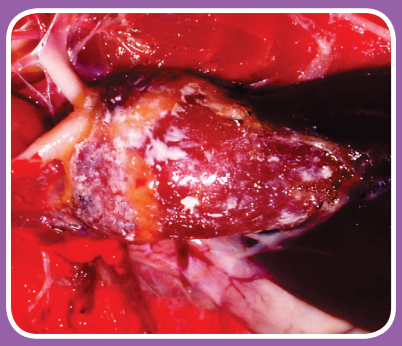
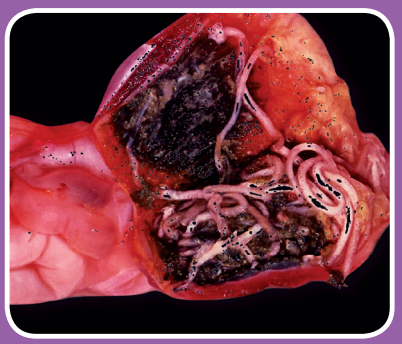
Second Edition

Pathology of Pet and Aviary Birds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WILEY Blackwell

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The Atrium, Southern Gate, Chichester, West Sussex, PO19 8SQ, UK
9600 Garsington Road, Oxford, OX4 2DQ, UK

Library of Congress Cataloging-in-Publication Data

Schmidt, Robert E. (Robert Eugene), 1939-, author.
Pathology of pet and aviary birds / Robert E. Schmidt, DVM, PhD, Zoo/Exotic Pathology Service, Citrus Heights, California, Drury R. Reavill, DVM, Zoo/Exotic Pathology Service, Citrus Heights, California, David N. Phalen, DVM, PhD, Faculty of Veterinary Science, University of Sydney, Australia. – 2nd edition.
p. ; cm
Includes bibliographical references and index.
ISBN 978-1-118-82809-0 (cloth)
1. Birds–Diseases. I. Reavill, Drury R., author. II. Phalen, David N., author. III. Title.
[DNLM: 1. Bird Diseases–pathology. SF 994]
SF994.S36 2016
636.5′0896–dc23 2015014448

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

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

Set in 9.5/12pt MinionPro by Aptara Inc., New Delhi, India

Printed in Singapore

1 2015

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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.

Pathology of Pet and Aviary Birds, Second Edition. Robert E. Schmidt, Drury R. Reavill and David N. Phalen.

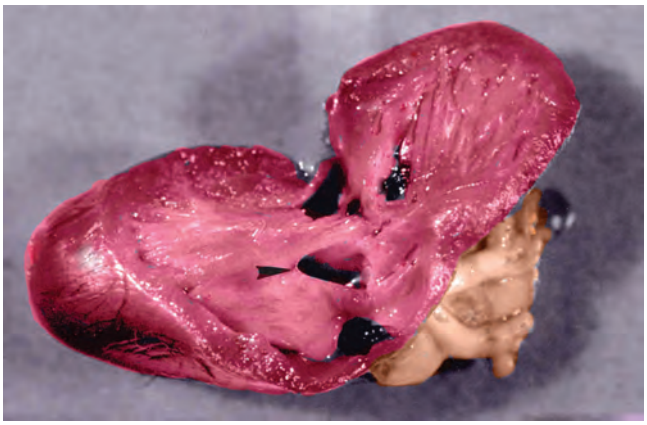

Figure 1.1 Interventricular septal defect (arrowhead).

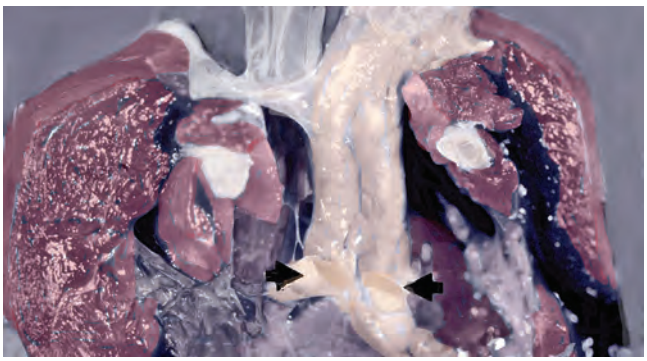

Figure 1.2 Marked distension of pulmonary veins (arrows) secondary to right-sided and left-sided failure in a bird with an interventricular septal defect.

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.

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.

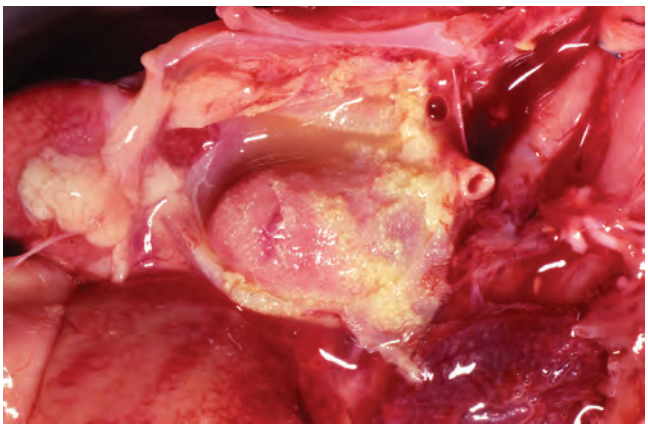

Figure 1.4 Epicarditis and pericarditis due to a systemic infection by *Chlamydia*. Grossly differential diagnoses include a variety of bacterial infections.

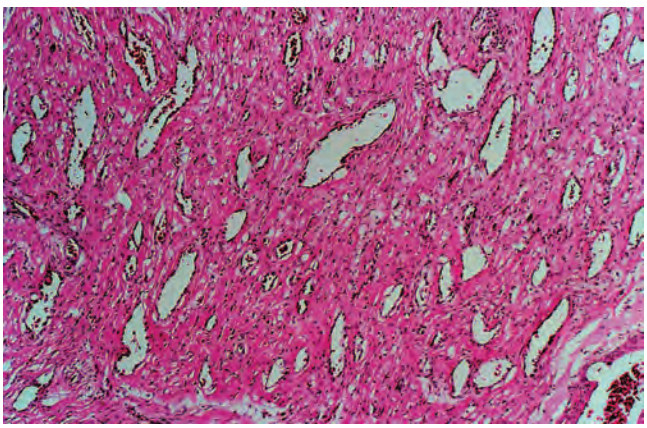

Figure 1.3 Congenital myocardial lesion comprised of irregular, dilated vascular channels.

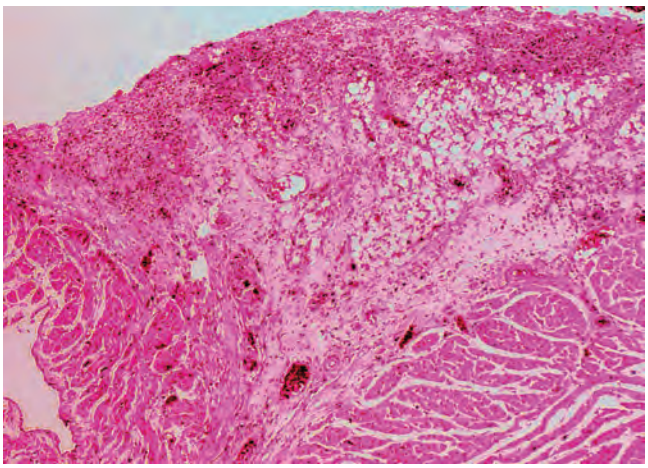

Figure 1.5 Chronic pericarditis/epicarditis. Note the diffuse inflammatory reaction and adherence of the pericardial tissue to the epicardium.

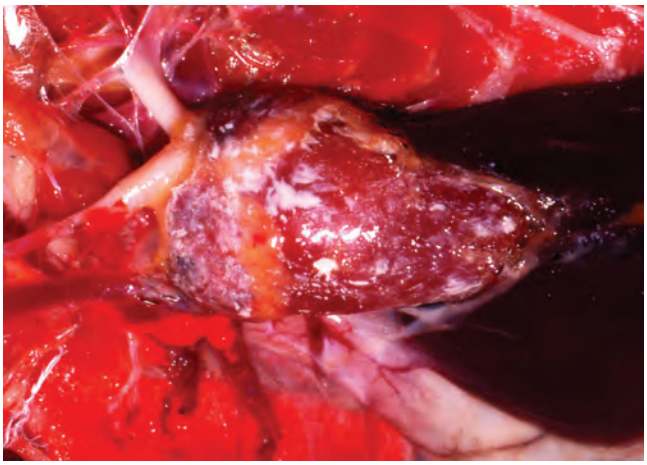

Figure 1.6 Severe pericardial and epicardial urate deposition. The lesion must be differentiated from infection.

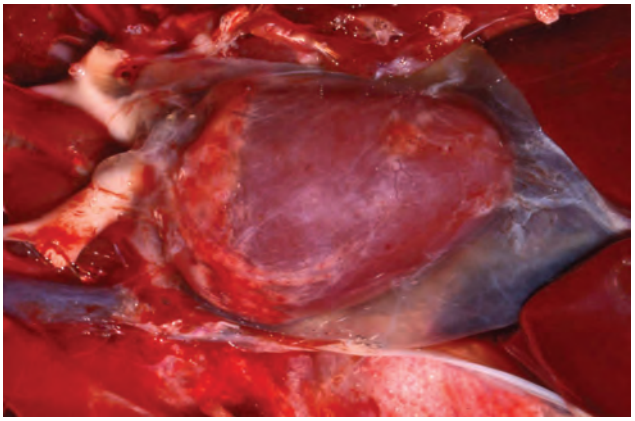

Figure 1.7 A large amount of partially coagulated proteinaceous fluid in the pericardial sac leading to cardiac tamponade. The fluid accumulation can be due to a number of causes.

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.

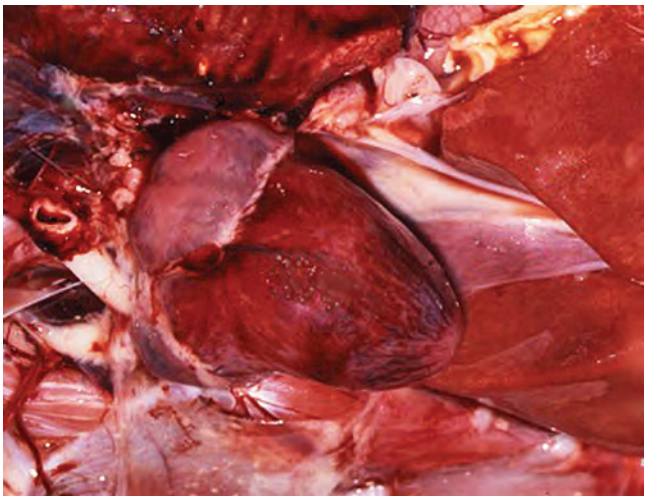

Figure 1.8 Polyomavirus infection causing patchy epicardial and myocardial hemorrhage.

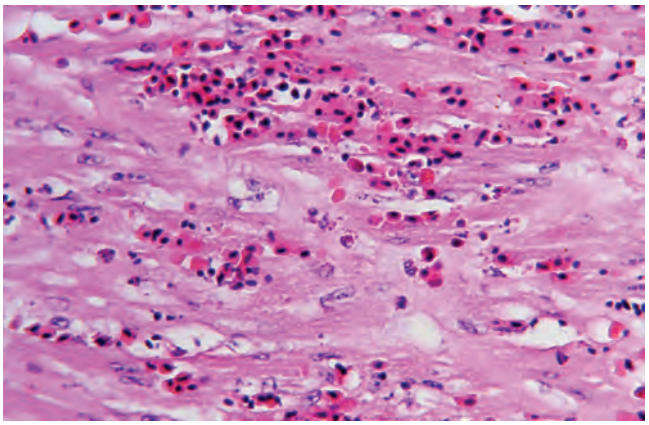

Figure 1.10 Severe myocardial degeneration and hemorrhage due to polyomavirus infection.

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

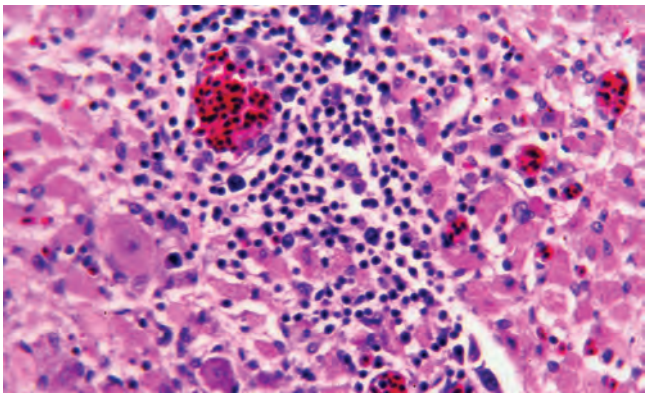

Figure 1.11 Nonsuppurative myocarditis in a bird with polyomavirus infection. Inflammation is seen infrequently in the heart in this disease.

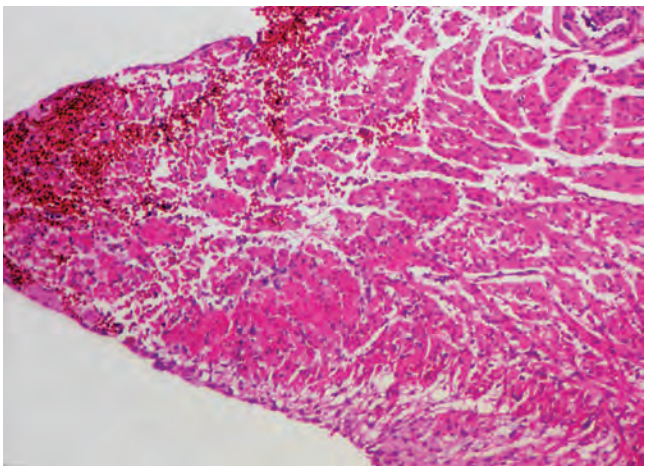

Figure 1.9 Focus of epicardial and myocardial hemorrhage in a bird with polyomavirus infection.

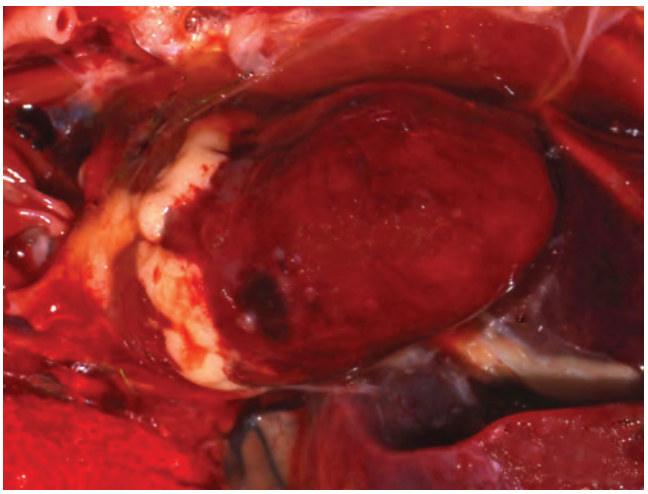

Figure 1.12 Foci of discoloration in the myocardium and asymmetrical dilatation of the ventricles of the heart of a bird with proventricular dilatation disease (Bornavirus infection). A focus of agonal hemorrhage is also seen, but hemorrhage is not a typical feature of this condition.

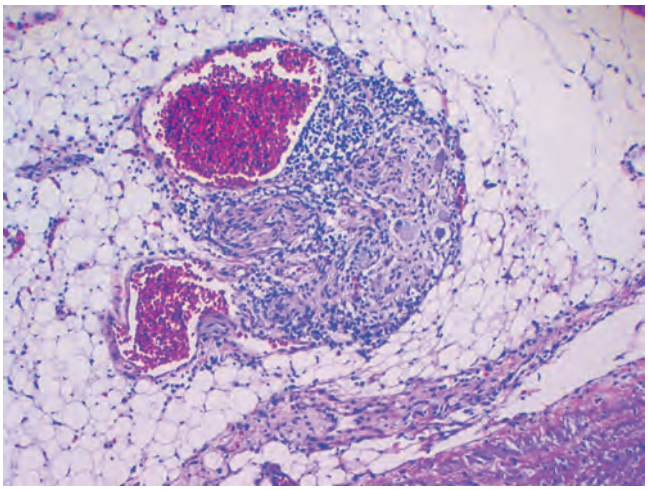

Figure 1.13 Epicardial ganglioneuritis in a bird with proventricular dilatation disease. A lymphoplasmacytic infiltrate is visible.

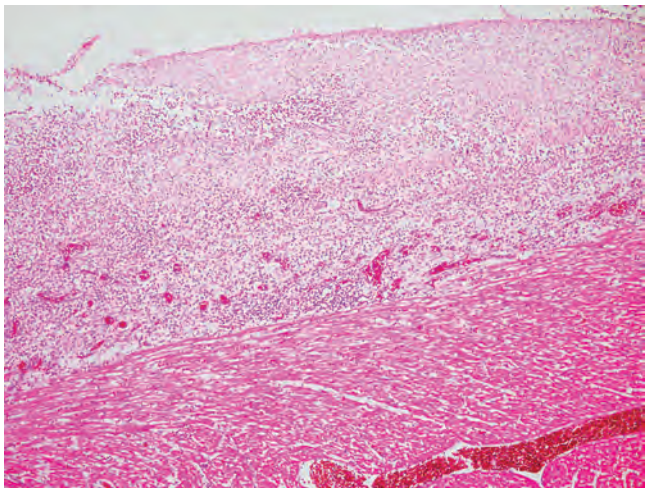

Figure 1.15 Severe chronic epicarditis in a pigeon with systemic salmonellosis. Nearly identical lesions can be caused by fungal infections and other bacterial infections.

lymphoplasmacytic and histiocytic infiltrates are seen in nerve ganglia (Fig. 1.13) and in the epicardium and myocardium, particularly near cardiac conduction fibers (Fig. 1.14). The conduction system may be involved, and, when severe, these lesions may cause the bird to die suddenly. Myocyte necrosis and, less commonly, fibrosis are seen.

Togavirus (eastern equine encephalomyelitis) is thought to be the etiologic agent of a disease described as avian viral serositis. Heart lesions in this disease include a fibrinous gray-yellow epicarditis. There may also be excessive cloudy pericardial fluid. Histologic lesions include the infiltration of lymphocytes, plasma cells, and histiocytes. Inclusion bodies are not seen, but ultrastructurally viral nucleocapsids are noted near cytoplasmic and intracytoplasmic membranes.

There is one report of a parvoviral myocarditis in canaries. A nonsuppurative myocarditis and viral particles were seen on electron microscopy. Myocardial necrosis is reported in systemic poxvirus infection but this is uncommon. The diagnostic features of the disease involve other organ systems. Myocarditis has been seen in cases of West Nile Virus infection. Grossly there may be gray-white foci and histologically a nonsuppurative myocarditis is present.

Bacterial infection of the heart can result in endocarditis, including valvular endocarditis, myocarditis, or epicarditis (Fig. 1.15), although in most cases at least two areas are affected. Bacterial heart disease may be the result of hematogenous spread of infection or direct extension from air sacs or adjacent tissues.

Primary gross changes in myocarditis are multifocal to confluent yellow-white foci (Fig. 1.16) that extend into the myocardium when sectioned. In advanced cases, large yellow nodules may be seen and must be differentiated from other types of infectious disease and neoplasia. Endocarditis may involve the wall and/or valves. Lesions are usually friable and vary from red-gray to yellow. Lesions may be seen on the chordae tendineae.

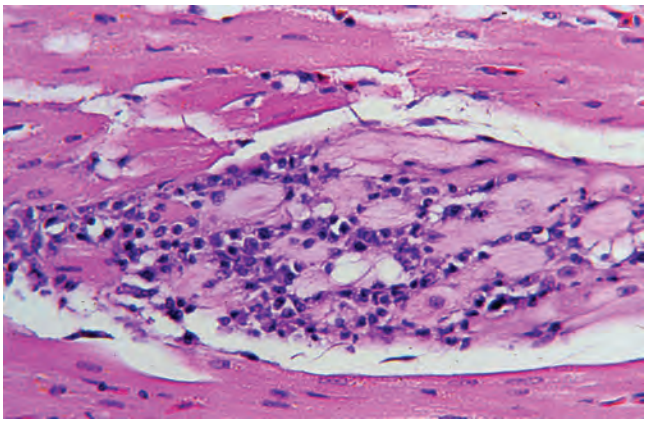

Figure 1.14 Lymphoplasmacytic inflammation in cardiac conduction fibers in a bird with proventricular dilatation disease.

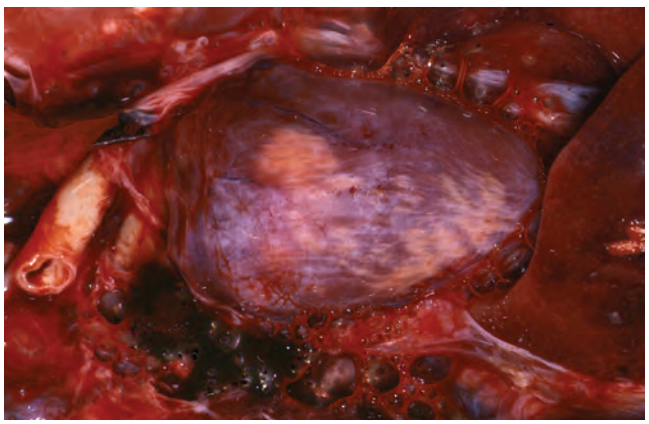

Figure 1.16 Severe bacterial myocarditis.

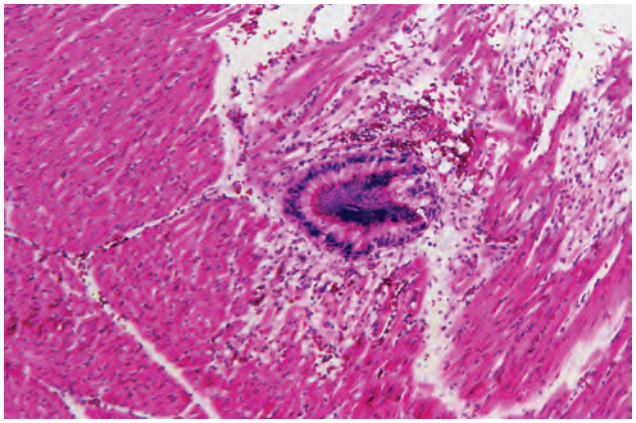

Figure 1.17 Bacterial myocarditis. Bacterial colonies are present in necrotic foci. A pleocellular inflammatory infiltrate is seen.

Valvular endocarditis is relatively rare, and, in our experience, the left AV valve is generally the only valve affected.

Histologically, bacterial infections vary with age. In early infections, there is acute necrosis and heterophilic reaction, and organisms may be seen (Fig. 1.17). As the lesion becomes more chronic, necrotic foci become surrounded by increasing numbers of macrophages, plasma cells, lymphocytes, and giant cells (Fig. 1.18). Organisms are usually seen in the center of these lesions. In endocardial lesions, in particular, fibrosis may occur as mural thrombi are organized. In cases of endocarditis, septic emboli may form, leading to disseminated infection in any other organ.

Mycobacterial infections are usually secondary to hematogenous dissemination or extension from cervical or thoracic air sacs. They usually involve the aorta or pericardium at the base of the heart and have been previously described. If there is myocardial extension, the lesion is similar grossly and histologically to those seen in the pericardium (Fig. 1.19).

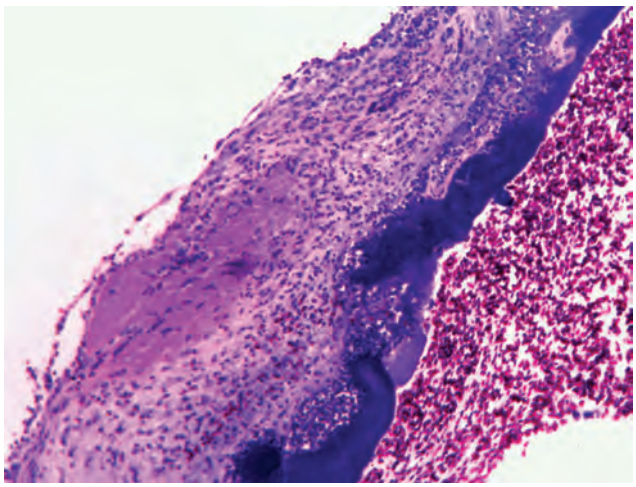

Figure 1.18 Severe valvular endocarditis. Large numbers of bacteria and a chronic-active inflammatory response are seen.

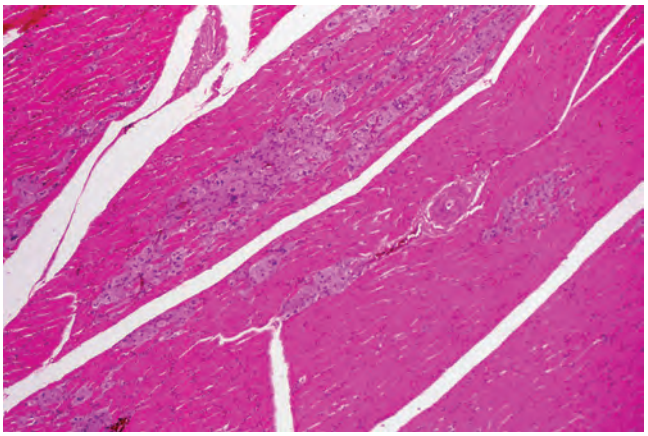

Figure 1.19 Mycobacterial infection of the myocardium. Numerous large macrophages are infiltrating the area. Granular material in the macrophage cytoplasm represents bacterial organisms.

We have occasionally seen myocarditis due to an intracellular bacteria that seems to be unique and unclassified. It is a Gram negative organism that has some morphologic features similar to *Helicobacter* sp. and also the organism called epitheliocystis in fish. There was no gross lesion seen. Histologically there was minimal inflammation and scattered myofibers contained small basophilic structures (Fig. 1.20). Ultrastructurally the organisms were within cytoplasmic vacuoles in degenerative myocytes. The organism has an undulating outer membrane, and a nucleoid with dense central masses and occasionally polar flagella (Figs. 1.21 and 1.22). The degree of inflammation associated with this infection can vary from little to moderate.

Mycotic infections of the heart are infrequent and usually the result of disease extension from air sacs. They usually involve the epicardium and superficial myocardium. Gross lesions are nodular or diffuse, gray-white, and friable (Fig. 1.23). If the fungal infection extends from an adjacent air sac, the fungus will sometimes produce conidia, giving the fungal plaque a green

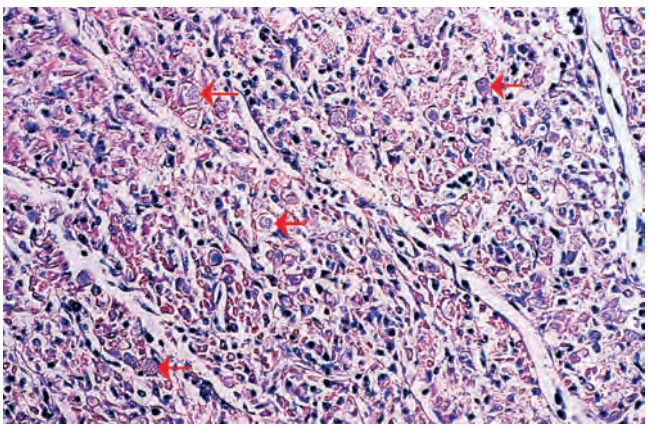

Figure 1.20 Myocarditis due to an unclassified intracellular bacterium. Note small organisms with myocardial fibers (arrows).

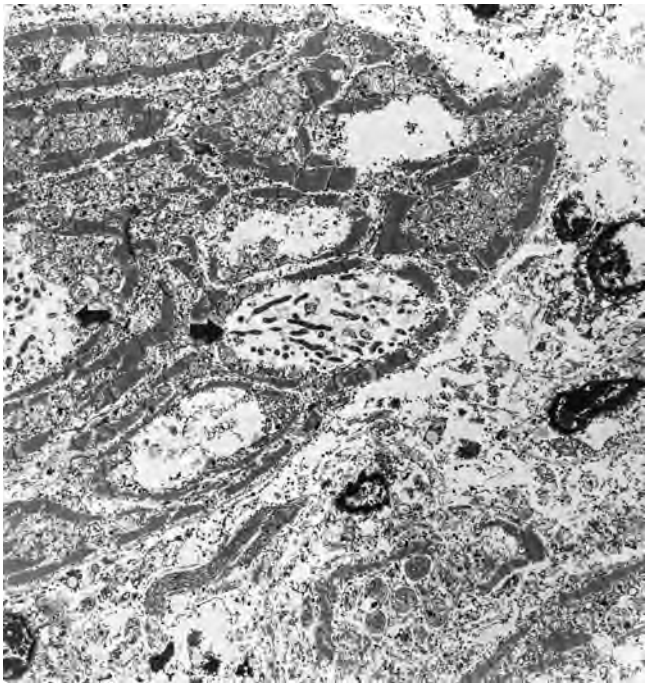

Figure 1.21 Unclassified intracellular organisms from Fig. 1.20. Ultrastructurally the organisms are within cytoplasmic vacuoles in degenerative myocytes.

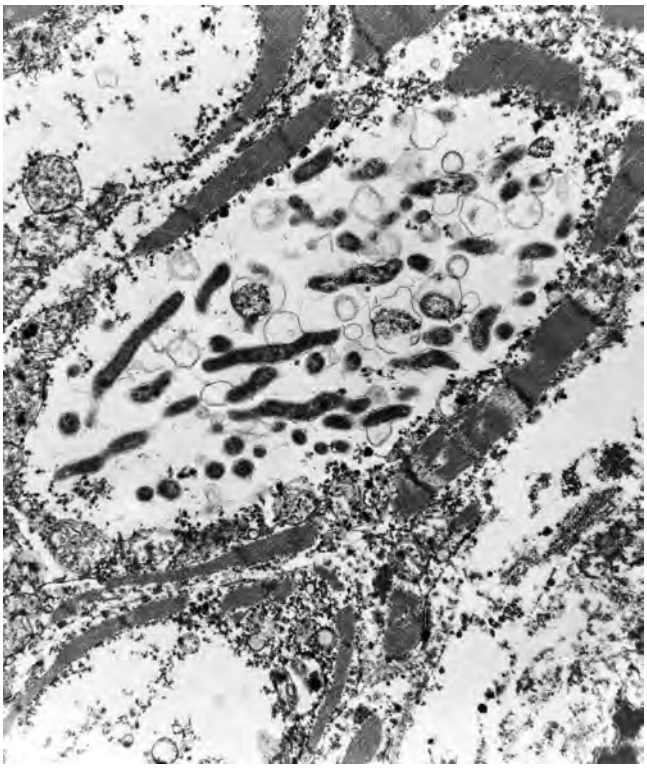

Figure 1.22 The organism has an undulating outer membrane, and a nucleoid with dense central masses and occasionally polar flagella.

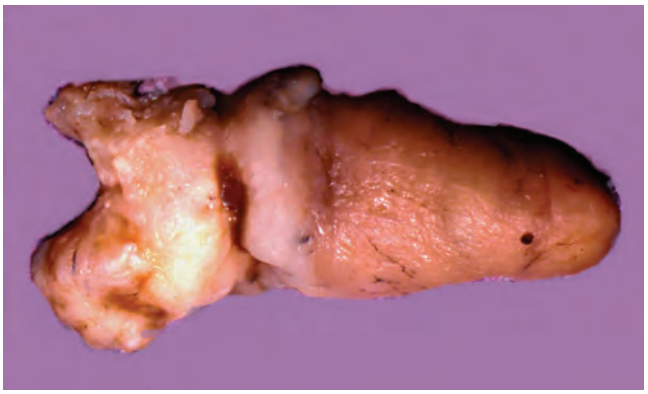

Figure 1.23 A large granuloma due to infection by *Aspergillus* sp. involving the atria and base of the large arteries. The lesion is not grossly specific, but should be suspected when more classical lesions of Aspergillosis are seen.

or black color. Histologically the lesions are similar to bacterial infections, with a pleocellular exudate whose character depends on chronicity. Fungal hyphae must be found for an exact diagnosis. *Aspergillus* is the most common organism involved, but a specific etiologic diagnosis requires that the organism be cultured or identified by molecular techniques.

Disseminated infection by *Aspergillus* sp., other mycelia fungi, and *Candida* sp. can develop in immunocompromised hosts, resulting in hematogenous dissemination to the heart. These lesions are grossly similar to bacterial infections of the heart, but fungal organisms are seen in necrotic foci. The inflammatory response is variable and involves both granulocytes and mononuclear cells.

Protozoal myocarditis is seen in some cases of systemic infection by *Sarcocystis* sp. *Sarcocystis falcatula* is a common cause due to the wide range of the definitive host, the Virginia opossum. The disease in most New World psittacine birds (macaws and conures) is usually subclinical, and the only evidence of infection is the incidental histologic finding of protozoal cysts in the myocardium at necropsy. Old World psittacine birds and some Amazon parrots have an acute disease with pneumonia and widespread dissemination of the organisms. Gross myocardial lesions are often not seen, but small white foci and streaks may be present in severe cases. Histologically there is a spectrum of myofiber necrosis, hemorrhage, and an inflammatory response comprised of lymphocytes, plasma cells, and macrophages. Newly formed cysts may be found (Fig. 1.24). Along with the brain and the lung, the heart is one of the common organs targeted by *Toxoplasma gondii* infections in birds. Bradyzoites and tachyzoites can be found in muscle fibers associated with inflammation (Fig. 1.25) similar to that caused by *S. falcatula*. In some instances inflammatory changes are seen but organisms are not found in the heart. Infections in cage birds are relatively rare but occur worldwide. Infections in poultry are common but rarely cause disease. Infections in wild birds are common, but the number of infections that result in disease is generally small.

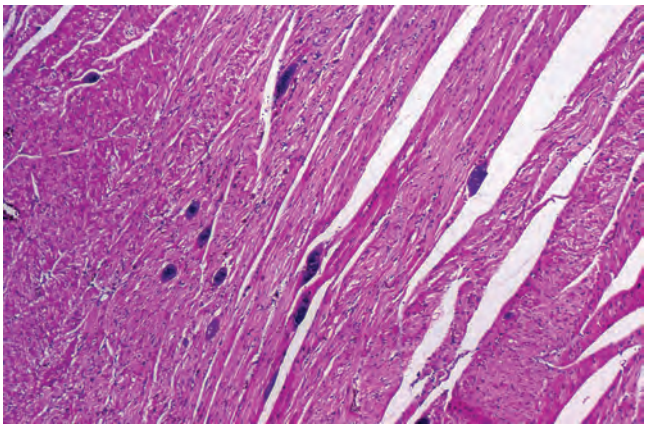

Figure 1.24 Multiple cysts of *Sarcocystis* sp. in the myocardium. Inflammation is usually not seen.

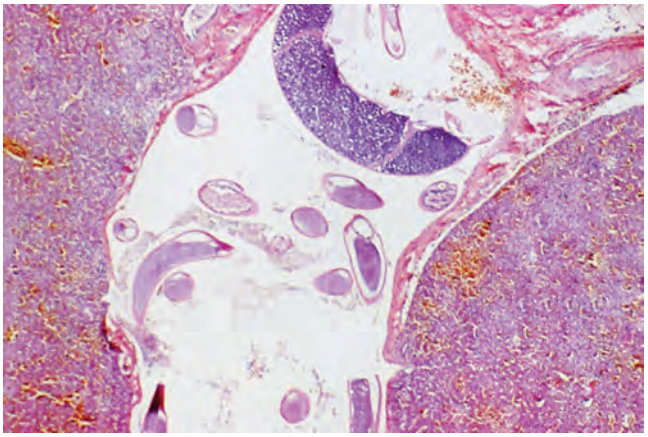

Figure 1.26 Filarid worms in a hepatic portal vein.

Filarid nematodes are an occasional necropsy finding in wild-caught cockatoos and occasionally other species and are particularly common in wild-caught African storks. These 1.5-cm white worms may be found in the right heart or in other vessels (Fig. 1.26). Histologically, adult nematodes may be found in hepatic and renal veins, and microfilaria are seen intravascularly throughout the body. In addition, focal endocardial hypertrophy and intimal hypertrophy of intramural vessels are noted. The intimal changes may be due to partial blood flow blockage by adults or a large number of microfilaria. Rarely, microfilaria are associated with embolic disease (Fig. 1.27).

Noninfectious disease

Inflammatory disease

Deposition of urates in the epicardium or occasionally myocardium results in grossly noted white-gray foci or streaks. Similar material may be seen in the pericardial fluid. Histologically the urates can be crystalline or amorphous and may elicit an inflammatory reaction comprised primarily of heterophils, although urate deposition without inflammation is also seen.

Nonseptic valvular endocarditis with formation of nodules and accumulation of inflammatory cells and fibrin has been seen as a secondary condition in cases of severe frostbite.

Inflammatory disease of undetermined etiology

Nonsuppurative myocarditis with no obvious cause occurs sporadically in birds. The possibility of autoimmune or immune-mediated disease should be considered in these cases even though not documented. It is known that certain peptides produced by *Chlamydia* mimic murine heart muscle-specific alpha-myosin heavy chains that can lead to nonsuppurative perivascular inflammation of the heart of mice. Since pet birds have a

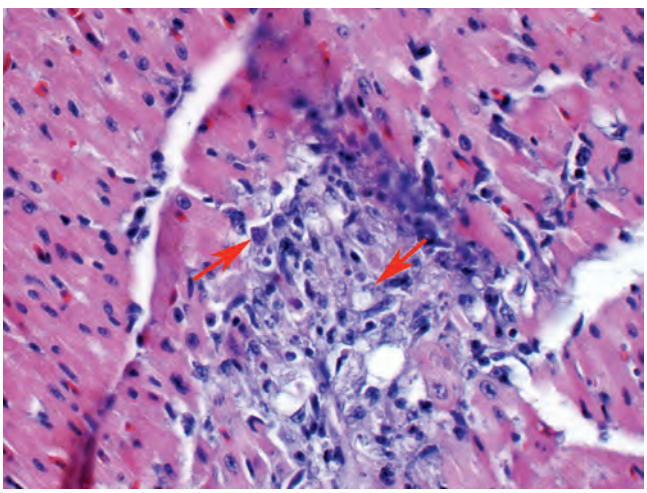

Figure 1.25 Myocardial necrosis and chronic inflammation associated with aggregates of Toxoplasma organisms (arrows).

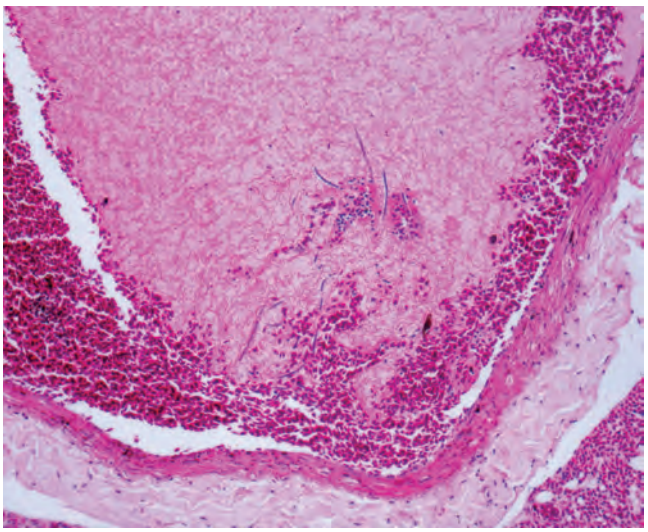

Figure 1.27 Microfilaria in a pulmonary thrombus.

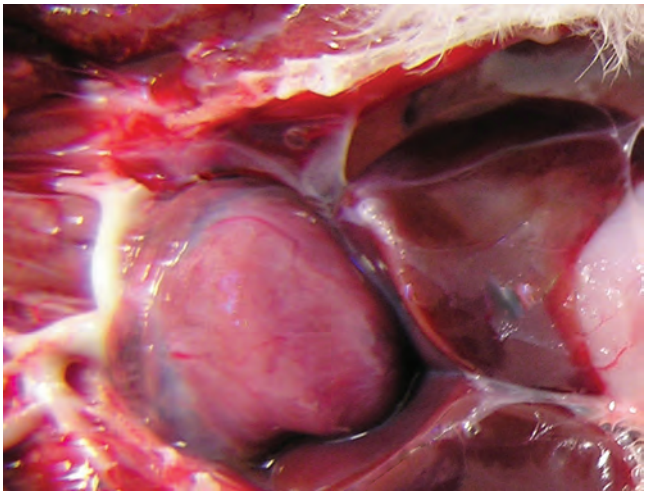

Figure 1.28 Serous atrophy of fat at the base of the heart. This change is consistent with a recent or longer term negative energy balance.

moderate incidence of chlamydial infection, perhaps the same mechanism that is seen in mice may be operational in birds.

Noninflammatory disease

Serous atrophy of fat

In cachectic birds, fat in the coronary grooves and epicardium may appear clear and watery as well as being reduced in amount (Fig. 1.28). Histologically adipocytes are small, and proteinaceous fluid may be present. Loss of heart fat is one of the first changes in birds experiencing a negative calorie balance, and it may even precede pectoral muscle atrophy.

Mineralization

Deposition of mineral may occur for several reasons, including dietary calcium/phosphorous imbalance, renal disease, and vitamin D_3 toxicity. It may also occur with excessive egg laying, but the pathogenesis is unclear. Mineral can be deposited in areas of myocarditis or myofiber necrosis of any etiology.

Grossly there are gray-white streaks and patches in the pericardium, epicardium, and/or myocardium. Gross differentiation from urates may not be possible, and both may be present in some cases. A spectrum of histologic changes can be seen, depending on the duration of the lesion. Evidence of primary inflammation or degeneration can coexist with myofibers containing a fine basophilic stippling along the cross striations. In some areas, there may be almost complete effacement of myofibers by mineral (Figs. 1.29 and 1.30).

Fat infiltration

Epicardial fat with some infiltration into the myocardium can be seen in birds and is not considered significant. It can, however, be excessive, and excessive fat is usually associated with obesity. Grossly the fat appears normal, but histologically there can be deep infiltration of the myocardium. Excessive amounts of fat may be a factor in heart failure, and the condition is seen in birds that die suddenly with no other morphologic change to explain death (Fig. 1.31).

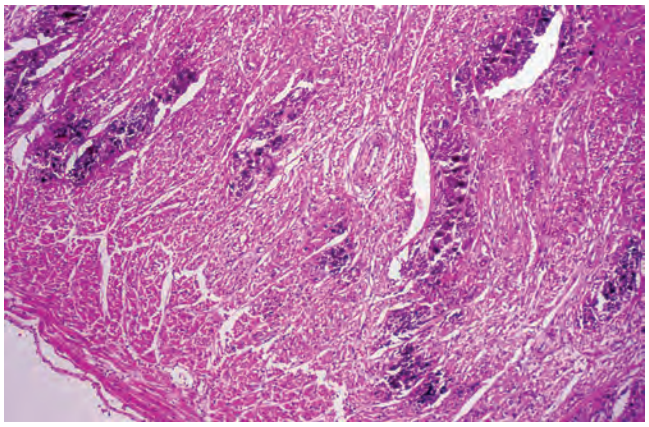

Figure 1.29 Multiple foci of myofiber mineralization in a bird with vitamin E deficiency.

Fibrosis

Fibrosis can occur after any insult to the heart that does not result in the immediate death of the bird (Fig. 1.32). Myocardial fibrosis is reported to occur in birds with atherosclerosis even though there is not necessarily coronary artery disease.

Lipofuscin

This is an intralysosomal pigment associated with excessive oxidation and polymerization of unsaturated fatty acids. It may accumulate in cells, including cardiac myocytes, secondary to a variety of disease processes. Although usually indicating emaciation or chronic disease, it is sometimes seen in young birds with acute clinical disease, possibly indicating a more chronic process than was expected. It is usually considered an incidental necropsy finding. If severe, the myocardium may have

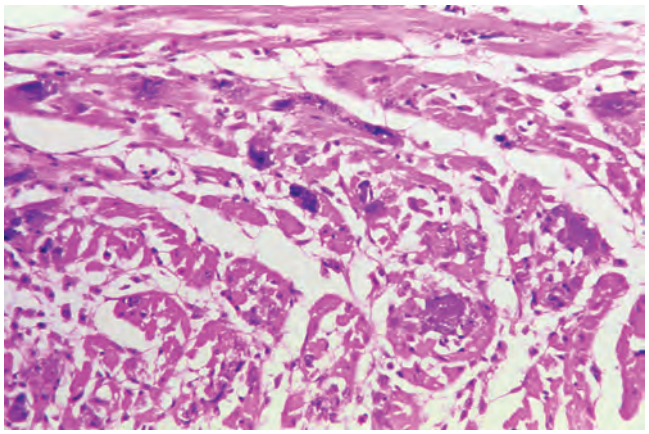

Figure 1.30 Myofiber necrosis and mineralization as a part of systemic changes in vitamin D toxicity.

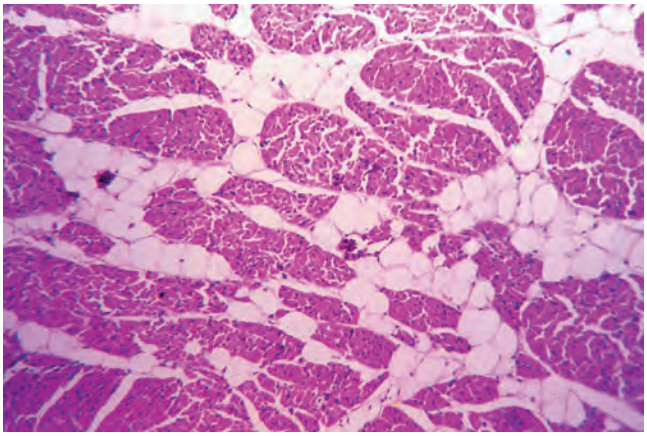

Figure 1.31 Severe myocardial fatty change probably associated with long-term feeding of a high-fat diet.

a brown discoloration. Microscopically, fine yellow-brown pigment is seen, primarily near the nucleus, but more diffuse in severe cases. One form of lipofuscin, ceroid, may occur in vitamin E deficiency.

Cardiomyopathy

Three forms of cardiomyopathy are described in mammals: hypertrophic, dilated, and restrictive. We have seen examples of the first two in pet birds. In both cases, the diagnosis is usually made on gross examination. Hypertrophic cardiomyopathy is characterized by ventricular thickening that leads to a diminution in ventricular volume. Dilated cardiomyopathy presents usually as a left-sided problem, with the left ventricle thin and flabby. In both cases, there may be no histologic change noted without quantitative morphometry and comparison to an age- and sex-matched bird of the same species. In some cases of dilated cardiomyopathy, the myofibers may appear obviously thin, with loss of sarcoplasmic detail, and, in some chronic cases, there has been evidence of fibroplasia, possibly indicating a previous insult. Restrictive cardiomyopathy is characterized by endocardial disease and fibrosis and could theoretically follow a variety of endocardial diseases, but we have not seen well-documented cases in pet birds.

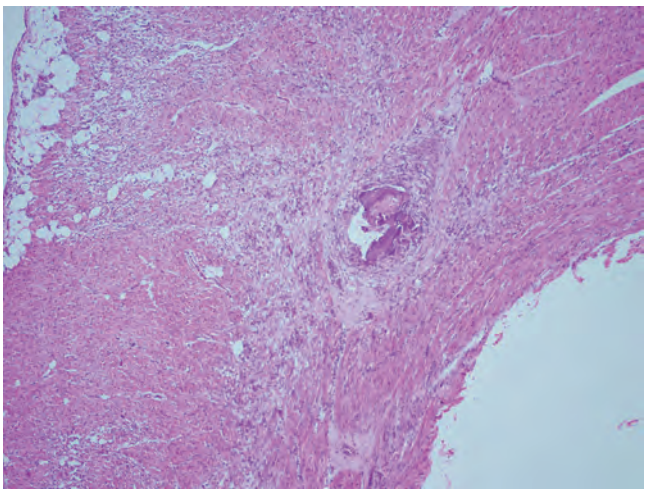

Figure 1.32 Mineralization, fibrosis, and fatty degeneration in a pigeon's heart. The cause of this lesion is not known, but may be the result of vascular disease resulting in ischemia.

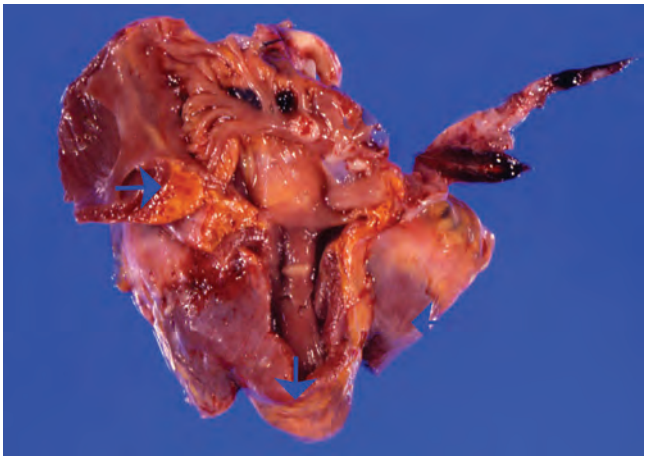

Figure 1.33 Myocardial degeneration due to vitamin E deficiency. Pale streaks and foci extend into the myocardium and on the epicardial and endocardial surfaces (arrows).

Myocardial degeneration

This may be the result of a vitamin E and/or selenium deficiency, vascular problems, and some toxicities. In many pet bird cases, the exact underlying problem is not determined. The gross appearance of an affected heart varies from having white streaks and patches to large pale areas (Fig. 1.33). In some chronic cases, the foci may appear as depressed areas. If there has been mineralization, affected areas are gritty when cut. Hydropericardium may be present.

Early histologic changes include contraction band formation, cross-striation loss, swelling, and hyalinization (Fig. 1.34). With progression, there is granulation, necrosis, and segmental fragmentation of myofibers. Microscopic mineralization may be seen, and, in chronic lesions, myofiber shrinkage and fibrous connective tissue proliferation are noted. No appreciable inflammatory response is seen (Fig. 1.35).

Myocardial degeneration is a prominent feature of a fatal disease of great-billed parrots. Also affected are the white matter and Purkinje cells of the cerebellum and skeletal muscles. The etiology is not known, but the lesions closely resemble those seen in poultry with vitamin E deficiency.

Endocardiosis

Noninflammatory swelling of true heart valves occurs occasionally. The cause is usually not determined. Affected valves are grossly swollen and usually smooth and firm. Histologically

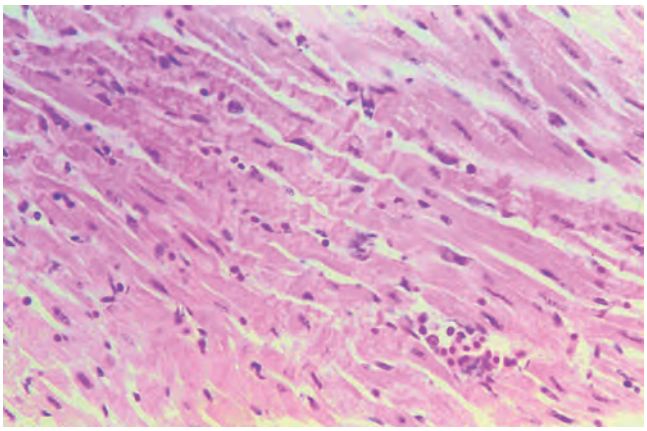

Figure 1.34 Contraction band formation and early myofiber fragmentation.

there may be hemorrhage, fibrous tissue proliferation, deposition of mucinous material, and cartilaginous metaplasia. Myxomatous degeneration of the left AV valve has been reported in an Indian ring-necked parakeet resulting in heart failure.

Heterotopic bone

This is seen sporadically in the myocardium. Its cause is usually not determined. Grossly the myocardium may feel gritty and histologically there is well-differentiated bone (Fig. 1.36).

Myocardial toxicity

Although a variety of drugs and chemicals are potentially cardiotoxic in birds, there are very few documented cases. Natural and experimental poisoning by avocados is seen in ostriches, canaries, cockatiels, and budgerigars. Gross lesions include subcutaneous edema and hydropericardium. Histologic lesions include myofiber degeneration and variable inflammation.

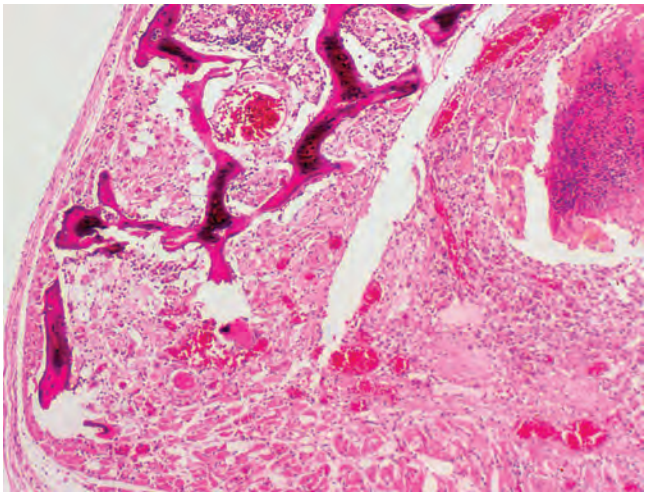

Figure 1.36 Diffuse production of heterotopic bone in the atrium of the heart.

Heterophils are seen in affected ostriches, but the lesion in canaries is characterized by nonsuppurative inflammation (Fig. 1.37). The toxic principle has not been determined.

Proliferative disease of the myocardium

Hypertrophy

Hypertrophic cardiomyopathy as a specific condition has already been discussed. Sporadic cases of myofiber hypertrophy are seen secondarily as compensatory responses to conditions that lead to an increased preload. These changes include pulmonary disease, vascular disease (especially atherosclerosis), congenital anomalies, and possibly chronic renal disease. Grossly the affected portion of myocardium is thickened and the lumen of the affected ventricle(s) is reduced. Histologically myofibers may appear to be normal, and without

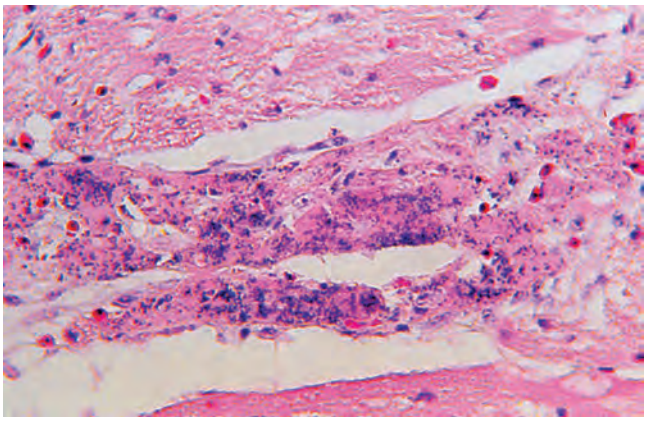

Figure 1.35 Necrosis of myofibers and severe mineralization in chronic nutritional myodegeneration.

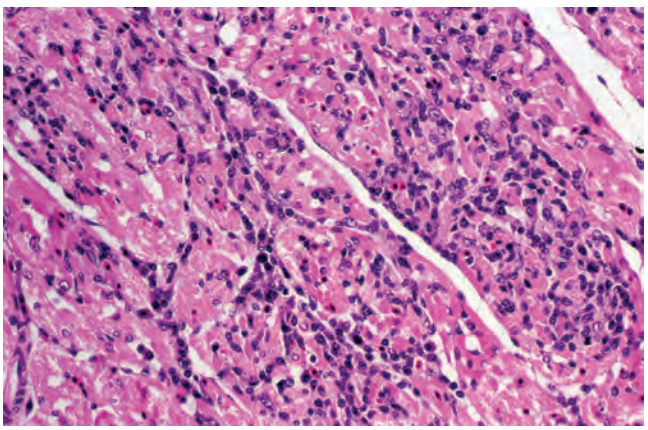

Figure 1.37 Myodegeneration, sarcolemmal proliferation, and nonsuppurative inflammation in a bird with avocado toxicity.

quantitative morphometry change can be difficult to discern. In more advanced cases, fibers may be thickened and lose their typical parallel appearance, and their nuclei may be enlarged.

Neoplastic disease

Several types of tumor are seen in the heart of birds. Rhabdomyoma or rhabdomyosarcoma is usually pale and firm grossly and may be multiple. Microscopically, strap and fusiform cells and cross striations are seen in routine sections of benign tumors. Sarcomas contain cells that may be fusiform, stellate, or strap-like, and cross striations are usually not present with hematoxylin-eosin-stained sections. Immunohistochemistry is often needed to prove the tumor is of striated muscle origin. Cell nuclei are enlarged and vesicular and may be multilobulated. Giant cells are often present. Rhabdomyosarcomas are infiltrative into surrounding myocardium.

Hemangiomas and hemangiosarcomas are found in the myocardium as red-black masses that may be friable and bleed easily. Histologically, benign tumors are comprised of well-differentiated vascular channels. Although histologically benign, these lesions interfere with normal cardiac function and do not have a benign behavior. Sarcomas are less well differentiated and may contain vascular channels lined by poorly defined endothelium, as well as solid foci.

We have seen primary fibrosarcomas of the myocardium. These tumors present as firm gray-white masses comprised of interlacing bundles and whorls of fibroblasts. Mitotic figures are typically abundant (Fig. 1.38).

Lymphosarcoma may involve the myocardium alone, or the heart may be involved as part of a generalized disease. Grossly the tumor is yellow-white or gray and may be diffuse or in multiple masses. Histologically lymphosarcoma is comprised of moderately undifferentiated pleomorphic lymphoid cells with variable mitotic activity (Fig. 1.39). They form infiltrative sheets in the myocardium. Occasionally, varieties appear to be primarily histiocytic (Fig. 1.40). The tumors caused by Marek's disease in chickens commonly infiltrate the heart (Fig. 1.41).

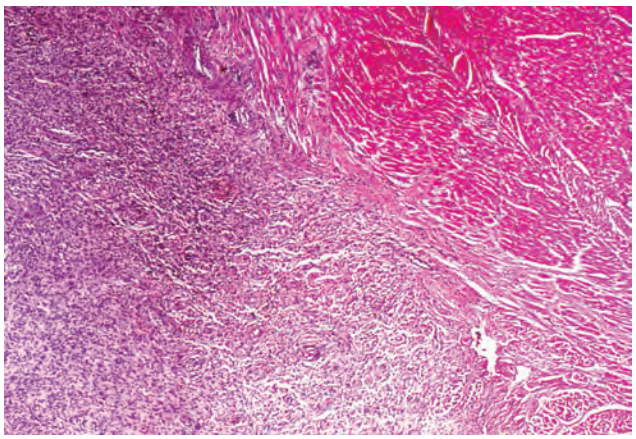

Figure 1.38 Fibrosarcoma replacing myofibers of the ventricle.

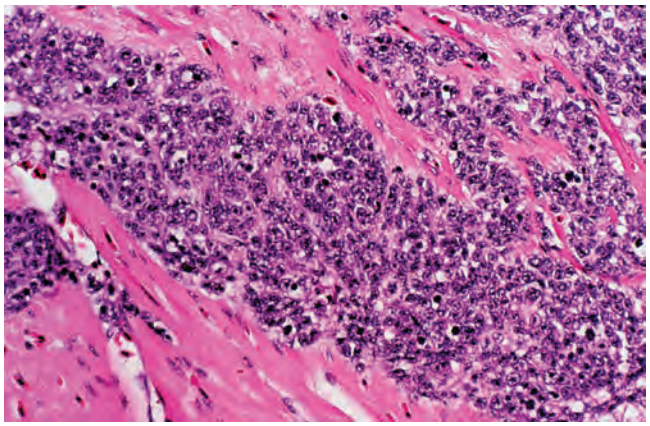

Figure 1.39 Lymphosarcoma infiltrating and effacing the myocardium.

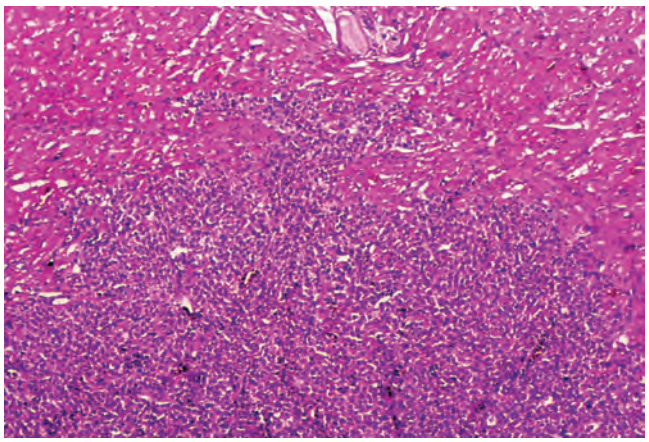

Figure 1.40 Myocardial histiocytosis. Compare the cell morphology with Figure 1.38.

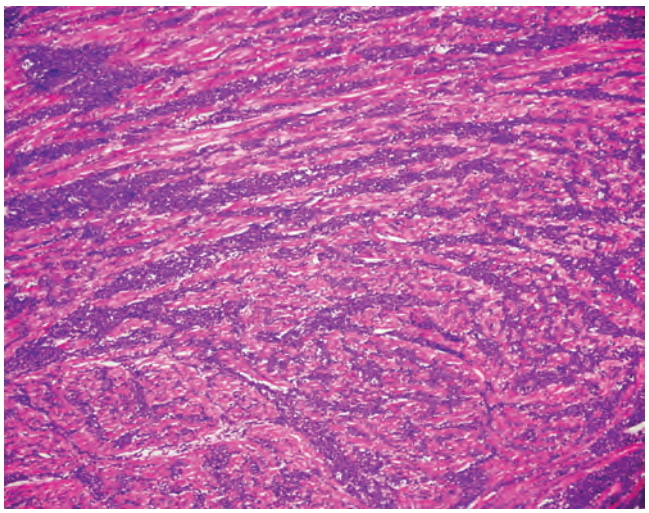

Figure 1.41 Marek's disease in a chicken. Neoplastic lymphocytes are seen infiltrating between muscle bundles.

Malignant melanoma may be found in the avian heart and is usually metastatic. These tumors are brown-black and may be multiple. Microscopically, poorly differentiated melanocytic cells form nests and sheets within the myocardium. Other metastatic tumors are rarely seen in the avian heart, but we have noted metastatic proventricular carcinoma in the epicardial lymphatics in a few birds.

The morphology of heart failure

A variety of conditions have been described that lead to heart lesions and subsequent death in birds. In addition, there have been many cases of pet birds whose presenting clinical sign was sudden death. In many of these cases, there is no gross or microscopic lesion. Sudden cardiac failure may be the underlying cause in many of these cases, and a careful and complete necropsy, as well as an investigation into the environment and husbandry of the bird, may be necessary to try and reach a conclusion as to possible cause.

The mechanism leading to acute cardiac death in any animal is often the creation of ventricular fibrillation or asystole. Factors influencing fibrillation include a long QT interval, hypokalemia, acidosis, imbalance in sympathetic/parasympathetic stimulation with a sympathetic dominance, and emotion. With sympathetic dominance, there is an exaggerated catecholamine reaction, reduced oxygen supply to the myocardium, and muscle spasm. The cardiac conduction system is described as anaerobic, with every cell functioning in an all-or-nothing capacity. Only a few cells are required for functioning, and problems can persist for some time until some insult leads cardiac failure. In chickens and turkeys, abnormal calcium regulation plays a part in the pathophysiology of heart failure. Although studies have not been done in pet birds, the mechanism may be similar.

Internal factors to consider in evaluating possible acute heart failure include disease in almost any other organ. In particular, there may be a relationship between the adrenal gland and the heart that leads to sudden death. With stress, there is an increase in interrenal cell (avian analog of cortical cells) hormone production that increases target organ sensitivity to the beta-adrenergic effects of epinephrine, leading to cardiotoxicity.

External factors may be obvious, such as a high-fat or other improper diet leading to obvious heart lesions. Less obvious is the possibility of the water supply being artificially softened, possibly causing electrolyte (potassium and magnesium) imbalances. In humans, soft water is apparently associated with cardiac problems. Although not documented in birds, evaluation of the water supply could be considered in ruling out unexplained sudden death.

The oculocardiac reflex results from pressure on or within the eyeball or stretching of ocular muscles. This results in a trigeminovagal reflex that leads to slowing of sinus rhythm and decreased conduction and contractility. This reflex has caused sudden cardiac failure in a pet bird.

Nonspecific stress is difficult to quantify and may be of different types and intensity. It is a factor in birds, particularly those in large aviary situations. Physical stress may be suspected if the owner is a good observer, but the possibility of mental or emotional stress as documented in humans is difficult to affirm. If environmental conditions include overcrowding, noise, and species/size mixture, some of the birds may certainly become stressed and die suddenly of no apparent cause.

In growing chickens there is a condition called sudden death syndrome (SDS). Affected chickens die suddenly and there is no apparent cause. Although the pathogenesis is not completely understood, some of the birds may have a predisposition to arrhythmia that leads to death. In some chickens there have been degenerative changes in cardiac myocytes and Purkinje cells.

Vascular disease

Inflammatory disease

Arteritis, phlebitis, and lymphangitis are infrequently encountered in pet birds. Bacterial infections can result in associated vasculitis in any organ. Histologically there is necrosis of the vessel wall and a response comprised primarily of heterophils. Microorganisms may or may not be present.

Mycobacterial arteritis is seen associated with lesions involving the pericardium and base of the heart. Lesions are similar to those that are seen in other parts of the heart (Figs. 1.42 and 1.43).

Fungi, such as *Aspergillus* sp., commonly invade the vasculature, resulting in the wide dissemination of fungal emboli that can cause vasculitis within any organ. In addition to necrosis and a pleocellular inflammatory infiltrate involving the vessel wall, organisms are usually present in the lumen and wall of the involved vessel or vessels.

Although not common in pet birds, paramyxovirus 1 (PMV-1) and togavirus (western equine encephalo-myelitis or WEE)

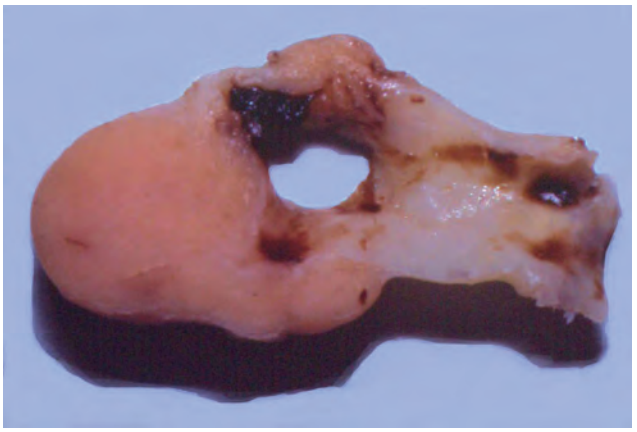

Figure 1.42 Chronic aortitis due to *Mycobacteria* sp. There is a large granuloma involving the wall of the aorta. Note the similarity of the lesion to that due to *Aspergillus* infection (Fig. 1.24).

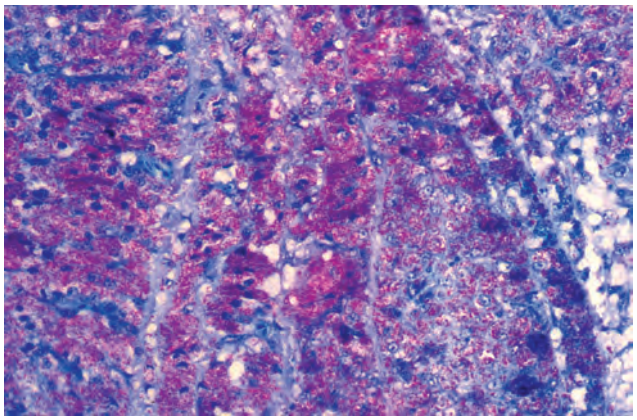

Figure 1.43 Acid-fast stain of diffuse infiltration of the artery wall by large macrophages containing acid-fast bacteria in a bird with mycobacteriosis.

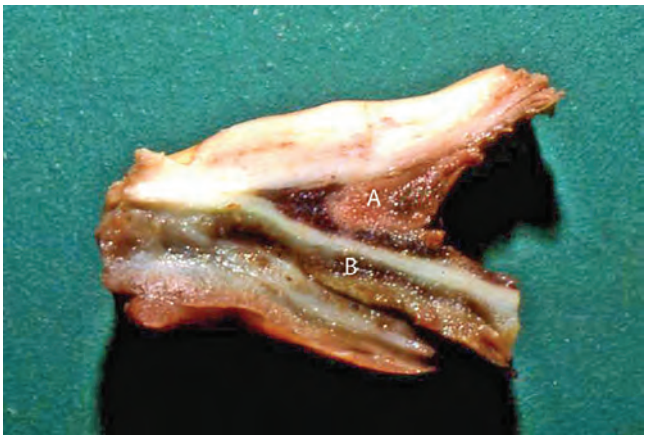

Figure 1.45 Separation of the aortic wall in a dissecting aneurysm. Original lumnen (B) and wall dissection (A).

can cause vasculitis. Grossly, in both infections, there will be hemorrhages, particularly in the serosa of the gastrointestinal tract. Histologically, vascular lesions of both infections are similar. Hemorrhage and edema are associated with hyalinization and degeneration of blood vessel walls, endothelial necrosis, possible thrombosis, and a variable mononuclear inflammatory infiltrate (Fig. 1.44).

Noninflammatory disease

Aneurysmal dilatation of blood vessels is not common in pet birds (Fig. 1.45). Occasional uncomplicated aneurysms are noted as variably sized dilatations in arteries. Histologically they are characterized by attenuation of the media, and there is usually no indication as to underlying cause, but they may be associated with atherosclerotic plaques.

Dissecting aneurysm is found in many avian species but is most often seen in the turkey and occasionally seen in the ostrich. These lesions are usually considered to be associated with a copper deficiency. A copper-dependent enzyme, lysly oxidase, is needed for connective tissue cross-linking of collagen and elastin in artery walls. Aneurysms begin with necrosis of elastin and arterial smooth muscle in the media, with subsequent hemorrhage and longitudinal dissection within the artery. Grossly there is dilatation and hemorrhage, and, on section, the dissecting band of hemorrhage is noticeable. Affected arteries may rupture (Figs. 1.46 and 1.47), with hemorrhage and clots noted in adjacent tissue and spaces. Thrombosis may also be present. Microscopically there is elastic tissue necrosis, acid mucopolysaccharide material deposition, hemorrhage, and variable inflammation that separate the arterial media.

Atherosclerosis

Atherosclerosis is perhaps the most common vascular lesion seen in parrots, especially aging populations of parrots. It can be found in any species of parrot and has been reported in many other species of birds. It is seen most often in Amazon parrots, particularly the blue-front Amazon parrot, African grey parrots, cockatiels, cockatoos, and macaws. It is also our experience that eclectus parrots are likely to develop atherosclerosis. Birds can be of any age, but most are 8 or more years old and many are more

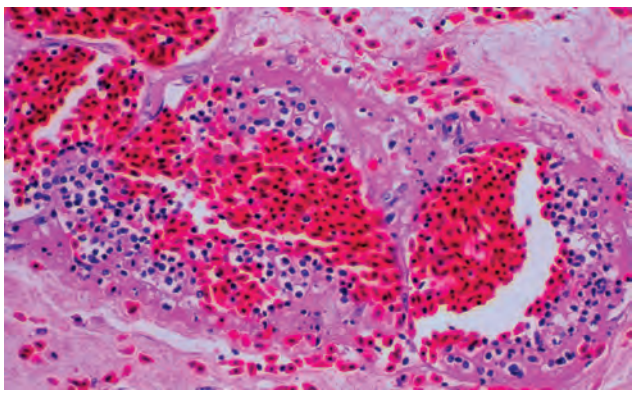

Figure 1.44 Vasculitis and degeneration of the vessel wall due to Togavirus infection.

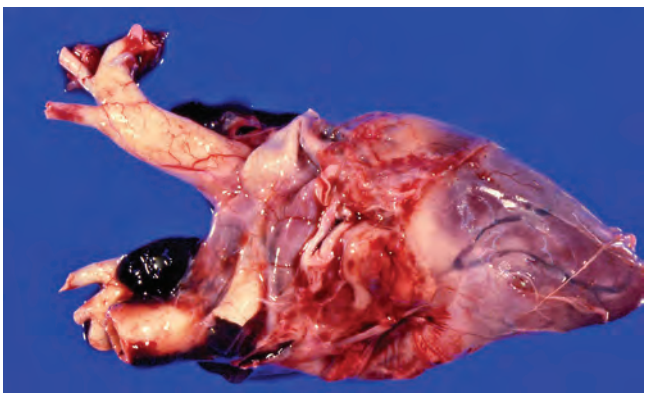

Figure 1.46 Ruptured aortic aneurysm with associated severe hemorrhage.

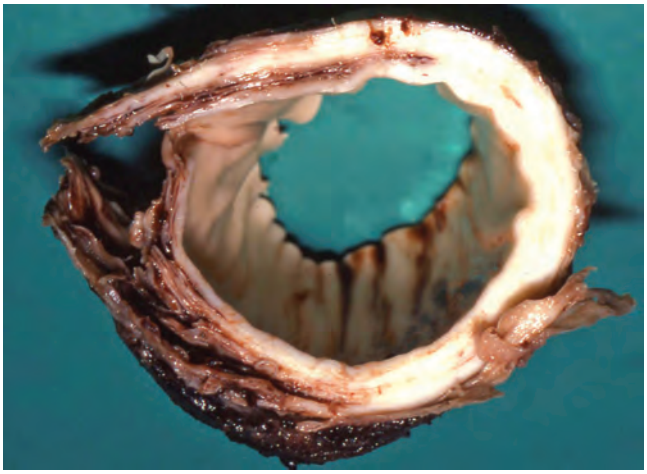

Figure 1.47 Rupture of the aortic wall. This is often the sequella of a dissecting aneurysm.

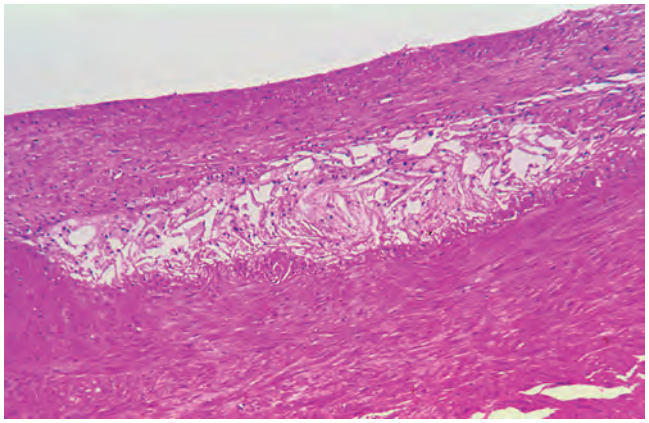

Figure 1.49 Early atherosclerotic lesion with foam cell and cholesterol cleft formation.

than 15 years old. The severity of lesions appears to increase dramatically in birds aged 20–30 years old. The prevalence of disease is very similar between sexes, but female birds may be more prone to develop severe lesions. Birds may die suddenly and be in excellent condition or be obese. Less commonly, atherosclerosis may cause a chronic disease that results in a loss of condition. Often there is a history of birds going through periods of a loss of awareness of their surroundings in the days or weeks prior to their death. Many birds have a history of being fed a diet rich in fat.

Lesions can be found in the aorta, brachiocephalic trunks, and pectoral and carotid arteries up to the level of the thyroids. Pulmonary arteries may also be involved but less commonly. Aortic lesions predominate in the ascending aorta and are uncommon in the descending aorta. Atherosclerosis of the coronary arteries is rare. Grossly the affected arterial wall is variably thickened and yellow (Fig. 1.48) and contains roughened yellow intimal plaques. Grading scales for atherosclerotic lesions have been extensively investigated and two have been developed. They are similar but not identical. Readers are referred to references by Beaufrère *et al*. and Fricke *et al*. for more detailed descriptions of these grading schemes. Microscopically the appearance of these plaques depends on the chronicity of the condition. Early lesions are characterized by fragmentation of the elastica and cell proliferation, and the deposition of extracellular substances resulting in thickening of the media. At approximately at the same time, fat-filled macrophages (foam cells) can be seen in the intima accompanied by an increase in extracellular matrix (Fig. 1.49). As the lesions progress, the number of foam cells increases, and as these cells die, there is the development of extracellular lipid that can include cholesterol clefts. Advanced lesions bulge into the lumen of the artery, have marked disruption of elastic layers, and form a fibrous cap. Fibrous changes continue in as the lesions mature, the atheroma is highly fibrous, and may contain little lipid. Mineralization also increases in the media of the diseased artery as the lesions become more severe. Chrondocytes are present in advanced lesions and may replace the smooth muscle cells in large areas of the media. Again in advanced lesions, there can be considerable narrowing of the vessel (Fig. 1.50). Microhemorrhage occurs variably.

Figure 1.48 Typical atherosclerosis. The aorta, brachiocephalic arteries, and carotid arteries are rich yellow colored and are thickened.

Many birds die because of a decreased blood supply to the brain as a result of severe narrowing of the carotid arteries. Infarction of the pectoral muscles occurs but is relatively rare. Likewise, it is rare to see ischemic disease of the heart. Atherosclerosis can lead to aneurysmal dilatation of the arteries. More commonly, it causes increased arterial resistance that affects the heart. Early changes in the heart include hypertrophy of the left ventricle, followed by left ventricular dilation, dilation of the left atria, right-heart dilation, and right-heart failure. Right-heart failure causes congestion, atrophy, and subsequently cirrhosis of the liver and is commonly accompanied by acites (Fig. 1.51).

Medial hyperplasia of vessels of the heart, liver, lung, and kidney have been associated with atherosclerotic changes in the

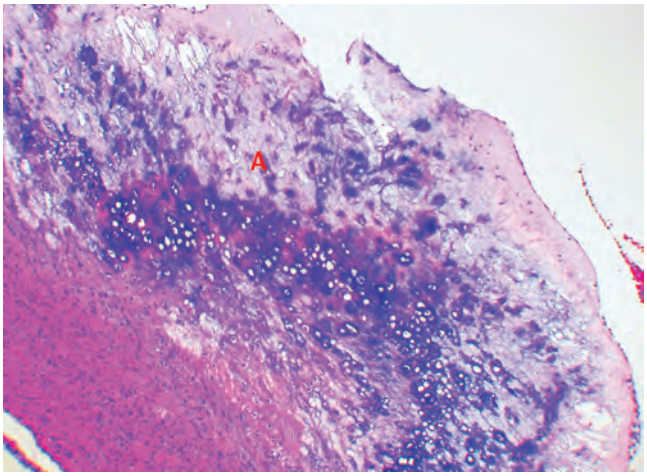

Figure 1.50 Severe arterial thickening due to atherosclerosis. Note the early chondroid metaplasia (A).

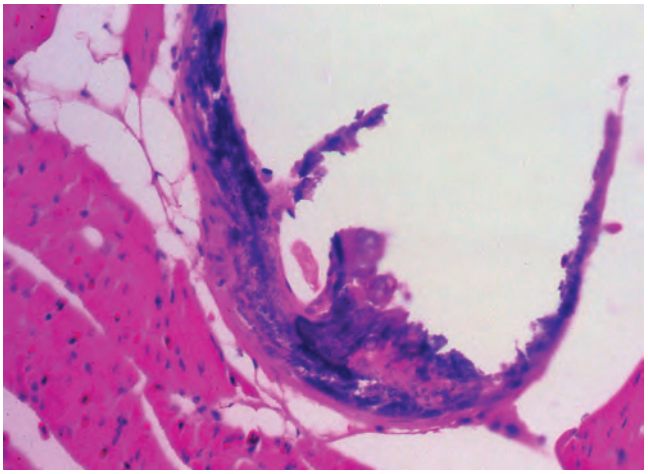

Figure 1.52 Mineralization of the arteriolar wall associated with a generalized problem due to chronic renal disease.

aorta and brachiocephalic trunks. Fibrosis of the myocardium has also shown a positive correlation with the severity of atherosclerosis of these vessels.

Mineralization

Mineralization of blood vessels with no other morphologic change is seen in cases of severe renal failure, chronic dietary imbalance of calcium and phosphorus, and vitamin D_3 toxicity. The change is typically in arteries or arterioles and can be found in any organ or tissue. The only gross indication may be a gritty feel to the tissue if the lesion is widespread or associated with other soft tissue mineralization. In larger arteries, raised, firm, irregular plaques may be seen that are usually gray-white and may have a shiny appearance. Histologically, all or part of the vessel wall may be affected (Fig. 1.52).

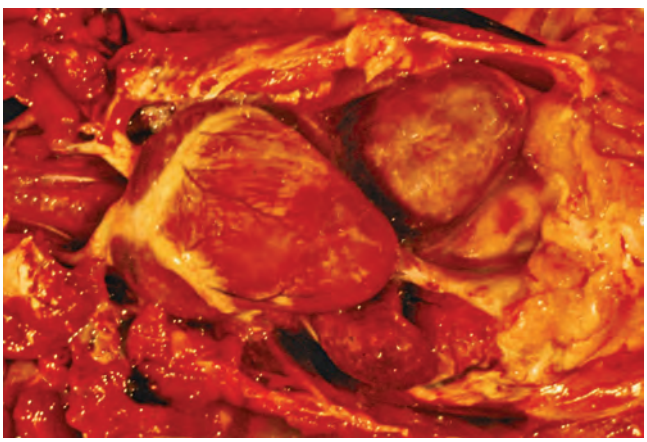

Figure 1.51 Left- and right-sided heart failure in a macaw with atherosclerosis. The spaces between the liver and adjacent air sacs were filled with a transudate. The liver is rounded and histologically was undergoing cirrhosis.

Amyloidosis

Among pet birds, amyloidosis is more common in the small passerines species but can occasionally be seen in other birds. Amyloid is deposited in a number of soft tissues and, in some cases, is found in the walls of blood vessels. This condition is usually not detected grossly. Histologically, affected vessels have a thickened media that has an amorphous, smooth appearance that is eosinophilic or amphophilic on hematoxylin-eosin stain. As in mammals, it is birefringent when stained with Congo red and viewed with polarized light (Fig. 1.53).

Thrombosis

Septic and nonseptic thrombi may be found in any tissue, depending on their cause (Fig. 1.54). Septic thrombi are often associated with valvular endocarditis, and bacteria may be present. Fungal infection that involves blood vessels may also lead to thrombi (Fig. 1.55). Secondary changes include infarcts and infection/inflammation in the involved tissue. Bone

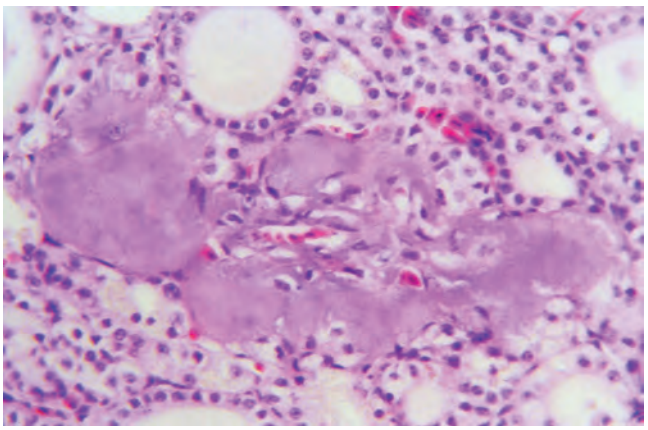

Figure 1.53 Amyloidosis of the arteriolar walls in the thyroid gland.