 + 28.6 (119-252)	145 ± 6.3 (141-159)	—	9.5 ± 6.2 (0.0-18.5)
Potassium (mmol/L)	4.9 ± 1.7 (3.3-7.9)	2.4 ± 0.8 (1.7-4.7)	2.7 ± 0.5 (1.9-3.2)	—	50.0 ± 12.0 (34.5-70.6)
Chloride (mmol/L)	102 ± 11.1 (83-120)	101 ± 9.1 (75-122)	103 ± 8.3 (91-120)	96 ± 4.8 (90-108)	10.5 ± 3.9 (5.8-18.3)
Calcium (mmol/L)	2.2 ± 0.4 (1.9-2.9)	2.3 ± 0.4 (1.7-3.2)	2.3 ± 0.3 (2.0-2.9)	—	17.8 ± 7.2 (8.1-32.5)
Phosphorus (mmol/L)	3.0 ± 0.4 (2.3-3.4)	4.5 ± 1.1 (3.0-6.2)	4.1 ± 1.2 (2.4-5.8)	—	12.1 ± 5.1 (1.8-19.6)
Magnesium (mmol/L)	1.2 ± 0.5 (0.9-2.0)	1.0 ± 0.2 (0.7-1.4)	1.3 ± 0.5 (0.9-2.2)	—	1.6 ± 0.5 (0.6-2.4)

Source: Modified from: Polo FJ, Peinado VI, Viscor G, et al: Hematologic and plasma chemistry in captive psittacine birds, *Avian Dis* 42:523–535, 1998.
Mean ± SD (minimum-maximum).
ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine phosphokinase; IU, International Units; LDH, lactic dehydrogenase.

Serum Biochemistry Values for Juvenile Eclectus Parrot (*Eclectus roratus*)

Blood Chemistry Values	30 Day	90 Day	All
Na (mmol/L)	141 (2)	154 (3)	148 (6)
K (mmol/L)	2.9 (1.0)	2.7 (0.6)	2.8 (0.7)
Cl (mmol/L)	105 (3)	115 (3)	111 (5)
Ca (mmol/L)	2.37 (0.12)	2.77 (0.1)	2.32 (0.1)
Phosphorus (mmol/L)	2.55 (0.25)	1.84 (0.29)	2.19 (0.38)
Urea (mmol/L)	0.25 (0.38)	0.33 (0.51)	0.28 (0.40)
Creatinine (mmol/L)	26.5 (8.84)	35.3 (8.84)	35.3 (8.84)
UA (mmol/L)	47.5 (53.5)	231.9 (89.2)	118.9 (95.1)
Cholesterol (mg/dL)	181 (43)	300 (69)	268 (80)
Glucose (mmol/L)	13.8 (0.88)	14.7 (1.05)	14.5 (0.99)
LDH (IU/L)	235 (145)	268 (70)	228 (101)
AST (IU/L)	85 (21)	216 (47)	140 (58)
ALT (IU/L)	4 (3)	7 (3)	4 (3)
ALP (IU/L)	421 (85)	565 (217)	489 (159)
GGT (IU/L)	5 (2)	2 (1)	4 (2)
CK (IU/L)	555 (164)	643 (262)	616 (472)
TP (g/L)	26 (4)	29 (04)	29 (5)
ALB (g/L)	12 (2)	13 (2)	13 (3)
GLOB (g/dL)	13 (3)	16 (3)	15 (3)
A:G ratio	0.9 (0.1)	0.8 (0.1)	0.9 (0.2)
ALB (Elect; g/L)	18 (5)	21 (4)	22 (4)
GLOB (Elect; g/L)	7 (2)	7 (2)	8 (2)

Source: Modified from: Clubb SL, Schubot RM, Joyner K, et al: Hematologic and serum chemistry reference intervals in juvenile eclectus parrots, *J Assoc Avian Veterinarians* 4:218–225, 1990.
Mean (SD).
ALB, Albumin; ALT, alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; GGT, γ-glutamyl transferase; GLOB, globulin; IU, International Units; LDH, lactic dehydrogenase; TP, total protein.

Serum Biochemistry Values for Juvenile Cockatoos (*Cacatua* spp.)

Blood Chemistry Values	30 Day	90 Day	All
Na (mmol/L)	139 (3)[a]	139 (3)[c]	145 (6)
K (mmol/L)	4.0 (0.8)[a]	3.10 (0.4)[b]	3.6 (0.7)
Cl (mmol/L)	105 (4)[a]	115 (4)[c]	110 (6)
Ca (mmol/L)	2.3 (0.15)[a]	2.3 (0.25)[a,b]	2.4 (0.17)
Phosphorus (mmol/L)	2.26 (0.19)[a]	1.64 (0.38)[c]	1.97 (0.35)
Urea (mmol/L)	0.26 (0.31)[a]	0.43 (0.41)[b]	0.33 (0.36)
Creatinine (mmol/L)	27.4 (5.30)[a]	37.1 (6.1)[a,b]	35.3 (8.84)
UA (mmol/L)	71.3 (53.5)[a]	303 (107)[c]	172.4 (136.8)
Cholesterol (mmol/L)	4.26 (0.82)[a]	9.05 (3.1)[b]	6.49 (2.71)
Glucose (mmol/L)	13.7 (1.11)[a]	13.8 (1.60)[a,b]	14.0 (1.33)
LDH (IU/L)	393 (348)[a]	367 (218)[a]	371 (285)
AST (IU/L)	98 (54)[a]	195 (73)[c]	143 (79)
ALT (IU/L)	2 (2)[a]	3 (3)[a,b]	2 (3)
ALP (IU/L)	593 (202)[a]	478 (167)[c]	579 (239)
GGT (IU/L)	2.35 (1.75)[a]	2.79 (1.54)[a,c]	2.55 (1.67)
CK (IU/L)	595 (205)[a]	368 (156)[b]	510 (235)
TP (g/L)	22 (4)[a]	31 (6)[b]	28 (7)
ALB (g/L)	8 (2)[a]	12 (3)[b]	11 (3)
GLOB (g/L)	13 (4)[a]	19 (4)[b]	17 (5)
A:G ratio	0.6 (0.2)[a,b]	0.6 (0.1)[b]	0.6 (0.2)
PRE-ALB (g/L)	4 (1)[a]	5 (2)[b]	5 (2)
ALB (Elect; g/L)	11 (3)[a]	7 (5)[b,c]	15 (5)
α-GLOB (g/L	2 (1)[a]	3 (2)[c]	2 (1)
β-GLOB (g/L)	3 (2)[a]	3 (1)[a]	3 (1)
γ-GLOB (g/L)	2 (1)[a]	3 (1)[b]	3 (1)

Source: Modified from: Clubb SL, Schubot RM, Joyner K, et al: Hematologic and serum biochemical reference intervals in juvenile cockatoos, *J Assoc Avian Veterinarians* 5:16–26, 1991.
Mean (SD).
[a,b,c] = Values for parameters are statistically different when letters are different.
ALB, Albumin; ALT, alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; GGT, γ-glutamyl transferase; GLOB, globulin; IU, International Units; LDH, lactic dehydrogenase; TP, total protein.

Serum Biochemistry Values for Juvenile Umbrella Cockatoos (*Cacatua alba*)

Blood Chemistry Values	30 Day	90 Day	All
Na (mmol/L)	139 (1.78)[s]	149 (2.33)[n]	145
K (mmol/L)	4.23 (0.57)[n]	3.13 (0.44)[n]	3.54
Cl (mmol/L)	107 (2.8)[s]	115 (3.2)[n]	111
Ca (mmol/L)	2.41 (0.09)[s]	2.35 (0.32)[n]	2.44
Phosphorus (mmol/L)	2.09 (0.14)[s]	1.51 (0.28)[n]	1.79
Urea (mmol/L)	0.16 (0.29)[n]	0.32 (0.40)[s]	0.26
Creatinine (mmol/L)	30.0 (6.1)[n]	29.1 (3.53)[s]	32.7
UA (mmol/L)	49.3 (21.4)[s]	294.4 (99.9)[n]	162.3
Cholesterol (mmol/L)	4.65 (0.95)[s]	11.0 (1.81)[s]	7.52
Glucose (mmol/L)	13.5 (1.0)[n]	13.1 (1.56)[s]	13.5
LDH (IU/L)	326 (394)[n]	341 (174)[n]	325
AST (IU/L)	84 (17.7)[n]	187 (39.2)[n]	136
ALT (IU/L)	1.8 (1.7)[n]	2.69 (1.58)[n]	2.11
ALP (IU/L)	426 (100)[s]	404 (104)[s]	440
GGT (IU/L)	1.95 (1.73)[n]	2.81 (1.33)[n]	2.66
CK (IU/L)	629 (193)[n]	395 (115)[n]	517
TP (g/L)	24.7 (4.1)[s]	32.5 (5.9)[n]	30.3
A:G ratio	0.6 (0.1)	0.62 (0.08)	0.64
PRE-ALB (g/L)	4.3 (1.2)[n]	4.9 (1.3)[n]	4.5
ALB (Elect; g/L)	12.7 (2.7)[s]	18.6 (3.5)[n]	16.9
α-GLOB (g/L)	1.7 (0.5)[n]	2.9 (1.9)[n]	2.6
β-GLOB (g/L)	3.9 (1.6)[n]	3.4 (1.4)[n]	3.8
γ-GLOB (g/dL)	2.3 (0.6)[n]	3.1 (1.1)[n]	2.9

Source: Modified from: Clubb SL, Schubot RM, Joyner K, et al: Hematologic and serum biochemical reference intervals in juvenile cockatoos, *J Assoc Avian Veterinarians* 5:16–26, 1991.
Mean (SD).
s, means statistically different ($p > 0.05$) from the same parameter in all juvenile cockatoos; n, means it is not statistically different ($p < 0.05$) from the same parameter in all juvenile cockatoos.
ALB, Albumin; ALT, alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; GGT, γ-glutamyl transferase; GLOB, globulin; IU, International Units; LDH, lactic dehydrogenase; TP, total protein.

Plasma Biochemical Reference Values in Captive Adult, Clinically Normal Indian Peafowl (*n* = 69)

Parameter	P2.5-P97.5*	Mean	SD	Range
Alkaline phosphatase (IU/L)	140-457	290	95	130-467
Alanine aminotransferase (IU/L)	0.0-6.4	1.7	1.7	0.0-7.2
Amylase (IU/L)	1364-2555	1810	300	1193-2617
Aspartate aminotransferase (IU/L)	49-198	124	38	19-222
Bile acids (μmol/L)[†]	25.0-88.0	51.1	18.0	22.0-90.0
Total bilirubin (mg/dL)	0.0-0.3	0.2	0.1	0.0-0.5
Blood urea nitrogen (mg/dL)	0.0-4.4	1.2	1.2	0.0-6.6
Calcium (mg/dL)	11.7-15.7	13.4	1.2	11.6-17.2
Cholesterol (mg/dL)	105-202	147	24	103-212
Creatinine (mg/dL)	0.6-1.3	0.8	0.2	0.6-1.3
Creatine kinase (IU/L)	906-2784	1482	453	830-3063
Glucose (mg/dL)	324-492	413	44	321-496
Gamma glutamyltransferase (IU/L)	0.0-7.3	1.6	2.3	0.0-7.7
Lactate dehydrogenase (IU/L)	98-437	232	77	74-508
Iron (μg/dL)	93-192	137	27	84-209
Phosphorus (mg/dL)	3.9-12.7	6.5	2.1	3.3-14.1
Uric acid (mg/dL)	0.8-5.8	3.0	1.2	0.5-6.1

Source: Samour J, Naldo J, Rahman H, et al: Hematologic and plasma biochemical reference values in Indian Peafowl (*Pavo cristatus*), *J Avian Med Surg* 24(2):99–106, 2010.
*Recommended reference ranges.
[†]n = 68.

Interspecies comparison of Mean Values in Several Biochemical Parameters in Crested and Common Coots

Biochemical Parameters	Crested Coot*	Common Coot*	*p*-Value[†]	1–β[‡]
Uric acid (mg/dL)	2.22 ± 1.14 (28), 51.35%	7.43 ± 2.47 (21), 33.24%	0.000	1.00
Urea (mg/dL)	8.74 ± 3.35 (19), 38.32%	43.3 ± 17.6 (20), 40.64%	0.000	1.00
Cholesterol (mg/dL)	157.25 ± 44.45 (24), 28.26%	251.61 ± 52.85 (21), 21%	0.000	1.00
Triglycerides (mg/dL)	116.42 ± 46.49 (27), 39.93%	172.23 ± 68.06 (21), 39.51%	0.001	0.95
Lactate dehydrogenase (IU/L)	575.66 ± 451.97 (23), 78.51%	985.73 ± 394.22 (14), 39.99%	0.004	0.78
Creatine kinase (IU/L)	189.16 ± 274.06 (16), 144.88%	1841.93 ± 1450.19 (12), 78.73%	0.000	0.99

Source: Rubio MD, Ildefonso N, Aguera RJ, et al: Plasma biochemistry and hematology of crested coots (*Fulica cristata*) and common coots (*Fulica atra*) from Spain. Comparative Clinical Pathology, 2012.
*Values shown are mean ± standard deviation (sample size), coefficient of variation.
[†]*p*-value of the ANOVA was obtained after conducting 10,000 Monte Carlo simulations with the original data set (see Manly, 1997). Significant values are shown in bold type.
[‡]1–β power of the test (probability of rejecting the null hypothesis when it is in fact false (Zar, 1999).

Plasma Biochemistry Values of Captive Horned Guan (*Oreophasis derbianus*; *n* = 32) Calculated Using the Abaxis Vet Scan VS2

Analyte	Mean or Median*	RI
Albumin		
g/L	24	19-29
g/dL	2.4	1.9-2.9
Albumin : Globulin (g/dL)	2.3	0.8-3.7
Aspartate transaminase (IU/L)	54.5*	31-212
Bile acids (μmol/L)	14.0*	0.0-141
Calcium		
mmol/L	2.6	2.2-3.0
mg/dL	10.3	8.8-11.9
Ca : P	2.6	1.1-4.2
Creatine kinase (IU/L)	1,172*	506-3,741
Glucose		
mmol/L	15	12-19
mg/dL	280	217-344
Globulins		
g/L	10*	6.0-2.3
g/dL	1.0	0.6-2.3
Potassium (mmol/L)	3.9	2.4-5.3
Sodium (mmol/L)	154	145-164
Phosphorous		
mmol/L	1.4	0.5-2.2
mg/dL	4.3	1.7-6.9
Total protein		
g/L	35	25-45
g/dL	3.5	2.5-4.5
Uric acid		
μmol/L	484	7.1-955.8
mg/dL	8.2	0.12-16.1

Source: Cornejo J, Richardson D, Perez JG: Hematologic and plasma biochemical reference values of the horned guan, *Oreophasis derbianus, J Zoo Wildlife Med* 45(1):15–22, 2014.
RI indicates the reference interval. Ca:P ratio calculated in mg/dL.

Blood Chemistry for Canary Finches (*Serinus canaria*)

Blood Chemistry Values	Mean	SD	P2.5-P97.5
Ca (mmol/L)	1.99	0.46	1.27-3.35
P (mmol/L)	1.05	0.39	0.51-1.80
Na (mmol/L)	139.2	8.18	125-154
Cl (mmol/L)	108.88	8.85	93-123
K (mmol/L)	3.58	0.69	2.7-4.8
Glucose (mmol/L)	19.19	1.68	16.1-21.7
Triglycerides (mmol/L)	2.08	0.62	1.35-3.52
Creatinine (mmol/L)	42.4	22.1	8.84-88.4
NH_3 (mmol/L)	221.18	110.42	87-467
ALT (IU/L)	11.58	7.92	2-30
AST (IU/L)	98.93	34.73	45-170
LDH (IU/L)	1582.63	325.72	1580-1816[m] 1300-1632[f]
ALP (IU/L)	265.05	79.62	146-397
Cholesterol (mmol/L)	4.27	1.15	2.84-7.39
Amyl (IU/L)	481.78	141.84	277-787
CK (IU/L)	302.1	106.94	177-556
TP (g/L)	2.84	0.75	2.0-4.4
Uric acid (mmol/L)	531.1	196.8	255.7-880.3

Source: Modified from: Schöpf A, Vasicek L: Blood chemistry in canary finches (*Serinus canaria*), *Proceedings of the European Association of Avian Veterinarians*, Vienna, 1991, pp 437–439.
Kodak Ektachem –25°C.
m = male; f = female.
ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; IU, International Units; LDH, lactic dehydrogenase; TP, total protein.

Age-Related Blood Chemistry Values in Captive Masai Ostrich (*Struthio camelus massaicus*), (*n* = 24)

Variable	*p*	Months 1	2	3	4	5	6	7	8	9	10	11	12
Bilirubin total (μmol/L)	<0.001	1.19 ± 0.34*	0.68 ± 0.34	2.05 ± 0.34	0.51 ± 0.17	1.19 ± 0.51	1.88 ± 3.93	0.51 ± 0.17	1.36 ± 0.17	2.05 ± 0.34	2.56 ± 0.34	2.22±0.34	2.56 ± 0.34
		0.00-5.13†	0.00-6.49	0.00-5.13	0.00-1.71	0.00-5.47	0.00-5.13	0.00-1.71	0.00-3.93	0.17-8.20	0.68-8.03	0.17-7.01	0.17-6.84
BUN (mmol/L)	<0.001	0.03 ± 0.05	0.14 ± 0.02	0.22 ± 0.05	0.12 ± 0.12	0.10 ± 0.01	0.09 ± 0.00	0.73 ± 0.01	0.04 ± 0.01	0.27 ± 0.04	0.06 ± 0.02	0.29±0.03	0.21 ± 0.02
		0.00-1.35	0.02-0.56	0.01-1.28	0.03-0.99	0.03-0.20	0.01-0.15	0.10-0.27	0.00-0.21	0.00-0.55	0.00-0.34	0.01-0.50	0.00-0.45
Calcium (mmol/L)	0.004	2.30 ± 0.10	2.20 ± 0.10	4.97 ± 0.10	2.95 ± 0.15	2.67±0.02	2.97 ± 0.07	3.32 ± 0.02	2.87 ± 0.05	3.07 ± 0.07	3.60 ± 0.05	3.67 ± 0.05	3.52 ± 0.05
		1.10-3.72	0.25-2.50	1.90-3.75	2.10-4.15	2.45-2.92	2.50-3.65	2.97-3.60	2.65-3.65	2.65-4.20	3.30-4.12	3.17-4.15	3.05-4.17
Cholesterol (mmol/L)	<0.001	3.80 ± 0.18	3.23 ± 0.19	5.31 ± 0.34	4.10 ± 0.20	4.02 ± 0.23	4.01 ± 0.21	3.14 ± 0.16	3.22 ± 0.15	2.53 ± 0.17	2.93 ± 0.17	2.52 ± 0.16	2.16 ± 0.13
		2.51-5.95	0.74-4.93	2.33-8.22	2.41-5.98	2.22-6.34	1.85-5.98	1.84-4.46	1.78-4.41	0.64-3.81	1.31-4.45	1.00-4.08	1.08-3.29
Creatinine (μmol/L)	<0.001	15.91 ± 1.76	18.36 ± 5.30	22.10 ± 1.7	29.17 ± 3.53	22.10 ± 0.88	27.40 ± 0.88	29.17 ± 1.76	30.05 ± 0.88	55.69 ± 2.65	65.41 ± 0.88	46.85 ± 0.88	43.31 ± 1.76
		0.00-34.47	0.88-132.6	1.06-51.27	17.68-87.51	17.68-26.50	18.56-35.36	0.88-44.20	23.86-33.70	37.12-84.86	55.69-77.79	38.01-58.34	33.59-70.72
Amylase (U/L)	<0.001	1,801.5 ± 82.2	1,851.4 ± 157.5	1,811.9 ± 160.4	2,342.9 ± 162.1	3,503.1 ± 248.0	4,132.8 ± 435.7	7,241.3 ± 577.2	7,540.1 ± 623.7	6,950.5 ± 674.8	7,010.9 ± 247.6	6,749.7 ± 239.7	7,980.2 ± 582.6
		560.1-4,147.9	488.5-3,319.0	900.1-3,643.3	1,198.9-4,378.5	1,548.1-6,543.1	570.4-10,251.4	2,677.1-14,901.4	2,682.1-16,426.9	766.7-15,216.6	3,859.8-8,362.9	4,147.8-8,789.2	2,815.3-13,884.3
ALP (U/L)	<0.001	1,042.5 ± 55.8	827.7 ± 44.7	644.5 ± 57.0	506.4 ± 29.3	387.0 ± 21.3	329.3 ± 17.2	349.9 ± 31.5	532.9 ± 30.4	485.0 ± 41.6	482.6 ± 34.4	615.8 ± 50.1	473.3 ± 34.9
		688.1-1,851.6	460.5-1,272.4	211.1-1,310.0	312.6-869.6	181.6-654.3	163.2-530.6	162.2-847.0	250.6-771.6	64.0-883.8	249.9-843.0	199.6-1221	164.6-816.2
CK (U/L)	<0.001	3,476.5 ± 288.9	2,936.3 ± 137.4	2,997.0 ± 185.2	3,074.9 ± 113.1	3,544.1 ± 247.0	4,919.9 ± 458.5	5,293.6 ± 393.1	6,333.4 ± 653.0	4404.0 ± 623.1	2741.0 ± 231.8	3300.9 ± 147.3	7504.9 ± 840.0
		455.0-6,676.4	1,954.8-4,603.3	1,168.9-4,593.6	2,116.2-4,131.9	1,383.4-7,678.0	2,314.8-10,815.1	3,255.0-9,717.0	2,326.6-14,402.0	586.6-12,096.3	330.8-3,867.4	1,330.3-4,094.1	3,288.4-17,788.2
GGT (U/L)	<0.001	1.45 ± 0.18	0.78 ± 0.19	1.26 ± 0.22	1.25 ± 0.21	1.29 ± 0.15	1.88±0.48	2.29 ± 0.33	1.03 ± 0.23	0.36 ± 0.16	6.02 ± 0.23	5.34 ± 0.30	5.84 ± 0.25
		0.00-2.90	0.00-2.93	0.00-3.85	0.00-3.90	0.00-3.00	0.00-11.10	0.00-4.00	0.00-3.50	0.00-2.58	2.74-7.75	1.79-7.25	3.22-8.60
AST (U/L)	<0.001	602.1 ± 14.0	597.4 ± 21.7	592.7 ± 24.5	552.1 ± 15.6	505.7 ± 11.8	453.8 ± 10.7	480.0 ± 14.6	421.8 ± 22.7	189.3 ± 10.2	172.3 ± 11.4	221.5 ± 31.3	177.1 ± 9.7
		497.4-779.5	344.8-832.7	347.3-867.4	347.8-736.2	417.7-627.8	342.1-546.6	378.5-673.6	315.15-817.4	111.0-260.4	42.2-251.9	109.0-602.4	75.3-257.8

Source: Samour J, Naldo J, Libanan N, et al: Age-related hematology and plasma chemistry changes in captive Masai ostriches (*Struthio camelus massaicus*), *Comp Clin Pathol* 20:659–667, 2011.

*Mean ± standard error of the mean.

†Inner limits of the percentiles $P_{2.5}$-$P_{97.5}$ with a probability of 95% confidence interval.

ALB, Albumin; ALT, alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CK, creatine kinase; GGT, γ-glutamyl transferase; GLOB, globulin; IU, International Units; LDH, lactic dehydrogenase; TP, total protein.

Serum Chemistry and Enzyme Values for Nonreproductive Adult Mallards (*Anas platyrhynchos*)

Assay	Male	Female
TP (g/L)	38 ± 7	42 ± 5
ALB (g/L)	15 ± 4	17 ± 2
GLU (mg/dL)	185.0 ± 47.0	215.0 ± 34.0
AMY (IU/L)	2631.0 ± 630.0	2766.0 ± 684.0
CHE (IU/L)	794.0 ± 249.0	812.0 ± 197.0
ALT (IU/L)	26.3 ± 8.0	29.9 ± 9.9
AST (IU/L)	16.2 ± 4.3	15.8 ± 4.7
GGT (IU/L)	7.7 ± 4.2	8.0 ± 4.8
ALP (IU/L)	26.3 ± 8.0	44.2 ± 22.7
LDH (IU/L)	199.0 ± 83.0	147.0 ± 80.0
Ca (mmol/L)	2.35 ± 0.47	2.45 ± 0.27
Mg (mmol/L)	1.8 ± 0.4	1.8 ± 0.3
PHOS (mmol/L)	0.93 ± 0.32	0.96 ± 0.32
UA (mmol/L)	237 ± 77.3	267.6 ± 107

Source: Modified from: Fairbrother A: Changes in mallard (*Anas platyrhynchos*) serum chemistry due to age, sex and reproductive condition, *J Wildlife Dis* 26:67–77, 1990.
Mean ± SD.
ALB, Albumin; ALT, alanine transaminase; ALP, alkaline phosphatase; AMY, amylase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CHE, cholesterol; CK, creatine kinase; GGT, γ-glutamyl transferase; GLOB, globulin; IU, International Units; LDH, lactic dehydrogenase; TP, total protein; UA, uric acid.

Serum Chemistry and Enzyme Values for Adult Female Mallards (*Anas platyrhynchos*) of Differing Reproductive States

Assay	Pre-Egglaying	Egglaying	Incubating	Molt
TP (g/L)	56 ± 29	63 ± 12	44 ± 6	45 ± 12
ALB (g/L)	20 ± 3	23 ± 2	16 ± 2	17 ± 2
GLU (mmol/L)	13.21 ± 1.16	14.3 ± 2.83	11.71 ± 2.94	11.04 ± 1.66
AMY (IU/L)	3058.0 ± 527.0	3821.0 ± 741.0	2700.0 ± 626.0	2346.0 ± 1012.0
CHE (IU/L)	1337.0 ± 280.0	1563.0 ± 592.0	1002.0 ± 266.0	894.0 ± 219.0
ALT (IU/L)	31.0 ± 10.3	34.2 ± 19.4	30.6 ± 13.1	41.1 ± 17.1
AST (IU/L)	18.0 ± 3.4	23.7 ± 6.7	22.1 ± 7.4	22.6 ± 12.6
GGT (IU/L)	19.8 ± 19.8	199.6 ± 283.0	7.5 ± 4.7	20.8 ± 36.9
ALP (IU/L)	63.6 ± 56.8	124.9 ± 56.7	34.3 ± 15.8	36.0 ± 18.1
LDH (IU/L)	165.0 ± 50.0	177.0 ± 57.0	215.0 ± 107.0	268.0 ± 2.2
Ca (mmol/L)	3.5 ± 1.02	5.47 ± 1.4	2.57 ± 0.5	2.63 ± 1.05
Mg (mmol/L)	2.3 ± 0.5	3.6 ± 0.8	1.6 ± 0.3	1.6 ± 0.5
PHOS (mmol/L)	1.48 ± 0.54	2.61 ± 0.77	1.19 ± 0.32	1.32 ± 0.71
UA (mmol/L)	309 ± 65.4	541.2 ± 303	327 ± 101.1	291 ± 101.1

Source: Modified from: Fairbrother A: Changes in mallard (*Anas platyrhynchos*) serum chemistry due to age, sex, and reproductive condition, *J Wildlife Dis* 26:67–77, 1990.
Mean ± SD.
ALB, Albumin; ALT, alanine transaminase; ALP, alkaline phosphatase; AMY, amylase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CHE, cholesterol; CK, creatine kinase; GGT, γ-glutamyl transferase; GLOB, globulin; IU, International Units; LDH, lactic dehydrogenase; TP, total protein; UA, uric acid.

Serum Chemistry and Enzyme Values for Adult Male Mallards (*Anas platyrhynchos*) of Differing Reproductive States

Assay	Pre-Egglaying	Egglaying	Incubating	Molt
TP (g/L)	46 ± 6	45 ± 8	42 ± 5	39 ± 8
ALB (g/L)	18 ± 2	16 ± 2	17 ± 3	15 ± 3
GLU (mmol/L)	15.7 ± 1.83	12.9 ± 1.77	11.0 ± 1.44	10.2 ± 1.60
AMY (IU/L)	3123.0 ± 583.0	2869.0 ± 614.0	3203.0 ± 785.0	2991.0 ± 748.0
CHE (IU/L)	1326.0 ± 344.0	1380.0 ± 399.0	984.0 ± 470.0	983.0 ± 452.0
ALT (IU/L)	34.6 ± 9.4	35.8 ± 13.1	27.6 ± 12.1	28.4 ± 19.2
AST (IU/L)	17.3 ± 4.0	20.5 ± 8.0	20.8 ± 15.7	18.1 ± 8.1
GGT (IU/L)	8.5 ± 7.6	10.6 ± 12.6	9.3 ± 6.0	16.5 ± 36.0
ALP (IU/L)	40.2 ± 25.3	44.1 ± 44.8	38.4 ± 48.0	35.3 ± 44.2
LDH (IU/L)	168.0 ± 66.0	219.0 ± 107.0	263.0 ± 203.0	202.0 ± 152.0
Ca (mmol/L)	2.72 ± 0.25	2.75 ± 0.47	2.47 ± 0.25	2.32 ± 0.55
Mg (mmol/L)	2.0 ± 0.2	2.0 ± 0.4	1.8 ± 0.4	1.8 ± 0.9
PHOS (mmol/L)	1.19 ± 0.24	0.9 ± 0.22	0.7 ± 0.12	0.77 ± 0.35
UA (mmol/L)	309.2 ± 71.3	309.2 ± 8.92	339.0 ± 113.0	279.5 ± 136.8

Source: Modified from: Fairbrother A: Changes in mallard (*Anas platyrhynchos*) serum chemistry due to age, sex, and reproductive condition, *J Wildlife Dis* 26:67–77, 1990.
Mean ± SD.
ALB, Albumin; ALT, alanine transaminase; ALP, alkaline phosphatase; AMY, amylase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CHE, cholesterol; CK, creatine kinase; GGT, γ-glutamyl transferase; GLOB, globulin; IU, International Units; LDH, lactic dehydrogenase; TP, total protein; UA, uric acid.

Serum Chemistry and Enzyme Values for Juvenile Mallards (*Anas platyrhynchos*)

Assay	Age 5 d	Age 18 d	Age 42 d	Age 58 d
TP (g/L)	34 ± 6	43 ± 13	40 ± 8	32 ± 10
ALB (g/L)	14 ± 2	15 ± 3	16 ± 4	14 ± 4
GLU (mmol/L)	13.2 ± 2.99	11.9 ± 5.16	10.4 ± 1.49	10.3 ± 2.49
AMY (IU/L)	3230.0 ± 760.0	3984.0 ± 1297.0	3005.0 ± 302.0	2395.0 ± 699.0
CHE (IU/L)	1423.0 ± 696.0	984.0 ± 559.0	827.0 ± 253.0	818.0 ± 248.0
ALT (IU/L)	21.3 ± 9.1	30.5 ± 10.5	26.1 ± 7.0	23.9 ± 7.1
AST (IU/L)	22.3 ± 7.4	88.5 ± 54.1	9.4 ± 5.1	17.4 ± 5.7
GGT (IU/L)	1.2 ± 2.8	4.6 ± 3.6	5.3 ± 5.7	6.1 ± 3.6
ALP (IU/L)	411.0 ± 89.0	386.0 ± 194.0	217.0 ± 32.0	185.0 ± 47.0
LD-LH (IU/L	425.0 ± 153.0	629.0 ± 251.0	169.0 ± 70.0	233.0 ± 83.0
Ca (mmol/L)	3.25 ± 2.57	2.4 ± 0.42	2.72 ± 0.4	2.1 ± 0.45
Mg (mmol/L)	2.8 ± 0.8	1.8 ± 0.7	2.0 ± 0.2	1.6 ± 0.5
PHOS (mmol/L)	2.55 ± 0.90	2.45 ± 0.41	2.0 ± 0.41	1.61 ± 0.54

ALB, Albumin; ALT, alanine transaminase; ALP, alkaline phosphatase; AMY, amylase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CHE, cholesterol; CK, creatine kinase; GGT, γ-glutamyl transferase; GLOB, globulin; IU, International Units; LDH, lactic dehydrogenase; TP, total protein; UA, uric acid.

Reference Values (Mean ± SD) for Selected Blood Chemistry Parameters in Nestling (<105 Days Old, *n* = 21), Immature (3-6 yr old, *n* = 14), Adult (>6 yr old, *n* = 12), and Free-Living (Immature and Adult Pooled Together, *n* = 26) Bearded Vultures

Parameter	Nestling	*n*	Immature	*n*	Adult	*n*	Free-Living	*n*
TSP (g/dL)*	2.78 ± 0.28 (2.3-3.3)	12	3.36 ± 0.4 (2.60-3.85)	8	3.49 ± 0.33 (2.90-3.9)	11	3.43 ± 0.36 (2.6-3.9)	19
TPP (g/dL)	3.60 ± 0.49 (2 8-4.3)	13	3.71 ± 0.51 (2.85-4.80)	14	3.98 ± 0.45 (2.90-4.8)	12	3.84 ± 0.49 (2.85-4.8)	26
GLU (mg/dL)	249.09 ± 55.26 (164-394.2)	20	224.15 ± 42.66 (148.9-299.8)	14	248.74 ± 45.23 (188.7-326.7)	12	235.50 ± 44.75 (148.9-326.7)	26
UA (mg/dL)*	4.81 ± 1.63 (2.3-7.7)	16	2.85 ± 1.75 (1.10-6.95)	14	3.49 ± 1.96 (1.6-7.5)	11	3.13 ± 1.83 (1.1-7.5)	25
U (mg/dL)*	17.68 ± 8.88 (3.5-32.1)	17	11.51 ± 6.58 (4.10-32.0)	14	11.98 ± 5.06(4.8-22.4)	12	11.73 + 5.82 (4.1-32)	26
U:UA ratio	3.81 ± 2.58 (0.46-9.23)	16	4.97 ± 2.96 (1.78-11.09)	14	4.75 ± 3.05 (2.18-9.74)	11	4.88 ± 2.93 (1.78-11.09)	25
CREA mg/dL)	0.20 ± 0.18 (0.01-0.7)	21	0.20 ± 0.08 (0.10-0.35)	14	0.25 ± 0.13 (0.03-0.54)	12	0.22 ± 0.11 (0.03-0.54)	26
Ca (mg/dL)	9.68 ± 1.20 (7.10-12.7)	20	8.73 ± 1.03 (7.99-9.9)	5	9.64 ± 0.67 (8.75-11.05)	11	9.44 ± 0.81 (7.99-11.05)	16
Phos (mg/dL)*	6.46 ± 2.05 (2.9-9.72)	15	3.02 ± 0.91 (1.26-4.2)	14	2.83 ± 0.84 (1.6-3.8)	7	2.96 ± 0.87 (1.26-4.2)	21
Chol (mg/dL)	239.23 ± 62.58 (165.1-391)	20	211.67 ± 52.66 (127.0-300)	14	198.03 ± 57.58 (124.3-327)	12	205.38 ± 54.30 (124.3-327)	26
Trig (mg/dL)*	152.54 ± 67.51 (26-278)	20	56.92 ± 18.35 (30-91)	13	50.53 ± 19.64 (17.5-87)	10	54.14 ± 18.76 (17.5-91)	23
Mg (mg/dL)*	1.77 ± 0.53 (0.98-2.8)	19	2.66 ± 0.47 (2.1-3.5)	8	2.55 ± 0.52 (1.6-3.5)	11	2.60 ± 0.49 (1.6-3.5)	19
AST (UI/L)*	110.19 ± 33.50 (52-211)	21	149.64 ± 40.54 (78-220)	14	170.3 ± 59.33 (57.5-239)	12	159.17 ± 50.14 (57.5-239)	26
LDH (UI/L)*	3,099.48 ± 1,437.47 (778-7.5)	21	1,552.86 ± 579.48 (891-2,557)	14	1,170.4 ± 386.04 (752-2,055)	12	1,376.35 + 527.24 (752-2,557)	26
CK (UI/L)*	2,645.10 ± 933.74 (729-3,967)	20	1,350.25 ± 646.19 (539-2.850)	12	1,254.1 ± 585.88 (625.0-2,335.0)	10	1,306.55 ± 606.82 (539-2,850)	22
ALP (UI/L)*	658.52 ± 395.71 (153-1.234)	21	61.5 ± 39.41 (25-115)	10	59.86 ± 25.83 (21.0-98.0)	11	60.64 ± 32.14(21-115)	21
AMY (UI/L)*	1,179.22 ± 421.06 (568-2.11)	18	591 ± 150.33 (456-753)	5	745.9 ± 250.22 (423.0-1,184.0)	10	710.19 ±± 235.24 (423-1,184)	15
LIP (UI/L)*	62.22 ± 54.26 (14-221)	18	953 ± 67 (876-998)	5	(955.1 ± 221.38) (442-1,155)	10	954.58 ± 193.66 (442-1.155)	15
CHE (UI/L)	739.28 ± 375.26 (568-2.11)	13	723.21 ± 155.54 (420-965)	14	688.1 ± 195.1 (412.2-1,062)	11	707.77 ± 171.11 (412.2-1,062)	25

Hernandez M, Margalida A: Hematology and blood chemistry reference values and age-related changes in wild bearded vultures, *J Wildlife Dis* 46(2):390–400, 2010.

*Values significantly different between nestlings, immatures, and adults (analysis of variance, $p < 0.05$) and between nestlings and free-living bearded vultures (*t*-test, $p < 0.05$).

ALB, Albumin; ALT, alanine transaminase; ALP, alkaline phosphatase; AMY, amylase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CHE, cholesterol; CK, creatine kinase; GGT, γ-glutamyl transferase; GLOB, globulin; GLU, glucose; IU, International Units; LDH, lactic dehydrogenase; TPP, total plasma protein; TSP, total serum protein; UA, uric acid.

Age-Related Blood Chemistry Changes in Captive Buff-Crested Bustards (*Eupodotis ruficrista gindiana*)

Assay	2-8 wk	*n*	9-16 wk	*n*	17-24 wk	*n*	>1 yr	*n*
Glucose (mmol/L)	14.93 ± 0.45 (14.11-15.67)	3	18.17 ± 0.81 (15.83-22.17)	8	16.32 ± 0.68 (13.94-17.78)	5	20.36 ± 0.55 (15.28-28.83)	25
Uric acid (mmol/L)	392.86 ± 47.14 (172.61-589.28)	7	337.5 ± 39.58 (113.1-726.19)	14	573.21 ± 47.29 (238.1-779.76)	12	533.93 ± 38.97 (202.38-928.57)	26
Total protein (g/L)	18.14 ± 1.37 (11.0-22.0)	7	26.88 ± 1.76 (13.0-39.0)	17	30.67 ± 1.56 (23.0-42.0)	12	32.81 ± 0.83 (25.0-41.0)	26
Albumin (g/L)	nd		nd		nd		12.91 ± 0.37 (10.0-16.0)	23
Globulin (g/L)	nd		nd		nd		19.61 ± 0.64 (14.0-26.0)	23
Albumin : globulin ratio	nd		nd		nd		0.67 ± 0.02 (0.52-0.79)	23
ALP (IU/L)	975.33 ± 367.33 (511-2926)	3	1080.81 ± 143.73 (392-2132)	11	561.5 ± 143.94 (155-790)	4	475.16 ± 45.71 (160-868)	26
ALT (IU/L)	26 ± 7 (19-40)	3	28.25 ± 6.93 (17-76)	8	23.2 ± 2.48 (18-32)	5	nd	
AST (IU/L)	248.2 ± 20.17 (184-310)	5	256.46 ± 20.33 (151-388)	13	269 ± 29.51 (154-469)	9	334.96 ± 11.91 (217-482)	26
LDH (IU/L)	381.5 ± 183.5 (198-565)	2	447.57 ± 79.65 (220-831)	7	415 ± 130.1 (278-675)	3	373.96 ± 29.72 (152-788)	26
CK (IU/L)	nd		nd		nd		361.65 ± 41.27 (115-797)	26
Magnesium (mmol/L)	nd		nd		nd		1.05 ± 0.03 (0.83-1.36)	26
Calcium (mmol/L)	1.49 ± 0.11 (1.23-1.73)	5	1.61 ± 0.09 (1.1-1.95)	8	2.05 ± 0.19 (1.23-2.83)	7	2.55 ± 0.06 (2.02-3.51)	26
Cholesterol (mmol/L)	nd		nd		nd		3.64 ± 0.15 (2.19-5.37)	26

Source: Bailey TA, Wernery U, Howlett J, et al: Age-related plasma chemistry findings in the buff-crested bustard (*Eupodotis ruficrista gindiana*), *J Vet Med* 45:635–640, 1998.
Mean ± standard error of the mean (minimum-maximum).
ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CPK, creatine phosphokinase; GT, glutamyl transferase; IU, International Units; LDH, lactic dehydrogenase; nd, not determined.

Age-Related Blood Chemistry Changes in Captive Kori Bustards (*Ardeotis kori*)

Assay	*p*-Value	4-8 wk *n* = 24	9-16 wk *n* = 6-8	17-24 wk *n* = 7-14	25-32 wk *n* = 6-13	33-40 wk *n* = 8-10	41-52 wk *n* = 6-7	1 yr (Adult), *n* = 28
Glucose (mmol/L)				17.74 ± 0.45 (15.43-19.87)				
Uric acid (μmol/L)	0.057							
Total protein (g/L)				35.86 ± 1.96 (24.0-51.0)		37.0 ± 1.64 (28.0-43.0)		30.0 ± 0.82 (23.0-40.0)
Albumin (g/L)	nd	nd	nd	nd	nd	nd	nd	11.0 ± 0.3
Globulin (g/L)	nd	nd	nd	nd	nd	nd	nd	1.9 ± 0.06
Albumin : globulin ratio	nd	nd	nd	nd	nd	nd	nd	0.58 ± 0.01 (0.29-0.73)
ALP (IU/L)				147.5 ± 16.35 (55-219)				85.9 ± 2.6 (37-98)
ALT (IU/L)	0.566	27 (*n* = 1)		28 ± 2.27 (20-34)	33.7 ± 1.54 (20-39)		34.4 ± 3.43 (20-10)	
AST (IU/L)		221.5 ± 8.5 (213-230)						207 ± 7.1 (168-369)
LDH (IU/L)	0.001	1114 ± 17 (1024-1158)						
CK (IU/L)	nd	nd	nd	nd	nd	nd	nd	
Magnesium (mmol/L)	nd	nd	nd	nd	nd	nd	nd	1.05 ± 0.02
Calcium (mmol/L)		0.83 ± 0.1 (0.7-1.03)	1.41 ± 0.12 (1.0-1.8)	1.42 ± 0.07 (1.1-1.83)	1.67 ± 0.06 (1.25-2.2)	1.98 ± 0.1 (1.5-2.4)	1.94 ± 0.1 (1.5-2.28)	2.34 ± 0.07 (1.71-3.44)
Cholesterol (mmol/L)	nd	nd	nd	nd	nd	nd	nd	3.7 ± 0.13

Source: Bailey TA, Wernery U, Howlett J, et al: Age-related plasma chemistry changes in houbara and kori bustards, *J Wildlife Dis* 33:31–37, 1998.
Mean ± standard error of the mean (minimum-maximum).
ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CPK, creatine phosphokinase; GT, glutamyl transferase; IU, International Units; LDH, lactic dehydrogenase; nd, not determined.

Age-Related Blood Chemistry Changes in Captive Houbara Bustards (*Chlamydotis undulata macqueenii*)

Assay	*p*-Value	4-8 wk *n* = 10-11	9-16 wk *n* = 24-26	1 yr (Adult) *n* = 28
Glucose (mmol/L)	0.016	20.70 ± 2.45 (14.93-39.63)	16.90 ± 0.56 (13.21-24.37)	16.89 ± 0.26 (14.04-19.59)
Uric acid (mmol/L)	0.186	393.76 ± 55.61 (190.34-713.76)	402.32 ± 42.75 (107.1-856.51)	432.42 ± 39.79 (202.23-1005.21)
Total protein (g/L)	0.006	32.0 ± 0.97 (27.0-36.0)	33.12 ± 1.07 (23.0-48.0)	37.93 ± 0.9 (30.0-48.0)
Albumin (g/L)	nd	nd	nd	14.5 ± 0.28 (11.0-18.0)
Globulin (g/L)	nd	nd	nd	2.38 ± 0.09 (1.7-3.7)
Albumin : globulin ratio	nd	nd	nd	0.64 ± 0.03 (0.32-0.84)
ALP (IU/L)	0.044	622.8 ± 82.43 (257-1131)	278.72 ± 23.97 (122-622)	80.39 ± 7.24 (17-175)
ALT (IU/L)	0.018	21 ± 1.31 (11-26)	22.2 ± 1.33 (14-42)	45.14 ± 3.27 (22-97)
AST (IU/L)	0.007	376.36 ± 16.21 (293-466)	342.2 ± 15.08 (247-528)	467.9 ± 24.93 (246-774)
LDH (IU/L)	0.006	934.6 ± 69.54 (676-1284)	690.72 ± 46.31 (406-1467)	609.57 ± 43.42 (246-774)
CK (IU/L)	0.01	228.1 ± 42.76 (55-427)	141.04 ± 24.52 (14-479)	778.4 ± 122.2 (12-2309)
Magnesium (mmol/L)	0.015	0.75 ± 0.05 (0.41-1.03)	0.80 ± 0.02 (0.58-1.03)	1.01 ± 0.03 (0.81-1.27)
Calcium (mmol/L)	0.013	2.07 ± 0.13 (1.5-2.75)	1.81 ± 0.08 (1.1-2.73)	2.49 ± 0.08 (1.46-3.01)
Cholesterol (mmol/L)	0.273	6.29 ± 0.33 (4.65-7.76)	6.08 ± 0.28 (1.09-8.83)	6.78 ± 0.33 (4.06-10.65)

Source: Bailey TA, Wernery U, Howlett J, et al: Age-related plasma chemistry changes in houbara and kori bustards, *J Wildlife Dis* 33:31–37, 1998.
Mean ± standard error of the mean (minimum-maximum).
Values are significantly different among the groups ($p < 0.05$).
ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CPK, creatine phosphokinase; GT, glutamyl transferase; IU, International Units; LDH, lactic dehydrogenase; nd, not determined.

Age-Related Blood Chemistry Changes in Captive White-Bellied Bustards (*Eupodotis senegalensis*)

Assay	4-8 wk	n	9-16 wk	n	17-24 wk	*n*	1 Year (Adult)	*n*
Glucose (mmol/L)	15.21 ± 1.19 (13.1-18.09)	4	16.97 ± 0.29 (16.65-17.54)	3	16.54 ± 0.43 (14.99-17.59)	5	19.09 ± 1.05 (16.32-23.04)	6
Uric acid (mmol/L)	400.0 ± 30.81 (356.88-487.74)	4	303.35 ± 38.66 (261.71-380.67)	3	457.99 ± 88.63 (273.61-808.93)	6	408.63 ± 15.39 (356.88-481.79)	7
Total protein (g/L)	16.4 ± 2.42 (10.0-24.0)	5	24.25 ± 5.41 (13.0-37.0)	4	30.33 ± 1.36 (27.0-34.0)	6	30.0 ± 1.71 (25.0-35.0)	6
Albumin (g/L)	nd		nd		nd		12.0 ± 0.9 (9.0-15.0)	6
Globulin (g/L)	nd		nd		nd		18.0 ± 0.89 (15.0-20.0)	6
Albumin : globulin ratio	nd		nd		nd		0.66 ± 0.03 (0.56-0.75)	6
ALP (IU/L)	187.75 ± 23.99 (137-244)	4	139 ± 4 (135-143)	2	70.75 ± 8.89 (53-91)	4	44.17 ± 10.53 (23-86)	6
ALT (IU/L)	21.25 ± 2.32 (17-27)	4	24 ± 3 (21-27)	2	26.33 ± 5.17 (16-32)	3	nd	
AST (IU/L)	293 ± 32.5 (225-375)	4	397.67 ± 100.1 (256-591)	3	372.33 ± 82.57 (206-718)	6	444.67 ± 42.34 (266-574)	6
LDH (IU/L)	1235.25 ± 152.95 (944-1620)	4	1452 ± 231 (1221-1683)	2	978.67 ± 174.33 (704-1302)	3	755.17 ± 80.18 (474-985)	6
CK (IU/L)	410.5 ± 32.56 (335-492)	4	180 ± 16 (164-196)	2	378 ± 142.49 (164-648)	3	377.14 ± 42.77 (170-521)	7
Magnesium (mmol/L)	0.65 ± 0.04 (0.53-0.74)	4	0.69 ± 0.04 (0.66-0.74)	2	0.78 ± 0.02 (0.74-0.82)	3	1.01 ± 0.07 (0.84-1.32)	
Calcium (mmol/L)	1.41 ± 0.19 (1.03-1.93)	4	1.59 ± 0.45 (1.48-1.7)	2	1.99 ± 0.09 (1.73-2.3)	5	2.42 ± 0.05 (2.25-2.56)	6
Cholesterol (mmol/L)	nd		nd		nd		3.30 ± 0.35 (2.66-4.97)	6

Source: Modified from: Bailey TA, Wernery U, Naldo J, et al: Normal blood chemistry and age-related changes in the white-bellied bustards, *Comp Haematol Int* 8:61–65, 1998.
Mean ± standard error of the mean (minimum-maximum).
ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; CK, creatine kinase; GT, glutamyl transferase; IU, International Units; LDH, lactic dehydrogenase; nd, not determined.

Reference Urine Chemistry Values in Clinically Normal Falcons

Assay	Mean (Minimum-Maximum)	Median (95% CI for the Median)
Chloride (mmol/L)	41.85 (0.3-121.64)	37.75 (20.81-52.46)
GGT (IU/L)	42.65 (2.41-426.11)	26.07 (17.82-40.73)
Glucose (mmol/L)	1.34 (0.26-1.85)	0.91 (0.87-1.02)
Total protein (g/L)	27 (0-12)	2 (2-3)
ALP (IU/L)	80.15 (0-889.3)	36.4 (26.02-52.88)

Source: Modified from: Tschopp R, Bailey TA, Di Somma A, et al: Urinalysis in Falconidae, *J Avian Med Surg* 2l(1):1–7, 2007.
ALP, Alkaline phosphatase; GGT, γ-glutamyl transferase; IU, International Units.

Vitamin A, B1, C, and E levels in the Blood of Clinically Normal Captive Houbara Bustards (*Chlamydotis undulata*)

Vitamin B1 (μg/L)	Vitamin E (μmol/L)	Vitamin C (mg/L)	Vitamin A (μmol/L)
45.83 ± 1.87	17.81 ± 1.03	4.06 ± 0.32	5.42 ± 0.23
(33-60)	(11.9-30.9)	(1.3-5.9)	(4.1-7.6)

Source: Bailey TA: *Diseases and medical management of houbara bustards and other Otididae*, Chapter 5, United Arab Emirates, 2008, Avian Research Center, Environment Agency Abu Dhabi, pp 71–80.
Mean ± standard error of the mean (minimum-maximum).

Plasma α-Tocopherol and Cholesterol Concentrations in Captive Bustards (*Chlamydotis undulata*)

Species	Age	α-Tocopherol (mg/mL)	*n*	Cholesterol (mg/mL)	*n*	α-Tocopherol:Cholesterol (mg/mg)
Houbara bustards *(Chlamydotis undulata)*	Adult	11.07 ± 0.41	32	1.93 ± 0.10	12	6.09 ± 0.44
	Juvenile	6.33 ± 0.48	12	2.08 ± 0.09	11	2.94 ± 0.22
Kori bustards *(Ardeotis kori)*	Adult	4.43 ± 0.42	21	1.23 ± 0.25	20	3.67 ± 0.44
	Juvenile	4.46 ± 0.26	11	1.28 ± 0.11	11	3.71 ± 0.36
Buff-crested bustards *(Eupodotis ruficrista)*	Adult	6.64 ± 0.33	19	1.22 ± 0.05	18	5.56 ± 0.32
White-bellied bustards *(Eupodotis senegalensis)*	Adult	7.75 ± 0.81	8	1.35 ± 0.13	8	5.83 ± 0.43
Black bustards *(Eupodotis afra)*	Adult	10.08 ± 0.06	2	1.37 ± 0.04	2	7.36 ± 0.17
Heuglin's bustards *(Neotis heuglinii)*	Adult	6.08 ± 0.64	4	1.16 ± 0.09	4	5.39 ± 0.86

Source: Anderson SJ, Dawodu A, Patel M: Plasma concentrations of vitamin E in six species of bustard (Gruiformes: Otididae), *J Wildlife Dis* 38:414–419, 2002.
Mean ± standard error of the mean.
Adult: ≥12months; juvenile: 6-12 mo.

Blood Gas Values for Eight Clinically Normal Houbara Bustards (*Chlamydotis undulata*)

Assay	Range
pH	7.44 ± 0.02 (7.39-7.53; 8)
Pco_2 (mm Hg)	24.44 ± 0.91 (21.3-28.7; 8)
Po2 (mm Hg)	48.13 ± 1.39 (43.7-55.3; 8)
So_2%	61.21 ± 2.45 (54-73.2; 8)
Na (mmol/L)	151.95 ± 0.69 (150.2-156; 8)
Ca2+ (mmol/L)	1.49 ± 0.02 (1.39-1.6; 8)
Glucose (mg/dL)	243.5 ± 3.73 (225-256; 8)
BEecf (mmol/L)	–7.71 ± 0.79 (10.6-3.2; 8)
HCO3 (mmol/L)	16.69 ± 0.63 (14.1-19.7; 8)
Tco_2 (mmol/L)	17.438 ± 0.6497 (14.7-20.4; 7)
BEB (mmol/L)	–5.06 ± 0.736 (7.5-0.8; 7)
SBC (mmol/L)	19.55 ± 0.63 (17.6-23.3; 7)
A (mm Hg)	118.89 ± 1.09 (113.8-122.7; 7)
A-aDo2 (mmHg)	71.343 ± 1.77 (65.1-78.1; 7)
a/A	0.41 ± 0.01 (0.4-0.5; 7)
RI	1.5 ± 0.08(1.2-1.8; 7)
P50 (mm Hg)	43.09 ± 0.47(41.7-45; 7)
O2Cap (mL/dL)	19.37 ± 0.58(17.7-22.4; 7)
O2Ct (mL/dL)	12.09 ± 0.66(10-15.2; 7)
nCa (mmol/L)	1.54 ± 0.03(1.46-1.64; 7)

Source: Bailey TA: *Diseases and medical management of houbara bustards and other Otididae*, Chapter 5, United Arab Emirates, 2008, Avian Research Center, Environment Agency Abu Dhabi, pp 71–80.
Mean ± standard error of the mean (minimum-maximum; n).
Samples were collected from a brachial vein of manually restrained birds.
A, Alveolar saturation; a/A, arterial alveolar oxygen tension ratio; A-aDo2, arterial alveolar oxygen tension gradient; Beb, base excess of blood; BEecf, base excess extracellular fluid; nCa, ionized calcium normalized to pH 7.4; O2Ct, oxygen content; P50, the Po2 of a sample at which the hemoglobin is 50% saturated with oxygen at pH 7.4; Pco_2, partial pressure of carbon dioxide; Po_2, partial pressure of oxygen; SBC, standard bicarbonate concentration; So_2, oxygen saturation; Tco_2, total carbon dioxide content.

I-STAT Blood Values in Clinically Normal Gyrfalcons (*Falco Rusticolus*) and gyr Hybrid Falcons (*Falco rusticolus-Falco Cherrug, Falco rusticolus-Falco peregrinus*)

Assay	Range (*n* = 70)
Glucose (mmol/L)	18.79 ± 1.43 (15.7-22.5)
BUN (mmol/L)	2.45 ± 0.06 (2.14-5.71)
Na (mmol/L)	150.33 ± 2.30 (146-157)
K (mmol/L)	2.90 ± 0.69 (2.0-4.1)
Cl (mmol/L)	119.86 ± 2.22 (15.0-125.0)
Tco_2 (mmol/L)	26.41 ± 2.24 (21.0-30.0)
AnGAP (mmol/L)	6.76 ± 2.70 (1.0-14.0)
Hct (%)	46.57 ± 4.48 (40.0-57.0)
Hb (g/dL)	15.79 ± 1.51 (14.0-19.0)
pH log10	7.47 ± 0.04 (7.4-7.6)
Pco_2 (mmHg)	34.96 ± 4.20 (27.0-49.9)
HCO_3 (mmol/L)	25.34 ± 2.19 (26.0-29.0)
BEecf (mmol/L)	1.77 ± 2.41 (–3.0-6.0)

Source: McKinney P: Clinical applications of the I-STAT blood analyzer in avian practice, *Proceedings of the European Association of Avian Veterinarians*, Tenerife, 2003, pp 341–346.
Mean ± SD (minimum-maximum).
AnGAP, Anion gap; BEecf, base excess extracellular fluid; BUN, blood urea nitrogen; Hb, hemoglobin; Hct, hematocrit; Pco_2, partial pressure of CO; Tco_2, total CO_2.

I-STAT Blood Values in Clinically Normal Red-Tailed Hawks (*Buteo jamaicensis*)

Assay	Range (*n* = 40)
Glucose (mmol/L)	20.5 ± 2.4
Na (mmol/L)	151.7 ± 2.1
K (mmol/L)	3.06 ± 0.47
Cl (mmol/L)	119.2 ± 3.0
Tco_2 (mmol/L)	18.65 ± 4.25
AnGAP (mmol/L)	17.63 ± 4.8
Hct (%)	36.8 ± 3.2
Hb (g/dL)	12.65 ± 0.98
pHlog10	7.43 ± 0.07
Pco_2 (mm Hg)	26.78 ± 4.6
HCO3 (mmol/L)	18.1 ± 4.25
BEecf (mmol/L)	–6.36 ± 5.22

Source: Heatley JJ, Demirjian SE, Wright JC, et al: Electrolytes of the critically ill raptor, *Proceedings of the Association of Avian Veterinarians*, Monterey, 2005, pp 23–25.
Mean ± SD.
AnGAP, Anion gap; BEecf, base excess extracellular fluid; Hb, hemoglobin; Hct, hematocrit; Pco_2, partial pressure of CO; Tco_2, total CO_2.

Serum Copper, Magnesium and Zinc Levels in Six Species of Captive Bustards and Stone Curlew

Species	Houbara Bustard (*n* = 56)	Kori Bustard (*n* = 45)	White-Bellied Bustard (*n* = 33)	Buff-Crested Bustard (*n* = 31)	Black Bustard (*n* = 3)	Heuglin's Bustard (*n* = 4)	Stone Curlew (*n* = 3)
Scientific Name	***Chlamydotis undulata***	***Ardeotis kori***	***Eupodotis senegalensis***	***Eupodotis ruficrista***	***Eupodotis afra***	***Neotis heuglinii***	***Burhinus oedicnemus***
Copper (mg/dL)	86.05 ± 0.71 (76.7-98.1)	82.91 ± 0.88 (67.8-101.6)	84.20 ± 0.59 (77.4-93.3)	81.61 ± 0.82 (70.9-89.9)	83.7 ± 0.81 (82.4-85.2)	82.43 ± 4.14 (71.6-91)	81.5 ± 1.28 (76-88.6)
Magnesium (mmol/L)	1.2 ± 0.04 (0.63-1.95)	1.08 ± 0.05 (0.63-1.84)	1.12 ± 0.04 (0.83-1.72)	1.01 ± 0.03 (0.71-1.27)	1.03 ± 0.09 (0.93-1.21)	1.12 ± 0.05 (0.97-1.22)	0.88 ± 0.03 (0.75-1.0)
Zinc (mg/dL)	161.55 ± 2.53 (111.1-215)	159.38 ± 5.53 (104-293.4)	175.5 ± 4.33 (121.5-220.2)	174 ± 3.37 (129.7-205)	171.9 ± 8.84 (156.3-186.9)	201.33 ± 4.92 (192-215.2)	180.56 ± 7.89 (147.4-231.7)

Source: Bailey TA, Silvanose C, Combreau O, et al: Normal blood concentrations of copper, magnesium and zinc in stone curlews and five species of bustards in the United Arab Emirates, *Proceedings of the European Association of Zoo and Wildlife Veterinarians*, Ebeltoft, 2004, pp 297–301.
Mean ± SD(minimum-maximum).

Results of Plasma Biochemical Analyses in Clinically Normal Elegant-Crested Tinamou (*n* = 19)

Analyte	Mean ± SD (SEM)	Reference Interval	Median
Sodium (mEq/L)	165.44 ± 3.05 (1.02)	162.00-176.00	165.00
Chloride (mEq/L)	123.20 ± 3.06 (0.97)	108.00-128.00	124.00
Potassium (mEq/L)	2.15 ± 0.58 (0.28)	0.60-6.80	2.15
Calcium (mg/dL)	13.70 ± 0.58 (0.18)	12.40-22.00	13.70
Phosphorus (mg/dL)	4.22 ± 0.90 (0.25)	2.20-15.10	4.40
Creatine phosphokinase (U/L)	728.83 ± 466.01 (134.52)	63-4756	610
Aspartate aminotransferase (U/L)	111.69 ± 25.19 (6.99)	75.0-306.0	104.0
Glucose (mg/dL)	331.42 ± 28.20 (8.14)	38.0-479.0	34.0
Total bilirubin (mg/dL)	0.21 ± 0.10 (0.03)	0.10-0.50	0.20
Uric acid (mg/dL)	4.83 ± 1.70 (0.47)	3.30-21.00	4.00

Source: Black P, Mack M, Tieber A, Weber M: Reference values for hematology, plasma biochemical analysis, plasma protein electrophoresis, and *Aspergillus* serology in elegant-crested tinamou (*Eudromia elegans*), *J Avian Med Surg* 27(1):1–6, 2013.
SD, Standard deviation; SEM, standard error of the mean.

Blood Chemistry Values for Selected Ratites

Species	Ostrich	Emu	Cassowary
Scientific Name	***Struthio camelus***	***Dromiceius novaehollandiae***	***Casuarius sp.***
Total protein (g/L)	37 ± 7	42 ± 05	61 ± 5
Osmolality (mOsmol/kg)	286.0 ± 49.0	—	—
Glucose (mmol/L)	13.8 ± 3.88	8.7 ± 1.22	11.54 ± 2.63
Triglycerides (mmol/L)	1.01 ± 0.50	3.66 ± 6.67	2.03 ± 0.81
Cholesterol (mmol/L)	2.50 ± 1.16	2.68 ± 0.80	2.06 ± 0.41
BUN (mmol/L)	1.71 ± 0.42	1.78 ± 0.64	6.64 ± 0.42
Uric acid (mmol/L)	487.7 ± 160.5	279.5 ± 118.9	356 ± 35.6
Calcium (mmol/L)	2.3 ± 0.6	2.62 ± 0.32	2.85 ± 0.05
Phosphorus (mmol/L)	1.55 ± 0.38	1.74 ± 0.32	1.61 ± 0.03
Sodium (mmol/L)	147.0 ± 34.0	—	149.0 ± 2.1
Potassium (mmol/L)	3.0 ± 0.8	—	4.1 ± 1.0
Chloride (mmol/L)	100.0 ± 16.0	—	108.0 ± 0.0
Magnesium (mmol/L)	2.2 ± 0.8	—	2.3 ± 0.3
ALP (IU/L)	575.0 ± 248.0	84.0 ± 44.0	—
ALT (IU/L)	2.0 ± 1.7	15.4 ± 4.3	80.0 ± 21.0
AST (IU/L)	131.0 ± 31.0	104.0 ± 24.0	698.0 ± 532.0
GGT (IU/L)	1.5 ± 2.9	4.4 ± 3.4	—
LDH (IU/L)	1565.0 ± 660.0	240.0 ± 91.0	1060.0 ± 516.0
CK (IU/L)	688.0 ± 208.0	264.0 ± 170.0	—

Modified from: Stewart JS: Husbandry, medical and surgical management of ratites, part 2. *Proceedings of the Association of Zoo Veterinarians*, Greensboro, 1989, pp 119–122.
ALT, Alanine transaminase; ALP, alkaline phosphatase; AST, aspartate aminotransferase; BUN, blood urea nitrogen; CK, creatine kinase; GGT, γ-glutamyl transferase; IU, International Units; LDH, lactic dehydrogenase.

Summary of the Physiology and Effects of Changes in Vitamin Levels in Avian Species

Vitamin	Physiology in Avian Species	Causes and Effects of Changes in Vitamin Levels
A	Fat-soluble vitamin essential for growth and differentiation of epithelial tissues, mucopolysaccharide formation, stability of cell membranes, growth of bones, and normal reproduction. Also improves the immune system. It is stored in the liver and has the potential to act as an accumulative toxicant. Deficiencies can result from insufficient dietary fat, insufficient antioxidant protection, or disorders that interfere with fat digestion or absorption. Liver disease may reduce the bird's ability to store vitamin A	Deficiency—Embryo mortality and abnormalities, susceptibility to respiratory infections, visual disorders, squamous metaplasia of mucous membranes, hyperkeratosis, decreased testes size and testosterone levels, urate deposits in the kidneys and ureters, egg binding, poorly formed eggs. Toxicity—bone abnormalities, spontaneous fractures, conjunctivitis, enteritis, suppressed keratinization, internal hemorrhages, fatty liver and kidneys, and secondary deficiencies of other fat-soluble vitamins
D3	Fat-soluble vitamins essential for the absorption of calcium and consequently normal bone and egg shell formation. It is destroyed by excess radiation with ultraviolet light and oxidation in the presence of rancidifying fatty acids. There are two forms of this vitamin, ergocalciferol (D2), a plant derivative, and cholecalciferol (D3) produced in the bird's body. Vitamin D3 is synthesized in avians kin exposed to ultraviolet light and is 30-40 times more potent than vitamin D2. A dietary source of vitamin D3 is needed by animals that do not have access to ultraviolet light	Deficiency—thin, soft-shelled eggs, embryonic abnormalities and mortality, metabolic bone disease, leg weakness, seizing, pathologic bone fractures, and poor feathering. Can be induced by high dietary vitamin A or E levels. Toxicity—reduced fertility, decreased egg shell quality, soft tissue calcification, renal and artery calcification, bone demineralization, and muscular atrophy
E	Fat-soluble vitamin that provides natural antioxidation protection for cells, fatty acids, and other fat-soluble vitamins. Working in conjunction with vitamin E are several metalloenzymes that incorporate manganese, zinc, copper, iron, and selenium. The selenium-containing glutathione peroxidase is the most important of these enzymes. Because of their activity, selenium and vitamin E tend to have a sparing effect on each other. Vitamin E is active in several metabolic systems including cellular respiration, normal phosphorylation reactions, ascorbic acid synthesis, sulfur amino acid synthesis. It also has effects on immunity by increasing phagocytosis and antibody production as well as stimulating macrophage and lymphocyte activity	Deficiency-low fertility, embryonic mortality, low hatchability, immunosuppression, testicular degeneration, and specific clinical abnormalities such as encephalomalacia, exudative diathesis, and muscular myopathies. May be predisposed by giardiasis. Toxicity-enlarged fatty liver, waxy feathers. High levels can cause secondary deficiency signs of bone demineralization n or blood clotting failure if vitamins D3 and K are marginal
K	Fat-soluble vitamin essential for normal blood clotting. It comes from three sources, namely: green plants, bacteria, and synthetic forms. The microbial synthesis in the intestinal tract is significant in most species. The requirements of this vitamin vary according to the extent to which different species use the synthesized vitamin K and to which they practice coprophagy. It is destroyed by oxidation, alkaline conditions, strong acids, ultraviolet light, and some sulfa drugs. Vitamin K also requires the presence of dietary fats and bile salts for absorption from the gut, so decreased pancreatic and biliary function can impair normal absorption	Deficiency—embryonic mortality, hemorrhaging, anemia, and altered bone metabolism. Can be induced by high dietary levels of vitamins A or E or by prolonged antibiotic treatment. Toxicity—high levels can cause chick mortality and anemia
B1 Thiamine	Water-soluble vitamin essential for enzyme activity and cellular respiratory control as well as being involved in nerve activity. It is common in plant and animal food sources but generally at low concentration. Several compounds in nature possess antithiamine activity. These include amprolium, which inhibits thiamine absorption from the intestine; thiaminases, which are found in raw fish; and thiamine antagonists such as tannic acid. Thiamine is not stored in the body for long	Deficiency—embryonic mortality, muscular paralysis, ataxia, convulsions, neurologic signs, and organ atrophy. Toxicity—not studied in birds. High levels in mammals can cause depression of the respiratory center and blockage of nerve transmission
B2 Riboflavin	Water-soluble vitamin essential for enzyme activity, carbohydrate utilization, cellular metabolism and respiration, uric acid formation, amino acid breakdown, and drug metabolism. It is destroyed by ultraviolet light and alkaline solutions. Very little riboflavin is stored in the body and it is rapidly excreted	Deficiency—embryonic abnormalities and mortality, chick mortality, curled toe paralysis and other neuromuscular disorders, dermatitis, poor feather pigmentation, splayed legs, fatty liver, and dermatitis Toxicity—not reported in birds. Toxicity not thought to be a risk because it is not well absorbed from the gut

Continued

Summary of the Physiology and Effects of Changes in Vitamin Levels in Avian Species—cont'd

Vitamin	Physiology in Avian Species	Causes and Effects of Changes in Vitamin Levels
B6 Pyridoxine	Water-soluble vitamin involved in a number of enzyme systems as a coenzyme. It is required in all areas of amino acid utilization, the synthesis of niacin and the formation of antibodies. It is destroyed by oxidation	Deficiency-reduced hatchability, ataxia, neuromuscular disorders, perosis, hemorrhaging, and gizzard erosion Toxicity-not reported in birds
B12 Cyanocobalamin	A product of bacterial biosynthesis and therefore must be obtained by consuming a bacterial source or animal tissues that accumulate the vitamin. It is a critical component of many metabolic pathways and is involved in the synthesis of nucleic acids and protein as well as carbohydrates and fats. Most vitamin B12 in the body is found in the liver, with secondary stores in the muscles. Vitamin B12 is stored efficiently with along biological half-life of 1 year in humans	Deficiency—embryo abnormalities and mortality, chick mortality, gizzard erosion, and poor feathering. Toxicity—not reported in birds

Source: Bailey TA: *Diseases and medical management of houbara bustards and other Otididae*, Chapter 5, United Arab Emirates, 2008, Avian Research Center, Environment Agency Abu Dhabi, pp 71–80.

Summary of the Physiology and Effects of Changes in Vitamin Levels in Avian Species—cont'd

Vitamin	Physiology in Avian species	Causes and Effects of Changes in Vitamin Levels
Biotin	Water-soluble vitamin that is an active part of four different carboxylase enzymes in the body in the metabolism of energy, glucose, lipids, and some amino acids. It is destroyed by strong acids and bases, oxidizing agents, and the protein avidin in raw egg albumin. Biotin is widely distributed in foods at low concentrations. The synthesis of biotin by intestinal microflora may be important	Deficiency—embryo abnormalities and mortality, poor growth, dermatitis, perosis and leg abnormalities, fatty liver-kidney syndrome. Toxicity—not reported in birds
Choline	Water-soluble vitamin with four important metabolic functions: (1) as a component of phospholipids and therefore in maintaining cell integrity, (2) maturation of the cartilage matrix of bone, (3) fat metabolism in the liver, and (4) acetylated to form the neurotransmitter acetylcholine. Although most animals synthesize choline, young animals cannot synthesize enough to meet the demands of growth	Deficiency-reduced hatchability, perosis, and enlarged hocks hepatic steatitis, fatty liver syndrome. Toxicity—not reported in birds
Folic acid	Water-soluble vitamin involved in amino acid metabolism and bioconversion and in the synthesis of nucleotides. It is involved in red blood cell maturation, white cell production, functioning of the immune system, and uric acid formation. It is also essential for normal growth. Some sulfa drugs increase folic acid requirements. Zinc deficiency can decrease the absorption of folic acid by reducing activity of the mucosal enzyme that creates an absorbable form of folic acid. Enzyme inhibitors are present in some foods such as cabbage, oranges, beans and peas	Deficiency—embryo abnormalities and mortality, perosis, macrocytic anemia, poor feathering, and loss of feather pigmentation. Toxicity—not reported in birds
Niacin	Water-soluble vitamin that is an important component of coenzymes NAD and NADP ,which are involved in carbohydrate, fat, and protein metabolism	Deficiency—dermatitis, perosis, stomatitis, perosis, and enlarged hocks, anemia, digestive disorders, general muscular weakness. Toxicity-coarse dense feathering and anteriorly directed short legs in chickens
C Ascorbic acid	Has not been demonstrated to be a required nutrient for most avian species. It is easily manufactured in the liver and kidneys of birds, but biosynthesis can be inhibited by deficiencies of vitamins A, E, and biotin. Ascorbic acid is involved in the synthesis of collagen, is an excellent antioxidant, and can regenerate vitamin E.	Deficiency—Signs of vitamin C deficiency have not been documented in birds
Pantothenic acid	Water-soluble vitamin that is a structural component of coenzyme A, one of the most critical coenzymes in tissue metabolism. As such it is involved in fatty acid biosynthesis and degradation, and the formation of cholesterol, triglycerides, phospholipids, and steroid hormones. It is destroyed by heat, acids, and bases	Deficiency—embryonic mortality, dermatitis, perosis, poor feathering, poor growth, fatty liver-kidney syndrome, ataxia, and reduced semen volume and fertility. Toxicity—not reported in birds

Source: Adapted from: Anderson S: Bustard micronutrient review. National Avian Research Centre External Report No. 4, Abu Dhabi, 1995; Brue RN: Nutrition. In: Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine and surgery: principles and applications*, Lake Worth, FL, 1994. Wingers Publishing, pp 63–95; McWhirter P: Malnutrition. In Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine and surgery: principles and applications*, Lake Worth, FL, 1994, Wingers Publishing, pp 842–861. Bailey TA: *Diseases and medical management of houbara bustards and other Otididae*, Chapter 5, United Arab Emirates, 2008, Avian Research Center, Environment Agency Abu Dhabi, pp 71–80.

Plasma Protein Electrophoresis in Clinically Normal Birds of Prey

Species	Bonelli's Eagle *n* = 18	Golden Eagle *n* = 33	Peregrine Falcon *n* = 29	Imperial Eagle *n* = 20	Griffon Vulture *n* = 17	Booted Eagle *n* = 12	Eagle owl *n* = 13	Barred Owl *n* = 11
Scientific Name	***Hieraaetus fasciatus***	***Aquila chrysaetos***	***Falco peregrinus***	***Aquila heliaca***	***Gyps fulvus***	***Hieraaetus pennatus***	***Bubo bubo***	***Strix varia***
Total protein	3.61 ± 0.41 (2.79-4.17)	3.76 ± 0.39 (3.19-4.4)	3.19 ± 0.68 (2.13-5.32)	3.56 ± 0.39 (3.12-4.36)	4.36 ± 0.94 (2.73-5.6)	5.13 ± 0.45 (4.6-5.6)	4.14 ± 0.65 (3.38-5.25)	3.35 ± 0.87 (2.16-2.49)
Prealbumin	0.12 ± 0.04 (0.06-0.23)	0.12 ± 0.03 (0.09-0.19)	0.09 ± 0.03 (0.05-0.2)	0.11 ± 0.02 (0.06-0.15)	0.16 ± 0.05 (0.24-1.14)	0.22 ± 0.04 (0.17-0.27)	0.10 ± 0.03 (0.05-0.14)	0.10 ± 0.04 (0.06-0.7)
Albumin	1.55 ± 0.20 (1.26-1.92)	1.48 ± 0.17 (1.22-1.85)	1.09 ± 0.25 (0.65-1.6)	1.43 ± 0.19 (1.17-1.85)	2.06 ± 0.37 (1.35-2.35)	2.10 ± 0.33 (1.63-2.52)	1.53 ± 0.31 (1.13-2.05)	1.31 ± 0.36 (0.87-1.79)
Alpha 1	0.22 ± 0.04 (0.15-0.29)	0.22 ± 0.02 (0.17-0.27)	0.10 ± 0.03 (0.04-0.17)	0.22 ± 0.04 (0.15-0.28)	0.38 ± 0.09 (0.22-0.52)	0.34 ± 0.04 (0.3-0.4)	0.15 ± 0.04 (0.09-0.14)	0.19 ± 0.06 (0.13-0.27)
Alpha 2	0.82 ± 0.09 (0.61-0.98)	0.93 ± 0.15 (0.63-1.25)	1.01 ± 0.28 (0.59-1.92)	0.90 ± 0.13 (0.65-1.23)	0.77 ± 0.22 (0.49-1.16)	1.40 ± 0.17 (1.21-1.67)	1.07 ± 0.12 (0.89-1.24)	0.78 ± 0.17 (0.57-1.01)
Beta	0.66 ± 0.12 (0.5-0.86)	0.73 ± 0.15 (0.48-1.04)	0.53 ± 0.19 (0.34-1.05)	0.66 ± 0.11 (0.49-0.9)	0.51 ± 0.19 (0.29-0.83)	0.73 ± 0.09 (0.59-0.82)	1.02 ± 0.18 (0.78-1.32)	0.71 ± 0.28 (0.39-1.13)
Gamma	0.25 ± 0.04 (0.16-0.33)	0.28 ± 0.05 (0.19-0.4)	0.38 ± 0.12 (0.23-0.78)	0.26 ± 0.05 (0.17-0.34)	0.47 ± 0.15 (0.28-0.72)	0.35 ± 0.10 (0.25-0.39)	0.26 ± 0.06 (0.19-0.33)	0.26 ± 0.06 (0.16-0.32)
A:G ratio	0.72 ± 0.19 (0.0-0.92)	0.66 ± 0.09 (0.5-0.82)	0.52 ± 0.10 (0.3-0.67)	0.67 ± 0.08 (0.59-0.86)	0.92 ± 0.12 (0.72-1.03)	0.69 ± 0.10 (0.55-0.82)	0.59 ± 0.06 (0.5-0.67)	0.64 ± 0.09 (0.54-0.79)

Extracted from: Blanco JM, Hofle U: Plasma protein electrophoresis as diagnostic and prognostic tool in raptors, *Proceedings of the European Association of Avian Veterinarians*, Tenerife, 2003, pp 256–262.
Mean ± SD (minimum-maximum); values expressed in g/dL.

Serum Protein Values (g/dL) for Clinically Healthy Falcons (Group 1) and for Falcons with Confirmed Aspergillosis (Group 2)

Variable	Mean (SD)	95 % CI	Median	Interquartile Range	Reference Interval	*P* Value
Group 1 (n = 73)						
TP, g/dL (biuret)	3.18 ± 1.78	2.77-3.60	2.7	2.20-3.93	0-6.46	
Albumin, g/dL (BCG)[a]	1.14 ± 0.48	0.10-1.28	1.2	0.70-1.50	0.14-2.12	
A:G ratio[a]	1.36 ± 0.37	0.48-2.24	1.43	1.14-1.60	0.63-2.15	
Total albumin, g/dL	1.74 ± 0.80	1.55-1.93	1.59	1.23-2.14	0-3.22	
Prealbumin, g/dL (SPE)	0.68 ± 0.32	0.60-0.75	0.65	0.49-0.82	0-1.27	
Albumin, g/dL (SPE)	1.08 ± 0.53	0.9.6-1.21	0.99	0.73-1.23	0-2.03	
Alpha globulin, g/dL (SPE)	0.48 ± 0.42	0.38-0.58	0.39	0.29-0.54	0-1.22	
Beta globulin, g/dL (SPE)	0.45 ± 0.25	0.39-0.50	0.36	0.31-0.52	0-0.91	
Gamma globulin, g/dL (SPE)	0.53 ± 0.49	0.41-0.64	0.37	0.26-0.59	0-1.38	
Group 2 (n = 32)						
TP, g/dL (biuret)	2.66 ± 1.57	2.10-3.23	2.4	1.65-3.05		.06
Albumin, g/dL (BCG)	0.92 ± 0.52	0.73-1.11	0.88	0.56-1.18		.03[b]
A:G ratioa	1.22 ± 0.54	1.02-1.42	1.14	0.78-1.61		.14
Total albumin, g/dL	1.32 ± 0.76	1.04-1.59	1.24	0.87-1.52		.002[b]
Prealbumin, g/dL (SPE)	0.43 ± 0.34	0.29-0.56	0.34	0.22-0.53		<.001[b]
Albumin, g/dL (SPE)	0.93 ± 0.52	0.75-1.12	0.83	0.63-1.12		.09
Alpha globulin, g/dL (SPE)	0.44 ± 0.40	0.30-0.59	0.28	0.21-0.54		.12
Beta globulin, g/dL (SPE)	0.42 ± 0.33	0.30-0.54	0.34	0.19-0.48		.17
Gamma globulin, g/dL (SPE)	0.49 ± 0.42	0.34-0.64	0.33	0.24-0.52		.42

Source: Kummrow M, Vorbruegen S, Silvanose C, et al: Serum protein electrophoresis in healthy and Aspergillus sp. infected falcons, *J Avian Med Surg* 26(4):213–220, 2012.
[a]Normally distributed (Kolmogorov-Smirnov test).
[b]Statistically significant ($P < .05$).
CI indicates confidence interval; TP, total protein; A:G, albumin : globulin; BCG, bromocresol green; SPE, serum protein electrophoresis. SI conversion factor: To convert g/dL to g/L, multiply by 10.

Plasma Protein Electrophoresis in Selected Psittacine Species

Species	Hyacinthine Macaw *n* = 8	Blue and Yellow Macaw *n* = 11	Green-Winged Macaw *n* = 10	Scarlet Macaw *n* = 12	Military Macaw *n* = 11
Scientific Name	***Anodorhynchus hyacinthinus***	***Ara ararauna***	***Ara chloroptera***	***Ara macaw***	***Ara militaris***
Total protein	30.3 ± 3.9 (23.8-38.5)	29.2 ± 7.0 (17.9-46.5)	36.7 ± 9.8 (18.0-59.9)	38.3 ± 6.4 (28.6-47.5)	29.6 ± 8.2 (17.2-44.4)
Prealbumin	5.9 ± 5.7 (0.0-14.9)	10.1 ± 6.3 (3.0-21.8)	9.3 ± 7.2 (0.0-17.2)	10.2 ± 4.8 (0.0-14.3)	9.5 ± 6.2 (0.0-18.5)
Albumin	58.0 ± 9.9 (45.9-69.2)	54.6 ± 3.0 (51.4-59.2)	64.1 ± 5.1 (55.3-70.5)	55.6 ± 11.8 (34.6-66.6)	50.0 ± 12.0 (34.5-70.6)
Alpha	7.5 ± 3.2 (3.3-12.2)	7.8 ± 5.1 (1.7-15.9)	6.7 ± 3.6 (2.1-11.9)	8.8 ± 8.2 (2.0-28.5)	10.5 ± 3.9 (5.8-18.3)
Beta	14.4 ± 5.3 (5.6-20.3)	17.0 ± 7.0 (11.3-31.6)	12.3 ± 6.0 (4.9-24.3)	14.9 ± 9.1 (5.2-37.7)	17.8 ± 7.2 (8.1-32.5)
Gamma	14.3 ± 8.5 (2.2-25.6)	10.5 ± 4.7 (2.6-16.4)	7.5 ± 4.6 (1.7-16.5)	10.4 ± 5.6 (1.5-20.1)	12.1 ± 5.1 (1.8-19.6)
A:G ratio	1.9 ± 0.6 (1.3-3.0)	1.9 ± 0.5 (1.3-2.8)	2.9 ± 0.8 (1.6-4.1)	2.3 ± 1.1 (0.6-4.1)	1.6 ± 0.5 (0.6-2.4)

Source: Modified from: Polo FJ, Peinado VI, Viscor G, Palomeque J: Hematologic and plasma chemistry in captive psittacine birds, *Avian Dis* 42:523–535, 1998.
Mean ± SD (minimum-maximum).

Plasma Protein Electrophoresis in Selected Psittacine Species—cont'd

Species	Yellow-Shouldered Amazon (*n* = 29)	Red-Browed Amazon (*n* = 18)
Scientific Name	***Amazona barbadensis***	***Amazona rhodocorytha***
Total protein	4.8 ± 6.1	45.06 ± 3.26
Prealbumin	11.42 ± 3.67 (24.99 ± 6.87)	7.74 ± 1.21 (17.28 ± 3.16)
Albumin	23.79 ± 3.86 (53.31 ± 5.60)	27.30 ± 3.27 (60.35 ± 3.61)
Alpha	2.88 ± 0.66 (6.37 ± 1.24)	2.68 ± 0.73 (5.97 ± 1.60)
Beta	3.19 ± 0.96 (7.11 ± 2.20)	3.39 ± 0.46 (7.4 ± 1.07)
Gamma	3.74 ± 1.22 (8.23 ± 2.33)	4.0 ± 1.1 (8.83 ± 2.21)
A:G ratio	3.80 ± 1.04	3.56 ± 0.69

Source: Bürkle M, Silveira Viera L, Dreisörner CJ, et al: Electrophoresis in Psittaciformes normal values and selected cases. *Proceedings of the European Association of Avian Veterinarians*, Tenerife, 2003, pp 253–255.
Mean ± SD (minimum-maximum); values expressed in g/dL, percentage in parentheses.

Plasma Protein Electrophoresis in Selected Birds

Species	Bar-Headed Goose, (*n* = 62)	White Stork, (*n* = 42)	Domestic Pigeon, (*n* = 36)	Jackdaw, (*n* = 44)	Black Kite, (*n* = 34)	African Grey Parrot, (*n* = 13)	Black Smith Plover, (*n* = 8)
Scientific Name	***Anser indicus***	***Ciconia ciconia***	***Columba livia***	***Corvus monedula***	***Milvus migrans***	***Psittacus erithacus***	***Vanellus armatus***
Total protein	4.11 ± 0.51	4.10 ± 0.34	3.68 ± 0.49	3.60 ± 0.55	4.01 ± 0.32	3.71 ± 0.40	3.61 ± 0.40
Albumin	2.53 ± 0.26	1.60 ± 0.17	1.06 ± 0.15	1.41 ± 0.22	1.76 ± 0.24	1.99 ± 0.24	1.55 ± 0.26
Alpha 1	0.23 ± 0.05	0.84 ± 0.17	0.08 ± 0.01	0.09 ± 0.27	0.29 ± 0.13	0.06 ± 0.01	0.19 ± 0.04
Alpha 2	0.75 ± 0.17	0.60 ± 0.11	0.13 ± 0.17	0.81 ± 0.15	1.03 ± 0.23	0.90 ± 0.19	0.93 ± 0.19
Beta	0.57 ± 0.16	0.43 ± 0.09	0.97 ± 0.27	1.05 ± 0.27	0.61 ± 0.16	0.57 ± 0.09	0.63 ± 0.20
Gamma	0.20 ± 0.07	0.53 ± 0.11	0.33 ± 0.10	0.20 ± 0.08	0.31 ± 0.08	0.23 ± 0.04	0.29 ± 0.05
A:G ratio	0.13 ± 0.02	0.06 ± 0.00	0.04 ± 0.01	0.06 ± 0.01	0.07 ± 0.01	0.11 ± 0.02	0.07 ± 0.01

Source: Modified from: Ordonneau D, Roman Y, Chaste-Duvernoy D, Bomsel MC: Plasma electrophoresis reference ranges in various bird species. *Proceedings of the European Association of Avian Veterinarians*, Arles, 2005, pp 283–287.
Mean ± SD (minimum-maximum); values expressed in g/dL.

Results of Protein Electrophoresis for Clinically Normal Elegant-Crested Tinamou (*n* = 19)

Analyte	Mean ± SD (SEM)	Reference Interval	Median
Total protein (g/dL)	5.19 ± 0.68 (0.16)	4.00-7.00	5.2
Albumin : globulin	1.36 ± 0.27 (0.06)	0.86-2.01	1.33
Prealbumin			
Absolute (g/dL)*	0.04 ± 0.09 (0.02)	0.00-0.28	0.00
Relative (%)	0.60 ± 1.40 (0.32)	0.00-4.00	0.00
Albumin			
Absolute (g/dL)	2.94 ± 0.45 (0.10)	2.02-3.90	2.90
Relative (%)	56.61 ± 4.71 (1.08)	46.46-63.23	56.14
α-1 globulins			
Absolute (g/dL)*	0.21 ± 0.10(0.02)	0.12-0.46	0.18
Relative (%)	4.00 ± 1.56 (0.36)	2.60-7.67	3.46
α-2 globulins			
Absolute (g/dL)	0.72 ± 0.10 (0.02)	0.51-0.93	0.69
Relative (%)	13.99 ± 2.14 (0.49)	9.29-17.22	13.89
β-globulins			
Absolute (g/dL)*	0.79 ± 0.14 (0.03)	0.61-1.15	0.74
Relative (%)	15.33 ± 2.24 (0.51)	11.73-21.25	15.56
γ-globulins			
Absolute (g/dL)	0.50 ± 0.13 (0.03)	0.30-0.78	0.52
Relative (%)	9.56 ± 2.38 (0.55)	6.13-14.44	9.63

Source: Black P, Macek M, Tieber A, Weber M: Reference values for hematology, plasma biochemical analysis, plasma protein electrophoresis, and Aspergillus serology in elegant-crested tinamou (Eudromia elegans), *J Avian Med Surg* 27(1):16, 2013.
*Data are not normally distributed; mean, SD, and SEM listed for comparison.
SD, Standard deviation; SEM, standard error of the mean.

Plasma Electrophoresis Reference Values in Captive Adult, Clinically Normal Indian Peafowl (n = 69)

Parameter	$P_{2.5}$-$P_{97.5}$*	Mean	SD	Range
Total protein (g/dL)	2.88-5.23	4.11	0.62	2.72-5.43
Albumin (g/dL)	1.47-2.46	2.03	0.27	1.33-2.47
Alpha-1 globulin (g/dL)	0.00-1.15	0.65	0.3	0.00-1.30
Alpha-2 globulin (g/dL)	0.02-1.78	0.89	0.46	0.00-2.04
Beta globulin (g/dL)	0.00-1.43	0.38	0.51	0.00-1.49
Gamma globulin (g/dL)	0.00-1.31	0.15	0.40	0.00-1.79
A : G ratio	0.67-1.20	0.99	0.13	0.63-1.36

Source: Samour J, Naldo J, Rahman H, Sakkir M: Hematologic and plasma biochemical reference values in Indian Peafowl (*Pavo cristatus*), *J Avian Med Surg* 24(2):99–106, 2010.
*Recommended reference values.

3 APPENDIX

Selected Avian Literature

Merle M. Apo

Altman RB, Clubb SL, Dorrestein GM, Quesenberry K: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders. ISBN 978-0721654461.

Altman RB, Forbes NA: *Self-assessment color review of avian medicine*, Ames, IA, 1998, Iowa State University Press. ISBN 978-0813823393.

Arnall L, Keymer IF: *Bird diseases: an introduction to the study of birds in health and disease*, Neptune City, NJ, 1975, THF Publications. ISBN 978-0876669501.

Atkinson CT, Nancy J, Thomas NJ, Hunter DB: *Parasitic diseases of wild birds*, Hoboken, NJ, 2008, Wiley-Blackwell. ISBN 978-0813820811.

Beer JV: *Diseases of gamebirds and wildfowl*, Fordingbridge, Hampshire, UK, 1988, Game Conservancy. ISBN B0000EHJ1D.

Beynon PH, Forbes NA, Harcourt-Brown NH: *Manual of raptors, pigeons and waterfowl*, Cheltenham, Gloucestershire, UK, 1996, British Small Animal Veterinary Association. ISBN 978-0813828763.

Beynon PH, Forbes NA, Lawton MPC: *Manual of psittacine birds*, Cheltenham, Gloucestershire, UK, 1995, British Small Animal Veterinary Association. ISBN 978-0905214306.

Brearley MJ, Cooper JE, Sullivan M: *A colour atlas of small animal endoscopy*, London, UK, 1991, Wolfe Publishing. ISBN 978-3211285411.

Burr EW: *Diseases of parrots*, Neptune City, NJ. USA, 1982, TFH Publications. ISBN 978-0876668436.

Burr EW: *Companion bird medicine*, St. Louis, MO, 1995, Mosby. ISBN 978-0813803623.

Campbell TW: *Avian hematology and cytology*, ed 2, Ames, IA, 1988, Iowa State University Press. ISBN 978-0813800646.

Campbell TW: *Clinical cases in avian and exotic animal hematology and cytology*, Hoboken, NJ, 2010, Wiley-Blackwell. ISBN 978-0813816616.

Canon JM: *A guide to basic health and disease in birds: their management, care and well-being: their management, care and breeding*, ed 2, Australia, 2002, ABK Publications. ISBN 978-0957702455.

Capua I, Alexander DJ: *Avian influenza and new castle disease: a field and laboratory manual*, Philadelphia, PA, 2009, Springer. ISBN 978-8847008250.

Carpenter JW: *Exotic animal formulary*, ed 4, Philadelphia, PA, 2012, WB Saunders. ISBN 978-1437722642.

Chitty J, Lierz M: *BSAVA manual of raptors, pigeons and passerine birds*, Gloucester, UK, 2008, Quedgeley. ISBN 978-1905319046.

Clark P, Boardman W, Raidal S: *Atlas of clinical avian hematology*, Hoboken, NJ, 2009, Wiley-Blackwell. ISBN 978-1405192484.

Coles BH: *Essentials of avian medicine and surgery*, Oxford, UK, 2007, Blackwell Publishing. ISBN 978-1405157551.

Coles BH, Krautwald-Junghanns ME: *Avian medicine: self-assessment picture test in veterinary medicine*, St. Louis, MO, 1998, Mosby. ISBN 978-0723430087.

Cooper JE: *Veterinary aspects of captive birds of prey*, Saul, Gloucestershire, UK, 1978, Standfast Press. ISBN 978-0904602043.

Cooper JE: *Disease and threatened birds*, Guildford, Surrey, UK, 1989, Anagram Editorial Service. ISBN 978-0946888184.

Cooper JE: *Birds of prey: health and disease*, ed 3, Oxford, UK, 2002, Blackwell Science. ISBN 978-0632051151.

Cooper J, Cooper M: *Captive birds in health and disease*, Fordingbridge, Hampshire, UK, 2003, World Pheasant Association, Hancock House Publishers. ISBN 978-0888395382.

Cooper JE, Greenwood AG: *Recent advances in the study of raptor diseases*, Keighley, West Yorkshire, UK, 1981, Chiron Publications. ISBN 978-0950771601.

Doneley R: *Avian medicine and surgery in practice: companion and aviary birds*, Boca Raton, FL, 2010, CRC Press. ISBN 978-1840761121.

Drenowatz C: *Ratite encyclopedia: ostrich, emu and rhea*, San Antonio, CA, 1995, Ratite Record. ISBN 0064294021.

Fowler ME: *Zoo and wildlife animal medicine*, Philadelphia, PA, 1978, WB Saunders. ISBN 978-0721665597.

Fowler ME: *Zoo and wild animal medicine*, ed 2, Philadelphia, PA, 1986, WB Saunders. ISBN 978-0721610139.

Fowler ME: *Zoo and wild animal medicine: current therapy*, ed 3, Philadelphia, PA, 1993, WB Saunders. ISBN 978-0721636672.

Fowler ME: *Wild animal medicine: current therapy*, ed 4, Philadelphia, PA, 1999, WB Saunders. ISBN 978-0721686646.

Fowler ME, Miller RE: *Wild animal medicine: current therapy*, ed 5, Philadelphia, PA, 2003, WB Saunders. ISBN 978-0721694993.

Fowler ME, Miller RE: *Wild animal medicine: current therapy*, ed 6, Philadelphia, PA, 2007, WB Saunders. ISBN 978-3642034534.

Friend M: *Field guide to wildlife diseases: general field procedures and diseases of migratory birds*, Washington, DC, 1987, United States Government Printing. B00BXV00OQ.

Fudge AM: *Laboratory medicine: avian and exotic pets*, Philadelphia, PA, 2000, WB Saunders. ISBN 978-0721676791.

Gage LJ, Duerr RS: *Hand-rearing birds*, Hoboken, NJ, 2008, Wiley-Blackwell. ISBN 978-0813806662.

Ginn HB, Melville DS: *Moult in birds*, Thetford, Norfolk, UK, 2000, British Trust for Ornithology. ISBN 978-0903793025.

Girling SJ: *Veterinary nursing for exotic pets*, ed 2, Hoboken, NJ, 2013, Wiley-Blackwell. ISBN 978-0470659175.

Harcourt-Brown N, Chitty J: *BSAVA manual of psittacine birds*, ed 2, Gloucester, UK, 2005, British Small Animal Veterinary Association. ISBN 978-0905214764.

Harrison GJ, Harrison LR: *Clinical avian medicine and surgery*, Philadelphia, PA, 1986, WB Saunders. ISBN 978-0721612416.

Harrison L, Johnson-Delaney C: *Exotic companion medicine handbook for veterinarians* (2 vol), Lake Worth, FL, 1996, Wingers Publishing. ISBN 978-0963699640.

Harrison GJ, Lightfoot TL: *Clinical avian medicine*, Palm Beach, FL, 2006, Spix Publishing. ISBN 978-0975499405.

Heidenreich M: *Birds of prey: medicine and management*, Oxford, UK, 1997, Blackwell Science. ISBN 978-0632041862.

Jacobson ER, Kollias GV Jr: *Exotic animals: contemporary issues in small animal practice*, NY, 1988, Churchill Livingstone. ISBN 978-0443084089.

King AS, McLelland J: *Birds: their structure and function*, ed 2, Eastbourne, East Sussex, UK, 1984, Baillière Tindall. ISBN 978-0702008726.

Klasing RC: *Comparative avian nutrition*, Wallingford, Oxfordshire, UK, 2000, CAB International. ISBN 978-0851992198.
Lumeij JT: *Clinical investigative methods for birds*, Rijksuniversiteit Utrecht, The Netherlands, 1987, Vakgroep Geneeskunde van het Kleine Huisdier Faculteit der Diergeneeskunde. ISBN 90-9001845-X.
Lumeij JT, Remple JD, Redig PT, et al: *Raptor biomedicine III, including bibliography of diseases of birds of prey*, Lake Worth, FL, 2000, Zoological Education Network. ASIN B000P1JWLM.
Mayer J, Doneley T: *Clinical veterinary advisor: birds and exotic pets*, Philadelphia, PA, 2012, WB Saunders. ISBN 978-1416039693.
McLelland J: *A colour atlas of avian anatomy*, London, UK, 1990, Wolfe Publishing. ISBN 978-0723415756.
Miller RE, Fowler ME: *Fowler's zoo and wild animals medicine: current therapy*, ed 7, Philadelphia, PA, 2011, WB Saunders Elsevier. ISBN 978-1437719864.
Miller RE, Fowler ME: *Fowler's zoo and wild animal medicine: current therapy*, ed 8, Philadelphia, PA, 2014, WB Saunders Elsevier. ISBN 978-1455773978.
Mitchell M, Tully TN: *Manual of exotic pet practice*, Philadelphia, PA, 2008, WB Saunders. ISBN 978-1416001195.
Muller GM: *Practical handbook of falcon husbandry and medicine*, Hauppauge, NY, 2009, Nova Science Publishers, Inc. ISBN 978-1607416081.
Olsen J: *Caring for birds of prey*, Springville, CA, 1990, Wild Ones Animal Books. ISBN 978-1886013001.
Olsen GH, Orosz SE: *Manual of avian medicine*, St Louis, MO, 2000, Mosby. ISBN 978-0815184669.
Orosz SE, Ensley PK, Jaynes CJ: *Avian surgical anatomy. thoracic and pelvic limbs*, Philadelphia, PA, 1992, WB Saunders. ISBN 978-0721636542.
Patisson M, MacMullin PF, Bradbury JM, Alexander DJ: *Poultry diseases*, ed 6, Philadelphia, PA, 2007, Saunders Ltd. ISBN 978-0702028625.
Petrak ML: *Diseases of cage and aviary birds*, ed 2, Philadelphia, PA, 1982, Lea & Febiger. ISBN 978-0812106923.
Randall CJ: *A Colour atlas of diseases and disorders of the domestic fowl and turkey*, ed 2, London, UK, 1991, Wolfe Publishing. ISBN 978-0723408277.
Randall CJ: *Diseases and disorders of the domestic fowl and turkey*, ed 2, London, UK, 1997, Mosby–Wolfe. ISBN 978-0813803760.
Randall C, Reece RL: *Color atlas of avian pathology*, London, UK, 1996, Mosby–Wolfe. ISBN 978-0723420873.
Redig PT, Cooper JE, Remple DJ, Hunter DB: *Raptor biomedicine*, Minneapolis, MN, 1993, University of Minnesota Press. ISBN 978-0816622191.
Riddell C: *Avian histopathology*, ed 2, Saskatoon, Saskatchewan, Canada, 1996, American Association of Avian Pathologists. ISBN 978-0915538034.
Ritchie BW: *Avian viruses: function and control*, Lake Worth, FL, 1995, Wingers Publishing. ISBN 978-0963699633.
Ritchie BW, Harrison GJ, Harrison LR: *Avian medicine: principles and application*, Lake Worth, FL, 1994, Wingers Publishing. ISBN 978-0963699602.
Rosenthal KL, Forbes N, Frye FL, Lewbart GA: *Rapid review of exotic animal medicine and husbandry: pet mammals, birds, reptiles, amphibians and fish*, Boca Raton, FL, 2008, CRC Press. ISBN 978-1840760552.
Rosskopf W, Woerpel R: *Diseases of cage and aviary birds*, ed 3, Philadelphia, PA, 1996, Williams & Wilkins. ISBN 978-0683073829.
Rupley AE: *Manual of avian practice*, Philadelphia, PA, 1997, WB Saunders. ISBN 978-0721640839.
Saif YM, Barnes HJ, Glisson JR, et al: *Diseases of poultry*, Hoboken, NJ, 2008, Wiley-Blackwell. ISBN 978-0813807188.
Samour J: *Exotic animal medicine: review and test*, Philadelphia, PA, 2012, WB Saunders Elsevier. ISBN 978-0702044441.
Samour J, Naldo LJ: *Anatomical and clinical radiology of birds of prey: including interactive advanced anatomical imaging*, Philadelphia, PA, 2006, WB Saunders. ISBN 978-0702028021.
Schat KA, Kaspers B, Kaiser P: *Avian immunology*, ed 2, London, UK, 2013, Academic Press. ISBN 978-0123969651.
Schmidt RE, Reavill DR, Phalen DN: *Pathology of pet and aviary birds*, Ames, IA, 2003, Iowa State Press. ISBN 978-0813805023.
Silverman S, Tell L: *Radiology of birds: an atlas of normal anatomy and positioning*, Philadelphia, PA, 2009, WB Saunders. ISBN 978-0721606354.
Silvy NJ: *The wildlife techniques manual: volume 1, research; Volume 2: management* (2 vols), ed 7, Baltimore, MD, 2012, Johns Hopkins University Press. ISBN 978-1421401591.
Stark JM, Ricklefs RE: *Avian growth and development*, Oxford, UK, 1998, Oxford University Press. ISBN 978-0195106084.
Stroud R: *Diseases of canaries*, Worcestershire, UK, 2006, Home Farm Books. ISBN 978-1406795394.
Thomas NJ, Hunter DB, Atkinson AT: *Infectious diseases in wild birds*, Hoboken, NJ, 2007, Wiley-Blackwell. ISBN 978-0813828121.
Tudor DC: *Pigeon health and disease*, Ames, IA, 1991, Iowa State University Press. ISBN 978-0813812441.
Tully TN, Dorrestein G, Jones A: *Handbook of avian medicine*, ed 2, Philadelphia, PA, 2009, WB Saunders. ISBN 978-0702028748.
Tully TN, Lawton MPC, Dorrestein GM: *Avian medicine*, Oxford, UK, 2000, Butterworth-Heinemann. ISBN 978-0750635981.
Tully TN, Shane SM: *Ratites: management, medicine and surgery*, Malabar, FL, 1996, Krieger Publishing. ISBN 978-0894648748.
Wernery R, Wernery U, Kinne J, Samour J: *Colour atlas of falcon medicine*, Hannover, Germany, 2004, Schlütersche. ISBN 978-3899930078.
Whiteman CE, Bickford AA: *Avian diseases manual*, ed 3, Saskatoon, Saskatchewan, Canada, 1989, American Association of Avian Pathologists. ISBN 978-0840357953.
Whittow GC: *Sturkie's avian physiology*, ed 3, London, UK, 2000, Academic Press. ISBN 978-0127476056.

Legislation and Codes of Practice Relevant to Avian Medicine

Margaret E. Cooper

It is useful and, indeed, important for those who work in avian medicine to be aware of the legislation relevant to their work, their clients, and their patients.

Legislation is very largely produced by individual countries and therefore is subject to variation, on any particular topic, from nation to nation. Even within a country that has a federal constitution, law-making powers may be given to provincial authorities, creating further variety. Consequently, in an international publication such as this, it may be more helpful to indicate the types and fields of legislation that are most commonly encountered and most significant to the avian practitioner than to discuss specific pieces of national legislation. Individuals may use this summary to identify the sort of laws that are appropriate and then seek in their own jurisdiction for specific legal provisions.

In modern times, many countries make their legislation available on the Internet. The actual laws and additional guidance on their implementation can often be found on government websites (see Table of Websites and Literature).

LEGISLATION

Legislation is made at several levels

Level	Source	Examples
International	Global treaties	Convention on the International Trade in Endangered Species of Wild Fauna and Flora (CITES) Convention on the Conservation of Migratory Species of Wild Animals (CMS)
Regional	European Union (EU): 28 countries	EU regulations: CITES Regulations Directives: Birds Directive; Habitats Directive
	Council of Europe (CoE): 47 countries	Conventions on conservation, pets, animal transport, animal research
National	Individual countries (see examples of national law databases in Table of Websites and Literature)	Primary and secondary legislation, for example, statutes, orders, regulations, decrees.
Provincial	Within a country, for example, state, province, canton, land.	
Local	District, city	Bye-laws

Legislation on particular topics may be found at several levels; for instance, trade in endangered species is subject to global, regional and national legislation although, in practice, the effective administration and enforcement of the laws is usually implemented at national or provincial level.

In some countries the keeping and use of birds is highly regulated, yet in others the legislation is not well developed or little control is exercised and/or laws are not effectively enforced.

In some situations, there are voluntary, nonlegal provisions, often in the form of guidelines, codes of practice or rules. These are followed on a voluntary basis as a matter of self-regulation or because no legislation is in place.

It should always be remembered that legislation can change from time to time and it is important that the reader should ensure that he/she has up-to-date information on the law.

FIELDS OF LAW RELEVANT TO AVIAN MEDICINE

The veterinarian and veterinary practice

Professional

Most countries regulate the practice of veterinary medicine and surgery. This is to ensure:

- That the veterinarians are properly trained and qualified to practice medicine and surgery in animals.
- That they can provide and maintain adequate standards of treatment and facilities.
- That they follow ethical standards such as client confidentiality, advertising, and animal welfare.
- That these requirements are overseen by a veterinary board or other authority.
- That it is responsible for registering and authorizing veterinarians to practice.
- That it has powers to take action if veterinarians do not comply with the national veterinary law or standards. The level of enforcement varies from country to country, from very strict to nil.

The veterinary legislation defines which activities are included in medicine and surgery and which animals are covered. Activities not included may be carried out by non-veterinarians and, likewise, species excluded from the veterinary law may be treated by lay persons.

Veterinarians normally have to be registered in the country where they practice. Veterinarians whose qualifications or examinations are not recognized in the country where they wish to work may have to satisfy additional requirements including taking an examination or restrictions on the type of veterinary work that they can do.

This may cause difficulties for nonregistered veterinarians who are asked to treat a bird because of their particular skills or because there is no one else available. Some countries are very strict about this issue; others, for example, Kenya, are beginning to enforce their laws. On the other hand, registration may not always be practicable, the law not enforced, and a bird may be in serious need of help. An unregistered veterinarian should be very careful when invited to treat a bird and should assess his/her position and the legislation and level of enforcement before carrying out treatment. It may be better to train a local veterinarian who is authorized to practice, thus avoiding prosecution and also leaving skills in country for the future.

Malpractice/Negligence

A veterinarian should practice to a level of competence that is required by the law of the land. If not, he/she may risk being sued for compensation by the owner of the animal treated. The person complaining must show that there was a contract or some agreement between the parties as to the veterinary treatment, that normal standards were not met, and that the person suing has suffered loss (e.g., the animal is harmed or died unnecessarily).

In some counties, it is normal for a veterinarian to be insured against claims and other liabilities, but at the other extreme there is no concept of professional indemnity insurance. It is wise for a veterinarian to have insurance, particularly where his/her work includes animals of high value such as raptors and rare species or when providing veterinary services for a captive breeding business, zoo, or other valuable collection. Insurance may also cover legal expenses and other costs in the event of disciplinary investigations. Insurance schemes often provide advice and support in such circumstances.

Medicines

Most countries have legislation that controls the production, supply, and administration of medicinal products. This often includes especially strict rules on the possession and supply of dangerous/controlled drugs. This law may be very carefully enforced as in the European Union, United States, Canada, Australia, and New Zealand. In Africa, Kenya is rapidly tightening the enforcement of its laws.

On the other hand, there are countries where regulation either does not exist or is not enforced, making it easy for anyone to obtain drugs or to supply illegal or unauthorized medicinal products of doubtful quality.

Health and Safety

Some countries have extensive legal provisions setting high standards of safety at work and for the well-being of employees. This usually extends also to volunteers and other visitors to the workplace. This includes regulations for specific situations such as precautions against fire, the hazards of dangerous substances and equipment, as well as the working environment. Health and safety are managed by way of risk assessments that evaluate risks of any procedure or situation and devise the means of preventing the hazards involved.

There are still many countries where such legislation either does not exist or is not enforced. Those working in such situations can still voluntarily make their own risk assessments and set standards and provide training and appropriate protection. This is particularly important when working in remote areas or in situations where standards are low or not understood by workers.

Animal Legislation

Animal Welfare

Many countries have legislation that makes it an offense to treat animals (including birds) cruelly or to cause them unnecessary suffering. There is also a growing trend in modern animal welfare legislation to impose a legal "duty of care" on the person responsible for an animal to provide properly for the animal's welfare. This may include providing appropriate accommodation, food and water, and health care. A person may be prosecuted for failing to provide the needs of an animal even if no unnecessary suffering has occurred. There may be additional legislation relating to the welfare of birds while they are being transported. If birds are used for research, whether in captivity or in the wild, special authorization is often required by the laws regulating scientific research or wildlife conservation.

Wildlife Conservation

Most countries have legislation that is designed to protect their wildlife, including birds. The laws are likely to regulate the taking, killing, or injuring of wild birds and their young. Nests and eggs are also protected. There may be additional restrictions or penalties when rare and vulnerable species are concerned. Licenses may be issued to permit certain activities such as pest control or the protection of public health or safety.

In modern legislation, it is often illegal to release into the wild "alien," that is, nonindigenous species, including birds, because of the threat they may pose to indigenous species. Hunting with birds may be controlled or forbidden (see in the following sections).

Animal Health

Animal health legislation includes:

- Controls on the measures that are taken to prevent the spread of disease in animals within a country. Although this is primarily aimed at farm animals, including poultry, some of the laws also apply to birds (and other animals) kept for other purposes. It includes action to be taken to control a disease outbreak such as avian flu, including restrictions on the movement of animals.
- The regulation of importation and exportation or animals (see in the following sections).

LAW ENFORCEMENT

The veterinarian with special skills in avian medicine may be asked to assist with law enforcement investigations, particularly regarding animal welfare or wildlife conservation. Other occasions may involve requests to provide factual or expert evidence to support a prosecution or a claim for compensation, or in a dispute about ownership, insurance, or in a disciplinary action.

This may include the assessment of a bird's condition to provide evidence of its health or welfare. Assessments are also used to indicate whether the bird is wild caught or captive bred, the cause of wounds, or its psychological condition.

Attendance at a crime scene may mean that the veterinarian is asked to examine birds found there (or birds that have been confiscated), taking DNA samples, and providing a form of identification for birds and specimens. Forensic investigation may include the assessments of birds for evidence to indicate whether they were captive bred or wild caught. In these situations, the veterinarian is likely to be asked to prepare a report and/or give evidence in court. The report has to be presented and the evidence has to be given according to the rules of the court where the case is heard (see Cooper and Cooper, this volume).

BIRDS

Several areas of law are applicable to captive or free-living birds.

- *In captivity*, birds may be kept as pets, for show, sport, breeding, trade, research, treatment or rehabilitation. Authorization (e.g. permit/license) may be required: to keep a bird privately; for commercial or sporting purpose; to take a wild bird into captivity; or to study them in the wild. Responsibility must be taken for the welfare of a bird in captivity: in its day-to-day life, when being transported. If birds are used for research, whether in captivity or in the wild, special authorization is often required by the laws regulating scientific research or wildlife conservation.
- *Many species of free-living bird* are protected against: hunting, killing, injury or capture, and the use of certain methods of killing or capture. Eggs, young and nests may also be protected. Habitat protection (e.g. national parks, forests, etc.) restricts access to, and protects, free-living birds. Authorization (permit, license) may allow certain activities for purposes such as possession, breeding, research, zoological collections, the rehabilitation of sick or injured birds, and commerce. Legal attitudes to falconry can vary from total prohibition (e.g. Norway) to not being regulated by law as a sport (as in the United Kingdom [UK]), although possession of the birds, taking the quarry and the sale of quarry, which count as game, are regulated. In the United States of America (USA), falconry training and permits are required, in most cases under state law. Falconry clubs may impose rules and standards. In the UK they are responsible for a measure of self-regulation in respect of bird licensing.
- *Trade in birds*: at a national level trade in protected species may be controlled as part of wildlife legislation—permits to sell may be restricted to captive-bred specimens. Likewise, a permit may be required to export indigenous species of birds (in addition to authorization under the Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES) and the animal health legislation)
- *International trade and movement of birds*: the movement of endangered species between countries is regulated by CITES (see below) legislation implemented by individual countries. Permits are required to import and export species listed on Appendices I, II and III of CITES. Permits are not given for primarily commercial purposes for Appendix I species. Import and export for commercial and non-commercial purposes of Appendix II species is allowed under permit. This applies to whole birds, parts or derivatives (e.g. diagnostic samples) of a bird. CITES has made special provision for a simplified procedure for some types of diagnostic samples in the CITES Resolution Conf. 12.3 (Rev. CoP16). This is implemented by the European Union (EU) in Article 18 of Regulation (EC) No 865/2006.

Some countries apply stricter controls or list more species than does CITES itself. CITES management authorities (usually the government department responsible for the environment, but sometimes for agriculture) may issue guidance notes for permit applicants. In the EU, CITES is operated under EU Regulations that apply uniformly amongst all 28 member states, whereas national laws deal only with enforcement. Once a CITES bird is legally present in a member state, it may be moved freely within the EU. A wide range of birds is listed in the Convention Appendices, including most birds of prey. The EU CITES Regulation lists and upgrades many additional birds in its Annexes A–D. From 1 July 2007, it is illegal to import wild-caught birds into the EU on both disease risk and welfare grounds.

- *Animal health*: birds moving from one country to another are likely to need a veterinary health check and certificate and, in some countries, to go into quarantine (around 35 d) on arrival. This is normally regulated by the government department responsible for agriculture. Special restrictions may operate when there is a risk of a pandemic as in the case of Highly Pathogenic Avian Influenza (H5N1).
- *Customs control and charges* apply to imported birds. CITES and animal health importation documentation should be presented at customs on arrival, even if carrying samples and derivatives in personal baggage.

EUROPE

EU: European Union: The EU has 28 member states. It has legislation (regulations or directives) on a number of subjects relevant to avian medicine. Regulations (e.g. on CITES) need no further legislation and one refers to the provisions of the actual regulations. Directives (e.g. on veterinary laws, medicinal products, animal health, health and safety, wild birds, habitat conservation) take the form of directives and require national legislation to implement their provisions. Enforcement (including powers, offenses and penalties), even for regulations, is normally a matter for national law. The Council of Europe has 47 Member States and is primarily concerned with social and cultural issues. It has produced Conventions on (*inter alia*) conservation, pets, animal transport, and animal research.

Table of Websites and Literature

Topic	Website	Information/Comment
Convention on International Trade in Endangered Species of Wild Fauna and Flora (CITES)	ww.cites.org	CITES
		Structure of CITES, Convention, and other official documentation
	http://www.cites.org/eng/res/12/12-03R16.php	CITES: Resolution Conf. 12.3 (Rev. CoP16)
		Species information
	http://www.cites.org/eng/resources/transport/index.php	CITES (2013) *Guidelines for the nonair transport of live wild animals and plants* (CoP16, Bangkok, 2013)
Convention on the Conservation of Migratory Species of Wild Animals (CMS)	http://www.cms.int/	CMS
	http://www.cms.int/en/cms-instruments/agreements	Agreements and MOUs on the Conservation of Albatrosses and Petrels, African-Eurasian Migratory Waterbirds, Raptors, Siberian Cranes, and other species
	http://www.cms.int/en/cms-instruments/mou	
Council of Europe (CoE)	www.conventions.coe.int	CoE: Treaty Office Conventions
	http://conventions.coe.int/Treaty/en/Treaties/Html/104.htm	Convention on the Conservation of European Wildlife and Natural Habitats (CETS No.: 104)
European Union (EU)	http://eur-lex.europa.eu/homepage.html	EUR-lex. EU legislation database
	http://ec.europa.eu/environment/cites/legislation_en.htm	EU CITES legislation information and links
	http://ec.europa.eu/environment/cites/pdf/2007_referenceguide2_en.pdf	EC/TRAFFIC (2013). *Reference Guide to the European Union Wildlife Trade Regulations*. European Commission and TRAFFIC, Brussels, Belgium
	http://ec.europa.eu/environment/cites/pdf/trade_regulations/short_ref_guide.pdf	*Wildlife Trade Regulations in the European Union*. Short guide
	http://ec.europa.eu/environment/cites/pdf/national_legislation.	EU Member States national legislation.
	http://eur-lex.europa.eu/legal-content/EN/TXT/PDF/?uri=CELEX:32006R0865&from=EN	EU: COMM. RE.G. (EC) No 865/2006 Article 18. Biological samples
	http://ec.europa.eu/environment/nature/legislation/birdsdirective/index_en.htm	Birds Directive information
	http://ec.europa.eu/environment/nature/legislation/habitatsdirective/index_en.htm	Habitats Directive information
GOV.UK	www.gov.uk	Information portal for all UK government services
UK legislation	http://www.legislation.gov.uk/	UK legislation database
International Air Transport Association (IATA)	www.iata.org	IATA (annual) *Live Animals Regulations*. International Air Transport Association, Montreal and Geneva
	http://www.iata.org/publications/Pages/live-animals.aspx	
International Union for Conservation of Nature (IUCN)	www.iucn.org	IUCN/SSC (2013). *Guidelines for Reintroductions and Other Conservation Translocations*. Version 1.0. Gland, Switzerland: IUCN Species Survival Commission
	https://portals.iucn.org/library/efiles/documents/2013-009.pdf	
UK wild bird legislation	www.rspb.org.uk/Images/WBATL_tcm9-132998.pdf	RSPB (2010) *Wild Birds and the Law, England and Wales*. Royal Society for the Protection of Birds, Sandy, Bedfordshire (downloadable booklet)
	www.rspb.org.uk/Images/WbatlScotland_tcm9-202599.pdf	RSPB (2008) *Wild Birds and the Law Scotland. Royal Society for the Protection of Birds*, Sandy, Bedfordshire (downloadable booklet)
Raptor rehabilitation	http://www.raptorrescue.org.uk/rehabilitation/	Raptor Rescue (2010) *The Raptor Rescue Rehabilitation Handbook and Code of Practice*
Veterinary forensics		Cooper ME (2007) Importance and application of animal law. In: *Introduction to Veterinary and Comparative Forensic Medicine*. Cooper JE and Cooper ME (2007). Blackwell Publishing, Oxford, UK.
		Cooper ME (2013) Legislation. In: Cooper JE and Cooper ME. *Wildlife Forensic Investigation. Principles and Practice*. CRC Press, Taylor and Francis, Boca Raton, FL, USA.

Continued

Table of Websites and Literature—cont'd

Topic	Website	Information/Comment
US field research	http://www.nwhc.usgs.gov/publications/field_manual/ http://www.nwhc.usgs.gov/publications/field_manual/field_manual_of_wildlife_diseases.pdf http://www.nwhc.usgs.gov/publications/field_manual/chapter_6.pdf	USGS-National Wildlife Health Centre (1999). Friend M and Franson JC (eds.). *Field Manual of Wildlife Diseases: General Field Procedures and Diseases of Birds* *Guidelines for Proper Care and Use of Wildlife in Field Research* For a summary of US, UK, and Canadian laws relating to raptors, see: Millsap BA, Cooper ME, Holroyd G (2007) Legal considerations. In: *Raptor Research and Management Techniques*. Bird DM and Bildstein KL (eds.). Hancock House Publishers Ltd., Surrey, Canada and Blaine, USA.
United States Fish and Wildlife Service (USFWS)	http://www.fws.gov/le/laws-regulations.html http://www.ncwildlife.org/portals/0/License/Documents/Falconry_Federal_Regulations.pdf http://www.fws.gov/le/businesses.html	USFWS manages and enforces the US federal wildlife legislation and falconry is regulated by federal legislation, the Code of Federal Regulations Title 50 § 21.29, but this responsibility is delegated to States that have legislation of the same standard. Its Office of law Enforcement deals with CITES matters, including permits
Canada	http://laws.justice.gc.ca/eng/ http://www.ec.gc.ca/default.asp?lang=En&n=E826924C-1	Canada's Justice Laws Website for federal law. See also individual Province law Websites Environment Canada Wildlife Legislation
EU Member States	https://e-justice.europa.eu/content_member_state_law-6-en.do	EU portal to Member States' national legislation databases
Australia	http://www.comlaw.gov.au/Home	Australian Commonwealth laws. See also individual State law websites
New Zealand	http://www.legislation.govt.nz/	New Zealand legislation website
Kenya	http://www.kenyalaw.org:8181/exist/kenyalex/index.xql	Laws of Kenya

APPENDIX 5

Organizations and Electronic Resources Relating to Avian Medicine

F. Joshua Dein, VMD, MS

The original edition of this Appendix was conceived and produced at a time when Web resources and tools were much less advanced than they are today. As a result, many of the references listed in the previous editions are no longer available, have been merged with other sites, or their Web locations (URLs) have changed. Electronic reference works and devices to access such resources have also proliferated. Given the development of this evolving and dynamic information exchange context, it seems clear that a static printed list of resources by URL is no longer appropriate for the digitally connected avian practitioner. Therefore, a more flexible and easily updated collection has been created using Zotero, a free and open-source reference management tool. It can be found at https://www.zotero.org/groups/samour3appendix/items.

There are also Zotero desktop and portable versions, as well as mobile apps. Because the Zotero Group is free and open for all to read and comment, it is hoped that readers of this book will also become members of the Group, contribute their favorite sites, and add entries as new resources become available.

The following considerations apply to the initial collection:

- It cannot be a complete list of all Web sites related to birds and avian medicine, but is a starting point to be expanded and refined through community involvement as described previously.
- It contains primarily English language sites. Sites in other languages are welcome.
- A priority has been given to sites that contain lists of links to other sites within specific subject areas.
- The categories are general. Sites may cover more than one category.
- Although efforts have been made to use a broad and global context, sites included may reflect the North American and personal experience bias of the author, so contributions from other perspectives will be of great value.

Below are the abstracted examples of the eight categories and 100 sites included in the initial collection, selected to demonstrate the range of content. When a creator is not listed, it should be assumed to be the same as the title of the site.

Web Site	Creator
Aviculture	
Aviculture Europe	
International Aviculture Organizations	Birdmag.com
Model Aviculture Program, Inc.	
Pet Bird Clubs & Associations	PetStation
The Society for Conservation in Aviculture	
Conservation, Distribution, and Identification	
Avibase	Denis Lepage
Biological Resources—Non-waterfowl Birds	USGS Northern Prairie Wildlife Research Center
BIRDNET	Ornithological Council
International Crane Foundation	
Videos, Photos, and Sound Recordings of all the Birds of the World	Internet Bird Collection
Backyard Poultry	
Backyard Poultry Information Centre	Backyard Poultry
Poultry Organizations List	PoultryHelp.com
Small and Backyard Flocks	eXtension
The Poultry Site	
Urban Chickens	
Museums and Zoos	
Avian Scientific Advisory Group	
Bird type specimens online in 3D	Zoological Museum Amsterdam
Electronic inventory of European bird collections	Natural History Museum, London
ORNIS	
Zoo Associations	Zoos Worldwide
Pet Birds	
BirdChannel	I-5 Publishing
Hotspot for Birds	Advin Systems
Pet Parrots & Exotic Bird Associations & Bird Clubs	Birds n Ways
The Gabriel Foundation	
The Pet Bird Page (Parrots)	Grant Yoshimori
Reference Books and Resources	
Atlas of Avian Diseases	Cornell University
Avian Medicine: Principles and Applications	Branson Ritchie, et al.

Continued

Web Site	Creator
Backyard Poultry Medicine and Surgery	Cheryl Greenacre and Teresa Morishita
Birds—Educational Modules	Montana Science Partnership
Field Manual of Wildlife Diseases	Milton Friend, et. al.
The Grey Parrot Anatomy Project	Avian Studios
Veterinary	
Abu Dhabi Falcon Hospital	
American Association of Wildlife Veterinarians	
Association of Avian Veterinarians	
Avian Specialty	European College of Zoological Medicine
World Poultry Veterinary Association	
Wild Birds	
African Bird Club	
American Birding Association	
Birding Organizations	Wildbirds
Birdingonthe.Net	jsiler
BirdLife International	

APPENDIX 6

Pharmaceutical Products Commonly Used in Avian Medicine

Thomas A. Bailey, Merle M. Apo

Antibiotics

Generic and Trade Names	Formulation	Route of Administration	Dosage	Activity	Comment
Ampicillin trihydrate (Omnipen, Wyeth-Ayerst; Polycillin Apothecon)	250-mg and 500-mg capsules 125 mg/5 mL and 250 mg/5 mL oral suspension	PO	11-15 mg q8h—ratites 25-100 mg/kg q12-24h—pigeons 100-200 mg/kg q6-8h—psittacines 170 mg/L drinking water—game birds 1000-2000 mg/L drinking water—canaries/aviary use 2000-3000 mg/kg soft food—canaries/aviary use 1000 mg/L drinking water—Galliformes flock	Broad-spectrum, poor GI absorption and Gram activity of common bacterial isolates of birds; may be effective when organism is sensitive	
	125-, 250-, 500-mg, and 1-, 2-, and 10-g phials	IM	15 mg/kg q12h—raptors 100 mg/kg q4h—psittacines and most species 155 mg/kg q12-24h—pigeons 100 mg/kg q12h—cranes 4-7 mg/kg q8h—ratites (except emus) 15-20 mg/kg q12h—emus		Amoxicillin favored over ampicillin for IM use in pigeons Also SC Also SC
Apramycin i.e., Apralan (Elanco)	Soluble powder 1-g and 50-g packs	PO	25-50 g/100 L	Primarily of use against *Salmonella* spp	Licensed for poultry
Azithromycin Zithromax (Pfizer)	Powder for reconstitution 100 mg/5 mL and 200 mg/5 mL oral suspension	PO	45 mg/kg q24h—most species 10-20 mg/kg q48h for five treatments—blue/yellow macaws non-intracellular infections 40 mg/kg q24h for 30 treatments—blue/yellow macaws intracellular infections	Recommended for intracellular infections (e.g., *Toxoplasma* spp, *Plasmodium* spp, *Chlamydia psittaci*, and *Cryptosporidium* spp)	

Continued

Antibiotics—cont'd

Generic and Trade Names	Formulation	Route of Administration	Dosage	Activity	Comment
Azithromycin Zithromax (Pfizer)	100 mg/5 mL, 200 mg/5 mL oral suspension	PO	43-45 mg/kg q24hr		Use with ethambutol and rifabutin
Bactrim (Roche), Septra (Burroughs Welcome)	Trimethoprim and sulfamethoxazole	PO	25 mg/kg q24h—toucans, mynahs (*Coccidia*) 320-525 mg/L drinking water—poultry (*Coccidia*)		
Cefazolin Ancef (SmithKline Beecham) A first-generation cephalosporin	500-mg, 1-g, and 10-g phials	IM	25-75 mg/kg q8-1 h—most species 50-100 mg/kg q12h—raptors		Administration PO is also an acceptable means of treating raptors
Cefadroxil Cefa-Tabs (Fort Dodge) A first-generation cephalosporin	50-, 100-, 200-, and 500-mg and 1-g tablets 50 mg/mL or 100 mg/mL oral suspension	PO	20 mg/kg q12h—ratites 100 mg/kg q12h for 7 d—most species, including pigeons		
Cephalothin Kefzol (Lily) A first-generation cephalosporin	1-g phials	IM	100 mg/kg q6-8h—most species, emus 30-40 mg/kg q6h—ratites 100 mg/kg q 2-3h—quail, ducks 100 mg/kg q 2-6 hr—passerines		
Cephradine Cephradine (Biocraft) A first-generation cephalosporin	125 mg/5 mL oral suspension 250- and 500-mg capsules	PO	35-50 mg/kg q4-6h—most species 100 mg/kg q4-6h—pigeons, emus, cranes		
Cefoxitin Mefoxin (Merck) A second-generation cephalosporin	1-g phial	IM	50-100 mg/kg q6-12h—most species	Broad-spectrum activity against Gram-negative and Gram-positive organisms	
Ceftazidime Ceptaz, Fortaz (GlaxoSmithKline) A third-generation cephalosporin	Powder for reconstitution 1- and 2-g phials	IM	50-100mg/kg q4-8h—most species		Penetrates CSF and CNS
Ceftiofur Naxcel (Pharmacia) A third-generation cephalosporin	Powder for reconstitution 1- and 4-g vials	IM	50-100 mg/kg q4-8 h—most species 10 mg/kg q8-12h—orange-winged Amazon parrots 10 mg/kg q4h—cockatiels	Broad-spectrum activity against Gram-negative and Gram-positive organisms	

Antibiotics—cont'd

Generic and Trade Names	Formulation	Route of Administration	Dosage	Activity	Comment
Clindamycin Dalacin (Upjohn)	150 mg/mL, 2- and 4-mL injectable	IM	100 mg/kg q12h for 7 d—psittacines		
Cosumix Plus (Ciba), many other preparations	Sulphachloropyridazine 100 g and trimethoprim 20 g	PO	2.4 mg/kg for addition to drinking water or food	Reported to be effective against *Mycoplasma gallisepticum* and *Escherichia coli*	
	Oral powder		400 mg/kg feed—geese		
Ciproxin (the primary metabolite of Enrofloxacin; Bayer)	500-mg tablet (human preparations only)	PO	380 mg/L drinking water 10-20 mg/kg q12h—most species 50 mg/kg q12h—raptors 3-6 mg/kg q12h—ratites 250 mg/L drinking water × 5-10 d—pigeons		Slightly better activity against Gram-negative organisms; has a bitter taste. A 50-mg/mL suspension can be compounded by crushing a 500-mg tablet and mixing with sterile water or flavored compounding solution.
Ciprofloxacin Cipro (Bayer)	250-, 500-, and 750-mg tablets	PO	80 mg/kg q24h 15 mg/kg q12h		As combination therapy for mycobacterium.
Clarithromycin Biaxin (Abbott)	250- and 500-mg tablets, 125 mg/5 mL 250 mg/5 mL granules for reconstitution	PO	55-85 mg/kg q24h		
Cycloserine Seromycin (Lilly)	250-mg capsules	PO	5 mg/kg q12h		
Devoprim (Mycopharm) Duphatrim (Solvay-Duphar)	Sulphadiazine 100 mg, trimethoprim 20 mg per tablet	PO	½ tablet per pigeon q12h		
Di-Trim (Fort Dodge)	Sulphadiazine 50 mg and trimethoprim 10 mg per dose of 1.1 mL	PO	0.55 mL dose per pigeon q12h		Raptors with *Sarcocystis* spp infection treat for at least 6 wk.
	Suspension		30 mg/kg q8-12h—most species 60 mg/kg q12h for 3-d treat, 2-d off medication, 3-d treat—raptors, waterfowl (*Coccidia*)		

Continued

Antibiotics—cont'd

Generic and Trade Names	Formulation	Route of Administration	Dosage	Activity	Comment
Doxycycline Ronaxan (Rhône-Mérieux)	20- and 100-mg tablets	PO	40-50 mg/kg, i.e., 0.16 tablet per Amazon parrot and cockatoos 25-30 mg/kg, i.e., 0.1 tablet per African grey parrot		Is much less effected by cations than other tetracyclines so that absorption from the GI tract is almost 100% and, in contrast to the water-soluble tetracyclines, only 30% is excreted via the kidneys. If regurgitation occurs, reduce the dose by 25%. 70% of doxycycline is excreted in the bile and some undergo enterohepatic recirculation, which helps maintain plasma levels. The tablets are really too large except for the larger birds.
Duphatrim (Solvay-Duphar)	Sulphaquinoxaline 500 mg/g and trimethoprim 165 mg/g, 500-g pack Oral granules for addition to drinking water or food	PO	30 mg/kg for broiler chickens and turkeys more than 21 d of age		
Engemycin (Mycofarm), Oxytetrin (Mallinckrodt Veterinary)	As the hydrochloride for injection 5%, i.e., 50 mg/mL, 50-mL, and 100-mL phials	IM	58 mg/kg q24h for birds weighing more than 400 g; 100 mg for birds weighing less than 400 g		Do not be tempted to use the higher concentrations available for large farm animals (e.g., 100 mg/mL instead of 50 mg/mL) in an attempt to reduce the bulk of the injection as this only results in tissue necrosis.
Isoniazid Niazid (Duramed)	300-mg tablets 50 mg/5 mL elixir 25 mg/mL injection	PO IM	30 mg/kg q24h		Human anti-tuberculosis drug May be hepatotoxic and may cause GI disturbance.
Kanamycin Kantrim (Fort Dodge)	50 mg/mL injectable solution	IM PO	10-20 mg/kg q12h—most species 13-65 mg/L drinking water for 3-5 d made fresh daily—most species		Primary use in enteric infections.

Antibiotics—cont'd

Generic and Trade Names	Formulation	Route of Administration	Dosage	Activity	Comment
Norfloxacin Noroxin (Merck), Veriflox 20% oral solution (Lavet, Ltd.)	400-mg tablets	PO	10 mg/kg q24h—chickens, geese 10 mg/kg q6-8h—turkeys 3-5 mg/kg q12h—ratites 175 mg/L drinking water × 5 d—chickens		
Oleandomycin Amimycin, Matromycin, Romicil (Pfizer)		PO IM	50 mg/kg q24h—passerines 25 mg/kg q24h—passerines		
Piperacillin sodium/ tazobactam sodium Zosyn (Lederle)	2.25, 3.375, 4.5 g phials	IM	100 mg/kg q12h—most species	Tazobactam sodium is an antibiotic potentiator to increase the drug's effectiveness against *Staphylococcus* spp and *Streptococcus* spp	For severe or polymicrobic bacterial infections, preoperative orthopedic or coelomic surgery
Rifabutin Mycobutin (Pharmacia)	150-mg capsules	PO	15-45 mg/kg q24h		
Sarafloxacin SaraFlox (Abbott)	50 mg/mL aqueous formulation	PO	10 mg/kg q8h—chickens 20-40 mg/L drinking water × 5 d—chickens (colibacillosis) 30-50 mg/L drinking water × 5 d—turkeys (colibacillosis)		
Streptomycin Spectam (AgriLabs)	100 mg/mL injectable	IM	30 mg/kg q12h		See toxicity of the aminoglycosides (Appendices Table 1.4)
Ticarcillin Ticar (Link)	1- and 5-g phials of powder for reconstitution	IM	75-100 mg//kg q4-6h—Amazon parrots (for blue-fronted Amazon parrots give q2-4h) 200 mg/kg q6-12h—most species	More active against *Proteus* spp than carbenicillin	Ticarcillin is broken down by β-lactamase produced by some strains of *Pseudomonas*, it has therefore been combined with clavulanic acid (see below)
Ticarcillin and clavulanic acid, i.e., Timentin (SmithKline Beecham)	Powder for reconstitution Ticarcillin 1.5 g, clavulanic acid 100 mg, 1.6-g phials	IM IV	100-200 mg/kg q12h—most species	Effective against many Gram-negative organisms, particularly against resistant strains of *Pseudomonas*	

Continued

Antibiotics—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Amikacin sulfate	Amiglyde 50 mg/mL, 240 mg/mL, (Aveco); Amikin (Bristol Labs)	Most species	IV, IM	10-15 mg/kg bid	Indicated against acute infections. Active *in vitro* Gram-negative bacteria including *Pseudomonas* spp., *E. coli*, *Proteus* spp., *Klebsiella* spp., and *Enterobacter* spp. and Gram-positive bacteria including *Staphylococcus* spp. and *Streptococcus* spp. The risk of nephrotoxicity increased with dehydrated patients. Synergistic effect when used with third-generation penicillins and cephalosporins. Risk factors predisposing to aminoglycoside nephrotoxicosis: prior renal insufficiency, advanced age, increased dose or frequency, hepatic disease, hypovolemia, dehydration, metabolic acidosis, exposure to other nephrotoxins, severe sepsis, and endotoxemia
Amoxicillin trihydrate	Amoxinsol 150 Injection (Univet)	Pigeons	IM or SC	150 mg/kg daily for 5 d; every other day if using long-acting preparation	Broad-spectrum antibiotic active against a wide range of Gram-positive and Gram-negative microorganisms. However, many bacterial organisms affecting birds are resistant to amoxicillin, and antibiotic sensitivity tests should be conducted before treatment is embarked on. Injections of large volumes should be given SC in valuable athletic birds such as falcons to avoid any possible complications associated with muscle necrosis
	Amoxinsol LA (Univet) Amoxinsol 50 soluble powder (Univet)	Waterfowl	PO	1 g per 3 L of drinking water. Provide on alternate days for 3 d	
	Use injection (SmithKline Beecham) 150 mg/mL	Bustards	IM or SC	100 mg/kg bid	Pharmacologic studies on houbara bustards indicate that therapeutic levels may be maintained for 5-7 d after a dose of 250 mg/kg IM. If doses of 100 mg/kg IM are used, therapeutic levels greater than 2 μg/mL are maintained for 72 h.
	Clamoxyl LA (SmithKline Beecham) 150 mg/mL	Bustards	IM or SC	100-250 mg/kg every 3 to 5 days	
	Betamox 40-mg tablets (Norbrook) Amoxypen 40-mg tablets (Mycofarm)	Pigeons	PO	40 mg/kg bid for 5 d	
	Vetremox powder for poultry/pigeons (Vetrepharm)	Pigeons	In drinking water	1-1.5 g/L of drinking water for 5-7 d	
Amoxicillin/ clavulanic acid	Synulox Ready to Use Injection (Pfizer); Synulox Palatable Drops (Pfizer); Clavamox (SmithKline Beecham)	Psittacines	IM or PO	7 mg amoxicillin/ 1.75 clavulanic acid per kilogram bid	Broad-spectrum antibiotic active against a wide range of Gram-positive and negative organisms. Effective against β-lactamase-producing bacteria, among them *Staphylococcus aureus*, *E. coli*, and *Proteus* spp. Fewer cases of resistance than amoxicillin alone. Injection can cause renal failure in dehydrated birds
	Synulox (Pfizer); Clavamox (SmithKline Beecham)	Raptors		150 mg/kg bid for 5-7 d	

Antibiotics—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Ampicillin	Polyflex 100 mg/mL (Fort Dodge)	Psittacines Galliformes	PO IM PO	100-200 mg/kg qid 100 mg/kg q4h 250 mg/8 oz of drinking water	Broad-spectrum antibiotic active against a wide range of Gram-positive and Gram-negative organisms. However, minimum activity for the common Gram-negative infections of birds. Poor GI absorption. May be useful for treating sensitive pathogens restricted to GI tract.
Carbenicillin	Pyopen (Link); Geopen (Roerig) Pyopen (SmithKline Beecham)	Raptors Psittacines	IM IM or IV	100-200 mg/kg tid for 3-5 d 200 mg/kg bid	Effective against Gram-negative organisms, especially *Pseudomonas* spp. and *Proteus* spp. resistant to other antibiotics. Synergistic with aminoglycosides. May also be given as IT injection 100 mg/kg sid
Cefotaxime	Claforan Injectable Solution, variable concentrations (Hoechst-Roussel)	Most species	IM, IV	75-100 mg/kg q6h	Broad-spectrum activity for many avian Gram-positive and Gram-negative pathogens. Penetrates blood-brain barrier. For best result qid therapy is recommended. Reconstituted vial stable for 13 wk if frozen
Ceftiofur	Naxcel Injectable Solution, variable concentrations (Upjohn)	Most species	IM	50-100 mg/kg qid	Similar treatment regimen to other third-generation cephalosporins
Ceftriaxone	Rocephin Injectable Solution, variable concentration (Roche)	Most species	IM, IV	75-100 mg/kg qid or every 4 hr	Can be reconstituted into 10-250 mg/mL concentrations. When reconstituted into lower concentrations, the Rocephin should be administered IV. Reconstituted solution is stable for 10 d when refrigerated. Useful for Gram-positive and Gram-negative bacteria, including some activity against *Pseudomonas* spp.
Cefuroxime	Zinacef 250-mg vials (Glaxo)	Most species	IM, IV	100 mg/kg tid	Bactericidal cephalosporin antibiotic resistant to β-lactamases active against Gram-positive and Gram-negative organisms. Highly effective against *S. aureus*. 100 mg/kg tid is the dose rate of other cephalosporins
Cephalexin	Ceporex (Mallinckrodt) Keflex (Dista)	Raptors, bustards, and other species	IM or PO	40-100 mg/kg for 3-5 d tid or qid	Active against many Gram-positive and Gram-negative bacteria. Active against *E. coli* and *Proteus* spp., but not *Pseudomonas* spp. Useful for *Staphylococcus* spp. dermatitis. Reconstituted suspension stable for 14 d if refrigerated.
Chloramphenicol	Chloramphenicol Injection (Willows Francis, Fort Dodge, Parke-Davis) Chloramphenicol Injection (Willows Francis, Fort Dodge, Parke-Davis); Intramycetin (Upjohn)	Raptors Budgerigar	IM IM PO	50 mg/kg tid For 3-5 d 200 mg/kg, bid for 5 d 50 mg/kg tid or qid	Effective in flock treatment for *Salmonella* spp. infections. May be useful for cases of enteritis in young birds. Use with caution on patients with renal or kidney disease. Causes bone marrow suppression in humans. Associated with temporary infertility in male pigeons

Continued

Antibiotics—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Chlortetracycline	Aureomycin Soluble Powder (Cyanamid)	Pigeons	PO	130 mg activity/L drinking water for 5-8 d (2.4 g powder/L)	Broad-spectrum antibiotic active against a wide range of Gram-positive and Gram-negative bacteria
			PO	400 mg activity/L drinking water for 5-8 d (7.25 g powder/L)	
			PO	400 mg activity/L drinking water for 21 d (7.25 g powder/L)	
		Waterfowl	PO	1000 ppm (18.2 g/kg) in feed for 45 d	
Clindamycin	Aureomycin Ophthalmic Ointment (Cyanamid)	Pigeons	Topical	Apply to affected eye bid for 7 d	
	Antirobe Capsules (Upjohn)	Raptors	PO	50 mg/kg bid for 7-10 d	Indicated for osteomyelitis and tendon sheath infections. Has been used for up to 12 wk without deleterious effects
		Psittacines	PO	100 mg/kg sid	
		Pigeons	PO	100 mg/kg sid	Recommended for osteomyelitis. Monitor renal and hepatic function during long-term use for secondary yeast infections (has been noted in mammals).
					Recommended for bone and joint infection
Clofazimine	Lamprene (Ciba)	Psittacines	PO	1.5 mg/kg sid	Recommended for the treatment of mycobacteriosis
Cloxacillin	Ampicox Syrup/ Capsules (SmithKline Beecham); Cloxapen (SmithKline Beecham); Tegopen (Bristol)	Raptors	PO	250 mg/kg bid for 7-10 d	Recommended in the treatment of infected bumblefoot
Cycloserine	Seromycin Pulvules (Lilly)	Raptors	PO	5 mg/kg bid for 3 months to 1 year	Tuberculosis in combination with enrofloxacin, cyclofazamine and ethambutol. Beware zoonotic risk of *M. avium* infection
Doxycycline	Ronaxan tablets (Rhône Mérieux, Henry Schein, Roerig)	Raptors	PO	50 mg/kg bid for 3-5 d (45 d for chlamydiosis)	Drug of choice for the treatment of chlamydiosis. The agent has greater activity, less immunosuppression, and fewer side effects including fungal overgrowth and disturbance to the normal bacteria of the GI tract than other tetracycline preparations. Monitor feces for *Candida* spp. infections
	Vibravenos (Pfizer); β-Ronaxan tablets (Rhône Mérieux, Henry Schein, Roerig)	Waterfowl	PO	50 mg/kg bid for 3-5 d (45 d for chlamydiosis)	
				240 ppm in feed for 45 d	
		Pigeons	PO	1 tablet daily for 5-7 d (40 mg/kg)	Respiratory infections
		Psittacines	IM	10 mg/kg sid	Treat for 45 d for chlamydiosis. Some injectable doxycycline preparations can cause myositis. Vomiting has been reported in macaws
			IM	75-100 mg/kg every 5-7 d	
			PO	25-50 mg/kg bid	
		Bustards	IM	100 mg/kg once every week	Pharmacokinetic studies in houbara bustards have shown that therapeutic levels are maintained in birds given a dose of 100 mg/kg IM every 7 d. Doxycycline hydrate may be given IV once at a dose of 22-44 mg/kg for initial severe cases of chlamydiosis followed by PO or IM treatment

Antibiotics—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Enrofloxacin	Baytril 2.5% or 5% Injection, Baytril 2.5% Oral Suspension, Baytril 10% Solution (Bayer)	Raptors Waterfowl	IM or PO Baytril 10% Solution can be used either in water or PO undiluted in food	10-15 mg/kg bid for 5-7 d	Broad-spectrum antimicrobial, bactericidal in action and effective against a wide range of Gram-positive and Gram-negative bacteria including *Pseudomonas* spp. and *Klebsiella* spp. as well as *Mycoplasma* spp. Higher doses (15 mg/kg bid) are more effective for less susceptible organisms such as *Pseudomonas* spp. IM or PO route can cause emesis in some species (e.g., falcons) if the bird has eaten in previous 6 hr. The injectable solution may be administered orally.
	Baytril tablets (Bayer) Baytril 2.7% (Haver/ Diamond)		Nasal flushing	4 mg in 20 mL saline for a 1-kg bird daily for 10 d 500 ppm in feed for 45 d (for chlamydiosis)	Useful for bacterial hepatitis or septicemia in neonates. Used widely in growing chickens and poultry of all ages without incidence of articular cartilage problems: at normal therapeutic levels (10-15 mg/kg bid), it is unlikely to produce joint deformity in neonatal birds (or in pigeons or waterfowl). Baytril 10% Solution can be used either in water or PO undiluted in food; the water dose should be based on the water drinking habits of the species
	Baytril 10% Oral Solution (Bayer)	Pigeons	PO	150 mg/L drinking water for up to 10 d; may need 300 mg/L to prevent reappearance of infection	
	Baytril 2.7% (Haver/ Diamond)	Bustards	PO	10-15 mg/kg bid for 5-7 d	
	Baytril 10% Injection (Bayer)	Pigeons	IM or SC	20 mg/kg initially, followed by oral treatment	
	Baytril 2.5% or 5% Injection, Baytril 2.5% or 10% Oral Solution (Bayer)	Psittacines	IM	5-15 mg/kg bid	Treat for 21 d for chlamydiosis, use 15 mg/kg or 2 mL/L
			PO	1-2 mL 10% solution/L drinking water	
	Baytril 2.7% (Haver/ Diamond)	Bustards	IV or IM	15 mg/kg	
Erythromycin	Erythrocin Soluble (11.56 g erythromycin activity/70 g sachet) (Sanofi, Lextron)	Pigeons	PO	13-26 mg erythromycin activity per liter drinking water for 3 d (1.5 g powder/L)	Gram-negative bacteria affecting birds are resistant to this antibiotic. May be effective in sinusitis and air sacculitis caused by *Mycoplasma* spp.
		Psittacines	PO	10-20 mg/kg bid 500 mg/4.5 L drinking water	
Ethambutol	Myambutol tablets (Lederle)	Raptors	PO	20 mg/kg bid for 3 mo to 1 yr	Tuberculosis in combination with enrofloxacin, cyclofazamine, and cycloserine. Beware zoonotic risk of *M. avium* infection.

Continued

Antibiotics—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Ethambutol Gentamicin	Myambutol (Lederle) Gentacin (Nicholas, Butler, Schering-Plough)	Psittacines	PO	15-20 mg/kg bid	Treatment of mycobacteriosis Bactericidal antibiotic that is most effective against Gram-negative pathogens, especially *Pseudomonas* spp. Effective in respiratory tract. Inject between tracheal rings in the neck region. The drug is nephrotoxic and can cause a transient polyuria. It is better to use amikacin if available. May be given IV and nebulized for treatment of upper respiratory tract infections
		Psittacines	IM	5-10 mg/kg tid for 5 d	
Lincomycin	Lincocin Injection/ Tablets (Upjohn)	Raptors	IM or PO	50-75 mg/kg	This drug has a poor activity against most Gram-negative bacteria, but it has a good activity for many Gram-positive bacteria. Useful for bumblefoot, chronic dermatitis, and respiratory infections caused by mycoplasmas. Has been used in raptors for up to 12 wk without ill effect. Patients should be monitored for secondary yeast infections.
		Intra-articular injection		bid for 7-10 d 0.25-0.5 mL daily for 7-10 d in injectable form	
		Psittacines	IM	100 mg/kg bid	
			PO	75 mg/kg bid	
	Lincocin Soluble Powder (Upjohn)	Waterfowl	PO	10 g/5 L drinking water for 5-7 d	
Lincomycin/ spectinomycin Lincomycin HCl 33.3% spectinomycin 66.7%	Linco-Spectin 100 Soluble Powder (Upjohn)	Pigeons	PO	50 mg (16.7 mg lincomycin/33.3 mg spectinomycin)/kg q24h for 3-7 d, e.g., 1 g powder/L drinking water	Susceptible infections, mycoplasmal infections
		Waterfowl	PO	3 g/4 L drinking water for 3-7 d	Mycoplasmal tenosynovitis, sinusitis
	Linco-Spectin Soluble Powder (Upjohn)	Psittacines	PO	0.125-0.25 teaspoonfuls powder per 568 mL drinking water	Water-soluble treatment for enteritis and mycoplasmal sinusitis
Marbofloxacin	Marbocyl 2% and 10% Injection (Univet) Marbocyl 5-mg and 20-mg Tablets (Univet)	Raptors	IV, IM, PO	5-10 mg/kg bid for 5-7 d 15 mg/kg sid	As for enrofloxacin but less likely to cause emesis. Pharmacokinetic studies in buzzards have indicated that doses of 2 mg/kg IV bid can maintain therapeutic plasma levels
Metronidazole	Flagyl Tablets (Rhône Poulenc Rorer); Flagyl (Searle)	Raptors	PO	50 mg/kg sid 5 d	Anaerobic infections (and giardiasis and trichomoniasis)
Oxytetracycline	Various standard preparations and tablets	Raptors	IM or PO	25-50 mg/kg tid for 5-7 d	A broad-spectrum bacteriostatic antibiotic, although high prevalence of resistant bacteria
	Long-acting injection	Raptors	IM	50-200 mg/kg every 3-5 d	IM injection may cause significant muscle necrosis
	Terramycin Soluble Powder (Pfizer)	Waterfowl	PO	37 g/15 L drinking water 5-7 d	Pasteurellosis and other sensitive bacterial infections

Antibiotics—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Oxytetracycline dihydrate	Oxycare 50-mg tablets (Animalcare)	Waterfowl	PO	One tablet daily for 5-7 d	*Chlamydia psittaci* infections
	Terramycin (Pfizer)	Psittacines	IM	50 mg/kg sid	For birds weighing more than 700 g, use 20 mg/kg.
	Terramycin Injection (Pfizer)	Pigeons		For birds under 400 g use 80 mg/kg. 0.1-0.5 mL/kg on alternate days for 21 d in conjunction with oral tetracycline	Birds under treatment with long-acting oxytetracycline need to be monitored for secondary yeast infections
Piperacillin	Pipril (Lederle); Pipracil (Lederle)	Raptors	IV or IM	100 mg/kg bid for 5-7 d	Broad-spectrum bactericidal penicillin. Recommended for the treatment of systemic and local infections caused by susceptible Gram-negative and Gram-positive aerobic and anaerobic organisms, particularly *Pseudomonas* spp., *Proteus* spp., *Klebsiella* spp., *Enterobacter* spp., *Serratia* spp., *E. coli*, and *Haemophilus* spp.
		Psittacines	IV or IM	100-200 mg/kg bid or tid	
Rifampin (rifampicin)	Rimactane (Ciba); Rifadin (Marion Merrell Dow)	Psittacines	PO	15 mg/kg bid	Used for the treatment of avian tuberculosis. It has been associated with hepatitis, central nervous system signs, depression, and vomiting
Sodium amoxicillin	Amoxil (sodium amoxicillin) (SmithKline Beecham)	Bustards	SC/IM/IV	100 mg/kg	Pharmacokinetic studies in houbara bustards using 100 mg/kg have shown that administrations every 8 h (IV) or 4 h (IM) will maintain levels above 2 μg/mL
Spectinomycin	Spectam (Sanofi, Syntex)	Psittacines	IM	10-30 mg/kg bid, tid	Has been used to treat enteritis in Galliformes caused by Gram-negative bacteria. It can be also given into sinuses at 35 mg/kg: one third into each sinus and the rest IM
Sulfachlorpyridazine	Vetisulid Water-Soluble Powder (Solvay)	Most species	PO	0.25-tsp/gallon 5-10 d	Excellent flock treatment for *E. coli* infections. Check sensitivity
Tetracycline HCl 80%	Tetsol 800 (C-Vet, Generic)	Pigeons	PO	1 g/1.5 L drinking water for 5-7 d (60 mg/kg)	*Chlamydia psittaci* infections
Tiamulin	Tiamutin 12.5% Solution (Leo Laboratories)	Pigeons	PO	225 mg (2 mL)/L drinking water for 6 d	Mycoplasmal infections
Tobramycin	Nebcin Injection (Lilly)	Raptors	IM	5-10 mg/kg bid for 5-7 d	Least nephrotoxic of all current aminoglycosides; should be used only for severe infections caused by resistant *Pseudomonas* spp. Also useful to treat joint injections by direct irrigation of toe joints in raptors. Use with caution in neonatal and geriatric birds, birds with neuromuscular disorders, and preexisting renal disease
		Pheasants	Intra-articular flush	0.25-0.5 mL daily for 7-10 d	
		Cranes	IM	2.5-5 mg/kg bid	
	Nebcin (Lilly), Tobradex (Alcon)	Psittacines	IM or topical	2.5-10 mg/kg tid	Septic arthritis

Continued

Antibiotics—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Trimethoprim/ sulfonamide	Borgal (Hoechst); Duphatrim (Solvay Duphar); Cosumix Plus (Ciba); Bactrim (Roche)	Psittacines	IM	8 mg/kg bid	Good lipid solubility and well distributed in sulfonamide tissues. Bacteriocidal for Gram-positive and Gram-negative pathogens including *E. coli*, *Pasteurella* spp., *Proteus* spp., *Salmonella* spp., and *Listeria* spp.
			PO	20 mg/kg bid or tid Cosumix Plus should be given at a rate of 1 g/L drinking water daily for 5 d	Recommended for the treatment of nephritis, bacterial hepatitis, and septicemia in neonates. May also be effective for treating some forms of coccidiosis. Regurgitation is reported to occur in some species. Do not use in dehydrated birds or patients with liver disease or bone marrow suppression. Cosumix is a water-soluble powder and is useful for providing sulfachlorpyridazine in the drinking water.
	Cosumix Plus Soluble Powder (Ciba); Duphatrim Poultry Suspension (Solvay Animal Health); Tribrissen Piglet Suspension (Mallinckrodt); Bactrim (Roche)	Most species	PO	12-60 mg/kg bid (combined constituents for 5-7 d)	
	Cosumix Plus Soluble Powder (Ciba); Duphatrim	Waterfowl	PO	1 mL/5 L drinking water for 5-7 d	
	Poultry Suspension (Solvay Animal Health); Bactrim (Roche)	Raptors	SC	30 mg/kg bid for 5-7 d	
	Duphatrim 24% Injection (Solvay Duphar)	Bustards	IM	8-30 mg/kg bid for 5-7 d	
Tylosin	Tylan Injection (Elanco) (Butler)	Raptors	IM	30 mg/kg bid for 3 d	60 mg/kg tid for birds of 50-250 g 25 mg/kg tid for birds 250-1000 g
	Tylan (Elanco) (Butler)	Psittacines	IM	20-40 mg/kg tid	15 mg/kg tid for birds weighing more than 1000 g
			PO	2 teaspoonfuls/4.5 L drinking water	Well distributed in tissues. Upper respiratory tract infections. *Mycoplasma* spp., *Pasteurella* spp., and *Chlamydia psittaci*
	Tylan 50 or 200 Injection (Elanco, Butler)	Waterfowl	IM	20-30 mg/kg tid for 3-7 d; or 100 mg in 10-mL saline daily nasal flush for 10 d	Most Gram-negative organisms are resistant to this drug
	Tylan Soluble Powder (100-g tylosin activity per bottle) (Elanco, Butler)	Pigeons	PO	550-mg tylosin activity/L drinking water for 3 d (activity approx. 100%)	
		Waterfowl	PO	2.5 g/5 L drinking water for 3 d	

Antimycotic

Generic and Trade Names	Formulation	Route of Administration	Dosage	Activity	Comment
Clotrimazole Mycelex solution (Schering-Plough)		IT	2 mg/kg q24h × 5 d—psittacines (tracheal granulomas)	Antifungal agent with broad spectrum activity	May be nebulized or administered topically on lesions in air sac.
Fluconazole Diflucan (Roerig)	200-mg tablets	PO	2-5 mg/kg q24h × 7-10 d—most species (candidiasis) 5-15 mg/kg q12h × 14-60 d—most species (aspergillosis) 100 mg/kg soft food—Gouldian finches (candidiasis) 150 mg/L drinking water—Gouldian finches (candidiasis)	*In vitro* × 100 more potent than ketoconazole. Excellent against yeasts but variable activity against *Aspergillus* spp.	Very soluble, readily absorbed PO. Widely distributed in the body including CNS. Eliminated via the kidneys. Toxicity low with mild GI upset and CNS signs (but only reported in humans). Because liver enzymes are sometimes elevated, it would be wiser to monitor these during treatment. Take care if used with potentially nephrotoxic drugs.

Antimycotic Agents

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Amphotericin B	Fungizone (Squibb, Bristol-Meyers)	Raptors	IV	1.5 mg/kg tid for 7 d	Organisms with an MIC <1 m/mL are considered susceptible. There is good correlation between MIC values and clinical response. Treatment failure caused by fungal resistance is rare in humans, but *Aspergillus* spp. are the most frequently reported resistant fungi. Give by slow IV administration with 10-15 mL/kg fluids for 7 d. Absorption from the lungs following aerosol administration is poor and this route is used to treat pulmonary aspergillosis. Improved clinical efficacy when used in combination with flucytosine or azole antifungal agent. Poorly absorbed from the GI tract so must be given IV, IO, or IT. Most important clinical toxicosis is nephrotoxicity, which is dose related and seen clinically as increases in BUN and creatinine in mammals. Dosing every other day, electrolyte loading, and slow infusion of amphotericin B decrease severity and rate of development of renal toxicity. Other adverse effects include phlebitis, fever, nausea, vomiting, and hypokalemia with resulting cardiac arrhythmia. Measures to prevent vomiting include giving antiemetic drugs before infusion. In dogs, doses higher than 5 mg/kg resulted in death caused by cardiac abnormalities; doses of 2-5 mg/kg occasionally caused cardiac problems, but doses lower than 1 mg/kg were without effect on the heart. Treatment protocols should include pretreatment with sodium chloride and it should be infused at a slow rate. It can be mixed with 5% dextrose solution during infusion. Administration of amphotericin B in 0.45% saline with 0.5% dextrose SC in dogs/cats is a way of administering large quantities of amphotericin B without producing the marked azotemia associated with IV injection
		Psittacines	IT	1 mg/kg bid for 12 d, reduce to every other day for 5 wk	
			Nebulized	1 mg/mL saline solution bid × 20 min	
			IV	1.5 mg/kg tid together with 1 mg/kg, IT bid for 3-5 d	
	Fungilin Suspension (Squibb)	Psittacines	PO	1 mL/kg bid for 3-5 d	Candidiasis. Especially in young neonates as they are not absorbed from the alimentary tract.
Amphotericin B	Abelcet	Raptors	IV	1.5 mg/kg sid	New, less-toxic lipid-based formulation, has lipid complex been used in humans. Can be given at higher doses with less toxicity. In humans, daily dose is 3-5 mg/kg daily, whereas the dose of the conventional form is 0.5-1 mg/kg every 48 hr. Indicated for aspergillosis. The pharmacokinetics of Abelcet and conventional amphotericin B are different. In dogs, peak blood levels and kidney levels are lower after Abelcet administration. Appears well tolerated in raptors and can be given SC
	Amphotericin B Lipid Complex (Squibb)				

Antimycotic Agents—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Caprylic acid	Kaprycidin A Capsules 325 mg, (Ecological Formulas)	Most species	PO	¼ capsules/300 g	Adjunct treatment of antifungal therapy using imidazole
Chlorhexidine	Nolvasan Solution 20 mg/mL (Fort Dodge)	Most species	PO	10-30 mL/gallon drinking water 0.5% as a wound cleanser	Disinfectant. For flock or individual treatment of mild GI candidiasis. Toxic to finches. Use properly. This drug may also slow the spread of viral diseases. Give for 2-4 wk duration in drinking water. Not absorbed from the gut. May irritate eyes or mucous membranes
			Topical		
Enilconazole	Imaverol (Janssen)	Raptors	IT	Dilute 1:10, administer 0.5 mL/kg daily for 7-14 d	Excellent antifungal activity. Residual effect after topical application and used to treat topical dermatophyte infections. Indicated for dermatomycoses such as *Trichophyton* spp. and *Microsporum* spp. Treatment of choice for nasal aspergillosis in dogs and local infusion is used in the treatment of guttural pouch mycosis in the horse. Can be given topically or nebulized
	Clinafarm (Sterwin)		Topical	Dilute 1:10, apply topically bid for 3-4 weeks	
			Nebulized	1 mL in 9 mL saline solution	
		Psittacines	IT or topical	1:10-1:100 dilution	Clinafarm-EC formulation is used in the environmental decontamination of poultry facilities and equipment to prevent aspergillosis. Indicated for dermatomycoses such as *Trichophyton* spp. and *Microsporum* spp. Dilute solution in 50 parts of water and apply topically 3-4 μd. Also used intratracheally for aspergillosis treatment—dilute 1:20 and give 0.5-1.0 mL/kg IT q8h. Used as a nebulizing agent at 0.5 mL in 25 mL saline for systemic and topical fungal infections. Corrosive and may cause damage to the eyes
Flucytosine 5-Fluorocytosine	Alcobon (Roche), Ancobon (Roche)	Raptors	PO	20-30 mg qid for 20-90 d 40-50 mg/kg tid	Flucytosine has a narrow spectrum of activity and few *Aspergillus* sp. strains are susceptible. Strains with MIC <16 mg/mL are susceptible. Two-thirds of fungal isolates change from susceptible to resistant during treatment, so flucytosine should only be used in combination with other antifungal agents. Combination with amphotericin B is synergistic because amphotericin B increases fungal permeability to flucytosine. Total dose of 120 mg/kg/d divided into 18-30 mg/kg q6h. Used as a preventative agent for aspergillosis. May be indicated for the long-term treatment of aspergillosis infections or severe candidiasis infections that are resistant to nystatin. Toxic to the bone marrow.
		Psittacines	PO	20-75 mg/kg bid for 21 d	Generalized yeast or fungal infections
F10	F10 (Health & Hygiene)	Most species	Nebulized	1:250 dilution 1-3 × d	Complete-spectrum virucidal, bactericidal, fungicidal, and sporicidal but aldehyde-free compound of six synergistic active ingredients. Has been tested against every significant animal/human pathogen and has outperformed other disinfectants during efficacy testing, over a range of temperatures, in the presence of organic material, at low concentrations, short contact times, without any corrosive effects on infrastructure, metal nozzles, or any tissue irritation on workers and animals. Can be used to disinfect animal environments in their presence, lowering the environmental pathogen challenge significantly, with no side effects. Using either a nebulizer or a "smogger" unit, has been used to treat both individuals and groups of falcons with aspergillosis. Nebulized at a concentration of 1:250 and can also be added at the same concentration to drinking water, where it may limit the spread of bacterial or viral diseases
Gentian violet	Gentian Violet Powder or Solution 16 mg/mL	Psittacines	Topical	Apply affected area with cotton swabs	Topical application as drying agent. Excellent for crop candidiasis and skinfold candidiasis in hyacinth macaw chicks

Continued

Antimycotic Agents—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Griseofulvin	Grisovin 125-mg tablets (Mallinckrodt)	Pigeons	Crop tube	10 mg/kg q12h for 21 d	Dermatophytosis
	Grisol-V Powder (Univet)	Pigeon	PO	10 mg/kg q12h for 7 d	Dermatophytosis
Itraconazole	Sporanox capsules (Janssen)	Raptors	PO	10 mg/kg sid for 7-10 d Therapeutic dose: 10-15 mg/kg bid for 4-6 wk	Prophylactic dose: Drug of choice to treat aspergillosis infections. Organisms with MIC <0.12 m/mL are susceptible; those with MIC 0.25-0.5 m/mL are susceptible depending on dose and those with MIC >1 m/mL are considered resistant. Absorbtion is increased in an acidic environment and when taken with meals. Bioavailability increases from 40% after fasting to 99.8% when given with food. Extensively distributed throughout the body. Hepatometabolized and eliminated mainly in the bile. Therapeutically active concentrations maintained longer in tissues than in plasma. Used to treat superficial and systemic infections and to treat and prevent aspergillosis in birds. As oral absorbtion is pH dependent, dosage adjustments are necessary if gastric pH is increased. Oral capsules should be given with food but the oral suspension is better absorbed on an empty stomach. Higher tissue concentrations are reported when itraconazole is dissolved in acid and gavaged with orange juice in pigeons. Treatment of serious infections should be prolonged (>3 mo) and relapses occur. Better tolerated than ketoconazole. Fewer side effects are seen than with other antifungal agents. Beads dissolve best in 5% acetic acid (cola-based fizzy drinks or orange juice may also be used) left to stand overnight. Diarrhea and inappetence has been associated with the use of this drug in juvenile kori bustards. Dose of 10 mg/kg for 1 mo considered prophylactic. Sporanox liquid (10 mg/mL) is often more convenient.
		Waterfowl	PO	Prophylactic dose: 10 mg/kg sid for 7-10 d Therapeutic dose: 10 mg/kg bid for 4-6 wk	
		Psittacines	PO	5-10 mg/kg q24h for 3 weeks to 3 months	Adverse reaction observed in certain species, in particular African gray parrots
Ketoconazole	Nizoral (Janssen)	Raptors	IM	25 mg/kg bid for 7 d	Used for treatment of candidiasis when other therapies have been ineffective.
	Nizoral Suspension (Janssen)	Raptors	PO	60 mg/kg bid	Treatment for 14 d. Effective against *Candida* spp. *Mucor* spp. and *Penicillium* spp. Can also use ketoconazole tablets, dissolving a 200-mg tablet in 1.2 L of water. Reported to be nephro- and hepatotoxic after long-term application in mammals
		Pigeons	Crop tube	3 mg/kg daily for 7-21 d	
		Psittacines	PO	10 mg/kg bid	
Miconazole	Daktarin (Janssen-Cilag)	Raptors	Topical	bid for 3-5 d	Highly effective against candidiasis infections in the oropharynx and crop of falcons
		Psittacines			

Antimycotic Agents—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Nystatin	Nystatin Oral Suspension (Lagap)	Raptors	PO	300,000 U (3 mL/kg) bid for 7 d	Antifungal agent active against yeast infections localized to the alimentary tract, including *Candida albicans*. For oral and intestinal *Candida* spp. infections, give 1-3 × d for 7-14 d. Not absorbed after oral application, so very safe. Not effective against *Aspergillus* spp. May be used in conjunction with antibiotic therapy to prevent yeast overgrowth
		Waterfowl	PO	300,000 U (3 mL) bid for 7 d	
		Pigeons	Crop tube	20,000 U daily for 7 d	
		Psittacines	PO		
	Nystatin Feed Premix Myco 20 (Squibb)	Most species	With feed	300,000 U/kg bid for 10 d	
STA Solution	Salicyclic acid (3 g), tannic acid 3 g and ethyl alcohol to 100 mL	Most species	Topical	As needed	For fungal dermatitis
Terbinafine	Lamisil, Terbinafine 125-mg Tablet (Novartis)	Raptors	Nebulized	1 mg/mL solution q8-12h 10-15 mg/kg bid	Synthetic allylamine drug, highly fungicidal. Allylamines decrease the fungal synthesis of ergosterol and cause fungal death by disrupting the cell membrane. Active against *Aspergillus* spp. but to date clinical use has been limited in veterinary medicine, although preliminary nebulization results are promising. Oral and topical antifungal primarily used for dermatophytic infections. Use with caution if liver or kidney disease present
			PO		
Voriconazole	Vfend 50-mg tablets (Pfizer)	Raptors	Nebulized	10-15 mg/kg bid	New triazole with *in vitro* activity against a wide range of fungi and potential for use in the treatment of avian aspergillosis. Human studies have shown that the MIC of voriconazole at which 90% of *Aspergillus fumigatus* isolates is inhibited is lower (1 mg/L, range 0.25-2.0 mg/L) than those of amphotericin B and itraconazole. Highly efficacious experimentally in prevention and treatment of *Aspergillus* endocarditis in guinea pigs and superior to itraconazole. Appears to be well tolerated in falcons
			PO		

Antiprotozoal Agents

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Albendazole	Valbazen 113-116 mg/mL Suspension (SmithKline Beecham)	Ratites	PO	1 mL/50 lb bodyweight bid for 3 d. Repeat in 2 wks	For protozoal infections in ratites
Amprolium	Corid, Amprol plus (MSD, Ag Vet)	Raptors	PO	30 mg/kg daily for 5 d	Indicated for coccidiosis. Some coccidial strains of mynahs and toucans may be particularly resistant Ideal for flock treatment for 5-7 d
		Most species	PO	2-4 mL/gallon (5-100 mg/L) for 5-7 d	
Amprol	Coccoid 3.4% Solution (Harkers); Corid (MSD, Agvet)	Pigeons	PO	28 mL/4.5 L drinking water for 7 d, i.e., 25 mg/kg. Use half strength for extended regimen.	Coccidiosis
Aminothiazole	Tricoxine (Fabry)	Pigeons	PO	5 mL/L drinking water for 7 d	Susceptible trichomonads, avoid overdosing

Continued

Antiprotozoal Agents—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Carnidazole	Spartrix 10-mg tablets (Harkers, Wildlife Laboratories, Janssen)	Raptors	PO	20-25 mg/kg once	Treatment for trichomoniasis, hexamitiasis, and histomoniasis. Highly effective as a single dose but use lower dose for juvenile birds. Use together with dimetridazole in rest of the loft, susceptible trichomonads. Has been used in other species, although single doses have not always been effective in falcons and in bustards with advanced infections in the Middle East; use with caution
		Pigeons	PO	0.5-1 tablet once, e.g., 12.5-25 mg/kg	
		Psittacines	PO	20-30 mg/kg once	
Clazuril	Appertex (Harkers, Janssen)	Raptors	PO	5-10 mg/kg every 3rd day; give three times	Indicated for coccidiosis
		Waterfowl	PO		
		Pigeons	PO	1 tablet/bird single dose, e.g., 6.25 mg/kg	
		Psittacines	PO	7 mg/kg 3 d, 2 d off, 3 d on	
Co-trimazine (trimethoprim+sulfadiazine)	Cosumix Plus	Raptors	PO	60 mg/kg (combined constituents) bid 3 d on, 2 d off, 3 d on	Indicated for coccidiosis; do not use with dehydrated birds
	Soluble Powder (Ciba); Duphatrim Poultry Suspension (Solvay Animal Health); Bactrim (Roche)	Waterfowl	PO	60 mg/kg (combined constituents) bid 3 d on, 2 d off, 3 d on	
	Duphatrim 24% Injection (Solvay Duphar)	Raptors	SC	30 mg/kg bid 3 d on, 2 d off, 3 d on 30 mg/kg bid 3 d on, 2 d off, 3 d on	
		Waterfowl	SC		
Chloroquine	Resochin R tablet 500 mg (Bayer); Arlen (Winthrop)	Penguins	PO	10 mg/kg once then 5 mg/kg at 6, 18, and 24 hr	Primarily used to treat *Plasmodium* spp. usually in combination with primaquine. Overdose may result in death
Chloroquine phosphate Aralen (Sanofi)	68 mg/mL syrup 40 mg/mL injection, 5-mL ampule		PO	10 mg/kg once then 5 mg/kg at 6, 18, and 24 hr—penguins 25 mg/kg body weight, then 15 mg/kg at 12, 24, and 48 hr—most species, raptors, use in conjunction with 0.75-1.0 mg/kg primaquine at 0 hr	Active against *Plasmodium* spp Bitter taste, so crush tablet and dissolve in fruit juice or with honey. Low therapeutic index, *overdose fatal*. Rapidly absorbed from GI tract. May cause mucous membranes to turn yellow. Best used in combination with primaquine. Used in penguins, raptors, and psittacines.

Antiprotozoal Agents—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Dimetridazole	Harkanka 40% Powder (Harkers); Emtryl Prescription (40%) Soluble Powder (Rhône-Mérieux)	Pigeons	PO	666 mg powder/L drinking water for 7-12 d	Soluble powder indicated for water treatment against giardiasis, trichomoniasis, and hexamitiasis. Also used at a dose of 2.5 g/kg food in bustards. Drug of choice for flock treatment of trichomoniasis in bustards housed in naturalistic aviaries. Dimetridazole also has some activity against some anaerobic bacteria. Low therapeutic index. Toxic to Pekin robin and some other Passeriformes. Very toxic if overdosed in parrots. Acute hepatitis reported in fledging birds. Recommended not to be given in the breeding season. No problems associated with use of this drug in bustards.
		Bustards	PO	3 g/10 L water for 10 d; prevention 9 g/10 L for 5 d followed by 7 g/10 L for 10 d	
Metronidazole	Flagyl tablets (Rhône-Poulenc Rorer); Flagyl (Searle)	Raptors	PO	50 mg/kg daily for 3-5 d	Treatment of choice for trichomoniasis in raptors. In severe cases repeat after 7 d on completion of treatment.
	Metronidazole 200-mg tablets (Centaur); Flagyl (Searle)	Pigeons	PO	10 g powder/L drinking water for 5 d	Susceptible trichomonads; provides moderate control of hexamitiasis
	Metronidazole 25% Powder (Vetrepharm)	Pigeons	PO	100-150 mg in total divided over 5 d	
	Torgyl (Rhône Mérieux), Flagyl S Suspension (Rhône Poulenc Rorer); Flagyl (Searle)	Psittacines	PO	10-30 mg/kg bid for 10 d 10 mg/kg sid	Antiprotozoal
Primaquine Phosphate (Sanofi)	7.5-mg tablets		0.03 mg/kg q24h × 3 d—game birds, penguins 1.25 mg/kg q24h—penguin prophylaxis therapy (*Plasmodium* spp)	Active against *Plasmodium* spp	Used in penguins. Used in conjunction with chloroquine.
Pyrimethamine	Daraprim (Glaxo-Wellcome)	Psittacines	PO	0.5 mg/kg bid	For treating *Plasmodium* spp., *Sarcocystis* spp. and *Toxoplasma* spp.
		Raptors	PO	0.25-0.5 mg/kg bid for 30 d	Indicated for *Sarcocystis* spp., toxoplasmosis
		Waterfowl	PO	0.25-0.5 mg/kg bid for 30 d	

Continued

Antiprotozoal Agents—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Pyrimethamine/ sulfaquinoxaline	Microquinox (C-Vet Livestock Products)	Waterfowl	PO	60 mg/L drinking water, 3 d on, 2 d off, 3 d on	Indicated for coccidiosis
Quinacrine HCl Atabrine (Sanofi)	100-mg tablets	Pigeons	PO	7.5 mg/kg q24h × 7-10 d—most species (atoxoplasmosis) 5-10 mg/kg q24h × 7-10 d—most species 26-79 mg/L drinking water × 10-21 d	Hepatotoxicity a concern with overdosage Active against *Plasmodium* spp, *Atoxoplasma* spp
Ronidazole	Ronidazole 10% Powder (BP)	Pigeons	PO	1 g powder/L drinking water for 6 d, e.g., 12.5 mg/kg/d	Indicated in the treatment of trichomoniasis. Recommended flock treatment dose 60 g/100 L water for 5-7 d. Preventive dose 40 g/100 L water for 5-7 d
Sulfaquinoxaline/ pyrimethamine	Microquinox (Microbiologicals)	Psittacines	PO	15 mL/10 L drinking water, 3 d on, 2 d off, 3 d on, 2 d off	Coccidiostatic
Sulfadimidine sodium 33.3%	Vesadin (Rhône Mérieux); Intradine (Norbrook); Bimadine (Bimeda)	Pigeons	PO	10-20 mL/L drinking water for 5 d (or 3 d on, 2 d off, 3 d on, 2 d off, 3 d on)	Coccidiosis. May be effective against toxoplasmosis
Tetracycline plus furaltadone		Pigeons	PO	400 mg tetracycline + 400 mg furaltadone/L drinking water for 7 d	Avoid in adults feeding young less than 10 d of age; indicated for trichomoniasis and hexamitiasis
Toltrazuril	Baycox (Bayer, Bayvet)	Raptors	PO	10 mg/kg three times on alternate days or 15-25 mg/kg daily for 2 consecutive days	Treatment of choice for coccidiosis in falcons. Bitter taste. Mixing in equal parts (e.g., 1 mL to 1 L) with a soft drink (e.g., cola-based) prevents spitting of medication
	Baycox 2.5% Waterfowl Solution (Bayer, Baycox, Bayvet)		PO	1 mL of 2.5% solution/2 L drinking water for 48 hr	
		Pigeons	PO	5 mL/L drinking water for 5 d, e.g., 10 mg/kg	

Anthelmintics

Generic and Trade Names	Formulation	Route of Administration	Dosage	Activity	Comment
Fenbendazole Panacur (Hoechst)	2.5% (25 mg/mL) Oral suspension	PO	20-100 mg/kg once—most species 1.5-3.9 mg/kg q24h × 3 d—chickens 15-45 mg/kg—ostriches 5-15 mg/kg q24h × 5 d—waterfowl 10-12 mg/kg q24h × 3 d—pigeons 10-50 mg/kg repeat in 14 d—raptors 15 mg/kg q24h × 5 d—psittacines 30 mg/kg once—bustards 33 mg/kg q24h × 3 d—psittacines, passerines, and raptors (microfilaria and trematodes)	Broad spectrum Active against adult and larval nematodes Ovicidal; also active against tapeworms and some flukes	A tasteless and odorless drug. The suspension has been used for finches in the drinking water at a dose of 10 mg/L, although quite how it remains in suspension has not been stated. Even at this low dose, it can be toxic for finches. Also reported to be toxic in marabou storks and some species of vulture. Should not be used when birds are actively growing feathers. Shown to be teratogenic in sheep, otherwise when used at the recommended dose a relatively safe drug. Resistant strains of nematode can develop
Flubendazole Flutelmium 7.5% (Janssen—Cilag)	25 mg/g oral powder	PO for incorporation in food	30 mg/kg feed × 7 d—poultry 60 mg/kg feed × 7-14 d—partridges, pheasants		The use of this agent in species other than those for which it is licensed is not documented
Niclosamide Yomesan (Bayer)	Tablet containing 0.5 g of niclosamide	PO crop tube in feed	220 mg/kg repeat in 10-14 d—most species 50-100 mg/kg repeat in 10-14 d—ostriches 250 mg/kg q14d as needed—cranes 500 mg/kg q7d × 4 wk—finches	Active against tapeworms	Tablets are not soluble, must be suspended in water or mixed in mash. Death has occurred in pigeons and some Anseriformes at recommended doses
Thiabendazole Thibenzole (Merck)	176 mg/g oral suspension	PO	40-100 mg/kg q24h × 7 d—most species 100 mg/kg q once, repeat in 10-14 d—ratites 100-500 mg/kg once—most species 425 mg/kg feed × 14 d—pheasants, cranes	Roundworms, gapeworm, and some flukes	Not a very safe drug for birds, although it has been used in the past Toxic to ratites, ducks, and cranes
Wormex (Hoechst) (Mebendazole)	4% Oral powder (40 mg/mL) for incorporation in food		50 mg/kg q24h × 5 d—most species (*Capillaria*) 40 mg/kg of dry food used for grouse, formulated in grit-like pellets (trematodes)		
	Capsules containing 8 mg (licensed for pigeons)		One capsule per pigeon older than 2 mo of age		
	2% (20 mg/mL) Oral suspension for incorporation in feed 300 mL Pack licensed in UK for game birds		7-10mg/kg body weight q24h		

Continued

Anthelmintics—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Cambendazole	Ascapilla 30-mg capsules (Chevita); Equiben (MSD, Agvet)	Pigeons	PO	75 mg/kg on 2 consecutive days	Ascariasis and capillariasis
Carbaryl	Sevin 5% Powder, (Southern Agricultural Insecticides, Inc.)	Most species	Topical	Light dusting on feathers	Recommended for ectoparasite control when lightly dusted on to feathers. Add 1-2 teaspoons, depending on size of box, to nesting material to control insects
Clorsulon	Curatrem (MSD, Agvet)	Raptors	PO	20 mg/kg 3 times at 2-wk intervals	Control trematodes and cestodes
	Waterfowl		PO	20 mg/kg	Three times at 2-wk intervals
Crotamiton	Eurax Cream 10% and Lotion 10%, (Westwood Squibb)	Psittacines	Topical	Apply to affected area	Cnemidocoptic mite infestation areas
Cypermethrin	Dy-Sect (Deoson)	Raptors Psittacines	Spray	Dilute to 2% (avoid contact with bare skin)	Treatment of premises infested with *Dermanyssus* spp.
Cypermethrin 5% concentrated	Barricade (Lever)	Pigeons	Spray or dip	1:100 dilution	Lice, mites
Dichlorophen		Pigeons	PO	10 mg per pigeon	Cestodicide
Doramectin 10 mg/mL injection (Pfizer)	Dectomax	Raptors, Bustards	SC, IM	1 mg/kg	Used to treat alimentary tract nematodes, lungworms, eyeworms, and mites
Febantel	Avicas 15-mg tablets (Ortho Pharma) Rintal Suspension (Miles)	Pigeons	PO	37.5 mg/kg single dose	Ascariasis and capillariasis
Fenbendazole	Panacur 2.5% or 10% Liquid, 8-mg capsules (Hoechst)	Raptors	PO	100 mg/kg once	Ascarids, some microfilariae, capillariasis, other nematodes, and trematodes. May be effective against *Syngamus* spp.; not effective against the gizzard worms that infect finches. Can cause feather abnormalities during molt and also have adverse effect if used during the breeding season.
			PO	20 mg/kg daily for 14 d	
			PO	20 mg/kg daily for 5 d	
	Panacur 2.5% or 10% Liquid, 8-mg capsules (Hoechst)	Waterfowl	PO	20 mg/kg once	
	Panacur 8-mg capsules (Hoechst)	Pigeons	PO	One capsule/pigeon >8 wk old, single dose	20-50 mg/kg daily for 5 d for capillariasis. Do not use during molt.
	Panacur (Hoechst)	Psittacines	PO	15 mg/kg daily for 5 d; or 20-50 mg/kg once and repeat after 10 d	Medicate feed for 7 consecutive days. Broad-spectrum anthelmintic. Dosed according to bodyweight has produced no side effects in bustards
		Bustards	PO	30 mg/kg	Used, in addition to ivermectin treatment, to treat *Serratospiculum* spp. in falcons at a dose of approximately 20 mg/kg/d for 14 d. Dose of 60 mg/kg PO used in pheasants. Use all benzimidazole anthelmintics with care in molting birds—feather stunting has been reported in other species. Appears to be well tolerated by bustards.

Anthelmintics—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Fipronil	Frontline (Rhône Mérieux)	Raptors	Topical	Spray direct on to skin. One treatment usually sufficient. Repeat after 1 mo if required	All ectoparasites. Beware drying action of alcohol on feather structure—may reduce durability of feathers
Levamisole	Levacide (Norbrook), Ripercol-L (American Cyanamid)	Raptors	SC	10-20 mg/kg once	Control nematodes, including *Capillaria* spp.
			PO	20 mg/kg once	
			PO	40 mg/kg once	
	Levacide 7.5% Injection (Norbrook)	Waterfowl	SC	25-50 mg/kg once	Low therapeutic index. Do not use in debilitated birds. Toxic if given parentally to finches.
		Pigeons	IM	0.1 mL once. Can repeat after 7 d	Immunostimulant 2 mg/kg IM or SC every 14 d three times
			PO	300-400 mg/L drinking water as sole source of water over 24 hr. Repeat 7 d later	Loft treatment of capillariasis and ascariasis. Follow up to parenteral treatment
	Nilverm (Mallinckrodt); Levasole (Pitman-Moore)	Psittacines	PO	Use 1:40 dilution of 7.5% solutions: 20-50 mg/kg (5-15 mL/4.5 L) for 1-3 d	Has a low therapeutic index, therefore beware use as a wormer
			IM or SC	2-5 mg/kg, repeated 10-14 d on three occasions as an immunostimulant	
Mebendazole	Mebenvet (Janssen); Telmin (Pitman-Moore)	Raptors	PO	20 mg/kg daily for 14 d	Broad-spectrum ovicidal anthelmintic but primarily use for *Capillaria*. May be given by oral gavage but most commonly administered in the food of Galliformes and waterfowl. Toxic in pigeons, cormorants, finches, and raptors. Has been associated with hepatitis in some mammals and raptors. Appears to be well tolerated by bustards
		Waterfowl	PO	5-15 mg/kg daily for 2 d	
			PO	120 ppm (1.2 g/ton) in feed for 14 d	
Moxidectin	Imox tablets 1000 mg/tablet (Vetafarm)	Raptors	PO	0.5 to 1 mg/kg once	For the treatment of internal and external parasites by oral dosing. Drug recommended by J. Samour for the control of *Serratospiculum seurati*, *Capillaria* spp., *Acanthocephalan* spp., *Paraspiralatus sakeri*, and *Physaloptera alata* in falcons
Piperazine	Wazine 34 (Salisbury)	Pigeons	PO	1 g/L drinking water (12.5 mg/kg)	Has been used for ascarids in gallinaceous birds. Not effective in psittacines and finches. Repeat the dose every 10-14 d

Continued

Anthelmintics—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Praziquantel	Droncit 50-mg tablets (Bayer, Bayvet), Droncit Injectable 56.8 mg/mL	Raptors	SC or PO	5-10 mg/kg once	Active against cestodes and trematodes
			SC or PO	5-10 mg/kg for 14 d	May need to repeat treatment 14 d after first dose
		Bustards		1 mg/kg	Has been used in bustards. Cestodes are expelled within 24 hr. Injectable form may be toxic in finches and has been associated with depression and death in some species. Appears to be well tolerated by bustards
		Waterfowl	SC or PO	10-20 mg/kg. Repeat after 10 d	
			SC or PO	10 mg/kg daily for 14 d	
		Psittacines	IM	9 mg/kg	For tapeworms repeat after 10 d
			PO	10-20 mg/kg	
			IM or SC	10 mg/kg daily for 3 d, then 10 mg/kg daily for 11 d PO	
		Pigeons	PO	20 mg/kg single dose	For flukes
Pyrantel	Strongid (Pfizer); Nemex (Pfizer)	Raptors	PO	20 mg/kg once	Control of nematodes
		Psittacines	PO	4.5 mg/kg. For nematodes, repeat after 10 d	
Thiabendazole	Equizole 4 mg/30 mL	Most species	PO	250-500 mg/kg repeat 10-14 d	Treatment for ascarids. For treatment of helminth parasites especially *Syngamus trachea*. Maybe toxic to cranes, ratites, and diving ducks
	Suspension, (MSD agent)			100 mg/kg sid for 7-10 d	

Ectoparasiticides

Generic and Trade Names	Formulation	Route of Administration	Dosage	Activity	Comment
Coumaphos Negasunt (Bayer)	Powder containing 3% w/v coumaphos 2% w/v propoxur 5% w/v sulphanilamide	External application	Dust onto feathers	Active against all ectoparasites	Contains carbamate propoxur Useful for fly-blown wounds
Derris powder Derris 2% (Vet Drug)	Powder containing 2% w/v derris	External application	Dust onto feathers	Active against all ectoparasites	Safe and effective; brush out excess from hand-held bird
Fipronil Frontline (Merial UK)	0.25% solution in alcoholic spray	External application	Lightly wipe feathers with cotton wool moistened with spray	Blocks GABA neurotransmitters in all ectoparasites	Do not use the spot-on preparation. Best not to spray directly onto the feathers as the alcoholic preparation may damage plumage.

Ectoparasiticides—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
High-cis permethrin	Harker's Louse Powder (Harker, Generic)	Raptors	Topical application		Ectoparasite control
		Psittacines	Topical dusting		
Ivermectin	Ivomec 1% Cattle Injection (MSD, Agvet)	Raptors	SC	1-2 mg/kg. Repeat after 14 d	Some nematodes, feather mites, lice, and *Cnemidocoptes* spp. May be effective also for some coccidia, microfilariae, *Syngamus* spp., *Capillaria* spp., and *Sternostoma* spp. Has been used undiluted in raptors by IM injection for the control of *Serratospiculum* spp. May be toxic when given by injection in some small birds, some finches, and budgerigars. Appears to be well tolerated by bustards. Percutaneously used in canaries, finches, and budgerigars. Control nematodes and nasal or duck leeches. Dilute 1:9 with sterile water and use 0.2 mL/kg. Active against nematodes, feather mites, lice, and *Cnemidocoptes* spp. Repeat 7-10 d after first dose. Can also be administered topically or orally
		Psittacines	IM, SC, and PO	200 μg/kg	
		Pigeons	IM	Dilute 1:9 in sterile water just before use. Give 200 mcg (0.2 mL)/kg	Lice, mites
	Ivermectin 0.8% w/v in propylene glycol (Vetrepharm)	Pigeons	Topical	Apply one drop to the skin once a week for 3 wk	
Malathion	Duramitex (Harkers, Generic)	Raptors	Paint or spray on to perches	Dilute to 0.93%	Treatment of premises infested with *Dermanyssus* spp.
Piperonyl butoxide/ pyrethrin	Rid-mite Powder (Johnson)	Raptors	Topical dusting	Repeat after 3 wk	Control lice, hippoboscids
		Psittacines	Topical	Apply to plumage. Repeat after 10 d	
Permethrin	Companion Flea Powder (Battle, Haywood & Bower, Generic)	Pigeons	Dusting powder		Fleas, lice
Permethrin/piperonyl butoxide/methoprene	Avian Insect Liquidator (Vetafarm)	Most species	Ready-to-use spray, concentrate	Apply to plumage, spray cages, aviaries, bird rooms, and surroundings	Fleas, lice, mites, flies, mosquitoes, and moths

Sedatives/Tranquilizers/Anesthetics

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Alphaxalone/ Alphadolone	Saffan (Mallinckrodt)	Psittacines	IV–IM/IP	5-10 mg/ kg/36 mg/kg	May cause a transient apnea
		Cranes, flamingoes, and bustards	IV	4-8 mg/kg	Surgical anesthesia lasts for 8-10 min
					Practical and effective for short surgical procedures, e.g., surgical sexing
Atipamezole	Antisedan (Pfizer)	Psittacines	IM	250-380 mcg/kg	Used to reverse xylazine or medetomidine
Diazepam	Valium (Roche)	Raptors	IV or IM	0.5-1 mg/kg bid or tid as required	Control of fits
		Waterfowl	IV or IM	0.5-1 mg/kg bid or tid, as required	
Halothane	Halothane (Rhône Mérieux), Fluothane (Mallinckrodt; Fort Dodge)	Psittacines	Inhalation agent		Slower recovery than with isoflurane, marked respiratory depression, and moderate myocardial depression. Metabolism is impaired with liver disease, in particular fatty liver in obese captive birds
Isoflurane	Isoflo (Mallinckrodt)	Raptors	Inhalation anesthetic		Anesthetic of choice in avian species
	Isoflurane (Abbots Laboratories) Aerrane (Anaquest)	Psittacines	Inhalation agent		
Ketamine	Ketaset (Willows Francis); Vetalar (Upjohn); Ketaset (Fort Dodge/Ave, co.); Ketalar (Parke-Davies)	Raptors	PO	100 mg/kg in a 30 g piece of meat	Sedation to capture an escaped bird.
			IM	5-30 mg/kg	Reversible anesthetic in combination with medetomidine
		Psittacines	IV, IM, or SC	20-50 mg/kg	Ketamine on its own does neither produce good muscle relaxation nor adequate analgesia
Medetomidine	Domitor (Pfizer)	Raptors	IM	150-350 mcg/kg	Reversible anesthetic (by equal volume of Antisedan) in combination with ketamine. Use atipamezole to reverse
		Psittacines	IM	60-85 mcg/kg	
Midazolam	Hypnovel (Roche); Versed (Roche)	Raptors	IV or IM	0.5-1 mg/kg tid	Control of fits; shorter duration than diazepam
		Psittacines	IM or SC	0.2 mg/kg	For use in combination with ketamine
Propofol	Rapinovet (Mallinckrodt); Diprivan (Stuart)	Psittacines	IV	1.33 mg/kg	Useful to induce anesthesia
Sevoflurane	Ultane (Abbot)	Psittacines	Inhalation anesthetic		Expensive, requires specific vaporizer. Induction and recovery faster than isoflurane
	Raptors				
Tiletamine	Telazol (Robins; Fort Dodge)	Psittacines	IM	5-10 mg/kg	Provides good immobilization, also contains zolazepam
Xylazine	Rompun (Bayer, Miles); Virbaxyl (Virbac)	Raptors	IM or IV	1-2.2 mg/kg	Xylazine in combination with ketamine (1:3 or 1:5) is still widely used in raptors by many veterinarians in developing countries. Its effect can be reversed using yohimbine hydrochloride 0.1-0.2 mg/kg IV
		Psittacines	IV or IM	1-2.2 mg/kg	

Behavior Modifier

Drug and Trade Name	Formulation	Route of Administration	Dosage	Comment
Hydroxyzine Atarax (Roerig)	10-, 25-, 50-, and 100-mg tablets 10 mg/5 mL syrup	PO	2.0-2.2 mg/kg q8h—Amazon parrots 34-40 mg/L drinking water—most species	Used for feather picking and self-mutilation in psittacine patients
Nortriptyline Nortriptyline HCl (Pharmaceutical Associates)	10 mg/5 mL oral solution	PO	16 mg/L drinking water	A tricyclic antidepressant used to treat feather-picking birds. Has antiinflammatory properties

Behavior Modifier—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Amitriptyline	Lentizol (Upjohn); Elavil (Stuart)	Psittacines	PO	1-5 mg/kg sid or bid	Behavior modifier. Useful with some feather pluckers.
Clomipramine	Anafranil (Geigy, Baker Cummings)	Psittacines	IM	0.5-1 mg/kg sid or bid	Behavior modifier
Doxepin	Sinequan (Pfizer)	Psittacines	PO	0.5-1 mg/kg bid	Behavior modifier. Useful in some feather pluckers.
Fluoxetine	Prozac (Dista)	Psittacines	PO	0.4 mg/kg sid	Antidepressant. Useful in some feather pluckers
Haloperidol 2 mg/mL solution	Dozic (RP Drugs); Haldol (Henry Schein)	Psittacines	PO	0.4 mg/kg sid	For feather plucking
Naltrexone	Nalorex (Du Pont); Trexan (Du Pont)	Psittacines	PO	1.5 mg/kg bid	An opioid antagonist used to prevent self-mutilation.
Paroxetine			PO	1 to 2 mg/kg sid or bid	Blocks serotonin uptake in the brain resulting in antidepressive activity
Phenobarbital	Phenobarbital (Poythress)	Psittacines	PO	3 mg/kg bid	Used in cases of feather plucking. May cause deep sedation and inability to perch

Analgesic/Antiinflammatory

Drug and Trade Name	Formulation	Route of Administration	Dosage	Comment
Cimetidine Tagamet (SmithKline Beecham)	300 mg/2 mL injectable solution	IM	5 mg/kg q8-12h—psittacines 5-10 mg/kg q12h—ratites	Used for ingluvial inflammation and gastric ulceration
Diphenhydramine Benadryl (Pfizer)	Multiple oral formulations (tablets, liquid medication)	PO	1-4 mg/kg q8h—macaws, Amazon parrots 20-40 mg/L drinking water—most species	Inflammatory skin and respiratory reactions

Analgesic/Antiinflammatory—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Carprofen	Zenecarp (C-Vet)	Psittacines	IV, IM, or SC	2-10 mg/kg sid	Nonsteroidal antiinflammatory drug with analgesic and antipyretic properties. Used in mammals for the control of postoperative pain and inflammation after orthopedic and tissue surgery (including intraocular surgery). Second dose can be administered 24 hr after the first.
Diclofenac	Voltarol 25-mg tablets (Geigy)	Pigeons	PO	12.5 mg as a single dose	Arthritis Residue in carcasses responsible for the decline of vultures in the Indian sub-continent. Use with caution
Flunixin	Finadyne (Schering-Plough); Bonamine (Schering-Plough)	Raptors	IM	1-10 mg/kg sid for 1-5 d only	Potent nonsteroidal non-narcotic analgesic agent with antiinflammatory, anti-endotoxic, and antipyretic properties. Not recommended to treat for more than 5 consecutive days in mammals. Reported to cause vomiting.
Ketoprofen	Ketofen (Rhône Mérieux) (Fort Dodge/Aveco) Ketofen 1% Injection (Rhône Mérieux, Fort Dodge/Aveco)	Raptors Waterfowl Pigeons Psittacines	IM IM IM or SC IM	1 mg/kg sid for 1-10 d 1 mg/kg sid for 1-10 d 0.1 mL sid or bid on 2 consecutive days 2 mg/kg	Pain relief, arthritis. Antiinflammatory and analgesic Do not use in vultures
Meloxicam	Metacam 1.5 mg/mL suspension (Boehringer Ingelheim) Metacam 5 mg/mL injection (Boehringer Ingelheim)	Most species	PO IM, SC	0.5 to 1 mg/kg sid or bid	Nonsteroidal anti-inflammatory drug with analgesic and antipyretic properties. Used in mammals for the control of postoperative pain and inflammation following orthopedic and tissue surgery (including intraocular surgery).
Piroxicam			PO	0.5 mg/kg q12h	Inhibition of COX enzymes decreasing prostaglandins used in inflammation
Prednisolone	Prednicare (Animalcare); Solu-Delta-Cortef (Upjohn)	Psittacines	PO	2 mg/kg bid	Antiinflammatory and analgesic
Tolfenamic acid	Tolfedine 4% injectable (Vetoquinol)	Raptors	IM, SC	2-4 mg/kg	Nonsteroid anti-inflammatory drug with analgesic and antipyretic properties.
Tramadol		Eagles Psittacines		5 mg/kg q12h 30 mg/kg q6h	Management of mild-moderate pain

Hormones

Generic and Trade Names	Formulation	Route of Administration	Dosage	Comment
Delmadinone Tardak (Syntex)	10 mg/mL aqueous suspension	IM	1 mg/kg (0.02 mL/30 g) once	An anti-androgen. Sometimes effective for neurotic regurgitation in budgerigars. Well tolerated.
Dexamethasone Dexadreson (Intervet) Azium (Schering-Plough)	10 mg/mL clear aqueous suspension	IM, IV Topical	0.2-1 mg/kg once or q12-24h × 2-7 d, then q48h × 5 d—most species (antiinflammatory) 2-4 mg/kg q12-24h—most species, ratites (shock, trauma) 2-8 mg/kg q12-24h—cranes Mixed in 50% solution with DMSO	The action of corticosteroids is subtly different in birds compared with mammals (Westerhof, 1995). Birds are much more sensitive to their action. To reduce the inflammatory response and combat shock. Preferably given with appropriate antibiotics. If possible, always use NSAIDs
Dexamethasone sodium phosphate Dexaject SP (Vetus)	4 mg/mL injectable vial	SC, IM, IV	2-4 mg/kg q6-24h—most species	Faster acting dexamethasone product for head trauma and shock.

Hormones—cont'd

Generic and Trade Names	Formulation	Route of Administration	Dosage	Comment
Levothyroxine		PO q12 to 24 h	0.2 mg/kg	Treatment of hypothyroidism. Induction of molt—may cause feather dystrophy if molt induced too rapidly
Suprelorin	4.7 mg implant	SC	One implant per bird regardless of size,	Desensitizes GNRH receptors and decreases release of FSH and LH. Used in birds for behavioral and reproductive conditions (esp chronic egg laying). Variable results.
Testosterone Durateston (Intervet)	50 mg total esters/mL oily solution for injection containing four esters of testosterone	IM, SC	2-8 mg/kg once	Prolonged activity over 2 weeks.

Hormones—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Buserelin	Receptal	Psittacines	IM	0.5-1 μg/kg q48h up to 3x	Used to suppress chronic egg laying
Cabergoline	Galastop	Psittacines	IM	10-50 μg/kg q12-24h	May have beneficial effect in chronic egg laying
Dinoprost	Lutalyse (Upjohn)	Raptors	Topical	0.02-0.1 mg/kg on to cloacal mucosa, once	Egg binding
		Waterfowl	IM or topical	0.02-0.1 mg/kg on to cloacal mucosa, once	
		Psittacines	IM or per cloacum	0.02-0.1 mg/kg once	
Leuprolide acetate	Lupron (TAP Pharmaceutical)	Cockatiels	IM	0.375 mg/kg once	Prevention of ovulation
Medroxy-progesterone	Promone-E (Upjohn)	Psittacines	IM or SC	5-50 mg/kg every 4-6 weeks: 150 g bird—0.05 mg/g; 300-700 g bird—0.03 mg/g; >700 g bird—0.025 mg/g	Used for excessive egg production, especially in cockatiels. Can cause lethargy, inappetence, polydipsia, and fatty liver syndrome
Nandrolone laurate	Laurabolin 50 mg/mL (Intervet)	Psittacines, raptors, bustards	IM or SC	0.4 mg/kg once	Testosterone derivative recommended as part of treatment after chronic and debilitating diseases. Give every 3 wk. Can cause liver disease
Oxytocin					No longer recommended for birds
Thyroxine	Soloxine tablets (Vet-2-Vet, Butler)	Raptors	PO	Birds weighing 750-1000 g, e.g., female red-tailed hawk: 25 μg sid for 7 d 50 μg sid for 7 d 75 μg sid for 7 d 50 μg sid for 7 d 25 μg sid for 7 d	Stimulate molt. Scale dose up or down by up to 50% for larger or smaller birds
		Psittacines	PO	100 mg/kg bid (i.e., 0.025 mg/100 mL water) for 4 wk	Used to induce molt and treat hypothyroidism
Levothyroxine		Psittacines, raptors	PO	0.2 mg/kg q12-24h	Treatment of hypothyroidism. Induction of molt—may cause feather dystrophy if molt induced too rapidly

Vitamins, Minerals, and Nutritional Supplements

Drug	Formulation	Route of Administration	Dosage	Comment
Ace-High (Vetark)	Powdered vitamin supplement with high levels of vitamins A, C, and E, plus B vitamins and minerals including zinc	Orally in food	Follow directions on pack	As an aid in the treatment of stress or infectious disease, particularly viral infections. *Do not use with other vitamin supplements*
Critical Care Formula (Vetark) also Emeraid Critical Care (Lafeber, Co., USA)	Each 5 g contains 0.72 g protein, 18 Kcal of energy	Orally	Make up 1:2 with water Do not make up more concentrated and give orally by gavage	To stop weight loss in severely ill birds. Provides easily digestible calorific support as mixture of short-chain maltodextrin plus concentrated protein and amino acids
Human invalid foods Farlene Complan (Farley Health Foods) Build Up (Carnation Foods) Vita Food (Boots Ltd) Milupa	Human invalid foods that contain approximately 400 Kcal/100 g of food Fruit salad, tropical fruit	By crop tube	100 g/kg daily 7.5 g/30 g daily	Best results obtained in the severely debilitated bird by dividing daily dose and giving at hourly intervals. Add a probiotic, e.g., Avipro. Useful in an emergency if no specific avian product is readily available
Multivitamin and mineral preparations SA 37 (Intervet) Vionate (Squibb)	See maker's data and information sheets	Oral	500 mg/kg body weight	May be wasteful when mixed with seed Needs to be given in a mash
Multivitamin injections (C-Vet; Norbrook) (Arnolds)	See maker's data and information sheets	IM	0.5 mL/kg supplying 7,500 IU vitamin A and other vitamins	*Vitamin A overdose is toxic* and can produce skeletal abnormalities and damage to membranes *Use with caution in macaws and African grey parrots*
Multivitamin oral drops Abidec (W-L, human preparation) vitamins A, B group, C and D	See maker's data and information sheets	In drinking water	Five drops/0.3 mL/30 g bird, every third day	
Duphalyte (Duphar)	An injectable solution of electrolytes, vitamins, amino acids, and dextrose	SC, *very slowly* IV	10 mL/kg q12h	Given in the groin or at base of neck with hyaluronidase
Natural yogurt	Contains *Lactobacillus acidophilus*	Oral	2 mL/kg body weight	May help to restore gut flora after antibiotic therapy. Controversial but will do no harm
Nutrobal (Vetark)	Powdered vitamin/mineral supplement with calcium: phosphorus ratio of 40-50:1	Orally in food	Follow directions on pack	Designed to balance diets low in calcium; also contains 150 IU vitamin D_3/g Valuable supplement for fast-growing young birds and laying hen birds *Do not use with other vitamin supplements*

Viruses Prophylactic Protection Against Viruses

Virus	Formulation	Effectiveness and Comment
Fowl pox	Vaccine Freeze-dried live virus powder for reconstitution Poxine (Solvay)	Licensed in the UK for use in poultry. Effective against turkey pox, quail pox, and falcon pox. Does not protect peacocks against peacock pox virus
Duck virus hepatitis enteritis	Duck virus hepatitis (Animal Health Trust) Duck plaque virus (Intervet)	Animal Health Trust vaccine only available to members of Duck Producers Association. Intervet vaccine—Anseriformes/modified live virus (MLV) may be used during outbreak
Infectious laryngotracheitis (ILT)	Freeze-dried live vaccine ILT Vaccine (Solvay)	Licensed for use in UK in poultry, not pheasants. May be effective against Amazon tracheitis, but there is risk of an undesirable reaction
Marek's disease virus	Freeze-dried live vaccines: • Delvax (Mycofarm) • Marexin (Intervet) • Md-Vac (Solvay) • Also cell-associated live vaccines available.	Licensed for use in UK in poultry. Before use, consult manufacturers and DEFRA
Corona viruses		
Avian infectious bronchitis	Live virus vaccines for addition to drinking water IBMM (Solvay) IB Nobilis H-52 and H-120 and Strain MA5 (Intervet) Poulvac H52 and H120 (Solvay)	Licensed for use in UK in poultry but *may* be effective in pheasants, guinea fowl, psittacines, pigeons, ostrich, and Japanese quail, providing an exact diagnosis has been made. Consult manufacturer and DEFRA before use
Abro viruses		
Avian encephalomyelitis	Live virus vaccine for addition to drinking water AE-Vac (Solvay) AE-Nobilis (Intervet)	Licensed for use in UK in poultry but *may* be effective in other species, providing a specific diagnosis has been made. Consult manufacturer and DEFRA before use
Louping ill virus	Inactivated + oil adjuvant louping ill vaccine (Pitman-Moore)	Licensed for use in UK in cattle, sheep, and goats but may be effective in susceptible avian species, i.e., grouse, capercaillie, and pheasant. Consult manufacturer and DEFRA before use
Eastern equine encephalitis (EEE) and western equine encephalitis	Triple-E (Solvay) EEE vaccine (Fort Dodge)	Emus, eclectus parrots, and palm cockatoos should be vaccinated and boosted annually in endemic areas
West Nile virus	West Nile—Innovator (Fort Dodge)	Full equine dose recommended if at all possible. Inconsistent and unsubstantiated immunity from available equine vaccine in avian species. Not licensed for avian use
Papova viruses		
Polyomavirus	Avian Polyoma Vaccine (Biomune)	Thickened skin may be noted at vaccination site

Vaccines

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Canary pox virus strain Herzberg vaccine	Poulvac P Canary (Duphar, Holland)	Houbara bustards	Topical (wing-web)		Recommended for use in houbara bustards following research at the National Wildlife Research Center, Taif, Saudi Arabia
Famciclovir	25 mg/kg	Ducklings	PO	Q 12h	Inhibits viral replication (viral DNA polymerase of herpesviruses)
Newcastle Disease Vaccine (NDV)—Living	NDV Hitchner B1 (Living)	Bustard chicks	Intraocular or intranasal	1-2 Drops	For the primary vaccination of young growing bustards at 4, 8, and 20 wk. Does not result in detectable serum hemagglutinating antibodies in bustards
NDV—Inactivated	Newcavac Nobilis (Inactivated)	Bustards	SC	1.0 mL/kg	For the secondary vaccination of bustards. Administered at 32 wk of age. Must be used only when birds have been primarily vaccinated with NDV Hitchner B1 living vaccine. Annual vaccination will result in detectable serum HI antibodies. Response after 14 d
Paratyphoid vaccine	Bespoke (Specialist Laboratories)	Pigeons	IM	Usually 0.25 mL	Persistent loft problem with paratyphoid
Pigeon pox vaccine	Pigeon Pox Vaccine (Living) Nobilis (Intervet, Vetrepharm)	Pigeons, falcons	Topical	Topical application on to 6-8 exposed feather follicles	Pigeon pox. Vaccinate on lower legs. Pluck 6-8 feathers and stretch skin to open feather follicles. Brush vaccine onto open follicles. For routine prophylaxis, vaccinate young birds before racing: vaccinate old birds at least 6 wk before pairing. Vaccinated birds are infectious until the vaccine lesions have healed. Vaccinate every year in early autumn
PMV-1 Vaccine	Colombovac PMV (Solvay Duphar) Nobi-Vac Paramyxo (Intervet, Vetrepharm)	Pigeons	SC SC SC	0.2 mL 0.2 mL 0.25 mL	PMV-1. Choose vaccination site carefully. Avoid vascular trauma
Psittimune PDV (Pacheco's disease vaccine, killed virus)	Biomune Co	Psittacines	SC, IM		Susceptible psittacine birds before the breeding season, before fledging, during quarantine, or before exposure to other psittacines
Psittimune APV (Avian polyomavirus vaccine, killed virus)	Biomune Co	Psittacines	SC	Dose rate for larger birds 200 g is 0.5 mL. Dose for birds <200 g is 0.25 mL	Susceptible psittacine birds before the breeding season, during quarantine or before exposure to other psittacines. Young birds are vaccinated at 5 wk of age
Poximune C (Canary pox vaccine, MLV)	Biomune Co	Canaries	Wing web method	0.01 mL per bird	Susceptible canaries at least 4 wk old
Reovirus vaccine	Nobilis Reo Inac (Intervet International, France)	Bustards	SC		Inactivated reovirus vaccine used in some bustard collections

Topical

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Aloe vera, sodium lauryl sulfate, sodium dodecyl, and benzene sulfonate	Bird Rain (Avian Care Products)	Cage and aviary birds	Topical	Spray on to feathers	Revitalize skin and feathers
Aloe vera, ammonia solution	Soother Ointment (Avian Care Products)	Cage and aviary birds	Topical	Apply on to affected areas	Promote healing of skin irritation and superficial lesions. Do not use in birds with kidney disorders
Dimethylsulfoxide	Fluvet DMSO (Univet); DOMOSO (Syntex)	Pigeons, raptors	Topical application		A method of reducing swelling and a vehicle for carrying some antibiotics and antiinflammatories into difficult-to-reach sites of infection, particularly in the hock and foot region. Avoid contact with human skin
Triamcinolone/neomycin/ thiostrepton/nystatin	Panolog Ointment (Ciba)	Pigeons	Topical	One drop into the eye bid for 3-5 d	Conjunctivitis, "one-eyed cold"

Topical Preparations

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Oil of proflavine	Proflavine Cream (Loveridge)	Most species	Topical	Apply to wounds sid or bid to effect	Very safe. Stimulates granulation. May cause yellow coloration of urates
Povidone-iodine	Pevidine Surgical Scrub (BK); Vetasept (Animalcare)	Most species	Topical	Apply and wash off after 3 min	Very safe cleansing agent for open wounds
Propylene glycol, malic acid, benzoic acid, and salicylic acid	Dermisol (Pfizer)	Most species	Topical	Apply sid or bid to effect	Removes skin debris, scabs, and crusts. Antiseptic properties. Very safe
Sodium fusidate	Fucidin	Most species	Topical	Apply sparingly bid to effect	Antibacterial, particularly against *Staphylococcus* spp. Infections. Useful for mild/early bumblefoot and other skin lesions. Reported to penetrate intact skin
Sodium fusidate/ hydrocortisone	Fucidin H (Leo)	Raptors	Topical	Apply sparingly bid to effect	Antibacterial, particularly against *Staphylococcus* spp. infections plus antiinflammatory action. Reported to penetrate intact skin. Excess application will cause polydipsia

Miscellaneous Drugs

Drug and Trade Name	Formulation	Route of Administration	Dosage	Comment
Aminophylline Roxane (Watson)	105 mg/5 mL oral solution	PO	4-5 mg/kg q6-12h—most species, psittacines 8-10 mg/kg q6-8h—raptors, ratites	Bronchodilator
Bisolvon (Boehringer)	8-mg tablet (human medical product) 10-mg/g oral powder for addition to drinking water			Bronchial mucolytic
Bromhexine hydrochloride	3 mg/mL solution for injection	IM	3-6 mg/(1-2 mL)/kg, 0.1 mL/30 g divided into two or three doses daily	Mucolytic. May help better penetration of antibiotics and gamma globulins into respiratory tract. Well tolerated. The injection is water based so can be given orally or in daily water intake (Ahlers, 1970)
Carboplatin		IV over 3 minutes	5 to 10 mg/kg	For neoplastic disease 5 mg/kg intralesional to make concentrate of 10 mg/ml for squamous cell carcinoma

Continued

Miscellaneous Drugs—cont'd

Drug and Trade Name	Formulation	Route of Administration	Dosage	Comment
Chlorambucil		PO for 2 weeks	1 to 2 mg/kg	Management of some lymphoproliferative disease
Colchicine		PO q12 hrs	0.04 mg/kg	Management of hepatic fibrosis and renal amyloidosis
Cyclophosphamide		IO q7 d	200 mg/m^2	Treatment of lymphoproliferative disease
Deferoxamine, Desferal		PO IM PO, IM, SC	20 mg/kg 40 mg/kg 100 mg/kg	Hemochromatosis of softbills in a wide range of doses
Diphenhydramine injectable (Elkins-Sinn)	50 mg/mL injectable solution	IV, IM	2-4 mg/kg q12h—most species	
Enalapril Enacard (Merck)	1-, 2.5-, 5-, 10-, and 20-mg tablets	PO	0.25-0.5 mg/kg q24-48h—psittacines	Treatment for cardiomyopathy. Not recommended if patient has renal disease
Ferric chloride solution BP also powder (non proprietary)		Apply topically to bleeding areas		To arrest minor hemorrhage.
Gabapentin		PO	10 mg/kg q12-24h	Adjunct therapy in the tx of seizures
Glibenclamide BP (nonproprietary)	2.5-mg tablets	Oral in drinking water	Dissolve one-fourth tablet (0.62 mg) in 1 L	For non–insulin-dependent diabetes mellitus in psittacines, particularly budgerigars
Glycerine		Oral per cloacum	5 mL/kg q24h	For impaction of the cloaca and egg binding
Liquid paraffin mineral oil		Oral per cloacum	4 mL/kg q24h	For impaction of the cloaca and egg binding
Low molecular weight heparin		IV	300 iu/kg once	Anticoagulant that inhibits factor Xa and thrombin. Used in birds to treated PTFE toxicosis
Mannitol		Slow IV	0.2-2 mg/kg	Reduction of intracerebral pressure (most effective in acute elevations of IC pressure. Used in acute renal failure and cerebral edema
Naloxone		IV	2 mg/kg	Treatment of opioid overdose
Norcuron (Organon-Teknika)			Use one drop only, every 5 min for 15 min until pupil dilates	
Propranolol		Slow IV	0.04 mg/kg	Nonselective beta blocker to manage cardiac arrhythmias. This dose has been used in cases of supraventricular tachycardia
Pancreatic enzyme supplements		PO	1 capsule/kg bodyweight per day, sid	Treatment of exocrine pancreatic insufficiency
Potassium iodide		In drinking water	100-200 mg/100 mL	As a palliative in the treatment of chronic respiratory disease. Should not be given for periods longer than a week
Silver nitrate	Stick or pencil	Apply to points of minor hemorrhage		To arrest localized bleeding by chemical cautery. Flush the area with saline solution to form silver chloride when bleeding has ceased to neutralize the cauterizing action
Sodium iodide	20% sterile solution for injection (200 mg/mL)	IM	0.01-0.03 mL (2-6 mg)/30 g bird 0.33-1.0 mL (66-133 mg)/kg body weight	Secondary thyroid hyperplasia caused by iodine deficiency. Considerable improvement to respiratory obstruction seen within 3 d in budgerigars
Sucralfate		PO	25 mg/kg q8h	Stimulation of mucosal defenses. Used in the therapy of gastric or duodenal ulcers
Sucrose in water	30% solution	Oral	Up to 10 mL/kg q12h	Mild purgative
Vecuronium bromide	10-mg ampule powder for reconstitution	Topical	Reconstitute in 2.5-mL water	Used as an alternative to tubocurarine as a nondepolarising muscle relaxant to cause dilation of the pupil (mydriasis) because in birds this is under CNS and not parasympathetic control (as in mammals)

Miscellaneous

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Aciclovir	IV Solution Powder, 200-mg capsule, 50 mg/mL, 40 mg/mL Suspension, Gel (Zovirax, Burroughs, Wellcome)	Most species	PO, IM, IV Topical	80 mg/kg tid up to 240 mg/kg of food	Muscle necrosis if given IM. Phlebitis and neurologic signs if given IV. Has been shown to affect sperm development and fetal development in mammals. Herpesvirus DNA polymerase inhibitor. Use to control Pacheco's disease virus. Most effective when administered before clinical signs occur. Also suggested that it can be used topically in pox virus infections
Activated charcoal	Forgastrin (Arnolds); Toxiban (Vet-A-Mix)	Psittacines	PO	2-8 g/kg as required	Used to absorb ingested toxins, including insecticides, heavy metals, and chemotherapeutic agents from the alimentary tract. Can be mixed with hemicellulose to act as a bulk laxative and aid in the passage of ingested toxins
Allopurinol	Zyloric (Glaxo-Wellcome)	Psittacines	PO	Dissolve one 100-mg tablet in 10 mL water—1 mL of diluted solution/30 mL drinking water	Used to treat gout. Well absorbed from the gut. Functions to inhibit purine catabolism, which prevents the formation of uric acid. Provide fresh drinking water and ensure that birds are well hydrated
Aminoloid	Aminoloid (Schering)	Raptors	IM	0.25-0.75 mg/kg repeat 10-14 d	Induction of molt in raptors
Aminopentamide hydrogen sulfate	Centrine Injectable Solution (Aveco)	Most species	IM, SC	0.05 mg/kg bid five doses maximum	Antiemetic. Antidiarrheal, slows gastrointestinal (GI) motility
Apple cider vinegar	Apple Cider Vinegar (organic; Avian Care Products)	Cage and aviary birds	PO	1 teaspoonful per pint of water as the sole drinking water for 10-14 d	Helps restore normal intestinal flora. Control of many Gram-negative bacterial and yeast problems
Atropine	Atropine Injection (C-Vet)	Raptors Waterfowl Psittacines	IV or IM IV or IM IM or SC	0.1 mg/kg every 3-4 hr 0.1 mg/kg every 3-4 hr 0.05 mg/kg repeat hourly	Acetylcholinesterase poisoning, e.g., carbamates. Not recommended as preanesthetic in avian species. Thicker respiratory secretions, which may block endotracheal tube. Used in organophosphate poisoning. Does not dilate pupils in avian species
Avipro	Contains *Lactobacillus*, *Streptococcus*, *Saccharomyces*, electrolytes, trace minerals and vitamins, amylase, cellulase, and proteases (Vetark)	Psittacines, raptors, bustards	PO	1 scoop/200 mL water; for stressed birds, use 1 scoop/100 mL	Probiotic combination of bacteria, enzymes, electrolytes, and vitamins. Highly palatable. Can be added to drinking water
Avipro Pediatric	Contains *Lactobacillus*, *Streptococcus*, *Saccharomyces*, amylase, cellulose, and proteases	Psittacines, raptors, bustards	PO	¼ teaspoon per 60 g food	An enzyme and probiotic blend for hand rearing or feeding convalescent birds. Use Avipro starter packs at 6 hr of age to start the chick off
Biotin	Biotin Tablets (Arnolds); (Generic)	Raptors	PO	50 mcg/kg daily for 30-60 d	Aid in beak or claw regrowth
Bismuth subsalicylate	Bismuth Subsalicylate Oral Suspension 1.75% subsalicylate	Most species	Oral	2 mL/kg bid	For GI irritation. May help to remove ingested toxins
Brewer's yeast	Dried Yeast Tablets BP 300 mg (Lloyds Chemists)	Pigeons	PO	One tablet crushed over feed/10 birds/d during the molt	For the treatment of brittle feathers if suspected to be nutritional in origin
Buprenorphine	Vetergesic (Animalcare); Buprenex (Norwich Eaton)	Psittacines	IV or IM	0.1 mg/kg bid	Antiinflammatory and analgesic

Continued

Miscellaneous—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Butorphanol	Torbugesic (Willows Francis); Torbutrol (Bristol Laboratories)	Psittacines	IV or PO	3-4 mg/kg tid	Antiinflammatory and analgesic. Use with caution in liver-compromised patients
Calcium gluconate or borogluconate 10%	Various Calcium Sandoz Injection (10-mL ampules; Sandoz, SmithKline Beecham, Fort Dodge)	Raptors Pigeons	IV or SC SC or slow IV	1-5 mL/kg slowly, once 0.1-0.2 mL (25-50 mg)/kg injection	Initial treatment of hypocalcemia, hypocalcemic fits, and egg binding. Also useful for bone healing
	Calcium borogluconate 20%, Calcibor CBG20 (Arnolds)	Psittacines	IV or IM	0.5-1 mL/kg	
	Calcium gluconate 20% (Sandocal, Sandoz, SmithKline Beecham, Fort Dodge)	Psittacines	IV IM or SC	50-100 mg/kg slowly to effect 5-10 mg/kg bid as required	
Cisapride	Propulsid (Janssen)	Psittacines	PO	0.5-1.5 mg/kg tid	Used to stimulate GI motility
Clofazimine	Lamprene Tablets (Geigy)	Raptors	PO	1-5 mg/kg sid for 3 mo to 1 yr	Tuberculosis in combination with enrofloxacin, cycloserine, and ethambutol. Beware zoonotic risk of *Mycobacterium avium* infection
Copper sulfate	Caustic powder 51% (Phoenix Butler)	Most species	Topical	Apply to affected area as needed	Treatment of cases of ulcerative dermatitis
Dandelion	Dandelion root (Avian Care Products)	Cage and aviary birds	PO	Mix 5 drops in ½ oz lactulose and use 1 drop/100 g bodyweight bid	Liver stimulant
D-penicillamine	Distamine (Dista); Cuprimine (Merk); Depen (Wallace); Titratabs (Wallace)	Raptors Waterfowl	PO PO	55 mg/kg bid for 7-14 d 55 mg/kg bid for 7-14 d	Heavy metal poisoning, e.g., copper, lead, and zinc
Dimercaprol	Bal Injectable Solution 100 mg/mL (Becton Dickinson)	Most species	PO	25-35 mg/kg bid 5 d per week for 3-5 wk	Painful injections IM. Less toxic and better at reducing blood lead levels than calcium EDTA
D-tubocurarine	D-tubocurarine Solution 3 mg/mL	Raptors	Ophthalmic drops	Every 5 min × 3 times	Mydriatic
Doxapram	Dopram Injection (Willows Francis, Fort Dodge) Dopram Injection (Willows Francis, Robins) Dopram-V (Willows Francis, Fort Dodge)	Raptors Waterfowl Psittacines	IV IV IV or IM	10 mg/kg once 10 mg/kg once 5-10 mg/kg once	Respiratory stimulant. Reversal of respiratory depression associated with overdose of general anesthetic, hypnotic, and sedative drugs. Speed up recovery from ketamine/xylazine anesthesia. Be aware with excessive doses hyperventilation may lead to cerebral vasoconstriction and cerebral hypoxia
Echinacea	Echinacea Solution (Bio-botanica) Echinacea (Avian Care Products)	Cage and aviary birds	PO	2.5 drops/kg, 5 drops/cup of water. Mix five drops in ½ oz lactulose and use 1 drop/100 g bodyweight bid	Immunostimulant especially in viral infections. Holistic use
EDTA-TRIS	Lysozyme Solution Mix 3.07 g Trizma HCl, 3.17 g Trizma base, 1.12 g disodium EDTA in 100 mL water	Most species	Topical	Used intratracheally, intranasally, or for wound lavage	Helps antibiotics penetrate bacterial wall

Miscellaneous—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Electrolytes, vitamins, amino acids, dextrose	Duphalyte (Solvay Duphar)	Psittacines, raptors, bustards	IV, SC, PO	10 mL/kg	Indicated for the prevention and treatment of dehydration, electrolyte imbalance, and hypoproteinemia. Administer SC in the groin or administer PO by stomach tube. Use very slow IV
Electrolyte solution	Tyrode's Solution (Avian Care Products)	Cage and aviary birds	PO	Add envelope to 1 quart drinking water. Use as sole source of drinking water	Electrolyte imbalance, cases of polyuria and polydipsia.
Epinephrine	Epinephrine injection 1 : 1000 epinephrine (Elkins-Sinn)	Most species	IV, IO, IT, IC	Emergency drug. Dilute with 10 parts Lactated Ringers Solution and use 0.5-1.0 mL/kg.	Clinical indications for attempts to restore cardiac function in cases of peracute death from anesthesia. Use drug with caution in birds. The therapeutic index for this drug is low.
Essential fatty acid	Dermplus Liquid (C-Vet)	Raptors	PO	0.5 mL/kg daily for 50 d or indefinitely	Pruritic dermatitis (atopy)
		Pigeons	PO	5 mL per 1 kg feed once weekly	Improve feather quality
Ferric subsulfate	Monsel's Solution, liquid or powder	Most species	Topical	As needed to stop bleeding, especially after nail trim	May cause feather cysts if applied into damaged follicles
		Psittacines	IM	1-10 mg/kg	
Furosemide	Lasix Injection (Hoechst)	Raptors	IM	1.5 mg/kg qid as required	Diuretic. Beware of overdose, which causes dehydration and electrolyte abnormalities
		Psittacines	IM, SC	0.15-2 mg/kg, sid or bid	Toxic reactions characterized by neurologic signs and death
Goldenseal	Goldenseal (Avian Care Products)	Cage and	PO	Mix 5 drops in aviary birds ½ oz lactulose and use 1 drop/100 g bodyweight bid	Immune stimulation. Short-term use only, i.e., <5 d
Glucose polymer		Pigeons	PO	15 g/L drinking water for 4 d	Racing pigeon tonic
Glucose 5%	Aquapharm No. 6 (Animalcare)	Most species	IV, SC, or PO	50 mg/kg (1 mL/kg)	Isotonic solution. Useful in the treatment of hypoglycemia and dehydration. IV slowly
Heparin	Heparin sodium 25,000 IU/mL (Leo Labs)	Most species	IV		Use for flushing IV locks and IV catheters Use diluted at 100 IU/mL in saline for flushing Heparin therapy should be given with caution to patients about to undergo surgery and those with impaired renal or hepatic function
Hyaluronidase	Hyalase, 150 IU/mL (CP Pharmaceuticals)	Most species		150 IU/L fluids	Increases the rate of absorption of subcutaneous fluids
Iodine and trace minerals	Budgie Builder (Avian Care Products)	Cage and aviary birds	PO	Dilute content to 1 gallon of water. Use as only source of drinking water	Nutritional supplement
Iodine	Lugol's	Psittacines	PO	Two parts iodine + 28 parts water—add 3 drops to 100 mL drinking water	For treatment of goiter in budgerigars

Continued

Miscellaneous—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Iron dextran	Vet Iron Injection (Animalcare, Butler, Lextron, Vedco)	Raptors	IM	10 mg/kg. Repeat after 1 wk if required	Hemopoiesis
		Waterfowl	IM	10 mg/kg. Repeat after 1 wk	
		Psittacines	IM	10 mg/kg, repeat in 7-10 d as required	
Isoxsuprine	Navilox (Univet)	Raptors	PO	5-10 mg/kg sid for 20-40 d	Wing-tip edema, dry gangrene syndrome
Kaolin	Kaogel (Upjohn); Kaolin (Vet-A-Mix, Evsco)	Psittacines	PO	15 mL/kg bid or tid	For treatment of nonspecific diarrhea
Lactated Ringer's solution	Hartmann's Solution, Aquapharm No. 11 (Animalcare)	Most species	IV, IO, or SC	10 mL/kg/min	Calculate fluid deficit from PCV. Give over 2-d period plus 50 mL/kg/d. Give the calculated daily requirement in four equal volumes during the day.
Lactulose	Duphalac (Solvay Duphar); Cephulac (Marion Merrell Dow)	Psittacines	PO	0.2-1 mL/kg q8-12h	Can be administered for a period of many weeks. To decrease toxin absorption from the alimentary tract and/or CNS symptoms from liver damage, stimulate appetite, and improve intestinal flora
Lipoform Tabs	Lipo-form Chewable Tablet (Vet-A-Mix)	Most species	PO	500 mg sid	Prevent liver damage in face of viral infection
Liquid iron		Pigeons	PO	2 mL per 2 kg feed daily for 3 d, then once weekly	Racing pigeon tonic
Magnesium sulfate crystals	Magnesium Sulfate (various)	Raptors	PO	0.5-1 g/kg sid for 1-3 d	Increase gut motility to aid passage of lead if present in intestine
		Waterfowl	PO	0.5-1 g/kg sid for 1-3 d	
Metoclopramide	Emequell (Pfizer); Reglan (Robins)	Raptors	IV or IM	2 mg/kg tid as required	Anti-emetic. Control of GI stasis, e.g., sour crop
		Waterfowl	IV or IM	2 mg/kg tid as required	
		Psittacines	IM, IV, or PO	0.5 mg/kg, repeat q8h as required	
Milk thistle	Milk Thistle (Avian Care Products)	Cage and aviary birds	PO	Mix 5 drops in ½ oz lactulose and give 1 drop/100 g bodyweight bid	Supports and protects the liver and helps restore liver function. Antioxidant
Monoglycerides of edible fatty acids in organic denude oil	Booster (Avian Care Products)	Cage and aviary birds	PO	1 drop of the liquefied product/100 g bodyweight	Stimulant of immune system
N-butyl-cyanoacrylate	Vet-Seal Tissue Adhesive (Braun)	Topical			Surgical glue. Indicated for the closure of skin wounds, surgical incisions, mucosa in oral surgery, and skin punctures. Useful for closing incision after surgical pinioning of young birds
Nutrobal	Per gram, 200 mg Ca, 150 IU Vitamin D_3 and other vitamins and minerals (Vetark)	Psittacines, raptors, and bustards	PO	Sprinkle on food at a rate of 1 pinch/kg of animal being supplemented	Calcium balancer and multivitamin supplement to help during growth and breeding in all birds. High potency: do not exceed normal levels
Oseltamivir phosphate	Tamiflu (Roche)	Bustards	PO	8-10 mg/kg bid	Antiviral agent active against influenza virus in humans. Has been used in white-bellied bustards by one of us (T.A.B.) with confirmed avian influenza. Clinical signs resolved and no side effects were observed

Miscellaneous—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Organic denude oil	Sunshine Factor (Avian Care Products)	Cage and aviary birds	PO	1 teaspoon of the liquefied product with 1 lb of food	Indicated for dry flaky skin, balding feet, lack of sheen, and improper feather color, especially in older birds
Oxyglobin	Hemoglobin glutamer-200 (Oxyglobin, Biopure)	Raptors	IV	10 mL/kg once	Provides oxygen-carrying support to dogs, improving clinical signs of anemia for at least 24 hr, independent of the underlying conditions. Has been used as a one-off treatment in anemic falcons
Pedialyte	Pedialyte (Abbott Laboratories): water, dextrose, sodium chloride, potassium citrate, and sodium citrate	Most species	PO		Pedialyte is used in the maintenance of body water and electrolytes in neonatal birds with mild or moderate diarrhea and dehydration
Penicillamine	Distamine (Dista); Cuprimine (Merk)	Psittacines	PO	50-55 mg/kg bid	Chelating agent that binds copper, zinc, mercury, and lead
Plasma substitute	Haemaccel 3.5% Colloid Infusion (Hoechst)	Most species	IV	10 mL/kg	Indicated as a plasma volume substitute in cases of hypovolemic shock caused by hemorrhage, burns, and water and electrolyte loss from persistent vomiting or diarrhea. Should be administered IV in a volume approximately equal to the estimated blood loss to restore circulatory volume
Poly-Aid	Poly-Aid Nutrient Supplement (Vetafarm)	Psittacines, raptors, and bustards	PO	10 g/100 g bodyweight/d in two divided doses	A sustained release of carbohydrate and protein supplement with vitamins and electrolytes for debilitated birds. Add 5 mL water to 10 g Poly-Aid and make into slurry
Pralidoxime chloride	Protopam (Wyeth-Ayerst)	Raptors	IM	100 mg/kg. Repeat once after 6 hr	Organophosphate and acetylcholinesterase poisoning, e.g., carbamates. Contact National Poisons Bureau regarding availability
Pralidoxime mesylate	Mesylate (Ayerst); Protopam (Wyeth-Ayerst)	Waterfowl	IM	100 mg/kg. Repeat once after 6 hr	Organophosphate and acetylcholinesterase poisoning, e.g., carbamates
		Psittacines	IM	100 mg/kg, repeat once after 6 hr	
Propentofylline	Vivitonin (Hoechst)	Raptors	PO	5 mg/kg bid for 20-40 d	Wing-tip edema, dry gangrene syndrome
Sodium bicarbonate	8.4% Sodium Bicarbonate 1 mEq/mL (Abbott Laboratories)	Most species	IV	1 mEq/kg q15-30 min to a maximum of 4 mEq/kg total dose	Emergency drug. Used to treat severe metabolic acidosis. Contraindicated in respiratory and metabolic alkalosis
Sodium chloride	Aquapharm No.1 (Animalcare) 0.9% sodium chloride	Psittacines, raptors, and bustards	SC, PO, or IV	50 mL/kg/d for maintenance	To correct water and electrolyte depletion. Indicated in severe vomiting of acute onset, or where lodgment of foreign bodies interferes with ingestion, e.g., where there is vomiting and/or endotoxic shock. Sodium overload may occur in cases with myocardial or renal damage
Sodium chloride and glucose	Aquapharm No.3 (Animalcare) sodium chloride 9 g and anhydrous glucose 50 g	Psittacines, raptors, and bustards	IV, SC, or PO	50 mL/kg/d for maintenance	For the treatment of dehydration to correct water and electrolyte depletion where the patient's carbohydrate store is considered to be depleted
Sodium lactate solution	Aquapharm 11 (Animalcare) sodium chloride 6.0 g, potassium chloride 0.4 g, calcium chloride dihydrate 0.27 g, and molar sodium lactate 28.9 g	Psittacines, raptors, and bustards	IV, SC, or PO	50 mL/kg/d for maintenance	Give to severely diseased patients with diarrhea, dehydration, and vomiting to combat metabolic acidosis. Sodium overload may occur in cases with myocardial and renal damage

Continued

Miscellaneous—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Sodium calciumedetate	Strong (Animalcare); Calcium Disodium Versenate (3M Pharmaceuticals)	Raptors	IV or IM	10-40 mg/kg bid for 5-10 d	Lead and other heavy metal poisoning such as copper, lead, and zinc. Dilution not required up to 100 mg/kg bid IM has been used in falcons without any deleterious effect.
		Waterfowl	IV or IM	10-40 mg/kg bid for 5-10 d	
		Psittacines	IV or IM	20-40 mg/kg bid for 5 d	
Soluble multivitamins		Pigeons	PO	1 g/L drinking water for 5-7 d or 1 d/wk	Support in infectious disease. Nutritional dermatitis
Soluble multivitamins	Soluvet (Vetafarm) raptors, and bustards	Psittacines	PO	4 g in 400 mL water 1 g in 100 mL food	Support in infectious diseases and stress
Spark	Spark carbohydrate and oral electrolyte supplement (Vetafarm)	Psittacines, raptors, and bustards	PO (water)	3 g/150 mL water	High-calorie electrolyte for use in birds that need extra energy and body salts. Use before birds are to be translocated and give for 3 d before and 2 d after any move
Stanozolol	Winstrol V Tablet, Injectable Solution 50 mg/mL	Most species	IM	25-50 mg/kg	Increases weight gain in anorectic cases Monitor patients with hepatic or renal problems May not achieve desired results
Silymarin		Most species	PO	50 to 75 mg/kg q12h	Adjunct therapy for liver disease
Thiamine	Thiamine compound tablets (Rhône Poulenc Rorer, Generic)	Raptors	PO	1-50 mg/kg sid for 7 d or indefinitely	For control of thiamine-responsive fits
Testosterone	Androject (Intervet, Henry Schein, Upjohn)	Psittacines	IM	8 mg/kg weekly as required	Use with great care. Usually contraindicated. May affect spermatogenesis
Vitamin A	Various injectable preparations	Raptors	IM	Maximum 20,000 IU/kg weekly	Hypovitaminosis A. To increase skin healing, e.g., in bumblefoot. Supplemental therapy for avian pox infections, sinusitis, and ophthalmic disorders
Vitamin A, D, E	Various injectable preparations	Psittacines	IM	0.1-0.2 mL (10,000-20,000 IU)/300 g, weekly as required	Useful in the treatment of vitamin A + D deficiencies, reproductive disorders, and bone healing
Vitamin B complex	Various injectable preparations	Raptors	IM	Sufficient to give 10-30 mg/kg of thiamine. Repeat control of thiamine-responsive fits weekly as required	Stimulate appetite. General health, neuromuscular disease, and hepatic disorders
		Psittacines	IM	To give 1-3 mg thiamine/kg every other day	Acute toxicity after administration of high doses of B6 has been reported in falcons. Doses of 10-20 mg/kg IM and 75-50 mg/kg PO produced acute hepatitis, splenitis, nephritis, and moderate-to-severe gastritis resulting in death of several gyrfalcons. Acute toxicity has also been reported in pigeons after oral administration of a human B complex preparation. The lethal dose was estimated at 90-100 mg/kg PO
Vitamin B_{12}	Cyanocobalamin (Butler)	Psittacines	IM	250-500 mcg/kg weekly	Indicated in anemias and convalescence. Has been reported to produce pink droppings in other species but this has not been observed in bustards
Vitamin E/ selenium	Dystosel (Upjohn); Seletoc (Schering-Plough)	Raptors	SC	0.05 mg selenium + 3.4 IU vitamin E. Repeat once after 72 hr	Vitamin E or selenium deficiency. For prevention and treatment of muscular weakness, capture myopathy. Given to birds before transportation during translocation procedures

Miscellaneous—cont'd

Generic Name	Trade Name	Species	Route	Dosage	Remarks
Vitamin E	Dystosel (Intervet); Seletoc (Schering-Plough)	Psittacines	IM	0.06 mg/kg weekly	
Vitamin K	Konakion (Roche, Butler, Phoenix, Vet-A-Mix)	Most species	IM	0.2-2.5 mg/kg daily as required	For hemorrhagic disorders and to prevent such problems when amprolium and sulfa drugs are administered or after long-term tetracycline treatment
Yeast Cell Derivatives	Preparation H Ointment (Whitehall Laboratories)	Most species	Topical	As needed	Stimulate epithelial healing, especially abrasions and lacerations

bid, Twice a day; BUN, blood urea nitrogen; CNS, central nervous system; CSF, cerebrospinal fluid; DEFRA, Department for Environment, Food and Rural Affairs; DMSO, Dimethyl sulfoxide; EDTA, ethylenediaminetetraacetic acid; GABA, γ-aminobutyric acid; HI, hemagglutination inhibiting; IC, intracardiac; IM, intramuscular; IO, intraosseus; IP, intraperitoneal; IT, intratracheal; IV, intravenous; MIC, minimum inhibitory concentration; PO, by mouth; qid, four times a day; SC, subcutaneous; sid, once a day; tid, thrice a day.

Sources:

Anonymous: *Compendium of data sheets for veterinary products 1995-1996*. National Office of Animal Health, 1995, Enfield, Middlesex.
Bailey TA: *A contribution to preventive medicine and therapeutics of avian captive breeding programmes with special reference to the houbara bustard (Chlamydotis undulata macqueenii)*. PhD Thesis, Durrell Institute of Conservation and Ecology, 1999, University of Kent, Canterbury.
Bailey TA: Aspergillosis: therapy and prevention in zoo animals with emphasis on birds, *Falco* 20:18–22, 2002.
Bailey TA, Sullivan T: Aerosol therapy using a novel disinfectant to treat upper and lower respiratory tract infections in birds, *Exotic DVM Magazine* 3:17, 2001.
Bailey TA, Samour JH, Sheen RS, Garner AG: Analysis of amoxicillin in houbara bustard plasma by HPLC after administration of a long-acting formulation, *J Vet Pharmacol Ther* 19:313–315, 1996.
Bailey TA, Sheen RS, Silvanose C, et al: Clinical pharmacology and pharmacokinetics of enrofloxacin after intravenous, intramuscular and oral administration in houbara bustard (*Chlamydotis undulata macqueenii*), *J Vet Pharmacol Ther* 21:288–297, 1998.
Bailey TA, Manvell R, Gough RE, et al: A review of Newcastle disease in bustards: presentation, diagnosis and control; results of vaccination trials, *Proceedings of the European Association of Avian Veterinarians*, Munich, 2001, pp 202–207.
Bonn, Flammer K, Aucoin DP, Whitt DA: Intramuscular and oral disposition of enrofloxacin in African grey parrots following single and multiple doses, *J Vet Pharmacol Ther* 14:359–366, 1991.
Carpenter JW, Mashima TY, Rupiper DJ: *Exotic animal formulary*, Philadelphia, PA, 2001, WB Saunders.
Clubb SL: Therapeutics. In: Harrison GJ, Harrison LR, editors: *Clinical avian medicine and surgery*, Philadelphia, PA, 1986, WB Saunders, pp 327–355.
Coles BH: *Avian medicine and surgery*, ed 2, Blackwell Science, 1997, Oxford, UK, pp 240–279.
Debuf Y: *Veterinary formulary*, London, UK, 1991, Pharmaceutical Press.
Di Somma A, Bailey TA: Aspergillosi: la malattie che sfida i veterinari aviari [Aspergillosis: the challenging disease], *Proceedings of SIVAE*, Rome, 2003.
Dorrestein GM: The pharmacokinetics of avian therapeutics. *Vet Clin North Am Small Anim Pract* 21:1241–1263, 1991.
Dorrestein GM: Antimicrobial drug use in pet birds. In: Prescott JF, Baggot JD, editors: *Antimicrobial therapy in veterinary medicine*, Ames, IA, 1993, Iowa State University Press, pp 490–506.
Dorrestein GM, Van Miert ASJAM: Pharmacotherapeutic aspects of medication of birds, *J Vet Pharmacol Ther* 11:33–44, 1988.
Dorrestein GM, Verburg E: Pharmacokinetics of enrofloxacin (Baytril) in homing pigeons after different administration routes, *Proceedings of the Fourth Congress of the European Association for Veterinary Pharmacology and Toxicology*, Budapest, 1988, p 172.
Flammer K: An overview of antifungal therapy in birds, *Proceedings of the Association of Avian Veterinarians*, Nashville, 1993, pp 1–4.
Forbes NA, Harcourt-Brown NH: Formularies. In: Forbes NA, Harcourt-Brown N, editors: *Manual of raptors, pigeons and waterfowl*, Cheltenham, 1996, British Small Animal Veterinary Association, pp 334–352.
Greth A, Gerlach H, Gerbermann H, et al: Pharmacokinetics of doxycycline after parenteral administration in the houbara bustard (*Chlamydotis undulata*), *Avian Dis* 37:31–36, 1993.
Heidenreich M: Clinical therapy. In: *Birds of prey medicine and management*, Oxford, 1997, Blackwell Science, pp 91–101.
Krautwald-Junghanns ME: Avian therapeutics. In: *Proceedings of the European Association of Avian Veterinarians*, Vienna, 1991, pp 30–39.
Lawrence K: Therapeutics. In: Price CJ, editor: *Manual of parrots, budgerigars and other psittacine birds*, British Small Animal Veterinary Association, 1988, Cheltenham, pp 175–176.
McDonald SE: Summary of medications for use in psittacine birds, *J Avian Med Surg* 3:124–127, 1989.
Martínez MR, Diaz MJ, Fernández-Cruz ML, et al: Pharmacokinetics of marbofloxacin in broiler chickens after intravenous administration, *J Vet Pharmacol Ther* 20(Suppl 1):197, 1997.
Michell AR: Small animal fluid therapy 1. Practice principles, *J Small Anim Pract* 35:559–565, 1994.
Michell AR: Small animal fluid therapy 2. Solutions and monitoring, *J Small Anim Pract* 35:613–619, 1994.
Michell AR: Oral rehydration for diarrhoea: symptomatic treatment or fundamental therapy, *J Comp Pathol* 118:175–193, 1998.
Naldo JL, Bailey TA, Samour JH: Trichomonosis: a review of clinical pathology and control in bustard collection. In: *Proceedings of the European Association of Zoo and Wildlife Veterinarians*, Paris, 2000, pp 347–352.

Continued

Ostrowski S, Dorrestein G, Burger L, et al: Cross-protection of an avian poxvirus isolated from houbara bustards, *Avian Dis* 40:762–769, 1996.
Peeters N, Viaene N, Devriest L: Acute toxicity of high doses of Vitamin B6 (Pyridoxine) in pigeons, *Vlaams Diergeneeskundig Tijdschrify* 46(4):268–269, 1977.
Plumb DC: *Veterinary drug handbook*, ed 4, Iowa State University Press, 2002, Ames, IA.
Pokras MA, Karas AM, Kirkwood JK, Sedgwick CJ: An introduction to allometric scaling and its uses in raptor medicine. In: Redig PT, Cooper JE, Remple JD, Hunter DB, editors: *Raptor biomedicine*, University of Minnesota Press, Minneapolis, MN, 1993, pp 211–224.
Pugh DM: Blood formation, coagulation and volume. In: Brander GC, Pugh DM, Bywater RJ, Jenkins WL, editors: *Veterinary applied pharmacology and therapeutics*, Baillière Tindall, London, UK, 1991, pp 166–186.
Redig P: Fluid therapy and acid-base balance in the critically ill avian patient. In: *Proceedings of the Association of Avian Veterinarians*, Boulder, 1984, pp 59–74.
Ritchie BW, Harrison GJ: Formulary. In: Ritchie BW, Harrison GJ, Harrison LR, editors: *Avian medicine: principles and application*, Wingers Publishing, 1994, Lake Worth, FL, pp 457–478.
Robertson EL: Farmaci antinematodici. In: Booth NH, McDonald LE, editors: *Farmacologia e Terapeutica Veterinaria Italia*, ed 6, Iowa State University Press, 1991, Ames, IA.
Rosskopf WJ, Woerpel RW: Practical avian therapeutics with dosages of commonly used medications, *Vet Clin North Am Small Anim Pract* 21:1265–1271, 1991.
Rupiper DJ, Carpenter JW, Mashima TY: Formulary. In: Olsen GH, Orosz SE, editors: *Manual of avian medicine*, St Louis, 2000, Mosby, pp 553–589.
Rupley AE: Formulary. *Manual of avian practice*, Philadelphia, 1997, WB Saunders, pp 502–518.
Samour J: Acute toxicity after administration of high doses of Vitamin B6 (Pyridoxine) in falcons, *Proceedings of the European College of Zoological Medicine Scientific Meeting*, Wiesbaden, Germany. 2013.
Sedgwick CJ: Allometric scaling and emergency care: the importance of body size. In: Fowler ME, editor: *Zoo and wildlife medicine: current therapy,* ed 3, Philadelphia, 1993, WB Saunders, pp 34–37.
Tully TN: Formulary. In: Altman RB, Clubb SL, Dorrestein GM, Quesenberry K, editors: *Avian medicine and surgery*, Philadelphia, PA, 1997, WB Saunders, pp 671–688.
Tully TN: Formulary. In: Coles B, editor: *Essentials of avian medicine and surgery*, ed 3, Hoboken, NJ, 2007, Wiley-Blackwell, pp 219–264.

ACKNOWLEDGMENTS

The authors are grateful to Dr. Paolo Zucca from the Laboratory of Animal Cognition and Comparative Neuroscience, Department of Psychology, University of Trieste, Italy for providing most of the information on antiparasitic drugs.

INDEX

Page numbers followed by "*f*" indicate figures, "*b*" indicate boxes, and "*t*" indicate tables.