



Avian cardiology

Anneliese Strunk, DVM^{a,*},
G. Heather Wilson, DVM, Dipl. ABVP (Avian)^b

^a*University of California, Veterinary Medical Teaching Hospital,
One Shields Avenue, Davis, CA 95616-8747, USA*

^b*Exotic Animal, Wildlife and Zoological Medicine, Department of Small Animal Medicine,
University of Georgia College of Veterinary Medicine, Athens, GA 30602, USA*

The avian cardiovascular system is highly developed to accommodate the specialized requirements of various species' abilities to fly, run, and/or swim [1,2]. The unique anatomy and physiology of the avian heart allows for highly efficient blood circulation and oxygen delivery [2]. Although avian patients with heart disease can present diagnostic and therapeutic challenges, the clinical approach to these cases should be as thorough as in other companion animals. Once diagnosed, treatment becomes the greatest challenge, as little pharmacokinetic or toxicity data exist for cardioactive drugs in avian species. The avian cardiac disease patient will be discussed, with an emphasis on anatomic and physiologic features, clinical presentation, history, physical examination, diagnostics, disease processes and finally, therapeutics and monitoring.

Anatomy and physiology

Functional anatomy

The avian heart is situated cranially within the coelomic cavity on the ventral midline. The right and left lobes of the liver enclose the apex of the heart on each side (Fig. 1) [3]. As in mammals, the heart has four chambers [3]. The muscle wall of the left ventricle is two to three times thicker than that of the right ventricle, and becomes thinner towards the apex [3,4]. The right ventricle wraps around the left ventricle, forming a crescent-shaped cavity that does not extend to the apex [2]. The right cranial vena cava and

* Corresponding author.

E-mail address: anneliesevet@yahoo.com (A. Strunk).

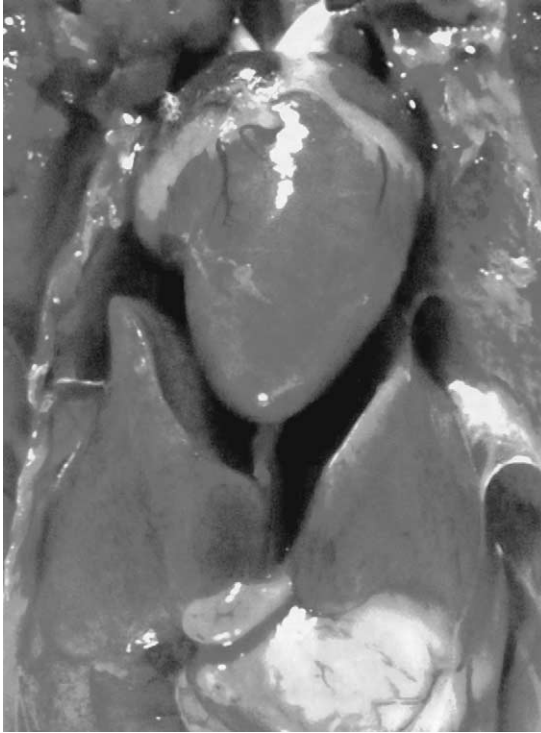


Fig. 1. There is no diaphragm in birds, and the right and left liver lobes enclose the apex of the heart. Photo courtesy of B. Ritchie.

caudal vena cava enter the right atrium separately from the left cranial vena cava via a poorly defined sinus venosus [3]. The right atrioventricular valve is unique in that it is a spiral muscular flap without chordae tendinae [2]. The other valves generally correspond with their mammalian counterparts. There is a muscular ring around the cusps of the left ventricular outflow tract, to which the middle Purkinje bundle branch connects [2]. The pulmonary veins join in the left atrium as a single vessel protruding into the left atrioventricular orifice, which helps to guide blood flow into the left ventricle and prevent regurgitation [3]. The aorta is derived from the right aortic arch, and thus curves to the right in birds. The pericardium consists of a tough fibrous membrane that contains a small volume of fluid for lubrication [2].

Avian cardiac muscle cells are one-fifth to one-tenth the diameter of mammalian cardiac myocytes, and lack the M-band and transverse tubules (T-tubules) found in mammalian cardiac muscle [2,5]. The smaller diameter of the avian myocardial cells provides a greater surface area, precluding the need for T-tubules and allowing for rapid myocardial depolarization [2].

Conduction system

The conduction system of birds is relatively similar to that of mammals [2,5]. Rings of fibrous tissue electrically separate the atria from the ventricles, preventing generalized spread of depolarization throughout the heart [3]. The sinoatrial node is located near the opening of the caudal and right cranial vena cavae into the right atrium, and functions as the pacemaker of the avian heart [2,3]. Impulses from the sinoatrial node conduct via Purkinje cells and normal cardiac muscle cells to the atrioventricular (AV) node where the wave of excitation is delayed to allow the atria to empty completely [2].

The impulse continues from the atria through the ventricles via the Purkinje system, which consists of an atrioventricular Purkinje ring, the bundle of His and three bundle branches [2]. The right and left bundle branches move down the interventricular septum and form a network in the subendocardium of the right and left ventricles, penetrating the myocardium along the tracts of the coronary arteries, which results in very rapid depolarization of the thick muscle of the left ventricle [2]. Unique to birds, the AV Purkinje ring runs up and around the right AV valve [2,3]. The middle bundle branch separates from the others and runs around the base of the aorta to the variably developed truncobulbar node, then connects with the AV ring, forming a figure of eight [2,6,7]. The sequence of depolarization begins subepicardially, spreading through the myocardium to the endocardium, starting with the apex of the right ventricle and continuing throughout the ventricles in a coordinated fashion [9].

Cardiac output

The avian heart is proportionally larger than mammals relative to body mass [3]. The proportion of body weight taken up by the heart increases as the size of the bird decreases [3]. Heart size may also vary within a species based on activity. Before migration, the heart size of some species, such as the barnacle goose (*Branta leucopsis*), increases dramatically [2]. The heart rate of birds is quite variable and dependent on body size, level of activity, and special physiologic needs [3]. Rapid heart rates in combination with large heart sizes contribute to higher cardiac outputs when compared with mammals [3]. Total peripheral resistance in birds is lower than in mammals; thus, a higher arterial pressure is needed to maintain a high cardiac output [3]. The mean avian arterial pressure ranges from 108 to 250 mmHg, depending on the species [3,8,9]. Blood pressure is influenced by age, gender (higher in males), environment, and diet [10]. In a recent study of mean arterial pressure in doves (*Columba livia*) anesthetized with isoflurane, 70 to 100 mmHg was apparently the normal range (C.M. Trim, personal communication, August 2002). Indirect blood pressure may be measured in larger birds by using a Doppler ultrasound flow meter and a small blood pressure cuff on the tibiotarsus [11]. Alternatively, direct blood pressure measurements may

be obtained through the catheterization of peripheral arteries [11]. Another characteristic of the avian heart is that the ventricles empty almost completely with every beat [12]. The aforementioned functional specializations of the avian cardiovascular system work synergistically to make up an efficient system of oxygen transport, contributing to the remarkable exercise capacity for which birds are known [2,3,9].

Clinical presentation

Heart disease should be suspected when patients are presented with dyspnea, coughing, weakness, lethargy, exercise intolerance, collapse/syncope, or coelomic distention [13]. Early in the course of cardiac disease, birds may present without any overt signs. Additionally, birds may be presented for acute death with no history to suggest the presence of heart disease [13,14]. However, a thorough history is important to help determine risk factors that may contribute to heart disease [13].

Signalment

Species, age, and gender are factors that may predispose birds to heart disease [4,9,15,16]. Older Amazon (*Amazona sp.*) and African grey (*Psittacus erithacus*) parrots are more susceptible to atherosclerosis [16]. Male poultry have been shown to have a higher blood pressure that can be lowered by the administration of estrogen [2].

Diet

Diet may be a factor in the development of heart disease in companion avian species [17]. Among medical disorders linked to obesity, those that pertain to the cardiovascular system include atherosclerosis, hypertension, and cardiovascular dysfunction [18]. Broilers (*Gallus domesticus*) that are bred for a high meat yield often develop pulmonary hypertension and ascites syndrome, a spontaneous cardiomyopathy thought to develop secondary to the increased demand placed on the cardiovascular system by the large breast muscle mass and rapid growth [19]. Copper deficiency may be linked to the formation of dissecting aortic aneurysms in turkeys and ostriches (*Struthio camelus*) [20]. Similar to the mechanism in mammals, a link between plasma taurine levels and heart failure has been described in broilers [21]. Feeding only seeds or other high-fat diets to sedentary, nonflighted birds leads to obesity and subsequent strain on the heart [17].

Activity level

Determination of the patient's ability to fly as well as the bird's general activity level is important in the determination of general physical fitness. Any changes related to exercise tolerance should be noted.

Physical examination

The physical examination is an important diagnostic tool in avian medicine [22]. If possible, the patient should first be evaluated in the cage to assess how the bird looks at rest. If there is any indication of dyspnea, the transport carrier may be placed in an oxygen cage to minimize handling and undue stress, with the examination continued in stages. Otherwise, the general appearance and responsiveness of the bird should be evaluated and the resting respiratory rate recorded. Some birds present in an advanced stage of debilitation, rendering it difficult to perform a complete physical examination. If the patient is dyspneic or appears weak before or at any time during the exam, it is advisable to place the bird in a humidified oxygen cage and continue the exam in stages, administering oxygen as needed.

Information regarding hydration and perfusion may be obtained by several methods. The color of the mucus membranes of the oral cavity is not reliable in most species due to pigmentation. Refill time of the ulnar or basilic vein may be prolonged (greater than 1–2 seconds) in patients with greater than 7% dehydration or patients with poor peripheral perfusion or venous return [23]. In species in which capillary beds are visible through facial skin or the keratin of nails and beaks, it may be possible to observe a change in color related to cyanosis due to poor oxygenation. Palpation of the peripheral pulses may help give an indication of peripheral perfusion.

Auscultation

Auscultation of the heart is best performed on the left and right ventral thorax at the base of the sternum [9]. Muffled heart sounds may indicate pericardial effusion or hepatomegaly [13]. If possible, the patient should be ausculted with minimal restraint [13]. Due to rapid heart rates, murmurs may be difficult to auscult in conscious birds. Alternatively, briefly placing the bird in a light plane of anesthesia with isoflurane may slow down the rate enough to appreciate subtle murmurs [9,24].

The lungs are best ausculted on the right and left sides of the dorsum between the shoulder blades as the lungs are attached to the body wall on either side of the spine [9]. Muffled lung sounds may indicate pleuroperitoneal effusion [9,13].

Coelomic palpation

Gentle palpation of the coelomic cavity caudal to the base of the sternum may reveal coelomic distention with fluid (ascites, peritonitis) or organomegaly. Ascites in pet birds is most frequently associated with liver disease, but may be due to heart disease [25,26]. Ascites with associated cardiac disease is more prevalent in domestic poultry and waterfowl [25]. Coelomic (and/or pericardial) fluid should be sampled and characterized based on specific gravity and cell/protein content. It should also be submitted for microbial culture [13,27]. For abdominocentesis, using standard aseptic technique, a needle or butterfly catheter (21 to 25 gauge) is inserted caudal to the base

of the sternum on the ventral midline and directed to the bird's right side to avoid puncturing the ventriculus [25,27]. Ultrasound may be used to ensure that coelomic structures are avoided. Removal of more than a diagnostic sample may result in hypoproteinemia and hypovolemia [25]. However, removal of a considerable volume of ascitic fluid may be necessary to alleviate clinical symptoms caused by the pressure of the fluid on the airsacs and the heart [25]. Ultrasound or endoscopic guided pericardiocentesis under anesthesia allows for characterization of pericardial effusion (S.J. Hernandez-Divers, personal communication, August 2002).

Diagnostic tests

Clinical pathology

Complete blood cell count

The complete blood cell count may reveal changes in red and white cell parameters that could support a diagnosis of cardiac disease. Polycythemia is a common finding in turkeys and broilers that develop chronic heart failure [19]. A leukocytosis may support the diagnosis of infectious causes of cardiac disease such as bacterial or fungal endocarditis [28,29]. Viral or overwhelming bacterial infections may lead to leukopenia [28].

Biochemical analysis

The plasma levels of muscle enzymes such as creatine kinase may be elevated secondary to cardiac muscle damage [30]. Heart-associated creatine kinase isoenzyme levels have been shown to rise secondary to furazolidone cardiac toxicity in Pekin ducklings (*Anas platyrhynchos*) [31]. Liver enzymes may be elevated from hepatic congestion secondary to right heart failure or hypoxic hepatocellular damage associated with congestive heart failure [31,32]. In turkeys with furazolidone-induced cardiomyopathy, hypoproteinemia from hypoalbuminemia and hypoglobulinemia were explained by blood volume expansion, and secondarily by inanition and possibly transudative loss into body cavities [32]. Hyperuricemia can lead to pericardial gout [33]. Hypokalemia is a common cause of bradycardia and arrhythmias. Hypocalcemia can cause arrhythmias in susceptible species such as African grey parrots [33]. Isoflurane anesthesia may exacerbate the hypocalcemia and thus the arrhythmia [34].

Additional lab tests

A variety of assays (polymerase chain reaction, serology, virus neutralization, etc.) may be used to support a diagnosis of cardiac disease due to specific infectious processes. In addition to direct blood smears and blood filtration tests, a commercial ELISA-based canine heartworm antigen test has been used to support the diagnosis of filarial nematodes (*Paronchocerca*

ciconarum) in two saddle-billed storks (*Ephippiorhynchus senegalensis*) [35]. Although the sensitivity and specificity values of these tests for avian filariae are unknown, the data from this case suggest that some cardiovascular filariae may conserve antigens across their genera, producing a positive result [35].

Radiography

A general understanding of normal avian radiographic anatomy is needed to accurately interpret radiographs (Fig. 2) [13,36,37]. Pertinent findings on survey radiographs may include enlargement of the cardiac silhouette, microcardia, enlargement of the hepatic silhouette, increased soft tissue density within the coelom due to coelomic effusion, increased density of the great vessels, congestion of pulmonary vessels, or pulmonary edema [13,36]. An enlarged heart shadow may be caused by cardiomegaly secondary to



Fig. 2. This ventrodorsal radiograph of a macaw demonstrates the hourglass silhouette formed by the heart and liver of most birds. Photo courtesy of B. Ritchie.

dilatation or hypertrophy, the presence of a mass, or pericardial effusion [36]. Microcardia is indicative of severe dehydration or hypovolemia [13,36]. Increased density or widening of the great vessels may be seen in cases of atherosclerosis [9,36]. An enlarged hepatic silhouette may be caused by hepatomegaly or any coelomic (gastrointestinal, reproductive, etc.) structure caudal, or lateral to the liver [36]. Presumed hepatomegaly and increased soft tissue density within the coelom should be further evaluated with gastrointestinal contrast radiography to confirm the course of the gastrointestinal tract, thereby clarifying the source of the soft tissue density. Anesthesia may be necessary to minimize the stress of manual restraint and allow for proper positioning of the patient for good quality films [36,37].

Heart size on the ventrodorsal radiograph may be evaluated by measuring the heart base at the level of the atria, which should be about 50% of the width of the coelom measured at the level of the fifth thoracic vertebrae [36]. Statistical analysis of radiographic heart shadow widths in relation to widths of the sternum and cranial coelomic cavity in asymptomatic individuals of several wild avian species has produced equations expressing theoretical normal heart width values for those species [38]. Future studies will be needed to develop equations, and thus theoretical normal values, for additional species [38].

Electrocardiography

The electrocardiogram (ECG) is a useful tool in avian medicine as it can be utilized to measure heart rate and to detect arrhythmias, cardiac chamber enlargement, and electrical conductance abnormalities (Tables 1 and 2) [9,39]. Auscultation of an arrhythmia or heart murmur during physical examination or visualization of an enlarged cardiac silhouette on radiographs are indications for performing an ECG. There have been several studies undertaken to document normal ECG values in a number of pet, production, and free-living avian species [40–47,115]. To evaluate morphologic features of the avian ECG, it is important to have an ECG machine that has a speed of 100 mm/s or greater (may need 200 mm/s in very small patients) [9]. At slower speeds the ECG wave forms are too close together to interpret but the tracing may be useful as a monitoring device during anesthesia and may be used to detect arrhythmias [9].

The ECG demonstrates the summed electrical activity of the heart [2]. In birds, the mean electrical axis is negative (and thus the QRS wave is inverted in lead II); however, in many other respects the avian ECG is similar to mammals [2]. The standard bipolar limb lead II is commonly used to evaluate ECG wave forms in birds (Figs. 3 and 4) [9].

For a diagnostic ECG, the machine is standardized at 1 cm = 1 mV [9]. The sensitivity of the ECG should be adjusted to produce wave forms that fit fully within the tracing paper, yet do not exceed the edges [9]. In psittacines, isoflurane anesthesia is recommended to take ECG tracings unless the

Table 1
Avian ECG waveform analysis [2,9,44,90,115]

| | |
|------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| General comments | The average mean electrical cardiac axis is negative (between -83° and -162°) Low-voltage ECG: suggestive of pericardial effusion, adipose tissue, or space occupying masses |
| P-wave | Atrial depolarization Normally upright (may be bifid in cockatoos) Tall and peaked: right atrial hypertrophy (P pulmonale) Too wide: left atrial hypertrophy (P mitrale) (may be associated with dyspnea from tracheal obstruction) Tall and wide: suggestive of biatrial enlargement (eg, influenza virus in gallinaceous birds) |
| PR interval | Represents short delay in the atrioventricular node prior to ventricular contraction Small T _a -wave within PR interval: repolarization of the atria (normal in pigeons and asymptomatic psittacines/gallinaceous birds) |
| QRS complex | Represents ventricular depolarization and contraction Q-wave variable: generally regarded to be absent in most birds Duration: measured from start of R-wave to end of S-wave S-wave amplitude: measured from baseline downwards QRS Too wide/too deep: suggestive of left ventricular hypertrophy Prominent R-waves: suggestive of right ventricular hypertrophy |
| ST-segment | Represents repolarization of the ventricles Very short/absent: clinically asymptomatic racing pigeons and psittacines |
| T wave | If present, may be elevated above baseline: normal in birds Represents repolarization of the ventricles Normally in opposite direction to the main vector of QRS complex Always positive in lead II Inverted polarity: suggestive of myocardial hypoxia Progressive increase in size: suggestive of myocardial hypoxia (eg, during anesthesia) Increased amplitude: suggestive of hyperkalemia |
| QT Interval | Prolongation may be associated with anesthesia or electrolyte disturbances (hypokalemia, hypocalcemia) Prolongation may occur in clinically normal African grey and Amazon parrots |

patient is tolerant of the lead placement and only mild restraint is required [9,13]. Isoflurane anesthesia causes only an increase in the QT interval, making interpretation of ECGs recorded from anesthetized birds possible [44]. Needle electrodes placed subcutaneously, or 25-gauge needles placed through the proptagium of each wing and through the skin of each thigh, onto which the alligator clips are attached, are superior to alligator clips alone for use in avian patients as they help to prevent artifact and skin damage (Fig. 5) [12,48]. A handheld ECG recorder without leads (Biolog ECG Recorder, CVM Resources, Dallas, TX) has been used with apparent success in both awake and anesthetized birds that weigh more than 100 grams [49].

Table 2

Common avian arrhythmias

| | |
|-------------------------------------------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| Sinus rhythms | Normal rhythm established by sinoatrial (SA) node Increased vagal activity may decrease heart rate Decreased vagal activity may increase heart rate |
| Normal/physiologic | Sinus arrhythmia: heart rate may increase during inspiration/ decrease during expiration (S-S interval may change) Sinus arrest: exaggerated form of sinus arrhythmia; diagnosed if the pause is greater than twice the normal S-S interval Sinoatrial block: when electrical impulses from SA node fail to activate the atria; pauses are exactly twice the S-S interval Wandering pacemaker: continuous shifting of the pacemaker site in SA node |
| Sinus bradycardia | Vagal stimulation Various anesthetics (eg, halothane, xylazine, and acepromazine) although isoflurane and halothane may increase heart rate [8]; may be potentiated by hypothermia Atropine responsive Pathologic causes: hypokalemia, hyperkalemia, thiamine deficiency, hypovitaminosis E, toxins (eg, Organophosphates, polychlorinated biphenyls) Atrioventricular nodal escape rhythm: noted in hypokalemic ducks with sinus bradycardia |
| Reflex vagal bradycardia | May occur secondary to neoplasms and space occupying lesions impinging on the vagal nerve Atropine responsive |
| Atrial tachycardias Sinus tachycardia | Caused by pathologic conditions of the atria May see P-wave superimposed on T-wave (P on T phenomenon) Has been recorded in 16% of normal Amazon parrots and in 6% of normal African grey Parrots [44]. Must differentiate junctional tachycardia (negative P-waves) from ventricular tachycardia/atrioventricular dissociation (normal P-waves not followed by a QRS complex) as digoxin may potentiate ventricular fibrillation in birds with ventricular arrhythmias |
| Atrial fibrillation | When electrical impulses are generated in the atrium in a rapid and irregular way Atria are in a state of permanent diastole Ventricular rhythm is irregular ECG is characterized by the absence of normal P-waves, normal QRS complexes, and irregular S-S intervals Digoxin is treatment of choice but prognosis is guarded because of the presence of marked cardiac pathology |
| Ventricular arrhythmias Supraventricular tachycardias | Sinus tachycardia originates from SA node Atrial tachycardia originates from atrium Junctional tachycardia originates from junctional area Differentiation of atrial vs. sinus tachycardia; P-P interval equidistant in atrial tachycardia, irregular in sinus tachycardia (due to vagal effects) |

Table 2 (continued)

| | |
|-------------------------------------------|------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------------|
| | Junctional tachycardias: diagnosed by presence of inverted P-waves in lead II Most common cause of sinus tachycardia is nervousness (may also be precipitated by stress and pain) |
| Ventricular premature contractions (VPCs) | Characterized by QRS complexes that are unrelated to the P-waves Bigeminy: alternating normal beats and VPCs Trigeminy: two normal beats followed by one VPC Causes of VPCs: hypokalemia, thiamine deficiency, Hypovitaminosis E, Newcastle disease/avian influenza viruses, myocardial infarction due to lead toxicosis and digoxin toxicity Ventricular tachycardia often characterized by: ventricular capture beats (normal P-QRS complexes between abnormal VPCs) ventricular fusion beats (QRS complex intermediate between a normal P-QRS complex and a bizarre QRS complex; formed by simultaneous discharge of ectopic ventricular focus and normal AV node) VPCs, ventricular tachycardia, and ventricular fibrillation may occur secondary to hypoxia and the use of halothane During anesthesia monitoring, changes in the T-wave may be related to myocardial hypoxia, potentially leading to more severe ECG abnormalities |
| Atrioventricular dissociation | Special form of ventricular tachycardia Atrial and ventricular rhythms are independent of each other: atrial rate is slower than the junctional or idioventricular rate |
| Atrioventricular node arrhythmias | May be caused by serum electrolyte concentrations and acid-base balance [44] |
| First-degree heart block | Increased PR interval caused by delayed impulse through AV node May be caused by halothane and xylazine PR interval may increase to three to four times normal May cause severe bradycardia; reversible with atropine |
| Second-degree heart block | Majority of P-waves are followed by a QRS complex, but some impulses do not reach the ventricles Mobitz type 1 (Wenckebach phenomenon: PR interval lengthens progressively until a ventricular beat is dropped) [114]; physiologic phenomenon in 5% of trained racing pigeons [115] seen occasionally in asymptomatic psittacines and raptors |
| Third-degree heart block | Characterized by independent activity of atria and ventricles: frequency atrial depolarizations is higher than ventricular depolarizations Ventricular complexes may have a normal configuration or may be idioventricular depending on site of ventricular impulse formation Complete AV block has been seen in chickens with hypokalemia |

Ultrasound

Ultrasound can help to evaluate the coelomic cavity for effusion and soft tissue masses. In addition, ultrasound can be useful for sampling coelomic or pericardial fluid via aspiration. Ultrasound examination of the coelomic

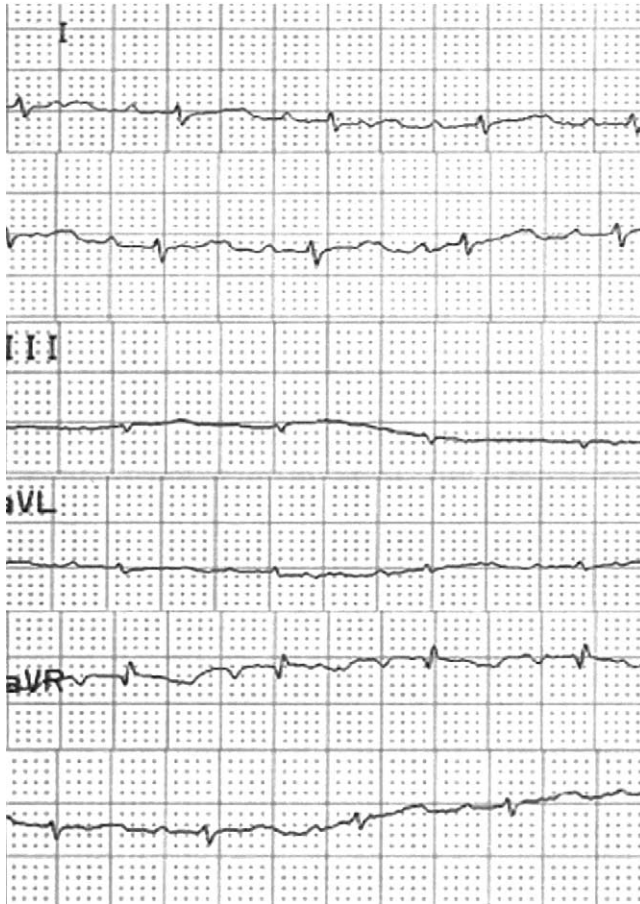


Fig. 3. Electrocardiogram tracings from a normal chicken. From top to bottom: leads I, II, III, aVL, aVR, and aVF. 1 cm = 1 mV; paper speed 25 mm/s.

cavity can help elucidate the source of cardiomegaly identified radiographically by verifying the presence of pericardial effusion, chamber enlargement, and masses associated with the heart.

Krautwald-Junghanns et al [50] have established a detailed protocol for the ultrasonographic examination of avian hearts to develop standard views and reference values for avian echocardiography. For thorough examinations, simultaneous electrocardiographic recording and ECG triggered sonography were used to record the stage of the cardiac cycle [50].

Ultrasound is a useful tool, but poses some difficulty in birds because internal organs are surrounded by airsacs. However, there are “windows” on the avian body that provide adequate space for visualization of various body systems. A period of fasting may be necessary [51]. The contact area

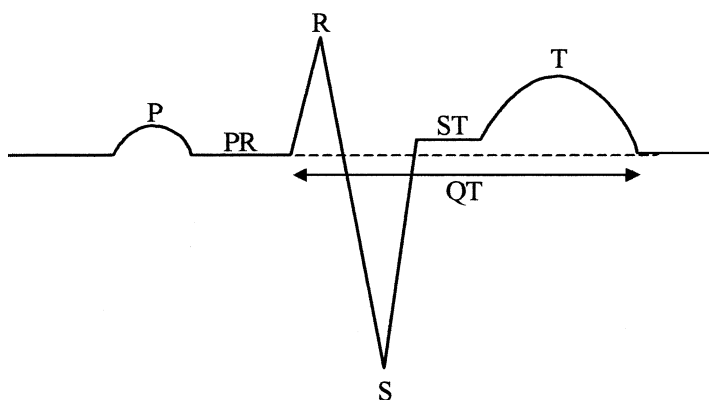


Fig. 4. Schematic representation of a normal lead II electrocardiographic complex of a bird, labeled with commonly measured parameters. P wave (P), PR interval (PR), ST segment (ST), T wave (T), and QT interval (QT).

on which the probe can be applied is typically very small; thus, probes with small footprints are necessary. A 7.5-MHz or faster transducer is preferred for appropriate resolution. Sector transducers produce a fan shaped field of view, which helps maximize the field of view while minimizing contact area. A stand off pad may be used to examine smaller patients [50].

The most common approach is ventromedial, just caudal to the sternum, viewing the heart through the liver. Sedation is typically not necessary for ultrasound examination. Patients may be held in partial upright dorsal recumbency or completely vertical position for this approach. There is a natural aperture in this area in most psittacines, where the feathers can be easily parted and acoustic gel applied (Fig. 6) [51]. A right lateral, parasternal approach has been developed for poultry and other deep-chested or long-keeled birds. The probe is placed in the space caudal to the last right rib. The patient may be held in right lateral recumbency or remain standing for this approach [50].

The echocardiographic examination, together with color flow Doppler technology, allows for identification of the four chambers of the heart and their relationship to one another. It may also be used to evaluate the contractility of the ventricular walls, determine internal systolic and diastolic dimensions, measure wall thickness, evaluate septal, valvular, and ventricular outflow tracts, and identify changes in echogenicity of the pericardium or presence of pericardial fluid [50].

Other diagnostic tests

Nonselective angiography has been reported to evaluate the heart and the great vessels by taking rapid sequence serial radiographs after the administration of a bolus of contrast medium in the basilic vein [52]. Limitations



Fig. 5. Proper positioning for an ECG in an African grey parrot (*Psittacus erithacus*). Note that 25-gauge needles are placed through the proptagium of each wing and through the skin of each thigh, onto which the alligator clips are attached.

of this modality are related to patient size and intravenous access. Specialized equipment may be needed to observe all four chambers in patients with very rapid heart rates. Because no published references are available for all species, this procedure may require a clinically normal bird of the same species for comparison. Both ventrodorsal and lateral views should be taken to fully evaluate the heart and vessel anatomy. This technique illustrated dilatation and abnormal function of the right side of the heart and decreased perfusion of the aorta and brachiocephalic arteries in a macaw with congestive heart failure secondary to atherosclerosis [52].

The Doppler ultrasound flow meter may be used for detecting peripheral perfusion, detecting arrhythmias and measuring indirect blood pressure. It is used most frequently for monitoring of peripheral pulses during anesthesia. Several arteries may be utilized for this purpose, including the carotid, recurrent ulnar, and medial metatarsal arteries [11,53].

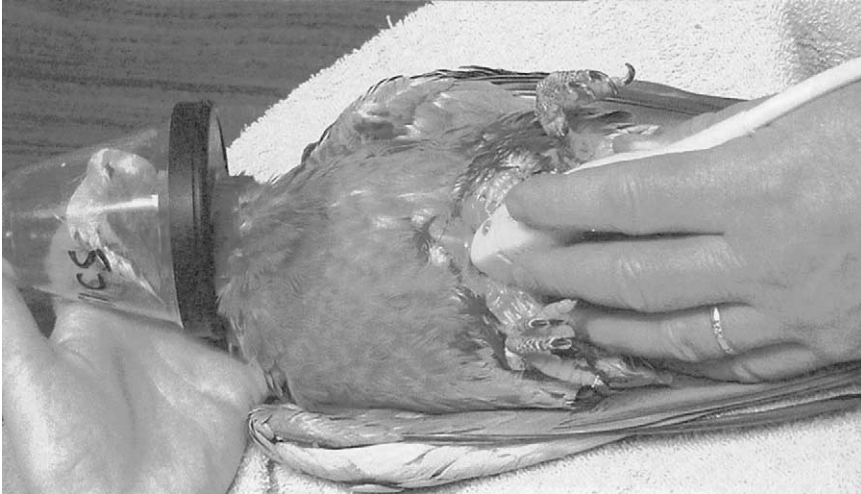


Fig. 6. Echocardiogram in a galah cockatoo (*Eolophus roseicapillus*). Note the placement of the probe at the base of the sternum in the natural apteric region.

Pulse oximetry is frequently utilized for measuring tissue oxygenation in mammals. However, a study evaluating the pulse oximeter (placed on the gastrocnemius and tibialis cranialis muscles) in comparison with arterial blood gas measurements in several species of birds has shown that the oxygen saturation values given by the pulse oximeters in birds are not accurate due to the different hemoglobin dissociation curve seen in bird blood. Although not completely accurate, the pulse oximeter may be useful for monitoring avian patients during anesthesia, as decreasing values can be positively interpreted as a fall in oxygen saturation [54].

Disease processes

Heart disease in birds poses a diagnostic challenge, and therefore, can be readily missed. Companion birds are more likely to develop heart disease secondary to captive husbandry [4]. Lack of exercise, inappropriate diet, and abnormal environments are risk factors associated with captivity. Retrospective studies of necropsy findings illustrate the prevalence of heart lesions in psittacines. Oglesbee and Oglesbee [55] found that 9.7% of 269 psittacine birds evaluated at necropsy had cardiac lesions and 58% of these showed evidence of congestive heart failure. Straub et al [56] found the prevalence of cardiac lesions to be over 30% in over 200 psittacine birds evaluated.

Congenital disease

Congenital disorders have been demonstrated in birds, but typically lead to embryonic or fledgling death due to the normal high demands placed on

the avian cardiovascular system. Ventricular septal defects are common in poultry, but have been reported in other species, including a houbara bustard (*Chlamydotis undulata macqueenii*) [57]. Most of these defects are functionally closed, but a study indicated that 2% of evaluated cases were associated with congestive heart failure [9,58]. Multiple congenital defects, including persistent truncus arteriosus, aortic hypoplasia, and ventricular septal defects, were diagnosed with radiography and echocardiography in two young cockatoos presenting with heart murmurs, tachycardia, and bounding pulses [58].

Endocardial diseases

Endocardial disease has been reported in a variety of species, and may be caused by several different etiologies, including inflammatory or infectious, parasitic, degenerative, and less frequently, idiopathic disease [9,35]. Vegetative endocarditis of the aortic and mitral valves may cause valvular insufficiency with or without an associated systolic murmur, lethargy, and dyspnea. It is most common in birds with chronic bacterial infections such as salpingitis, hepatitis, and pododermatitis [9,29,59]. Blood cultures should be considered with patients presenting with murmurs of sudden onset.

Myxomatous degeneration of the left AV valve leading to chronic heart failure has been reported in an Indian ringneck parakeet (*Psittacula krameri*) [60]. Mitral insufficiency of idiopathic origin leading to congestive heart failure has been diagnosed using ECG and echocardiography and treated in an Indian Hill Mynah (*Gracula religiosa*) [61]. Endocardial fibrosis and cardiomegaly have been reported in a secretary bird (*Sagittarius serpentarius*) [62].

Thromboembolic disease has been linked with vegetative valvular lesions in several cases [59]. Endocardial lesions may be caused by the presence of circulating filarial nematodes. Filariae of the genus *Paronchocerca* (Nematoda: Filarioidea) have been identified in the right heart chamber and/or the pulmonary artery of numerous species of free living avian species [35].

If the cause of the endocardial disease can be determined to be of infectious origin and is acute, appropriate treatment may allow for a full recovery. Once changes in the myocardium secondary to valvular insufficiency have occurred, management is similar to the treatment of congestive heart failure.

Myocardial diseases

Infectious myocarditis can occur secondary to viral, bacterial, mycotic, and protozoal infections [9,63]. Other disease processes may result in myocarditis. Amyloid deposition has been reported in various tissues (including the heart) in a group of laying Japanese quail (*Coturnix japonica*) [64].

Myocarditis due to toxoplasmosis has been reported in free-ranging turkeys [65]. Infection in a 3-week-old red lory (*Eos bornea*) showing depression

and poor growth caused multifocal necrotizing myocarditis and similar lesions in the liver and lungs when evaluated at necropsy [66].

Case reports have also documented cardiac sarcocystis in a limited number of free-living and companion avian species [67–73]. A study has defined the pathogenesis of *Sarcocystis falcatula* in the budgerigar (*Melopsittacus undulatus*), with sudden death during the acute phase of the infection being the predominant clinical finding [71].

Cardiomyopathy in poultry and other birds has been associated with rapid growth, hypoxia, high altitude, furazolidone, monensin and sodium toxicities, vitamin E and selenium deficiencies, rancid fat in the diet, and inbreeding [9,19,74–76]. The pathogenesis of cardiomyopathy and myocarditis in birds is similar to that in mammals [9]. Spontaneous turkey cardiomyopathy occurs in turkeys 1–4 weeks of age, and is characterized by marked dilation of the right ventricle with extreme thinning of the ventricular wall and pulmonary congestion [19]. This disease is associated with rapid growth and hypoxic conditions, which lead to pulmonary hypertension and cor pulmonale [19]. Round heart disease in backyard poultry refers to acute cardiac failure due to myocardial degeneration [19].

Myocardial infarctions may be caused by emboli originating from valvular endocarditis or heavy metal poisoning [9]. Degenerative, inflammatory or neoplastic conditions of the myocardium or aneurysms of the myocardial vessels may lead to rupture of the myocardium [9]. A link between copper deficiency and aortic aneurysm development and rupture is suggested in turkeys and ostriches (*Struthio camelus*) [20].

Rupture of the heart apex has been documented in three young capercaillie hens (*Tetrao urogallus*). The similarity of the lesions among the birds suggests an anatomic predisposition to rupture in this species [77,78].

Neoplasia

When masses are found associated with the heart, cytology or histopathology is required to differentiate between granulomas and neoplasia. Reported primary cardiac tumors include lymphoma, hemangioma/hemangiosarcoma, and rhabdomyoma/rhabdomyosarcoma. In chickens, the development of skeletal muscle tumors has been linked to oncogenic viruses [79].

Tumors may decrease chamber lumen diameter, interfere with contractility, and/or affect conduction due to neoplastic and necrotic foci within the myocardium and conduction system. Diagnosis may be made ante mortem through radiography, ECG, and echocardiography or postmortem through gross necropsy and histopathology [79–81].

Toxins

Numerous toxins, including those of plant and fungal origin, may cause either temporary or permanent cardiac dysfunction. Although birds tend be

less sensitive to plant toxins, as a general guideline, most toxins affect birds similarly to mammals, and may be treated similarly [82].

Cardiac glycosides in lily of the valley (*Convallaria majalis*) and oleander (*Nerium oleander*) have digitalis-like effects. Taxines in the yew (*Taxus media*) inhibit ionized calcium currents in the heart. Alkaloids of nightshade (*Solanum nigrum*) and other *Solanum* spp. including potatoes (*Solanum tuberosum*) (most concentrated in sprouts and skin of potatoes) may cause fatal arrhythmias, and chronic exposure may lead to calcification of the vascular system. Other cardiotoxic plants include jimsonweed (*Datura* spp.) and milkweed (*Aesclepias* spp.). The toxicity of the avocado (*Persea americana*) is controversial. No toxic compound has been found, although cardiac damage and sudden death associated with avocado ingestion have been reported [82–85]. Chocolate ingestion may cause tachycardia and arrhythmias [82].

Birds with hemochromatosis may have excessive iron deposition within the myocardium that may lead to cardiomegaly [9,13,86]. The toxic effects of furazolidone produces a syndrome in turkeys that is indistinguishable from spontaneous turkey cardiomyopathy, providing a model for heart disease in humans [19,87]. Furazolidone induces cardiomyopathy in other avian species as well [9,31,88]. Moniliformin, a toxin produced by *Fusarium moniliforme*, has been shown to cause cardiomegaly and death in poultry [89].

Epicardial and pericardial diseases

Pericardial effusion may result from cardiac or systemic disease and may be inflammatory or noninflammatory in nature [13]. Clinical signs associated with pericardial disease include lethargy, decreased exercise tolerance, and dyspnea. Pericardial effusion will lead to insufficient filling of the ventricles in diastole due to atrial compression by the pericardial fluid [9]. Non-infectious pericardial effusion is associated with congestive heart failure and hypoproteinemia [9]. Pericardial effusion has been reported secondary to mycotoxicosis from aspergillosis [90]. Hemopericardium is typically associated with trauma, rupture of the left atrium, or neoplasia [9].

Fibrinous pericarditis is the most common cause of pericardial effusion, and may lead to adhesions of the epicardium to the pericardium and subsequent constrictive heart failure [9]. Etiologic agents include bacterial, viral, mycotic, parasitic, mycoplasmal, and mycobacterial infections [4,9,90,91]. Pericardial effusion secondary to a mycobacterial heart base granuloma has been reported in a Gang Gang cockatoo (*Callocephalon fimbriatum*) [92]. Generalized trichomonas infection in a group of pigeons that led to a granulomatous pericarditis has also been reported [93]. Uric acid deposits on the pericardium may be seen in birds as a degenerative process related to visceral gout [4,9].

Treatment of an underlying cause can help resolve clinical signs of pericardial effusion. This may include pharmaceuticals, although a pericardio-

centesis or pericardectomy may be indicated if infection and/or neoplasia are present [9].

Epicardial steatosis, or lipid deposition over the surface of the heart is well documented in humans, and is associated with obesity. However, its relationship with other aspects of cardiovascular disease in birds is unknown [52].

Atherosclerosis

Atherosclerosis is a diffuse or local degenerative condition of the internal and medial tunics of the walls of muscular and elastic arteries [9,16]. Calcium deposits may be present, causing an increased radiodensity and widening of the aorta in affected birds on radiographs. These luminal changes result in decreased elasticity, variable degrees of lumen narrowing, aneurysmal ballooning of the vessel wall and increased thrombogenic potential [16,94]. Atherosclerotic lesions in the aorta and brachiocephalic trunk leading to lumen narrowing and decreased compliance may cause increased afterload, which ultimately may lead to left heart failure [52].

Atherosclerotic lesions are found in various vessels, but are most commonly found in the abdominal aorta and brachiocephalic trunk [9,94]. A gender susceptibility for female birds has been suggested, but may be variable [9,15]. Infection with Marek's disease in chickens induces atherosclerosis [95]. It has been reported that birds fed a diet low in vitamin A have the highest incidence of coronary atherosclerosis and the most severe aortic atherosclerosis [95].

In a retrospective study of pet, exotic, and wild birds presented for pathologic examination, atherosclerotic lesions were present in 53 of 12,072 birds [16]. In that study, the prevalence of atherosclerosis increased in birds over 5 years old. Additionally, Amazon parrots (especially Blue-fronted Amazon parrots, *Amazona aestiva*) seem to be specifically prone, especially if allowed to become obese [16,18].

Cases of atherosclerosis are most commonly associated with sudden death [9,16]. Reported subtle and intermittent signs include dyspnea, weakness, and neurologic signs. These signs may be attributed to decreased blood flow to the central nervous system or peripheral tissues [14,16,96]. Biochemical analysis may reveal elevated plasma cholesterol [9]. Radiographs may reveal increased density and size of the right aortic arch [9]. An aneurysm of the right coronary artery in a 16-year-old male white cockatoo (*Cacatua alba*) was diagnosed using echocardiography, confirmed using nonselective angiography, and was found to be associated with atherosclerosis on necropsy [95].

Congestive heart failure

As in mammals, congestive heart failure in birds is an end-stage condition associated with inappropriate fluid flow dynamics from chronic inadequate

cardiac output that is unable to meet the demands of the body. This condition may be caused by numerous etiologies [9].

The pathophysiology of congestive heart failure involves both backward and forward failure. Chronic heart failure can occur secondary to failure of either or both ventricles. Concurrent failure of both ventricles is common because failure of one ventricle quickly leads to failure of the other. Pulmonary edema secondary to increased pulmonary venous pressure is the major finding in isolated left ventricular disease. Because birds have a constant-volume parabronchial lung, pulmonary vascular resistance increases more with increases in cardiac output when compared to mammals [97]. Avian lungs are rigid and nondistensible; thus, the pulmonary vasculature is not able to expand to accommodate an increase in blood flow [97]. Additionally, because the plasma colloid osmotic pressure in birds can be less than half of the value of mammals while the capillary pressures remain the same, the effect of volume loading on the accumulation of extravascular water in the lung interstitium is greater in birds than in mammals. As a result, pulmonary arterial hypertension develops quickly and leads to right ventricular pressure overload. Chronic passive congestion of the liver, ascites, and systemic edema due to increased portal hydrostatic pressure are potential sequelae of right ventricular disease [26]. The muscular right AV valve thickens with the right ventricle secondary to an increased workload, which may predispose birds to insufficiency of the right AV valve and right-sided heart failure [9].

Clinical findings of right-sided heart failure include hepatomegaly, splenomegaly, ascites, and subsequent dyspnea. In left-sided heart failure, predominant signs include pulmonary edema and congestion, pericardial effusion, dyspnea, lethargy, exercise intolerance, coughing, syncope, weight loss, and episodic weakness [96]. Once congestive heart failure has been diagnosed, the prognosis for long-term survival is usually guarded.

Treatment

Therapeutic regimens for cardiac disorders in avian patients have not been well established. Currently, most therapeutic regimens are extrapolated from protocols developed for other companion animals. A number of human cardiac disease treatment studies use avian species as models, which show promise for treatment of avian cardiac disease. Ideally, treatment should be based on etiology, but if it is unknown, treatment should be geared towards alleviating clinical signs.

Diuretics

Diuretic therapy is indicated where volume overload is a problem and should be instituted when respiratory distress from pulmonary edema or excessive coelomic fluid is apparent. Furosemide may be initiated at a

dosage of 1–2 mg/kg every 12 to 24 hours [98]. A dosage of 2.2 mg/kg every 12 hours has been reported in an Indian Hill Mynah [61]. Lories tend to be sensitive to diuretic therapy and may be easily overdosed [12]. Response to therapy is usually rapid. The patient should be weighed daily to evaluate the degree of fluid loss. Side effects of diuretic treatment may include anorexia, vomiting, hypovolemia, and hypokalemia [99]. In the authors' experience, spironolactone, which is potassium sparing, has been successfully used at a dosage of 1–2 mg/kg orally every 12 to 24 hours for both psittacine birds and chickens. Treatment is initiated with furosemide, and once the patient is stable, spironolactone is added and the dose of furosemide is then decreased. Electrolyte levels (especially potassium) should be carefully monitored in all patients undergoing diuretic therapy [99].

Cardiac glycosides

Cardiac glycosides are indicated in cases of chronic heart failure, especially when accompanied by atrial fibrillation. Arterial pressure, cardiac output, and stroke volume are increased towards normal, while venous pressure is decreased. Treatment with glycosides may be contraindicated when ventricular tachycardia is occurring due to their arrhythmogenic side effects [9]. Signs of toxicity include cardiac arrhythmias and gastrointestinal signs including anorexia [9,100].

There have been digoxin protocols and pharmacokinetic studies performed in birds [61,101–103]. A dosage of 0.02 mg/kg every 24 hours was considered safe and produced satisfactory plasma levels of digoxin in budgerigars [101]. However, signs of toxicosis were seen in Indian Hill Mynah at this dosage [61]. Case management should include regular measurement of serum digoxin levels. The therapeutic range for serum digoxin in mammals is 0.8 to 2.4 ng/mL, and may be used as a guideline [61,100,101].

ACE inhibitors and beta blockers

ACE inhibitors have not been adequately studied in birds, but do have effects that may make them useful in the treatment of avian heart disease in the future. Although the renin–angiotensin system in birds is slightly different than in mammals, ACE inhibitors do have measurable effects [104]. For example, a study in Japanese quail demonstrated that captopril decreases intake of drinking water [105]. As in mammals, it is important to monitor electrolyte levels in birds treated with ACE inhibitors [106].

A study of the effects of beta blockers and ACE inhibitors on blood pressure in roosters revealed that beta-blockers (propranolol, atenolol, and practolol) decreased mean arterial pressure and heart rate. The ACE inhibitor did not cause a dramatic decrease in mean arterial pressure [10]. A study evaluating the effects of the beta-blocker carteolol on broad-breasted white turkey poults (*Meleagris gallopavo forma domestica*) with furazolidone

induced dilated cardiomyopathy showed an increased survival time, reversal of contractile abnormalities and induction of cardiac cellular remodeling [107]. In furazolidone-treated turkeys given propranolol, myocardial morphology and morphometry were maintained, thereby preventing the structural sequelae of furazolidone-induced cardiomyopathy. Digoxin did not show any protective effects in this study [108]. High blood pressure and high catecholamine levels play a role in the development of vascular hypertrophy in naturally hypertensive turkeys. Labetalol, an alpha and beta blocking agent, significantly lowered blood pressure and heart rate and minimized tunica media thickening in a group of turkeys [109].

A comparison of cardioprotective effects of chronic administration of a beta 1-selective antagonist (atenolol), nonselective beta antagonist (propranolol), alpha-receptor antagonist (phenoxybenzamine), and two calcium channel blockers (nifedipine and verapamil) was undertaken in turkey poults fed furazolidone. Maximum cardioprotection was found when the poults were treated with either propranolol or nifedipine. All other agents were more protective than phenoxybenzamine [110]. Further studies are needed to evaluate long-term safety of the use of beta blockers in birds.

Calcium channel blockers

Calcium channel blockers in avian cardiac patients have not been fully evaluated, but show promise for future use. Pranidipine, a dihydropyridone calcium antagonist (calcium channel blocker), prevented progressive left ventricular dilatation and wall thinning in broad-breasted white turkey poults (*Meleagris gallopavo forma domestica*) fed furazolidone to induce dilated cardiomyopathy. These turkeys also showed significantly smaller left ventricular dimensions [87].

Atherosclerosis treatment

Although there have been numerous studies evaluating therapeutics for use in humans with atherosclerosis, an effective therapy for birds with atherosclerosis has not been developed. Treatment is aimed at relieving clinical signs with diuretics, cardiac glycosides, and nutritional supplements such as carnitine [52]. Because diet and activity may play a large role in the development of atherosclerotic lesions, providing a balanced diet and the ability to exercise are indicated for the prevention of these cases [52,111,112].

A study evaluating atherosclerosis development in cholesterol fed roosters infected with Marek's disease virus revealed that pravastatin, a 3-hydroxy-3-methylglutaryl-coenzyme A reductase inhibitor, and quinapril, an angiotensin converting enzyme inhibitor, each decreased areas of plaque formation in study birds. However, these drugs had no effect on the lymphocytic infiltration of the vessels typically seen with Marek's disease [112]. Studies evaluating the use of beta blockers such as labetalol in turkeys, have shown them to have a preventative effect against the development of lesions

in predisposed birds and may become more widely used after further testing [109].

Supportive and symptomatic treatment

Other symptomatic therapy has been used to support affected birds. Once inflammatory disease had been ruled out, theophylline (2 mg/kg every 12 hours) and guaifenesin (0.8 mg/kg every 12 hours) were used for their bronchodilation effects and mild diuresis in a dyspneic severe macaw (*Ara severa*) with right heart enlargement [52].

In general, supportive care, including the judicious use of fluids, nutritional therapy, and vitamin supplementation should be instituted when any cardiac disorder is diagnosed. Dietary modification may include sodium restriction, supplementation of vitamins and amino acids, or a shift to a low-fat, higher fiber diet for weight loss. Care should be taken when modifying the diet for weight loss to keep the rate of loss between 1 to 3% of the body weight per week [113].

Conclusion

This article is intended to be as up to date as possible. However, in light of the expanding knowledge base of avian cardiology, new data will continually become available. By constantly gaining awareness of current information regarding variable clinical manifestations, diagnostic capabilities, and available treatment options for avian cardiac disease, the practitioner may provide optimum care for affected birds.

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