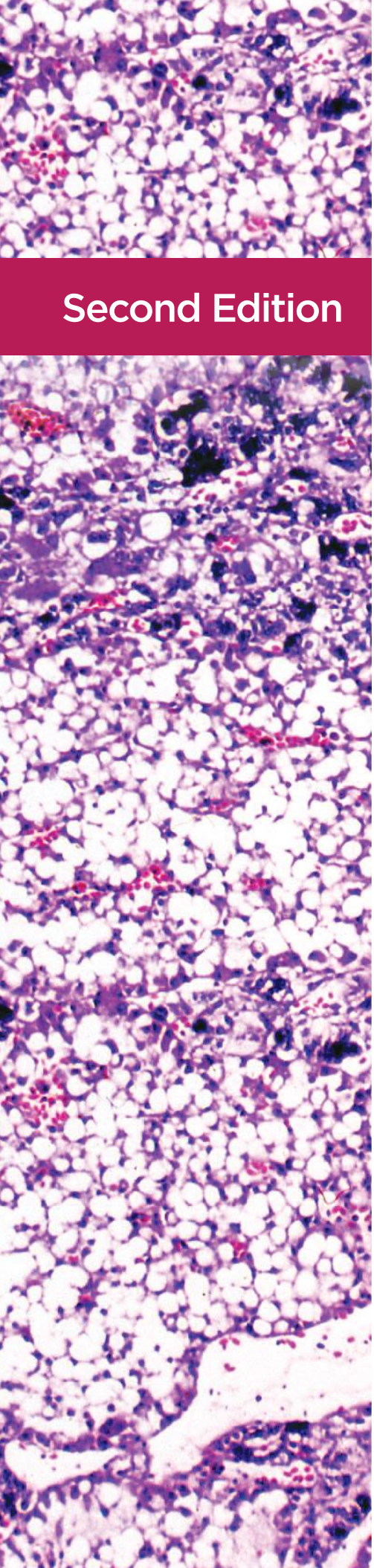
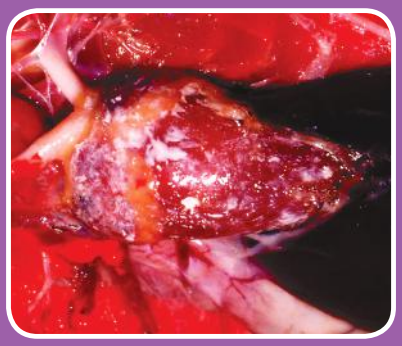
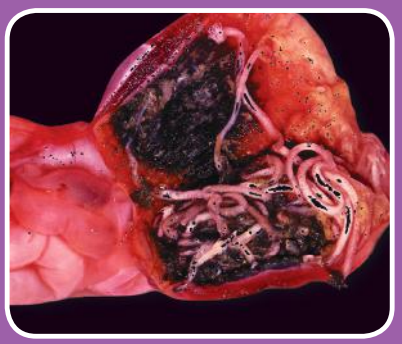


Second Edition

# Pathology of Pet and Aviary Birds


Robert E. Schmidt
Drury R. Reavill
David N. Phalen

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# Pathology of Pet and Aviary Birds

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## Second Edition

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**Robert E. Schmidt**, DVM, PhD, is a veterinary pathologist and consultant for Zoo/Exotic Pathology Service, Citrus Heights, California. He received his BS and DVM degrees from the University of California, Davis; his MS in veterinary pathology from Michigan State University; and his PhD in veterinary comparative pathology from Oklahoma State University. He is a Diplomate of the American College of Veterinary Pathologists and has been involved in diagnostic pathology of pet and exotic birds for more than 45 years. His professional affiliations include the Association of Avian Veterinarians, Exotic Mammal Veterinarians, The American Veterinary Medical Association, and the American Association for the Advancement of Science. He is a recipient of the Avian Pathology Award from the Association of Avian Veterinarians.

**Drury R. Reavill**, DVM, is a 1986 graduate of the College of Veterinary Medicine, Colorado State University. She is a Diplomate of the American Board of Veterinary Practitioners (Avian and Reptile and Amphibian Practice) and Diplomate of the American College of Veterinary Pathologists. Dr. Reavill has 28 years of experience in avian and exotic animal clinical medicine, clinical laboratory diagnostics, and exotic animal pathology. She currently owns and operates Zoo/Exotic Pathology Service and is a consultant for Veterinary Information Network (VIN) in fish medicine. She has received the VIN Special Service Award 1996, 2000, and 2004; the Association of Reptilian and Amphibian Veterinarians Presidential Service Award 2011; and Association of Exotic Mammal Veterinarians President's Award 2014.

**David N. Phalen**, DVM, PhD, earned his BA at the University of Chicago, his DVM from Cornell University and his PhD from Texas A&M University. He has been an avian practitioner for 32 years and is a Diplomate of the American Board of Veterinary Practitioners in avian practice. Dr. Phalen has spent the last 27 years studying the epizootiology, diagnosis, and control of diseases of aviary, companion, and wild birds and has published extensively in this field. He is currently an Associate Professor in the Faculty of Veterinary Science at the University of Sydney. He has received the Excellence in Avian Research Award from the American Veterinary Medical Foundation and was awarded the TJ Lafeber Practitioner of the Year in 2009.

# Preface to the First Edition

The number of birds in captivity, as pets and breeders, and in ornamental and zoological collections has increased dramatically in the past 30 years. In many cases, wild populations of some of these species are threatened or have disappeared entirely, leaving the survival of the species to captive-bird breeding programs. With the growth in the bird-owning public has come a commensurate growth in the number of veterinarians providing care for birds and an enormous increase in the knowledge of the husbandry and diseases of these birds, including several comprehensive textbooks of avian medicine and surgery. Since birds are now common mainstream pets, there is also a need for diagnostic veterinary pathologists to be familiar with the diseases of these species.

The necropsy and related diagnostic services are an integral part of avian medicine. Both private and public collections are often large and closely housed. The death of a bird may be the first indication of a serious infectious disease, nutritional disease, or other management-related problem. Avian veterinarians and bird owners depend on pathologists to make an accurate diagnosis and provide advice on the significance of their findings.

Diseases of pet and aviary birds differ significantly from those of poultry. They also differ from many of the common diseases seen in wild birds, even wild birds of the same species. Much of the literature on the disease of pet and aviary birds is widely scattered in individual articles and in proceedings that most pathologists would not routinely review. Additionally, much information has never been published in any form. The goals of this book are to bring together in one volume a comprehensive review of the gross and histologic features of the diseases of pet and aviary birds and to provide a guide to ancillary diagnostics and a context in which to interpret the pathologic findings. While we feel this book will be a valuable reference for practitioners and students of avian medicine, helping them to understand the pathogenesis of the clinical manifestations of disease.

We have organized this material in a systemic format, so that pathologists faced with a diagnostic challenge involving a particular organ can hopefully go to the appropriate chapter rather than having to search through extraneous listings under etiology or by bird species.

For the most part, this book deals with diseases of common, and a few uncommon, pet birds. However, the authors have also included material relating to other avian species that private practitioners and pathologists might occasionally be expected to encounter.

# Preface to the Second Edition

Eleven years have passed since the first edition of this book was published. During that time there have been many exciting advances in the fields of avian pathology and the medicine of pet and aviary birds. Additionally, the nature of avian medicine has changed. Veterinarians are now likely to be treating pigeons, backyard chickens, and other species of poultry, as well as, traditionally kept pet bird species. Veterinarians are also more likely to be treating birds with diseases associated with aging. The role and importance of the veterinarian in regard to aviculture continues. In general, the avian species that we now have in captivity cannot be replaced by birds from the wild so that maintaining their health and maximizing their breeding success is essential. Increasingly, captive breeding is also the last line of defense against extinction requiring significant veterinary input to maintain the health of small numbers of vulnerable birds.

Tissue biopsies and postmortem examination are an integral part of avian medicine. Biopsies inform treatment options and prognosis. Gross and microscopic postmortem assessments are essential if the impact of disease and inappropriate management practices are to be minimized. The second edition of *Pathology of Pet and Aviary Birds* is designed to assist the modern avian veterinarian and the avian pathologist so that they can maximize the information that they obtain from tissue biopsies and post mortem examinations. To this end the number of illustrations is increased and the figures are in color. The written content is also greatly expanded. These changes will allow practicing veterinarians and the avian pathologists in identifying the common and not-so-common diseases in the case material presented to them and understand the pathogenesis and epizootiology of the diseases they identify across a wide range of species.

# Acknowledgments

The authors thank the many veterinarians who have contributed material that has led to this book. In particular, we would like to thank the following for the contribution of photos: Drs. Kristin Alhgrim, Duane Belote, Jennifer Blair, Scott Ford, Alan Fudge, Chris Griffin, Gregg Harrison, Irv Ingram, Isabella Langlois, Teresa Lightfoot, Douglas Mader, Tracy McNamara, Michael Murry, Chiara Palmieri, Brian Speer, Rhoda Stevenson, and Colin Walker.

# 1 Cardiovascular System

## Normal structure

The bird's heart sits squarely in the middle of the coelomic cavity just caudal to the thoracic inlet. The axis of a normal heart deviates only slightly from the midline. Enlargement of any of the chambers may result in a change in the heart axis. The cranial ventral surface of the heart is in contact with the sternum, and the liver lobes cover the apex of the ventral surface.

The thin-walled atria have a scalloped surface and margins and are symmetrically located at the base of the heart. The right atrium is somewhat larger than the left. The right atrioventricular (AV) valve is a single muscular flap and is not membranous. The right ventricular free wall wraps around the heart from the caudal right lateral aspect of the heart to the cranial ventral surface of the heart. The wall of the right ventricle is approximately one-third to one-half the thickness of the interventricular septum and the free wall of the ventricle. This ratio, however, varies to some degree with the species, between individuals within species, and also varies depending on what level of the heart the measurements are taken.

The pulmonary and aortic values are essentially the same as those found in mammals. The left AV valve is membranous but is a continuous sheet and does not have clearly defined cusps. The valve is connected to papillary muscles by chordae tendineae. The brachiocephalic trunks immediately branch off the aorta as it leaves the heart. The first arteries to leave the brachiocephalic trunks are the carotids, which are relatively thin walled and narrow. The aorta arches to the right in the bird, as opposed to the left in mammals. Birds have a larger heart compared with body mass than do mammals. Myocytes have a smaller diameter (approximately one-fifth to one-tenth) than those found in mammalian hearts and a more rapid depolarization leading to a faster heart rate and relatively greater cardiac output. Purkinje fibers of the conduction system are relatively large as compared to those found in mammals.

## Congenital anomalies

Most of the literature on avian heart anomalies concern chickens. Congenital lesions in pet birds are rarely described. Ventricular septal defects appear to be relatively common in umbrella cockatoos, and one of the authors (D.N.P.) has also seen them in cockatiels and an African grey parrot. The defects between the ventricles are typically 1–3 mm in diameter and are located in the interventricular septum just below the pulmonary and aortic valves (Fig. 1.1). Right- and left-sided heart failure typically develops in these birds between 1 and 3 years of age. Dilation of both ventricles is common, and the pulmonary veins are markedly distended (Fig. 1.2). Perihepatic effusion and cirrhosis of the liver with dilation of the hepatic veins may be present secondary to right-heart failure. Interventricular septal defects have also been associated with a truncus ateriosis in an umbrella cockatoo and aortic hypoplasia in a Moluccan cockatoo (*Cacatua moluccensis*).

Congenital aneurysms of the left ventricle are uncommon. One of us (D.N.P.) has seen several of these in cockatiels. All were small, typically 2–4 mm in diameter. A large left ventricular aneurysm (2 cm in diameter) was found in a mature blue and gold macaw. All of these emanate from the apex of the heart. There was no other evidence of heart disease in these birds and the lesion was not thought to impact the heart function.

An epicardial keratinaceous cyst presented as a yellow nodule containing caseous material. Histologically it was lined by stratified squamous epithelium, and the grossly noted material was laminated keratin. Based on the gross appearance, the differential diagnosis for this type of lesion would be an abscess. We have seen an African grey parrot with a focus of capillary proliferation in the myocardium (Fig. 1.3) that was considered to be congenital telangiectasis or possibly an example of a hamartoma.

In chickens, cardiac anomalies are thought to be associated with stress during organogenesis, including increased temperature and hypoxia. Vitamin deficiencies may also be responsible for these malformations in chickens. Aortic anomalies are reported in chickens and have been associated with excessively high or low humidity during incubation. Given that ventricular septal defects are seen most frequently in umbrella cockatoos, a genetic defect may be to blame for this anomaly in this species.

## Pericardial disease

Pericardial lesions can be a manifestation of infectious, noninfectious, or neoplastic diseases.

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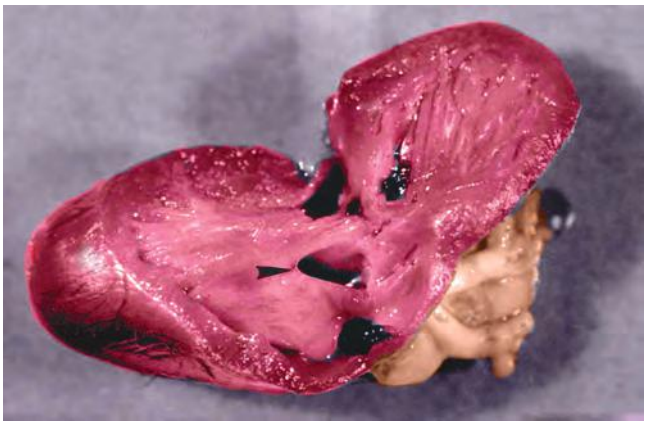

Figure 1.1 Interventricular septal defect (arrowhead).

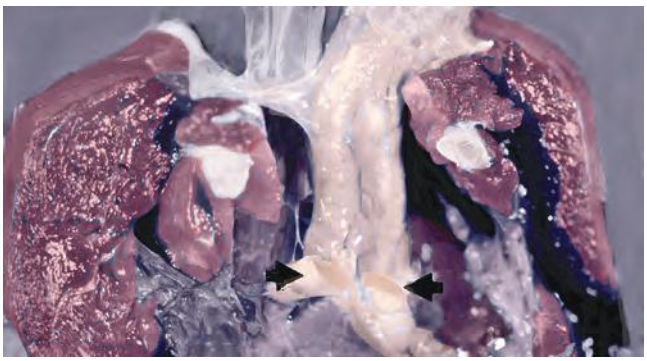

Figure 1.2 Marked distension of pulmonary veins (arrows) secondary to right-sided and left-sided failure in a bird with an interventricular septal defect.

## Infectious disease

Infectious disease of the pericardium can be localized to the pericardium or may be just one manifestation of a systemic disease. A variety of organisms have been found to cause pericarditis, including numerous bacteria, including members of the Enterobacteriaceae, Mycobacteria, and *Chlamydia psittaci*, fungi and, occasionally, avian polyomavirus.

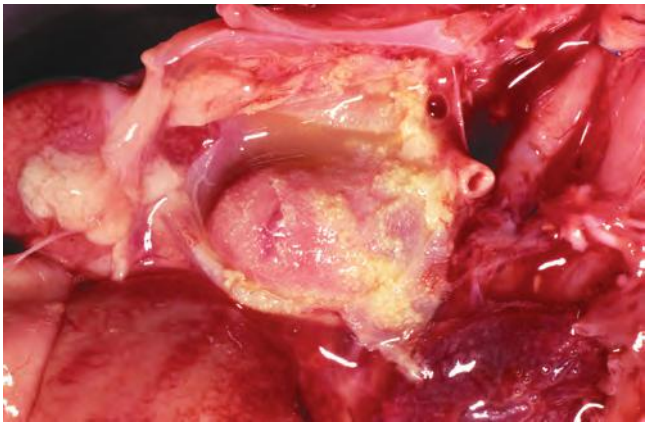

Figure 1.4 Epicarditis and pericarditis due to a systemic infection by *Chlamydia*. Grossly differential diagnoses include a variety of bacterial infections.

Pericarditis causes the pericardium to be variably thickened and gray to yellow-white, with red foci seen occasionally. The pericardium may have a shaggy appearance. In less severe cases, multifocal plaques are seen. There may be adhesions to the epicardium (Fig. 1.4). Pericardial fluid is increased, gray-yellow, and cloudy and may be flocculent. Histologically, bacterial and fungal infections cause edema, fibrin deposition, and an initial purulent response containing numerous heterophils and macrophages. Relatively more lymphocytes and plasma cells may be found in fungal infections. The pericardium may be adhered to the epicardium (Fig. 1.5).

With chronicity, there can be abscess formation. Macrophages and possibly giant cells as well as a more pleocellular response surround a central necrotic area. In both acute and chronic conditions, specificity depends on finding organisms that may be present.

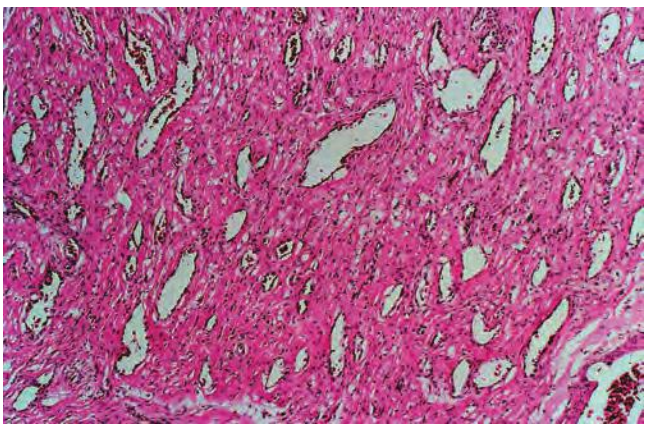

Figure 1.3 Congenital myocardial lesion comprised of irregular, dilated vascular channels.

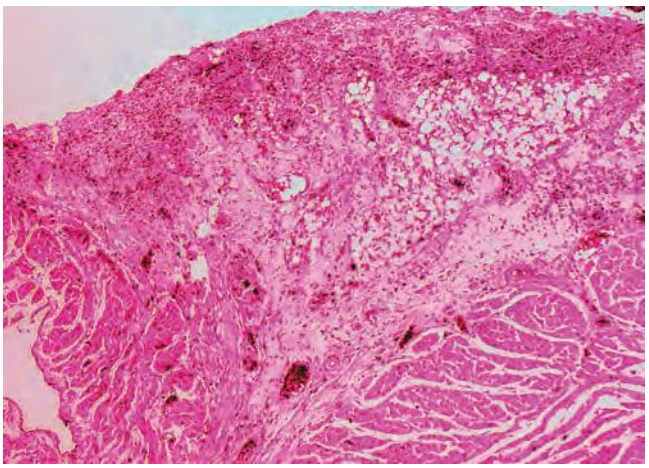

Figure 1.5 Chronic pericarditis/epicarditis. Note the diffuse inflammatory reaction and adherence of the pericardial tissue to the epicardium.

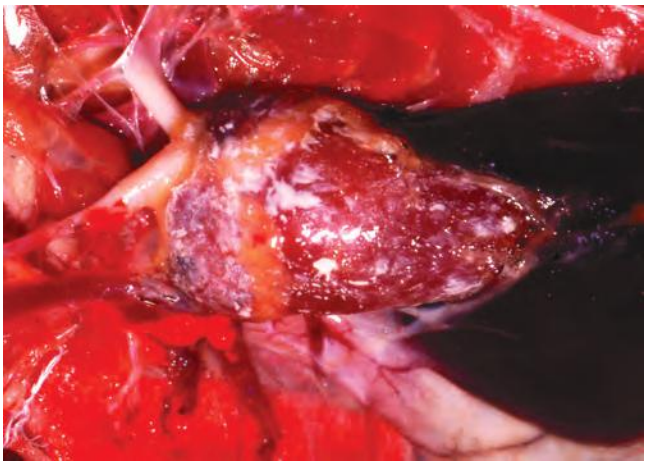

Figure 1.6 Severe pericardial and epicardial urate deposition. The lesion must be differentiated from infection.

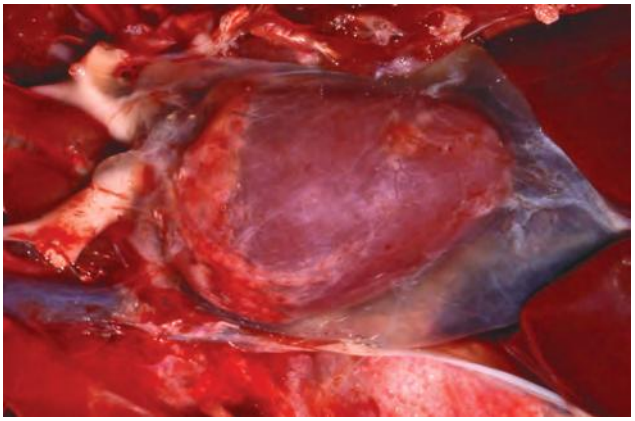

Figure 1.7 A large amount of partially coagulated proteinaceous fluid in the pericardial sac leading to cardiac tamponade. The fluid accumulation can be due to a number of causes.

Mycobacterial infections usually present grossly as large irregular masses that can mimic neoplasia. They are relatively firm, gray-white, and most often near the heart base. Early mycobacterial infections elicit a response of heterophils and macrophages. Organisms may be present infrequently. In advanced mycobacterial disease, the response will be primarily large macrophages with abundant light basophilic cytoplasm. Organisms can be seen within the cytoplasm with acid-fast stains.

### Noninfectious disease

The pericardium is a common site of visceral urate deposition (gout). Grossly the lesion can be similar to an infectious pericarditis, with a thickened membrane containing gray-white plaques. However, pericardial thickenings associated with gout are typically white, smooth, and shiny as opposed to the yellowish, roughened, and dull exudates seen in infectious conditions. Flocculent material, along with an excess of turbid fluid, may be present in the pericardial sac (Fig. 1.6).

Histologically, urates may be crystalline or amorphous and are lightly basophilic on hematoxylin-eosin stains. Although the crystals dissolve in formalin, the remaining characteristic needle-shaped spaces can be found in most cases. Alcohol fixation and special staining can be used if there is any doubt that the lesion is gout. Depending on the duration of the urate deposition, there will be an inflammatory response comprised primarily of heterophils. Focal necrosis may also be seen.

### Neoplastic disease

In mammals, sarcomas and mesothelioma have been reported in the pericardium. Primary pericardial tumors are not documented in pet birds, and we have not seen any examples of them.

### Pericardial effusion

Effusion may accompany primary heart and pericardial diseases, as already discussed, and may be a part of systemic problems, including anything leading to right-sided heart failure or hypoproteinemia. Effusions may be transudates, modified transudates, exudates, or hemorrhage. The gross appearance will depend on the composition of the fluid. Within several hours of death, high-protein effusions will often become gel-like. In some instances the amount of pericardial fluid may be massive (Fig. 1.7).

## Heart disease

Diseases of the heart can be divided into traumatic inflammatory, noninflammatory, and neoplastic. Infectious disease can be further divided into viral, bacterial, mycobacterial, fungal, and protozoal infections. Most diseases of the heart are confined to the myocardium, but, less commonly, lesions can also be seen in the epicardium and endocardium.

### Trauma

Traumatic injuries to the heart are rare in cage birds, but extremely common in wild birds. Bruising of the myocardium is very common in birds that have been hit by cars or have had other blunt force trauma. Infrequently an atrium will be ruptured as the result of a proximal oblique coracoid fracture. Atrial rupture generally leads to a fatal bleed.

### Infectious disease

Several viruses are known to cause myocardial lesions in pet birds. Polyomavirus is seen in a variety of psittacine birds and can also cause heart disease in finches. In budgerigars, gross lesions include hydropericardium, cardiomegaly, and hemorrhage. The myocardium may have patchy pale areas. Histologically there is coagulative myofiber necrosis and variable nonsuppurative inflammation and hemorrhage. There may be karyomegaly of myocyte nuclei, with margination of chromatin and inclusion body formation. Polyomavirus inclusions are usually pale or almost clear, or granular and basophilic.

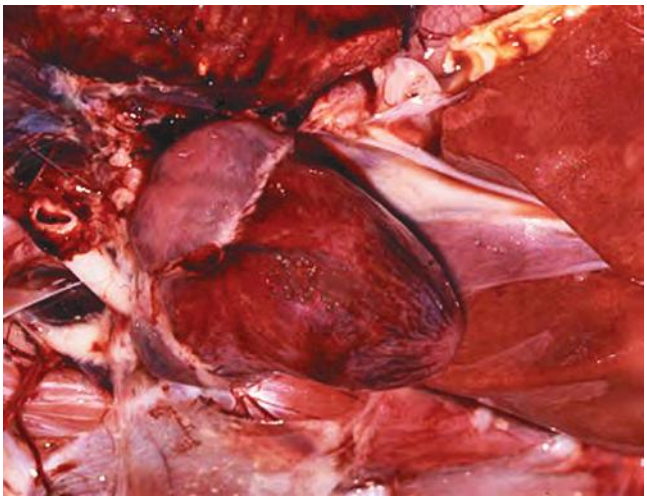

Figure 1.8 Polyomavirus infection causing patchy epicardial and myocardial hemorrhage.

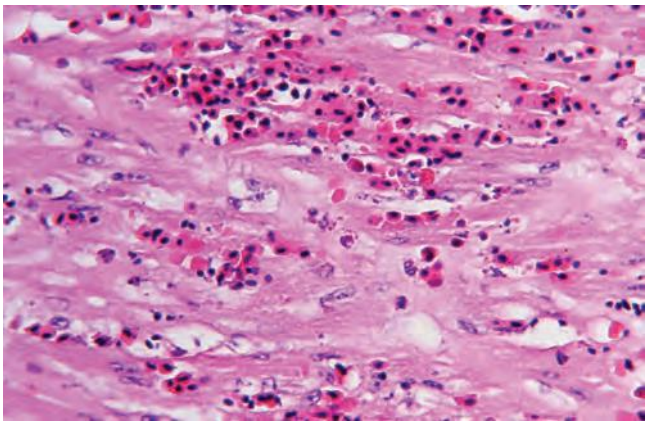

Figure 1.10 Severe myocardial degeneration and hemorrhage due to polyomavirus infection.

In nonbudgerigar psittacines, gross and histologic lesions vary somewhat from those seen in budgerigars (Figs. 1.8, 1.9, 1.10, and 1.11). Hemorrhage is a much more prominent feature of this disease and can be seen in subcutaneous tissues and serosal surfaces. Petechial and ecchymotic hemorrhages are often present on the surface of the epicardium. As the result of blood loss, birds are very pale and their muscles exhibit an unusual orange hue. If there is an inflammatory reaction, it is primarily lymphoplasmacytic. In finches, necrosis, inflammation, and inclusion bodies have been reported.

Avian Bornavirus infection resulting in proventricular dilatation disease affects a wide variety of psittacine and nonpsittacine birds, and heart lesions are relatively common. Grossly there may be slight dilatation of the ventricles, and occasional pale foci and streaks are seen (Fig. 1.12). Histologically, multifocal

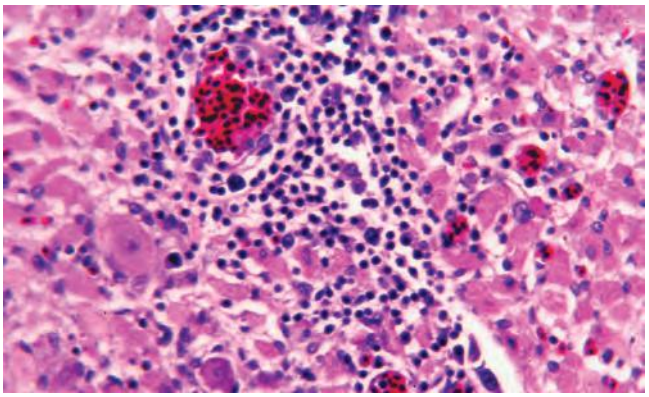

Figure 1.11 Nonsuppurative myocarditis in a bird with polyomavirus infection. Inflammation is seen infrequently in the heart in this disease.

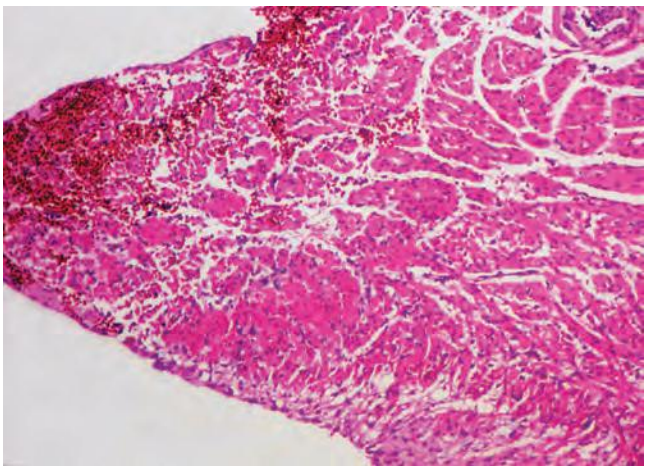

Figure 1.9 Focus of epicardial and myocardial hemorrhage in a bird with polyomavirus infection.

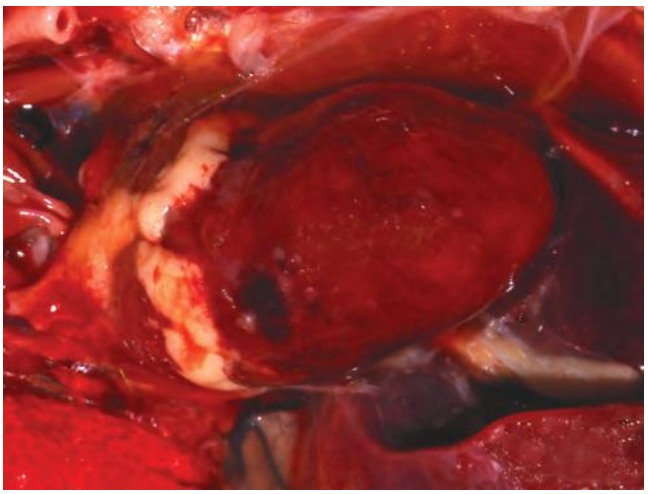

Figure 1.12 Foci of discoloration in the myocardium and asymmetrical dilatation of the ventricles of the heart of a bird with proventricular dilatation disease (Bornavirus infection). A focus of agonal hemorrhage is also seen, but hemorrhage is not a typical feature of this condition.