Patrick T. Redig, John E. Cooper, J. David Remple, and D. Bruce Hunter, editors
Trudi Hahn, technical editor

Raptor BIOMEDICINE

Raptor Biomedicine

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On the cover: The golden eagle (*Aquila chrysaetos*) is the most widely distributed eagle of the genus *Aquila*. The species is found in North America, Europe, northern Africa, the Middle East, and Asia.

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Contents

List of Abbreviations

ALT	alanine aminotransferase
AP	anterior-posterior
AST	aspartate aminotransferase
ATP	adenosine triphosphate
BID	twice a day
bpm	beats per minute
CBC	complete blood count
C	centigrade
cc	cubic centimeters
cm	centimeter
CNS	central nervous system
dl	deciliter
DPI	days postinoculation
ECG	electrocardiogram
ED_{50}	effective dosage for 50% of animals
EF	external fixtor
ft	foot
g	gram
hr	hour
HR	heart rate
Hz	hertz, cycles per second
IAVD	incomplete atrioventricular dissociation
i.d.	inside diameter
IM	intramuscular
in	inch
IU	international units
IU/l	international units per liter
IV	intravenous
kcal	kilocalorie
kJoule	kilojoules
kg	kilogram
km	kilometer
km^2	square kilometer
kva	kilovolt-amperes
kvp	kilovoltage peak
kv	1,000 volts
K-wire	Kirschner wire
l	liter
L	levo-rotary
LD_{50}	lethal dose for 50% of animals
LDH	lactate dehydrogenase
m	meter
mA	milliampere
meq	milliequivalent
mg	milligram
mm	millimeter
mm^3	cubic millimeter
mm/sec	millimeters per second
msec	milliseconds
min	minute
ml	milliliter
mon	month
μ	micron
μg/kg	microgram per kilogram
μm	micrometer
μV	microvolt
mV	millivolt
n	number
NAD	nicotinamide adenine dinucleotide
n.d.	no date
n.s.	nonsignificant
p	probability value for statistical evaluation
PO	per os, orally
ppm	parts per million
PR	PR interval of an ECG
PCV	packed cell volume, expressed as a percentage
QID	four times a day
r	correlation coefficient
rpm	revolutions per minute
RR	respiratory rate
sec	second
SGOT	serum glutamic-oxaloacetate amino transferase (also AST)
SID	once a day
sp., spp.	species, singular and plural
SQ	subcutaneous
ST	S-T segment of an ECG
T_b	body temperature
TID	three times a day
V	volt
VD	ventral-dorsal
wbc	white blood cells
wk	week
wt	weight
yr	year

Standard Abbreviations for Raptor Species as Used in This Text

BDO	barred owl (*Strix varia*)
BE	bald eagle (*Haliaeetus leucocephalus*)
BWH	broad-winged hawk (*Buteo platypterus*)
CSH	Cooper's hawk (*Accipiter cooperii*)
GE	golden eagle (*Aquila chrysaetos*)
GHO	great horned owl (*Bubo virginianus*)
GSH	northern goshawk (*Accipiter gentilis*)

HAR	northern harrier (*Circus cyaneus*)
KES	American kestrel (*Falco sparverius*)
LEO	long-eared owl (*Asio otus*)
PEF	peregrine falcon (*Falco peregrinus*)
PRF	prairie falcon (*Falco mexicanus*)
RLH	rough-legged hawk (*Buteo lagopus*)
RTH	red-tailed hawk (*Buteo jamaicensis*)
SSH	sharp-shinned hawk (*Accipiter striatus*)
SWO	saw-whet owl (*Aegolius acadicus*)

List of Addresses of Contributors

Adnan A. Al-Ashbal, A.M.R.C.Path., Dip. Histopath.
Dept. of Histopathology
New Dubai Hospital
P.O. Box 7272
Dubai
United Arab Emirates

Cheryl Bartlett, B.Sc., Ph.D.
Dept. of Mathematics and Natural Sciences
University College of Cape Breton
Sidney, Nova Scotia
Canada

Diane Blais, D.V.M.
Faculté de Médecine Vétérinaire
Université de Montréal
C.P. 5000
Saint Hyacinthe, Québec
Canada J2S 7C6

Steven Boros, M.D.
Infant Pulmonary Research Center
St. Paul Children's Hospital
St. Paul, MN 55102
USA

Kenneth A. Bruecker, D.V.M., M.S.
Veterinary Surgical Consultants
P.O. Box 3146
Ventura, CA 93006
USA

Naomi L. Burtnick, M.T. (A.S.C.P.)
Cardiology Section
Veterinary Teaching Hospital
University of Minnesota
1365 Gortner Avenue
St. Paul, MN 55108
USA

Terry W. Campbell, D.V.M., Ph.D.
Sea World of Florida
7007 Sea World Drive
Orlando, FL 32821
USA

Richard J. Cawthorn, Ph.D.
Dept. of Pathology and Microbiology
Atlantic Veterinary College
University of Prince Edward Island
550 Charlottetown, Prince Edward Island
Canada C1A 4P3

Susan B. Chaplin, Ph.D.
Dept. of Biology
Mail #4369
University of St. Thomas
2115 Summit Avenue
St. Paul, MN 55105
USA

John E. Cooper, D.T.V.M., F.I.Biol., M.R.C.Path., F.R.C.V.S.
Faculty of Veterinary Medicine
P.O. Box 3021
Sokoine University of Agriculture
Morogoro, Tanzania
East Africa
Contact address in Europe:
DICE
The University
Canterbury
Kent CT2 7NX
England

Roy D. Crawford, B.S.A., M.S., Ph.D.
Dept. of Animal and Poultry Science
University of Saskatchewan
Saskatoon, Saskatchewan
Canada S7N 0W0

Laurel A. Degernes, D.V.M.
Dept. of Companion Animals and Special Species
College of Veterinary Medicine
North Carolina State University
Raleigh, NC 27606
USA

Gary Duke, Ph.D.
Dept. of Small Animal Clinical Sciences
College of Veterinary Medicine
University of Minnesota
St. Paul, MN 55108
USA

M. Finnegan, D.V.M.
Zoological Medicine
Veterinary Medical Teaching Hospital
University of California
Davis, CA 95616
USA

Guy Fitzgerald, M.Sc., D.V.M.
UQROP
C.P. 5000
Saint Hyacinthe, Québec
Canada J2S 7C6

Neil A. Forbes, B.Vet.Med., M.R.C.V.S.
Clockhouse Veterinary Hospital
Wallbridge, Stroud
Gloucestershire GL5 3JD
England

Margaret Gordon, R.R.T.
St. Paul Children's Hospital
St. Paul, MN 55102
USA

Edna Gorney-Labinger, M.Sc.
George S. Wise Faculty of Life Sciences
Dept. of Zoology
Tel Aviv University
Ramat-Aviv, Tel Aviv 69978
Israel

Fritz Grimm, Ph.D.
Institut für Geflügelkrankheiten
Ludwig-Maximilians-Universität München
Veterinärstrasse 3
D-8042 Oberschleissheim
Germany

Dieter Haas, Dr.Med.
Charlottenstrasse 21
7470 Albstadt-Tailfingen
Germany

David D. Herrick, D.V.M.
Cortez Animal Clinic
Cortez, CO 81321
USA

John Hoolihan, B.S.
Abu Dhabi Falcon Research Hospital
P.O. Box 77
Abu Dhabi
United Arab Emirates

C. Stuart Houston, M.D., D.Litt.
863 University Drive
Saskatoon, Saskatchewan
Canada S7N 0J8

D. Bruce Hunter, D.V.M., M.Sc.
Ontario Veterinary College
University of Guelph
Guelph, Ontario
Canada N1G 2W1

Fiona Ion, B.Sc.
183 Weaponess Road
Wembley Downs
6019 Western Australia
Australia

Alicia M. Karas, D.V.M.
8710 Woodbrook Road
Dallas, TX 95243
USA

Mats Karlbom
Institute of Zoophysiology
Box 560
751 22 Uppsala 1
Sweden

G. E. Kaufman, D.V.M.
Tufts University
200 Westboro Road
North Grafton, MA 01536
USA

Robert E. Kenward, D.Phil.
Institute of Terrestrial Ecology
Furzebrook Research Station
Wareham, Dorset BH20 5AS
England

James K. Kirkwood, B.V.Sc., Ph.D., M.R.C.V.S.
Institute of Zoology
Zoological Society of London
Regent's Park
London NW1 4RY
England

Julie S. Kreeger, D.V.M.
Dept. of Pathobiology
College of Veterinary Medicine
University of Minnesota
St. Paul, MN 55108
USA

Terry J. Kreeger, M.S., D.V.M., Ph.D.
BallistiVet, Inc.
8990 Springbrook Drive, Suite 130
Minneapolis, MN 55433
USA

K. M. Langelier, D.V.M.
Island Veterinary Hospital
1621 Townsite Road
Nanaimo, British Columbia
Canada V9S 1N3

Keith Lawrence, B.V.Sc., Ph.D., F.R.C.V.S.
Elanco Products Ltd.
Dextra Court, Chapel Hill
Basingstoke
Hampshire RG21 2SY
England

Paula J. Lind, A.H.T., B.S.
Minnesota Zoo
13000 Zoo Blvd.
Apple Valley, MN 55124
USA

J. T. Lumeij, D.V.M., Ph.D., Dipl. Int. Med. Comp. Animals (R.N.V.A.)
Division of Avian and Exotic Animal Medicine
Dept. of Companion Animal Clinical Sciences
Utrecht University
Yalelaan 8, 3584 CM Utrecht
The Netherlands

Mark Mammel, M.D.
Infant Pulmonary Research Center
St. Paul Children's Hospital
St. Paul, MN 55102
USA

Vidar Marcström, Ph.D.
Institute of Zoophysiology
Box 560
751 22 Uppsala 1
Sweden

Howard D. Martin, D.V.M.
6626 Tamarind Street
Agoura, CA 91301
USA

Katherine McKeever
Owl Rehabilitation and Research Foundation
Vineland, Ontario
Canada

Lori R. Mueller, M.A.
The Raptor Center
University of Minnesota
1920 Fitch Avenue
St. Paul, MN 55108
USA

I. Newton, B.Sc., D.Phil., D.Sc.
Institute of Terrestrial Ecology
Monks Wood Experimental Station
Abbots Ripton, Huntingdon
Cambridgeshire PE17 2LS
England

J. Lindsay Oaks, Jr., D.V.M.
Dept. of Veterinary Microbiology and Pathology
Washington State University
Pullman, WA 99164-7040
USA

Lynn W. Oliphant, B.S., M.S., Ph.D.
Dept. of Veterinary Anatomy
University of Saskatchewan
Saskatoon, Saskatchewan
Canada S7N 0W0

David W. Osgood, Ph.D.
P.O. Box 3386
Sunriver, OR 97707
USA

J. R. Paul-Murphy, D.V.M., Dip. A.C.Z.M.
School of Veterinary Medicine
University of Wisconsin-Madison
Madison, WI 53711
USA

Mark A. Pokras, B.S., D.V.M.
Wildlife Clinic
Tufts University School of Veterinary Medicine
200 Westboro Road
North Grafton, MA 01536-1895
USA

Stuart L. Porter, D.V.M.
Wildlife Center of Virginia
P.O. Box 98
Weyers Cave, VA 24486
USA

Marc R. Raffe, D.V.M., M.S.
C311 Veterinary Training Hospital
College of Veterinary Medicine
University of Minnesota
St. Paul, MN 55108
USA

Patrick T. Redig, D.V.M., Ph.D.
The Raptor Center
University of Minnesota
1920 Fitch Avenue
St. Paul, MN 55108
USA

J. David Remple, D.V.M.
P.O. Box 13919
Dubai
United Arab Emirates

Kenton E. Riddle, B.S., D.V.M.
Abu Dhabi Falcon Research Hospital
P.O. Box 77
Abu Dhabi
United Arab Emirates

Christine Ringdahl, R.P.T.
1201 Twin Peaks Circle
Longmont, CO 80503

Richard Rushton, D.V.M.
64105 Tanglewood
Bend, OR 97701
USA

Judy Scherpelz, M.S.
Director, Rocky Mountain Raptor Program
VTH, Colorado State University
200 W. Drake
Fort Collins, CO 80523
USA

Charles J. Sedgwick, B.S., D.V.M., A.C.L.A.M., A.C.Z.M.
Wildlife Clinic
Tufts University School of Veterinary Medicine
200 Westboro Road
North Grafton, MA 01536-1895
USA

T. Smit, D.V.M.
Balkeruieg 66
77 PB Witharen
The Netherlands

Stephen A. Smith, D.V.M., Ph.D.
Dept. of Pathobiology
Virginia-Maryland Regional College of Veterinary Medicine
Virginia Polytechnic Institute and State University
Blacksburg, VA 24061-0442
USA

T. J. Spierenburg, Ir.
Ingenieursburo Oranjewoud
Laboratorium Lelystad
Maerlant 13
8224 AC Lelystad
The Netherlands

David L. Suarez, D.V.M.
National Animal Disease Center
2300 Dayton Road
Ames, IA 50010
USA

Ina Westerhof, D.V.M.
Division of Avian and Exotic Animal Medicine
Dept. of Companion Animal Clinical Sciences
Utrecht University
Yalelaan 8, 3584 CM Utrecht
The Netherlands

Eric B. Wheeldon, B.V.M.S., Ph.D., M.R.C.Path.
ICI Central Toxicology Laboratory
Aderley Park
Macclesfield, Cheshire SK10 4TJ
England

Colette L. Wheler, B.Sc., D.V.M., M.Vet.Sc.
Dept. of Veterinary Internal Medicine
Western College of Veterinary Medicine
University of Saskatchewan
Saskatoon, Saskatchewan
Canada S7N 0W0

Wendy Wilkens, B.A., Vet.M.B., M.R.C.V.S.
Ditchetts Cottage
Witheridge, Tiverton
Devon EX16 8AH
England

List of Addresses of Peer Reviewers

Dr. I. K. Barker
Dept. of Pathology
Ontario Veterinary College
University of Guelph
Guelph, Ontario
Canada N1G 2W1

Dr. Peter Bloxham (M.R.C.V.S.)
5 George Street
Teignmouth
Devon TQ14 8AH
England

Dr. David Brown
College of Veterinary Medicine
University of Minnesota
St. Paul, MN 55108
USA

Dr. Susan B. Chaplin
Dept. of Biology
Mail #4369
University of St. Thomas
2115 Summit Avenue
St. Paul, MN 55105
USA

Dr. Laurel Degernes
Dept. of Companion Animal
and Special Species Medicine
College of Veterinary Medicine
North Carolina State University
Raleigh, NC 27606
USA

Dr. Josh Dein
U.S. Fish and Wildlife Service
Wildlife Health Laboratory
6006 Schroeder Road
Madison, WI 53711
USA

Dr. Gary Duke
College of Veterinary Medicine
University of Minnesota
St. Paul, MN 55108
USA

Dr. Doris Dyson
Dept. of Clinical Studies
Ontario Veterinary College
University of Guelph
Guelph, Ontario
Canada N1G 2W1

Dr. Larry Felice
College of Veterinary Medicine
University of Minnesota
St. Paul, MN 55108
USA

Dr. A. Fernando
Dept. of Pathology
Ontario Veterinary College
University of Guelph
Guelph, Ontario
Canada N1G 2W1

Miss Moya Foreman
Zoological Society of London
Regent's Park
London NW1 4RT
England

Dr. Mark Fuller
Patuxent Wildlife Research Center
U.S. Fish and Wildlife Center
Laurel, MD 20811
USA

Dr. Helga Gerlach
Mittenheimer Str. 54
D-8042 Oberschliessheim
Germany

Dr. James Gessaman
UMC 5305
Utah State University
Logan, UT 84322
USA

Dr. David Graham
Dept. of Microbiol/Parasitol
Texas A & M University
College Station, TX 77843
USA

Dr. R. Hampson
Veterinary Services Laboratory
Ontario Ministry of Ag & Food
Box 3612
Guelph, Ontario
Canada N1G 2W1

Dr. Nigel Harcourt-Brown (M.R.C.V.S.)
30 Crab Lane
Bilton, Harrogate
North Yorkshire HG1 3BE
England

Dr. Darryl Heard
5400 NW 39th Avenue, #E29
Gainesville, FL 32606
USA

Dr. Bruce Hunter
Dept. of Pathology
Ontario Veterinary College
University of Guelph
Guelph, Ontario
Canada N1G 2W1

Dr. James Kirkwood (M.R.C.V.S.)
Zoological Society of London
Regent's Park
London NW1 4RT
England

Dr. George V. Kollias
Dept. of Small Animal Clinical Sciences
College of Veterinary Medicine
University of Florida
Gainesville, FL 32610
USA

Dr. P. Laughtenslauger
Veterinary Services Laboratory
Ontario Ministry of Ag & Food
Box 3612
Guelph, Ontario
Canada N1H 6R8

Joyce Mordenti
Genentech, Inc.
460 Point San Bruno Blvd.
South San Francisco, CA 94080
USA

Dr. Maurice Nicholson (M.R.C.V.S.)
P.O. Box 77
Abu Dhabi
United Arab Emirates

Dr. Philip Ogburn
College of Veterinary Medicine
University of Minnesota
St. Paul, MN 55108
USA

Dr. Victor Perman
College of Veterinary Medicine
University of Minnesota
St. Paul, MN 55108
USA

Dr. Marc Raffe
College of Veterinary Medicine
University of Minnesota
St. Paul, MN 55108
USA

Dr. Rod Reece (M.R.C.V.S.)
Houghton Laboratory
Houghton, Huntingdon
Cambridgeshire PE17 2DA
England

Dr. Stan Wiemeyer
Patuxent Wildlife Research Center
U.S. Fish and Wildlife Center
Laurel, MD 20811
USA

Preface

This book is a refereed collection of papers presented at the Second International Symposium on Biomedical Research in Raptors. This meeting was convened in October 1988 in Minneapolis, Minnesota, USA, in conjunction with the annual meeting of the Raptor Research Foundation, Inc.

The editing of this work has been a significant endeavor by five individuals (four scientific editors and one technical editor) whose main task was to bring terminology and grammar into a conventional form—a need that derived from the multinational authorship of this work. English was not the first language for many of our participants, who included most of the world authorities and specialists in the field of raptor diseases and many of their colleagues with overlapping interests in biomedical research and field biology.

The invited papers were designed to complement the proceedings of the First International Symposium on Raptor Biomedicine (Cooper and Greenwood, 1981). As such, neither this volume nor the proceedings from the 1980 London symposium are meant to be an all-inclusive treatise of raptor diseases or biology. However, between the two works, the current status of most medically relevant issues about raptors is covered.

In the editing of this volume, we focused on avoiding tampering with content, conclusions, and, to some extent, writing style. Since the medical component of the field of raptor biomedicine is relatively embryonic, none of us felt we could render severe judgment on approaches and recommendations made by individuals. It is our hope that inconsistencies or variations in approach and interpretation may serve as challenges to the many young scientists now exploring raptor biomedicine.

In standardizing scientific names of birds, we used the checklists of the American Ornithologists' Union wherever possible. Names of medical compounds are provided generically; brand names and supply sources in the United States and Europe are included. Units of measure were standardized largely according to the style manual of the Council of Biology Educators (CBE Style Manual Committee, 1983).

Some of those who reviewed our full manuscript were critical of the emphasis on the role of falconers, and especially falconers who are veterinarians, in advancing the cause of raptor medicine. No slight to our nonfalconer participants or readers is intended by our emphasis. However, three of the four scientific editors of this work as well as many of the authors of the individual papers, both veterinarians and field biologists, can directly trace their present interest in raptor biomedicine to their interest in falconry. Partly because of the intimate knowledge of behavior we have gained by managing raptors for falconry purposes, and because of the frustration we felt when medicine could not help our own birds or those of our falconry associates that were injured or ill, we ourselves were motivated to dedicate our professional lives to studying these subjects. Beyond these associations, amateur falconers have played and continue to play key roles in the global conservation of birds of prey in concert with many biologists professionally engaged in this work. Given the negative publicity often directed at falconers, it is important to recall these roots.

Although a page of acknowledgments is included in this volume, as senior editor I would like to thank the following people in particular.

A very special thanks is given to Trudi Hahn, a copy editor for the *Star Tribune* newspaper of Minneapolis, Minnesota, and senior technical editor of this volume, whose persistence is appreciated. She was assisted by several volunteers of the Raptor Center in retyping of manuscripts and preparation of graphic materials.

Thanks also to Barbara Coffin and Julie Surma of the University of Minnesota Press, who helped us establish a publishing and distribution pathway by which we could complete this volume, and to the many others associated with the production who entered the process in the later stages.

To the many hundreds of falconers who have called at all hours of the day and night and flown their precious birds across the country to entrust them to the care of a person they knew only as a voice on the phone: Thanks for your faith; I've learned a lot.

Thanks to Don Hunter of Centerville, South Dakota, well-known North American falconer, breeder, and founder of the Raptor Research Foundation, Inc., who took the time to respond to a letter from a restless 13-year-old boy consumed with a passion for raptors and falconry and badly needing some advice on how to proceed.

Thanks to Gary Duke, to the administrators, faculty, and staff of the College of Veterinary Medicine, and to all who have seen fit over the years to provide the environment for the science of raptor biomedicine and myself to develop and flourish professionally and academically at the University of Minnesota.

I would like to thank the coeditors of this work for their diligence and patience in staying in contact with the many contributors and for carefully critiquing the manuscripts. Finally, I would like to express my most sincere gratitude to

H.H. Sheikh Hamdan Bin Rashid Al Maktoum of Dubai for his generous input of funds that supported this publishing effort.

This book has been a long time in the making. The time passage has been due in part to the factors competing for attention among the various editors and authors, and in part to the time required for the peer review. In the years since the 1988 symposium, the authors of the individual papers have responded to repeated requests to revise and continue to update their papers during the publishing process, so as to keep the information presented as current as a book-publishing schedule permits; their cooperation is gratefully acknowledged.

My coeditors and I are delighted to be able to bring this work forth at this time, and to call for the planning of the Third International Symposium on Raptor Biomedicine in the very near future.

Patrick T. Redig
The Raptor Center
University of Minnesota

References

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Many other people gave indirect help at the symposium or subsequently. We thank them also.

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Part I

Introduction

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CHAPTER 1

A Decade of Progress in Raptor Biomedicine

PATRICK T. REDIG, D.V.M., Ph.D.

The Raptor Center
University of Minnesota
St. Paul, Minnesota, U.S.A.

The treatment of sick and injured raptors is as old as falconry itself, but application of modern treatment, involving high-performance inhalant anesthetic agents, pathogen-specific antibiotic preparations, and complex surgical methods, is relatively recent. While the oral traditions handed down by Persian falconers more than a thousand years ago and the writings of European falconers of the thirteenth to the nineteenth centuries describe many of the same medical problems seen today in raptors, the remedies proffered are of little more than historical interest.

The decline of falconry for all but small enclaves such as the Olde Hawking Club of England, and the outright hostility expressed toward birds of prey at the beginning of this century in America, impeded progress in veterinary techniques for raptors commensurate with those for domestic and companion animals. The first significant modern English-language text on medical treatment of raptors was John E. Cooper's book, *Veterinary Aspects of Captive Birds of Prey* (1978). Cooper's introduction gave a detailed summary of the history of raptor medicine and provided bridging terminology between writings of authors from previous centuries and present-day practitioners.

Cooper's book appeared two years before the first international symposium on biomedical studies of raptors convened in London, England. The papers presented were published in a book titled *Recent Advances in the Study of Raptor Diseases* (Cooper and Greenwood, 1981). A second symposium was convened in the United States in 1988 at the University of Minnesota, and the chapters in this volume represent the content of that symposium. These symposia have served to keep raptor medicine and surgery globally in pace with advances occurring in other areas of veterinary science. Avian medicine experienced explosive growth in the 1980s, and raptor medicine, a forerunner to much of this growth, continues to be a major contributor as well as a beneficiary.

A profluence of several factors emerged in the past decade that propelled avian and especially raptor medicine to a highly refined specialty, including the following: (1) legislative protection of raptors throughout North America and much of Europe, (2) a resurgence in the practice of falconry, (3) the formal organization of falconers, (4) professional inputs by several falconer-veterinarians, (5) establishment of treatment protocols for several major raptor health problems, (6) research catalyzed by the need for preserving endangered species, (7) a substantial increase in the volume of published literature, (8) emergence of a professional association of avian veterinarians, (9) construction of hospitals specifically designed for treatment of raptors, and (10) increasing acceptance of a comparative medicine approach to teaching clinical medicine and pathology at veterinary schools.

In the wake of the catastrophic decline of several raptor populations, notably the peregrine falcon (*Falco peregrinus*) and the bald eagle (*Haliaeetus leucocephalus*), from pesticide poisoning in the 1940s, 1950s, and 1960s, several international legislative actions such as the Migratory Bird Treaty Act (MBTA, United States) and the Convention on International Trade in Endangered Species (CITES) were enacted by many Eastern and Western Hemisphere governments. The passage of legislation reflected a substantial change in human attitudes about raptors and the value placed on their ecological niche.

A key provision in the MBTA was to allow the use of wild and domestically produced raptors, including endangered species such as the peregrine falcon, for falconry purposes. As a result, a federal framework of regulations followed, to which falconers themselves were key contributors, that firmly established falconry in North America. At the same time, a similar renaissance was under way throughout much of Europe. More recent developments have raised the practice of falconry to a level rivaling, if not exceeding, the practice of this art at any previous time in the world's history.

Additionally, the resurgence of the practice of falconry has been enhanced by the successful captive propagation of peregrine falcons and other raptors in Europe and North America, providing birds to a greater number of falconers than at any other previous time. Falconers have become highly organized and have taken a keen interest in protecting

the health of their birds with professional assistance from the veterinary community. Health and medical matters are regularly discussed through publications such as the *Hawk Chalk* of the North American Falconers Association, the publications of the British Falconers Club, and the *Journal of Raptor Research*, published by the Raptor Research Foundation, Inc.

The active participation of several veterinarians in the sport of falconry in the past 20 years has integrated professional knowledge with firsthand experience.The measure of falconers' confidence in veterinarians has improved so that counsel and assistance are sought rather than regarded distrustfully in the event of a health crisis.

State-of-the-art approaches to diagnosis and treatment of diseases by falconer-veterinarians have removed the aura of distrust and skepticism that biased the attitudes of past falconry authors toward the subject of raptor health problems (Cooper, 1978). It is now well-known that raptors can be completely cured of diseases such as "sour crop," "bumblefoot," and even aspergillosis (Remple and Remple, 1987; Redig, 1979, 1981, 1987), and orthopedic repair of fractures occurs with predictably favorable results (Redig, 1986). Anesthesia with isoflurane is now routine and may be approached with the same expectation for uncomplicated recovery as in other areas of veterinary or human medicine. Applications of endoscopy, ultrasound evaluation, electrocardiography, and radiology are covered in this text or were addressed in the proceedings of the previous international meeting, bringing the practice of raptor medicine to an unprecedented level of sophistication.

In the late 1960s and early 1970s, field biologists and laboratory researchers began focusing on the peregrine falcon, bald eagle, and other raptorial species because of their endangered status and the role played by toxic substances and disease factors in their demise. The result has been a greater understanding of the physiology of these birds' gastrointestinal tracts, immune systems, heme synthesizing enzymes, and central nervous system sensitivities to toxic substances (Duke et al., 1976; Lawler and Redig, 1984; Lawler et al., 1991; Redig et al., 1991). Field biologists have conducted radiotelemetry studies on released raptors that were captively propagated and rehabilitated to determine their survival (Martell and Redig, 1991). Integrated programs involving captive breeding and management of free-living populations, including health monitoring, have increasingly become features of raptor conservation. Thus, a triangle consisting of falconer-veterinarians, researcher-rehabilitators, and field biologists has emerged that has vastly enhanced our ability to protect raptors, both captive and wild, from unwanted loss and destruction in many parts of the world (Jones and Owadally, 1985; Cooper, 1987).

Raptor medicine has been featured in prominent veterinary texts, including entire sections in the first two editions of Fowler's *Zoo and Wild Animal Medicine* (1978, 1986) and *Kirk's Current Veterinary Therapy XI* (Redig, 1992). *Clinical Avian Medicine and Surgery*, by Harrison and Harrison (1986), which is dedicated entirely to medical care of birds, has sections on orthopedic surgery, mycotic diseases, and anesthesia that were written on the basis of materials derived almost entirely from experiences gained in providing health care for raptors. Beebe and Webster's *North American Falconry and Hunting Hawks* (1989)—with a fifth edition expected in 1993, making it the most published reference book on falconry—contains an extensive section on medical care of raptors, as do recent British books on falconry (Woodford, 1987; Parry-Jones, 1988).

Complementary to the standard texts and publications dealing with raptor medicine has been the regular inclusion for more than two decades of specific paper sessions on this topic at professional meetings such as those of the Raptor Research Foundation (RRF) and the American Association of Zoo Veterinarians (AAZV). The information base for knowledge about medical care of raptors has broadened further and substantially because of the establishment and explosive growth of the Association of Avian Veterinarians, which was founded in the early 1980s. Now more than 2,000 members strong worldwide, the association, like its predecessors, the RRF and the AAZV, exists to foster communication among veterinarians and biologists involved in the study of birds as well as to promote research and conservation of birds. All three organizations publish quarterly journals that contain high-quality, refereed articles on topics that relate to the medicine, surgery, and pathology of raptors as well as psittacines and other species of birds.

Finally, in the last decade, hospital complexes have been constructed that are designed explicitly for the care of raptors held for falconry as well as for free-living casualty victims. Included are facilities at the University of Minnesota in the United States and falcon hospitals in Dubai and Abu Dhabi in the United Arab Emirates (see Riddle and Hoolihan, chapter 33, this volume). It seems entirely proper and fitting that the region whose people were among the earliest and most persistent in embracing the sport of falconry more than 4,000 years ago should also give birth to two of the world's most sophisticated and technologically modern health management facilities for birds of prey, the Dubai Falcon Hospital and the Abu Dhabi Falcon Research Hospital.

In the eight years between the London symposium and the Minnesota symposium, vast amounts of information were added to our biomedical knowledge base about raptors. The information was derived from a mixture of basic, field, and clinical research, benefiting all raptors, captive as well as wild populations. One of the main objectives of the Minne-

sota symposium was to bring together laboratory investigators, field biologists, and raptor clinicians to develop our information base further through the amalgamation of experiences from disparate areas. The chapters in this book deal with topics ranging from strictly clinical approaches to the treatment of specific diseases in individual birds to approaches for assessing the impact of morbidity- and mortality-causing factors on entire raptor populations.

Who should read this book? Anyone involved in the study or maintenance of raptors, whether in the wild or in captivity, requires the information compiled here. Veterinarians will benefit because of the information that is directly applicable to health care delivery. Similarly, falconers and managers of captive birds will gain useful information that will promote their understanding and practice of preventive medicine, as well as an appreciation of the knowledge base from which veterinarians operate when presented with medical problems. Field biologists and rehabilitators will acquire useful perspectives not only on clinical materials, but also on disease and environmental factors that determine survival of casualty birds and influence population dynamics of wild raptors.

Any reader will gain a sense of the tremendous dynamics and accomplishments that have occurred as a direct result of the interdisciplinary involvement of many professionals and lay persons, experts in their own right, who have contributed to the recent advances in the study of raptor diseases.

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CHAPTER 2

The Need for Closer Collaboration between Biologists and Veterinarians in Research on Raptors

JOHN E. COOPER, D.T.V.M., F.I.Biol., M.R.C.Path., F.R.C.V.S.

Department of Pathology
The Royal College of Surgeons of England
London, England

ABSTRACT: Raptor researchers come from a variety of disciplines. Regrettably, many opportunities for collaboration among them are overlooked. For example, when investigating deaths in free-living birds of prey, biologists have tended to focus on nutritional status, predation, competition, and poisons, and to ignore infectious disease and parasites. Veterinarians, for their part, have generally been concerned with pathological and microbiological findings and have not always been attentive to the roles of other factors. Biologists and veterinarians can both benefit from working more closely together. Their two fields are usually complementary and can be synergistic. Biologists can teach their veterinary counterparts a great deal about ecology, including population dynamics and behavior. Veterinarians can contribute expertise on medicine, surgery, anesthesia, and pathology. Greater cooperation between biologists and veterinarians will enhance the development and recognition of raptor biomedicine as a bona fide scientific discipline, and will do much to promote the welfare and conservation of birds of prey.

KEY WORDS: raptor, veterinarian, collaboration, raptor biologist.

The title of the symposium that led to this book included the term *biomedical*, a hybrid word—a mixture of Greek and Latin. We in Britain are accustomed to new words evolving (and on occasion being created) in North America, and some of these cause us problems, but in this case the term *biomedical* is correct: it occurs in both the *Oxford Dictionary* and *Webster's*!

Biomedical implies a blending of two disciplines, biology and veterinary medicine. Those of us who work with raptors are, of course, all biologists in the broadest sense in that we deal with living things. But in the context of this chapter, I prefer to use the term *raptor biologist* in a more specialized sense of the trained person, usually a graduate, who studies birds of prey but who does not have any specific training in medicine or pathology.

I am keen on, and familiar with, the concept of a multidisciplinary approach to research because I am a veterinarian who works in a medical college and who also maintains close links with zoologists. The Royal College of Surgeons of England (RCSE), where I am based, is surprisingly broad in its approach. The teaching and research staff of the college are from various disciplines, including human medicine, dentistry, veterinary science, biochemistry, pharmacology, physics, and engineering. They work together to unravel problems relating to human and animal health and disease. That the RCSE owns and maintains Down House, where Charles Darwin lived from 1842 to 1882, adds testimony to the college's genuine interest in the biological sciences. I have always found the atmosphere within the college stimulating, and it prompts me to encourage a multidisciplinary approach to research projects in which I am involved. In this chapter I would like to discuss ways in which greater collaboration between biologists and veterinarians might lead to important advances in raptor medicine.

Discussion

My thesis is this: raptor biomedicine is now a bona fide subject in its own right, but if it is to flourish it needs multiple input from people in disciplines other than veterinary medicine.

First of all, I would like to draw attention to two terms: *complementary* and *synergistic*. The first comes from the Latin *complementum*, meaning "completing, complementing each other." *Synergistic* is from the Greek *synergos*, and means "working together": it implies the production of a combined effect that exceeds the sum of the individual effects.

Raptor biologists and veterinarians are sometimes complementary in their work, as the chapters in this volume demonstrate, but synergism appears to be unusual. Relations between the two professions are characterized often by indifference, and occasionally by jealousy and antagonism. This lack of cooperation has three potential effects: divergence, false or misleading research results, and conditions that may compromise the welfare of the birds.

I use the term *divergence* ("genetic drift") to describe what happens when an aspect of raptor biomedicine becomes fragmented, with two or more distinct lines of development. There is a tendency for the gap to widen with time and for work to be unnecessarily repeated. An example of

this with which I have had contact concerns endoscopy, the examination of internal body organs with fiber-optic instruments. Zoologist colleagues at one research institute in Britain spent several months developing a laparoscopic technique for seabirds they were studying in the South Atlantic. They had not read journals other than those concerned with ornithology, and as a result were unaware of advances in veterinary endoscopy. The researchers needlessly repeated work on equipment selection, surgical techniques, and anesthetic procedures that had already been developed by veterinarians, and published a paper that included no references to veterinary literature (Hector, 1984).

This incident is an example of how developments in avian medicine can be hampered when people from different disciplines pull in separate directions. Awareness of mutual benefits has led to closer collaboration in recent years in the field of avian endoscopy (Brearley et al., 1991).

An important and prevalent example of *false or misleading results* concerns postmortem examinations. The diagnosis made as a result of an examination will depend on the investigations that are carried out, and these in turn hinge upon who performs the examination and on his or her investigative focus. An instance arose following the unexpected deaths of large numbers of owls at the London Zoo some years ago (Jones, 1977). Many veterinary pathologists participated in the investigations; extensive microbiological, histopathological, and other investigations were carried out, initially to no avail. It was toxicologists, involved at a later stage of the investigation, who were finally able to demonstrate that the deaths were due to chlorinated hydrocarbon pesticide poisoning.

Many surveys of mortality in birds of prey are incomplete in that they are carried out by personnel from only one discipline. As a result, wrong diagnoses are sometimes made. Pathologists may fail to look for poisons, while toxicologists do not routinely conduct microbiological analysis or histopathological examinations. The situation will be remedied only when a multidisciplinary approach becomes standard.

Finally, *the welfare of birds may be compromised* by a lack of cooperation between raptor biologists and veterinarians. Ignorance of state-of-the-art techniques for handling or treating birds can result in needless pain, injury, or death. For instance, biologists may use unsatisfactory and outdated anesthetic agents because they have not read the relevant veterinary literature or consulted veterinarians. Likewise, veterinarians may not know how to handle, house, feed, or manage birds of prey properly. We live in an age in which animal welfare is attracting much attention, and it behooves us to be sensitive to the well-being of the raptors with which we work.

Examples of potentially harmful effects deriving from lack of collaboration abound. For instance, one of the last California condors (*Gymnogyps californianus*) to hatch in the wild was killed by biologists who were attempting to weigh and measure other basic physiological parameters. They failed to recognize and appreciate the signs of handling stress the bird was exhibiting, and it died in their hands.

Proposals

The veterinarian and the biologist have much to offer each other, and through their cooperation, synergism may result. The veterinarian can benefit particularly from the biologist's knowledge of the following areas:

1. *Classification and taxonomy*: Veterinary graduates often are unfamiliar with the correct scientific terms for raptors, and frequently misidentify birds or their prey species.
2. *Behavior*: Most veterinarians still have very limited knowledge of, or training in, ethology. They will benefit greatly from the assistance of experienced biologists. Very recent publications in the veterinary literature have begun to address this deficit (Marder and Voith, 1991), but on the whole, avian species have received relatively little attention in this arena (Rosskopf and Woerpel, 1991). For the raptor biomedical professional, the falconry literature is still the most important source of information about raptor behavior.
3. *Population dynamics*: This is a field in which the expertise of field biologists is preeminent. Few veterinarians have had formal training in such matters as numbers and densities of birds or feeding strategies. The benefits of biological training are admirably demonstrated in Ian Newton's (1979) excellent book on population ecology of raptors.

It is clear to me that a veterinarian should not be pontificating on (for example) population dynamics any more than a biologist should be performing orthopedic surgery. There are many other fields in which biologists are often authorities—for example, the identification of parasites, as graphically illustrated in this volume by Cawthorne (chapter 4), the use of radiotelemetric monitoring (see Rushton and Osgood, chapter 34, this volume), and physiological assessment and physical therapy (see, respectively, Chaplin et al., chapter 29, and Martin et al., chapter 36, this volume).

How can the biologist benefit from the veterinarian?

1. *Clinical pathology*: Biologists can utilize the expertise of the veterinarian in clinical or postmortem diagnosis, particularly in the taking of blood, fecal, and biopsy samples and the interpretation of results (Cooper, 1978).
2. *Surgical management*: Few biologists would contemplate operating on a cataract, but it is surprising how many embark upon taking biopsies or performing surgical treatment of bumblefoot, even if they have little experience in surgery. If such a procedure is performed without adequate knowledge of surgical or anesthetic techniques, the bird may experience pain or unnecessary distress, or the procedure may fail entirely. In some countries, such undertakings by unlicensed individuals may be illegal (Cooper, 1987);

even if no specific legislation exists, however, increasing public concern over animal welfare dictates that biologists should take every precaution to prevent unnecessary suffering in avian patients. Veterinary involvement is clearly required, not only for performance of surgical techniques but also because of the advantages afforded by the application of new biomedical techniques—for example, new anesthetic agents developed for domesticated animals may have relevance to work with birds of prey.

Does cooperation between veterinarians and biologists really work? The answer is yes, as some scientists from both disciplines can testify. Examples of where such an approach has proved fruitful in my own career include joint work with the late Leslie Brown in Kenya on harrier hawks (*Polyboroides typus*) (Cooper, 1980), studies with other field biologists on blood parasites in Africa (Cooper, 1978), characterization of bacterial flora in peregrine falcons (*Falco peregrinus*) from Greenland (Cooper et al., 1980), collaboration with scientists of many disciplines on threatened species of Mauritius (Cooper et al., 1986), and cooperative studies with toxicologists and others in Britain on mortality factors in birds of prey (Cooper and Petty, 1988). Elsewhere, cooperation has also proved invaluable. The thrust of raptor conservation in the last 15 years has involved captive propagation, management, and release. In a number of cases this work, largely performed by biologists, has been enhanced by veterinary involvement. Examples include the following:

1. *Peregrine falcon rescue*: The Raptor Center at the University of Minnesota has rescued and recycled several peregrine falcons that became injured or ill during the hacking phase of a restoration project operated by biologists from state and federal agencies in the United States. Through immediate, proper, and expert veterinary care, these birds were returned to their hack groups within the time frame of the same release season so they could continue to develop with their own cohort.

2. *Peregrine falcon screening*: Physical examinations, blood work, and radiographs of young peregrine falcons enroute from breeder facilities to hack sites have detected problems such as severe scoliosis, osteomyelitis of vertebral bodies, coccidiosis, and trichomoniasis. As a result, the wasting of other resources that would have been consumed in the attempt to release unfit birds has been prevented. In other cases, medical treatment permitted recovery and release of birds that otherwise would have died.[1]

Conclusion

Those concerned with raptors come from many different backgrounds and disciplines. There is a need for us to recognize the special talents of the different groups and to utilize these fully. If biologists and veterinarians collaborate, much can be achieved. If, however, differences and divisions persist, research on raptors will suffer. As a result, raptor biomedicine may begin to exhibit features reminiscent of birds with a high inbreeding coefficient and lack of genetic diversity.

The breadth of interdisciplinary participation in the Minnesota symposium is an indication that research in the field of raptor biomedicine is capable of flourishing. We must ensure that it continues to grow and to metamorphose by working together, regardless of our backgrounds and training. We should aim for synergism—or perhaps it should be termed hybrid vigor—that will carry us through to the next symposium in a few years' time.

Acknowledgments

I am grateful to Dr. Patrick Redig for inviting me to present this paper at the 1988 symposium, and to the many friends and colleagues—zoologists and veterinarians, amateur and professional—who have contributed to my understanding and enjoyment of raptors. Ms. Trudi Hahn made helpful comments on an early draft of this chapter; I found that my subject matter proved easier to cover in a lecture than on paper!

Note

1. Personal communication with Dr. Patrick Redig, director, the Raptor Center, St. Paul, Minnesota.

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Part II

Pathology and Microbiology

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CHAPTER 3

Cytodiagnosis in Raptor Medicine

TERRY W. CAMPBELL, D.V.M., Ph.D.

Department of Surgery and Medicine
College of Veterinary Medicine
Kansas State University
Manhattan, Kansas, U.S.A.

ABSTRACT: Avian cytodiagnosis is a useful tool that can be developed easily by a practitioner once he or she has gained familiarity with pertinent pathology, avian clinical medicine, and techniques required for the investigative procedures. Veterinarians involved in raptor rehabilitation can use cytology as a simple, rapid, inexpensive diagnostic tool. The primary goal of the cytologist is to evaluate the cellular response in samples taken from the patient. The samples may be characterized as normal tissue or as a cellular response such as inflammation, tissue hyperplasia (or benign neoplasia), malignant neoplasia, or mixed cellular response (inflammatory and noninflammatory response). Identification of an etiologic agent, if present, should also be made. Detailed descriptions of normal and abnormal avian cellular responses (Campbell, 1988) will assist the cytologist in his or her examination of specimens obtained from raptors.

KEY WORDS: raptor, cytology, cytodiagnosis, microscopic examination.

Material and Methods

Common cytologic specimens examined in raptor medicine are those obtained from blood collection, fine-needle aspiration biopsy, contact smears, swab samples, and tracheal wash procedures. Other specimens that are less frequently obtained include those from bone marrow aspiration (or core) biopsy, abdominocentesis, sinus aspiration, conjunctival scraping, and air sac swab.

The equipment needed for cytologic evaluation of specimens includes a microscope with a good oil-immersion (100X) lens, microscope slides, coverslips, and stain. The Romanowsky-type stains are commonly used in veterinary cytology and the quick stains are especially useful under veterinary practice conditions. New methylene blue is a vital stain that can be used to evaluate cytologic specimens on air-dried or wet mount preparations. A variety of special stains are also commonly used in veterinary cytology; these include acid-fast stains for tubercle bacilli, Gram's stain for bacteria, and Macchiavello's or Gimenez stains for chlamydial inclusions.

Interpretation

Microscopic evaluation of a cytologic sample results in the classification of the cellular response into one of the following categories: normal cytology, inflammation, hyperplasia/benign neoplasia, or malignant neoplasia. Occasionally, a nondiagnostic sample may be obtained in which no interpretation is possible. The cytologist must be acquainted with the normal cytology of the particular tissue being examined in order to detect abnormal cellular responses.

The cytology of inflammatory lesions is characterized by the predominant type of inflammatory cells present. The inflammatory cells of birds, including raptors, are heterophils, eosinophils, monocyte/macrophages, lymphocytes, and plasma cells (Campbell, 1988; Hawkey and Dennett, 1989; Dieterlen-Lievre, 1988). The inflammatory lesion can be classified as heterophilic, eosinophilic, mixed-cell, or macrophagic inflammation, depending upon the predominant cell types.

Heterophilic inflammatory lesions show a predominance of heterophils (that is, greater than 70% of the inflammatory cells in the cytologic specimen). The heterophils may appear degenerate or nondegenerate, depending upon the presence of toxins, such as bacterial toxins, in the microenvironment. Degenerate heterophils may show varying degrees of nuclear hyalinization and swelling, karyorrhexis, karyolysis, and pyknosis and cytoplasmic basophilia and vacuolization.

Eosinophilic inflammation appears to be rare in birds because eosinophils may be difficult to differentiate from heterophils in the cytologic specimen using routine cytologic stains. Avian eosinophils appear to behave differently from mammalian eosinophils; therefore, avian eosinophilic inflammation may result from conditions that differ from those that produce mammalian eosinophilic inflammation (Dieterlen-Lievre, 1988).

Mixed-cell inflammation is represented by a mixture of inflammatory cells, although heterophils often predominate (that is, they are greater than 50% of the inflammatory cells). Because of the rapid influx of macrophages into in-

flammatory lesions, mixed-cell inflammation is common in birds (Montali, 1988).

The necrotic center of heterophilic inflammatory lesions produces necrotoxins that are chemotactic for macrophages, and a granuloma quickly develops. Macrophagic inflammation is characterized by a predominance of large, vacuolated macrophages that later develop into multinucleated giant cells. It is common in certain diseases of raptors, such as mycobacterial and fungal infections and cutaneous xanthomatosis. Inflammatory lesions can result from tissue invasion by living organisms (for example, bacteria, fungi, and viruses), as a response to nonliving agents (for example, traumatic, thermal, or chemical), or neoplasia. Often the etiologic agent associated with the inflammatory response can be seen in the cytologic specimen.

Tissue hyperplasia and benign neoplasia cannot be differentiated by cytology. Cytological examination of tissue hyperplasia often reveals a uniform population of cells with increased cytoplasmic basophilia and pale, vesicular nuclei. The cells have a uniform nucleus to cytoplasm (N:C) ratio. Examples of tissue hyperplasia most often encountered in raptors are the normal fibrous tissue of epithelial cell proliferation that occurs in areas adjacent to chronic inflammatory lesions.

Neoplasia is rare in raptors (Cooper et al., chapter 7, this volume). Neoplastic lesions reveal abnormal pleomorphic cells. The characteristics of the cells permit classification as follows: epithelial cell (for example, carcinoma), connective tissue cell (for example, sarcoma), round cell (for example, lymphoid neoplasia), or poorly differentiated neoplasm (characterized by abnormal cells that are difficult to classify).

Abdominal effusions occur infrequently in raptors. Hemorrhagic effusions resulting from traumatic injury in the abdomen can occur. Hemorrhagic effusions are characterized by the presence of erythrocytes and erythrophagocytosis by leukocytes. The lack of erythrophagocytosis and the presence of thrombocytes and erythrocytes are suggestive of peripheral blood contamination of the sample. Therefore, erythrophagocytosis is the key feature for the identification of a hemorrhagic effusion. Another type of abdominal effusion that occasionally occurs in raptors is an inflammatory exudate resulting from peritonitis. Transudate and modified transudate may also be present.

Inflammatory lesions involving the oral cavity of raptors are frequently seen (Cooper, 1978). Gross inspection of these lesions may reveal plaques, nodules, ulcers, and abscessation. A cytologic sample can be obtained by cotton swab, scraping, or fine-needle aspiration. A sterile microscope slide and cotton swab will allow sampling of the lesion for cytologic and microbiologic evaluations. Trichomoniasis (''frounce'') causes lesions in the upper alimentary tract of raptors. Cytological examination of these lesions reveals piriform, flagellate protozoa with anterior flagella, and prominent undulating membranes. An inflammatory response is usually present. Other disorders of the upper alimentary tract of raptors include candidiasis, capillariasis, and septic ingluvitis or stomatitis. Candida appear as narrow-based budding yeast and are usually not associated with an inflammatory response. Capillariasis may be detected by the presence of the characteristic double-operculated ova of the nematode parasite from a swab or wash sample of the esophagus or crop.

Disorders of the respiratory tract of raptors may involve the upper respiratory sinuses, trachea, syrinx, lungs, or air sacs. Septic inflammation is indicated by the presence of an inflammatory response with bacterial phagocytes. Mycotic lesions reveal fungal elements, such as hyphae, spores, and condidiophores. A macrophagic inflammatory response with giant cells is usually present with mycotic lesions. Chlamydiosis (*Chlamydia psittaci* infection) is a rarely reported disease of raptors (Halliwell and Graham, 1986). If suspected, the chlamydia inclusions are best found in sinus exudates or air sac swabs in living birds, or imprints of the spleen, liver, pericardium, and air sacs of necropsy specimens. Special stains, such as Gimenez or Macchiavello's, may aid in the identification of chlamydial inclusions.

Birds of prey are often presented with skin or subcutaneous lesions. The more common lesions are bacterial infections involving traumatized skin, poxvirus lesions, foreign-body granulomas, and hematomas. Less common are cutaneous xanthomatosis, feather cysts, and neoplasia. Bacterial infections reveal numerous inflammatory cells with leukocytic phagocytosis of the bacteria. Avian poxvirus often causes raised, scablike lesions around the eyes, beak, feet, and legs. Cytologic examination of these lesions may reveal swollen squamous epithelial cells with ballooning degeneration. The large cytoplasmic vacuoles push the nuclei to the margin of the cells. Tiny, round, pale eosinophilic inclusions may be seen within the vacuoles with Wright's stain. Foreign-body reactions reveal evidence of macrophagic or mixed-cell inflammation with giant cell formation. Lesions showing numerous erythrocytes and erythrophagocytosis are representative of hematomas. Cutaneous xanthomatosis may occur in the skin of birds where previous hemorrhage has occurred. Xanthomatosis is characterized by a thick, friable, yellow skin in which there is a mononuclear leukocyte response (primarily macrophages) with multinucleated giant cells and cholesterol crystals. Feather cysts are cystic lesions involving feather follicles that contain feathers that have failed to grow through the skin. These lesions contain keratinous debris, and cytologic examination reveals a hemorrhagic lesion in acute lesions or mixed-cell inflammation in chronic lesions.

Conjunctivitis and keratoconjunctivitis caused by bacteria (either primary or secondary) are characterized by nu-

merous inflammatory cells showing phagocytosis of bacteria and degenerate epithelial cells. Chronic lesions show an increase in mononuclear leukocytes.

Occasionally, raptors are presented with injury to joints resulting in an increase in the amount of synovial fluid. Evaluation of the synovial fluid involves a simple viscosity test, micromucin clot test, and cytologic evaluation. Septic arthritis is confirmed by the presence of inflammatory cells exhibiting bacterial phagocytosis. Traumatic arthritis may reveal numerous leukocytes (primarily heterophils) and possibly synovial lining cells. If hemorrhage has occurred, many erythrocytes and possible erythrophagocytosis will be seen. The presence of cartilage cells (chondrocytes) or osteoblasts is indicative of erosion of the articular cartilage, which may expose the underlying bone.

Cytologic evaluation of internal organs can be provided from samples obtained by biopsy in the living bird or from necropsy specimens. Normal liver cytology shows uniformly appearing hepatocytes, erythrocytes, and lymphocytes with occasional macrophages, heterophils, and plasma cells. Hepatic hematopoiesis can be seen occasionally and is detected by the presence of developing forms of the various blood cells. Inflammatory lesions involving the liver will show numerous leukocytes. These lesions can be differentiated from focal granulopoiesis by the presence of only mature leukocytes (primarily heterophils). Avian tuberculosis may result in granulomatous lesions involving the liver, spleen, intestines, and occasionally lungs. The majority of the leukocytes are mononuclear cells with multinucleated giant cell formation. The background contains numerous bacilli that do not stain with Romanowsky stains and therefore must be identified using acid-fast staining and microbial culture. Avian chlamydiosis frequently involves the liver, and cytology reveals an increase in plasma cells and macrophages. Chlamydial inclusions within macrophages appear as small intracytoplasmic blue spheres with Wright's stain and red spheres with Gimenez and Macchiavello's stains. Hepatic lipidosis is not common in raptors (Forbes and Cooper, chapter 10, this volume), but when present is characterized by swollen hepatocytes with cytoplasmic vacuolation and fat droplets in the noncellular background. Occasionally, developing stages of blood parasites (for example, *Haemoproteus, Plasmodium,* and *Leucocytozoon*) may be found in liver imprints.

Normal splenic cytology reveals numerous erythrocytes and lymphocytes. Macrophages showing various degrees of erythrophagocytosis are common. The spleen provides a good cytologic sample for the demonstration of chlamydia inclusions, systemic infections, and developmental stages of blood parasites. Excessive iron pigment and erythrophagocytosis may be seen in splenic samples from birds with hemolytic anemias.

Conclusion

Cytology can be a useful antemortem and postmortem diagnostic tool in raptor medicine. It often gives a rapid definitive or strong presumptive diagnosis that helps provide quick, specific therapy for the raptorial patient.

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CHAPTER 4

Cyst-forming Coccidia of Raptors: Significant Pathogens or Not?

RICHARD J. CAWTHORN, Ph.D.

Department of Pathology and Microbiology
Faculty of Veterinary Medicine
University of Prince Edward Island
Charlottetown, Prince Edward Island, Canada

ABSTRACT: Several genera of protozoan parasites (*Caryospora* spp., *Eimeria* spp., *Frenkelia* spp., and *Sarcocystis* spp.) use raptors as definitive hosts. Relatively few data are available on the developmental cycles and pathogenicity of these parasites in raptors. The disease "coccidiosis" in raptors is difficult to define—that is, clinical signs can be vague, and often parasitic infections are concomitant with other major illnesses in the birds.

Coccidia with heteroxenous (indirect) life cycles use predator-prey or scavenger-carrion relationships among intermediate and definitive hosts to ensure transmission of the parasites. Apparently some species of *Frenkelia* and *Sarcocystis* use cannibalism and congenital transmission to enhance vertical transmission within populations of intermediate hosts. These phenomena can increase prevalence of infection within prey populations. Infections with species of *Sarcocystis* or with *Toxoplasma gondii* can cause behavioral alterations of intermediate hosts, increasing prey susceptibility to aerial predation.

Usually, definitive hosts (i.e., raptors) of cyst-forming coccidia develop little disease when infected even after repeated experimental infections. However, some protective immunity results as manifested by shorter patent periods and reduced sporocyst or oocyst output in feces. In naturally infected free-flying birds, it is difficult to determine the health effects of coccidial infections. In rehabilitation clinics, parasitic infection is usually a complication (which may be debilitating) of unrelated illness. Stress probably exacerbates the effects of coccidial infections on raptors, as it can with other avian and mammalian hosts.

KEY WORDS: raptor, coccidia, *Sarcocystis*.

Review and Discussion of the Literature

Several genera of protozoan parasites, including species of *Caryospora*, *Eimeria*, *Frenkelia*, *Sarcocystis*, and *Toxoplasma*, use raptors as intermediate or definitive hosts. Relatively little information is available that describes the developmental cycles, systematics, pathogenesis of infection, or epidemiology of these parasites in raptors. The purpose of this review is to provide critical comments on the significance of cyst-forming coccidia as pathogens of raptors.

At present the impact of infectious (i.e., parasitic) diseases on individuals or populations of raptors in either laboratory or field situations is not well understood. However, although disease is often difficult to detect or quantify as causing mortality in wildlife populations, infection and disease have a major impact on the health status of wildlife populations (Croze, 1981; Scott, 1988). Parasitic disease can have various effects on hosts, affecting survival, reproduction, movement (patterns), population, community structure, and ecosystems (Scott, 1988). The host-parasite relationship demonstrates a continuum of effects ranging from subclinical infection to death of the host; even subtle effects (i.e., reduced bodily condition or reproductive output) can have significant impact on the population dynamics of raptors (Holmes, 1982). Numerous factors impinge on host-parasite interaction (Holmes, 1982); this relationship is difficult to monitor in raptors. A major concern is to determine whether parasitic disease causes selective mortality; if so, is the mortality additive or compensatory (Hassell et al., 1982; Holmes, 1982)? Consequently, Koch's postulates that a pathogenic agent in one individual is pathogenic in another individual must be reevaluated as they pertain to host-parasite interactions in wildlife populations to determine the role of parasitic diseases in the varying environment of wildlife populations (Hanson, 1988).

Within the phylum Apicomplexa, life cycles are similar, comprising three stages: schizogony or merogony (asexual multiplication of haploid zoites, with mitosis), gametogony (sexual multiplication producing haploid gametes, diploid zygotes), and sporogony (asexual multiplication with meiosis-mitosis to form haploid sporozoites) (Corliss, 1974; Levine, 1982). In homoxenous (direct) life cycles (see Figs. 4.1a, 4.1b, 4.1d), hosts are infected by ingesting sporulated oocysts; after excystation, sporozoites enter host cells to undergo a species-specific number of schizogonous generations. End-stage merozoites form gamonts, macrogametes

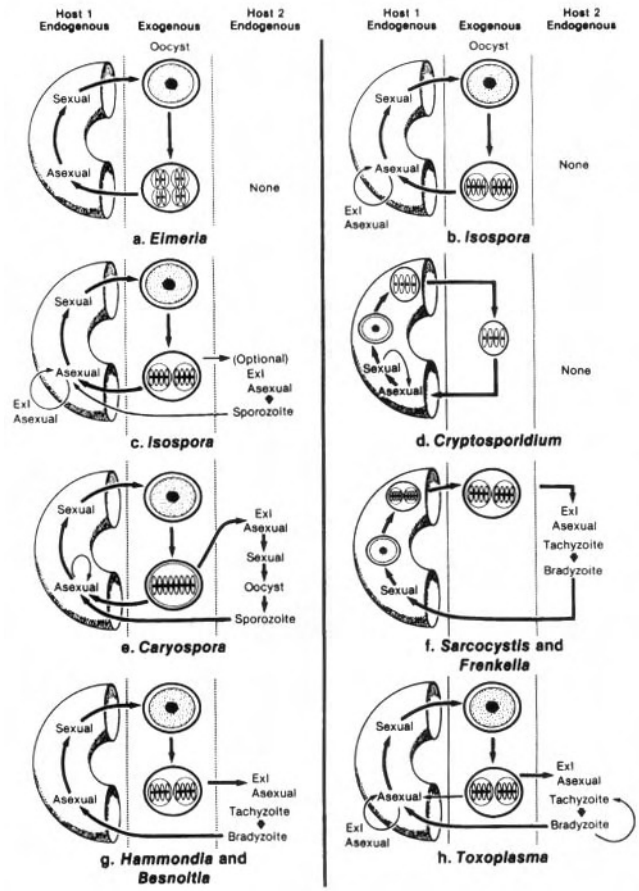

Figure 4.1. Simplified life cycles of coccidian genera. For each genus, schematic representation of the intestinal tract appears on the left under "Host 1" (definitive host); oocyst structure is in the center, under "Exogenous"; and extraintestinal stages (ExI) that develop in intermediate host(s) are listed in order of appearance at the right under "Host 2." In some genera (b, c, h) extraintestinal stages also appear in the definitive host. (Reprinted with permission from P. L. Long [ed.], *Coccidiosis of Man and Domestic Animals*. CRC Press, Boca Raton, Florida, 1990, p. 13.)

(no multiplication), and microgametes (by asexual multiplication) in new host cells. Syngamy and zygote formation occur when the motile, flagellated microgamete actively penetrates the stationary macrogamete. The oocyst, or walled zygote, undergoes sporogony either within or outside the host. In coccidia with heteroxenous (indirect) life cycles (see Figs. 4.1c, 4.1f, 4.1g, 4.1h), schizogony and cyst formation occur only in intermediate hosts, while gametogony (and also schizogony or sporogony) occur in definitive hosts (see the discussion of *Caryospora* below). The disease coccidiosis, caused by the "true" coccidia (Phylum Apicomplexa, Class Sporozoasida, Subclass Coccidiasina, Order Eucoccidiorida, Suborder Eimeriorina) (Levine, 1982) is highly variable. The various manifestations of disease are dependent on species of coccidium involved, stage(s) (i.e., schizonts versus gamonts) of parasite causing disease, age of host at infection, and strain of parasite. The disease coccidiosis can be difficult to diagnose in raptors, especially in hospitalized hosts. Clinical signs are often vague, and coccidial infections (and disease) are usually secondary to other major problems in the raptors.

Raptors can act as intermediate hosts of cyst-forming coccidia, although the infections are apparently not clinically significant. Cysts of *Sarcocystis*-like organisms (see Fig. 4.1f) have been reported in the musculature of various raptors, including the Australian black-shouldered kite (*Elanus notatus*), brown falcon (*Falco berigora*), brown goshawk (*Accipiter fasciatus*), and the great horned owl (*Bubo virginianus*) (Munday et al., 1979; Vande Vusse, 1966). There was no degeneration or inflammation surrounding sarcocysts in the myocardium of a bald eagle (*Haliaeetus leucocephalus*) (Crawley et al., 1982). However, single (or different) individuals of a given species of host can act as both intermediate and definitive hosts of species of *Sarcocystis* (Matuschka and Bannert, 1987). Thus, cannibalism or scavenging may ensure transmission of *Sarcocystis* spp. among raptors (reviewed in Cawthorn and Speer, 1990). Raptors probably act as intermediate hosts of *Toxoplasma gondii* (see Fig. 4.1h), although this is unconfirmed and untested (reviewed in Dubey and Beattie, 1988). Clinical toxoplasmosis in birds probably results from stress of captivity and handling, which facilitates reactivation of a latent infection (Parenti et al., 1986).

Raptors act as definitive hosts of various cyst-forming coccidia, including species of the genera *Caryospora*, *Eimeria*, *Frenkelia*, and *Sarcocystis* (Table 4.1). Species of *Caryospora* have uniquely facultatively heteroxenous life cycles in which schizogony and gametogony are completed in (1) the intestinal epithelium of primary hosts (predatory reptiles and birds) and (2) dermal connective tissues of secondary hosts (rodents), also liver and other tissues, as stated below (Fig. 4.1e) (Upton et al., 1986). Also, sporogony is completed in rodents, and sporozoites are often found singly in caryocysts—that is, individual sporozoites enclosed in a parasitophorous vacuole in a macrophagelike cell. Overall, species of *Caryospora* form distinct types of oocysts, sporocysts, and sporozoites in each of the primary and secondary hosts (Upton et al., 1986). Transmission can occur three ways: predator-prey via carnivorism, predator-predator directly via sporulated oocysts, and prey-prey via cannibalism.

Although more than 30 species of *Caryospora* have been described, only seven are named from birds; the remainder are named from reptiles (Table 4.1) (Upton et al., 1986). Of the avian species, four occur in raptors: *C. bubonis* in great horned owls; *C. falconis* in peregrine falcons (*Falco peregrinus*); *C. henryae* in black kites (*Milvus migrans*), eagle owls (*Bubo bubo*), northern hobby falcon (*F. subbuteo*), Eu-

Table 4.1
Named species of coccidia with raptors as definitive hosts

Species	*Definitive host*	*Intermediate host*
Caryospora bubonis	great horned owl (*Bubo virginianus*)	house mouse (*Mus musculus*)
C. falconis	peregrine falcon (*Falco peregrinus*)	?
C. henryae	black kite (*Milvus migrans*) eagle owl (*Bubo bubo*) northern hobby falcon (*F. subbuteo*) European kestrel (*F. tinnunculus*) turkey vulture (*Cathartes aura*)	?
C. strigis	barn owl (*Tyto alba*)	?
Eimeria accipitris	European sparrowhawk (*Accipiter nisus*) lesser kestrel (*F. naumanni*) booted eagle (*Hieraaetus pennatus*)	
E. asturi	goshawk (*Accipiter gentilis*)	
E. atheni	spotted owlet (*Athene brama*)	
E. bubonis	great horned owl eagle owl	
E. spectytoi	burrowing owl (*Speotyto cunicularia*)	
E. strigis	tawny owl (*Strix aluco*)	
Frenkelia microti	buzzard (*Buteo buteo*) red-tailed hawk (*Buteo jamaicensis*)	various rodents, i.e., short-tailed vole (*Microtus agrestis*)
E. glareoli	buzzard	bank vole (*Clethrionomys glareoli*)
Sarcocystis accipitris	goshawk	passeriform birds
S. cernae	European kestrel	vole (*Microtus arvalis*)
S. dispersa	barn owl masked owl (*T. novaehollandiae*) long-eared owl (*Asio otus*)	house mouse
S. rauschorum	snowy owl (*Nyctea scandiaca*)	varying lemming (*Dicrostonyx richardsoni*)
S. scotti	tawny owl	house mouse
S. sebeki	tawny owl	house mouse wood mouse (*Apodemus sylvaticus*)

ropean kestrels (*F. tinnunculus*), and turkey vultures (*Cathartes aura*); and *C. strigis* in barn owls (*Tyto alba*). Great horned owls have been reported as natural and experimental primary hosts of *Caryospora bubonis* (Cawthorn and Stockdale, 1982a). In direct (owl-to-owl via sporulated oocysts) transmission studies, the prepatent period was 12 days, with maximal oocyst production at 12 to 17 days after infection and a patent period of 4 to 11 days. Schizogony and gametogony occurred in villar epithelial cells of the posterior third of the small intestine; sporogony was exogenous. In indirect transmission studies, mice (*Mus musculus*) were suitable secondary hosts. Pyogranulomas were observed primarily in liver tissues and occasionally in the brain and lungs of mice. However, dermal connective tissues were not examined and the stage of parasite was not identified (Cawthorn and Stockdale, 1982a). Chickens (*Gallus gallus*) were not suitable secondary hosts. In great horned owls fed mice infected with *C. bubonis*, the prepatent period was shorter at 8 to 10 days with maximal oocyst production at 10.5 to 11.5 days after infection and a patent period of 5 to 11 days. None of the great horned owls was ill while infected with *C. bubonis*. Great horned owls likely have little contact with their own feces; consequently, transmission of *C. bubonis* would be based on predator-prey relationships (Cawthorn and Stockdale, 1982a).

More than 1,160 species of *Eimeria* have been described from various hosts, including raptors (reviewed in Levine, 1988). Of the approximately 160 species occurring in avian hosts, six occur in raptors: *E. accipitris* in European sparrowhawks (*Accipiter nisus*), lesser kestrels (*Falco naumanni*), and booted eagles (*Hieraaetus pennatus*); *E. asturi* in goshawks (*Accipiter gentilis*); *E. atheni* in spotted owlets (*Athene brama*); *E. bubonis* in great horned owls and eagle owls; *E. speotytoi* in burrowing owls (*Speotyto cunicularia*); and *E. strigis* in tawny owls (*Strix aluco*) (Table 4.1) (Levine, 1988). *Eimeria bubonis* utilizes great horned owls as natural and experimental definitive (i.e., primary) hosts (Fig. 4.1a) (Cawthorn and Stockdale, 1982b). In direct transmission studies in owls, the prepatent period was 5 to 6 days; maximal oocyst production occurred 24 to 48 hr after infections became patent, with highly variable patent periods (as short as 4 to 6 days). Schizogony and gametogony occurred in villar epithelial cells of the anterior half of the

small intestine. Sporogony was exogenous. No illness was observed in owls infected with *E. bubonis*. In indirect transmission studies, chickens and mice were not suitable paratenic (i.e., secondary) hosts (Cawthorn and Stockdale, 1982b). Although pyogranulomas were observed in murine liver, parasites were not found and transmission attempts to owls were unsuccessful. However, as discussed above with *Caryospora bubonis*, coccidia of raptors are more likely transmitted in nature via carnivory of various rodents.

Only two species of *Frenkelia* have been named, both from raptors (Table 4.1) (reviewed in Dubey et al., 1989). Species of *Frenkelia* have obligatorily heteroxenous life cycles (Fig. 4.1f). Gametogony and sporogony occur in the intestine of definitive hosts, raptors. In intermediate hosts, small rodents, schizogony occurs in hepatocytes and Kupffer cells; cyst formation (containing metrocytes and bradyzoites—only the latter are infectious to definitive hosts) occurs in neural tissues (primarily the brain) (Frenkel, 1977). Transmission mostly occurs via carnivorism (predator-prey relationships), although congenital (i.e., transplacental) transmission and transmission by cannibalism (among intermediate hosts) can occur (Krampitz et al., 1976; Tadros and Laarman, 1976).

Frenkelia microti utilizes buzzards (*Buteo buteo*) as natural and experimental definitive hosts and red-tailed hawks (*B. jamaicensis*) as experimental definitive hosts (Tadros and Laarman, 1982). Recently, *Frenkelia*-like sporocysts were reported from a naturally infected red-tailed hawk in the United States (Lindsay et al., 1987). Intermediate hosts are numerous species of small rodents (reviewed in Dubey et al., 1989). In experimentally infected buzzards, the prepatent period was 7 to 9 days, with a patent period to 60 days (Krampitz and Rommel, 1977; Tadros and Laarman, 1982). None of the buzzards became ill. In the short-tailed vole (*Microtus agrestis*) schizogony occurred in hepatic tissues at 5 to 8 DPI. Schizogonic development was accompanied by transient necrosis and inflammation of the liver, lymphoid hyperplasia, and inflammation of the heart, pulmonary veins, skeletal musculature, and brain (Geisel et al., 1979). Cysts (containing only metrocytes) were present as early as 23 DPI. Developmental time to infectious bradyzoites is unknown; cysts become macroscopic (to 1 mm diameter).

Frenkelia glareoli also uses buzzards as natural and experimental definitive hosts; however, this species has a narrow range of intermediate hosts, developing only in bank voles (*Clethrionomys glareoli*) (Krampitz et al., 1976; Rommel et al., 1977). In buzzards the prepatent period was 7 to 9 days, with a patent period of 5 to 57 days (Rommel and Krampitz, 1975). Gametogony occurred in epithelial cells of the anterior half of the small intestine, and oocyst formation and sporogony occurred in the lamina propria of the intestine. None of the buzzards became ill. In bank voles, schizogony occurred in hepatocytes at 5 to 8 DPI. Schizogonic development of *F. glareoli* was accompanied by focal hepatic necrosis, splenomegaly, lymphoid hyperplasia, and inflammation of the liver, heart, and brain (Geisel et al., 1977). Cyst formation (containing only metrocytes) was evident 18 DPI (Tadros and Laarman, 1982). Developmental time to infectious bradyzoites was undetermined; cysts were 360 μm in diameter at 180 DPI (Tadros and Laarman, 1982). Cyst formation caused encephalitis and disseminated focal necrosis associated with tissue compression (Geisel et al., 1977).

More than 130 species of *Sarcocystis* have been described from various intermediate and definitive hosts (birds, fish, mammals, and reptiles); life cycles are known for approximately 60 species (Fig. 4.1f) (reviewed in Cawthorn and Speer, 1990; Dubey et al., 1989; Levine, 1986). Species of *Sarcocystis* have obligatorily heteroxenous life cycles. Gametogony and sporogony occur in the intestine of definitive hosts. In intermediate hosts, one or more generations of schizogony occur in various tissues; sarcocyst formation (containing metrocytes and bradyzoites—the latter infectious to definitive hosts) occurs primarily in muscle and occasionally in neural tissue (Dubey et al., 1989). Transmission occurs primarily via carnivorism (predator-prey relationships) or scavenging. A given host species can act as both intermediate and definitive host of a *Sarcocystis* sp. (Cawthorn and Speer, 1990; Matuschka and Bannert, 1987).

Relatively few species of *Sarcocystis* have been transmitted using raptors as definitive hosts (Table 4.1). *Sarcocystis accipitris* uses goshawks as definitive hosts and passeriform birds as intermediate hosts (Cerna and Kvasnovska, 1986). As reviewed in Cawthorn et al. (1984), the following species utilize small rodents as intermediate hosts with various raptors as definitive hosts: *S. cernae*, European kestrel as definitive host; *S. dispersa*, barn owl, masked owl (*T. novaehollandiae*), long-eared owl (*Asio otus*); *S. rauschorum*, snowy owl (*Nyctea scandiaca*); *S. scotti*, tawny owl; and *S. sebeki*, tawny owl. Additionally, a *Sarcocystis* sp. has been described with northern saw-whet owls (*Aegolius acadicus*) as definitive hosts and deer mice (*Peromyscus maniculatus*) as intermediate hosts (Espinosa et al., 1988). A *Sarcocystis* sp. uses cape vultures (*Gypes coprotheres*) and white-backed vultures (*G. africanus*) as definitive hosts and impala (*Aepyceros melampus*) as intermediate hosts, illustrating transmission by scavenger-carrion relationships (Markus et al., 1985).

Sarcocystis rauschorum uses snowy owls as natural and experimental definitive hosts and varying lemmings (*Dicrostonyx richardsoni*) as experimental intermediate hosts (Cawthorn et al., 1984). In experimental studies with snowy owls, the prepatent period was 7 days and the patent period 12 to 19 days. Although the location of gametogony

was not determined, sporogony occurred in the lamina propria throughout the length of the small intestine. Owls were not clinically ill, although repeated infections with bradyzoites of *S. rauschorum* did produce patent infections. At necropsy, there were no lesions grossly or histologically (Cawthorn and Brooks, 1985a). As reviewed in Cawthorn et al. (1984), the pathogenicity of *Sarcocystis* spp. to raptors as definitive hosts is unclear. Tadros and Laarman (1982) strongly suggest that *Sarcocystis* spp. may be pathogenic (lethal) in raptors, especially *S. cernae* in kestrels and *S. sebeki* in tawny owls. These authors indicate the parasites to be pathogenic in both juvenile and adult birds. Similarly, young, emaciated snowy owls may be found at roadside locations, outside the normal winter range of the birds, with heavy infections with *S. rauschorum*, especially in years of low prey populations.

Apparently, *S. rauschorum* has a relatively narrow range of intermediate hosts, developing only in varying lemmings (one of six species of rodents tested) (Cawthorn et al., 1984). Overall, species of *Sarcocystis* with raptor-rodent cycles have a broader range of definitive hosts than intermediate hosts. In lemmings infected with *S. rauschorum* there is one schizogonous generation, which occurs in hepatocytes at 4 to 6 DPI. Sarcocyst formation begins, in striated muscle primarily, at 9 DPI (Cawthorn and Brooks, 1985b). Bradyzoites are present as early as 21 DPI (Friesen et al., 1989). Lethal doses of sporocysts (> 500) were relatively low in lemmings; all animals became moribund and died within 12 hr of becoming clinically ill at 5 to 6 DPI. Hepatic necrosis and purulent inflammation began 4.5 DPI, peaking at 6 DPI (time of maximum hepatic schizogony), and subsiding at 11 DPI; hepatic normality was apparent 15 DPI (Stackhouse et al., 1987). Lemmings died from hepatic insufficiency or release of toxic parasite metabolites when schizonts ruptured. Sarcocyst formation elicited purulent inflammation, necrosis, and sarcolemmal proliferation in striated and cardiac muscle beginning 9 DPI. Lesions persisted to 42 DPI. Additionally, unassociated with parasitic stages, multifocal nonsuppurative meningoencephalitis developed in several lemmings 11 to 15 DPI. Apparently the latter lesions may increase lemming susceptibility to aerial predation by enhancing inappropriate exploratory activity and lessening the fear response (Quinn et al., 1987). Overall, *Sarcocystis* spp. with raptor-rodent cycles develop very rapidly in intermediate hosts to infectious bradyzoites and relatively quickly to sporocysts in definitive hosts. Consequently, this rapid development could ensure parasite transmission between short-lived, relatively mobile prey as intermediate hosts and long-lived, highly mobile predators as definitive hosts (Cawthorn and Brooks, 1985b).

Although most cyst-forming coccidia have little effect on their definitive hosts, the parasites can cause illness of varying degrees and modify behavior in intermediate hosts. Behavioral deficits of mice infected with *Toxoplasma gondii* could enhance transmission of the parasite to terrestrial or aerial predators (Hay and Hutchison, 1983). Other genera of cyst-forming coccidia may have similar effects on their intermediate hosts.

Apparently, clinical coccidiosis is uncommon in free-living birds of prey. The following measures have been proposed to prevent coccidiosis in captive birds: regular cleaning of mews to prevent buildup of feces; locating perches and feeding platforms to minimize the birds' contact with feces; regular cleaning of the feeding platform to prevent fecal buildup and fecal contamination of feed; routine examination (for parasites) of mutes of breeding birds prior to breeding season, and treatment of birds infected with coccidia; and regular visual examination of mutes of young birds and microscopic examination of feces when mutes are watery, mucoid, or bloody. Coccidiosis can be treated using sulfadimethoxine a at a dosage of 25-55 mg/kg body weight once daily for 5 to 7 days.[1] In addition to treatment of birds, the mews must be thoroughly cleaned to prevent oocyst buildup.

Conclusion

Overall, most cyst-forming coccidia of raptors are not significant pathogens of the birds. However, although we recognize that parasites can be lethal to individual birds, the impact of parasites on the population dynamics of raptors has not been well studied. The effects of stress on parasitic infection in birds is not well understood; conversely, we also understand little about the effects of parasitic stressors on birds (Esch et al., 1975; Ould and Welch, 1980). Parasite burdens are often increased, and perhaps immunological responses are impaired, in stressed birds. Consequently, much remains to be studied of the relationships between cyst-forming coccidia and their raptor hosts.

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Note

1. Personal communication with Dr. B. Hunter, director of the Raptor Clinic, University of Guelph, in Ontario, Canada.

Product List

[a]Bactrovet, Pitman-Moore, 421 East Hawley, Munderlein, Illinois 60060 USA

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CHAPTER 5

Diagnosis and Treatment of Helminths in Birds of Prey

STEPHEN A. SMITH, D.V.M., Ph.D.

Department of Pathobiology
Virginia-Maryland Regional College of Veterinary Medicine
Virginia Polytechnic Institute and State University
Blacksburg, Virginia, U.S.A.

ABSTRACT: Both wild-caught and captive Falconiformes and Strigiformes are commonly found to be infected with internal helminths. The helminths include nematodes, digenetic trematodes, cestodes, and acanthocephalans. This chapter presents the most common helminths known to occur in North American birds of prey, and discusses currently available anthelmintics.

KEY WORDS: internal parasite, helminth, raptor, nematode, cestode, trematode, acanthocephalan, anthelmintic.

Birds of prey are commonly parasitized by a wide variety of helminths, including numerous species of nematodes, trematodes, cestodes, and acanthocephalans. Infections usually cause little or no distress to healthy individuals in the wild. However, parasites can become a significant problem in birds held in prolonged captivity and confined housing, as well as in birds that have recently come into captivity that are stressed by illness, injury, and/or acclimatization to new surroundings. Thus, it is important to recognize and control those helminth species capable of producing disease in the host. The purposes of this chapter are (1) to discuss the antemortem and postmortem diagnosis of helminth parasites as it pertains to birds of prey, (2) to summarize the results of a fecal survey of birds of prey from Ohio, (3) to review briefly the literature concerning the most common helminths of North American birds of prey, and (4) to mention some of the anthelmintics and treatment regimens commonly used to eliminate parasites from birds of prey.

Materials and Methods

Fresh fecal material (mutes) from 115 Falconiformes (representing 15 species) and 102 Strigiformes (representing 8 species) were collected for analysis. Species of birds of prey and numbers examined are listed in Table 5.1. Samples were collected from birds housed in private and state-run facilities throughout Ohio and from birds presented to the Raptor Rehabilitation Program at Ohio State University College of Veterinary Medicine. Birds were categorized as being either free-living or captive, with *captive* defined as any bird that was captive-bred, captive-reared, or in captivity longer than 6 mon. Standard techniques of flotation—using a saturated solution of sodium nitrate—and sedimentation were used to examine each sample (Sloss, 1970, pp. 20-21). All slides were examined at 100× for the presence of helminth ova.

Table 5.1
Falconiformes and Strigiformes examined by fecal analysis

Species	*Number*
Falconiformes (15 species)	
Red-tailed hawk (*Buteo jamaicensis*)	47
Rough-legged hawk (*Buteo lagopus*)	5
Broad-winged hawk (*Buteo platypterus*)	4
Red-shouldered hawk (*Buteo lineatus*)	2
Harris's hawk (*Parabuteo unicinctus*)	2
American kestrel (*Falco sparverius*)	24
Prairie falcon (*Falco mexicanus*)	1
Merlin (*Falco columbarius*)	1
Cooper's hawk (*Accipiter cooperii*)	7
Sharp-shinned hawk (*Accipiter striatus*)	2
Turkey vulture (*Cathartes aura*)	8
Northern harrier (*Circus cyaneus*)	1
Bald eagle (*Haliaeetus leucocephalus*)	7
Golden eagle (*Aquila chrysaetos*)	3
Osprey (*Pandion haliaetus*)	1
Total	115
Strigiformes (eight species)	
Great horned owl (*Bubo virginianus*)	44
Screech owl (*Otus asio*)	30
Barn owl (*Tyto alba*)	11
Barred owl (*Strix varia*)	10
Short-eared owl (*Asio flammeus*)	3
Saw-whet owl (*Aegolius acadicus*)	2
Snowy owl (*Nyctea scandiaca*)	1
Burrowing owl (*Speotyto canicularia*)	1
Total	102

Results

Table 5.2 summarizes the results of the fecal survey, based on the free-living or captive status of the bird. Of the total 115

Table 5.2
Helminths found in fecal samples of free-living and captive birds of prey

	infected		*Nematoda*		*Trematoda*		*Cestoda*		*Acanthocephala*	
	n	%	*n*	%	*n*	%	*n*	%	*n*	%
Falconiformes										
Free-living (n = 71)	50	70	42	59	33	46	3	4	1	1
Captive (n = 44)	12	27	12	27	4	9	0	0	0	0
Total (n = 115)	62	54	54	47	37	32	3	3	1	1
Strigiformes										
Free-living (n = 72)	61	85	58	81	26	36	5	7	4	6
Captive (n = 30)	7	23	6	20	2	7	1	3	0	0
Total (n = 102)	68	67	64	63	28	27	6	6	4	4

Table 5.3
Nematodes of free-living and captive birds of prey

	Capillaria spp.		*Ascarids*		*Spirurids*		*Syngamus* sp.		*Serratospiculum* sp.	
	n	%	*n*	%	*n*	%	*n*	%	*n*	%
Falconiformes										
Free-living (n = 71)	38	54	15	21	11	15	1	1	1	1
Captive (n = 44)	9	20	0	0	5	11	0	0	0	0
Total (n = 115)	47	41	15	13	16	14	1	1	1	1
Strigiformes										
Free-living (n = 72)	56	78	22	31	9	13	4	6	0	0
Captive (n = 30)	2	7	2	7	4	13	0	0	0	0
Total (n = 102)	58	57	24	24	13	13	4	4	0	0

Falconiformes and 102 Strigiformes examined, 62 (54% of the total) and 68 (67%), respectively, were found to be infected with one or more classes of helminth parasite. In both the Falconiformes and Strigiformes, helminth parasites were more common in the free-living than in the captive birds.

In the Falconiformes, nematodes were the most common helminth, appearing in 54 (47%) of the birds. Nematode eggs observed during fecal analysis were further classified on the basis of egg morphology into the following groups: *Capillaria* spp., ascarids, spirurids, *Syngamus* sp., and *Serratospiculum* sp. The results for both the Falconiformes and Strigiformes are shown in Table 5.3. In the Falconiformes, *Capillaria* spp. were found in 47 (41%) of the birds examined. Differentiation of oral from intestinal infections of *Capillaria* spp. was impossible by fecal analysis because all capillarids produce a similar lemon-shaped double-operculate egg (Fig. 5.1, part 1). Scrapings of suspect oral lesions sometimes aided in the identification of oral capillariasis by demonstrating the typical capillarid egg in a clump of mucus (Fig. 5.1, part 2). Ascarid ova were found in 15 (13%) of the Falconiformes examined. Two types of ascarid ova were observed: one resembling the classical ascarid ovum of *Ascaridia* spp. (Fig. 5.1, part 3), and the other a bipolar ovum typical of *Porrocaecum* spp. (Fig. 5.1, part 4). The small, thin-shelled, larvated ova of spirurid nematodes were found in 16 (14%) of the Falconiformes (Fig. 5.2, part 5). The large bipolar-operculate ovum of *Syngamus* sp. was found in only 1 (1%) free-living American kestrel

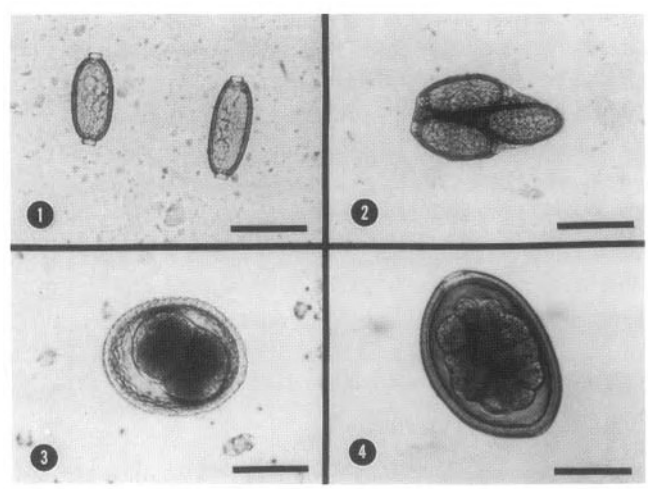

Figure 5.1. (1) *Capillaria* sp. ovum in fecal material from a red-tailed hawk. (2) *Capillaria* sp. ova in an oral scraping from a great horned owl. (3) *Ascaridia* sp. ovum in fecal material from an osprey. (4) *Porrocaecum* sp. ovum in fecal material from a red-tailed hawk. Bar = 50 μm in each section of the figure.

(*Falco sparverius*) (Fig. 5.2, part 6), and the very small, thin-shelled ovum of *Serratospiculum* sp. was found in a single free-living prairie falcon (*Falco mexicanus*) (Fig. 5.2, part 7).

Trematodes were the second most prevalent group of helminths in the Falconiformes, occurring in 37 (32%) of the birds. The large single-operculate ova of strigeid and

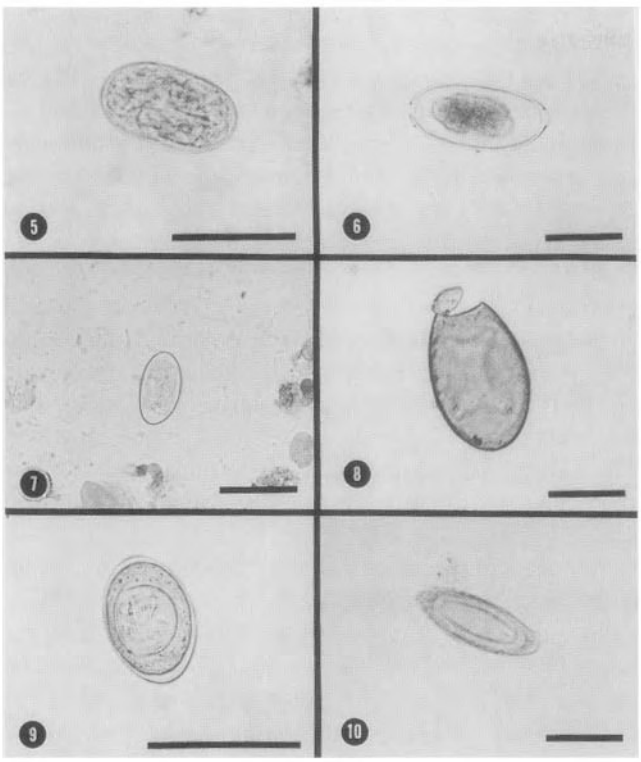

Figure 5.2. (5) Larvated ovum of a spirurid "stomach worm" in fecal material from a red-tailed hawk. (6) *Syngamus* sp. ovum in fecal material from an American kestrel. (7) Larvated ovum of *Serratospiculum* sp. in fecal material from a prairie falcon. (8) Trematode ovum with attached operculum in fecal material from a great horned owl. (9) Cestode ovum in fecal material from a screech owl. (10) Acanthocephalan ovum in fecal material from a red-tailed hawk. Bar = 50 μm in all sections of the figure.

diplostomatid trematodes were best identified during fecal analysis using a sedimentation technique (Fig. 5.2, part 8). The smaller ova of dicrocoelid trematodes were not found during fecal analysis of any of the birds examined. Cestodes and acanthocephalans were found to be rare in the Falconiformes examined, occurring in 3 (3%) and 1 (1%) of the birds, respectively. Cestode ova were of the typical taeniad type, being spherical and containing a hexacanth embryo with three pairs of hooks (Fig. 5.2, part 9). Ova of acanthocephalan parasites (Fig. 5.2, part 10) were found in fecal material from a single red-tailed hawk (*Buteo jamaicensis*) and typically had thick outer eggshells surrounding the acanthor larvae.

Among the Strigiformes, 64 (63%) of the birds were found to be infected with nematodes. *Capillaria* spp. were found in 58 (57%) of the owls examined, ascarids in 24 (24%), spirurids in 13 (13%), and *Syngamus* sp. in 4 (4%). A total of 28 (27%) of the owls were found to be infected with trematodes. As in the Falconiformes, cestodes and acanthocephalans were found to be rare in the Strigiformes, occurring in only 6 (6%) and 4 (4%) of the birds, respectively.

Discussion

Antemortem identification of helminth parasites from fecal material is best accomplished using both flotation and sedimentation techniques. Flotation techniques, using either a saturated solution of sucrose or sodium nitrate, are effective in the identification of a wide variety of parasites, including nematodes, cestodes, and acanthocephalans. Sedimentation is the technique of choice for determining the presence of trematode eggs in the feces of a host. While neither technique determines the intensity or pathogenicity of a parasitic infection, both provide information on the types of helminths present. Based on information obtained with these techniques, the clinician must decide whether or not treatment with an appropriate anthelmintic is necessary.

Postmortems for internal parasites should always include evaluation of the dead bird by standard necropsy procedures and evaluation by histopathology. The entire gastrointestinal tract should be opened and examined grossly, with portions examined with a dissection microscope to observe those helminths too small to be seen with the naked eye. Additional organs that should be included in the gross examination for parasites include the air sacs, heart, eyes, trachea, and bile ducts. Histopathology can then be used as an adjunct to the gross necropsy to visualize and identify parasites that reside in the tissue. All of these techniques help provide a basis for determining whether a particular parasite may be causing harm to the bird.

In the fecal survey of raptors from Ohio, 54% of the combined free-living and captive Falconiformes and 67% of the combined free-living and captive Strigiformes were parasitized by helminths. Infection rates in free-living Falconiformes and Strigiformes were higher than infection rates in captive raptors. Although the infection rates were different, the proportionate distribution of the four major groups of helminths was similar between the free-living and captive birds of prey. In addition, there was little evidence—except in the case of two juvenile great horned owls (*Bubo virginianus*) that died of heavy ascarid infections—that helminths were causing any significant pathology other than unthriftiness in any of the birds of prey examined. However, a complete postmortem would have made possible a more accurate assessment of the lesions caused by these helminths.

Common Helminths of North American Birds of Prey

Nematodes are the most common, most diverse, and most potentially pathogenic helminth parasite affecting birds of prey. The most common nematodes infecting raptors include capillarids, ascarids, spirurids, and tracheal and air sac nematodes. *Capillaria* sp. (threadworms) appear to be the most common type of nematode in both captive and free-

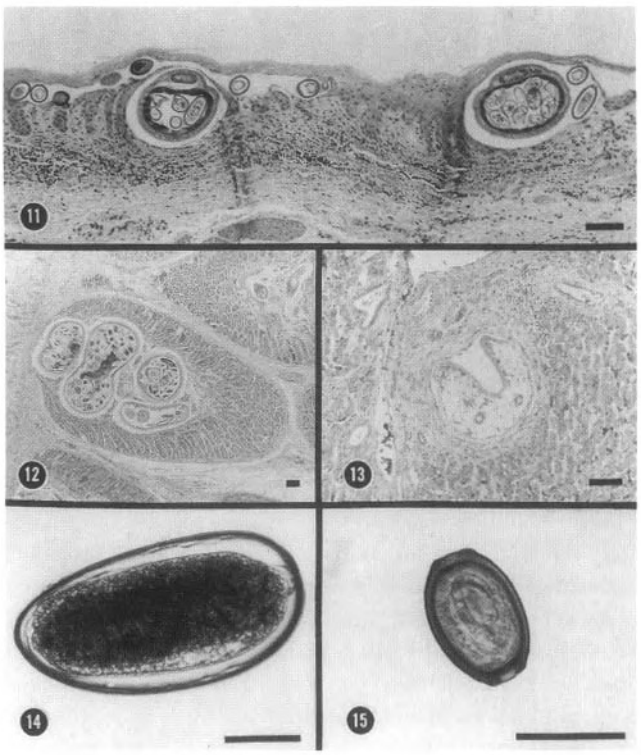

Figure 5.3. (11) *Capillaria* sp. adults and ova embedded in epithelium of the esophagus from a red-tailed hawk; hematoxylin and eosin stain. (12) *Microtetrameres* sp. in glands of proventriculus from a red-tailed hawk; hematoxylin and eosin stain. (13) Unidentified trematode in the bile duct of an immature screech owl; hematoxylin and eosin stain. (14) Unidentified mite egg in fecal material from a red-tailed hawk. (15) Larvated ovum of *Trichosomoides crassicauda*, a pseudoparasite in fecal material from a great horned owl. Bar = 50 μm in all sections of the figure.

living birds of prey. Included in this genus are species that infect the mouth, oropharynx, esophagus, crop, small intestine, and cecum of raptors. Intestinal infections by *Capillaria* spp. are usually asymptomatic, but birds with heavy infections in the intestinal tract may demonstrate clinical signs of diarrhea, anorexia, emaciation, and listlessness. Other species of capillarids can produce significant tissue damage by burrowing into the mucosal lining of the mouth, oropharynx, esophagus, and crop, causing varying degrees of inflammation and edema (Fig. 5.3, part 11).

Cooper (1969) attributed two cases of "frounce" (a disease characteristically caused by the protozoan *Trichomonas* sp.) in falcons to *Capillaria contorta*. He reported the presence of a white necrotic exudate and diphtheritic membranes in the buccal cavity and pharynx of a peregrine falcon (*Falco peregrinus*) and a red-headed merlin (*Falco chicquera ruficollis*) caused by these parasites. A similar observation was made by Trainer et al. (1968), who found a "caseous growth" in the throat of two gyrfalcons (*Falco rusticolus*) to be the result of a *Capillaria* sp. infection. Clausen and Gudmundsson (1981), in an examination of the cause of death of 36 gyrfalcons from Iceland, found 13 (36%) had died of an upper alimentary tract infection caused by *C. contorta*.

Ascarids are relatively common in birds of prey, and include the genera *Ascaridia*, *Porrocaecum*, and *Contracaecum*. These are the largest nematodes infecting raptors and generally inhabit the small intestine, but may also be found in the proventriculus, ventriculus, and large intestine. In small numbers they usually are not pathogenic, causing only occasional unthriftiness and weight loss. However, they can produce overt clinical disease and even death if their numbers are sufficiently large to cause intestinal obstruction (Wehr, 1971). In addition, perforation of the small intestine has been observed in two juvenile great horned owls in which each bird had more than 120 mature ascarids in the ventriculus and small intestine (S. A. Smith, unpublished data).

A number of species of spirurid "stomach worms" occur in the lumen and submucosa of the proventriculus and ventriculus of raptors. *Habronema* sp. was recorded from the ventriculus of hawks (Chandler, 1941), kestrels (Johnston and Mawson, 1941), eagles (Kocan and Locke, 1974), and owls (Ortlepp, 1934), while *Hatertia* sp. was found in the proventriculus of an immature prairie falcon (Ward and Fairchild, 1972). Several species of *Microtetrameres* were reported in the proventriculus of the great horned owl, goshawk (*Accipiter gentilis*), and golden eagle (*Aquila chrysaetos*) (Schell, 1953), and snowy owl (*Nyctea scandiaca*) (Mawson, 1956; Ramalingam and Samuel, 1978). Red-tailed hawks are commonly infected with an unidentified species of *Microtetrameres* in the proventriculus (S. A. Smith, unpublished data). The adult female worm resides in the glands of the proventriculus, apparently causing little or no inflammatory reaction (Fig. 5.3, part 12).

Tracheobronchial infections with *Syngamus* sp. and *Cyathostoma* sp. in hawks and owls have been reported (Cooper, 1978, pp. 82-96; Hunter et al., chapter 11, this volume), but their prevalence in free-living North American raptors appears to be very low. These parasites are usually few in number, but can cause inflammation and sometimes partial or complete obstruction of the trachea, producing clinical signs of dyspnea and open-mouthed breathing. Younger birds and smaller-sized birds are generally more severely affected than are larger adult birds.

Serratospiculum sp. in falcons is often found coiled within the air sacs, on serous membranes of the viscera, or in the connective tissue of the abdominal or thoracic cavity. *S. amaculata* has been reported from the peregrine falcon and prairie falcon (Wehr, 1938; Bigland et al., 1964; Ward and Fairchild, 1972; Croft and Kingston, 1975), the bald eagle (*Haliaeetus leucocephalus*) (Kocan and Locke, 1974), and, most recently, a Cooper's hawk (*Accipiter cooperii*) (Sterner and Espinosa, 1988). This parasite does not appear

to be pathogenic in low numbers. However, Bigland et al. (1964) reported five cases of *S. amaculata* in prairie falcons in which this parasite was considered the major contributing cause of death. Smears from necrotic foci in the liver, spleen, and heart, and of necrotic lesions in the crop and esophagus, all contained large numbers of larvated ova and free larvae of *S. amaculata*. Ward and Fairchild (1972) reported several cases of *Serratospiculum* sp. infection in prairie falcons. In one case, the air sacs of a dyspneic bird were filled with hundreds of the adult nematodes. Clinical signs may include respiratory difficulty due to inflammation and thickening of the air sacs, or general unthriftiness and emaciation due to inflammation of the gastrointestinal tract.

Other nematodes that have been reported in birds of prey include *Physaloptera* sp. in the ventriculus of the red-tailed hawk (Morgan, 1948; Smith and Miller, 1988, p. 55) and the sharp-shinned hawk (*Accipiter striatus*) (Hassall, 1931), filarial nematodes in the heart of a peregrine falcon (Crisp, 1854), and *Thelazia* sp. in the orbit of the eye of a Swainson's hawk (*Buteo swainsoni*) (Herde, 1942).

Trematode parasites, especially strigeid and diplostomatid trematodes, are common in birds of prey. These helminths are generally found in the duodenum and are seldom considered pathogenic, even in large numbers. Dedrick (1965), however, described a fatal syndrome characterized by poor condition and diarrhea associated with a trematode infection in a prairie falcon. Smith (1978) reported emaciation and death in a bald eagle due to a massive trematode infection, and Greenwood et al. (1984) concluded that the death of a saker falcon (*Falco cherrug*) was caused by severe intestinal trematodiasis. In addition to the strigeid and diplostomatid trematodes, several species of dicrocoelid trematodes have been reported in raptors, especially from the liver and bile ducts of the American kestrel (Schell, 1957). An unidentified species of trematode has also been observed in the bile ducts of a 6-wk-old free-living screech owl (*Otus asio*) (S. A. Smith, unpublished data) (Fig. 5.3, part 13). Though rarely diagnosed by fecal examination, these liver and bile duct trematodes produce an ovum that is smaller than the typical trematode ovum and may often be asymmetrical, with a distinct protuberance at one end (Ward, 1986).

Cestodes are uncommon in birds of prey. Small numbers of tapeworms are generally well tolerated by a healthy bird, and clinical signs of a heavy cestode infection range from no obvious clinical signs to mild diarrhea and weakness. Cestodes are generally located in the small intestine with the scolex attached to the intestinal mucosa, but rarely occur in numbers large enough to cause intestinal obstruction or death of the bird. In addition to fecal analysis, diagnosis of a cestode infection can often be made by observing the small, light-colored proglottids in the feces or around the vent of the bird.

Infections with acanthocephalans in raptors are rare, with the notable exception of one study in Louisiana in which 57% and 60% of the free-living Falconiformes and Strigiformes, respectively, were infected (Nickol, 1966). Kocan and Locke (1974) reported that 7% of 59 necropsied bald eagles were infected with acanthocephalans, and Kollias et al. (1987), using fecal analysis, found 29% of a number of species of birds of prey to be infected. These parasites are generally found in the distal small intestine with the proboscis firmly embedded in the mucosa. Except for heavy infections, there is little evidence that acanthocephalans cause clinical disease other than localized inflammation at the site of attachment. Keymer (1972) reported that Jennings attributed the cause of death of a lanner falcon (*Falco biarmicus*) to a severe infection of an unidentified acanthocephalan.

Fecal analysis of a bird of prey will often reveal ova from parasites that are not actually infecting the gastrointestinal tract or internal organs of the bird. Mite eggs are often observed during routine fecal examination (Fig. 5.3, part 14). These eggs might be swallowed by the bird during normal preening or may represent contamination of the fecal material from the cloacal area, and are an indication that the bird should be examined for a mite infestation. A second example of pseudoparasitism involves the ability of parasite ova of prey that has been eaten by a raptor to be passed unchanged through the bird's digestive system. The ova of *Trichosomoides crassicauda*, a parasite of the urinary bladder of rats, are frequently seen during fecal examination of captive raptors fed laboratory rats (Fig. 5.3, part 15). To determine the source of a questionable egg found in raptor fecal material, the researcher should repeat the fecal examination in two to three days. If the egg originated from prey, it should no longer be found in the fecal material, assuming the diet of the bird has been changed. If the egg originated from parasites of the bird of prey, it will most likely be present in subsequent fecal examinations.

Treatment of Parasitism in Birds of Prey

Various anthelmintics and treatment schedules have been suggested for the removal of helminths from birds of prey. However, little research is available on the use and efficacy of these drugs in raptors, with most dosages being extrapolated from nonraptorial species of birds. The physical condition and health of the raptor must be taken into consideration before treatment. Caution should be exercised when treating a sick, debilitated, stressed, or immature bird.

Table 5.4 lists a variety of anthelmintics used to treat nematodes. Both thiabendazole[a] and levamisole[b] have been reported effective against *Capillaria* spp., but a second treatment after 7 to 10 days is often necessary to eliminate the infection. Two treatments of ivermectin[c] at 3 to 4 wk apart

Table 5.4
Anthelmintics used for the treatment of helminth parasites in birds of prey

Anthelmintic	*Treatment*
Nematodes	
Thiabendazole	100 mg/kg PO, repeated in 10-14 days
Levamisole	10-20 mg/kg PO, for 2 consecutive days
Ivermectin	0.4 mg/kg IM, repeated in 3-4 wk
Ivermectin	200 mg/kg PO or IM, dilute Ivomec with water 1:9 and use 0.2 ml/kg
Piperazine	100 mg/kg PO
Mebendazole	50 mg/kg PO, repeated in 10-14 days
Fenbendazole	25 mg/kg PO, for 3 consecutive days
Trematodes	
Rafoxanide	10-15 mg/kg PO
Praziquantel	50 mg/kg PO
Cestodes	
Praziquantel	50 mg/kg SQ
Acanthocephalan	
No known effective treatment	

have been used successfully to treat capillarid infections in raptors (Kollias et al., 1987). Ascarids in birds of prey have been successfully treated with piperazine,[d] thiabendazole, and mebendazole.[e] Treatment of dual infections of *Capillaria* spp. and ascarids can be accomplished by the administration of levamisole or ivermectin. Stehle (1977) has reported that fenbendazole[f] is effective in eliminating both capillarids and ascarids from birds of prey. Mebendazole has been used to control stomach worms in waterfowl and has been suggested as a treatment for *Microtetrameres* sp. and other spirurid nematodes in raptors. Infections of *Serratospiculum amaculata* have been effectively treated with either thiabendazole or levamisole, and infections of *Syngamus* sp. have been treated with either thiabendazole or mebendazole.

Most anthelmintics appear relatively safe and effective for use against common nematodes in raptors. In my experience, either levamisole or ivermectin works best in birds of prey, although levamisole may be slightly more efficacious in the treatment of ascarids. Either anthelmintic should be administered orally and repeated if necessary at the proper time interval. The anthelmintic should be given when the upper digestive tract is empty, to decrease the amount of nonspecific binding or inactivation of the drug and to reduce the possibility of vomiting. I have also used levamisole and ivermectin successfully to terminate the passage of eggs of spirurid nematodes and *Serratospiculum* sp. in the fecal material of raptors, though it was not known if the nematodes were killed and/or eliminated from the birds. The success of anthelmintic treatment of the less common nematodes known to infect birds of prey (that is, *Physaloptera* sp. and *Thelazia* sp.) has not been investigated.

Since both trematodes and cestodes rarely cause clinical disease, treatment of infected raptors may be more palliative than necessary. However, Greenwood et al. (1984) have suggested that control of trematodes in captive birds of prey may be indicated, given that severe or fatal infections do occasionally occur in these birds. Trematodes have been effectively and safely treated with rafoxanide[g] or praziquantel.[h] In addition, praziquantel is effective against cestode infections. At present, there is no known effective treatment for infection of acanthocephalan parasites.

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Product List

[a]TBZ, MSG AGVET, Division of Merck and Co. Inc., 126 E. Lincoln Ave., Rahway, New Jersey 07065-4687 USA

[b]Levasole, Pitman-Moore, 421 E. Hawley St., Mundelein, Illinois 60060 USA

[c]Ivomec, MSG AGVET, Division of Merck and Co. Inc., 126 E. Lincoln Ave., Rahway, New Jersey 07065-4687 USA

[d]Sergeants Worm Away, Conagra Pet Products, 3902 Leavenworth, Omaha, Nebraska 68105 USA

[e]Telminic, Pitman-Moore, 421 E. Hawley St., Mundelein, Illinois 60060 USA

[f]Panacur, Hoechst-Roussel Pharmaceuticals Inc., P.O. Box 2500, Somerville, New Jersey 08076 USA

[g]Ranide, MSG AGVET, Division of Merck and Co. Inc., 126 E. Lincoln Ave., Rahway, New Jersey 07065-4687 USA (Editors' Note: Ranide is not available in the United States.)

[h]Droncit, Haver Lockhart, 5101 Speaker Rd., Turner, Kansas 66106 USA

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CHAPTER 6

Parasitological and Other Studies on Migrating Raptors

JOHN E. COOPER, D.T.V.M., F.I.Biol., M.R.C.Path., F.R.C.V.S.

Department of Pathology
The Royal College of Surgeons of England
London, England

EDNA GORNEY-LABINGER, M.Sc.

George S. Wise Faculty of Life Sciences
Department of Zoology
Tel Aviv University
Ramat-Aviv, Tel Aviv, Israel

FIONA ION, B.Sc.

Animal Health Laboratory
The Royal College of Surgeons of England
London, England

ABSTRACT: Despite interest in the migration of raptors and studies on various aspects of their biology, relatively little research has been carried out on the possible role of these birds in transporting disease or infection from one area to another. Migrating raptors live-trapped in Israel were sampled. Each bird was examined, weighed, and measured. Blood smears were taken, stained with Giemsa, examined for blood parasites, and retained for differential blood counts. Ectoparasites, mainly Mallophaga, were collected and identified. Suggested protocols for the monitoring of migrating birds of prey are presented.

KEY WORDS: parasite, raptor, Israel, migration.

Migration is a time when raptors are subject to many dangers, particularly starvation, persecution, collisions with objects such as power lines and vehicles, and poisoning. As has been emphasized previously (Cooper, 1989a, 1989b) relatively little has been published on diseases of migrating birds of prey other than studies by Redig et al. (1980) on migrating goshawks and a recent search for *Salmonella* spp. by Kirkpatrick and Trexler-Myren (1986). In this chapter, preliminary findings from migrating raptors in Israel are presented.

Israel lies on the path of one of the world's largest migration routes. More than a million raptors have been observed in a single spring season at Eilat, in the southern part of Israel (Leshem, 1989). From 1987 through 1988, a raptor-banding project at Eilat resulted in the handling of more than 2,500 birds of 21 species.

A visit to Israel in the spring of 1987 to attend a meeting of the ICBP World Working Group on Birds of Prey yielded an opportunity to observe the trapping and banding (ringing) of migrating raptors. The senior author (JEC) suggested that samples could usefully be taken from the birds at the time of banding and that analysis of these would contribute to our knowledge of the species. In order to help implement this, two Israeli biologists were taught to take blood samples and to prepare smears.

Project Goals

Goals of the project included the following:

1. to collect as many samples as possible from each bird so long as this was compatible with (a) the aims of the main Israel-based project, (b) the welfare of the birds, and (c) legal restrictions on the importation of pathological samples into Britain (Cooper, 1987)
2. to promote the concept of screening migrating raptors and to draw up protocols for the guidance of other investigators

Materials and Methods

Sampling

Migrating raptors were trapped using a number of techniques, but mainly a Bal-Chatri trap baited with laboratory mice was used (Clark et al., 1986). All birds were weighed and measured. Blood samples were taken from the brachial (basilic) vein following routine techniques (Cooper, 1978, p. 25). Two smears were prepared from each bird, air dried, and fixed with alcohol. Throat and cloacal swabs were taken from some birds, but this was not part of the current study and therefore will not be discussed here. Likewise, in 1988,

Table 6.1
Raptors trapped/sampled at Eilat, Israel, spring 1984 to spring 1988

Total	*2,517 birds of 21 species*
Most frequently trapped species	
Buzzard (*Buteo buteo*)	1,456
Levant sparrowhawk (*Accipiter brevipes*)	425

Table 6.2
Birds from which ectoparasites were removed, 1986 and 1987

Species	*Number*
Buzzard (*Buteo buteo*)	140
Levant sparrowhawk (*Accipiter brevipes*)	4
Marsh harrier (*Circus aeruginosus*)	2
Booted eagle (*Hieraaetus pennatus*)	4
Black kite (*Milvus migrans*)	1
European kestrel (*Falco tinnunculus*)	4
Honey buzzard (*Pernis apivorus*)	2

Table 6.3
Specimens of ectoparasites collected, 1986 and 1987

Lice
Various species in the genera *Laemobothrion*, *Colpocephalum*, *Kurodaia*, *Craspedorrhynchus*, *Degeeriella*, and *Falcolipeurus*

Ticks
Amblyomma lepidum
Rhipicephalus turanicua

Hippoboscids
Pseudolynchia canariensis
Icosta sp.

Table 6.4
Frequency of body lice in 109 buzzards, 1986 and 1987

Number of lice	*Number of birds*	*% of total birds*
0-29	62	56.9
30-69	27	24.8
70-250	20	18.3

blood was collected from buzzards for determination of total body water, packed cell volume, and plasma osmolality, but these results are not included in this chapter.

Ectoparasites were collected by placing the body of each live bird in a plastic bag containing ether for 5 min. The bag was then removed and dead and narcotized parasites were brushed off onto a collecting tray.

Processing

Fixed blood smears were transported to the Royal College of Surgeons, London, for staining and examination. Staining was carried out initially with a modified Giemsa stain, subsequently with a May-Grünwald. Stained smears were examined microscopically for parasites (protozoa and microfilariae) and for hematological abnormalities. One set of

Table 6.5
Blood samples taken and parasites identified, 1987 and 1988

Species	*Number sampled*
Buzzards	60
Levant sparrow hawks	9
Honey buzzards	3
Booted eagle	1
Barbary falcon	1
Blood parasites identified to date	
Leucocytozoon toddi	
Haemoproteus sp. (*buteonis*?)	
Plasmodium sp.	
Of 66 blood smears examined to date	
No parasites seen	24
Leucocytozoon sp.	27
Haemoproteus sp.	21

slides was sent to Dr. Michael Peirce of the International Reference Centre for Avian Haematozoa for a second opinion. Blood cells were also used for differential blood-cell counts using the methods described by Hawkey and Dennett (1988).

Ectoparasites were preserved in alcohol and counted before identification. Some were identified in Israel by Dr. A. Friedberg, Tel Aviv University; others by Dr. J. E. Keirons and Dr. K. C. Emerson, Biosystemics and Beneficial Insects Institute of the U.S. Department of Agriculture; and the majority of the lice by Professor R. D. Price of the University of Minnesota.

Results

The number of birds trapped during the period 1984-88 is given in Table 6.1, together with a breakdown of the samples collected. Birds yielding ectoparasites are listed in Table 6.2. Determinations to date of parasites are given in Table 6.3, and body-lice frequency in buzzards in Table 6.4. Parasites found in blood smears are listed in Table 6.5.

In addition to these screening results, occasional cases of clinical disease and accidental damage were observed. These included overgrown/damaged beaks, pox, foot lesions, oiled birds (Clark and Gorney, 1987), and a kestrel with feather damage that proved on scanning electron microscopy to have been caused by electrocution.

Discussion

The majority of the birds sampled were considered to be in good health, and thus the study is best considered a screening program rather than a diagnostic exercise.

The data presented are incomplete, as many parasites remain to be identified and the hematological examinations are still in progress. Nevertheless, the results to date illustrate the extent of information that can be obtained from

Table 6.6
Protocol for clinical screening of migrating raptors

Basic

Observation and examination

1. Presence or absence of
   a. clinical signs of disease
   b. injuries or external lesions
   c. ectoparasites
2. Measurements for
   a. body weight
   b. carpal length
   c. condition score
3. Gross appearance of
   a. feces
   b. pellets

Laboratory tests

1. Presence or absence of protozoan and metazoan parasites in feces
2. Presence or absence of parasites or cellular abnormalities in blood smears

Additional investigations, if personnel and facilities permit

1. Bacteriological examination of swabs from
   a. trachea
   b. cloaca
2. Examination of blood (in anticoagulant) with particular reference to
   a. PCV (hematocrit)
   b. total protein
3. Examination of serum for antibodies (serology)

Table 6.7
Postmortem investigation of migrating raptors

Basic

1. Gross examination
   a. body weight
   b. carpal length
   c. condition score
   d. appearance of internal organs
   e. presence or absence of fat
   f. presence or absence of ectoparasites on plumage
   g. presence or absence of endoparasites in alimentary or respiratory tract
2. Toxicology: submission or retention (frozen) of carcasses or tissues for analysis (e.g., for chlorinated hydrocarbon pesticides, heavy metals)

Additional investigations, if personnel and facilities permit

1. Bacteriology
   a. heart blood
   b. intestinal contents
   c. any significant lesions
2. Histopathology
   a. lung
   b. liver
   c. kidney
   d. any significant lesions
3. Other tests: submission or retention (frozen/fixed) of tissues for virology, mycoplasmology, electron microscopy, etc.

a systematic study of migrating raptors. There is clearly a need for similar work on migrating birds of prey in other parts of the world. Protocols for screening raptors were proposed in a previous paper (Cooper, 1989a) and are reproduced here (Tables 6.6 and 6.7). We welcome comments and constructive criticism on these from colleagues.

Much remains to be learned about migrating raptors and their susceptibility to disease. Such research may also throw light on the possible role of such birds in the spread of infectious agents or arthropod vectors.

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Tables 6.6 and 6.7 were reprinted with permission from R. D. Chancellor, ICBP, from *Raptors in the Modern World* (World Working Group on Birds of Prey, 1989).

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CHAPTER 7

Four Cases of Neoplasia in Birds of Prey

JOHN E. COOPER, D.T.V.M., F.I.Biol., M.R.C.Path., F.R.C.V.S.

Department of Pathology
The Royal College of Surgeons of England
London, England

WENDY WILKENS, B.A., Vet.M.B., M.R.C.V.S.

Westmoor Veterinary Centre
Tavistock, Devon, England

KEITH LAWRENCE, B.V.Sc., Ph.D., F.R.C.V.S.

23 Woodside Gardens, Chineham, Basingstoke
Hampshire, England

ABSTRACT: Neoplasms were diagnosed in four birds of prey: a renal carcinoma in a common buzzard (*Buteo buteo*), squamous-cell carcinomas in a Barbary falcon (*Falco pelegrinoides*) and European eagle owl (*Bubo bubo*), and a mesothelioma in a ferruginous hawk (*Buteo regalis*). The cases illustrate three points: (1) the value of taking biopsies from clinical cases, (2) the importance of routinely carrying out histopathological examination of tissues of free-living birds that are examined postmortem, and (3) the possible role of raptors as important sentinels for the detection of environmental carcinogens.

KEY WORDS: raptor, neoplasia, biopsy, carcinoma, postmortem histopathology, environmental sentinel.

Neoplasms have been reported infrequently from birds of prey (orders Falconiformes and Strigiformes) despite the numbers of these species that are kept in captivity and examined postmortem (Cooper, 1978b). In this chapter we report four neoplasms in birds of prey: a renal carcinoma in a common buzzard (*Buteo buteo*), squamous-cell carcinomas in a Barbary falcon (*Falco pelegrinoides*) and European eagle owl (*Bubo bubo*), and a mesothelioma in a ferruginous hawk (*Buteo regalis*).

Histories

The common buzzard was not seen alive. It was one of three submitted frozen to a Ministry of Agriculture Veterinary Investigation Centre for postmortem examination. A farmer had found all three dead in the same area during the previous three weeks.

The Barbary falcon was a captive bird with a history of a chronic ulcerative skin lesion on the flank and medial aspect of the leg. A number of species of bacteria had been isolated, including *Pseudomonas aeruginosa*. The bird had been given antibiotics and various other forms of topical therapy. Following referral to one of the authors (JEC), a skin biopsy was taken. Therapy was about to commence when the bird died.

The European eagle owl was a captive adult female bird that developed a skin lesion over its pectoral muscles. The lesion was excised and submitted to JEC for histological examination. Following surgery the bird made a full and uneventful recovery.

The ferruginous hawk was a captive bird that had been imported as a subadult four to five years earlier. It was presented to one of the authors (KL) with a displaced, self-mutilated wing. On clinical examination under general anesthesia, a traumatized swelling was located proximal to the elbow. Radiography revealed a marked periosteal reaction involving the joint. The wing was amputated through the shoulder joint and the bird made an uneventful recovery.

Pathological Examination

Only two of the birds, the common buzzard and the Barbary falcon, received full postmortem examinations. Standard procedures (Cooper, 1987) were followed. With the European eagle owl and ferruginous hawk, fixed tissues were received for histopathological investigation.

Tissues from all four birds were fixed in buffered formol saline, embedded in paraffin wax, and sectioned. Stains used included hematoxylin and eosin, Gram, periodic acid Schiff, and hematoxylin van Gieson. Skin lesions from the Barbary falcon were also fixed in glutaraldehyde and examined by transmission electron microscopy.

Pathological Findings

Buzzard

The bird was in poor condition, dehydrated and debilitated. Hemorrhage was present throughout the body cavity. The mucosa of the proventriculus-ventriculus was hyperemic and there were three nodules present, each with a caseous necrotic center. Smears from the nodules revealed no parasites or acid-fast organisms. There were *Capillaria* spp. worms and eggs in the intestinal tract.

Both kidneys were abnormal—enlarged, very pale, and infiltrated throughout with multiple small yellowish-white foci and large white "fibrous" lesions. Histopathological examination of the kidneys showed a sheet of tissue consisting of acinar structures characteristic of a renal adenocarcinoma.

Barbary Falcon

The biopsy sample taken when the bird was still alive showed ulceration of the skin and dermal masses of keratin surrounded by nests of moderately well differentiated stratified epithelial cells. This lesion was identified as a squamous-cell carcinoma.

Postmortem examination revealed a very thin and dehydrated bird with extensive ulceration in the left groin. The only other gross finding of note was a cloacal calculus measuring 1 cm × 1.5 cm. No internal or external parasites were detected.

Histopathological examination confirmed a squamous-cell carcinoma. There was no evidence of metastasis, but the skin lesion also contained bacteria and cholesterol crystals. Medial calcification was present in the great vessels and renal arteries, while the myocardium showed chronic inflammatory cells and cholesterol deposits. Transmission electron microscopy revealed, in addition to the features above, abnormal clusters of collagen.

European Eagle Owl

The biopsy revealed skin ulceration. The dermis contained islands and strands of well-differentiated epithelial cells together with whorls of keratin. On the basis of these findings a diagnosis of squamous-cell carcinoma was made.

Ferruginous Hawk

Histological examination of the soft tissue swelling revealed a papillomatous, branching tumor; the lining cells were cuboidal, with round nuclei. A diagnosis of mesothelioma was made.

Discussion

Although these tumors were diagnosed in four species of birds in two different orders, they add to the relatively sparse data on neoplasms in birds of prey. Of particular interest are the buzzard with the renal adenocarcinoma and the ferruginous hawk with the mesothelioma. Only two previous neoplasms of possible renal origin have been reported in raptors. Both were in buzzards, *Buteo* spp., in Britain (Cooper, 1978a; Wadsworth and Jones, 1980). The common buzzard (Cooper, 1978a) was a free-living bird from Cornwall, the county adjacent to Devon, where the case in this chapter originated.

The mesothelioma appears to be only the second reported in a raptor and, like the renal tumors, both were in Britain. The case described by Cooper and Pugsley (1984) was also in a ferruginous hawk. The captive ferruginous hawk population in Britain is small and highly inbred, and both mesotheliomas occurred in birds in the county of Oxfordshire.

Taken as a group, these cases illustrate three important points. First, researchers should always consider taking biopsies when dealing with birds with skin or other lesions, especially if the latter prove intractable to treatment or show unusual features. Neither squamous-cell carcinoma would have been diagnosed in life unless biopsy samples had been taken and examined. Had the Barbary been biopsied earlier, therapy might have been possible. Second, the diagnosis of a renal adenocarcinoma in the buzzard illustrates the value of carrying out histopathological investigation of tissues from any wild birds examined postmortem, especially if gross abnormalities are noted. Finally, although the role of birds of prey as environmental sentinels is well recognized, their importance has largely been considered in terms of poisons and certain infectious diseases; they might also prove of value in the study of neoplasia.

Acknowledgments

We are grateful to the Ministry of Agriculture, Fisheries, and Food for permission to report on the buzzard, and to our colleagues David Beckingham and Michael Williams for submitting material from the eagle owl and Barbary falcon, respectively. Ian Keymer very kindly gave an opinion on a biopsy from the latter bird. Staff of the Royal College of Surgeons carried out routine microbiological and histopathological investigations and assisted with postmortem examinations.

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CHAPTER 8

Pathological Studies on the Barn Owl

JOHN E. COOPER, D.T.V.M., F.I.Biol., M.R.C.Path., F.R.C.V.S.

Department of Pathology
The Royal College of Surgeons of England
London, England

ABSTRACT: The barn owl (*Tyto alba*) is a widely distributed member of the Strigiformes, but has suffered a decline in recent years in the United Kingdom. During an investigation into the status of the barn owl in Britain and Ireland by the Hawk Trust, the opportunity arose to examine 67 dead birds. These were subjected to full postmortem examination with supporting laboratory tests. Carcasses and/or tissues of several were submitted for toxicological investigation. The pathological findings in these birds are reported and discussed. Postmortem examination revealed that 30 deaths were associated with traffic accidents or other trauma and 14 with inanition. Other findings included endoparasites, fatty infiltration of the liver, peritonitis, and pancreatitis. In 25.3% of the birds there was no diagnosis, nor were significant lesions detected.

KEY WORDS: barn owl, *Tyto alba*, Britain, Hawk Trust.

The barn owl (*Tyto alba*) is the most widespread bird in the world but is believed to have declined in recent years in the United Kingdom, other parts of Europe, and the northern United States. It is commonly kept in captivity, where it breeds readily (Bunn et al., 1982).

In 1987 the results of the 1982-1985 Barn Owl Survey of Britain and Ireland, conducted by the Hawk Trust, were published (Shawyer, 1987). The conclusions reached were that the British barn owl had declined by about 70% over the last 50 years, largely because of new agricultural practices coupled with climatic changes. Part of the study involved the examination of dead barn owls submitted by members of the public, mainly in reponse to specific requests by the Hawk Trust. In this chapter the postmortem findings in 67 of these birds are reported and discussed. A more extensive veterinary report is in progress and will include data on owls that were examined alive as well as pathological findings on eggs and embryos.

Materials and Methods

The majority of specimens were received by mail. In a few cases the carcasses had been frozen, but most were either fresh or had been chilled at 4°C. Full histories were always requested and sometimes received.

On arrival at the Animal Health Laboratory at the Royal College of Surgeons of England, each carcass was given a daybook number. A standard investigative technique was then followed, regardless of whether the bird had been free-living or captive:

external examination, including measurement of body weight (mass) and carpus (wing chord) length

collection of ectoparasites for identification

whole-body radiography (selected cases only)

gross postmortem examination

histopathological examination of lung, liver, kidney, and any abnormalities, plus "touch preparations" (impression smears) for cytological evaluation (see Campbell, chapter 3, this volume)

selected aerobic bacteriology

microscopical examination of wet preparations of intestinal tract plus salt (concentrated NaCl) flotation (selected cases only)

carcass/tissues (liver) frozen for subsequent toxicological investigation

In interpreting the results, the emphasis was not primarily on ascertaining the cause of death. Instead, the various findings were collected to provide more background data on this species and to elucidate the factors that might contribute to disease, reproductive failure, or death.

Although some distinction is made in the analysis between free-living and captive birds, the difference was often not clear-cut because some of the owls examined were free-living birds that had been brought into captivity as "casualties" and tended for varying periods of time. In Table 8.1 all cases are listed as free-living unless they had been in captivity for more than a month.

Results

The main findings in 67 barn owls (55 free-living, 12 captive) are summarized in Table 8.1. Some birds yielded find-

Table 8.1
Findings in 67 barn owls

	Free-living	*Captive*	*Total*
Road accident or other trauma	28	2	30
Inanition ("starvation")	14	0	14
Coccidia	7	0	7
Fatty liver	0	6	6
Chilling	3	0	3
"Shock"	1	0	1
Dehydration (?)	1	0	1
Pancreatitis	0	1	1
Peritonitis	0	1	1
Pneumonia/air sacculitis	0	1	1
Ticks	1	0	1
No diagnosis to date	11	6	17

Note: Some birds show two or more findings, thus the total number of findings exceeds 67.

ings in two or more categories; thus the total number of findings exceeds 67.

A total of 30 birds, 28 of them free-living, showed traumatic injuries; many of these appeared to be the result of traffic accidents. In most cases they had been found on or near a road. Injuries detected ranged from subcutaneous bruising and ruptured organs (usually liver) to extensive fractures and crushing of the head or body. One free-living bird was probably killed by a domestic cat (*Felis catus*).

Two birds, nestlings, were found dead beneath their nests. They had fractures of the legs and there was strong evidence that they had been killed intentionally by a person. The barn owl is protected in the United Kingdom (Cooper, 1987) under the Wildlife and Countryside Act of 1981.

Of the free-living birds, 14 showed emaciation, a complete absence of internal fat, an empty stomach, and, in most cases, bile staining of viscera. These findings, coupled with the absence of significant histopathological or microbiological findings, were considered consistent with a diagnosis of inanition.

Coccidial oocysts (*Isospora* sp.) were detected in large numbers (more than 100 per low power field) in the intestinal contents of 7 free-living birds. In no case was it possible to diagnose "coccidiosis" rather than merely detect "coccidia" because postmortem autolysis severely limited histopathological examination. In the majority of these cases the bird was also in poor condition. One owl showed bruising of organs, possibly due to trauma, as well as a high coccidial oocyst count.

Six captive barn owls (two groups of three birds) showed extensive fatty change of the liver characterized by gross enlargement and pallor. Histopathological examination confirmed marked vacuolation of the cytoplasm of hepatocytes. Three of these owls had been fed on a diet rich in fat and starch, and this was assumed to be contributory. The cause in the other three was unclear.

One free-living bird showed marked dehydration, but it was impossible to determine to what extent this was an antemortem or postmortem change. The bird was in good bodily condition but not suitable for histopathological investigation. A captive owl was found to have chronic pancreatitis, and peritonitis was diagnosed in a free-living bird. In both cases the lesions were confirmed on histopathological examination. Pneumonia and air sacculitis were likewise diagnosed in one captive bird.

Postmortem autolysis made bacteriological investigation of many of the owls impossible, and the results were of doubtful relevance in others. However, no significant isolates were obtained.

"Chilling" was diagnosed on the basis of clinical history and nonspecific postmortem findings in three free-living nestling owls whose male parent had died as a result of trauma. The one case of "shock" was a bird in poor condition that died soon after admission to a veterinary hospital. The diagnosis was a clinical one; inanition may have contributed to death, and hypoglycemia was also a possibility. Ticks were found on one free-living bird that had been taken into captivity. These are awaiting identification. There was no obvious cause of death.

For 17 of the 67 owls (25.3%), 11 free-living and 6 captive, no diagnosis has yet been made. In most of these birds there was no evidence of gross or microscopic lesions. Carcasses or tissues have also been submitted for toxicological analysis, and this may yield further information.

Discussion

There have been few pathological studies on the barn owl other than case reports and surveys of captive birds (Cooper, 1978; Keymer, 1972), investigation of *Salmonella* spp. in free-living nestlings (Kirkpatrick and Colvin, 1986), and a recent paper on free-living barn owls (McOrist, 1989). Most of the work on mortality in this species has been carried out by biologists, who have paid particular attention to such factors as availability of food (Honer, 1963; Morton and Martin, 1979; Wilson et al., 1986) and nest sites (Colvin, 1983; Shawyer, 1987) and mortality during cold weather (Marti and Wagner, 1985). Newton et al. (1991), in a survey of 627 barn owls examined during the period 1963 to 1989, concentrated on the possible role of pesticides. They refer in their paper to "diseased birds" (less than 3% of total) with "obvious lesions," but give no indication that any supporting laboratory tests were performed that might have confirmed or refuted their diagnoses.

The Hawk Trust survey suggested that the British barn owl had declined largely as a result of changing agricultural practices and adverse climate (Shawyer, 1987). Significance was also attached to the regional importance of road casualties and the potential for secondary poisoning by some ro-

denticides. Shawyer points out, however, that widespread surveys of this kind can provide only a general indication about this decline and that other factors, as yet unidentified, may also play an important role.[1]

Among other such factors may be toxic chemicals. Newton et al. (1991) describe poisoning by organochlorine pesticides, especially aldrin/dieldrin, and the susceptibility of *Tyto alba* to poisoning by this group of chemicals has been confirmed by studies on captive owls (Jones et al., 1978). Second-generation rodenticides such as difenacoum and brodifacoum have been suggested as possible causes of death in barn owls (Shawyer, 1985, 1987), and brodifacoum can certainly prove lethal when fed to captive owls (Newton et al., 1990), but Hegdal and Blaskiewicz (1984) state that the potential in this species for mortality by brodifacoum "appears to be low." Nevertheless, many of the birds examined in the present study are being analyzed for brodifacoum, and the findings will be included in the final survey.

It appears the role of infectious and parasitic disease in free-living barn owls has never been fully investigated. There has, however, been interest in the subject for some time. Bunn et al. (1982) refer to a note by Williams (1917), who reported that "some disease has attacked barn owls over a great part of Ireland." They point out, however, that the signs described by Williams are almost certainly attributable to low temperatures and starvation. Bunn et al. give other examples of diseases and/or organisms that might contribute to morbidity and mortality and conclude that "although disease is unlikely to be a major cause of a decline in barn owl numbers . . . that is not to say that such a situation is impossible."

In this context the work of Honer (1964) is relevant. Honer rejects the idea that the coccidian *Isospora buteonis*, which is commonly found in barn owls (Pellérdy, 1974), is of significance, even when present in large numbers. However, Bunn et al. (1982) attribute to S. Davies the hypothesis that "coccidiosis could well be a secondary feature following stress and starvation." The findings in the present survey suggest that such a role warrants further investigation.

No *Salmonella* spp. were isolated in this study. In the United States, Kirkpatrick and Colvin (1986) swabbed the cloaca of 94 owls and cultured *Salmonella* spp. (three serotypes) from 8 (8.5%). They suggest that "salmonellosis may be yet another factor which affects common barn-owl survival and reproduction." Salmonellosis has certainly been diagnosed as a cause of ill health and death in young captive barn owls in Britain (Cooper, unpublished data) and was implicated in the death of a free-living specimen in the United States (Locke and Newman, 1970).

It is clear from this survey that pathological investigations can play a useful part in studies on mortality in the barn owl. Even if such investigations do not reveal a cause of death, they provide background information on the biology of this species. They can help determine whether, for example, the bird killed by a car was struck because of underlying disease; in none of the owls examined in this study did that appear to be the case. For optimum results, carcasses should be received first by the pathologist and then forwarded to the toxicologist. In this way pathological examination can be carried out on tissue that has not been subjected to freezing and thawing, with the attendant damage and artifact.

A multidisciplinary approach to the decline of the barn owl is desirable. As Hunter et al. (1987) write:

> Disease processes may affect species population dynamics in more subtle ways than killing the host. Subclinical disease may be an important factor in reproductive success and livability of wild owl populations. Our knowledge of naturally occurring disease in owls is very limited and it would be highly desirable for field biologists and veterinary pathologists to develop cooperative studies to investigate this fascinating aspect of owl biology.

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Note

1. Personal communication with C. R. Shawyer of the Hawk and Owl Trust, Zoological Society of London.

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CHAPTER 9

Pathological Studies on Eggs and Embryos

JOHN E. COOPER, D.T.V.M., F.I.Biol., M.R.C.Path., F.R.C.V.S.

Department of Pathology
The Royal College of Surgeons of England
London, England

ABSTRACT: Despite the interest in reproductive success of raptors, examination of unhatched eggs and embryos has largely been restricted to work on free-living populations, and even here the emphasis has tended to be on toxicological rather than pathological investigations. The methods used to examine unhatched eggs (both fertile and infertile) of falconiform and strigiform birds are outlined in this chapter. A considerable amount of extrapolation is possible from work carried out in the domestic fowl. Techniques that are of value in helping to elucidate the cause of death in embryonated eggs include gross pathological examination, microbiology, and histopathology. An integral part of any examination is the full collation of data relating to the environment, including a careful analysis of the conditions under which the eggs were incubated or stored. There is a need for more work on embryonic mortality in raptors, and standard methods of investigation and recording should be established.

KEY WORDS: egg, embryo, raptor.

There have been many developments in the captive breeding of raptors in the last few years, as well as notable advances in their veterinary care. Nevertheless, remarkably little attention has been paid to the investigation of eggs and embryos of these species other than studies, usually of a toxicological nature, on free-living populations. The latter have provided useful baseline data on, for example, egg size, weight, and shell thickness (Burnham et al., 1984), but little on pathological aspects.

In this chapter I outline the techniques I have used to examine unhatched eggs (both fertile and infertile) of falconiform and strigiform birds.

Materials and Methods

In order to investigate mortality in embryos and newly hatched chicks, adequate laboratory facilities are required, and the veterinarian must be prepared not only to inspect aviaries, incubators, and brooders, but also to cooperate closely with the owner of the bird and others with experience in captive breeding (Cooper, 1987a, 1987b, 1988). Essential laboratory equipment for egg examination includes, in addition to routine items, a candler, an accurate balance, callipers, and a micrometer. For work in the field a portable candler is helpful (Sobkowiak and Bird, 1984). A dissecting microscope or binocular loop will greatly facilitate examination of small specimens.

All data are recorded. The standard forms I use for this purpose are reproduced as Figs. 9.1-9.3. The form shown in Fig. 9.3 provides a convenient way to summarize findings in a clutch or batch of eggs. A camera or drawing may be used to obtain a permanent record of markings or any external abnormalities.

Examination of Eggs

The techniques used are summarized in Fig. 9.4. Examination must be accompanied by adequate history and detailed information on any incubation technique used. Eggs are given a reference number, and this is written in pencil or water-resistant ink on the shell. The external appearance of the egg is described, including the presence of dirt or cracks, and, where appropriate, the egg is sketched or photographed. The egg is candled, and on the basis of this the egg is categorized as being (1) probably infertile or (2) probably fertile. In the latter case, a sketch (drawing) is made of the size and position of the embryo and air space as seen on candling. Eggs in the former category are sketched if anything unusual or significant is noted.

All eggs, whether considered infertile or fertile, are weighed, measured, and opened in an identical fashion. First the shell is cleaned with 70% alcohol (methanol). It is important to let this evaporate before opening the shell. An elliptical piece of the shell is removed parallel to the long axis; the portion removed is retained for subsequent study. The interior of the egg is examined and sketched or photographed. At this stage the protocol varies depending on whether the egg is believed to be fertile or infertile. If there is any doubt, it is put into the fertile category.

The two approaches are as follows:

1. *Probably infertile*: The contents may be cultured for

REQUEST FOR PATHOLOGICAL EXAMINATION OF EGGS/EMBRYOS

To A.H.L., Date submitted: .....
Royal College of Surgeons of England,
35-43 Lincoln's Inn Fields, London WC2A 3PN.

Species: ..... Owner/Origin: .....

Address: .....

.....

Nature of specimen: (please tick) Infertile ( ) Exploded ( )

Early fertile ( ) Embryo dead in shell ( )

Other (please specify): .....

Please give information concerning:

Date laid: ..... Clutch and egg number:.....

Incubated by parents YES/NO For what period .....

Fostered YES/NO For what period .....

Artificially incubated YES/NO For what period .....

Incubator type .....

Temp. of incubator ..... Relative humidity .....

Was the egg dipped YES/NO In what .....

Was the egg turned YES/NO Was it candled YES/NO

Results of candling FERTILE/INFERTILE

Other observations .....

Length of time incubated ..... Normal incubation period .....

Date/time of collection ..... Storage .....

Weight of egg at death ..... Weight loss .....

Any other relevant information:

PLEASE LABEL AND PACKAGE SPECIMEN CORRECTLY

Figure 9.1. Standard form for request for pathological examination of eggs and embryos.

PATHOLOGICAL EXAMINATION OF EGGS/EMBRYOS

For lab use:

A.H.L., Royal College of Surgeons of England,
35-43 Lincoln's Inn Fields, London WC2A 3PN.

Ref. no:
Received:
Ack.:
Examined:
Method of packing:

FINDINGS AND REPORT

EGG/EMBRYO EXAMINATION
(to be completed for each specimen)

Species: .....

Owner/Origin: .....

Weight of whole egg: ..... **Length:** ..... **Width:** .....

Weight of dried eggshell: ..... **Thickness:** .....

External appearance (see Fig. 1):

Appearance on candling (see Fig. 2):

Embryo

Air cell

Blood vessels

Fluids

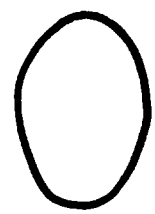
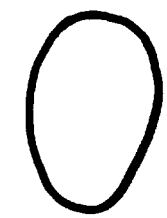


Fig. 1



Appearance when opened (see Fig. 3):

Contents:

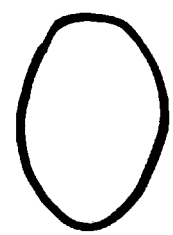


Fig. 2

Embryo: **Length (crown-rump)**

Amniotic cavity

Allantoic cavity

Yolk sac

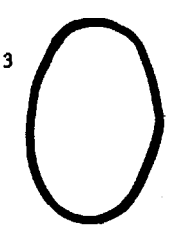


Fig. 3

Other comments:

Microbiology:

Histopathology:

Other tests:

Samples sent elsewhere:

Samples stored:

COMMENTS:

Date: ..... **Signature:** .....
J.E. COOPER MRCPath, FRCVS

Figure 9.2. Standard form for the pathological examination.

Figure 9.3. Standard form for summarizing findings.

SUMMARY OF FINDINGS - EGG EXAMINATION

Our ref: AHL:
Your ref:

Ref.	Species	Egg no:	Wght.	Lgth.	Wdth.	Fertile	Comments on embryo	Bact.	Isolates	Other comments

COMMENTS

ANIMAL HEALTH LABORATORY

Date: .....

.....
J.E. COOPER MRCPath, FRCVS

EXAMINATION OF UNHATCHED EGGS

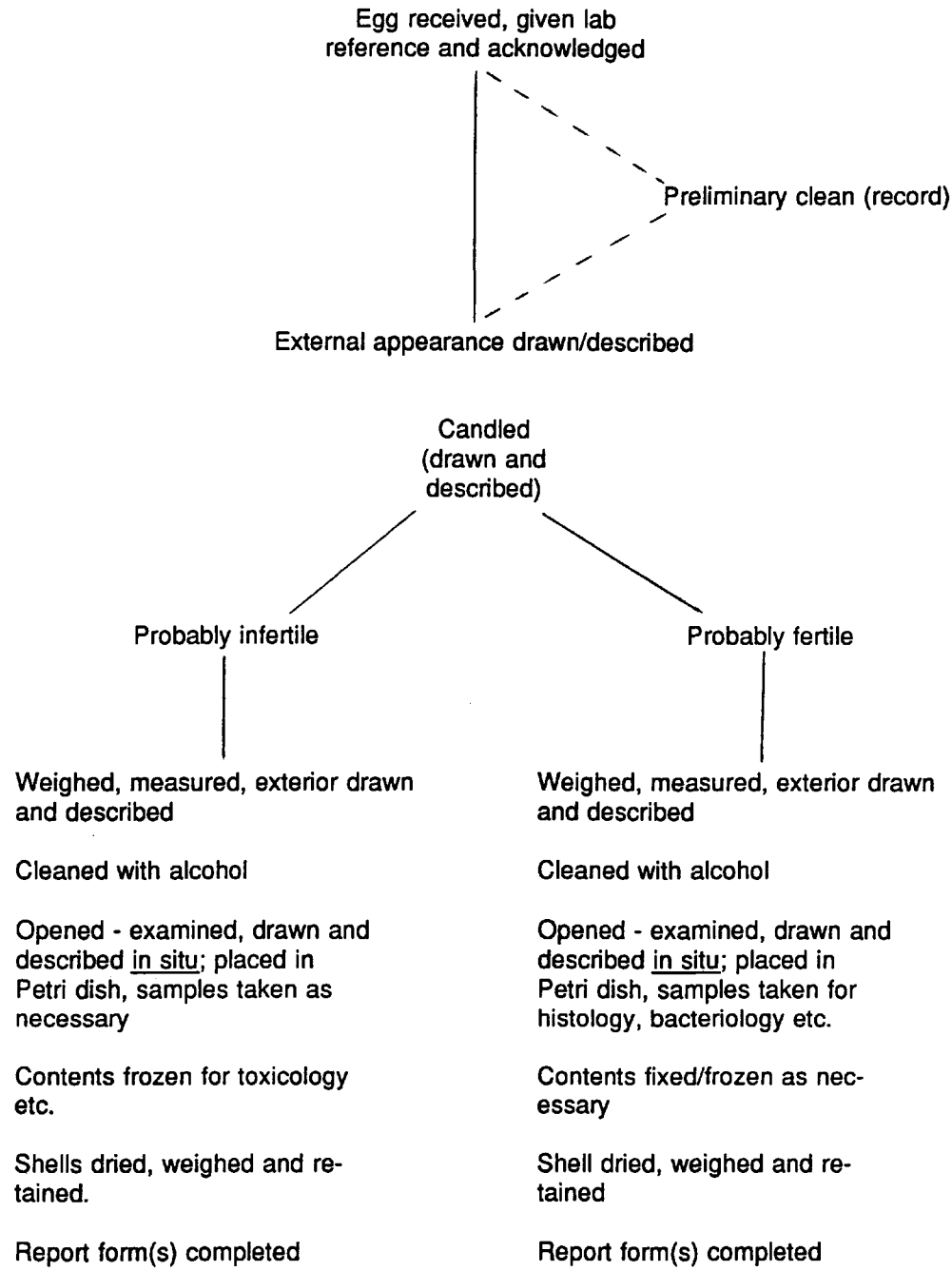


Figure 9.4. Techniques used in the examination of unhatched eggs.

bacteria and frozen for toxicological examination or discarded. The shell is dried for seven days, weighed, and either retained for measurements and other studies or forwarded to the Institute of Terrestrial Ecology, Monks Wood, England, where shell indices are calculated and other tests are performed.

2. *Probably fertile*: Depending on the degree of embryonic development, the contents may either be cultured *in toto* or swabs taken from specific sites, for example, amniotic fluid. The embryo is examined *in situ* for evidence of malpositioning. It is then removed, inspected for developmental abnormalities (for example, hydrocephalus, prognathia), measured, and weighed. It may be dissected and tissues taken for histopathology, bacteriology, mycology, or other tests. Small embryos are fixed and sectioned whole. Any remaining embryonic tissue is fixed in buffered formol-saline and retained for further study. The shell is dried and weighed as described for the infertile egg.

It is important to complement microbiological findings with histopathological investigations. For example, strains of *Staphylococcus aureus* can be associated with both gross and histological changes in chickens (Khamas and Waites, 1987).

If serious doubt exists as to whether an egg is fertile, histological examination of any suspicious material can be carried out and may distinguish between an early embryo and (for example) inspissated yolk. It is for this reason that doubtful eggs should be put into the "probably fertile" category. Decomposing eggs can present particular problems of examination and interpretation (see Houston et al., chapter 11, this volume).

Toxicological tests on eggs may yield useful results. Standard analyses are those for chlorinated hydrocarbons and heavy metals.

Selenium toxicity has been associated with poor reproductive success, including embryonic deformities, in aquatic birds (Ohlendorf et al., 1986) and may be significant in raptors. Material for all these toxicological investigations is usually stored frozen at $-20°C$.

Interpretation of Findings

Interpretation is not easy. The researcher must take account of such factors as the history, method of management, and age of the egg, and conditions and duration of storage. It is important to ascertain whether the eggs were washed or disinfected prior to setting. Management factors may contribute substantially to embryonic death, as is well recognized in galliform birds and suggested in recent work on budgerigars by Baker (1988). It may be necessary to sample nest boxes, incubators, brooders, and other items.

The health of the female bird is important, and clinical examination, including laparoscopy, may be advisable. Reproductive disorders of raptors have been well documented (Cooper, 1978; pp. 177-180; Keymer, 1980), and a number of the conditions reported may influence egg production or quality.

Culture of bacteria does not necessarily mean that the isolates killed the embryo. They may be secondary invaders, the embryo having died from some other cause. The mechanisms by which bacteria can penetrate and colonize eggs were investigated more than 25 years ago (Board, 1966), but there remains doubt over the significance of many isolates, even from fertile eggs (Orajaka and Mohan, 1985). Conversely, failure to isolate organisms need not preclude an infection. Albumen tends to inhibit growth, and for this reason Helga Gerlach recommends culturing the vitelline membrane.[1]

Kiessling and Gerlach (1989) draw attention to the possible significance of bacterial L-forms and urge those examining birds or eggs to search for them. This has not to date been my practice, and therefore I may have missed these organisms.

Care must also be taken over other findings. A malpositioned embryo may have died not because it was unable to hatch (some malpositionings are nonlethal), but because of overheating or other environmental factors during incubation. Researchers should exercise particular caution when developmental abnormalities are suspected; for example, the hypertrophic neck muscles of an embryo, which are normal, can easily be thought to be pathological. Reference material and baseline data can prove invaluable, although there is a dearth of such information. There is also a paucity of information on specific gross and histopathological lesions in poultry embryos, and therefore interpretation of changes in raptors often must be speculative.[2]

The possible interaction between toxic chemicals and other factors can be difficult to determine: Newton (1979, pp. 128-149) discusses this in the context of breeding failures in free-living birds of prey. It is clearly important that toxicologists and pathologists cooperate closely.

Conclusions

There is a need for more research on causes of mortality in embryos and newly hatched chicks (Cooper, 1987a; Langenberg, 1989). Most of the work to date has been carried out on fowl, game birds, and certain waterfowl, but increasing numbers of raptor eggs are being submitted to laboratories for investigation, and this provides an opportunity to develop the subject.

One urgent need is for baseline data on normal embryonic development. Very little work appears to have been carried out other than the studies by Bird et al. (1984) on the

American kestrel (*Falco sparverius*) and Desai and Malhotra (1980) on the pariah kite (*Milvus migrans*). This is a disturbing situation considering the numbers of raptors, such as peregrines, that have now been successfully produced in captivity.

There is also a dearth of information on the prevalence and importance of transovarial infection in raptorial species. Even the role of bacteria isolated from dead embryos must often remain speculative. Research is needed similar to that already published on passerine birds (Pinowski et al., 1988).

Those who breed birds of prey in captivity could help tackle the problems of embryonic and chick morbidity and mortality by keeping better records and by collaborating more closely with veterinarians and avian pathologists.

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Notes

1. Personal communication with Helga Gerlach, University of Munich.
2. Personal communication with R. L. Julian, University of Guelph, Guelph, Ontario, Canada.

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CHAPTER 10

Fatty Liver-Kidney Syndrome of Merlins

NEIL A. FORBES, B.Vet.Med., M.R.C.V.S.

Clockhouse Veterinary Hospital
Wallbridge, Stroud, Gloucestershire, England

JOHN E. COOPER, D.T.V.M., F.I.Biol., M.R.C.Path., F.R.C.V.S.

Department of Pathology
The Royal College of Surgeons of England
London, England

ABSTRACT: Twelve European merlins (*Falco columbarius*) were examined postmortem. Four were birds taken from the wild; the remainder were captive-bred. All birds had been in captivity for at least three months at the time of death. All the birds had lesions consistent with those previously described as fatty liver-kidney syndrome (FLKS) of merlins. The exact cause of the disease remains a mystery, although a number of etiological factors can be excluded. At present FLKS is the largest single cause of death in captive merlins in Britain. As such, and in view of the great pressures on merlins in the wild, this condition is worthy of further research.

KEY WORDS: merlin, *Falco columbarius*, fatty liver-kidney syndrome, raptor, Britain, fat metabolization, diet for captive birds.

The merlin (*Falco columbarius*) is a small bird of prey (160 to 240 g) in the family Falconidae, order Falconiformes. In the wild it is found in North America and Eurasia, including Britain. Although the species has a widespread breeding area, the overall population density is low.

About 400 merlins currently live in Britain.[1] This ground-nesting species is under threat as traditional grouse moors decline. Roberts and Green (1983) describe the typical pitiful plight of a moor that once harbored a high density of nesting merlins, but that has become depleted. The only glimmer of hope appears to be that merlins are now found in increasing numbers nesting off the ground in the perimeter of young coniferous forests, itself perhaps a signal of a return to a pristine nesting condition.

Fatty liver-kidney syndrome (FLKS) of merlins was first recognized by Cooper and Forbes (1983). In a subsequent paper on morbidity and mortality in merlins, Cooper and Forbes (1986) found that FLKS of merlins was the single largest cause of death. FLKS may occur in the wild but has never been reported; this may be due to the paucity of postmortem material from free-living birds, or it may be that the condition is a problem associated with nutrition under conditions of captive management.

Fatty liver syndromes have been reported in captive parrots, parakeets, cockatoos, cockatiels, lovebirds (Arnall and Keymer, 1975; Baker, 1980; Minsky and Petrak, 1982), and hummingbirds (Ingram, 1978). In a large survey, Wadsworth et al. (1984) found fatty livers in penguins, sacred ibises, avocets, and masked weavers, as well as in cockatoos and parakeets. They found a higher incidence in psittacine birds than in other species, although in only one of their cases was the kidney affected as well as the liver.

Materials and Methods

Between 1978 and 1989, 12 cases were submitted to us either for gross postmortem examination or as specimens for histopathological evaluation. These formed part of a larger survey of morbidity and mortality in merlins (Cooper and Forbes, 1986).

The techniques for postmortem examination were as described by Cooper (1978). Routine laboratory diagnostic aids included bacteriology, histopathology, and parasitology. Toxicology was carried out on some individuals, but not virology or serology. The livers of four birds were weighed.

Data were retrospectively compiled by circulating a questionnaire to the keepers of all the birds. This covered the merlins' whole life span and sought information on a range of topics, including diet, fitness, activity, and previous illness.

Results

The findings are summarized in Table 10.1. Of the 12 birds affected, 4 had either been taken from the wild under license or were injured (casualty) birds. These birds were from diverse geographical areas. Their period in captivity ranged from 3 mon to 5 yr.

Table 10.1. Details of merlins that died of FLKS

Case	*Sex*	*Age (in years)*	*Time in captivity (in years)*	*Month of demise*
1 (CB)	M	1	1	May
2 (CB)	M	1	1	June
3 (CB)	M	2	2	June
4 (WB)	M	6	6	April
5 (CB)	F	3	3	November
6 (CB)	M	3½	3½	December
7 (CB)	M	1	1	March
8 (CB)	M	2	2	March
9 (WB)	M	7	5	August
10 (WB)	F	4	¼	January
11 (WB)	F	1	1	March
12 (CB)	F	6½	6	January

Note: CB = captive-bred; WB = wild-bred.

The remaining birds were all captive-bred. Many were related; this was probably due to the small numbers that were bred from 1975 to 1985. Descendants of these birds have not been affected in the period 1985 to 1990; hence, a hereditary or congenital weakness is thought unlikely.

All birds were in good body condition except two that had been suffering from severe bumblefoot. The birds' body weights postmortem were, with one exception, all slightly above (5%) their normal body weights. Some birds had been previously flown for falconry, but none was being flown (i.e., not in a reduced body weight) at the time death occurred. All were housed in aviaries.

In one case the bird was mildly off-color the previous day; however, in all other cases the birds were fit and had been carefully checked no more than 12 hr previously. In all cases death was sudden, for example, while eating or while with the keeper; the bird was seen to collapse and die immediately.

Two of the cases were birds with histories of nonfunctional preen glands, while five were birds that either were currently affected by unilateral or bilateral bumblefoot or had been so affected within the previous 2 mon.

Many, but not all, of the birds had been fed predominantly on a diet of day-old chicks for several months before death.

Although affected birds were from multiple-merlin "households," there was never more than one bird lost from a collection during any one period.

The four livers weighed were all 30% heavier than those of normal birds of the same size and sex.

In all cases the kidney and spleen were affected as well as the liver. The organs appeared grossly enlarged and white, and the disease could be differentiated from visceral gout only on histological examination. Rounding of the liver edges was observed.

Histological sections of liver showed extensive vacuolation of the hepatocytes and parenchyma. Similar vacuolation was present in the renal tubular epithelium and within the parenchyma of the spleen. Special stains confirmed the presence of lipid.

Discussion

In a healthy liver, fat is metabolized as follows. Fatty acids arrive in the liver via the plasma from two sources, as triglycerides from fat deposits around the body and as chylomicrons from the intestine (Thompson, 1978). These fatty acids are either used in the liver for metabolic processes or bound to protein produced in the endoplasmic reticulum and secreted from the liver into the plasma. They may leave as phospholipid or cholesterol compounds. Disturbances in synthesis or secretion may lead to accumulation of visible fat droplets in the hepatocytes or an increase in triglycerides in the blood, resulting in deposition in organs such as the liver, spleen, and kidney.

Thus, fatty change will occur as a result of any of the following:

1. Specific or nonspecific damage to hepatocytes. In addition, some nutritional deficiencies may interfere with protein production in the endoplasmic reticulum; lipoproteins cannot be formed and, as a result, the lipid cannot be secreted from the cell and therefore accumulates.
2. Interference with the release of lipoproteins from the cell.
3. Impaired combination of lipid with protein.
4. Blockage of oxidation of fatty acids.
5. Greater-than-normal amounts of fatty acid presented to the liver following absorption from the intestine or release from adipose tissue around the body.

Although fatty liver has a multitude of etiologies, it is possible to group the pathogenic mechanisms into those that involve an imbalance of nutrition or metabolic factors, those with a toxic basis, and those that are a result of cellular anoxia (Jubb et al., 1985). Apart from starvation diets, those that are low in protein or high in fat or carbohydrates have been shown to cause fatty liver.

In the poultry industry, FLKS is a metabolic disorder affecting immature chicks. It is characterized by sudden onset

of lethargy and paralysis, rapidly followed by death. Numerous studies of the nutritional and biochemical aspects of FLKS in poultry have been carried out (Pearce and Balnave, 1978), and it has been shown that the syndrome may be prevented by ensuring that the birds have an adequate supply of dietary biotin. In this situation the condition occurs due to impaired hepatic gluconeogenesis, which is associated with decreased specific activity of the key gluconeogenic enzymes (Bannister et al., 1975). It is the biotin-containing enzyme pyruvate carboxylase that is most severely affected, a situation readily reversed by supplementation with biotin.

In mammals it has been shown that the type of dietary lipid has significant effects on hepatic lipogenesis, with polyunsaturated fatty acids having a greater effect than saturated fatty acids (Bartley and Abraham, 1972). Fewer studies have been carried out with birds, but similar results have been obtained.

Apart from upset of the metabolism caused by external dietary factors, fatty change can be caused by endogenous imbalances, especially those involving deficiencies of insulin, thyroxine, or anteriohypophyseal hormones, for example, hypothyroidism and diabetes in dogs and cats, respectively.

There are numerous toxic factors known to cause fatty degeneration of the liver; examples include bacterial toxins, phytotoxins, and chemical poisons.

Frequently, when hepatic function is impaired, the increase in hepatic fat is accompanied by a lowering of plasma lipid levels. In an alternative situation, triglycerides may be synthesized at an increased rate. Usually the rate of triglyceride release is related to the level of usage. However, a hyperlipemia can occasionally be found concurrently with a fatty liver; in this situation increased triglyceride synthesis or reduced usage is likely.

In the majority of species the severest lipidosis is seen in metabolic diseases such as diabetes mellitus in dogs, ketosis in cattle, or pregnancy toxemia in sheep. Severe lipidosis is also seen in poisoning caused by phosphorus and plants of the species *Lupinus* (Jubb et al., 1985).

The significance of fatty liver depends on its cause, severity, and duration. Animals with fatty livers have increased susceptibility to superimposed toxic injury. In dogs, a rapid buildup of fat will sometimes lead to rupture of the fat-laden cells, release of fat droplets into the sinusoids, and widespread fat embolism.

Animals with fatty change without concurrent hepatic necrosis do not show clinical jaundice, and the detoxifying functions of the liver are not impaired (Jubb et al., 1985).

Conclusions

FLKS of merlins is a noncontagious, probably noninfectious, illness of merlins, which to date has been characterized clinically by sudden death. It is possible that less severely affected birds show no clinical signs and recover without treatment; biochemical analysis may elucidate this in the future.

In this study, there was no statistical difference in prevalence between male or female birds. The ages of birds affected varied from 1 to 7 yr, but more cases were in the 1- to 3-yr range, probably due to the short life span of merlins in captivity. Deaths occurred during all seasons of the year, with no predilection for any particular period. FLKS of merlins therefore appears to affect all ages and sexes of birds, at any time of the year. Various etiologies seem possible.

Unlike fatty liver hemorrhage syndrome of laying hens, no significant hemorrhage was found in any cases. In common with fat distribution in young chicks with FLKS, fat was present in the liver, kidney, and spleen. A biotin deficiency is implicated as one predisposing factor in young chicks with FLKS, and this could be a factor in merlins as well. In all but one case, the birds had been on a diet high in day-old chicks for longer than 2 mon. Day-old chicks are thought to contain an appreciable level of avidin,[2] which in turn will bind any biotin present, rendering the dietary biotin deficient, leading to reduced hepatic gluconeogenesis. However, in poultry the onset of signs of disease is more gradual than that seen in merlins.

Another factor that may be significant in merlins is the relatively high proportion of fat to protein in the diet eaten by captive birds. There could also be other deficiencies, such as riboflavin, pantothenic acid, choline, manganese or magnesium, leading to reduced oxidation of fatty acids.

A toxic or anoxic change to the liver that in turn retards triglyceride uptake from the plasma would also result in the same condition and might be responsible for the periacinar infiltration. There is histological evidence that there is hepatocyte rupture into the sinusoids, which would lead to fat embolism. This may account for rapid death following a primary fatty change in the liver.

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Notes

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2. Personal communication with C. Dawes, Royal Veterinary College, London, England.

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CHAPTER 11

Unhatched Eggs in Raptor Nests in Saskatchewan

C. STUART HOUSTON, M.D., D.Litt.

Department of Medical Imaging
University of Saskatchewan
Saskatoon, Saskatchewan, Canada

ROY D. CRAWFORD, B.S.A., M.S., Ph.D.

Department of Animal and Poultry Science
University of Saskatchewan
Saskatoon, Saskatchewan, Canada

LYNN W. OLIPHANT, B.S., M.S., Ph.D.

Department of Veterinary Anatomy
University of Saskatchewan
Saskatoon, Saskatchewan, Canada

ABSTRACT: In visits to 3,852 active nests of 10 diurnal and 3 nocturnal raptor species in Saskatchewan since 1966, we recorded the numbers of unhatched eggs. Among Falconiformes, from 6.3% to 16.7% of nests contained unhatched eggs. Among Strigiformes, only 4.1% of nests of great horned owls (*Bubo virginianus*) contained unhatched eggs. Great horned owls had unhatched eggs more commonly when food was scarce at the bottom of the 10-year cycle for snowshoe hares (*Lepus americanus*). Swainson's hawks (*Buteo swainsoni*) had unhatched eggs more commonly in years of heavy grasshopper infestation.

KEY WORDS: unhatched egg, infertility, embryo death, pesticide, great horned owl, *Bubo virginianus*, Swainson's hawk, *Buteo swainsoni*, raptor.

Remarkably little has been written about hatching failure—infertility, embryo death, or death during hatching (Ricklefs, 1969)—in North American raptors. The numbers of unhatched eggs in nests of the red-tailed hawk (*Buteo jamaicensis*), northern goshawk (*Accipiter gentilis*), prairie falcon (*Falco mexicanus*), and eastern screech owl (*Otus asio*) have been reported as 4%, 10%, 11%, and 18%, respectively (Luttitch et al., 1971; McGowan, 1975; Klaas and Swineford, 1976; Ogden and Hornocker, 1977). Long-term studies of the occurrence of unhatched eggs and their variation with the food supply have been performed in Europe, where addled eggs of the common buzzard (*Buteo buteo*) accounted for 4% of all incubated eggs in the best food years and 22% in the poorest years (Mebs, 1964).

We posed two questions for study in 1986 and 1987:

1. Does a "sloshing" ("addled") egg represent early embryo death or an infertile egg? Will candling establish the differentiation?

2. In a raptor nest in the wild, probably heavily contaminated with bacteria, will an infertile egg "dry down" or will it "slosh"?

Methods

The senior author and his many volunteer assistants recorded presence or absence of unhatched eggs in visits to active nests (with one or two parents present) of 10 diurnal and 3 nocturnal raptor species (Table 11.1) in Saskatchewan from 1966 through 1987. Eggs in nests with tiny young were not disturbed, since hatching is staggered in raptors and such eggs may hatch later. Eggs in deserted nests, with no parent birds present, were not counted.

If the smallest nestling was 1 or more wk old, the egg was shaken. Eggs that had an audible "slosh" from air and fluid content (suggesting cell death, autolytic breakdown of membranes, mixing of liquefied contents, and secondary bacterial overgrowth) were classed as "addled" ("rotten") and taken from the nest.

Two other categories of unhatched eggs are possible. When an embryo that is nearly fully formed almost fills the egg, there is sometimes a modest "slosh," but more commonly the egg feels solid. "Solid" eggs were taken from the nest only if the youngest nestling was at least 2 wk old.

Between 1966 and 1985, all addled eggs were collected, frozen, and sent to the Canadian Wildlife Service in Ottawa at the end of the season. Few of these eggs have been ana-

Table 11.1
Percentage of addled eggs for 13 species of Saskatchewan raptors, 1966-87

	1 *Number active nests c/young*	*2* *Number young banded*	*3* *Nests unhatched eggs only*	*4* *Total number active nests*	*5* *Number nests unhatched eggs*	*6* *Percentage nests unhatched eggs*	*7* *Number eggs in nests c/young*	*8* *Number eggs in nests s/young*	*9* *Total number unhatched eggs*	*10* *Eggs plus young*	*11* *Percentage unhatched eggs*
Diurnal raptors											
Swainson's hawk (*Buteo swainsoni*)	892	1,780	29	921	92	10.0	73	41	114	1,894	6.0
Ferruginous hawk (*Buteo regalis*)	360	1,095	8	368	39	10.6	41	10	51	1,146	4.5
Red-tailed hawk (*Buteo jamaicensis*)	171	287	2	173	14	8.1	13	3	16	303	5.3
Osprey (*Pandion haliaetus*)	97	195	2	99	12	12.1	10	3	13	208	6.3
Golden eagle (*Aquila chrysaetos*)	76	113	3	79	5	6.3	2	5	7	120	5.8
Northern harrier (*Circus cyaneus*)	71	286	0	71	6	8.5	8	0	8	294	2.7
Merlin (*Falco columbarius*)	72	291	0	72	6	8.3	9	0	9	300	3.0
Prairie falcon (*Falco mexicanus*)	66	258	1	67	7	10.4	8	1	9	267	3.4
Cooper's hawk (*Accipiter cooperii*)	16	49	0	16	2	12.5	2	0	2	51	3.9
Northern goshawk (*Accipiter gentilis*)	5	11	1	6	1	16.7	0	1	1	12	8.3
Total	1,826	4,365	46	1,872	184	9.8	166	64	230	4,589	5.0
Nocturnal raptors											
Great horned owl (*Bubo virginianus*)	1,863	4,207	12	1,875	76	4.1	67	20	87	4,294	2.0
Long-eared owl (*Asio otus*)	61	218	0	61	1	1.6	1	0	1	219	0.5
Short-eared owl (*Asio flammeus*)	44	142	0	44	2	4.5	2	0	2	144	1.4
Total	1,968	4,567	12	1,980	79	4.0	70	20	90	4,657	1.9

Notes: c = cum = with; s = sine = without; column 4 = column 1 plus column 3; column 6 = column 5 divided by column 4; column 9 = column 7 plus column 8; column 11 = column 9 divided by column 10. To calculate number of nests containing unhatched eggs *and* young, subtract column 3 from column 5.

lyzed chemically, but they have been kept frozen in a reference collection, available for retrospective study of pesticide content and eggshell thickness from that period and location. Once frozen, they are unsuitable for candling.

Because of the unusually wide variability in breeding activity and success in several raptor species, and because of some yearly variation in our effort with certain species, the number of addled eggs was compared with the number of active nests and with the total number of eggs produced each year.

In 1986 and 1987 we candled all eggs in the Animal and Poultry Science Laboratory, University of Saskatchewan, then opened them to look for evidence of embryonic development and to obtain samples for bacterial culture. The latter tests were performed in the Microbiology Department at the Western College of Veterinary Medicine.

In 1987, after 31 known infertile eggs from falcons at the University of Saskatchewan falcon-breeding facility had received full incubation, the eggs were kept under observation at room temperature for 6 wk, to see whether an infertile egg might dry down during incubation (as a hard-cooked egg does under sanitary laboratory conditions) or would proceed to the slosh stage.

Results

Most addled eggs remain unbroken in the nest while the nestlings grow to maturity. Addled eggs present at one visit usually remain at a second visit. They are not attractive to predators. All seven "solid" eggs encountered in nests of the Swainson's hawk (*Buteo swainsoni*) contained embryos.

The percentage of nests with unhatched eggs in our study

was in keeping with the findings of previously published studies for most diurnal raptors, falling between 6.3% for the golden eagle (*Aquilia chrysaetos*), and 16.7% for the northern goshawk (Table 11.1). In our five species with the largest sample sizes, there was a narrower variation: 6.3% for the golden eagle, 8.1% for the red-tailed hawk, 10.0% for the Swainson's hawk, 10.6% for the ferruginous hawk (*Buteo regalis*), and 12.1% for the osprey (*Pandion haliaetus*). Unhatched eggs were much less common among owls, and were present in only 4.1% of 1,875 active nests of the great horned owl.

Such rates of hatching failure are modest compared with those encountered in industrial poultry production, where accepted industry norms for chicken layer stock are a 90-95% hatch rate; for chicken broiler stock, 80-85%; and for turkey stock, only a 65-70% hatch rate (Crawford, unpublished data).

Annual samples were large and consistent enough to allow detection of statistically significant annual variations in two species, the great horned owl and Swainson's hawk. A dramatic change was seen in the ferruginous hawk in 1987 only.

The years when great horned owls had the most nests and most eggs per nest were the years with the lowest proportion of unhatched eggs, while years of fewer nests and fewer eggs per nest had a higher proportion of unhatched eggs. Regression analysis revealed a significant negative correlation ($p < 0.01$) between the percentage of addled eggs and the number of young plus unhatched eggs per nest (Houston et al., 1987). This correlation was only moderately strong, with an r value of 0.52. When the calculation was based on the percentage of nests that contained one or more addled eggs, the negative correlation remained significant but weaker, with an r value of 0.47. In a year of below-average owl success (Houston, 1975a, 1987), the increased percentage of addled eggs was only one of the measures of poor reproductive performance.

In the Swainson's hawk, comparing years of major use of grasshopper poison with years of little or no use, we found a statistically significant positive correlation between volumes of grasshopper biocides applied in the three prairie provinces of Alberta, Saskatchewan, and Manitoba (Sheehan et al., 1987) and the prevalence of addled eggs in our study area centered on Kindersley, Saskatchewan (Houston et al., 1991). Ferruginous hawks had a sudden but unexplained increase in the numbers of unhatched eggs in 1987, when there were 12 unhatched eggs in 7 of 53 (13.2%) of the active nests that year. Four eggs contained well-developed embryos—also unusual, in our experience. In one nest, two eggs, one dried down and one with the usual fluid content, were accompanied by a single young near fledgling.

Discussion

Except for three great horned owl eggs in which a tiny embryonic disc was recognizable, we were unable to differentiate between infertility and early embryo death in sloshing eggs. This is especially difficult in hot weather, perhaps because of increased destructive bacterial proliferation within such eggs. We therefore disagree with Campbell and Lack (1985, p. 3), who, without citing any authority for their decision, more narrowly define an addled egg as one "in which the developing embryo has died, as opposed to an infertile egg in which no development has taken place." Our use of the term *addled* refers to an egg that sloshes, and encompasses both infertile eggs and those with embryo death.

An infertile egg may indeed slosh. Of 31 infertile falcon eggs produced in captivity and left at room temperature after full-term incubation, 11 sloshed after 5 to 41 days. We broke open and examined 16 of these eggs; all were without embryos, but 9 had heavy mixed bacterial infection.

Results from random visits to raptor nests are not directly comparable to results of poultry science research. In the laboratory, a failed egg is studied promptly under sanitary conditions before decomposition sets in. In the field, when nestlings are of suitable age for banding, bacterial decomposition has probably been under way for 3 wk or more.

Only rarely does one encounter a dried-down, and hence assuredly infertile, egg in the field, perhaps in part because such eggs are more fragile as well as lighter in weight. We speculate that there may have been less bacterial contamination in such instances.

Once death of the embryo occurs, there is immediate failure of the normal bacteriostatic and immunologic defense mechanisms of the live organism; diversified microorganisms already residing on or in the eggshell soon proliferate in the excellent culture medium (Romanoff and Romanoff, 1949). Our bacteriology results showed mixed cultures of apparent contaminants (Houston et al., 1992). There was no evidence of intrinsic infection (a culture containing only one known avian pathogen) acquired within the oviduct of the adult female prior to egg laying (Cooper, 1987).

Reproductive failure in domestic fowl, a matter of great economic importance, has been studied for more than 100 years. In a classic paper, Riddle (1930), who studied doves, pigeons, and domestic fowl, reports that the embryos of all showed "two distinct periods of high mortality . . . very early in development and . . . very near the end of incubation." Insko and Martin (1935) found that for white leghorn chick embryos, the peaks of mortality fell on the second and nineteenth days, and for bronze turkey embryos, on the fourth and twenty-fifth days of incubation. We encountered the same two peaks in raptor hatching failure, the late peak being the less common of the two.

Yearly Variations in Percentages of Nests with Unhatched Eggs

Fluctuations in the annual occurrence of addled eggs were observed in two species. In the great horned owl, the incidence of addled eggs varies inversely with prey abundance. In the Swainson's hawk, the incidence of unhatched eggs increases when major grasshopper infestations occur and large quantities of grasshopper poisons are applied to district fields. Our recorded incidence of addled eggs in the northern harrier, short-eared owl (*Asio flammeus*), and long-eared owl (*Asio otus*) may be skewed downward because most of the nests of these species were found at the top of the vole cycle, in years of above-average breeding success, without adequate sampling of nests in low vole years.

In the great horned owl, which has a noticeable 10-year cycle roughly synchronous with that of its main prey species, the snowshoe hare (*Lepus americanus*) (Houston, 1987), there is an inverse relationship between prey species numbers and the number of addled eggs. In years when hares are scarce, fewer owls try to breed, and the number of owls produced per successful nest is low. In the years at the bottom of the hare and owl cycles, there is a statistically significant increased chance of finding addled eggs. At the top of the owl-hare cycle, there are fewer addled eggs. In 1986, the first year in which eggs were broken for direct examination, three contained small embryos, indicating failure early in incubation. One egg contained no recognizable embryonic material. In 1987, four owl eggs contained embryos, and four did not. In one instance each in 1986 and 1987, a fully developed dead owl embryo with pinfeathers and a prominent egg tooth was in malposition I of Landauer (1967), with the head lodged between the thighs, unable to gain access to the air space.

No correlation was observed between unhatched eggs and inclement weather. Indeed, a great horned owl was observed incubating two eggs successfully through temperatures as low as −34°C (Houston, 1965). We hypothesize that normal low temperatures might have adverse consequences only in the years at the bottom of the 10-year hare cycle. In such years the male might have greater difficulty in bringing sufficient food to the incubating female, who might be forced to risk leaving the eggs in below-zero temperatures to obtain some food for herself.

In Saskatchewan, great horned owls fledge by the end of May. On visits to owl nests in mid-May, addled eggs usually feel cool to the touch, indicating lack of recent incubation, and seem well preserved in cool spring weather (Houston, 1987). These cooler temperatures may account for better postmortem preservation and hence a higher percentage of identifiable embryos, perhaps due to less overwhelming secondary bacterial infection of the eggs in owls in contrast to hawks. Eggs of ducks, coots, and upland game, brought to the nests in the oviducts of prey species, are also found well preserved in owl nests (Houston, 1975b).

In contrast to the great horned owl, Saskatchewan young of the osprey and Swainson's hawk do not fledge until the last days of July and the first half of August. Is excessive bacterial overgrowth during hot days able to destroy some early dead embryos and thereby explain the decreased prevalence of embryonic tissue found in eggs of diurnal raptors, compared with owls (Houston et al., 1991)? Certainly, after several weeks of bacterial multiplication, candling is unable to differentiate between infertility and embryo death.

In 1986 we candled 17 eggs of the Swainson's hawk and 3 of the ferruginous hawk that had been exposed to ambient temperatures of up to 35°C. We could only report that most were "totally rotten," with no evidence as to whether an embryo had been present or not. Three with possible blood rings (possible first indications of embryo development), as determined at candling in 1986, failed to show embryonic tissue on histologic examination. In 1987 only 1 of 12 Swainson's hawk eggs had recognizable embryonic tissue.

The amounts of individual agricultural chemicals used in Saskatchewan are known to vary from year to year, but figures are generally not divulged in the province. Only the total number of hectares sprayed for neurotoxic, short-lived grasshopper poisons is available, and that only for the years 1974 to 1985 (Sheehan et al., 1987). Since most applications of these chemicals (carbaryl, dimethoate, chlorpyrifos, and deltamethrin) occurred after egg laying each year, a cause-and-effect relationship with increased addling of Swainson's hawk eggs is credible only if one postulates direct damage to the egg or an effect on adult behavior, for example, decreased hunting success by the adult male through the anticholinesterase effect of carbofuran. At present we merely note an apparent association between increased addled eggs on the one hand and increased grasshopper infestations and increased use of pesticides on the other.

Rigidly controlled studies have found some statistical evidence that carbofuran, the cheapest of the effective grasshopper poisons, is associated with drastic declines in reproductive success in the burrowing owl (*Athene cunicularia*) (James and Fox, 1987).

Conclusions

Future studies should search for a better means to differentiate between infertility and early embryo death, especially since some individual agricultural chemicals might cause one more than the other. We have not yet tested the 3-hydroxybutyric acid content of eggs as an indicator of embryonic development (Elenbaas et al., 1986). Since various

biocides have been shown to increase reproductive failure in some species of birds, further data collection should be encouraged. Knowledge of the normal range of frequency of addled eggs for each species may prove useful in appreciating any rise above these levels as an additional clue in early detection of damage by biocides.

Our retrospective data, collected incidental to a long-term banding effort, point to the need for more detailed studies, including histologic examination of dead embryos. We encourage others to take an interest in this neglected area.

Acknowledgments

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CHAPTER 12

Cyathostoma Infections in Screech Owls, Saw-whet Owls, and Burrowing Owls in Southern Ontario

D. BRUCE HUNTER, D.V.M., M.Sc.

Department of Pathology
Ontario Veterinary College
University of Guelph
Guelph, Ontario, Canada

KATHERINE McKEEVER

Owl Rehabilitation and Research Foundation
Vineland Station, Ontario, Canada

CHERYL BARTLETT, B.Sc., Ph.D.

Department of Zoology
University of Guelph
Guelph, Ontario, Canada

ABSTRACT: Nematodes of the genus *Cyathostoma* were identified as the cause of death in a wild saw-whet owl (*Aegolius acadicus*) and two wild screech owls (*Otus asio*) submitted to a rehabilitation facility and in four burrowing owls (*Speotyto cunicularia*) bred and raised in a captive-propagation project. The pathologic changes included granulomatous airsacculitis and pneumonia. Treatment of the surviving burrowing owls consisted of injections of ivermectin (200 μg/kg body weight) and control of intermediate and paratenic hosts by building an underground barricade and treating the soil within the compound with benomyl, a fungicide that is toxic for earthworms.

KEY WORDS: *Cyathostoma*, Strigiformes, saw-whet owl, *Aegolius acadicus*, screech owl, *Otus asio*, burrowing owl, *Speotyto cunicularia*, case history, captive-breeding projects, air sac, respiratory system, ivermectin, raptor.

Nematodes of the genus *Cyathostoma* parasitize the trachea and respiratory tract in a variety of species of birds, but are considered uncommon pathogens for birds of prey. Two species of Cyathostomes have been reported from birds of prey. *C. americana* (Chapin, 1925) has been identified in the eastern North American red-tailed hawk (*Buteo jamaicensis*) and in the long-eared owl (*Asio otus*) and goshawk (*Accipiter gentilis*) in the Soviet Union (Ryzhikov, 1980). *C. brodskii* has been reported from the marsh harrier (*Circus aeruginosus*) in Uzbekistan (Ryzhikov, 1980). Gapeworms of the genus *Cyathostoma* were reported to cause mortality in a wild eagle owl (*Bubo bubo*) in Switzerland (Mumcuoglu and Mueller, 1974).

The Owl Rehabilitation and Research Foundation (ORRF) is a rehabilitation center for injured owls and a research facility specializing in the captive breeding of nonreleasable North American owls. Approximately 100 to 150 injured owls are treated annually, and there is a resident breeding population of 92 birds of 15 species. This chapter describes fatal parasitic pneumonia in three injured wild North American owls—two screech owls (*Otus asio*) and a saw-whet owl (*Aegolius acadicus*)—that had been presented to the ORRF, and four juvenile burrowing owls (*Speotyto cunicularia*) bred and raised in captivity.

Case Histories

Case 1

A 5- to 6-week-old wild saw-whet owl suffering from assorted traumatic injuries was found near St. Catherines, Ontario, and taken to the ORRF. The bird was treated for nine days and appeared to be responding well, but on the tenth day became suddenly depressed, developed labored breathing, and died.

At necropsy the owl was in good body condition. The main lesions were pulmonary congestion and pneumonia. Occasional nematodes were observed in the trachea and main stem bronchi, and numerous parasites were present in the right cervical and anterior thoracic air sacs and in the ceolomic cavity. The walls of the air sacs that contained parasites were thickened and opaque. Samples of lung

and other tissues (liver, heart, spleen, kidney) were fixed in buffered 10% formalin for microscopic examination. Parasites were placed in formalin for further identification.

Case 2

An immature male screech owl was found with a fractured wing near Unionville, Ontario, and sent to the ORRF. The bird was placed in an indoor hospital facility and treated, but died on the eighth day of treatment.

Parasitic pneumonia and airsacculitis were evident at necropsy. Occasional nematodes were found in the trachea, and 10 adult parasites were located in the anterior thoracic and cervical air sacs.

Case 3

An immature screech owl was found in late November 1986 near Niagara Falls, Ontario, and sent to the ORRF. It was successfully treated for injuries and placed in an outdoor flight cage. One month after it was placed outside, it was found dead.

At necropsy, large numbers of nematodes were present throughout the abdominal cavity and thoracic and abdominal air sacs. Occasional parasites were in the trachea and main stem bronchi. The air sacs had opaque membranes that were discolored brown and contained fibrin and fluid. Parasites were fixed in warm 90% alcohol for further identification and selected organs fixed in formalin for microscopic examination.

Case 4

The 1987 report of the Committee on the Status of Endangered Wildlife in Canada considers the burrowing owl an endangered species in British Columbia and Manitoba and a threatened species in Saskatchewan and Alberta. A propagation project initiated at the ORRF in 1981 has successfully released more than 40 captive-bred burrowing owls into former habitat in the upper Okanagan Valley, British Columbia. In 1986, 17 young owls were fledged from four captive-breeding pairs. Four of these juveniles died from September to January of 1986, and in each of the owls there was severe pneumonia and airsacculitis (Fig. 12.1). Large numbers of nematodes were in the clavicular, thoracic, and abdominal air sacs and occasional worms were found in the trachea and main stem bronchi. Parasites were fixed in warm 90% alcohol and samples of selected organs were fixed in buffered formalin for histology.

Parasite Identification

The parasites from each of the species of owls in this report were identified, following Lichtenfels (1980), as belonging to the genus *Cyathostoma* and the subgenus *Hovorkonema*, but the state of preservation precluded identification of the species.

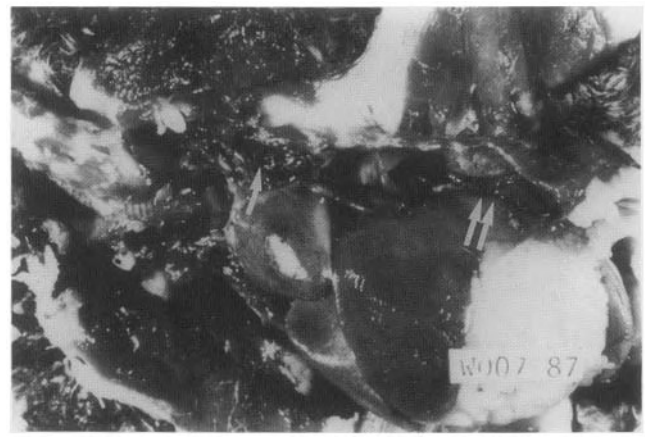

Figure 12.1. Burrowing owl dead with parasitic air sacculitis and pneumonia. Cyathostomes are present in cranial air sacs (*single arrow*) and posterior air sacs (*double arrow*).

Microscopic Findings

The microscopic findings in all owls were similar, but varied in severity. There was intense airsacculitis and pneumonitis, usually associated with either parasite eggs or adult nematodes. The pneumonia was severest in those parts associated with secondary bronchi that communicated with air sacs. Normal architecture of affected lung tissue was lost, and the lungs were congested and infiltrated with mixed inflammatory cells and fibrin. Golden pigment, likely hemosiderin, was common within macrophages and free in areas of inflammation. Parasite eggs and remnants of cuticle were present within areas of necrosis and often within the lumen of parabronchi (Fig. 12.2). Some areas of necrosis were walled off by macrophages and giant cells. Adult nematodes were numerous in air sacs, occasionally present within bronchi and parabronchi, but uncommon in either primary bronchi or trachea. The inflammatory reaction associated with anterior air sacs extended to cause inflammation of the adjacent esophagus. The primary bronchi and trachea were not affected, although cellular debris and parasite eggs were often present in the lumen of these structures.

Treatment and Control

The deaths of the wild owls (Cases 1, 2, and 3) were unexpected, and no treatment was given. Case 4 involved a captive-breeding colony of burrowing owls, and after the first death a rigorous treatment and control program was undertaken. All remaining adult and juvenile burrowing owls

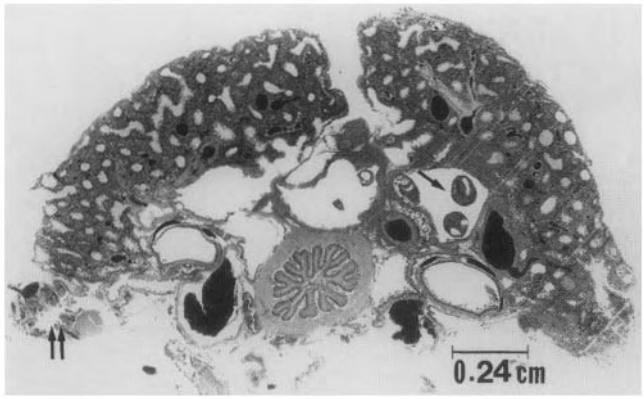

Figure 12.2. Cross section through cranial portion of the lungs from a burrowing owl infected with *Cyathostoma* sp. Cyathostomes are present within parabronchi (*single arrow*), and the surrounding tissue is consolidated and inflamed. Granulomatous lesions containing parasite eggs are present in the cranial air sacs (*double arrow*).

were taken out of the burrowing owl compound and treated with ivermectin[a] at a dose rate of 200 μg/kg body weight.

As earthworms are considered to be an important intermediate host of *Cyathostoma*, and were numerous within the compound and often found within water bowls, a program of earthworm control was instituted. A trench approximately 1 ft wide and varying between 3 and 4 ft deep was dug around the perimeter of the compound and filled with concrete. This earthworm barrier extended approximately 6 in above the ground. The ground within the compound was treated with benomyl[b] at 2 kg/acre to kill earthworms. The compound was left empty over winter, and owls were reintroduced in early spring.

Discussion

This chapter describes the pathology, treatment, and control of cyathostomiasis in a captive collection of burrowing owls, two wild screech owls, and a saw-whet owl. To our knowledge this is the first report of *Cyathostoma* sp. infection in these genera, and is evidence that this parasite is capable of causing mortality in juvenile wild owls in southern Ontario.

Lesions within the owls were severest in air sacs, particularly anterior air sacs, rather than in lung tissue. Although there was host reaction to larval and adult parasites, the severest lesions appeared to be a result of inflammatory response to the parasite ova. In geese infected with *Cyathostoma bronchialis*, the adult parasites reside in the bronchi and lesions are present mainly in the lung parenchyma (Fernando, Stockdale, and Ogungbade, 1973; Fernando, Hoover, and Ogungbade, 1973). Lesions in the air sacs of the geese were less common and thought to be a result of aspiration of parasite ova. In the present case studies, the large number of gravid adult parasites and parasite ova observed more often within the air sacs than within parabronchi or the trachea suggests that this parasite may not be well adapted to Strigiformes.

It is unfortunate that the poor preservation of the parasites did not allow species identification. It is possible that the parasite in the burrowing owls was *C. bronchialis* of waterfowl origin, as the ORRF is located on the bank of the Jordan estuary. Ryzhikov (1980) reported experimentally infecting a young red-tailed hawk with *C. bronchialis* originally isolated from a domestic goose. Further research is necessary to determine if *C. bronchialis* is unique to waterfowl.

The earthworm-control program appeared successful, as no mortality occurred in juvenile burrowing owls during the 1987-88 breeding season. The underground earthworm barricade also restricted entry into the owl compound of other insectivorous paratenic hosts, such as shrews and moles, which are common in the area.

The deaths in the burrowing owls demonstrate that disease can be a significant factor in the success of a captive-propagation project. It is important to establish efficient health and disease monitoring programs to ensure that disease-causing agents are not introduced into wild populations during the release of these birds to the wild.

Product List

[a]Ivomec, MSD AGVET, Division of Merck Frost Canada Inc., Box 1005, Pointe Claire, Quebec H9R 4P8, Canada

[b]Benlate, 50% wettable powder, E. I. du Pont de Nemours and Co. Inc., Wilmington, Delaware, 19898 USA

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CHAPTER 13

Causes of Death in Radio-Tagged Northern Goshawks

ROBERT E. KENWARD, B.A., M.A., D.Phil.

Institute of Terrestrial Ecology
Furzebrook Research Station
Wareham, Dorset, United Kingdom

VIDAR MARCSTRÖM, B.Sc., M.Sc., Ph.D.
MATS KARLBOM, J.M., S.M.

Institute of Zoophysiology
University of Uppsala
Uppsala, Sweden

ABSTRACT: From 1980 to 1987, wild (free-living) northern goshawks (*Accipiter gentilis*) were studied by radio-tagging on the Baltic island of Gotland. Of 220 juveniles and 132 adults, 67 were killed or found dead while they were being radio-tracked, and a further 89 banded (ringed) hawks were recovered dead between 1970 and 1988 without the aid of radio tags. Shooting and trapping accounted for 47% of the band recoveries, compared with only 36% of deaths among radio-tagged hawks. Other banding recoveries were significantly later in the biological year (July to June) than expected from the pattern of natural mortality.

Of the 67 radio-tagged hawks, 43 apparently died of natural causes, although 16 were radio-located too late for proper necropsy. Among the remaining 27 natural deaths, 33% died from predation (2) or with broken bones (7), and 37% (10) died of starvation with no sign of disease. Another 22% (6) starved, but pathological conditions (parasites, proventricular ulceration) may have contributed to their inanition. Only 2 of these birds probably died primarily from disease. There was no significant difference between juveniles and adults in the main causes of death, but starvation was recorded only in late winter (February-March) for adults, whereas juveniles starved throughout the autumn and winter. Levels of heavy metal and organochlorine residues were low.

We conclude that radio-tagging is an effective method for determining the cause of raptor deaths, partly because it minimizes recovery bias, and partly because it provides many carcasses fresh enough for autopsy. Our study suggested that disease was relatively unimportant as a primary cause of mortality among goshawks in Gotland.

KEY WORDS: northern goshawk, *Accipiter gentilis*, radiotelemetry, mortality, raptor death.

It is accepted practice in human medicine to direct most resources to combat those diseases that cause most mortality or morbidity. Registration of deaths or hospital admissions provide data for assessing the prevalence of each disease. Among captive raptors, too, there have been collections of autopsy findings (Keymer, 1972; Cooper, 1978; Cooper et al., 1981; Schröder, 1981) to indicate the most common agents of mortality, and complete "cohorts" of birds brought into captivity have been studied to determine the relative importance of disease or other factors in causing deaths (Kenward, 1981).

For wild (free-living) raptors, there have been reviews of autopsy records (Keymer et al., 1981), or of findings among incapacitated birds (Hurrell, 1967; Greenwood, 1977; Redig, 1978), complemented by cause-of-death records from banding returns (Coon et al., 1969; Glue, 1971; Weir, 1971; Newton; 1979). However, it is difficult to be sure how much the relative incidence of each mortality agent may have depended on recording bias. Newton (1981) notes that "a bird which kills itself by crashing through a kitchen window is almost certain to be noticed, whereas one which is eaten by a predator in the forest is almost certain to be missed," and the same "recovery bias" must act against birds that die of disease or starvation in places little visited by humans. Moreover, a fresh corpse in the kitchen or on the roadside is a better subject for autopsy than one rotting in the forest. For example, no less than 48% of sparrowhawks (*Accipiter nisus*) sent for analysis at the Institute of Terrestrial Ecology, Monks Wood, had died from collisions or other accidents (Newton et al., 1981). There may also be a greater tendency to submit a corpse for clinical examination if it is obviously diseased. In these cases, "reporting bias" can increase the effect of an original recovery bias.

There may be less recording bias if the live wild population is sampled for the occurrence of potential pathogens

(Needham et al., 1979; Redig et al., 1980; Peirce and Marquiss, 1983; Cooper et al., chapter 7, this volume), although diseases may be overrepresented if they reduce condition and thus increase the tendency to enter a trap (Mueller and Berger, 1970; Marcström and Kenward, 1981). Bias can be minimized only if individuals from a population can be sampled at random, or if complete cohorts can be followed to their deaths. We used the latter approach among goshawks on the Swedish island of Gotland, by radio-tagging juvenile hawks at the nest so that they could be tracked to record mortality and causes of death during their first year. These data are compared with material from radio-tagged adults that had been trapped, and from banding from Gotland within a decade of our study.

Methods

Gotland is an island of 3,100 km^2 in the center of the southern Baltic Sea. During the study period, from 1980 to 1987, it had 150 to 200 breeding pairs of the northern goshawk (*Accipiter gentilis*), and band recoveries showed that only 2-3% of the hawk population moved to or from the island (Marcström et al., 1990). A strip of 650 km^2 across the center of the island was used as an intensive study area, in which most if not all nests were found during the mortality study (1980-85). This area contained about 20 nests each year, and the young hawks were radio-tagged at 14 to 18 of them.

About 70% of the 220 radio-tagged young (114 males, 106 females) were fitted with 10- to 18-g leg-mounted tags[a] at 4 to 10 days before they left the nest. Most of these, and young without leg tags, were recaptured in box traps 3 to 5 wk after they left the nest. By this time their tails were fully grown, and they were fitted with 15-g tags across two rectrices. The antenna system of tail mounts, which were eventually molted, was more durable and more efficient than that of leg mounts, giving working ranges of 3 to 7 km from hawks in trees (occasionally 10 to 20 km) during the expected tag life of 9 to 12 mon.

The young hawks were radio-located every 1 to 3 days during the postfledgling period, and thereafter at 3- to 7-day intervals for the 1980-83 cohorts but at 2-mon intervals in 1984 and 1985. Checks for survival were mostly made at night, when live hawks were roosting in trees and thus radiating signals farther than if they had been eating a kill on the ground during the day. The checks were made in a minibus, using a six-element Yagi antenna mounted horizontally with a repeater compass on a hand-elevated mast (Cederlund and Lemnell, 1980) and RX-81 receivers.[b] The tail mounts contained mercury switches as posture sensors (Kenward et al., 1982), so that roosting hawks gave strong, slow signal pulses, whereas signals that were weak and (usually) fast suggested that a hawk had died or shed its tag. If there were several tags that could not be found from the minibus or from elevated points in the previous ranges of the hawks, the island was searched from the air. For further details of trapping, tagging, and tracking, see Karlbom (1981), Kenward et al. (1983), and Kenward (1987).

The aim was to tag all the young at each nest, but 10% with leg-mount tags escaped tagging with tail mounts (Kenward, forthcoming). Up to 20 box traps were set during autumn and winter at sites throughout the island to capture juvenile and adult hawks, of which 132 (60 male, 72 female) were radio-tagged. In the absence of death, tag failure, or premature molting of tags, young hawks could be tracked routinely from July to April. Most tags on adults, and some on juveniles, could be monitored until they were molted in the summer.

Provided that carcasses were fresh enough, hawks that were found dead were sent for necropsy at the Statens Veterinärmedicinska Anstalt (SVA) in Uppsala, Sweden. Records of these birds, and of those killed by humans (which was legal in defense of poultry), provided a minimum estimate of mortality rates. To estimate how often signals were lost because tags were damaged when hawks were killed, we compared subsequent recapture rates (1) for the hawks whose signals were lost without known cause and (2) for the hawks whose tags were known to have failed or to have been molted. The recovery rates in both categories were identical, which suggested that unaccountable losses were almost entirely due to tag failure that was not associated with death of the hawk (Kenward, forthcoming).

Results and Discussion

Mortality Patterns

Nestling goshawks at banding age had a mortality rate of 14% in their first 3 mon postfledgling, which rose to 31% by the end of 6 mon and was 48% for the year as a whole. The rates in each 3-mon period differed between the radio-tagged birds that died of natural causes and those killed by humans. Of the 15 deaths from shooting or trapping, only 4 (27%) were in July-September, compared with 9 (60%) in October-December, whereas 16 (53%) of the 30 natural deaths were recorded in the first 3 mon after fledgling and only 9 (30%) in October-December.

Among banded juvenile goshawks without radio tags, the seasonal pattern of deaths from shooting or trapping peaked in October. Similarly, 18 (75%) of the 24 radio-tagged hawks were killed by humans in October-December. There was a major difference, however, between banding and radio-tagging in the seasonal pattern of natural mortality. Of the 30 band recoveries, 18 (60%) were after December, with a peak of 10 in April-June, and this was significantly later than the recovery distribution expected

Table 13.1
Causes of death in 67 radio-tagged juvenile and adult goshawks on Gotland

	Juveniles N	%	*Adults* N	%	*Combined* N	%
Killed by humans	15	33	9	41	24	36
Killed by hawk	1	2	1	5	2	3
Other trauma	5	11	2	9	7	10
Disease as primary cause	2	4			2	3
Starvation with disease	3	7	3	14	6	9
Starvation as primary cause	8	18	2	9	10	15
Natural death, cause unknown	11	24	5	23	16	24
Total	45		22		67	

from radio-tagging (median test, two-tailed $p = 0.018$). We suspect that the finding of the banded hawks was strongly biased: ground cover probably reduced the detection of dead birds in late summer and autumn, so that many were not found until children and ramblers again took to the woods in the spring.

Causes of Death

The 67 radio-tagged hawks that died could be divided into seven categories, with no difference between adults and juveniles in the prevalence of each category (Table 13.1). Whereas 47% of the banded goshawks had been killed by humans, only 36% of the radio-tagged hawks were attributed to this group, as a result of being reported dead, found dead with shot damage to feathers and or tissues, or recovered from a burial site, dung heap, or garbage container by radio-tracking. Of the remaining hawks, 24% were found away from dwellings and without shot marks, and had therefore probably died naturally, but were recovered too late for a proper necropsy.

Twenty-seven hawks, 40% of the total, had died from identifiable natural causes. One radio-tagged adult male was killed and eaten by a female in February. The male was a second-year bird that also had been radio-tagged as a juvenile, and had weighed 975 g when caught and retagged as an adult the previous October. It was still in good condition, with a full crop of rabbit, when killed by the female, who was herself recaught on the kill and weighed 1,725 g. Since the mean weight of adult Swedish goshawks is about 840 g in October for males, with females at about 1,500 g in February (Marcström and Kenward, 1981), both birds were above average in weight. One young found near the nest shortly after fledging had injuries consistent with being killed by another hawk, possibly a sibling. Evidence of sibling aggression was seen at two nests that were receiving little food in 1987: one male was killed by its brother, and another starved the day after fighting with its sister. (These cases are omitted from Table 13.1 because mortality was not being monitored systematically in 1987. Also omitted is the nestling male of 140 g that we euthanized in 1981 after it was gravely injured by its sisters of 415 g, 480 g, and 520 g.) Fratricide and cannibalism among goshawks have been recorded by several other authors (Uttendörfer, 1952; Kollinger, 1962; Brüll, 1964). Our records of 2 among 27 deaths (7%) probably do not overestimate its importance. Eagle owls (*Bubo bubo*), which frequently kill and eat goshawks (Mikkola, 1976), did not breed on Gotland during the study period.

Of the 27 natural deaths with known causes, another 7 (26%) were of hawks that had broken bones or other injuries consistent with impact accidents. Adding the 2 killed by hawks, trauma thus accounted for one-third of the natural deaths, with the remainder attributable to starvation or disease (Table 13.2). In 10 of these hawks (37% of the 27) there were no pathological findings other than low weight, with means of 620 g (SE = 50 g, $N = 5$) in males and 880 g (SE = 90 g, $N = 5$) in females. Marcström and Kenward (1981) gave means of 830 g (SE = 60 g, $N = 222$) and 860 g (SE = 80 g, $N = 47$) for juvenile and adult male goshawks live-trapped during August to March in southern Sweden, with 1,150 g (SE = 90 g, $N = 151$) and 1,290 g (SE = 220 g, $N = 51$) for juvenile and adult females. They considered that males of 600 g or less and females at 950 g (or with less than 1% by weight of fat) were unlikely to survive.

In another six birds, death was apparently due to starvation, but other pathological factors may also have contributed. Three hawks had ulcerated proventriculi, a finding that is not uncommon in captive raptors (Cooper, 1978), and three other hawks either had severe infestations of nematodes (*Porrocaecum depressum*) or abundant coccidia (*Isospora* sp.) with associated enteritis (Table 13.2). These conditions may have predisposed the birds to starvation, or become prevalent during gradual loss of condition. Among

Table 13.2
Autopsy results from 18 radio-tagged goshawks that died of inanition or disease

Probable primary cause of death	*Number*
Starvation	
Inanition with no evidence of disease	10
Ulcerated proventriculus	3
Abundant coccidia (*Isospora*), few *Capillaria falconis* and *Porrocaecum depressum*	2
Abundant *Porrocaecum depressum*	1
Disease	
Inflamed thigh muscle, *Pasteurella multocida*	1
Acute circulation failure in heart, lungs, liver and limbs	1
Total	18

these and the other starved birds, we noted that all five adult deaths occurred in late winter (February and March), whereas juvenile starvation occurred from autumn onward, as noted in other raptors (Hirons et al., 1979; Newton, 1986).

In the absence of other indications, only two deaths appeared to result primarily from disease. One juvenile male was at a reasonable weight in its first August (695 g), but had suffered acute circulatory collapse with no identifiable cause. The other bird, a juvenile female, had starved to death in September (755 g), but had a severely inflamed upper leg from which the bacterium *Pasteurella multocida* was cultured. This infection may have been the result of an injury; Lumeij (1986) recently recorded a similar case in a parrot that had been bitten by a tame rat. *P. multocida* is recognized as part of the "normal" flora of the oral cavity of many species of mammal.

To complete the picture, residue levels of heavy metals and organochlorines were determined in a small number of hawk livers (Table 13.3). In all cases the contamination was too low to have contributed to the deaths of the birds concerned.

Conclusions

Among the natural deaths of radio-tagged hawks, one-third resulted from predation or accidents, with most of the remainder apparently due primarily to starvation. Although parasites in particular may have important roles to play during the fluctuations of dense game-bird populations (Hudson, 1986), disease seemed unimportant for this stable population of a reasonably abundant raptor, perhaps partly because there is reduced scope for conspecific infection in such a solitary predator, and partly because of strong selection for resistance to infectious diseases carried by prey.

The large number of deaths caused by humans shows that this goshawk population was experiencing a considerable cull. Nevertheless, because a high proportion of the natural deaths were by starvation, and no hawks bred in first-year plumage, as occurs when goshawks are under severe pressure (Looft and Busch, 1981; Link, 1986), it is probable that more birds would have starved in the absence of shooting or trapping.

Table 13.3
Geometric mean concentrations of heavy metals and organochlorines (ppm) in fresh liver tissue of goshawks killed or found dead on Gotland during 1980-85

	Mercury	*Lead*	*DDE*	*HCB*	*PCBs*
N of samples	20	3	5	5	5
Mean value	0.47	0.27	0.77	0.01	0.80
Range of values	0.01-4.16	0.14-0.70	0.16-1.86	0-0.26	0.26-8.70

Note: Cyclodiene concentrations never exceeded 0.01 ppm.

If patterns of mortality are to be used to determine crucial periods in the biological year, it is important that the findings not be biased by recoveries from periods when shooting or trapping was particularly intensive or by long delays before summer ramblers find hawks that died naturally the previous winter. Radio-tagging probably gives the least biased view of mortality causes and patterns.

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Product List

[a]Biotrack, Wareham BH20 5AJ, England

[b]Televilt HB, 71050 Stora, Sweden

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CHAPTER 14

Causes of Breeding Failure in Wild Raptors: A Review

I. NEWTON, B.Sc., D.Phil., D.Sc.

Institute of Terrestrial Ecology
Monks Wood Experimental Station
Abbots Ripton, Huntingdon
Cambridgeshire, England

ABSTRACT: The maximum possible breeding rate is greater in small raptor species than in large ones. To judge from published studies, most species produce less than half the maximum possible number of young per year. This is due partly to failure to lay eggs or to produce the maximum clutch, and partly to subsequent losses of eggs and young. Typically, small raptors raise 2 to 3 young per pair per year, medium-size raptors produce 1 to 2 young per pair per year, and large species 0.4 to 0.8. Within species, in the absence of human intervention, much of the variation in productivity is associated with variation in food supply. Experimental provision of extra food has advanced the laying date and improved the breeding performance of wild sparrowhawks and kestrels. Food shortage is often a predisposing factor in nest desertions and other failures.

Weather influences breeding success indirectly through its effects on prey availability and hunting success and directly through causing nest desertions and nestling deaths. Natural predation causes some losses irrespective of food supply, and varies in relation to the situation of the nest and other factors. With a few local exceptions, disease and parasites are generally unimportant causes of nestling deaths. Compared with most other birds, raptors show an unusually high frequency of egg addling and chick starvation, but suffer relatively little predation of eggs and chicks. Among populations affected by organochlorine pesticides, shell thinning and egg breakage are associated with DDE, a metabolite of the insecticide DDT. Mercurial compounds are also known to reduce the breeding rates of some raptor species.

KEY WORDS: raptor, breeding rate and food supply, breeding failure, clutch, brood size, nest desertion and food supply, weather, predation, disease, parasites, egg addling, organochlorine pesticides.

This review will present an examination of the extent and causes of breeding failure in diurnal raptors found in previously published studies. Thus, the focus will be on those factors that reduce reproductive rates below the maximum possible. These include the failure to lay eggs as well as the various mortality agents that subsequently reduce the number of eggs that give rise to independent young.

Some Generalizations

The Potential Breeding Rate

Different species of raptors vary greatly in their maximum possible breeding rate, depending largely on their body size. In general, the larger the species, (1) the later the age at which breeding begins, (2) the longer each successful attempt takes, and (3) the fewer the young produced. At one extreme, small falcons or sparrowhawks can start breeding in their first year. They lay 5 to 6 eggs per attempt and take only 80 days to produce independent young. With one brood per year, the maximum annual increase in population possible is thus 3.5 to 4.0 times the breeding population, if all birds survive. At the other extreme, the large condors do not begin breeding until they are more than 5 yr old. They lay only 1 egg at a time, and take about 485 days to produce independent young, so that annual breeding is impossible. Ignoring the nonbreeding immatures, the maximum likely increase in a condor population is 50% in 2 yr or 25% in 1 yr. These examples represent the extremes; other raptor species are intermediate in all respects, depending on their body size (Newton, 1979).

The Breeding Process

To produce young, a bird has to pass successfully through a number of stages. It must first establish a territory and acquire a mate. It must then proceed through nest building, on to egg laying, and thence to the hatching and rearing of young. Birds can fail at any stage in the process. Some pairs may hold a territory for only a few days or weeks, and may or may not build a nest, but get no further. Other territory holders may lay their eggs and then desert them, while others may hatch their young but not rear them. And not all successful pairs will lay the maximum possible number of eggs, hatch all the eggs they lay, or raise all the young they hatch. As a result of such complete and partial breeding failures, few of the raptor populations that have been studied have produced more than half the maximum possible number of young each year, if this number is defined as the number of territorial pairs times the maximum clutch size.

Typically, small kestrels and sparrowhawks produce 2 to 3 young per territorial pair per year, medium-sized raptors about 1 to 2 young per pair per year, and large vultures and eagles around 0.4 to 0.8 (Newton, 1979). These rates may be reduced further under exceptionally adverse circumstances.

Difficulties of Study

It is not easy to obtain unbiased estimates of the full extent of breeding losses in raptors, especially as some pairs move away or become inconspicuous after their nesting attempts have failed. So, unless observers are active from the start of the season, they will miss early failures, and their samples may be biased in favor of successful pairs that are still present at later visits. Similarly, unless one determines initial clutch size, it is impossible to assess the extent of egg and chick loss later in the cycle. Many of the published studies of breeding success in raptors are marred by such deficiencies, and only a minority give enough detail for a full assessment of breeding losses.

A second type of problem involves the distinction between ultimate and proximate causes of failure. The food supply is clearly the major factor influencing the breeding rate of most species that have been studied, with pairs producing more young per attempt in good food conditions than in poor ones. The effects of food shortage are manifested in various ways, however, including nonlaying, failure to produce the maximum clutch, desertion of clutches, and starvation of chicks. Moreover, starving chicks are vulnerable to other factors, and may suffer more predation, disease, and parasitism than healthy chicks. In all such cases, food shortage may be regarded as the underlying (ultimate) cause of breeding loss, and the other factors as the immediate (proximate) causes.

A third type of problem is that in some regions, a proportion of nest failures is due to direct human action, such as theft of eggs and chicks, or indirect human action, such as disturbance. Not only is the recorded nesting success then lower than it might otherwise have been, but it is sometimes hard to distinguish natural failures from human-induced ones. In any study, a full assessment of the causes of failures is always difficult, and, inevitably, some remain unexplained.

Influence of Food on Breeding Rates

As indicated above, much of the natural variation in the breeding rate of a species can often be related to variation in food supplies. The main evidence is circumstantial: (1) annual fluctuations in breeding rates are associated with annual fluctuations in prey numbers, while stable breeding rates are associated with stable prey numbers; (2) area differences in breeding rates are associated with area differences in prey numbers; and (3) sudden and long-term changes in breeding rates follow sudden and long-term changes in prey numbers.

Annual Differences

Annual differences in breeding rates are most marked among species that have restricted diets based on cyclic prey. Two main cycles are recognized: (1) an approximately 4-yr cycle of small rodents on the northern tundras and temperate grasslands, and (2) an approximately 10-yr cycle of snowshoe hares (*Lepus americanus*) in the boreal regions of North America, or of black-tailed jackrabbits (*Lepus californicus*) in the arid regions of western North America. Various game birds also cycle, but whereas in some regions they follow the 4-yr rodent cycle, in others they follow the 10-yr hare cycle. These various prey species influence the breeding of several raptors, but, as expected, they have their biggest impact on the species that most depend on them (Table 14.1). In extreme cases, such as the rough-legged hawk (buzzard) (*Buteo lagopus*) in Norway, reproduction has varied from 0.3 young per pair in years of rodent "troughs" to 2.8 in years of rodent peaks, a ninefold difference (Hagen, 1969).

Several of the species mentioned in Table 14.1 show stabler breeding rates in areas where their food supply is stabler (often through being more varied). This is evident from comparisons of goshawks (*Accipiter gentilis*) in northern and temperate Europe, of buzzards (*Buteo buteo*) in Germany and southern England, and of European kestrels (*Falco tinnunculus*) in northern and southern Britain.

In each case, the breeding rate fluctuates more from year to year in regions where prey is restricted and cyclic than where it is varied and relatively stable. This provides further circumstantial evidence for an overriding influence of food on the breeding rates of such species. Moreover, the whole range of variation in diurnal raptors is paralleled in owls, with the breeding output of several species fluctuating in response to rodent cycles, and that of the great horned owl (*Bubo virginianus*) in the boreal zone in response to hare cycles (Lockie, 1955; Keith, 1963; Southern, 1970; Lundberg, 1981; Village, 1981; Korpimaki, 1985). Stability in a breeding rate is usually associated with a wide food spectrum, presumably because the chances of total food supply changing violently are lessened the greater the range of prey species taken. However, even species whose breeding rate is normally fairly stable from year to year may have occasional bad years, associated with obvious food shortage or with adverse weather (see Ogden, 1975, for osprey [*Pandion halieatus*]).

Table 14.1
Variations in breeding output of raptors that are subject to fluctuating food supplies

Species that eat rodents	
Rough-legged hawk (Buzzard) (*Buteo lagopus*)	0.3-2.8 young reared per pair per year during 8 years, north Norway (Hagen, 1969)[a]
Common buzzard (*Buteo buteo*)	0.6-1.1 young reared per pair per year during 10 years, Germany (Wendland, 1952-53)
	1.4-2.1 young reared per nest per year during 6 years, Germany (Mebs, 1964)[a]
	0.4-2.1 young reared per nest per year during 12 years, Germany (Rockenbauch, 1975)[a]
Hen harrier (*Circus cyaneus*)	1.2-3.0 young reared per pair per year during 7 years, north Norway (Hagen, 1969)[a]
	1.6-3.3 young reared per nest per year during 10 years, Wisconsin (Hamerstrom, 1969)[a]
European kestrel (*Falco tinnunculus*)	1.4-3.7 young reared per pair per year during 6 years, north Norway (Hagen, 1969)[a]
	0.8-3.7 young reared per pair per year during 3 years, south Scotland (Village, 1980)[a]
	0 (small sample)-4.1 young reared per pair per year during 8 years, western Finland (Korpimaki, 1985)[a]
Species that eat game birds or hares	
Goshawk (*Accipiter gentilis*)	0.5-3.2 young reared per nest per year during 7 years, Sweden (Höglund, 1964)
	1.8-2.5 young reared per nest per year during 3 years, Alaska (McGowan, 1975)[a]
Gyrfalcon (*Falco rusticolus*)	0.9-2.9 young reared per nest per year during 5 years, Alaska (Swartz et al., 1974)[a]
Golden eagle (*Aquila chrysaetos*)	0.3-1.1 young reared per pair per year during 7 years, Utah (Murphy, 1974)[a]
Ferruginous hawk (*Buteo regalis*)	1.0 young per pair in a poor food year, 1.9 in a good one, Utah/Idaho (Howard and Wolfe, 1976)[a]
	0.6-3.0 young per pair per year during 8 years, Utah (Woffinden and Murphy, 1977)[a]

[a]Prey population also assessed and related to raptor breeding success.

Area Differences

Species whose breeding rates are relatively stable from year to year sometimes show area differences that are linked with food. This applies, for example, to the black eagles (*Aquila verreauxi*) of the Matopas Hills in Rhodesia, whose diet consisted of 98% hyraxes (Gargett, 1977). In areas where hyraxes were abundant, the overall production was 0.6 young per pair per year, while in adjacent areas where hyraxes were scarce, the equivalent was 0.3 young. The difference was due chiefly to more of the pairs in the poor areas failing to lay eggs each year. Similar area differences related to food supply have been noted among sparrowhawks (*Accipiter nisus*) (Newton, 1976) and golden eagles (*Aquila chrysaetos*) in Scotland (Dennis et al., 1984).

Long-Term Changes

The best documented case of a long-term change is that of the common buzzard in Britain, following the almost complete elimination of its main prey species, the rabbit, by disease. In one Devon valley, rabbits were still plentiful in 1954 but had gone by summer 1955. In 1954, 21 pairs of buzzards produced 29 to 33 young, but in 1955, among 14 pairs remaining, only 1 laid eggs, and none raised young (Dare, 1961). By the next year, the buzzards had turned more effectively to other prey and breeding was better than in 1955, but still well below normal. Similar changes happened over much of Britain, and 20 years later, neither rabbit numbers nor buzzard breeding output had reached the levels known in some areas before disease. On the other hand, in Australia the average clutch size of the little eagle (*Hieraaetus morphnoides*), the wedge-tailed eagle (*Aquila rapax*), and the goshawk (*Accipiter fasciatus*) became greater after introduced rabbits spread into their range (Serventy and Whittell, 1951).

Experimental Evidence

Evidence for the influence of food on breeding is based not only on associations found in field studies, but also on experiments. When carcasses of pigeons and other birds were left at perching places near sparrowhawk nests in a poor habitat, the hawks ate them, and nonlaying and clutch desertions were eliminated, laying was earlier, clutches were larger, and survival of young was improved (Newton and Marquiss, 1981). In the European kestrel, provision of extra food to captive and wild birds led to earlier egg laying and larger clutches (Dijkstra et al., 1982; Dijkstra, 1988).

Generalizations and Species Differences

From the studies available, certain generalizations can be drawn. In good, as opposed to poor, food conditions, (1) more of the territorial pairs laid eggs; (2) more of the birds that laid eggs succeeded in hatching their young; (3) mean laying dates were earlier; (4) clutches and broods were larger; (5) nestling growth rates and fledging weights were greater; (6) parental care was better, including defense against predators; and (7) repeat laying after failure was more frequent. Not all species showed all of these trends, but in some species not all aspects were studied. Also, the extent of annual fluctuations in breeding rate depended largely on the degree of dependence on a particular cyclic

prey, with less extreme variation among populations that had alternative prey.

These observations highlight food shortage as a major cause of nonbreeding by territorial pairs. Before laying, the female raptor has to accumulate massive body reserves, mostly of fat, and this requires a substantial increase in energy intake. In large raptors, the extra food required for body reserves may be much greater than that needed for egg production, and females that cannot accumulate such reserves do not lay (Newton, 1979; Dijkstra, 1988; Village, 1989). Moreover, in most species, the female does not hunt at this stage, but relies on the male for all her food. In effect, therefore, it is the ability of the male to acquire enough extra food in early spring, often a time of seasonal shortage, that determines whether a pair can breed. The success of the male is influenced by his hunting prowess and by the availability of prey in his range.

In most studies, more pairs failed near the start of the breeding cycle than near the end. This may have been due partly to the increase in hunting time (day length) with advance of season, and partly to an increase in food supply, but perhaps mainly to the poorer birds failing at an early stage, leaving only the good birds for later. At the same time, the most frequent kinds of breeding failure differed among species. In various buteonine hawks, nonlaying differed widely in extent between areas and between years, as did clutch sizes; in the kestrel, nonlaying by territorial pairs was rare and brood sizes varied negligibly between years, but desertions varied greatly. Because all types of failure were more prevalent in poor food conditions, food shortage could often be regarded as the ultimate cause of failure, and the egg addling, egg desertion, predation, and nestling mortality as the proximate causes.

Influence of Weather on Breeding Rates

Weather has at least three types of effect on the breeding rates of raptors. Long-term weather patterns, such as rainfall in arid regions, may influence the abundance and availability of prey. Thus black eagles in Zimbabwe bred more prolifically in years following below-average rainfall than in years following above-average rainfall, when prey were better hidden by vegetation (Gargett, 1977). Second, adverse weather during the breeding season itself may delay egg laying and reduce breeding success by lowering prey availability and the hunting success of adults. Among peregrines (*Falco peregrinus*) in Scotland, the proportion of clutches that hatched each year was inversely correlated with rainfall during incubation (Mearns and Newton, 1988); among honey buzzards (*Pernis apivorus*) in Germany, overall productivity was similarly reduced by high rainfall and low temperatures (Kostrzewa, 1987). Third, some nesting attempts of raptors fail as a direct response to extremes of weather, and some species have proved especially vulnerable when heavy rain occurs around hatch. Most nestling deaths among sparrowhawks and hen harriers (*Circus cyaneus*) in Scotland occurred on wet days, and annual variations in nestling mortality were associated with annual variations in the number of rain days in the nestling phase (Balfour, 1957; Moss, 1979). Some of the deaths occurred when young were chilled or soaked. At another extreme, 8 of 25 deaths among golden eagle nestlings in Idaho were attributed to overheating. The golden eagle is not especially adapted to heat, and these young were in exposed nests that caught the sun at the hottest time of day (Beecham and Kochert, 1975).

Influence of Natural Predation

Raptors are not immune to natural predation, though for obvious reasons they suffer less than most other birds. Avian predators on eggs or chicks include various crows, owls, and other raptors; mammal predators include mainly climbing species, such as cats, martens, raccoons, baboons, and monkeys. Some predation is associated with food shortage, as discussed above, and some with other factors, particularly the accessibility of the nest site and the proximity and abundance of particular predators.

Comparing species, some hole nesters seem to suffer little predation of nest contents; raptors that use open tree nests or cliffs are intermediate; and those that nest on the ground suffer most losses, presumably because their nests are vulnerable to the greatest range of predator species. Among merlins (*Falco columbarius*) in northern England, 63% of ground nests produced young, compared with 94% of tree nests (Newton et al., 1978); among prairie falcons (*Falco mexicanus*) in Idaho, 53% of nests accessible to mammalian predators were successful, compared with 88% of secure nests (Ogden and Hornocker, 1977); and among ferruginous hawks (*Buteo regalis*) in Washington state, ground nests produced an average of 0.6 young each, whereas tree nests produced 2.2 (Fitzner et al., 1977).

Large owls of the genus *Bubo* are especially important predators of young raptors. The eagle owl (*Bubo bubo*) has been noted as taking the young and adults of almost all the European raptors, up to the size of (and including) the female goshawk. In North America, the related great horned owl is important not only as a predator, but also as a usurper of newly built nests, even of species as large as the bald eagle (*Haliaeetus leucocephalus*) (Broley, 1947). Such owls ended 5% of 619 eagle nesting attempts (18% of all failures) recorded in Florida, and at least twice took over nests after the eagles had laid. The displaced hosts usually built another nest, but did not breed that year. Interactions between great horned owls and red-tailed hawks (*Buteo jamaicensis*) have been noted by several observers, and usually the

Table. 14.2
Parasites associated with nestling deaths in raptors

Organism	*Species*	*Reference*
Protozoan		
Trichomonas gallinae	grey hawk (*Buteo nitidus*)	Stensrude (1965)
	golden eagle (*Aquila chrysaetos*)	Beecham and Kochert (1975)
	northern goshawk[a] (*Accipiter gentilis*)	Cooper and Petty (1988)
Tick		
Ornithodoras aquilae	prairie falcon (*Falco mexicanus*)	Webster (1944)
Ixodes arboricola	peregrine falcon[a] (*Falco peregrinus*)	Schilling et al. (1981)
Fly		
Eusimulium clarum	red-tailed hawk[a] (*Buteo jamaicensis*)	Fitch et al. (1946)
Ornithomyia lagopodis	peregrine falcon (*Falco peregrinus*)	Ratcliffe (1980)
Passeromyia heterochaeta	black eagle (*Aquila verreauxi*)	Gargett (1975)

[a]Killed more than 10% of young observed.

hawks failed when the two species nested close together (Craighead and Craighead, 1956; Orians and Kuhlman, 1956; Hagar, 1957; Houston, 1975). In another study, Swainson's hawks (*Buteo swainsoni*) nesting close to either great horned owls or crows bred significantly less successfully than did those nesting in tree clumps lacking these predators (Dunkle, 1977).

In the studies mentioned above, the effects of predators were especially obvious. In other studies, predators may have accounted for some unexplained losses, but were generally unimportant compared with other factors.

Influence of Parasites and Diseases

Parasites and diseases seem to be of relatively minor importance as a primary cause of death among nestling raptors. They are no doubt present in many populations, but probably act mainly as contributory killers of young already weakened by starvation. Occasional mortality has been attributed to various bloodsucking ticks and flies and to trichomoniasis (frounce) caused by a flagellate protozoan parasite (Table 14.2). One of the most striking instances occurred among young peregrines in Germany infested with the tick *Ixodes arboricola* (Schilling et al., 1981). Out of 40 broods, 7 were infested, all of them in cave sites; broods on ledges were not affected. Up to 320 ticks were found on each chick, and 74% of infested chicks died. The few studies in which disease has been noted should be set against the dozens in which it has not. Disease may be underestimated, however, because freshly dead young are often eaten by their nest mates or parents, and so do not remain for long as evidence.

Some Other Causes of Breeding Failure

In addition to the causes discussed, some complete failures occur through accidents, such as the collapse of nest trees or the flooding or trampling of ground nests. Ospreys suffer more than most species from nests blowing down, partly because they often build on exposed dead treetops. In one study, 7% of 203 nests were destroyed in this way, and in another, nestling losses were reduced from 28% to 7% by the provision of stable platforms (Dunstan, 1968; Postupalsky and Stackpole, 1974).

Comparisons between Species

A selection of detailed results on nest failures is given in Table 14.3. Even allowing for unexplained failures, the main proximate causes differed widely among species. Egg and chick predation emerged as the main cause of loss in red-footed falcons (*Falco vespertinus*), which nested in rook (*Corvus frugilegus*) colonies; clutch desertion was the main cause of loss in buzzards, kestrels, and sparrowhawks; interactions with great horned owls were important in bald eagles. Egg addling was important in several species, but it was not recorded what proportion of the unhatched eggs were fertile.

Compared with the amount of egg addling in some other birds, the amount among raptors is often high. In part this

Table 14.3
Main causes of egg and chick losses in various raptor species

Species (sample size)	*Causes of failure (%)*	*Reference*
Kestrel, European	egg desertion (72)	Village (1989)
(*Falco tinnunculus*)		
(38% of 431 clutches)		
Sparrowhawk	egg desertion (31)	Newton (1986)
(*Accipiter nisus*)	egg breakage (18)	
(33% of 1,176 clutches)	chick starvation (7)	
Buzzard	egg desertion (32)	Tubbs (1971)
(*Buteo buteo*)	egg breakage (21)	
(23% of 645 clutches)	chick starvation (7)	
Bald eagle	adverse weather (45)	Broley (1947)
(*Haliaeetus leucocephalus*)	interaction with owl (31)	
(28% of 619 clutches)	egg addling (25)	
Red kite	egg addling (15)	Davies and Davis (1973)
(*Milvus milvus*)	human robbing (20)	
(59% of 231 clutches)	disturbance (6)	
Prairie falcon	egg addling (34)	Ogden and Hornocker (1977)
(*Falco mexicanus*)	egg predation (19)	
(39% of 302 eggs)	egg breakage (9)	
Red-footed falcon	egg predation (17)	Horvath (1955)
(*Falco vespertinus*)	chick predation (23)	
(54% of 105 eggs)	egg addling (14)	

addling is linked with food conditions, as mentioned, and perhaps with the nutritional state of the female. In buzzards, addled eggs formed 5% of all incubated eggs in good food years and 15% in poor ones (Mebs, 1964). In sparrowhawks, addled eggs were more frequent in poor than in good food areas, in late than in early clutches, and in repeat clutches than in firsts (Owen, 1926; Newton, 1976). In black kites (*Milvus migrans*), addled eggs were more frequent in small clutches than in large ones (Fiuczynski and Wendland, 1968). Other estimates of the proportions of incubated eggs that were addled (or infertile) included 7% in kestrel (Cavé, 1968), 9% in hobby (*Falco subbuteo*) (Fiuczynski, 1978), 10% in goshawk (McGowan, 1975), 11% in prairie falcon (Ogden and Hornocker, 1977), 12% in red-footed falcon (Horvath, 1955), 15% in sparrowhawk (Newton, 1986), 10% to 27% in different populations of Eleonora's falcon (*Falco eleonarae*) (Vaughan, 1961), and about 33% in hen harrier (Balfour, 1957). In most of these species, egg addling seldom affected the whole clutch, so was mainly a cause of partial loss.

Compared with other birds, raptors also have a high nestling mortality, again partly associated with food shortage. The evidence comes from comparisons of different years and different areas, and from comparisons of broods of different sizes. In buzzards, broods of two suffered 29% mortality in good food years, and 40% in poor ones (Schmaus, 1938), and in another part of Germany the corresponding figures for broods of three were 26% and 42% (Mebs, 1964). Other estimates of nestling mortality (mainly starvation) include the following, in order of frequency: 3% in hobby (Fiuczynski, 1978), 6% in buzzard (Picozzi and Weir, 1974), 14% in Galapagos hawk (*Buteo galapagoensis*) (de Vries, 1975), 13% and 17% in kestrel (Cavé, 1968; Village, 1989), 17% in sparrowhawk (Newton, 1986), 17% in broad-winged hawk (*Buteo platypterus*) (Rusch and Doerr, 1972), 17% in prairie falcon (Ogden and Hornocker, 1977), 32% in golden eagle (Beecham and Kochert, 1975), 27% and 38% in red-tailed hawk (Luttich et al., 1971; Fitch et al., 1946). Some of this mortality affected whole broods, but mostly it affected part broods. In certain eagle species that usually lay two eggs per clutch, one young kills the other (Gargett, 1970; Meyburg, 1974; Newton, 1979), so a minimum of 50% chick mortality is normal.

Effects of Organochlorine Pesticides

The previous examples were drawn from populations not known to have been affected seriously by organochlorine pesticides. In recent decades, other populations have suffered severe reductions in breeding rate as a result of contamination with DDE, the main metabolite of the insecticide DDT. This persistent, fat-soluble chemical accumulates in the birds from their food supply and causes shell thinning and egg breakage, thus reducing the breeding rate. Bird-eating and fish-eating species are the most vulnerable. The mechanism is incompletely known, but seems to involve interference with the enzymes carbonic-anhydrase and calcium adenosine triphosphatase (ATP-ase), which are responsible for the transport of relevant ions into the shell matrix during eggshell formation (Peakall, 1970). The re-

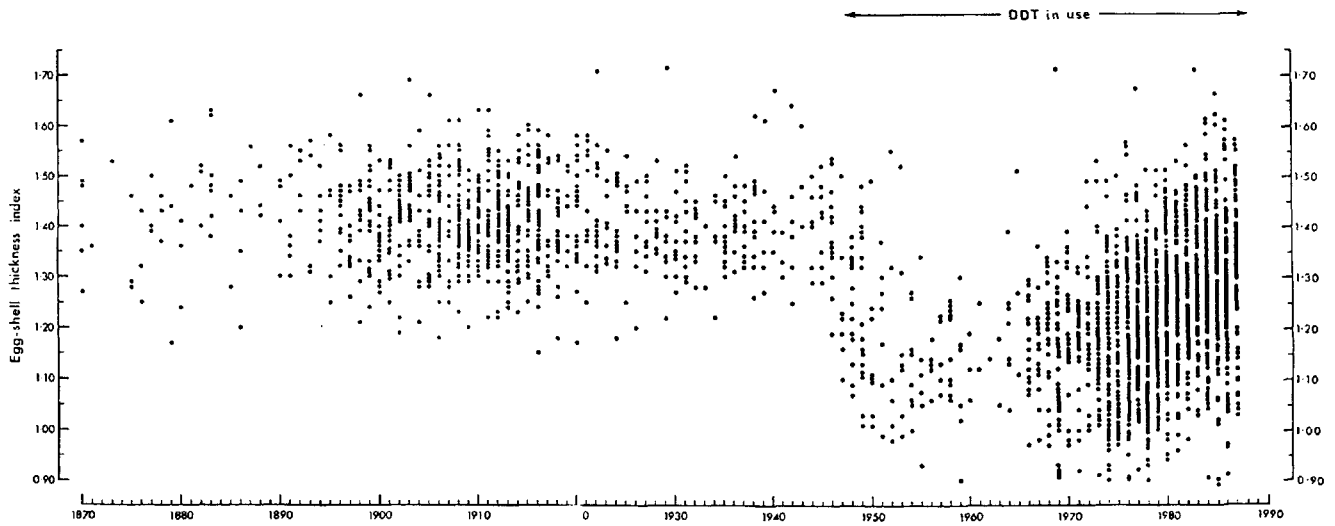

Figure 14.1. Eggshell thickness index of British sparrowhawks, 1870-1987. The shell index is measured as shell weight (mg)/shell length × breadth (mm) after Ratcliffe (1967). Each spot represents the mean shell thickness index of a clutch, and more than 2,500 clutches are represented from all regions of Britain. Shells became abruptly thinner after 1947, coincident with the widespread introduction of DDT in agriculture, and improved in recent years, following restrictions in DDT use. (Updated from Newton, 1986; reprinted with permission.)

sulting thin shells have a weak structure and break either on laying or later during incubation. Other thin shells may survive, but alter the normal rate of water loss, which sometimes also kills the embryo.

The field evidence for the involvement in DDE in shell thinning is of two kinds: (1) shell thinning has been restricted to the period of extensive DDT use, since the late 1940s, and has lessened as DDT has been withdrawn (Fig. 14.1); and (2) among individual eggs the shell thinning is inversely correlated with the DDE content (reflecting the level in the laying female) (Fig. 14.2). Such relationships between shell thickness and DDE have now been found in a wide range of species, including the peregrine falcon (Ratcliffe, 1970; Peakall et al., 1975; Newton et al., 1989), American kestrel (*Falco sparverius*) (Lincer, 1975), merlin (Hodson, 1975; Fox, 1979; Newton et al., 1982), and sparrowhawk (Newton, 1986). In each species, the relationship between shell thinning and DDE content is linear when DDE is plotted on a log scale; that is, it is similar to the dose-response relationship for many drugs. There is also much experimental evidence from captive birds, confirming that DDT-type compounds administered in the diet will cause shell thinning (Porter and Wiemeyer, 1969; McLane and Hall, 1972; Lincer, 1975).

Apparently, only chemicals of the DDT group have been widely found to cause shell thinning. Relationships between shell thinning and other chemicals have been found in wild eggs, but only when the levels of these other chemicals were in turn correlated with those of DDE.

Evidence from wild and captive birds has shown that different taxonomic groups of birds vary in their sensitivity to DDE. Thus, some 4 to 5 ppm of DDE in eggs is associated with about 15% shell thinning in some raptors, about 10% in herons, and less than 1% in gallinaceous birds and songbirds (Newton, 1979). Hence, raptors are especially vulnerable to DDE, first, because their feeding habits predispose them to accumulate large amounts, and second, because they are unusually sensitive to a given amount.

In some raptor populations, DDE contamination has assumed a major role in breeding failure. At its peak period of use, DDT may have reduced the success of some populations almost to nil, and even in recent years variations in production between populations of certain species have been correlated with the DDE contents of their eggs (Fig. 14.2).

Bird-eating raptors, such as the peregrine and sparrowhawk, were more influenced by DDE than were species that eat other prey, such as mammals. This is at least partly because birds are less able to metabolize organochlorine pesticides than are mammals, as indicated by lower activity levels of mono-oxygenase enzymes in the liver (Walker et al., 1987). Hence, birds in general accumulate higher levels of DDE in their bodies than do mammals, so predators of birds are correspondingly more vulnerable than are predators of mammals. Fish eaters are also vulnerable, partly because fish can accumulate large concentrations of DDE from water during respiration, as well as from their food. Oily fish species contain the biggest concentrations of DDE, especially in contaminated estuaries.

Other organochlorine pesticides, notably aldrin and dieldrin, have reduced the breeding rates of gallinaceous birds in captivity (Brown et al., 1965; Neill et al., 1971). Similar effects have not been demonstrated on wild raptors, though such toxic organochlorines are known to cause mor-

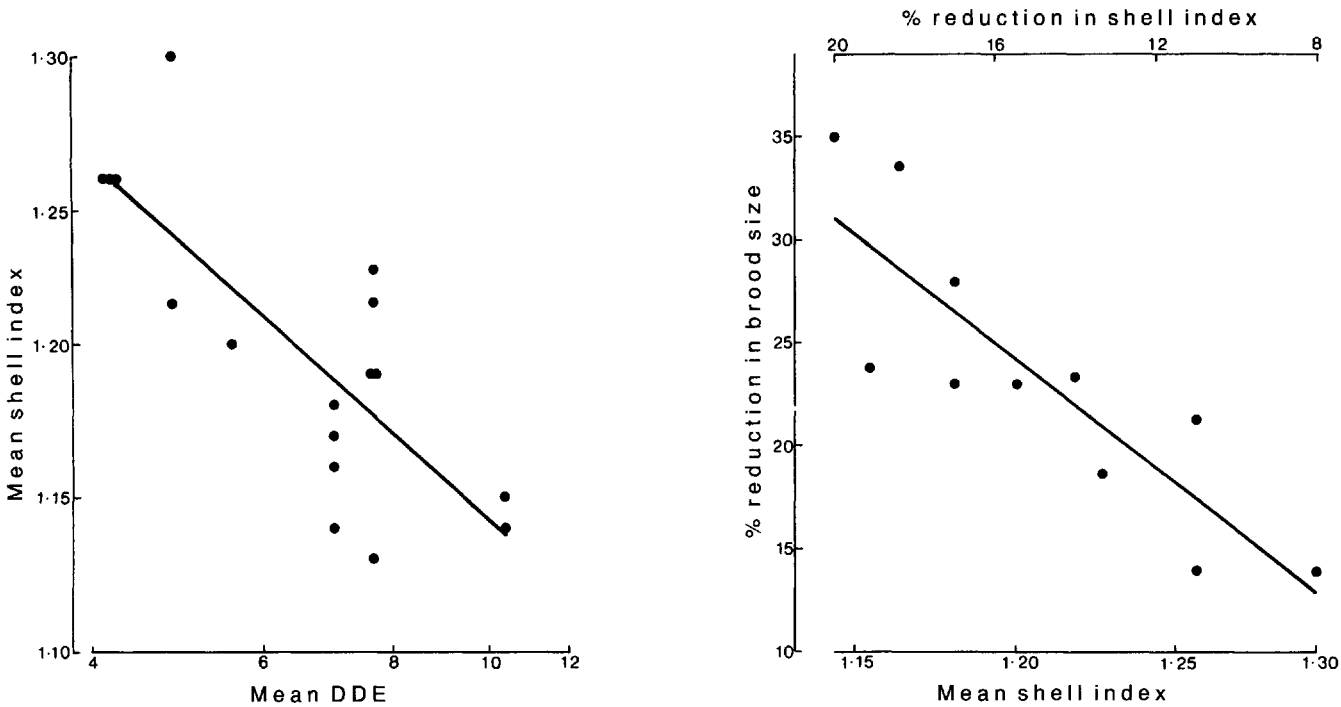

Figure 14.2. Relationships among DDE level, shell index, and breeding success of sparrowhawks. *Left*: Mean shell thickness index in relation to mean DDE level in the contents of eggs from different areas. DDE on log scale ($r = 0.78$, $p < 0.001$). *Right*: Percentage reduction in production of young in relation to mean shell thickness index in different areas. The poorer the shells, the greater the loss of productivity ($r = 0.85$, $p < 0.001$). (From Newton, 1986; reprinted with permission.)

tality of adult raptors, thus contributing to population decline (Newton, 1979, 1986). Various mercury compounds have been found to reduce the breeding rates of nonraptor species in captivity (Borg et al., 1969; Fimreite and Karstad, 1971), and have been implicated in reduced breeding success (Newton and Haas, 1988), increased mortality, and population decline of raptors (Borg et al., 1969). The mechanism of their effects on breeding is not understood, but it does not appear to involve shell thinning.

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CHAPTER 15

Immune and Inflammatory Responses in Falcon Staphylococcal Pododermatitis

J. LINDSAY OAKS, Jr., D.V.M.

Dubai Falcon Hospital
Dubai, United Arab Emirates

ABSTRACT: Falcon staphylococcal pododermatitis remains an important medical problem in captive falcons. Vaccination and immunomodulation have been used in the treatment of this disease, but clinical reports of effectiveness are inconclusive. There are limited data evaluating the immunological relationship between falcons and *Staphylococcus*. The purpose of this study was to test the hypothesis that insufficient or inappropriate host immune responses are involved in the pathogenesis.

Evaluated in the study were 31 clinical cases of naturally occurring staphylococcal pododermatitis in birds admitted for surgical treatment. Birds were clinically graded based on severity, and divided into treatment groups based on vaccination and immunomodulation. Systemic leukocyte counts, serologic responses, *in vitro* phagocytosis assays, and cytologic and histopathologic slides were evaluated. Hematology provided useful clinical management and prognostic information. Vaccination, with or without immunomodulation, had no clinical, hematologic, or serologic effects on the falcons in this study. Heterophils were the predominant inflammatory cell in this disease, and were capable of efficient *in vitro* phagocytosis. No specific immunological defect could be identified in this study, but the hypothesis warrants further investigation.

KEY WORDS: bumblefoot, falcon, pododermatitis, raptor.

Pododermatitis (bumblefoot) is a common disease of the large species of falcons maintained in captivity, often resulting in a chronic, progressive, invasive, disabling condition that is clinically resistant to medical and surgical therapy, and is frequently recurrent. Etiology and pathogenesis are unclear, but trauma and devitalization of the weight-bearing plantar structures of the foot with secondary bacterial infections are generally suggested to initiate the disease. *Staphylococcus aureus* is the most common pathogen isolated from affected feet (Halliwell, 1975; Cooper, 1978b; Riddle, 1980; Satterfield and O'Rourke, 1980; Remple and Remple, 1987).

Staphylococcus aureus is a complex organism with many antigenic determinants, endotoxins, and exotoxins that may serve as potential virulence factors (Anderson, 1986; Gerlach, 1986; Morse, 1980). *Staphylococcus* spp. in humans, mammals, and birds are responsible for a variety of superficial and invasive infections. This organism is generally considered an opportunist, and invasive infection is usually associated with a breakdown of host defense systems (Anderson, 1986; Gerlach, 1986; Morse, 1980; Mudd, 1970; Muller, 1983).

Insufficient or inappropriate host immune responses have been proposed as a factor in falcon staphylococcal pododermatitis. Satterfield and O'Rourke (1980) state that "avian staphylococcal disease is typified by a failure of the immune system to clear the antigen and its pathogenic products from the tissue, and failure to produce protective memory cells." The pathomechanism(s) for immunosuppression in avian species is poorly understood, but a potential role for it in falcon pododermatitis is an attractive hypothesis. Circumstantial evidence from our own practice suggests that generalized immunosuppression may occur in falcons, as diseases such as aspergillosis and chronic or heavy parasite burdens are frequently observed. Factors often associated with this disease include improper perching surfaces, trauma and puncture wounds to the feet, and malnutrition (Cooper, 1978b; Riddle, 1980; Harrison, 1986; Remple and Remple, 1987), all of which may compromise the first line of host defense against bacterial invasion: the intact skin. However, foot trauma in other species such as psittacines and turkeys may not result in the invasive infection often seen in falcons (Martland, 1984; Harrison, 1986), suggesting more generalized abnormalities in the falcons. Other potential environmental stressors encountered in this practice include ambient environmental temperatures up to 45°C and the maintenance of wild-caught falcons in captivity (psychological stress).

Based on this hypothesis, vaccination against *S. aureus* and immunomodulation with levamisole and Bacillus-Calmette-Guerin (BCG) have been suggested to be beneficial in the treatment of falcon staphylococcal pododermatitis. However, previous trials have been small and uncontrolled, and results have been contradictory (Cooper, 1978a; Satterfield and O'Rourke, 1980, 1981).

The interaction between *Staphylococcus* and the avian immune system leading to chronic, progressive, clinically unresponsive, and recurrent disease is complex and poorly

understood. There is only limited information available describing the local and systemic immune responses or responses to vaccination and immunomodulation in naturally occurring falcon pododermatitis, or the inherent resistance of uninfected falcons to *Staphylococcus*. This information is fundamental to understanding the pathogenesis and clinical management of this disease.

Cellular and humoral responses are important components of the avian immune system (Kollias, 1986). In mammals, the primary immunologic mechanism for the removal of *Staphylococcus* from tissues is the opsonic/phagocytic system. The heterophil leukocyte is an important cellular phagocytic element of the avian immune defense against bacterial infections (Kollias, 1986). The heterophil has been shown to be the predominant inflammatory cell in experimental acute inflammatory responses in chickens (Carlson and Allen, 1969) and in experimental staphylococcal pododermatitis in starlings and pigeons (Cooper, 1987). Lymphocytes and macrophages are also characteristic findings in avian inflammation, including pododermatitis (Carlson and Allen, 1969; Cooper, 1987; Satterfield and O'Rourke, 1980). Birds will respond to bacterial infections with a leukocytosis and heterophilia; the blood counts return to normal with appropriate therapy, and so the peripheral leukocyte count may be used as an indicator of bacterial infection and as a monitor of response to therapy (Dein, 1986).

Falcon staphylococcal pododermatitis, like any chronic disease, manifests different clinical and pathological stages in its development. In order to assess clinical, pathological, and physiological changes, a system for describing the stages of this disease is necessary. We have found that previous classification schemes (Cooper, 1978b; Halliwell, 1975) have been insufficient to describe the pododermatitis syndrome seen in our own practice. J. David Remple and I have developed a new classification system, which is used in this study (see Remple, chapter 27, this volume).

In this study, 31 cases of naturally occurring staphylococcal pododermatitis in saker (*Falco cherrug*) and peregrine (*Falco peregrinus*) falcons admitted for treatment were evaluated. These birds were clinically graded for severity and divided into three treatment groups based on vaccination and immunomodulation. Cellular, humoral, and local inflammatory responses of these birds during the course of treatment were evaluated. An attempt was made to correlate these responses to clinical severity, therapeutic and prognostic schemes, and incompetent immunological responses. The objectives of the studies were to provide useful clinical information for practitioners and to report some basic data on the immune and inflammatory response of falcons to *Staphylococcus* sp.

Materials and Methods

Clinical Grading

Remple and I have developed a five-part classification scheme for the pododermatitis syndrome seen in our practice. We feel that this grading system represents degrees of involvement of one disease, generally represents a chronological sequence of events, and has relevance to therapeutic and prognostic considerations (Remple, chapter 27, this volume).

Class I. Early devitalization of a prominent plantar area without disruption of the epithelial barrier; there are two subclasses:

A. hyperemia (bruise) or early ischemia (a blanched area with compromised capillary perfusion)
B. hyperkeratotic reaction (an early callus)

Class I carries the most favorable prognosis, as infection is not evident. These changes generally respond to conservative therapy, including changes in perching surfaces and application of topical emollients.[a]

Class II. Localized inflammation/infection of underlying tissues in direct contact with the devitalized area, with no gross swelling; there are two subclasses:

A. puncture wound
B. ischemic necrosis of epithelium (a penetrating callus or scab)

Class II carries a good prognosis, as infection is localized. These lesions respond to surgery with good results, as the infection is easily excised, epidermal defects are usually small, and the architecture of the plantar weight-bearing structures are intact. This class, although relatively superficial, generally will not respond to conservative therapy.

Class III. More generalized infection with gross inflammatory swelling of underlying tissues. The origin may be puncture wounds or ischemic necrosis, however, the initial insult becomes less significant as the predominant pathology is related to infection. This class may be further categorized in three ways based on the gross inflammatory response, which by this stage may be mixed (i.e., chronic/active inflammation):

A. serous (acute)—edema and hyperemia of tissues
B. fibrotic (chronic)—attempt at encapsulation and confinement
C. caseous—accumulation of necrotic debris

Class III carries a good to guarded prognosis, as infection has become established, and some structural changes of the foot have begun. Occasionally the epidermal defects are large enough that considerations of surgical technique, such as tension on the suture line, become significant.

Class IV. Established infection with gross swelling and involvement of deeper vital structures. Surgical exploration and radiology may be needed to differentiate Class III and Class IV. The latter is chronic, producing tenosynovitis and occasionally arthritis or osteomyelitis. When synovial structures are involved, the inflammatory response is often combined with a purulent synovial exudate. Class IV is a chronic extension of Class III with two subclasses—fibrotic and caseous.

Class IV carries a guarded to poor prognosis, as infection involves deeper vital structures, making surgical debridement difficult. Additionally, the chronic nature also leads to pockets of infected tissue that are insulated by fibrosis from host immune responses and antimicrobial drugs. Usually major changes of plantar architecture have occurred, predisposing the epithelium to poor healing and recurrent trauma. Frequently the surgical procedure is difficult, as epithelial defects are large and skin quality is poor. Occasionally, digit amputation is necessary to obtain skin grafts to close defects.

Class V. Extension of Class IV resulting in crippling deformities and loss of function.

Class V carries a grave prognosis, and birds in this class are generally euthanized without treatment.

Clinical Management

A total of 31 cases of naturally occurring pododermatitis of differing severity in 22 saker and 9 peregrine falcons were treated and evaluated for clinical response. These 31 birds had 58 affected feet, which represented all clinical classes; 27 birds had bilateral disease. The 58 feet included 3 in Class I, 12 in II, 13 in III, 28 in IV, and 2 in V (Table 15.1). None of these birds had Class I disease alone.

All falcons were given full physical examinations upon presentation in order to identify any concurrent medical problems. Craniocaudal and lateral-view radiographs of affected feet were taken to identify osteoarthritic involvement and to aid in clinical grading. Surgical therapy was indicated for falcons with Class II, III, IV, or V disease. Class I feet were treated conservatively.

Surgery was performed on 55 affected feet to excise devitalized skin and infected tissue, and to convert a chronic inflammatory process to an acute process capable of primary healing. The details of the anesthetic and surgical technique have been previously described (Remple and Remple, 1987; Riddle, 1980). Postoperatively, the feet were cast by Remple's technique (Remple and Remple, 1987) to prevent contamination and to remove pressure from the incision, thus encouraging healing.

During surgery, bacterial cultures were taken for aerobic bacterial isolation and identification. Based on sensitivity patterns, antimicrobial therapy was instituted and then discontinued when it was judged that resolution of infection had occurred. This assessment was based on reduction of swelling, healing of the incision, and a return to normal of the leukocyte count. Antimicrobials used included piperacillin[b] at 150 mg/kg body weight IM BID alone or in combination with amikacin[c] at 10 mg/kg body weight IM BID. Weekly injections of a multivitamin preparation[d] dosed to provide 30,000 IU vitamin A/kg body weight were given.

The cast was removed 14 to 16 days post-op, to examine the incision. If healing was apparent, half the sutures were removed and the foot rebandaged. The incision was reexamined 7 days later; if continued healing was apparent, then the remaining sutures were removed and the foot rebandaged. Finally, 5 to 7 days later, the foot was again examined. If the foot was healed, the falcon was returned to its owner. In the event of dehiscence at any stage, the entire process was repeated. If birds failed to respond after two or three surgical procedures, they were considered failures with a grave prognosis and were euthanized.

Immunomodulation Treatment Groups

The 31 falcons, with different classes of clinical severity, were randomly assigned to one of the three following treatment groups. Class III and IV feet/birds predominated in all groups, as this was the most common clinical presentation, although some Class I and II feet were also represented in each treatment group (Table 15.1).

Group 1: Control. The 15 birds in this group were treated as described in the above section on clinical management.

Group 2: Vaccination. The 7 birds in this group were vaccinated every 7 days, beginning several days after surgery and continuing through the treatment period. The vaccine used was a commercial *Staphylococcus aureus* bacterin containing lysed cultures of phage types I, II, III, and IV, and miscellaneous *S. aureus* groups.[e] Administration protocol was 0.05 ml vaccine intradermally for 1 to 2 wk, followed by 0.05 ml intradermally and 0.05 ml subcutaneously for another 2 to 3 wk. The vaccination site was the skin on the cranial aspect of the leg just proximal to the tibiotarsotarsometatarsal joint.

Group 3: Vaccination and immunomodulation. The 9 birds in this group received immunomodulation therapy with levamisole and BCG[f] in addition to vaccination. Falcons were vaccinated weekly as in Group 2, except that to each dose of vaccine 0.05 ml of BCG was added as adjuvant. These birds also received 2 mg/kg body weight levamisole[g] IM the day before vaccination.

Table 15.1
Summary: clinical cases, clinical management, and bacteriology

Treatment group	Case	Species[a]	Class		Bacterial isolate(s)		Clinical results, comments
			RT foot	LT foot	RT foot	LT foot	
Group 1, control	1	S	II	II	*S. aureus*	*S. aureus*	initial healing[b]
	2	S	II	II	*Staph.* sp.	N/G[c]	initial healing
	3	S		II		*S. aureus*	initial healing
	4	S	III		*S. aureus*		initial healing
	5	S	II	III	*S. aureus*	*S. aureus*	initial healing
	6	S	III	II	*S. aureus*, *Strep.*	*S. aureus*, *Strep.*	initial healing
	30	S	III	III	*S. aureus*	*S. aureus*	initial healing
	31	P	IV	IV	*S. aureus*	*S. aureus*	initial healing
	7	S	IV	II	*Strep.*	*Staph.* sp.	initial healing
	8	S	II	IV	*E. coli*	*S. aureus*	initial healing
	9	P	IV	III	*Staph.* sp.	N/G	initial healing
	10	P	IV	IV	*S. aureus*	N/G	initial healing
	11	S	IV	IVp[d]	*E. coli*	*S. aureus*	healing—2 surgeries
	12	S	II	IVp	*S. aureus*	*S. aureus*, *E. coli*	euthanized, falcon pox
	13	S	Vp	Vp	*S. aureus*	*S. aureus*, *Strep.* sp.	euthanized
Group 2, vaccination	14	S	I	III		*S. aureus*	initial healing
	15	P	III		*S. aureus*		initial healing
	16	S	IV	IV	*S. aureus*	*S. aureus*	initial healing
	17	S	IV	II	N/G	*S. aureus*	initial healing
	18	P	I	IV		*S. aureus*	initial healing
	19	S	IV	II	*S. aureus*	*Staph.* sp.	initial healing
	20	P	IV	IV	*S. aureus*, *E. coli*	*E. coli*	healing—2 surgeries
Group 3, vaccination and immunomodulation	21	S	III	III	N/G	*S. aureus*	initial healing
	22	S	III	III	N/G	*Staph.* sp.	initial healing
	23	S	IV	IV	*Staph.* sp.	*Staph.* sp.	initial healing
	24	S	IV		*Staph.* sp.		initial healing
	25	S	IV	IV	*S. aureus*	*S. aureus*	initial healing
	26	P	IV	I	*S. aureus*		initial healing
	27	S	IV	IV	*Staph.* sp.	*Staph.* sp.	initial healing
	28	P	IV	III	*S. aureus*	*Staph.* sp.	initial healing, aspergillosis
	29	P	IV	IV	*S. aureus*	*S. aureus*	initial healing

[a]S = saker, P = peregrine.
[b]Healed after one surgery.
[c]N/G = no growth.
[d]p = problem foot.

Bacteriology

Standard bacteriologic techniques were used to isolate and identify aerobic bacteria. *Staphylococcus* was defined as a catalase positive, Gram-positive coccus. *S. aureus* identification was based on a protein receptor capable of combining with fibrinogen.[h]

Hematology

Complete blood counts were performed on 27 of the 31 falcons at weekly intervals, beginning before treatment and continuing throughout the treatment period. Leukocyte responses were monitored for 12 birds from Group 1; 3 birds (Cases 30, 31, and 10) from this group were omitted from this section due to incomplete data. In Group 1, 3 Class II, 3 Class III, 5 Class IV, and 1 Class V birds were represented (assignment based on the worse foot when birds had bilateral disease). From Group 2, 7 birds were monitored: 2 with Class III and 5 with Class IV disease. From Group 3, 8 birds were monitored; 1 bird (Case 28) was omitted from this group because of concurrent aspergillosis. In this group there were 2 birds with Class III and 6 with Class IV disease (Table 15.2).

For each test, 1 ml of heparinized blood was collected from birds fasted for at least 12 hr to avoid lipemia. Heterophil enumerations were performed using a commercial phloxine-B system for manual determinations of mammalian eosinophil numbers,[i] and were counted on a hemacy-

Table 15.2
Total leukocyte counts × 1,000 by week

Treatment group	Case	Class[a]	Week 0	1	2	3	4	5	6
Group 1, control	2	II	10.6	14.4	18.7	18.8			
	1	II	12.9	16.1	28.3	11.6	8.2	12.5	8.9
	3	II	8.7	14.4	11.3	10.0	16.9		
	4	III	16.6	17.3	18.1	14.3			
	5	III	23.8	16.0	15.2	12.0			
	6	III	15.7	22.3	19.1	19.0	14.8		
	7	IV	18.9	25.8	36.8	20.6	10.1	10.8	
	8	IV	12.6	10.6	11.2	8.0	17.9		
	9	IV	9.2	8.6	5.9	11.8			
	12	IV	22.4	24.2	18.0	20.3			
	11	IV	13.6	38.4		26.0	11.2	16.4	11.2
	13	V	58.0	24.2	11.2	15.6	21.3	9.4	
Group 2, vaccination	14	III	15.1	11.1	8.3	13.4	12.8	8.7	
	15	III	17.7	17.1	12.4	24.2	10.0	13.6	16.2
	16	IV	19.2	10.6	15.1	10.2	8.3	11.1	
	17	IV	10.9	9.0	8.1	8.8			
	20	IV	22.9	22.7	28.2	25.3	17.1	28.9	14.7
	18	IV	12.6	18.2	24.6	12.7	12.0	10.9	
	19	IV	11.0	14.0	13.7	11.9	25.2	15.9	
Group 3, vaccination and immunomodulation	21	III	9.5	12.7	5.9	14.2	14.7	24.1	
	22	III	16.3	21.4	15.4	13.8	12.0	8.5	
	23	IV	14.8	14.8	8.0	14.6	10.8		
	24	IV	17.7	10.4	10.5	9.0	8.7		
	25	IV	19.3	8.3	14.9	12.8	13.3		
	26	IV	14.4	14.2	8.7	7.5	7.7	8.2	6.2
	27	IV	14.3	19.9	13.1	11.9	19.3	13.7	12.2
	29	IV	35.7	21.7	17.4	10.1			

[a]Based on worse foot.

tometer.[j] Differential counts were made on slides fixed in methanol and stained with a rapid Wright's type stain.[k] Total leukocyte counts were calculated routinely (Dein, 1986); absolute heterophil and mononuclear cell numbers were calculated from the differential slide percentages. Remaining blood was centrifuged; the plasma was recovered and frozen at minus 20°C until needed for further testing.

Reference values for leukocyte numbers were based on 35 clinically normal saker and peregrine falcons previously sampled in our practice using the above-described techniques.

The data obtained were tested statistically for significance using the Student's *t*-test with a significance level of p less than 0.05.

Complement Fixation Antibody Titers against S. aureus

A total of 78 plasma samples from 31 falcons were tested for the presence of complement-fixing (CF) antibodies against *Staphylococcus*. Of the 31, 6 falcons were normal controls with no clinical history of pododermatitis. The other 25 were clinical cases of staphylococcal pododermatitis from the study group. Clinical cases were titered at various times during treatment ranging from week zero (pretreatment) to week 13. The clinical cases included 9 birds from treatment Group 1, 7 from Group 2, and 9 from Group 3. Birds with Class II, III, and IV severity disease were represented (Table 15.3).

Plasma samples were thawed and diluted 1:10 in veronal buffer, pH 7.2. Diluted plasma was heat inactivated at 56°C for 30 min to remove endogenous complement. Sheep red blood cells (srbc) were washed three times in veronal buffer, and a 2% solution of srbc was made in veronal buffer. The hemolytic system was prepared by adding equal volumes of a 1:1000 solution of anti-srbc antibodies[l] in veronal buffer to the 2% srbc solution. Guinea pig complement[m] was used in a 4% concentration in veronal buffer. Antigen used was the polyvalent *S. aureus* bacterin used to vaccinate the falcons, diluted 1:10 in veronal buffer.

Using round-bottomed 96-well microtiter plates, 1:2 se-

Table 15.3
Complement-fixing (CF) and antihemolysin (AH) antibodies; reciprocal titers

Treatment group	Case	Class[a]	Toxigenic strains[b]	Week 0 CF AH	1 CF AH	2 CF AH	3 CF AH	4 CF AH	5 CF AH	6 CF AH	CF AH
Group 1, control	2	II			80 —[c]						
	3	II	h	80 —	80	80 —					
	4	III	h			80					
	6	III	h	80 —	160	40					
	30	III	h	80 20							
	31	IV	h	80 10							
	10	IV						80			80[d]
	8	IV	h			80	80 —				
	11	IV					80 —				80[e] —
Group 2, vaccination	14	III	h			80 —	40				
	15	III				80 —			80	80 —	
	16	IV				40 80	40	40 40			
	18	IV	h		40 —	40	40	40 10			
	19	IV				40 —	40 —				
	17	IV	h	40 —	80	80	80 —				
	20	IV	h		40 20	40		40		40 10	
Group 3, vaccination and immunomodulation	21	III			80 —	40	20	40 —			
	22	III	H		80 —	80	80	80 —			
	28	IV	h		80 10	80	80		80 10		
	25	IV			160 —	80	40 —				
	24	IV	H		80 —	40	80 —				
	23	IV	h			80	80 —				
	26	IV	h	40 10	40	20	40 —	40	80 —		
	27	IV		40 —	20	20	40 —	40	160 —		
	29	IV			160	160					
Normals	32			80 —							
	33			80 —							
	34			20 —							
	35			80 —							
	36			40 —							
	37			80 —							

[a]Based on worse foot.
[b]h = hemolytic strain of *Staphylococcus*.
[c]Negative.
[d]Week 13.
[e]Week 7.

rial dilutions of the 1:10 plasma samples were made in 0.05 ml volumes in veronal buffer. The last wells of each sample column were used as individual plasma controls, using 0.05 ml of 1:10 plasma. To all wells except the individual plasma controls, 0.05 ml of the 1:10 *S. aureus* antigen was added. To all wells, 0.05 ml of the 4% complement was added. Plates were then incubated at 35°C for 1 hr. After incubation, 0.1 ml of the hemolytic system was added to all wells and the plates were incubated at 35°C. Appropriate positive plasma, antigen, complement, and hemolytic system controls were also prepared. The test was interpreted after 1 hr; a positive result was considered to be the highest dilution showing significant inhibition of hemolysis. Positive plasma control was from a vaccinated falcon with clinical staphylococcal pododermatitis shown to be positive in preliminary assays.

Antibody Titers against Staphylococcal Hemolysins

A total of 45 plasma samples from 28 falcons were tested for the presence of antibodies against staphylococcal hemolytic toxin(s) at various times during the course of treatment, ranging from week zero (pretreatment) to week 13. Of the 28, 6 falcons were normal controls with no history of pododermatitis. The other 22 were clinical cases of staphylococcal pododermatitis from the study group, infected with

a mixture of hemolytic and nonhemolytic strains. The clinical cases included 7 birds from treatment Group 1, 7 from Group 2, and 8 from Group 3. Birds with Class II, III, and IV severity disease were represented (Table 15.3).

A double-zone hemolytic isolate of *S. aureus* from a pododermatitis lesion was incubated in nutrient broth for 12 days with a trace of magnesium sulfate, as the hemolytic reaction is magnesium dependent (Morse, 1980). The broth was centrifuged at 7,500 *g* for 15 min to sediment the bacteria. The supernatant containing the hemolytic toxin(s) (lysogenic solution) was recovered, and 10 mg piperacillin and 250 μg kanamycin^n per ml solution were added to inactivate any remaining viable bacteria. The solution was incubated overnight at room temperature and then plated onto blood agar to check sterility.

To determine the toxin concentration, 0.1 ml of lysogenic solution was added to the first well of a microtiter plate and 1:2 serial dilutions made in 0.05 ml volumes in 0.85% saline. To all wells, 0.05 ml of saline was added to compensate for the volumes of plasma used in the test in order to maintain dilutions. Then 0.05 ml of 5% srbc in saline was added to all wells. The plate was incubated for 12 hr at 35°C and then at room temperature for another 24 to 36 hr, as the hemolytic reaction is a "hot-cold" reaction (Morse, 1980). This protocol resulted in optimal hemolysis. One lysing unit was defined as the highest dilution of toxin in 0.05 ml causing significant hemolysis. Stock solution was then diluted appropriately to provide one to two lysing units in 0.05 ml for use in the test.

To test for toxin-neutralizing antibodies, plasma was diluted 1:10 in veronal buffer pH 7.2 and heat inactivated at 56°C for 30 min. Twofold serial dilutions of the plasma were made in 0.05 ml volumes in saline. Next, 0.05 ml of the lysing solution with one to two lysing units was added to all wells. The plates were incubated overnight at room temperature. After incubation, 0.05 ml of 5% srbc was added to all the wells. The plates were incubated for 12 hr at 35°C and then at room temperature for another 24 to 36 hr, at which time the test was interpreted. A positive result was considered to be the highest plasma dilution causing significant inhibition of hemolysis. Each plasma sample was checked for nonspecific hemolysis by mixing 0.05 ml of 1:10 plasma, 0.05 ml of normal sterile nutrient broth, and 0.05 ml of the srbc solution. The lysing solution was retitrated with the test to check toxin concentration and activity.

In Vitro *Phagocytosis Assays*

Ten falcons with staphylococcal pododermatitis from the study group and four clinically normal falcons with no history of pododermatitis were evaluated with respect to the ability of their leukocytes to phagocytose *S. aureus* cocci. Clinically affected falcons were not evaluated with regard to disease severity or vaccination and immunomodulation status.

To evaluate the ability of leukocytes (heterophils) to ingest bacteria, 0.25 ml of heparinized whole blood was placed into 1-ml tapered plastic cuvettes. Then 0.15 ml of a solution containing approximately 1.7×10^8 *S. aureus* diluted in phosphate-buffered saline (PBS) pH 7.2 was added to the blood. The blood-bacteria mixture was gently agitated to mix the contents and incubated at 35°C. After 5, 15, and 30 min, the mixture was agitated, a small volume was collected with a capillary tube, and a routine blood smear was made on a glass slide. Slides were fixed in methanol and stained with a rapid Wright's type stain. Then, 50 or 100 heterophils were counted randomly and recorded as either containing or not containing intracellular cocci.

To evaluate the role of plasma factors in phagocytosis, 1.0 ml of heparinized whole blood was collected, 0.5 ml aliquots were placed into two 10-ml flat-bottomed plastic tubes, and the cells in both tubes were washed twice in warm PBS pH 7.2 to remove plasma. To one tube, 0.1 ml of PBS was added; to the other, 0.1 ml of homologous plasma recovered earlier was added. Then, to both tubes, 0.15 ml of the concentrated bacterial solution was added. Tubes were gently agitated and then sampled at 5, 15, and 30 min. Slides were prepared and results recorded as above. The tube with plasma served as a normal control. In order to characterize the role of plasma factors further, this assay was performed also using plasma that was heat inactivated at 56°C for 30 min before being added to the system.

Cytologic Slides

Thirty cytologic slides were made from smears of purulent (synovial) exudates and from impression smears of inflammatory tissues at surgery. Slides were made before the tissues or exudates were contaminated with peripheral blood. Slides were fixed in methanol, stained with a rapid Wright's type stain, and examined microscopically for subjective evaluation.

Histopathology

Thirteen surgical biopsies of the epidermis, dermis, and inflammatory tissues were fixed in 10% buffered formal saline. Sections were prepared routinely, stained with hematoxylin and eosin, and examined microscopically for subjective evaluation.

Results

Clinical Management and Bacteriology

The breakdown of clinical cases, clinical grading, treatment

Table 15.4
Mean pretreatment leukocyte counts (cells/mm^3) based on clinical class

Class of disease[a]	*Number of cases*	*Mean wbc*	*Standard deviation*	*Range*	*% of cases with normal wbc*
Normals	35	10,600	±2,900	7,700-13,500	N/A[c]
I	0[b]	N/A	N/A	N/A	N/A
II	3	10,700	2,100	8,700-12,900	100
III	7	16,400	4,200	9,500-23,800	14
IV	16	16,800	6,500	9,200-35,700	31
V	1	58,000	N/A	N/A	0
Overall	27	17,600	9,900	7,700-58,000	33

[a]Based on worse foot.
[b]No falcons presented with Class I disease alone.
[c]Not applicable.

groups, and the results of the clinical management and bacteriology are outlined in Table 15.1.

Clinical success was achieved with 29 of 31 falcons (94%). Two falcons were euthanized: a Class V (both feet) in which treatment was attempted, and a Class IV (one foot) with osteomyelitis and concurrent falcon pox infection in which the majority of the plantar surface of the foot became necrotic, partially as a result of the pox lesions encroaching upon the incision. Two falcons, both Class IV, required two surgical procedures before healing was obtained. All other Class II, III, and IV feet healed uneventfully after one surgical procedure. All Class I feet healed with conservative therapy.

The 15 falcons in treatment Group 1 showed an 87% success rate, with 2 birds being euthanized (Cases 12 and 13). One falcon (Case 11) in Group 1 required a second surgery to obtain healing. The 7 falcons in Group 2 had a 100% success rate, with 1 falcon requiring two surgeries. All 9 falcons in Group 3 healed uneventfully after one surgery.

There were no adverse side effects noted related to the administration of levamisole or to the vaccination (with or without BCG) protocols used in this study.

Hematology

The raw hematology and relevant clinical information are presented in Table 15.2. The reference values for total leukocytes in this practice range from 7,700 cells/mm^3 to 13,500 cells/mm^3 and have a mean of 10,600 cells/mm^3 with a standard deviation of 2,900 cells/mm^3.

Staphylococcal pododermatitis in these falcons caused significant ($p < .01$) total leukocyte elevations when pretreatment (week zero) values were compared to reference (normal) values. The mean total leukocyte count for diseased falcons was 17,600 cells/mm^3 with a range of 8,700 cells/mm^3 to 58,000 cells/mm^3 and a standard deviation of 9,900 cells/mm^3. Falcons with Class II disease alone presented initially with mean normal total leukocyte counts (mean 10,700 cells/mm^3, range 8,700 cells/mm^3 to 12,900 cells/mm^3, standard deviation 2,100 cells/mm^3). Falcons with more advanced disease presented initially with mean elevated total leukocyte counts (Class III mean 16,400 cells/mm^3, range 9,500 cells/mm^3 to 23,800 cells/mm^3, standard deviation 4,200 cells/mm^3; Class IV mean 16,800 cells/mm^3, range 9,200 cells/mm^3 to 35,700 cells/mm^3, standard deviation 6,500 cells/mm^3; the single Class V bird had a total leukocyte count of 58,000 cells/mm^3). All 3 of the Class II diseased falcons (100%) presented with normal total leukocyte counts, 1 of the 7 Class III falcons (14%) presented with a normal leukocyte count, and 5 of the 16 Class IV falcons (31%) presented with normal counts (Tables 15.2 and 15.4).

To determine if vaccination with or without immunomodulation (different treatment groups) induced differences in leukocyte responses during the treatment period, mean values for falcons with Class IV disease from Groups 1, 2, and 3 that healed uneventfully were compared. Nonhealing was suspected to influence leukocyte counts, thus these birds were not included. Class IV birds were used, as this represented the largest number of individuals with similar disease severity (another potential leukocyte variable) within each group, and thus provided the most accurate mean values. Falcons in different treatment groups showed no significant variations in mean leukocyte responses related to vaccination with or without immunomodulation (Fig. 15.1).

To determine if clinical severity (classes of disease) affected the leukocyte response during the course of treatment, I compared falcons with different classes of disease that healed uneventfully. Since vaccination with or without immunomodulation did not induce significant changes in leukocyte values, it was possible to use birds of a given class from all treatment groups for comparison. Different degrees of clinical severity did not induce significant variations in mean leukocyte responses during the course of therapy after the week zero pretreatment values (Fig. 15.2).

To determine if there were differences in leukocyte responses between falcons showing initial positive clinical re-

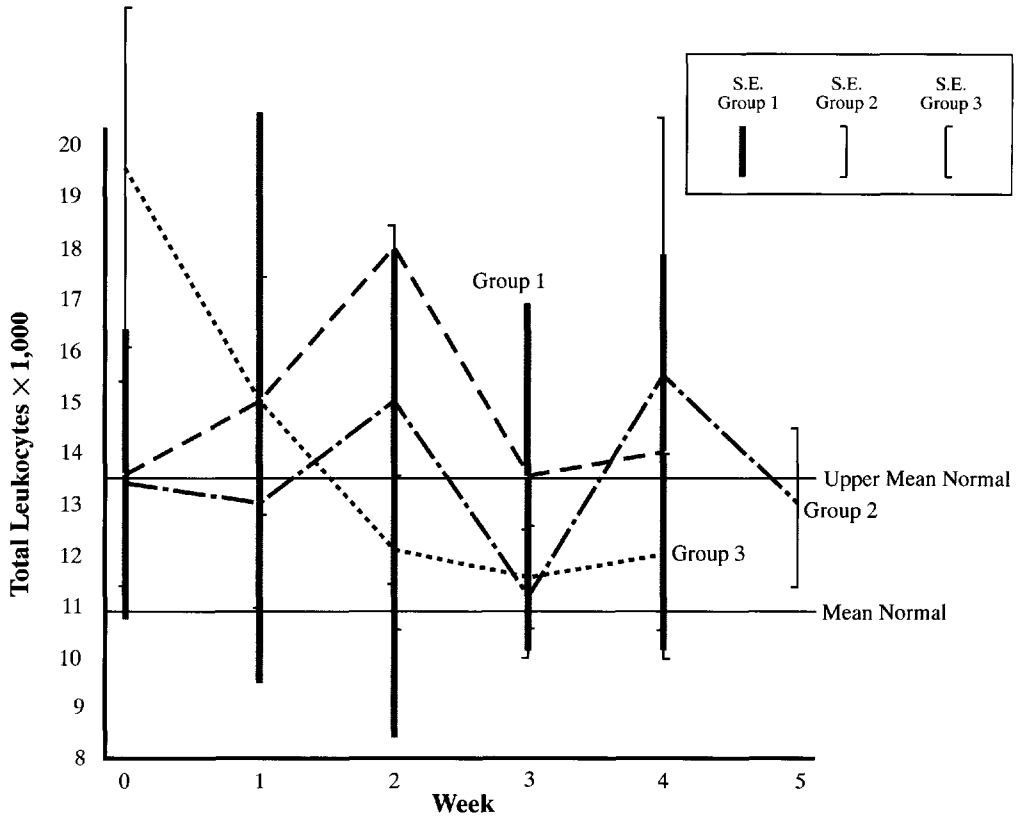

Figure 15.1. Mean leukocyte counts (±hr S.E.) by treatment groups with initial healing, Class IV.

sponses and those showing initial negative clinical responses to therapy (healing or not healing after the first surgery, respectively), I compared birds from all groups and classes showing initial success with those that were initial failures. The mean values for leukocyte responses showed that falcons that were not responding to treatment had values that were elevated, in contrast to falcons that were responding successfully. The mean leukocyte values of these two groups differed significantly at weeks zero ($p < .01$), 1 ($p < .001$), and 3 ($p < .001$); at week 2 there was not a significant difference ($p > 0.3$). Toward the end of the treatment period there was also no significant difference between these two groups: for week 4, $p > 0.2$; for week 5, $p > 0.1$ (Fig. 15.3).

During the course of treatment, the general pattern of leukocyte responses in affected birds was a decrease toward the normal range, which is graphically demonstrated in Fig. 15.3. However, these changes between week zero and week 5 were not statistically significant for birds showing either initial healing ($p > 0.1$) or initial failure ($p > 0.4$). As late as week 4, both of these groups of birds still had mean leukocyte counts that were significantly elevated (all $p < 0.05$), compared with reference values, although birds that appeared to be healing consistently had mean leukocyte counts lower than those of birds that were initial treatment failures (Fig. 15.3).

Five falcons presented with total leukocyte counts of more than 20,000 cells per ml; three (60%) were initial clinical failures (two euthanasias and one recurrence). Post-op peaks of more than 20,000 cells per ml (initial counts less than 20,000) did not appear to be as associated with failure, as only one of eight (13%) of the birds in this category was an initial failure.

Nine falcons presented with normal total leukocyte counts, seven of which showed leukocyte elevations above the normal range at approximately 1 to 2 wk after surgery followed by a decrease toward normal. This was best represented by all three Class II diseased falcons (Fig. 15.2). In the Class III birds, the one bird presenting with a normal leukocyte count did not develop elevated counts until weeks 3, 4, and 5. In the Class IV birds, five presented with normal leukocyte counts, three showing post-op elevations and two maintaining normal counts throughout the treatment period. There were seven falcons from different treatment groups that had elevated leukocyte counts late in the treatment course (weeks 4 and 5), although clinically their feet appeared to have healed uneventfully.

Complement Fixation Antibody Titers

The falcons tested, relevant clinical data, and individual titers are shown in Table 15.3. In the complement-fixation (CF) test, all the individual plasma controls showed no non-

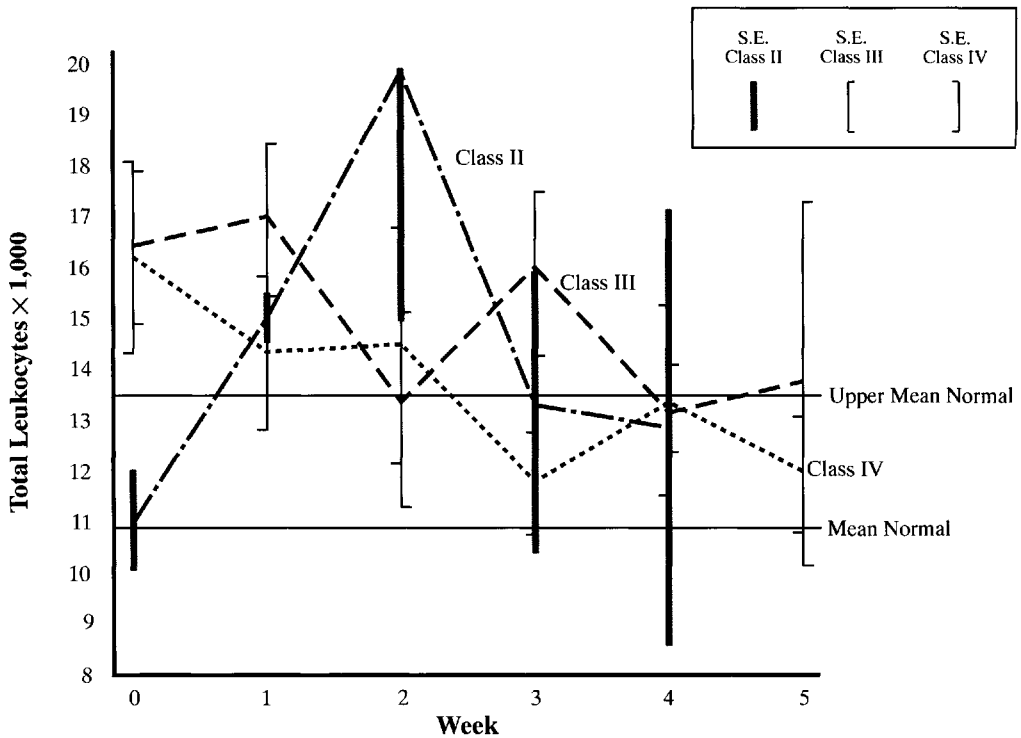

Figure 15.2. Mean leukocyte counts (±S.E.) by clinical classes with initial healing.

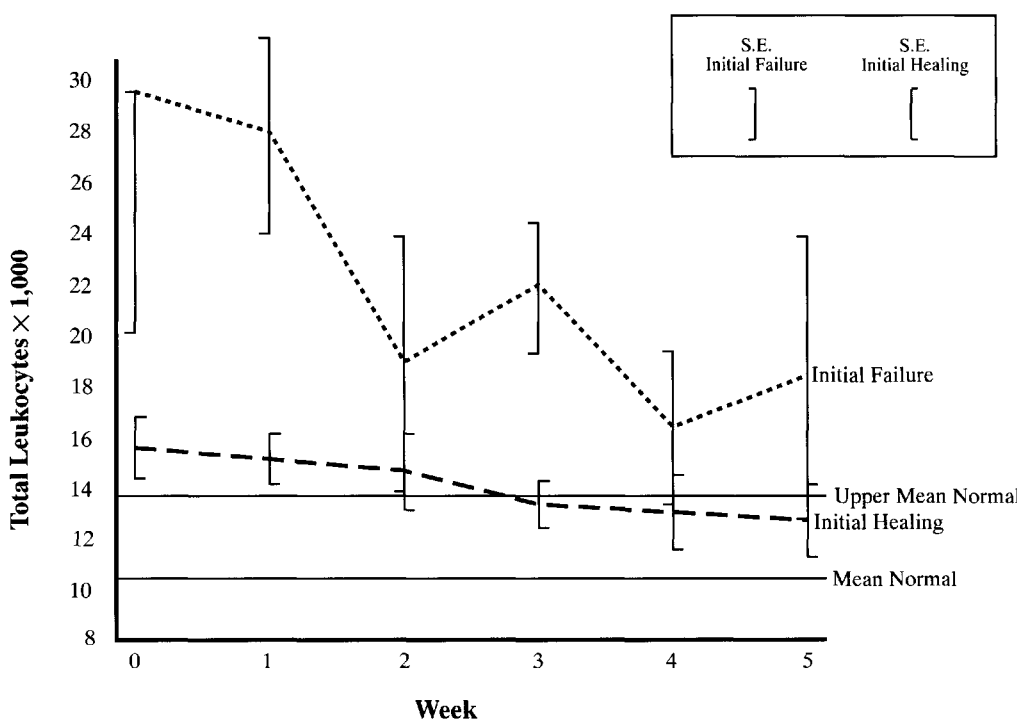

Figure 15.3. Mean leukocyte counts (±S.E.) by initial healing or initial failure.

specific complement fixation, and all other test controls reacted appropriately.

All plasma samples from all the falcons tested showed CF antibody titers against *S. aureus* antigens ranging in titer from 1:20 to 1:160. The geometric mean titer (GMT) for six clinically normal falcons was 56.6. The GMT for seven clinical cases at week zero prior to any treatment was 59.4. There was no apparent difference in titers between normal

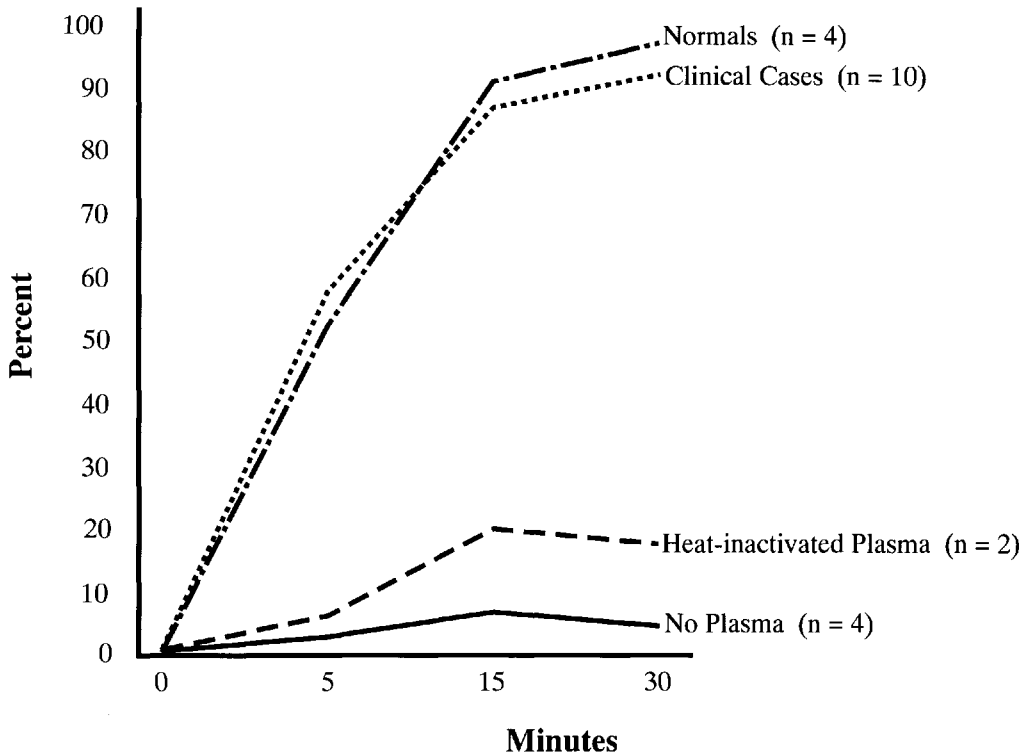

Figure 15.4. Percentage heterophils ingesting *S. aureus*.

and untreated, infected falcons. There was also no apparent relationship between clinical severity (class) and CF titers, nor were there any apparent significant changes in titers in individual falcons during the course of treatment. Many falcons had titers that fluctuated by a dilution from week to week with no apparent pattern. Vaccination, with (Group 3) or without (Group 2) immunomodulation, had no apparent significant effect on the GMT of these birds relative to the controls (Group 1) during the course of treatment. Additionally, the titers in the different treatment groups did not appear to vary significantly from the titers of clinically normal, or week zero, untreated falcons.

Antihemolysin Titers

The falcons tested, relevant clinical data, cases infected with hemolytic strains of *Staphylococcus*, and individual antihemolysin titers are shown in Table 15.3. Of the 22 falcons infected with *Staphylococcus*, 14 were infected with hemolytic strains and 8 were infected with nonhemolytic strains. Some isolates of *S. aureus* were nonhemolytic, and some isolates of other species of *Staphylococcus* were hemolytic. There was no apparent relationship between infection with hemolytic strains and the degree of clinical severity or lack of positive clinical response.

Of the 8 falcons from which nonhemolytic bacteria were isolated, 1 (13%) possessed antibodies against hemolysins. This bird, a vaccinate from Group 2, had relatively high titers of 1:80 at week 2 and 1:40 at week 4. Of the 14 falcons infected with hemolytic strains, 6 (43%) showed titers of 1:10 or 1:20. The vaccine, with or without immunomodulation, did not appear to induce antihemolysin antibodies consistently, if at all.

In Vitro *Phagocytosis Assays*

The raw data for this section are not presented, but the results are shown graphically in Fig. 15.4.

It was found that the heterophils in whole blood from all clinical cases and from normal falcons were able to phagocytose *S. aureus* efficiently, with 87% to 91% of the heterophils ingesting bacteria within 15 min.

The ability to ingest bacteria was plasma dependent. Washing the cells and removing the plasma markedly inhibited the ability of the heterophils to ingest bacteria in birds shown previously to have normal opsonic/phagocytic abilities. Only 6% of the heterophils of one clinical case and three normal falcons contained bacteria after 30 min of incubation.

It was also shown that the majority of the opsonic/phagocytic ability was dependent on heat-labile plasma factors, presumably, complement. In two cases where the plasma was heat inactivated prior to being added to the leukocyte and bacteria system, only 18% of the heterophils contained bacteria after 30 min of incubation.

Thrombocytes showed very active phagocytosis of bacteria; this activity was also plasma dependent.

Histopathology and Cytology

Histologically, the heterophil was the predominant inflammatory cell, with mononuclear cells seen only occasionally. Heterophils were found in large numbers intravascularly, perivascularly, and infiltrating into the connective tissue of the dermis in areas associated with or adjacent to epidermal ulcers and penetrating scabs. The scabs appeared to be aggregations of red blood cells, heterophils, and fibrous tissue. Bacteria were not seen in large numbers in inflammatory tissues; however, cocci could be seen colonizing the necrotic scab, and were occasionally present in the zone between the scab and inflammatory dermal tissue. For additional information on pododermatitis histopathology, see Remple and Al-Ashbal (chapter 17, this volume).

Cytologic slides of exudates also demonstrated that the heterophil was the predominant inflammatory cell. Large numbers of normal to toxic, pyknotic heterophils were invariably present, as well as moderate numbers of small and large lymphocytes and reactive, foamy macrophages. Bacteria were often present in variable numbers, ranging from a few to many. Even in the presence of large numbers of bacteria, phagocytosis by heterophils or macrophages was rarely observed. Occasional macrophages had ingested red blood cells and degenerate heterophils.

Discussion

Clinical Aspects

Surgical management of falcon pododermatitis has been described as the treatment of choice (Remple and Remple, 1987; Riddle, 1980). The overall success rate in this study of 94% confirms the good initial results of surgery. However, the long-term recurrence rates still need to be assessed critically. Long-term follow-up is difficult in our practice, and frequently when falcons are returned to their owners the birds are returned to the original management conditions that probably initiated the disease. Additionally, birds with advanced disease generally have sustained significant damage to the normal weight-bearing structures of the foot, such as the metatarsal pad, further predisposing them to foot trauma even under ideal environmental conditions. Thus, if a bird is returned with a recurrent infection, it may be difficult to determine if it is a recurrence of the primary infection or a new infection altogether. Certainly many primary infections do recur, and these infections apparently may stay in remission for many months. Similarly, there appear to be chronological variations among individual birds associated with the natural progression of pododermatitis; that is, some birds may exhibit Class II disease much longer than others before progressing to Class III. The interactive host and pathogen factors regulating the development of invasive infection are undoubtedly a key component to the overall pathogenesis of this disease.

Staphylococcal bacterin vaccination, with or without immunomodulation, did not appear to have any significant beneficial or detrimental clinical effects on the falcons evaluated in this study, nor was there any evidence of hypersensitivity reactions in the feet or at the vaccination sites in these birds. Although birds in the vaccination and immunomodulation groups had slightly better clinical response than the nonvaccinates, given the overall high clinical success rates, the differences are not significant.

The results of previous clinical reports of vaccination against *Staphylococcus* in pododermatitis differ widely, although it should be noted that the antigenicity of the vaccines used in the different studies, including the one reported in this chapter, also differed. Satterfield and O'Rourke (1980, 1981) have reported that toxoid vaccination may initially cause worsening of clinical signs consistent with a delayed-type hypersensitivity (DTH) response, but will result ultimately in rapid clinical improvement and reduce tissue damage associated with infection. Cooper (1978a) found no obvious benefits to vaccination with a toxoid. Conversely, in experimental staphylococcal synovitis in chickens, antistaphylococcal antibodies were shown to potentiate the disease (Forget et al., 1974). The role of vaccination in staphylococcal disease is not clear; this is discussed in more detail below.

In the hematology section of this chapter, I have attempted to assess the effects of disease, clinical severity of disease, clinical response, and vaccination with or without immunomodulation on systemic total leukocyte responses. I have found in preliminary work that heterophilia is the predominant response (Oaks, unpublished data). Thus, I have made no attempt here to distinguish among subgroups of leukocytes.

The data for the hematology were quite variable. Although some patterns of leukocyte responses were apparent, there were many individual cases that did not follow the expected or general patterns. Some of these anomalous cases have hypothetical explanations, while others could not be explained. It should be remembered that as a clinical tool, leukocyte values are of high sensitivity and low specificity (Dein, 1986), and there were many potential uncontrolled or unidentified variables in individual cases that may have affected leukocyte counts. These could include factors such as unilateral versus bilateral infections, mixed bacterial infections, individual temperaments and the consequent stress levels associated with handling and daily treatments, and unidentified concurrent diseases. These types of factors make critical analyses of the leukocyte data difficult, but

from a clinical standpoint I believe that the hematology information reported herein is relevant.

The systemic leukocyte responses to pododermatitis infections followed the expected patterns, and correlated well with the pathological and prognostic hypotheses outlined in the clinical classification scheme. Falcons with established infections had elevated leukocyte counts that returned toward normal with appropriate therapy. Vaccination with or without immunomodulation did not affect leukocyte responses with respect to controls. Leukocyte elevations were not apparent until infection was established (in Classes III, IV, and V), which is similar to the leukocyte changes found in Gentoo penguins (*Pygoscelis papua*) with pododermatitis (Hawkey et al., 1985). Falcons with early localized infections (Class II) presented with normal counts, most birds with more active infections (Class III) had elevated counts, and birds with more chronic infections (Class IV) had an approximately equal mixture of normal and elevated counts. In the more chronic Class IV, the fibrotic response may insulate the body from antigenic stimulation, although there are still viable bacteria present. The post-op elevations noted in Classes II and IV may be related to bacterial release and exposure during surgery, more active inflammation, and surgical stress. Persistent elevations of leukocyte counts throughout the treatment period could be correlated with initial clinical failure. Although not always dramatic or consistent, an elevated leukocyte count at weeks 3-4 may suggest a case warranting reevaluation even if other clinical parameters (swelling, incision healing) appear favorable. Additionally, a presenting leukocyte count of more than 20,000 cells per ml was a poor prognostic sign highly correlated with initial failure, which may be related to active and deep-seated infection. Most cases with osteomyelitis fell into this category.

The poor prognosis proposed with the more advanced Class IV and V disease was supported, in that clinical difficulties encountered in this study were all in these classes of feet.

Staphylococcus *and Falcon Immune Responses*

Staphylococcus, and *S. aureus* in particular, was the primary pathogen isolated in this study (Table 15.1). Its relationship to host immune defense systems, as in other species, is far from clear, although invasive infections are usually associated with some primary or secondary immunoincompetence (Mudd, 1970; Morse, 1980; Muller, 1983; Anderson, 1986; Gerlach, 1986). However, *Staphylococcus* also has considerable ability to evade immune responses in immunocompetent hosts (Remple and al-Ashbal, chapter 17, this volume). The opsonic/phagocytic system is the primary mechanism for removal of *Staphylococcus* (Anderson, 1986). There is no clear relationship between serum antistaphylococcal antibody titers and the clinical course of staphylococcal infections (Ekstedt, 1972), although in humans defective intracellular killing of bacteria by neutrophils can result in chronic staphylococcal granulomatous disease (Ammann and Fudenberg, 1980). *Staphylococcus* may also induce delayed-type hypersensitivity reactions (Morse, 1980) and Arthus reactions (Muller, 1983); this, along with the role of macrophages and lymphocytes in processing antigens, producing antibodies, and overall immune system regulation, would suggest that cell-mediated immunity is also important in resistance to or persistence of infection.

The pathomechanism(s) for immunosuppression in avian species is not known, although endocrine and metabolic abnormalities are generally implicated. In the fowl (*Gallus domesticus*), heat stress has been shown to suppress humoral immunity, possibly through altered thyroid or adrenal function (Chang and Hamilton, 1979; Thaxton, 1978). Serum T_3 and to a lesser extent T_4 have been shown to regulate antibody formation (Keast and Ayre, 1980; Kollias, 1986). Stress (social or environmental) results in increased secretion of adrenal cortical steroids through the hypothalamic/pituitary/adrenal axis (Ringer, 1976), which affects immune function (Thaxton, 1978; Kollias, 1986). Progesterone and testosterone are also important regulators of the avian immune system (Kollias, 1986). Nutritional deficiencies have also been shown to increase susceptibility to bacterial diseases (Cheville, 1979; Kollias, 1986).

Many of these factors could have been acting upon the falcons in this study. For example, peregrine falcons removed from the wild as juveniles (the case for all birds in this study) molt very poorly in captivity; these birds generally will molt more rapidly with thyroid hormone therapy (Oaks, unpublished data). Wild-caught falcons also breed very poorly in captivity, suggesting sex-hormone abnormalities. Falcons acquired as neonates (sexually and socially imprinted to humans) are presumably less psychologically stressed and tend to breed and molt normally in captivity. Additionally, the high incidence in our practice of other diseases generally associated with immunosuppression, such as pulmonary aspergillosis, supports the hypothesis that the falcons in this study may be immunologically deficient.

However, no specific immune defects were identified in this study. The presence of heterophils, lymphocytes, and macrophages in infected feet would suggest that at least fundamental immunoregulatory mechanisms are intact, although specific problems such as opsonization defects, defective intracellular killing, or hypersensitivity reactions cannot be ruled out. Remple and Al-Ashbal (chapter 17, this volume) present evidence that hypersensitivity reactions may be important in initiating this disease.

Poor *in vivo* and strong *in vitro* bacterial phagocytosis have been reported in normal chickens under experimental conditions (Carlson and Allen, 1969; Chang and Hamilton,

1979). The presence of large amounts of naturally occurring heat-labile opsonins (such as complement) for *Staphylococcus* that cannot be enhanced by vaccination (or clinical disease in this study) has been reported in normal humans and rabbits (Cohen, 1987). It appears that these phenomena are also present in falcons and do not necessarily represent abnormalities on the part of the host. Rather, the relatively poor *in vivo* responses seen may indicate successful evasive tactics on the part of *Staphylococcus*, either by direct immunological evasion or by physical isolation (that is, fibrosis).

Some researchers feel that continual natural exposure to *Staphylococcus* places most humans in a perpetual state of maximal immune responsiveness (Ekstedt, 1972), a concept that could account for the lack of complement-fixing antibody increases found in these vaccinated falcons, and for the high background titers observed in all normal and diseased falcons. This would suggest that these birds are capable of adequate antibody responses if stimulated.

There were no clinical or serologic responses to immunomodulation. Levamisole is known to potentiate immune responses in immunosuppressed humans and animals. It has been shown to modify and stimulate the action of T-lymphocytes, cell-mediated immunity, and responsiveness to antigens and mitogens. Levamisole has no effect on normal individuals (Brunner and Muscoplat, 1980). It has been reported to increase the protective effect of vaccines (antibody responses) in birds (Maheswaran et al., 1980; Panigraphy et al., 1979), and to result in clinical improvement of chronic human staphylococcal infections (Brunner and Muscoplat, 1980).

The cell-wall fraction of the BCG mycobacterium has adjuvant properties that nonspecifically stimulate cell-mediated immunity. The inflammatory response caused by BCG (stimulated T-cells and activated macrophages) may enhance the specific immune response to unrelated antigens (Bast et al., 1974), and is similar in principle to Freund's adjuvant. BCG is effective only in individuals capable of developing delayed-type hypersensitivities (Bast et al., 1974). The intradermal vaccination sites on the falcons in this study showed no clinical evidence of DTH; however, avian DTH responses to *Mycobacterium* sp. are not consistent (Cooper, 1978b). The lack of response to levamisole and/or BCG suggests that immune deficiencies are not present.

The role of hemolysins and other staphylococcal exotoxins in falcon pododermatitis, as in other species, warrants further investigation. In humans and falcons, high antitoxin titers may modify the development and outcome of disease (Ekstedt, 1972; Satterfield and O'Rourke, 1981). The falcons in this study had weak and variable antihemolysin titers, with no apparent correlation with infection with hemolytic strains. Vaccination did not alter antitoxin titers, although this may be related to the immunogenicity of the bacterin used. High antitoxin titers potentially may reduce tissue necrosis and fibrosis, a prominent feature of falcon pododermatitis, and permit improved immune clearance of bacteria.

Staphylococcus aureus is not normally a common inhabitant of the falcon foot (Cooper and Needham, 1976), yet it is by far the most common pathogen isolated from pododermatitis lesions, although many other species of bacteria may be isolated (Halliwell, 1975; Cooper, 1978b; Riddle, 1980; Satterfield and O'Rourke, 1980; Remple and Remple, 1987). Disruption of the podal integument would be expected to expose the underlying tissues to a wide variety of potential pathogens, as falcons perch in environments containing decaying food, feces, and soil. The overrepresentation of *S. aureus* as the pathogen does suggest a special relationship between the host and this organism. Generalized immunosuppression on the part of the host was the hypothesis evaluated in this study, and although no specific defect could be identified, this theory is far from being fully evaluated, given the complexity of the immune system, particularly when dealing with *Staphylococcus*. *S. aureus* infections may predominate because this organism is the most successful opportunist taking advantage of a host with only localized immune defects such as persistent epithelial trauma, that is, from pressure due to a scab or corn on the metatarsal pad. There is also the possibility that unadapted strains (i.e., human) of *Staphylococcus* in contact with or infecting the avian foot may elicit inappropriate immune responses such as interference antibodies or hypersensitivity reactions (Satterfield and O'Rourke, 1980).

Conclusions

The findings of this study do not support the hypothesis that systemic immunosuppression is a major factor in the pathogenesis of falcon pododermatitis. The interaction of environmental, host, and bacterial factors favoring invasive staphylococcal disease is complex. The imbalance of one or all of these factors, each with many variables, which may allow an often innocuous bacterium to become an invasive pathogen, is poorly understood in falcons, as it is in other species. It is still not clear if falcon pododermatitis represents *Staphylococcus* circumventing normal host immunity to gain access to tissues, or if an underlying host immune defect (either insufficient or inappropriate) is allowing progressive infection.

The methodology used in the study reported here was relatively crude, and would be unable to detect the types of subtle host and pathogen interactions that may be important etiological factors in this disease, such as hypersensitivity reactions, selective deficiencies of protective antibodies, production of interfering antibodies, or defective intracellular killing abilities. Identification of these interactions will allow the classification of these responses as host-deficient,

host-inappropriate, or primarily the result of bacterial virulence factors. The tremendous importance of the localized immune "defects" caused by persistent trauma to the feet should not be overlooked.

It is my hope that the fundamental information from this study will help direct more refined studies in the future and that the clinical information will assist practitioners faced with treating this difficult disease.

Acknowledgments

I would like to thank H. H. Sheikh Hamdan Bin Rashid Al Maktoum for financial support, and Dr. David Remple for his insight, assistance, and comments regarding this work. I am also indebted to Dr. Ulli Wernery for assistance with microbiology and serology, Dr. Adnan Al-Ashbal for histopathology interpretation, and Cheryl Remple for assistance with hematology.

Product List

[a]Vemorsul, Beecham Laboratories, Smith Kline Beecham Animal Health, Whiteside Business Park, 812 Springdale Dr., Exton, Pennsylvania 19341 USA

[b]Pipracil, Lederle Piperacillin Inc., Carolina, Puerto Rico 00630 USA

[c]Amiglide V, Aveco Co. Inc., 800 5th St. N.W., Fort Dodge, Iowa 50501 USA

[d]Injacom 100 plus B-complex, Roche Animal Health and Nutrition, Hoffmann-La Roche Inc., 340 Kingsland St., Nutley, New Jersey 07110 USA

[e]Lysigin, Bio Ceutic Laboratories, 2621 N. Belt Hwy., St. Joseph, Missouri 64502 USA

[f]Nomagen, Fort Dodge Laboratories Inc., 800 5th St. N.W., Fort Dodge, Iowa 50501 USA

[g]Levasole, Pitman-Moore, 421 E. Hawley St., Mundalein, Illinois 60060 USA

[h]Staphyslide Test, BioMerieux, Marcy-L'Etoile, 69260 Charbonnieres-Les-Bains, France

[i]Unopette Test 5877, Beckton-Dickson, P.O. Box 243, Cockeysville, Maryland 21030 USA

[j]Neubauer Haemocytometer, Weber, England

[k]Hemacolor, Diagnostic Systems, 100 E. Nasa Rd., Ste. 303, Webster, Texas 77598 USA

[l]Ambozeptor, Amboceptor-Behring, Postfach 1130, 3550 Marburg (Lahn), Germany

[m]Preserved guinea pig serum, Wellcome Diagnostics, P.O. Box 1887, Greenville, North Carolina 27835 USA

[n]Kantrim, Aveco Co. Inc., P.O. Box 717, Ft. Dodge, Iowa 50501 USA

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CHAPTER 16

Trichomoniasis in Owls: Report on a Number of Clinical Cases and a Survey of the Literature

MARK A. POKRAS, B.S., D.V.M.
Wildlife Clinic
Tufts University School of Veterinary Medicine
North Grafton, Massachusetts, U.S.A.

ERIC B. WHEELDON, B.V.M.S., Ph.D., M.R.C.Path.
Department of Experimental Pathology
Smith Kline & French Laboratories
Philadelphia, Pennsylvania, U.S.A.

CHARLES J. SEDGWICK, B.S., D.V.M.
Wildlife Clinic
Tufts University School of Veterinary Medicine
North Grafton, Massachusetts, U.S.A.

ABSTRACT: Cases of trichomoniasis in one barred owl (*Strix varia*) and eight barn owls (*Tyto alba*) are reported. Owls were emaciated, with ulcers in the oropharynx. Lesions caused destruction of both soft tissue and bone. Questioning of several raptor rehabilitators around the United States indicated that barn owls were most frequently affected, followed by great horned owls (*Bubo virginianus*) and barred owls. Trichomoniasis appeared more common and pathogenic in barn owls in the western part of the United States. The authors speculate on the small number of reports from owls compared with the Falconiformes, where the disease has been well documented. The importance of a thorough examination of the mouth in all owls presented for veterinary examination is stressed.

KEY WORDS: raptor, barred owl, *Strix varia*, barn owl, *Tyto alba*, trichomoniasis, frounce, United States.

Trichomoniasis is a well-documented condition of birds caused by the flagellated protozoan *Trichomonas gallinae*. The disease is considered to be enzootic in pigeons, and is often referred to as "canker" (Keymer and Arnall, 1980, pp. 126-28, 144-145). It also occurs in falcons and has been described for centuries by falconers as "frounce" (Stabler, 1954). The disease is transmitted to raptors through their feeding on infected pigeons. This chapter describes the occurrence of trichomoniasis in owls, based on one detailed clinic case, eight owls seen at postmortem, and extensive communication with owl researchers.

Case Report

An adult barred owl (*Strix varia*) was presented to the Tufts Wildlife Clinic, Tufts University School of Veterinary Medicine, in a moribund and emaciated condition. Physical examination revealed a cloacal temperature of 33.2°C (normal 40° to 41°C). The bird weighed 420 gm (normal adult weight range for males and females is 600 to 900 gm). The packed cell volume (PCV) was 18% (normal 45% to 50%) and the total serum protein (TSP) was 5.5 gm/dl (normal 3.0 to 5.0 gm/dl). The oral cavity had a foul odor and was filled with a tenacious saliva. A raised, yellow-tan plaque, approximately 2 cm in width, extended 2-3 cm along the roof of the mouth and totally obliterated the choanal opening (Fig. 16.1). Differential diagnoses included fungal lesions (*Candida* spp. or *Aspergillus* spp.), trichomoniasis, capillariasis, pox, and vitamin A deficiency. A saline wet mount of an oral swab was examined microscopically; numerous motile, flagellated protozoans identified as *Trichomonas* spp. were revealed.

The bird was warmed on a heating pad and 40 ml of a warmed 50:50 mixture of lactated Ringer's solution and 5% dextrose solutions was infused into the basilic (cutaneous ulnar) vein. Chloramphenicol[a] (30 mg) and dexamethasone[b] (1 mg) were added to the fluids.

Hydration was maintained with intravenous fluids. A 7-day course of intramuscular chloramphenicol, and injections of vitamins A, C, D_3, and B complex and iron dextran were administered. Oral metronidazole[c] (30 mg SID for 7 days) was used to treat the trichomoniasis. Fecal flotations

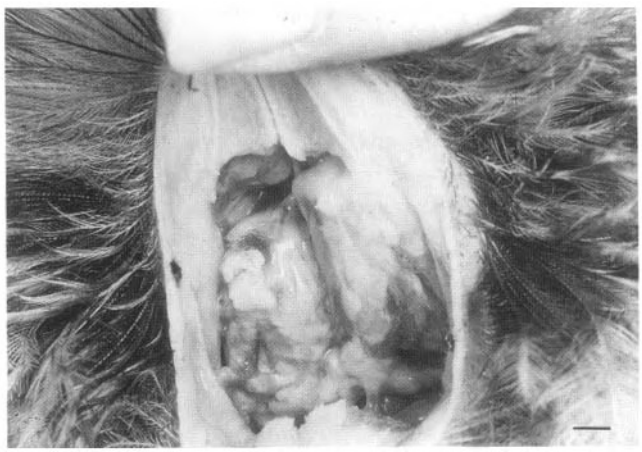

Figure 16.1. Oral cavity of barred owl upon admission. Note the raised, yellow-tan plaque obliterating the choanal opening with extensive necrosis of the mucosa and palate. Bar = 0.5 cm.

revealed gastrointestinal nematodiasis, which was treated with a single 0.5 mg dose of ivermectin[d] given SQ (a dose was calculated from 0.01mg/Kcal of basal metabolism).

Three 7-day courses of metronidazole were required to eliminate the protozoans. During each of the 7-day treatments, no organisms could be seen in repeated oral smears, but within 2 days of completing the first and second series of treatments, motile trichomonads were again found. There was no recurrence after the third series. During the first 30 days of captivity, the bird's weight increased from 430 gm to 764 gm. Its PCV/TSP increased from 18%/5.5 gm/dl to 44%/6.2gm/dl, and appetite and behavior appeared normal.

A week after the last metronidazole treatment, the oral lesion sloughed, leaving only the anterior 5 to 6 mm of the hard palate. The oral and nasal cavities and the infraorbital sinuses had become continuous.

During the next week, the bird's appetite declined and the white blood cell count (WBC) steadily increased. The owl ate irregularly and lost condition. Over the next few weeks, pieces of food and regurgitated pellets would regularly lodge in the enlarged oro-nasal cavity. Because of the poor prognosis, the owl was euthanized 74 days after admission at a weight of 480 gm.

A necropsy, performed immediately after euthanasia, revealed no protozoans or nematodes. Several small plaques were present on the posterior air sacs. Impression smears stained with a quick stain showed these to be inflammatory in nature, and no evidence of fungal infection was detected.

Necropsy Cases

Eight barn owls (*Tyto alba*) were seen at the University of California Veterinary School at Davis Raptor Center (Table 16.1). All cases were dead on presentation or were moribund and died soon thereafter. All received a complete necropsy. Typically, the birds had been found in a weak, emaciated condition, unable to fly. Only 1 bird (Case 7) had other clinical problems (multiple fractures). With the exception of Case 1, all birds had large distinct lesions in the roof of the mouth, which ranged from small nodules to extensive necrotic plaques. Figure 16.2 illustrates the lesions in Case 8.

Table 16.1
Clinical signs and lesions in barn owls (*Tyto alba*) infected with trichomoniasis

Case	*Species*	*Sex*	*Presenting clinical signs*	*Lesions*
1	barn owl	M	emaciation	raised nodules—floor of pharynx
2	barn owl	M	extreme emaciation	necrotic plaque—roof of mouth gut empty
3	barn owl	F	extreme emaciation comatose	necrotic plaque—roof of mouth gut empty
4	barn owl	F	very depressed	caseous plaque—roof of mouth trichomonads on smear
5	barn owl	M	depressed anorectic	small, raised nodules—roof of mouth
6	barn owl	?	emaciation	large plaque—roof of mouth gut empty
7	barn owl	M	severe emaciation multiple fractures	multifocal stomatitis
8	barn owl	?	emaciation	ulcerated lesion—roof of mouth

Microscopic examination of sections through the oral lesions revealed severe necrotizing, inflammatory lesions, often extending deep into underlying musculature and adjacent tissues, and occasionally involving the palatine bone.

Literature Review

Although there are numerous reports of trichomoniasis in Falconiformes, there are few published reports in Strigiformes. Tanabe (1925) reported the isolation of *T. gallinae* from an unidentified species of owl. Böttcher (1981) included an eagle owl (*Bubo bubo*) from West Germany with probable trichomoniasis in an analysis of 58 diseased raptors. Hardy et al. (1981) identified trichomoniasis at postmortem in 1 of 180 barn owls examined over a 10-yr period in England. Jessup (1980) presented several cases of tricho-

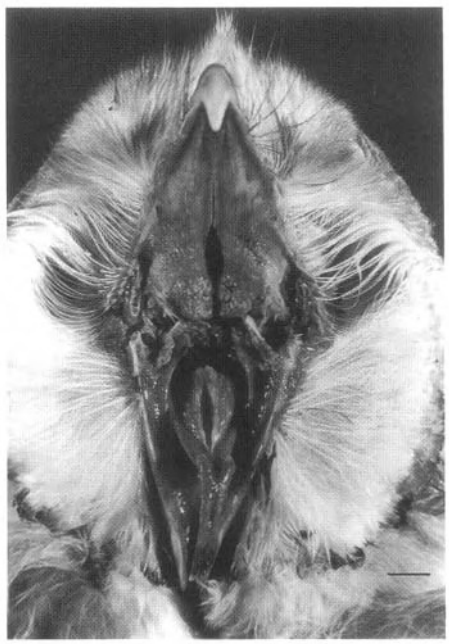

Figure 16.2. Oral cavity of barn owl (Case 8) at postmortem. Note the erosive lesion involving the mucosa lateral and caudal to the choanal opening. Bar = 1 cm.

moniasis in barn owls and great horned owls (*Bubo virginianus*) in California that resembled the cases in this report, and they proved refractory to treatment. Schulz (1986), in a survey of 1,638 barn owls (received at seven California rehabilitation centers), reported trichomoniasis to be the most common disease observed: of 72 diseased owls, 40 were diagnosed as having trichomoniasis. Schulz has reported that most of the cases seen were quite advanced, including thick oral pseudomembranous lesions, necrosis, and secondary infections.[1] Tappan examined the case records for 872 owls received between 1978 and 1987 by the Wild Bird Rehabilitation Center of the Audubon Park Zoo in New Orleans, Louisiana, and found only 3 records of trichomoniasis: 1 in a barn owl and 2 in barred owls.[2] Work over eight years on more than 200 barn owls being rehabilitated at several facilities in New Jersey revealed no diagnosed cases of trichomoniasis (Pokras, unpublished data).

Trichomonas gallinae is a flagellated protozoan (Class Zoomastigophorasida, Family Trichomonadidae). Pyriform in shape, it averages 10 × 5μ, slightly larger than an erythrocyte (Levine, 1985, pp. 72-74). Trichomoniasis is synonymous with "canker" of pigeons and with "frounce" of birds of prey. Although infection has been reported in psittacines, passerines, and raptors, most researchers consider columbids (pigeons and doves) to be the primary hosts and reservoirs for the organism (Whiteman and Bickford, 1979, pp. 142-144). Levine (1985, pp. 72-74) states that in pigeons the highest mortality occurs in the young, and that 80% to 90% of adults may be infected asymptomatically. Among columbids, vertical transmission occurs from adults to young via the crop milk. The feeding of crop contents may also maintain infection within groups of psittacines. Courtship feeding and the oral presentation of food to the young could play a role in disease transmission in a variety of taxa. Some researchers have speculated that pigeons could transmit *Trichomonas* spp. to other species by contaminating water sources (Stabler, 1954), although to our knowledge this has never been proven to occur. Raptors probably become infected with trichomoniasis by preying on columbids.

Although other trichomonads may occur in the distal gastrointestinal tract of birds, *T. gallinae* is most common in the oropharynx. It may extend down the esophagus to the crop and the proventriculus. In severe cases, caseous lesions will be present on many serosal surfaces and disseminated throughout the liver (Levine, 1985, pp. 72-74). Since the infection often results in dysphagia, clinical signs are primarily those of profuse salivation, emaciation, and weakness. Where penetrating cranial lesions have occurred, CNS signs may result, which include primarily a loss of balance (see note 1). Budgerigars (*Melopsittacus undulatus*) are said to vomit early in the disease (Baker, 1986). Later, they may exhibit esophageal obstruction and respiratory stridor (Keymer, 1982). Vomiting has not been reported in owls.

Diagnosis of trichomoniasis is based on the presence of characteristic lesion and demonstration of the organism in wet oral smears. The parasite is extremely delicate and is killed by drying or chilling. It is incapable of encystment (Stabler, 1954). The protozoan can be readily cultured, but this is rarely used for diagnostic purposes. Recently, a rapid field technique used for diagnosis of *Trichomonas foetus* infection in cattle has been modified for detecting avian trichomoniasis. The avian culture pouch[e] is designed to detect *T. gallinae*.

A number of antiprotozoal drugs have been used for the treatment of trichomoniasis. Campbell (1983) provides an excellent review of therapeutic options. Carnidizole,[f] a drug developed to treat trichomoniasis in pigeons, has recently become available in the United States. We have found this drug very effective in raptors at a dose rate of 0.28 mg per Kcal of basal metabolic rate.

Discussion

Trichomoniasis in owls has been reported less frequently than in diurnal raptors. The typical presenting signs include weakness and emaciation, which relate to a period of starvation.

Columbids are not a major food item for most species of owls, so we must consider that the small number of cases reported may be due to a low incidence of exposure to the

parasite. The barn owl probably preys on pigeons more than any other owl in North America because both species frequent buildings.[3] Other native owls that might take pigeon-sized prey, such as the barred owl and great horned owl, do not frequent areas in which pigeons roost. They are less likely to encounter the parasite and are therefore less likely to develop the disease, and/or to evolve resistance to it. No studies have been done with owls to determine if owl populations could be infected with nonpathogenic forms of the parasite, although such studies have been done with pigeons.

There are several possibilities why trichomoniasis seems to occur more frequently in barn owls in the western United States. Strains of *Trichomonas* spp. with increased pathogenicity may occur there, or, for as-yet-undefined reasons, one population of birds may be more susceptible than others. Trichomoniasis is not native to North America; it arrived with the introduction of pigeons and doves from the Old World by European settlers. Native species, being naive to the parasite, may have experienced large losses following this introduction. Hanson (1969) speculates that trichomoniasis may have played a major role in the extinction of the passenger pigeon (*Ectopistes migratorius*) in the United States.

Only a few cases of trichomoniasis have been reported from Europe and Japan, where pigeons and barn owls have lived together for centuries. On the East Coast of the United States, where they have coexisted for more than 200 years, no cases of the disease in barn owls have been reported. On the West Coast, however, trichomoniasis appears to be a significant cause of death for barn owls. If we assume that the parasite is a more recent arrival on the West Coast, the differential mortality seen today may be due to an adaptation over time of East Coast barn owl populations to the parasite.

Identification of the parasite antemortem using cytologic techniques is necessary for proper diagnosis. A thorough examination of the mouth should be an important part of any physical examination of raptors. Trichomoniasis should be suspected in any oral lesions seen.

Stabler (1954), in discussing the disease, states that birds that recover from the infection are immune. He also speculates that, since *Trichomonas gallinae* is known to occur in a variety of strains that vary widely in pathogenicity, infection with avirulent forms of the parasite may provide a lasting immunity to more virulent infections. This speculation suggests the possibility for immunization of birds being used for falconry, or those in endangered-species reintroduction projects.

Notes

1. Personal communication with T. Schultz, School of Veterinary Medicine, University of California at Davis.

2. Personal communication with A. Tappan, New Hampshire Fish and Game Department.

3. Personal communication with L. Soucy, the Raptor Trust, Millbridge, New Jersey.

Product List

[a]Chloromycetin Palmitate, Aveco Co. Inc., P.O. Box 717, Ft. Dodge, Iowa 50501 USA

[b]Vedco Inc., St. Joseph, Missouri 64504 USA

[c]Geneva Generics, 2599 W. Midway Blvd., Broomfield, Colorado 80020-1632 USA

[d]Ivomec, MSG AGVET, Division of Merck and Co. Inc., 126 E. Lincoln Ave., Rahway, New Jersey 07065-4687 USA

[e]InPouch-TG, BioMed Diagnostics, 1743 Hudson Dr., San Jose, California 95124 USA

[f]Spartrix, Wildlife Pharmaceuticals, Inc., 1401 Duff Dr., Ste. 600, Fort Collins, Colorado 80524 USA

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CHAPTER 17

Raptor Bumblefoot: Another Look at Histopathology and Pathogenesis

J. DAVID REMPLE, D.V.M.
Dubai Falcon Hospital
Dubai, United Arab Emirates

ADNAN A. AL-ASHBAL, A.M.R.C.Path., Dip. Histopath.
Department of Histopathology
New Dubai Hospital
Dubai, United Arab Emirates

ABSTRACT: Bacterial and histopathological investigations were carried out on the feet of 15 raptors afflicted with bumblefoot to elucidate some of the factors involved in the pathogenesis of this problem. *Staphylococcus aureus* was found associated with all afflicted feet. All feet also showed evidence of chronic granulomatous inflammation, although the exact appearance varied with stage of progression. Early cases of bumblefoot had plantar ulceration, mononuclear cell infiltrate, and evidence of perivascular necrotizing vasculitis. Later stages of the disease were characterized by hyperplastic fibrosis and a paucity of inflammatory cells. Since *S. aureus* is not prevalent on the feet of wild birds, transfer of human types of this bacteria may be involved in the pathogenesis. The etiologic pathway is hypothesized to involve, in order, repeated trauma to the foot, skin devitalization and ulceration, colonization by pathogenic *Staphylococcus* sp., phagosome inhibition by *Staphylococcus* toxins, host hypersensitivity reaction, and chronic granulomatous inflammation.

KEY WORDS: bumblefoot, pododermatitis, raptor.

Bumblefoot (pododermatitis) is characterized by local injury to integument of the avian foot, usually the plantar metatarsal pad or digital pads, which leads to scab formation and infection of the internal tissues of the foot (Halliwell, 1975; Cooper, 1978; Halliwell and Graham, 1978; Riddle, 1980; Sawyer, 1983; Remple and Remple, 1987). Infection appears to be seeded by two routes: direct inoculation by puncture from a contaminated talon or foreign object, and devitalization of the epithelium (a barrier to infection) permitting microbial entry into underlying tissues. The second route appears to relate to trauma to the plantar integument in the form of unrelenting pressure or persistent contusion. Once the process begins, spontaneous resolution rarely occurs; instead, a degenerative process leads to eventual destruction of the affected foot.

Initially, bacterial coagulase protects some pathogens from cellular immune response, while fibrosis shields other pathogens later in the inflammatory reaction. In bumblefoot, there appears to be a weakened or inadequate heterophil response during the acute phase of the inflammatory reaction (Glick et al., 1964). Eventually, a chronic granulomatous inflammatory reaction insulates the pathogens from humoral and cellular immune responses and systemic antibiotics (Topp and Carlson, 1972).

Factors predisposing to bumblefoot are those that favor trauma to the feet and those that suppress immune competence. Certain hypovitaminoses, poor nutritional status, heavy parasite burden, existing disease, and extremes in environmental temperature for a given species are factors that are known to reduce avian immune competence (Ringer, 1976; Eisen, 1980a; Satterfield and O'Rourke, 1980; Kollias, 1986). Vitamin A deficiency can weaken the epithelial barrier to infection (Ringer, 1976; Halliwell and Graham, 1978; Muller, 1983; Sawyer, 1983; Kollias, 1986). Deficiencies of vitamin E can reduce vascular integrity and predispose to bruising and weaken immune function (Kollias, 1986). Biotin deficiency can produce a dermatitis that affects the plantar surface of the foot and predisposes to bumblefoot. Trauma predisposes to bumblefoot (Halliwell, 1975; Cooper, 1978, 1987), and raptors that make a hard landing on their feet (large falcons and tethered raptors kept on hard surfaces) risk foot trauma from simple concussion. Birds kept on hard, smooth surfaces develop pressure necroses, perhaps analogous to human "bed sores," over prominent plantar areas of the feet and digits. Smooth perching surfaces favor keratin buildup resulting from inadequate desquamation, which can initiate and aggravate pressure ischemia. Sharp, overgrown talons facilitate accidental puncture with inoculation of pathogenic microbes (Cooper, 1987).

The pathogen most frequently isolated from bumblefoot is *Staphylococcus aureus* (Cooper and Needham, 1976; Riddle, 1980; Sawyer, 1983; Cooper, 1987; Remple, 1987). Cooper and Needham (1976) report a 90% prevalence,

which coincides with the findings in this report. However, *Streptococcus* spp., *E. coli*, *Proteus* spp., *Pasteurella* spp., *Pseudomonas* spp., *Klebsiella* spp., *Clostridium* spp., *Corynebacterium* spp., *Bacillus* spp., *Diplococcus* spp., *Nocardia* spp., *Actinobacillus* spp., *Actinomyces* spp., and *Aeromonas* spp. have all been isolated (Remple and Remple, 1987).

Objectives

The objectives of this study were (1) to review the predisposing factors to raptor bumblefoot and examine their relationship to bacterial introduction into the integument, (2) to compare histological sections of normal feet with biopsies from various raptor feet representing all stages of disease evolution in order to gain better insight into the initiation and pathogenesis of disease, and (3) to consider the possibility of bacterial hypersensitivity as a mechanism of pathogenesis of the disease within the raptor foot.

Materials and Methods

Bacterial Identification

The bacteria recovered from infected feet were isolated by standard methods. Four sterile swabs were taken from the surgical site. One was used to make two cytology smears for Gram's staining and Wright's staining. One swab was used for anaerobic culture. Another swab was plated on blood agar, brilliant green-phenol red-lactose agar, and nutrient agar. Casio broth was inoculated from the fourth swab. Identification was made by usual microbiological methods. When Gram-positive cocci were isolated, the identification of *Staphylococcus aureus* was confirmed by the following tests: growth on Baird-Parker medium with the production of black colonies, positive coagulase test, agglutination of sheep red blood cells (srbc) on the Staphyslide[a] test, and the reduction of mannitol. The results of microbiological culturing are reported by Oaks (chapter 15, this volume).

Histology and Histopathology

Elliptical sections of healthy plantar integument were taken from one female peregrine falcon (*Falco peregrinus*) (Fig. 17.1) and one female saker falcon (*Falco cherrug*), which had both died of diseases unrelated to bumblefoot. Sections measured approximately 8 mm by 2 mm and were taken across the middle of the metatarsal pad in a plane extending from the hallux to the second digit. These biopsies displayed healthy dermal papillae and showed no gross evidence of traumatic insult. They were prepared as references to be used for comparison with biopsy sections from diseased feet.

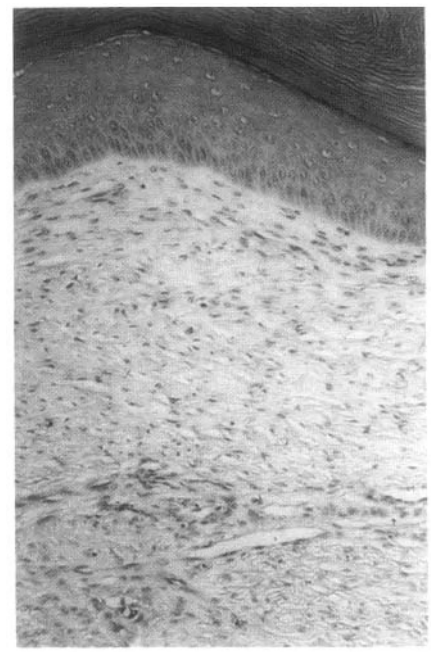

Figure 17.1. Normal foot integument for peregrine falcon. The apparent increase in the keratinized layer is normal, as the sample was taken over the metatarsal pad. Note the extensive capillary sinusoidal network within the superficial layer of the dermis.

Biopsies were taken from 15 affected feet representing all stages of disease evolution leading to thick scab formation and progressing to end-stage crippling disease. In all cases, incisions were made to incorporate all affected areas of disease (for example, scab, integument, subcutis, and inflammatory exudate) in the center of the sample so as to progress peripherally to relatively healthy integument. All surgical specimens were fixed in buffered formalin, paraffin embedded, and stained with hematoxylin and eosin, Gram's, Grocott's, and van Gieson stains.

Results

Histopathology

The 15 affected feet examined ranged clinically from early to late stages of disease. All cases showed ulceration of the skin. Ulcers were often covered by a laminated parakeratotic encrustation intermixed with acute inflammatory cellular exudate and heavily colonized by Gram-positive cocci. The adjacent epidermis showed hyperkeratosis (Fig. 17.2), patchy parakeratosis, and irregular acanthosis with prolongation of rete ridges or atrophic thinning. In early stages the dermis showed scanty perivascular cellular reaction, often consisting of a mixture of acute and chronic inflammatory cells; in the majority of cases the mononuclear inflammatory cells predominated. Several cases showed a perivascu-

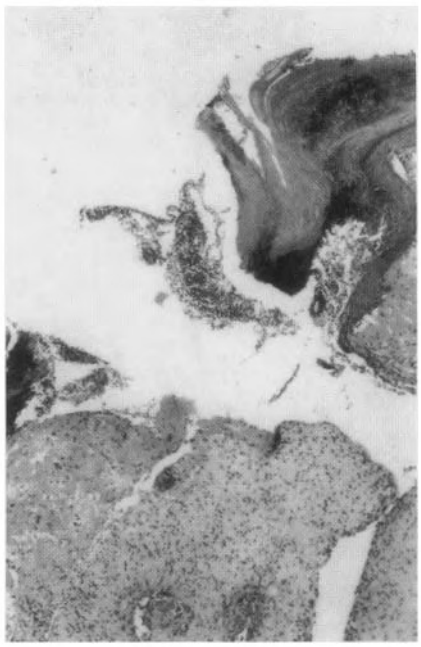

Figure 17.2. As a result of the underlying vascular insult, "punched-out" ulceration is seen within the epidermis of this peregrine falcon. There is hyperkeratosis and bacterial colonization within the fibrino-hemorrhagic crust. Within the dermis there is increased collagen and endothelial proliferation.

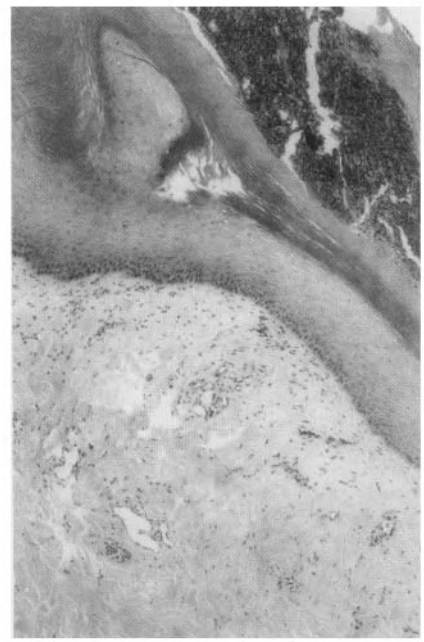

Figure 17.3. Advanced pathological change evident in the foot of a peregrine falcon. The epidermis has crust entrapped within the stratum corneum, acanthosis, and hyperkeratosis. The dermis shows advanced endarteritic obliterative vascular changes.

lar inflammatory infiltrate, edema, and degeneration of collagen, amounting to a necrotizing vasculitis. The inflammation affected the whole of the dermis and extended into the subcutis in most cases. In several cases, nonnecrotizing epithelioid and multinucleated, giant-cell granulomata were abundant.

Later stages of disease demonstrated a variable degree of diffuse and/or nodular hyperplastic fibrosis, an increase in dermal collagen and hyalinization, and sparse cellular inflammatory reaction. A remarkable feature in all cases examined was the nodular vascular proliferation/fibrous thickening, and endothelial and smooth-muscle hyperplasia with consequent luminal narrowing, representing endarteritic obliterative vascular changes (EOVC) (Fig. 17.3).

In summary, the histopathological features of the cases of raptor pododermatitis studied were as follows:

1. ulceration of the epidermis associated with acanthotic thickening and/or atrophic thinning
2. vascular injury, particularly within the capillary sinusoidal network of the superficial dermal stratum, presenting as perivascular inflammatory cellular infiltrate and/or necrotizing vasculitis and progressing to EOVC
3. scanty cellular inflammatory reaction within the dermis
4. heavy colonization by Gram-positive cocci at the level of superficial inflammatory crust and base of ulcer

Discussion

Histology

Avian skin has an epidermis and a dermis; however, the tissue organization warrants a different nomenclature from that of mammals (Sisson and Grossman, 1975). The epidermis consists of several layers. The stratum corneum is the outermost keratinized layer. The underlying living region of cells is divided into the stratum transitivum, a layer of flattened cells that lay down intercellular corneous laminae; a stratum intermedium, representing most of the germinal layer; and a stratum basale, which is a single layer of cells directly overlying the basement membrane. The basement membrane is a polysaccharide layer representing the dermoepidermal junction. The dermis is divided into the outermost stratum superficialis and the deep stratum profundum layers. The lamina elastica represents the basement of the dermis and delineates the boundary between dermis and subcutis. The epidermis tends to be thicker in specialized areas where pressure and friction are encountered, such as plantar pads. The connective tissue of the superficial dermal layer is packed with sinus capillaries, small capillaries, and other vessels. Sinus capillaries may have smooth muscle cells close to the endothelium. The elastic lamina may vary in thickness depending on the amount of collagen present.

Bumblefoot in Captivity

In recent studies one question continually arises: Why is raptor bumblefoot implicated as primarily a disease of captive raptors? Wild free-living raptors suffer from foot infections as well, but the incidence is lower. It is generally thought that cases seen in wild raptors appear to relate to inoculation of pathogens through mechanical injury, traps, foreign-body puncture, and the like, and the pathogens isolated are primarily nonstaphyloccal bacteria. One suggested explanation for the differences is that although wild birds are subject to harsher conditions, they have more control over landing impact than do tethered captives. Wild raptors also have a choice of perching surfaces with a variety of textures to stimulate the plantar integument and aid natural keratin debridement. Lastly, *Staphylococcus aureus*, the primary pathogen of captive raptor bumblefoot, has not been shown to be a common contaminant of wild raptor feet (Cooper and Needham, 1976; Satterfield and O'Rourke, 1980; Needham, 1981). This suggests that disease in captive raptors may arise from pathogen transfer from human handler to raptor (Satterfield and O'Rourke, 1980; Needham, 1981). It is possible that raptors are poorly adapted to human strains of *Staphylococcus aureus*, and that continual presentation of these strains may attenuate the immune function or elicit inappropriate immune responses within the avian foot (Satterfield and O'Rourke, 1980).

In this study, when the two routes to infection could be distinguished, devitalization of the integument consistently favored the recovery of *Staphylococcus* sp. either in pure culture or mixed infection, whereas puncture inoculation favored the recovery of other pathogens. This may imply a special route of infection for *Staphylococcus* sp. in the raptor foot.

Obstacles to Resolution

Advanced stages of both routes of infection (puncture inoculation and devitalization of integument) show plantar metatarsal scabs and can be identical in appearance; however, early stages show differences. Grossly, the puncture-related cases show a small healed hole (punctate scab) with thickened skin around the periphery of the lesion. Initially the surrounding skin displays normal color and capillary perfusion, is elastic, and shows good definition of plantar papillae. Early cases associated with integument devitalization present in a different way. Initially there appears to be a more generalized reaction involving the skin of a prominent area. At a very early stage the skin appearance may range from hyperemia, occasionally showing petechiation, to blanching with poor capillary perfusion. The early stages give way to a red-purple to brownish discoloration (reflecting capillary damage), hardening (loss of elasticity), and a gradual loss of plantar papillary definition as an early interpapillary crust begins to form. Later the crust may regress peripherally, as skin margins attempt to heal, but thicken centrally as degenerative processes continue. The result is a scab that penetrates the skin and exposes underlying tissues to infection.

The plantar scab is, by itself, a serious obstacle to healing. The pressure it exerts on tissues at the scab-tissue interface, compounded by the concussion of normal movement, causes ischemia and disrupts healing at lesion margins (Cooper, 1978; Riddle, 1980; Remple and Remple, 1987). Defects at the scab-tissue interface provide a portal of entry for contaminant pathogens into an existing lesion (Cooper, 1987; Remple and Remple, 1987).

In considering the microscopic obstacles to resolution of infection, it is necessary to look at the primary pathogen and the host immune response. The usual fate of unsuccessful bacteria is death by phagocytosis; therefore, survival entails avoidance of this fate. Successful pathogenic bacteria can kill phagocytic leucocytes, create insulating barriers against phagocytic cells, resist phagocytosis through attenuation of opsonization or other immune responses, and resist digestion and processing by phagocytic cells and thus survive within them (Playfair, 1982). *Staphylococcus aureus* can do all of the above.

The enzyme coagulase is utilized by *Staphylococcus aureus* in its resistance to phagocytosis. The formation of a fibrin barrier that is catalyzed by coagulase insulates the pathogen from phagocytosis and assists in the survival of some cocci after the acute inflammatory response subsides. Early fibrotic encapsulation can further insulate surviving pathogens from cellular immune responses and systemic antibiotics within 3 to 5 days of initial trauma (Cooper, 1978, 1987).

At present there is little doubt about the ability of avian heterophils to ingest *Staphylococcus* spp.; however, phagocytic activity and enzymatic degradation of antigen vary considerably between artificial and true inflammatory environments. Certain studies conducted *in vitro* have demonstrated that heterophils readily phagocytose *Staphylococcus* spp. and enzymatically degrade it within the heterophil phagosome (Topp and Carlson, 1972). However, the *in vivo* behavior of heterophils in the presence of *Staphylococcus* spp. appears to be markedly reduced (Glick et al., 1964; Carlson and Allen, 1969). This suggests a phagocytic paralysis that may result from staphylococcal toxins or a vague host immunosuppressive response somehow induced by the presence of the pathogen. Staphylococcal protein A, which binds to the F_c portion of host immunoglobulins, thereby preventing opsonization, may play a role in weakened phagocytic responses (Playfair, 1982).

An important disease in humans, which has not yet been reported in animals, relates to an inefficiency in neutro-

philic phagocytosis of organisms such as *Staphylococcus* spp. and coliforms. The granulocytic respiratory burst fails to occur, and the bactericidal efficiency is greatly impaired. The syndrome leads to chronic granulomatous disease (Tizard, 1987a). Impairment of the ability of raptor heterophils to phagocytose and degrade *Staphylococcus* sp. was strongly suggested in this study. Tissue sections from infected feet showed intact and degranulating heterophils in close contact with *Staphylococcus* spp., which apparently have had little or no observable effect on the cocci. The work of Oaks (chapter 15, this volume) supports the above finding. The relative inefficiency of raptor heterophils to phagocytose and degrade *Staphylococcus* sp. and possibly other pathogens commonly found in bumblefoot helps to explain the tendency of raptor bumblefoot to become a chronic granulomatous disease. This study and others have shown no significant differences in lesions produced by *E. coli* and *Proteus* spp. compared with *Staphylococcus aureus* within the avian foot (Cooper, 1987); decreased or weakened heterophil responses may account for this finding.

Studies of the cellular response indicate that the macrophage may be the predominant responding cell in avian staphylococcal disease, and suggest it may be instrumental in both resistance and pathogenicity (Satterfield and O'Rourke, 1980). After the heterophil response, monocytes and tissue macrophages are attracted to phagocytose antigen and debris. However, the ability of *Staphylococcus* spp. to survive in activated phagocytic cells leads to the continual recruitment of phagocytes, lymphocytes, and necrotic debris, all of which become circumscribed by mature macrophages (epitheloid cells and multinucleated giant cells). The continual activation leads to extensive tissue destruction and fibrin deposition. The resulting chronic granuloma shields *Staphyloccocus* spp. from specific immune factors and systemic antibiotics (Turk and Stamm, 1970).

Bacterial Hypersensitivity

Two routes of infection for bumblefoot have been discussed: inoculation and devitalization of the epithelial barrier to infection. In regard to devitalization, initial tissue damage is thought to relate more to an immune-mediated inflammatory response than to direct bacterial infection. The paucity of cellular inflammatory reaction within the dermis in the majority of cases examined, combined with the remarkable vascular changes observed, has led to the consideration of avian bacterial hypersensitivity as a likely cause of the pathological changes seen in this disease. The introduction of *Staphylococcus aureus* into the integument may initiate hypersensitivity reactions, with considerable cellular damage (Turk and Stamm, 1970; Scott et al., 1978; Satterfield and O'Rourke, 1980; Tizard, 1987b). Vasculitis is a prime feature of hypersensitivity reactions. The pathomechanism of most cutaneous vasculitides is assumed to involve Type III hypersensitivity reactions (Arthus) (Scott et al., 1978; Muller, 1983; Tizard, 1987b), and in such reactions, punched-out ulceration of the epidermis along associated vascular pathways is seen (Muller, 1983).

Multifactorial insults to the skin such as repeated trauma, malnutrition including hypovitaminosis A, and various stressors may act singly or in combination to provide pathogens, especially *Staphylococcus aureus*, with an opportunity to sensitize and colonize the skin. Damaged and compromised avian skin is a favorable and enhanced medium for bacterial growth (Ramdy and Barton, 1965). Such an environment favors the induction of hypersensitivity to bacterial antigens. In addition, this study showed the colonization of *Staphylococcus aureus* in pockets of fibrinohemorrhagic crust between the stratum corneum and stratum transitivum of plantar podal integument. It is possible that dermal sensitization could occur through antigen presentation by Langerhans cells within the epidermis or by antigens gaining access to the dermis through small breaks in the epidermis.

The local Arthus hypersensitivity reaction involves the formation of antigen-antibody complexes within tissues that activate the complement system. Antigen finding its way into tissues, by whatever mechanism, diffuses through tissue spaces toward small blood vessels, where it comes into contact with circulating antibody. Consequently, immune complexes are formed and deposited in proximity to endothelial cells. These complexes activate complement resulting in granulocyte accumulation, thrombocyte aggregation, and release of anaphylatoxins and vasoactive amines. During the process, large quantities of granulocytic enzymes are released into the tissues and cause damage. Enzyme release occurs through a number of processes, the most obvious of which is the death of granulocytes. Of greater importance in the Arthus reaction is the attachment of immune complexes to a nonphagocytosable structure such as a basement membrane, which results in granulocyte granular contents being secreted directly into surrounding tissues. The aggregating thrombocytes release procoagulants, which, in conjunction with the vascular damage, results in extensive thrombosis. Thrombosis and vascular damage cause impeded blood flow and hemorrhage into adjacent tissues. Localized patches of necrosis appear in small vessels (Eisen, 1980a; Wells, 1980; Tizard, 1987b). The vascular insult may eventually terminate in EOVC. The degenerative sequence of events appears to relate to the vascular narrowing and consequent ischemia. As a result, punched-out ulceration of the epidermis occurs along associated vascular pathways as well as degeneration and hyalinization of dermal collagen, ultimately leading to atrophy of the dermis (Muller, 1983).

Role of the Immune Response

The possibility that an avian Arthus reaction is involved in bumblefoot is based on two assumptions: first, that heterophils, and possibly thrombocytes, can produce tissue damage through release of granular enzymes similar to that caused by mammalian neutrophils; and second, that sufficient antibody is generated through previous exposure to result in the formation of immune complexes when reexposure to antigens occurs. An important enzyme within the neutrophil lysosome is myeloperoxidase. The release of myeloperoxidase from neutrophilic degranulation damages tissues (Eisen, 1980a; Wells, 1980; Tizard, 1987b). Although heterophils lack myeloperoxidase, they are capable of efficient intracellular microbiocidal activity through the activity of several enzymes (Topp and Carlson, 1972; Satterfield and O'Rourke, 1980). Avian thrombocytes are capable of ingesting *Staphylococcus* spp. also (Topp and Carlson, 1972), and possess lysosomes (Carlson and Allen, 1969). If heterophil enzymatic release and/or thrombocyte lysozyme release can produce tissue damage, there is every reason to assume that birds of prey can elicit Arthus-type hypersensitivity reactions. Further clinical studies are needed for confirmation.

The possibility of delayed-type hypersensitivity (DTH) to *Staphylococcus aureus* has been discussed elsewhere (Satterfield and O'Rourke, 1980). The Type IV or DTH response is cell mediated and occurs independent of antibody. T-lymphocytes play a central role in mediating immune responses. DTH occurs when T-lymphocytes are stimulated by repeated exposure to antigen, with a T-cell subpopulation, the T_{DTH} lymphocytes, being sensitized (Eisen, 1980b; Wells, 1980; Wing, 1980). T_{DTH} cells respond to further antigen contact with gamma interferon release, which attracts macrophages and stimulates them to increase their phagocytic and kill potential. Activated macrophages lack specificity, and their lysozyme spillover causes tissue damage. DTH is generally slower to appear than Arthus-type hypersensitivity, maximizing at 24 to 72 hr and then gradually regressing (Eisen, 1980b; Wing, 1980; Tizard, 1987b).

There is evidence that DTH can follow an Arthus reaction, or that Arthus and DTH can occur simultaneously after a single antigenic stimulation in the so-called bimodal reaction (Eisen, 1980b). Also, severe Arthus reactions not only overlap but can mimic DTH, producing a reaction that peaks at 24 to 72 hr, turns red-purple in the skin, necroses, and ulcerates (Scott et al., 1978). The gross and histopathological distinction between the two is obscure, and differentiation requires serum and lymphocyte transfer to naive recipients. From a clinical point of view, the distinction between the two may seem arbitrary—the point being that, with each exposure to antigen, an acute inflammatory reaction occurs, even in the absence of true infection. Although separate pathways are taken in each response, the end result is the same. The sensitized dermis, with its damaged capillary sinusoidal vasculature, reacts with a degenerative sequence of events eventually culminating in EOVC.

Conclusion

Devitalization of the integumental barrier to infection possible through Type III or Type IV hypersensitivity, or both, could provide an ideal portal of entry for infection to become established in underlying tissues. Further study is needed. Once seeded, by whatever mechanism, bacterial infection of the raptor foot tends to become chronic and granulomatous.

Bumblefoot appears to be more than a simple bacterial disease. There is much to be learned, and it is our hope that this chapter will stimulate further study of this complex disease.

Acknowledgments

We are grateful to Dr. Lindsay Oaks, who commented on earlier drafts of this chapter and who assisted in the surgical treatment and compilation of data. The first author also wishes to thank Cheryl Remple and Dr. Ulrich Wernery for their assistance in the microbiological analysis.

Product List

[a]Staphyslide Test, BioMerieux, Marcy-L'Etoile, 69260 Charbonnieres-Les-Bain, France

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CHAPTER 18

Diagnosis of Brachial Plexus Avulsion in Three Free-living Owls

STEPHEN A. SMITH, D.V.M., Ph.D.

Department of Pathobiology
Virginia-Maryland Regional College of Veterinary Medicine
Virginia Polytechnic Institute and State University
Blacksburg, Virginia, U.S.A.

ABSTRACT: Avulsion of the roots of the brachial plexus has been reported in both companion animals and birds. This study reports the use of electrodiagnostic techniques in the evaluation of three cases of brachial plexus avulsion in free-living owls. The use of electromyography and nerve-conduction studies allowed accurate diagnosis of the avulsion and an earlier determination of a poor prognosis in the birds.

KEY WORDS: electrodiagnosis, avulsion, great horned owl, *Bubo virginianus*, screech owl, *Otus asio*, raptor.

Avulsion or tearing of the roots of the brachial plexus is a well-established clinical syndrome in companion animals (deLahaunta, 1977, p. 340). Recently, the same condition has been described in birds (Smiley et al., 1988). In the latter cases, each bird was presented with pronounced atrophy of the muscles and paralysis of the affected limb. These signs, plus failure to regain function, were used to arrive subjectively at a presumptive diagnosis. In those cases a definitive diagnosis of a brachial plexus avulsion could not be confirmed until necropsy and histopathology were performed.

This chapter reports the use of electrodiagnostic studies in the evaluation of a brachial plexus avulsion in three free-living adult owls. Use of these techniques permits early diagnosis of brachial plexus avulsion in cases presented soon after injury, where atrophy of the limb musculature cannot be used as a diagnostic indicator.

Methods and Results

Two adult great horned owls (*Bubo virginianus*) and one adult eastern screech owl (*Otus asio*) were presented to the Raptor Rehabilitation Program at Ohio State University. The owls were found unable to fly and exhibited unilateral wing droop and paralysis.

Upon initial examination, the first great horned owl showed depression, lethargy, and an unresponsive drooping left wing. There was a slight ipsilateral ptosis to the left eye, along with paralysis of the left ear tuft (Fig. 18.1) and several spots of hemorrhage along the upper lid margins of both eyes. The remainder of the physical examination was normal, with no palpable fractures and no other signs of traumatic injury to the head or body. Each eye had a normal palpebral response and the owl responded appropriately to visual stimulation of each eye. The pupils were symmetrical and had normal direct pupillary light responses. The bird appeared to be in good flesh.

Figure 18.1. Ptosis of the left eyelid and paralysis of the left ear tuft in the first great horned owl.

The second great horned owl and the screech owl both had drooping and unresponsive right wings. Physical examination of each of these owls was otherwise unremarkable. None of the three owls demonstrated voluntary movement in the affected wing. Reflex withdrawal of the affected wing to noxious stimuli was not observed in any of the owls, nor was there any evidence of conscious perception to stimulus being applied to the affected wing.

Initial therapy in all three birds was similar and consisted of securing the affected wing to the body. The wings were checked daily for signs of edema or circulatory impairment and were rewrapped at 1 wk postpresentation. When the wing wraps were removed at 2 wk postpresentation, the affected wings were still drooping and unresponsive. In each

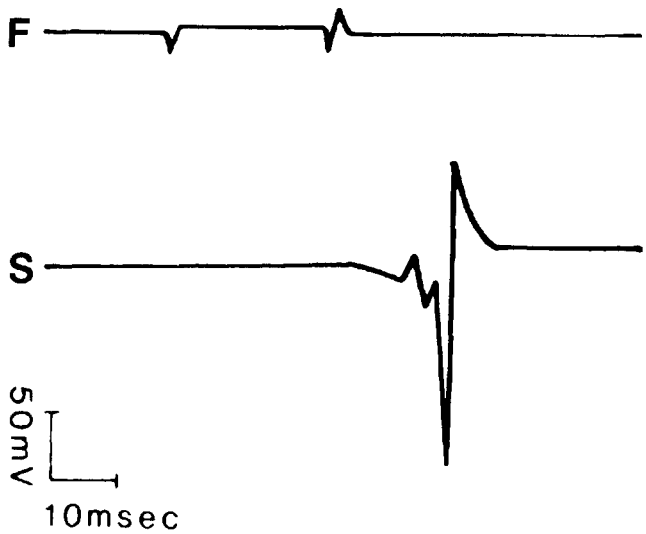


Figure 18.2. Representative tracing from electromyograph of the first great horned owl, showing positive sharp waves (S) and fibrillation potentials (F).

of the birds, there was noticeable atrophy of the intrinsic muscles of the affected wing, as well as atrophy of the ipsilateral pectoral muscles. The ipsilateral ptosis and ear-tuft paralysis were also still present in the first great horned owl.

Based on the clinical findings in each bird, a tentative diagnosis of an avulsion or tearing of the nerve roots of the brachial plexus was made, and electrodiagnostic studies were performed on all three birds.

The electrodiagnostic examination was performed under anesthesia using 20 mg/kg ketamine hydrochloride[a] injected IM (Redig, 1983). A needle electromyograph[b] (EMG) was performed on the muscles of the thoracic and pelvic limbs in all three birds, using techniques previously described for the dog (Chrisman et al., 1972; deLahaunta, 1977; Steinberg, 1979a). In the first great horned owl, normal insertional activity was seen in both pelvic limbs and in the right thoracic limb, and no abnormal potentials were seen in the muscles. The muscles of the left thoracic limb showed prolonged insertional activity, with persistent positive sharp waves and fibrillation potentials (Fig. 18.2). No normal motor-unit potentials were seen in the muscles of the left side of the thorax of the owl. These findings suggested widespread denervation of the left thoracic limb (Steinberg, 1979b).

Nerve-conduction studies were done on the right and left radial nerves using a technique previously described for the dog (Griffiths and Duncan, 1978; Steinberg, 1979a). The right radial nerve displayed a normal motor unit potential with an amplitude of 750 μv and a latency of 3.1 msec on stimulation, while no conduction could be elicited in the radial nerve of the left thoracic limb. Based on these findings, a poor prognosis for return of function in the left wing was given, and the bird was euthanized.

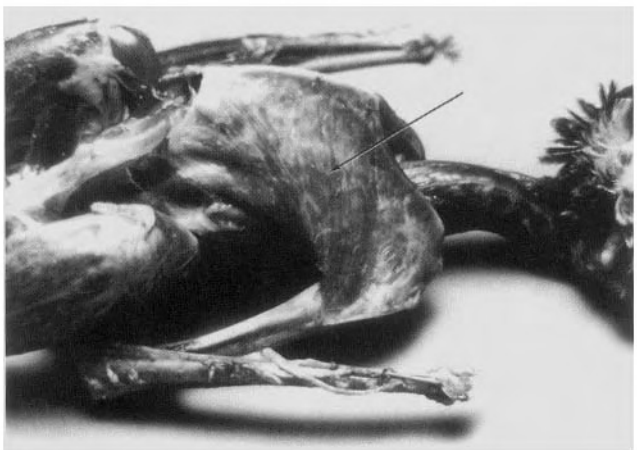
In the EMG of the second great horned owl, normal insertional activity was seen in both pelvic limbs and in the left thoracic limb, and no abnormal potentials were seen in the muscles. The muscles of the right thoracic limb showed prolonged insertional activity, with persistent positive sharp waves and fibrillation potentials. No normal motor-unit potentials were observed in the right thoracic limb. These findings suggested widespread denervation (Steinberg, 1979a). Nerve-conduction studies were performed on this owl, and again no radial nerve conduction could be elicited in the affected limb. Based on these findings, a poor prognosis for return of function in the affected wing was given. The affected wing was amputated and the bird placed in an educational program.

The EMG of the screech owl revealed a similar pattern of abnormal activity in the right wing and scapular muscles. All other appendicular and pectoral muscles in the screech owl were normal. These changes were consistent with widespread denervation of the affected limb. A nerve-conduction study was not performed on this bird, and the bird was euthanized.

Postmortem examination was conducted immediately following euthanasia of the first great horned owl and the screech owl. In both owls, gross necropsy revealed extensive diffuse unilateral atrophy of the pectoral, brachial, and antebrachial muscles of the affected wings (Fig. 18.3). The muscles of the unaffected side of the thorax, back, and pelvic limbs were unremarkable. The atrophic muscles were pale, with a streaked appearance, and were fibrous rather than fleshy in texture. Examination of the peripheral nerves revealed unilateral brownish discoloration and reduction in size of the nerves of the brachial plexus on the affected side in each bird (Fig. 18.4). Further dissection in the first great horned owl demonstrated involvement of the last two cervical and first thoracic roots, which were reduced to thin fibrous bands. Other tissues were unremarkable.

Microscopic findings in the first great horned owl confirmed those seen grossly. There was complete atrophy of both the ventral and dorsal roots of the left brachial plexus, with the affected spinal roots displaying a severe reduction in size and consisting of well-differentiated spinal cells without axons. The dorsal-root ganglion of the accessory brachial plexus had also degenerated. A collar of dense fibrous connective tissue surrounded the ventral roots in some sections. Microscopic evaluation of muscle sections confirmed the presence of skeletal muscle atrophy of neurogenic origin, as characterized by groups of small angular fibers, increased numbers of sarcolemmal nuclei, and focal infiltration of adipose tissue.

Thus, histopathological examination confirmed that peripheral nerve injury to the nerve roots of the left brachial plexus had occurred in the first great horned owl. Similar



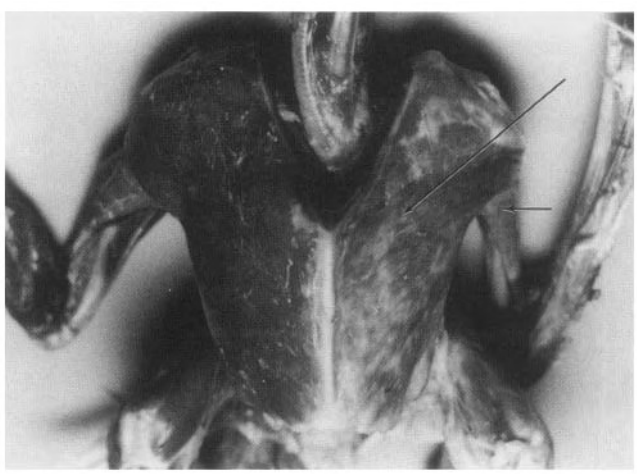


Figure 18.3. Macroscopic appearance of the first great horned owl at postmortem, showing unilateral atrophy and discoloration of both the left pectoral muscles and left wing muscles (*arrows*).

histopathological findings were observed in the screech owl.

Discussion

The anatomy of the brachial plexus is complex. The avian brachial plexus varies in both its location and the number of contributory nerve branches due to the variable number of cervical vertebrae among avian species (Bubien-Waluszewska, 1985). In general, the nerve roots contributing to the brachial plexus consist of the ventral nerve roots of four or five spinal nerves. In the raptors studied so far, these include spinal nerves 12 through 16—in conventional veterinary nomenclature, cervical spinal nerves 12-15 (C12-C15) and the first thoracic spinal nerve (T1). In some raptors, such as species in the genera *Buteo* and *Bubo*, there also exists an accessory brachial plexus, which consists of contributions from two or three of the caudal-most dorsal nerve

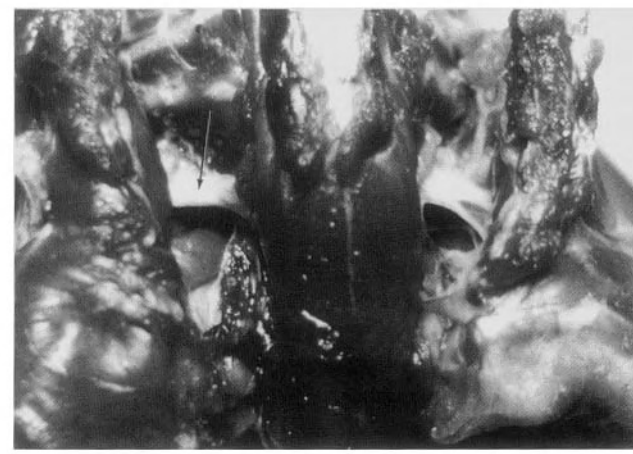


Figure 18.4. Dorsal view of the brachial plexus of the first great horned owl showing severe reduction in size and discoloration of the involved nerves (*arrow*) compared with those of the opposite normal side.

roots of the main brachial plexus (Baumel et al., 1979, pp. 490, 498; Bubien-Waluszewska, 1985).

In the great horned owl the major contributions to the brachial plexus consist of ventral and dorsal nerve roots 11 through 14, or spinal nerves C11-C13 and T1 (Fig. 18.5). It is hypothesized that an injury that displaces the shoulder and scapula caudally, putting excessive tension on the nerves of the brachial plexus, or an injury that causes severe abduction of the scapula and thoracic limb, may tear the roots of the brachial plexus from the spinal cord (Steinberg, 1979b). Thus, the most likely scenario to produce a brachial plexus injury in a flying bird would be flying into a stationary object such as a power line or a branch of a tree, or being struck by a moving vehicle.

Electrodiagnostic studies, such as electromyography and nerve-conduction studies, are useful in evaluating the electrical activity of a motor unit. Insertional activity is defined as electrical activity produced in a muscle cell due to excitation or mechanical irritation of the cell. This may be abnormally prolonged in cases of denervated muscle in which the muscle cells become hypersensitive to stimulation (Steinberg, 1979a). Persistent positive sharp waves are defined as electrical potentials that occur repeatedly in denervated or diseased muscle. Fibrillation potentials, which are usually seen in conjunction with positive sharp waves, generally indicate an advanced state of disease within the motor unit (Chrisman et al., 1972). The above findings, together with the absence of motor-unit potentials in the nerve-conduction studies of the affected limbs of the birds examined in this study, indicated severely denervated muscle in each of the birds and dictated a poor prognosis for return of function to the injured limbs.

The major advantage of using electrodiagnostic techniques is that severe neurological dysfunction, in this case

Figure 18.5. Dissected dorsal view of a normal brachial plexus of a great horned owl (*arrow*).

the denervation of the wing caused by an avulsion or tearing of the nerve roots of the brachial plexus, can be accurately diagnosed as early as 1 or 2 wk postinjury (Steinberg, 1979b). Therefore, a definitive diagnosis and a resulting prognosis can be made early and accurately without maintaining the bird in a medical facility for an extended period of time.

Although the signs of avulsion of the roots of the brachial plexus are predominantly those of radial nerve paralysis at the level of the shoulder, multiple nerve deficits may be seen (deLahaunta, 1977). In the first great horned owl, the signs of radial nerve paralysis were manifested as complete inability to extend the wing distal to the shoulder. Additionally, analgesia from the mid-humeral area distally and a partial Horner's syndrome were exhibited. Recommended therapy for an avulsion of the brachial plexus in domestic pet animals is usually amputation to prevent passive or self-induced trauma to the affected limb. Similar treatment of raptors renders them nonreleasable, and since two of the owls could not be placed in a suitable educational program, the birds were euthanized.

Acknowledgments

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Product List

[a]Ketaset, Aveco Co. Inc., P.O. Box 717, Ft. Dodge, Iowa 50501 USA

[b]Teca-B-2 Electromyograph, Teca Corporation, 3 Campus Dr., Pleasantville, New York 10570-1698 USA

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CHAPTER 19

Herpesvirus Disease in Raptors: A Review of the Literature

COLETTE L. WHELER, B.Sc., D.V.M., M.Vet.Sc.

Department of Veterinary Anesthesiology, Radiology and Surgery
Western College of Veterinary Medicine
University of Saskatchewan
Saskatoon, Saskatchewan, Canada

ABSTRACT: Several avian herpesviruses cause disease characterized predominantly by multifocal necrosis of the liver, spleen, and other organs with formation of intranuclear inclusion bodies. These include the herpesviruses of raptors, psittacines, pigeons, cranes, quail, and storks. Other known avian herpesviruses cause predominantly hemorrhagic or neoplastic disease, or no disease at all. This chapter reviews those herpesviruses causing hepatosplenitis, with particular emphasis on herpesvirus disease in raptors. Serological relationships are summarized, and implications for birds of prey are discussed.

KEY WORDS: herpesvirus, hepatosplenitis, virus or viral disease, raptor.

Herpesviruses are known to cause disease in mammals, birds, reptiles, amphibians, and fish (Roizman et al., 1981). The herpesvirus family is divided into three subfamilies, the alpha-, beta-, and gammaHerpesvirinae, based on biological properties of the virus, such as host range, duration of reproductive cycle, cytopathology, growth in tissue culture, and characteristics of latent infection (Roizman et al., 1981). Subfamily classification of the known avian herpesviruses is summarized in Table 19.1 (Kaleta, 1990). Viruses in the alpha- and gammaHerpesvirinae subfamilies are well characterized, probably due to commercial interest and economic necessity (Kaleta, 1990). The betaHerpesvirinae subfamily contains most of the remaining avian herpesviruses, some of which have tentatively been included pending more complete characterization (Kaleta, 1990). Several other herpesviruses isolated from passeriform birds remain, as yet, unclassified (Kaleta, 1990).

Table 19.1
Subfamilies of avian herpesviruses

AlphaHerpesvirinae	*BetaHerpesvirinae*	*GammaHerpesvirinae*
Anatid HV 1	Psittacid HV 1	Gallid HV2
Gallid HV 1	Psittacid HV 2	Meleagrid HV 1
	Psittacid HV 3	
	Columbid HV 1	
	Columbid HV 2	
	Strigid HV 1	
	Falconid HV 1	
	Acciprid HV 1	
	Phalacrocoraid HV 1	
	Gruid HV 1	
	Perdicid HV 1	
	Ciconiid HV 1	

Source: Kaleta (1990).

Apart from the subfamily designations, avian herpesviruses have also been divided into 11 different serogroups (Table 19.2) on the basis of cross-neutralization using either plaque reduction or micro-neutralization tests (Kaleta, 1990). The viruses in the first three groups cause disease of a hemorrhagic or neoplastic nature (Gallid HV 1 and 2,

Table 19.2.
Serogroups of avian herpesviruses

Serogroup[a]	*Herpesvirus*[b]	*Disease*
1	Gallid HV 2	Marek's disease
	Meleagrid HV 1	none
2	Anatid HV 1	duck viral enteritis
3	Gallid HV 1	infectious laryngotracheitis
4	Psittacid HV 1	Pacheco's disease
5	Psittacid HV 2	Pacheco's desease
6	Psittacid HV 3	Pacheco's disease
7	Columbid HV 1	inclusion body hepatitis ± splenitis
	Strigid HV 1	inclusion body hepatitis ± splenitis
	Falconid HV 1	inclusion body hepatitis ± splenitis
	Acciprid HV 1	inclusion body hepatitis ± splenitis?
8	Columbid HV 2	inclusion body hepatitis
9	Phalacrocoraid HV 1	none?
10	Gruid HV 1	inclusion body hepatitis ± splenitis
	Perdicid HV 1	inclusion body hepatitis ± splenitis?
11	Ciconiid HV 1	inclusion body hepatitis ± splenitis

Sources: Kaleta (1990), Kaleta, Heffels, et al. (1980), Kaleta, Marschall, et al. (1980).
[a]Numbering does not imply official terminology.
[b]Names proposed by Kaleta (1990).

Anatid HV 1), or are otherwise of importance to the commercial poultry industry (Meleagrid HV 1), and will not be discussed here (Kaleta, 1990). The Phalacrocoraid HV 1 is included for completeness; however, it is not known to cause disease. This virus was isolated in 1951 from a single nestling cormorant (*Phalacrocorax melanoleucos*) on Lake Victoria, Australia, during routine blood screening. It is unrelated serologically to any of the other avian herpesviruses and is not pathogenic for budgerigars (*Melopsittacus undulatus*) (French et al., 1973).

Nonraptor Herpesviruses Causing Hepatosplenitis

The remaining avian herpesviruses cause disease characterized predominantly by multifocal hepatosplenitis, although other lesions are often seen (Kaleta, 1990). The Ciconiid HV 1, isolated from captive black storks (*Ciconia nigra*), causes an inclusion body hepatosplenitis very similar to that seen in owls, but is not serologically related to the owl or other avian herpesviruses (Kaleta, Mikami, et al., 1980). Gruid HV 1, affecting cranes, and Perdicid HV 1 from quail are related serologically to each other (Foerster et al., 1989). Affected cranes have been recognized only in captivity (Docherty and Henning, 1980; Foerster et al., 1989). Clinical signs include isolation, depression, diarrhea, and hock sitting. Mortality is 35-100%, depending on the species of crane; death occurs in 2 days (Docherty and Romaine, 1983; Foerster et al., 1989). Pathological findings include multifocal necrosis of liver, spleen, kidney, and sometimes thyroid gland and bone marrow, diphtheritic membrane in the gastrointestinal tract, and intranuclear inclusion bodies of Cowdry type A near necrotic foci (Burtscher and Grünberg, 1979; Foerster et al., 1989). Disease is exacerbated by stress, overcrowding, poor hygiene, and interaction of different crane species (Docherty and Romaine, 1983). The serologically related quail virus, Perdicid HV 1, was isolated from bobwhite quail (*Colinus virginianus*) with concurrent *Clostridium colinum* infection (Kaleta, Marschall, et al., 1980). Additional information on the disease-causing potential of this virus is not yet available in the literature.

Three different herpesviruses have been isolated from psittacines showing multifocal necrosis of the liver with intranuclear inclusion bodies and other variable lesions (Rivers and Schwentker, 1932; Findlay, 1933; Burtscher and Sibalin, 1975; Simpson et al., 1975; Durham et al., 1977; Trapp et al., 1977; Hirai et al., 1979; Miller et al., 1979; Randall et al., 1979; Krautwald et al., 1988; Kaleta, 1990). These include Psittacid HV 1 (Pacheco's disease) and Psittacid HV 2 and 3 (Krautwald et al., 1988; Kaleta, 1990). Pacheco's disease was first described in 1931 in Brazil and was initially thought to be a form of psittacosis, since the pathological lesions were somewhat similar (Rivers and Schwentker, 1932). Further work performed in the United States, however, demonstrated the etiological agent to be a new virus of psittacines, causing focal necrosis of the liver and formation of intranuclear inclusion bodies (Rivers and Schwentker, 1932). In 1975, this disease was seen in parrots in Florida, and solid evidence identified the agent as a herpesvirus (Simpson et al., 1975). Kaliner (1975) reported multifocal necrosis and intranuclear inclusion bodies in a parrot from Kenya. In 1977, the first case of Pacheco's disease was reported in Michigan, and its similarity to herpesvirus disease of pigeons, falcons, and owls was noted (Trapp et al., 1977). The disease was reported in New Zealand in 1977, and in Scotland, Texas, and New York in 1979 (Durham et al., 1977; Hirai et al., 1979; Miller et al., 1979; Randall et al., 1979).

Clinical signs seen with Pacheco's disease include nasal discharge, anorexia, diarrhea, sneezing, coughing, lethargy, weight loss, neurological signs, or simply sudden death without obvious clinical signs (Rivers and Schwentker, 1932; Kaliner, 1975; Simpson et al., 1975; Cooper and Howard, 1977; Trapp et al., 1977; Miller et al., 1979; Randall et al., 1979). Death occurs 12-14 days after contact with infected birds (Simpson et al., 1975). Pathological lesions consist of focal necrosis in the liver and intranuclear inclusion bodies in affected cells (Rivers and Schwentker, 1932; Simpson et al., 1975; Trapp et al., 1977; Randall et al., 1979). Splenic, renal, pancreatic, intestinal, and neurologic lesions can also be seen (Simpson et al., 1975; Randall et al., 1979). In a review of the disease in 1977, Cooper and Howard concluded that it was present worldwide; only psittacines seemed susceptible; young, stressed birds were often the victims; and, in view of the known epidemiology, asymptomatic carriers were likely.

Recently, two additional herpesviruses have been recovered from psittacines with multifocal necrosis of liver and spleen and other variable pathological changes (Krautwald et al., 1988; Kaleta 1990). Results of cross-neutralization tests show the three psittacine herpesviruses, Psittacid HV 1, 2, and 3, to be serologically distinct from each other (Krautwald et al., 1988; Kaleta, 1990).

Two herpesviruses are known to infect pigeons, Columbid HV 1 and 2 (Kaleta, 1990). Very little information is available in the literature regarding Columbid HV 2 (Kaleta, 1990). Unlike the previously mentioned avian herpesviruses, Columbid HV 1 is serologically related to the three raptor herpesviruses, Strigid HV 1, Falconid HV 1, and Acciprid HV 1 (Boyle and Binnington, 1973; Maré and Graham, 1973a, 1973b; Purchase et al., 1973; Cho and McDonald, 1980; Kaleta, Heffels, et al., 1980; Kaleta, Marschall, et al., 1980; Kaleta, 1990).

Columbid HV 1 was first described in 1945, when a new viral disease causing focal necrosis of spleen, liver, and pan-

creas and formation of intranuclear inclusion bodies was seen in pigeons in the United States (Smadel et al., 1945). As in parrots, this disease was initially thought to be caused by the "psittacosis virus" (Smadel et al., 1945). Several pigeons were in fact concurrently infected with the two agents (Smadel et al., 1945). In 1967, another colony of pigeons in the United States was affected with an intranuclear inclusion disease, showing concurrent focal necrosis of liver, spleen, and pancreas. At about the same time, a herpesvirus was isolated from diseased birds in Scotland (Lehner et al., 1967; Cornwell et al., 1967). In the early 1970s, reports of this disease from Australia began to appear, and it is now known to occur worldwide in both domestic and free-ranging pigeons (*Columba livia*) (Ward et al., 1971; Boyle and Binnington, 1973; Surman et al., 1975; Saik et al., 1986). In pigeons, the clinical signs can include conjunctivitis, rhinitis, weakness, malaise, tremoring wings, ataxia, and reluctance to fly (Cornwell et al., 1967; Boyle and Binnington, 1973; Vindevogel and Pastoret, 1981). The clinical course can last up to a week, but occasionally no clinical signs are observed prior to death (Lehner et al., 1967; Boyle and Binnington, 1973). Pathological lesions consist of focal necrosis of viscera, including liver, spleen, and pancreas, with intranuclear inclusion bodies at the periphery of the necrotic foci (Smadel et al., 1945; Cornwell et al., 1967, 1970; Cornwell and Wright, 1970; Lehner et al., 1967; Boyle and Binnington, 1973; Surman et al., 1975). Peritonitis and diphtheritic areas in the pharynx and larynx are occasionally seen (Cornwell and Wright, 1970; Cornwell et al., 1970). Since the disease can occur in closed flocks and affects mainly young birds, horizontal transmission from adult carriers to squabs soon after hatching seems likely (Cornwell et al., 1967; Lehner et al., 1967; Ward et al., 1971).

Raptor Herpesviruses Causing Hepatosplenitis

Three distinct herpesviruses causing hepatosplenitis have been isolated from raptors, Strigid HV 1 from owls, Falconid HV 1 from falcons, and Acciprid HV 1 from eagles (Kaleta, 1990). The disease in owls was first reported by Green (1935) in a great horned owl (*Bubo virginianus*) exhibiting inclusion body hepatosplenitis in the United States. Fatal hepatosplenitis of suspected viral etiology, exhibiting intranuclear inclusion bodies, was described in Austria affecting several owls from 1915 to 1965, including the European eagle owl (*Bubo bubo*), long-eared owl (*Asio otus*), and snowy owl (*Nyctea scandiaca*) (Burtscher, 1965). Electron microscopic work on this virus suggested that it was a herpesvirus, and through further studies it appeared to be a new avian herpesvirus specific for owls (Burtscher and Schumacher, 1966; Bürki et al., 1973). In 1969, a herpesvirus was isolated from owls with hepatosplenitis in West Germany (Maré, 1975; Kaleta, 1990). A similar disease was described in a great horned owl in Canada in 1973, and experimental transmission to the little owl (*Athene noctua scopoli*) and Tengmalm's owl (*Aegolius funereus*) was documented in Austria (Burtscher and Sibalin, 1975; Sileo et al., 1975).

Paralleling the emergence of the Strigid HV 1, a similar disease characterized by hepatosplenitis and intranuclear inclusion bodies was first reported in a prairie falcon (*Falco mexicanus*) in the United States in 1971 (Ward et al., 1971). Reports of natural disease in the United States in a red-headed falcon (*Falco chicquera*) and a peregrine falcon (*Falco peregrinus*) soon followed, and the Falconid HV 1 was isolated in 1973 (Maré and Graham, 1973a, 1973b; Kaleta, 1990). In 1977, a naturally occurring case of inclusion body hepatosplenitis was reported for the first time in an American kestrel (*Falco sparverius*), and a herpesvirus was subsequently isolated (Kocan et al., 1977; Potgeiter et al., 1979). In 1982, herpesvirus disease was reported for the first time in raptors in Great Britain, affecting an American kestrel and a pair of European kestrels (*Falco tinnunculus*) (Veterinary Investigation Service, 1982).

The third recognized raptor herpesvirus, Acciprid HV 1, was isolated from an apparently healthy nestling bald eagle (*Haliaeetus leucocephalus*) and from a South American eagle showing typical hepatic and splenic lesions (Docherty et al., 1983; Kaleta, 1990). Further information on this virus is not yet available in the literature.

The raptor herpesviruses cause a fatal disease in birds of prey characterized by multifocal necrosis of the liver, spleen, and other organs, with formation of intranuclear inclusion bodies. The clinical course is very similar in both naturally occurring and experimentally induced disease. Several hours to several days prior to death, birds may show weakness, depression, and anorexia (Green and Shillinger, 1936; Maré and Graham, 1973a, 1973b; Sileo et al., 1975; Kocan et al., 1977). In many cases, no clinical signs are seen, and death is very sudden (Ward et al., 1971; Maré and Graham, 1973a, 1973b; Graham et al., 1975; Sileo et al., 1975; Kocan et al., 1977). Regurgitation, diarrhea, and weight loss are seen sporadically (Graham et al., 1975). In the experimentally induced disease, falcons die 3-6 days postinfection, whereas owls die 7-12 days postinfection (Green, 1935; Green and Shillinger, 1936; Maré and Graham, 1973a, 1973b; Graham et al., 1975; Sileo et al., 1975; Kocan et al., 1977). On gross examination, birds typically show multifocal tan-colored hepatic and splenic lesions (Green, 1935; Green and Shillinger, 1936; Maré and Graham, 1973a, 1973b; Graham et al., 1975; Sileo et al., 1975; Kocan et al., 1977). Similar lesions in the bone marrow, intestines, and pharynx are often seen (Maré and Graham, 1973a, 1973b; Graham et al., 1975; Sileo et al., 1975). Histologically, these tan-colored areas represent necrotic foci

without an associate inflammatory response (Maré and Graham, 1973a, 1973b; Graham et al., 1975; Sileo et al., 1975; Kocan et al., 1977). Cells at the periphery of these necrotic foci in the liver and spleen consistently contain Cowdry type A intranuclear inclusion bodies (Green, 1935; Green and Shillinger, 1937; Ward et al., 1971; Maré and Graham, 1973a, 1973b; Sileo et al., 1975; Graham et al., 1975; Kocan et al., 1977). Necrotic foci are also sporadically seen in the pancreas, lungs, and other tissues (Graham et al., 1975).

Diagnosis of this disease is confirmed by virus isolation in chicken embryo fibroblasts followed by serological identification (Maré and Graham, 1973b; Graham et al., 1975; Kaleta, 1990). In the final stages, an evaluation of white and red blood cell counts may be useful. Destruction of the bone marrow and lymphoreticular cells throughout the body causes a severe leukopenia without inflammatory cell response (Graham et al., 1975). Anemia is not seen, despite necrosis of erythropoietic tissue, because of the longer half-life of erythrocytes compared with leukocytes (Graham et al., 1975).

The host spectrum of the serologically related herpesviruses, Columbid HV 1, Strigid HV 1, Falconid HV 1, and Acciprid HV 1, is still not clearly defined. Natural infection of the Strigid HV 1 has been recognized in the great horned owl, European eagle owl, long-eared owl, snowy owl, and other unspecified owls (Burtscher, 1965; Burtscher and Sibalin, 1975). Experimentally, Strigid HV 1 has been transmitted to the great horned owl, screech owl (*Otus asio*), little owl, Tengmalm's owl, long-eared owl, European kestrel, American kestrel, ring-necked dove (*Streptopelia risoria*), and budgerigar (Green, 1935; Bürki et al., 1973; Burtscher and Sibalin, 1975; Maré and Graham, 1973a; Maré, 1975). The great grey owl (*Strix nebulosa*), tawny owl (*Strix aluco*), barn owl (*Tyto albo scopoli*), barred owl (*Strix varia varia*), and pigeon are resistant to experimental infection (Green, 1935; Green and Shillinger, 1936; Burtscher and Sibalin, 1975).

The Falconid HV 1 has a host range that includes the prairie falcon, peregrine falcon, red-headed falcon, gyrfalcon (*Falco rusticolus*), American kestrel, merlin (*Falco columbarius*), Cooper's hawk (*Accipiter cooperii*), Swainson's hawk (*Buteo swainsoni*), great horned owl, screech owl, ring-necked dove, muscovy duck (*Carina moschata*), green heron (*Butorides virescens*), budgerigar, and Amazon parrot (*Amazona ochracephala*) (Maré and Graham, 1973a, 1973b; Ward et al., 1971; Maré, 1975). The disease could not be experimentally transmitted to the domestic turkey, domestic fowl, red-tailed hawk (*Buteo jamaicensis*), or domestic pigeon (Maré and Graham, 1973a, 1973b; Graham et al., 1975). The virus isolated from an American kestrel, presumably Falconid HV 1, caused disease experimentally in the American kestrel and the barred owl (Kocan et al., 1977).

The Columbid HV 1 host range includes the domestic pigeon, ring-necked dove, cockatiel (*Nymphicus hollandicus*), and budgerigar (Maré and Graham, 1973a; Surman et al., 1975). The virus could not be experimentally transmitted to the American kestrel or great horned owl (Cornwell et al., 1970; Maré and Graham, 1973a). One isolate from a racing pigeon could not be experimentally transmitted to other pigeons, but was transmitted to cockatiels and budgerigars (Surman et al., 1975).

In raptors, the epizootiology of this virus can only be speculated upon (Graham et al., 1975). A popular suggestion is that prey species may be subject to mild or subclinical infection and serve as a source of virus (Ward et al., 1971; Graham et al., 1975; Maré, 1975). Natural infection by the oral route is substantiated by the prominence of intestinal and hepatic lesions, which are observed more commonly in field cases of the disease than in experimental infection by IM or intranasal inoculation (Graham et al., 1975; Maré, 1975). In addition, many naturally affected raptors were fed pigeon meat prior to succumbing to the disease (Ward et al., 1971; Kocan et al., 1977). Another suggestion is that the stress of captivity or concurrent disease may activate a latent virus, since natural cases involving Falconid HV 1 have occurred primarily in captive birds. In one report, 3 of 8 falcons that succumbed to Falconid HV 1 had concurrent disease (Graham et al., 1975; Maré, 1975). Experimental transmission studies from prey to predator species have been unsuccessful, even though close serological relationships exist among the different viruses (Kaleta, 1990). Despite this, it would seem prudent to avoid contact between pigeons and raptors, to reduce the stress of captivity as much as is feasible, and to avoid concurrent disease by sound husbandry and management practices.

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Part III

Surgery and Anesthesia

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CHAPTER 20

Electrocardiography on Fifty-nine Anesthetized Convalescing Raptors

NAOMI L. BURTNICK, M.T. (A.S.C.P.)

Cardiology Section
Department of Small Animal Clinical Sciences
University of Minnesota
St. Paul, Minnesota, U.S.A.

LAUREL A. DEGERNES, D.V.M.

The Raptor Center
Department of Veterinary Biology
University of Minnesota
St. Paul, Minnesota, U.S.A.

ABSTRACT: Standard and augmented limb leads were recorded on 20 bald eagles (*Haliaeetus leucocephalus*), 11 red-tailed hawks (*Buteo jamaicensis*), 8 great horned owls (*Bubo virginianus*), 8 American kestrels (*Falco sparverius*), 2 golden eagles (*Aquila chrysaetos*), 2 peregrine falcons (*Falco peregrinus*), 2 barred owls (*Strix varia*), 2 saw-whet owls (*Aegolius acadicus*), 2 Cooper's hawks (*Accipiter cooperii*), and 2 sharp-shinned hawks (*Accipiter striatus*) while they convalesced from traumatic injuries at the Raptor Center at the University of Minnesota. Ages ranged from hatch year to adult, and the sex was not determined in most species. The birds were anesthetized with ketamine/xylazine IV or IM and placed in dorsal recumbancy with wings folded. Four silver-platinum needle electrodes were placed subcutaneously over the distal lateral tarsometatarsus and over the carpal joint of each wing. The wave forms were analyzed on lead II at 50 mm/sec at 1 cm = 1 mV to determine P, PR, QRS, and QT durations and P and QRS amplitudes. The mean electrical axis for the frontal plane (MEA) was calculated using leads I and aVF, and the ST segment was assessed for elevation or depression. The mean, range, and standard deviation were measured for each parameter within each species. The polarity of each wave form was tabulated on all leads. A QS wave was the predominant wave form on leads II, III, and aVF, with an R wave on leads aVR and aVL. The mean QRS polarity of lead I was variable. The mean MEA never deviated more than 18° from an exact craniad direction of −90° for any species except the Cooper's hawk. Significant differences were observed among various age groups of bald eagles for the heart rate, P wave duration and amplitude, PR interval, and QRS amplitude. Statistical analyses of the electrocardiographic parameters indicate that the duration of P, PR, QRS, and QT increases with increasing species size.

KEY WORDS: raptor, ECG, EKG, electrocardiography.

Electrocardiography (ECG) has long been an effective diagnostic tool in determining cardiac arrhythmias, enlargements, and conduction disturbances in many mammalian and avian species (Czarnecki and Good, 1980; Hunsaker et al., 1971; Lumeij 1986b; Miller, 1986, 1987). Telemetry has been used on birds of prey to determine heart rates (Busch et al., 1984; Gessaman, 1978; Patton et al., 1984; Sawby and Gessaman, 1974), to assess the effects of handling stress (Busch et al., 1978), and as an indirect measure of metabolism (Gessaman, 1980). Electrocardiography has also been utilized to monitor anesthesia in eagles (Wingfield and DeYoung, 1972), goshawks (*Accipiter gentilis*) (Lumeij, 1986a), and red-tailed hawks (*Buteo jamaicensis*) (Degernes et al., 1988). Normal ECG values have previously been established on a limited number of raptors (Edjtehadi et al., 1977; Zenoble and Graham, 1979) due to the difficulty in obtaining large numbers of birds and the poor quality of ECG tracing seen with unanesthetized restraint.

In this study, electrocardiograms (ECGs) were recorded from anesthetized raptors that were convalescing from traumatic injuries. The purpose of this study was to obtain ECG data on various raptor species in order to determine what ECG differences exist among different species. Another goal was to evaluate the practicality and potential value of doing ECGs on raptors in a clinical setting.

Materials and Methods

Standard bipolar and augmented unipolar limb leads were recorded over a 1-yr period on 20 bald eagles (*Haliaeetus leucocephalus*) (BE), 11 red-tailed hawks (RTH), 8 great horned owls (*Bubo virginianus*) (GHO), 8 American kestrels (*Falco sparverius*) (KES), 2 golden eagles (*Aquila*

chrysaetos) (GE), 2 peregrine falcons (*Falco peregrinus*) (PEF), 2 barred owls (*Strix varia*) (BDO), 2 saw-whet owls (*Aegolius acadicus*) (SWO), 2 Cooper's hawks (*Accipiter cooperii*) (CSH), and 2 sharp-shinned hawks (*Accipiter striatus*) (SSH) while they convalesced from traumatic injuries at the Raptor Center. Ages ranged from hatch year to adult, and the gender was not determined in most species.

The birds were anesthetized with ketamine hydrochloride[a] and xylazine hydrochloride.[b] These drugs were combined and administered IV in the basilic vein in all birds except the KES, which, due to the small size of their veins, were injected in the pectoral muscle. The following 5:1 ketamine (KET)/xylazine (XYL) ratio dosages were used empirically: the BE, GE, and GHO received 15 mg KET combined with 3 mg XYL; the RTH received 10 mg KET with 2 mg XYL; the PEF received 7 mg KET with 1.4 mg XYL; the BDO received 6 mg KET with 1.2 mg XYL; the CSH received 4 mg KET with 0.8 mg XYL; and the KES, SWO, and SSH received 3 mg KET with 0.6 mg XYL. Any deviation from full immobilization was noted.

A pilot study of three raptors showed no difference in ECG values obtained with dorsal versus right lateral recumbancy, which supports other findings on birds of prey (Zenoble and Graham, 1979). The method chosen was dorsal recumbancy on a padded table with wings folded, and feet taped for operator safety.

Four silver-platinum electroencephalogram-type needle electrodes[c] were placed subcutaneously over the distal lateral tarsometatarsus and over the carpal joint of each wing (Fig. 20.1). Leads I, II, III, aVR, aVL, and aVF were run on a standard strip chart ECG unit[d] at 50 mm/sec with a standardization of 1 cm = 1 mV.

The P, PR, QRS, and QT durations and P and QRS amplitudes were measured on lead II and intervals were checked over three to four beats. The polarity of P, QRS, and T waves was tabulated for all leads. The mean electrical axis for the frontal plane (MEA) was calculated using two orthogonal leads: leads I and aVF, or occasionally leads II and aVL, or III and aVR if the lead I amplitudes were too small for accurate measurement. The ST segment was assessed for elevation or depression. Any arrhythmias were noted. To ensure consistency in the procedure, all of the ECG tracings were run and analyzed by the first author. The duration of measurements was accurate and repeatable up to 0.005 sec. Amplitude measurements were always repeatable.

The mean, range, and standard deviation were determined for each ECG parameter within each species. Box plots were generated and Spearman's rank correlation coefficient was determined for each ECG parameter to compare the BE, GHO, RTH, and KES. Of all the birds studied, these four species were the most desirable for statistical analyses because of the range in actual size of the birds and the relatively large amount of data for each species. This type of statistical analysis was chosen because the individual weights of the raptors were not known and ranking by relative size of the species was a viable option.

Figure 20.1. Raptors were positioned in dorsal recumbancy with wings folded. Needle electrodes were placed subcutaneously over the distal lateral tarsometatarsus and over the carpal joint of each wing.

Electrocardiograms were run on 3 nestling BE (8 wk old) to assess differences in the ECG relative to the age of the raptor. Box plots were generated and Spearman's rank correlation coefficient was determined for each ECG parameter to compare their values to the hatch-year, after-hatch-year, and adult BE in the study.

Serial tracings were run on three raptors, BE 4, BE 16, and an RTH, to assess the repeatability of their ECG values over time. On BE 4, a total of three ECG recordings were taken: the original, 3 wk after, and 12 wk after. On BE 16, two ECGs were recorded at 3-wk intervals, and for the RTH, two ECG tracings were recorded at 6-wk intervals.

Results

A representative ECG tracing is presented in Fig. 20.2. The lead II P, PR, QRS, and QT durations and heart rate for all birds are summarized in Table 20.1. The MEA and the lead II amplitudes for P wave, QRS complexes, and ST segment elevation or depression are summarized in Table 20.2 for all birds.

The raptors were divided into two groups based on the number of birds available for study. Group 1 had 8 to 20 birds evaluated per species (BE, GHO, RTH, and KES).

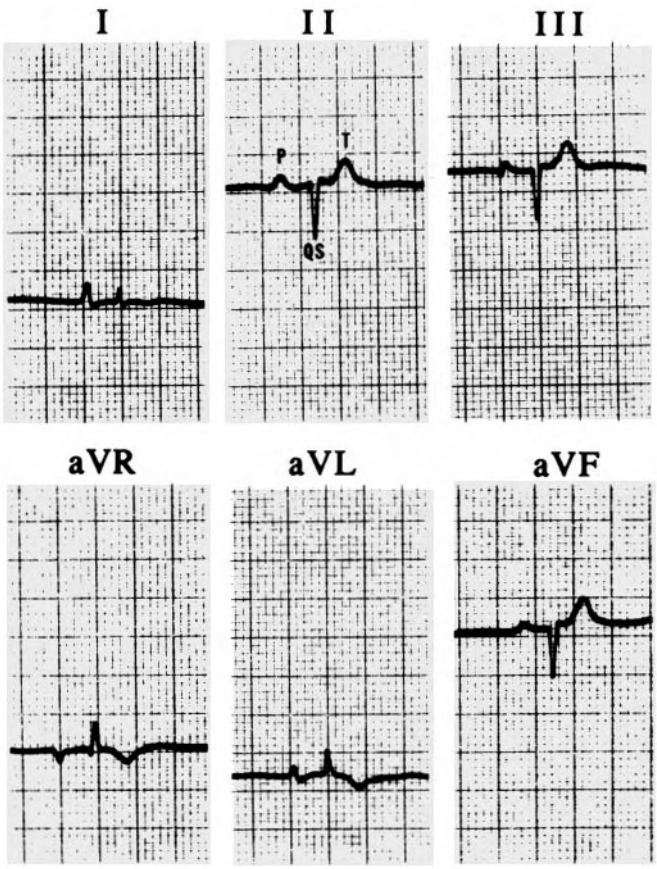

Figure 20.2. Electrocardiogram from a male, hatch-year BE with a fractured left ulna anesthetized with 15 mg ketamine and 3 mg xylazine. Leads I, II, III, aVR, aVL, and aVF are shown at 1 cm = 1 mV and at 50 mm/sec. The heart rate is 80 beats per min and the mean electrical axis is −80°. A QS wave is the major deflection in leads II, III, and aVF.

Group 2 had 2 birds evaluated per species (GE, PEF, BDO, SWO, CSH, and SSH).

Heart Rate

For Group 1 there was an increase in the heart rate (Spearman's $\rho = 0.73$, $p < 0.01$) as the relative size of the raptor became smaller (Fig. 20.3). The birds ranged in size from the largest to smallest—BE, GHO, RTH, and KES—and had respective mean heart rates (HR) of 82, 111, 122, and 158 beats per minute (bpm). The Group 2 birds had rates correspondingly appropriate for their size. The lowest heart rate of all 59 birds was recorded from a BE at 50 bpm. The highest heart rate of all 59 birds was recorded from an SWO at 280 bpm. Of the Group 1 birds, the GHO had the narrowest range at 100 to 130 bpm ($n = 8$), which is lower than previous findings of 180 to 360 bpm ($n = 6$) (Zenoble and Graham, 1979). The RTH had the broadest range at 80 to 220 bpm.

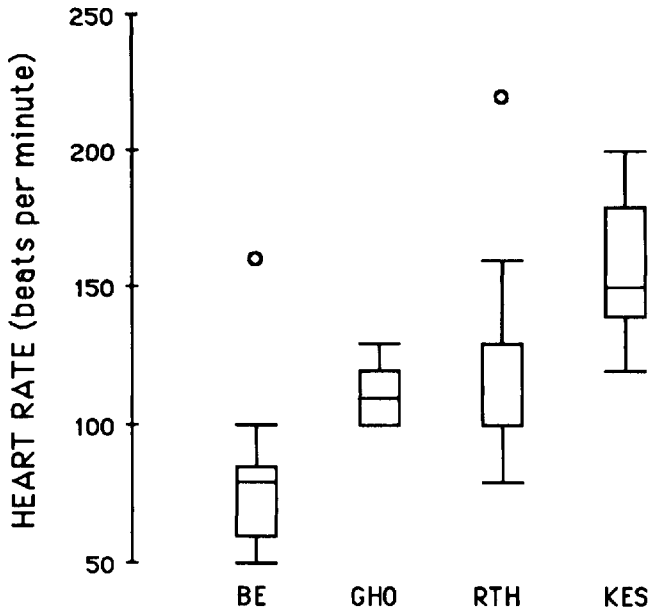

Figure 20.3. Comparison of heart rate to relative size of BE (largest bird), GHO and RTH (intermediate-sized birds), and KES (smallest bird) anesthetized with a 5:1 ratio of ketamine (mg)/xylazine (mg): BE and GHO 15/3; RTH 10/2; KES 3/0.6. Extreme data values were plotted with a small circle.

Rhythm

Incomplete atrioventricular dissociation (IAVD) was observed in two BE and both of the GE studied (Fig. 20.4). Sinoatrial block was occasionally present in one GHO. Sinus tachycardia was present in two RTH and one SWO. All other birds maintained a normal sinus rhythm versus sinus arrhythmia.

One of the GE with IAVD was only lightly sedated, with eyes open and needing slight restraint. A repeat ECG was run 5 days later with no anesthesia; at that time a normal sinus rhythm was observed. All three of the birds of prey showing sinus tachycardia were emerging from their anesthesia at the time of their ECG recording. The two RTH had initial HR of 400 bpm, which slowed to 160 and 140 bpm 2 min after reinjection of their KET/XYL doses. The SWO showed an HR of 280 bpm; whether it can truly be classified as sinus tachycardia is questionable. The other SWO studied showed an HR of 200 bpm, which is high compared to other raptors but may be appropriate for its small size. Only two other birds, both RTH, were emerging from their anesthesia during their ECGs, and both demonstrated normal sinus rhythm.

P Wave

The P wave (atrial depolarization) duration range on lead II for all 59 raptors was 0.01 to 0.06 sec. For Group 1 there is

Table 20.1
Lead II heart rate and duration of electrocardiogram intervals in 59 anesthetized[a] raptors

RAPTOR (n)[b]	*Heart Rate (bpm)*[c]	*P (sec)*	*PR (sec)*	*QRS (sec)*	*QT (sec)*
Bald eagles (20)					
(*Haliaeetus leucocephalus*)					
Mean	82	0.041	0.091	0.029	0.135
S.D.[d]	30.018	0.008	0.012	0.006	0.015
Range	50-160	0.030-0.060	0.070-0.110	0.020-0.040	0.110-0.165
Great horned owls (8)					
(*Bubo virginianus*)					
Mean	111	0.037	0.086	0.025	0.149
S.D.	12.464	0.008	0.009	0.004	0.015
Range	100-130	0.030-0.050	0.070-0.100	0.020-0.030	0.125-0.165
Red-tailed hawks (11)					
(*Buteo jamaicensis*)					
Mean	122	0.032	0.071	0.025	0.116
S.D	39.451	0.005	0.013	0.004	0.026
Range	80-220	0.020-.0350	0.050-0.090	0.020-0.030	0.080-0.165
American kestrels (8)					
(*Falco sparverius*)					
Mean	158	0.030	0.059	0.021	0.099
S.D.	27.124	0.003	0.004	0.003	0.014
Range	120-200	0.030-0.035	0.050-0.065	0.015-0.025	0.085-0.120
Golden eagles (2)					
(*Aquila chrysaetos*)					
Mean	100	0.043	0.090	0.025	0.142
S.D.	0	0.004	0.007	0	0.011
Range	100-100	0.040-0.045	0.085-0.095	0.025-0.025	0.135-0.150
Peregrine falcons (2)					
(*Falco peregrinus*)					
Mean	95	0.035	0.072	0.025	0.135
S.D.	7.071	0	0.004	0	0.014
Range	90-100	0.035-0.035	0.070-0.075	0.025-0.025	0.125-0.145
Barred owls (2)					
(*Strix varia*)					
Mean	120	0.055	0.105	0.027	0.145
S.D.	28.284	0.007	0.007	0.004	0.007
Range	100-140	0.050-0.060	0.100-0.110	0.025-0.030	0.140-0.150
Saw-whet owls (2)					
(*Aegolius acadicus*)					
Mean	240	0.018	0.055	0.020	0.102
S.D.	56.569	0.010	0.007	0	0.025
Range	200-280	0.010-0.025	0.050-0.060	0.02-0.020	0.085-0.120
Cooper's hawks (2)					
(*Accipiter cooperii*)					
Mean	160	0.027	0.062	0.025	0.098
S.D.	28.284	0.004	0.011	0	0.004
Range	140-180	0.025-0.030	0.055-0.070	0.025-0.025	0.095-0.100
Sharp-shinned hawks (2)					
(*Accipiter striatus*)					
Mean	220	0.030	0.060	0.020	0.110
S.D.	28.284	0	0.007	0	0.028
Range	200-240	0.030-0.030	0.055-0.065	0.020-0.020	0.090-0.130

[a]Anesthetized with a 5:1 ratio of ketamine (mg)/xylazine (mg): bald eagles, golden eagles, and great horned owls 15/3; red-tailed hawks 10/2; peregrine falcons 7/1.4; barred owls 6/1.2; Cooper's hawks 4/0.8; American kestrels, saw-whet owls, sharp-shinned hawks 3/0.6.
[b]n = number of birds.
[c]bpm = beats per minute.
[d]S.D. = standard deviation.

a relationship (Spearman's $\rho = -0.60$, $p < 0.01$) between P wave duration and the relative size of the raptor: the larger the bird, the longer the P wave duration (Fig. 20.5). In descending order of size, the BE, GHO, RTH, and KES had

Table 20.2
Frontal plane mean electrical axis and lead II amplitudes in 59 anesthetized[a] raptors

RAPTOR (n)[b]	*Axis (degrees)*	*P (mV)*	*QRS*[c] *(mV)*	*ST*[d] *(mV)*
Bald eagles (20)				
Mean	−86	0.182	0.723	↑0.017
S.D.[e]	26.222	0.078	0.363	0.061
Range	−30 to −150	0.050-0.325	0.150-1.450	↓0.050-↑0.200
Great horned owls (8)				
Mean	−88	0.069	0.303	0
S.D.	9.613	0.026	0.083	0
Range	−80 to −110	0.050-0.100	0.200-0.450	0
Red-tailed hawks (11)				
Mean	−84	0.08	0.482	↑0.068
S.D.	19.377	0.068	0.193	0.106
Range	−50 to −110	(−0.100)-0.175	0.300-0.900	↑0-0.300
American kestrels (8)				
Mean	−98	0.209	0.588	↑0.131
S.D.	7.709	0.058	0.195	0.119
Range	−87 to −110	0.150-0.325	0.350-0.900	↑0-0.300
Golden eagles (2)				
Mean	−85	0.100	0.375	0
S.D.	7.071	0	0.106	0
Range	−80 to −90	0.100-0.100	0.300-0.450	0
Peregrine falcons (2)				
Mean	−80	0.338	0.688	↑0.125
S.D.	28.284	0.088	0.442	0.035
Range	−60 to −100	0.275-0.400	0.375-1.000	↑0.100-0.150
Barred owls (2)				
Mean	−108	0.050	0.375	0
S.D.	10.607	0	0.035	0
Range	−100 to −115	0.050-0.050	0.350-0.400	0
Saw-whet owls (2)				
Mean	−90	0.100	0.550	0
S.D.	7.071	0.071	0.141	0
Range	−85 to −95	0.050-0.150	0.450-0.650	0
Cooper's hawks (2)				
Mean	−60	0.075	0.300	0
S.D.	7.071	0.035	0.141	0
Range	−55 to −65	0.050-0.100	0.200-0.400	0
Sharp-shinned hawks (2)				
Mean	−95	0.125	0.525	↑0.100
S.D.	0	0.035	0.601	0.141
Range	−95 to −95	0.100-0.150	0.100-0.950	↑0-0.200

[a]Anesthetized with a 5:1 ratio of ketamine (mg)/xylazine (mg): bald eagles, golden eagles, and great horned owls 15/3; red-tailed hawks 10/2; peregrine falcons 7/1.4; barred owls 6/1.2; Cooper's hawks 4/0.8; American kestrels, saw-whet owls, sharp-shinned hawks 3/0.6.
[b]*n* = number of birds.
[c]Lead II QRS complex measurements are the negative wave only (a QS or S wave).
[d]0 = isoelectric; ↑ = elevation from baseline; ↓ = depression from baseline.
[e]S.D. = standard deviation.

respective mean lead II P wave durations of 0.041, 0.037, 0.032, and 0.030 sec. In Group 2, the two BDO had P waves of 0.05 and 0.06 sec, both at the high end of the P wave duration range. The only other species with P waves as long as 0.06 sec was the BE, and that was only 1 bird out of 20.

The P wave was largest in amplitude on lead I in 73% of the birds, on lead II in 15%, and lead I equaled lead II on 12% of the birds. The lead II P wave amplitude range for all 59 raptors was −0.10 to 0.40 mV. There is no apparent relationship (Spearman's $\rho = -0.13$, n.s.) between P wave amplitude and size of the bird of prey, as shown by the high end of the range being shared by the large BE, the PEF, and the small KES. The eight GHO had a very narrow lead II P wave amplitude range of 0.05 to 0.10 mV.

On leads I, II, and aVL, the P wave was predominantly positive: 98%, 93%, and 94%, respectively. Only one bird, an RTH, showed a negative P wave on lead II. On

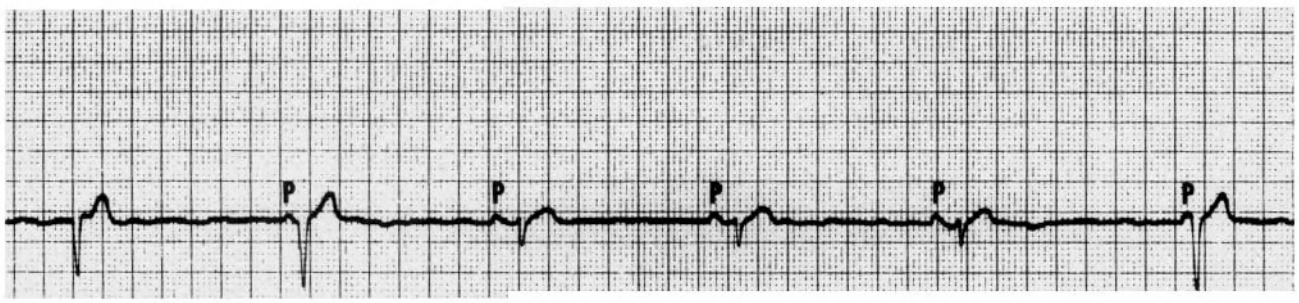

Figure 20.4. Incomplete atrioventricular dissociation in an adult GE with shotgun injuries to the body and one eye. It was anesthetized with 15 mg ketamine combined with 3 mg xylazine.

lead aVF, 62% of the P waves were positive and 23% were isoelectric or variably isoelectric and positive, with the remainder being negative, biphasic, or variably positive and negative. Lead aVR P waves were negative in 98% of the birds. Lead III P waves were negative in 44%, biphasic in 37%, and positive in 15% of the raptors, with the remainder being isoelectric or variably negative and isoelectric.

PR Interval

The PR interval is measured from the beginning of the P wave to the beginning of the QRS complex and represents atrioventricular conduction time. For Group 1 there is a relationship (Spearman's $\rho = -0.72$, $p < 0.01$) between the PR interval and the relative size of the raptor; the larger the bird of prey, the longer the PR interval (Fig. 20.6). The BE, GHO, RTH, and KES in descending order of size have respective mean lead II PR intervals of 0.091, 0.086, 0.071, and 0.059 sec. The range for all 59 birds was 0.05 to 0.11 sec. The high end of the range was shared by the BE and the BDO.

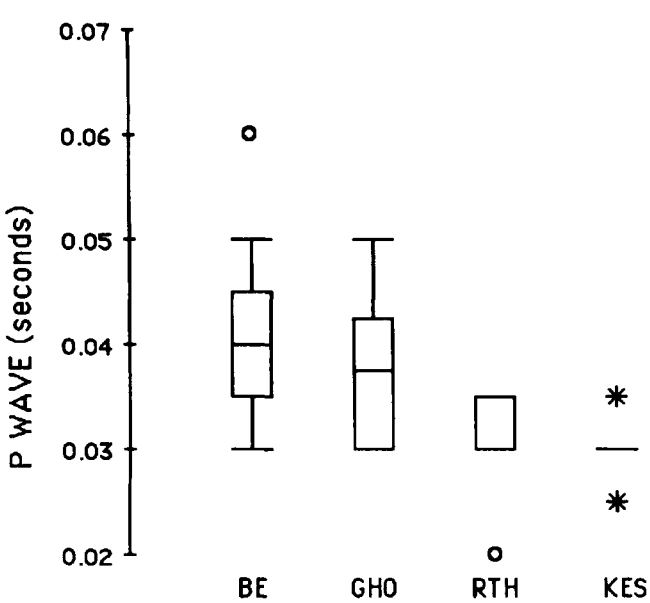

Figure 20.5. Comparison of P wave duration to relative size of BE (largest bird), GHO and RTH (intermediate-sized birds), and KES (smallest bird) anesthetized with a 5:1 ratio of ketamine (mg)/xylazine (mg): BE and GHO 15/3; RTH 10/2; KES 3/0.6. Extreme data values were plotted with a small circle and very extreme values with an asterisk.

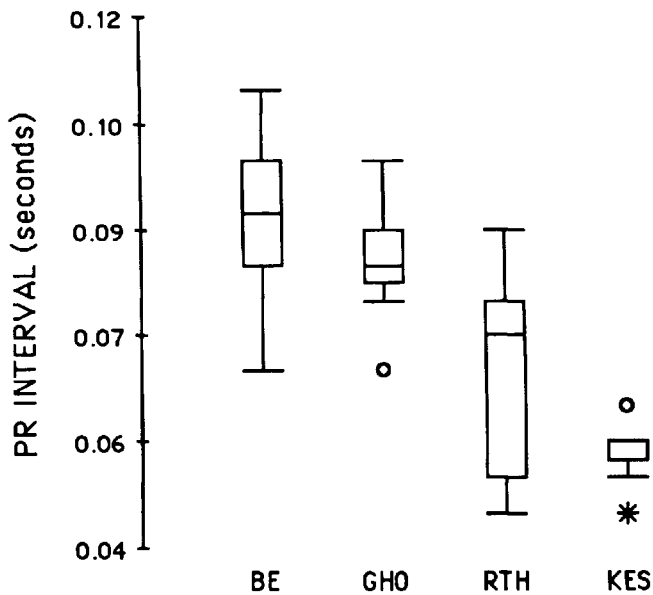

Figure 20.6. Comparison of PR interval to relative size of BE (largest bird), GHO and RTH (intermediate-sized birds), and KES (smallest bird) anesthetized with a 5:1 ratio of ketamine (mg)/xylazine (mg): BE and GHO 15/3; RTH 10/2; KES 3/0.6. Extreme data values were plotted with a small circle and very extreme values with an asterisk.

QRS Complex

The range for lead II QRS complex (ventricular depolarization) duration in all 59 raptors was 0.015 to 0.040 sec. Group 1 birds show a correlation (Spearman's $\rho = -0.57$, $p < 0.01$) with bird size to QRS duration (Fig. 20.7). The relatively larger the bird, the longer the QRS duration; the BE, GHO, RTH, and KES in descending order of size have respective mean lead II QRS durations of 0.029, 0.025, 0.025, and 0.021 sec.

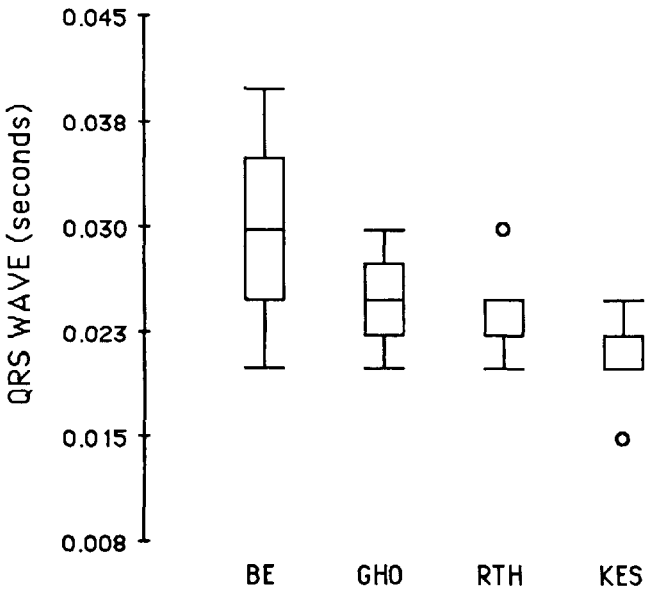

Figure 20.7. Comparison of QRS complex duration to relative size of BE (largest bird), GHO and RTH (intermediate-sized birds), and KES (smallest bird) anesthetized with a 5:1 ratio of ketamine (mg)/xylazine (mg): BE and GHO 15/3; RTH 10/2; KES 3/0.6. Extreme data values were plotted with a small circle.

The predominant wave form on leads II, III, and aVF was a QS wave: 75%, 88%, and 80%, respectively, with the remaining percentage being an rS wave with only rare Qr, R, or RS waves. Therefore, the lead II QRS complex amplitude values listed in Table 20.2 are the negative wave form only (that is, a QS, Q, or S wave). Lead I was almost equally divided among QS, R, and RS waves—38%, 32%, and 30%, respectively. Leads aVR and aVL were predominantly an R wave, 72% and 92%, respectively, with the remaining percentage being a qR wave with only occasional QS or RS waves.

The largest amplitude QRS complex was seen in lead II in 65% of the raptors, lead II amplitude equaled lead aVF amplitude in 21%, and lead aVF amplitude was largest in 14% of the raptors. The SSH, CSH, and BDO all showed the largest amplitude QRS on lead aVF only. Four of the eight GHO showed lead II as the largest amplitude QRS, with the other four having lead II equal lead aVF. The lead II QRS amplitude range for all 59 raptors was 0.10 to 1.45 mV. For Group 1 there was no correlation (Spearman's $\rho = -0.21$, n.s.) between size of raptor and QRS amplitude. The BE, the largest raptor, was at the high end of the QRS amplitude range, as might be expected, but the medium-sized RTH and small KES shared 0.90 mV as their upper limit. In Group 1 the GHO showed a narrow range of 0.20 to 0.45 mV. All of the Strigiformes—the BDO, SWO, and GHO—had a narrow lead II QRS amplitude range of 0.20 to 0.65 mV.

ST Segment

The ST segment (time from the end of the QRS interval to the onset of the T wave) was isoelectric in 67% of the raptors, elevated in 30%, and depressed in 3%. All of the Strigiformes—the GHO, BDO, and SWO—had isoelectric ST segments. Of the Falconiformes, only the GE and CSH were isoelectric. Greater than half of the KES and RTH and all of the PEF showed ST segment elevation—5 of 8, 6 of 11, and 2 of 2 birds, respectively. The only ST segment depression observed was in 2 of the 20 BE, while 3 of 20 BE showed ST segment elevation. Of the SSH, one showed ST segment elevation and the other showed ST depression.

QT Interval

The QT interval (summation of ventricular depolarization and repolarization) is measured from the beginning of the Q wave to the end of the T wave. The lead II QT interval range for all 59 raptors was 0.08 to 0.165 sec. As would be expected, the small KES and SWO had extremely short QT intervals. Although the BE and GHO shared the upper limit for QT interval duration with the RTH, there was a correlation with size of raptor to QT interval (Spearman's $\rho = -0.52$, $p < 0.01$), with the larger birds having longer QT intervals. The RTH had the widest range, encompassing the lowest and highest values.

T Wave

The T wave (ventricular repolarization) was positive in 95% of the birds of prey on leads II, III, and aVF, with the remaining 5% being either negative or biphasic. On lead I the T wave polarity was variable, with 57% of the raptors being positive, 22% negative, and the remaining 21% either isoelectric or combinations of isoelectric and positive or negative. The T waves on leads aVR and aVL were predominantly negative—90% and 88%, respectively—with the remaining proportion being either biphasic or variably isoelectric and negative. The T waves were discordant to the QRS complexes on all leads.

Mean Electrical Axis

The mean electrical axis (MEA) in the frontal plane was directed craniad in all 59 raptors (Fig. 20.8). The widest range of the 59 raptors studied was $-30°$ to $-150°$. Of the species studied, the BE had the widest range, encompassing the lowest and highest values. The breadth of this range is made clearer when compared with normal turkey poults at $-31°$ to $-121°$ (Hunsaker et al., 1971) and normal parakeets at $-90°$ to $-162°$ (Miller, 1987). Group 1 birds showed no significant difference (Spearman's $\rho = -0.23$, n.s.) in their MEA, with the mean MEA never more than 8° from an

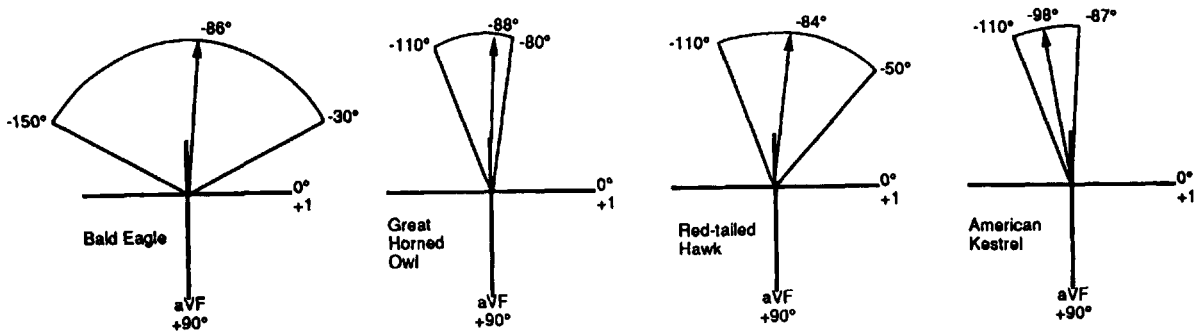

Figure 20.8. Comparison of mean electrical axis in the frontal plane to relative size of BE (largest bird), GHO and RTH (intermediate-sized birds), and KES (smallest bird) anesthetized with a 5:1 ratio of ketamine (mg)/xylazine (mg): BE and GHO 15/3; RTH 10/2; KES 3/0.6.

exact craniad direction of $-90°$ (Fig. 20.8). The MEA range for the GHO was $-80°$ to $-110°$, which is only slightly narrower than the previous findings of $-81°$ to $-127°$ (Zenoble and Graham, 1979). In Group 2, the CSH were the farthest to the left, with a mean MEA of $-60°$, and the BDO were farthest to the right, with a mean MEA of $-108°$. Except for the CSH, the mean MEA for Group 2 raptors was never more than 18° from an exact craniad direction of $-90°$.

Age Study

Of the 20 BE in the study, 5 were hatch-year, 4 were after-hatch-year, and 11 were adult birds. When these were compared with 3 nestling BE that were not included in the rest of the study, significant differences were found for the P wave duration and amplitude and the PR interval ($p \leq 0.01$), as well as the HR and QRS amplitude ($p < 0.05$). No significant differences were seen for the QRS duration, the QT interval, or the MEA.

Serial Study

In the small study to assess the stability of ECG values over time, serial tracings were run on three raptors, BE 16, BE 4, and an RTH. Two ECG tracings were recorded at 3-wk intervals for BE 16 and at 6-wk intervals for the RTH. Lead II P, PR, QRS, and QT durations varied no more than 0.005 sec. However, on the RTH the QRS amplitudes differed by 0.10 mV, while there was no difference in QRS amplitude on BE 16. ECG recordings were taken at 3 wk and 12 wk after the original ECG for BE 4. The P, PR, and QRS durations varied no more than 0.005 sec. However, the QT interval varied from 0.165 sec to 0.15 and 0.12 sec. The 12-wk ECG showed a large QRS amplitude increase of 0.30 mV. The MEA remained unchanged on all three birds.

Discussion

One of the goals of this study was to determine the practicality of running ECGs on raptors in a clinical setting. Most raptors require anesthesia at some point in their diagnostic workup, and the ECG can easily be incorporated at this time. All of the birds of prey in this study were anesthetized for radiology to assess various traumatic injuries, and their ECGs were run immediately after radiography. The time involved was brief: the actual recording of the ECG tracing took 2 to 3 min, including the time needed to apply electrodes. The short time commitment for ECG recording and analysis, combined with an affordable strip-chart recorder, the high-quality tracing obtained due to the anesthesia, and the use of needle electrodes all recommend the ECG as a practical tool for clinical use on raptors.

The potential diagnostic value of ECG on raptors is harder to assess. The need for more complete cardiac workups, including the ECG as a diagnostic tool, is apparent from the high number of previously undetected cardiac lesions in birds of prey observed during necropsy in the well-documented study by Cooper and Pomerance (1982). In our study, serious arrhythmias were noted that would otherwise have gone unobserved. These arrhythmias may have been due to the anesthesia or related to primary or secondary heart disease. The GE with IAVD, showing a normal sinus rhythm on the unsedated follow-up ECG, was being treated for a trap injury to his feet. Therefore, it is more likely that the anesthesia was the cause of the IAVD versus primary heart disease, trauma to the heart, or electrolyte disturbances. Because XYL causes cardiac depression in birds (Linn and Gleed, 1987; Mandelker, 1988), the KET-XYL anesthetic regimen may also be linked to the IAVD in the other GE tested, although trauma to the heart cannot be totally ruled out due to the multiple soft-tissue injuries observed in the bird. Further cardiac workup was indicated but was beyond the scope of this study. Although many species of owls respond favorably to the KET-XYL combination, the GHO and the snowy owl (*Nyctea scandiaca*) have both been known to show cardiac arrhythmias, inadequate muscle relaxation, and marked excitatory behavior on recovery when given levorotary isomer of KET (commercially avail-

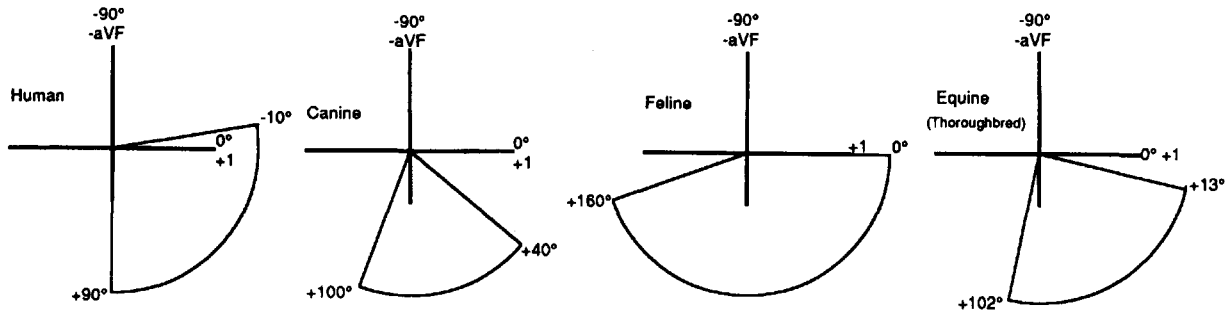

Figure 20.9. The predominantly sinistrad and caudad orientation of the mean electrical axes in the frontal plane in four mammalian species (human, canine, feline, and equine).

able ketamine contains equal amounts of both dextro- and levorotary forms) (Redig et al., 1984). Because KET does lower HR (Linn and Gleed, 1987), the KET-XYL anesthesia may also explain the narrower HR range (100 to 130 bpm) of the GHO in our study compared with the 180 to 360 bpm of the GHO in the Zenoble and Graham (1979) study, which used halothane/O_2. The one RTH showing a consistently negative P wave on lead II was also one of the two RTH with sinus tachycardia, which slowed to a normal sinus rhythm on reinjection of KET-XYL. It is therefore possible that the negative P wave was linked to the KET-XYL. It is also possible that atrial enlargement was present or that this is a normal variation.

When considering diagnoses such as atrial enlargement characterized by prolonged P waves or bundle branch blocks yielding prolonged QRS complexes, it will be necessary to consider the species of raptor in question because of the significant differences in P, PR, QRS, and QT durations relative to the size of the raptor. The long PR interval in the BE is not surprising due to the bird's large size, but in the case of the BDO it may be a reflection of the long P wave durations shown by the two BDO studied. The fact that statistically significant differences were observed on Group 1 birds for HR, P wave duration, PR interval, QRS duration, and the QT interval argues favorably for using the values in Tables 20.1 and 20.2 as guidelines for the separate species versus lumping all raptor ECG values together. That the GHO values existed within a very narrow range presents another good argument for keeping parameters separate for each species.

Major deviations from a normal MEA have been shown to be an extremely useful tool in identifying developing cardiomyopathies in turkey poults (Czarnecki and Good, 1980), round heart disease in turkey poults (Hunsaker et al., 1971; Jankus et al., 1971), vitamin E deficiency in chickens (Sturkie et al., 1954), and *Escherichia coli* infection in chickens (Gross, 1966). For clinical purposes it is ideal for the normal MEA ranges to be small to ensure that deviations from the norm are truly associated with pathology. A net MEA of approximately $-90°$ (craniad) is a distinctive feature of the avian ECG (Goldberg and Bolnick, 1980; Szabuniewicz and McCrady, 1974). This differs radically from the more sinistrad and caudad orientation of MEA in most mammalian species (Cooksey et al., 1977; Fregin, 1982; Tilley, 1985) (Fig. 20.9). This difference in MEA is due primarily to the disposition of the bundles of His within the myocardium. The bundles are distributed epicardially in avian species (Sturkie, 1986) and are usually located in the subendocardial region of the ventricular wall in mammals (Cooksey et al., 1977; Fregin, 1982; Tilley, 1985).

The widest range of the 59 raptors studied, $-30°$ to $-150°$, was seen in the BE. All other raptors in the study returned a much narrower range. Both CSH, although having a narrow range, $-55°$ to $-65°$, were the only raptors whose mean MEA deviated more than 18° from $-90°$, which is one more reason for keeping parameters separate for each species when determining axis deviations. It would be interesting to see more CSH evaluated to determine if all of their MEAs are oriented to the right.

It is questionable how much clinical significance should be placed on the ST segment changes observed. Although 30% of the birds of prey studied showed ST segment elevation, it has been shown to be normal in racing pigeons (Lumeij and Stokhof, 1985). ST segment changes can be an indicator of trauma to the heart muscle, myocardial infarction, pericarditis, and myocardial hypoxia in dogs and cats (Tilley, 1985), or associated with thiamine deficiency in chickens (ST segment depression) or vitamin E deficiency in chickens (ST segment elevation) (Sturkie, 1986).

The clinical value of the QT interval is also questionable. On BE 4 the QT interval varied from 0.165 sec to 0.15 and 0.12 sec. The QT interval is inversely proportional to the HR, but the HR was identical on the 0.12 and the 0.165 sec tracings, so this does not account for the differences seen. Changes in the QT interval may be seen with electrolyte disturbances, drug toxicity, hypothermia, and central nervous system disease in the dog and cat, although the QT interval alone is usually not enough to make a diagnosis (Miller and Tilley, 1988). Using a QT interval that has been corrected for HR may be more informative. The variation in data on

the small number of serial ECGs involving BE 4, BE 16, and an RTH suggest that more work needs to be done to determine the constancy of ECG values on raptors before assuming that differences in sequential tracings are meaningful for diagnostic purposes.

Two of many possible causes for the increased-amplitude R wave seen on the 12-wk ECG of BE 4 in the serial study could be a resolution of pericardial fluid, which could have been previously damping impulses, or, less likely, an increase in size of the cardiac chamber. Further cardiac evaluation was indicated but not pursued.

Although gender was not determined in most species of raptor tested, there may be sex differences in the raptor ECG. It has been shown that there are gender differences in the ECG of chickens, with females having more fusion of P and T waves on leads II and III and males having greater amplitudes on all waves (Sturkie and Hunsaker, 1957). Further work needs to be done to determine if gender differences exist on the ECG in raptors. The significant differences observed between nestling, hatch-year, after-hatch-year, and adult BE for the HR, P wave, PR interval, and QRS amplitude suggest that there are age differences in the ECG of raptors and that further work needs to be done to determine if this is true for other species.

The strong evidence of the existence of undetected cardiac lesions in birds of prey (Cooper, 1978; Cooper and Pomerance, 1982) and caged birds (Cooper, 1988) on necropsy indicates that most cases of cardiovascular disease in birds are diagnosed postmortem. The potential ability to determine heart enlargement with statistically significant values for each species of raptor and the capacity to identify arrhythmias and conduction disturbances highly recommend that the ECG be used routinely for the detection of cardiac disease in raptors.

Acknowledgments

We would like to thank Dr. Phillip Ogburn for the independence he encouraged, without which this study would not have been possible. We would also like to thank Diane Olson for her enthusiastic technical support.

Product List

[a]Ketaset, Aveco Co. Inc., P.O. Box 717, Ft. Dodge, Iowa 50501 USA; or Aescoket, Aesculaap, Mijlstraat 35, 5280AA Boxtel, Netherlands

[b]Rompun, Haver-Mobay Corp., Animal Health Division, P.O. Box 390, Shawnee, Kansas 66201 USA; or Rompun, Bayer, Nijverheidsweg 26, 3640AB, Mijdrecht, Netherlands

[c]Type E2, Grass Inc., P.O. Box 516, Quincy, Massachusetts 02169 USA

[d]Model 1500B, Hewlett Packard, 27 Industrial Ave., Chelmsford, Massachusetts 01824 USA

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CHAPTER 21

Raptor Orthopedics Using Methyl Methacrylate and Polypropylene Rods

LAUREL A. DEGERNES, D.V.M.
PAULA J. LIND, A.H.T., B.S.
PATRICK T. REDIG, D.V.M., Ph.D.

The Raptor Center
Department of Veterinary Biology
University of Minnesota
St. Paul, Minnesota, U.S.A.

ABSTRACT: A newly developed orthopedic technique using methyl methacrylate bone cement and polypropylene welding rods has been used in 28 raptor patients from 12 species at the Raptor Center. The orthopedic technique involves injection of methyl methacrylate bone cement into the medullary cavity on both sides of the fracture site, followed by shuttling a prepared polypropylene rod before the cement hardens. Additional cerclage wires, Kirschner-Ehmer apparatus, or external coaptation may be used to increase the stability of the fracture repair. The raptors ranged in body weight from a 220-g long-eared owl (*Asio otus*) to a 4,500-g bald eagle (*Haliaeetus leucocephalus*). The fractures repaired included humerus (13), ulna (9), major metacarpal (3), femur (2), and tibiotarsus (1), and the severity ranged from simple, closed fractures to open, comminuted or segmental fractures. The fractures healed in 25 (89.3%) of the cases, and 14 of the patients (50%) were released to the wild. Of the remaining raptors, 10 regained some degree of flight capability, but were not considered suitable for release. Further, 1 case is still pending, 2 cases developed a nonunion at the fracture site, and 1 raptor died 10 days after surgery, too soon to evaluate bone healing.

KEY WORDS: orthopedics, raptor, fracture, methyl methacrylate bone cement, polypropylene rod.

Orthopedics in raptors and other wild birds can be very challenging to the veterinary surgeon. The goals of orthopedic repair in raptors are to achieve perfect reduction and alignment of the fractured bone(s), rapid healing and return to functional use of the limb, and release to the wild as quickly as possible (Redig, 1986a, 1986b). These goals are often complicated by delays in receiving the bird following injury; severe comminution and/or contamination of bone fractures; compromised vascular supply; damaged skeletal muscles, ligaments, or tendons; fractures near joints; and inadequate postoperative immobilization. In some cases, prolonged limb immobilization necessary for healing may lead to arthritic joint changes, soft-tissue scarring, and severe muscle atrophy from disuse.

Many surgical and nonsurgical techniques have been used for repair of avian fractures, including external coaptation (Redig, 1986b), intramedullary pins (Newton and Zeitlin, 1977; Redig, 1986a), external fixation devices (Bush, 1977; Redig, 1986a), bone plating (Bush et al., 1976; Leeds, 1979), intramedullary polypropylene shuttle pins (MacCoy, 1983), cerclage wires, and combinations of one or more of these techniques. Some of the problems associated with these techniques include joint trauma, fractures at the pin or screw site, and inadequate fracture immobilization. New orthopedic techniques using methyl methacrylate bone cement (MMBC) have eliminated or reduced many of these complications. Methyl methacrylate bone cement has been used alone (Borman and Putney, 1978; Borman et al., 1978; Putney et al., 1983) and in conjunction with intramedullary polypropylene shuttle pins (Lind et al., 1988, 1989; Degernes et al., 1989), stainless steel intramedullary pins (Haas and Trah, 1988), and bone plates (Kuzma and Hunter, 1989a, 1989b). The bone cement does not act as an adhesive within the medullary canal; rather, it forms a mechanical or friction bond between bone and implant device.

The purposes of this chapter are to describe the use of methyl methacrylate bone cement[a] and polypropylene rods[b] (MMBC/PR), to recommend the indications and contraindications for such use, and to summarize the cases in which this technique has been used. The procedure described here is a modification of the technique originally developed by Lind et al. (1988).

Materials and Presurgical Preparation

Materials for this procedure were prepared according to the method used by Lind et al. (1988). Briefly, the powdered

portion of a two-component, sterile, radiopaque formulation of methyl methacrylate was aliquoted into 2-g portions in a laminar flow hood. Powdered cephalothin[c] (100 mg/2g) was added and the mixture stored in parafilm-sealed sterile plastic petri dishes. The liquid monomer was transferred to a sterile 10-ml glass tube, covered with aluminum foil for light protection, and stored in the refrigerator.

In addition to the common orthopedic and surgical instruments typically used for surgery, other required materials included a 12-ml curved-tipped irrigating syringe,[d] a sterile metal mixing spatula, a 3-ml syringe with 22-gauge needle, sterile polypropylene welding rod, 30-gauge stainless steel wire, 0.035-in K-wire with a trocar point, cotton-tipped applicators, and sterile saline for irrigation.

Surgical Procedure

The surgical site was prepared for aseptic surgery. Using the standard approaches described for avian orthopedic repair (Redig and Rousch, 1978), the fracture site was exposed and the fragments mobilized to allow for manipulation of the fracture. The medullary cavities of the fragments were swabbed or curretted as necessary to remove blood clots, bone marrow, excess fibrous callous material, and internal trabecular structures that would impair shuttling the intramedullary plastic rod. A polypropylene shuttle pin of sufficient diameter to fill approximately 50-70% of the medullary cavity was selected and prepared according to the method developed by Lind et al. (1988). The pin was inserted into the medullary cavity of the longest fragment, marked, removed, and cut to this length. A hole perpendicular to the long axis was drilled at or near the center of the pin, using a trocar-tipped 0.035-in K-wire. Three to four parallel rows of barbed projections were made using a scalpel blade held at an acute angle to the long axis of the pin, with the barbs pointing toward the center of the pin. The rows of barbs should occupy a minimum of 40% of each end of the pin (Fig. 21.1). A 20 cm length of 30-gauge stainless steel wire was passed through the small hole in the center of the pin. The barbed shuttle pin was temporarily inserted into the medullary cavity of the longest fragment to check the fit, then removed and set aside.

A surgical assistant then prepared the bone cement by adding 1.2 ml liquid monomer to 2 g powder in the petri dish and mixing it thoroughly for 15 to 30 sec. It was then ladled into the open barrel of the curved-tip irrigating syringe held by the surgeon. The plunger was replaced and the bone cement was injected into the medullary cavities of both fragments. The barbed shuttle pin was completely inserted into the longest fragment, and the fracture was reduced and held in alignment while the pin was shuttled across the fracture site by pulling the cerclage wire looped through the center of the pin. After the pin was in place, the wire was removed and excess bone cement at the fracture site and on the surrounding soft tissues was removed.

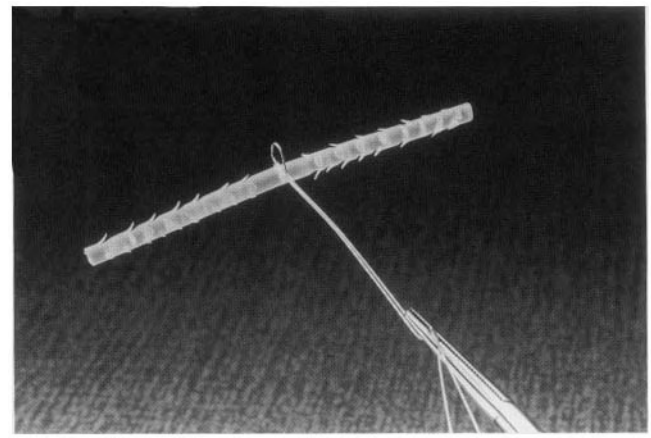

Figure 21.1. A plastic polypropylene intramedullary pin has been prepared for use with a hole drilled for a shuttle wire, and barbs have been cut into the surface.

The body temperature of the bird and the consistency of the bone cement determined the length of time for curing. In general, the mixture became too thick to work after 2 to 3 min, and was rigid within 5 to 10 min. The fracture was held in reduction until the bone cement had hardened. An exothermic reaction occurs during polymerization, but studies have shown that histologic changes attributable to thermal injury are transient and mild for the first 10 days following surgery (Putney et al., 1983). In this study, no difference was observed in the healing of fractures in which the surgical site was irrigated with cold saline during polymerization compared with those that were not irrigated. Following curing of the bone cement, the surgical site was closed and the limb was radiographed and immobilized with external coaptation.

Depending on the location and type of fracture and the temperament of the bird, the limbs were immobilized for 1 to 3 wk after surgery (Redig, 1986b). Radiographs were taken at 2, 4, and 8 wk after surgery to assess progress of healing. Use of the limb was permitted when clinical judgment warranted.

Indications, Advantages, Limitations, and Contraindications

Indications

MMBC/PR was used successfully in all avian long bones with a medullary canal, except the radius. The size of the medullary canal may limit the usefulness of this technique in birds smaller than 1,000 g. The lack of a medullary canal in the tarsometatarus prohibits the use of intramedullary pinning techniques.

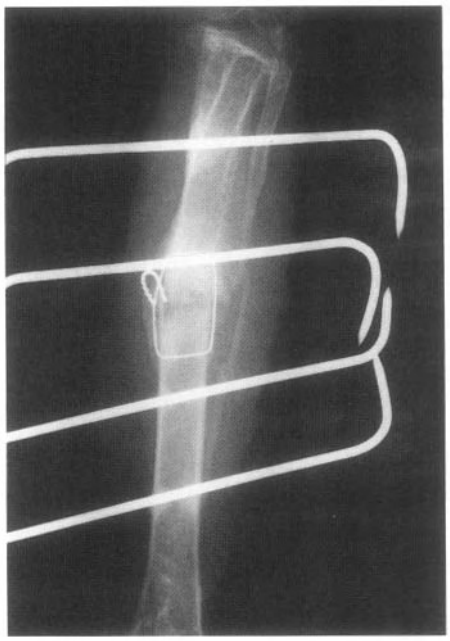

Figure 21.2. A tibiotarsal fracture in a red-tailed hawk (*Buteo jamaicensis*) was stabilized with an external fixator consisting of 0.062 in K-wires and hexcelite and an intramedullary polypropylene plastic rod.

Fractures near a joint may be successfully repaired if there is at least 1 to 2 cm of medullary canal in which to anchor the bone cement and shuttle pin. Segmental fractures may also be repaired by injecting bone cement into the two fracture ends, shuttling the polypropylene pin, and then injecting bone cement into the center segment.

For oblique, spiral, or comminuted fractures, application of three or more full cerclage wires is necessary to stabilize the fracture site further. A Kirschner-Ehmer (KE) apparatus (Redig, 1986a) was used in addition to the bone cement and polypropylene shuttle pin with a tibiotarsal fracture in a red-tailed hawk (*Buteo jamaicensis*) because additional stability at the fracture site was thought to be needed in a weight-bearing limb in a bird of this size (Fig. 21.2). External coaptation using a figure-of-eight wing bandage (with a body wrap with humerus fractures) or a Robert-Jones bandage for tibiotarsal fractures is highly recommended following surgery (Redig, 1986b).

Advantages

The advantages of this technique are (1) the rigid internal stability achieved, (2) the ability to stabilize fractures near joints without interfering with the joint integrity, (3) the reduction in the length of time needed for external coaptation, and (4) the rapid return to functional use of the limb. Another advantage is that there is no need for a second surgery to remove hardware (except when a KE apparatus is used). The reduction of time required for bandaging minimizes disuse atrophy of the muscles of the affected limb and shortens the rehabilitation process. The risks of secondary captive-management problems developing, such as pododermatitis or feather trauma, are thus reduced. Reduction in convalescent time in raptors such as accipiters or bald eagles is especially critical due to their self-destructive behavior while in captivity.

Limitations

The semiflexible polypropylene rods do not provide much resistance to lateral forces applied at the fracture site. Fractures that are overriding with extensive muscle contraction, or transverse fractures of the proximal femur or of the curved portion of the proximal humerus, have a tendency to bow out at the fracture site when polypropylene pins are used, making necessary external stabilization using a KE apparatus or a more rigid intramedullary pin (for example, a stainless steel intramedullary pin or K-wire) with the bone cement. This problem contributed to implant failures in two cases discussed below. Fractures with longitudinal fissures should be stabilized with three or more full cerclage wires to prevent breakdown of the fracture along the fissure lines.

Contraindications

MMBC is contraindicated in septic wounds; however, most of the open fractures in this group were undoubtedly contaminated. Pre- and post-op antibiotics and the addition of cephalothin to the powder may help to reduce problems with osteomyelitis. Human studies have shown antimicrobial activity for more than 20 days after surgery when cephalothin is added to the bone cement (Marks et al., 1976).

Clinical Case Discussion

MMBC/PR was used in a variety of raptor species and fracture types (Table 21.1). Humeral fractures were the most frequent subjects (n = 13), followed by fractures of the ulna (n = 9), major metacarpal (n = 3), femur (n = 2), and tibiotarsus (n = 1). Fracture healing was achieved in 89% (n = 25) of the attempts. All of the birds regained some degree of flight capability; however, 10 of the 24 could not fly well enough to be released to the wild and either were placed in captive programs for breeding or education or were euthanized.

Nearly 50% of the cases involved repair of a humeral fracture (Table 21.2). The MMBC/PR proved ideal in this application because of the excellent rotational stability afforded by the internal fixation. Of 13 humeral fractures, 10 healed (76.9%), and 4 of these birds were released to the

Table 21.1
Summary of 28 orthopedic cases managed with plastic shuttle pins and bone cement

Number	*Species*	*Bone*	*Location of fracture*	*Wound condition*	*Type of fracture*	*Age of fracture*	*Surgical management*	*Time to union (weeks)*	*Resolution (months)*
1	BDO	F	P	C	T	A	3SPBC/KE/IM	8	Pending
2	BDO	H	M	O	C	A	3SPBCW	—	DS1.5
3	BDO	H	M	C	O	B	3SPBCW	6	PD (retinal injury)
4	BDO	MC	D	O	O	A	2SPBCW	4	RL4
5	BDO	U	M	C	C	B	3SPBCW	5	RL4
6	BDO	U	D	C	O	B	3SPBCW	6	RL7
7	BE	H	M	C	O	A	8SPBCW	4	RL2.5
8	BE	MC	M	C	T	A	5SPBC	8	PD (unrelated elbow injury)
9	BE	U	D	O	C	B	5SPBCW	4	PD (self-inflicted injuries)
10	BWH	U	M	C	T	B	3SPBC	2.5	RL4
11	CSH	U	P	C	C	A	3SPBC	3	DS5
12	GHO	TT	P	C	C	C	3SPBCKE	4	RL5
13	GHO	U	P	C	C	B	5SPBC	5	RL6
14	GHO	U/R	M	C	C	B	3SPBCW	4	PD (arthritis)
15	GHS	H	M	C	O	B	3SPBCW	—	DS2
16	HAR	H	M	C	T	B	3SPBC	3	DS3
17	HAR	H	P	O	C	A	3SPBC	3	DS3
18	LEO	H	P	C	T	A	3SPBC	2	RL7
19	PEF	F	M	C	T	A	3SPBC	2	RL0.5
20	PEF	H	M	C	C	B	3SPBCW	—	DD (10 days post-op)
21	PEF	H	M	C	C	B	3SPBC	4	PD
22	PEF	U/R	M	C	T	A	3SPBCW	6	RL6
23	PRF	U	M	O	C	A	3SPBC	4	RL
24	RLH	H	P	C	T	A	3SPBC	3.5	RL2
25	RTH	H	M	C	C	B	3SPBCW	3	PD
26	RTH	H	M	O	O	C	3SPBCW	10	RL6
27	RTH	H	M	O	O	C	5SPBCW	12	DD (unrelated causes)
28	RTH	MC	M	C	C	A	2SPBC	10	RL

Note: Standard recognized species abbreviations are used here.
Bone: H = humerus, U = ulna, R = radius, MC = metacarpus, F = femur, TT = tibiotarsus.
Location: P = proximal, M = midshaft, D = distal.
Wound condition: O = open, C = closed.
Type: O = oblique, T = transverse, C = comminuted.
Age of fracture: A = <3 days, B = 4-10 days, C = >10 days.
Surgical management: Number = pin size (mm), SP = shuttle pin, BC = bone cement, W = cerclage wire, KE = Kirschner-Ehmer device, IM = intramedullary pins.
Time to union: determined by palpation and radiography.

wild (30.8%). Soft-tissue trauma associated with the original injury accounted for failure to achieve adequate function in the nonreleasable birds.

Of the nine ulna fractures repaired, seven did not have a concurrent radial fracture. Many cases involving a single ulna or radius fracture may be handled conservatively with external coaptation (Redig, 1986b). The decision to manage these cases surgically was made on the basis of the severity and location of the fracture and the temperament of the bird. All nine ulnar fractures healed, and six (66.7%) of these birds were released to the wild. Two of the nonreleasable raptors had joint-related damage that prevented adequate return to function, and one bald eagle developed self-destructive behavior shortly before he was to be released, which led to permanent damage to the carpometacarpal joints.

Three major metacarpal fractures were successfully repaired, and two of the birds (66.7%) were released. Union was delayed in two birds, which may have been caused by the lack of soft tissues covering these bones. Surgical repair of metacarpal fractures should be reserved for highly unstable fractures, since manipulation of the soft tissues may impair the vascular supply, leading to delayed or nonunion fracture healing.

MMBC/PR was used in only three cases of leg fractures. Two of these birds healed uneventfully and were released to the wild. The third case is flying but awaiting feather molt

Table 21.2
Resolution of avian fractures repaired with plastic shuttle pins and bone cement, based on fracture location

Fractures repaired	*Released to wild*	*Healed, but nonreleasable*	*Nonunion Fx*	*Pending*	*Died*
Humerus (13)	4	6	2		1
Ulna (9)	6	3			
Metacarpal (3)	2	1			
Femur (2)	1			1	
Tibiotarsus (1)	1				
Total (28)	14	10	2	1	1

prior to release. In one of these three cases, a tibiotarsal fracture in a great horned owl was provided additional stabilization with a KE. A KE may not be necessary in smaller patients, but further data should be collected to verify this assumption. In another case, involving a 44-day-old peregrine falcon rescued from a storm-toppled hacking tower, a femoral fracture was repaired with this technique and the falcon was returned to the repaired hack tower at 2 wk postop, from which it fledged normally. Prolonged recovery in captivity, which would have been necessary with other femoral-fixation techniques, would have precluded returning this bird to a hack site before it was too old to hack successfully.

Three failures of MMBC/PR technique were documented, including one femoral and two humeral fractures. One case involved an open humerus fracture with extensive soft-tissue trauma in a barred owl (*Strix varia*), where the vascular supply to the bone was probably disrupted. A second case occurred in a goshawk (*Accipiter gentilis*) with an old overriding proximal transverse humeral fracture. The lateral forces applied by the contracted muscles resulted in the polypropylene pin breaking at the site of the drilled hole. A KE device was applied in a second procedure, but the bird subsequently refractured the humerus at one of the KE pin sites and was euthanized (Fig. 21.3). A third case involved a transverse fracture of the proximal femur in a barred owl. The polypropylene pin fractured within one week of surgery. A KE device was applied, but failed when the wires pulled loose from the bone. The fracture was finally successfully managed with the placement of two intramedullary steel pins.

A retrospective study including band-return information would be useful to compare the outcome of bone cement orthopedic techniques to traditional avian orthopedic techniques. However, based on our experiences, there are many advantages for using methyl methacrylate bone cement in avian fractures.

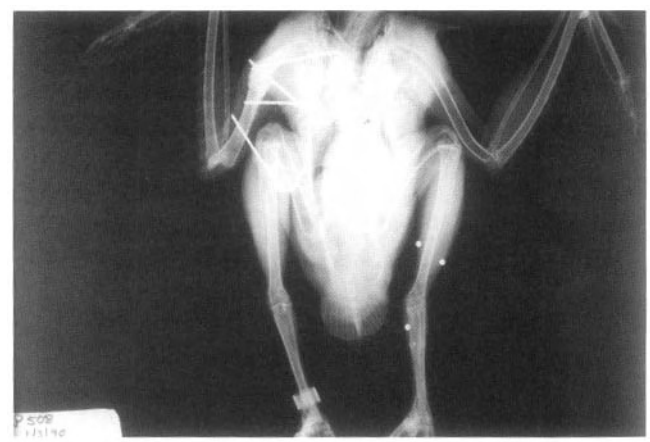

Figure 21.3. This MMBC/PR preparation with an external fixator on a humerus failed when the goshawk refractured the bone at one of the KE pin sites.

Acknowledgments

We would like to acknowledge the dedication and excellent care of Diane Olson, Lori Mueller, Barb Talbot, Jean Dunnette, Mark Martell, and the many Raptor Center volunteers in the rehabilitation of these raptors. Thanks also to the staff and volunteers of Carpenter Nature Center, Hastings, Minnesota, for their assistance in readying many of the patients for release to the wild.

Product List

[a]Surgical Simplex P, Howmedica, 359 Veterans Blvd., Rutherford, New Jersey 07070-2584, USA

[b]Commercial Plastics Inc., 601 N. Prior Ave., St. Paul, Minnesota, 55104 USA

[c]Keflin, Eli Lilly and Co., Lilly Corporate Center, Indianapolis, Indiana 46285 USA

[d]Monoject, Division of Sherwood, 1915 Olive St., St. Louis, Missouri 63103 USA

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CHAPTER 22

Inhalation Anesthesia in Birds of Prey

GUY FITZGERALD, M.Sc., D.V.M.
DIANE BLAIS, D.V.M.
Faculté de médecine vétérinaire
Université de Montréal
Montréal, Canada

ABSTRACT: The last decade has seen considerable improvement in avian anesthesia. Although several safe injectable anesthetic agents are available, inhalant anesthesia has several major advantages: rapid and smooth induction and recovery, excellent control of the depth and duration of anesthesia, and flexibility in regard to unfamiliar species. In this chapter, different inhalation agents and techniques are discussed, with emphasis on isoflurane anesthesia. Aspects of avian anatomy and physiology relevant to anesthesia are also reviewed. Finally, the importance of preanesthetic care and monitoring is discussed.

KEY WORDS: anesthetic techniques, raptor, inhalant anesthesia, preanesthetic care, avian respiratory physiology.

There is an increasing worldwide concern about birds of prey at several levels: rehabilitation, breeding programs, biomedical or fundamental research, and falconry. Raptor medicine requires an expertise in anesthesia for many routine diagnostic and therapeutic procedures. At the Faculté de médecine vétérinaire of the Université de Montréal, approximately 90% of the birds of prey admitted (more than 300 annually) through a provincial raptor rehabilitation network (Union québécoise de réhabilitation des oiseaux de proie) are anesthetized at least once. Many of these birds are in poor physical condition, and thus safe anesthetic techniques and adequate monitoring are prerequisite to success.

There are two major types of anesthesia, local and general. Anesthesia is used to relieve pain by producing analgesia and to provide chemical restraint for a diagnostic or therapeutic procedure. General anesthesia, achieved by either injectable or inhalation drugs, should be characterized by analgesia, unconsciousness, absence of voluntary movement, and muscle relaxation.

Some authors do not recommend the use of local anesthesia in birds (Hall and Clarke, 1983, pp. 354-357; Allamargot and Faublée, 1984). However, the toxicity of these drugs is similar to that seen in mammals, and toxic effects could be prevented through the use of appropriate concentrations and volumes (Klide, 1973; Coles, 1985). Nevertheless, physical restraint in the absence of unconsciousness may produce excessive stress and struggling (Linn and Gleed, 1987).

Several safe injectable anesthetic techniques exist (Camburn and Stead, 1978; Fedde, 1978; Kollias and McLeish, 1978; Kirkby, 1979; Altman, 1980; Haigh, 1981; Redig, 1982; Coles, 1985; Cooper, 1985; Paddleford, 1986; Taylor, 1987). They do not require expensive equipment, and IV induction is rapid. Supplemental oxygen can be administered, if needed, by endotracheal tube following induction of anesthesia. Individual and species responses to injectable anesthesia are variable, making it more difficult to control anesthetic depth and duration (Haigh, 1981; Elkins and Herron, 1982; Gandal and Amand, 1982; Allamargot and Faublée, 1984; Cooper, 1985; Harrison, 1986; Linn, 1986). Repeated doses, administered to prolong the duration of anesthesia, enhance the risk of complications (Hartsfield and McGrath, 1986; Sedgwick, 1986). Although antagonist drugs are currently being investigated in raptors (Allen and Oosterhuis, 1986; Degernes et al., 1988; Freed and Baker, 1989), the effects of an overdose of any of the commonly used injectable anesthetics cannot be reversed at the present time. Parenteral anesthetics have to be metabolized and excreted, which may lead to prolonged and sometimes violent recovery.

Recent advances in inhalation anesthesia and a better understanding of avian respiratory physiology now render this technique a method of choice in avian anesthesia. Although more expensive, inhalation anesthesia has more predictable effects among species or individuals and allows individual control of the depth of anesthesia. Usually, general anesthesia can be achieved without deleterious effects to the bird. A smooth and rapid recovery and better control of the duration of anesthesia are also major advantages of inhalation compared with injectable anesthetics (Frost, 1981; Farny and Lernould, 1985; Sedgwick, 1986; Mandelker, 1987; Taylor, 1987). This is particularly true for isoflurane.

Anatomical and Physiological Considerations

Birds have a very high metabolic rate (Arnall, 1961; Farner, 1982; Cooper, 1985; Ringer, 1986; Carter-Storm, 1987). This helps them to eliminate drugs rapidly, but may also increase the effects of toxic metabolites such as those produced by methoxyflurane[a] and halothane.[b] However, the basal metabolic rates of raptors and eutherian mammals are almost the same (Schmidt-Nielsen, 1984). Raptors possess relatively high glucogenic reserves. Plasma-glucose levels remain relatively high (> 330 mg/dl) in medium to large raptors even after a fasting of 24 hr, possibly because of a higher gluconeogenic capacity combined with low glucose utilization (O'Donnell et al., 1978).

The avian respiratory system is very different from that of mammals. It provides the most efficient gas exchange of all vertebrates (King and McLelland, 1984; Heard, 1988) and consequently a more rapid uptake and elimination of inhalant anesthetics. This highly specialized system depends on a nondistensible lung communicating with nine air sacs, eight paired and one single (Fig. 22.1). There are aspects of the anatomy and the physiology of the avian respiratory system that should be understood in order to enhance safe inhalation anesthesia techniques (Farner, 1982; King and McLelland, 1984; Nicolas and Brugère, 1984; Fedde, 1986; Ringer, 1986; Taylor, 1987).

Avian tracheal cartilages are full rings. If an endotracheal tube is used, special care should be taken not to damage the trachea by inflating the cuff of the endotracheal tube after intubation. Preferably, a cuffless tube should be used. In birds as in mammals, lungs are used to eliminate body heat and water. Thus, the anesthetic delivered in oxygen may contribute to hypothermia and dehydration of the bird during a lengthy anesthetic episode. Some authors suggest warming and moisturizing the inhaled gas to prevent these complications (Cooper, 1985; Evans, 1985; Carter-Storm, 1987). The gas-exchange surfaces (paleopulmonic and neopulmonic parabronchi) of the lungs are exposed to fresh air and anesthetic agent at most stages during inspiration and expiration. Indeed, the airflow is unidirectional throughout inspiration and expiration in the paleopulmo (Fig. 22.1). Furthermore, a cross-current arrangement between the gas flow in the parabronchi and the blood flow in the blood capillaries contributes to the efficiency of gas exchange. The humerus and the femur are pneumatic bones, and their fracture will allow escape of the anesthetic agent. Respiration is facilitated when the bird is kept on its breast or on its side when recumbent (Coles, 1985).

Inhalant Anesthetics

Several inhalation agents can be used to induce anesthesia in birds. Certain of these are no longer in use. For instance,

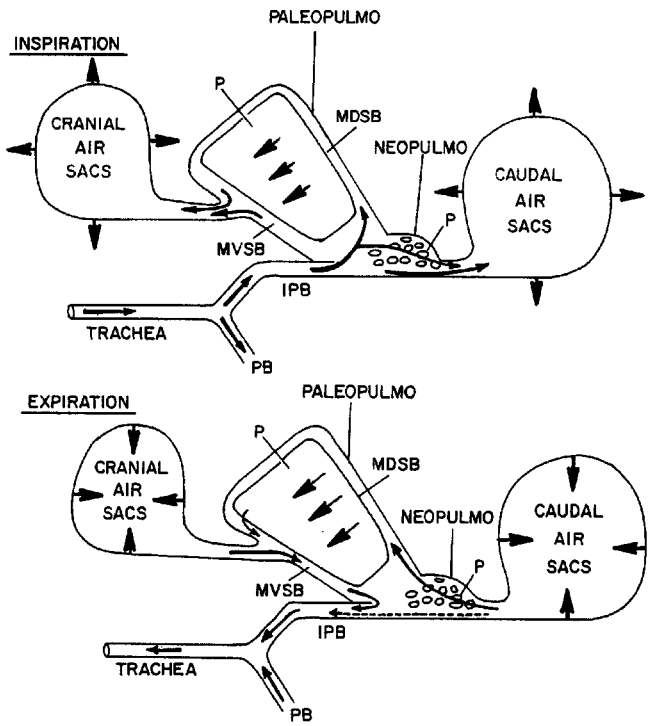

Figure 22.1. Air pathways in the avian respiratory system. Gas-exchange surfaces (parabronchi of the paleopulmo and the neopulmo) are exposed to fresh air and thus to anesthetic during most of the inspiration and expiration. PB = primary bronchus; IPB = intrapulmonary primary bronchus; MDSB = mediodorsal secondary bronchus; MVSB = medioventral secondary bronchus; P = parabronchi; caudal air sacs = abdominal and caudal thoracic air sacs; cranial air sacs = cervical, clavicular, and cranial thoracic air sacs (Farner, 1982; King and McLelland, 1984; Fedde, 1986).

chloroform should be used only for euthanasia; cyclopropane and ether are too highly explosive to provide a safe technique. Use of methoxyflurane is diminishing and highly discouraged. Halothane and isoflurane[c] are the noninflammable volatile agents currently in use in avian anesthesia. Different properties of methoxyflurane, halothane, and isoflurane are summarized in Table 22.1.

Methoxyflurane, a halogenated ether, produces long induction and recovery times compared with halothane and isoflurane. The use of the open drop or cotton technique with any of the existing inhalation agents can no longer be justified because of the ready availability of more appropriate and safer techniques (Harrison, 1986; Heard, 1988). When methoxyflurane is delivered through its specific vaporizer, the concentration must be increased gradually during the mask induction period. Anesthesia is usually maintained at about 1.0% methoxyflurane (Heard, 1988). Methoxyflurane, similar to the other inhalation anesthetics, produces dose-dependent cardiorespiratory depression. Respiratory depression is the most likely complication during and following methoxyflurane anesthesia, however. The

Table 22.1
Properties of inhalant agents

Properties[a]	*Methoxyflurane*	*Halothane*	*Isoflurane*	*Comments*
Vapor pressure (mm Hg, 20°C)	22.8	244.1	239.5	
Solubility coefficients (37°C)				It is better to have a low solubility of agent in
blood/air	15	2.5	1.4	the blood, for rapid induction and recovery;
fat/air	902	166	74	the body fat, for faster recovery;
rubber/air	630	120	62	the rubber parts of the circuit for faster induction and recovery.
Analgesia	+ + +	+	+ +	
Potency				
MAC in mammals	0.1-0.3%	0.7-1.0%	1.3-1.6%	MAC value represents the minimal alveolar concentration at which 50% of the individuals will not respond to a noxious stimulus. MAD represents the minimal anesthetic dose equivalent to MAC (Ludders et al., 1987, 1989).
MAD in birds	–	0.75-0.98%	1.32-1.34%	
Metabolism	43-50%	12-20%	0.2%	Minimal metabolism of the agent is desired because metabolites are often hepato- or nephrotoxic.
Myocardial sensitization to catecholamines	+/–	+	–	Sensitization increases the risk of catecholamine-induced cardiac arrhythmia.
Preservative	required	required	not needed	Preservatives may accumulate in the vaporizer and alter the calibration.
Toxic effects	liver, kidney	liver	not reported to date	Toxicity is enhanced by the high metabolic rate of birds.
Cost (Can$/ 100ml)	82	12	94	

[a]Eger (1985).

metabolites of methoxyflurane, fluoride ions, are nephrotoxic. Thus, this agent should not be used in combination with potentially nephrotoxic drugs, such as the aminoglycoside antibiotics, or in the presence of renal disease (Taylor, 1987). In the human, chronic low exposure to methoxyflurane has been associated with health problems (Harrison, 1986; Short, 1986; Taylor, 1987).

Halothane is a halogenated hydrocarbon. The induction and recovery periods are shorter than with methoxyflurane. Hypotension, bradycardia, and hypothermia are often observed during halothane anesthesia (Altman and Miller, 1979; Altman, 1980; Evans, 1985; Linn and Gleed, 1987). In spontaneously breathing birds, there is a narrow margin between respiratory arrest and cardiac arrest (Harrison, 1986; Taylor, 1987). Halothane is potentially hepatotoxic (McKeever, 1980; Harrison, 1985, 1986; Heard, 1988). Myocardial sensitization to endogenous catecholamines released during such circumstances as a stressful induction is possible when using halothane (Farny and Lernould, 1985; Taylor, 1987). A gradual increase of the halothane concentration up to 2.0% is recommended for mask induction of anesthesia (Farny and Lernould, 1985; Heard, 1988).

Isoflurane is a methyl ethyl ether, regarded by several authors as the safest volatile anesthetic agent currently available (Bednarski et al., 1985; Eger, 1985; Pascoe et al., 1985; Harrison, 1985, 1986; Clutton, 1986; Hartsfield and McGrath, 1986; Linn, 1986; Short, 1986; Janssen, 1987; Taylor, 1987; Werner, 1987; Heard, 1988). Harrison (1986) recommends the use of isoflurane for anesthetizing critical patients. It may be used in birds with hepatic or renal pathology, as to date no toxic metabolites have been identified. The relatively broad margin between respiratory arrest and cardiac arrest is an interesting quality (Harrison, 1986). Janssen (1987) reports that isoflurane gives the most rapid and complete recovery of any of the anesthetic agents available. Indeed, it is not uncommon to observe a bird eating less than 30 min after the end of anesthesia. Consequently, isoflurane is an excellent anesthetic agent, even for short procedures such as radiography.

Mask induction of anesthesia can be achieved smoothly with isoflurane by a gradual increase in the inspired concentration. Taylor (1988a) has reported variable responses in different species or individuals. However, more than 3.0% isoflurane is seldom necessary to induce anesthesia and allow endotracheal intubation (Heard, 1988). As the vapor pressure is similar to that of halothane, the use of a halothane-specific vaporizer is possible (Steffey et al., 1983). However, alternate use of these two different agents in the same vaporizer is contraindicated. The minimum anesthetic dose of isoflurane has been determined by Ludders

et al. (1986, 1987) in Pekin ducks (1.32%) and in sandhill cranes (*Grus canadensis*) (1.34%). The disadvantages of the use of isoflurane are hypotension and marked respiratory depression (Harrison, 1986; Short, 1986; Taylor, 1987; Werner, 1987). Intermittent positive pressure ventilation may have to be provided. Ludders et al. (1987, 1989) emphasize the marked disparity in cardiorespiratory function between ventilated and unventilated birds.

Nitrous oxide (N_2O), an anesthetic gas, has a low solubility in blood and fat, is not metabolized (Sedgwick, 1986), and does not cause cardiorespiratory depression. Because it cannot induce anesthesia when used alone, it must be combined with a volatile agent. The second-gas effect of N_2O accelerates the induction of anesthesia. In addition, combining N_2O with a volatile agent permits a reduction in the inspired concentration of the volatile agent, thus decreasing the cardiorespiratory depression produced by this agent (Sedgwick, 1986). To avoid hypoxia, it is preferable not to exceed the ratio 1:1 of N_2O:O_2 (Haigh, 1981; Coles, 1985; Evans, 1985; Paddleford, 1986). Heard (1988) suggests the use of a minimal total flow of 1.5 l/min (0.75 l/min O_2 and 0.75 l/min N_2O) with a nonrebreathing system. The use of nitrous oxide is not recommended in birds with respiratory problems (Hartsfield, 1982; Heard, 1988). Administration of N_2O should be discontinued 15 min before the end of anesthesia. The air sacs are not considered as closed gas spaces because they communicate freely with the airways of the lungs, so N_2O will not cause their expansion (Bednarski et al., 1985; Heard, 1988).

Preanesthetic Care

Before general anesthesia, a complete physical examination should be performed on any patient. A minimal data base consisting of packed cell volume (PCV), total solids, and blood glucose should be established (Steiner and Davis, 1981; Heard, 1988; Mandelker, 1988).

An intravenous catheter should be placed as soon as possible after the induction of anesthesia to allow the administration of fluid or emergency drugs. A solution of lactated Ringer's at a rate of 22 ml/kg/hr is recommended (Hartsfield and McGrath, 1986). Smaller birds may receive the same treatment in small IV boluses before and after the surgery at a rate of 10 ml/kg (Janssen, 1987). A bird with a PCV in excess of 55% should be rehydrated before the beginning of anesthesia. A PCV of less than 20% justifies postponing the anesthesia and considering a whole-blood transfusion. If a donor bird of the same species is not available, pigeon blood may be transfused once for a heterologous blood transfusion (Altman, 1982). A blood-glucose level below 200 mg/dl indicates hypoglycemia, and a solution of 5% dextrose in water should be administered intravenously before anesthesia (Altman, 1980).

Food should be withheld for 4 to 6 hr prior to anesthesia to ensure an empty crop (Haigh, 1981), prevent liquid regurgitation, and facilitate respiration. Indeed, a distended stomach will occupy an important volume of the abdominal cavity, thus compressing the air sacs. Considering the energy reserves of raptors in good physical condition (O'Donnell et al., 1978), a 12-hr fast should not be harmful.

The use of atropine as a preanesthetic is controversial. Many researchers recommend its administration to decrease the production of saliva and respiratory secretion and to diminish the risk of cardiac arrhythmias (Klide, 1973; Altman, 1980; Kollias, 1982; Harrison, 1985). In fact, its use may produce thicker secretions that are more likely to obstruct the endotracheal tube (Linn and Gleed, 1987). In addition, methoxyflurane, halothane, and isoflurane do not induce hypersalivation. An undesirable tachycardia may occur with the administration of atropine (Farny and Lernould, 1985). If bradycardia occurs during the anesthesia and does not respond to a decrease in the anesthetic concentration, atropine could be administered IV.

Tranquilizers may be required in nervous birds such as accipiters to reduce excitation and to facilitate restraint for face-mask induction. For pet birds, Heard (1988) suggests diazepam[d] at 2 mg/kg (< 500 g body weight [BW]) and 1 mg/kg (> 500 g BW) or midazolam[e] at 1.5 mg/kg (< 500 g BW) and 0.8 mg/kg (> 500 g BW) administered IV or IM.

Anesthetic Equipment and Technique

The standard equipment for inhalation anesthesia includes a specific precision vaporizer; a source of oxygen and nitrous oxide; flow meters for oxygen and nitrous oxide; and a breathing system including a reservoir bag, a manometer, a security (pop-off) valve, face masks of different sizes, and Cole or uncuffed Murphy endotracheal tubes of appropriate sizes. For field use, a portable modular anesthetic machine with an isoflurane vaporizer, an oxygen flow meter, a nonrebreathing Bain system, and an aluminum oxygen cylinder can be used (Taylor, 1988b).

The reservoir bag allows manual positive pressure ventilation. Hartsfield and McGrath (1986) suggest assisting the ventilation once or twice every minute. A pressure-controlled mechanical ventilator may also be used to assist or control the respiration (Sedgwick, 1979). The peak positive pressure in birds is at 12-24 cm H_2O (Sedgwick, 1980; Clutton, 1986; Hartsfield and McGrath, 1986; Carter-Storm, 1987; Janssen, 1987).

A nonrebreathing system such as a Bain or an Ayre's T-piece is recommended in birds because of their low airway resistance. Taylor (1987) prefers the Bain circuit because the expired gas flow warms and humidifies the delivered fresh air. A circle breathing system may be used for birds weighing more than 5 kg (Hartsfield and McGrath,

1986), and the Bloomquist infant circle system may be used in smaller raptors (Wingfield and DeYoung, 1972; Hartsfield, 1982; Wallach and Boever, 1983; Hartsfield and McGrath, 1986). An alternative technique consists of delivering the anesthetic directly through a needle or a catheter inserted in one or two air sacs. However, this method usually provokes apnea, and close monitoring of the heart is then very important (Whittow and Ossorio, 1970; Linn and Gleed, 1987; MacCoy, 1988).

Nonrebreathing systems necessitate a minimal oxygen flow of 660 ml/kg/min (Hartsfield, 1982; Hartsfield and McGrath, 1986; Janssen, 1987; Linn and Gleed, 1987), whereas the circle system necessitates 33 ml/kg/min (Hartsfield, 1982; Hartsfield and McGrath, 1986). To assure accurate delivery of anesthetic concentrations by the vaporizer, the oxygen flow meter should be set to at least 300 ml/min (Hartsfield, 1982; Hartsfield and McGrath, 1986). An oxygen flow of 0.5-2 l/min is recommended to avoid mixture of the agent with room air in the event of a pneumatic bone fracture or a punctured air sac (Guiraud, 1978, 1981; Genevois et al., 1983; Pionneau, 1985; Clutton, 1986).

Anesthesia can be induced either by a volatile anesthetic using a face mask or by an injectable drug. However, in the latter case, the synergistic effects of the different anesthetic agents should be considered. For instance, in mammals, the cardiovascular effect of xylazine[f] may be potentiated by a volatile anesthetic, resulting in severe bradycardia (Sedgwick, 1986). For anesthesia of short duration, the use of an injectable induction agent may prolong the recovery period. To induce anesthesia, a gradual increase in the inspired concentration of a volatile agent, such as 0.5% every 30 seconds, is preferable to the use of a constant high concentration of the agent (Wingfield and De Young, 1972; McKeever, 1980; Haigh, 1981; Kollias, 1982; Coles, 1985; Taylor, 1987, 1988a; Heard, 1988). It is also important to reduce stress during the induction to avoid the release of endogenous catecholamines, which may result in severe cardiac arrhythmias (Hartsfield and McGrath, 1986). For this reason, endotracheal intubation of a conscious bird is not recommended (Sedgwick, 1980).

Endotracheal intubation should always be performed during general anesthesia, even with parenteral anesthetics, since it allows control of ventilation in case of apnea, decreases the fresh-gas flow requirements, diminishes the dead space, and prevents liquid aspiration (Altman, 1980; Farny and Lernould, 1985; Hartsfield and McGrath, 1986; Heard, 1988). The largest possible diameter of endotracheal tube should be used to avoid an increased resistance to the airflow. Tubes smaller than 2.0 mm internal diameter are not recommended, because they increase airway resistance and can easily become obstructed (Farny and Lernould, 1985). In addition, the length of the endotracheal tube should be such as to minimize dead space (Altman, 1980; Heard, 1988). A short period of apnea may be observed following the intubation if the bird is too lightly anesthetized (Coles, 1985). In bald eagles (*Haliaeetus leucocephalus*), we have frequently noticed a transient period of apnea after endotracheal intubation.[1]

The recovery period is a critical part of the anesthesia. It is important to administer oxygen for at least 5 min after the inhalant anesthesia has ceased in order to eliminate the anesthetic agent from the air sacs. Then the bird should be placed in a warm and quiet cage. The recovering bird may be loosely wrapped in a towel and placed recumbent on its breast. An incubator providing a supplement of oxygen and heat (32-35°C) is suggested for the recovery of the critically ill bird (Altman, 1980; Kollias, 1982; Cooper, 1985). The patient should be continuously monitored until alert enough to stand.

Monitoring

Monitoring the anesthetized bird is an essential part of any general anesthetic procedure. The clinical parameters used to evaluate the depth of anesthesia are summarized in Table 22.2. These are guidelines, and must be assessed together, not individually. For instance, one should not conclude that the level of anesthesia is too deep based only on the loss of the corneal reflex. Other parameters must also be considered. Rate, depth and rhythm of the respiration, and response to pain (plucking of a feather) are probably more reliable signs of the plane of anesthesia (Sanford, 1971). The respiration should be slow, regular, and deep. The respiratory rate of a raptor under general anesthesia is between 10 and 16 breaths per min, depending on the size of the bird (Paddleford, 1986).

Several techniques that allow a more accurate monitoring of the anesthetized bird are also available. For instance, an electronic sensor inserted between the endotracheal tube and the breathing system allows audible monitoring of respiration (Coles, 1985; Cooper, 1985). A pneumogram may be obtained by using an impedance transducer and subcutaneous needles connected on each side of the thorax. Capnometry is a technique used in human and domestic animal anesthesia to monitor the expired end-tidal CO_2 (Moens and Verstraeten, 1982). The utility of its application in avian species remains uncertain, but deserves investigation.

Heart rates of anesthetized raptors vary between 120 to 240 bpm for medium-sized birds and 80 to 140 bpm for larger ones (Haigh, 1981; Hartsfield, 1982; Evans, 1985; Paddleford, 1986). Standard or esophageal stethoscopes are often used to monitor heart rate and rhythm. An electronic esophageal stethoscope that amplifies the audible signal of the heartbeat is also available. The ultrasonic Doppler allows audible monitoring of arterial blood flow (Kollias,

Table 22.2
Parameters monitored during different planes of inhalation anesthesia

	Plane of anesthesia			
Parameters	*Light*	*Medium*	*Deep*	*Comments*
Presence of voluntary movements	−	−	−	Fluttering may occur with painful surgical stimulation, even in light-medium plane of anesthesia.
Pattern of respiration	rapid, deep regular	slow, deep regular	slow, shallow, regular to irregular or apnea	Respiration is the most reliable indication of depth of anesthesia.
Presence of muscle tone				
neck	−	−	−	
toes	−/+	−	−	Relatively good indication of plane of anesthesia.
Presence of palpebral reflex	+/−	−	−	The palpebral and corneal reflexes are generally less reliable indicators of depth of anesthesia (Sanford, 1971; Steiner and Davis, 1981; Wallach and Boever, 1983).
corneal reflex	+	+/−	−	
pedal	+/−	−	−	The pedal reflex is less reliable in raptors than in other birds (Haigh, 1981).
Response to feather plucking	+	+/−	−	
Other observations		This plane of anesthesia is desired for most surgical procedures.	At this plane of anesthesia heart rate is slow, pulse is weak, and capillary refill time is slow.	

Source: Adapted from Arnall (1961).

1982; Clutton, 1986; Taylor, 1987; Heard, 1988). The electrocardiograph is another accurate monitoring technique and is used to detect cardiac arrhythmias. Silver-platinum needles placed subcutaneously on the limbs are less traumatic to birds than alligator clamps.

Measurement of direct arterial blood pressure and analysis of arterial blood gases are more sophisticated monitoring techniques. In larger birds, systolic arterial blood pressure can be measured by an indirect technique using an ultrasonic Doppler (Kollias, 1982; Heard, 1988). The pressure cuff must be of adequate width, approximately 40% of the circumference of the limb (Chalifoux et al., 1985). Measurement of direct arterial blood pressure involves an invasive technique and is thus more often used in research. The measure of the arterial blood gases and acid-base analysis give an accurate evaluation of the cardiorespiratory effects of the anesthetic (Kollias and McLeish, 1978; Neal et al., 1981; Kollias, 1982; Hartsfield and McGrath, 1986; Ludders et al., 1986, 1987) and can be compared with values in unanesthetized birds (Fedde, 1986).

Body temperature must be maintained during general anesthesia. Hypothermia can occur because of a depression of the thermoregulatory center by the anesthetic agent, heat loss via the respiratory system, muscle relaxation, absence of shivering, and peripheral vasodilation (Farny and Lernould, 1985). Thus, it is important to control heat loss during surgical preparation by minimizing feather plucking and the use of alcohol. The use of a circulating warm-water blanket is highly recommended (Carter-Storm, 1987; Heard, 1988).

Conclusion

There is no ideal anesthetic technique. It is a matter of experience and individual preference. Some procedures may require injectable agents. However, most of the time, inha-

lation anesthesia, particularly using isoflurane, offers major advantages. The time and money spent on endangered species such as some raptors justify the cost of these anesthetic techniques.

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Note

1. Editor's note: Application of a local anesthetic agent to the glottis or the end of the endotracheal tube eliminates this problem.

Product List

[a]Metofane, Pitman-Moore/MTC, 5100 Timberlea Blvd., Mississauga, Ontario, Canada L4W 2S5

[b]Fluothane, Ayerst, C.P. 6115, Montréal, Québec, Canada H3C 3J1

[c]AErrane, Anaquest, 2000 Argentia Rd., Mississauga, Ontario, Canada L5N 1V8

[d]Valium, Hoffmann-LaRoche, 401, West Mall, Etobicoke, Ontario, Canada M9C 5J4

[e]Versed, Hoffmann-LaRoche, 401, West Mall, Etobicoke, Ontario, Canada M9C 5J4

[f]Rompun, Bayvet/Haver, 1351 Matheson Blvd., Mississauga, Ontario, Canada L4W 2A1; or Bayer, Nijverheidsweg 26, 3640 AB Mijdrecht, Netherlands

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CHAPTER 23

Fracture Healing in Predatory Birds, with Special Consideration Given to a Modified External Fixator

FRITZ GRIMM, Ph.D.

Institute of Avian Diseases
Ludwig-Maximilians University
Munich, Germany

ABSTRACT: The care of bone fractures in predatory birds using intramedullary pins, plates, or stretch-support bandages does not always lead to desired results, especially with old or infected fracture sites, extensive bone and soft-tissue damage, or fractures near joints. This chapter evaluates a modified external fixator (Schanz screw) that can be employed on long tubular bones in birds weighing more than 600 g. In smaller birds the use of Schanz screws is not advantageous.

KEY WORDS: external fixator, orthopedics, Schanz screw, fracture.

The treatment of fractures of the long bones in birds can be achieved by different methods. For long tubular bones, intramedullary stabilization with various wires or pins has enjoyed popularity. Fixation with plates has gained widespread acceptance in the treatment of noninfected fractures and in corrective osteotomies, particularly in birds of more than 1,000 g body weight. Fracture treatment, especially of tarsometatarsal bones, may also be performed by means of suitable slings (Grimm, 1978; Redig, 1986; Grimm and Kösters, 1988).

External fixation is used when opening the fracture site is not appropriate, when an implant at the fracture site would hinder the healing process, or when infection in open fractures might be transmitted to noninfected areas by the technique. Treatment with external fixation is also the method of choice for fractures near joints.

The goals of this study were (1) to test patient tolerance of a modified external fixator, the Schanz screw[a] (Figs. 23.1, 23.2, and 23.3), (2) to assess the healing reaction of the patient bearing such a device, and (3) to test the mechanical strength of bones healed while stabilized with such a device.

Material and Methods

In previous studies on chickens, buzzards (*Buteo buteo*), and crows (*Corvus frugilegus* and *Corvus corone*), it was determined which version of the external fixator should be used and how a usable operative procedure for the avian patient should look (Grimm and Siebels, 1987). In addition, measurements of stiffness and stability were taken on roosters, chickens, and buzzards to which the external fixator had been applied, as already tested on the rabbit model by Siebels et al. (1986). The Schanz screws on the roosters and chickens were 2.5 and 2.0 mm, respectively, and 2.0 mm in buzzards and crows. The connecting bar, made of carbon-fiber-reinforced synthetics (CFK) with a 4 mm diameter and aluminum joints, was sufficiently stable for all types of birds tested in the study reported here.

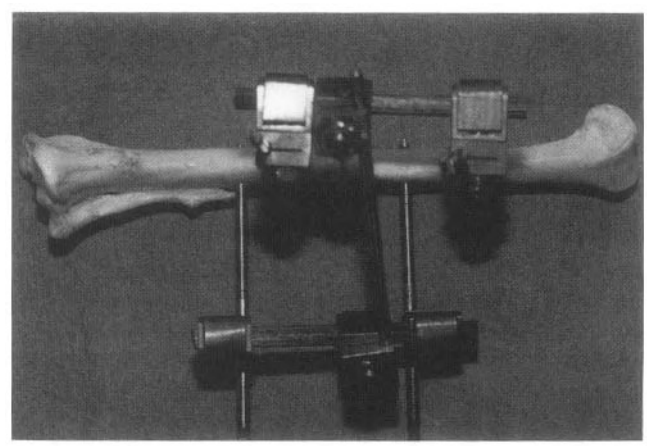

Figure 23.1. Frontal view of an external fixator affixed to a tibiotarsal bone. Note the connection of lateral and anterior frames with a bar.

By using connecting bars of synthetics (CFK) and aluminum joints, the weight of the fixator was reduced, for example, from 83.7 g to 31.6 g for the buzzard.

Surgical Procedure

After marking the location for the Schanz screw on the skin of the right tibiotarsal bone, the bone's diameter was determined with the needles inserted on both sides. A stab incision was made between the marked points with a scalpel. The tibiotarsal bones with diameters of 5.6 to 10 mm (buzzard and chicken) were drilled centrally and the Schanz

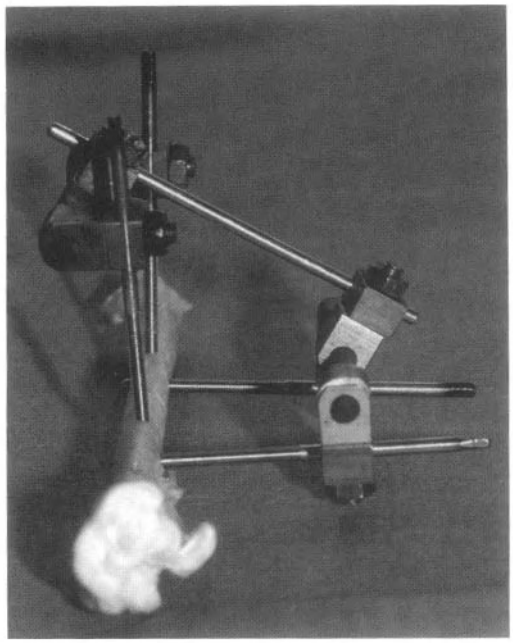

Figure 23.2. Top view of the external fixator depicted in Fig. 23.1.

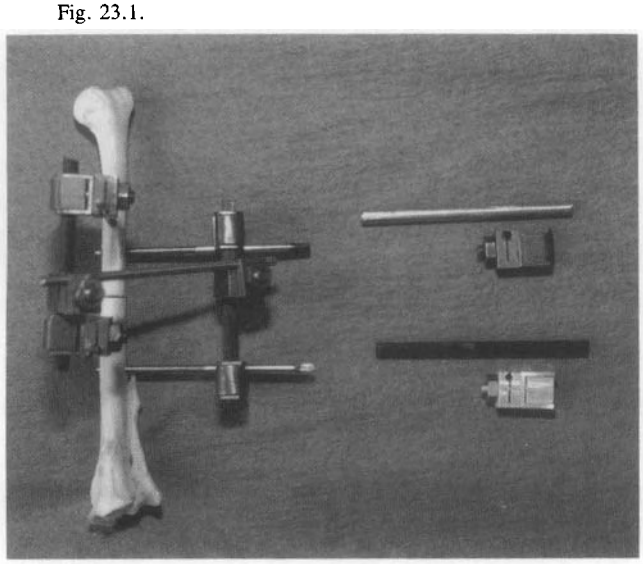

Figure 23.3. Components of an external fixator.

screw, 2.0 or 2.5 mm, inserted. Two screws were inserted in the anterior-posterior direction, so that they protruded 10 mm on the posterior side. The two lateral screws, inserted at 90° angles to the other two screws, were screwed in until they touched the opposite cortex.

The fixators with the aluminum compression joints and carbon-fiber-reinforced connecting rods were then attached. In addition, the tibia was exposed ventromedially via a small incision in the skin and then osteotomized in the middle of the shaft with a saline-cooled oscillating saw,[b] 0.4 mm thick. The wound was then routinely closed. The fixator

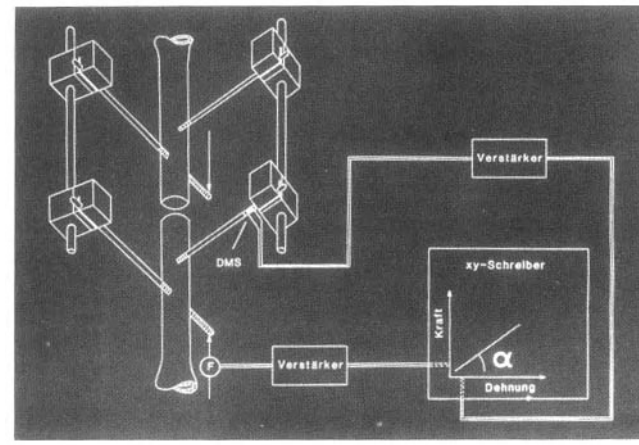

Figure 23.4. Diagram showing the devices used to measure strength of bone during the healing process. Verstärker = amplifier, xy-Schreiber = x-y plotter, Kraft = force, Dehnung = deformation, F = forceps, DMS = foil strain gauge.

so applied resulted in an initial axial compression stiffness of 15 Newton/mm in chickens.

Postoperative care was initially restricted to daily dressing change, cleansing of the protruding Schanz screws, and covering of the incision with wound powder[c] or wound spray.[d] Partial weight bearing was observed after 3 days postoperative, and full loading after 14 to 30 days. For 2 wk, every 2 days, calcium[e] at 2 ml/kg body weight and a multivitamin preparation[f] at 2 ml/kg body weight were injected IM into the *M. pectoralis profundus*.

Radiological Examination

Immediately following surgery, and then at weekly intervals, standard radiographs of the right tibiotarsal bones were taken in two planes (antero-posterior and lateral). The radiographs were employed during the experiment to aid in recognizing complications, such as fractures and/or loosening of screws. This was to ensure that a valid signal was obtained in the mechanical test, and that healing did not take place under abnormal biomechanical conditions.

Mechanical Testing

On the day of the operation, a foil strain gauge[g] with a measuring grid length of 0.6 mm was attached to the distal lateral Schanz screw near the fixator clamp. In order to measure stiffness *in vivo* and to load the osteotomized tibia in axial compression and tension, a forceps was attached to the posterior ends of both perforated screws (Fig. 23.4). By this means the manually induced force could be registered against the bending of the Schanz screws on an x-y plotting recorder (Fig. 23.5). Measurements were taken every 3

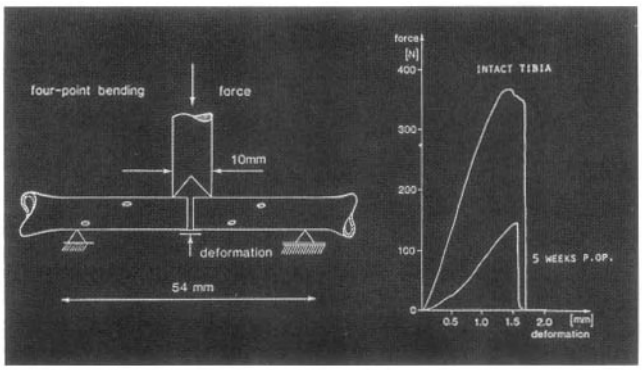

Figure 23.5. Graph showing the relationship between force and deformation of bone at 5 wk post-op.

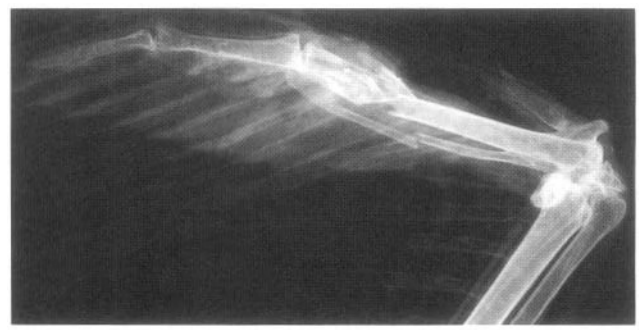

Figure 23.6. Radiograph showing a fracture of the metacarpus of a crane (*Grus grus*) caused by a gunshot.

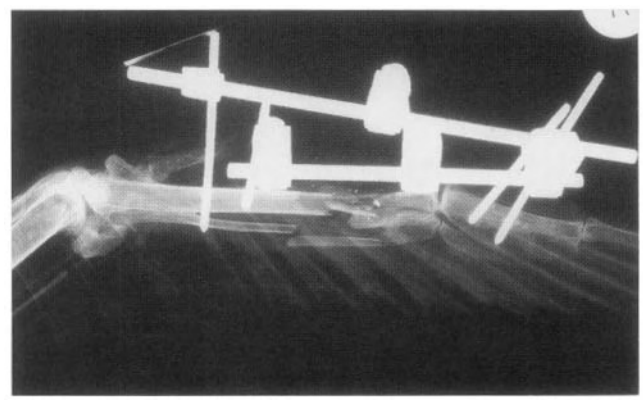

Figure 23.7. The metacarpal fracture in Fig. 23.6, stabilized with an external fixator.

days, every 2 days, and then every day at the end of the measurement.

As healing proceeded, the load transfer through the newly formed bone increased, hence the bending of the Schanz screws decreased, with comparable values of force applied through the forceps. This was reflected in an increased gradient of the force-strain curve. As the curve approached the vertical, the measuring method became invalid because even the use of extreme force no longer led to bending of the Schanz screw, and therefore did not generate a measurable signal; rather, stiffness of the bone still increased. On day 35 post-op, birds were euthanized with chloroform. All soft tissue was removed from both tibiotarsal bones, which were immediately tested to failure in a four-point bending test.

Results and Discussion

The usual fracture fixation devices, such as intramedullary wires or plate osteosynthesis, are not suited for fractures of long bones with massive shattering, for example, from gunshot wounds; for fractures near the joint; for fractures with large surface damage to the soft tissue; or for infected fractures. Even conservative procedures with stretch-support bandages or extension with a Thomas splint are often not suitable or fail for anatomical reasons.

The desire to fix the bone from the outside without affecting the wound area is traditional and has led to the construction and further development of a series of apparatuses that are used on both humans and mammals.

There is also the desire in the case of birds to treat fractured bones from the outside to bring about healing. The central problem was and still is, however, the stability of the structure while allowing weight bearing. The use of percutaneous osteofixation has already been described numerous times (Bush, 1977, 1982; Satterfield and O'Rourke, 1981; MacCoy, 1981; Redig, 1986). In all of these works, Kirschner wire in the form of a frame fixator was used, penetrating both sides. Frame fixators, because of their spatial structures, are limited to stabilizing relatively few avian fracture types. Their use is also limited because animals resist long-term confinement.

The employment of Schanz screws makes the use of a clamp fixator possible. This is less space-consuming than a K-wire frame fixator and is therefore universally applicable, even when a second layer is added at a different angle.

By using Schanz screws with a 2.0 mm diameter, it becomes possible to stabilize long tubular bones in birds of more than 600 g body weight. In the 400 to 600 g weight class, it is risky to use such Schanz screws, according to our own previous experiments. Here the use of weaker, penetrating drill wires in frame models, such as in Bush (1982) and Satterfield and O'Rourke (1981), supplied sufficient stability.

Data about the optimal strength of the drill wires or of the Schanz screws are nonexistent or imprecise in current literature. Bush (1982) finds appropriate the use of drill wires of 2.03 mm for the care of fractures of long tubular bones in medium-size and large predatory birds. But in the experiments performed here it was found that smaller screws created fewer complications. However, the stability of a 2.0 mm screw in the clamp model was insufficient for roosters

Figure 23.8. Gross view of the fixator device applied to the metacarpal fracture in Fig. 23.6.

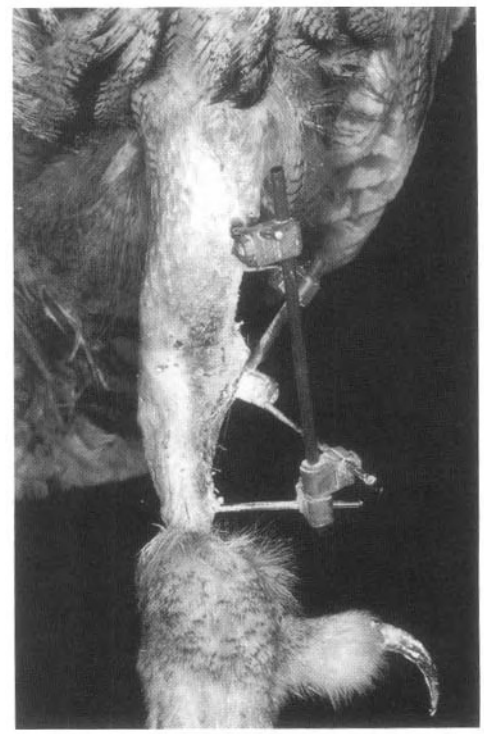

Figure 23.10. Gross view of the external fixator as applied to the tibiotarsal fracture in Fig. 23.9.

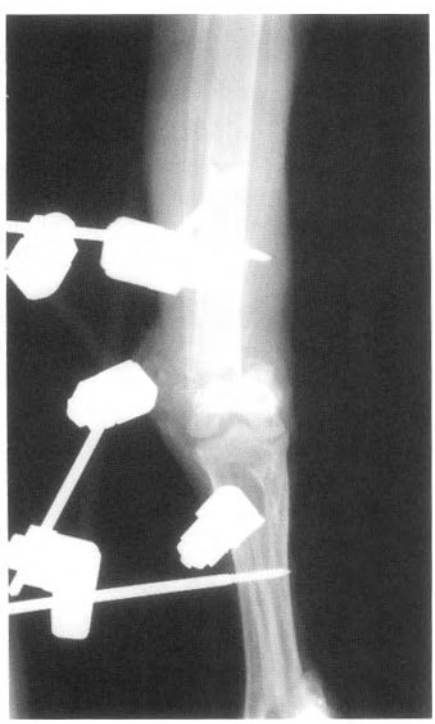

Figure 23.9. Radiograph showing a distal tibiotarsal fracture in an eagle owl repaired with an external fixator.

with a 2,000 g body weight. Swans with a humerus diameter of 16 and 18 mm bent the 2.0 and 2.5 mm Schanz screws, and an eagle owl (*Bubo bubo*) with a tibiotarsus diameter of 10 mm bent the 2.5 mm screws.

Bush's (1982) frame model, a two-sided fixation with 2.03 mm pins, showed itself to be stable even in cranes. But the 2.03 mm size pin that was used for a goshawk humerus would be far too weak for large predatory birds, for example, the sea eagles. This is true especially in the rear extremities, where there can be a considerable amount of strength in large predatory birds. At our institute a golden eagle (*Aquila chrysaetos*) weighing 3,600 g severely bent a compression plate fixed to the tibiotarsus.

Oehme and Kitzler (1975) calculate a 10- to 20-fold higher value for the endurance muscle power of various birds compared with the muscles of mammals. It can be concluded from their calculations that even for a short time, a considerably greater amount of force is applied to the bones of birds compared with those of mammals.

According to the experiments conducted here, the diameter of the screw, drilling wires, or pins must be 30% of the bone diameter for the modified external fixator, so that the stability of the fixation device surpasses the muscle-strength effect. Figures 23.6 to 23.10 show birds treated with modified external fixation.

Product List

[a]AO/ASIF-Minifixator, Synthes, Davos, Switzerland

[b]Aesculap, 7200 Tuttlingen, Germany

[c]Cicatrex-Puder, Wellcopharm, 3006 Burgwedel 1, Germany

[d]Leukospray Beiersdorf, 2000 Hamburg, Germany

[e]Calcium Sandoz 10%, Sandoz AG, Basel, Switzerland

[f]Tricrescovit, Rentschler Merieux, 8874 Leipheim, Germany

[g]Hottinger und Baldwin, 7000 Stuttgart, Germany

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CHAPTER 24

Immobilization of Raptors with Tiletamine and Zolazepam (Telazol)

TERRY J. KREEGER, M.S., D.V.M., Ph.D.

Department of Ecology and Behavioral Biology
University of Minnesota
Minneapolis, Minnesota, U.S.A.

LAUREL A. DEGERNES, D.V.M.

The Raptor Center
University of Minnesota
St. Paul, Minnesota, U.S.A.

JULIE S. KREEGER, D.V.M.

Department of Veterinary Biology
University of Minnesota
St. Paul, Minnesota, U.S.A.

PATRICK T. REDIG, D.V.M., Ph.D.

The Raptor Center
University of Minnesota
St. Paul, Minnesota, U.S.A.

ABSTRACT: Five great horned owls (*Bubo virginianus*) were given either 5 or 10 mg/kg of tiletamine and zolazepam (Telazol) intramuscularly (IM). Heart rates (HR) and respiratory rates (RR) were measured before and at 1, 5, 10, 15, and 20 min after anesthetic induction. Induction times were 8.8 ± 3.2 versus 3.6 ± 0.4 min for the 5 and 10 mg/kg dose, respectively ($p = 0.12$), and times to standing were 68.8 ± 8.1 versus 87.0 ± 6.4 min, respectively ($p = 0.16$). Inductions and recoveries were smooth. Heart rates for owls given 10 mg/kg Telazol were higher ($p < 0.03$) than for owls given the lower dose and tended to decrease over time, whereas HR at the lower dose remained constant. There was no difference in RR between groups ($p = 0.70$). Respiratory rates also decreased for the initial 20 min of anesthesia. Total recovery times for owls given the higher dose was 246.2 ± 36.6 min. Two screech owls (*Otus asio*) were also given 10 mg/kg Telazol IM. Induction time was 2.1 ± 0.6 min, and the time to when they first raised their heads was 61.5 ± 1.5 min. Total recovery time was greater than 5 hr. Three red-tailed hawks (*Buteo jamaicensis*) were given 10 mg/kg, 15 mg/kg, or 20 mg/kg, and one was given 40 mg/kg Telazol IM. None of these doses induced loss of consciousness. Both HR and RR increased during the test, and there was no difference between doses for HR ($p = 0.59$) or RR ($p = 0.36$). Recoveries were prolonged and characterized by catalepsy, opisthotonos, and ataxia. Thus, Telazol at a dose of 10 mg/kg provided satisfactory chemical restraint for at least 30 min in the two species of owls, but was not a satisfactory immobilizing agent in red-tailed hawks at any of the doses given.

KEY WORDS: tiletamine, zolazepam, anesthesia, raptor, great horned owl, *Bubo virginianus*, red-tailed hawk, *Buteo jamaicensis*, screech owl, *Otus asio*.

Telazol[a] (TEL) is an injectable anesthetic comprising equal weights of the arylcycloalkylamine, tiletamine hydrochloride (HCL), and of the pyrazolodiazepinone, zolazepam HCL. Tiletamine is an analogue of both phencyclidine and ketamine, having a relative potency of one-half that of phencyclidine and 2.5 times that of ketamine (Beck, 1972). When used alone, tiletamine produces convulsive seizures and clonic muscular reactions, while zolazepam alone causes aggressive behavior (Massopust et al., 1973). The combination of these two drugs, however, results in reduced convulsions, good muscle relaxation, and smoother recoveries (Massopust et al., 1973). Telazol is currently approved only for use in dogs and cats, but during its development over the last 15 years, TEL (previously identified as CI-744) was used on more than 200 nondomestic vertebrate species, including 37 avian species (Gray et al., 1974; Schobert, 1987). Controlled clinical evaluations, however, have been conducted only on dogs, cats, primates, and rodents. Evaluation of its use in birds is further limited by small samples (often $n = 1$) as well as use of

Table 24.1
Induction and recovery times of great horned owls anesthetized with either 5 or 10 mg/kg Telazol administered intramuscularly

Dose (mg/kg)	*Induction time (min) (mean ± SE)*	*Head up time (min) (mean ± SE)*	*Time to standing (min) (mean ± SE)*
5	8.8 ± 3.2	31.5 ± 5.6	68.8 ± 8.1
10	3.6 ± 0.4	42.2 ± 6.0	87.0 ± 6.4
p value	0.12	0.24	0.16

injured or otherwise compromised subjects (Gray et al., 1974).

The purpose of this study was to assess the efficacy, physiological responses, and safety of TEL on raptors under controlled conditions.

Materials and Methods

These studies were conducted in July-September 1988 in east-central Minnesota. All birds used were nonamputee permanent cripples housed either individually in cages or in 3.0 m × 4.0 m flight rooms. Birds were fed whole rodents daily and maintained on a 16:8 day:night cycle. All birds had been in captivity for at least 6 mon.

Telazol was reconstituted with 5 ml sterile water to provide a concentration of 50 mg/ml tiletamine and 50 mg/ml zolazepam. Injections were given IM in the breast muscle. Birds were weighed before the start of each trial. Induction time (IT), head-up time (HUT), and time to standing (ST) were recorded for each trial. Induction time was the time from injection of TEL to loss of consciousness; HUT was the time from injection to when the bird first opened its eyes and moved its head; ST was the time from injection to when the bird was first able to support itself in an upright posture.

Upon induction, birds were fitted with electrocardiogram (ECG) electrodes in a lead II configuration[b] and ECGs were taken at 1, 5, 10, 15, and 20 min after induction. Respiratory rates (RR) were visually counted at the same times.

Statistical analyses were by one-way analysis of variance (ANOVA) and ANOVA for repeated measures at a significance level of $p < 0.05$. Means are reported with standard errors (SE).

Group 1

Five great horned owls (GHO) (*Bubo virginianus*) of unknown sex were given either 5 or 10 mg/kg TEL IM at least one week apart. All birds received both doses. Preanesthetic ECGs and RR were taken in addition to the times listed above.

Group 2

Five red-tailed hawks (RTH) (*Buteo jamaicensis*) of unknown sex were given 5, 10, or 20 mg/kg TEL IM at least one week apart. Due to removal of some of the birds during the study, only three birds were tested at each dosage. In addition, one RTH was given 40 mg/kg TEL IM. No preanesthetic ECGs or RR were taken.

Group 3

Two screech owls (SO) (*Otus asio*) were given 10 mg/kg TEL IM. No ECGs or RR were taken due to the small sample size.

Results

Group 1

Both doses of TEL resulted in a rapid and smooth anesthetic induction with good muscle relaxation. Although not statistically different, the 10 mg/kg dose resulted in a shorter IT, but longer HUT and ST (Table 24.1). Anesthesia caused decreased heart rates (HR) for both doses of TEL, but the HR for the 10 mg/kg dose remained higher throughout the test ($p = 0.03$; Fig. 24.1). The HR was significantly lower than preanesthesia HR by 5 min postadministration ($p = 0.004$). Respiratory rates were likewise decreased upon induction but were not different between doses ($p = 0.70$; Fig. 24.2). Recoveries were uneventful and similar to recoveries from other cyclohexane drug combinations (e.g., ketamine and xylazine). Total recovery time for the 10 mg/kg was subjectively judged to be 246 ± 37 min.

Group 2

None of the four doses of TEL resulted in anesthesia in RTH. At the least, the state could be described as chemical restraint, as birds were conscious but ataxic and unable to perform coordinated, directed movements. Because the birds were never anesthetized, recovery times were deemed meaningless. Evidence of consciousness was supported by the sustained elevations of both HR and RR (Figs. 24.3, 24.4). These rates were similar to HR (416.5 ± 16.7 bpm) and RR (49.0 ± 3.8 breaths/min) of unanesthetized RTH measured in another study (Degernes et al., 1988). There

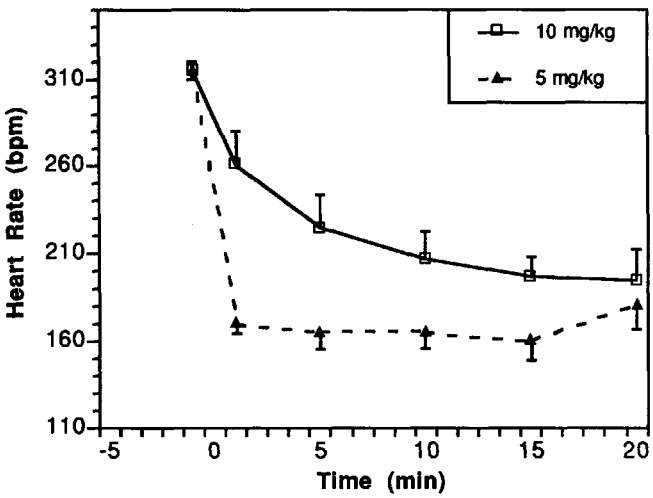

Figure. 24.1. HR of great horned owls anesthetized with either 5 or 10 mg/kg Telazol administered IM. Heart rate measured at the 10 mg/kg dose was significantly higher than at the lower dose (p = 0.03).

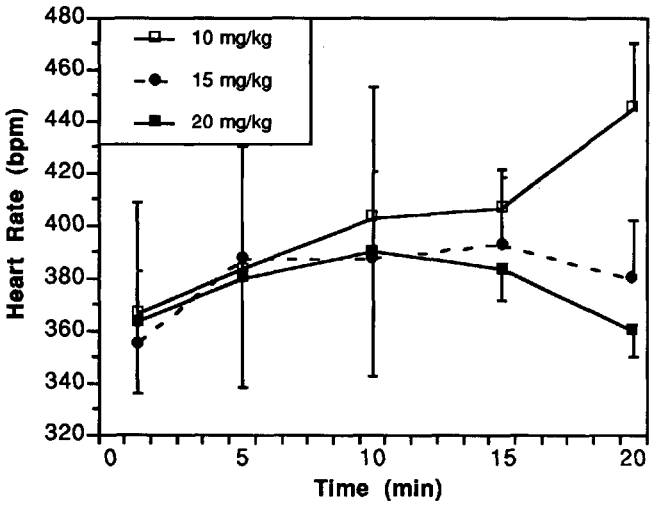

Figure 24.3. HR of red-tailed hawks given 10, 15, or 20 mg/kg Telazol. There were no differences in heart rates among doses (p = 0.59).

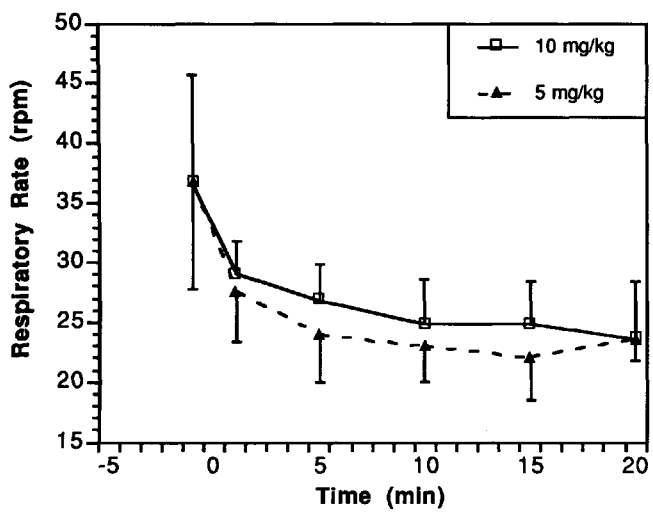

Figure 24.2. RR of great horned owls anesthetized with either 5 or 10 mg/kg Telazol administered IM. There was no difference in respiratory rates between doses (p = 0.70).

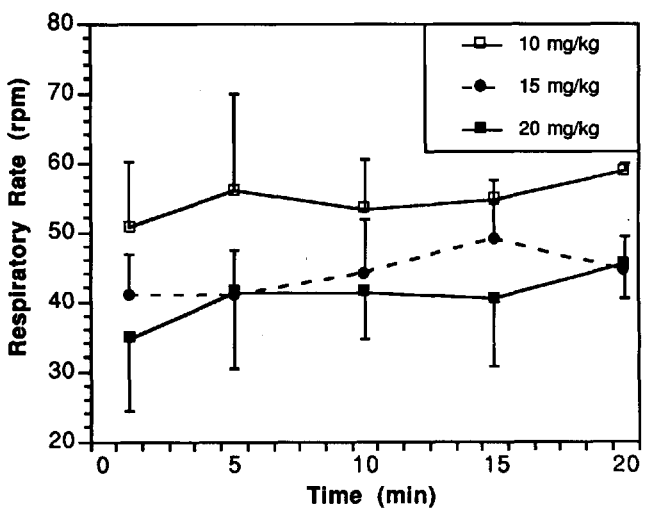

Figure 24.4. RR of red-tailed hawks given 10, 15, or 20 mg/kg Telazol. There were no differences in respiratory rates among doses (p = 0.36).

was no difference between doses for either HR (p = 0.59) or RR (p = 0.36). The birds reacted to both sound and movement throughout the test. Salivation and lacrimation were noted in at least one RTH at all dosages. Recoveries were generally characterized by ataxia, nystagmus, tremors, wing flapping, and opisthotonos.

Group 3

Telazol caused a rapid and smooth induction in both screech owls. Induction time was 2.1 ± 0.6 min and HUT was 61.5 ± 1.5 min. Neither bird was standing 4 hr after injection of TEL, but both were normal the following day.

Discussion

Group 1

Both doses of TEL provided satisfactory chemical immobilization in GHO. Both doses adequately supported cardiovascular and respiratory function. The relatively higher HR noted at the 10 mg/kg dose was most likely a function of the positive chronotropic properties of tiletamine (Chen et al., 1969). For nonpainful procedures of short duration, the 5 mg/kg dose should prove adequate. For surgical procedures of longer duration, at least 10 mg/kg should be used. If additional boosters of TEL are given to maintain anesthesia, recovery times will be extended. Although boosters were not tested in raptors, work with gray wolves has shown that boosters of ketamine at one-half the initial tiletamine dose

will satisfactorily maintain anesthesia and result in reduced recovery times, compared with tiletamine boosters (Kreeger et al., 1990).

Group 2

None of the TEL doses given RTH provided even satisfactory restraint, let alone anesthesia. The profound salivation could result in aspiration. Parasympatholytic agents, such as atropine, were not given to control salivation. However, the positive chronotropic effects of atropine (Adams, 1982) could be contraindicated in the presence of the existent tachycardia. The sustained, elevated HR coupled with the rapid RR suggested that the birds were aware of their surroundings. As demonstrated in Group 1, HR would be expected to decrease if the drugs had a sedative effect. Hawks anesthetized with ketamine and xylazine had HR of approximately 100 bpm 10 min after injection (Degernes et al., 1988). It is also possible that the increasing heart rates were due to a synergistic effect of the chronotropic properties of tiletamine coupled with the activation of the sympathetic nervous system (Porges, 1985). Thus, even if the chemical restraint imposed by TEL could be clinically useful in RTH, the humaneness of the use of any drug that causes akinesia without loss of consciousness is questionable.

Group 3

Although our sample size was small, our data indicate that TEL at a dose of 10 mg/kg provides excellent anesthesia in SO. Recovery periods were relatively prolonged and might have been reduced by using a lower dose.

Conclusion

We found TEL to be an effective and safe chemical immobilizing agent for both GHO and SO at doses of either 5 or 10 mg/kg. It is not a satisfactory agent for RTH, and other drug combinations have been shown to be more efficacious and potentially safer (Redig and Duke, 1976; Degernes et al., 1988).

Acknowledgments

We would like to thank Diane Olson for her assistance in this project and the Small Animal Clinical Sciences Department, College of Veterinary Medicine, University of Minnesota, for the use of the equipment. This work was supported by the Raptor Center, University of Minnesota, St. Paul, Minnesota.

Product List

[a]Telazol, A. H. Robins Co., 1407 Cumming Dr., Richmond, Virginia 23220 USA

[b]Datascope 871 Monitor, Datascope Corp., 14 Philps Parkway, Montvale, New Jersey 07645 USA

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CHAPTER 25

Effects of Ketamine-Xylazine Anesthesia on Adrenal Function and Cardiac Conduction in Goshawks and Pigeons

J.T. LUMEIJ, D.V.M., Ph.D., Dipl. Int. Med. Comp. Animals (R.N.V.A.)

Division of Avian and Exotic Animal Medicine
Department of Clinical Sciences of Companion Animals
Utrecht University
Utrecht, Netherlands

ABSTRACT: The effects of the anesthetic combination ketamine-xylazine in respective doses of 50 mg/kg and 4 mg/kg intramuscularly in goshawks (*Accipiter gentilis*) and racing pigeons (*Columba livia*) are reported. In goshawks this dose rate led to mortality, while in pigeons no satisfactory anesthesia could be induced, since marked excitatory behavior could be provoked by manipulation of the animals. In both species a marked atropine responsive sinus bradycardia accompanied by first-degree atrioventricular block was noted. It was demonstrated that the anesthetic combination ketamine-xylazine does not cause a reduced output of corticosterone by the adrenal gland. Use of ketamine-xylazine in the dose used in this experiment is not recommended for use in racing pigeons and is life threatening for goshawks.

KEY WORDS: raptor, goshawk, *Accipiter gentilis*, racing pigeon, *Columba livia*, ketamine, xylazine, anesthesia.

The combination of the anesthetic agents ketamine[a] and xylazine[b] is reported to be useful for anesthesia in diurnal birds of prey. Reported dosage regimens for intramuscular administration of these agents, however, vary considerably. Ketamine doses range between 2.5 and 170 mg/kg and doses of xylazine vary between 2 and 10 mg/kg (Borst and Vroege, 1972; Cooper, 1978, pp. 143-155; Gandal and Amand, 1982; Haigh, 1981; Steiner and Davis, 1981; Van Nie, 1972). The aims of this chapter are to document two fatalities that occurred in goshawks (*Accipiter gentilis*) after administration of the ketamine-xylazine combination in the medium dose range published and to present some experimental studies that were performed to clarify the cause of death in these two cases.

In both goshawks the deaths occurred as the birds were coming out of the anesthetic state. The times of deaths bear some resemblance to deaths reported in humans associated with etomidate narcosis (Finlay and McKee, 1982; Wagner and White, 1984) and with deaths reported in dogs associated with xylazine narcosis (Pascoe, 1985; Muir et al., 1975).

Increased mortality rates after etomidate narcosis in humans are associated with low plasma corticosteroid levels caused by etomidate-induced supression of adrenocortical response (Finlay and McKee, 1982; Wagner and White, 1984). Human patients receiving etomidate are incapable of responding to preoperative or postoperative stressful situations and show no adrenocortical response to ACTH stimulation. It was hypothesized that the same might occur in birds anesthetized with ketamine-xylazine, as Chiasson et al. (1973) have reported that poultry anesthetized with ketamine exhibit reduced weight and a lower number of cells throughout the interrenal tissue of their adrenals. These authors suggest that this might lead to some dramatic physiological changes in adrenal function. No functional studies of the avian adrenal gland after the use of this anesthetic have been reported, however. That was one of the aims of the present study.

In dogs, xylazine produces significant cardiovascular depression and sensitizes the myocardium to adrenalin-induced arrhythmias (Pascoe, 1985; Muir et al., 1975; Sands, 1981). When adrenalin is released due to stress in the recovery period, cardiac arrhythmias can occur. The second aim of the present study therefore was to investigate the effect of ketamine-xylazine anesthesia on cardiac conduction in birds. The effect of atropine on heart rate was also studied in a limited number of anesthetized birds.

Case Reports

The first case was a 13-week-old female goshawk that was presented with a fractured tibiotarsus. The bird was anesthetized with ketamine-xylazine in respective doses of 50 mg/kg and 4 mg/kg to treat the fracture. About 4 hr after anesthesia was induced, the bird regained some consciousness, but remained in sopor until the next morning when she died, about 24 hr after anesthetization.

The second case was an 8-year-old female goshawk that

was presented with a lesion on the rostral part of the tongue. A mucosal fold originating from the lingual frenulum was wrapped around the apex of the tongue and caused difficulties with eating. The bird was anesthetized in the same way as the first, and the mucosal fold was surgically removed. About 24 hr after anesthesia was induced, the bird had regained consciousness but remained drowsy. She had no strength in her legs and was unable to perch. This situation lasted until 50 hr after induction, when the bird died.

Experiments in Pigeons

Materials and Methods

Six healthy racing pigeons (*Columba livia*) and one goshawk, which was blind in one eye, were used as experimental animals. All birds were anesthetized with ketamine and xylazine at doses of 50 mg/kg and 4 mg/kg, respectively. An ACTH stimulation test was performed 4 hr after inducing anesthesia. Blood samples were collected before and 90 min after IM administration of synthetic $ACTH_{1\text{-}24}$[c] at a dose of 250 μg/kg. Plasma corticosterone concentrations were determined in these samples by radioimmunoassay (Lumeij et al., 1987). The response of the anesthetized pigeons to ACTH was compared with results obtained in a previous study in which ACTH was administered to unanesthetized pigeons (Lumeij et al., 1987). Electrocardiograms (ECGs) were recorded before and every 30 min during anesthesia. Electrocardiographic findings in the pigeons were compared with reference values published previously (Lumeij and Stokhof, 1985). Because reference values for ECG and ACTH stimulation tests in goshawks are lacking, and because only one goshawk was available, this bird served as its own control. Control values in this bird were established one week before the experiment.

In addition to the above, doses of atropine of 0.05, 0.5, and 1 mg/kg were administered to two pigeons anesthetized with the aforementioned mixture, and electrocardiographic effects were monitored.

Results

Plasma corticosterone concentrations in the anesthetized pigeons rose from baseline values of 1.47 ± 0.97 mg/dl (mean ± SD) to 14.57 ± 3.6 mg/dl in the poststimulation sample. In unanesthetized pigeons, plasma corticosterone changed from 0.27 ± 0.15 mg/dl to 8.23 ± 2.04 mg/dl (Lumeij et al., 1987). Thus, both baseline and poststimulation corticosterone concentrations were significantly higher in the pigeons that were anesthetized with ketamine-xylazine ($p < .01$).

In the unanesthetized goshawk a baseline corticosterone concentration of 5.2 mg/dl was measured, which rose to 25.6 mg/dl after stimulation with ACTH. During ketamine-xylazine anesthesia these concentrations were 2.34 mg/dl and 18.6 mg/dl, respectively.

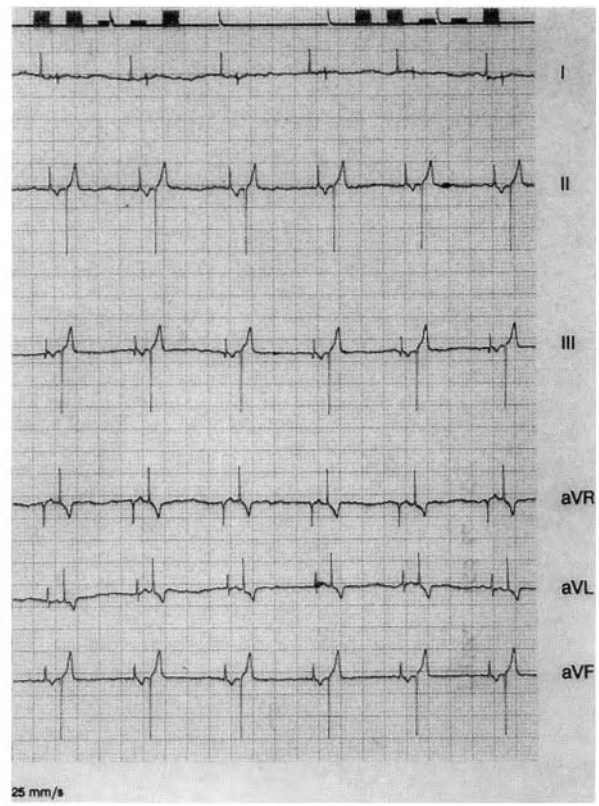

Figure 25.1. Tracing between 250 and 254 xx. Electrocardiogram of racing pigeon 2.5 hr after anesthesia induced with ketamine-xylazine, showing first-degree atrioventricular block. Paper speed 25 mm/sec; 1 cm = 1 mV. Heart rate 75 bpm; PR interval 0.14 sec (reference value 0.045-0.070 sec; Lumeij and Stokhof, 1985).

In all the pigeons that were anesthetized a marked sinus bradycardia accompanied by first-degree atrioventricular block was seen (Fig. 25.1). PR intervals ranging from 0.08 to 0.14 sec were observed 2.5 hr after induction (reference value 0.045-0.070 sec; Lumeij and Stokhof, 1985). The sinus bradycardia lasted for several hours. Heart rates before anesthesia ranged from 187 bpm to 375 bpm, and 2.5 hr after induction, heart rates vary between 58 bpm and 78 bpm (reference value 160-300 bpm; Lumeij and Stokhof, 1985). The recovery time to standing up was 6 hr. No real anesthetic state was achieved in the pigeons. Violent bouts of wing flapping and opisthotonus could be provoked by manipulation; however, the heart rate was unaffected and remained low.

In the goshawk the initial heart rate was 300 bpm and the lowest heart rate of 62 bpm was observed 1.5 hr after anesthesia was induced. The PR interval at that time was 0.1 sec (Fig. 25.2). There are no published resting ECG values available for goshawks. The goshawk exhibited good muscle relaxation and analgesia, which was established by ap-

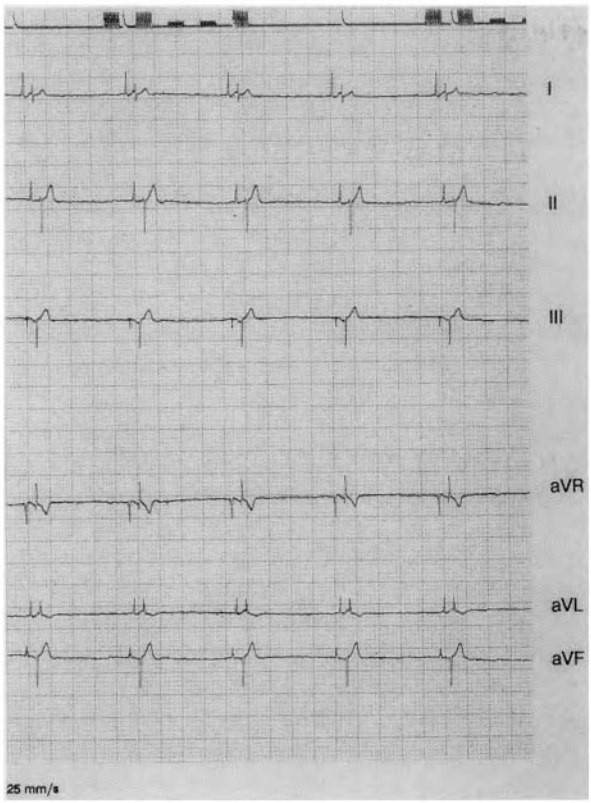

Figure 25.2. Tracing 138 xx. Electrocardiogram of goshawk 1.5 hr after anesthesia induced with ketamine-xylazine. Paper speed 25 mm/sec; 1 cm = 2 mV. Heart rate 62 bpm; PR interval 0.1 sec. Likely first-degree atrioventricular block (no reference values available).

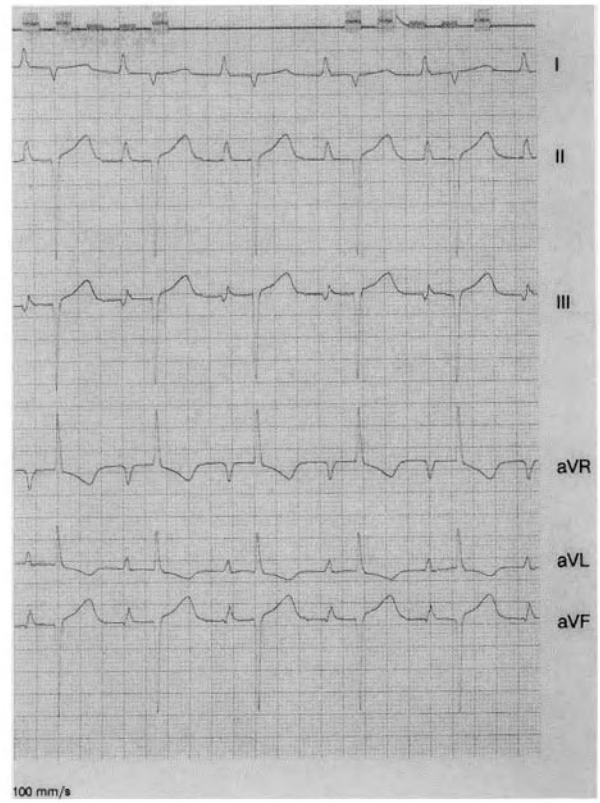

Figure 25.3. Tracing 575 xx. Electrocardiogram of a pigeon 2 hr after anesthesia induced with ketamine-xylazine. Paper speed 100 mm/sec; 1 cm = 1 mV. Heart rate 100 bpm; PR interval 0.08 sec. Bradycardia and first-degree atrioventricular block.

plying a mosquito forceps to the digits. It began to regain consciousness 6.5 hr after induction of anesthesia, but died 10 min later. The last electrocardiographic recording, which was made 20 min before the bird died, showed a heart rate of 107 bpm and the PR interval was not essentially different from that in Fig. 25.2.

To study the effects of atropine on ketamine-xylazine-induced bradycardia, atropine was administered in doses of 0.05, 0.5, and 1 mg/kg to two more pigeons that had received the aforementioned anesthetic mixture 2 hr before. A slight effect on heart rate could already be observed with the lowest dose, but a marked increase in heart rate occurred after the administration of 0.5 mg/kg (Figs. 25.3 and 25.4). Heart rate increased from 100 bpm to 272 bpm, with a simultaneous decrease in PR interval from 0.08 to 0.07 sec. No side effects were observed after the administration of atropine in a dose of 1 mg/kg.

Discussion

The results of this study clearly indicate that a diminished output of glucocorticoids (corticosterone) did not occur with ketamine-xylazine anesthesia in pigeons and goshawks. The fatalities seen in the goshawks could not therefore be explained by impaired function of the interrenal cells.

In the pigeons no satisfactory anesthetic state could be achieved with ketamine-xylazine in the doses used in this study. Marked excitatory behavior could be induced by manipulation. Other avian species that are known to show excitatory behavior after the administration of ketamine are gallinules (*Porphyrio* spp.), the water rail (*Rallus aquaticus*), the golden pheasant (*Chrysolopus pictus*), the Hartlaub's turaco (*Tauraco hartlaubi*), the crowned hornbill (*Tockus alboterminatus*), some European and African species of vultures (Samour et al., 1984), great horned owl (*Bubo virginianus*), and snowy owl (*Nyctea scandiaca*) (Redig et al., 1984). Redig et al. (1984) have demonstrated that in great horned owls the L-isomer of ketamine is responsible for the excitatory behavior. In mammals the D-isomer is three times more potent as an analgesic and one and a half times more potent as a hypnotic agent (White et al., 1982), while in great horned owls the hypnotic potency of the D-isomer is at least three times greater (Redig et al., 1984). It is likely that the excitatory behavior in the other

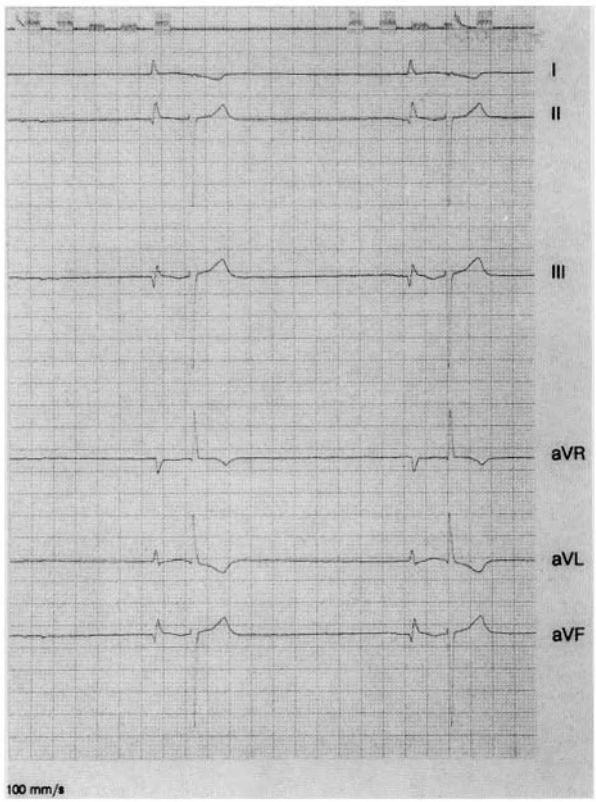

Figure 25.4. Tracing 610 xx. Same pigeon as in Fig. 25.3, 3 min after administration of atropine 0.5 mg/kg. Paper speed 100mm/sec; 1 cm = 1 mV. The heart rate has increased to 272 bpm and the PR interval has normalized (0.07 sec).

avian species mentioned (including the racing pigeon) is also caused by the L-isomer of the racemate of ketamine.

In the goshawks, the long recovery periods followed by death were more likely associated with the severe sinus bradycardia. In dogs, sinus arrhythmia, first- and second-degree atrioventricular block, and paroxysmal ventricular tachycardia have been reported after the use of xylazine. It has also been demonstrated that xylazine potentiates adrenaline-induced arrhythmias (Pascoe, 1985; Muir et al., 1975; Sands, 1981). The only conduction disturbance seen in the present study was a first-degree atrioventricular block. It cannot be ruled out, however, that in the goshawks myocardial sensitization occurred through adrenaline release when the birds started to wake up. In great horned owls the L-isomer of the racemic form of ketamine is known to cause a significant increase in heart rate for the first 1 to 2 min after induction. This increase is preceded by profound irregularity characterized by irregular stopping and starting of the heart in the first 30 to 45 sec, after which normal rhythms and notable slowing were observed (Redig et al., 1984). Heart rates after IV administration of 20 mg/kg of the racemate in great horned owls were at their lowest 10 min after injection (140 bpm) after which time they started to increase again. It is therefore not likely that the profound bradycardia that was observed in the present study for many hours after injection was caused by ketamine; it more likely resulted from xylazine. It is not clear whether the observed sinus bradycardia had any hemodynamic significance. If, however, the cardiac output was lowered as a result of the observed sinus bradycardia, this might have caused hypotension and diminished tissue perfusion, resulting in shock and metabolic acidosis.

The present study shows that atropine was effective in counteracting the anesthetic-induced bradycardia in a dose of 0.5 mg/kg IM. The reported value of atropine during avian anesthesia is controversial. Some researchers have considered it essential to reduce respiratory secretions in psittacines (Haigh, 1981), while one has reported 100% mortality in experimental birds (Heidenreich, 1978). In the present study no mortality was seen after the administration in doses up to 1 mg/kg. This drug seems to be indicated when severe bradycardia is encountered during avian anesthesia, starting with a dose of 0.05 mg/kg IM and giving supplementary doses when indicated. Routine administration of atropine as a preanesthetic agent might be considered. Glycopyrrolate might be a good alternative to atropine, with possibly fewer potential side effects (Churchill-Davidson, 1979), but its use in birds needs to be evaluated.

Although the exact cause of death of the goshawks anesthetized in this study with the combination ketamine-xylazine has not been established, it has been clearly demonstrated that the dosage used causes a marked atropine-responsive sinus bradycardia, accompanied by first-degree atrioventricular block. The dosage is too high to induce safe anesthesia in goshawks, while in pigeons no satisfactory level of anesthesia can be achieved.

Like many other avian hospitals worldwide, my own institution has changed in recent years to isoflurane[d] as the anesthetic of choice for birds (Bednarski et al., 1985; Harrison et al., 1985; Fitzgerald and Blais, chapter 22, this volume). Anesthesia is induced with a face mask and isoflurane 2.5% in a 50/50 mixture of oxygen and nitrous oxide or 100% oxygen. Maintenance of anesthesia can be achieved with 2% isoflurane. Intubation during anesthesia is optional, but is indicated when respiration is depressed. Induction and recovery with isoflurane are smooth and rapid in birds (including goshawks). Despite the availability of this superior anesthetic agent, inhalation anesthetic equipment is not always available and injectable anesthetics have to be used. For long-term surgical procedures in goshawks, however, no safe recommendations for injectable anesthetic agents can be made at present.

Empirical doses used for inducing anesthesia of short duration in most North American diurnal birds of prey at the Raptor Center of the University of Minnesota are 10 mg/kg ketamine and 2 mg/kg xylazine intravenously. Diazepam[e] in

a dose of 1.5 mg/kg IV is used as an alternative to xylazine (Redig and Duke, 1976; Redig et al., 1984). A combination of ketamine-climazolam[f] (25 mg/kg and 1.25 mg/kg) was successfully used to induce a 25-min anesthesia in pigeons. Respiratory and circulatory variables remained stable. The recovery period could be shortened with the benzodiazepine antagonist RO-15/1788, (flumazenil)[g] (Kummerfeld and Ganter, 1986). Some preliminary studies have been performed in raptors with medetomidine,[h] a highly potent and selective full alpha-2-adrenocepter agonist, in combination with ketamine. This combination is apparently safe, but no evaluations of respiratory or circulatory parameters are currently available. Atipamezole,[i] a potent alpha-2-adrenocepter antagonist, effectively shortens recovery times at doses three to six times that of medetomidine (Jalanka, 1991).

Product List

[a]Aescoket, Aesculaap, Mijlstraat 35, 5280 AA, Boxtel, Netherlands; or Ketaset, Aveco Co. Inc., 800 5th St. N.W., Ft. Dodge, Iowa 50501 USA

[b]Rompun, Bayer, Nijverheidsweg 26, 3640 AB, Mijdrecht, Netherlands; or Rompun, Haver-Mobay Corp., Animal Health Division, P.O. Box 390, Shawnee, Kansas 66201 USA

[c]Cortrosyn, Organon, Weth. v. Eschstraat 1, 5340 BH, Oss, Netherlands

[d]AErrane, Anaquest, 2005 W. Beltline Hwy., Madison, Wisconsin 53713-2318 USA; or Forene, Abbot, Maalderizl, 1185 ZB Amstelveen, Netherlands

[e]Valium, Roche, Division of Hoffman-LaRoche, Inc., 340 Kingsland St., Nutley, New Jersey 07110 USA

[f]Experimental drug of Hoffman-LaRoche Inc., 340 Kingsland St., Nutley, New Jersey 07110 USA. Closely related to midazolam or Dormicum, Hoffman-LaRoche or Roche BV, Nijverheidsweg 38, 3641 RR Mijdrecht, Netherlands

[g]Anexate, Roche BV, Nijverheidsweg 38, 3641 RR Mijdrecht, Netherlands

[h]Domitor, Orion Corp., Farmos, Turku, Finland

[i]Antisedan, Orion Corp., Farmos, Turku, Finland

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CHAPTER 26

Cardiorespiratory Effects of Ketamine-Xylazine in the Great Horned Owl

MARC R. RAFFE, D.V.M., M.S.

Department of Small Animal Clinical Sciences
College of Veterinary Medicine
University of Minnesota
St. Paul, Minnesota, USA

MARK MAMMEL, M.D.
MARGARET GORDON, R.R.T.

Infant Pulmonary Research Center
St. Paul Children's Hospital
St. Paul, Minnesota, USA

GARY DUKE, Ph.D.
PATRICK T. REDIG, D.V.M., Ph.D.

Department of Small Animal Clinical Sciences
College of Veterinary Medicine
University of Minnesota
St. Paul, Minnesota USA

STEVEN BOROS, M.D.

Infant Pulmonary Research Center
St. Paul Children's Hospital
St. Paul, Minnesota, USA

ABSTRACT: In recent years, ketamine has gained popularity for immobilization in many raptor species. Combining ketamine with other agents is necessary to overcome excitatory effects noted when ketamine alone is administered. The combination of xylazine and ketamine is effective in providing immobilization. Despite the popularity of this combination, little is known about its physiologic effects in raptor species.

This study evaluates the cardiopulmonary effects of the xylazine-ketamine (X-K) combination in great horned owls (GHO) (*Bubo virginianus*). We anesthetized 14 GHO using an X-K combination of 0.15 mg/kg X and 15 mg/kg K administered via pectoral muscle for immobilization. Following immobilization, owls were instrumented for measurement of heart rate, systemic blood pressure, and central venous pressure. Samples were collected from the arterial access for blood gas analysis to evaluate pulmonary function. Parameters measured include pH, pO_2, bicarbonate (HCO_3), and base excess (BE) values. All birds were permitted to breathe spontaneously on room air for all measurements. Baseline measurements were begun in the postinstrumentation period when arousal from the initial dose of X-K was noted. Following baseline measurements, the X-K combination was readministered at the original dose schedule. Measurements were collected at 5-min intervals following X-K readministration. Sampling was discontinued at the termination of the trial.

All raptors responded favorably to the initial X-K dose. Satisfactory restraint was noted in all birds within 5 min following administration. Transient apnea was noted during the initial 5 min of immobilization. This reverted to normal ventilatory rate by 10 min postinjection. Heart rates were consistent for all birds and remained in the range of 140 to 165 beats per min (bpm) following initial administration. Supplemental doses frequently resulted in heart rate reduction to 80 to 100 bpm. Blood pressure parameters were consistent for each bird throughout the procedure. Systolic blood pressure ranged from 154 to 290 mm Hg. Diastolic blood pressure ranged from 77 to 160 mm Hg. Mean blood pressure ranged from 106 to 250 mm Hg. Central venous pressure averaged 8 cm water. Time-weighted blood gas values were as follows: pH, 7.55; pO_2, 96 torr; pCO_2, 21 torr; HCO_3, 18 meq/l; and base excess, -3.

Based on the results, we feel that X-K is well tolerated in the great horned owl. We advise caution with repeat doses by IV or pectoral route, as deterioration in cardiopulmonary stability may be noted.

KEY WORDS: raptor, anesthesia, ketamine, xylazine, great horned owl, *Bubo virginianus*.

Injectable anesthetic drugs have been popular for restraint and anesthesia in avian species. Ketamine has been shown to be relatively safe for use in raptors; however, spontaneous movements and muscle rigidity associated with administration are undesirable side effects associated with its administration (Clark et al., 1982; Allen et al., 1986). Tranquilizers or sedative drugs have been combined with ketamine to overcome these undesirable qualities (Clark et al., 1982). Of the agents reported, xylazine has become the most popular because of its widespread availability and ease of administration. However, it has been reported to have significant effects on cardiorespiratory and endocrine status in many species (Haskins et al.,1986; Kollias and McLeish, 1978). Because little information is available regarding the effects of these drugs in raptor species (Redig and Duke, 1976), because of the widespread use of the xylazine-ketamine combination, and because of the scarcity of information regarding the cardiopulmonary effects following xylazine-ketamine administration, we elected to evaluate the cardiorespiratory effects of xylazine-ketamine in great horned owls (GHO) (*Bubo virginianus*).

Materials and Methods

Fourteen GHO weighing 1080 ± 35 g were used for the study. The owls were presented to the Raptor Center at the University of Minnesota for musculoskeletal injuries and were not rehabilitable for release. They were kept under conditions of controlled temperature and photoperiod and fed freshly killed rodents daily. On the day of trial, each bird was transported to the laboratory in a transport crate. The bird was restrained and administered an initial dose of xylazine-ketamine (X-K) at 3 mg/kg X[a] and 15 mg/kg K[b] IM in the pectoral muscle. Following restraint, the femoral vessels were surgically isolated and cannulated with 5 French pediatric vascular catheters.[c] Following cannulation, the femoral arterial catheter was connected to a pressure transducer and oscilloscope monitoring device[d] for display and recording of blood pressure waveforms and values. The external jugular vein also was surgically exposed and a 5 French pediatric catheter was inserted to the level of the right atrium by observing pressure waveforms following connection to a separate pressure transducer. External electrodes were attached to monitor a lead II electrocardiogram.[e] Spontaneous ventilation was permitted on room air during the instrumentation period.

Following instrumentation, baseline values were collected for heart rate (HR); systolic (SYS), diastolic (DIA), and mean (M) blood pressure; central venous pressure (CVP); respiratory rate (RR); and arterial blood gas values (pH, pO_2, pCO_2, bicarbonate [HCO_3], and base excess [BE]). The birds were permitted to recover from the initial dose of X-K until emergence was indicated by ocular reflexes, muscle tone, and spontaneous wing activity.

Table 26.1
Respiratory parameters following xylazine-ketamine administration

Parameter	*Oxygen level* 21% (room air)	40% (oxygen-air)
Respiratory rate	32 ± 6	21 ± 2*
pH	7.53 ± 0.11	7.68 ± 0.04
pO_2 (mm Hg)	90.44 ± 6	143.4 ± 11*
pCO_2 (mm Hg)	22.03 ± 7	12.2 ± 0.2*
HCO_3 (meq/l)	17.8 ± 0.25	16.7 ± 0.6
BE	−6	−7

*Significant ($p < 0.05$) difference between groups.

X-K was readministered via femoral vein using the initial dose schedule. Following administration, a 4.0 mm i.d. endotracheal tube[f] was placed through the glottis into the proximal airway. Following readministration of X-K, all parameters were reevaluated at 5-min intervals until emergence was noted. Criteria for emergence were the same as indicated above. Two levels of oxygen support, room air and a 40% oxygen-air mixture,[g] were provided to assess the response to enriched oxygen administration. Each bird was evaluated at both room air and enriched oxygen in a crossover design.

All parameters were analyzed by two-way analysis of variance, evaluating the variables of drug treatment and time. Significant findings were further analyzed by Student's t-test to evaluate the significance of the observation.

Results

Chemical restraint and anesthesia were induced in all birds by the initial dose of X-K. Loss of righting reflexes, mental awareness, and voluntary motor activity were produced at 5.02 ± 0.35 min following X-K administration. The duration of X-K effect was 18.45 ± 0.7 min. Transient apnea, initially associated with X-K administration, was resolved by 10.0 ± 1.5 min following administration. Following airway intubation, 45% of birds in the study exhibited apnea; however, spontaneous ventilation returned within 2.3 ± 0.8 min following intubation.

RR was higher on room air (32 ± 6 breaths per min) than with supplemental oxygen (21 ± 2 breaths per min). Gas exchange, as measured by arterial blood gas analysis, demonstrated significant differences in pH, pO_2 and pCO_2 values between room air and supplemental oxygen groups (Table 26.1). Bicarbonate and BE values were not significantly different between groups.

Cardiovascular parameters were stable in all birds initially following anesthetic induction. Elevation in SYS, DIA, M, and CVP and a reduced HR were noted following

Table 26.2
Cardiovascular parameters following xylazine-ketamine administration

Parameter	Oxygen level	
	21% (room air)	*40% (oxygen-air)*
Heart rate	120 ± 8*	151 ± 13
Systolic BP (mm Hg)	173 ± 23*	235.2 ± 14
Diastolic BP (mm Hg)	103.78 ± 25*	159.2 ± 13
Mean BP (mm Hg)	135.78 ± 28*	197.4 ± 8
CVP (cm H_2O)	6.78 ± 2.3	5.2 ± 1

*Significant ($p < 0.05$) difference between groups.

X-K administration. Progressive reduction in HR and SYS, DIA, and M values, as well as increased CVP level, were noted in all birds during the room air trial (Table 26.2). Significantly higher HR and SYS, DIA, and M values and lower CVP values occurred with supplemental oxygen. The trends noted with enhanced oxygen levels were consistent whether the first trial included supplemental oxygen or oxygen was supplemented in the crossover trial. Supplemental X-K doses frequently resulted in a 35-50% reduction in HR. In all cases, supplemental oxygen improved cardiorespiratory performance.

Discussion

The combination of xylazine-ketamine has been shown to produce predictable chemical restraint and anesthesia in a variety of domestic and nondomestic species (Clark et al., 1982; Allen et al., 1986; Haskins et al., 1986). The dosages for xylazine and ketamine selected for the current study were based on clinical experience in GHO. In the current study, several observations are consistent with those previously reported with X-K administration in other species (Clark et al., 1982; Allen et al., 1986; Haskins et al., 1986). The onset of X-K action was predictable and the quality of response was smooth for all birds in the study. The duration of chemical restraint induced by a single X-K dose is similar to that reported for ketamine in other raptor species, but shorter than expected for mammals (Kollias and MacLeish, 1978). Recovery was generally smooth, but some excitement was noted on emergence. This excitement may be associated with the intrinsic effects of the X-K combination or may reflect residual drug actions present at emergence from multiple-dose administration. A single injection of X-K for chemical restraint apparently produces emergence excitement in raptors that appears to be independent of time and dose (Redig and Duke, 1976; Redig et al., 1984). On the basis of these observations, we believe that the recovery reflects the residual effects of the ketamine component and not the redosing response (Redig et al., 1984).

Cardiorespiratory effects of X-K in GHO do not appear to be significantly different from those reported in other species (Clark et al., 1982; Allen et al., 1986; Haskins et al., 1986). Blood pressure values recorded in the present study were comparable to previous reports. Several factors may contribute to elevated blood pressure values following X-K administration. The alpha adrenergic action of xylazine has been shown to induce a marked increase in total peripheral vascular resistance, which produces vasoconstriction and contributes to elevated blood pressure values (Klide et al., 1975; Muir and Piper, 1977; Haskins et al., 1986). Increased cardiac output and blood pressure are also associated with ketamine (Haskins et al., 1985). Although cardiac output has been shown in dogs and cats to be reduced following X-K administration, hypertension has been documented, indicating significant vasoconstriction as the primary component (Clark et al., 1982; Allen et al., 1986; Haskins et al., 1986). Handling and restraint may liberate catecholamines that produce additional positive inotropic and chronotropic actions on the heart and an increase in peripheral vascular resistance. In the present study, all these factors may have contributed to the high blood pressure values noted. Unfortunately, predrug blood pressure values were not obtained for comparison because of the invasive nature of the measurement technique and behavior of the owl. Therefore, we cannot comment on the individual effects of stress and X-K on blood pressure.

HR changes following X-K administration parallel those in other species in that the rate was slower following X-K administration. Bradycardia following X-K administration may be related to drug effect, body positioning, and/or hypoventilation. As noted above, xylazine has potent cardiovascular effects that can produce bradycardia. The degree of bradycardia is reduced because ketamine attenuates the heart-slowing effects of the xylazine (Clark et al., 1982; Allen et al., 1986; Haskins et al., 1986).

Central venous pressure values parallel heart rate and blood pressure. Elevation in central venous pressure accompanies periods of bradycardia and hypertension. This elevation most likely reflects reduced cardiac output following X-K administration. This trend was reversed following supplemental oxygen administration, and increased heart rate, indicating bradycardia, was associated with hypoxemia.

Respiratory parameters also were changed following X-K administration. The effects of anesthetic agents on respiratory control have been widely documented and are characterized by hypercapnia secondary to hypoventilation. Higher respiratory rates in the control (room air) group indicate a response to anesthesia-induced hypoventilation. Oxygen partial pressures were significantly lower following X-K administration in owls, compared with values reported in mammalian species (Clark et al., 1982; Allen et al., 1986; Haskins et al., 1986). Hypercapnia was noted in all birds following X-K administration. The presence of hypercapnia in all birds following X-K indicates that hypoventi-

lation was principally responsible for the lower oxygen and higher carbon dioxide levels noted in the study. Reduced blood pH values reflect acidosis of respiratory origin, most likely produced by elevated carbon dioxide values. Apparently, hypoxemia contributed to increased respiratory rates because supplemental oxygen lowered rates and produced higher partial pressures of oxygen in the blood. Transient hypoxemia likely developed based on the interactive effects of body positioning, X-K administration, and absence of supplemental oxygenation (Don, 1981). Based on these observations, lateral recumbency should not be maintained for an extended time in X-K anesthestized birds without supplemental oxygen administration.

Conclusion

Based on this study, we conclude that X-K can be safely administered to GHO. The quality of restraint/anesthesia is good, with duration of effect for the doses evaluated shorter than expected on a comparative species basis. The cardiorespiratory actions of X-K in these owls are similar to those reported for other species. It appears that GHO are more sensitive to hypoxemia associated with restraint and recumbency. This may be related to respiratory physiology and function that differs from mammalian species. Supplemental oxygen should be considered for birds in dorsal or lateral recumbency or for those cases demonstrating pulmonary disease.

Product List

[a]Rompun, Haver-Mobay Corp., Animal Health Division, P.O. Box 390, Shawnee, Kansas 66201 USA; or Rompun, Bayer, Nijverheidsweg 26, 3640 AB Mijdrecht, Netherlands

[b]Ketaset, Aveco Co. Inc., P.O. Box 717, Ft. Dodge, Iowa 50501 USA; or Aescoket, Aesculaap, Mijlastraat 35, 5280 AA, Boxtel, Netherlands

[c]Edwards and Co., 110 N. Main St., Ste. 708, Santa Ana, California 92701 USA

[d]Model 7833, Hewlett Packard, 1700 S. Baker St., McMinnville, Oregon 97218 USA

[e]Model 78333, Hewlett Packard, 1700 S. Baker St., McMinnville, Oregon 97218 USA

[f]Sherwood Medical Co., 1915 Olive St., St. Louis, Missouri 63103-1791 USA

[g]Baby Bird I, Bird Corp., 3101 E. Alejo Rd., Palm Springs, California 92264 USA

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CHAPTER 27

Raptor Bumblefoot: A New Treatment Technique

J. DAVID REMPLE, D.V.M.

Dubai Falcon Hospital
Dubai, United Arab Emirates

ABSTRACT: The pathogenesis of bumblefoot in raptors has three distinct phases: initiation, microbial complication, and inappropriate host response. The lesion that develops is a chronic granuloma that effectively shields the pathogens from humoral and cellular immune responses as well as systemic antibiotics. This chapter describes a new five-step classification for bumblefoot, based on elements of pathogenesis, that ranks cases on a scale from mild, superficial tissue devitalization with a good prognosis for recovery to deep tissue inflammation and destruction with a correspondingly poor prognosis for recovery. Successful treatment is predicated on interrupting the pathogenic processes identified above, and involves surgical debridement of antigenic and inflammatory debris, creation of a wound capable of first-intention healing, and a new method of foot casting that protects the bottom of the foot from mechanical forces that would disrupt healing.

A total of 157 raptors (representing 266 cases of bumblefoot) were classified and treated by the methods described. Specific recovery rates by class were as follows: Class I, 100%; Class II, 86.4%; Class III, 81.6%, (18.6% of cases required repeat surgery); and Class IV, 32.8%. All raptors in Class V received no treatment and were euthanized due to loss of pedal function.

KEY WORDS: bumblefoot, pododermatitis, foot, feet, raptor.

Bumblefoot (pododermatitis) has been recognized for centuries as one of the most common diseases of captive raptors. The earliest known English reference to the disease appears in the *Boke of St. Albans*, published in 1486, and the earliest reference to surgical treatment appears in *Falconry, or the Falcons Lure and Cure*, published in 1615 (Cooper, 1980). The disease is characterized by local injury to the pedal integument, usually the plantar metatarsal pad or plantar digital pads, which leads to scab formation and infection of the internal tissues of the foot (Halliwell, 1975; Halliwell and Graham, 1978; Cooper, 1980; Riddle, 1980; Sawyer, 1983; Remple and Remple, 1987). Infection appears to be seeded by two routes: direct inoculation of pathogenic microbes by puncture of the integument, or devitalization of the epithelium (a barrier to infection) permitting microbial entry into underlying tissues. Once the process begins, spontaneous resolution rarely occurs; instead, the pathological sequence of events leads to eventual destruction of the foot unless determined treatment is begun. Untreated disease may lead to crippling deformity and occasionally death (Halliwell, 1975).

The tenacity of established infection and the difficulty of treatment relate to the location of the lesion, physical problems centering on the plantar metatarsal scab itself (Remple and Remple, 1987), and the raptor's inadequate or inappropriate immune response (Satterfield and O'Rourke, 1980). At the plantar location, pressure, movement of injured tissues, contusion, and contamination are persistent obstacles to healing. The plantar scab places pressure on underlying tissues, with resulting ischemia, and contamination is usually present at the scab-tissue interface (Remple and Remple, 1987). Predisposing factors of bumblefoot and pathogens commonly associated with it are discussed in Remple and Al-Ashbal (chapter 17, this volume).

Treatment failure for bumblefoot is common. Relapse after apparent success is frequent, often after quiescent periods ranging from weeks to years. Previous treatment regimes have been diverse and have included hypertonic foot baths and poultices, topical dimethylsulfoxide-antibiotic-corticosteroid application (Stefanatos, 1974), radiation (Bird and Lague, 1975), immunomodulation and vaccination (Satterfield and O'Rourke, 1980), surgery with secondary healing by granulation (Gandal, 1969; Arnall and Keymer, 1975), and surgery with first-intention healing (Cooper, 1980; Riddle, 1980). All procedures have met with varying degrees of success. In my opinion, surgery represents the treatment of choice for debulking the antigen load and inflammatory debris and stimulating immune responses for the elimination of infection; however, it is not without risk and complications. Undesirable sequelae are anesthetic complications, postoperative wound contamination, nonhealing, and reseeding of infection. All but the anesthetic complications are consequences of pressure and periodic trauma sustained during the postoperative healing period (Riddle, 1980).

Bumblefoot lesions have been classified according to two different schemes. One emphasizes the location and extent of lesions (Halliwell, 1975); another focuses on degen-

erative pathological changes and chronicity as a basis for classification (Cooper, 1980).

Objectives

The objectives of this chapter are (1) to describe a method of alleviating pressure, contusion, movement, and contamination from the affected plantar surfaces during the healing period; (2) to evaluate it as adjunctive therapy to surgical treatment; (3) to propose a new classification for bumblefoot that is broader in scope than earlier systems; and (4) to describe methods of prevention derived from extensive experience in managing this problem.

Materials and Methods

Raptors

The raptors examined in this study were of five species: gyrfalcon (*Falco rusticolus*), saker falcon (*Falco cherrug*), peregrine falcon (*Falco peregrinus*), Barbary falcon (*Falco pelegrinoides*), and goshawk (*Accipiter gentilis*). The majority of raptors were female saker falcons (about 70%) and female peregrine falcons (about 30%). The total number of raptors receiving treatment was 157, which represented 266 individual cases of bumblefoot.

Classification Scheme

The raptors investigated were each placed into one of five classifications of disease. This grading system was based on the extent and severity of pododermatitis, with each class representing a different stage of disease evolution. Each raptor was assigned to a class during the surgical procedure.

Class I cases were characterized by a lesion of the integument only, with no infection of underlying tissues. There were 15 raptors (8.7%) in this class; 6 were treated conservatively and 9 received surgical treatment.

Class II raptors displayed infection of the subcutaneous tissues but with no gross swelling of affected feet. There were 22 raptors (12.7%) in this class. All were treated surgically and with postoperative foot casting.

Class III raptors displayed infected, swollen, and painful feet without apparent damage to deep vital structures. There were 59 (34.1%) in this class.

Class IV raptors displayed infection of deep vital structures (tendons and bones) but still retained pedal function. There were 61 raptors in this class (35.3%).

Class V raptors displayed end-stage disease with loss of pedal function. There were 16 (9.2%) in this class. These birds received no treatment and were euthanized.

Surgical Treatment

Raptors with bumblefoot were thoroughly evaluated prior to anesthetic or surgical procedure. All patients were given a physical examination and complete blood count. Fecal examination was performed to evaluate parasite burdens. Whole-body and foot radiographic examination was performed to rule out concurrent systemic disease, to isolate and evaluate degenerative changes, and to help establish a prognosis. Patients were routinely given a multivitamin injection[a] equivalent to 30,000 IU vitamin A/kg body weight to correct possible deficiencies.

A preanesthetic mixture of ketamine HCl[b] at 5 mg/kg body weight, xylazine[c] at 0.25 mg/kg body weight (Haigh, 1980), and atropine at 0.05 mg/kg body weight was given as a bolus into the medial tarsal vein. Upon loss of consciousness, the raptor was intubated, and surgical anesthesia was accomplished by administering 0.5-2% halothane[d] or isoflurane[e] in 60% oxygen and 40% nitrous oxide at a flow rate of 1 l/min in a nonrebreathing system.

The goals of surgery were to reduce the antigen load, to debride the infected area of as much inflammatory exudate as possible, and to convert an infected necrotic area into a fresh vascular incision capable of being brought into perfect apposition for first-intention healing (Cooper, 1980; Riddle, 1980; Remple and Remple, 1987). The surgical procedure has been described by others (Cooper, 1980; Riddle, 1980), however, the modifications described below can streamline the procedure and improve success rate.

A thorough scrub of the surgical site with a stiff toothbrush was performed to remove the likelihood of a hyperkeratotic epidermal reaction to the inflammatory process, which could impede healing of plantar integument. Surgery was performed under an illuminating magnification loupe to enhance visualization.

Surgery was facilitated by a padded device that supported the foot while the raptor was in dorsal recumbency. The device attached to the surgical table and gently grasped the foot at the tarsometatarsus (Fig. 27.1). The digits were held in hyperextension with towel clamps, and the plantar surface of the foot was readily accessible without the need of an assistant (Remple and Remple, 1987).

Prior to being incised, the integument was carefully evaluated. The thickness and vascularity as well as the creases and folds of the plantar skin determined the direction and extent of the elliptical incision. If a scab was present, it was removed after the scrub. After the incision was made, the deep internal tissues were cultured. Care was taken to avoid areas in contact with the scab periphery so as to minimize secondary contamination (Remple and Remple, 1987).

Removal of fibrotic and exudative material was accomplished with dull spoon curettes, which allowed vigorous curettage without damage to vital structures. Curettage was

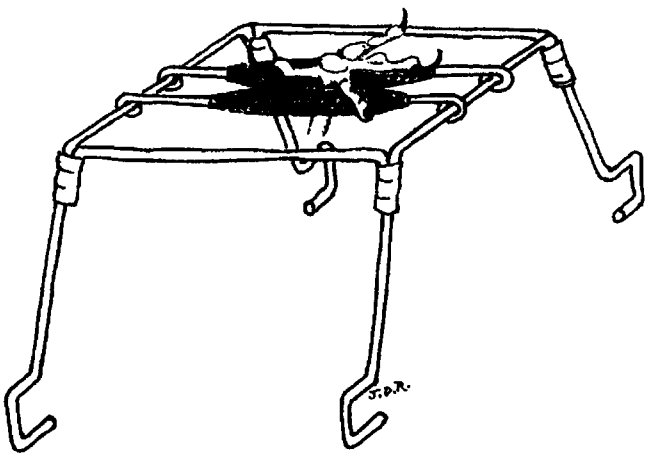

Figure. 27.1. The foot-holding device is fashioned from small-diameter aluminum orthopedic casting rod. Base design and flexibility permit attachment to a variety of surgical-table surfaces. The padded sliding tarsometatarsus-grasping bars are taped to each other to hold the foot gently during the surgical procedure.

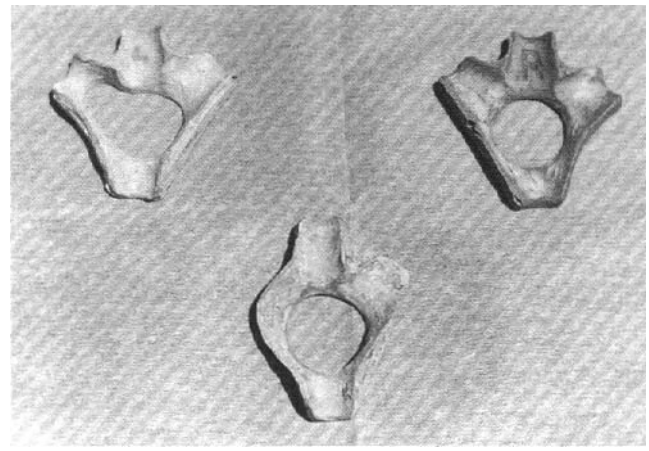

Figure 27.2. Preformed styrene plastic polymer casts can be formed prior to surgery to shorten casting time. The hole in the sole circumscribes the incisional area and as much of the infected area as possible without placing excessive pressure on adjacent healthy metatarsal and digital integument.

followed by chymotrypsin[f] irrigation into the wound to enhance the removal of necrotic, inflammatory debris (Remple and Remple, 1987). Enzymatic debridement was followed by irrigation with a broad-spectrum penicillin such as ticarcillin[g] or piperacillin.[h]

Great care was taken with closure. Incision edges were cut back to a full-thickness level and were actively bleeding. A near-perfect apposition of skin edges with little or no pressure on the suture line was attempted. Closure was accomplished with 4-0 monofilament nylon. A pattern of simple interrupted sutures alternating with vertical mattress sutures was used, starting and ending with simple interrupted sutures. The simple sutures were placed so as just to oppose the skin edges, while the mattress sutures lent slight eversion to the incision edges.

Foot Casting

Following surgery, nitrofurazone soluble dressing[i] was applied to the closed incision and a nonadhesive sterile dressing[j] affixed over the sutures. The operated foot was gently wrapped with a nonadhesive elastic wrap.[k] The wrap extended approximately 2.5 cm distally on all digits. Care was taken when wrapping so as not to constrict the vasculature extending along the lateral and medial aspects of the digits. Casts were then fashioned to protect the incision from pressure, contusion, and contamination. The raptors in this study were fitted with casts made from a thermoplastic orthopedic tape[l] (Remple and Remple, 1987). Following this study, the original technique was modified as described next; the newer method has come to be the preferred one for making such casts.

In the newer technique, a preformed styrene plastic polymer[m] "sole" cast is fitted to the plantar aspect of the foot. (The material is commonly used as a filler in automotive body repair.) The cast is fashioned to conform to the contour of the foot, so as to distribute the pressure evenly over the healthy areas of plantar pedal integument, but alleviate pressure from the incision and the immediate infected area (Fig. 27.2). Forming the cast to the contour of the foot is accomplished with the aid of a hobby drill (Fig. 27.3). The contoured cast is then fixed to the plantar aspect of the foot by means of freshly mixed (unhardened) styrene plastic polymer used as a glue. The semiliquid polymer adheres strongly to the hardened cast and to the nonadhesive elastic wrap. In my experience, all raptors have stood in these casts within minutes of recovery from anesthesia without showing any signs of awkwardness or distress (Fig. 27.4).

Postsurgical Management

The feet and cast(s) were checked daily. If digital swelling occurred, it was usually obvious within 24 hr of casting and was alleviated by loosening the offending digital wrap with scissors. The dead space between the cast and the incision area of the foot was checked periodically to assure that debris from perching surfaces did not accumulate there. Casts remained in place for 14 to 21 days. At the end of this time the patient was anesthetized (see above for anesthetic used) and the cast and wrap were removed. Incision edges were carefully debrided of dried blood and loose keratin so that incisional healing could be properly evaluated. If healing was satisfactory, the simple interrupted sutures were removed, leaving the mattress sutures in place. The foot was rewrapped with nitrofurazone soluble dressing.

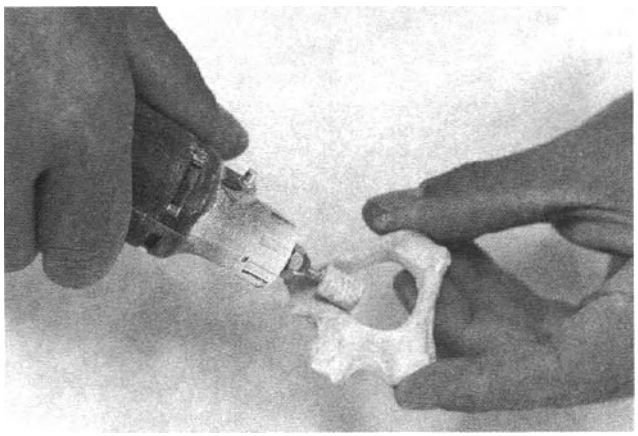

Figure 27.3. Casts are contoured with the aid of a hobby drill.

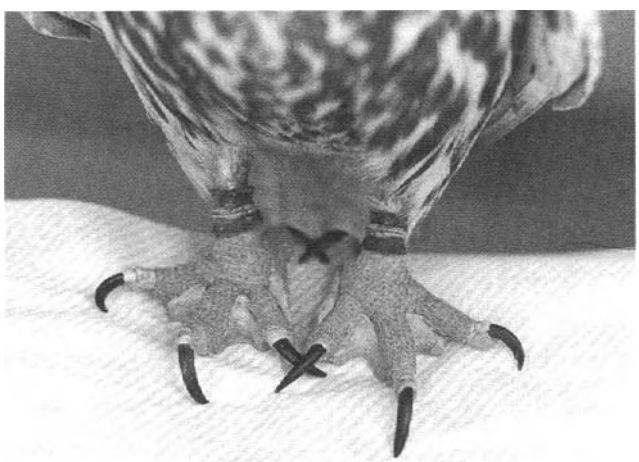

Figure 27.4. Finished casts are "glued" to the nonadhesive elastic foot wraps.

This precaution guarded against weakening and/or dehiscence of the incision because of renewed weight stress on a freshly healed scar. During the following week a certain degree of remodeling (morphallaxis) occurred as weight was placed on healing areas. This consisted of relaxation of some aspects of the scar and renewed fibroplasia in response to weight stress (Remple and Remple, 1987). Seven days after the removal of simple interrupted sutures, a short-acting anesthetic was given prior to further evaluation and final suture removal. Following removal of the mattress sutures, the foot was wrapped for an additional 7 days, and antibiotic dressing was discontinued.

Following surgery and foot casting, raptors were injected with ticarcillin or piperacillin at 200 mg/kg body weight IM BID until the results of culture and sensitivity testing were available. When sensitivity testing showed susceptibility to several antibiotics, a bactericidal one was favored over one that was bacteriostatic. Broad-spectrum penicillins were favored over aminoglycosides, owing to the latter's potential for renal toxicity and impairment of immune function and healing (Kollias, 1986). Problematic infections often dictated the use of synergistic combinations such as a penicillin and an aminoglycoside (given separately). The aminoglycoside (amikacin,[n] tobramycin[o]) was discontinued at 14 days, but the penicillin was maintained for a full 21 days or longer. When the final wraps were removed and antibiotics were discontinued, raptors were moved to corrective perching surfaces.

Results

Among the 173 raptors examined in this study (a total of 298 affected feet), bumblefoot lesions ranged from early insult of an area of plantar integument (bruise, corn, and so on) to functionless feet displaying necrosis of vital structures, tenosynovitis, crippling arthritis, and osteomyelitis. The total number of raptors receiving treatment was 157, which represented 266 affected feet.

Of the 15 Class I cases, 6 were treated conservatively, using topical softening and stimulating ointments,[p] corrective perching surfaces, and/or foot casting. All responded with complete healing; however, treatment time often exceeded 6 wk. The other 9 were treated surgically, and all healed completely within 3 wk. There were no reported recurrences within 1 yr, thus there was a 100% success rate for this class.

There were 22 raptors (12.7%) in Class II. All were treated surgically and with postoperative foot casting. Of this group, 3 raptors returned within 12 mon displaying diseased feet. It was impossible to distinguish between recurrence and new disease, because the raptors returned to the same conditions that initiated disease. Since 3 of 22 returned within 1 yr, the initial success rate for this class was 86.4%.

There were 59 raptors (34.1%) in Class III. Of these, 7 required one or more repeated surgical and casting treatments within 3 mon of initial treatment, and 4 required repeated surgical and casting treatments within 12 mon of initial treatment. The initial success rate for this class was 81.6%.

There were 61 raptors in Class IV (35.3%). Of these, 24 required one or more repeat surgical and casting treatments within 3 mon of initial treatment, and 17 relapsed within 12 mon of initial treatment. The total number of raptors in this class with "known" recurring disease (see the following discussion) was 41 of 67 (67.2%). Of the 41, 7 were eventually euthanized after they failed to respond to multiple surgical treatments. The success rate for this class was taken to be 32.8%.

Discussion

Raptor bumblefoot (pododermatitis) arises from pathogenic microbes gaining entry into the internal tissues of the foot.

Entry is gained by two routes: puncture of the integument (a barrier to infection) with inoculation of deeper tissues, and devitalization of the integument, which permits pathogens to cross the barrier to infection into the internal tissues. Advanced stages of both routes of infection demonstrate plantar metatarsal scabs and can be identical in appearance.

The metatarsal scab is, grossly, the most obvious obstacle to spontaneous healing. The location of the lesion and its relationship to pressure, contusion, movement, and contamination present significant barriers to healing. The scab increases pressure on underlying tissues, causing ischemia with spread of infection (Cooper, 1980; Remple and Remple, 1987). At the plantar location, tissues move each time the foot moves, which impairs healing. Repeated contusion at any time retards healing (Riddle, 1980) and can further seed encapsulated infection by breaking down fibrotic barriers. Contamination of the lesion is a continual threat to healing, since raptors stand on surfaces contaminated with food and feces. Cracks and crevices at the scab-tissue interface of an existing lesion provide easy access for contaminant pathogens into an existing lesion (Remple and Remple, 1987).

The ischemic, fibrotic, and granulomatous inflammatory response of avian bumblefoot is an impairment in itself to resolution of infection and healing. Fibroplasia is likely to occur within 96 hr of infection in birds (Cooper, 1980). The causative pathogen can become insulated from immune factors and chemotherapeutic penetration. Due to the rapid avian "insulating" fibroplastic response, other studies have suggested that chemotherapy of *acute* bumblefoot be carried out promptly (Cooper, 1987).

Surgical Treatment

Surgical management is preferred because it decreases antigen load and inflammatory debris; it converts a chronic, fibrotic inflammatory reaction to a fresh acute reaction, thereby stimulating healing; and it assures adequate vascular perfusion and antibiotic delivery to tissues damaged by an ischemic inflammatory response. Fresh incisions heal more rapidly than primary injury or second-intention responses because there is no need for autolysis and phagocytosis, and fibroplasia can begin immediately (Remple and Remple, 1987).

Our experience has shown that the long-term use of antibiotics is necessary. Prior to this study, all raptors displaying infected feet and treated without surgery generally relapsed within 14 days of discontinuance of systemic antibiotics, regardless of length of prior treatment. Similarly, raptors undergoing surgical treatment tended to relapse if antibiotics were discontinued before 14 days. Staphylococcal disease of skin and underlying tissues requires long-term antibiotic treatment, in part because of the chronic granulomatous inflammatory response it generally elicits (Scott et al., 1978).

Foot Casting

Following surgery, casting yielded many advantages over other postoperative management regimes. It offers convenience in daily maintenance to both raptor and handler and greatly facilitates healing. In this study, raptors were allowed to eat on their own, to perch on surfaces of their choice, and even to fly free in an enclosed area, because their feet were completely protected from contamination and trauma. Pressure was removed from the operated area and transferred to the proximal plantar surfaces of the digits, well away from the incision. This minimized ischemia and micromovement of the incision edges, and fibrosis could proceed uninterrupted until healing was complete. Reseeding of encapsulated infection (not completely removed by surgery) was minimized because the incision area was protected from trauma. Reinfection of the incision was minimized by the large, readily accessible air space that insulated the bandaged foot from contaminated surfaces. Finally, and perhaps most important, casting eliminated the pathological changes that played a role in initiating disease for the duration of the healing period (Remple and Remple, 1987).

Results of Treatment

The success of surgical debridement and postoperative foot casting in the treatment of bumblefoot is clearly related to the stage of disease at the time of treatment initiation. One method of evaluating success is to estimate the percentage of raptors in a given class with recurring disease within a given time frame (for example, 12 mon) of initial treatment. Since there were no recurrences in Class I within 1 yr, this represented a 100% success rate. All raptors in Class V were euthanized; therefore, the success rate was 0%. Classes II, III, and IV fell between the two. The "known recurrences" weighed heavily on our statistics, yet it was impossible to assess accurately the present condition of all raptors in this study. The following variables had to be considered. Undoubtedly, more raptors had posttreatment disease than we knew. We could not be sure that many "known recurrences" were not the result of a return to conditions that initiated disease rather than an initial treatment failure. We knew that of seven raptors that were constantly under our care for 12 mon following initial treatment, only one relapsed. That failure had a *Pseudomonas* infection and relapsed within 2 wk of initial treatment, but was asymptomatic for more than 1 yr after the second operation. Therefore, in an attempt to place everything in perspective, we feel the "known recurrences" represented a reasonably accurate balance between unknown and/or unreported recurrent disease arising from improper conditions and those cases erroneously reported as recurrent disease.

Prevention

Prevention is the primary goal of disease management. A high nutritional plane assuring adequate vitamin A should be provided for all captive raptors. Perching surfaces should be cushioned, yet uneven and slightly abrasive. Unevenness assures that the weight of the bird will not be unrelentingly imposed over a single prominent area, but instead different areas of the foot will be involved each time the bird shifts its weight. Abrasiveness assists in the normal debridement of keratin buildup and assists in keeping talon length constant. Nervous raptors that constantly bate should bate onto a soft, cushioned surface to minimize contusive trauma to the feet.

Proposed New Classification of Bumblefoot

Depending on the stage of the disease, bumblefoot may range from barely perceivable tissue insult to the gross deformities of end-stage, crippling disease. A new classification is proposed (Table 27.1) that is based on the chronological sequence of events, the inflammatory exudative response, and the extent and depth of tissue destruction, all of which correlate with the ease of surgical debridement and efficacy of chemotherapeutic penetration into affected tissues. Since the effectiveness of surgical debridement and chemotherapeutic delivery decreases with each stage of advancing disease, the percentage of cases recurring is expected to increase with each stage. Using this as a criterion, together with the extent and depth of tissue disease, the new classification is intended to serve as a guideline for treatment and prognosis.

Bumblefoot is not a simple microbial-induced disease. Although the rate of success associated with surgery and postoperative foot casting is encouraging, much information regarding all aspects of bumblefoot remains to be discovered and addressed before we can feel confident about the cause, treatment, and prevention of this disease.

Table 27.1
A guide to classification and assessment of bumblefoot in raptors

Prognosis	*Class*	*Inflammatory response*
Excellent	Class I	early insult or lesion of a prominent plantar area with no apparent underlying infection A. bruise or early ischemia (a blanched area with poor capillary perfusion, before surface alterations appear) B. hyperkeratotic reaction (callus, corn)
Good	Class II	infection of underlying tissues in direct contact with the surface lesion but with no gross swelling A. puncture (with localized infection) B. local ischemic necrosis (a penetrating corn or scab)
Good to guarded	Class III	infection with gross pedal inflammatory swelling A. serous (usually resulting from a puncture in which an acute inflammatory reaction follows) B. fibrotic (a chronic, encapsulating reaction) C. caseous (a chronic, necrotic inflammatory reaction)
Guarded to poor	Class IV	infection with swelling of underlying tissues involving deep vital structures; chronic, producing tendosynovitis, arthritis, and/or osteomyelitis; when synovial structures involved, tissue exudate often combined with purulent synovium A. fibrotic B. caseous
Grave	Class V	extension of Class IV A, B, resulting in crippling deformity and loss of function

Acknowledgments

I am grateful to Dr. Lindsay Oaks, who assisted in the surgical treatment and casting procedures and in the compilation of data. His work is reflected in the proposed bumblefoot classification. Additionally, I wish to thank Cheryl Remple and Dr. Ulrich Wernery for their assistance in microbiological analysis. Finally, I am grateful to Mr. Clayton Smith for his assistance in the development and refinement of the "Bondo shoe" cast.

Product List

[a]Injacom 100 plus B-complex, Roche Animal Health and Nutrition, Hoffmann-La Roche Inc., 340 Kingsland St., Nutley, New Jersey 07110 USA

[b]Ketaset, Aveco Co. Inc., P.O. Box 717, Ft. Dodge, Iowa 50501 USA; or Aescoket, Aesculaap, Mijlstraat 35, 5280 AA, Boxtel, Netherlands

[c]Rompun, Haver-Mobay Corp., Animal Health Division, P.O. Box 390, Shawnee, Kansas 66201 USA; or Rompun, Bayer, Nijverheidsweg 26, 3640 AB Mijdrecht, Netherlands

[d]Fluothane, Ayerst Laboratory, 685 Third Ave., New York, New York 10017-4071 USA

[e]Forane, Anaquest, 2005 W. Beltline Hwy., Madison, Wisconsin 53713-2318 USA

[f]Chymar, Armour Pharmaceutical Co. Ltd., Eastbourne, England

[g]Ticar, Beecham Laboratories, 501 Fifth St., Bristol, Tennessee 37620 USA

[h]Pipracil, Lederle Pipracillin Inc., Carolina, Puerto Rico 00630 USA

[i]Nitrofurasone, Norwich Eaton Pharmaceuticals Inc., 13-27 Eaton Ave., Norwich, New York 13815-0231 USA

[j]Telfa, Colgate-Palmolive, 300 Park Ave., New York, New York 10022-7990 USA

[k]Vetwrap, 3M Animal Care Products, 225-5S 3M Center, St. Paul, Minnesota 55144 USA

[l]Hexcelite, Hexcell Medical Products, 11555 Dublin Blvd., Dublin, California 94568-2854 USA

[m]Bondo, Dynatron/Bondo Corp., 3700 Atlanta Industrial Park N.W., Atlanta, Georgia 30331-1098 USA

[n]Amiglide V, Aveco Co. Inc., P.O. Box 717, Ft. Dodge, Iowa 50501 USA

[o]Nebcin, Eli Lilly Industries Inc., Carolina, Puerto Rico 00630 USA

[p]Vemorsul, Beecham Laboratories, 501 Fifth St., Bristol, Tennessee 37620 USA

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CHAPTER 28

A Form-fitting, Composite-casting Method for Avian Appendages

KENTON E. RIDDLE, B.S., D.V.M.
JOHN HOOLIHAN, B.S.
Abu Dhabi Falcon Research Hospital
Abu Dhabi, United Arab Emirates

ABSTRACT: A new casting method with unique applications in birds of prey was developed using a combination of materials that provided precise anatomical fit and strength without bulk. The case was lightweight, rigid, and fast setting. For a variety of reasons, other casting materials have proven unsatisfactory for small species and for small or complex anatomical sites such as toes and feet. The method described here employs a soft, conforming, elastic, self-adherent wrap as substrate/padding material and an epoxy resin as a hardening agent.

From April 1986 to July 1988, 251 casts were applied to falcons. Of those, 214 were total-foot casts used as an adjunctive therapy in the treatment of bumblefoot. Casts were constructed so that pressure was removed from the plantar surface of the foot. The remaining 37 casts were applied in the treatment of fractures, severed tendons, self-inflicted trauma of the lower limb and phalanges, and wing sprains. This method allowed meticulous application of substrate/padding and precise positioning and conformity to anatomical parts without regard to time constraints prior to and during epoxy application. The casts were well tolerated in most cases and were of ideal form, consistency, strength, and weight. Casts were removed after 7 to 31 days by using a cast cutter or mechanical shears.

The ease of using this material has enabled us to train technicians in its various applications. The materials are also readily available, inexpensive, and easily adapted to a wide variety of treatment and research applications.

KEY WORDS: avian orthopedics, raptor, self-adherent bandage, falcon.

A variety of casting materials have been used in the management of avian fractures, dislocations, soft-tissue reconstructive surgery, and orthopedics (Brinker, 1965; Halliwell, 1980). Most veterinary casting materials are adaptations of medical products designed for humans. Recent notable exceptions that are lightweight, fast setting, durable, malleable, and nonadhesive to feathers include acrylics and thermoplastic casting materials that attempt to meet special requirements of avian patients (Halliwell, 1980; Harrison, 1986). However, these materials may not be ideal for conditions involving small or complex anatomical sites such as toes and feet or for small avian coaptation splints. The fast-setting characteristic of impregnated materials hardened by temperature or catalytic reaction can be a handicap. With such products, the operator is in a race against the setting time of the material while attempting a precise anatomical fit. In many products the amount of material exceeds the need, so that cost and wastage are important considerations.

This report presents a new casting method with unique applications to raptors. The method incorporates two readily available materials, an elastic self-adherent bandage[a] and a fast-setting epoxy resin[b] in a two-stage process.

Materials and Methods

The self-adherent bandage stretches by virtue of a porous elastic fabric gathered on parallel elastic fibers; it has a wrinkled surface in the contracted state. It is available in different colors and widths from 2.5 cm to 10 cm. Its supple elastic qualities make it the bandage of choice for anatomically complex appendages, especially in avian species. The wrinkled surface is ideal for receiving the liquid epoxy coating in preparation of the composite cast.

The clear, super-fast-setting epoxy adhesive, which combines an epoxy resin with a polymercaptan hardener, sets in 5 min.

In our practice a bandage 1.66 cm wide was needed for many of the specialty casts used in toe and foot casting. The 10-cm material was cut into six equal widths by using a scalpel blade and cutting through the entire wrap to the paper tube center (see Fig. 28.1). The parallel fibers provided a guide for making uniform cuts. The wrap was applied using minimal stretching to prevent constriction of the tissues or flattening of the wrinkled texture of the fabric. This preserved the padding and epoxy-holding qualities of the wrap.

The adhesive was set within 5 min after application. The bird was allowed to place weight on the cast after 15 min, and full bond strength occurred in $\frac{1}{2}$ to 1 hr.

The adhesive was prepared by thoroughly mixing equal parts of the resin and the hardening agent on a piece of paper. A wooden applicator stick was used to apply the adhesive to the wrap. Two coats were initially applied in

Figure 28.1. Materials required for composite foot cast.

succession, with care taken to avoid dripping. Leaving an area of epoxy-free wrap where the cast met the skin assured comfort.

For leg casts, stockinette and cast padding were followed by several thicknesses of elastic bandage/epoxy. A composite leg cast used in conjunction with a Schroeder-Thomas splint improved stability. Prior to the epoxy application, the wrap was adjusted, molded, or cut to provide a perfect fit, thereby eliminating potential pressure points.

When casting toes and feet, the wrap was usually the only padding necessary or desirable. Small strips of cotton padding were used to cover pressure points when casting toes that had suffered ruptured tendons. For these cases the cast incorporated the distal tarsometatarsus, thereby forming angular support and stability to prevent flexion or extension of the part.

For applications to wings, figure-of-eight splints were applied in the usual manner and then coated on the external surface to prevent removal or destruction of the bandage.

A detailed description of the composite foot cast for large falcons follows. Required materials are elastic self-adherent

Figure 28.2. An elastic self-adherent bandage (2.54 cm wide and 7.5 cm long) encircles the toe two times tangent to the foot.

bandage strips 1.66 cm and 2.54 cm wide, a cotton ball, epoxy, and a wooden applicator (Fig. 28.1).The falcons are anesthetized during the procedure.

Step 1: A strip of elastic, self-adherent bandage 2.54 cm wide is wrapped loosely around the proximal toes tangent to the foot, so that it encircles each toe two complete turns (Fig. 28.2). *Step 2*: A small amount of epoxy (approximately 1 cc) is thoroughly mixed. The ventral surface of each toe wrap is coated with an oval spot of epoxy. This serves as a buttress or support upon which the bird's weight is to be distributed, and prevents undue point pressure and impairment of circulation.

Step 3: A cotton ball is encircled five times with an elastic self-adherent bandage 2.54 cm wide, and formed into a compact cylinder by molding with the fingers. *Step 4*: This cotton bridge is placed over the ball of the foot so that it rests on each of the previously glued and hardened toe wraps (Fig. 28.3). *Step 5*: A strip of fabric 1.66 cm wide is used to anchor the cotton bridge to the foot while completing at least two thicknesses of the interdigitating bandage from mid-toe to approximately the lower fourth of the tarsus (Fig. 28.4).

Step 6: Fresh epoxy is prepared (approximately 6 cc) and applied in two coats, allowing for setting between coats. The distal toe wrap and the wrap on the metatarsus are not coated with epoxy, so as to provide a soft, padded transition between cast and skin (Fig. 28.5). An attempt is made to apply the thickest coating to the sides of the foot and cotton bridge.

The bird is allowed to recover as soon as the material has set. A weight-bearing cast is ready in 15 min. Within the following 24 hr, the bottom of the cotton bridge is removed with a cast cutter and the cotton and composite cast cylinder removed with a hemostat (Fig. 28.6).

The cast is usually left on for 17-21 days. Removal is accomplished using a cast cutter. Shallow longitudinal cuts are made in the dorsal aspect of each toe cast and continuing across the dorsal aspect of the foot cast. Bandage scissors

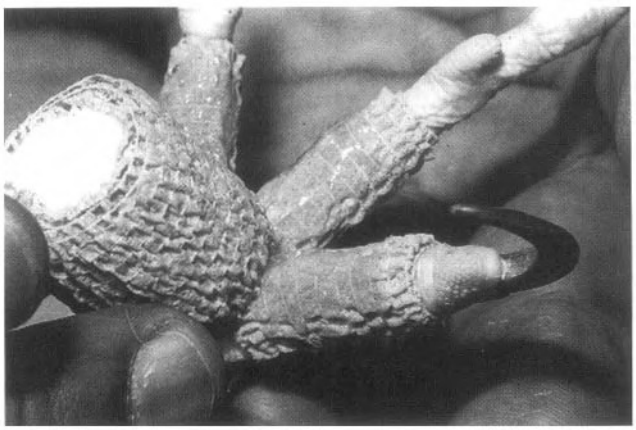

Figure 28.3. The bandage-cotton cylinder is placed over the oval spots of epoxy on the ventral aspect of each toe wrap.

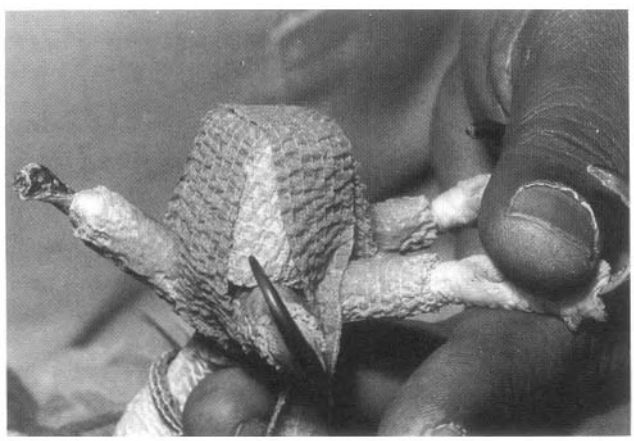

Figure 28.4. A 1.66-cm strip of elastic, self-adherent bandage is used to anchor the cotton bridge to the foot by an interdigitating wrap.

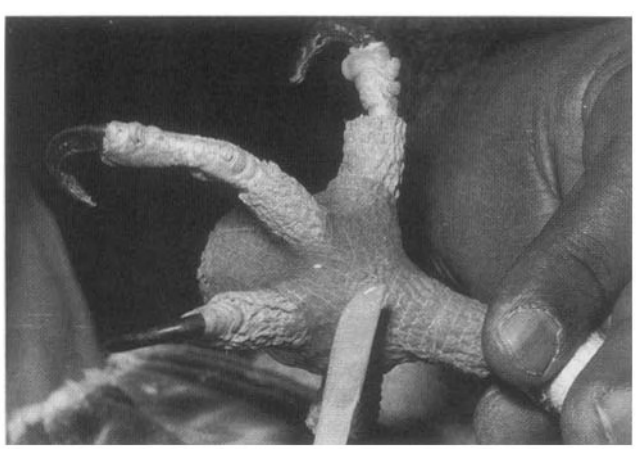

Figure 28.5. The bandage-cotton cylinder, proximal toe, and foot wraps are coated with several epoxy layers. Note that the wrap extends well beyond the area of epoxy coating.

follow the cutter line; the cast is spread, and the foot is removed through the top of the cast.

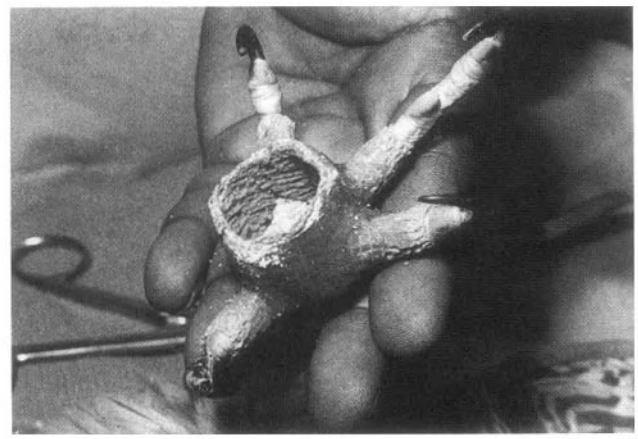

Figure 28.6. The bottom of the cast has been removed with a cast cutter, and the cotton bandage surrounding it has been removed. Note that the foot may be seen through the bottom of the cast.

When using a stryker saw to remove casts, care should be taken to avoid cutting the skin, since the saw blade will adhere to the scales and result in a ragged laceration. Bandage scissors complete the shallow stryker cut.

Results

From April 1986 to July 1988, 251 casts were applied to falcons. In 37 cases, composite coaptation splints were used to immobilize fractures of the wings and feet, wing sprains, and digits with ruptured tendons, and to guard against self-mutilation of the toes and feet. Toe and leg splints were lightweight and well tolerated. A total of 214 composite foot casts were used as adjunctive therapy in the surgical treatment of bumblefoot (Riddle, 1980; Remple and Remple, 1987). The foot casts effectively eliminated pressure from the plantar surfaces of involved feet.

Casts were well tolerated in most cases and were of ideal form, weight, and consistency. Early experience during the development of the technique resulted in a few cases of postcasting constrictive toe edema. Occasionally, a raptor's picking at the cast resulted in the necessity of repeat casting. The above problems were associated with faulty technique and now are rarely encountered.

The falcons postured normally, walked, stood, and handled food with ease; casts appeared to be comfortable. Daily inspection was facilitated by direct visualization (or palpation) through cast opening. Procedures such as dressing changes or suture removal were possible through the cast opening. Casts used in the treatment of toe fractures and ruptured tendons were well tolerated and resulted in healing in all cases.

Discussion

A casting material that is ideal for all circumstances in a

wide variety of animal species may not be found. There are unique advantages offered by the two components employed in the casting technique described above. The fact that components are used in two separate stages allows precise placement of the elastic self-adherent bandage, which achieves anatomical fit without regard to time constraints inherent in other materials. The process is completed by the simple application of liquid epoxy, which sets in 5 min, is semimoldable for an additional 3 to 5 min, and becomes rigid in 15 min. Strength and thickness can be varied for raptor size and anatomical parts by using more or less of the two components.

The technique follows routine casting principles and is easily accomplished by technical staff. The casts are clean and formfitting, unlike ball casts, which have been previously recommended for use on small avian patients (Redig and Roush, 1978; Wallach, 1975). The application of a total-foot cast, which is the most time-consuming and complex technique reported here, requires a total of 15 min. Epoxy-resin composite casts are inexpensive. The monetary cost of casting materials for a large falcon's leg or applying a total-foot cast is the cost of one-half tube of epoxy (14 g) and 50 cm of elastic self-adherent bandage, 1.66 wide.

The composite cast offers the advantage of variation when desirable, such as the incorporation of "operative windows" or metallic appliances. When a porous cast is desired, epoxy is simply omitted from certain aspects of the cast.

It is our hope that the epoxy composite cast described here will meet the requirements of the avian clinician for innovative casting solutions to emerging avian problems.

Product List

[a]Vetrap or Coban, 3M Co., 225-5S 3M Center, St. Paul, Minnesota 55144 USA

[b]5 Minute Epoxy, Devcon Corp., 30 Endicott St., Danvers, Massachusetts 01923 USA

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Part IV

Medicine and Therapeutics

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CHAPTER 29

Physiological Assessment of Rehabilitated Raptors prior to Release

SUSAN B. CHAPLIN, Ph.D.

LORI R. MUELLER, M.S.

Department of Veterinary Biology
University of Minnesota
St. Paul, Minnesota, U.S.A.

LAUREL A. DEGERNES, D.V.M.

The Raptor Center
University of Minnesota
St. Paul, Minnesota, U.S.A.

ABSTRACT: Blood lactate levels were measured for 10 min following standardized indoor exercise (10 hallway flights) in rehabilitated red-tailed hawks (*Buteo jamaicensis*), rough-legged hawks (*Buteo lagopus*), and great horned owls (*Bubo virginianus*) in order to determine (1) whether lactate response to exercise was a good indicator of flight fitness and (2) if blood lactate and postexercise respiratory rate were correlated.

Blood lactate levels peaked at 100 to 120 mg/dl at 2 min postexercise and gradually decreased to or below preflight levels (50 to 80 mg/dl) within 10 min in well-conditioned raptors. In contrast, unconditioned raptors exhibited peak (2 min) blood lactate levels over 200 mg/dl, and recovery was not complete for 15 min. There was no correlation between postflight respiratory rate and blood lactate levels during the flight conditioning period. It was concluded from these studies that raptor patients admitted for mild injuries whose medical treatments lasted 1 mon or less could be reconditioned by daily flights in just 3 wk. Patients experiencing long-term cage confinement required 6 or more wk of flight conditioning.

KEY WORDS: blood lactate level, respiratory rate, red-tailed hawk, *Buteo jamaicensis*, rough-legged hawk, *Buteo lagopus*, great horned owl, *Bubo virginianus*, flight conditioning, physiological assessment, exercise, performance testing, raptor.

Approximately 6,000 raptors have been treated at the Raptor Center (TRC) at the University of Minnesota for injuries or other medical problems since the inception of the program in 1972. The average time spent by raptor patients in convalescence and rehabilitation at TRC was 10 to 12 wk, but some birds remained in the clinic for 6 mon to 1 yr before release. In other performance athletes (humans, dogs, thoroughbred horses), lack of exercise and training quickly results in a marked decline in physiological fitness (Newsholme and Leech, 1983; Thornton et al., 1983). We assumed that a prolonged period of cage rest must result in a decline in flight fitness in raptors as well. The primary goal of the rehabilitation period is to assist releasable individuals in regaining their previous level of fitness through exercise. It is imperative that these birds be reconditioned as closely as possible to the performance level of a wild individual. If their flight fitness and stamina are inadequate, they will be inferior competitors in the wild.

Certain behavioral and physiological responses to exercise are routinely monitored during the course of the rehabilitation program at TRC. Some of the criteria for determining readiness for release are (1) willingness to fly, (2) ability to sustain flight and gain altitude, (3) exhibition of normal range of motion in both wings and in legs, (4) packed (red) blood cell volume of 40-45%, (5) plasma protein levels of 3.2-4 mg/dl, and (6) a decrease in respiratory rate of 25-40% within the first 2 min postexercise (Martell and Redig, 1986). These assessments may provide an index of "readiness," but, with the exception of blood parameters and respiratory rate, they are subjective and are not a practical test of flight performance.

In this study, we devised a performance test for some of the common raptor species that involves sprint-exercise conditioning of birds over a standardized course and measurement of their blood lactate response to the exercise. Accumulation of blood lactate in response to exercise reflects dependence on anaerobic metabolic pathways and thus indicates inadequacy of the oxygen supply to working muscles. Race trainers have found that as fitness increases in thoroughbred horses with endurance conditioning, their aerobic capacity improves, and levels of lactic acid produced during moderate to high levels of work remain rela-

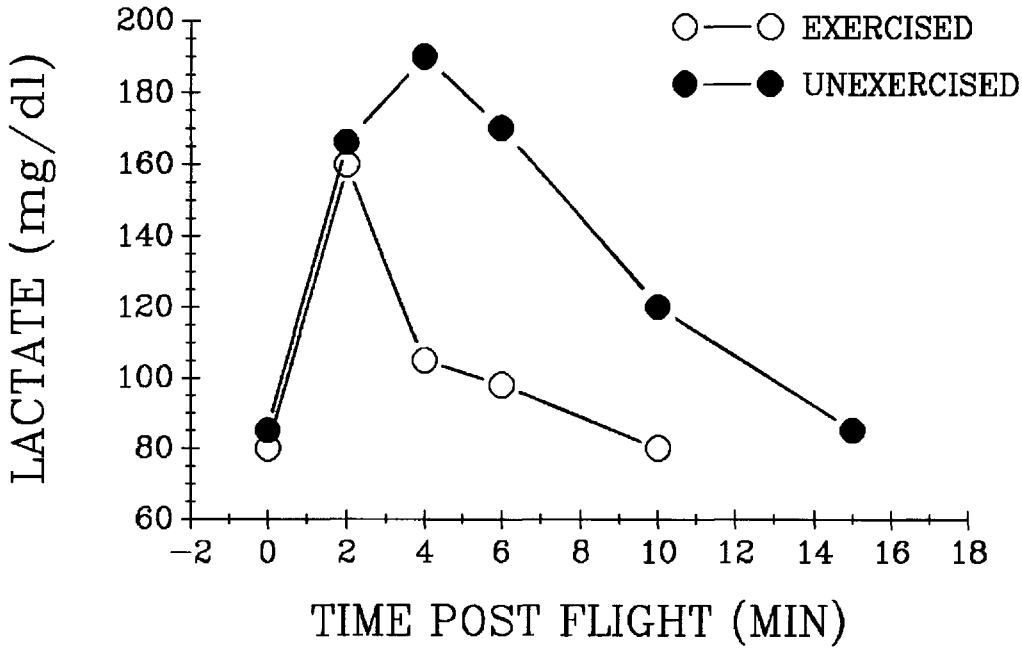

Figure 29.1. Blood lactate concentration (mg/dl) of two red-tailed hawks as a function of time (min) following standardized flight exercise. Open circles are data from a well-conditioned bird just prior to release. Closed circles are data from a hawk that had been housed in a flight room but never exercised. (Reprinted with permission from *Wildlife Journal*.)

tively low (Rose et al., 1983; Persson, 1983; Thornton et al., 1983). In unconditioned individuals, however, the levels of lactic acid rise rather quickly at these same work levels, and, eventually, the acidotic condition of the muscle limits (or stops) the work. The purpose of this study was to determine whether lactate response to exercise could be used as an indicator of physiological fitness in raptors; additionally, we hoped to determine whether there was any correlation between blood lactate level and respiratory rate in response to exercise and conditioning.

Methods

Experimental Animals

Seven red-tailed hawks (*Buteo jamaicensis*), two rough-legged hawks (*Buteo lagopus*), and three great horned owls (*Bubo virginianus*) were used in this study. All had been admitted to TRC for treatment and had been confined to cages for 3 to 30 wk prior to beginning flight training. All of the birds were maintained in a large flight room (5.5 m × 3 m × 3 m), where they were fed rats, mice, or guinea pigs every other day.

Exercise Protocol

The birds were trained to fly down a 25 m corridor with free-standing perches (0.6 m high) at each end. Each bird was expected to fly 10 lengths of the corridor three times a week, but was not pushed to fly the entire distance if it became overheated or overly tired. The time between successive corridor flights was 20 sec. At the conclusion of the exercise, respiratory rate was counted at min 1, 3, 5, and 10 during recovery, two days a week, and blood samples were taken at 2 and 10 min following the third exercise period.

Once the birds could fly 10 lengths of the corridor easily, they were taken outdoors and flown 50 to 100 m on a light nylon rope, two to three times a week, alternating with the days on which corridor flights were done. No blood or respiratory data were taken following this exercise.

Body Temperature Measurement

Body temperatures were measured during and following exercise with temperature transmitters[a] that were force-fed to one red-tailed hawk and one great horned owl. The transmitters had been calibrated in a water bath over a temperature range of 30° to 40°C (accuracy to 0.1°C) prior to use. Body temperatures were monitored concurrently with measurements of respiratory rate at min 1, 3, 5, and 10 following the conclusion of exercise.

Lactate Analysis

Blood lactate was determined from whole blood, using a Sigma assay kit.[b] Whole blood (0.1 to 0.2 ml) was extracted

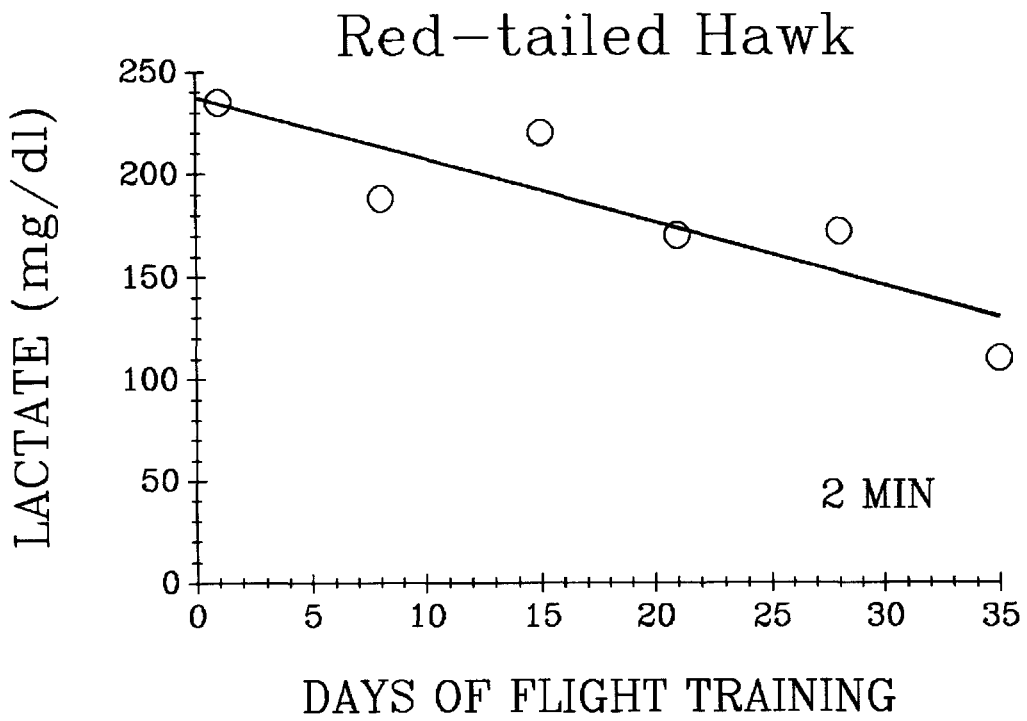

Figure 29.2. Blood lactate concentration (mg/dl) 2 min postexercise as a function of days of flight training in a red-tailed hawk. A best-fit linear regression of individual data points was obtained by computer. (Reprinted with permission from *Wildlife Journal*.)

with twice the volume (0.2 to 0.4 ml) of ice-cold 8% perchloric acid to precipitate plasma proteins. Samples were maintained on ice for 10 min, then centrifuged for 10 min at 2,500 rpm. The supernatant was pipetted off and refrigerated in a sealed tube until assayed. The lactate assay was performed by adding buffer, water, NAD, and LDH enzyme to 0.1 ml of the supernatant and incubating in a 37°C water bath for 30 min. The absorbance of the mixture was then read at 340 λ (UV) on a spectrophotometer.[c] Plasma lactate in mg/dl was obtained by multiplying the corrected sample absorbance by 65.1 (as per assay instructions).

From a pilot study, we determined that lactate levels in the blood peaked at 2 min and returned to preflight levels by 10 min in the well-conditioned bird (Fig. 29.1). Therefore, in all other flight trials, we took blood samples prior to exercise and at 2 and 10 min postexercise. Blood samples were taken from a wing vein while the bird was restrained in dorsal recumbency. Care was taken to ensure that samples were obtained within 30 sec of the desired 2 and 10 min sampling time. Blood sampling following exercise was done once a week during the course of the bird's rehabilitation until the time of release.

Blood was also taken from an additional two red-tailed hawks and two great horned owls, anesthetized with a mixture of a 4.4 mg/kg ketamine[d] and 2.2 mg/kg xylazine,[e] in order to determine the basal levels of blood lactate in these species. The dosage used was that given for short-term anesthesia of raptors by Degernes et al. (1988). Following IV administration of the anesthetic, 0.1 ml blood was drawn from a wing vein at 10 to 20 min postinduction.

A red-tailed hawk that had been trained for falconry and was currently being used for hunting was exercised over the standardized test course, in the same way as the rehabilitation patients. Blood samples were drawn at 2 and 10 min and lactate analyses were performed as described above. This individual represented our ideal endpoint of a physically fit hawk, and its postexercise lactate values were used as standards with which to compare the rehabilitated hawks.

Results

A steady decrease in both the 2 and 10 min lactate levels was observed in individuals of all three species during the course of their flight training. Linear regressions were fitted to the data by computer[f] so that individuals could be compared (Fig. 29.2). Generally, we found that the decline in 2 and 10 min blood lactate levels over days of flight training was steeper in birds that required more than 3 wk of flight training. Some of the red-tailed hawks that were admitted with complicated wing fractures required months of mild exercise to regain use of the wing and flight fitness. These individuals typically exhibited 2 min blood lactate levels over 200 mg/dl and 20 min lactates over 100 mg/dl early in

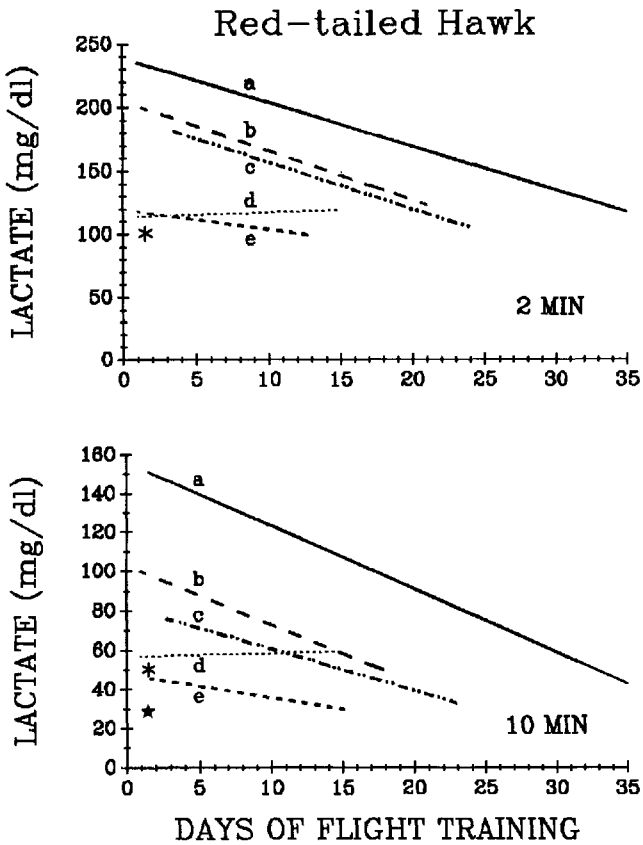

Figure 29.3. Best-fit linear regressions of blood lactate concentration (mg/dl) 2 and 10 min postexercise as a function of days of flight training in five red-tailed hawks (a to e). The asterisk represents data from a falconry-trained red-tailed hawk, a "fit" bird; the star represents mean lactate concentration of two red-tailed hawks anesthetized with mixture of ketamine-xylazine for 10 min. (Reprinted with permission from *Wildlife Journal*.)

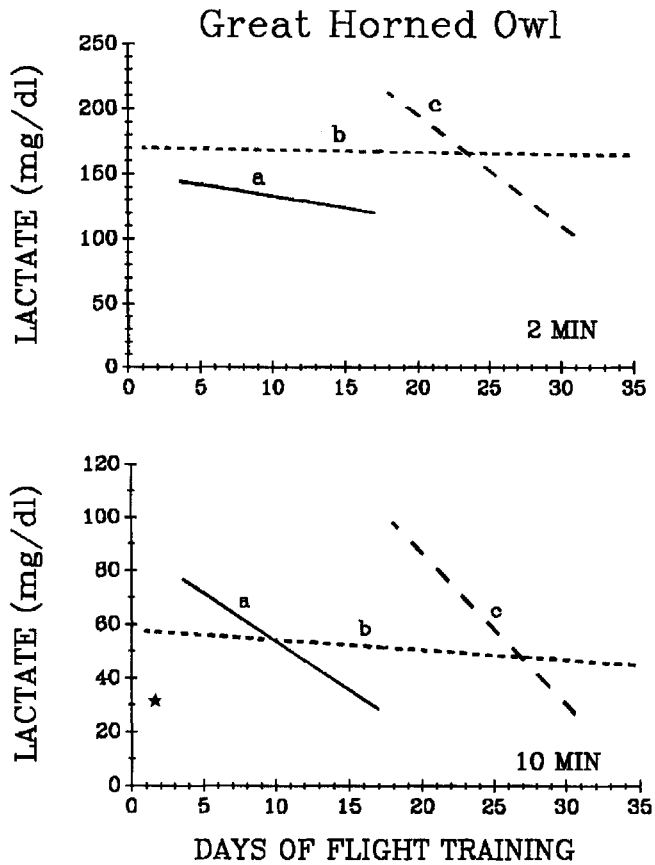

Figure 29.4. Best-fit linear regressions of blood lactate concentration (mg/dl) 2 and 10 min postexercise as a function of days of flight training in three great horned owls. The star represents mean lactate concentration of two great horned owls anesthetized with a mixture of ketamine-xylazine for 20 min.

the training program (for example, Fig. 29.3, bird a). In contrast, red-tailed hawks admitted with minor injuries recovered much more quickly during convalescence, lost much less fitness, and consequently had a shorter reconditioning period (for example, Fig. 29.3, bird e). Some individuals required no more than 2 wk of flight conditioning prior to release.

The relationship of recovery of flight fitness and severity of injury in great horned owls was similar to that observed in red-tailed hawks. One owl failed to make any progress in 2 min lactate levels (Fig. 29.4, bird c), although the 10 min lactate levels were reasonably low throughout the training period. This individual was admitted with an open fracture of the left humerus and had an old, healed left ulna fracture. During flight training, her flight became progressively more labored, her left wing extension never improved, and she was eventually dropped from the training program.

The time course for reconditioning of the two rough-legged hawks was much the same as that for the other species. One individual suffered a compound fracture of the ulna (Fig. 29.5, bird a) and required a much longer flight conditioning program than the other bird, which had sustained a soft-tissue injury to the wrist.

The 2 and 10 min lactate levels of a falconer's hawk postexercise were used as standards for comparing the progress of rehabilitated birds: lactate level 2 min postexercise was 99 mg/dl, at 10 min it was 47 mg/dl in this bird. After 3 to 6 wk of flight training, all of the hawks and owls exhibited lactate levels at 2 min postexercise of 100-120 mg/dl, which was considered to be the endpoint of the training. In well-conditioned hawks and owls, lactate levels 10 min postexercise (30-50 mg/dl) were well below the preflight lactate levels (40-80 mg/dl), and were closer to that of anesthetized hawks (24 mg/dl) and owls (30 mg/dl). Therefore, we feel that the 10 min lactates represent a basal level for these birds and can be used as a standard for recovery from exercise.

Postexercise respiratory rates of the hawks and owls used in this study did not change significantly with flight training. We determined the percentage change in respiratory rate from 1 min to 3 min postflight and compared individu-

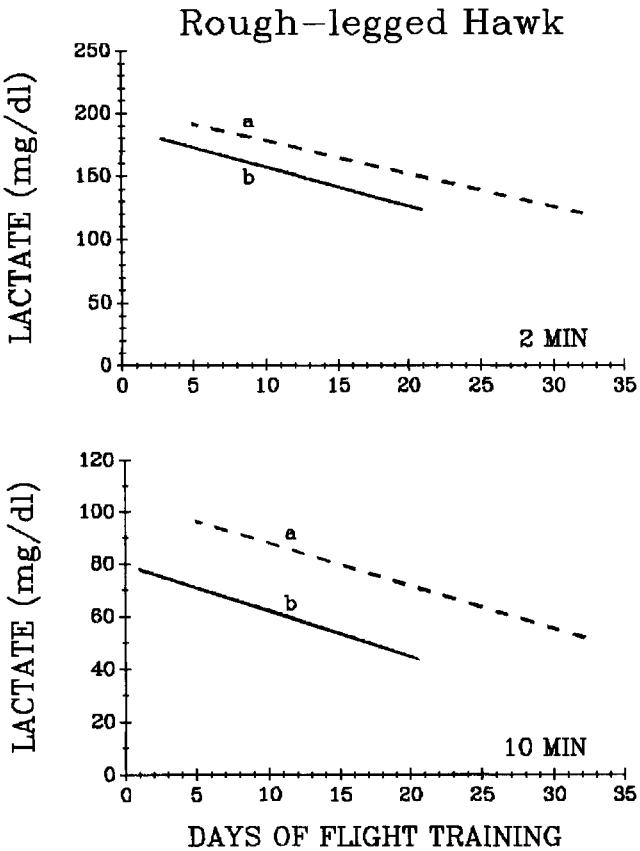

Figure 29.5. Best-fit linear regressions of blood lactate concentration (mg/dl) 2 and 10 min postexercise as a function of days of flight training in two rough-legged hawks.

als during the early and late phases of their flight training (Table 29.1). In almost all cases, the percentage change was the same or higher during the late phase, compared with the early phase. In contrast, blood lactate levels in these individuals decreased throughout their training period.

One reason for the elevated respiratory rates observed postexercise in the hawks could be their need to dissipate heat by panting. We confirmed that both hawks and owls experienced a mild hyperthermia (body temperatures of 42-43°C, compared with normal hawk T_b of 41.1°C and normal owl T_b of 39.5°C; Chaplin et al., 1984) for 10 min postexercise (Fig. 29.6). Simultaneous observations of rapid, shallow breathing in the hawks (>100/min) and elevated body temperature support the idea that respiratory rate of hawks postexercise has more to do with thermoregulation than with repayment of oxygen debt and metabolism of lactate.

In the owls, heat dissipation by gular flutter was continuous for the first 3 to 5 min postexercise and frequent for the next 5 min. The gular flutter mechanism is independent of respiratory movements, and therefore in the owls respiratory rate declined more quickly to near resting levels than in the hawks during recovery from exercise (Fig. 29.7).

Table 29.1
Percentage decrease in respiratory rate from 1 min to 3 min postexercise in red-tailed hawks and great horned owls, determined early and late in the flight training program

Species ID	*Early*	*Late*
RTH 183	44.6	38.7
RTH 165	42.5	39.5
RTH 154	21.9	31.8
RTH 135	36.6	48.1
RTH 625	15.8	9.0
Mean (S.D.)	32.2 (12.7)	33.4 (14.8)
RTH (Martell and Redig, 1986)	—	40.5
GHO 174	29.6	14.9
GHO 618	34.0	23.8
GHO 604	23.2	34.7
Mean (S.D.)	28.9 (5.4)	24.5 (9.9)
GHO (Martell and Redig, 1986)	—	24.0

Discussion

The change in lactate levels in the blood following exercise in raptors is very similar to that observed in mammals, with a sharp peak occurring immediately after exercise and a gradual decline to basal levels (Newsholme and Leech, 1983). Lactate accumulation in response to exercise is regarded as an indicator of the degree of conditioning, because it reflects the dependence on anaerobic instead of aerobic pathways and, thus, the adequacy of the oxygenation of working muscle.

Endurance training (aerobic exercise) promotes slower glycogen utilization and lower muscle and blood lactate production during submaximal exercise (Persson, 1983). However, in sprint exercise, which we have used in flight training raptors in this study, the initial high demand for ATP in the working muscle must be met by anaerobic metabolism (Newsholme and Leech, 1983). Sprint training in humans can actually improve the rate of ATP production 25% more than continuous aerobic training when short bursts of intense exercise alternate with periods of rest (Newsholme and Leech, 1983). Furthermore, sprint exercise improves phosphocreatine and myoglobin stores in the muscle (Newsholme and Leech, 1983). After a period of this type of training, blood supply to the muscle increases due to increased capillary circulation and cardiac output, and this further enhances oxygen supplies, which, in turn, facilitate aerobic oxidation of muscle energy stores (Newsholme and Leech, 1983). Therefore, training using the kind of short-distance exercise employed in this study encourages development of aerobic conditioning. Improvement in aerobic capacity is evidenced from the decline of 2 and 10 min lactate levels, and thus a decline in dependence on anaerobic metabolism, throughout flight training in all three species examined.

Linear regressions of 2 and 10 min lactate data over the

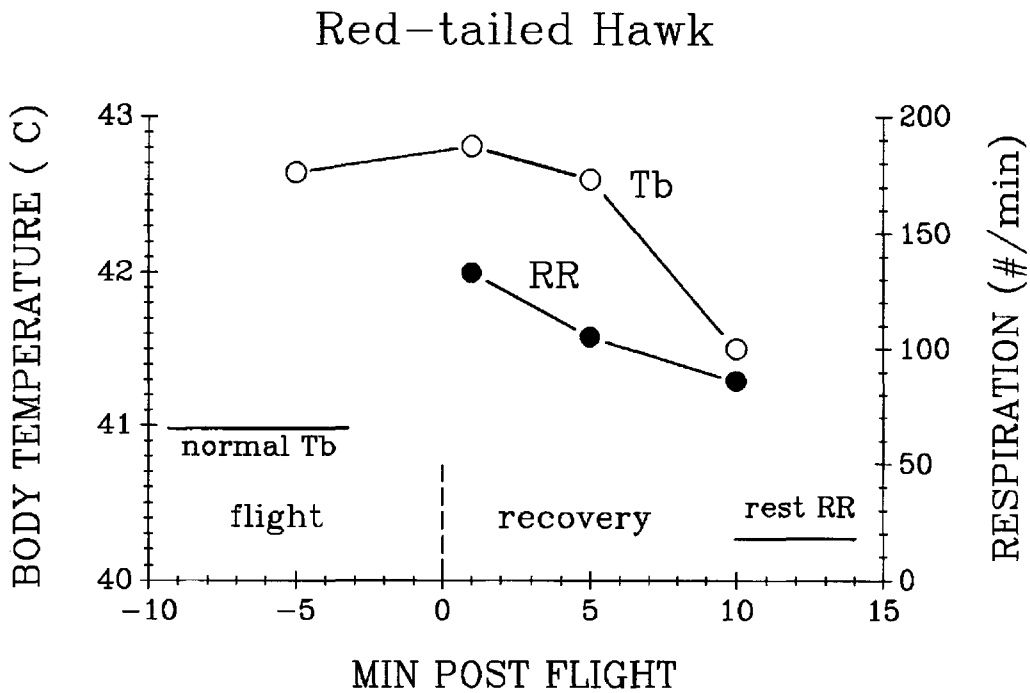

Figure 29.6. Body temperature (T_b, open circles) and respiratory rate (RR, closed circles) of a red-tailed hawk during and after flight exercise. Normal daytime T_b of a hawk is 41°C (Chaplin et al., 1984).

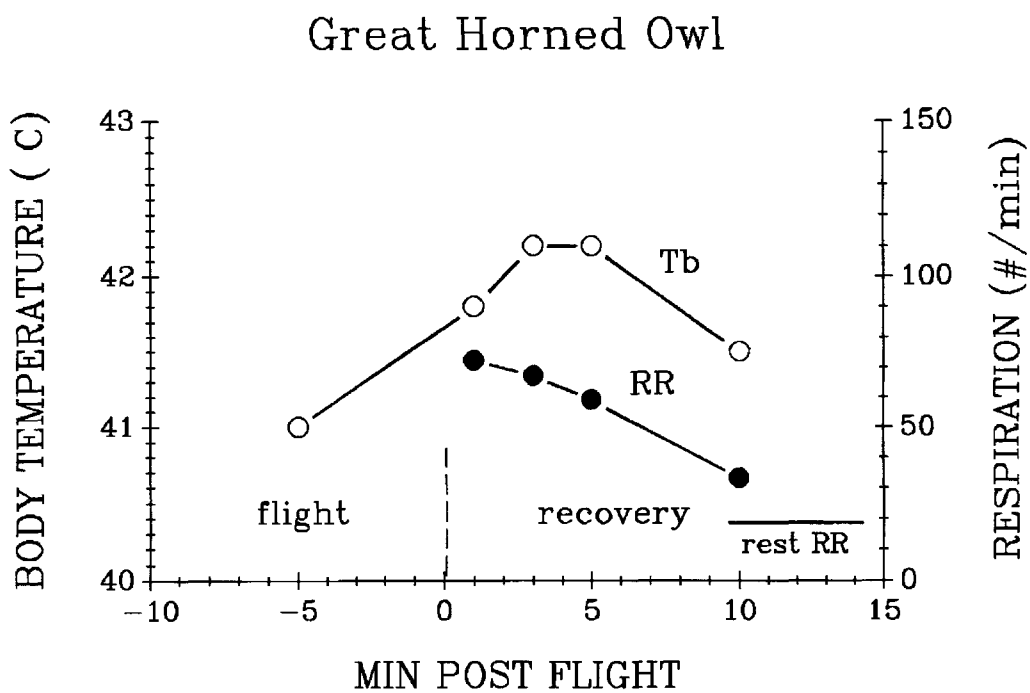

Figure 29.7. Body temperature (T_b, open circles) and respiratory rate (RR, closed circles) of a great horned owl during and after flight exercise. Normal daytime T_b of an owl is 39.5°C (Chaplin et al., 1984).

training period provide some useful information about the progress of the training. The difference between the initial 2 min lactate level and the desired endpoint (100 mg/dl) gives an indication of how much training will be needed. For example, birds with complicated wing fractures that need a long period of immobilization will typically have initial 2 min blood lactate concentrations in excess of 200 mg/dl. Such an individual will probably require more than 4 wk of

flight training. In contrast, a bird admitted for bruises or skin abrasions, which require a shorter period of treatment for full recovery, will typically have initial 2 min lactate levels around 150 mg/dl. Such individuals should approach the 100 mg/dl endpoint within 2 to 3 wk of flight training.

The slope of the regression of blood lactate versus days of training indicates how rapidly the conditioning is proceeding; if the slope is too flat and the 2 min lactate concentrations are much higher than the 100 mg/dl endpoint, then the bird may have a problem with the mechanics of the joints, bones, or muscles of one or both wings, or the training itself may be inadequate (i.e., too few flights, or too few exercise periods per week).

Finally, and most important, the 2 and 10 min lactate levels determine when good aerobic condition has been achieved, and at what point the bird should be considered a candidate for release. In this study we found that after a standardized exercise of 10 flights (for a total of about 300 m), well-conditioned great horned owls and red-tailed and rough-legged hawks had 2 min lactate levels of 100-120 mg/dl, returning to basal levels (30 mg/dl) within 10 min.

The lactate response to exercise should be evaluated only in birds that are accustomed to the exercise technique and the blood sampling regime. Naive, well-conditioned birds will probably yield higher lactate values than expected because of the stress of the exercise trial.

Respiratory rate of hawks and owls postexercise was a poor indicator of physiological fitness in this study, and we do not feel that it should be a criterion for release. Respiratory rate is complicated by thermoregulatory demands for heat dissipation in large-bodied raptors. Further, no improvement in recovery of respiratory rate could be demonstrated throughout the flight training period, even though there was significant improvement in the 2 and 10 min lactate levels of the same individuals.

We have only scratched the surface of this look into raptor exercise physiology by examining the response to short-distance sprint exercise in rather sedentary large-bodied raptors. Much more information could be added to this baseline by measuring (1) the lactate responses to exercise in raptors of different sizes, wing area to body weight ratio, or flight speed and foraging style; (2) the lactate response to "work" over different distances; and (3) the effect of line versus cage flight exercise on development of aerobic capacity. Eventually we hope to use the lactate data to program the type of exercise required for rapid reconditioning in particular raptor species.

Acknowledgments

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Product List

[a]Mini-Mitter Model X-M, Mini-Mitter Co. Inc., P.O. Box 3386, Sunriver, Oregon 97707 USA

[b]Pyruvate-lactate assay No. 826 UV, Sigma Chemical Co., P.O. Box 14508, St. Louis, Missouri 63178 USA

[c]Beckman DU 20, Beckman Instruments, Inc., 2500 Harbor Blvd., Fullerton, California 92634 USA

[d]Ketaset, Aveco Co. Inc., P.O. Box 717, Ft. Dodge, Iowa 50501 USA; or Aescoket, Aesculaap, Mijlstraat 35, 5280 AA, Boxtel, Netherlands

[e]Rompun, Haver-Mobay Corp., Animal Health Division, P.O. Box 390, Shawnee, Kansas 66201 USA; or Rompun, Bayer, Nijverheidsweg 26, 3640 AB Mijdrecht, Netherlands

[f]Sigma-Plot software, Jandel Scientific, 65 Koch Rd., Corte Madera, California 94925 USA

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CHAPTER 30

Soft-Tissue Wound Management in Avian Patients

LAUREL A. DEGERNES, D.V.M.
PATRICK T. REDIG, D.V.M., Ph.D.

The Raptor Center
Department of Veterinary Biology
University of Minnesota
St. Paul, Minnesota, U.S.A.

ABSTRACT: Management of soft-tissue injuries constitutes a majority of the treatments for injured wild raptors presented for rehabilitation. Proper wound management will improve the rate and quality of healing and the chances for successful release. Initial stages of wound management are similar to those used in mammalian medicine, and include patient stabilization; removal of feathers from the area surrounding the wound; debridement; wound flushing; wound closure, if indicated; application of dressing material; and immobilization and protection of the injury site with bandaging. Synthetic, nonadherent, moisture/vapor-permeable dressings developed for use in humans have been found to improve the quality of avian soft-tissue wound management greatly and to decrease wound healing time and complications.

KEY WORDS: raptor, soft-tissue injuries, wound healing, topical antibiotics, occlusive dressings, semiocclusive dressings.

Phases of Wound Healing

Wound healing is a complex interaction of host responses to an injury leading to regeneration of connective tissue, vascular supply, and epithelium. The three basic phases of wound healing are inflammatory (exudative), collagen, and maturation (Bojrab, 1981). It is important to understand the principles of wound healing in order to assess the wound and devise a treatment plan for optimal results.

Inflammatory Phase

The hemodynamic and cellular responses of the acute inflammatory response in birds have been studied in chickens (Carlson and Allen, 1969; Jortner and Adams, 1971; Awadhiya et al., 1980; Jain et al., 1982). Chemical mediators, such as bradykinin, histamine, and 5-hydroxytryptamine, initiate the acute inflammatory response immediately after the injury (Awadhiya et al., 1980). The response is similar in mammalian and chicken skin in the first 12 hr, with immediate vasoconstriction to control hemorrhage, followed by vasodilation within 30 min (Jortner and Adams, 1971). Large numbers of polymorphonuclear leukocytes (heterophils and basophils) and monocytes infiltrate the margins of the injured and necrotic tissue within the first 2 to 6 hr (Carlson and Allen, 1969; Jortner and Adams, 1971), causing active phagocytosis of necrotic cellular debris and bacteria. Increased vascular permeability of the capillaries and venules is thought to be mediated by histamine released from basophils and mast cells (Jain et al., 1982).

By 12 hr postinjury, the ratio of polymorphonuclear to mononuclear cells begins to shift toward a predominance of mononuclear cells (Jortner and Adams, 1971). During the next 36 hr, necrotic leukocytes that were active in phagocytosis accumulate at the periphery of the necrotic tissue, and are in turn phagocytized by macrophages and multinucleated giant cells. Fibroblasts appear in the wound during this period and continue to proliferate during the next few days, signaling the end of the first phase of the healing process.

Collagen Phase

There has been little work done in avian species to document the later stages of wound healing, necessitating extrapolation from mammalian studies. The collagen phase begins on the third or fourth day in chickens (Carlson and Allen, 1969) and approximately the fifth day in mammals (Bojrab, 1981) and lasts approximately 2 wk. During this phase, the fibroblasts synthesize collagen in the form of microfibrils, which aggregate into larger fibers over time. Capillaries develop from bud-like structures from nearby vessels and penetrate the wound. Wound contraction occurs and epithelial cells proliferate and migrate across the wound surface (Johnston, 1975, 1977).

Maturation Phase

The final phase of wound healing is marked by remodeling of the collagen bed and a decrease in the number of fibroblasts (Johnston, 1977). The weak, poorly developed collagen is replaced by collagen fibers that are thicker and stronger. The fibers become more oriented relative to the normal tension on the wound margins. In mammals, this phase may take weeks to months.

Impediments to Wound Healing

There are many factors that can potentially impair or prevent normal wound healing (Bojrab, 1981). Dehydration, starvation, severe protein deficiency, and chronic anemia may have adverse effects on wound healing. Necrotic tissue or blood clots may harbor bacteria and physically impede epithelial cell migration. Infection by pathogenic bacteria may significantly delay wound healing. Dirt, debris, dead bone, and even suture material may cause host reaction, leading to the development of fistulous tracts.

Tissue destruction resulting from desiccation, severe trauma in crushing or projectile injuries, or poor surgical technique will delay healing. Wounds of the distal extremities, especially the toes, tend to heal more slowly, possibly due to poor vascular supply. Nonimmobilized injuries over joint surfaces or those subjected to continual abrasion also heal more slowly.

Wound Assessment

Before deciding on the course of treatment for a soft-tissue injury, one must assess both the condition of the patient and the wound. Dehydrated, emaciated, and/or anemic patients should be stabilized before the wounds are treated. In raptors and other wild birds, this is especially critical, since there is often a delay of many hours to days following injury before the bird is presented for treatment.

After patient stabilization, a number of factors should be assessed before determining the course of treatment. These include the cause and location of the injury, the condition of the vascular supply to the area and associated orthopedic, muscle, and nerve damage, the age of the wound, and the extent of contamination present. The types of soft-tissue injuries in birds include puncture wounds, lacerations, abrasions, crushing wounds, thermal and chemical burns, and frostbite.

Every diagnostic workup should include a history, if available, and a thorough physical examination. Other diagnostic tests that may be useful in assessing the injury include microbiological cultures, hematology, radiology, and ophthalmologic examination. Vascular integrity may be evaluated by palpating the warmth of the limb, checking the capillary refill time of the skin, clipping a talon, or pricking the skin. Neurological tests modified from those used in mammals can be used to assess pain perception, proprioception, motor control, and so on. A green discoloration of the soft tissues may be present in wounds older than two to three days, due to accumulation of hemoglobin-breakdown products. Biopsies may be indicated in chronic, nonhealing, or self-inflicted wounds.

Wound Treatment

Ideally, fresh, uncomplicated wounds should be managed by first-intention healing (Bojrab, 1981). However, as many avian wounds are older than 8 hr or contaminated, this is often impossible and most soft-tissue healing is by second intention. In some instances, once a healthy granulation bed is established and the infection is under control, the wound may be sutured (third-intention healing).

Wound Preparation

The three basic considerations of wound management are cleansing, closing, and covering (Bojrab, 1981). If the wound is extensive and the bird is stable, these procedures should be done under isoflurane anesthesia.[a]

The feathers surrounding the wound should be carefully plucked, or trimmed if the skin is very friable. There should be a 2- to 3-cm feather-free zone of healthy skin around the wound. The wound should then be irrigated under a gentle stream of warm sterile saline or with an irrigating syringe to remove gross contaminants, debris, and blood clots. In cases of suspected bacterial infections, cultures for bacterial isolation and antibiotic sensitivity should be taken after surface contaminants have been removed. A 0.05% chlorhexidine diacetate solution[b] or 0.5-1.0% povidone-iodine solution[c] should be used to lavage the wound and provide antibacterial activity (Swaim and Lee, 1987). Hydrogen peroxide has been shown ineffective for bacterial infections, but may be effective as a sporicide in cases of suspected clostridial infections, or for initial cleansing of dirty wounds (Baldry, 1983). Chemical burns secondary to contact with caustic solutions, acids, or other irritants should be thoroughly washed and the compound neutralized by either sodium bicarbonate solution for acidic compounds or dilute vinegar for alkaline compounds (Perry, 1987).

Necrotic tissue should be surgically debrided and hemostasis achieved. In complicated or older wounds, the debridement process may have to be repeated over a period of days.

Primary wound closure should be done in fresh, uncomplicated lacerations, and in other selected cases, based on the nature of the wound and the surgeon's judgment. Most absorbable suture materials can be used in birds (2-0 to 5-0 size, with an atraumatic tapered needle). Closure may be appropriate in larger wounds that were allowed to heal by second intention, once a healthy granulation bed is established and the infection is under control.

Treatment options for soft-tissue wounds following this stage involve choices for topical medications, dressings, and bandaging materials. The treatment regimen is dependent on the type of wound, anatomical location, available materials, and preferences of the clinician. The purpose of

the following section is to outline the various choices and indications for each.

Topical Medications

The use of topical antibiotics may be beneficial. When non-water-soluble medications or ointments are used, the wound should be covered with dressing and bandaging material to prevent soiling of the feathers. Bacitracin, neomycin, and polymyxin are three antibiotics that may be used in combination topically against a wide spectrum of pathogenic bacteria (Swaim and Lee, 1987). One percent silver sulfadiazine[d] in a water-soluble cream has a broad antibacterial spectrum, and is indicated for topical treatment of thermal burns and other wounds (Perry, 1987). Nitrofurazone products slow epithelialization in mammals (Eaglstein and Mertz, 1981) and are probably not indicated for use in birds. Gentamicin sulfate cream has been shown to impair wound contraction in dogs (Lee et al., 1984), and should be avoided in birds. However, gentocin-containing ophthalmic preparations are effective for corneal abrasions.

Topical use of live yeast-cell derivative[e] has been shown to stimulate epithelialization and collagen synthesis for wounds in humans (Kaplan, 1984) and canines (Swaim and Lee, 1987). In raptors, this product has been used topically in granulating wounds over carpal joints and in granulating pododermatitis (bumblefoot) lesions (Degernes et al., 1990). Other topical medications used for the treatment of bumblefoot include camphor spirits and tincture of benzoin, and dimethyl sulfoxide (DMSO) in combination with dexamethasone and an antibiotic (Redig, 1987; Degernes et al., 1990).

Bandaging

Properly applied bandages provide an optimal environment for rapid epithelialization and wound contraction with the fewest complications. The functions of bandaging material are as follows (Swaim and Wilhalf, 1985):

1. to apply pressure in order to reduce dead space, swelling, edema, and hemorrhage
2. to protect the wound from pathological microorganisms
3. to immobilize the wound (and underlying fractures if present)
4. to protect the wound from desiccation and additional trauma from abrasions or self-mutilation
5. to absorb exudate and help debride the wound surface
6. to provide comfort for the patient

Bandages are typically composed of three layers—a primary or contact layer or dressing; the secondary or intermediate layer, consisting of an absorbent material; and the tertiary or outer layer, consisting of tape or elastic bandaging material.

Primary Layer

The primary or contact layer is the most critical layer for normal wound healing. This layer, which should be sterile, ideally remains in place even with patient movement, provides a moist wound environment, is comfortable, and assists with the debridement process (Swaim and Wilhalf, 1985).

Two basic groups of dressings include adherent and nonadherent dressings. Adherent dressings, such as fine-mesh or open-weave gauze pads, should be used only during the initial phase of wound treatment, when there is an excessive amount of necrotic debris that cannot be surgically debrided, or with excessive exudate production. Wet-to-dry bandage techniques (Bojrab, 1981; Cambre, 1984) involve the application of sterile saline-soaked warm gauze pads over the wound surface. The exudate and necrotic tissue will be mechanically removed with daily dressing changes during the first few days of treatment. The disadvantages of using wet-to-dry dressings routinely are that tissue maceration may occur with the very moist environment, and some disruption of the healing wound surface occurs with each dressing change. As a result, the dressing should be changed to a nonadherent dressing as soon as the exudation and necrosis have been controlled.

Nonadherent dressings are used during the granulation and epithelialization phase of wound healing, and by definition do not adhere to the healing wound surface (Lee et al., 1987). Two nonadherent dressings widely used in veterinary medicine include cotton nonadherent film dressings[f] and petrolatum-impregnated fine mesh gauze.[g] These products allow excess fluid to be absorbed into the secondary layer, while maintaining a moist wound surface. Although petrolatum dressings are superior for transfer of excess fluid to the absorbent layer, they have been shown to slow epithelialization in dogs (Lee et al., 1987) and are not recommended for use in birds because of feather soiling and loss of insulation. Empirical observations in avian wound management using cotton nonadherent film dressings have been that these dressings do in fact adhere to the wound surface when left in place for more than two to three days between dressing changes. This results in bleeding and disruption of the healing surface when removed.

Increased understanding of wound healing processes has resulted in the development of many new synthetic nonadherent dressings (Barnett et al., 1983a, 1983b; Eaglstein, 1984; Eaglstein et al., 1988). These dressings keep the wound surface moist and prevent scab formation, which significantly increases the rate of re-epithelialization, compared with air-exposed and wet-to-dry gauze dressings (Al-

varez et al., 1983). These dressings are either "occlusive" or "semiocclusive." Occlusive dressings are impermeable to moisture/vapor, while semiocclusive dressings are moisture/vapor permeable.

Occlusive dressings include flexible hydrocolloid dressings,[h,i] which are adhesive, opaque, and impermeable to moisture/vapor and oxygen. These hydrophilic dressings absorb fluid and exudate from the wound surface to produce a gelatinous, moist cover for the healing surface. They have been used successfully in zoo mammals and birds (Cambre, 1984; Gonzales-Tirado, 1987), as well as in a variety of wounds in raptors at the Raptor Center. Hydrocolloid dressings are commonly used for bumblefoot lesions (Degernes et al., 1990) and for slow-healing, granulating wounds over the carpal joints or keel. Despite adhesive tendencies, it may be necessary to use additional bandaging material to hold such dressings in place.

Semiocclusive dressings[j,k,l] are thin, transparent, flexible polyurethane membranes that are moisture/vapor and oxygen permeable, yet impermeable to liquids and bacteria, and allow fluid to collect under the dressing (Mertz et al., 1985). They produce a moist aerobic environment that promotes active phagocytosis of the necrotic debris and bacteria by the leukocytes. Prevention of wound desiccation and scab formation allows for unimpeded epithelial cell migration from the wound margins, and more rapid wound healing (Alvarez et al., 1983; Barnett et al., 1983a). When applied to an aseptically cleansed wound, these materials will maintain a sterile wound surface as long as the dressing margins remain adhered to the healthy skin surrounding the wound. Additionally, studies in human burn patients have shown a substantial decrease in wound pain with these dressings because exposed nerve endings are protected from desiccation (Barnett et al., 1983a).

Semiocclusive dressings have been used in hundreds of patients at the Raptor Center during the past three years, including more than 20 species of raptors, several species of swans, and a number of caged birds (Degernes, 1989). The flexible membrane can be effectively conformed to nearly every part of the avian anatomy, including difficult-to-bandage structures such as heads. In most cases they are used in combination with additional bandaging material to immobilize the injury site and to prevent removal by the patient.

The adhesives used with semiocclusive dressings have excellent properties when applied to clean, dry, detergent-free skin, yet do not adhere to the moist healing wound surface and therefore do not disrupt the healing process upon removal, nor do they leave a residue on the skin or feathers. The transparent film allows for visual monitoring of the wound healing and assessment of the quantity and quality of exudate production. There is often some accumulation of pus under the membrane as a normal by-product of phagocytosis. The amount of exudate production and the wound appearance should dictate the frequency of dressing changes. If the amount of exudate is such that leakage around the perimeter of the dressing occurs, the dressing should be changed to prevent bacterial invasion into the wound. Use of the product should be temporarily discontinued if there is gross evidence of infection, such as excessive redness, swelling, and odor. In general, it is recommended that the dressing be changed every one to three days during the initial healing stages, and then as infrequently as weekly, once a healthy granulation bed is established.

Avian wounds covered with semiocclusive dressings appear to heal much more rapidly than those covered with conventional dressings (for example, cotton, nonadherent film dressings). The rapid healing and fewer bandage changes result in less stress and fewer complications for the bird and savings of time and materials for the clinician.

Secondary Layer

The functions of the secondary layer are to absorb fluids and wound exudate, to pad the wound from trauma, and to immobilize the wound during the healing phases. Conforming gauze bandages[m] are the most suitable for figure-of-eight wing bandages and other types of bandages in birds. For figure-of-eight wing bandages, the bulkiness of the gauze should provide the padding and immobilization, rather than the tightness of the bandage (Redig, 1987).

Tertiary Layer

The tertiary, or outer, layer serves to hold the other layers of bandages in place. Conforming stretch tapes with adhesive surfaces[n] or without[o] work well as avian bandages. Self-adherent bandage material is preferred at the Raptor Center because the material conforms well to avian limbs, sticks to itself without leaving tape residues on feathers, is lightweight and porous, and is well tolerated by most species of raptors. If a raptor patient has a history of shredding its bandage, the tertiary layer is usually composed of white adhesive tape.

Conclusions

Recently introduced wound-care products that can interact with and enhance the physiological processes associated with wound healing have been used in avian species and found to decrease complications and wound healing time significantly. Initial stages of wound management are similar to those used in mammalian medicine and include patient stabilization, removal of feathers from the area surrounding the wound, debridement, wound flushing, application of dressing material, and immobilization and protection of the injury site with bandaging. Fresh wounds

may be sutured; however, most traumatic wounds are managed by secondary-intention healing. Application of topical antibiotics is not recommended; however, compounds with live yeast-cell derivative can enhance epithelialization in a granulating wound. Synthetic, nonadherent dressings of the occlusive and semiocclusive type are the most recent introductions and have greatly increased the efficiency and effectiveness of management of avian wounds.

Acknowledgments

We gratefully acknowledge 3M Animal Care Products for their generous donation of Tegaderm dressings and Vetrap bandages for use at the Raptor Center.

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Product List

[a]AErrane, Anaquest, 2005 W. Beltline Hwy., Madison, Wisconsin 53713-2318 USA

[b]Nolvasan, Fort Dodge Laboratories Inc., 800 5th St. N.W., Ft. Dodge, Iowa 50501 USA

[c]Betadine, Purdue Frederick Co., 100 Connecticut Ave., Norwalk, Connecticut 06856 USA

[d]Silvadene, Marion Laboratories Inc., Product Surveillance Dept., P.O. Box 8480, Kansas City, Missouri 64114 USA

[e]Preparation H, Whitehall Laboratories, Inc., Division of American Home Products Corp., 685 Third Ave., New York, New York 10017 USA

[f]Telfa Pads, Kendall Health Care Products Co., International Division Hospital Products, 1 Federal St., Boston, Massachusetts 02110 USA

[g]Nu Gauze Sponges, Johnson & Johnson Products, 501 George St., New Brunswick, New Jersey 08903 USA

[h]Dermaheal, Squibb, P.O. Box 4500, Princeton, New Jersey 08543-4500 USA

[i]DuoDERM, Squibb, P.O. Box 4500, Princeton, New Jersey 08543-4500 USA

[j]Tegaderm, 3M Animal Care Products, 225-5S 3M Center, St. Paul, Minnesota 55144 USA

[k]Op-Site, T. J. Smith and Nephew, Welwyn Garden City, Hertfordshire, England

[l]Bioclusive, Johnson & Johnson Products, 501 George St., New Brunswick, New Jersey 08903 USA

[m]Kling Gauze, Johnson & Johnson Products, 501 George St., New Brunswick, New Jersey 08903 USA

[n]Elasticon, Johnson & Johnson Products, 501 George St., New Brunswick, New Jersey 08903 USA

[o]Vetrap, 3M Animal Care Products, 225-5S 3M Center, St. Paul, Minnesota 55144 USA

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CHAPTER 31

Clinical Signs and Treatment of Large Birds Injured by Electrocution

DIETER HAAS, Dr.Med.

Stromtod-Dokumentationsstelle
des Naturschutzbundes Deutschland e.V.
Albstadt, Germany

ABSTRACT: Electrocution of large birds on the poles of medium-high-voltage power lines is a severe problem in Europe. Among raptors and storks, electrocution causes more losses than collisions with power lines. Such high losses may have contributed to local extinction of some endangered species. A considerable number of electrocuted birds do not die at once, but suffer severe injuries that, without early treatment, lead to irreversible damage or death. Symptomatology and successful therapies are described. Literature on the subject is scarce, most research having been done in Germany. More knowledge of this subject is considered essential for bird protection programs.

KEY WORDS: raptor, electrocution.

Among those raptors brought to the Bird Rehabilitation Centre in Albstadt, Germany, the main causes of injury are electrocution (Fig. 31.1), poisoning, and collisions. The relative frequency of electrocution in injured live birds is shown in Table 31.1. Often, electrocution can be determined as the cause only after careful examination and thorough collation of a bird's history.

Damage from exposure to high voltage may arise as a primary injury from the effects of current traversing the body or from secondary trauma associated with the fall from the power pole. Some birds may die outright from cardiac arrest caused by ventricular fibrillation (Haas, 1980). Others injured by current leakage may suffer muscle convulsions or be rendered unconscious. They may then suffer fatal trauma from the fall (Lenz and Zimmerman, 1990) or sustain nonfatal traumatic injuries such as skull and brain damage, long bone fractures, or fractures of the vertebral column with paraplegia (Fig. 31.2).

Other injuries associated with electrocution include necrotic wings and legs with burn marks, incineration of the talons, and severe burns to the mouth and beak (see Figs. 31.3-31.7). Permanent, secondary changes often accompany the trauma. Often the extremities become necrotic and mummified, or are damaged seriously otherwise because of local ischemia associated with shock and cardiac insufficiency. These types of injuries are listed in Table 31.2. Another possible secondary effect of electrocution is renal gout (Kiel, 1986), which is potentially fatal.

Survival following severe electrocution injuries is possible in some cases if appropriate emergency treatment is provided (Cooper, 1978; Redig, 1992). After electrocution, a bid usually sits apathetically at the base of the power pole in a state of shock. Several days may pass before it is found.

Figure 31.1. Electrocuted birds in the Bird Rehabilitation Centre at Albstadt, Germany, August 31, 1988. Live birds being held, from right: black stork (*Ciconia nigra*), red kite (*Milvus milvus*), peregrine falcon (*Falco peregrinus*). In front of the woman on the left are birds that had to be euthanized after being presented alive with extensive necrosis, including a European eagle owl (*Bubo bubo*), heron (*Ardea cinerea*), barn owl (*Tyto alba*), and European kestrels (*Falco tinnunculus*). On the table are white storks (*Ciconia ciconia*) that were dead when submitted. Photo: D. Haas.

When the bird is ultimately admitted to the clinic, it must receive standard emergency medical care (Redig, 1992). Briefly, this consists of rehydration with oral and intravenously administered fluids such as lactated Ringer's solution or 5% dextrose. For oral fluid administration, I prefer a 50/50 mixture of water and bovine serum[a] administered at rates up to 10% of the bird's body weight BID to TID. This is given with a syringe to which is affixed a small-diameter polyethylene or latex rubber tube that is inserted into the bird's esophagus while the head is extended obliquely. Additional shock treatment is provided by giving long-acting steroids[b] either IM or IV at 2 mg/kg.

Table 31.1
Number of electrocuted birds found damaged and still alive (January 1, 1984, to September 30, 1988)

Species	*Damaged by electrocution*		*All other cases*	*Total*	*Percentage of definite electrocution among total cases*
	Definitely	*Probably*			
Total	42	14	187	243	17
Heron (*Ardea cinerea*)	1	1	5	7	14
Red kite (*Milvus milvus*)	2	1	1	4	50
Black kite (*Milvus migrans*)	1	—	2	3	33
Buzzard (*Buteo buteo*)	17	4	69	90	18
Peregrine falcon (*Falco peregrinus*)	3	—	7	10	30
Hobby (*Falco subbuteo*)	—	1	2	3	0
European kestrel (*Falco tinnunculus*)	13	5	41	59	22
Long-eared owl (*Asio otus*)	—	2	6	8	0
Tawny owl (*Strix aluco*)	1	—	9	12	8
Little owl (*Athene noctua*)	—	—	3	3	0
Barn owl (*Tyto alba*)	2	—	5	7	28

Note: The birds shown here were all received by the Bird Rehabilitation Centre at Albstadt, Germany.

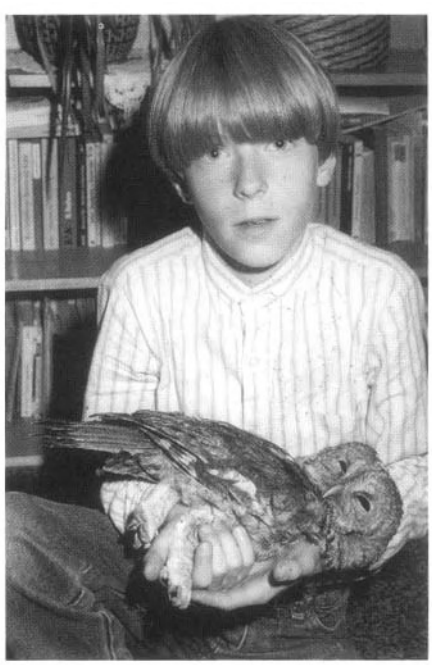

Figure 31.2. Electrocuted tawny owl (*Strix aluco*) with broken spine and paraplegia. Photo: D. Haas.

A careful medical examination follows, which should include radiographs to detect fractures. The burn marks are treated with antiseptic povidone iodine[c] or 1% silver sulfadiazine[d] in a water-soluble cream. Systemic antibiotics such as amoxicillin[e] are also provided as needed for wound treatment (Degernes and Redig, chapter 30, this volume).

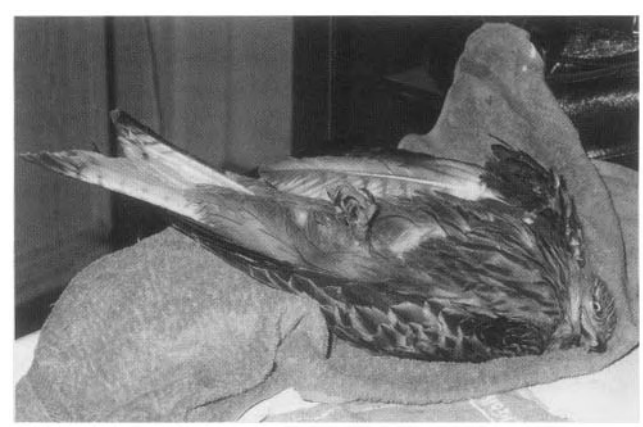

Figure 31.3. Female red kite found sometime after electrocution with necrotic feet and a necrotic wing. Note the scorched feathers on right alula. Myiasis, an infestation with dipterous larvae, was present. Photo: D. Haas.

Cautious force-feeding with ground, fatless meat is the next step. To avoid all unnecessary stress, the bird is housed in an isolation cage for several days. If the treatment is continued correctly, the bird will feed spontaneously after a few days. Rehabilitation will proceed as needed for several weeks for complete recovery from the secondary injuries.

Table 31.2
Major findings in larger birds that were electrocuted and found alive

	Damage by electrocution (electropathology)	*Other kinds of damage (mainly collisions with wires and vehicles)*
Main fractures	spinal fractures (with paraplegia), mainly dorsal and lumbar vertebrae	extremities, skull, bill, etc.
Plumage lesions	burnt little gaps in the plumage, often very discrete, difficult to find; rarely, widely scorched plumage areas	traumatic plumage damage
Skin lesions	very small burns on entry and exit locations of electric current; later, without treatment, large necrotic areas in the limbs through which electric current flowed	mechanical lesions (laceration, contusion); later, necrosis, infection, etc.
Follow-up damages on extremities	large necroses, often totally necrotic extremities in the areas through which current flowed; myiasis (see Fig. 31.3)	necrosis generally in the area of complicated injuries, infections
General condition	first, traumatic shock; later, without treatment, damage by necrotic limbs	shock, local damage by injury

Note: Electrocution damage to feet is sometimes mistakenly diagnosed as bumblefoot.

Discussion

Most birds that suffer electrocution on medium-high-voltage power lines die immediately. Any damage to the power lines is so negligible that the power companies do not record such events, although extensive damage to the lines occurs occasionally. Current leakage of 5 to 10 mA produces muscular paralysis, and current leakage greater than 10 mA causes death (Winfield, n.d.).

In southern Germany and many other parts of Europe, electrocution is a main mortality factor, which by itself might cause extinction of rare species (Fiedler and Wissner, 1980; Haas, 1980; VDEW, 1991). Though medical treatment can save many birds that survive electrocution, the most important measure that can be taken to reduce electrocution fatalities is to eliminate the construction of dangerous power-pole configurations and to change the existing ones. The most dangerous poles are constructed of prestressed concrete or of metal in combination with pin-type insulators in medium-high-voltage power lines (10 to 30 kv), but other poles are also dangerous. Haas (1980) gives a detailed description of dangerous poles in Europe, illustrated by graphs.

Figure 31.4. Electrocuted European kestrel with necrotic feet and a necrotic wing. The wing has already been reduced to the bone by myiasis. Photo: D. Haas.

Figure 31.5. Electrocuted peregrine falcon with damaged wing. The skin and feather loss were sequelae to electrocution. The bird was found in winter, and there was no myiasis. Photo: D. Haas.

In Germany there have been some important developments in the fight to protect birds from electrocution. A new "bird protection" paragraph in the guidelines for construction of power lines forbids the installation of new poles in dangerous configurations, and specifies that existing dangerous poles shall be made safe.

Until recently, in Germany the insulation of dangerous poles by bird-protector caps had been done mainly in the range of the European eagle owl (*Bubo bubo*) and of the white stork (*Ciconia ciconia*)—both highly endangered species.

Figure 31.6. Burnt feathers and early necrosis at the base of the carpus and necrotic foot of a European kestrel, found still alive, in summer, some days after electrocution. Photo: D. Haas.

Figure 31.7. Heavily scorched feather of a white stork killed by electrocution. Photo: D. Nill.

Many of the most dangerous poles in Germany have been insulated in the last few years, but in other parts of Europe the situation is unchanged. VDEW (1986) gives detailed descriptions of bad poles and proven safeguarding methods used in Middle Europe.

Conclusion

Information on electrocution of birds is still very sparse. In particular, detailed descriptions are missing. Because of its relevance to the preservation of endangered species and in raptor pathology, this subject warrants more attention in the veterinary literature.

Product List

[a]Boviserin, Hoechst Veterinar GmbH, 8044 Unterschleissheim, Germany

[b]Azium, Schering Corp., Animal Health Division, Kenilworth, New Jersey 07033 USA

[c]Betaisodona, Mundipharma GmbH, 6250 Limburg/Lohn, Germany; or Betadine, Purdue Frederick Co., 100 Connecticut Ave., Norwalk, Connecticut 06856 USA

[d]Silvadene, Marion Laboratories, Inc., Product Surveillance Dept., P.O. Box 8480, Kansas City, Missouri 64114 USA

[e]Amoxitabs, Beecham Laboratories, 501 Fifth St., Bristol, Tennessee 37620 USA

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Editor's note: Copies of the following can be ordered free from the author: Fiedler and Wissner, 1980; Haas, 1980; Kiel, 1986; Lenz and Zimmermann, 1990; VDEW, 1991.

CHAPTER 32

Preliminary Evaluation of the Effects of Dexamethasone on Serum Hepatic Enzymes, Glucose, and Total Protein in Red-tailed Hawks

G. E. KAUFMAN, D.V.M.
J. R. PAUL-MURPHY, D.V.M., Dip.A.C.Z.M.
M. FINNEGAN, D.V.M.

Zoological Medicine
Veterinary Medical Teaching Hospital
University of California
Davis, California, U.S.A.

ABSTRACT: Dexamethasone sodium phosphate (DexNaP) was administered IM and IV to red-tailed hawks (*Buteo jamaicensis*). Blood samples were taken before and at periodic intervals up to 120 hr after steroid administration. Packed cell volume and total plasma solids were unaffected, while glucose levels increased sixfold within the first 24 hr. Plasma concentrations of alanine aminotransferase and aspartate aminotransferase were elevated following intramuscular injection, but not after intravenous injection.

KEY WORDS: dexamethasone, red-tailed hawk (*Buteo jamaicensis*), steroid, liver enzyme, raptor.

A study was conducted to observe the magnitude and duration of changes in serum aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase, glucose, and total protein in captive red-tailed hawks (*Buteo jamaicensis*) following a single administration of dexamethasone sodium phosphate (DexNaP) at a dose of 4 mg/kg (Harrison and Harrison, 1986).

Corticosteroids have been used traditionally in the treatment of shock in mammals. Their use is believed to aid in the stabilization of cellular membranes, thereby minimizing the leakage of potentially damaging cellular components such as lysosomal enzymes and myocardial depressant factors. Corticosteroids produce sustained vasodilation, resulting in improved tissue perfusion and cardiac output, in addition to protecting and stabilizing capillary endothelium. Their use also increases available cellular energy through stimulation of gluconeogenesis, largely in the liver (Booth and McDonald, 1982). For these reasons, and due to their wide acceptance in mammalian medicine, corticosteroids are also commonly used in the treatment of severely debilitated avian patients. These patients are presumed to be experiencing a shocklike phenomenon, although shock has not been clearly defined in birds.

In mammals, corticosteroids are conjugated in the liver and excreted by the kidneys. A single injection has been known to produce hepatopathy in dogs and rabbits, characterized by ballooning degeneration, centrilobular necrosis, and leakage of serum hepatic enzymes (predominantly alkaline phosphatase in the dog). These effects are felt to be reversible on withdrawal of the drug. Rats, mice, and humans appear to be relatively resistent to this effect (Rogers and Ruebner, 1977; Booth and McDonald, 1982). The occurrence of such hepatopathies in birds has not been reported.

At the University of California, Davis, Zoological Medicine Service, emergency therapy for birds routinely includes IV fluids and IV or IM administration of dexamethasone sodium phosphate. Clinical observations have indicated that serum hepatic enzyme levels may be elevated 24 to 48 hr following initial hospitalization and emergency treatment of injured raptors. The objective of this study was to investigate the possible effects of corticosteroid administration on hepatic enzymes.

Materials and Methods

Sixteen mature red-tailed hawks were selected from the resident nonreleasable birds at the University of California, Davis, Raptor Center. These birds were randomly divided into four groups of four birds each:

Group 1 received a single IV injection of 4.0 0.9% NaCl.

Group 2 received a single IV injection 1 ml/kg normal saline.

Group 3 received a single IM injection of 4.0 mg/kg DexNaP.

Group 4 received a single IM injection of 1 ml/kg 0.9% NaCl.

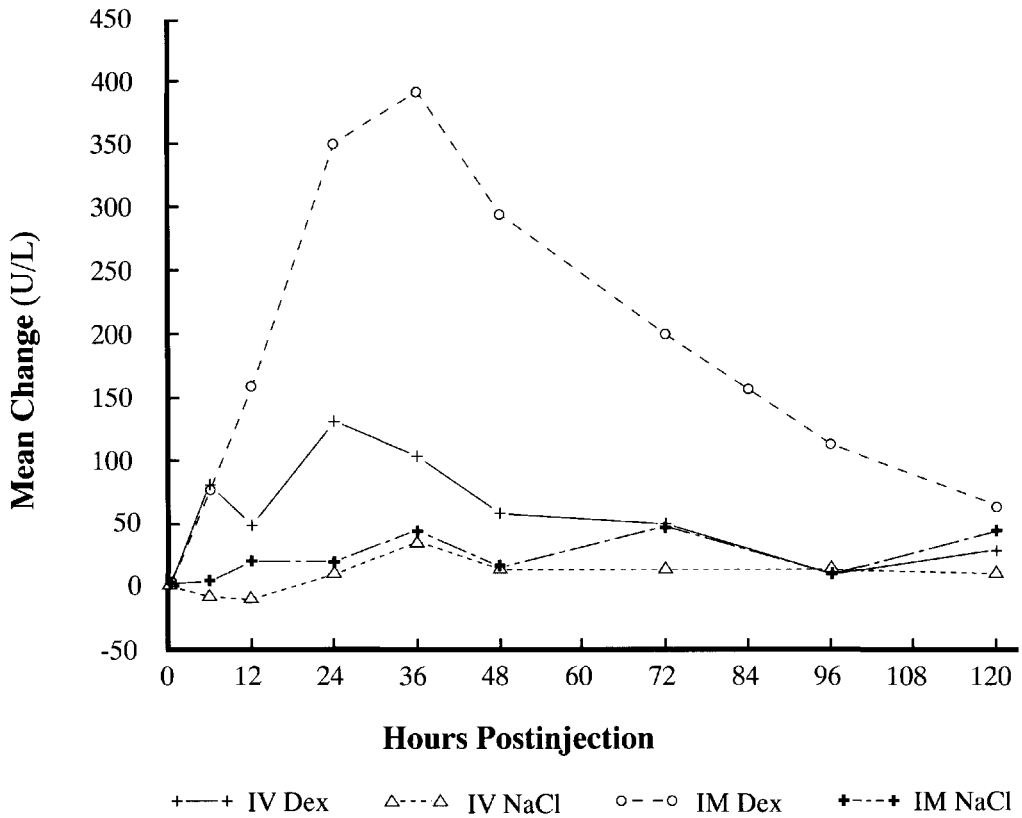

Figure 32.1. Mean changes in serum aspartate aminotransferase (AST) activity (units per liter) in red-tailed hawks following a single injection of dexamethasone or normal saline.

Blood sampling was performed on all birds from either the cutaneous ulnar or cranial tibial veins at a maximum volume of 0.5 ml per sample. Blood was collected prior to injection and at 6, 12, 24, 36, 48, 72, 96, and 120 hr postinjection. Centrifugation and separation of each blood sample was performed within 1 hour of collection. Serum samples were then frozen until the conclusion of the experiment. Samples were initially analyzed for AST, ALT, alkaline phosphatase, glucose, and total protein. Alkaline phosphatase and total protein were later dropped from the analysis due to lack of any observable response. Packed cell volume and refractometer measurements of total solids were used to monitor blood loss and hydration status.

Results

Serum values of measured parameters were normalized to reflect the mean positive or negative changes from the baseline preinjection value. Individual baseline values were within reference ranges (Campbell, 1986). One bird in Group 1 (IV DexNaP) died suddenly during the fifth blood collection due to a metabolic disorder unrelated to the study.

The most noteworthy changes were seen in values for serum AST, ALT, and glucose (see Figs. 32.1, 32.2, and 32.3). Alkaline phosphatase values did not change appreciably in the initial sample analysis and were therefore not pursued in the remaining samples. Packed cell volume and total protein values varied only slightly throughout the trial.

In Group 3 (IM DexNaP), serum AST values increased steadily to a peak at 36 hr, averaging 2.6 times baseline. Levels slowly declined, approaching baseline values at 120 hr postinjection. Again in Group 3, serum ALT values rose markedly to reach a peak at 24 hr, averaging 3.8 times baseline. They also slowly declined, but remained on average 1.3 times baseline levels even at 120 hr. The other three groups did not show appreciable changes in these enzymes.

Serum glucose became elevated in Groups 1 and 3, reaching a peak of 1.3 times baseline levels at 12 hr and rapidly dropping to baseline values at 36 hr.

Discussion

In this preliminary study, IM administration of corticosteroids appeared to induce an elevation of serum hepatic enzymes, AST and ALT, for up to five days. Elevations of both enzymes are of a magnitude similar to those seen in naturally occuring hepatic disease and to those reported when hepatic disease was induced in raptors (Campbell, 1986). However, IV administration of the same dose did not appear to induce similar enzyme elevations. The elevation in AST and ALT following IM dexa-

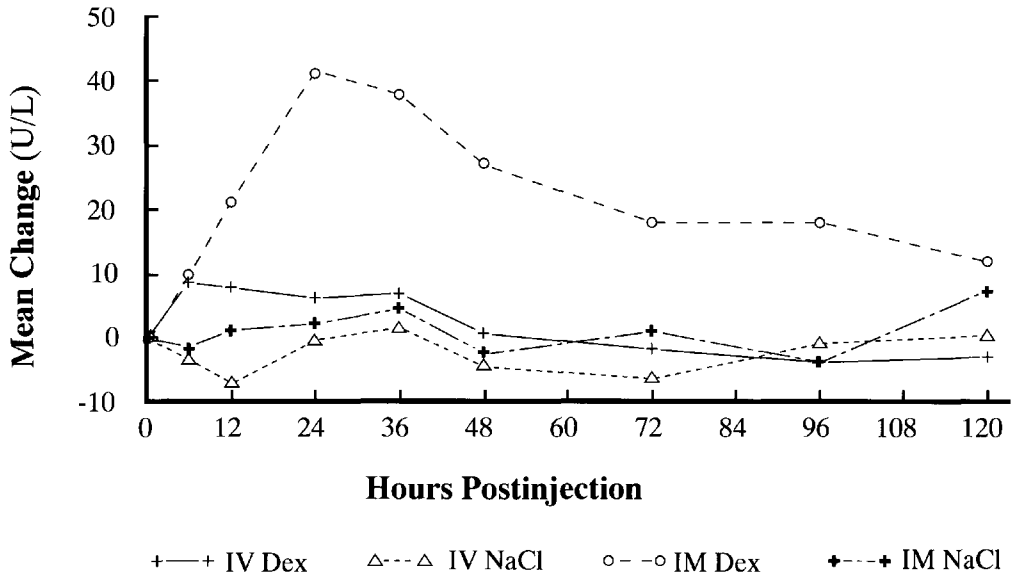

Figure 32.2. Mean changes in serum alanine aminotransferase (ALT) activity (units per liter) in red-tailed hawks following a single injection of dexamethasone or normal saline.

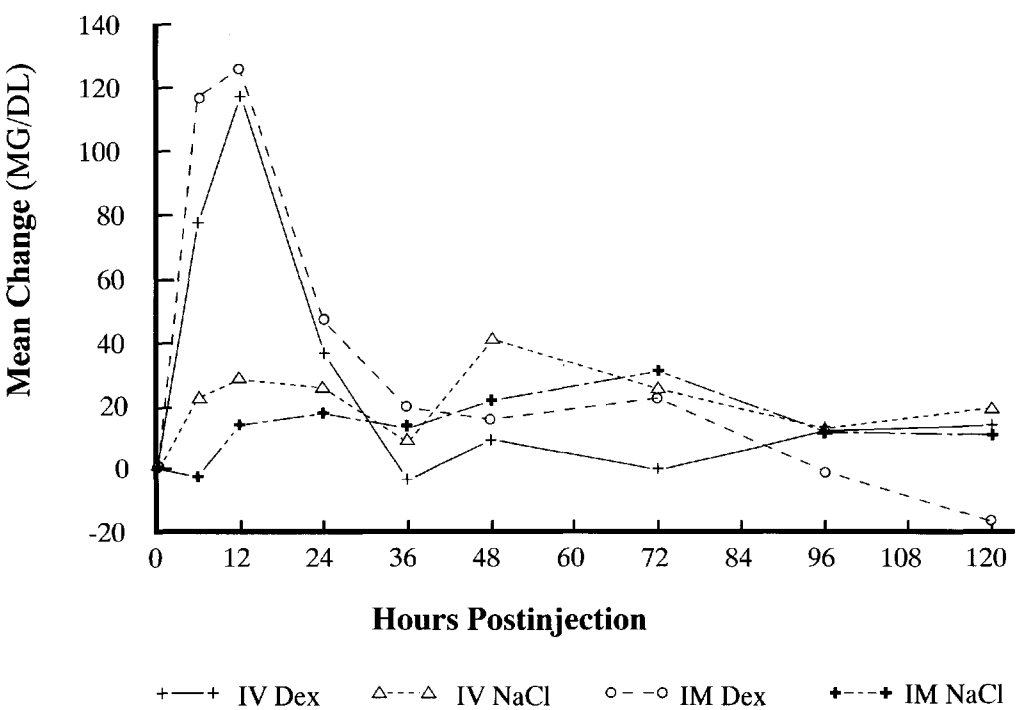

Figure 32.3. Mean changes in serum glucose concentration (milligrams per deciliter) in red-tailed hawks following a single injection of dexamethasone or normal saline.

methasone injection may be attributed to a steroid hepatopathy, or could be due to release of these same enzymes from muscle tissue at the injection site. AST and ALT have been shown to originate from both tissues (Lumeij, 1988). It has recently been shown that bile-acid analysis is a very specific measure of hepatocellular damage in birds, whereas creatinine phosphokinase is a very specific measure of muscle damage in birds (Lumeij, 1988). These two chemical tests could be used to distinguish hepatic from muscle changes observed in this type of study. Further investigation into the absorption rates of intravenous or intramuscular administration of dexamethasone may also provide some answers.

Serum glucose levels were rapidly elevated following both intravenous and intramuscular administration of dexametha-

sone. This observed response is presumably due to induction of gluconeogenesis. It is worthwhile to note the equally precipitous drop in glucose 36 hr following injection. This effect may exaggerate a state of hypoglycemia in an anorexic patient.

In evaluating the raw data from this study, it is important to note the variation recorded within each group. This may be explained by individual differences in the ability to handle the stress of the experimental protocol and subsequent variation in endogenous glucocorticoid release. Overall trends in the data were clearly not affected by the variation.

This pilot study helps to direct further investigation into the effects of dexamethasone administration on serum AST, ALT, and glucose in red-tailed hawks. The use of steroids in raptors as an initial treatment for shock is widespread. Accurate appreciation of the effects of this drug on liver enzymes and glucose levels is essential for evaluation and diagnosis of raptor patients.

Acknowledgments

We wish to thank Terry Schultz, the volunteers and employees of the Raptor Center, and the birds of the University of California, Davis, Raptor Center for their generous time and cooperation in completion of this study.

Product List

[a]Azium, Schering Corp., Animal Health Division, Kenilworth, New Jersey 07033 USA

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CHAPTER 33

A Research Hospital for Falcons: Design, Operation, and Admissions Summary

KENTON E. RIDDLE, B.S., D.V.M.
JOHN HOOLIHAN, B.S.
Abu Dhabi Falcon Research Hospital
Abu Dhabi, United Arab Emirates

ABSTRACT: A multipurpose hospital and research facility was designed and constructed to meet the unique requirements of a large population of captive falcons. The complex provides space for clinical examination, surgery, radiology, clinical pathology, and necropsy. Surgical and medical wards provide automated control of all environmental parameters. Facilities include quarantine, open and closed weathering areas, convalescent wards, sterile supply, pharmacy, diet kitchen, client waiting area, meeting rooms, administrative offices, and research library. On-site tack room and maintenance shop as well as personnel accommodations are also provided. The facilities are capable of accommodating up to 75 inpatient falcons, depending on medical status and grouping configurations. Outpatient visits can number 50 per day, and inpatient population averages 53 falcons.

Statistics were tabulated on 2,865 clinical cases (including 535 cases admitted to field hospitals during expeditions) for the period September 1985 to April 1988. Bumblefoot accounted for the largest number of medical admissions, 327 (11.4%). Following in frequency were parasites, 285 (10%), avian pox, 160 (5.6%), and aspergillosis, 138 (4.8%). Feather repair (imping), a nonmedical problem, accounted for 575 (20%) of the admissions. The remaining clinical conditions are tabulated with morbidity and mortality figures. There were 172 deaths or euthanasias recorded. Of those, aspergillosis accounted for 103 (59.8%) of the total. By contrast, bumblefoot accounted for 16 (9.3%) deaths.

Development of this facility has made it possible to provide comprehensive medical care and an educational effort for a large falconry community. Hospital admissions totaling more than 2,800 cases to date offer a significant opportunity for investigative study. In-depth collaborative research programs are concentrating on selected problems in avian surgery, medicine, and pharmacology.

KEY WORDS: falcon, hospital, raptor.

In the spring of 1985, the president of the United Arab Emirates and ruler of Abu Dhabi, His Highness Sheikh Zayed Bin Sultan Al Nahayan, commissioned the construction of a veterinary research hospital for hunting falcons. This farsighted project was the culmination of a major commitment to the management of a culturally valuable wildlife resource. Conceived by Sheikh Zayed before the 1976 International Falconry Conference held in Abu Dhabi, the multifaceted facility was designed to provide modern avian medical care and promote advancement of this discipline through veterinary medical research and client education. A major goal was to ensure the welfare of falconry traditions, important elements in the heritage and culture of the people of Abu Dhabi (Al Nahayan, 1976). Construction of the 733.61 m^2 facility (Simmonds, 1973) began in July 1985 and was completed in September 1987.

Figure 33.1. The hospital was constructed on a hilltop site in Abu Dhabi, United Arab Emirates.

Description of the Facility

The facility was constructed on a hilltop site of 0.8 ha located between Al Ain and Abu Dhabi (Fig. 33.1). The mean maximum temperature in July is 42.2°C, with a mean relative humidity of 71%. The mean minimum temperature in January is 11.6°C, with a mean relative humidity of 56%. Average annual rainfall is 51.3 mm.

The facility design provides three types of housing to meet the requirements of veterinary diagnosis and medical

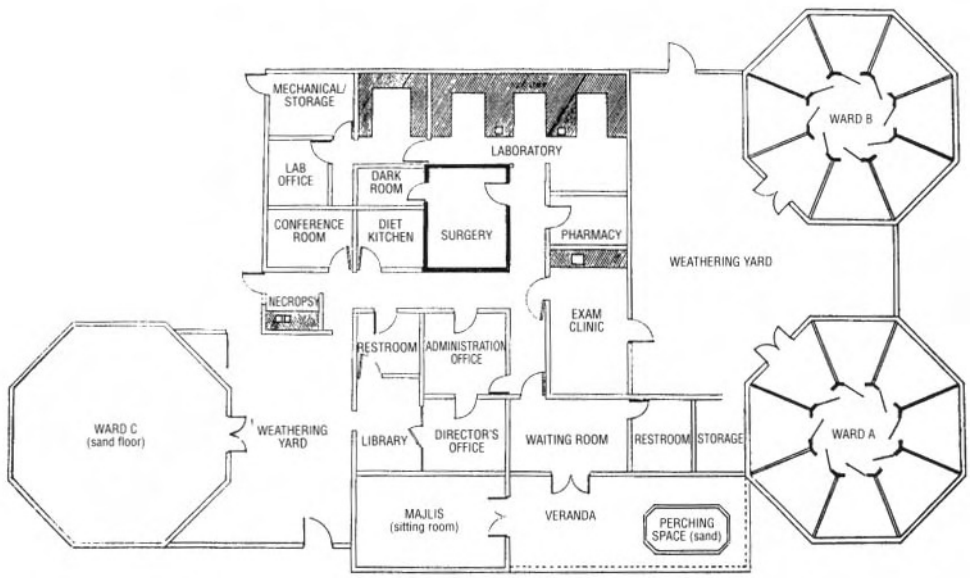

Figure 33.2. Indoor and outdoor perching areas are indicated on the floor plan for each functional area.

care: quarantine and isolation, longer-term housing, and research. (Copich, 1972; Enfield, 1972; Miner and Lundeen, 1972; Riddle et al., 1982). Design and management assured our objectives of providing modern Western-style veterinary care for predominantly wild-caught hunting falcons. Key psychological, environmental, and management requisites were met in the design of the facility to minimize stress to the falcons and to prevent cross-contamination and nosocomial infection. The facility is designed to accommodate up to 75 inpatient falcons, depending on medical status and grouping configurations.

Indoor and outdoor perching areas are provided in each functional area (Fig. 33.2). A marble rectangular sand trap for block perches is provided on the veranda to accommodate nonpatient falcons while clients are engaged with hospital business. Stainless steel disk-based perches topped with removable wood blocks covered with a synthetic turf are located in specific areas throughout the hospital. They are easily cleaned and sterilized, resist corrosion, and provide an ideal perching surface. Perch bases and surrounding floor areas are covered with artificial grass[a] carpets that are changed daily, or with sand, where that substrate exists. Receiving perches are located in the reception area, clinic, and weathering lawn. Outdoor weathering yards provide a protected perching area for sunning and a temporary holding area for large numbers of falcons during physical examination/vaccination clinics.

The facility is constructed of concrete masonry blocks with smooth mortared walls and a concrete roof. The ceiling in the service and administrative areas is made of suspended fiber tiles. Ceilings in bird rooms are epoxy-painted smooth concrete. The floors are acid-resistant ceramic tiles throughout the medical service areas. Ward surfaces are epoxy-painted smooth concrete. Hallway width (1.8 m) and door width (1.2 m) permit carriage of falcons without mishap to feathers or wings. The roof of the central hospital area is red-clay Spanish tiles, while the roofs of the wards are custom epoxy fiberglass panels shaped to resemble tents.

Electric supply to the hospital is 220 v 60 Hz with additional 110 v supply in clinic, surgery, and laboratory. Recessed fluorescent lighting in ceiling panels provides 100 footcandles of light at desk level throughout hospital service areas. Wards are provided with 200 footcandles of light at floor level. Two diesel-powered generators (50 kva) provide standby energy for power failures.

Water sources to the hospital are of two types. Irrigation water for landscaping is local well water. Hospital water is desalinated processed seawater. Emergency reserve for hospital supply is stored underground in a 45,600-l fiberglass tank. Laboratory reagent-grade water[b] is provided in the clinical pathology laboratory. Pretreatment, filtration, and softening of the hospital laboratory water supply are necessary.

All air-handling equipment is mounted in the ceiling crawl space. Chilled water from air-conditioning units near the personnel facility is piped to fan-coil units in the hospital. All main building air is recirculated, except the air in the surgical suite.

An underground septic tank located 0.5 km from the hospital receives nontoxic domestic waste water. Laboratory, surgery, and darkroom drains are collected in an acid-resistant biohazard tank that can be emptied by pump truck.

The core hospital provides administrative and support services, including a research library (Fig. 33.3). A *majlis* (sitting room) is located in the front of the hospital for special receptions and honored visitors (Fig. 33.4). The clinic

Figure 33.3. A research library is included in the area for administrative and support services.

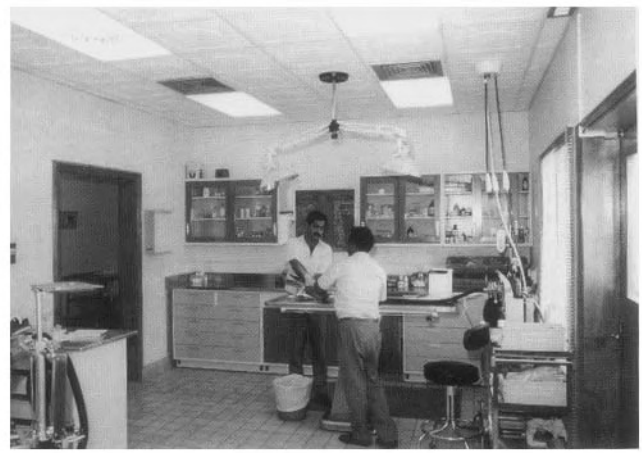

Figure 33.5. A falcon is examined in the examination and treatment room in the clinic.

Figure 33.4. Clients can wait on the veranda adjacent to the client reception area. The carving depicts falcons on the double doors to the *majlis*.

Figure 33.6. The clinical pathology laboratory has work bays for research.

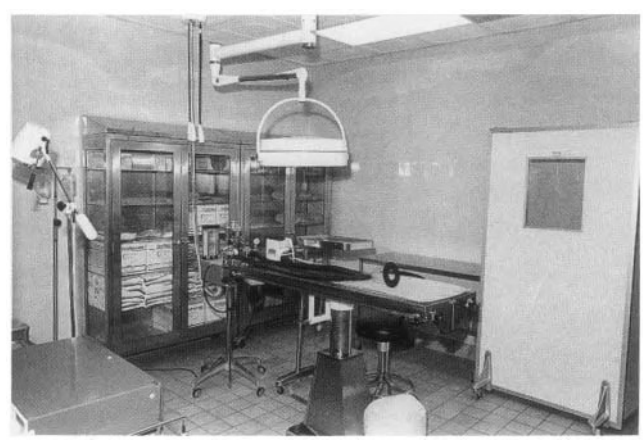

Figure 33.7. The surgery room provides equipment for electrosurgery, orthopedics, and endoscopy.

examination room also serves for intensive care and feather imping (Fig. 33.5). The clinic is equipped with an undercounter refrigerator, electronic balances for weighing falcons, custom-designed intensive care units,[c] and portable inhalation anesthetic machines supplied with piped-in oxygen, nitrous oxide, and sterile air.[d] A custom-designed workstation for feather imping is equipped with an anesthetic machine, examination light with magnifying lens, organizers, and drawers and numbered receptacles for an extensive feather bank.

A clinical pathology laboratory has work bays for parasitology, microbiology, hematology, serum chemistries, and histology, and provides automated diagnostic and research support (Fig. 33.6). Surgery/radiology is equipped with piped-in medical gases. Surgical equipment provides capabilities for electrosurgery, orthopedics, and endoscopy (Fig. 33.7). Radiology is provided by a 450-mA portable neonatal X-ray machine[e] and automatic film processor[f] (Fig. 33.8). A combined central sterile supply and diet kitchen is equipped with autoclave, microwave/convection oven with countertop stove, refrigerator, freezer, and food processors. A phar-

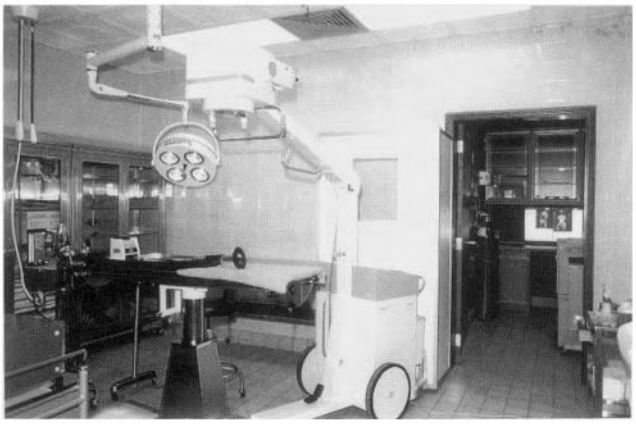

Figure 33.8. A portable X-ray machine adds radiology to the functions of the surgery room. A darkroom opens off the main area.

macy stores hospital items as well as replacement falconry equipment (Fig. 33.9).

A necropsy room is equipped with extra-wide doors (1.5 m) to facilitate entry of equipment, cages, and large animal species, if necessary. It is equipped with a necropsy table, weighing balance, freezer, glass-front storage shelves, and pathology display area.

Patient wards are located in octagonal-shaped buildings (Fig. 33.10). Eight wedge-shaped rooms with a central octagonal corridor provide seven wards and one holding room in the center (Fig. 33.11). Each ward has an adjacent treatment room with examination table and exam light, anesthetic machine, and supply cabinets. Ward doors are equipped with one-way viewing windows. Controls in individual rooms allow independent regulation of environmental parameters: temperature, humidity, and photoperiod, with rheostatic control of light intensity. Control panels are located to the right of each door. The air supply in each room is provided through individual ceiling-mounted fan-coil units and outside air-handling units. The supply is 100% nonrecirculated air at 15 to 18 changes per hour. Wards are balanced for negative pressure with respect to the common central corridor. High/low temperature alarms for wards are located in the hospital.

Falcons are maintained on the floor with or without the use of block perches. A 5-cm foam pad measuring 1.5 m by 1.5 m is covered by a full-sized artificial grass carpet and placed under the falcon. Carpets are changed daily, cleaned by high-pressure water hose, and disinfected on a specially designed sloping concrete pad (4.5 m by 7 m) with central drain. Orientation of the drying racks are north/south, which allows direct sanitizing sun rays on each side, morning and evening. Carpets are dry by the end of the workday and stacked ready for changing the next morning.

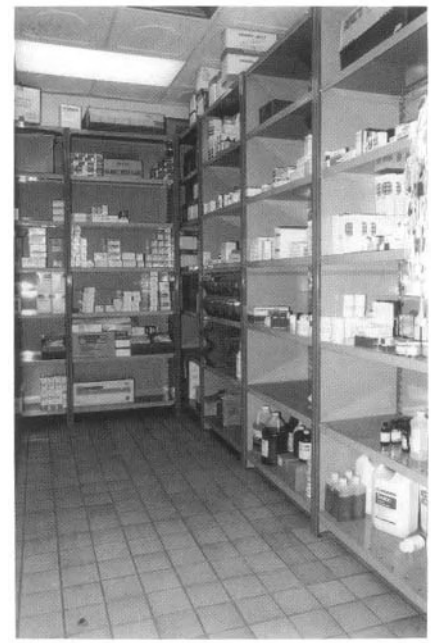

Figure 33.9. Hospital items and replacement falconry equipment are stored in the pharmacy.

Figure 33.10. A weathering lawn fronts the octagonal medical/surgical wards.

An open-air, octagonal, covered ward provides shaded perching space for convalescent or healthy falcons. Birds are maintained on stainless steel disk-bottomed perches with synthetic turf surfaces. Floors are of clean sand. Aluminum-barred windows are screened on the outside to prevent entry of insects. Electronic insect-killing lights are used in all outdoor perching areas.

Record of Care

The hospital provides care for the hunting falcons of the ruling family of Abu Dhabi and many local citizens of the region. Of 2,865 clinical cases seen from September 1985 to

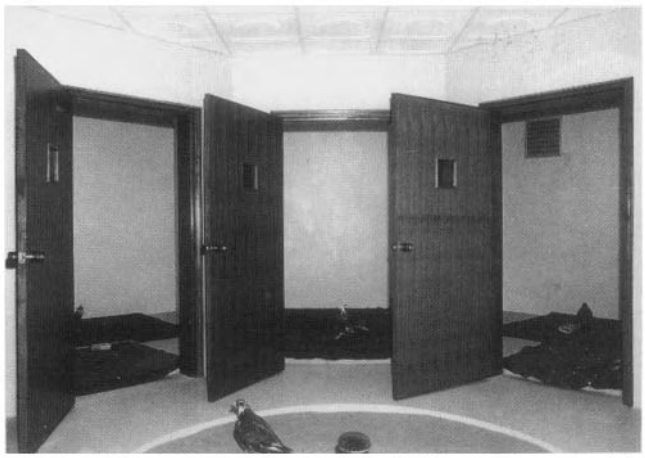

Figure 33.11. A central holding room links the bird rooms within the octagonal wards.

April 1988, 535 were admitted to field hospitals during hunting expeditions (see Table 33.1). Outpatient visits may number 50 per day, and inpatient populations range from 40 to 88 (average 52.9) during September to June. In the September 1985-April 1988 period, bumblefoot accounted for the largest number of medical admissions, 327 (11.4%); following in frequency were parasites, 285 (10%); avian pox, 160 (5.6%); and aspergillosis, 138 (4.8%). Feather repair (imping), a nonmedical problem, accounted for 575 (20%) of admissions. There were 172 deaths or euthanasias recorded. Of those, aspergillosis accounted for 103 (59.8%) of the total. By contrast, bumblefoot accounted for 16 (9.3%) deaths. Remaining hospital admissions are tabulated in Table 33.1.

Table 33.1
Falcon hospital admissions, September 1985-April 1988

Disease or condition	*Number of cases*	*Incidence (% of all admissions)*	*Number of deaths*	*% dying of specific condition*
Avian pox	160	5.6	4	2.5
Trichomoniasis	53	1.8	3	5.6
Meningitis	7	0.2	4	57.0
Aspergillosis	138	4.8	103	74.6
Tuberculosis	1	0.03	1	100.0
"Sour" crop	145	5.0	2	1.4
Sinusitis	20	0.7	1	5.0
Stomatitis	53	1.8	0	0.0
Eye infection	104	3.6	1	0.1
Coccidiosis	130	4.5	3	2.3
Internal parasite	128	4.4	0	0.0
Bumblefoot	327	11.4	16	4.8
External parasite	175	5.4	0	0.0
Lead poisoning	16	0.5	3	19.0
Bone fracture	18	0.6	3	17.0
Miscellaneous trauma	44	1.5	4	9.0
Cachexia	149	5.2	17	11.4
Wing sprain	33	1.1	0	0.0
Diarrhea	15	0.5	0	0.0
Restraint injury	19	0.6	0	0.0
Jess injury	18	0.6	0	0.0
Self-mutilation	27	0.9	0	0.0
Laceration	18	0.6	0	0.0
Abscess	21	0.7	0	0.0
Foot injury	58	2.0	0	0.0
Preventive medicine	268	9.3	0	0.0
Attach tail mount	42	1.4	0	0.0
Feather imp	575	20.1	0	0.0
Cope beak, talons	113	3.9	0	0.0
Anesthetic death	8	1.3	8	100.0
Total	2,865		173	6.0%[a]

[a]Percentage of deaths among all admissions.

Discussion

Actual aspergillosis cases were undoubtedly much higher than the above count shows; however, documented cases reflect clinical admissions of cachexia and dyspnea with incomplete necropsy results. A clinical diagnosis of aspergillosis often resulted in the owner releasing the bird to the wild rather than electing euthanasia and confirmation by necropsy.

Development of the Abu Dhabi Falcon Research Hospital has made it possible to provide comprehensive raptor medical care and educational benefits for a large Arab community. Hospital design and traffic flow provide separation of functional areas where desirable (i.e., laboratory, surgery, and radiology from clinics, wards, and necropsy), yet the centrally located clinic work center provides efficient access to service areas and wards. The combination surgery/radiology room has drawbacks, as one facility may interfere with the function of the other. Critical flexibility in housing is met by the application of easily moved, cleaned, and disinfected perching accessories and variable climate-control parameters within rooms and housing areas. On-site personnel accommodations are crucial to providing 24-hour coverage for admissions, medications, and security.

Hospital admissions total more than 2,800 cases thus far, offering a significant opportunity for investigative study. In-depth collaborative-research programs are concentrating on selected problems in avian medicine, surgery, pharmacology, and diagnostics.

Acknowledgments

We would like to recognize the excellent work of the Abu Dhabi Municipality, Agriculture Section, for architectural, engineering, and construction performance on this project. Special recognition is due M. R. Sorouri for his leadership and integrity throughout the period of construction.

Product List

[a]Astroturf, Balsam Corp., 809 Kenner St., Dalton, Georgia 30721 USA; or Balsam AG, Bisamweg 3, Postsach 1143, Steinhagen 4803, Germany

[b]Mili-RO and Mili Q, Millipore Co., 80 Ashby, Bedford, Massachusetts 01730 USA

[c]Snyder Intensive Care Cage, Snyder Mfg. Co., 500 E. Pacific Pl., Denver, Colorado 80222 USA

[d]Mediline, Ohmeda Co., Derbyshire, England

[e]Siemens Mobillette, Siemens Ela AB, Solna, Sweden

[f]X-Omat M-35 Processor, Eastman Kodak Co., 343 State St., Rochester, New York 14650-0001 USA

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CHAPTER 34

Physiological Monitoring of Raptors Using an Automated Biotelemetry System

RICHARD RUSHTON, D.V.M.
DAVID W. OSGOOD, Ph.D.

Sunriver Nature Center
Sunriver, Oregon, U.S.A.

ABSTRACT: An automated biotelemetry system was used to monitor core temperature, heart rate, and ECG waveforms in nonreleasable raptors. With the bird under anesthesia, a radio transmitter was implanted intraperitoneally. Telemetered signals were picked up by a receiver located under the bird's cage and carried via cables to a computer located in a separate room away from the bird. All data were collected and stored by an automated data-collection system controlled by microcomputer. Data were then collected continuously for 21 days without disturbing the bird. A distinct circadian rhythm was noted in temperature and heart rate, as well as elevations in these parameters when the birds were fed, handled, or otherwise disturbed.

KEY WORDS: biotelemetry, raptor, automated data-collection system, stress, circadian rhythm.

Monitoring of body temperature and heart rate in raptors by conventional methods requires handling and restraint of the birds by the investigator. Stress-induced changes in temperature and heart rate have been documented in studies of laboratory animals (Schwen and Jones, 1984; Peris and Cunningham, 1987). Raptors by their very nature are quite stressed by human presence and handling. In addition to the stresses of restraint, attempts to make continuous measurements significantly disturb the light-dark cycle and the resting state of the bird.

We were interested in employing a biotelemetry system to observe diurnal rhythms of body temperature and heart rate. Using the automated data-collection system described here,[a] we could measure responses to feeding, human disturbance, and the stress of handling.

Materials and Methods

Birds that were nonreleasable and unsuitable for educational purposes were used in this study. The initial feasibility study was done using a Swainson's hawk (*Buteo swainsoni*). A prairie falcon (*Falco mexicanus*) was then implanted and monitored for 3 wk. Limited temperature data were obtained from a great horned owl (*Bubo virginianus*). In this case the transmitter was placed in the crop and replaced whenever it was regurgitated. This method involved no surgery and was employed to test the feasibility of postsurgical monitoring by noninvasive means.

The birds were housed singly in a large fiberglass cage located in a closed room that was maintained with a 12L:12D photoperiod. The subjects were disturbed as little as possible. Feeding and cage cleaning were the only direct contacts by humans with the birds.

A good plane of surgical anesthesia was obtained using a dosage of 25 mg/kg of ketamine[b] with 2 mg/kg of xylazine.[c] (*Editor's note*: A dosage of 10 mg/kg ketamine with 2 mg/kg xylazine is more usually recommended.) The transmitter was implanted in the peritoneal cavity via a ventral abdominal midline incision, using sterile surgical technique. The leads for ECG sensing were directed subcutaneously onto the thoracic wall using a blunt probe and a slit plastic sleeve. Connective tissue secured the leads in place after 3 to 4 days. Lead placement corresponded to lead II on an ECG machine. Silastic tabs on the transmitter were sutured to the abdominal wall during closure to anchor it in place.

The implantable transmitter[d] weighed 9 gm and was 3.5 cc in volume. It was battery powered and contained a sensor and electronics to combine temperature, ECG, and heart rate into a multiplex signal in the AM band. Calibration of the transmitter required measuring the frequency output of the transmitter at five different temperatures in a water bath. Calibration values at two temperatures were entered into the computer and the Dataquest III software used these values to convert the telemetered frequency information to temperature data.

Receiver circuitry detected, amplified, and decoded the multiplex signal into two trains of logic-level pulses, one for temperature and another for heart rate. The receiver measured heart rate by detecting the interval between pulses triggered by R waves in the ECG waveform.

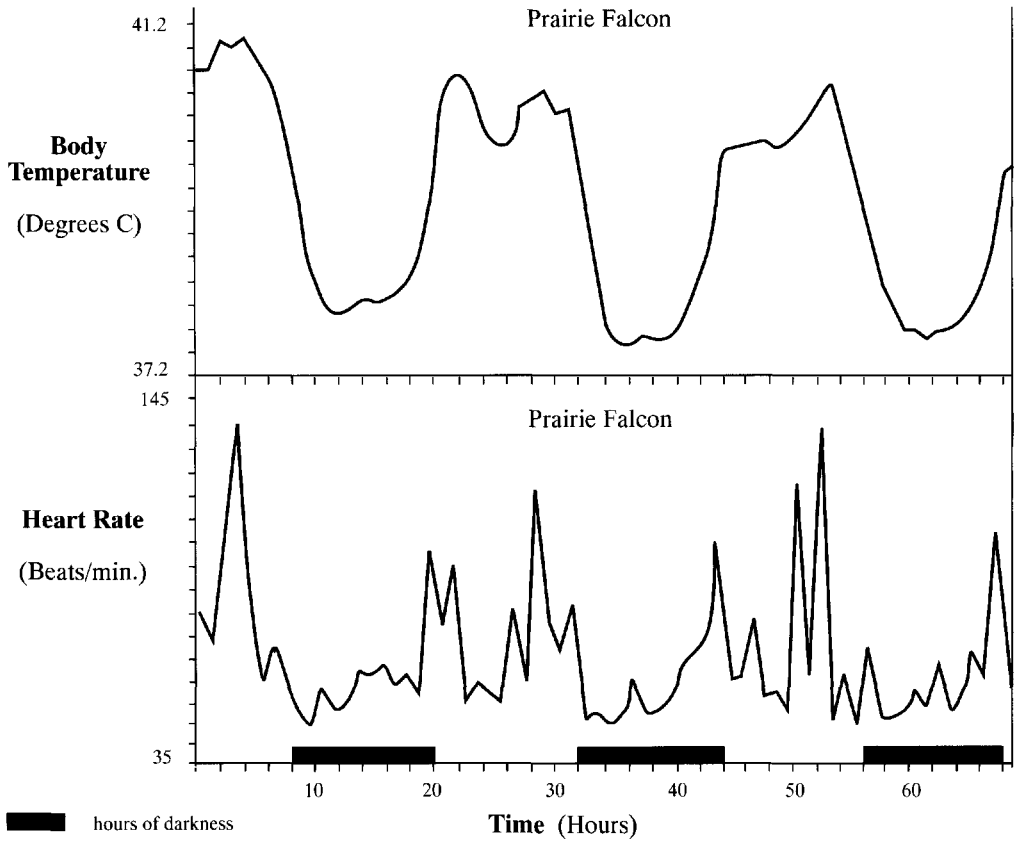

Figure 34.1. Diurnal rhythm of body temperature and heart rate.

The automated data-collection system allowed continuous monitoring of the instrumented bird's temperature, ECG waveform, and heart rate. A 5-min sampling interval was used for this study. Data were collected and stored for later analysis using programs within the system, while an "instant data" mode also allowed observation of current values.

Results

Body temperature and heart rate measurements of the prairie falcon were collected continuously at 5-min intervals and stored over a period of 3 wk. The owl's temperature was followed for several periods of 12 to 18 hr. Plots of the data were made for time periods that ranged from several hours to 10 days. Feeding and disturbance events were plotted over several hours, while diurnal rhythms were plotted over 3- to 10-day periods.

A distinct diurnal rhythm of body temperature and heart rate can be seen in the plotted data for the prairie falcon (Fig. 34.1). Fluctuations in body temperature and heart rate were very closely correlated. Over a period of 3 days, body temperature ranged from a low of 37.5°C to a high of 40.8°C with a mean of 39.0°C. Heart rate ranged from 45 to 136 bpm. The mean heart rate was 65 bpm. Peaks occurred at midday. Lows were reached about 8 hr later and began to rise again after 6 hr. The low resting heart rate of this bird (45 to 50 bpm) seems to indicate that the higher values usually recorded manually may be elevated by stress. The amplitude of temperature variation during a given 24-hr period was 2.8° to 3°C. A temperature plot over a 10-day period (Fig. 34.2) illustrates the continuance of the circadian rhythm over a longer time period. The more limited owl data showed similar daytime body temperature peaks and nighttime decrease.

During the study birds were fed dead ground squirrels. Feeding increased heart rate and body temperature (Fig. 34.3). Heart rate increased from 70 to 345 bpm and temperature increased from 39.9°C to 41.6°C during the feeding event illustrated in Fig. 34.3. The temperature increase began within 5 min and peaked about 40 min after the feeding. Similar results were seen at other feeding events.

The effect of handling on body temperature is shown in Fig. 34.4. During this trial the falcon was captured and restrained, and a thermometer was placed in the cloaca for conventional body temperature measurement. During this procedure, the bird was kept near the telemetry receiver. The bird's body temperature increased steadily over a 15-min period of human exposure and handling. The temperature increase continued after the bird was returned to the

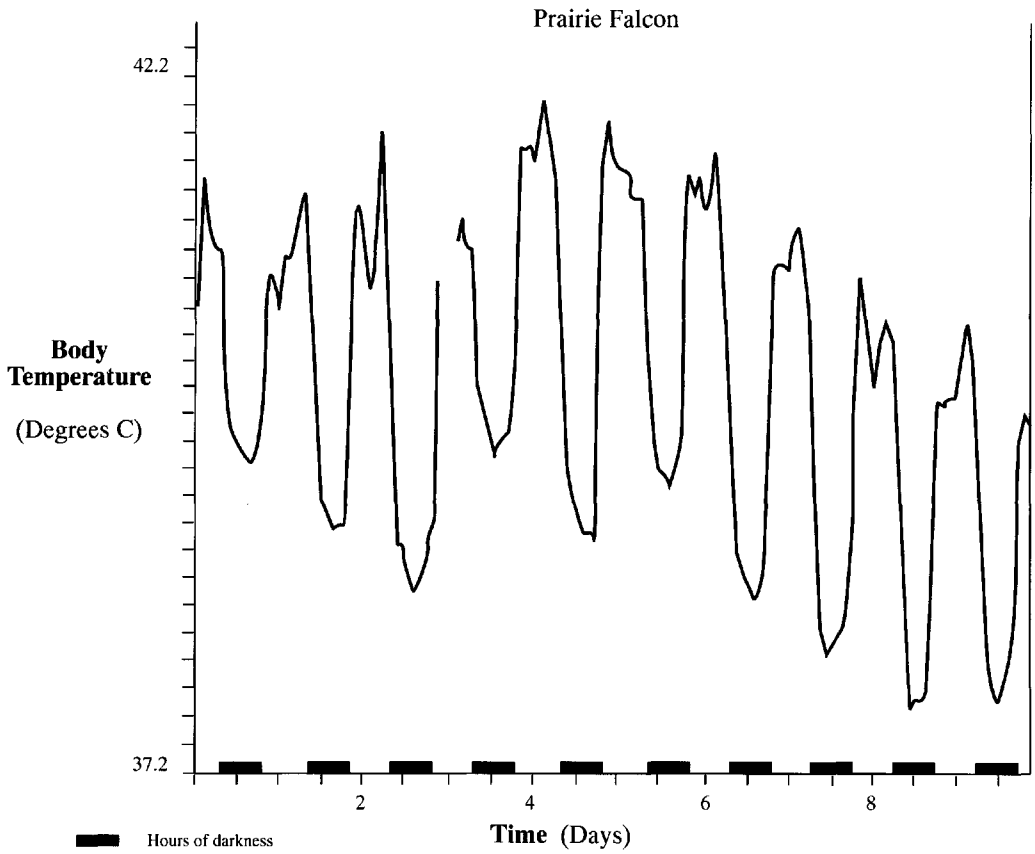

Figure 34.2. Body temperature over 10 days.

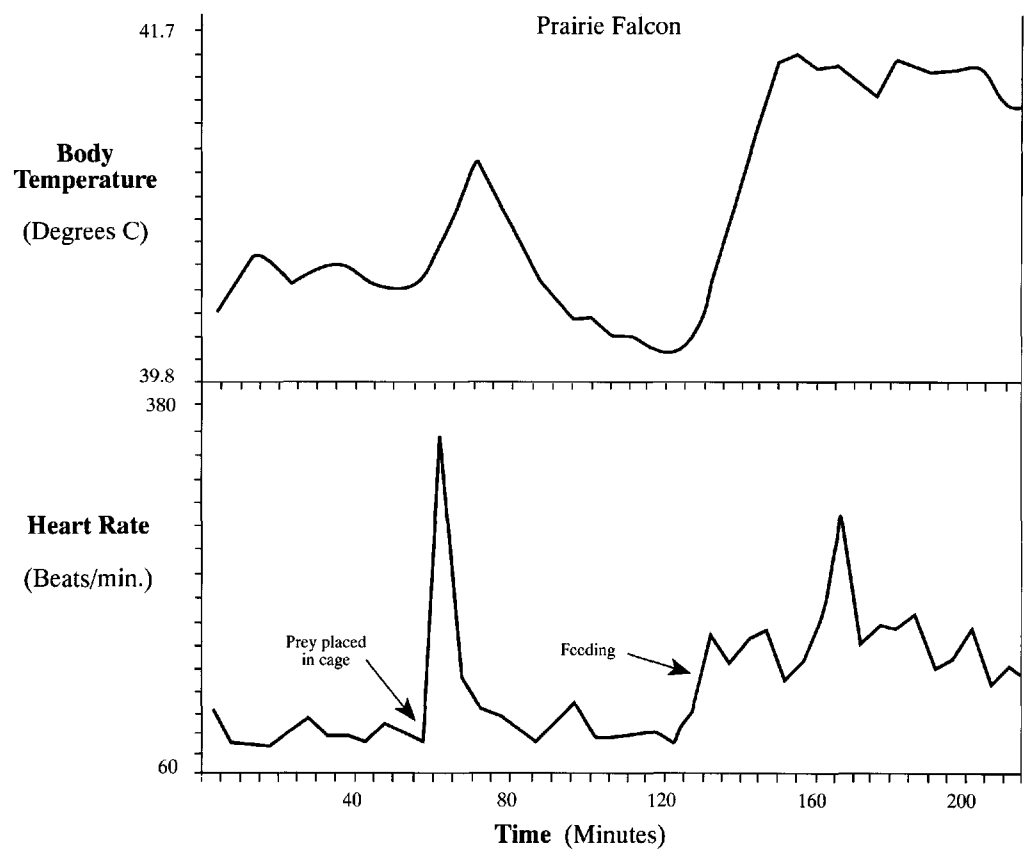

Figure 34.3. Body temperature and heart rate at feeding.

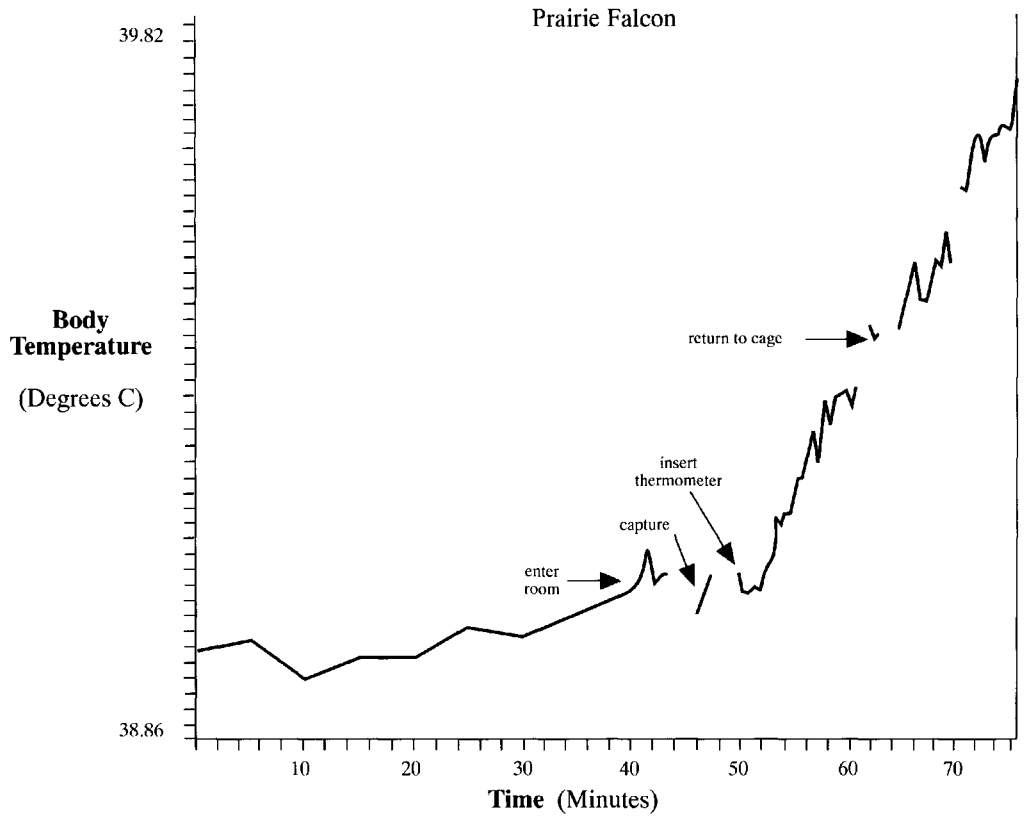

Figure 34.4. Effect of handling on body temperature.

cage, with a peak about 30 min after initial human exposure.

Discussion

We have described the use of biotelemetry in conjunction with an automated data-collection system to monitor body temperature and heart rate in raptors. This initial study indicates that biotelemetry would be an ideal method for monitoring physiological parameters in raptors.

In this study we demonstrated a distinct circadian rhythm of body temperature and heart rate by continuous, remote monitoring. Body temperature cycles of this magnitude have not been demonstrated in other raptor species except in red-tailed hawks (*Buteo jamaicensis*) that have been food deprived or exposed to cold (Chaplin et al., 1984).

We have also demonstrated the magnitude of stress-induced changes in body temperature and especially heart rate that occur when raptors are handled and restrained. The data obtained raise questions about the validity of what we consider "normal" values for raptor species.

Further studies of raptor physiology using biotelemetry are certainly suggested, including investigations of drug responses, pyrogenicity, postsurgical monitoring, and stimulus-response behavior. This knowledge would greatly enhance our understanding of the physiology of these magnificent birds.

Acknowledgments

We wish to thank the Oregon Department of Fish and Wildlife and Jay Bowerman of the Sunriver Nature Center for access to the birds used in this project.

Product List

[a]Dataquest III, Mini-Mitter Co. Inc., Sunriver, Oregon 97707 USA; and Data Sciences Inc., 2678 Patton Rd., Roseville, Minnesota 55113 USA

[b]Ketaset, Aveco Co. Inc., P.O. Box 717, Ft. Dodge, Iowa 50501 USA; or Aescoket, Aesculaap, Mijlstraat 35, 5280 AA, Boxtel, Netherlands

[c]Rompun, Haver-Mobay Corp., Animal Health Division, P.O. Box 390, Shawnee, Kansas 66201 USA; or Rompun, Bayer, Nijverheidsweg 26, 3640 AB Mijdrecht, Netherlands

[d]Model CTT85-SA, Data Sciences Inc., 2678 Patton Rd., Roseville, Minnesota 55113 USA

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Peris, J., and C. L. Cunningham. 1987. Stress enhances the development of tolerance to the hypothermic effects of ethanol. Alcohol and Drug Research 7:187-193.

Schwen, R. J., and L. C. Jones. 1984. Non-stressful measurement of morphine and caspaicinoid-induced hypothermia in the rat. Pharmacologist 26:140.

CHAPTER 35

Elbow Luxations in Raptors: A Review of Eight Cases

HOWARD D. MARTIN, D.V.M.
KENNETH A. BRUECKER, D.V.M., M.S.

Department of Clinical Sciences
College of Veterinary Medicine and Biomedical Sciences
Colorado State University
Fort Collins, Colorado, U.S.A.

DAVID D. HERRICK, D.V.M.

Cortez Animal Clinic
Cortez, Colorado, U.S.A.

JUDY SCHERPELZ, M.S.

Fort Collins Regional Raptor Program
Veterinary Teaching Hospital
Colorado State University
Fort Collins, Colorado, U.S.A.

ABSTRACT: Consequences of elbow luxations of eight raptors in six different species are presented. Initial examination often revealed a dropped wing with the elbow abducted and extended about 90 degrees, crepitus of the elbow joint, swelling, and associated wounds. Diagnoses were confirmed by radiographs. Of the eight cases, four (50%) were treated and released. Releasable birds had no fractures or penetrating joint injuries. Two cases (a red-tailed hawk [*Buteo jamaicensis*] and a bald eagle [*Haliaeetus leucocephalus*]) responded to manual reduction of the luxation and supportive bandages for one week. The other two cases (a great horned owl [*Bubo virginianus*] and a prairie falcon [*Falco mexicanus*]) were treated by reduction and stabilization of the elbow with an acrylic transarticular external fixation device for 7 and 12 days, respectively. Following removal of bandages or external fixators, physical therapy was provided for 16 to 36 days. Techniques included a progressive three-stage process: (1) passive range-of-motion, (2) active and active assisted range-of-motion, and (3) free flight for endurance.

Birds were judged to be releasable following assessment for (1) range of motion of elbows, shoulders, and carpi; (2) full extension of the wing; (3) palpation for crepitus; (4) total lift in flight before release; (5) follow-up radiographs; (6) follow-up blood work; (7) overall appetite and ability to apprehend prey; and (8) total body weight.

Elbow luxations are a relatively common injury to wild raptors. Those with no external wounds may carry a good prognosis for release, even with extensive soft-tissue injuries initially resulting in marked joint instability postreduction. In these cases, rigid stabilization with external fixators is recommended. Those with open joint wounds carry a poor prognosis.

KEY WORDS: elbow luxation, luxation, raptor, external fixator, physical therapy

Techniques have been described for treating orthopedic injuries in the avian species (Roush, 1980; Redig, 1982, 1986). Few, however, address joint injuries, and those are sketchy and discouraging regarding results. Consequently, luxations and other joint injuries in birds carry a poor prognosis in wildlife rehabilitation circles and often terminate in euthanasia. This results in fewer attempts at luxation repair, which perpetuates the myth of a grim prognosis. This report details elbow luxations in eight raptors of six different species. Cases range from chronic open luxations to acute closed luxations. Species included one great horned owl (*Bubo virginianus*), one golden eagle (*Aquila chrysaetos*), one bald eagle (*Halieaeetus leucocephalus*), two prairie falcons (*Falco mexicanus*), two sharp-shinned hawks (*Accipiter striatus*), and one red-tailed hawk (*Buteo jamaicensis*).

Materials and Methods

Injured birds of prey were presented to the Veterinary Teaching Hospital at Colorado State University and the Cortez Animal Clinic in Cortez, Colorado, for evaluation of inju-

Table 35.1
Summary of results of treatment for elbow luxations in raptors

Case	*Species*	*Sex*	*Body weight*	*Wing affected*	*Direction of luxation*	*Open or closed*	*Total WBC*	*Treatment*	*Time wing immobilized*	*PT[a] time*	*Final results*
1	great horned owl (*Bubo virginianus*)	?	1.25 kg	right	caudal	closed	—	external fixator chloramphenicol	7 days	16 days	released
2	prairie falcon (*Falco mexicanus*)	F	700 g	right + fx radius	caudal	open	14,000	external fixator + IM pin trimethoprim sulfa	14 days	17 days	arthritis; nonreleasable
3	prairie falcon	M	468 g	right	caudodorsal	closed	4,081	external fixator trimethoprim sulfa	12 days	31 days	released
4	golden eagle (*Aquila chrysaetos*)	F	5 kg	left + fx ulna	caudodorsal	open	69,000	curettage, flush trimethoprim sulfa amikacin	--	—	euthanized (day 58)
5	bald eagle (*Haliaeetus leucocephalus*)	M	3.5 kg	right	caudal	closed	24,400	bandaged	8 days	36 days	released
6	sharp-shinned hawk (*Accipiter striatus*)	F	185 g	left	caudodorsal	open	—	trimethoprim sulfa	—	—	died (3 hrs)
7	sharp-shinned hawk	M	160 g	left + fx ulna	caudodorsal	open	—	trimethoprim sulfa	—	—	euthanized (day 1)
8	red-tailed hawk (*Buteo jamaicensis*)	?	?	right	caudodorsal	closed	—	bandaged	15 days	15 days	released

[a]PT = length of physical therapy from time of removal of bandages or fixators.

ries from April 1986 through December 1987. Of 65 raptors examined, 8 were found to have elbow luxations. General information was obtained when possible as to circumstances under which the birds were found.

A general physical examination was performed on each bird. In five cases, blood was drawn for routine complete blood counts and clinical chemistry panels. Initial treatment consisted of wound dressing, administration of intravenous or oral fluids, immobilizing the wing by bandaging it to the body, and systemic antibiotics in five cases with open injuries. One bird died before any other treatment could be carried out. Radiographs were taken within 48 hr of presentation, usually under heavy sedation with ketamine hydrochloride[a] (20 to 30 mg/kg IM). Reduction of luxations was attempted within 72 hr. In two cases, luxations were reduced manually (closed technique) under gas anesthesia and the elbow was immobilized with a bandage. In two other cases, luxations were reduced manually (closed technique) under gas anesthesia, and external fixation devices were applied. One case was reduced by open surgical technique, which included repair of a proximal radial fracture as well as application of an external fixation device. One case resulted in the bird being euthanized following the initial surgical approach and reassessment of the extent of soft-tissue damage. Recovery from surgery and anesthesia was in quiet, darkened cages. Convalescing birds were provided a diet of rats and mice.

Birds were examined daily. Elbows were immobilized for 7 to 15 days. Physical therapy was begun within 3 to 5 days following removal of immobilization devices (see Martin, Ringdahl, and Scherpelz, chapter 36, this volume). This consisted of passive range of motion (to increase range of motion and soft-tissue flexibility), progressing to active and active assisted range-of-motion exercises and finally free flight. Birds were assessed for (1) range of motion of elbows and shoulders, (2) full wing extension, (3) total wing lift in flight, (4) overall appetite, (5) ability to apprehend prey, and (6) total body weight. Follow-up radiographs were taken in all cases, and elbows palpated for crepitus upon flexion and extension.

Results

In most cases, the causes of initial injuries were unknown. Likely possibilities included being hit by a car, hitting

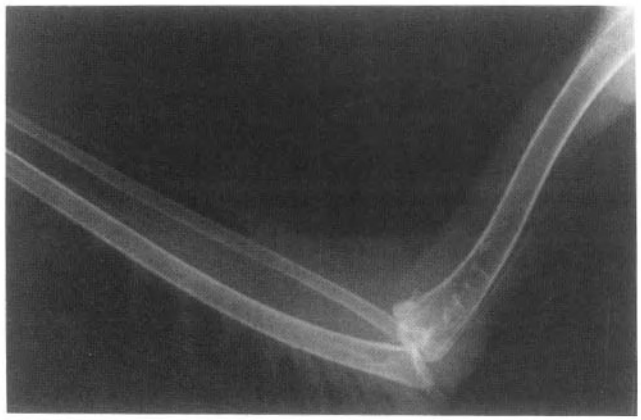

Figure 35.1. Radiograph of a great horned owl (GHO), VD view, demonstrating a luxated elbow.

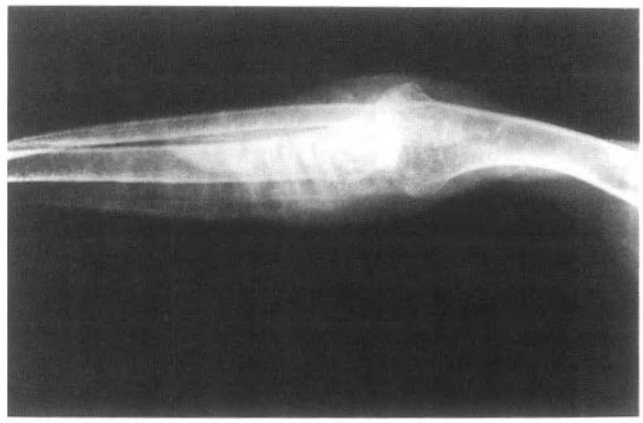

Figure 35.2. GHO radiograph, AP view, demonstrating that luxation is in a caudal direction.

power lines, gunshot wound, cat attack, or flying into windows. Results in the eight cases described below are summarized in Table 35.1.

Case 1

A mature great horned owl was presented with a drooping wing following an encounter with a domestic cat. Physical examination revealed swelling over the medial aspect of the right elbow. The owl was depressed but in good physical condition. Initial treatment included chloramphenicol sodium succinate[b] (50 mg/kg IM BID), bandaging the affected wing to the body, and oral fluids. The following day the owl was anesthetized using isoflurane[c] via a nonrebreathing circuit, radiographs were taken, and the luxation reduced (Figs. 35.1 and 35.2). An external fixation device was applied (Figs. 35.3 and 35.4) that consisted of three Kirschner (K) wires (diameter 0.062 in) in the distal humerus and three similar wires in the proximal ulna with one K-wire (diameter 0.035 in) in the proximal radius. A Type I external fixator was formed by bending the wires at right angles and encasing the long axis in methyl methacrylate to form a crossbar over the dorsal aspect of the wing.

The external fixation device was removed 7 days later under anesthesia. At that time the elbow was stable, with a range of motion approximately 80% of normal. Passive range-of-motion exercises were initiated and continued twice a day until active and active assisted range-of-motion exercises were begun 8 days later. Overall flight and range of motion were constantly assessed and judged to be felt at least 95% of normal when the bird was released. The owl was released following 16 days of physical therapy. The bird flew well at that time.

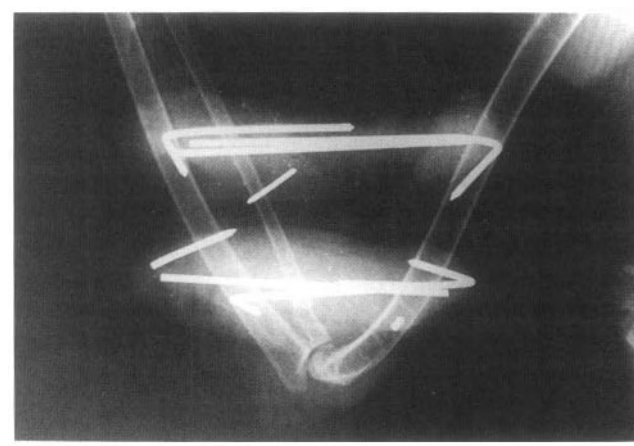

Figure 35.3. GHO radiograph, VD view, with the external fixator applied. Connecting bar is of methyl methacrylate.

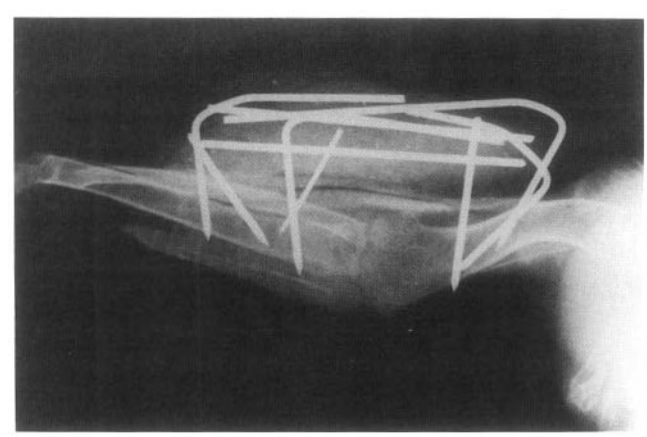

Figure 35.4. GHO radiograph, AP view, with the external fixator in position.

Case 2

A female prairie falcon was presented with open wounds of the right elbow joint, a fracture of the proximal radius, and a caudal luxation of the ulnar humeral joint (Fig. 35.5). No other abnormalities were detected by physical examination, radiographs, CBC, or plasma chemistry profiles. The falcon was

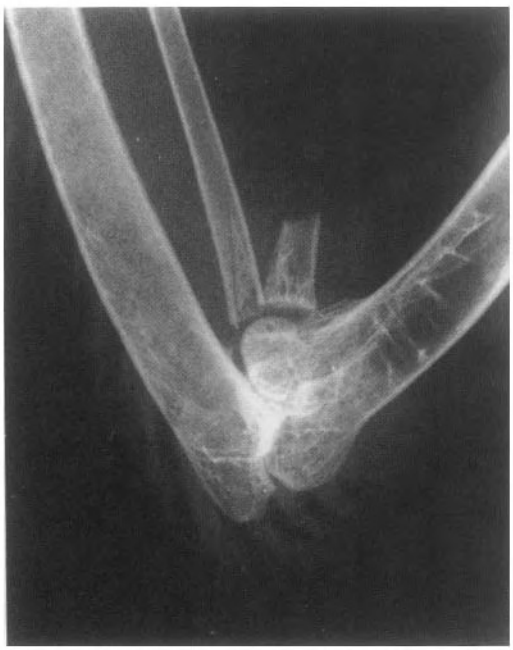

Figure 35.5. Radiograph of a female prairie falcon (PRF), VD view, demonstrating loss of joint space (caudal luxation of ulna) and fracture of the radius.

started on trimethoprim sulfa[d] (50 mg/kg orally BID), the wounds were cleaned, and the wing was immobilized by bandaging it to the body. Two surgeries (3 days apart) were required before the luxation and fracture could be stabilized. A short intramedullary pin and cerclage wire were used to repair the fractured radius, and an external fixator was applied to stabilize the elbow. The external fixator consisted of one K-wire (diameter 0.062 in) placed in the humerus and one K-wire (diameter 0.045 in) in the ulna, connected dorsally and ventrally with a methyl methacrylate crossbar. The fixation device was left in place for 14 days (Fig. 35.6).

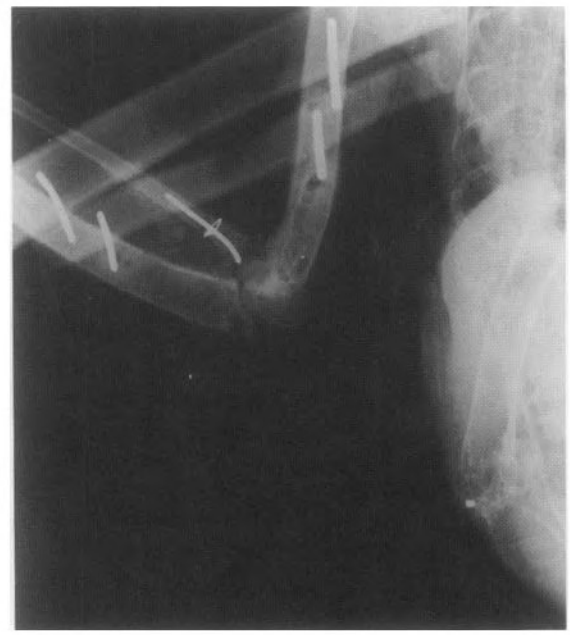

Figure 35.6. Female PRF radiograph, VD view, 2 wk postoperative. An external fixator with four pins connected by methyl methacrylate bars is seen. An IM pin in conjunction with cerclage wire (to stabilize a fissure fracture) stabilizes the radial fracture.

Physical therapy was not aggressive in this case, beginning 40 days after removal of the external fixation device. At that time the wing drooped from the shoulder and range of motion was restricted to 90 degrees. Full extension or flexion of the elbow was not possible. After little progress and radiographic evidence of degenerative joint disease (Fig. 35.7), physical therapy was discontinued and the bird retained for teaching purposes as a permanent cripple.

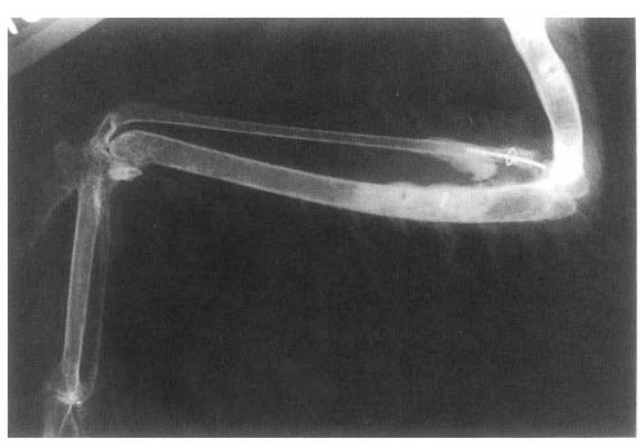

Figure 35.7. Female PRF radiograph, VD view, demonstrating degenerative joint disease with sclerosis of the distal humerus and proximal ulna. There is also bridging between the radius and ulna.

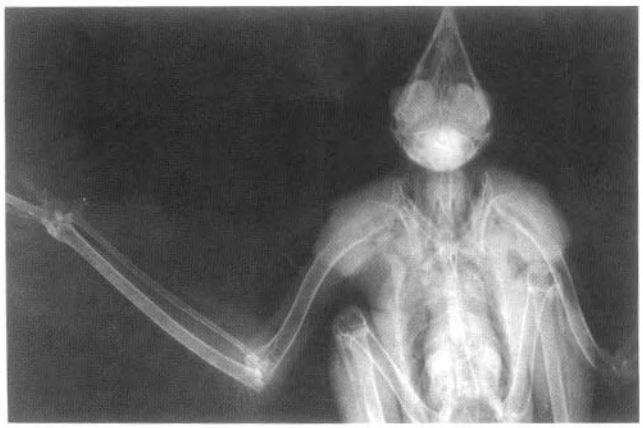

Figure 35.8. Radiograph of a male PRF, VD view, demonstrating the elbow luxation.

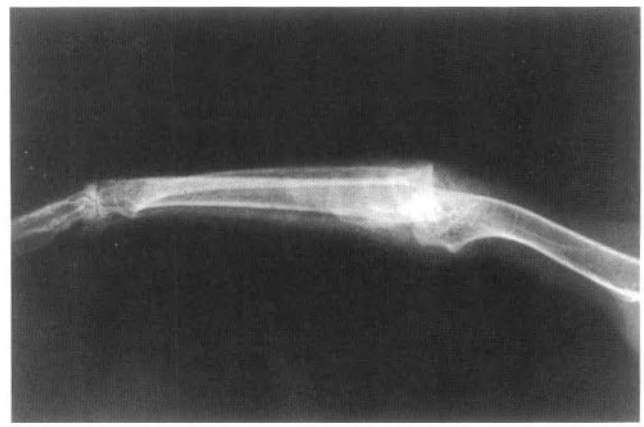

Figure 35.9. Male PRF radiograph, AP view, demonstrating dorsocaudal direction of the luxation.

Case 3

A male prairie falcon was examined and found to have an abrasion and laceration on the dorsal surface of the right elbow as well as a caudodorsal luxation of the elbow joint. It was anesthetized the following day with isoflurane on a nonrebreathing circuit. Radiographs were taken (Figs. 35.8 and 35.9) and the luxation reduced. One K-pin (diameter 0.062 in) was placed in the distal humerus and one in the proximal ulna and connected by methyl methacrylate crossbars to form a Type II external fixator (Figs. 35.10 and 35.11). After 12 days, the falcon was anesthetized again, the external fixator was removed, and radiographs were taken (Fig. 35.12 and 35.13). The elbow was palpated and determined to be stable with approximately 120 degrees range of motion (75-80% of normal). The wing was bandaged to the body for two additional days. Immediately following bandage removal, passive range-of-motion exercises were begun. Active and active assisted range-of-motion exercises were started 5 days after removal of the external fixator and free flight was encouraged 1 wk later. The bird was reassessed 31 days following removal of the external fixator. Range of motion at that time was 95% of normal (approximately 140-145 degrees of extension). There was no evidence of joint swelling or crepitus. The falcon was released and flew extremely well.

Case 4

A female golden eagle was presented two weeks after it had been found alongside a road. Physical examination revealed severe atrophy of the left pectoral muscle, an open caudodorsal luxation of the left elbow, and a healed fracture of the proximal ulna. The elbow was swollen, hot, and painful. A CBC revealed a leukocytosis (69,000 WBC/mm^3), and a swab taken for culture and sensitivity from the elbow yielded a heavy growth of *E. coli*. Initial treatment included

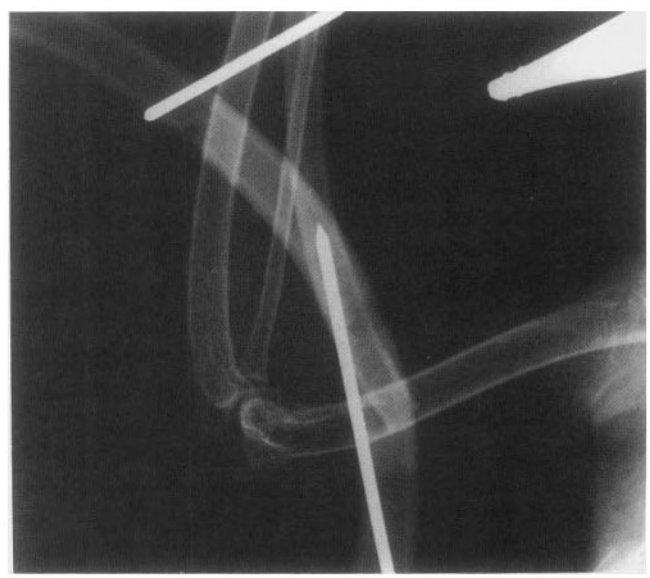

Figure 35.10. Male PRF radiograph, VD view, external fixator in place.

injectable trimethoprim sulfa (30 mg/kg IM BID) in the pectoral muscles and daily flushing of the wound, including two surgical attempts to debride and flush the elbow. Following results of sensitivity testing, the antibiotic therapy was changed to amikacin[e] (20 mg/kg IM BID). In spite of vigorous therapy resulting in slight improvement, evidenced by decreased swelling and pain, the bird was euthanized due to persistence of an *E. coli* osteomyelitis.

Case 5

An adult male bald eagle was found beneath heavy power lines and presented with caudal luxation of the elbow. Physical examination revealed no other problems. A CBC revealed a leukocytosis (24,400 WBC/mm^3) and all plasma chemistry parameters were normal. Approximately 48 hr later the eagle was anesthetized with isoflurane on a non-

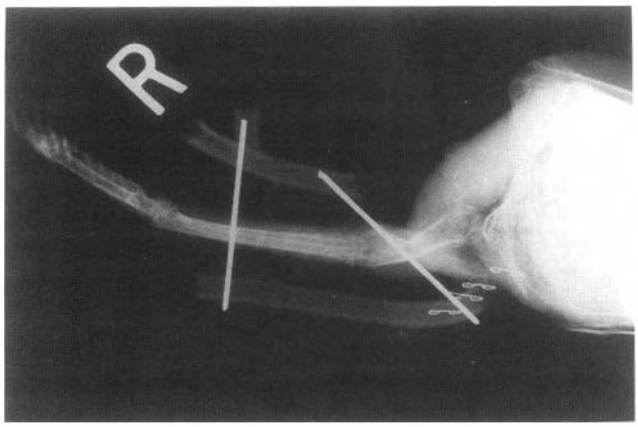

Figure 35.11. Male PRF radiograph, AP view, external fixator in place.

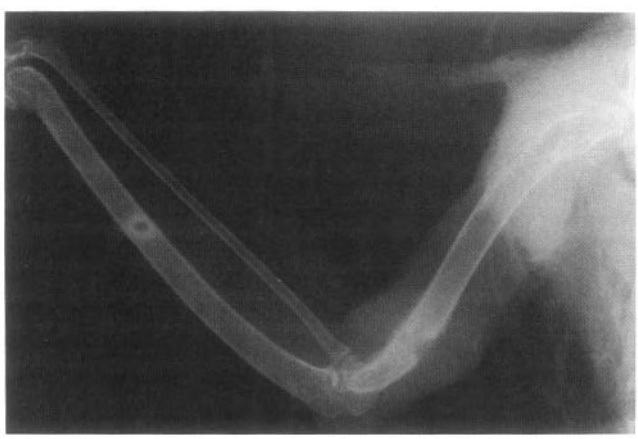

Figure 35.12. Male PRF radiograph, VD view, following removal of external fixator.

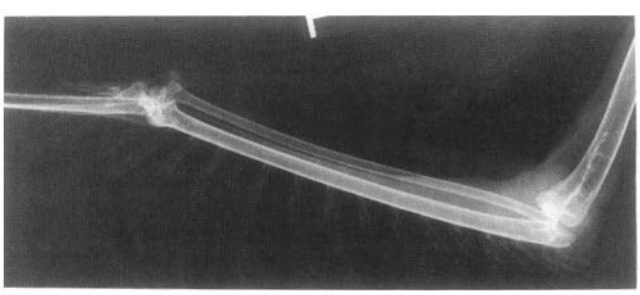

Figure 35.13. Male PRF radiograph, AP view, following removal of external fixator.

rebreathing circuit. Radiographs confirmed the luxation (Fig. 35.14), and it was manually reduced. Reduction was accomplished by flexion of the elbow and stabilization with a heavy cotton bandage. The elbow was bandaged in approximately 90 degrees of flexion. The bandage was removed 8 days later under anesthesia and the wing radiographed and palpated (Fig. 35.15). Range of motion at this time was 90% of normal and there was no evidence of crepitation or instability. Three days later, passive range-of-

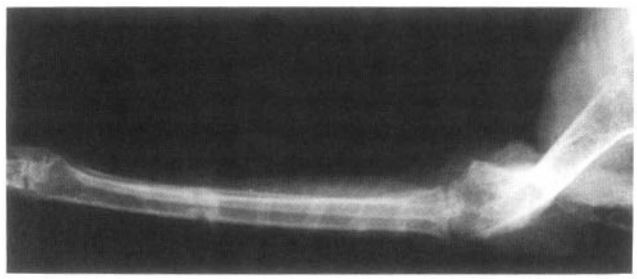

Figure 35.14. Radiograph of a bald eagle (BE), VD view of luxated elbow. Note the soft-tissue swelling.

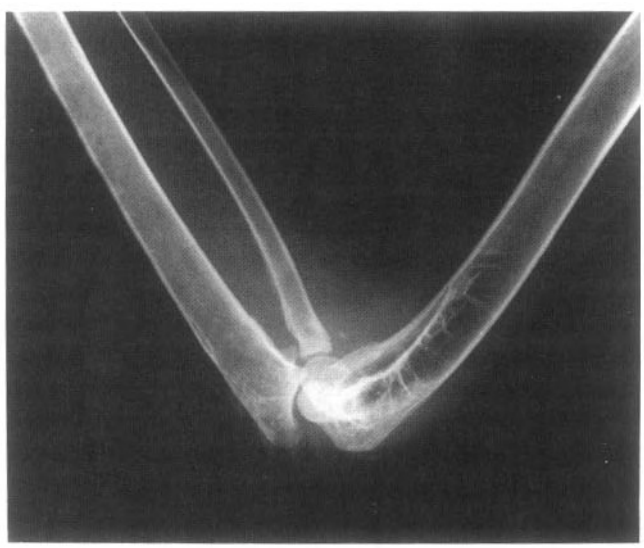

Figure 35.15. BE radiograph, VD view, following removal of bandage.

motion manipulations were begun twice a day for 5 to 10 minutes and progressively increased to 10 to 15 minutes twice a day. After 5 days the bird was transferred to a flight facility. The bald eagle was released 36 days following removal of the bandage, at which time he demonstrated 95% normal range of motion with no evidence of crepitation, swelling, or pain. Upon release, the bird sustained flight for more than three miles and appeared strong and normal.

Case 6

Physical examination of a female sharp-shinned hawk revealed her to be alert and responsive, with wounds on the left pectoral area and the left lateral elbow region. The left elbow was unstable, crepitus was evident, and a caudodorsal elbow luxation was diagnosed. The wounds were flushed and the wing was bandaged to the body. The bird was given 20 mg trimethoprim sulfa IM. Oral alimentation consisted of .5 cc warm water with strained-meat baby food by gavage tube. The hawk became progressively more depressed over the next few hours and died approximately 3 hr following

presentation. Postmortem revealed severe contusions and pulmonary hemorrhage.

Case 7

A male sharp-shinned hawk was presented to the Cortez Animal Clinic. Upon physical examination the bird was found to be bright, alert, responsive, and in good flesh, with a caudodorsal luxation of the elbow. Dense metal fragments seen on radiographs suggested a gunshot wound. The bird was transferred to Colorado State University. Treatment was begun and included trimethoprim sulfa (50 mg/kg IM BID). At the time of surgery it was determined that the extent of the soft-tissue damage over the distal two-thirds of the radius and ulna warranted a grave prognosis for return to functional flight, and the bird was euthanized.

Case 8

An adult red-tailed hawk had been found alongside power lines and was presented to the Cortez Animal Clinic. The bird was found to be in good condition and excellent health, except for a caudodorsal elbow luxation. Anesthesia was accomplished in a chamber with methoxyflurane and the luxation reduced by flexing the elbow and compressing the joint lateromedially. Reduction was evaluated radiographically and the elbow felt fairly stable. Both wings were taped to the body using stretch tape.[f] The bird recovered well and began eating on its own. After 15 days with its wings immobilized with tape, the bird was placed in a small cage to prohibit attempts at flight, although it could fully stretch its wings. Five days later the bird was placed in a flight cage and demonstrated flight capability, and 10 days following flight exercise the bird was released with full range of motion and normal even flight.

Discussion

Elbow luxations are a relatively common injury to raptors (approximately 12% of cases in this study). This injury may be caused in a number of ways, but severe blunt trauma to the wing is necessary to damage the ligamentous structures without causing a fracture. The anatomy of the elbow and the relationship between the dorsal and ventral condyles of the distal humerus limit the direction of radial head luxation to a dorsal or straight caudal position. If the radius is fractured, ventral luxation is possible (Baumel, 1979). As expected, the presence of open wounds involving the joint correlates with a poor prognosis because of the increased incidence of both septic arthritis and osteomyelitis. Both birds that responded to stabilization with bandages probably had relatively little ligamentous damage compared with those whose unstable elbows required rigid fixation. The decision to use a bandage technique or rigid external fixation was based upon the degree of stability in postreduction palpation.

Clinical signs of elbow luxations in raptors are easily recognized and include a drooped wing with elbow held in an open abducted position and extended about 90 degrees. Palpation usually reveals crepitation, swelling, and pain. Care should be taken in conducting a physical examination to part the feathers in order to examine the skin closely for wounds that may communicate with underlying bones or joints. The head of the radius is easily palpated over the dorsal or lateral epicondyle of the humerus. Anesthesia is usually required for good positioning when taking radiographs. Both lateral and AP views should be taken. Positioning is easily accomplished for the lateral view. Proper positioning for the AP view requires directing the beam at the anterior or posterior surface of the humerus with the wing held in extension.

Reduction of elbow luxations is accomplished under deep anesthesia. The elbow is manipulated with one hand on the proximal radius and ulna and the other on the distal humerus as the elbow is flexed. This manipulation counters the forces of the scapulotriceps muscle, which tends to pull the ulna caudally. The radius and ulna are rotated internally with pressure on the proximal radius to bring the radial head into alignment with the dorsal condyle (laterally). If the radius and ulna and their transverse ligament are intact, the ulna will "pop" into place as the elbow is extended. This is often subtle and suggests that most of the ligamentous attachments of the joint are still intact. With severe ligamentous damage, the "pop" may not be evident, the elbow remains unstable, and rigid external fixation is recommended.

Ideally, it would be constructive to follow "successful" cases over a long period of time to assess secondary joint changes. Currently no such data exist. In the dog, osteoarthritis of the elbow is relatively rare following a reduction of elbow luxations and is most often associated with cases involving fractures of the joint surface or luxations of long-standing duration requiring open reduction procedures (Campbell, 1971; Brinker et al., 1983). To date, little has been published regarding joint injuries in avian species, and so there is an inadequate comparison base. This report illustrates that joint involvement does not necessarily warrant a poor prognosis. Many elbow luxations, particularly closed ones, may not only be repairable but may heal well enough to enable full flight function, with minimal loss of range of motion.

Product List

[a]Ketaset, Aveco Co. Inc., P.O. Box 717, Ft. Dodge, Iowa 50501 USA; or Aescoket, Aesculaap, Mijlstraat 35, 5280 AA, Boxtel, Netherlands

[b]Chloromycetin Palmitate, Aveco Co. Inc., P.O. Box 717, Ft. Dodge, Iowa 50501 USA

[c]AErrane, Anaquest, 2005 W. Beltline Hwy., Madison, Wisconsin 53713-2318 USA

[d]Bactrim, Rugby Laboratories, Inc., Rockville Center, Long Island, New York 11570 USA

[e]Amiglide V, Aveco Co. Inc., P.O. Box 717, Ft. Dodge, Iowa 50501 USA

[f]Elasticon, Johnson & Johnson Medical Equipment, Arlington, Texas 76004-0130 USA

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CHAPTER 36

Physical Therapy for Specific Injuries in Raptors

HOWARD D. MARTIN, D.V.M.

Department of Clinical Sciences
College of Veterinary Medicine and Biomedical Sciences
Colorado State University
Fort Collins, Colorado, U.S.A.

CHRISTINE RINGDAHL, R.P.T.
JUDY SCHERPELZ, M.S.

Fort Collins Regional Raptor Program
Veterinary Teaching Hospital
Colorado State University
Fort Collins, Colorado, U.S.A.

ABSTRACT: Rehabilitation of an injured raptor may be a long process of medical stabilization, prevention of tissue deformity, and promotion of normal function. The physical therapy program is directed by the medical diagnosis and becomes an integral component of recovery. The vast majority of raptor injuries involve the limbs, with fractures of the humerus, radius, and ulna most frequent. Anatomical repercussions, especially concomitant joint injury, are reviewed for these and other common injuries. Exercise techniques for passive range-of-motion, active assisted range-of-motion, progressive strengthening, and muscular and cardiovascular endurance training are described. Qualitative and quantitative guidelines are provided for modulation of exercise intensity, frequency, and duration in order to ensure a safe yet appropriately aggressive rehabilitation program. Signs of patients' intolerance of exercise are reviewed, and sensitivity to stress is discussed.

KEY WORDS: raptor, physical therapy, exercise.

The treatment of a raptor is a process of medical stabilization, restoration, prevention of tissue deformity, and promotion of normal physical function. Rehabilitation includes assessment and stabilization, medical treatment and physical therapy, and, finally, evaluation for release. Physical therapy conditions a bird for return to the wild after successful medical treatment. The evaluation of fitness for release is the final task of the veterinarian. Considerable research has been conducted on early assessment and medical stabilization (Pokras, 1986; Redig, 1984; Sparrow and Turner, 1982; and Taylor, 1983). Evaluation of recovery for release is outlined by Martell and Redig (1985), but the progressive physical therapy program has not been adequately described. Horowitz et al. (1983) describe basic methods of physical therapy and exercise in raptor rehabilitations. Development of a progressive physical therapy regimen should ensure the least stress and the most rapid and complete recovery (Patton and Crawford, 1985). This chapter identifies specific therapy goals and describes progression of physical treatments for specific injuries of birds of prey.

Diagnosis-Related Rehabilitation

Etiologies of injuries for raptors are many, but the resultant nature of injury can be categorized based on three types of tissue damage: (1) musculoskeletal, (2) neuromuscular, and (3) cardiovascular. The vast majority of injuries occur in the first category, including soft-tissue damage and fractures to the humerus, radius, and ulna.

Initial treatment includes thorough examination followed by medical stabilization, wound debridement, possible surgery with immobilization, adjunctive drug therapy, and monitoring of signs of intolerance to captivity. Recovery depends upon normal use of injured tissues, thus physical therapy is often necessary. Physical therapy goals must be identified based on specific anatomical injuries, and these determine the therapy techniques to be used. The therapist must also be aware of species characteristics, individual needs, and the bird's physical and behavioral responses to each therapy session in order to direct the progression of the therapy program.

Exercise Techniques

The condition and rate of recovery of the bird will direct the specific exercise techniques required for rehabilitation. In general, passive range-of-motion (PROM) exercise is per-

Table 36.1
Physical therapy program according to types of injuries

Type of injury	*Therapy goal*	*Therapy*
Joints and connective tissues (ligaments, tendons, joint capsule, cartilage)	minimize loss of range of motion and prevent changes in soft-tissue flexibility	passive range-of-motion active assisted range-of-motion
Muscular system	prevent changes in soft-tissue flexibility, improve muscular strength, enhance muscular and cardiovascular endurance	active assisted range-of-motion active range-of-motion
Cardiovascular system	enhance muscular and cardiovascular endurance	active range-of-motion activities of daily living
Neurological system	neuromuscular reeducation of balance, coordination for normal active daily living	active range-of-motion activities of daily living

formed by the therapist by firmly grasping the limbs on either side of the joint and gently moving the joint through its range of motion to provide a slow stretch, to gain motion of the joint, and to increase soft-tissue flexibility. Care is taken to palpate tissue tautness and stretchability. When inflammation or fibrosis of the joint capsule exists, joint capsule mobilization can be increased as the therapist passively moves the joint. The therapist should note the "end feel" of the joint and whether the motion is open, spongy, or solidly blocked at the end of travel. This technique is used during the more acute phase, when tissue fragility is high and the range of motion of joints and soft-tissue flexibility must be maintained or increased.

Active assisted range-of-motion (AAROM) exercise is used when mobility of the tissue is reduced. It allows the beginning of muscular strengthening to occur early in the rehabilitation program. AAROM exercise involves cooperative maneuvers between therapist and bird. The therapist assists the bird's wingspread by smoothly raising and lowering the bird while holding the legs. Speed of movement and trajectory of the movement of the bird can be modulated to increase the difficulty of the exercise. Muscle strengthening, soft-tissue flexibility, range of motion of joints, and circulation are facilitated. The therapist's attention is directed toward the quality of symmetrical movement, the extent of guarding, and the bird's overall response to stress.

Active range-of-motion (AROM) exercise is selected and progressively worked to develop muscular strength, full range of motion of joints, and endurance during mid to late stages of recovery. The exercises can be systematically graded to build cardiovascular and muscular endurance as well as coordination. AROM exercises (such as graded flight) are performed by the bird. They are structured and evaluated by the therapist, who assesses the bird's performance and quality of integrated movement. Signs of weakness, range-of-motion deficits, and cardiovascular intolerance are noted. As the bird accomplishes increasingly higher levels of active exercises, supervised hunting and killing can be practiced as the final exercise mode requested by the clinician.

Table 36.1 describes the physical therapy goals for general categories of tissue injury. In all this work, signs of exercise intolerance such as severe fatigue, incoordination of movement, open-mouth breathing, dyspnea, vocalization, drooping wings, and perching incapacities must be recognized. A certain degree of stress behavior in response to simple handling and pain is normal. Recognition of the unique responses of each bird allows identification of excessive signs of exercise intolerance. Behavioral and physical acceptance of physical therapy is crucial before progression can be attempted without increasing the bird's risk of reinjury or excessive stress.

Modulation of the therapeutic exercise can be achieved gradually by varying the parameters that describe exercise frequency, duration, and intensity. *Frequency* is how often the raptor undergoes exercise. One to three times per day is optimal for short bouts of moderate exercise. Exercise *duration* describes the length of the exercise session. A session of 5-20 min is recommended, and depends largely on the species, acceptance of handling, and exercise tolerance. *Intensity* is the rigor or amount of work required. Intensity is graded by increasing the amount and speed of the voluntary effort required of the bird. Adding more repetitions tends to build strength and endurance. Guidelines for exercise progression include the following:

1. Vary one parameter at a time (frequency, duration, or intensity).
2. Keep frequency to one to two times daily in early rehabilitation. It may be increased to three times daily, and then reduced to once daily or every other day for high-intensity, longer-duration exercise sessions such as flight.
3. Standardize frequency, depending on the individual bird, and then increase intensity as tolerated by the bird. Duration of the exercise should then be increased at maximum intensity to 20 min. Return to a shorter duration when increasing the exercise intensity to the next level.

Table 36.2
Common injuries, medical treatments and their consequences in raptors

Injury	*Primary anatomical consequences of injury*	*Stabilization techniques*	*Secondary consequences of stabilization*
Luxations Wing (shoulder, elbow, carpal joints) (elbow most commonly seen) Leg (coxofemoral, stifle, and tarsal joints)	associated fractures common with luxations of the carpal and tarsal joints, avulsion of the ventral tubercle of the humerus with shoulder luxations direct injuries to tendons, nerves, and muscles avulsion of nerves decreased range of motion in affected joint potential of septic arthritis and degenerative joint disease decreased strength and flexibility in injured muscles	open or closed reduction and immobilization of the affected joint for 5-14 days by bandaging external fixators transarticular pins	decreased range of motion of all joints immobilized potential degenerative joint disease decreased strength and flexibility of immobilized muscles decreased cardiovascular and muscular endurance
Fractures Wing (carpometacarpus, radius, ulna, humerus, coracoid) Leg (tarsometatarsus and phalanges, tibiotarsus, femur)	direct injuries to tendons, nerves, and muscles injuries to nearby joints resulting in decreased range of motion potential for osteomyelitis decreased strength and flexibility in injured muscles damage to trachea and large blood vessels with coracoid fractures	open or closed reduction and immobilization of fractures by bandaging to immobilize joints above and below the fracture splints (Schroeder-Thomas splints recommended for fractures of the tarsometatarsus, spica-type splints for tibiotarsus and femur) external fixators (allow for free motion of joints) IM pins ± wires, external fixators bone plates cage rest may be used to manage fractures of the radius and minor metacarpal bones when ulna and major metacarpal remain intact	decreased range of motion of all immobilized joints decreased strength and flexibility of immobilized muscles decreased cardiovascular and muscular endurance

4. The interplay of repetition and intensity yields the best response when the bird is worked up to 20-25 repetitions. When introducing greater intensity, drop repetitions to 5-10 times and work the patient up to 20-25 repetitions until the next higher work load is again introduced.

5. Do not increase the intensity of the exercise if the patient cannot repeatedly perform the exercise exactly.

6. Reassess exercise intolerance and quality of movement at all times. Do not assume that the bird will tolerate the same exercise it performed well the previous day.

7. Observe the whole bird, not just the injured area. Heart rate, respiratory rate, and overall intolerance of exercise are easily monitored. In general, if the bird can perform a task 15 to 20 times with good quality of movement for two to three days and a frequency of two to three times daily, then a new demand can be made. When ready,

the bird should be kept where the environment facilitates independent active movement and natural skills. These exercises, such as perching, eating, short flights, hopping, and walking, are termed *activities of daily living* (ADL).

Discussion

Paramount to rehabilitation is strong sensitivity of the clinician to the bird's behavioral and physical responses to captivity, handling, and therapy. This sensitivity depends on an understanding of avian anatomy and kinesiology. A systematic and goal-oriented program of therapeutic techniques is highly recommended.

The primary goal of physical therapy is identified based on the tissues injured, the extent of the damage, the type and duration of immobilization, and the nature of the individual bird. The primary anatomical consequences of the injuries are important in planning the therapeutic regimen as well as the physical therapy program. However, of equal importance are those effects of the repair and mobilization techniques on joints, muscles, and tendons. These secondary consequences must be addressed in the physical therapy regimen (Table 36.2). Once these factors are evaluated, the appropriate physical therapy program may be detailed.

Consideration of the physical therapy required should begin at the time of initial examination. Attention to these details will minimize time required for rehabilitation and enhance return of normal function. The most common consequence of orthopedic repairs is loss of range of motion of joints. This may be minimized through the use of external fixators and initiation of physical therapy as soon as possible.

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CHAPTER 37

An Introduction to Allometric Scaling and Its Uses in Raptor Medicine

MARK A. POKRAS, B.S., D.V.M.
ALICIA M. KARAS, B.A., M.S., D.V.M.
Tufts University School of Veterinary Medicine
North Grafton, Massachusetts, U.S.A.

JAMES K. KIRKWOOD, B.V.S.C., Ph.D., M.R.C.V.S.
Institute of Zoology
Zoological Society of London
London, England

CHARLES J. SEDGWICK, B.S., D.V.M.
Tufts University School of Veterinary Medicine
North Grafton, Massachusetts, U.S.A.

ABSTRACT: Allometric scaling provides a method for examining the structural and functional consequences of changes in size or scale among otherwise similar organisms (Schmidt-Nielsen, 1984). It has been found that many physiological and life-history variables can be related to body mass by simple mathematical relationships. For example, when the resting metabolic rates of members of a taxonomic group of animals are plotted against body mass on a log-log plot, the points fall close to a straight line. This relationship has been expressed for nonpasserine birds (including raptors) as $Y = 78\ (M)^{0.72}$ (Lasiewski and Dawson, 1967), where Y is the animal's resting energy output in kilocalories per 24 hours, and M is the bird's mass in kilograms. The exponent 0.72 is not significantly different for 0.75 and thus variable Y is said to "scale" with mass to the 3/4 power (Schmidt-Nielsen, 1984).

The finding that energy requirements increase with the 3/4 power of the mass of the animal has become well established in the practice of comparative animal nutrition (Robbins, 1983). Size exerts its influence upon many other areas of importance to veterinary medicine (such as pharmacokinetic half-lives, heart rate, and clutch size), but this has yet to gain common acceptance in veterinary medicine. The purpose of this review is to outline methods by which certain biomedical values may be estimated from an animal's body weight using an allometric approach. Areas of specific interest to raptor biology are emphasized, including scaling of resting metabolism to body size, scaling of the capacity of transport organs (such as cardiac stroke volume), scaling of cyclic frequencies (heart rate, respiratory frequency, and so on), the concept of metabolic time, and the applications of scaling to such topics as nutrition, anesthesia, and clinical pharmacology.

KEY WORDS: raptor, allometric scaling, metabolic relationship, chemotherapeutics, anesthesia.

Modern veterinary practice had its roots in the care of domestic animals. With this limited number of species, it was possible to consider the physiology of each species independently. For this reason, the influence of size on biology was not appreciated, nor was a knowledge of it deemed necessary. Today the profession is becoming involved with a much wider range of species (there are 20,000 or so terrestrial vertebrates, spanning a size range from 2 g to 5,000 kg), and medicine is becoming very complex. An understanding of the influence of body size on biology and the rates of physiological processes is essential, because this millionfold difference in weight causes variations in physiology that obscure the basic patterns of similarity. Even among raptors, there is more than a 100-fold variation in size.

The finding that energy requirements increase with approximately the 3/4 power of body weight has become well established as a key principle in the science and practice of animal nutrition (Robbins, 1983), but similar weight relationships have yet to gain common acceptance in practice in other branches of veterinary science. As biological parameters have become established for animals of a range of body sizes, the allometric relationships of many of these parameters have been examined (Calder, 1984). The nature of these relationships offers clues as to how animals "work." They can also be of immediate practical value in predicting response to captive management.

In this chapter, the methodology of size scaling and its value in various fields of veterinary science and practice are

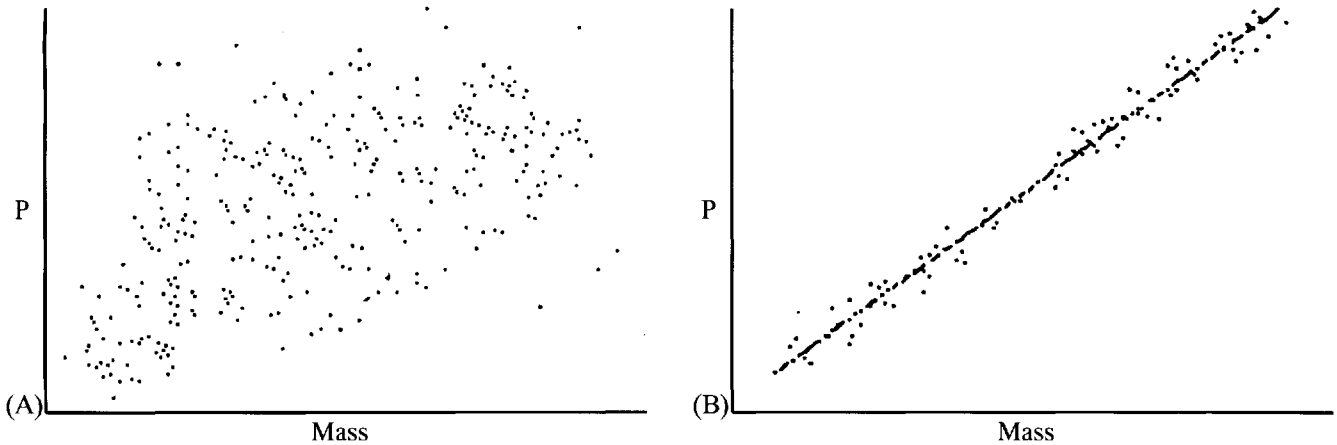

Figure 37.1. In allometry, many parameters (P), such as metabolic rate, life span, and kidney size, are found to vary between species with body mass in a curvilinear way (A). When logarithmically transformed, the points fall close to a straight line (B).

described. It must be borne in mind, first, that allometric relationships are not biological laws, but simply useful characterizations of data; and, second, that this field is in its infancy. As more data are gathered, new relationships will be found, and some of our existing theories will prove to be incorrect.

The Allometric Equation

Derivation

The allometric equation is a formula that can be used to summarize the relationship between the magnitude of any biological parameter (such as heart rate) and body weight. If the mean magnitude of the parameter P is plotted against mean body mass M for a variety of species, the points are often observed to cluster about a curved line (Fig. 37.1A). If these data are logarithmically transformed and then plotted, the points fall close to an approximately straight line (Fig. 37.1B). This line can be expressed in the traditional way ($y = bx + a$) by fitting a least squares linear regression equation:

$$\log P = \log a + b \log M$$

In the equation, b is the slope and log a is the y intercept. This then can be rearranged to the allometric equation:

$$P = aM^b$$

Although this equation was originally devised to describe the variation of P with M during the growth of an individual (Huxley, 1972), in this chapter we are concerned with its use in describing the variation of P among adults of different species. The following list summarizes several of the important mathematical techniques required to manipulate allometric equations (let A be equal to any number):

1. $A^m \times A^n = A^{m+n}$
2. $A^m + A^n = A^{m-n}$
3. $(A^m)^n = A^{mn}$
4. $A^o = 1$
5. $1/A^n = A^{-n}$
6. $A^{1/n} = \sqrt[n]{A}$
7. $A^{m/n} = \sqrt[n]{A^m}$

Examples and Interpretation

Some examples of allometric equations describing a selection of biological parameters are shown above. It is useful to have an insight into the significance of the values of b in the allometric equations. Fig. 37.2 includes two diagrams that show the pattern of the relationship of a parameter P to body weight M for various values of b on a generalized log-log scale.

Linstedt and Calder (1981) describe the various ways in which physiologic parameters are found to scale. Generally, the capacity of transport organs, such as blood volume, lung volumes, and cardiac stroke volume, tend to scale linearly with M ($b = 1$), so that their volume remains approximately in constant proportion to M as body size increases (Table 37.1).

Physiological volume rates such as oxygen consumption (ml O_2/min), glomerular filtration rate (GFR) (ml/min), and respiratory minute volume (l/minute), which have in common units of volume per time, tend to scale with $M^{0.75}$. These rates increase as animals increase in size, but note that the $b < 1$ slope (Fig. 37.2) means that a 5-kg animal will have a *lower* O_2 consumption per unit of body mass compared with a 50-g animal.

The frequency of cyclic events ranging from individual cycles (heart rate, respiratory rate, and so on) to population cycles (rate of population increase [r_{max}] or number of births/female/year, and so on) tend to scale with an exponent of approximately $M^{-0.25}$. For example, the heart rate

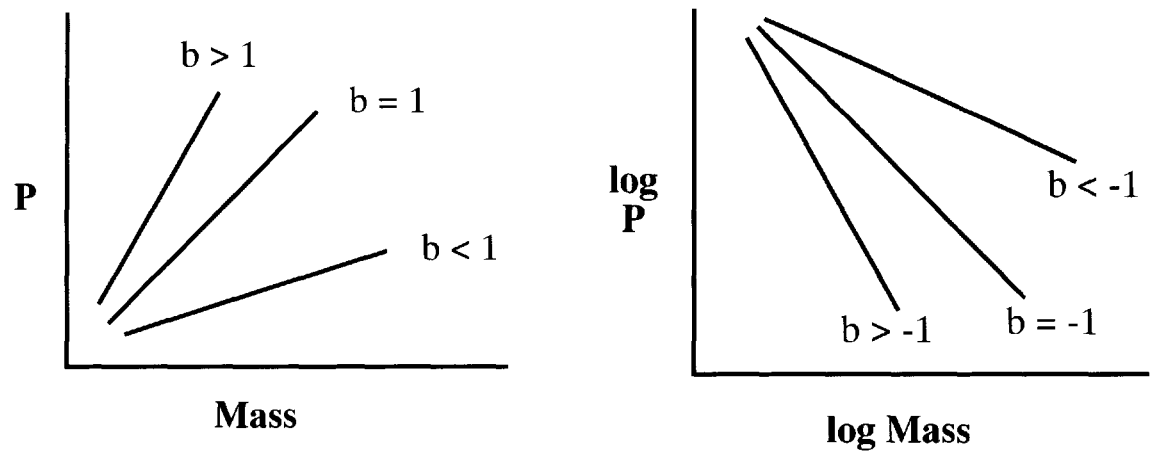

Figure 37.2. Patterns of the relationship of the magnitude of the parameter P to an animal's mass for various values of the exponent b.

of a large animal tends to be lower than that of a smaller animal.

Since cyclic frequencies vary with $M^{-0.25}$, the time to complete one cycle varies with $M^{0.25}$ (that is, $b = +0.25$). This means that the duration of one cardiac or respiratory cycle, the time to burn one unit of energy (kilocalorie, or kcal), or the time to excrete a given amount of a drug (the half-life) will tend to increase in a predictable fashion with the increasing size of the organism. The durations of many biological processes (time, for example, longevity or gestation period) tend to scale with $M^{0.25}$ (Fig. 37.3) (Linstedt and Calder, 1981).

Manipulation of Allometric Equations

If the correlation coefficients are very high, allometric equations can be manipulated as if they were simple equations, that is, as if they were exact rather than best-fit expressions. For example, in birds the growth time (t) is proportional to $M^{1/3}$ ($t \propto M^{1/3}$), and the length of the tarsometatarsus bone (L) is proportional to $M^{1/3}$ ($L \propto M^{1/3}$). The L/t (growth rate) is then $M^{1/3}/M^{1/3}$, which is equal to $M^{1/3-1/3}$, which is equal to M^0, which is 1. Thus, L/t is found to be proportional to a constant and independent of mass. This result is supported by Kirkwood et al. (1989), who found that tarsometatarsus growth rate was related to $M^{0.03}$ in a sample of 87 species of birds. The exponent 0.03 is not significantly different from 0.

Some Caveats

How useful is this equation as a general predictor of physiological, pharmacological, and other values? The points in Fig. 37.1 do not fall right on the line, but are scattered about it, so the equation provides an estimate of the value of P for a given M. Depending on the correlation coefficient, which provides an index of scatter about the line, the estimate may be good or may be of little value in practice. The confidence limits of an estimate of P for a given M can be calculated using techniques described in standard statistical texts.

Two important issues have a bearing on the value of the equations for predictive purposes. These methodological points have been discussed by Harvey and Mace (1982), Calder (1984), and others:

1. *The nature of the database*: The values of intercept a and slope b are wholly dependent on the data points (species) chosen or available for inclusion in deriving the regression equation. The line obtained may not represent the best fit through the data for the entire class or other subsets of it. The larger the data base and the greater the size range of the species included, the less likely it is that the line will be seriously biased. This point is particularly important when one is considering nonmammalian vertebrates, such as raptors. For many categories of data (physiological, population, and so on), our present extrapolations are based on small data sets from a limited number of avian species. Since one purpose of constructing allometric equations is to estimate values for an unknown species from a data set about its taxonomic group, the quality of the data determines the accuracy of the estimate. For this reason, it is imperative that additional, carefully collected, numerical data describing normal biological processes in healthy birds make their way into the literature.

2. *Statistical methods*: The equation also depends on the statistical method used in its derivation. There are a number of ways of fitting lines through scattergrams of data: least squares linear regression, major axis, or reduced major axis analyses. The pros and cons of these methods are described by Feldman and McMahon (1983), Smith (1984), and Calder (1984). When the correlation coefficient is high, the lines produced by these techniques are more or less superimposed and of equal predictive value in practice. But when

Table 37.1
Allometric formulae for nonpasserine birds

Parameter	*Formula*	*Reference*
Life span in captivity	yr = 28.3 $M^{0.19}$	Calder (1984)
Life span in the wild	yr = 17.6 $M^{0.20}$	Calder (1984)
Incubation period	days = 28.9 $M^{0.20}$	Calder (1984)
Length of respiratory cycle	sec = 3.22 $M^{0.33}$	Calder (1984)
Length of cardiac cycle	sec = 0.39 $M^{0.23}$	Calder (1984)
Heart rate (# per min)	155.8 $M^{-0.23}$	Lindstedt and Calder (1981)
Respiratory frequency (# per min)	17.2 $M^{-0.31}$	Lindstedt and Calder (1981)
Daily food intake (KJoule/day)	917 $M^{0.69}$	Farlow (1976)
Glomerular filtration rate (ml/min)	2.00 $M^{0.73}$	Calder and Braun (1983)
Tidal volume	13.2 $M^{1.08}$	Calder (1984)
Births per female per year (prop.)	$\propto M^{-0.22}$ to $M^{-0.33}$	Calder (1984)
Oxygen consumption in birds (ml/min)	11.3 $M^{0.72}$	Schmidt-Nielsen (1984)

Note: M = body mass in kg. Basic requirements for every 100 kcals of MEC (for any species): water = 80-120 cc; sodium = 2.8 mEq; potassium = 2.2 mEq; chloride = 1.8 mEq.

the correlation coefficients are low, the prediction is influenced by the method used to draw the best-fit line.

Metabolic Rate and Body Size

Early in this century, comparative physiologists measured the metabolic rates of a variety of vertebrate species (Kleiber, 1947; Brody, 1945). As far as possible, these measurements were made on thermoneutral, postabsorptive, nonstressed animals. When the results of these studies were plotted, it was found that the energy required for minimal, resting body functions, including breathing, heart beat, peristalsis, and muscular support, did not increase linearly in proportion to increase in mass between species, but was proportional to body mass raised to about the 3/4 (or 0.75) power, indicating that smaller species had higher metabolic rates per unit mass than larger ones. Gessaman (1987) provides a good explanation of measuring the metabolism of raptors: the value of the exponent depends upon the species included in the analysis and the statistical techniques used. Typically, values in the range of 0.67 to 0.80 are found (Bennett and Harvey, 1987). Those who work with birds realize that these organisms both are smaller and have faster metabolic processes than domestic mammals. Observation reveals that birds also eat more food and excrete wastes more frequently than their equivalently sized mammalian counterparts.

When the lines relating metabolic rate to mass for various taxa have been compared, roughly parallel lines having a slope of about 0.75, but differing only in their Y intercepts, have been found (Fig. 37.4) (Hainsworth, 1981; Kleiber, 1947; Schmidt-Nielsen, 1984). It has been suggested that for various taxa these lines can be described by the following equation:

$$Y = K\,(M)^{0.75}$$

In the equation, Y = the resting animal's energy output in kcals per 24 hr, K = a taxonomically dependent constant, and M = an animal's body mass in kilograms. The following values of K have been suggested: passerine birds, 129; nonpasserines, 78; placental mammals, 70; marsupials, 49; and reptiles, 10 (at a species' preferred optimum temperature).

In practice, this equation allows us to estimate the basal energy expenditure for a wide range of species we might encounter. We refer to this basal energy expenditure as MEC, or minimum energy cost (Sedgwick et al., 1986). Two sample calculations will clarify the relationships:

1. The MEC of a 1,200 g owl can be estimated:
   $78 \times (1.2)^{0.75} = 89$ kcal/day.
2. The MEC of a 45 g passerine can be estimated:
   $129 \times (0.045)^{0.75} = 12.6$ kcal/day.

Mass-specific Metabolic Rate

When allometric rates are expressed per unit of body mass, they are termed mass specific or specific (SMEC). Mass specific scaling exponents are formed by dividing the rate equation by M:

$$Y = a(M)^{0.75}/\,M^{1} = a(M)^{-0.25}$$

Thus, the mass-specific metabolic rate of an animal is a measure of the energy cost per unit mass to carry out its essential metabolic functions. In general, we can say that smaller animals require more energy per unit mass than large animals. For example:

The MEC of a 100-g raptor:
$MEC = 78 \times 0.100^{0.75} = 13.5$ kcals/day,
and therefore:
SMEC = 13.5/0.1 = 135 kcals/kg.
The MEC of a 1,000-g raptor:
$MEC = 78 \times 100^{0.75} = 78$ kcals/day,
and therefore:
SMEC = 78/1 = 78 kcals/kg (Fig. 37.5).

This can also be used to illustrate differences between taxa. For example, comparing the SMEC of an 850-g passerine bird with that of a placental mammal of the same mass:

The MEC of an 850-g passerine bird:
$MEC = 129 \times 0.85^{0.75} = 114$ kcal/day,

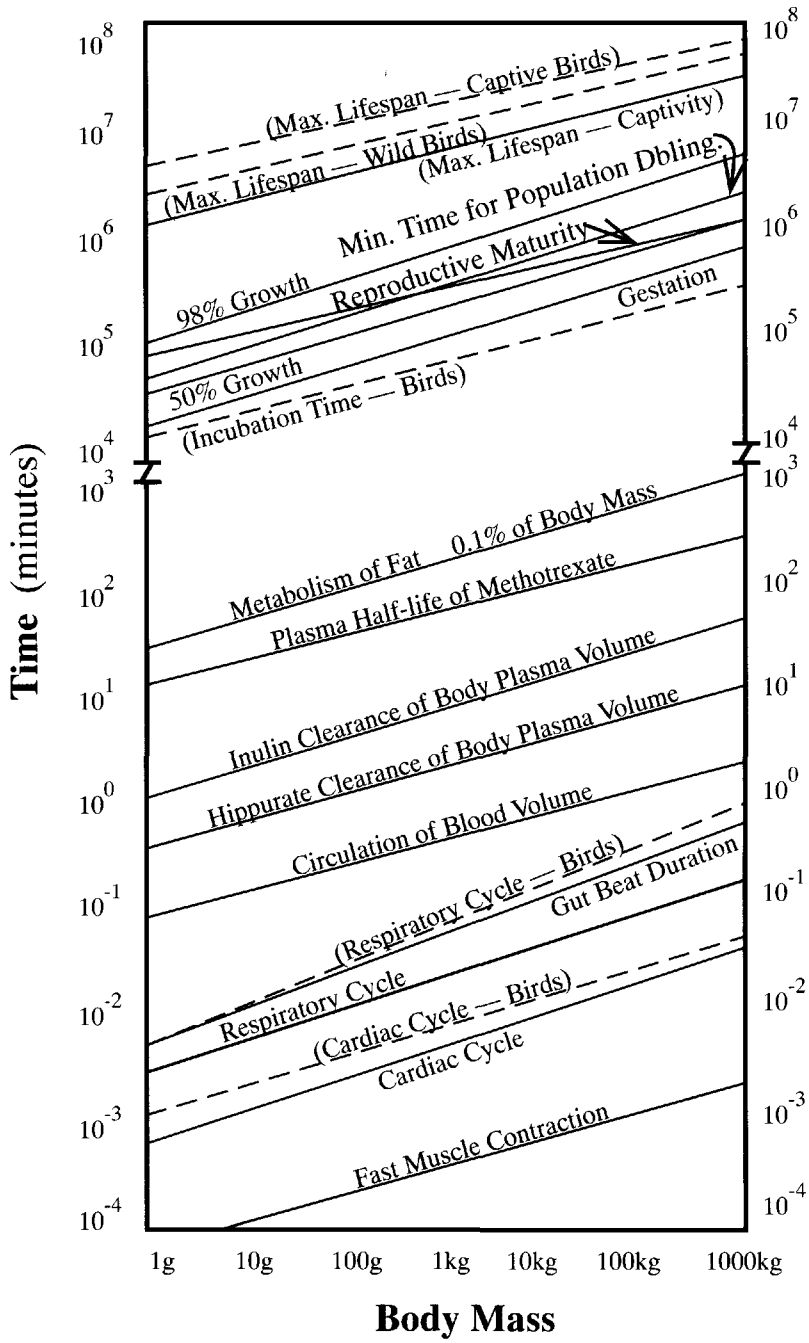

Figure 37.3. The relation between body mass and lengths of biological periods or cycles for mammals (solid lines) and for birds (dashed lines). Individual equations have been extrapolated to span the range of body sizes shown. Used with the permission of the *Quarterly Review of Biology* (Lindstedt and Calder, 1981).

and therefore:

$$\text{SMEC} = 114/0.85 = 134 \text{ kcal/kg/day.}$$

The MEC of an 850-g placental mammal:

$$\text{MEC} = 70 \times 0.85^{0.75} = 61.9 \text{ kcal/day,}$$

and therefore:

$$\text{SMEC} = 61.9/0.85 = 73 \text{ kcal/kg/day.}$$

Passerine birds have nearly twice (1.8 ×) the metabolic rate of a eutherian mammal of the same mass. This can also be seen by dividing the equations for MEC for the two taxa:

$$\frac{129\ M^{0.75}}{70\ M^{0.75}} = 1.8$$

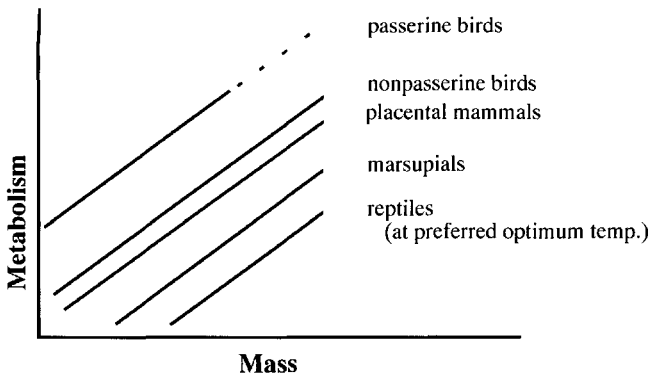

Figure 37.4. Regression lines representing the log-log plot of daily basal energy expenditure versus body mass for taxa of terrestrial vertebrates (after Hainsworth, 1981; Schmidt-Nielsen, 1984).

Allometric Calculation of Dietary Energy Requirements

Minimum energy cost is the amount of energy expended in sustaining vital functions when a resting animal is in a thermally neutral environment. The daily energy requirements exceed this (Kirkwood, 1981; Robbins, 1983; Calder, 1984). The amount by which requirements exceed MEC can be estimated under a variety of conditions as follows:

animal at rest	1.3-1.5 × MEC
animal with injury	1.5-2.5 × MEC
after surgery	1.5–2.5 × MEC
actively exercising	2-6 × MEC (depends on amount of exercise)
growing young	2-5 × MEC (depends on rate of growth)

Metabolic Scaling and Nutrition

We have already shown that the daily energy requirements of raptors are easily estimated. Food requirements can be estimated after consulting Fowler (1986), Exler (1987), Kirkwood (1981), or Watt and Merrill (1963) to find out how much metabolizable energy is contained in given food items. For example:

1. For a bald eagle weighing 5,000 g:
   $MEC = 78\ (5)^{0.75} = 260$ kcal/day.
2. Assume a low level of activity in captivity:
   $260 \times 1.5 = 390$ kcal/day.
3. How many adult rats should it be eating daily?
4. Rats contain 1.5 to 2 kcal/g of metabolizable energy (Fowler, 1986; Kirkwood, 1981).
5. Therefore, the bird is likely to require about 195 g of rat per day just for maintenance in captivity.

Sedgwick (1973, 1988a) and others have introduced to veterinary medicine the concept that specific nutrient requirements can also be scaled according to metabolism. For example, work by several authors (cited in Robbins, 1983) on water turnover rates has shown that for a variety of avian species, water turnover (Y = liters per day) scales to body mass with an exponent of 0.75 ($Y = 0.119\ M^{0.75}$) (Walter and Hughes, 1978; cited in Robbins, 1983).

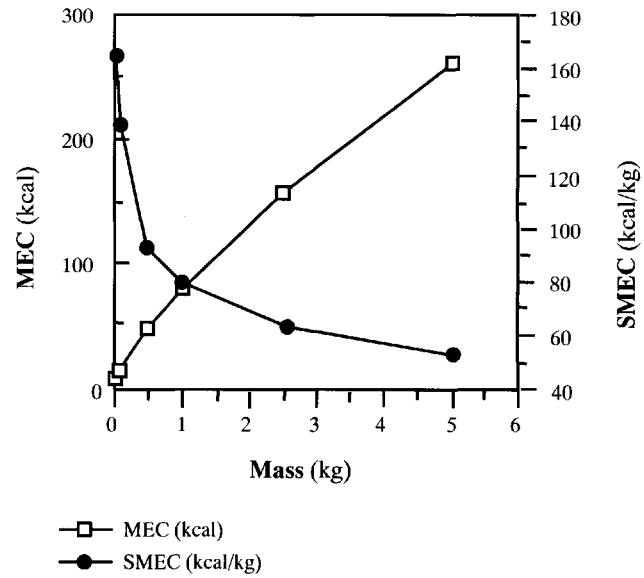

Figure 37.5. Graphic illustration of calculated minimum energy costs and specific metabolic rates for raptors from 50 g to 5 kg.

Other nutrients, both macro and micro, may tend to be utilized in proportion to the metabolic needs of an animal. Therefore, once the MEC for an animal has been calculated, an approximation of the daily requirement of other nutrients, including water, can be estimated allometrically per 100 kcal of daily energy cost (Table 37.2).

Physiologic (Metabolic) Time

Biologists have long made the association that large animals tend to live longer than small ones. In addition, larger animals tend to have slower heart rates and to reach sexual maturity at later ages. In 1945, Brody originated the term "physiological time," recognizing that smaller animals seem to "live" according to a faster time scale than large animals. It is helpful to look at a comparison of the specific metabolic rates of two birds of different weight. Returning to an example used in the previous section:

1. The MEC of a 100-g raptor:
   $MEC = 78 \times 0.100^{0.75} = 13.5$ kcals/day,
   therefore:
   $SMEC = 13.5/0.1 = 135$ kcals/kg/day.
2. The MEC of a 1,000-g raptor:
   $78 \times 100^{0.75} = 78$ kcals/day,
   therefore:
   $SMEC = 78/1 = 78$ kcals/kg/day.

Table 37.2
Suggested nutrients for mammalian or avian parenteral alimentation (per 100 kcal daily energy cost) [Reprinted with permission from the American Animal Hospital Association.]

Nutrient	*Adult*	*Infant*
Protein as essential amino acids (g)	3-7	5-9
Fat as essential fatty acids (g)	3-7	5-9
Carbohydrate as simple sugars (g)	10-13	3-6
Water (ml)	50-95	95-100
Vitamins		
A (IU)	300	300
D3 (IU)	25-35	50-75
E (IU)	2	3
C (mg)	10-15	10-15
K (mg)	3-6	15
Folic acid (μg)	25-50	25-50
Thiamin (μg)	100	200-800
Riboflavin (μg)	100-200	200-400
Niacin (mg)	1-2	800-1000
Pyridoxine (μg)	100-200	90-200
Cyanocobalamin (μg)	0.5	0.5
Biotin (μg)	10-20	10-20
Choline (mg)	20-100	20-100
Inosital (mg)	5	5
Pantothenic Acid (μg)	600-900	600-900
Minerals		
Calcium (mg)	5-100	100-200
Phosphorus (mg) (Ca:P = 1:1 to 2:1)	3-66	50-100
Sodium (mg)	75-100	35-50
Potassium (mg)	150	120
Chloride (mg)	75-100	50
Iron (mg)	1.1	2
Magnesium (mg)	25	10
Copper	0.1	0.1
Zinc (mg)	0.94	0.94
Iodine (μg)	10	10
Manganese (mg)	0.2	0.2

It could be said that the smaller raptor is "living" at nearly twice (1.7×) the rate of the larger. Linstedt and Calder (1981), in their extensive review of the literature on physiological time, remark that for homeotherms, a large number of developmental, physiological, and ecological cycles tend to scale in proportion to the quarter power of the body mass (about $M^{0.75}$) (Fig. 37.3). This elegant statement from Mordenti (1985) is useful:

> Physiologic time is measured by biologic clocks which use internal, physiologic parameters such as heartbeats, breath cycles or blood circulation velocities as units of measurement. When physiologic events in different mammals are measured by biological clocks, they occur in equivalent physiologic time. . . . Thus, the lifespan of an elephant and a mouse is the same when measured with a biological clock (i.e., heartbeats), although their life spans vary significantly when measured in years.

In the section on metabolic scaling and anesthesia below, we include an example of how one may use heartbeats as a measure of physiologic time. Fig. 37.6 is a graphic representation of the difference between chronological and physiological time, from Mordenti (1985).

Metabolic Scaling and Chemotherapeutics

Traditionally, veterinary and human medicine have calculated drug dosage and nutritional requirements based on body weight. Estimations of dose, treatment interval, toxicity, tissue levels, and routes of excretion are based upon measurements made in common, economically important domestic animals. No linear trend among these variables has been demonstrated; various authors have commented on the "random" nature of variables (Baggott, 1977). This approach has been acceptable where extrapolation occurs between species of similar size and metabolic rates, although variation in other factors (for example, species differences in drug metabolism) is known to occur.

For avian, wildlife, and zoo veterinarians, the traditional means of extrapolation based on weight have proven to be less than satisfactory. We must all too often apply drug therapy to patients by extrapolation from a nebulous array of sources and therapeutic philosophies. This section introduces a metabolic scaling approach to the problem of interspecies drug extrapolation based upon physiological principles.

The Rationale of Pharmacokinetic Scaling

We have reviewed some of the ways in which allometry allows us to predict structural and functional characteristics of organisms. Allometry has proved to be of practical significance in the field of comparative nutrition (Robbins, 1983; Sedgwick, 1988a).

Newer and more controversial is the application of the power function to studies of pharmacology and toxicology. The action of toxins or drugs is linked to physiological mechanisms such as the blood supply to organs of excretion and the metabolic processes of cells for removal from the body. It seems reasonable to think that since we can form allometric equations to describe blood flow and metabolic rate, we may in a similar way describe the drug-disposition parameters that are dependent upon them. The relevance of body size to variation between species in the pharmacokinetic parameters describing elimination rates has been demonstrated for a range of drugs (Kirkwood and Merriam, 1990).

When a drug is administered to an animal, it first undergoes the processes of absorption from the site of administration and distribution to tissues. These processes are controlled by blood flow and extent of tissue perfusion (as

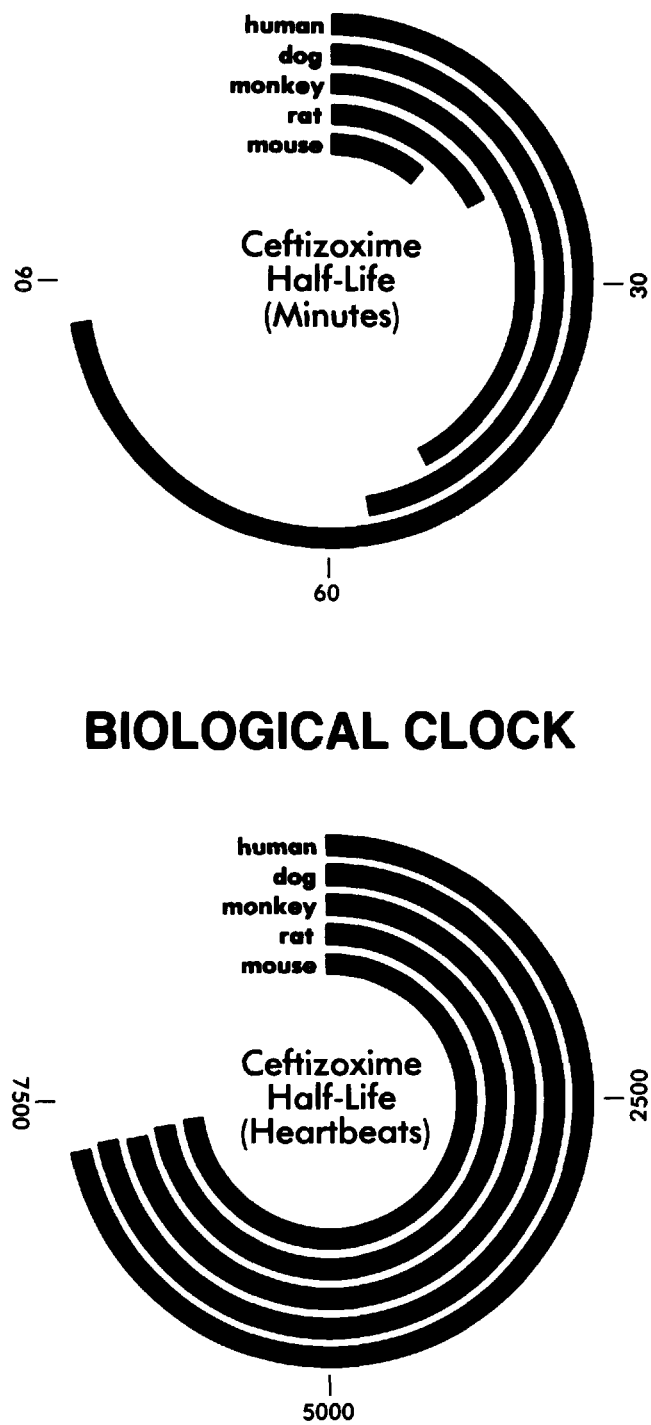

Figure 37.6. Perceived differences in the half-life of ceftizoxime in various mammals depend on the reference system used to denote time. (A) When half-lives are reported in minutes, the smaller mammals eliminate 50 percent of the drug more rapidly than the larger species. (B) When half-lives are reported in heartbeats, all mammals eliminate 50 percent of the drug in an equivalent time. Adapted from Mordenti (1985) with permission of the American Society for Microbiology.

well as by the local factors of pH, protein binding, and solubility). The drug may be taken up by various organs and either biotransformed or excreted unchanged. Rates of biotransformation are thought to be a function of such factors as membrane transport, enzyme kinetics, and concentration gradient. Although drugs vary as to the extent to which they are biotransformed, disposition kinetics are influenced by hemodynamics and rates of cell metabolism.

Based on the differences in physiology and metabolic rate between passerine birds and mammals, we form an intuitive sense of how drug disposition will vary between the two taxa. The 1-kg bird may have more rapid absorption and distribution of a drug from an injection site than the 1-kg mammal. Thus, the time to produce an effect is expected to be shorter in the bird. If the duration of action is governed by the rate of clearance of the drug from the plasma, then we may expect that the duration of effect will tend to be shorter in the bird than in the mammal (assuming they start with the same peak level). If the therapeutic object is to obtain some steady-state level of the drug, then doses may need to be given to the bird more frequently, or at higher levels, than to the mammal. However, this is not necessarily the case (see the discussion below concerning the difference between mammals and birds in glomerular filtration rate).

The little work that has been done in avian pharmacokinetics (Kirkwood and Merriam, 1990) provides evidence to suggest that relative volumes of distribution (V_ds) of drugs will vary with the type of drug. For most classes of drugs, it is not even known how V_d varies among orders of birds (Dorrestein et al., 1984).

Regarding routes of elimination, Dorrestein and van Miert (1988) remark: "In general, there is minimal variation among species in the manner by which polar compounds (like gentamicin) are handled by excretory mechanisms. This is in contrast with the wide interspecies variations and unpredictability associated with the biotransformation of extensively metabolized drugs (like chloramphenicol and sulfonamides)." The lack of information for avian species means that pharmacokinetic scaling in birds is in a more rudimentary stage than in mammals. Interspecies scaling in mammals is beginning to find its way into practical consideration (Mordenti, 1985; Mordenti and Chappell, 1989).

The following section presents (1) an introduction to interspecific scaling using an example of the drug inulin, (2) a basic scheme for scaling therapeutic regimens as outlined by Mordenti (1986; Mordenti and Chappell, 1989) in mammals, and (3) a method proposed by Sedgwick (1988b; Sedgwick et al., 1986) for scaling from mammals to birds.

Inulin Clearance, Scaling Therapeutic Regimens, from Mammals to Birds

A good first example is inulin, a drug that is filtered and excreted unchanged by the kidney. Edwards (1975) determined from published values of inulin clearance in mammals that

$$C_{in} = 5.36\ M^{0.72}$$

where C_{in} is the inulin clearance in ml/min, and M is the weight of the animal in kg. (The exponent 0.72 is not considered to be significantly different from 0.75.) Clearance is a drug-disposition parameter that has the units of a volume per time, just as cardiac output, minute ventilation, and oxygen consumption do; and clearance appears to vary with size in the same way. The data that Edwards used were collected from 26 species of mammals ranging in size from 0.15 to 590 kg.

Calder and Braun (1983) estimated inulin clearance from data on eight species of birds and found that

$$\text{avian } C_{in} = 2.00\ M^{0.73}.$$

Note that the constant 2.00 is lower than that for mammals (5.36), which implies that birds have a lower rate of inulin clearance than mammals of the same size (Edwards, 1975; Calder and Braun, 1983). Since inulin clearance is proportional to glomerular filtration rate (GFR), this suggests that birds also have a lower GFR than mammals of the same size. Although there are few data at the present time for birds, this finding could have important consequences in avian pharmacotherapeutics.

The lower clearance of inulin and perhaps lower GFR in birds compared with mammals seems in contrast with our expectation that clearance rates might be higher in birds due to their higher weight-specific metabolic rate. Perhaps it will be found that drugs that are excreted by simple glomerular filtration are more slowly cleared in birds than in mammals of the same size. This appears to be the case with gentamicin (Kirkwood and Merriam, 1990). The areas of avian physiology and pharmacology are certainly in need of an expanded data base.

A Basic Therapeutic Scaling Scheme

Mordenti and Chappell (1989) remark that, in mammals, the allometric exponents for the clearance of drugs usually range between 0.6 and 0.8. An exponent of less than 1.0 signifies that the mass-specific clearance, or Cl/kg, is lower in a large animal than in a smaller animal. Thus, one should expect that the same mg/kg dosage given to a 5,000-g eagle will result in a much longer "duration of action" than in a 100-g kestrel. To maintain effective drug levels, one must give the drug to the smaller animal more frequently.

This scaling principle may be applied toward three basic pharmacotherapeutic goals in approximating dosing schedules for an "unknown" subject based on pharmacokinetic data from a model animal (Mordenti and Chappell, 1989).

Goal 1 is the achievement of average equivalent steady-state concentrations. This is the basic ambition of most antibiotic/antifungal drug therapy and many other therapeutic agents. The plasma concentrations will oscillate around a constant average value. Mordenti reports an equation to predict dose for a subject animal (in this case the "subject" is a human) based on a dose in a "model" (animal) such that their steady-state concentration (C_{ss}) will be the same:

$$D_{human} = D_{animal}\left(\frac{F_{animal}}{F_{human}} \times \frac{T_{human}}{T_{animal}} \times \frac{W_{human}}{W_{animal}}\right)^{0.70}$$

D is the dose (mg, μg, and so on), F the bioavailability, T the treatment interval (min, hr, or the like), and W the body mass in kg. If T and F were the same for both animals, then the smaller of the two would require higher doses, which would result in higher peak concentrations (assuming equivalent V_d in the two animals). Mordenti uses the value for the exponent of 0.70 as a compromise between proponents of the 0.67 and 0.75 values.

In veterinary medicine, this means that if the smaller animal can tolerate the higher dose, and thus peak concentration, then the treatment interval may not need to be altered. If the predicted dose (mg/kg) is above the accepted toxic threshold, then the equation can be used to shorten the treatment interval, thus lowering the peak dose while maintaining "pharmacokinetic similarity." Although the above equation has many terms, it is a relatively simple matter to insert the values from a known situation to derive a dose for an animal for whom nothing is known but its weight.

Goal 2 is the achievement of similar peak concentrations. There are two instances in which calculation of doses to achieve equivalent minimum or maximum concentrations would be desirable. The first is if it is known that exceeding a certain concentration produces toxicity, as in the case of the drug gentamicin. The second is if a certain minimum concentration needs to be obtained. The equation given by Mordenti to achieve equivalent peak concentrations is as follows:

$$D_{human} = D_{animal}\left(\frac{F_{animal}}{F_{human}}\right)\left(\frac{V_{human}}{V_{animal}}\right) = D_{animal}\left(\frac{W_{human}}{W_{animal}}\right)^{1.0}$$

where V is the volume of distribution (ml, l, and so on), an estimate of the extent of distribution of the drug within the body (Baggot, 1977). The reduced equation assumes F is similar and volume scales as $W^{1.0}$.

Goal 3 is the achievement of similar "area under the curve" (AUC). AUC is important when cumulative dose becomes an issue, such as with the relatively toxic antineoplastic drugs. The equation given for this, in which hum = human and an = animal, resembles the equation given for Goal 1 above:

$$\#\text{doses/day}_{hum} = \#\text{doses/day}_{animal}\left(\frac{F_{an}}{F_{hum}} \times \frac{D_{an}}{D_{hum}} \times \frac{W_{hum}}{W_{an}}\right)^{0.70}$$

These three equations are based on the work of Mordenti (1986) and Mordenti and Chappell (1989) and apply to extrapolations within the taxonomic group of placental mammals. Extrapolation between taxonomic groups based on variations in metabolic rates (K factors) rather than body size are considered in the next section.

A Proposed Method of Scaling from Mammals to Birds

Sedgwick et al. (1986), Sedgwick (1988b), and Kirkwood (1983a, 1983b) have proposed that if drug disposition is influenced by metabolic rate over a range of sizes within a taxon, then the K factor constants (mentioned above in the section on metabolic rate and body size) should also be considered when one is predicting dosage. In other words, the K factors should relate parameters that scale with the 0.75 exponent, as dose for equivalent C_{ss} does. If all else is equal, we might expect dose (mg) for C_{ss} per unit of energy expenditure to be roughly constant between species. The following example shows how dose can then be calculated, remembering that K for nonpasserine birds is 78 and for mammals is 70:

1. Using a species for which reliable laboratory data are available (model species), find a reliable dose and treatment interval.

Example: Cephalothin, 2.0 g IM QID for a 70 kg "model" human (Sanford, 1988). Then, 2,000 mg × 4 = 8,000 mg/day for the total dose.

2. Calculate the MEC for this species:

$$MEC = K \times M^{0.75},$$

or

$$MEC = 70 \times 70^{0.75} = 1{,}694 \text{ kcal/day}.$$

M is the weight of the animal in kg and K is the K factor constant for placental mammals, or 70.

There are currently two models for how the above quantities can be used to calculate an allometric, or MEC, dose. Methods 1 and 2 are illustrated in Steps 3 through 5. Conceptually, these methods differ in that Method 1 concludes that smaller species with higher metabolic rates must *both* receive an increased dose of a drug (mg/kg) and receive these doses at more frequent intervals. Method 2 concludes that simply increasing the frequency of administration should be sufficient to account for metabolic variations.

Method 1 gives numerical results that are in agreement with Mordenti and Chappell's (1989) pharmacokinetic data for several mammalian taxa. Proponents of Method 1 believe that scaling drug dosages is most appropriately performed by extrapolating from the quantity of drug a model species is given per treatment interval, whereas proponents of Method 2 advocate that extrapolations should be performed from the 24-hr total drug dose received by the model species. As more pharmacokinetic data become available, we anticipate that one of these models will be verified. At present the technique illustrated by Method 1 is in use at the Tufts Wildlife Clinic.

Method 1

3. Divide the per treatment dose (mg) by the MEC and get a dosage in mg/kcal:

$$2{,}000/1{,}694 = 1.18 \text{ mg per kcal.}$$

Assume that this dosage rate is applicable to any species.

4. Calculate the appropriate dose for an 890-g peregrine falcon (subject species):

a. Calculate the MEC:

$$78 \times 0.89^{0.75} = 71 \text{ kcal.}$$

b. Multiply by 1.18 mg/kcal to get the dose:

$$71 \times 1.18 \text{ mg/kcal} = 83.78 \text{ mg/treatment.}$$

5. Calculate a metabolically appropriate dosing interval:

a. Calculate specific metabolic rate (SMEC, expressed as kcal/kg) for both model species and subject species:

Human: 1,694 kcal/70 kg = 24.2 kcal/kg.

Falcon: 71 kcal/0.89 kg = 79.7 kcal/kg.

b. Utilize these values in the following formula:

$$\text{Dose Interval}_{\text{subject}}\ (\text{hours}) = \left(\frac{\text{SMEC}_{\text{subject}}}{\text{SMEC}_{\text{model}}} + \text{Dose Interval}_{\text{model}}\right)^{-1}$$

For this subject (peregrine) this would work out in this manner:

$$\text{Dose Interval} = [(79.7/24.2) + 6]^{-1} = 1.8 \text{ hrs.}$$

This means that ideally the falcon's daily dose should be divided into 12 doses of 84 mg to be given once every 2 hr, or 1,008 mg/day.

Method 2

3. Divide the total daily dose (mg) by the MEC and get a dosage in mg/kcal:

$$8{,}000/1{,}694 = 4.72 \text{ mg per kcal.}$$

4. Calculate the appropriate dose for an 890-g peregrine falcon (subject species):

a. Calculate the MEC:

$$\text{MEC} = 78 \times 0.89^{0.75} = 71 \text{ kcal.}$$

b. Multiply by 4.72 mg/kcal to get the dose:

$$71 \times 4.72 = 335 \text{ mg/day.}$$

5. Calculate a metabolically appropriate dosing interval: The calculation for dosing interval is identical to that in Method 1, however, subsequent reasoning differs. We now must take the total daily dose of 335 mg and divide it into equal intervals to be given every 1.8 hr. When rounded to unit hours, this means that the falcon's daily dose should be divided into 12 doses of 28 mg to be given once every 2 hr.

Obviously, such brief treatment intervals are impractical under most circumstances. If a drug were safe, it would probably be appropriate simply to give a larger dose less often. It is possible to use the equation for steady-state concentrations from Mordenti and Chappell (1989) for a longer and more practical treatment interval, as long as the new dose is not toxic. The toxic dose can be estimated from the model animal, if available, by using the peak-concentration equation given above from Mordenti and Chappell. The following list summarizes the differences in the two methods:

Step	*Method 1*	*Method 2*
3. mg/kcal dose	1.18	4.72
4. dose for 0.89 kg falcon	83.8 mg/ treatment	335 mg/day
5. determine interval	84 mg every 2 hr for 12 treatments per day = 1,008 mg/day	28 mg every 2 hr for 12 treatments per day = 335 mg/day

The dramatically different results obtained from these two models point clearly to the need for further laboratory studies of avian pharmacokinetics. Clearly, until pharmacokinetic studies are performed in a broader range of species, one must be very cautious about the use of drugs with known tissue toxicity in small species with high metabolic rates. We hope that this knowledge about the effects of metabolic scaling on pharmacokinetics will provide the impetus for the development of new techniques for drug administration in small wildlife species. In theory, such techniques as implantable osmotic pumps, slow-release drug-impregnated polymers, and adhesive patches for transcutaneous absorption might provide the slow, constant levels that small species appear to require.

The rates and efficiency of absorption from the digestive tract of birds appear to be quite variable. This is an area in which it is very unreliable to apply mammalian dosing schedules to birds, owing to the great variation in avian gastrointestinal anatomy and function. Dorrestein and van Miert (1988) present a good review of the methods of drug administration in birds.

Table 37.3
Calculated doses for nonpasserine birds based on an MEC dose of 0.23 mg/kg of ketamine compared with published data for empirically derived doses of ketamine

Body weight (kg)	*MEC (kcal)*	*Total ketamine dose (mg)*	*Calculated dose (mg/kg)*	*Empirical dose (mg/kg)*[a]
0.040	6.9	1.6	40	40
0.100	13.8	3.1	31.7	30
1.187	88.7	20.4	17.1	20
2.0	131	30.1	15	16
4	220.6	50.7	12.6	16
8	371	85.3	10.6	16

[a]Samour et al. (1984).

Extensions of Scaling Principles to Other Aspects of Raptor Biomedicine

Metabolic Scaling and Anesthesia

In a retrospective study of several injectable anesthetics, Samour et al. (1984) examined the use of these drugs in 154 species of birds ranging from 40 g to more than 8 kg. Table 37.3 includes a sampling of their data for a variety of nonpasserines.

The allometric (or MEC) dose of 0.23 mg/kcal utilized in Table 37.3 was derived as described in the above section on chemotherapeutics. By comparing the last two columns of Table 37.3, one can see that the doses predicted by allometric scaling closely approximate those derived empirically. Kirkwood and Wathes (1984) plotted Samour's data for ketamine allometrically and found that doses of ketamine required for adequate sedation scale with body mass to 0.78. This suggests that the dose rates are a function of the animals' metabolic rates, although other explanations are possible. However, the principles of allometric scaling may assist in making reasonable approximations for the use of injectable anesthetic agents on unknown species before subjecting any patients to an actual trial.

Studies at the Wildlife Clinic of Tufts University School of Veterinary Medicine (Sedgwick et al., 1986; Sedgwick, 1988b) have found that a combination of equal volumes of the injectable anesthetic agents ketamine (100 mg/ml) and xylazine (20 mg/ml) also scales allometrically for a wide variety of species. The ketamine-xylazine (50:50 mixture by volume) MEC dose of between 0.0022 and 0.0044 ml per kcal of basal energy requirement will chemically immobilize most vertebrates, including raptors, from small kestrels to the largest eagles. The lesser rates of administration should be used in the larger species, otherwise considerable respiratory depression may occur.

Metabolic Scaling and Heart Rate

One of the most useful indicators for measuring depth of anesthesia is heart rate. Knowing that the heart rate of raptors scales according to $156\ M^{-0.23}$ (Schmidt-Nielsen, 1984) allows us to calculate what the resting heart rate of any species should be. The Tufts Wildlife Clinic uses a functional definition of bradycardia as any rate more than 20% below the calculated rate.

Metabolic Scaling and Surgical Issues: Heart Rate and Oxygen Consumption

A knowledge of metabolic time is also critical for successful anesthetic technique. For example, common knowledge has it that humans can live for 5 to 6 min without oxygen before sustaining brain damage. Most veterinary students learn that dogs can survive for about 4 min under the same conditions. According to metabolic time, this should indicate to us that any vertebrate should be able to live for about 500 heartbeats before serious damage occurs. If this is so, what does it mean for our raptor patients? We already know that birds have slower heart rates than mammals of the same mass (Calder, 1984). Calculating the heart rate for a 6,000-g raptor reveals that it is likely to have a resting rate of approximately 100 beats per min. A 50-g raptor, on the other hand, would have a calculated rate of about 310 beats per min. We contend, therefore, that, all else being equal, the large raptor should survive about 5 min unharmed, but that our smaller patient might suffer irreversible damage in less than 2 min.

Hypothermia

Smaller patients have a proportionately greater surface area from which to lose heat than large ones. A surgical plane of anesthesia eliminates the patient's ability to generate additional heat by movement, shivering, or other adaptive actions. Additionally, ventilation is conducted with relatively cool, low-humidity gases. So even before an initial incision is made on a small avian patient, the bird may be approaching hypothermia.

Once the bird's body is opened surgically, we have not only eliminated the insulative protection of its feathers, but we have drastically increased the surface area available for cooling. So it is apparent that the provision of external sources of heat, an ocassionally used option for larger patients, is critical to surgical success with small birds.

In metabolic time terms, every minute on the surgical table for a 6-kg eagle is metabolically equivalent to 3 min of surgical time for a 50-g owl. Therefore, in planning the length of stressful procedures with small patients, attention must be paid to the metabolic clock—*not* the one on the wall. A 1-hr surgery on the tiny owl could be expected to cause the same amount of physiological stress as subjecting the eagle to a 3-hr procedure.

Conclusion

As stated at the outset, this chapter provides only a brief introduction to allometric scaling. We have not addressed such topics as population biology (where scaling has been used to explain the differences in population cycles of several species), island biogeography (where scaling has been useful in explaining the distribution of variously sized animals), the evolutionary limits of growth (where scaling has contributed strong support for the theory of the warm-blooded dinosaurs), and dozens of others. Schmidt-Nielsen (1984) and McMahon and Bonner (1983) provide a basic introduction to allometric scaling, while Calder (1984) gives a much more detailed and mathematically more rigorous approach.

For anyone wishing to try scaling drug dosages and periodicities, but intimidated by the mathematics entailed in allometric scaling, Pokras (1987), Pokras and Sedgwick (1987), and Sedgwick et al. (1986) offer simplified introductions to working with these concepts.

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CHAPTER 38

Appetite Stimulation in Raptors

DAVID L. SUAREZ, D.V.M.

Southeastern Raptor Rehabilitation Center
Auburn University College of Veterinary Medicine
Auburn, Alabama, U.S.A.

ABSTRACT: The effective management of anorexia in raptors requires identification of the causes and use of the appropriate therapy for its control. Successful management consists of a blend of good husbandry practices, presentation of familiar foods, familiarization to novel diets, and the use of pharmacological agents. B-complex vitamins, corticosteroids, anabolic steroids, probiotics, and benzodiazapine derivatives have been employed to stimulate appetite where provision of reduced-stress environments and familiar foods have been insufficient. Based on a mix of largely unpublished experiences of various avian clinicians as well as extrapolations from human and other areas of veterinary medicine, the use of B-complex vitamins at a rate of 10 mg/kg thiamine, dexamethasone at 2-4 mg/kg, stannizol at .5 mg/kg, diazepam at .25-.5 mg/kg, and Lactobacillus cultures are recommended for pharmacological management of anorexia in raptors.

KEY WORDS: raptor, anorexia, appetite stimulation.

Anorexia can be a significant clinical problem in wild birds, and a concerted effort is often required by the clinician to induce feeding. Factors inhibitory to a bird's feeding behavior include reaction to the stress of injury and captivity, debilitation, injury, and organic disease. Anorexia is managed through recognition of the underlying cause(s); good husbandry practices, such as provision of a reduced-stress environment and recognizable food; and medical therapy. Little specific information exists on methods for medically managing anorexia in birds. The purpose of this chapter is to summarize the major clinical trends in managing this problem in birds based on the limited published information, personal communications, my own experiences, and extrapolation of the use of procedures used in human and other areas of veterinary medicine.

Management of Anorexia by Traditional Methods

Captivity may cause stress to wild animals. Among raptors, buteos, falcons, and owls generally are more tolerant of captive environments, while bald eagles (*Haliaeetus leucocephalus*), accipiters, and especially ospreys (*Pandion haliaetus*) exhibit poorer responses. Ospreys seldom feed spontaneously in any but the most stress- and disturbance-free environments. Astute attention to individual response to stress is important for inducing spontaneous feeding.

The state of health and injury of birds at admission will have an impact on the appetite of the patient. Two different situations are commonly encountered: (1) acute injuries without debilitation, and (2) chronic injury with debilitation. The former is readily managed in most instances, since the patient, once stabilized, can be offered and will accept an appropriate diet if favorable husbandry practices (see below) are employed. The starving bird, on the other hand, often cannot digest a solid diet and should receive easily digested preparations (Redig, 1984).

The base of such diets usually consists of an electrolyte solution with simple sugars and amino acids. Products available for human uses[a] may be used for raptors, as well as several homemade solutions (Redig, 1984; McKeever, 1979, pp. 30-34). A commercially available avian product[b] may also be chosen. In severely emaciated birds, the simple diet is fed by gavage tube exclusively for the first day at a rate totaling about 10% of the bird's weight in 2 to 4 divided doses (see Pokras et al., chapter 37, this volume, for further information on establishing amounts to feed). The volume of the simple diet is gradually decreased as a more complex or solid-meat diet is accepted by the bird. This transitional process continues until the bird is taking a whole-animal diet, which may take more than a week.

Force-feeding may be required to induce recognition and acceptance of the novel diets that are often substituted for natural foods. Such items as ground-meat products[c] or day-old cockerels or turkey necks or white mice may be substituted (Garcelon and Bogue, 1977). The sight of another raptor feeding may also help stimulate feeding behavior.[1]

Pharmacological Solutions to Anorexia

Husbandry solutions to anorexia are not always successful, and medical therapy may be employed. The use of drugs should not suspend the search for a possible organic cause of anorexia, however, because the anorexia may only be a

sign of a specific disease. The use of various agents with appetite-stimulating properties may be attempted. The drug groups recommended are the B-complex vitamins, corticosteroids, anabolic steroids, benzodiazepines, and the probiotics.

B-complex vitamins are commonly used to stimulate appetite in mammals (Buffington, 1986; Jenkins, 1982) and birds (Redig, 1992). Vitamins are regulatory enzymes for various physiologic processes. They are divided into fat-soluble and water-soluble forms. The fat-soluble vitamins (K, A, D, and E) are usually stored in large enough quantities to survive periods of injury or disease, but the water-soluble forms, which cannot be stored in great amounts, must be constantly ingested. Animals that are injured or diseased will require higher levels of vitamins, but it is often at this time that the ingestion of vitamins is at its lowest point (Buffington, 1986). The combination of increased need, decreased intake, and rapidly diminishing reserves makes the use of vitamins logical (Lewis et al., 1987). The B-complex vitamins are thought to be the most helpful vitamins for anorexia, however, their mechanism of action is poorly understood (Jenkins, 1982). The dosages of B-vitamins in raptors have not been specifically established, and prescribed dosages are extrapolations from other species. Fortunately, the use of B-complex vitamins, except in very large doses, is considered harmless. Amounts of B-complex vitamins administered are determined as a function of the thiamine content of the preparation, so that the dosage rate of 10 mg/kg given daily by IM injection for the duration of the anorexia is recommended (Clubb, 1986).

The second group of drugs to be considered for treating anorexia are the corticosteroids (Lewis et al., 1987; Jenkins, 1982; see Kaufman et al., chapter 32, this volume). Corticosteroids are widely used in veterinary medicine and are commonly administered in emergency raptor care (Redig, 1992; Kaufman et al., chapter 32, this volume). Corticosteroids are usually given in a single large dose by intramuscular or intravenous routes and are not repeated except in cases where continued anti-inflammatory action is needed, such as cranial trauma. The side effects of steroid use include immunosuppression, decreased wound healing, and iatrogenic Cushing's disease, but a single dose is rarely contraindicated. The dose of dexamethasone for emergency use is 2 to 4 mg/kg (Redig, 1992). In mammals, the corticosteroid appetite-stimulating effect is thought to be the result of its ability to create a euphoric effect that may relieve depressive states and therefore stimulate food intake (McDonald, 1982).

Several researchers suggest the use of anabolic steroids (stanazolol[d]) as another alternative for anorexia management in mammals (Lewis et al., 1987; Jenkins, 1982). The action of anabolic steroids involves their tissue-building capabilities, and they are used mainly in animals recovering from debilitating diseases. A dosage recommended by Clubb (1986) for stanazolol for use in birds is 25 mg/kg once or twice weekly.

Benzodiazepines, tranquilizers with both anticonvulsant and antianxiety activities, are commonly used for appetite stimulation in small animals (Lewis et al., 1987; Lulich and O'Brien, 1988; Jenkins, 1982). Two theories of mechanism of action have been proposed. Benzodiazepines may inhibit the satiety center or activate the hunger center in the brain (Jenkins, 1982; Baile and McLaughlin, 1979). The second theory invokes suppression of emotional influences that inhibit feeding (Baile and McLaughlin, 1979). The benzodiazepines used for stimulation include diazepam,[e] oxazepam,[f] and flurazepam hydrochloride[g] (Lewis et al., 1987; Lulich and O'Brien, 1988). Degree of appetite stimulation, induction of ataxia, and length of effect vary among these compounds.

In one study, oxazepam proved to be the most successful drug in cats of those tested, but it is available only in oral form (Fratta et al., 1976). Oxazepam has been shown to continue its appetite stimulation for up to 12 hr after ingestion (Fratta et al., 1976). Flurazepam hydrochloride has been reported to last for 4 to 7 days in cats (Lulich and O'Brien, 1988). Diazepam, although not as powerful a stimulant as some of the other drugs, is used commonly in cats and is readily available in a variety of dosage forms.

Benzodiazepines are effective in overcoming many causes of feeding inhibition, including heat stress and food additives with unfamiliar or aversive flavors. Their ability to overcome anorexia with many disease states in a variety of species has been reported (Baile and McLaughlin, 1979). Different species also show different responses to the various drugs. Cats are several times more sensitive than dogs to the effects of the benzodiazepines (Della-Fera et al., 1980). Interspecific response variation among raptors is likely.

In my experience, diazepam has been an effective appetite stimulant in raptors and is the recommended benzodiazepine because of availability, oral and parenteral forms of administration, and safety with raptors. The drug has been given both IM and IV in dose ranges of .25 mg/kg to .5 mg/kg. A single dose is often effective, but dosing at daily intervals for 2 or 3 days can also be helpful.

Red-tailed hawks (*Buteo jamaicensis*), red-shouldered hawks (*Buteo lineatus*), great horned owls (*Bubo virginianus*), bald eagles, and several nonraptorial species have responded to diazepam therapy, although favorable results were not obtained in all situations. Common side effects have been sedation and ataxia; IV administration has produced more marked side effects than has the IM dose. Response time following either type of administration ranged from almost immediate to several hours. Further work is

needed to determine optimal doses and definition of circumstances leading to failure of ingestive behavior.

Probiotics have been recommend for management of anorexia and diarrhea (Flammer, 1986). A common cause of diarrhea in birds is antibiotic use with resultant intestinal upset (Clubb, 1986). The antibiotics are believed to kill or inhibit certain intestinal organisms, thereby favoring Gram-negative organisms, which are often pathogens. These organisms can in turn cause clinical disease or gastrointestinal irritability. The feces of cage birds are often Gram stained to categorize the bacteria by Gram reaction and morphology (Harrison, 1986). The clinician can often detect problems with the bird by a change in the bacterial flora (Harrison, 1986; Sable, 1986). If the normal flora can be reestablished, the patient often will resume eating normally. This is one of the uses of probiotics in trying to reestablish beneficial bacteria. The bacteria that are considered important are those that generate lactic acid, in the genera *Lactobacillus* and *Streptococcus*. Their mode of action is unknown, but the theories for their beneficial action include production of lactic acid, hydrogen peroxide, or antibiotics, and competitive antagonism to pathogens (Wren, 1987).

Probiotics have been shown effective at correcting antibiotic-induced diarrhea in clinical cases in humans (Sandine et al., 1972). Some research has been done with probiotics in poultry, but none has been reported with raptors and psittacines. In chickens, *Lactobacillus acidophilus* organisms have been shown to inhibit *Escherichia coli* if given in sufficient numbers, but they have proved only mildly beneficial in protecting against *Salmonella typhimurium* (Fuller, 1977; Weinack et al., 1985; Soerjadi et al., 1981). In the inhibition of *E. coli*, low pH from production of lactic acid and competitive crop adherence by *Lactobacillus* bacteria are regarded as the most important factors (Fuller, 1977). Of the strains of *Lactobacillus* used in one study in chickens, only chicken-derived strains were able to attach to the crop wall. A rat strain and human strain were both unable to adhere to the crop wall in exposed chickens (Fuller, 1977). It is unlikely that commercial strains of *Lactobacillus* would adhere to crop mucosa, but lactic acid production may be the more important factor. The *Lactobacillus* organisms do have to be present in sufficient numbers before lactic acid production is beneficial (Fuller, 1977). There is some concern that the strains used in commercial products may not become established in the gastrointestinal tract of the alien species (Flammer, 1986; Harrison, 1986). The *Lactobacillus* organism may not be able to colonize permanently, but may initially alter the pH, which allows more beneficial bacteria to grow (Clubb, 1986). This type of therapy is controversial, but it seems to be helpful and, at the least, is not harmful. Clearly, further investigation is needed to establish its utility.

Conclusion

Brief anorexia occurs in a majority of raptors admitted to rehabilitation programs. The period of anorexia commonly lasts only two to three days, but can be much longer. The control of anorexia should be initiated as quickly as possible, for the well-being of the bird. The clinician may choose among husbandry, food presentation, and pharmacological agents to manage the problem.

Product List

[a]Vivonex, Norwich Eaton Pharmaceuticals Inc., 13-27 Eaton Ave., Norwich, New York 13815-0231 USA

[b]Emeraid 1, Lafeber Co., 7278 Milwaukee Ave., Niles, Illinois 60648 USA

[c]Zupreem, Nebraska Brand, Central Nebraska Packing, P.O. Box 550, North Platte, Nebraska 69103 USA

[d]Winstrol-V, Winthrop Veterinary, Sterling Animal Health Products, Division of Sterling Drug, Inc., 90 Park Ave., New York, New York 10016, USA

[e]Valium, Roche, Division of Hoffmann-La Roche Inc., 340 Kingsland St., Nutley, New Jersey 07110 USA

[f]Serax, Wyeth-Ayerst, P.O. Box 8299, Philadelphia, Pennsylvania 19101 USA

[g]Dalmane, Roche Products Inc., Manati, Puerto Rico 00674 USA

Note

1. Personal communication with Dr. Patrick Redig, director, the Raptor Center, St. Paul, Minnesota.

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Part V

Environmental Toxicity

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CHAPTER 39

Barbiturate Poisoning in Twenty-nine Bald Eagles

K. M. LANGELIER, D.V.M.

Island Veterinary Hospital
Nanaimo, British Columbia, Canada

ABSTRACT: In January 1988, 29 free-living bald eagles (*Haliaeetus leucocephalus*) were found poisoned after sodium pentobarbital ingestion from the carcass of a euthanized cow. Levels of consciousness varied from light sedation to deep anesthesia. Clinical signs varied depending on the level of consciousness, but included sluggish corneal reflex, decreased heart and respiration rates, cyanosis, and hypothermia. The crops of all birds were manually emptied. Treatment included warming, intravenous saline infusion, intramuscular doxapram (5 to 10 mg/kg body weight), oxygen therapy, oral activated charcoal, and prophylactic antibiotics (amoxicillin at a dosage of 22 mg/kg body weight PO) as supportive therapy. Of the 29 eagles, 3 were found dead, and 2 died within an hour of presentation. Only 3 were mature, 4 were almost mature, and the remaining eagles were immature. This case demonstrates the importance of proper disposal of carcasses euthanized with barbiturates.

KEY WORDS: barbiturates, sodium pentobarbital, poisoning, bald eagle, *Haliaeetus leucocephalus*, disposal of euthanized carcasses, British Columbia, scavenging, raptor.

Large numbers of bald eagles (*Haliaeetus leucocephalus*) winter in the area of Nanaimo, British Columbia. A survey on January 11, 1988, estimated a wintering population of 787 bald eagles. In winter, many immature eagles are in a negative caloric balance and rely heavily on piracy and scavenging to obtain food (Fischer, 1985). A readily available food source, such as the carcass of a euthanized cow, would soon be eaten.

History

On January 2, 1988, an immature bald eagle was presented to the Island Veterinary Hospital with signs of moderate sedation. The eagle was in good flesh, with the only clinical signs being lethargy and sluggish corneal reflexes. Radiographs and a complete blood count revealed no abnormal findings. The following day, three more eagles were found exhibiting more profound sedation. Two of the birds had fed on the carcass of a cow that had been euthanized with an overdose of the barbiturate sodium pentobarbital. The farmer who regularly fed the eagles did not realize the effect the drug could have if ingested. Although the eagles had been feeding on the muscles of the carcass for three weeks, the amount of food ingested and the concentration of the drug in the skeletal muscles was apparently not sufficient to result in clinical signs. When the carcass of the cow was opened and the eagles fed on internal organs, barbiturate intoxication resulted. The following day another 22 bald eagles were found suffering from barbiturate poisoning: 5 unconscious bald eagles were found at the bottom of one tree, 1 eagle was found unconscious on the railway tracks, 1 was found 21 kilometers south of the cow carcass, and the remaining birds were found in the field near the carcass. Three more mildly affected eagles were found over the next two days. Of the birds found, 21 were deeply sedated.

Clinical Signs and Treatment

Clinical signs ranged from mild sedation to complete unconsciousness. Most of the eagles were hypothermic, with cloacal temperatures as low as 38.1°C. Corneal reflexes were sluggish. Heart rates were generally depressed, and birds with heart rates less than 80 bpm often had cardiac arrhythmias, occasional missed beats, and cyanosis. Respiratory rates were depressed occasionally to less than 20 breaths per min. Full crops were found in the most heavily sedated eagles. An eagle that had ingested rumen contents was not as heavily sedated as those that had ingested liver.

Treatment began with emptying the crop manually after endotracheal intubation to prevent aspiration of crop contents. Up to 1 liter of ingesta was removed in some cases. One bird that was found dead had suffocated on crop contents.

Blood was withdrawn for serology and hematology before treatment. Most of the eagles were given warmed normal saline intravenously on presentation. Some were unconscious or semiconscious for up to four days, so fluids were essential to prevent dehydration.

Warm-water bottles were placed against the axillary area. One hypothermic, unconscious eagle was alert and able to stand 40 min after body temperature was restored. Deeply anesthetized birds required warm-water bottles for the entire time they were unconscious. Heart rates of less than 80 bpm were often characterized by skipped and irregular beats, so when heart rates were lower than 100 bpm or

Table 39.1
Postadmission recovery time of bald eagles poisoned with sodium pentobarbital

Recovery time (to standing)	<24 hr	24-48 hr	48–72 hr	72—96 hr
Number of birds	7	8	5	1

when respiration rates were less than 20 breaths per min, doxapram[a] was administered at a rate of 5 to 10 mg/kg body weight. When doxapram was given intravenously, arousal was instantaneous, as evidenced by head shaking, vocalization, and increased heart and respiratory rates. IM injection produced arousal in less than 30 sec and had longer-lasting effects. Deeply anesthetized birds required IM doxapram injections every 30 min to 4 hr. Oxygen therapy was used on birds with slow heart or respiratory rates and on those exhibiting cyanosis.

Due to the concern over the possibility of aspiration of crop contents, birds that were conscious and able to stand were given prophylactic antibiotics (amoxicillin[b] at a dosage of 22 mg/kg body weight PO). Activated charcoal was given orally to conscious birds to help prevent absorption of any drug remaining in the intestinal tract. B vitamin and iron supplement injections (0.05 mg/kg) were given.

All but one eagle recovered smoothly. That particular eagle flapped wildly at any stimulation. The bird was wrapped in a large blanket and confined to a small kennel to prevent self-inflicted injury. The area was kept dark and quiet, and handling of the bird was kept to a minimum.

Recovery time was variable for the 21 birds requiring treatment (Table 39.1). Birds that took the longest to recover had slow corneal reflex, decreased heart and respiration rates, hypothermia, and full crops.

Complete blood counts after recovery revealed adequate packed cell volume (mean 42.8%, range 37 to 48). Birds that took the longest to recover showed elevations in SGOT (mean 1,182 IU/1).

Discussion

Although surveys reveal that approximately 65% of British Columbia's wintering population of bald eagles are mature birds (Farr and Dunbar, 1987), the majority of eagles affected by the barbiturate poisoning were immature birds. Of the 29 eagles affected, only 4 were mature (see Table 39.2). Many immature eagles that die in the winter months are in a negative caloric balance (Stalmaster and Gessaman, 1984). Research has shown that 50% mortality occurs in the first year of life, and 30% mortality has been recorded for the second and third years. Immature eagles are more likely to engage in piracy to obtain food (Fischer, 1985).

Table 39.2
Age of mortality distribution of bald eagles poisoned with sodium pentobarbitol

Age	1 yr	2 yr	3 yr	4 yr	5 yr	>5	Total
Number affected	9	9	2	2	3	4	29
Number died	0	0	2	0	1	2	5

Of the 29 bald eagles in this case, 3 were found dead, and 2 died within an hour of presentation. One of the immature birds suffocated on crop contents, and the other possibly drowned.

Clinically, the corneal reflex was the most reliable indicator of depth of anesthesia. (Care should be taken to prevent trauma to the cornea when making this assessment.) Slow heart rates often became irregular and erratic, especially when associated with cloacal temperatures below 40°C and cyanosis. Hypothermic animals given pentobarbital are very susceptible to death from ventricular fibrillation (Booth, 1977). Doxapram injections appeared to result in an increase in heart and respiratory rates. IV doxapram produced an immediate response, whereas IM injections took 5 to 30 sec to produce a similar effect.

Amoxicillin was administered rather than chloramphenicol, sulfonamides, or doxycycline, because the use of these antibiotics with barbiturates is contraindicated (Booth, 1977).

These poisonings emphasize the importance for veterinarians to inform their clients of the proper disposal of barbiturate-euthanized carcasses. Burial or cremation is necessary to prevent secondary poisoning of scavengers.

Product List

[a]Dopram-V Injectable, A. H. Robbins Co., 1407 Cummings Dr., Richmond, Virginia 23220 USA

[b]Amoxil-125 Suspension, Ayerst, 1025 Laurentian Blvd., St. Laurent, Quebec, Canada H4R 1J6

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CHAPTER 40

Diagnosis and Treatment of Poisoning in Raptors from the Netherlands: Clinical Case Reports and Review of 2,750 Postmortem Cases, 1975-1988

J. T. LUMEIJ, D.V.M., Ph.D., Dipl. Int. Med. Comp. Animals (R.N.V.A.)
INA WESTERHOF, D.V.M.

Division of Avian and Exotic Animal Medicine
Department of Companion Animal Clinical Sciences
Utrecht University
Utrecht, Netherlands

T. SMIT, D.V.M.
T. J. SPIERENBURG, Ir.

Central Veterinary Institute
Lelystad, Netherlands

ABSTRACT: The clinical approaches to a goshawk (*Accipiter gentilis*) and a common buzzard (*Buteo buteo*) that were found in comas are reported. A tentative diagnosis of poisoning with a hypnotic drug was made in both birds. Analyses of ingluvial and ventricular contents, which were removed by ingluviotomy and gastrotomy, respectively, confirmed the diagnosis. Both birds recovered and were released to the wild. For the differential diagnosis of poisoning in raptors, a review of 597 confirmed poisoning cases in birds of prey is presented, based on postmortem findings from 2,750 raptors found in the wild between 1975 and 1988 in the Netherlands. The types of poisons most commonly encountered were cholinesterase inhibitors, strychnine, and α-chloralose. Based on the situation in the Netherlands, potential methods of diagnosis and treatment for poisoned raptors in avian clinical practice are discussed.

KEY WORDS: Falconiformes, poisoning, organophosphorous compounds, carbamates, aldicarb, parathion, cholinesterase inhibitors, strychnine, α-chloralose, lead, the Netherlands, goshawk, *Accipiter gentilis*, common buzzard, *Buteo buteo*, raptor.

Many substances are known to have poisoned wild birds of prey. The most extensive reductions in raptor populations in the Netherlands were caused in the 1950s and 1960s by organocholorine and methylmercury agricultural compounds (Koeman et al., 1971). For this reason, the agricultural use of aldrin, dieldrin, heptachlor, and the majority of methylmercury compounds was completely prohibited in the Netherlands in 1969 (Koeman et al., 1971). Since then, raptor populations in the Netherlands have been increasing in numbers, and some populations are larger than ever before, such as the honey buzzard (*Pernis apivorus*), goshawk (*Accipter gentilis*), European sparrowhawk (*Accipter nisus*), and common buzzard (*Buteo buteo*) (SOVON, 1987; Bijlsma, 1989).

Accidental poisoning by other chemicals used for agricultural purposes and intentional poisoning of wild raptors, however, still occur. Van Ooijen (1985) reported that of 1,948 raptors examined postmortem, 356 (18%) were poisoned. Most poisoned birds are found dead, but some are still alive when found and may be presented to veterinarians or wild-bird hospitals for examination.

This chapter reports the diagnosis and treatment of two separate cases of *a*-chloralose poisoning in two raptor species that were submitted to the Avian and Exotic Animal Division of the Utrecht University Department of Clinical Sciences of Companion Animals. Furthermore, a review of poisoning cases in raptors from the Netherlands examined postmortem between 1975 and 1988 by the Central Veterinary Institute, Lelystad, is presented in the hope that it will be useful to practitioners in diagnosing and treating raptors suspected of being poisoned.

Case Histories

Case 1

A juvenile female goshawk was presented in August 1985 to the department. The animal had been found in a nearby forest in a coma. Apart from severe mental depression, dry mucous membranes, and a subnormal cloacal temperature (36°C), no abnormalities were detected on physical examination. The heart rate as monitored by an electrocardiograph was 260 bpm. The condition of the bird was good. A soft mass was palpated in the cervical region, indicating a food-

filled ingluvies. This was verified by a total-body radiograph, which was made to rule out trauma or the presence of lead shot. The ventriculus was relatively empty. Since no signs of trauma were seen, a tentative diagnosis of poisoning with a hypnotic chemical, probably α-chloralose, was made, possibly complicated by shock. The bird was treated for shock by the rapid intraveneous administration of lactated Ringer's solution, according to Redig (1984), and dexamethasone[a] (1 mg/kg). Heat was provided with a heat lamp. The ingluvial content was removed by ingluviotomy and analyzed. The following day, the bird regained consciousness. Further recovery was uneventful and the bird was released to the wild after showing normal feeding behavior for one week.

Case 2

An adult common buzzard was admitted to the department in a stupor in January 1987. The bird had been found in a meadow near the clinic. Apart from a subnormal cloacal temperature (38°C) no abnormalities were found on physical examination. The ingluvies was slightly filled. On radiological examination, the proventriculus and ventriculus appeared extremely filled with food. A tentative diagnosis of poisoning with a hypnotic chemical was made. The ingluvies, proventriculus, and ventriculus were evacuated by ingluviotomy and ventriculotomy. The latter was performed through a midline incision in the abdominal wall. The ventriculus was closed with three layers of interrupted sutures.[b] Ingluvial and ventricular contents were analyzed. Extra heat was provided during the recovery period by a heat lamp. The following day, the bird was standing. As in Case 1, further recovery was uneventful.

Toxicology

Chemical Analysis of Ingluvial and Ventricular Contents

Ingluvial and ventricular contents of the two clinical cases were analyzed for the possible presence of α-chloralose. Since α-chloralose is quickly metabolized into trichloroethanol and trichloroacetic acid, the concentration of α-chloralose was calculated from the concentration of organically bound chlorine. Ingluvial or gizzard contents were dried with sodium sulfate and extracted with diethyl ether, evaporated to dryness, and taken up with alcohol. Organically bound chlorine was determined microcoulometrically.[c] The ingluvial content of the goshawk contained 1,500 mg/kg organically bound chlorine, that is, 4,400 mg/kg α-chloralose. The α-chloralose concentrations of the ingluvial and stomach contents of the common buzzard were 650 and 870 mg/kg, respectively.

Chemical Analysis of Dead Raptors

Dead raptors (n = 2,750) submitted between 1975 and 1988 to the Netherlands Working Group on Wild Bird Mortality were examined by the Central Veterinary Institute, Lelystad. Diagnosis of poisoning with various cholinesterase-inhibiting pesticides was made using thin-layer chromatography or high-performance liquid chromatography (Spierenburg et al., 1987; Zoun and Spierenburg, 1989). Lead concentrations in wet-digested animal tissue were determined by graphite furnace absorption spectrometry (van Beek et al., 1987).

Nicotine and strychnine were extracted according to Stewart et al. (1937) and analyzed based on the detection by potassium iodoplatinate (Drost and Reith, 1967) by thin-layer chromatography using alkaline aluminium oxide as the stationary phase and acetonytril as the mobile phase. α-Chloralose was hydrolyzed in an acid environment to chloralhydrate and analyzed using gas chromatography with electron capture detection after purification by steam distillation. The lower limits of reportable residues (limit of quantification) were 0.4 ppm for nicotine and strychnine and 0.2 ppm for α-chloralose. The average recoveries from gastrointestinal content varied between 87% and 92% for nicotine and strychnine, and between 73% and 85% for α-chloralose.

Results of the toxicological studies are presented in Table 40.1. Cholinesterase inhibitors detected (total 477) included parathion (435); mevinphos (8); other organophosphorous compounds (3); carbamoyloximes, especially aldicarb (30); and a carbamate (1).

High lead levels were found in only two specimens, one European sparrowhawk liver (34 ppm; kidney 48 ppm) and a hen harrier (*Circus cyaneus*) (kidney 41 ppm). Lead concentrations in this study are expressed as dry-weight values. The approximate moisture content of liver is 75% and of kidney 80%. Therefore, the estimated values on a wet-weight basis for the European sparrowhawk are 8.5 ppm for the liver and 9.6 ppm for the kidney, and for the hen harrier, 8.2 ppm for the kidney.

Discussion

In this study, the finding of poisonous chemicals such as cholinesterase (ChE) inhibitors, strychnine, α-chloralose, and nicotine in ingesta or tissues of raptors that were found dead in the wild was considered diagnostic for the cause of death. For a diagnosis of lead poisoning, lead concentrations in the liver or kidney and necropsy findings of lead-related pathology were taken into consideration. Although the lethal level of lead in European sparrowhawks and hen harriers is not known, and probably depends on the general condition of the individual involved, Longcore et al. (1974)

Table 40.1
Confirmed cases of poisoning in raptors from the Netherlands as established by the Central Veterinary Institute, Lelystad, 1975 to 1988

Species	*Number examined*	*Type of poison*					*Total poisoning*
		ChE inhibitor	*strychnine*	*α-chloralose*	*lead*	*nicotine*	
Red kite	31	25	1	1	0	0	27
(*Milvus milvus*)		(81%)	(3%)	(3%)			(87%)
Marsh harrier	67	2	2	12	0	0	16
(*Circus aeruginosus*)		(3%)	(3%)	(18%)			(24%)
Hen harrier	24	2	0	2	1	0	5
(*Circus cyaneus*)		(8%)		(8%)	(4%)		(21%)
Goshawk	205	36	6	7	0	1	50
(*Accipiter gentilis*)		(18%)	(3%)	(3%)		(0.5%)	(24%)
European sparrowhawk	403	0	1	0	1	0	2
(*Accipiter nisus*)			(0.2%)		(0.2%)		(0.5%)
Common buzzard	1,485	402	58	27	0	0	487
(*Buteo buteo*)		(27%)	(4%)	(2%)			(33%)
Rough-legged buzzard	22	2	0	0	0	0	2
(*Buteo lagopus*)		(9%)					(9%)
European kestrel	513	8	0	0	0	0	8
(*Falco tinnunculus*)		(1.6%)					(1.6%)
Total raptors	2,750	477	68	49	2	1	597
		(17%)	(2.5%)	(1.8%)	(0.07%)	(0.04%)	(22%)

concluded from experimental studies, and from results reported in the literature for lead-poisoned waterfowl, that 6 to 20 ppm lead (wet wt) in the liver of mallards indicated acute exposure to lead. The lower concentrations associated with mortality due to lead poisoning in a variety of species of Falconiformes were 6.8 ppm in liver and 5.2 ppm in kidney (wet wt) (Wiemeyer et al., 1988). The values found in the present study are therefore well within the range associated with lead poisoning in birds.

Theoretically, poisoning from ChE inhibitors requires more than just chemical analysis of gastrointestinal tract contents or tissues. Determination of brain acetylcholinesterase (AChE) activity in birds that were suspected of having died of anti-ChE poisoning enhances the diagnosis. A reduction of 20% of normal activity indicates exposure, while an AChE inhibition of 50% or more is considered the diagnostic threshold for determining the cause of death (Ludke et al., 1975).

The postmortem toxicological studies that are reported show that poisoning is an important cause of death in birds of prey in the Netherlands (Table 40.1). The types of poisons most commonly encountered are ChE inhibitors (especially parathion and aldicarb, a carbamoyloxime), strychnine, and α-chloralose. Lead and nicotine were found only in isolated cases.

Secondary poisoning with ChE inhibitors of raptors depredating on the poisoned birds and rodents has been reported (Shlosberg, 1976; Mendelssohn and Paz, 1977; Hill and Mendenhall, 1980). The toxicity of organophosphorous and carbamate compounds in relation to wildlife, including raptors, has been reviewed recently (Smith, 1987).

The majority of cases in the present study were caused by intentional poisoning by means of poisoned bait or eggs, although there were also cases of secondary poisoning due to ingestion of other birds that had been eating poisoned grains. Some common buzzards were poisoned through ingestion of crane-fly larvae that had been controlled with parathion (van Ooijen, 1985).

ChE inhibitors have been classified in one group because in a clinical setting it is not possible to differentiate between the various organophosphorous compounds and carbamates. The poisons owe their toxicity to their ability to inhibit AChE, the enzyme that hydrolyzes acetylcholine (ACh). Clinical signs can be explained by the persistence of ACh at nerve endings, where it normally exists only momentarily during the passage of a nerve impulse. Carbamates are known as reversible ChE inhibitors (Clarke and Clarke, 1967; Smith, 1987). It should be stressed that although aldicarb is a carbamate and therefore a reversible ChE inhibitor, it is the most toxic of the anti-ChE pesticides, with an extremely low acute oral LD_{50} (Smith, 1987).

Clinical signs reported for secondary monocrotophos (an organophosphorous compound) poisoning in raptors include flaccid paralysis of legs and usually wings, while the head was held erect and the birds were alert. A pronounced bradycardia was present. Salivation, diarrhea, and miosis were not observed. Many birds were found lying on the ground below trees, presumably because roosting birds developed paralysis during the night and fell to the ground (Shlosberg, 1976). Butyrylcholinesterase (BChE; Enzyme Commission Number 3.1.1.8), also called pseudocholinesterase, is found in blood sera; although its physiological

function has not been well defined, it is a useful indicator of exposure to ChE inhibitors such as organophosphates and carbamates in the living bird (Sherman et al., 1964; Ludke et al., 1975) and can be used to confirm a diagnosis in a bird that shows the clinical signs of anti-ChE poisoning. It should be kept in mind that certain pathological conditions and heavy metals such as mercury and lead can also depress plasma BChE activity (Hill and Fleming, 1982).

A good response to administration of pralidoxime iodide (chloride)[d] (100 mg/kg), given by IM injection to raptors poisoned with organophosphorous compounds has been reported (Shlosberg, 1976). Administration of atropine (0.2-0.5 mg/kg) is indicated in poisoning with organophosphorous compounds and carbamates.

Strychnine is nearly always associated with deliberate poisoning. In the Netherlands, it is still available as a repellent for moles (*Talpa curepaea*) but is used illegally in eggs and other bait to destroy predators, including raptors. Strychnine has also caused mortality of bald eagles (*Haliaeetus leucocephalus*) and golden eagles (*Aquila chrysaetos*) (Cromartie et al., 1975; Kaiser et al., 1980; Redig et al., 1983; Reichel et al., 1984). Since the effect of primary strychnine poisoning is acute death, it is not very likely that cases of primary strychnine poisoning in wild raptors will be seen in clinical practice. Garlough and Ward (1932) have suggested that secondary poisoning with strychnine in raptors is very unlikely, but Redig et al. (1982) have reported secondary strychnine poisoning in gulls and owls. Strychnine causes convulsions through its direct action on the spinal cord. Death results from asphyxia due to prolonged spastic paralysis of respiratory muscles (Clarke and Clarke, 1967). Treatment should be aimed at controlling the convulsions. Administration of benzodiazepines has been suggested (Clarke and Clarke, 1967). External stimuli should be avoided, since these are known to induce convulsions. Removal of ingluvial and gastric contents, after general anesthesia has been induced, seems indicated. Potassium permanganate has been suggested for oxidizing the strychnine, and tannic acid to precipitate it (Clarke and Clarke, 1967). In dogs and cats the former is diluted 1:10,000 and given in a dose of 2 to 4 ml/kg orally (Clarke and Clarke, 1967). Strychnine is metabolized in the body within 10 hr (Clarke and Clarke, 1967).

α-Chloralose is well known for its use for the control of bird pests such as feral sparrows, feral pigeons, and crows (Murton et al., 1963; Thearle, 1965; Knapp and Russel, 1973; Lees, 1973). The Forestry Commission of the Netherlands uses it for the control of gulls.[1] Primary poisoning in raptors can occur after the ingestion of poisoned rodents or birds. Keymer et al. (1981) report possible chloralose poisoning in European kestrels in the United Kingdom. Poisoning of common buzzards by α-chloralose has previously been reported in the Netherlands (van Nie, 1975). α-Chloralose causes a profound narcosis, and affected birds are unable to maintain their body temperature. In a cold environment, narcotized birds will become hypothermic and die. The LD_{50} for rats is 400 mg/kg, while the LD_{50} for birds ranges from 32 to 180 mg/kg (Schafer, 1972). Treatment of birds poisoned with α-chloralose should consist of providing extra heat and supportive care to prevent dehydration.

Lead poisoning in raptors usually occurs after ingestion of prey or carrion containing lead shot (Lumeij et al., 1985). Bald eagle mortality in the United States has been associated with ingestion of waterfowl killed or crippled by hunters (Kaiser et al., 1980; Reichel et al., 1984). Lead poisoning was an important cause of mortality in free-living California condors (*Gymnogyps californianus*). In this species, ingestion of bullet fragments lodged in dead mule deer, coyotes, and California ground squirrels was the most likely source of the poison (Janssen et al., 1986; Wiemeyer et al., 1988).

Apart from the two cases reported in this chapter (Table 40.1), one more case of lead poisoning in a raptor from the Netherlands has been reported (Lumeij et al., 1985). Symptoms included behavioral changes; lethargy; anorexia; paralysis of the esophagus, proventriculus, or ventriculus; vomiting; diarrhea; ataxia; paralysis of the neck, legs, or wings; amaurosis; convulsions; anemia; edema of the head and nictitating membrane; and emaciation. A tentative diagnosis of lead poisoning can be made by demonstrating radiopaque densities suggestive for lead shot in the ventriculus, while a definitive diagnosis can be made by finding an elevated blood lead concentration, a reduced blood δ-aminolevulinic acid dehydratase activity, or increased blood protophorphyrin IX concentrations (Lumeij, 1975). Treatment of lead-poisoned raptors should consist of administering calcium disodium ethelyne diamine tetra-acetic acid[e] ($CaNa_2EDTA$) (20 mg/kg, IM, TID for 5 days) and, if possible, removing the lead from the ventriculus. In raptors that still show some appetite it might be helpful to give some extra casting material to clear the gizzard of lead. If this fails, an attempt can be made to remove the lead by aspiration with a stomach tube (Redig, 1980) or by using an endoscope with grasping forceps under fluoroscopic control (Lumeij et al., 1985). Gastrotomy should be used only as a last resort if lead pellets remain in the ventriculus after 5 days of medical treatment.

Although common buzzards and goshawks were most commonly poisoned during the years 1975 to 1988, their populations have been increasing considerably during the study period (SOVON, 1987; Bijlsma, 1989). The latter can be explained by an increase in prey populations and an increase in potential breeding habitat (Bijlsma, 1989).

One species, the red kite (*Milvus milvus*), seems to be especially vulnerable to poisoning (Table 40.1; 87% of car-

casses examined), since this species is notorious for eating carrion. Marked decreases in range and numbers of this species have been noted due to human persecution. Humans have been responsible for the extirpation of the red kite in large parts of Europe (Bijleveld, 1974). Since the species is limited to Europe and shows a continuous decline, it must be considered endangered (Bijleveld, 1974; Cramp et al., 1980). A number of poisoned red kites have been found with nearly fully developed eggs in the oviduct; therefore, it has been postulated that an increase of the breeding populations in the Netherlands (estimated to be three to five pairs) has been prevented by their illegal persecution (SOVON, 1987). The great number of red kites examined postmortem (31 in 13 years), despite such a small breeding population, can be explained by a larger number of migrating birds in the Netherlands.

The low incidence of poisoning in European sparrowhawks (0.5%) is striking when compared with other raptor species from the Netherlands. A high mortality (50% to 70%) of young sparrowhawks in the first year of life due to shortage of food seems to be normal (SOVON, 1987). An important differential diagnosis for a comatose sparrowhawk is trauma, since European sparrow hawks, because of their way of hunting, fly against objects regularly.

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Product List

[a]Azium, Schering Corp., Animal Health Division, Kenilworth, New Jersey 07033 USA

[b]Vicryl 3-0, Ethicon, 200 Norderstedt, Germany

[c]Model UCMK3, Euroglas BV, 2614 GS Delft, Netherlands

[d]2-PAM, Whitehall Laboratories, Inc. Division of American Home Products Corp., 685 Third Ave., New York, New York 10017 USA

[e]Calcium versanate, 3M Co., 225-5S 3M Center, St. Paul, Minnesota 55144 USA

Note

1. Personal communication with T. Smit, Central Veterinary Institute, Lelystad, Netherlands.

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CHAPTER 41

Pesticide Poisoning in Birds of Prey

STUART L. PORTER, V.D.M.

Wildlife Center of Virginia
Weyers Cave, Virginia, U.S.A.

ABSTRACT: The use of pesticides to enhance agricultural production adversely affects many species of wildlife, especially raptors. The principal compounds are organochlorines (DDT, endrin, chlordane, dieldrin, and heptachlor epoxide) and cholinesterase inhibitors of the chemical classes organophosphates and carbamates. Organochlorines are persistent in the environment and were responsible for eggshell thinning and subsequent population declines among bald eagles (*Haliaeetus leucocephalus*), ospreys (*Pandion haliaetus*), and peregrine falcons (*Falco peregrinus*) during the 1950s and 1960s. Cholinesterase inhibitors are short-lived in the environment, but highly toxic to birds. Raptors poisoned with these compounds exhibit a variety of central nervous system disorders that respond to treatment with atropine sulfate in many cases. The Environmental Protection Agency estimates that carbofuran, a carbamate compound, may be responsible for more than 2 million bird deaths a year in the United States. The effects of cholinesterase inhibitors on raptor populations have not been determined, and individuals involved with rehabilitation of these birds can play an important role in documenting the occurrence of these toxins in raptors.

KEY WORDS: pesticide, organochlorines, organophosphates, carbamates, poisoning, raptor, Virginia, cholinesterase inhibitors.

The products referred to as pesticides include herbicides, fungicides, rodenticides, avicides, and insecticides. There is little information available on the toxicity of herbicides, fungicides, and avicides to raptors. Some of the newer, more potent rodenticides, such as brodifacoum, have been reported to cause owl toxicity, but still little has been published (Hegdal and Colvin, 1988). The Environmental Protection Agency (EPA) has registered 45,000 pesticide products containing 600 active ingredients and 1,200 inert ingredients (Auerbach, 1988). Most of the pesticide problems reported in raptors are caused by insecticides (Newton, 1988; James and Fox, 1987; Henny et al., 1985, 1987; Franson et al., 1985; Stone et al., 1984; Stone, 1987; Stone and Okoniewski, 1988; Ludke et al., 1975; Mendelsohn and Paz, 1977; Blus et al., 1983; Balcomb, 1983; Stickel, 1973; Porter, 1987).

There are four main chemical classes of insecticides: organochlorines, organophosphates, carbamates, and pyrethroids. Only the pyrethroids are relatively nontoxic to vertebrates (Blodgett, 1988). The majority of the insecticides are used in agriculture and are sprayed or deposited as granules in the environment. Raptors are end-stage predators and are at risk for coming into contact with these products either by being sprayed directly or by eating contaminated prey such as insects and small vertebrates.

Insecticides can affect free-living wildlife in five basic ways: (1) by causing direct mortality and/or behavioral abnormalities, (2) by causing decreased reproduction, (3) through other sublethal effects, (4) by altering the habitat, and (5) by altering or decreasing the food supply. Most of the reported effects on raptors have been limited to direct mortality, behavioral abnormalities, and decreased reproduction (Newton, 1988; James and Fox, 1987; Henny et al., 1987; Franson et al., 1985).

This chapter discusses the different classes of insecticides and the effects of each on raptors, with emphasis on diagnosis and treatment.

Organochlorines

The organochlorines were the first insecticides to be widely used, starting in the 1940s with DDT. DDD and DDE are the main breakdown products of DDT (Stickel, 1973). DDE is more prevalent because it is more resistant to degradation in animals and nature (Stickel, 1973). DDE caused eggshell thinning and reproductive failure in a wide variety of avian species, including the osprey (*Pandion haliaetus*), peregrine falcon (*Falco peregrinus*), and bald eagle (*Haliaeetus leucocephalus*). The general use of DDT was severely restricted in 1972 by the EPA, and the populations of osprey and bald eagles have increased since then (Grier, 1982; Spitzer and Poole, 1980).

Unfortunately, DDT is still a threat to wildlife because of its persistence in the environment and its use in parts of Central and South America, where many raptors and their prey spend the winter. Other organochlorines were or are still in use (Table 41.1). Organochlorine insecticides are stored in body fat, and birds exposed sublethally may accumulate large amounts that can then be mobilized during times of food stress and result in poisoning (Stickel, 1973).

Table 41.1
Organochlorine Insecticides

Common name	*Trade name*
Aldrin	Aldrite
BHC	Benzahex
Chlordane	Velsicol
DDT	many exist
Dicofol	Kelthane
Dieldrin	Dieldrite
Endrin	Endrex
Heptachlor	Drinox-H34
Lindane (gamma BHC)	Lintox
Mirex	Dechlorane
Methoxychlor	Marlate
Toxaphene	Strobane T

Source: Bohmont (1983).

A number of studies have demonstrated that North American raptors still contain organochlorines in their bodies (Wiemeyer and Cromartie, 1981; Wiemeyer et al., 1984, 1987, 1988, 1989; Blus et al., 1989; Sundlof et al., 1986; Reichel et al., 1984; Stickel, 1973; Stickel et al., 1979, 1983, 1984; Pattee et al., 1985; Grier, 1982; Johnston, 1978; Kredl and Kiren, 1986; Peakall et al., 1975; Stone, 1981).

At the Wildlife Center of Virginia (WCV), two to four cases of "acute" organochlorine toxicity have been diagnosed annually since 1987. The affected birds rarely have had injuries and usually have been found on the ground away from roads and buildings. All have been emaciated and have exhibited tonic-clonic convulsions, especially when being handled. A normal pupillary reflex to light has been present. The birds have appeared blind, have exhibited abnormal posture, and usually could not stand. The complete blood count has been unremarkable except for low packed cell volumes (<15%) and very low total solid values (<1.0 gm/dl).

These birds have been treated with intravenous fluids, intraosseous blood transfusions, oral hyperalimentation formulas, vitamin B complex, and other supportive measures. Some researchers have recommended diazepam[a] or barbiturates to control the convulsions (see, e.g., Davis, 1982; Redig, 1992). There is no specific antidote for organochlorine toxicity. Of the cases presented since 1987, only one bird has survived; the others died 2 to 3 days after admission.

The definitive diagnosis of organochlorine poisoning is made by postmortem examination. Gross necropsy findings are nonspecific, except for emaciation. Histopathology is unremarkable. Tissues must be submitted for pesticide analysis by gas chromatography. Specific residue concentrations in the brain are diagnostic (Stickel, 1973). In the "normal" bird the highest levels will be found in body fat, although blood can also be analyzed (Wiemeyer et al., 1989).

Table 41.2
Organophosphorous Insecticides

Common name	*Trade name*
Acephate	Orthene
Amidithion	Thiocron
Azinphos-ethyl	Ethyl Guthion
Azinphos-methyl	Guthion
Bomyl	Bomyl
Bromophos	Nexion
Carbophenothion	Trithion
Chlorfenvinphos	Sapecron, Supona
Chlormephos	Dotan
Chlorpyrifos	Dursban, Lorsban
Coumaphos	Co-Ral
Crotoxyphos	Ciodrin
Crufomate	Ruelene
Cythioate	Cyflee
DDVP	Vapona
Dialifor	Torak
Diazinon	Spectracide
Dichlofenthion	Mobilawn
Dichlorvos	Vapona
Dimefox	Hanane
Dimethoate	Cygon, De-Fend
Dioxanthion	Delnav
Disulfoton	Di-Syston
EPN	EPN
Ethion	Ethanox
Ethoprop	Mocap
Etrimphos	Ekamet
Famphur	Warbex
Fenitrothion	Agrothion, Sumithion
Fensulfothion	Dansanit
Fenthion	Baytex, Tiguvon
Fonofos	Dyfonate
Iodofenphos	Nuvanol N
Isofenphos	Amaze
Leptophos	Velsichol
Malathion	Cythion
Mephosfolan	Cytrolane
Methamidophos	Monitor
Methidathion	Supracide
Methyl parathion	many
Mevinphos	Phosdrin
Monocrotophos	Azodrin
Naled	Dibrom
Omathoate	Folimat
Oxydemeton-methyl	Meta-Systox R
Parathion	many
Phenthoate	Cidial
Phorate	Thimet
Phosfolan	Cyolane
Phosmet	Imidan Prolate
Phosolone	Zolone
Phosphamidon	Dimecron
Phoxim	Baythion
Profenphos	Curacron
Quinalphos	Bayrusil
Schradan	OMPA
Sulfotep	Bladafume
Sulprofos	Bolstar

Temophos	Abate
TEPP	Vapatone
Terbufos	Counter
Tetrachlorvinphos	Gardona, Rabon
Thiometon	Ekatin
Trichlorfon	Dylox, Neguvon, Dipterex
Trichloronate	Agritox

Source: Bohmont (1983).

Interpretation of the levels found may be difficult, as toxic levels vary among species and there is evidence of synergism among different chemicals (Stickel, 1973). DDT and its degradation products DDD and DDE are best assessed together, using the formula ppm DDE/15 + ppm DDD/5 + ppm DDT = DDT equivalents (ppm in wet weight). Lethally poisoned birds have brain concentrations exceeding 20 DDT equivalents (Stickel, 1973; Stickel et al., 1984). Chlordane poisoning should be suspected whenever the sum of the brain levels of heptachlor epoxide and oxychlordane exceeds 4.0 ppm (Stickel, 1973). Brains of heptachlor-poisoned birds contain heptachlor epoxide concentrations exceeding 9.0 ppm (Stickel, 1973). Lethal concentrations of chlordane begin at 5.0 ppm (Stickel et al., 1983). Residues of 0.8 ppm of endrin in the brain cause death (Stickel et al., 1979). Dieldrin is considered toxic when brain levels exceed 3 to 4 ppm (Stickel, 1973).

Raptors tested at the Wildlife Center of Virginia have been found to have detectable levels of nine different organochlorines, most in small amounts. The highest levels have been found in the great horned owls (*Bubo virginianus*). One of these had 54.31 ppm of DDE in its fat after two years in captivity on a diet of laboratory rodents.

Although much less prevalent than it once was, organochlorine toxicity in raptors still occurs, and levels in the wild should continue to be monitored.

Organophosphorous Compounds

As organochlorines were phased out, they were replaced by the organophosphorous and carbamate pesticides. These neurotoxicants inactivate the enzyme acetylcholinesterase (AChE), which is responsible for the breakdown of acetylcholine, the neurotransmitter at cholinergic nerve endings and myoneural junctions (Meerdink, 1989). This enzyme inhibition allows the buildup of acetylcholine, which results in continued stimulation and fatigue of cholinergic end-organs and muscles (Meerdink, 1989). Death is due to respiratory failure. While these compounds are not as persistent in the host or the environment as organochlorines, they are more acutely toxic to mammals and especially to birds (see Table 41.4) (Stone, 1987; Rattner and Franson, 1983; Hill and Fleming, 1982).

There are 36 different organophosphates found in 2,972 different products on the market,[1] with uses ranging from nematicides to external parasiticides (Table 41.2). They come in spray, powder, and granular forms. Granular forms are readily consumed by and are often toxic to songbirds, which are then eaten by raptors (Balcomb et al., 1984). The effects of organophosphates on raptors have been well documented (Henny et al., 1985, 1987; Franson et al., 1985; Stone et al., 1984; Smith, 1987; Mendelsohn and Paz, 1977; Keymer et al., 1981; Hill and Fleming, 1982; Porter, 1987; Rattner and Franson, 1983; Stone and Okoniewski, 1988; Stone, 1987).

The clinical signs described for mammals, which resemble overstimulation of the parasympathetic nervous system (Meerdink, 1989), may not be seen in poisoned raptors (Porter, 1987). In three acutely poisoned bald eagles seen at the Wildlife Center of Virginia, the clinical signs exhibited were ataxia, inability to stand, opisthotonus, and a spastic nictitans. Other less obvious signs seen in red-tailed hawks (*Buteo jamaicensis*) included a detached attitude, inability to fly unrelated to any injury, and a full crop. Most of the affected birds were in good flesh. Some of these birds had other more obvious injuries, such as fractures. Other reported clinical signs include rigid paralysis with tightly clenched talons, rapid respiration, salivation, muscle twitching, alternating miosis and mydriasis, and absence of the pupillary light reflex (Balcomb, 1983; Henny et al., 1985, 1987; Shlosberg, 1976).

Some organophosphates, such as EPN and leptophos, have been reported to cause a delayed neuropathy syndrome that takes eight days to develop. A secondary metabolite has been demonstrated that affects peripheral axons and myelin sheaths, resulting in sensory and motor neuropathy (Meerdink, 1989). This syndrome has been produced experimentally in chickens,[2] but has not been reported in raptors.

Treatment is often initiated based on history and clinical signs and prior to a definitive diagnosis based on depressed plasma cholinesterase levels (Hill and Fleming, 1982; Hill, 1988; Ludke et al., 1975; Meerdink, 1989; Tharwat and Zinkl, 1985). Specific treatment includes atropine sulfate at 0.5 mg/kg, given intramuscularly, or one-fourth this dose given intravenously and the rest IM (Porter, 1987; Meerdink, 1989). Atropine has no effect on the insecticide enzyme bond, but rather blocks the muscarinic and some central nervous system effects at the nerve endings (Meerdink, 1989). If some reduction in the severity of clinical signs is not seen in 15 minutes, the atropine dose or the diagnosis should be reviewed (Meerdink, 1989). Diphenhydramine[b] at 4 mg/kg IM or orally has been shown to block the effects of the nicotine-receptor overstimulation and appears to enhance nerve function and prevent receptor paralysis (Meerdink, 1989). Its usefulness in birds has not yet been reported.

The use of pralidoxime chloride[c] is controversial. This drug, given at 20 mg/kg IM, will break the organophosphate-acetylcholinesterase bond, but it is most effective when administered within 24 hr of intoxication; an excess of the drug may itself inhibit the enzyme (Meerdink, 1989; Fikes, 1988). This drug was used successfully to treat a golden eagle (*Aquila chrysaetos*) at the WCV.

Supportive therapy is also very important, including oral, intraosseous, or intravenous fluids. If the crop is full, it should be emptied. If the bird has ingested the pesticide recently, activated charcoal may be useful. The birds should be kept in a warm, quiet place and given nutritional support until they start eating on their own. Birds that are treated usually survive, although their return to the wild may be delayed. The affected golden eagle noted above was released in two weeks, while an affected bald eagle needed 10 months to recuperate.

Organophosphate poisoning can be definitively diagnosed in the live bird either by measuring plasma or serum cholinesterase or by detecting the pesticide in the crop or gastrointestinal contents through gas chromatography (Hill and Fleming, 1982). Little has been published on normal serum cholinesterase values in raptors (Porter, 1987; Hill, 1988). Another problem in diagnostic testing is caused by an additional cholinesterase, butyrylcholinesterase, found in varying amounts in different species of birds. Butyrylcholinesterase constitutes only 3% of the total cholinesterase activity in the serum of the prairie falcon (*Falco mexicanus*), but makes up 88% of the total in the burrowing owl (*Speotyto cunicularia*). Although poisoning depresses both cholinesterases, different reagents used in the testing procedures will give different results (Hooper, 1988). Also, sample collection, storage, and the laboratory method used will affect the results (Fairbrother and Bennett, 1988; Hill, 1989; Hill and Fleming, 1982). Hemolysis, icterus, and lipemia in the sample will all result in elevated values (Hill, 1989).

Other factors may depress cholinesterase levels, including repeated freezing and thawing of samples or carcass, end-stage liver disease, and certain drugs with which wild raptors would not come into contact (Hill, 1989; Fairbrother and Bennett, 1988). However, plasma cholinesterase is stable if kept frozen (Hill, 1989).

At the WCV, heparinized plasma was analyzed with a colorimeter-based kit[d] and the colorimeter results reported in Rappaport units/ml (Ru/ml). A preliminary study done in 1987 on samples from 62 raptors, 52 of which were red-tailed hawks, revealed two groups: one returning values of 7 to 11 Ru/ml was considered abnormal and the other returning values of 17 to 26 Ru/ml was regarded as normal (Porter, 1987). Raptors with plasma cholinesterase levels below 17 Ru/ml were considered poisoned at the WCV (Porter, 1987). Outside laboratories report values in international units/l (iu/l) and micromole/ml/min, which are equivalent. A crude conversion factor is iu/l $\times$ 33.3 = Ru/ml.[3] Normal plasma cholinesterase values in red-tailed hawks (n = 9) were 0.566-1.009 micromoles/min/ml. A new, rapid, cholinesterase slide test[e] utilizing the Kodak Ektachem DT analyzer requires only 10 µl of plasma and the results are reported in 5 min. Preliminary work at the WCV suggests that values less than 0.9 units per ml are depressed. In the absence of known normal values, the suspect's values may be compared with those of a long-term captive.

The diagnosis of organophosphate poisoning in dead birds requires a relatively fresh carcass (Hill and Fleming, 1982; Hill 1988). Crop and gastrointestinal contents can be analyzed by gas chromatography for specific chemicals, or, more commonly, the brain is analyzed for cholinesterase levels. Normal values are not readily available for many species, so normal controls are often run at the same time (Hill and Fleming, 1982; Hill 1988). Brain cholinesterase levels depressed by 20% indicate exposure. Levels less than 50% of normal are diagnostic, and most poisoned birds have levels depressed by 70% (Fairbrother and Bennett, 1988; Hill, 1988; Hill and Fleming, 1982).

Carbamates

There are 46 different carbamate compounds available, found in 1,626 different products,[4] and their toxicity to birds varies (Tables 41.3 and 41.4). The carbamate insecticides also inhibit the enzyme acetylcholinesterase, resulting in the buildup of acetylcholine and the continued stimulation and fatigue of cholinergic end-organs and muscles (Meerdink, 1989). The major difference between organophosphates and carbamates is that the latter may separate from the enzyme, resulting in reactivation (Meerdink, 1989). The clinical signs, diagnosis, and treatment are the same as for organophosphate toxicity, except that pralidoxime chloride may not be useful (Meerdink, 1989; Porter, 1987). Differentiation between carbamate and organophosphate poisoning is difficult, but there is a 2-PAM reactivation technique that can be used to detect organophosphate poisoning in plasma or brain tissue on samples less than 48 hr postintoxication (Hooper, 1988).

Carbofuran, a commonly used carbamate, is extremely toxic to birds. The state of Virginia has banned the granular formulation of carbofuran, while the EPA has reached an agreement with the manufacturer to phase out the granular formulations nationwide over five years. This product has been linked to deaths in many bald eagles from Chesapeake Bay. An Endangered Species Impact Response by the U.S. Department of the Interior states that the recovery of this population will be slowed by the impact of this product (Larsen, 1987). An EPA special review team report states

Table 41.3
Carbamate Insecticides

Common name	*Trade name*
Aldicarb	Temik
Allyxcarb	Hydrol
Aminocarb	Matacil
Bendiocarb	Ficam
Bufencarb	Bux
Carbaryl	Sevin
Carbofuran	Furadan
Carbosulfan	Advantage
CPMC	Etrofol
Dimetilan	Snip
Ethiophencarp	Croneton
Mecarbam	Murfotox
Mercaptodimethur	Mesurol
Metalkamate	Bux
Methomyl	Lannate, Nudrin
Mexacarbate	Zectran
MIPC	Etrofolan
Pirimicarb	Pirimor
Promecarb	Carbamult
Propoxur	Baygon

Source: Bohmont (1983).

that granular carbofuran may be responsible for up to 2 million bird deaths annually in the United States. Most of these deaths involve songbirds, but secondary poisoning has been seen in many species of raptors, including the bald eagle.

Research from Saskatchewan has shown that aerial application of carbofuran to control grasshoppers resulted in a 54% decrease in the number of burrowing owl young per nest and a 50% decrease in the proportion of pairs that raised one or more young when the insecticide was applied within 400 meters of the nest burrow (James and Fox, 1987). This impact is thought to be a result of alterations in the parental behavior of the adult burrowing owls. Carbofuran may affect other insectivorous raptors where it is used in broad-scale applications.

Discussion

Birds of prey are still suffering mortalities and other effects from pesticides, although few direct mortalities that are caused by today's commonly used cholinesterase inhibitors have been documented in raptors. Most of the affected individuals die in the wild. In the period 1984-1991 there were 103 confirmed or suspected bald eagle poisonings by cholinesterase inhibitors diagnosed at the National Wildlife Health Research Center.[5] The drastic effects of the organochlorines on raptor reproduction have not been observed with today's cholinesterase inhibitors.

There are four critical aspects that impinge on our ability to develop a clear picture of the extent of the impact of cholinesterase inhibitors on raptors. First, there are many causes of neurological signs in wild raptors, including trauma, infectious agents, and other toxins. A thorough history, including where and under what circumstances the bird was recovered, along with a thorough medical workup, is required for accurate diagnosis. Second, assays to identify compounds causing pesticide intoxication can be performed only by specially equipped laboratories, and the costs of such analyses are high. A tissue screen for pesticides often costs more than $100 (U.S.). Third, there is a paucity of normal values and patterns of response to pesticides for enzymes in raptors. Most of the research has been conducted on mallard ducks (*Anas platyrhynchos*) and corturnix quail (*Colinus virginianus*). Finally, the sublethal effects of these pesticides on nontarget species is virtually unstudied, and the effects of the inert ingredients in these preparations have not been assessed even in humans, much less in wildlife species.

The documentation of large numbers of nontarget species being killed by the application of cholinesterase inhibitors, as well as the finding of raptors becoming secondarily poisoned, gives strong indication that much closer scrutiny of the regulations and application procedures for these compounds must be undertaken.

Table 41.4
Acute oral toxicities of carbamate and organophosphorous pesticides in the mallard (*Anas platyrhynchos*)

Chemical	LD_{50} *(mg/kg)*
Aldicarb	3.4
Baygon	11.9
Carbaryl	>2179
Carbofuran	0.4
DDVP	7.8
Demeton	7.2
Diazinon	3.5
Dicrotophos	4.2
Disulfoton	6.5
Ethoprop	12.6
Famphur	9.9
Fensulfothion	0.7
Fenthion	5.9
Fonophos	16.9
Malathion	1485
Methamidophos	8.5
Methidathion	23.6
Methiocarb	12.8
Methyl parathion	10.0
Mevinphos	4.6
Monocrotophos	4.8
Parathion	2.4
Phorate	2.6
Phosmet	1830
Phosphamidon	3.8
Trichlorfon	36.8

Sources: Buck and Osweiler (1976), Hudson et al. (1984).
Note: An $LD_{50} > 20$ mg/kg is considered highly toxic.

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Product List

[a]Valium, Roche, Division of Hoffman-La Roche Inc., 340 Kingsland St., Nutley, New Jersey 07110 USA

[b]Benadryl, Parke Davis, Division of Warner Lambert Co., 20 Tabor Rd., Morris Plains, New Jersey 07950 USA

[c]2-PAM, Whitehall Laboratories, Inc., Division of American Home Products Corp., 685 Third Ave., New York, New York 10017 USA

[d]Cholinesterase kit (catalog 420-CC), Sigma Chemical Co., P.O. Box 14508, St. Louis, Missouri 63178 USA

[e]Cholinesterase test, Eastman Kodak Co., 343 State St., Rochester, New York 14650-0001 USA

Notes

1. Personal communication with Arnold Aspelin, chief, Economic Analysis Branch, Office of Pesticide Programs, U.S. Environmental Protection Agency, Washington, D.C.

2. Personal communication with Dr. Marion Ehrich, Virginia Maryland Regional College of Veterinary Medicine, Virginia Tech, Blacksburg, Virginia.

3. Personal communication with Sigma Chemical Co. technical services personnel, St. Louis, Missouri.

4. See note 1.

5. Personal communication with Dr. Nancy Thomas, National Wildlife Health Research Center, Madison, Wisconsin.

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List of Editors and Primary Authors

Naomi L. Burtnick was board certified with the American Society of Clinical Pathologists as a medical technologist in 1975. Since then she has been with the cardiology section of the Veterinary Teaching Hospital of the University of Minnesota. Currently she holds a scientist position and is responsible for performing, interpreting, and teaching electrocardiography and echocardiography on both small and large animals.

Terry W. Campbell received a D.V.M. degree in 1979 and a Ph.D. in veterinary clinical pathology in 1987 from Kansas State University. From 1986 to 1989 he was an assistant professor in the Dept. of Surgery and Medicine at KSU, specializing in exotic and wildlife medicine. Since 1989 he has been staff veterinarian at Sea World of Florida. His book *Avian Hematology and Cytology* was published by Iowa State University Press in 1988.

Richard J. Cawthorn is professor of parasitology in the Faculty of Veterinary Medicine, University of Prince Edward Island. He has extensively studied the coccidial parasites of raptors, and continues a strong teaching commitment to wildlife parasitology. More recently, he has developed an extensive research program on developmental cycles, pathogenesis of infections, immunological responses, systematics, and epidemiology of apicomplexan parasites of livestock and economically important shellfish and finfish.

Susan B. Chaplin received her Ph.D. in physiology from Cornell University in 1973. Her research interests include physiological adjustments of birds to cold, water stress, nutritional stress, and physiological changes in birds following recovery from injury. She initiated studies of avian exercise physiology at the Raptor Center at the University of Minnesota while doing postdoctoral work in the Veterinary School there.

John E. Cooper graduated from Bristol University, U.K., in 1966. From 1967 to 1968 he studied tropical veterinary medicine at Edinburgh University. He spent five years in East Africa (Tanzania and Kenya) with both wild and domestic animals, and has recently returned to Tanzania as professor of veterinary medicine at Sokoine University of Agriculture. He is an honorary research fellow, University of Kent, U.K.; member of the Royal College of Pathologists, with specialist status in pathology from the Royal College of Veterinary Surgeons; and an elected fellow of the Institite of Biology. His professional interests include medicine and pathology of wildlife and captive exotic animals.

Laurel A. Degernes graduated from the University of Minnesota College of Veterinary Medicine in 1981 and worked in private practice for four years. From 1985 to 1990 she was staff veterinarian at the Raptor Center at the University of Minnesota, where she gained extensive experience in trauma medicine and surgery, especially in the areas of wound management and orthopedics. She completed a residency at the College of Veterinary Medicine at North Carolina State University, concentrating in other areas of avian medicine (primarily with psitticine species), and in 1992 became a clinical instructor in avian medicine at NCSU.

Guy Fitzgerald received his D.V.M. degree from the University of Montreal in 1987 and his M.Sc. degree on avian anesthesiology, isoflurane MAD in red-tailed hawks and pigeons, in 1990. He founded the Raptor Clinic at the University of Montreal in 1986 and the Provincial Raptor Rehabilitation Network (UQROP) in 1987. He is currently coordinating UQROP, which admits over 300 birds of prey of 26 different species annually.

Neil A. Forbes grew up with the benefit of a family interest in falconry and ornithology. In 1981, while still an undergraduate at the Royal Veterinary College, he met J. E. Cooper, and has had the opportunity to study under and work with Professor Cooper at that time and since. In recent years he has developed a busy first- and second-opinion avian practice, with particular emphasis on raptors.

Fritz Grimm is associate professor at the Institute for Bird Diseases of the University of Munich. He has been director of the avian clinic since 1980. He has published more than sixty manuscripts, and is coauthor of *Vogelkrankheiten* (Bird health) and *Altas der Röntgendiagnostik bei Heimtieren* (Atlas of radiographic diagnosis of small domestic animals).

Dieter Haas is a practicing physician and leader of the Diagnostik- und Therapiezentrum für Vögel gefährdeter Arten (Diagnostic and therapy center for birds of threatened and endangered species). He is also leader of the Stromtod-Dokumentationsstelle (Documentation place for electrocution of birds) of the Naturschutzbund Deutschland (NABU). He treats about 400 bird patients per year, mainly nondomestic large birds.

Trudi Hahn is a graduate of the University of Wisconsin-Oshkosh and has been a copy editor since 1971 for the *Minneapolis Tribune* and its successor, the *Star-Tribune*. Since 1990, she has been editorial assistant to Dr. Patrick Redig at The Raptor Center at the University of Minnesota, and in 1993 joined TRC's contract staff to present eagle education programs to large groups.

C. Stuart Houston is a professor of medical imaging at the University of Saskatchewan. His avocational interests include the history of ornithology, bird distribution, and reproductive biology of raptors. He has banded over 103,500 birds, with over 2,750 recoveries. He has published three books (*Birds of the Saskatchewan River, To the Arctic by Canoe, Arctic Ordeal*) and more than 200 natural-history papers. He has been the recipient of 10 conservation awards, including two national awards in Canada (Roland Michener Conservation Award in 1986 and Douglas J. Pimlott Conservation Award, jointly with his wife, Mary, in 1988) and the Eugene Eisenmann medal in 1990.

D. Bruce Hunter is an avian pathologist with the Dept. of Pathology, Ontario Veterinary College. He is coordinator of the clinical wildlife program and has a special interest in the pathology and epizootiology of diseases of wild birds, raptors in particular. His research interests include the pathophysiology of diseases of cardiovascular and respiratory systems of birds.

Gretchen E. Kaufman graduated from Tufts University School of Veterinary Medicine in 1986. She worked in a small animal practice in Boston until 1987, when she began a two-year residency in zoological medicine at the University of California at Davis. On completion of the residency she returned to Tufts to create and run the Exotic Animal Medicine Service in the small animal teaching hospital.

Robert E. Kenward graduated in zoology at Oxford University, and stayed at the Edward Grey Institute of Field Ornithology to complete a doctoral study of goshawk predation on wood pigeons. His postdoctoral work (with his coauthors in this volume, Vidar Marcström and Mats Karlbom) developed into a major radio-tagging study of goshawk population dynamics on Gotland, Sweden. Dr. Kenward now works on raptors and squirrels for the Institute of Terrestrial Ecology in Dorset, England, but has cooperative projects in North America and is a director-at-large of the Raptor Research Foundation.

Terry J. Kreeger is a wildlife veterinarian experienced with several predatory species. He has authored over 20 scientific articles and book chapters on chemical immobilization of wildlife. Currently he is the manager of pharmaceutical research and development for BallistiVet, Inc., a manufacturer of remote-delivery devices.

K. M. Langelier is a veterinarian practicing in Nanaimo, British Columbia. He is the past chairperson of the Wildlife Committee of the B.C. Veterinary Medical Association and was awarded the 1989 Canadian Veterinary Medical Association Humane Award for his wildlife work.

J. T. Lumeij graduated in 1979 from the Faculty of Veterinary Medicine of Utrecht University, where he subsequently completed an internship and a residency in companion animal internal medicine, followed by a residency in avian and exotic animal medicine. In 1987 he received his Ph.D. degree and in 1991 became a Diplomate Internal Medicine of Companion Animals, Royal Netherlands Veterinary Association. Since 1986 he has been the head of the Division of Avian and Exotic Animal Medicine of the Dept. of Clinical Sciences of Companion Animals at Utrecht University.

Howard D. Martin graduated from the University of California at Davis in 1982. He completed a small animal medicine and surgery internship, worked in private practice, and completed a residency in zoo and wildlife medicine at the University of Florida. He established the zoological medicine program at the College of Veterinary Medicine and Biomedical Sciences, Colorado State University, and directed and performed veterinary services for the Rocky Mountain Raptor Program at CSU. Currently he is in private practice.

Ian Newton is head of the avian ecology section at Monks Wood Experimental Station in England. He has studied birds for 25 years, and has published four books and more than 150 scientific papers. He is best known for his work on the population ecology of raptors.

J. Lindsay Oaks, Jr. has been a practicing falconer since 1973. He received a B.S. in biological sciences in 1982 and a D.V.M. in 1986, both from Colorado State University. For three years he worked in the United Arab Emirates at a private practice caring for falcons and horses. Currently he is pursuing a combined residency/Ph.D. program in clinical microbiology at Washington State University.

Mark A. Pokras received his B.S. in biology from Cornell University in 1972. He taught ornithology and marine biology at Stockton State College from 1974 through 1980. In 1984 he received his D.V.M. from Tufts University School of Veterinary Medicine, and later he returned to Tufts to complete a residency in wildlife and zoologic medicine under Dr. Charles Sedgwick. He is currently an assistant professor of wildlife medicine at Tufts, and his research interests include the surgical anatomy of birds and reptiles,

allometric scaling, and the biomedicine and environmental pathology of aquatic birds.

Stuart L. Porter is cofounder and director of veterinary services for the Wildlife Center of Virginia, a private, nonprofit teaching hospital for injured and orphaned wildlife. He is also professor of veterinary technology at Blue Ridge Community College in Weyers Cave, Virginia, and adjunct assistant professor at the University of Virginia, Dept. of Comparative Medicine, and at the Virginia-Maryland Regional College of Veterinary Medicine, Virginia Polytechnic Institute and State University.

Marc R. Raffe is associate professor of anesthesia/critical care at the College of Veterinary Medicine, University of Minnesota. He received his D.V.M. degree from the University of Illinois and his M.S. degree from Purdue University. He holds specialty certification in both anesthesia and emergency/critical care medicine boards.

Patrick T. Redig is the cofounder of the Raptor Center at the University of Minnesota and has served as its director since 1986. He has been involved with birds of prey as a falconer and veterinary clinician for over 30 years, and has treated over 6,000 birds since 1970. His research interests revolve around finding ways to treat and prevent aspergillosis and developing orthopedic techniques for use in raptors. He is the author of more than 75 papers, book chapters, and abstracts dealing with raptors. His goals include training veterinary professionals as well as lay individuals in the care, management, and conservation of raptors.

J. David Remple earned a B.A. in zoology from the University of Colorado and a D.V.M. from Colorado State University. He has been a falconer for 40 years, and while in private practice conducted extensive raptor rehabilitation programs in conjunction with state and federal agencies. He was medical consultant to the Peregrine Fund, Inc., for 10 years. He has authored several manuscripts on raptor diseases and is writing a book on falconry and birds of prey in the Arabian Gulf. In 1983 he became the founding veterinarian of the Dubai Falcon Hospital, the first medical facility dedicated solely to the care of hunting falcons. He is currently director of that facility, which examines thousands of falcons annually and conducts research into improved methods of diagnosis, treatment, and prevention for raptor diseases.

Kenton E. Riddle is the director of the Abu Dhabi Falcon Research Hospital, Abu Dhabi, United Arab Emirates. Since 1985 he has designed and managed a unique falcon research hospital for the ruling family of Abu Dhabi. He is also an adjunct associate professor of veterinary medicine and surgery at the University of Texas System Cancer Center. His background includes 10 years of raptor fieldwork studies in Greenland, Alaska, and the Texas gulf coast; peregrine falcon migration, banding, radio-tagging, and toxicology studies; and research specialties in nonhuman primates and Great Ape medicine and surgery. He is a master falconer with 40 years of experience.

Richard Rushton received his D.V.M. degree and worked in small animal veterinary medicine, focusing on raptor rehabilitation. He has been a volunteer raptor biologist at the Snake River Birds of Prey Natural Area near Boise, Idaho. His current position with Mini-Mitter Co., Inc., allows opportunities to work with others in designing research studies using biotelemetry.

Stephen A. Smith earned his B.S., M.S., and D.V.M. degrees at Ohio State University, where he organized and subsequently directed the OSU Raptor Rehabilitation Program from 1983 to 1986. He earned his Ph.D. from North Carolina State University in 1990. He has published numerous articles on the radiographic and gross anatomy of pet and wild birds, and is coauthor of *Atlas of Avian Radiographic Anatomy*. He is presently on the faculty of the Virginia-Maryland Regional College of Veterinary Medicine.

David L. Suarez received his D.V.M. from Auburn University and conducted the clinical work for the chapter in this volume in association with the Southeastern Raptor Rehabilitation Center at the College of Veterinary Medicine at Auburn University. He worked for three years in a small animal/exotic practice in Anniston, Alabama, and currently is pursuing a Ph.D. in veterinary microbiology at Iowa State University.

Colette L. Wheler received her D.V.M. from the Western College of Veterinary Medicine in Saskatoon, Saskatchewan, in 1984. After three years in private practice, she returned to this school to obtain her M.Sc. degree, concurrent with a residency in avian and caged pet medicine. Currently she is a clinical associate in avian and caged pet medicine and supervisor of the students' raptor rehabilitation facility at the Western College of Veterinary Medicine.

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Compiled by Dr. Patrick T. Redig and Trudi Hahn