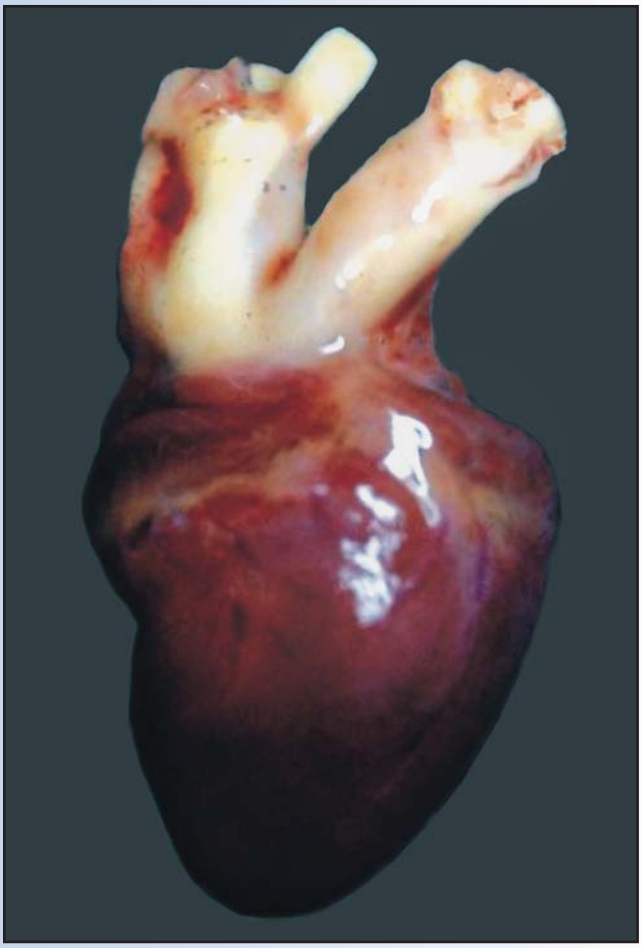
CHAPTER
12

Evaluating and Treating the Cardiovascular System

MICHAEL PEES, Dr med vet, German vet spec for birds/reptiles;
MARIA-ELISABETH KRAUTWALD-JUNGHANNS, Prof Dr med vet, Dr habil, Dipl ECAMS, German vet spec for birds/reptiles;
JENS STRAUB, Dr med vet, German vet spec for birds/reptiles



Anatomical Considerations

The avian heart is located within the cranial part of the thoracoabdominal cavity parallel to the spine in close proximity to the sternum. No diaphragm is present and the apex of the heart is surrounded by the liver lobes. The pericardium covers the heart and normally encloses a small quantity of fluid within the pericardial space.

The anatomy of the avian heart is similar to the mammalian heart, although some morphological peculiarities exist. As in mammals, it is four chambered and functionally divided into the right and the left sides each consisting of an atrium and a ventricle. The right ventricle is sickle-moon shaped and surrounds the left ventricle. The wall thickness of the ventricles changes from the base to the apex of the heart (for measurements see Table 12.6). The triangular right atrioventricular (AV) valve is muscular and unlike the right atrioventricular valve in the mammalian heart, does not contain *chordae tendinae*. Contraction assists emptying of the right ventricle during systole. In some species, the right cranial and the caudal *V. cava* form a *Sinus venosus* that is separate from the right atrium. In contrast, the ascending aorta curves to the right in birds rather than the left side of the body in mammals.[2,25,26,41,44,45,53]

Diagnostics

Diagnosis of cardiovascular diseases in cage and aviary birds is complicated by several factors. Only a few systematic investigations on post mortem diagnosis of cardiac diseases have been conducted.[4,24] All descriptions of ante mortem diagnostics are case reports, based on individual experience. Documented normal reference values are rare. Clinical signs of cardiac disease in birds are often nonspecific and may be accompanied by other concurrent disease conditions disguising the clinical picture. Diagnostic techniques in living birds are limited by the size of the patients and high heart rates. Fortunately, within recent years, modern imaging techniques (like echocardiography) have been evaluated in birds and initial reference values for the assessment of cardiac function are now available. Since these techniques require special equipment and experience, they are not commonly performed in practice. Nevertheless, the practitioner should be aware of the diagnostic potential of ultrasound and other imaging techniques in avian cardiac disease.

Birds with acute circulatory problems have to be handled as emergency cases. Handling should be kept to a minimum and diagnostic procedures be carefully selected. The bird should be maintained in an upright position to prevent circulatory failure. Echocardiography may therefore be less stressful in these patients than radiographic examination.

CLINICAL EXAMINATION

The clinical diagnosis of cardiovascular disease in living birds can be difficult. There is no palpable pulse in birds. In addition, auscultation, an important standard technique in mammals, is difficult to interpret in birds.

Birds suffering from cardiac disease are often presented to the veterinarian with a history of weakness and lethargy. In some cases, cardiovascular failure can be suspected on the basis of bluish discoloration of the periorbital skin (especially in African grey parrots) and abdominal distension. Nonspecific symptoms like dyspnea and exercise intolerance may also lead to a tentative diagnosis of a cardiac problem and provide an indication for further diagnostic procedures.

STANDARD RADIOLOGY

Radiology is a commonly performed and well established imaging technique in avian medicine. It may (often by chance) disclose signs of cardiovascular disease and indicate the need for further diagnostics, especially echocardiography. Position, size and shape of the heart and other internal organs as well as the radiodensity of the large vessels should be assessed.

Alterations of cardiac shape and size are often seen as an enlargement of the heart silhouette. This can be caused by different etiologies (eg, hypertrophy, dilatation, pericardial effusion, aneurysm, inflammation or neoplasia). Radiographic differentiation between these etiologies is difficult. In cases of an existing pericardial effusion, the conventional radiograph may reveal cardiomegaly with an irregular cardiac silhouette.

An increased radiodensity of the large heart vessels can be seen on the ventrodorsal projection as roundish shadows superimposed on the base of the heart and in the lateral projections as an enlarged and radiodense aortic shadow. These findings may indicate atherosclerosis, whereas their lack does not prove the absence of pathological alterations.

Furthermore, radiographs may reveal secondary changes in other organs, such as increased radiodensity of the lungs, enlargement of the hepatic silhouette, or ascites in the case of congestive heart failure (see Fig 12.10). Ascites can also mask the radiographic detail of the thoracocoelomic cavity and the air sac shadows may be decreased and/or displaced.

In birds, measurements of the length and width of the cardiac silhouette on radiographs are limited. Initial examinations have been performed in Canada geese and psittacines.[12,46] Measuring the length of the cardiac silhouette is often complicated by superimposition of the apex of the heart on the liver on both the lateral and ventrodorsal projections or the ventral aspect of the heart on the sternum on the lateral projection. Measurements of the width of the cardiac silhouette (see "h" in Fig 12.1) should be performed on the ventrodorsal radiograph with exact superimposition of the keel and the spine. To assess the width of the cardiac silhouette, the measured value should be taken at the level of the maximum width of the cardiac silhouette, and compared to the maximum width of the thorax (see "t" in Fig 12.1). The sternal length should be taken on the radiograph or on the patient using a standard sliding calliper. In medium sized psittacines (approximately 200 to 500 g) the width of the cardiac silhouette should be approximately 36 to 41% of the length of the sternum or 51 to 61% of the width of the thorax respectively. There is a strong correlation between the width of the cardiac silhouette and the width of the thorax.[46] In Canadian geese the width of the cardiac silhouette is 47 to 57% of the width of the thorax.[12] This study also showed that the movements of the thorax due to respiration are not as important as might be expected.

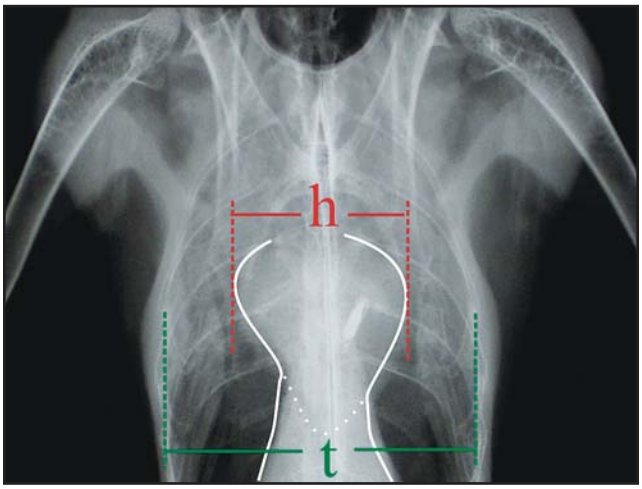


Fig 12.1 | Schematic illustration of the distances to be measured for the evaluation of the avian heart on ventrodorsal radiographs (h = maximum width of the heart size, t = width of the thorax at the level of the maximum width of the cardiac silhouette (microchip can be seen in the left pectoral muscle).

ANGIOCARDIOGRAPHY

Although few reports about the use of angiocardiography in birds exist, the authors' initial experiences show a high potential value for its use in diagnosing cardiac problems. Angiocardiography cannot replace echocardiography but it might give additional information, especially in birds with insufficient detail demonstrated on echocardiographic examination. Examination of the heart vessels is another indication for angiocardiography. In one report, an aneurysm of the right coronary artery was demonstrated in a white cockatoo (*Cacatua alba*) using angiocardiography.[52]

Angiocardiography should be performed under anaesthesia. A venous catheter is placed within the jugular or basilic vein. Iodinated contrast media (eg, Iopamidol, 510 mg/ml adequate 250 mg iodine per ml) is used according to mammalian angiocardiography, but the dosage in birds (2-4 ml/kg of pamidol ie, 1,020-2,040 mg body mass) is twice as high as commonly used in mammals. Administration rate in medium sized birds should be approximately 1 to 2 ml of contrast medium per second.

Due to the rapid heart rate in birds, assessment of contractility may be difficult to evaluate, but hypertrophy (ventricles), dilatation (ventricles, atria), stenosis (vessels, valves) and aneurysm (vessels) can be detected (see Fig 12.2).

ECHOCARDIOGRAPHY

In veterinary medicine, echocardiographic examination has become a very important diagnostic tool, and is indicated for assessment of cardiac function and the structure of the heart. For a long time, due to anatomical peculiarities (especially the position of the airsacs),

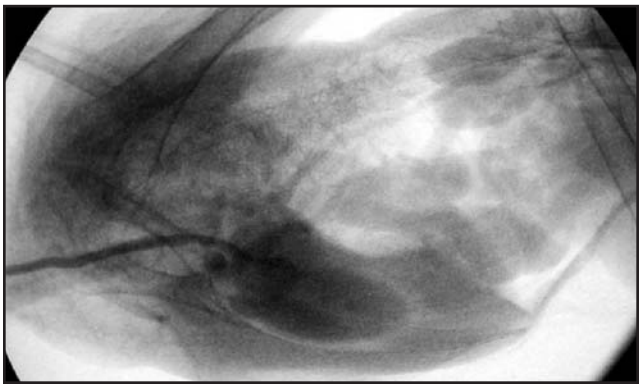


Fig 12.2 | Angiocardiography: filling of the heart in a common buzzard (*Buteo buteo*).

echocardiography has been considered inaccurate and difficult in birds. Bone and air block the passage of ultrasound waves. Standardized views and protocols for mammals recommended by the American College of Veterinary Internal Medicine[50] cannot be used in birds and comparison of the measurements with those in mammal or human medicine are not valid.[15] However, in recent years, case reports regarding the use of echocardiography for diagnosis of cardiac disease in birds have demonstrated the potential of this diagnostic procedure.[29,30,47,52] Initial studies have been conducted and standardized protocols for echocardiographic examination in avian patients have been established.[15,34,46,47]

Improved technology has produced ultrasound equipment that is effective and affordable for avian echocardiography. Parameters of these machines should include
1) minimum 100 frames/second
2) Doppler function
3) microcurved or phased array probes
4) minimum 7.5 MHz frequency

On avian patients that are tolerant of restraint, echocardiography can be performed awake; stress-sensitive birds should be anesthetized.

Because the food-filled gastrointestinal tract might be interposed between the scanner and the heart ideally the GI-tract should be empty. Therefore it is recommended to fast the bird before ultrasound examination. Granivorous birds should be fasted for about 2 (small psittacines) to 12 (pigeons) hours. Raptors should generally be fasted for a longer period.

Echocardiographic measurements should be done in relation to the patient's size. Two parameters should be determined: first, the bird's weight (although this might be misleading in seriously ill birds that are emaciated), and second, the length of the sternum, taken on the radiograph or on the patient using a standard sliding calliper. Simultaneous ECG recording and triggering is

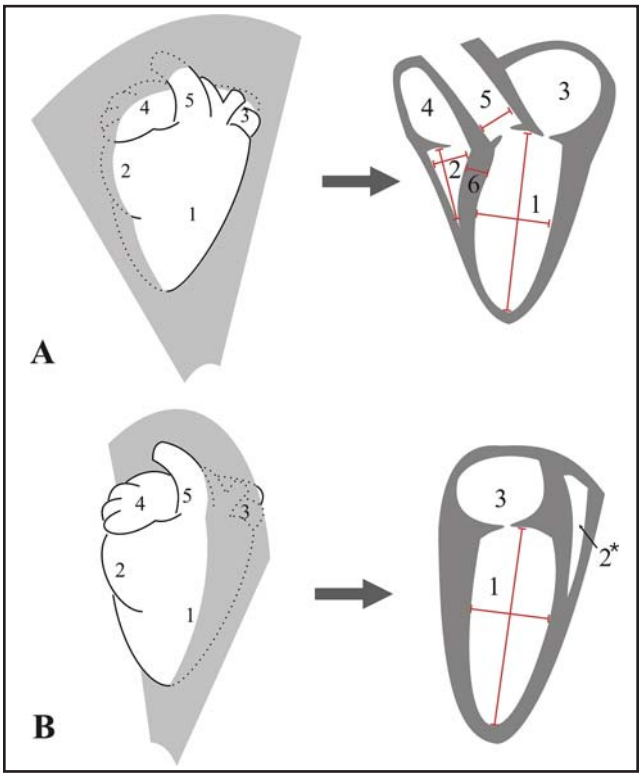


Fig 12.3 | Two-dimensional echocardiography, schematic views: ventromedian approach, horizontal (a) and vertical (b) view. Important measurement points, values see Tables 12.1 and 12.2. (1 = left ventricle, 2 = right ventricle, 3 = left atrium, 4 = right atrium, 5 = aortic root, 6 = interventricular septum, * = no assessment possible in B view)

recommended to allow measurements at defined stages of the heart cycle. For the examination, birds should be held in an upright position in order to minimize stress and its influence on the cardiovascular system.

Because of the avian anatomy, suitable echocardiographic windows to the heart are limited. There are two possibilities: the ventromedian and the parasternal approach.[15] In psittacines and raptors, the ventromedian approach is routinely used. With this approach the homogenous liver tissue serves as an acoustic window. The scanner is placed on the midline directly behind the sternum and the beam plane is directed craniodorsally. In birds with sufficient space between the last ribs and the pelvis (eg, pigeons), the parasternal approach can be used. The transducer is placed on the right side of the body, since in the left part of the thoracoabdominal cavity, the grit-filled ventriculus is present and often makes visualization of the heart impossible. The probe is placed behind the last ribs and the plane is directed craniomedially.

With the ventromedian approach, two longitudinal (comparable to "long axis" in mammal echocardiography) views of the heart are obtained (Fig 12.3). The vertical view (corresponding to the "two-chamber view") shows the heart lying on the inner surface of the sternum. The horizontal view ("four-chamber view") is produced by a counter-clockwise 90 degree rotation of the scanner.

B-mode-echocardiography (2-D-echocardiography)

Due to the fact that only long axis views are possible with the ventromedian approach, M-Mode echo can not be used in birds to evaluate contractility and wall thickness. Therefore, measurements have to be taken on the 2-D-echocardiograph.

Longitudinal Vertical View ("Two Chamber View")

The vertical view shows the left ventricle and the left atrium (which is often not completely distinguishable from the surrounding tissue) as well as the left atrioventricular valve. Sometimes border areas of the right ventricle may also be seen next to the total reflexion of the sternum (Fig 12.4) but cannot be used for assessment. In this view, artifacts caused by reflexions of the sternal bone may produce a second "heart" lying outside the thoracoabdominal cavity (mirror picture artifact).

Longitudinal Horizontal View ("Four Chamber View")

In the four-chamber view, both ventricles, the interventricular septum, both atria, the atrioventricular valves and the aortic root with the aortic valves can be visualized (see Fig 12.4). In this view it is difficult to outline the borders of the atria.

Measurements in 2-D-echocardiography

Measurements have to be taken from 2-D images, since there is no possibility for a suitable cross section for M-Mode technique. Additionally, the images are of very small structures. Therefore the interpretation of measurements is limited and small changes in size may not be detected with the current techniques.

To measure specific cardiac parameters, the position of the transducer must be adjusted until the maximum expansion of the chamber is visible. Measuring points are demonstrated in Fig 12.3. All parameters should be measured in systole and diastole at the point of the widest distance using the inner edge method.[56] Measurements of the left ventricle are possible in both views, but are easier to obtain in the vertical view. In psittacines, there is a strong correlation between the sternal length/the body mass and the length of the left ventricle (LVL); the expected length can be estimated with the formula LVL = 0.5 + 0.33 x sternal length.[32] Measuring the length of the right ventricle is more difficult because it is visible as an acute-angled triangle. Additionally, the muscular right atrioventricular valve is — due to its motility

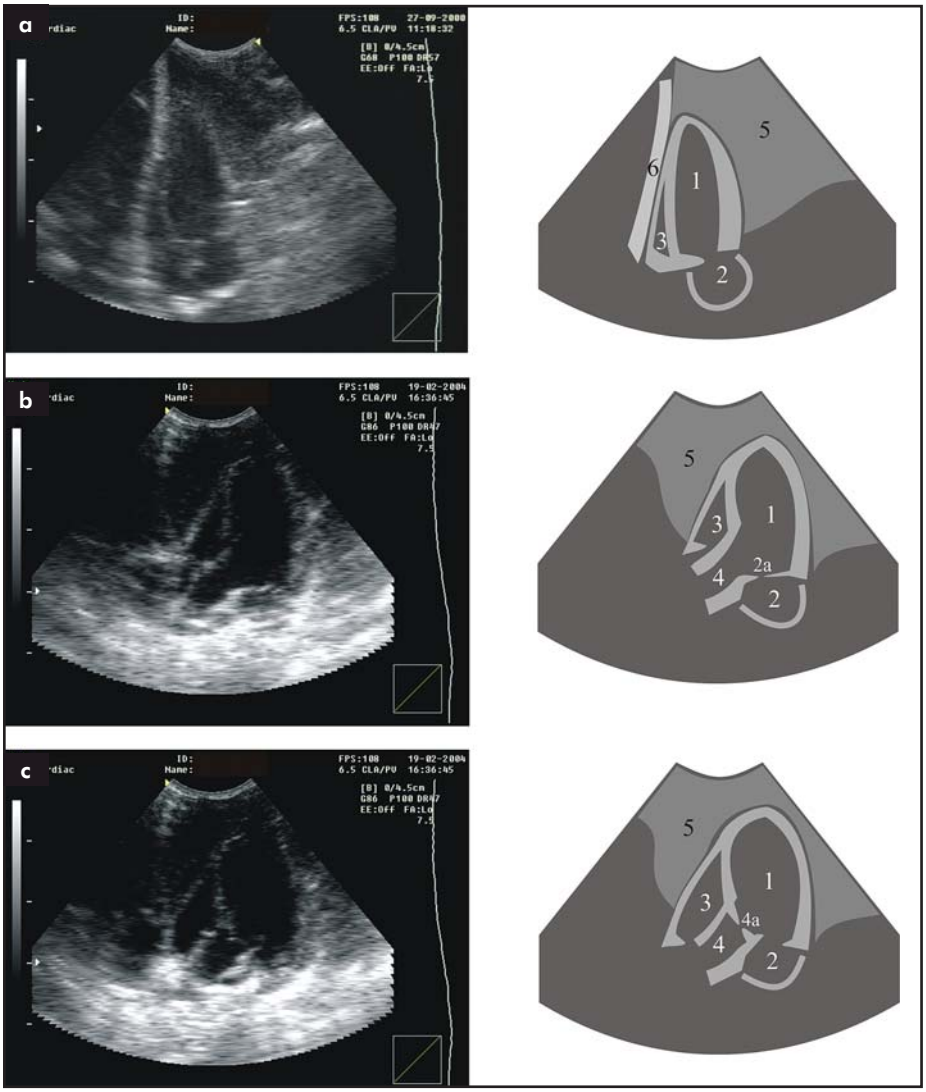


Fig 12.4 | Two dimensional echocardiography, normal hearts. a: vertical view, blue fronted Amazon (*Amazona aestiva*), b: horizontal view, systole, carrion crow (*Corvus corone*), c: horizontal view, diastole, carrion crow (*Corvus corone*). (1 = left ventricle, 2 = left atrium, 2a = left atrioventricular valves, 3 = right ventricle, 4 = aortic root, 4a = aortic valves, 5 = liver tissue, 6 = sternal reflexion).

— an imprecise margin for right ventricular measurement. The outer walls of the heart are often difficult to distinguish from the surrounding tissue. The thickness of the interventricular septum may give information about ventricular hypertrophy. Measurements of the atria are imprecise and therefore of limited use. The importance of atrial contraction for blood flow into the ventricles is limited, therefore assessment may not be important.[42]

Calculations for Functional Assessment (Tables 12.1-12.3)

On the basis of the measurements obtained, the fractional shortening (FS) can be calculated. This is one functional parameter for evaluating the capability of ventricular contraction. It is calculated as the relative reduction of the width of the transverse left ventricular diameter during systole in relation to the diastolic diameter (%FS = (diastole - systole) 100/diastole). The values are different than those in mammals, which might be due to anatomical peculiarities (see above). Contractility of the right ventricle is significantly higher than the value found for the left one.

The relationship of the width of the ventricle to the length can give information about dilatation. For psittacines, a relatively constant value (systolic 0.33, diastolic 0.40) (see Table 12.3) has been noted.[32,33,34]

Volume calculations, based on simplified models of the chamber volume of the left ventricle, have not yet been documented as diagnostic.[34]

Preliminary reference values for morphometry and assessment of cardiac function have been established for psittacines,[32,33,34] birds of prey[3] and pigeons[15] (Tables 12.1-12.3).

Doppler-echocardiography

To date, there only a few reports of Doppler echocardiography in birds. The principles are the same as in mammalian echocardiography, but specific problems exist. The frame rate when color Doppler mode is used is significantly lower in most ultrasound devices. This makes it difficult to assess blood flow. Also, pulsed-wave Doppler has limitations due to the high velocity of flow in the avian heart.

Color-Doppler can be used for the detection of valvular insufficiencies (reported in an Indian Hill Mynah Bird).[39] It is also helpful for the positioning of the gate for spectral Doppler-analysis to determine the degree of valvular insufficiency. In one case, the use of color Doppler for diagnosis of an aneurysm in a coronary artery has been reported.[52] With the standard ventromedian approach, the diastolic inflow into the ventricles is displayed red (flowing towards the transducer) and the systolic outflow blue (flowing away from the scanner) (see Fig 12.5).

Table 12.1 | Longitudinal and Transverse Diameter of the Left Ventricle in Systole and Diastole (mm)*

	LVLS^{V}	LVLD^{V}	LVTS^{V}	LVTD^{V}	LVLS^{H}	LVLD^{H}	LVTS^{H}	LVTD^{H}
Psittacus erithacus erithacus	22.2 ± 1.9	23.9 ± 1.9	7.0 ± 1.1	9.1 ± 1.5	22.5 ± 1.9	24.0 ± 1.9	6.8 ± 1.0	8.6 ± 1.0
Psittacus erithacus timneh	18.4 ± 1.9	19.5 ± 1.9	6.9 ± 0.8	8.9 ± 1.4	17.9 ± 2.4	18.6 ± 3.0	5.9 ± 0.2	7.6 ± 1.3
Amazona spp.	20.7 ± 1.5	21.8 ± 1.9	6.7 ± 1.1	8.7 ± 1.2	21.1 ± 2.3	22.1 ± 2.2	6.7 ± 1.2	8.4 ± 1.0
Cacatua spp.	18.9 ± 1.7	19.4 ± 1.8	6.6 ± 1.7	8.8 ± 1.8	19.0 ± 1.3	19.9 ± 1.6	6.4 ± 1.7	8.3 ± 1.5
Poicephalus s. senegalus	14.5 ± 1.1	15.1 ± 1.0	5.2 ± 0.9	6.9 ± 1.0	14.4 ± 1.2	15.1 ± 2.0	4.6 ± 0.3	5.9 ± 0.5
Diurnal raptors (male)	17.7 ± 1.2	19.3 ± 1.6	6.6 ± 0.9	7.5 ± 1.0	14.7 ± 2.0	16.5 ± 1.8	6.1 ± 0.8	7.4 ± 1.0
Diurnal raptors (female)	18.2 ± 4.7	20.1 ± 5.2	7.7 ± 1.8	8.9 ± 2.1	14.7 ± 4.5	16.3 ± 4.5	6.8 ± 1.7	8.3 ± 1.8
Pigeons (parasternal approach)	—	—	—	—	17.9 ± 1.0	20.1 ± 1.4	5.2 ± 0.4	7.4 ± 0.6

*Psittaciformes according to Pees,[32,33,34] birds of prey according to Boskovic,[3] pigeons according to Krautwald-Junghanns[15]
V = vertical view; H = horizontal view
LVLS = left ventricle, longitudinal diameter systole; LVLD = left ventricle, longitudinal diameter diastole;
LVTS = left ventricle, transverse diameter systole; LVTD = left ventricle, transverse diameter diastole

Table 12.2 | Longitudinal and Transverse Diameters of the Right Ventricle in Systole and Diastole (mm)*

	RVLS^{H}	RVLD^{H}	RVTS^{H}	RVTD^{H}	IVSS^{H}	IVSD^{H}	AOS^{H}	AOD^{H}
Psittacus erithacus erithacus	9.2 ± 1.4	11.5 ± 1.9	2.8 ± 0.9	4.8 ± 1.1	2.9 ± 0.5	2.5 ± 0.3	3.6 ± 0.4	4.0 ± 0.6
Amazona spp.	9.4 ± 1.8	10.3 ± 1.3	3.1 ± 0.7	5.2 ± 1.3	2.2 ± 0.1	2.1 ± 0.4	3.0 ± 0.5	3.4 ± 0.6
Cacatua spp.	10.3 ± 1.2	11.3 ± 2.3	2.3 ± 0.0	3.5 ± 0.5	1.9 ± 0.3	1.7 ± 0.4	—	—
Poicephalus s. senegalus	7.5 ± 1.1	7.6 ± 0.2	2.5 ± 0.4	3.3 ± 0.3	1.9 ± 0.3	1.7 ± 0.2	2.5 ± 0.3	2.4 ± 0.0
Diurnal raptors (male)	12.6 ± 1.9	13.8 ± 1.8	2.1 ± 0.5	2.5 ± 0.7	1.8 ± 0.4	1.9 ± 0.4	—	2.7 ± 0.4
Diurnal raptors (female)	13.0 ± 4,6	14.2 ± 4.2	2.2 ± 0.8	2.5 ± 1.1	2.0 ± 0.8	2.0 ± 0.7	—	2.9 ± 0.4
Pigeons (parasternal approach)	—	9.9 ± 0.8	—	4.0 ± 0.5	3.8 ± 0.1	3.3 ± 0.2	—	3.0 ± 0.1

*Mean value ± standard deviation (Psittaciformes according to Pees,[32,33,34] birds of prey according to Boskovic[3], pigeons according to Krautwald-Junghanns[15])
H = horizontal view
RVLS = right ventricle, longitudinal diameter systole; RVLD = right ventricle, longitudinal diameter diastole; RVTS = right ventricle, transverse diameter systole; RVTD = right ventricle, transverse diameter diastole; IVSS = interventricular septum, thickness systole; IVSD = interventricular septum, thickness diastole; AOS = aorta, diameter systole; AOD = aorta, diameter diastole

Table 12.3 | Calculated Parameters in Psittacines*

	Relation Width to Length of the Ventricles				Fraction Shortening %	
	LVS^{V}	LVD^{V}	RVS^{H}	RVD^{H}	LVT^{H}	RVT^{H}
Psittacus erithacus erithacus	0.32 ± 0.05	0.39 ± 0.07	0.31 ± 0.08	0.43 ± 0.1	22.6 ± 4.4	40.8 ± 11.9
Amazona spp.	0.32 ± 0.04	0.40 ± 0.05	0.35 ± 0.11	0.51 ± 0.13	22.8 ± 4.2	34.1 ± 3.7
Cacatua spp.	0.35 ± 0.08	0.45 ± 0.09	0.23 ± 0.03	0.32 ± 0.07	25.6 ± 7.0	33.3 ± 10.3
Poicephalus s. senegalus	0.36 ±0.06	0.46 ± 0.07	0.30 ± 0.07	0.44 ± 0.04	24.9 ± 3.1	37.1
All examined psittacines	0.33 ± 0.05	0.4 ± 0.07	0.31 ± 0.08	0.43 ± 0.1	23.1 ± 4.6	39.6 ± 11.4

*Mean value ± standard deviation (Pees[32,33,34])
V = vertical view; H = horizontal view
LVS = left ventricle systole; LVD = left ventricle diastole; RVS = right ventricle systole;
RVD = right ventricle diastole; LVT = left ventricle, transverse diameter; RVT = right ventricle, transverse diameter

Spectral Doppler-echocardiography is the standard method for noninvasive blood flow velocity measurements in mammalian medicine. Signals are displayed as two-dimensional time versus velocity graphs. Two different types of spectral Doppler are of interest: pulsed-wave (PW) and continuous-wave (CW). PW Doppler is limited to velocities lower than 2 m/second but allows measurements within an operator-specified sample volume or "gate". Diastolic inflow into the left and the right ventricle, as well as systolic aortic outflow, have been measured in psittacines and raptors using PW Doppler (Fig 12.6).[5,43,47,49] For the mitral inflow, two peaks could be recorded (E-wave corresponding to ventricular filling, A-wave to atrial contraction); E:A was positive for macaws and African grey parrots, whereas in cockatoos it was negative.[5] Blood flow within the pulmonary artery could only be differentiated in a few cases.[5] Measurements are given in Table 12.4.

CW Dopplers are useful for the detection of very high blood flow velocities but do not allow the measurement of the flow velocity at a certain point. No studies regarding the use of CW Dopplers in birds have been published so far.

Birds under stress significantly increase their intracardial blood flow velocity. It is recommended that Doppler-echocardiography be performed under anesthesia in these patients.

ELECTROCARDIOGRAPHY (ECG)

Although electrocardiography was the first

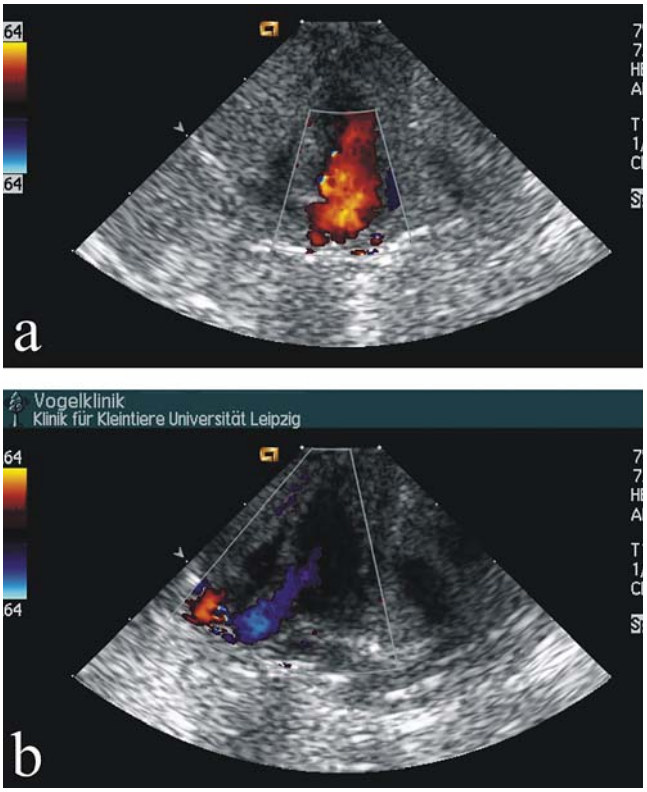
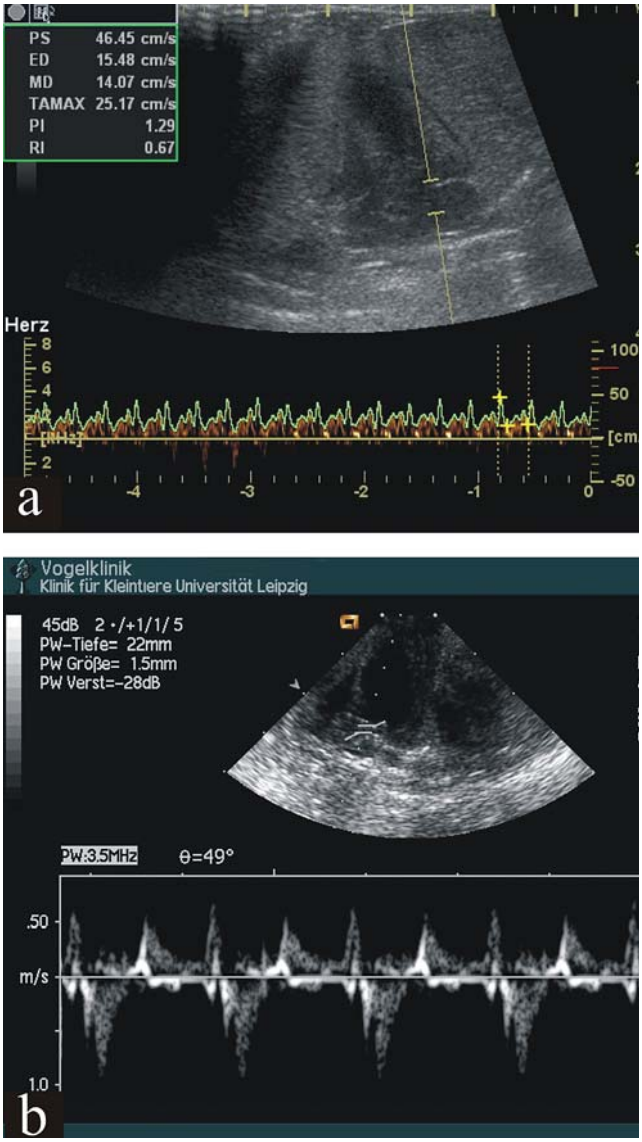


Fig 12.5 | Color-Doppler echocardiography, carrion crow (*Corvus corone*): **a)** Filling of the left ventricle (red inflow). **b)** Systolic outflow into the aorta (blue). Red colored areas are caused by fluid moving towards the transducer, blue areas show movement away from the transducer.

described technique for ante mortem diagnosis of cardiac diseases, it is used less frequently in birds compared to mammals. Difficulties may occur with the connection of the leads to the skin and stress may cause alterations of the recorded ECG. Additionally, no reference values have been published for many of the bird species.

The main indication for ECG is diagnosis and control of arrhythmias and conduction disorders. It may also be valuable for detecting enlargement of the ventricles and metabolic disorders.[38] ECG can be used for monitoring cardiac function during anesthesia and for recording cardiac stages (systole, diastole) while performing other imaging techniques (eg, echocardiography). Another indication for performing ECG is monitoring cardiac disease therapy.

There are many reports on how to perform ECGs in birds. Some authors recommend examination under anesthesia, others prefer recording the ECG in the awake bird. Anesthesia may induce alterations in the ECG. With isoflurane anesthesia, arrhythmias have been described, such as second- and third degree AV-block, sinus arrest, T-wave depression and atrial premature contraction.[1]



I. Kiefer

Fig 12.6 | Pulsed wave Doppler echocardiography: **a)** Diastolic inflow into the left ventricle, channel-billed toucan (*Ramphastos vitellinus*). **b)** Systolic aortic blood flow, carrion crow (*Corvus corone*).

Table 12.4 | Doppler Derived Intracardial Blood Flow Velocities in Birds*

Species	Diastolic Inflow Left Ventricle (m/s)	Diastolic Inflow Right Ventricle (m/s)	Systolic Outflow Aortic Root (m/s)
Amazona spp.[A,43,48,49]	0.18 ± 0.03	0.22 ± 0.05	0.83 ± 0.08
Cacatua galerita[A,5]	0.32 ± 0.15	—	0.78 ± 0.19
Psittacus erithacus[A,5]	0.39 ± 0.06	—	0.89 ± 0.13
Ara sp.[A,5]	0.54 ± 0.07	—	0.81 ± 0.16
Buteo buteo[A,43,48,49]	0.14 ± 0.01	0.14 ± 0.02	1.18 ± 0.05
Buteo buteo[C,43,48,49]	0.22 ± 0.03	0.19 ± 0.03	1.36 ± 0.16
Parabuteo unicinctus[C,43,48,49]	0.19 ± 0.03	0.21 ± 0.03	1.09 ± 0.17
Tyto alba[C,43,48,49]	0.2 ± 0.03	0.22 ± 0.06	1.08 ± 0.12
Falco spp.[C,43,48,49]	0.21 ± 0.03	0.21 ± 0.04	0.95 ± 0.07
Falco spp.[C,43,48,49]	0.28 ± 0.05	0.27 ± 0.05	1.25 ± 0.09

*Straub;[43,48,49] Carrani[5]
A = anesthetized
C = accustomed to handling, conscious

For the clinical application of an ECG in avian patients, six leads, as commonly performed in mammals, can be used. Due to the high cardiac heart rate, electrocardiographs in avian medicine must be able to run at a paper speed of at least 100 mm/second. Leads are attached on the right wing (RA), left wing (LA) and left leg (LL), and the right leg is connected to the ground (Lumeij and Ritchie, 1994). If the ECG is used to monitor anesthesia or to trigger echocardiographic images, bipolar leads (following the electrical heart axis; with one attached cranially to the sternum and slightly paramedian on the right side of the body, and the other one attached caudally to the sternum, slightly on the left side) may be sufficient.

Several techniques for attachment of the leads to the skin have been described (subcutaneous needles, alligator clips, specially constructed feather clips). In the authors' experience, alligator clips can be effectively used in birds, when clipped on the proximal part of the rachis of the feathers. By using alligator clips without sharp teeth there is no damage to the feathers. To get electrical contact with the skin, water soluble ECG gel or small amounts of alcohol are applied between the skin and clip.

The avian ECG is quite different from the mammalian ECG. A schematic lead II-ECG-complex typical for a healthy bird is shown in Fig 12.7. Reference values exist for racing pigeons,[21] Amazons and African grey parrots[23] and some macaw species.[6] A complete overview of the electrophysiologic evaluation of the avian heart and the clinical use of the ECG in birds is given by Lumeij and Ritchie.[22]

POST MORTEM MORPHOMETRY

Whilst reproducible measurements of the thickness of the atrial myocardium are nearly impossible, the thickness of the ventricular myocardium is readily detectable. Due to the huge number of different species that are seen in avian practice, the establishment of reference values for each species is impossible. Fortunately, current investigations could prove that relative values (as a %) rather than absolute values (in mm) are of significance in the assessment of the avian myocardium.

Dissection of the heart under standardized conditions is a basic requirement for performing measurements of the ventricular myocardium thickness at necropsy. The heart should be placed on its left side on a cutting board. A longitudinal cut running through the apex of the heart, the uppermost region of the right ventricular wall and the center of the interventricular septum has to be performed. All blood clots must be removed and the mass of the heart should be determined. Measurements of the thickness of the myocardium at three regions of the left

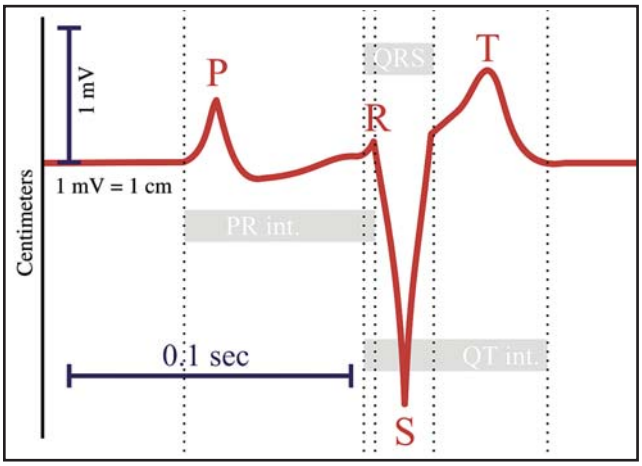


Fig 12.7 | Schematic view of a normal ECG-complex, lead II, in a healthy macaw. Measurement points following Lumeij.[22]

and the right ventricular free wall as well as the interventricular septum should be made using precision callipers. Additionally, measurements of the length of the left ventricle should be ascertained (Fig 12.8).

To date, the amount of scientific data concerning the morphometry of the avian heart is limited. Scientific studies have been performed in two psittacine species (*Melopsittacus undulatus, Alisterus scapularis*) and the common buzzard (*Buteo buteo*).[44,45] Interestingly, by comparing the relative values (thickness of the myocardium in relation to the length of the sternum and the length of the left side of the interventricular septum, respectively) of these different species, it is speculated that the hearts of most avian species follow a set morphological pattern. The myocardium of the ventricles as well as the interventricular septum shows changes of the thickness from the base to the apex of the heart. Whilst the thickness of the left ventricular free wall decreases in the direction of the apex the interventricular septum and the right ventricular free wall become thicker from the base to the middle region and then decrease in thickness towards the apex (Fig 12.8 and Table 12.6).

Therapy of Cardiovascular Diseases

The therapy of avian cardiac disease is still in its infancy. Only a few scientific studies exist, and many drugs routinely used in mammals have not as yet been tested in caged and aviary birds. Overdosing may cause severe side effects; and the pharmacodynamics of drugs in birds is often different due to physiological differences.

Nevertheless, the principles of cardiac therapy are the same as in mammal medicine. Stabilization of the

Table 12.5 | Reference Values for Electrocardiograms in Selected Avian Species*

Parameter	*Columbia* sp.[21]	*Psittacus erithacus*[22]	*Amazon* sp.[22]	*Macaw* sp.[23] Small sp.	*Macaw* sp.[23] Large sp.
	Inner limits for $P_{2.5}$ and $P_{97.5}$ with a probability of 90%,			Mean value ± standard deviation	
Heart rate (1/min)	160-300	340-600	340-600	389 ± 85	275 ± 72
Heart axis (°)	(-83) - (-99)	(-79) - (-103)	(-90) - (-107)	-97 ± 5	-98 ± 8
P-wave duration (s)[M]	0.015-0.02	0.012-0.018	0.008-0.017	0.016 ± 0.002	0.019 ± 0.002
P-wave amplitude (mV)[M]	0.4-0.6	0.25-0.55	0.25-0.60	0.34 ± 0.11	0.24 ± 0.05
PR interval duration (s)[M]	0.045-0.07	0.04-0.055	0.042-0.055	0.050 ± 0.010	0.053 ± 0.009
QRS complex duration (s)[M]	0.013-0.016	0.01-0.016	0.01-0.015	0.017 ± 0.002	0.018 ± 0.002
R amplitude (mV)[M]	0-0.5	0-0.2	0-0.65	0.04 ± 0	0.08 ± 0.04
S amplitude (mV)[M]	1.5-2.8	0.9-2.2	0.7-2.3	0.624 ± 0.234 (QRS-ampl.)	0.624 ± 0.234 (QRS-ampl.)
T amplitude (mV)[M]	0.3-0.8	0.18-0.6	0.3-0.8	0.4 ± 0.09	0.25 ± 0.1
QT Interval Duration					
Anaesthetized (s)[M]	—	0.039-0.07	0.038-0.055	0.081 ± 0.006	0.104 ± 0.018
Unanaesthetized (s)[M]	0.06-0.075	0.048-0.08	0.05-0.095	—	—

*Lumeij and Stokhof;[21] Nap;[23] Casares[6]
M = measurements taken in lead II

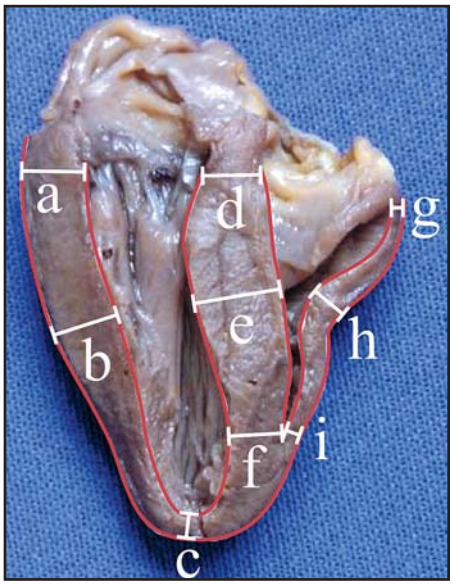


Fig 12.8 | Heart of a common buzzard (*Buteo buteo*). Illustration of the measuring points for the post-mortem evaluation of the myocardial thickness: a to c = left ventricular free wall, a = basal, b = middle, c = apical; d to f = interventricular septum, d = basal, e = middle, f = apical; g to i = right ventricular free wall, g = basal, h = middle, i = apical; distance d to c = length of the left ventricle close to the interventricular septum.

Table 12.6 | Approximate Thickness of the Ventricular Myocardium in Relation to the Length of the Sternum and the Length of the Left Ventricle in *Melopsittacus undulatus*, *Alisterus s. scapularis* and *Buteo buteo*

Myocardium	% of Sternal Length			% of the Length of Interventricular Septum*		
	Basal (Point a)	Middle (Point b)	Apical (Point c)	Basal (Point a)	Middle (Point b)	Apical (Point c)
Left ventricle	7-10%	7-10%	2-4%	20-30%	20-30%	5-10%
Interventricular septum	5.5-7.5%	6.5-9.0%	5-7%	12-22%	16-30%	12-21%
Right ventricle	1.5-2.7%	1.8-3.0%	1.3-2.5%	4.5-8.0%	5.0-8.5%	3.5-6.5%

Straub[44,45]
*Measured on the left side of the interventricular septum points a, b, c, etc. are ref. in Fig 12.8.

patient is essential, and besides symptomatic therapy, the underlying disease has to be diagnosed and treated.

Because cardiac disease in birds is often not diagnosed until the heart decompensates, the veterinarian commonly has to deal with advanced changes. Due to weakness, emaciation and high-grade circulatory problems, these birds are often presented as emergencies. Stress during handling, examination and treatment may be fatal.

ACCOMPANYING THERAPY

Accompanying therapy is essential to maintain circulatory stability and organ function. Liver and kidney function may be affected by circulatory problems. The liver may be congested, renal blood flow may be reduced, and the resulting increase of toxic metabolites and urinary excreted substances may affect the general condition of the bird. The function of the lung can be reduced as a direct result from left heart failure causing pulmonary congestion. Fibrotic lungs will increase the work load of the right ventricle. In addition, reduced air sac volume can lead to dyspnea and hypoxemia damaging the heart.

Cardiac therapy's goals are to reduce congestion (diuretics, ACE inhibitors) and improve cardiac output (ACE inhibitors) which can improve renal/hepatic function. The following supportive measures should also be taken:

- Incubation in a warm environment (80° F (25° C) or above), with sufficient air humidity (60% relative humidity or above) in order to improve circulation and to reduce energy loss.
- Reduction of stress: handling should be reduced to a minimum. Positioning of the patient in ventrodorsal recumbency (eg, for radiology) is a risk and should be considered carefully. Diagnostic procedures in an upright position should be performed (eg, echocardiography). The environment should be calm and temporarily darkened.
- Fluid administration is essential to prevent circulatory collapse and shock and to prevent dehydration due to increased diuresis due to therapy with ACE-inhibitors, and furosemide. Therapy should be

started intravenously or via intraosseus canula. Maintenance administration may be subcutaneous or (preferably) orally. Careful monitoring must be done so the patient is not volume overloaded which would increase congestion.
- Addition of electrolytes, vitamins, amino acids and buffer solution may also be indicated.
- Fluid aspiration from the thoracoabdominal cavity is easily performed in avian patients (if possible, do it ultrasound-guided) and helps to reduce dyspnea due to ascites. For pericardiocentesis, see pericardial effusion.

DRUGS FOR CARDIAC THERAPY
(see Table 12.7)

Heart Glycosides (Digoxin)

Heart glycosides have a positive inotropic effect on the contraction of cardiac muscle. Also relaxation of cardiac muscle will be improved and the heart rate will decrease. These effects lead to a reduction of oxygen demand and an improvement in circulation of the cardiac vessels. Therefore digoxin works best for cardiac diseases that involve volume overloads (insufficient valves), decreased contractility (dilatative cardiomyopathy), or supraventricular tachycardia. The increased cardiac function decreases ascites and edema. To date, there are only two scientific studies on the use of glycosides in pet birds. Pharmacokinetics of digoxin has been examined in sparrows (*Passer domesticus*), budgerigars (*Melopsittacus undulatus*), and quaker (*Myiopsitta monachus*).[11,54]

The therapeutic margin of cardiac glycosides is small, and half-life varies greatly between species. Overdosing may lead to accumulation of cardiac glycosides, and side effects include arrythmias. Contraindications are ventricular tachycardia, second and third degree atrioventricular heart block, hypercalcemia, potassium deficiency and stenotic valves. Cardiac side effects are documented with the combination of glycosides and ketoconazole (therapy of aspergillosis) in humans.

In the authors' experience, cardiac glycosides are useful for emergencies. Chronic administration may be problematic due to difficulties in controlling the side effects and plasma levels. An initial recommended dose of digoxin is 0.02 to 0.05 mg/kg (20 to 50 μg/kg) q 12 h. Maintenance should be dosed carefully at 0.01 mg/kg (10 μg/kg) q 12 h.[11,54]

Angiotensin Converting Enzymes (ACE-) Inhibitors (Enalapril)

Angiotensin II is responsible for the constriction of arterial and venous vessels and the retention of sodium and water by the kidneys. The inhibition of this hormone leads to diuresis (and indirectly to an improvement of renal function), and a decrease of blood pressure. The effect on the heart is a decreased pre- and afterload so that cardiac workload is eased and the cardiac muscle cells may be able to recover.

Although scientific studies regarding the pharmacokinetics of enalapril in birds are lacking, clinical experiences with this drug exist. The results indicate that its use in birds with cardiovascular disease may be beneficial. Empirical dosage used for treatment of birds is 5 mg/kg/day, with reduction to 1 mg/kg/day following improvement of cardiac function. Tolerance is much better when compared to cardiac glycosides and long term administration is possible. Observed side effects after high-dose therapy (5 mg/kg/day) were an increase of PCV and signs of dehydration. These effects were not present when the dose was reduced to 1 mg/kg BID orally.[29,47]

Diuretics (Furosemide)

Indications for furosemide are pulmonary edema, ascites, and pericardial effusion as well as an increased pre- and afterload. The recommended dosage used in birds is 0.15 to 2.0 mg/kg/day PO/IM.[36] Long term administration may lead to a potassium deficiency and therefore cause heart arrhythmias. Since there is a risk of dehydration, especially in small birds, additional careful fluid administration is essential.

In the authors' experience, the main use for diuretics is in the initial therapy of cardiac failure with fluid accumulation (ascites, pericardial effusion) in combination with ACE-inhibitors or glycosides.

Antiarrhythmics (ß-blockers)

A protective effect against the development of atherosclerotic plaques has been demonstrated using oxprenolol in poultry (2 mg/kg/day).[27] Other uses, such as for supraventricular or ventricular arrhythmias, have not been described.

Before using antiarrhythmics, it is important to exclude metabolic causes for the arrhythmia (eg, potassium deficiency, see diuretics). The safety margin of these drugs is small, and the half-life is normally very short, but the clinical effects can last longer, especially if cardiac failure is present.

Calcium Sensitizers (Pimobendan)

Calcium sensitizers are substances with a positive inotropic effect. The pharmacokinetic mechanism is unknown and there are no scientific reports of the use of calcium sensitizers in birds.

Table 12.7| Cardiac Medications in Birds*

Drug	Indication	Application / Dose	Remarks / Side effects
Digoxin	Systolic myocardial failure	PO 0.02-0.05 mg/kg BM BID initially, following 0.01 mg/kg BM BID	• Low therapeutic index • Overdosing: bradycardia, arrhythmia, diarrhea, vomitus
Enalapril	Myocardial failure, increased pre-/afterload	PO 2.5 mg/kg BM BID initially, after one week 1 mg/kg BM BID	• High therapeutic index • Side effect: dehydration
Furosemide	Pericardial effusion, edema, increased pre-/afterload	PO, IM 0.15-2.0 mg/kg BM SID/BID	• Risk of dehydration esp. in smaller birds (lorikeets) • Side effect: arrhythmia caused by potassium deficiency
Oxprenolol	Cardioprotection, prevention from arteriosclerotic plaques (poultry)	PO 2 mg/kg BM SID	• Possible side effects: hypotonia, arrhythmia, tachycardia, AV-block
g-Strophantin (only available in EU)	Circulatory system stimulant	PO drop-wise, to effect IV, IM 1 ml/kg BM	• In case of long-time overdosage, heart hypertrophy especially in smaller birds possible
Atropine	Conduction disturbances, bradycardia	IM 0.01-0.1 mg/kg BM SID	• Only short-time • Overdosing: arrhythmia, gastrointestinal stasis
Etilefrine (only available in EU)	Hypotonia	PO drop-wise, to effect	• Possible side effects: tachycardia, arrhythmia, hypertonia

*Hamlin and Stalnaker;[11] Wilson;[54] Lumeij and Ritchie;[22] Ritchie and Harrison;[36] Pees;[29] Krautwald-Junghanns and Kummerfeld.[19]
SID = 1x/day, BID = 2x/day, PO = orally, IM = intramuscular, BM = body mass

Pathology of Cardiovascular Diseases

Caged birds, in comparison with free living birds, are frequently compromised by restricted exercise, nutritional deficiencies and abnormal climactic conditions. Combined with the bird's natural physiologically high blood pressure, the risk factor for cardiovascular disease in pet birds is significant.

Recent post mortem studies show the frequency of cardiac pathology in pet birds. According to these studies, heart disease occurs commonly in avian species. Oglesbee and Oglesbee[24] found gross and histological evidence for cardiac disease in 26 of 269 psittacine birds. A study of 107 psittacines submitted for routine necropsy found macroscopic lesions of the heart and/or the large vessels in more than one third of the birds[4,20,42] (Table 12.8). In 99% of these 107 birds, at least low-grade histologic changes were present (predominantly inflammatory mixed-cellular infiltration, bacterial infiltration, and/or fat cell accumulation within the myocardium).

Diseases of the heart may occur as the result of congenital, infectious, toxic or idiopathic etiologies. No data currently exist on age-related cardiovascular diseases in pet birds. A variety of cardiac abnormalities occur secondarily as acquired disease and/or compensation/decompensation due to other organ failure (ie, lung and liver), neoplasia or systemic infections.[16,17,20]

CAUSES OF (CONGESTIVE) HEART FAILURE

Heart failure is the clinical syndrome resulting from abnormalities of cardiac function (eg, myocardial failure, valvular regurgitation).

Table 12.8 | Incidence of Selected Cardiac and Vascular Diseases in 107 Psittacine Birds. Macroscopic Findings (Findings in 39 Birds (36.4 %)*

	Findings	Cases
Pericardium	Pericarditis	16 (14.9%)
	Pericardial effusion	6 (5.6%)
Myocardium	Hypertrophy/dilatation	16 (14.9%)
	Petechial bleeding	6 (5.6%)
Endocardium	Endocarditis *valvularis*	1 (0.9%)
Aorta/A. *pulmonalis*	Yellow discoloration/hardening	11 (10.3%)

*Braun;[4] Krautwald-Junghanns[20]

The syndrome "congestive heart failure (CHF)" results from inadequate cardiac output and an increased preload. Fluid retention is caused by elevated venous and capillary pressures. In left heart failure, an increase of the pressure in the left atrium and the pulmonary veins leads to pulmonary edema. In birds with right heart failure, ascites, liver congestion and hydropericardium are commonly seen.

CONGENITAL DISEASES

Due to the high physiological load of the avian heart, congenital cardiovascular disorders often lead to early embryonic or fledgling death. They are rarely presented as clinical diseases in birds, ante mortem diagnosis is rare and the cause is normally found only at necropsy. In one case, evidence for a congenital defect of the muscular right atrioventricular valve could be found in a 35-year-old Amazon diagnosed with dilatation of the right ventricle.[30]

PERICARDIAL DISEASES (PERICARDITIS, PERICARDIAL EFFUSION)

Pericardial changes frequently found in birds include inflammation (pericarditis) and effusion (hydropericardium, hemopericardium).

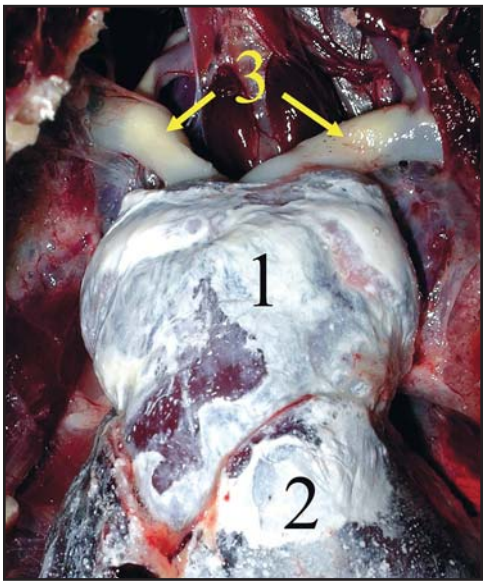


Fig 12.9 | Necropsy, blue fronted Amazon (*Amazona aestiva aestiva*), pericarditis urica. Uric acid deposits can be seen on pericardium (1) that covers the heart and the serosa of the liver (2). The yellowish discoloration of the large vessels (3) is an indication for arteriosclerosis.

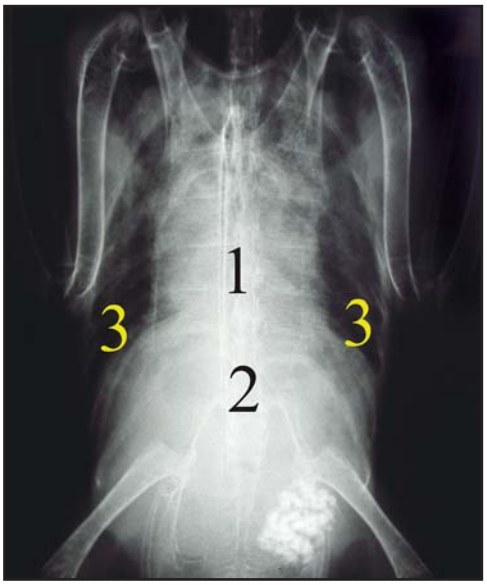


Fig 12.10 | Radiograph, ventrodorsal view, African grey parrot (*Psittacus erithacus*), tentative diagnosis of cardiac disease (corresponding to Fig 12.11). The heart (1) and the liver (2) shadow are enlarged. Detail recognition in the thoracoabdominal cavity is reduced; the air sacs (3) are compressed.

Inflammation of the pericardium may develop in the process of infectious diseases of surrounding tissue. Common causes are generalized trichomonas infections in pigeons, mycotic infections originating from the respiratory tract, and bacterial infections including mycobacteriosis. Deposits of uric acid in the pericardium may be seen with visceral gout (Fig 12.9).

Hydropericardium is the cardiac change most frequently diagnosed ante mortem. It may develop with infectious exudative pericarditis, but can also occur due to congestion in cardiac failure, metabolic disorders (protein deficiency) or as an idiopathic syndrome. Hemopericardium is generally found following trauma or sclerosis with subsequent rupture of vessels, and is almost always fatal.

Clinical symptoms in birds with pericarditis are often nonspecific. Diagnosis is usually confirmed at necropsy. Radiography may reveal cardiomegaly with an irregular cardiac silhouette. Echocardiography is of limited use for diagnosis if there is no pericardial effusion. Endoscopy is the most reliable ante mortem diagnostic aid, although there is an increased risk during anaesthesia (see Chapter 24, Diagnostic Value of Endoscopy and Biopsy). In cases of acute visceral gout, plasma uric acid levels may be increased.

Common symptoms in birds with pericardial effusion are abdominal distension, dyspnea and exercise intolerance. Hydropericardium is often combined with congestion of the liver and ascites. The typical radiographic findings are enlargement of the heart and liver shadow as well as loss of detail (Fig 12.10). ECG may show low voltage,[22] but allows only a tentative diagnosis (adipose tissue and space-requiring processes like tumors can cause low voltage on the ECG as well).[29] The only reliable ante mortem diagnostic test for pericardial effusion is echocardiography. Hydropericardium can be visualized as an anechoic area between the myocardium and the pericardium (Fig 12.11). For further examinations (cytology, microbiology) an ultrasound guided aspirate from the pericardial space may be taken. In most cases, pericardial effusion is non-infectious and clear. Nevertheless, inflammatory cells may indicate infection, and culture may help to identify bacterial or fungal causes.

Treatment of exudative pericarditis, in addition to treating the underlying condition, may include administration of furosemide or another diuretic. Treatment for visceral gout is generally unrewarding (see Chapter 16, Evaluating and Treating the Kidneys). For treatment of hydropericardium due to congestive heart failure, diuretics in combination with ACE inhibitors are indicated.[29,47] Pericardial effusion may result in diastolic heart failure, ie, in an insufficient filling of the ventricles in diastole due to compression of the atria by the fluid in the pericardial cavity. This is a contraindication for the use of glycosides. An ultrasound guided pericardiocentesis may be performed to remove fluid from the pericardial space. Repeated echocardiography can be used to evaluate success of the therapy.

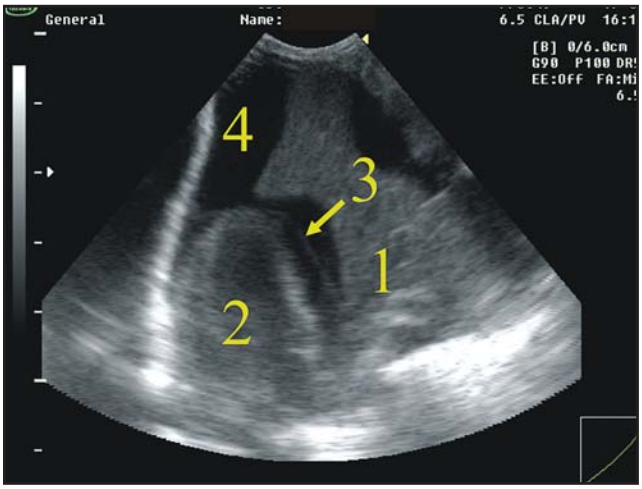


Fig 12.11 | 2-D-Echocardiography, African grey parrot (*Psittacus erithacus*), pericardial effusion and ascites (4) (corresponding to Fig 12.10). The pericardial effusion (1) can be seen as an anechoic area between the heart (2) and the pericardium (3).

MYOCARDIAL DISEASES (MYOCARDIAL FAILURE)

A decreased myocardial contractility (myocardial failure) may be primary, idiopathic (dilatative cardiomyopathy), or secondary, as a result of systemic diseases of infectious (myocarditis), toxic, metabolic (lipomatosis cordis, arteriosclerosis) and neoplastic origin. Frequently, an increased workload (eg, due to arteriosclerosis and pulmonary hypertension) leads to hypertrophy of the ventricles and can eventually result in decompensation and dilatation. Valvular insufficiencies can also lead to ventricular dilatation and eventually failure.[30] In psittacines, myocarditis is described as a complication in neuropathic proventricular dilatation disease.[40,51] Iron deposition in the myocardium combined with dilatation of the ventricles occurs in mynah birds with iron storage disease.[9] After prolonged transport of wild birds, myocardial necrosis may be seen as neurogenic arrhythmias as a consequence of metabolic disturbances.

Mostly myocardial failure is right-sided. Systemic lung diseases (ie, chronic mycosis) frequently result in dilatation of the right ventricle (including the right, muscular AV valve) and the right atrium and therefore the venous part of the circulatory system. Isolated left-sided myocardial failure is rare, since the resulting pulmonary congestion also affects the right ventricle and leads to a secondarily increased afterload and eventually right-sided CHF.

Although one case report suggests that myocardial compensation of valvular insufficiencies may occur for years, decompensation and development of clinical signs seem to develop more quickly in birds than they do in mammals.[30]

Clinical signs of myocardial failure of any cause can present as generalized weakness and as respiratory impairment. Hepatic congestion and ascites often cause abdominal distension.

Radiographs may demonstrate a consolidated, enlarged and sharply delineated shadow of the entire heart and liver. The appearance of the lung fields may demonstrate homogenous or non-homogenous increased opacity of the honeycomb pattern of the lungs which indicates pulmonary edema.

In birds with dilatative cardiomyopathy, the ECG may show an increased R-wave and a negative P-wave. The heart axis can change to between 0° and -170°. Arrhythmias are often seen with cardiomyopathy and myocarditis.[22]

In 2-D echocardiography, altered wall thicknesses and diameters of the ventricles as well as decreased contractility can be seen (measurements see Tables 12.1-12.3). In birds with right heart failure, the right ventricle is often as large as the left (Fig 12.12). Accompanying common findings are hydropericardium, ascites and congested liver parenchyma (see dilated vessels, Fig 12.13).

In case of suspected cardiomyopathy, blood-chemistry evaluation should include AST, CK, uric acid, LDH and electrolytes.

In addition to treatment of the underlying cardiac disease, supportive care including cage rest and diet change is important. Diuretics are indicated when edema (eg, pulmonary edema) or ascites are present. In the authors' experience, a combination of glycosides, ACE-inhibitors and diuretics seems to be the most effective initial therapy for cases of acute heart failure. For long-term therapy, ACE-inhibitors are preferable.[29,47]

ENDOCARDIAL DISEASES (ENDOCARDITIS, VALVULAR INSUFFICIENCY, VALVULAR STENOSIS)

Alterations of the endocardium, in particular the valves, may be idiopathic.[30] However, these changes are found more frequently secondary to infections with streptococci, staphylococci, *Pasteurella multocida* or *E. coli*.[20,22,35]

Independent of the etiology, functional damage of the AV valves leads to regurgitation and to left heart failure with pulmonary edema (small circulatory cycle) and/or right heart disease with liver congestion (large circulatory cycle) and can cause general cardiac failure. Thickening of the right AV valve is normally caused by muscular hypertrophy. It may be a consequence of valvular insufficiency or due to hypertrophy of the whole right ventricle (see Fig 12.12). This alteration occurs

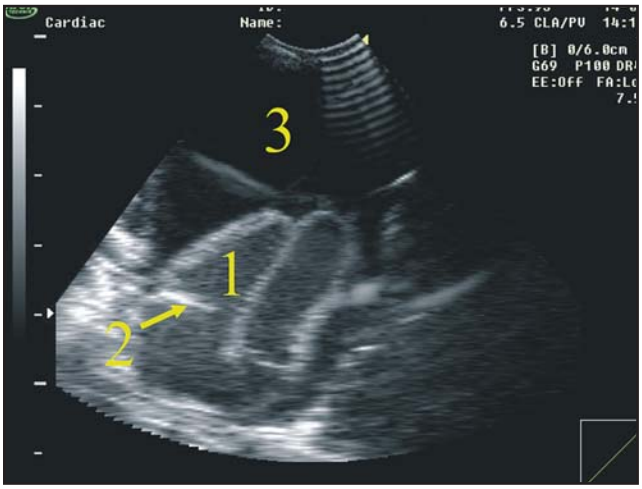


Fig 12.12 | 2-D-Echocardiography, yellow-crowned Amazon (*Amazona ochrocephala*), right heart failure. The right ventricle (1) is as large as the left one, the muscular right atrioventricular valve (2) is clearly thickened (3 = ascites).

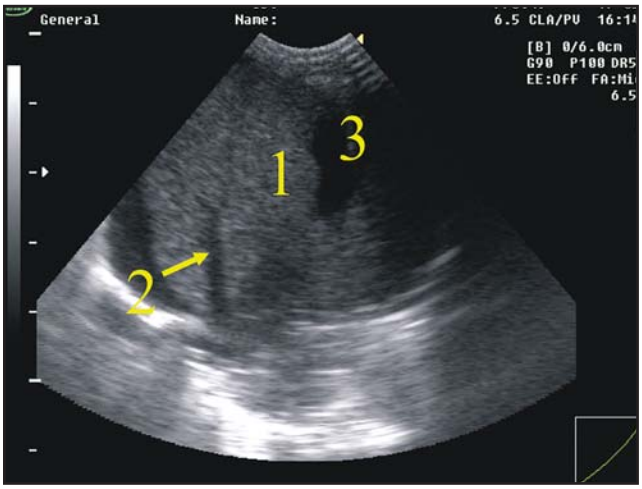


Fig 12.13 | 2-D-Echocardiography, African grey parrot (*Psittacus erithacus*). Liver (1) congestion and ascites (3). Dilated vessels (2) can be demonstrated in the liver tissue.

more often than alterations of the left AV valve. Chronic inflammation of the semilunar valves can also result in stenosis.[20]

Clinically, nonspecific symptoms (weakness, dyspnea, distended abdomen) may be noticed.

Radiographic investigation gives information about cardiac size and congestion of organs (see Myocardial Diseases section) but radiographic diagnosis of endocardial alterations is not possible.

2-D-Echocardiography can show accompanying myocardial thickening and changes of the valves, especially thickening of the right atrioventricular valve (see Fig 12.12).[30,35] Doppler echocardiography is the imaging tool of choice for demonstration of valvular regurgitation in mammals. Although initial examinations have been performed in birds (see Echocardiography), little has been published about the use of Doppler echocardiography in case of suspected valvular damage. In an Indian hill mynah, color flow and spectral Doppler have been used for demonstration of mitral regurgitation.[37,39]

A good prognosis may be given in cases of acute endocarditis with aggressive treatment of the causal agent (Myocardial Diseases section).

In patients with valvular regurgitation, glycosides can be used, whereas in case of stenosis, these drugs are contraindicated. A decrease in the heart rate to a normal range and decreased congestion may be interpreted as therapeutic success.

ARTERIOSCLEROSIS

Arteriosclerosis is the most frequently described pathologic change of the vessels in psittacine birds.[4,10] Different etiological factors are discussed; the most frequent causes are hyperlipidemia, endothelial inflammation, toxins, immune complexes, hypertonia and/or stress factors. Age and nutritional deficiencies over many years, as well as lack of exercise seem to play a role in the development of arteriosclerosis.[13] Psittacines commonly affected by arteriosclerosis are amazons (especially blue fronted Amazons), African grey parrots and cockatoos.[4,13,20]

Macroscopic changes include arterial wall thickening, intimal roughening, induration and yellowish discoloration.[10] Calcification can cause plaque-like or diffuse hardening of the larger arteries like the aorta and brachiocephalic trunk (Fig 12.14). Arteriosclerosis cannot be diagnosed by gross findings alone, but requires histological examination, especially in earlier stages.[10,20]

A diagnosis of arteriosclerosis is often made at necropsy. Clinical findings are usually absent or may only lead to a tentative diagnosis. Possible symptoms include lethargy, neurological signs (tremor, paralysis of the legs), decreased exercise tolerance and dyspnea.[8,13,14] Acute death may occur.

Radiologically, an increased radiodensity and widening of the aorta may be seen in advanced cases, sometimes in connection with left atrial or left ventricular enlargement. Echocardiography may be useful for diagnosis of the arteriosclerotic processes of the large vessels close to the heart, but no systematic studies have been done to date. In a white cockatoo, an aneurysm of a coronary artery associated with arteriosclerosis has been diagnosed by echocardiography.[52]

Effective therapy for arteriosclerosis is not known for birds. In poultry, ß-blockers (eg, oxprenolol) proved to have a protective effect against the development of

arteriosclerotic plaques. Unfortunately it is not possible to remove plaques which are already present.[27,28] Diuretics may be indicated as well as ACE-inhibitors if heart failure occurs.[7] Balanced food supply and sufficient opportunities to exercise are important preventive factors. Proper flight exercise and diet will prevent obesity, a major contributor to this cardiac disease.

CIRCULATORY DISTURBANCES (CIRCULATORY COLLAPSE, SHOCK)

Physiologically, the avian circulatory system has a large load capacity. Unexpected deaths, after catching small birds, can sometimes be attributed to asphyxia (strong pressure on the sternum). Long restraint of fractious birds may lead to circulatory collapse, especially in birds with dyspnea, ascites (resulting in displacement of the air sacs) or cardiac failure. Hypovolemic, septic (toxic-infectious) and neurogenic shock as well as anaesthetic complications may also be a cause. Overcrowding of carriers or transporting in temperatures near or above 100° F (38° C) may lead to circulatory collapse.

Clinical symptoms of circulatory collapse start as respiratory signs with spread wings/ legs and tachypnea/ dyspnea. This is followed rapidly by respiratory arrest, convulsions with opisthotonus, and a loss of consciousness.

Further diagnostics should only be done after stabilization of the patient. If possible, the bird should be kept upright for examination (eg, circulatory risk during positioning for radiology). The treatment for shock is always handled as an emergency situation. The bird must immediately be brought to a calm and darkened area. A volume substitution with warmed (100-103° F) lactated

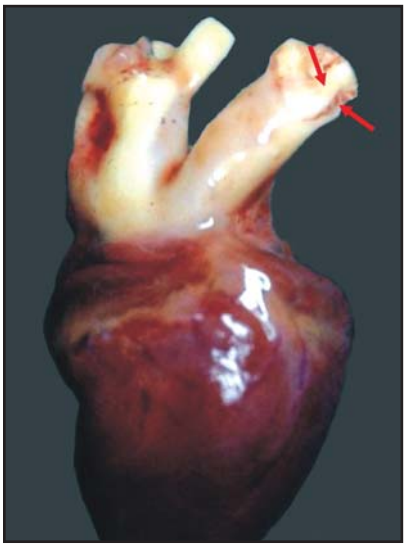

S. Braun

Fig 12.14 | Necropsy, African grey parrot (*Psittacus erithacus*), with arteriosclerosis. Thickening and discoloration of the wall of the large vessels (arrows).

Ringer's solution (LRS) or half-strength LRS + 2.5% dextrose (IV, intraosseus cannula, for maintenance also SC) is indicated. Infusions with sodium bicarbonate solution should be given in case of metabolic acidosis, recommended dosage is 1 mEq/kg (=1 ml/kg), in intervals of 15 to 30 minutes up to a maximum of 4 mEq/kg.[55] Additionally, oxygen supply is indicated. If faced with respiratory arrest administration of doxapram (respiratory stimulant, orally dropwise to effect) may be useful. Oral /IV/IM administration of g-strophantin (see Table 12.7) may successfully stabilize patients presenting in circulatory failure. Sympathomimetics (etilifrine) may be administered orally to increase the stroke volume in case of heart failure (in particular, in case of cardiogenic shock).

Parts of this chapter are copied or paraphrased from Proc Assoc Avian Vet, 2001, pp 225-330. Permission granted by AAV 2003.

References and Suggested Reading

1. Aguilar RF, Smith VE, Ogburn P, Redig PT (1995) Arrhythmias associated with isoflurane anaesthesia in bald eagles (*Haliaeetus leucocephalus*). J Zoo Wildl Med 26, 508-516.
2. Bezuidenhout AJ (1983) The valva atrioventricularis dextra of the avian heart. Zentralbl Vet Med C Anat Histol Embryol 12, 104-108.
3. Boskovic M, Krautwald-Junghanns ME, Failing K, et al. (1999). Möglichkeiten und Grenzen echokardiographischer Untersuchungen bei Tag- und Nachtgreifvögeln (Accipitriformes, Falconiformes, Strigiformes). Tierärztliche Praxis 27, 334-341.
4. Braun S, Krautwald-Junghanns ME, Straub J. (2002) Zur Art und Häufigkeit von Herzerkrankungen bei in Deutschland in Gefangenschaft gehaltenen Papageienvögeln. Deutsche Tierärztliche Wochenschrift 109, 253-292.
5. Carrani F, Gelli D, Salvadori M, Aloisi M (2003). A preliminary echocardiographic initial approach to diastolic and systolic function in medium and large parrots. Proc 7th Europ AAV Conf, Puerto de la Cruz, 145-149.
6. Casares M., Enders F. Montaya JA. (1999). Electrocardiography in some macaw species (genus *Anodorhynchus* and Ara). Proceedings of the 5th European AAV meeting, Pisa, 158-163.
7. Chamontin B, Amar J (2001). Prevention in cardiovascular pathology. Therapy 56, 119-124.
8. Dorrestein GM, Zwart P, Borst GHA, Poelma FG, Buitelaar MN (1977). Ziekte-en doodsoorzaken van vogels. Tijdschr Diergeneesk. 102, 437-447.
9. Dorrestein GM, Van Der Hage MH (1988). Veterinary problems in mynah birds. Proc Assoc Avian Vet, 263-274.
10. Fricke C, Dorrestein GM, Straub J, Krautwald-Junghanns ME (2003). Macroscopic and microscopic changes in blood vessels of psittaciformes. Proc 7th Europ AAV Conf, Puerto de la Cruz, 137-144.
11. Hamlin RL, Stalnaker PS (1987). Basis for use of digoxin in small birds. J Vet Pharmacol Therap 10, 354-356.
12. Hanley SH, Helen GM, Torrey S, et al. (1997). Establishing cardiac measurement standards in three avian species. J Avian Med Surg 11,15-19.
13. Johnson HJ, Phalen DN, Kondik VH, Tippit T, Graham DL (1992). Atherosclerosis in psittacine birds. Proc Assoc Avian Vet, New Orleans, 87-93.
14. Kempeneers P (1987). Atherosklerose bij de papegaai. Utrecht, Utrecht University.
15. Krautwald-Junghanns ME, Schulz M, Hagner D, Failing K and Redmann T (1995). Transcoelomic two-dimensional echocardiography in the avian patient. J Avian Med Surg 9,19-31.
16. Krautwald-Junghanns ME, Straub J, Pees M, Braun S (2001a). Anatomy and pathology of the psittacine heart. Proceedings of the 4th Scientific ECAMS Meeting, Munich, 14-15.
17. Krautwald-Junghanns ME, Straub J (2001b). Avian Cardiology: Part 1. Proceedings of the Assoc Avian Vet, Orlando, 323-330.

18. Krautwald-Junghanns ME, Pees M, Schütterle N (2001c). Echokardiographische Untersuchungen an unsedierten Brieftauben (*Columbia livia f. domestica*) unter besonderer Berücksichtigung des Trainingszustandes. Berliner und Münchner Tierärztliche Wochenschrift 114, 1-4
19. Krautwald-Junghanns ME, Kummerfield N (2003). Arzneimittelverzeichnis. *In:* Kaleta EF, Krautwald-Junghanns ME: Kompendium der Ziervogelkrankheiten. Schluetersche Verlagsanstalt, Hannover.
20. Krautwald-Junghanns ME, Braun S, Pees M, Straub J, Valerius HP (2004). Investigations on the anatomy and pathology of the psittacine heart. J Avian Med Surg, 18,2-11.
21. Lumeij JT, Stokhof AA (1985). Electrocardiogram of the racing pigeon (*Columbia livia forma domestica*). Res Vet Sci 38, 275-278.
22. Lumeij JT, Ritchie BW (1994). Cardiovascular System. *In:* Ritchie BW, Harrsion GJ, Harrsion LR (eds): Avian Medicine, Principles and Application, Brentwood, TN, HBD Int'l Inc, 694-722.
23. Nap AMP, Lumeij JT, Stokhof AA(1992). Electrocardiogram of the African Grey (*Psittacus erithacus*) and Amazon (*Amazona* sp.) parrot. Avian Pathol 21, 45-53.
24. Oglesbee BL, Oglesbee MJ (1998). Results of post-mortem examination of psittacine birds with cardiac disease: 26 cases (1991-1995). JAVMA 212, 1737-1742.
25. Orosz S (1997). Anatomy of the cardiovascular system. *In:* Altman RB, Clubb SL, Dorrestein GM, Quesenberry K (eds): Avian medicine and surgery. WB Saunders Co, Philadelphia, 489-490.
26. Owen R (1866). Anatomy of vertebrates. Vol II. Birds and mammals. Longmans, Green, and Co., London.
27. Pauletto P, Pessina AC. Pagnan A, Thiene G, Semplicini A, Vescovo G, Mazzucato A, Scannapieco G, Angelini A, Dal Palu C (1985). Evidence of reduced atherosclerotic lesions in broad breasted white turkeys treated with oxprenolol. Artery 12, 220-233.
28. Pauletto P, Vescovo G, Scannapieco G, Pessina AC, Dal Palu C. (1990). Cardioprotection by beta-blockers: molecular and structural aspects in experimental hypertension. Drugs Exp Clin Res. 16,123-128.
29. Pees M, Straub J, Krautwald-Junghanns ME (2000). Pericardial effusion in birds: Demonstration of clinical cases. Proceedings of the Assoc Avian Vet, Portland, 189-191.
30. Pees M, Straub J, Krautwald-Junghanns ME (2001). Insufficiency of the muscular right atrioventricular valve in the heart of a blue-fronted Amazon (*Amazona aestiva aestiva*). Vet Rec 148, 540-543.
31. Pees M, Straub J, Krautwald-Junghanns ME (2003a). Echokardiographische Untersuchungen bei Psittaciformes, Teil 1: Durchführung und gattungsspezifische Unterschiede. Tierärztl Praxis 31(K): 126-31.
32. Pees M, Straub J, Krautwald-Junghanns ME (2003b). Echokardiographische Untersuchungen bei Psittaciformes, Teil 2: Sonografisch ermittelte Messwerte zur Herzanatomie und errechnete Parameter der Herzfunktion. Tierärztl Praxis 31(K), 180-187, 2003.
33. Pees M, Straub J, Krautwald-Junghanns ME (2003c). Echocardiographical examinations of healthy psittacine birds under special consideration of the African Grey parrot (*Psittacus erithacus erithacus*). Proc 7th Europ AAV Conf, Puerto de la Cruz, 161-167.
34. Pees M, Straub J, Krautwald-Junghanns ME (2004). Echocardiographical examinations of psittacine birds with special reference to the African grey parrot. Vet Rec, 155,73-76.
35. Randolph J, Moise S, Graham D, Murphy C (1984). Bacterial endocarditis and thromboembolism of a pelvic limb in an emu. J Am Vet Med Assoc 11, 1409-1410.
36. Ritchie BW, Harrison GJ (1994). Formulary. *In:* Ritchie BW, Harrison GJ, Harrison LR (eds): Avian Medicine, Principles and Application, Brentwood, TN, HBD Int'l Inc, 457-478.
37. Rosenthal K and Stamoulis M (1992). Congestive heart failure due to mitral regurgitation in an Indian hill mynah bird. Proc of the Assoc Avian Vet, New Orleans, 171-173.
38. Rosenthal K, Miller M (1997). Cardiac disease. *In:* Altman RB, Clubb SL, Dorrestein GM, Quesenberry K (eds): Avian medicine and surgery. WB Saunders Co, Philadelphia, 491-500.
39. Rosenthal K and Stamoulis M (1993). Diagnosis of congestive heart failure in an Indian hill mynah bird (*Gracula religiosa*). J Assoc Avian Vet 1, 27-30.
40. Shivaprasad HL, Barr BC, Woods LW, Daft BM, Moore JD, Kinde H, Anderson ML, Drougal R (1995). Spectrum of lesions (pathology) of proventricular dilatation syndrome. Proc Assoc Avian Vet, California, 505-506.
41. Smith FM, West NH, Jones DR (2000). The cardiovascular system. *In:* Avian Physiology, 5th edition (GC Whittow ed) Academic Press.
42. Straub J, Pees M, Krautwald-Junghanns ME (2000). Heart diseases in birds: Rare or just not diagnosed? Proc Assoc Avian Vet, Portland, 285-289.
43. Straub J, Pees M, Schumacher J, Gompf RE, Baade H, Krautwald-Junghanns ME (2001). Doppler-echocardiography in birds. Proceedings of the 6th European AAV Main Conference, Munich, 92-94.
44. Straub J, Valerius KP, Pees M, Krautwald-Junghanns ME (2002a). Morphometry of the heart of budgerigars (*Melopsittacus*), Alisterus parrots (*Alisterus s. scapularis*) and common buzzards (*Buteo buteo*). Research in Veterinary Science 72, 147-151.
45. Straub J, Valerius KP, Pees M, Krautwald-Junghanns ME (2002b). Untersuchungen zur Myokarddicke bei Wellensittichen (*Melopsittacus undulatus*) und Königssittichen (*Alisterus s. scapularis*). Berliner und Münchener Tierärztliche Wochenschrift 115, 440-444.
46. Straub J, Pees M, Krautwald-Junghanns ME (2002c). Measurement of the cardiac silhouette in psittacines. Journal of the American Veterinary Medical Association 221, 76-79.
47. Straub J, Pees M, Enders F, Krautwald-Junghanns ME (2003a). Pericardiocentesis and the use of enalapril in a Fischer's lovebird (*Agapornis fischeri*). Vet Rec 152, 24-26.
48. Straub J, Forbes N, Pees M, Krautwald-Junghanns ME (2003b). Effect of handling-induced stress on the results of spectral Doppler-echocardiography in falcons. Res Vet Sci 74,119-122.
49. Straub J, Forbes N, Pees M, Krautwald-Junghanns ME (2004). Pulsed-wave Doppler derived velocity of diastolic ventricular inflow and systolic aortic outflow in diurnal and nocturnal raptors. Veterinary Record 154,145-147.
50. Thomas WP, Gaber CE, Jacobs GJ, Kaplan PM, Lombard CW, Moise NS, Moses BL (1993). Recommendations for standards in transthoracic two-dimensional echocardiography in the dog and cat. J Vet Int Med 7, 247-252.
51. Vice C, Cazayoux A (1992). Myocarditis as a component of psittacine proventricular dilatation syndrome in a Patagonian conure. Avian Diseases 35, 1117-1119.
52. Vink-Nooteboom M, Schoemaker NJ, Kik MJL, Lumeij JT, Wolvekamp WTC (1998). Clinical diagnosis of aneurysm of the right coronary artery in a white cockatoo (*Cacatua alba*). J Small Anim Pract 39: 533-537.
53. West NH, Langille BL, Jones DR (1981). Cardiovascular system. *In:* King, AS, McLelland J (eds): Form and Function in Birds (Vol. 2). Academic Press, London, New York, 235-251.
54. Wilson RC, Zenoble RD, Horton CR, and Ramsey DT (1989). Single dose digoxin pharmacokinetics in quaker conure. J Zoo Wildl Med 20, 432-434.
55. Wilson H (2003). Avian Emergency and Critical Care. Proceedings of the Assoc Avian Vet, Pittsburgh, 261-268.
56. Wyatt HL, Heng MK, Meerbaum S (1979). Cross-sectional echocardiography I. Analysis of mathematic models for quantifying mass of the left ventricles in dogs. Circulation 60, 1104-1113.