FIVE MINUTE VETERINARY CONSULT

Jennifer E. Graham

BLACKWELL'S FIVE-MINUTE VETERINARY CONSULT: AVIAN

Includes client education handouts, algorithms, and procedure guides on a companion website

WILEY Blackwell

Blackwell's Five-Minute Veterinary Consult: Avian

Blackwell's Five-Minute Veterinary Consult

Avian

Jennifer E. Graham, DVM, DABVP (Avian/ECM), DACZM
Assistant Professor of Zoological Companion Animal Medicine,
Department of Clinical Sciences,
Tufts Cummings School of Veterinary Medicine,
North Grafton, Massachusetts, USA

WILEY Blackwell

Editorial offices: 1606 Golden Aspen Drive, Suites 103 and 104, Ames, Iowa 50010, USA
The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK
9600 Garsington Road, Oxford, OX4 2DQ, UK

Library of Congress Cataloging-in-Publication Data

Names: Graham, Jennifer E. (Jennifer Erin), 1974- , editor.
Title: Blackwell's five-minute veterinary consult. Avian / [edited by] Jennifer E. Graham.
Other titles: Five-minute veterinary consult. Avian | Avian
Description: Ames, Iowa : John Wiley and Sons, Inc., 2016. | Includes bibliographical references and index.
Identifiers: LCCN 2015039615 | ISBN 9781118934593 (cloth)
Subjects: LCSH: Birds–Diseases. | MESH: Bird Diseases. | Veterinary Medicine–methods.
Classification: LCC SF994 .B63 2016 | NLM SF 994 | DDC 636.5/0896–dc23
LC record available at http://lccn.loc.gov/2015039615

A catalogue record for this book is available from the British Library.

Wiley also publishes its books in a variety of electronic formats. Some content that appears in print may not be available in electronic books.

Set in 9/10pt GaramondPro by Aptara Inc., New Delhi, India

1 2016

This book is dedicated to my heroes: my dear 'ol dad, Lewis, and my sister, Amy.

My father had me convinced at one point that he was Dr. Bob, with a side job as a brain surgeon (this was a total lie). This resulted in me telling an entire busload of children about his craniotomy adventures on the ride home from school and making the bus driver wait outside the house so he could wave to them all. He turned bright red and didn't tell so many stories after that day as I recall.

Amy-sis—thanks for always being there. I am so lucky to have you as "my person".

PREFACE

As a veterinary student and new graduate, I remember reaching for my *Blackwell's Five-Minute Veterinary Consult: Canine and Feline* text on a regular basis. The concise and thorough coverage organized by topic was exactly what I needed to refresh memories from lectures and make sure I was not forgetting an important aspect of a case. Although I have since left canine and feline practice behind, I still find myself reaching for this invaluable text when collaborating with colleagues, consulting on a case, or if I am in need of a quick overview of a particular disease process. It probably comes as no surprise that I am also a fan of the *Blackwell Five-Minute Veterinary Consult: Ferret and Rabbit* text.

When Wiley contacted me about working on a Five-minute Veterinary Consult: Avian text, I had two thoughts. The first thought was surprise at the realization that this had not already been done. The second was the fact that it was a complete no-brainer, that I would love to be the one to head up such a worthy project. The other no-brainer was who I would be inviting to write chapters. I have been fortunate to get an amazing group of talented contributors. All are leaders in the field of avian medicine and eminently qualified to address the topics included in this text.

This book is divided into 123 topics covering a wide range of diseases and syndromes in avian patients, seven appendices and accompanying algorithms, and a companion website that includes client handouts and descriptions and pictures of common clinical procedures. The approach on topics is focused on practical clinical knowledge and organized to offer fast access to essential information.

A helpful aspect of the table of contents is multiple listings of topics based on common terminology. For example, the topic of "viral neoplasms" can also be found under the headings: Marek's disease, lymphoid leukosis, and reticuloendotheliosis. Topics have been chosen to encompass the majority of syndromes and diseases seen in avian practice. Species covered in the text include psittacine birds, passerines, poultry, raptors, ratites, and waterfowl. The template design of topic layout ensures quick access to information without the need to read the entire section—you can jump to any section on an as-needed basis. At the end of each section is a "see also" section which lists similar diseases or syndromes.

The appendices are broken down into user-friendly tables. The formulary, Appendix 1, includes drugs mentioned throughout the book and lists dosing recommendations along with indications for use. Hematology and biochemistry reference ranges are outlined in Appendix 2. Appendix 3 lists common laboratory tests available for avian species and laboratory contact information. Viral diseases are outlined in Appendix 4 with information supplied on species affected, common lesions, and transmission routes. Zoonotic diseases of concern and personal protective guidelines are listed in Appendix 5. Appendix 6 outlines common plant toxins along with systems affected and treatment recommendations. Appendix 7 includes clinical algorithms for 15 common clinical presentations ranging from non-specific signs like "sick bird syndrome" and to specific presentations like "feather damaging behavior" and "lameness".

The companion website will be a helpful resource for practitioners. The website includes client education handouts that can be downloaded and edited for distribution to clients. Common procedures with accompanying descriptions and pictures are also available on the website.

We are hopeful this text will be a useful addition to your library—whether you see birds occasionally or as a regular part of your practice caseload.

Jennifer E. Graham

Acknowledgments

There are many individuals whose contributions helped make this book happen. To Hugues Beaufrère, who helped with modification and improvement of the table of contents along with some fabulous topic summaries—thank you for always being willing to lend a hand and for sharing your brilliance. To Erika Cervasio—thanks so much for the extra help with contributions to the online procedures and helping create some last-minute topic summaries that were originally omitted. To Lauren Powers—your images for online reference material are much appreciated. George Messenger—even though you are supposedly "retired", all the more time for you to help with future endeavors—thanks for your willingness to help with this project. To all my amazing colleagues who have contributed to the topics in this text—thank you for the time and effort you dedicated to this book. To Nancy, Erica, Shalini, Gayle, and the rest of the wonderful team at Wiley—working with you is a pleasure. And last but not least—to my husband James—thank you for your continued support, encouragement, and attendance at all the "boring vet events"; may our love outlast even our tattoos.

CONTRIBUTORS

ANDREW D. BEAN, DVM, MPH, CPH
Veterinarian
Pet Care Veterinary Hospital
Virginia Beach, Virginia, USA

HUGUES BEAUFRÈRE, Dr.Med.Vet, PhD, ECZM (Avian), DABVP (Avian), DACZM
Service Chief, Avian and Exotics Service
Health Sciences Centre
Ontario Veterinary College
University of Guelph
Guelph, Ontario, Canada

JOAO BRANDAO, LMV, MS
Assistant Professor, Zoological Medicine Service
Department of Veterinary Clinical Sciences
Center for Veterinary Health Sciences
Oklahoma State University
Stillwater, Oklahoma, USA

JAMES W. CARPENTER, MS, DVM, DACZM
Professor, Zoological Medicine
Veterinary Medical Teaching Hospital
Kansas State University
Manhattan, Kansas, USA

ERIKA L. CERVASIO, DVM, DABVP (Avian)
North Paws Veterinary Center
Mansfield, Massachusetts, USA

SUE CHEN, DVM, DABVP (Avian)
Gulf Coast Avian & Exotics
Gulf Coast Veterinary Specialists
Houston, Texas, USA

LEIGH ANN CLAYTON, DVM, DABPV (Avian, Reptile-Amphibian)
Director Animal Health
National Aquarium
Baltimore, Maryland, USA

SUSAN L. CLUBB, DVM, DABVP (Avian)
Rainforest Clinic For Birds and Exotics, Inc.
Hurricane Aviaries, Inc.
Loxahatchee, Florida, USA

ROB L. COKE, DVM, DACZM, DABVP (Reptile & Amphibian), CVA
Senior Staff Veterinarian
San Antonio Zoo
San Antonio, Texas, USA

GREGORY J. COSTANZO, DVM
Stahl Exotic Animal Veterinary Services
Fairfax, Virginia, USA

LORENZO CROSTA, DVM, GP CERT (Exotic Animal Practice), PhD
FNOVI Accredited Veterinarian in Avian Medicine and Surgery and Zoo Animal Medicine and Surgery and Zoo Management
ECZM
Veterinari Montevecchia
Montevecchia (LC), Italy

JULIE DECUBELLIS, DVM, MS
Calgary Avian and Exotic Pet Clinic
Calgary, Alberta, Canada

MARION DESMARCHELIER, DMV, MSc, DACZM
Clinical Instructor
Zoological Medicine Service
Department of Clinical Sciences
Faculté de medicine vétérinaire
Université de Montréal
Saint-Hyacinthe, Quebec, Canada

RYAN DEVOE, DVM, MSpVM, DACZM, DABVP (Reptile & Amphibian)
Clinical Veterinarian
Department of Animal Health
Disney's Animal Kingdom
Bay Lake, Florida, USA

STEPHEN M. DYER, DVM, DABVP (Avian)
NorthPaws Veterinary Center
Greenville, Rhode Island, USA

THOMAS M. EDLING, DVM, MSpVM, MPH
Vice President, Veterinary Medicine
Petco Animal Supplies, Inc.
San Diego, California, USA

SHANNON FERRELL, DVM, DABVP (Avian), DACZM
Veterinarian Zoo de Granby 525
Granby, Quebec, Canada

SUSAN G. FRIEDMAN, PhD
Department of Psychology
Utah State University
Logan, Utah, USA

ALAN M. FUDGE, DVM, DABVP (Avian)
Mobile Veterinary Services, Birds & Fish
Greenville, South Carolina, USA

JENNIFER E. GRAHAM, DVM, DABVP (Avian/ECM), DACZM
Assistant Professor of Zoological Companion Animal Medicine
Department of Clinical Sciences
Cummings School of Veterinary Medicine
Tufts University
North Grafton, Massachusetts, USA

DAVID E. HANNON, DVM, DABVP (Avian)
Avian and Exotic Animal Veterinary Services
Memphis Veterinary Specialists
Memphis, Tennessee, USA

KENDAL E. HARR, DVM, MS, DACVP
Pathologist
Florida Veterinary Pathology Consultants Inc.
Bushnell, Florida, USA

LISA HARRENSTIEN, DVM, DACZM
Zoological Medicine Consultant
Portland, Oregon, USA

J. JILL HEATLEY, DVM, MS, DABVP (Avian, Reptile & Amphibian), DACZM
Associate Professor, Zoological Medicine
College of Veterinary Medicine
Texas A&M University
College Station, Texas, USA

CHRISTINE T. HIGBIE, DVM
Department of Clinical Sciences
College of Veterinary Medicine
Kansas State University
Manhattan, Kansas, USA

HEIDI L. HOEFER, DVM, DABVP (Avian)
Island Exotic Veterinary Care
Huntington Station, New York, USA

MICHAEL P. JONES, DVM, DABVP (Avian)
Associate Professor of Avian & Zoological Medicine
Department of Small Animal Clinical Sciences
College of Veterinary Medicine
University of Tennessee
Knoxville, Tennessee, USA

ERIC KLAPHAKE, DVM, DABVP (Avian), DACZM, DABVP (Reptile-Amphibian)
Associate Veterinarian
Cheyenne Mountain Zoo
Colorado Springs, Colorado, USA

LAURA M. KLEINSCHMIDT, BS, DVM
Department of Small Animal Clinical Sciences, Zoological Medicine Service
College of Veterinary Medicine and Biomedical Science, Texas A&M University
College Station, Texas, USA

ISABELLE LANGLOIS, DMV, DABVP (Avian)
Clinical Instructor, Zoological Medicine Service
Department of Clinical Sciences
Faculty of Veterinary Medicine
Université de Montréal
Saint-Hyacinthe, Québec, Canada

DELPHINE LANIESSE, Dr. Med. Vet, IPSAV
Ontario Veterinary College
University of Guelph
Guelph, Ontario, Canada

ANGELA M. LENNOX, DVM, DABVP (Avian, Exotic Companion Mammal)
Avian and Exotic Animal Clinic
Indianapolis, Indiana, USA

SHACHAR MALKA, DVM, DABVP (Avian)
The Avian and Exotics Service
The Humane Society of New York
New York, New York, USA

RUTH MARRION, DVM, DACVO, PhD
Ophthalmologist
Bulger Veterinary Hospital
North Andover, Massachusetts, USA

KEMBA L. MARSHALL, DVM, DABVP (Avian)
Director of Merchandising Pet Quality and Education
PetSmart SSG
Phoenix, Arizona, USA

GEORGE MESSENGER, DVM, DABVP (Avian)
Concord, New Hampshire, USA

MAUREEN MURRAY, DVM, DABVP (Avian)
Clinical Assistant Professor
Wildlife Clinic
Department of Infectious Disease and Global Health
Cummings School of Veterinary Medicine
Tufts University
North Grafton, Massachusetts, USA

HEIDE M. NEWTON, DVM, DACVD
Southern Arizona Veterinary Specialty & Emergency Center
Dermatology for Animals
Tucson, Arizona, USA

GEOFFREY P. OLSEN, DVM, DABVP (Avian)
Associate
Medical Center for Birds
Oakley, California, USA

DAVID N. PHALEN, DVM, PhD, DABVP (Avian)
Associate Professor
Faculty of Veterinary Science
University of Sydney
Sydney, New South Wales, Australia

ANTHONY A. PILNY, DVM, DABVP (Avian)
Medical Director
The Center for Avian and Exotic Medicine
New York City, New York, USA

CHRISTAL POLLOCK, DVM, DABVP (Avian)
Lafeber Company Veterinary Consultant
Cornell, Illinois, USA

LAUREN V. POWERS, DVM, DABVP (Avian/ECM)
Service Head, Avian and Exotic Pet Service
Carolina Veterinary Specialists
Adjunct Assistant Professor
School of Veterinary Medicine
North Carolina State University
North Carolina, USA

DRURY R. REAVILL, DVM, DABVP (Avian/Reptile & Amphibian Practice), DACVP
Zoo/Exotic Pathology Service
Carmichael, California, USA

GREGORY RICH, DVM, BS Medical Technology
Owner
West Esplanade Veterinary Clinic
Metairie, Louisiana, USA

SHANNON M. RIGGS, DVM
Director of Animal Care
Pacific Wildlife Care
Morro Bay, California, USA

VANESSA ROLFE, DVM, ABVP (Avian)
The Bird & Exotic Hospital, Inc.
Greenacres, Florida, USA

KAREN ROSENTHAL, DVM, MS
Dean, School of Veterinary Medicine
St. Matthew's University
Grand Cayman, Cayman Islands, British West Indies

PETRA SCHNITZER, DVM, GP Cert (Exotic Animal Practice)
ECZM Resident (Avian)
Veterinari Montevecchia
Montevecchia (LC), Italy

NICO J. SCHOEMAKER, DVM, PhD, DECZM (Small mammal, Avian), DABVP (Avian)
Associate Professor
Division of Zoological Medicine
Department of Clinical Sciences of Companion Animals
Faculty of Veterinary Medicine
Utrecht University
Utrecht, The Netherlands

ELISABETH SIMONE-FREILICHER, DVM, DABVP (Avian)
Senior Clinician
Avian and Exotic Animal Medicine Department
Angell Animal Medical Center
Boston, Massachusetts, USA

KRISTIN M. SINCLAIR, DVM, DABVP (Avian)
Staff Veterinarian
Kensington Bird and Animal Hospital
Kensington, Connecticut, USA

KURT K. SLADKY, MS, DVM, DACZM, DECZM (Herpetology)
Clinical Associate Professor
Zoological Medicine
Department of Surgical Sciences
School of Veterinary Medicine
University of Wisconsin
Madison, Wisconsin, USA

DALE A. SMITH, DVM, DVSc
Professor
Department of Pathobiology
Ontario Veterinary College
University of Guelph
Guelph, Ontario, Canada

MARCY J. SOUZA, DVM, MPH, DABVP (Avian), DACVPM
Assistant Professor
Director of Veterinary Public Health
Department of Biomedical & Diagnostic Sciences
College of Veterinary Medicine
University of Tennessee
Knoxville, Tennessee, USA

BRIAN SPEER, DVM, DABVP (Avian), DECZM (Avian)
Medical Center for Birds
Oakley, California, USA

JONATHAN STOCKMAN, DVM, DACVN
Senior Research Scientist
Effem Foods Co., Ltd.
Wyong, New South Wales, Australia

ANNELIESE STRUNK, DVM, DABVP (Avian)
Medical Director
The Center for Bird and Exotic Animal Medicine
Bothell, Washington, USA

JOHN H. TEGZES, MA, VMD, DABVT
Professor of Toxicology
College of Veterinary Medicine
Western University of Health Sciences
Pomona, California, USA

FLORINA S. TSENG, DVM
Associate Professor
Department of Infectious Disease and Global Health
Director, Tufts Wildlife Clinic
Cummings School of Veterinary Medicine
Tufts University
North Grafton, Massachusetts, USA

YVONNE VAN ZEELAND, DVM, MVR, PhD, DECZM (Avian, Small Mammal)
Associate Professor
Division of Zoological Medicine
Department of Clinical Sciences of Companion Animals
Faculty of Veterinary Medicine
Utrecht University
Utrecht, The Netherlands

KENNETH R. WELLE, DVM, DABVP (Avian)
College of Veterinary Medicine
University of Illinois
Urbana, Illinois, USA

AMY B. WORELL, DVM, ABVP (Avian)
Avian Veterinary Services
Wet Hills, California, USA

NICOLE R. WYRE, DVM, DABVP (Avian)
Attending Clinician – Exotic Companion Animal Medicine and Surgery
Matthew J. Ryan Veterinary Hospital
University of Pennsylvania
Philadelphia, Pennsylvania, USA

ASHLEY ZEHNDER, DVM, PhD, DABVP (Avian)
Postdoctoral Fellow, Program in Epithelial Biology
Stanford University
Stanford, California, USA

About the Companion Website

This book is accompanied by a companion website:

www.fiveminutevet.com/avian

The website includes:

- Client Education Handouts
- Procedures
- Algorithms

The password for the companion website is the first word on page 72 (first word in the bulleted list at the start of the page). Please use all lowercase.

Client Education Handouts

Airborne Toxins
Angel Wing
Arthritis
Aspergillosis
Atherosclerosis
Avocado/Plant Toxins
Cardiac Disease
Chlamydiosis
Chronic Egg Laying
Cloacal Diseases
Cystic Ovarian Disease
Feather Damaging Behavior
Heavy Metal Toxicity
Hypocalcemia (and Hypomagnesemia)
Liver Disease
Macrorhabdosis (Avian Gastric Yeast)
Nutritional Imbalances
Obesity
Ovarian Neoplasia
Overgrown Beak and Nails
Pododermatitis
Polyomavirus Infection in Psittacines
Problem Behavior: Ten Things Your Parrot Wants You to Know about Behavior
Problem Behavior: Top Ten List of Behavior Tips
Proventricular Dilatation Disease
Regurgitation and Vomiting
Renal Disease
Rhinitis and Sinusitis
Sick Bird Syndrome
Trauma

Procedures

Air Sac Cannula
Beak and Nail Trimming
Blood Transfusion
Bone Marrow Aspiration
Choanal Swab
Cloacal Swab
Celiocentesis
Conjunctival Swab
Deslorelin Implant
E-Collars
Fecal Wet Mount and Gram's Stain
Figure-of-Eight Bandage
Handling and Restraint
Indirect Blood Pressure Monitoring
Ingluvial Gavage
Intramuscular Injection
Intraosseous Catheter
Intravenous Catheter
Microchip Placement
Nasal Flush
Oral Medications
Sinus Aspiration
Subcutaneous Injection
Tracheal Swab or Wash
Venipuncture
Ventplasty
Wing Trimming

Contents

Client education handouts are available at www.fiveminutevet.com/avian for you to download and use in practice

Client education handouts are available at www.fiveminutevet.com/avian for you to download and use in practice

Client education handouts are available at www.fiveminutevet.com/avian for you to download and use in practice

Client education handouts are available at www.fiveminutevet.com/avian for you to download and use in practice

ADENOVIRUSES

BASICS

DEFINITION

Adenoviruses are double-stranded nonenveloped DNA viruses. Specific adenoviruses are known to infect and cause disease in passerine birds, psittacine birds, pigeons (aka rock doves), falcons, hawks and owls, and gallinaceous birds.

PATHOPHYSIOLOGY

• Not all adenovirus infections result in disease. • When they do, most cause a systemic infection and may cause considerable morbidity and mortality. • Diseased birds that survive and subclinically infected birds may remain infected for life and be a persistent source of infection. • The Egg Drop Syndrome Virus in chickens replicates extensively in the oviduct causing abnormalities in egg shells and the production of unshelled eggs.

SYSTEMS AFFECTED

• Gastrointestinal—falcons, finches, hawks, owls, pigeons, psittacine birds, turkeys. • Hemic/Lymphatic/Immune—falcons, finches, pheasants, pigeon, psittacine birds, owls, pigeon, turkeys. • Hepatobiliary: Necrosis—pigeons, psittacine birds, falcons, hawks, owls. • Renal/Urologic—finches, pigeons, psittacine birds. • Reproductive—chickens. • Respiratory—pheasants. • Respiratory—quail.

GENETICS

• At least one falcon adenovirus is believed to subclinically infect peregrine falcons and is more likely to cause disease in other species. • The adenoviruses causing Marbled Spleen Disease in pheasants and Hemorrhagic Enteritis in turkeys are asymptomatically carried by waterfowl. • It is likely that many outbreaks of adenoviruses in mixed collections of birds are caused by cross species infection.

INCIDENCE/PREVALENCE

• Falcons: Rare outbreaks have been described. Prevalence of infection in peregrine falcons may be high. • Hawks and owls: Two outbreaks have been described. Prevalence is unknown. • Pigeons: Outbreaks occur sporadically, prevalence is unknown, but subclinical infections are likely to be common. • Psittacine birds: Prevalence is variable. There have been extensive outbreaks in Europe in Budgerigars. Individual infections and outbreaks in other psittacine birds are sporadic. • Chickens: Prevalence is variable, but can be high. • Turkeys: Prevalence is variable, but can be high. • Pheasants: Prevalence is variable. • Quail: High prevalence of infection.

GEOGRAPHIC DISTRIBUTION

• Finches: Described in North America. • Falcons: Described in North America. • Hawks and owls: Described in the United Kingdom. • Pigeons: Worldwide. • Psittacine birds: Outbreaks have occurred on multiple continents, not all adenoviruses have been adequately characterized. The distribution of each adenovirus therefore is not fully known. • Chickens: Worldwide, but not in North America. • Turkeys: Worldwide. • Pheasants: Worldwide. • Quail: Worldwide.

SIGNALMENT

• Finches: Adult finches, multiple species, both sexes. • Falcons: Nestling northern aplomado falcon, peregrine falcon, Taita falcon, and orange-breasted falcon, and adult American kestrel, both sexes. • Hawks and owls: Harris hawk, Bengal eagle owl, Verreaux's eagle owl, both sexes various ages. • Pigeons: Less than one year old, both sexes. • Psittacine birds: Most common in budgerigars, lovebirds and Poicephalus species; occurs sporadically in other parrot species. • Chickens: Laying hens. • Turkeys: Growing birds of both sexes 6–12 weeks old. • Pheasants: Three to eight months old, both sexes. • Quail: One to six weeks old, both sexes.

SIGNS

Historical Findings

• Finches: Unexpected deaths in a flock. • Falcons: Death after a short duration of nonspecific signs. • Hawks and owls: Death without premonitory signs or a short duration of nonspecific signs. • Pigeons: • Type 1: Vomiting, watery diarrhea and depression, rapid spread through the loft, increased mortality; • Type 2: Multiple unexpected deaths. • Psittacine birds: Unexpected mortality in nestling parrots. • Chickens: Sudden drop in egg production, abnormally colored eggs, shell-less eggs. • Turkeys: Sudden onset of hemorrhagic enteritis and depression. • Pheasants: Dyspnea and death. • Quail: Sudden and dramatic increase in mortality, nonspecific signs of illness, increased respiratory effort and increased respiratory sounds.

Physical Examination Findings

• Finches: N/A • Falcons: N/A • Hawks and owls: Birds die before they can be presented for examination. • Pigeons: ◦ Type 1: Vomiting, watery diarrhea, depression, weight loss; ◦ Type 2: N/A • Psittacine birds: N/A • Chickens: Abnormally colored eggs, shell-less eggs. Chickens appear normal. • Turkeys: Bloody diarrhea and depression. • Pheasants: Dyspnea, cyanosis. • Quail: Nasal discharge, open-mouthed breathing, respiratory sounds.

CAUSES

Three genera of adenoviruses (*Aviadenovirus*, *Siadenovirus*, and *Atadenovirus*) have been shown to cause disease in birds. The signs associated with infection depend on the organ targeted by the virus and the host's immune response.

RISK FACTORS

• Failure to quarantine new birds. • Housing multiple species together in the same collection. • High stocking densities. • Pheasants, turkeys, chickens: exposure to waterfowl. • Pigeons: Concurrent infection with pigeon circovirus. • Quail: Exposure to infected birds.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

All species: Other systemic viral infections, septicemia, gross management errors.

CBC/BIOCHEMISTRY/URINALYSIS

In birds experiencing hepatitis, elevations in the aspartate aminotransferase are expected.

OTHER LABORATORY TESTS

• Falcons: A virus neutralization assay has been developed that can detect serological evidence of virus infection. • Chickens: Antibodies can be detected by hemagglutination inhibition and enzyme-linked immunoassays.

IMAGING

Pigeons: Hepatomegaly and splenomegaly would be expected.

DIAGNOSTIC PROCEDURES

N/A

PATHOLOGIC FINDINGS

• Finches: Grossly, liver and spleen enlargement. Microscopically, multiple round-to-irregular pale tan (necrotic) foci. Hepatic, splenic, and intestinal mucosal necrosis with varying numbers of large intranuclear basophilic to amphophilic inclusion bodies. • Falcons: Grossly, liver and spleen enlargement. Microscopically, hepatic and splenic necrosis with a mild-to-moderate lymphoplasmacytic inflammatory response. Varying numbers of large intranuclear basophilic to amphophilic inclusion bodies are present. • Hawks and owls: Grossly, liver and spleen enlargement. Microscopically, hepatic necrosis and mild-to-moderate inflammatory response, splenic necrosis, and proventricular and ventricular and necrosis resulting in ulceration. Varying numbers of large intranuclear basophilic to amphophilic inclusion bodies are present in all affected tissues. • Pigeons: ◦ Type 1: Grossly, fibrinous and hemorrhagic enteritis, variable liver enlargement with necrotic foci. Microscopically, villus atrophy of the duodenum, characteristic inclusion bodies are found in intestinal epithelial cells. Hepatic necrosis may occur, but it is infrequent. Inclusion bodies are infrequently found in the liver ◦ Type 2: Grossly, hepatic and possibly splenic enlargement are seen. There may be

A

ADENOVIRUSES (CONTINUED)

multifocal discoloration of the liver. Microscopically there is a moderate to massive necrosis of the liver with intranuclear eosinophilic inclusion bodies. • Psittacine birds: Lesions depend on the virus and species of bird. Grossly there may be evidence of one or more of: conjunctivitis, hepatitis, pancreatitis, enteritis, and splenic enlargement. The virus causes necrosis of the affected tissues, which will be accompanied by inflammation depending on how long the bird lives after the lesions develop. Intranuclear inclusions are generally common, but may be difficult to find. Inclusions in the tubular epithelial cells of the kidneys may be incidental findings in birds dying of other causes. • Chickens: Grossly, inactive ovaries and atrophied oviducts. Microscopically, severe chronic active inflammation of the shell gland with intranuclear inclusion bodies in the epithelial cells. Microscopically there is expansion of the histiocytic population surrounding the sheathed arteries of the spleen with lymphoid necrosis with pannuclear inclusion bodies. Digestive tract lesions include epithelial sloughing, hemorrhage within the villi and the submucosa, a variable degree of inflammation which can include heterophils and mononuclear cells and the presence of intranuclear inclusion bodies. Lesions are most severe in the duodenum. • Turkeys: Grossly, well muscled but pale, may have still been eating, hemorrhage into the intestine, hepatomegaly and splenomegaly. Lesions resemble those seen in the chicken, but do not involve the digestive tract. • Pheasants: Pulmonary edema and enlarged mottled spleens. Lesions resemble those seen in the chicken, but do not involve the digestive tract. • Quail: Exudate in the nasal passages and in the trachea with tracheal mucosal thickening. Exudate may extend into the mainstem bronchi. Microscopically there is necrosis and sloughing of the tracheal epithelium and the presence of intranuclear inclusion bodies and nuclear enlargement. There will be varying degrees of inflammation, which may be complicated by secondary bacterial infections. Multifocal hepatic necrosis may also occur.

TREATMENT

NURSING CARE

• Pigeons: ◦ Type 1: Supportive care with fluids, supplemental heat and assist feeding of easily digested food. Broad spectrum antibiotics to prevent secondary *E. coli* enteritis and sepsis. • Quail: Supportive care. • All other species: N/A

ACTIVITY

Pigeons who survive Type 1 infections may take months to return to racing condition.

DIET

N/A

CLIENT EDUCATION

N/A

SURGICAL CONSIDERATIONS

N/A

MEDICATIONS

DRUG(S) OF CHOICE

N/A

CONTRAINDICATIONS

N/A

PRECAUTIONS

N/A

POSSIBLE INTERACTIONS

N/A

ALTERNATIVE DRUGS

N/A

FOLLOW-UP

PATIENT MONITORING

N/A

PREVENTION/AVOIDANCE

• Falcons: Do not raise other species of falcons with peregrine falcons. • Chickens, turkeys, pheasants: Avoid contact with waterfowl. • Chickens: Disease has been eradicated from laying stock. Infection is prevented by strict quarantine and hygiene methods. Inactivated vaccines have been developed and used effectively. • Turkeys: Vaccination by water administration. • Quail: Strict biosecurity measures.

POSSIBLE COMPLICATIONS

Pigeons, turkeys: Secondary *E. coli* infections.

EXPECTED COURSE AND PROGNOSIS

• Finches: Diseased birds die, low level or sporadic mortality. • Falcons: Most of the diseased birds will die. • Hawks and owls: The only known infected birds died. • Pigeons: ◦ Type 1: High levels of morbidity (up to 100%), low mortality unless secondary *E. coli* infections occur. ◦ Type 2: Sporadic mortality, most birds that develop the disease die. • Psittacine birds: Birds with disease die. It is likely that there are many subclinically infected birds. Nestling deaths may occur in subsequent clutches. • Chickens: A 10–40% reduction in egg production. • Turkeys: Average mortality of 10–15%, but may be higher. Secondary *E.coli* infections may increase the morbidity and mortality. • Pheasants: Flock mortality ranges from 2 to 15%. • Quail: Mortality rates may exceed 50% of susceptible birds.

MISCELLANEOUS

ASSOCIATED CONDITIONS

N/A

AGE-RELATED FACTORS

N/A

ZOONOTIC POTENTIAL

N/A

FERTILITY/BREEDING

N/A

SYNONYMS

N/A

SEE ALSO

Appendix 3: Laboratory Testing
Colibacillosis
Herpesviruses
Liver disease
Viral disease

ABBREVIATIONS

N/A

INTERNET RESOURCES

N/A

Suggested Reading

Marlier, D., Vindevogel, H. (2006). Viral infections in pigeons. *The Veterinary Journal*, **172**:40–51.

Oaks, J.L., Schrenzel, M., Rideout, B., Sandfort, C. (2005). Isolation and epidemiology of Falcon Adenovirus. *Journal of Clinical Microbiology*, **43**:3414–3420.

Saif, Y.M. (ed.) (2008). *Diseases of Poultry*, 12th edn. Oxford, UK: Blackwell Publishing.

Schmidt, R., Reavell, D., Phalen, D.N. (2003). *Pathology of Exotic Birds*. Ames, IA: Iowa State University Press.

Zsivanovits, P., Monks, D.J., Forbes, N.A., Ursu, K., Raue, R., Benko, M. (2006). Presumptive identification of a novel adenovirus in a Harris hawk (*Parabuteo unicinctus*), a Bengal eagle owl (*Bubo bengalensis*), and a Verreaux's eagle owl (*Bubo lacteus*). *Journal of Avian Medicine and Surgery*, **20**:105–112.

Author David N. Phalen, DVM, PhD, DABVP (Avian)

BASICS

DEFINITION
Airborne toxins are defined as particles or chemicals that are inspired and cause damage to various tissues of the body.

PATHOPHYSIOLOGY
The avian respiratory tract is particularly sensitive to airborne toxins because of specific anatomic and physiologic features that allow them to absorb oxygen more efficiently than can mammals. These include a cross-current flow of air and blood that allows the potential for blood oxygen levels to be higher than the oxygen levels in the expired breath. With this ability also comes the risk of absorbing higher amounts of toxins from the air, causing them to reach toxic levels sooner than would mammals.

SYSTEMS AFFECTED
• Respiratory system—direct exposure to the toxin. • Nervous system—secondary to hypoxia. • Cardiovascular system—secondary to a compromised respiratory system. • Ocular and upper gastrointestinal—inflammation and irritation.

GENETICS
N/A

INCIDENC/PREVALENCE
N/A

GEOGRAPHIC DISTRIBUTION
N/A

SIGNALMENT
• No sex or age predilections have been described. • PTFE—smaller birds like budgerigars may be more susceptible than larger birds. • COPD (hypersensitivity syndrome)—macaws are more susceptible than other species.

SIGNS
• Increased respiratory effort, open mouth breathing, exercise intolerance, cyanosis of facial skin, depression, ataxia, weakness, tail bobbing. • CO toxicity—cherry red mm. • Acute death or coma. • Weight loss, sneezing and coughing may occur with COPD of macaws. • The onset clinical signs of acute smoke inhalation may be delayed several hours after the exposure.

Historical Findings
• Recent toxin exposure. • Acute death or coma may occur after PTFE or carbon monoxide poisoning. • Sneezing and nasal discharge. • Smoking habit of the owner. • Multiple species of birds housed nearby (COPD).

Physical Examination Findings
• Open mouth breathing. • Cyanosis. • Sneezing and/or coughing. • Increased respiratory effort. • Ataxia, incoordination. • Cherry red mm. • Dyspnea. • Lethargy, depression. • Nasal discharge on nares and feathers of the face. • Weight loss.

CAUSES
Many types of toxins may be encountered and include the following:
• PTFE found on the surface of nonstick cookware, irons and ironing boards, heat lamps and self-cleaning ovens produce acidic fluorinated gases and particles. • Feather dander and dust from powder-down-producing birds like cockatoos (*Cacatua* spp), cockatiels (*Nymphicus hollandicus*), and African grey parrots (*Psittacus erithacus*) that can cause hypersensitivity reactions known as COPD of macaws or macaw hypersensitivity syndrome. • Smoke—solid or liquid material released into the air by pyrolysis (combustion). • CO, CO_2. • Nicotine, butadiene and other chemicals released in cigarette smoke. • Many other airborne toxins can have variable effects on birds, including air fresheners, scented candles, aerosols, methane, gasoline fumes, glues, paint fumes, self-cleaning ovens, solvents, bleach, ammonia, propellants and grooming products (nail polish, hair products).

RISK FACTORS
• Presence and use of nonstick cookware or other source of PTFE. • Presence of powder-down-producing birds in immediate environment of a macaw. • Cigarette smoking by the owner. • Housework involving painting or cleaning with aerosol producing chemicals. • Recent fire or other event releasing smoke into the environment.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
• Respiratory compromise caused by trauma and secondary air sac rupture, bacterial, fungal or viral infections, neoplasia, ascites or hypovitaminosis A with secondary sinusitis. • Primary heart disease, artherosclerosis causing left heart failure, congenital heart disease. • Avocado toxicity. • Ataxia and weakness secondary to other neurologic disease (see neurologic conditions), metabolic derangements or systemic disease.

CBC/BIOCHEMISTRY/URINALYSIS
• Hemogram—in most cases, the hemogram will not show any consistent changes, except with polycythemia of COPD. The PCV can be as high as 80%. • Biochemistry profile—varied based on the systems affected.

IMAGING
• Radiographs may be useful in ruling out causes of respiratory disease and to evaluate the heart and lungs for secondary complications. Radiographic changes are often not apparent until the disease is advanced. • COPD–Often unremarkable. Occasionally right sided heart failure is seen due to chronic polycythemia. • CT scan may show smaller lesions not readily identifiable on radiographs.

DIAGNOSTIC PROCEDURES
Coelomic endoscopic examination and lung biopsy may reveal consistent histologic changes associated with damage to the lungs caused by airborne toxin (see pathologic findings). It may also help elucidate the presence of other secondary diseases such as aspergillosis or bacterial infections that may require specific treatment.

PATHOLOGIC FINDINGS
• PTFE toxicity—Grossly, red, wet lungs, eosinophilic fluid filled bronchi, and multifocal to confluent hemorrhage. Microscopic changes include air capillary collapse, congestion, hemorrhage and edema. • Chronic smoke inhalation may cause tertiary bronchi obliterans. • COPD of macaws—Grossly, firm and "rubbery" lungs. Microscopic changes include eosinophilic infiltration of the interstitium, proliferative fibrous connective tissue, and a mixed cellular infiltrate. Tertiary bronchi may be obstructed due to hypertrophy of smooth muscle. These lesions are usually well advanced by the time polycythemia has occurred. • Artherosclerotic plaques may results from chronic exposure to butadiene in cigarette smoke.

TREATMENT

APPROPRIATE HEALTH CARE
• Inpatient intensive care management is often required. • Administer bronchodilators and antianxiety analgesic, then place in oxygen. • Administer diuretics if heart failure is present and antimicrobials for potential secondary infections.

NURSING CARE
• Oxygen therapy—78–85% O_2 at a flow rate of 5 L/min. • HEPA filtration. • Fluid therapy to maintain hydration or correct dehydration

ACTIVITY
• Acute—Exercise restriction until symptoms have resolved. • Chronic—Lifelong exercise restriction due to permanent respiratory system damage.

DIET
Ingluvial gavage for anorectic patients.

CLIENT EDUCATION
• Prognosis varies based on the level of exposure and chronicity of disease. • Educate owners on the sources of airborne toxins and their role in removing these toxins from the environment. • Separate macaws from powder-down-producing bird species. HEPA filtration can be helpful.

A

AIRBORNE TOXINS (CONTINUED)

SURGICAL CONSIDERATIONS
Caution in patients with chronic respiratory system damage.

MEDICATIONS

DRUGS OF CHOICE
• Antianxiety analgesic—Butorphanol at 0.5–2 mg/kg IM. • Terbutaline 0.01 mg/kg IM q6–12h or 0.1 mg/kg PO q12–24h. • Eye ointment if ocular irritation. • Nonsteroidal anti-inflammatories (NSAIDs)–Meloxicam at 0.5 mg/kg PO q12–24h. • Short acting corticosteroids—Use is controversial; but some will use for smoke inhalation and COPD: ◦ dexamethasone may be considered at a dose of 0.2–1.0 mg/kg IM once or q12–24h; ◦ dexamethasone sodium phosphate at 2 mg/kg once or q6–12h during the acute phase.

CONTRAINDICATIONS
Housing New World and Old World species in the same space without adequate ventilation/filtration.

PRECAUTIONS
Use caution if using corticosteroids in birds; consider concurrent antibiotic and antifungal therapy.

POSSIBLE INTERACTIONS
N/A

ALTERNATIVE DRUGS
• Midazolam: 0.5–1.0 mg/kg IM may be used to reduce anxiety if butorphanol is not sufficient. • Other bronchodilators include theophylline or aminophylline. These may be less effective at bronchodilation in birds, but clinical improvement has been noted with their use: ◦ theophylline: 2 mg/kg PO q12h; ◦ aminophylline: 10 mg/kg IV q3h, 4 mg/kg IM q12h, or 5 mg/kg PO q12h; ◦ albuterol nebulization: 2.5 mg in 3 cc saline q4–6h during acute clinical signs.

FOLLOW-UP

PATIENT MONITORING
• COPD—frequent monitoring of PCV to assess treatment effectiveness. • Radiographs to evaluate lungs and air sacs, and heart size and to check for the presence of atherosclerotic plaques.

PREVENTION/AVOIDANCE
Airborne toxicosis a complication of captivity. Elimination of the potential toxins before exposure is often possible and carries the best prognosis.

POSSIBLE COMPLICATIONS
Heart failure can result if polycythemia or pulmonary fibrosis is significant.

EXPECTED COURSE AND PROGNOSIS
• In the case of exposure to PTFE and clinical signs are present, the prognosis is usually very poor. • In the case of COPD, the condition can be improved with medication, HEPA filtration, and elimination of allergens from the environment, but even with good control, the condition will often shorten the normal lifespan of the patient.

MISCELLANEOUS

ASSOCIATED CONDITIONS
N/A

AGE-RELATED FACTORS
N/A

ZOONOTIC POTENTIAL
N/A

FERTILITY/BREEDING
Birds with COPD may have decreased breeding success.

SYNONYMS
N/A

SEE ALSO
Air sac rupture
Appendix 6: Common Avian Toxins
Aspergillosis
Avocado/plant toxins
Hemorrhage
Pneumonia
Respiratory distress
Tracheal/syringeal diseases

ABBREVIATIONS
PCV—packed cell volume
COPD—chronic obstructive pulmonary disease
PTFE—polytetrafluoroethylene
CO—carbon monoxide
CO_2—carbon dioxide
MM—mucus membranes
O_2—oxygen
HEPA—high-efficiency particulate air

INTERNET RESOURCES
N/A

Suggested Reading

Lichtenberger, M. (2006). Emergency case approach to hypotension, hypertension, and acute respiratory distress. *Proceedings of the Association of Avian Veterinarians Annual Conference*, San Antonio, TX, pp. 281–290.

Lightfoot, T.L., Yeager, J.M. (2008). Pet bird toxicity and related environmental concerns. *Vetinary Clinics of North America. Exotic Animal Practice*, **11**(2):229–259.

Orosz, S.E., Lichtenberger, M. (2011). Avian respiratory distress: Etiology, diagnosis and treatment. *Vetinary Clinics of North America. Exotic Animal Practice*, **14**(2):241–255.

Phalen, D.N. (2000). Respiratory medicine in cage and aviary birds. *Vetinary Clinics of North America. Exotic Animal Practice*, **3**(2):423–452

Schmidt, R.E. (2013). The Avian Respiratory System. *Proceedings of the Western Veterinary Conference*, Las Vegas, NV.

Author Stephen M. Dyer, DVM, DABVP (Avian)

Acknowledgement Erika L. Cervasio, DVM, DABVP (Avian)

Client Education Handout available online

AIR SAC MITES

BASICS

DEFINITION
Infection with mites (including *Sternostoma tracheacolum* and less commonly *Cytodites nudus, Ptilonyssus spp*.) in the upper and lower respiratory systems (including nasal passages, trachea, smaller air passages, and air sacs). Some mite species (especially *Cytodites*) have also invaded other visceral areas including the coelom.

PATHOPHYSIOLOGY
Mites transmitted from infected birds travel throughout the respiratory system, restricting air flow by being present within narrowed spaces as well as causing inflammation and increased mucous production.

SYSTEMS AFFECTED
• Respiratory (because of a foreign body response, inflammation, and increased fluid/mucous production). • Behavioral (bird's reaction to the respiratory system being affected).

GENETICS
None known other than species predilections.

INCIDENCE/PREVALENCE
Common.

GEOGRAPHIC DISTRIBUTION
Worldwide both in captive and wild individuals.

SIGNALMENT
• **Species:** Finches (especially Australian species including Gouldian finches), canaries, pigeons, small psittacine birds (budgerigars and cockatiels), poultry and waterfowl. Society finches may be resistant to infection. • **Mean age and range:** N/A • **Predominant sex:** N/A

SIGNS
Historical Findings
• No signs may have been noted by the owner. • Vocalization changes (cessation or tonal change of singing, clicking sounds). • Nonspecific signs of illness (lethargy, fluffed feathers, reduced appetite), • Head shaking and frequent swallowing, coughing or sneezing, • Open-mouth breathing and tail bobbing,

Physical Examination Findings
• Normal in mild cases, • Dyspnea (open-mouth breathing, tail bobbing, increased respiratory rate and effort). • Click (may be variously loud; may require bird being close to ear to hear this sound). • Frequent swallowing motions and increased oral mucous. • Beak rubbing (upper and lower together or both on perch), head shaking. • Nasal discharge. • Moist breathing sounds ausculted. • Weight loss. • Various degrees of general lethargy/reduced activity/"fluffing" of feathers. • Death.

CAUSES
• Mites or mite eggs eliminated in sneezes, coughs, and feces of infected birds. • Waste, food, and environment may become contaminated. • Infected parents feeding chicks. • No intermediate host required.

RISK FACTOR
Poor quarantine and flock management.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
Other causes of respiratory distress:
• bacterial respiratory infection (*Enterococcus fecalis*); • fungal infection (aspergillosis); • other parasitic infection (*Syngamus* or *Trichomonas* infection); • viral infection (poxvirus); • space-occupying lesions in or around the respiratory system (including obesity, dystocia); • airborne irritants and toxicants (PTFE).
Susceptible species have higher infection rate and mites are more likely with respiratory signs.

CBC/BIOCHEMISTRY/URINALYSIS
There are usually no changes in biochemical or urinalysis tests; reported hematological changes include eosinophilia and/or a basophilia with infection.

OTHER LABORATORY TESTS
The eggs of the mites, or the mites themselves, may be seen in oral swab samples or in fecal samples (via direct wet mount microscopy). Test results may be false negative.

IMAGING
A respiratory mite infection may have generalized nonspecific radioopacity changes to the pulmonary and air sac fields with radiology.

DIAGNOSTIC PROCEDURES
• Transilluminate the neck/trachea with bright light source after moistening skin with alcohol. • Dark specks can be seen in tracheal lumen. • The mites may be in the lower respiratory areas instead and not seen in trachea. • Tracheal endoscopy (using 1.2-mm endoscope) may be useful.

PATHOLOGIC FINDINGS
At necropsy dark specks may be seen in the mucous at any location of the respiratory system. Pneumonia, thickened and opaque air sac membranes and tracheitis can be seen. Mites embed their legs into the tissue and live in the mucous layer. Histopathology may show mucous epithelial necrosis, mucosal hyperplasia, and inflammation.

TREATMENT

APPROPRIATE HEALTH CARE
• Mild-to-moderate signs: Home care is usually sufficient. • Very dyspneic, lethargic, inappetant, or thin/weak birds: Hospital care may be required.
Infection with concurrent primary or secondary infectious agents may need additional therapy.

NURSING CARE
If severe dyspnea is present, oxygen and humidity supplementation might be useful. If weight loss and/or reduced self-feeding is present, the bird may require supplemental/ assisted feeding and crystalloid fluid administration (usually delivered subcutaneously).

ACTIVITY
If respiratory distress is present, the bird's exercise range should be limited and agitation/ stress should be carefully limited/ monitored.

DIET
Although nutritional needs must be met in all species, caloric intake must be aggressively maintained in the species with high metabolic rate that may have decreased intake with illness, as well as addressing any longer-term nutritional deficiencies that might be concurrent.

CLIENT EDUCATION
The owner should be advised to expect possible exaggerated symptoms after therapy as it has been reported with heavy infections; the massive die-off of the mites may cause symptoms to worsen shortly after treatment, before improving.

SURGICAL CONSIDERATIONS
It is best to fully treat these mites before surgery; in the case of emergency surgical needs, there is a higher risk of airway maintenance difficulties because of the increased mucous production and mite blockage of the respiratory passageways. The species affected tend to be small, which may affect endotracheal tube use.

MEDICATIONS

DRUG(S) OF CHOICE
Drugs of the avermectin class, especially Ivermectin at 0.2 mg/kg treated topically, orally, or parenterally repeated as often as weekly, for as long as several months. Other avermectins used include moxidectin and doramectin of unknown regimen.

CONTRAINDICATIONS
None known.

A

AIR SAC MITES (CONTINUED)

PRECAUTIONS
Drugs that depress respiration should be used with caution such as sedatives and opiods.

POSSIBLE INTERACTIONS
None likely.

ALTERNATIVE DRUGS
Some texts describe the use of a dichlorvos or a "no pest" strip near the affected birds, or an aerosol of rotenone or pyrethrin sprays. These pesticides have risks of toxicity, as precise dosing is impossible.

FOLLOW-UP

PATIENT MONITORING
Recheck examination performed within weeks for mild symptoms; sooner or later as symptom degree necessitates. Hemogram changes can be followed.

PREVENTION/AVOIDANCE
Quarantine, examine and treat new arrivals into the flock to prevent spread to birds already treated.

POSSIBLE COMPLICATIONS
• Acaricide may worsen symptoms due to mite die-off. • Insufficient therapy or resistance may allow recurrence of symptoms if all mites are not killed. • Secondary infections may progress even with resolution of primary issue. • Anesthesia and tracheoscopy higher risk in very symptomatic birds.

EXPECTED COURSE AND PROGNOSIS
Resolution of symptoms with therapy; prognosis is good unless symptoms are severe.

MISCELLANEOUS

ASSOCIATED CONDITIONS
N/A

AGE-RELATED FACTORS
N/A

ZOONOTIC POTENTIAL
N/A

FERTILITY/BREEDING
Heavy air sac mite infections can decrease breeding success. Some finch breeders will use surrogate parents of less-susceptible species to foster the chicks of more-susceptible species to limit the spread of this organism to new chicks.

SYNONYMS
Tracheal mite, respiratory mite, visceral mite, respiratory acariasis

SEE ALSO
Airborne toxicosis
Aspiration
Respiratory distress
Sick-bird syndrome
Tracheal/syringeal disease

ABBREVIATIONS
N/A

INTERNET RESOURCES
www.petmd.com/bird/conditions/respiratory/c_bd_respiratory_parasites-air_sac_mites

Suggested Reading

Ritchie, B., Harrison, G., Harrison, L. (1994). *Avian Medicine: Principles and Application*. Lake Worth, FL: Wingers Publishing.

Rosskopf, W., Woerpel, R. (1996). *Diseases of Cage and Aviary Birds*, 3rd edn. Baltimore, MD: Williams and Wilkins.

Samour, J. (ed.) (2000). *Avian Medicine*. London, UK: Mosby Publishing.

Altman, R., Clubb, S., Dorrestein, G., Quesenberry, K. (1997). *Avian Medicine and Surgery*. Philadelphia, PA: WB Saunders.

Author Vanessa Rolfe, DVM, ABVP (Avian)

BASICS

DEFINITION
The cervicocephalic, abdominal, or caudal thoracic air sacs can contribute to subcutaneous air accumulation when ruptured. Affected birds show emphysematous enlargement of various body parts depending on which air sac is leaking.

PATHOPHYSIOLOGY
This condition occurs when the air sac lining is disrupted secondary to a traumatic event, allowing air to accumulate under the skin. The location of the rupture is generally not identifiable. The cervicocephalic air sacs appear most commonly involved, with subcutaneous emphysema affecting the head, neck and extending over the dorsum/ventrum in severe cases. The cervicocephalic air sacs are the only air sacs that do not communicate directly with the pulmonary system. They communicate with the infraorbital sinuses. No oxygen exchange occurs within the cervicocephalic air sacs.

SYSTEMS AFFECTED
• Respiratory—rupture of the cervicocephalic air sacs and sometimes the abdominal/caudal thoracic air sacs. • Skin—subcutaneous emphysema leading to skin expansion over the affected area; the degree of skin tension varies with the quantity of air present in the subcutaneous space. • Musculoskeletal—air accumulation under the skin may restrict body movement.

GENETICS
None.

INCIDENCE/PREVALENCE
Unknown, but relatively common.

GEOGRAPHIC DISTRIBUTION
N/A

SIGNALMENT
• No specific species, age or sex predilection. • More commonly reported in Amazon parrots, macaws and cockatiels.

SIGNS
General Comments
• Rupture of an air sac is not life-threatening in most cases. However, fatalities may occur. • Subcutaneous emphysema causes discomfort to the avian patient and likely affects the bird's quality of life.

Historical Findings
• Traumatic event reported by the owner. • "Ballooning" under the skin of various body parts, most often affecting the head, neck, ventrum, and dorsum.

Physical Examination Findings
• Subcutaneous emphysema – in large species, the accumulation of air is most often confined to the dorsal aspect of the neck, whereas generalized subcutaneous emphysema may be seen more commonly in small bird species.

CAUSES
• Traumatic—air sacs generally rupture secondary to a traumatic event. Fractures of pneumatized bones may cause or contribute to the emphysema. • Infectious—chronic upper respiratory infection may also be involved. A pathologic process in the vicinity of the narrow connecting passage between the infraorbital sinus and the cervicocephalic air sac can act as a one-way valve, trapping air in the lumen of the air sac. • Nutritional—nutritional deficiencies such as hypovitaminosis A may predispose birds to respiratory infection.

RISK FACTORS
• Environmental—birds free-flying outside their cage present a higher risk of traumatic events, especially if the environment presents some dangers (ceiling fan, large mirror, wide unprotected windows, etc.). • Medical conditions—malnutrition may predispose to upper respiratory disease.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS
• Fracture of a pneumatized bone. • Luxation or subluxation of the humero–scapular joint causing disruption of the clavicular air sac. • Distension of the cervicocephalic air sacs without rupture—with upper respiratory disease involving the infraorbital sinuses, air may become entrapped within the cervicocephalic air sacs leading to their distention. • Infection with gas-producing bacteria.

CBC/BIOCHEMISTRY/URINALYSIS
• No specific abnormalities are seen with air sac rupture. • CBC/biochemistry—indicated to rule out concurrent disease.

OTHER LABORATORY TESTS
• Bacterial/fungal culture—to evaluate the presence of respiratory disease. The rostral aspect of the choana may be sampled. Nasal or sinus flush may be considered to obtain a more representative sample of the upper respiratory tract microbial flora. Skin culture if subcutaneous infection is suspected. • *Chlamydia* testing—may be considered if respiratory signs are present. • *Aspergillus* testing—may be considered if respiratory signs are present.

IMAGING
• Whole body radiographs—to identify musculoskeletal abnormalities. • Computed tomographic examination—to assess the infraorbital sinuses for disease processes; to identify musculoskeletal abnormalities.

DIAGNOSTIC PROCEDURES
No additional diagnostic procedures are indicated.

PATHOLOGIC FINDINGS
Air sac distention with concurrent signs of inflammation.

TREATMENT

APPROPRIATE HEALTH CARE
• Outpatient medical management—patient otherwise normal; diagnostic approach may require brief hospitalization. • Inpatient medical management—patient presenting with severe subcutaneous emphysema with depression/lethargy or concurrent disease that requires close monitoring. • Surgical management if relapse occurs with air aspiration

NURSING CARE
• Air aspiration—air can easily be removed using a syringe and hypodermic needle to decrease skin tension. However, the space often quickly refills as the patient breathes since the breach within the respiratory system is still patent. The procedure may be repeated multiple times. This technique may be used initially, especially if severe emphysema is present to decrease skin tension and improve patient's comfort. • Elizabethan collar—may be considered if a Teflon dermal stent is used in an area that the bird can reach with its beak.

ACTIVITY
Exercise may exacerbate the subcutaneous emphysema. Activity level recommendations should be adjusted to each individual avian patient.

DIET
Suboptimal diets should be improved.

CLIENT EDUCATION
Spontaneous resolution is possible but problem may be chronic and recurrent.

SURGICAL CONSIDERATIONS
• Fistula: Using a 2–6 mm skin biopsy punch or a scapel blade, a piece of skin is removed over the inflated area and the wound is left to heal by second intention. This opening will allow the skin to lie against the traumatized tissue and allow the rupture site to heal properly. In many cases, there is no reinflation of the subcutaneous space by the time the skin wound has healed. • Teflon® dermal stent (McAllister Technical Services, Coeur d'Alene, ID): Nonabsorbable sutures are preplaced in the four pairs of holes found around the stent. Then, a skin incision is performed over the distended skin ideally in an area that the bird is unable to reach with its beak. The bird's skin will deflate once the skin is incised and skin tension will decrease. The incision size should be just large enough to

A

AIR SAC RUPTURE (CONTINUED)

insert the stent into the subcutaneous space. A hypodermic needle is used to retrieve the sutures through the skin and tie the stent in place. The four sutures should be placed two on each side of the incision and two at both ends of the incision. A purse-string suture may be placed around the rim of the stent that remains above the skin. The stent is generally left in place permanently.
• Cervicocephalic–clavicular air sac shunt: Possible when one of these two air sacs is ruptured. A skin incision is performed in the left lateral thoracic inlet area to avoid the esophagus. A small endotracheal tube is inserted in the hyperinflated air sac. The tube is then directed caudally along the esophagus to the cranial aspect of the clavicular air sac. The tube is sutured to the *longus coli* muscle to prevent migration before skin closure. The shunt is generally left in place permanently.

MEDICATIONS

DRUG(S) OF CHOICE
• Broad-spectrum antibiotic—consider perioperatively and until cleaning of the stent is minimal postoperatively. • Nonsteroidal anti-inflammatory medication (meloxicam 0.5 mg/kg PO/IM q12h) may be considered to alleviate inflammation at the surgical site. • Opioids (butorphanol 1–4 mg/kg q4–8h) is recommended perioperatively.

CONTRAINDICATIONS
None.

PRECAUTIONS
None.

POSSIBLE INTERACTIONS
N/A

ALTERNATIVE DRUGS
Doxycycline: Consider to attempt pleurodesis. Air sac lining of birds is similar to the pleural lining of mammals. Medication instilled in the air sac will cause irritation between the air sac surfaces, closing off the space between them and preventing further air from accumulating. This procedure may be considered for cervicocephalic air sac rupture only to avoid dissemination of the instilled product to the lower respiratory system. Since pleurodesis has only been described in one Amazon parrot thus far, this technique should be used in last resort.

FOLLOW-UP

PATIENT MONITORING
• Aspiration: Recurrence of emphysema indicates the rupture site is not healed. The procedure may be repeated or surgical approach may be considered. • Fistula: Recurrence of emphysema indicates the rupture site is not healed. Depending on the location, placement of a dermal stent or cervicocephalic–clavicular air sac shunt may be considered. • Teflon® dermal stent: Postoperative care requires cleaning of the stent opening to prevent obstruction by debris and tissue fluids. Sterile swabs or needles are recommended to decrease bacterial contamination. Initially, cleaning twice daily is recommended. The cleaning frequency should be adjusted to each patient and decrease progressively. Recurrence of emphysema indicates the stent is no longer patent. • Cervicocephalic–clavicular air sac shunt: Recurrence of emphysema may indicate the shunt is no longer patent and surgical exploration is indicated.

PREVENTION/AVOIDANCE
Environment—ensure the bird's environment is safe to decrease the likelihood of trauma.

POSSIBLE COMPLICATIONS
• Teflon® dermal stent: Occlusion of the stent opening by debris and tissue fluids is common initially. On rare occasions, bird may pick at the stent. • Cervicocephalic-clavicular air sac shunt: Occlusion of the shunt. • Unable to achieve complete resolution of the emphysema despite all therapeutic measures.

EXPECTED COURSE AND PROGNOSIS
• Aspiration: If performed rapidly following the traumatic event, chances that the air will continue to accumulate in the subcutaneous space may be decreased. This technique is expected to provide only a temporary relief in chronic cases of air sac rupture. • Fistula: If performed rapidly following the traumatic event, chances that the air will continue to accumulate in the subcutaneous space are decreased. This technique is expected to provide only a temporary relief in chronic cases of air sac rupture. • Teflon® dermal stent: Complete resolution of the subcutaneous emphysema in most birds, partial resolution of the subcutaneous emphysema in some birds, minimally invasive procedure associated with a good prognosis. • Cervicocephalic–clavicular air sac shunt: Complete resolution of the subcutaneous emphysema in most birds based on scant literature, moderately invasive procedure associated with a good prognosis.

MISCELLANEOUS

ASSOCIATED CONDITIONS
None.

AGE-RELATED FACTORS
None.

ZOONOTIC POTENTIAL
None.

FERTILITY/BREEDING
Avoid teratogenic antibiotics in laying hen.

SYNONYMS
N/A

SEE ALSO
Bite wounds
Fracture/luxation
Respiratory distress
Rhinitis and sinusitis
Tracheal/syringeal diseases
Trauma

ABBREVIATIONS
None.

INTERNET RESOURCES
http://avianmedicine.net/content/uploads/2013/03/41.pdf

Suggested Reading

Antinoff, N. (2008). Attempted pleurodesis for an air sac rupture in an Amazon parrot. *Proceedings of the Association of Avian Veterinarians Annual Conference*, August 11–14, Savannah, GA, p. 437.
Bennett, R.A., Harrison, G.J. (1994). Soft tissue surgery. In: Ritchie, B.W., Harrison, G.J., Harrison, L.R. (eds), *Avian Medicine: Principles and Application*. Lake Worth, FL: Wingers Publishing, pp.1096–1136.
Harris, J.M. (1991). Teflon dermal stent for the correction of subcutaneous emphysema. *Proceedings of the Association of Avian Veterinarians Annual Conference*, September 23–28, Chicago, IL, pp. 20–21.
Levine, B.S. (2005). Cervicocephalic-clavicular air sac shunts to correct cervicocephalic-clavicular air sac emphysema. *Proceedings of the Association of Avian Veterinarians Annual Conference*, August 9–11, Monterey, CA, pp. 59–60.
Petevinos, H. (2006). A method for resolving subcutaneous emphysema in a griffon vulture chick (*Gyps fulvus*). *Journal of Exotic Pet Medicine*, **15**(2):132–137.

Author Isabelle Langlois, DMV, DABVP (Avian)

BASICS

DEFINITION

The literal translation of the word "anemia" is lack of blood. In practice, anemia is more commonly defined by a decrease in the red blood cell population or in a decrease in the normal quantity or quality of hemoglobin in the red blood cells. In other words, anemia can also be defined as a decrease in the oxygen-carrying capacity of the blood. Anemia can be further delineated by the cause of the condition. This can be grouped into three large categories:

1. Impairment of red blood cell production leading to decreased red blood cells or improperly functioning red blood cells.
2. Increased destruction of red blood cells (hemolytic anemia).
3. Blood loss (both acute and chronic).

Finally, one further way of classifying anemia is based on the number of circulating immature red blood cells (reticulocytes). In this scheme, anemia is either regenerative (excess of reticulocytes) or non-regenerative (lack of reticulocytes) anemia.

PATHOPHYSIOLOGY

The pathophysiology depends on the type of anemia that is present. The pathophysiology of blood loss is self-explanatory. Hemolytic anemia occurs when healthy red blood cells are being destroyed at a faster rate than normal and red blood cell production cannot keep pace with destruction. Hemolysis can occur for a variety of reasons including if there are antibodies directed against the red blood cells. Red blood cells can be phagocytized by macrophages (extravascular hemolysis) or destroyed in the blood vessels (intravascular hemolysis). Impairment of red blood cell production can occur for a number of reasons. Intrinsic causes of decreased red blood cell production are due to a defect in the bone marrow and erythrocyte stem cells are not produced. Extrinsic causes also cause disease in the bone marrow leading to decreased production of or defective red blood cells but are due to conditions outside of the bone marrow. In either case, not enough functioning erythrocytes are produced. Without enough competent, hemoglobin-rich cells, the tissues of the avian body lack sufficient oxygen to properly carry out necessary functions leading to an overall weakness and lethargy of the patient. Depending on the chronicity and severity of the anemia, the lack of oxygen to tissues can lead to eventual organ failure and death of the patient.

SYSTEMS AFFECTED

• Behavioral—anemia may not change the behavior, *per se*, of the patient, but if the patient is weakened from anemia, the typical behaviors of the pet may be dampened or non-existent due to lack of energy.
• Cardiovascular—chronic anemia can lead to increased cardiac output, increased blood flow, and cardiac murmurs. • Endocrine/Metabolic—metabolic functions may be affected if anemia is severe and/or chronic due to the lack of oxygen • Hepatobiliary—the liver and/or spleen may enlarge in cases of hemolytic anemia.
• Musculoskeletal—skeletal tissue may appear pale due to decreased erythrocyte population. Weakness may also be present.
• Ophthalmic—conjunctiva will appear pale with anemia. Retinal hemorrhage can occur with some forms of anemia.
• Renal/Urologic—hemoglobinuria can occur with hemolytic anemia and urine and urates can have a darker color.
• Reproductive—breeding hens may have a disruption in breeding cycle or decreased production of chicks if anemia is present.
• Respiratory—with severe anemia, respiratory rate and depth may be increased significantly. • Skin—the skin and mucous membranes can take on a very pale appearance with severe anemia.

GENETICS

In the avian literature, there is a disease condition termed, "conure bleeding syndrome" but no etiology was ever discovered although vitamin (e.g., vitamin D or K) or mineral (i.e., Ca) deficiency was suspected in certain lines of conures.

INCIDENCE/PREVALENCE

Anemia of chronic disease is common in birds. Blood loss due to trauma or parasites is also common. Hemolytic anemias are the least common types of anemia in birds.

GEOGRAPHIC DISTRIBUTION

There is no geographic distribution of anemia.

SIGNALMENT

• **Species:** All species of birds can have anemia. • **Mean age and range:** All ages are susceptible to anemia. • **Predominant sex:** Both sexes are equally affected.

SIGNS

General Comments

It is possible that no signs of anemia will be apparent to the owner, there will be no significant aspect of the history, and anemia may be found as an incidental finding during veterinary visit for an annual physical examination and minimum database.

Historical Findings

If anemia is due to blood loss, owners may report trauma and blood loss due to trauma. If there is hemolytic anemia, owners may report darkened urine and urates in the droppings. Although unusual, frank blood or melena can be seen in the fecal portion of the droppings. Or there may be frank blood not associated with the droppings but seen emanating from the cloaca as could be the case with ulcerated lesions associated with cloacal papilloma disease.

Physical Examination Findings

• Pale mucous membranes including conjunctiva. • Overall body weakness.
• Increased respiratory rate and effort.
• Evidence of trauma and subsequent blood loss. • Puncture wounds if trauma. • Blood in or around the cloaca. • Color change or blood in feces, urine and urates. • Petechia and ecchymosis.

CAUSES

Blood Loss Anemia

• Trauma • Gastrointestinal bleeding: neoplasia, bacterial infection, viral infection, parasitic infection, toxins • Reproductive disease in hens • External and internal parasites • Anticoagulant toxicosis.

Hemolytic Anemia

• Zinc toxicosis • Copper toxicosis
• Aflatoxicosis • Transfusion reaction
• *Plasmodium* infection • Viral diseases
• Immune mediated hemolytic anemia-idiopathic • Other toxins • Septicemias.

Impaired Erythrocyte Production

• Heavy metal toxicosis, especially lead
• Chronic disease: common diseases include: Chlamydia, aspergillosis, mycobacteria, egg yolk coelomitis, circovirus • Neoplasia
• Corticosteroid administration • Iron deficiency • Nutritional deficiencies
• Chronic organ disease: Renal disease, liver disease • Ingestion of petroleum products.

RISK FACTORS

Exposure to toxins, exposure to predators, chronic disease, red blood cell parasites, iron or folic acid deficiency, neoplasia.

DIAGNOSIS

DIFFERENTIAL DIAGNOSIS

Iatrogenic hemolysis of the red blood cells due to poor venipuncture technique or sample handing before analysis.

CBC/BIOCHEMISTRY/URINALYSIS

• PCV below the normal reference interval.
• Hemoglobin below the normal reference interval. • Increased polychromasia and anisocytosis in regenerative anemia.
• Increased reticulocytes in regenerative anemia.

OTHER LABORATORY TESTS

• Specific tests for infectious diseases. • Heavy metal concentration in blood samples.
• Examination of droppings for red blood cells. • Examination of blood drop on slide for evidence of agglutination. • Fecal parasitic examination.

IMAGING

Whole body radiographs may be necessary depending on the differential diagnoses such

A

ANEMIA (CONTINUED)

as: heavy metal ingestion, coelomitis, reproductive disease, splenic disease, liver disease.

DIAGNOSTIC PROCEDURES
Additional diagnostics may be necessary depending on the rule out list.

PATHOLOGIC FINDINGS
Dependent on the cause of the anemia. Unless there is erythrophagocytosis in the bone marrow, liver or spleen, there may be no pathologic findings present with anemia except for the lack of red blood cells.

TREATMENT

APPROPRIATE HEALTH CARE
Management will depend on the cause of the anemia.

NURSING CARE
Despite the cause, the patient may need supportive care until the cause of the anemia can be determined and treated. Nursing care may consist of:
• Fluids, especially if due to blood loss. Fluids may consist of a physiologically balanced replacement and/or a plasma expanding solution such as one containing hydroxyethyl starch. • Blood transfusion. Blood should only be transfused from a member of the same species. • Assisted feedings may be necessary.

ACTIVITY
In severe cases, activity should be limited until oxygen carrying capacity is restored to normal.

DIET
If dietary deficiencies led to anemia, the diet should be corrected.

CLIENT EDUCATION
N/A

SURGICAL CONSIDERATIONS
If anemia is due to heavy metal ingestion, in some cases, surgical or endoscopic retrieval of the metal objects can be performed. Some cases of trauma, especially causing internal bleeding, may require surgical intervention.

MEDICATIONS

DRUG(S) OF CHOICE
• Erythropoietin. • Iron dextran 10 mg/kg IM once, repeat if necessary.

CONTRAINDICATIONS
Iron supplementation in species prone to hemochromatosis.

PRECAUTIONS
Any drug that has the potential side effect of causing further anemia should be avoided.

POSSIBLE INTERACTIONS
N/A

ALTERNATIVE DRUGS
N/A

FOLLOW-UP

PATIENT MONITORING
PCV should be closely monitored and can be checked on a daily basis during the acute phase of treatment. The entire CBC should be evaluated on a regular basis until the patient is stable.

PREVENTION /AVOIDANCE
This is dependent on the cause of anemia.

POSSIBLE COMPLICATIONS
Severe anemia can lead to death.

EXPECTED COURSE AND PROGNOSIS
Depends on the cause of the anemia.

MISCELLANEOUS

ASSOCIATED CONDITIONS
N/A

AGE-RELATED FACTORS
Young chicks, geriatric birds will be more susceptible to the severe complications of anemia than healthy adult birds.

ZOONOTIC POTENTIAL
Depends on the cause of anemia.

FERTILITY/BREEDING
Anemia can disrupt egg production.

SYNONYMS
N/A

SEE ALSO
Anticoagulant rodenticide
Circoviruses
Coagulopathies
Heavy metal toxicity
Hemoparasites
Hemorrhage
Liver disease
Nutritional deficiencies
Oil exposure
Polyomavirus
Renal disease
Sick-bird syndrome
Urate/fecal discoloration
Trauma
Viral disease
Appendix 7, Algorithm 7. Anemia

ABBREVIATIONS
N/A

INTERNET RESOURCES
N/A

Suggested Reading

Campbell, T. (2004). Hematology of birds. In: Thrall, M., *Veterinary Hematology and Clinical Chemistry*. Philadelphia, PA: Lippincott Williams, and Wilkins, pp. 225–258.

Fudge, A. (2000). *Laboratory Medicine: Avian and Exotic Pets*. Philadelphia, PA: WB Saunders.

Tully, T., Lawton, M., Dorrestein, G. (2000). *Avian Medicine*. Woburn, MA: Butterworth-Heinemann.

Author Karen Rosenthal, DVM, MS

BASICS

DEFINITION
Angel wing is a commonly-used term to describe the condition of carpal valgus in avian patients resulting from malnutrition and captive mismanagement.

PATHOPHYSIOLOGY
Angel wing is the result of the plumage developing at a faster rate than the musculoskeletal structures of the wing. The immature musculoskeletal structures of the wing are not strong enough to support the weight of the blood-filled quills of the rapidly developing plumage. The weight of the developing feathers increasingly pulls the wing into a deformed position.

SYSTEMS AFFECTED
• Musculoskeletal. • Integument.

GENETICS
There is information to suggest a genetic predisposition to angel wing in certain lines of birds. In a study of white Roman geese, angel wing severity was worse in certain lines of birds regardless of diet.

INCIDENCE/PREVALENCE
Angel wing is typically a disorder of captive birds or those being fed artificial diets (such as geese and ducks in parks that receive significant amounts of bread from well-meaning bird lovers). Incidence is rare in well-managed captive animals. Occasionally, outbreaks of angel wing are noted in the nestlings of certain wild populations.

GEOGRAPHIC DISTRIBUTION
N/A

SIGNALMENT
• **Species:** All species. Birds belonging to the orders anseriformes and otidiformes are most frequently affected. Slower growing species from temperate regions of the world are especially susceptible. • **Mean age and range**: Varies according to species. Occurs when initial set of primary wing feathers develop. • **Predominant sex**: No sex predilection.

SIGNS
Angel wing presents as unilateral or bilateral drooping of the wing at the carpi and elbows with outward (valgus) rotation of the wing distal to the carpi. The primary remiges are often deviated dorsally and laterally with the wing in a relaxed, flexed position. In young birds with immature musculoskeletal structures, the wing(s) can usually be manually manipulated into normal conformation. In older birds, bone mineralization and maturation of soft tissue structures result in permanent deformities that cannot be corrected manually.

CAUSES
Carpal valgus occurs when the weight of the developing primary feathers exceeds the musculoskeletal structure's ability to hold the wing in a normal position. Diets containing excessive protein and carbohydrates result in inappropriately rapid feather development leading to angel wing. This often occurs in populations of birds in parks that are fed large amounts of high-energy foods such as bread. Lack of exercise and musculoskeletal fitness can also result in angel wing even when the diet is appropriate.

RISK FACTORS
There are a number of other factors thought to be involved in the development of angel wing. Excessive dietary protein and energy are most commonly associated with angel wing. However, lack of adequate exercise, genetic predisposition, interruption of egg incubation, excessive heat during early development, vitamin D, E and manganese deficiency are also implicated in some cases.

DIAGNOSIS

DIFFERENTIAL DIAGNOSES
Occasionally fractures (traumatic or pathologic) of the distal wing(s) will mimic angel wing.

CBC/BIOCHEMISTRY/URINALYSIS
N/A

OTHER LABORATORY TESTS
N/A

IMAGING
Radiography of the affected wing(s) may help rule out traumatic injury, osteopenia, etc.

DIAGNOSTIC PROCEDURES
The diagnosis of angel wing in birds is usually quite straightforward and is made via signalment, history, physical examination and sometimes radiography. Rarely will other diagnostic modalities be necessary.

PATHOLOGIC FINDINGS
Abnormalities are limited to the gross anatomic deformities. Histologic lesions are not typically appreciated.

TREATMENT

APPROPRIATE HEALTH CARE
• Early intervention is key to correction of angel wing in young, growing birds. • In cases where clinical signs are limited to a mild wing droop and valgus has not begun to develop, trimming of the primary feathers to relieve weight on the distal wing can be corrective. • In more severe cases where valgus of the distal wing has begun to develop, intervention usually requires fixing the wing in a normal resting position with or without weight relief by primary feather trimming. The wing is placed into a normal, resting position and a light figure-of-eight bandage applied. In some cases taping the humerus, radius/ulna, and phalanges in line is sufficient; other cases require a more substantial bandage such as a figure-of-eight. A body wrap is not usually required. Holding the wing in a normal position for 3–5 days is typically sufficient to correct the deformity. • If not caught early and musculoskeletal maturation has progressed to the point where the wing cannot be manually manipulated easily back into normal position, treatment via wing taping is usually not effective.

NURSING CARE
N/A

ACTIVITY
Activity and exercise are encouraged in young birds to stimulate development of strong wings capable of supporting mature plumage. Typically exercise can be encouraged simply by providing spacious quarters for the young birds to move about in.

DIET
• A balanced diet with appropriate protein and energy levels for the species is paramount to preventing angel wing and other development deformities in young, growing birds • Relatively slow-growing avian species should not be fed high-energy and high-protein diets as it can create mis-matches between musculoskeletal and plumage development leading to angel wing. In waterfowl, northern/arctic species are adapted to essentially feed around the clock on high quality foods in order to maximize growth in the small window of time available. Conversely, temperate/tropical species typically have a much longer window of time in which to achieve the necessary growth, therefore they do not have need for a constant intake of high energy diet. • Dietary protein levels between 8 and 15% are recommended during the first three weeks of life in slow-growing waterfowl.

CLIENT EDUCATION
Clients who are interested in breeding bird species predisposed to developing angel wing should be well versed in their dietary and husbandry requirements. Exact diet and husbandry requirements will, of course, depend on the species in question, but in general protein and energy levels should be kept at the minimum acceptable level and opportunity for exercise maximized.

SURGICAL CONSIDERATIONS
• Mature birds with angel wing typically require surgical correction. Phalangeal amputation ("pinioning") or osteotomy with rotation and fixation are typically required to create a comfortable and cosmetic wing.

A

ANGEL WING (CONTINUED)

• Pinioning of older birds can result in significant complications as the resultant stumps are often prone to injury.

MEDICATIONS

DRUG(S) OF CHOICE
Although typically not indicated, analgesic and/or anti-inflammatory medications may be prescribed for relief of discomfort at the discretion of the clinician.

CONTRAINDICATIONS
Birds affected by angel wing are usually young, but otherwise there are usually no unique contraindications for analgesic/anti-inflammatory medications.

PRECAUTIONS
N/A

POSSIBLE INTERACTIONS
N/A

ALTERNATIVE DRUGS
N/A

FOLLOW-UP

PATIENT MONITORING
• Wing bandages/tape should not be left in place beyond 72 hours if possible. If bandaging is going to be successful, it will only need to be in place for 48–72 hours prior to removal. • In adult birds with mature deformities, primary wing feathers can be periodically trimmed to make the abnormal conformation less obvious.

PREVENTION/AVOIDANCE
Most cases of angel wing can be easily prevented with appropriate diet and husbandry. Proper nutrition during early development is key to prevention of angel wing. Neonatal birds should be provided with adequate opportunity to exercise and strengthen the developing musculoskeletal system. Removal from the breeding population of affected birds or birds with significant occurrence of the deformity in offspring may be warranted.

POSSIBLE COMPLICATIONS
• If intervention is not initiated early enough, usually within 3–5 days of first noticing clinical signs, bandaging will fail to correct carpal valgus. • In cases of permanent deformity, the wing can sometimes become traumatized if the bird cannot manipulate it appropriately.

EXPECTED COURSE AND PROGNOSIS
• Birds with angel wing typically are incapable of normal flight as adults despite intervention. • Angel wing deformities are not life-threatening and many birds will survive and thrive if the deformities are not corrected. • The birds will obviously continue to display the abnormal conformation and will be unable to fly. Complications can occur if the bird is not able to manipulate the wing to keep it from being traumatized.

MISCELLANEOUS

ASSOCIATED CONDITIONS
Angular limb deformities of the pelvic limbs are sometime encountered in conjunction with angel wing deformities.

AGE-RELATED FACTORS
Angel wing only occurs in young birds with developing plumage.

ZOONOTIC POTENTIAL
N/A

FERTILITY/BREEDING
As the role genetics play in the occurrence of angel wing is not understood, in some cases it may be prudent to avoid breeding affected birds.

SYNONYMS
Carpal valgus, airplane wing, dropped wing, crooked wing.

SEE ALSO
Arthritis
Feather disorders
Fracture/luxation
Lameness
Metabolic bone disease
Nutritional deficiencies
Splay leg/slipped tendon
Trauma

ABBREVIATIONS
N/A

INTERNET RESOURCES
N/A

Suggested Reading

Olsen, J.H. (1994). Anseriformes. In: Ritchie, B.W., Harrison, G.J., Harrison, L.R. (eds), *Avian Medicine: Principles and Application*. Lake Worth, FL: Wingers Publishing, pp. 1237–1275.

Author Ryan S. De Voe, DVM, DABVP (Avian, Reptile/Amphibian), DACZM

Client Education Handout available online