

Cardiovascular Diseases

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PREVALENCE AND RISK FACTORS

Prevalence

The prevalence of cardiac disease in the general feline population is not currently determined. Stalis and coworkers⁸³ found that myopathic heart disease was identified in approximately 9% of 1472 feline necropsies from 1986 to 1992 at the University of Pennsylvania. More recently, two small studies (approximately 200 cats in total) have examined the prevalence of cardiac disease in apparently healthy cats.^{18,61} Côté and colleagues¹⁸ examined the prevalence of heart murmurs in apparently healthy cats and detected murmurs in 22 of 103 cats examined. Of these 22 cats, seven had echocardiographic evaluations, and six were considered to have evidence of myocardial hypertrophy (one was normal). Paige and coworkers⁶¹ examined 103 apparently healthy cats: 16 of 103 had murmurs, and five of these had evidence of myocardial hypertrophy. Additionally, 11 of 103 cats had evidence of myocardial hypertrophy but no murmurs.

On the basis of these small epidemiologic studies, approximately 20% of apparently healthy cats examined at random will have cardiac murmurs, and a similar percentage might have myocardial hypertrophy. Of these, half will have murmurs, and half will have occult disease. Similarly, 50% or more of the cats with murmurs will not have identifiable cardiac disease; dynamic physiological murmurs likely account for some of these. It is

important to note that both studies examined small numbers of cats. Furthermore, no longitudinal evaluation was performed to determine if the myocardial hypertrophy was transient (e.g., secondary to dehydration, thyroid disease or as yet unidentified causes of transient hypertrophy) or persistent. Only the latter would be consistent with hypertrophic cardiomyopathy. Thus the prevalence of cardiac disease in the general feline population remains unknown. However, a large longitudinal study of cats presenting to shelters in London is currently under way and may better define the prevalence of feline myocardial diseases.

Relative prevalence of cardiac disease has been examined by Harpster³¹ at a single referral institution. Of 500 cats presenting to the cardiology department at Angell Memorial Animal Hospital from 1987 to 1989, 22% had hypertrophic cardiomyopathy, 15% had unclassified cardiomyopathy, 14% had mitral valve disease, 12% had dilated cardiomyopathy, 10% had thyrotoxic heart disease, and approximately 7% had congenital diseases. Systemic hypertension was identified in 1%. It should be obvious that these percentages do not represent true prevalence (or incidence) but rather describe the distribution of heart diseases in patients presenting for evaluation of heart disease. Additionally, substantial changes in feline nutrition (namely taurine supplementation) and early detection and management of hyperthyroidism have greatly reduced the percentage of cats presenting with either dilated cardiomyopathy or thyrotoxic heart disease.

Similarly, the prevalence of congenital cardiac diseases in cats is much less comprehensively reported than that of dogs. Frequency of congenital heart disease has not been examined in the last 30 years. Buchanan¹¹ estimated that atrioventricular valve malformations were the most common congenital defect in cats, followed by ventricular septal defect (VSD), endocardial fibroelastosis and patent ductus arteriosus (PDA).

Côté and Jaeger¹⁷ examined the incidence of structural heart disease in 106 cats presenting with ventricular arrhythmias. Almost all cats with ventricular tachyarrhythmias had echocardiographic evidence of structural heart disease (102 of 106). Prior studies by Fox and associates²⁶ and Fox and Harpster²⁵ suggested substantially lower incidence rates of ventricular tachyarrhythmias in cats with hypertrophic cardiomyopathy (HCM) (10% to 40%). However, both studies reported all arrhythmias in cats with HCM at much higher rates (25% to 70%).

Risk Factors

Certain risk factors are associated with some feline heart disease. HCM has specific breed predispositions, and at least one identified genetic cause in each of two breeds (Maine Coons and Ragdolls).^{53,55} Sphynx, Norwegian Forest Cats, American Shorthairs, Scottish Folds, Persians, Siamese, Abyssinians, Himalayans, and Birman cats are all breeds considered to be predisposed to cardiomyopathies.²³ Whether there are any gender differences in expression of genetic traits or in prevalence of congenital disorders is not well defined.

Taurine deficiency was identified as a major cause of dilated cardiomyopathy in cats in the mid 1980s.⁶⁴ Subsequent supplementation of commercial diets with taurine has led to the almost complete disappearance of taurine-deficient myocardial failure in cats. However, homemade diets can still occasionally lead to taurine deficiency, resulting in dilated cardiomyopathy.

Hyperthyroidism is a risk factor for cardiac disease in cats. However, the prevalence of thyrotoxic heart disease has likely decreased since hyperthyroidism was first recognized, as clinicians have become more adept at identifying cats with hyperthyroidism earlier in the disease course, often before the development of severe cardiac remodeling and high-output heart failure. Other risk factors, such as acromegaly, appear to be extremely uncommon, and descriptions of these are limited to small case series or case reports.

HISTORY AND PHYSICAL EXAMINATION

Although history taking can offer insights to the clinician about the patient, the uncanny ability of cats to mask their disease status until the condition is critical

prevents many owners from providing diagnostically useful information. Owners might report findings such as panting, hiding, or reluctance to participate in usual activities in the days preceding presentation for severe disease. With mild subclinical disease, no changes will be apparent to the owners. Dietary history is useful only if taurine deficiency is suspected; however, a homemade diet might be a clue to the clinician to examine the patient for taurine deficiency.

Systemic thromboembolism is often accompanied by a history of acute paralysis or paresis and apparent excruciating pain. Owners often report that their cat screamed or yowled loudly at the onset of the event, without any apparent evidence of trauma. Cats presenting later in the course of the disease often have a history of being missing for a period of time and being found paralyzed or paretic.

Murmurs

The physical examination of cats with heart disease is often only modestly revealing. Many cats with cardiac disease have no indicative clinical signs or physical examination findings. In one small study, only 5 of 16 of cats with cardiomyopathy had murmurs at initial examination; 11 had occult disease.⁶¹ This number increased to 11 of 16 cats when dynamic murmurs (not necessarily auscultated at the time of examination but provoked during echocardiographic evaluation) were examined. Conversely, many cats with a murmur have no identifiable heart disease. Paige and coworkers⁶¹ also identified murmurs in 16 of 103 healthy cats but found cardiac disease in only 5 of these; 11 had no evidence of structural disease.

Dynamic murmurs are common findings in cats with and without heart disease. Paige and coworkers⁶¹ identified dynamic murmurs in 28 of 103 apparently healthy cats. Dynamic murmurs change in intensity or appear only after provocation (e.g., fear, aggression). They are generally parasternal murmurs (either right or left) and can be extremely transient, lasting only a few beats in some cats. Rishniw and Thomas⁷¹ identified a dynamic right ventricular outflow tract obstruction in 50 cats between 1994 and 1996 that was only occasionally associated with structural heart disease. The most common diseases associated with this physiologic murmur were chronic kidney disease and nasal squamous cell carcinoma (SCC), but these cats were examined in California, where nasal SCC is highly prevalent. Cats younger than 4 years of age with dynamic right ventricular outflow tract obstruction most often had HCM.

Systolic anterior motion of the mitral valve and the associated dynamic left ventricular obstruction account for most of the remainder of identifiable dynamic murmurs in cats. This phenomenon is observed predominantly in cats with HCM but can occasionally be

observed in cats without any identifiable structural heart disease. Midventricular obstructions have also been identified in cats with HCM or other feline cardiac diseases⁴⁹ and might account for some dynamic murmurs in cats. In one study only 36% of provable murmurs had an identifiable etiology; thus many dynamic murmurs might not have an easily identifiable cause.⁶¹

Heart Sounds and Arrhythmias

The most commonly observed abnormal heart sound in feline heart disease (excluding murmurs) is the gallop sound. This can be intermittent or sustained and results from an increased intensity of the third or fourth heart sound (or a summation of the two). A true gallop sound is indicative of severe heart disease in cats, associated with marked diastolic dysfunction. However, because of the almost identical systolic and diastolic time intervals in cats, feline gallop sounds are auscultably indistinguishable from systolic clicks. Systolic clicks are uncommon, and, as in dogs, they are thought to be associated with mild mitral valve disease in older cats. They can be distinguished from gallop sounds only by high-fidelity phonocardiograms that have electrocardiographic timing, which demonstrate that the extra heart sound occurs in midsystole. Finally, ventricular extrasystoles (ventricular bigeminy) can sometimes produce a gallop sound if the ventricular extrasystolic beat occurs close to the sinus beat. In these cases the mitral valve opens and then closes during the extrasystole, but the aortic valve fails to open (causing only one heart sound from the extrasystole and two heart sounds from the preceding sinus beat). This can be identified by electrocardiography (ECG). Thus the presence of an additional heart sound in a cat warrants further diagnostic investigation.

Arrhythmias occur frequently in cats with heart disease. In one retrospective study, 96% of cats with ventricular tachyarrhythmias had echocardiographic evidence of structural heart disease. Thus auscultation of extrasystoles warrants further investigation. It is, however, more difficult to define sustained tachyarrhythmias in cats presenting to clinicians for physical evaluation. Feline heart rates can easily reach 240 to 260 beats per minute (bpm) in stressful situations and can do so in a matter of seconds. Cats stressed by a hospital visit or because of other systemic disease can have sustained heart rates above 220 bpm.¹ The astute clinician should note heart rates from prior visits in regular patients to determine whether the rate is appropriate for that patient. Unexpectedly high heart rates, especially those that deviate from rates obtained at prior visits, might warrant further investigation.

Bradyarrhythmias are less commonly auscultated but occur especially in older cats. The author considers any heart rate persistently lower than 130 bpm in a cat

during a clinical examination to be unexpectedly low, warranting further diagnostic testing. However, healthy young (mostly) male cats occasionally appear to have low resting heart rates.

Sinus arrhythmias are uncommon in cats in the hospital environment and have been associated mostly with extracardiac disease.⁶⁹ However, some healthy young cats can have a mild sinus arrhythmia as an incidental finding. Additionally, most cats exhibit brief periods of sinus arrhythmia during sleep.⁸⁶

Clinical Signs of Congestive Heart Failure

Cats are extremely adept at hiding signs of heart disease until they reach a critical stage. Often, clinical signs such as mild tachypnea and reduced activity are not apparent to the owner, and cats present to the clinician with profound dyspnea. Thoracic auscultation might reveal signs of congestive heart failure (CHF). A murmur or gallop sound coupled with dyspnea increases the index of suspicion for CHF. Muffled or absent breath sounds or dorsally displaced breath sounds (absent ventrally) are suggestive of pleural effusion. On the other hand, coughing and gagging, wheezing, or auscultable crackles are rarely associated with CHF but are almost always indicative of primary respiratory disease. Extremities may be somewhat cool because of vasoconstriction that occurs with CHF, but this is an unreliable sign of CHF.

Physical Examination Procedures of Limited Value in Diagnosis of Feline Heart Disease

Peripheral Pulse Quality

With the exception of systemic thromboembolic disease, assessment of peripheral pulses in cats with heart disease offers little in the general cardiovascular assessment of the patient. Pulses are rarely altered with most feline cardiac disease, and clinicians' ability to discern minor changes in pulse quality is limited. Thus the author advises evaluating feline pulses only if paresis or paralysis of limbs is suspected.

Mucous Membrane Color and Capillary Refill Time

Most cats have mucosal color that is somewhat "anemic" or "cyanotic looking." Severe heart disease and even CHF often fail to alter mucosal color or capillary refill time, and interpretation of the findings is questionable enough that performing these procedures in cats with suspected heart disease is not recommended.

Murmur Localization and Characterization

Although localization of murmurs in dogs can help with identification of the underlying heart disease, this

approach is much more difficult in cats. First, many clinicians use stethoscopes with large diaphragms, and the area of the diaphragm is similar to the area of the cardiac silhouette. This effectively limits the ability to localize to an area smaller than the entire heart. Second, many cats with (and without) heart disease have parasternal murmurs, which are often dynamic. These can occur for various reasons and do not help in further defining the nature of the heart disease.

In some instances murmur localization and description can assist with diagnosis. VSDs and tricuspid valve defects are generally ausculted on the right side, whereas PDA murmurs are supraventricular continuous murmurs.

Thus clinicians should, in practice, limit their auscultation to detection of a murmur and possible description of location but not expect to derive a diagnosis solely on the basis of this physical examination procedure.

DIAGNOSIS OF FELINE HEART DISEASE

As previously explained, the history and physical examination, although important, generally fail to provide a definitive diagnosis of heart disease or the type of heart disease. In most cases, when heart disease is suspected, additional diagnostics are required to confirm the suspicion before any therapy can be instituted.

Electrocardiography

ECG is largely limited to diagnosis of arrhythmias and conduction disturbances in cats. It is best reserved for those patients that have auscultable arrhythmias. Arrhythmias are relatively uncommon in cats, with the exception of sinus tachycardia. However, their frequency increases substantially with the presence of heart disease.

In eupneic cats the ECG is recorded in right lateral recumbency. However, sternal recumbency alters few electrocardiographic parameters of clinical interest.^{29,32} Therefore assessment in sternal recumbency in fractious, dyspneic, or fragile patients is acceptable.

Continuous 24-hour ambulatory ECG (Holter) monitoring has historically been less successful in cats than dogs, largely because of the size of the recording systems. New digital Holter systems are small enough to be attached to the cat with adhesive bandaging. Holter monitoring can provide diagnostic information in cats with syncope.²² Additionally, small event recorders can be surgically implanted into syncopal patients to increase the probability of arrhythmia detection.^{20,38} Holter monitors should not be used on cats with severe structural heart disease or CHF because the stress of monitoring can result in the death of the patient.

Electrocardiography as a Screening Test for Subclinical Heart Disease

ECG is ineffective as a screening tool for occult cardiac disease in cats. The basis for using ECG as a screening tool relies on its ability to detect either chamber enlargement or shifts in the mean electrical axis (MEA). However, ECG is extremely insensitive and relatively imprecise in detecting chamber enlargement (or myocardial concentric hypertrophy), and although it can identify deviations in the MEA, these occur relatively infrequently in the general population and can occur in cats with and without underlying structural disease. Only one study has examined the ability of ECG to identify left atrial enlargement in cats.⁷⁶ This study showed poor sensitivity (12% to 60%) and good specificity (72% to 100%), suggesting that very few cats with p-wave abnormalities have normal left atria. No equivalent studies exist that specifically examine the sensitivity and specificity of ECG in detecting ventricular enlargement in cats; however, studies in humans and other species suggest sensitivities of approximately 50% and specificities of 80% (similar to those found by Schober and coworkers⁷⁶ for left atrial enlargement). Two studies have examined ECG abnormalities in cats with heart disease. Ferasin and coworkers²¹ identified 106 cats with varying degrees of HCM; of these 41 (39%) had no identifiable ECG abnormalities. Riesen and coworkers⁶⁸ examined 395 cats with various symptomatic heart diseases, including 169 cats with HCM; of these 35 (21%) had no identifiable ECG abnormalities. Riesen and coworkers identified morphologic changes (i.e., chamber enlargement patterns) in only 15 of 169 (10%) cats with HCM, whereas Ferasin and coworkers found morphologic changes in 30 of 61 (50%) cats with HCM. If these data are combined, morphologic changes indicative of chamber enlargement occur in fewer than 20% of cats with HCM. However, this may be an overestimation, because individual animals in these studies might have had more than one morphologic change; we have assumed that each observation is independent, which gives the "best-case scenario." Thus the sensitivity of ECG in detecting morphologic changes consistent with chamber enlargement or HCM, on the basis of these two studies, is 20%. If one assumes that 15% of the general feline population has heart disease, the positive predictive value of morphologic changes on the ECG is approximately 15%, and the negative predictive value is approximately 85%. Thus a clinician is six times as likely to find a false-positive result as a true-positive result when screening cats by ECG, resulting in substantial expense to clients in pursuit of nonexistent disease. With lower prevalence the probability of a false-positive finding only increases. A negative result would strongly suggest that the cat is "unaffected" because most cats examined are going to be normal. However, most cats

with HCM that are examined will also have normal ECG results; these will not be identified.

Presence of pathologic arrhythmias (ventricular premature contraction [VPC], atrial premature contraction [APC], atrial fibrillation) occurred in 17 of 169 (10%) of HCM cats in one study,⁶⁸ and 8 of 106 (8%) in another study²¹—again, whether these were independent observations or whether multiple arrhythmias were present in the same cat was not apparent. However, this again results in a sensitivity of 10%, preventing the clinician from effectively ruling out the presence of HCM in the absence of arrhythmias.

Radiography

Radiography has been used for diagnosis of feline heart disease since the early 1970s. More recently, it has been supplanted by echocardiography for diagnosis of heart disease, but it is still a valuable diagnostic test for identification of CHF or discrimination of causes of dyspnea in cats.

Identification of cardiomegaly from radiographs in cats is difficult. Enlargement patterns most amenable to radiographic evaluation are *left atrial or biatrial enlargement* and *left ventricular volume overload*. Mild enlargement (as defined echocardiographically) is generally not detectable radiographically; chambers must be at least moderately enlarged before they are radiographically detectable. Right-sided heart changes are both uncommon and difficult to identify in cats (and dogs). Similarly, left ventricular concentric hypertrophy, as occurs with HCM, is not radiographically identifiable; cats can have profoundly thickened left ventricular walls that are radiographically undetectable.

Both lateral and dorsoventral (DV) or ventrodorsal (VD) views are required for the diagnosis of feline heart disease because atrial enlargement is best appreciated in the DV/VD view. There is little difference between VD or DV views. Cats that are dyspneic or tachypneic are best imaged in sternal recumbency to reduce the stress of restraint, which can result in severe clinical deterioration. End-inspiratory films are preferred, although this is not essential in most cases. In the author's experience of evaluating feline thoracic radiographs obtained by general practitioners, most cats provide films of sufficient quality for interpretation. Obese cats can be problematic because of their reluctance to take deep breaths; in such patients, interpretation of the pulmonary parenchyma can be problematic.

Most traditional rules of cardiac mensuration (measurement) are of little value in cats. Comparisons of the cardiac silhouette to the thoracic cavity or degree of cardiosternal contact have no value in assessing feline thoracic radiographs for cardiac disease. Sternal contact is prominent in many cats and increases with age in cats with normal hearts.^{57,59} Similarly, aortic "redundancy" or "undulation," wherein the ascending aorta forms a

prominent silhouette on thoracic films, along with a more sternally positioned heart, is commonly observed in older cats and is an incidental finding.⁵⁷ One study suggested that this finding is associated with systemic hypertension.⁶⁰

Vertebral heart scale (VHS) has been developed for assessment of cardiac size in cats⁴⁶ and can help with identification of atrial enlargement or generalized cardiomegaly. A VHS greater than 8.1 is consistent with cardiomegaly in the cat (Figures 20-1 and 20-2). However, clinicians should recognize that the most common adult-onset feline disease (HCM) often does not cause radiographically detectable ventricular enlargement, so a normal VHS does not rule out the presence of significant heart disease in cats.

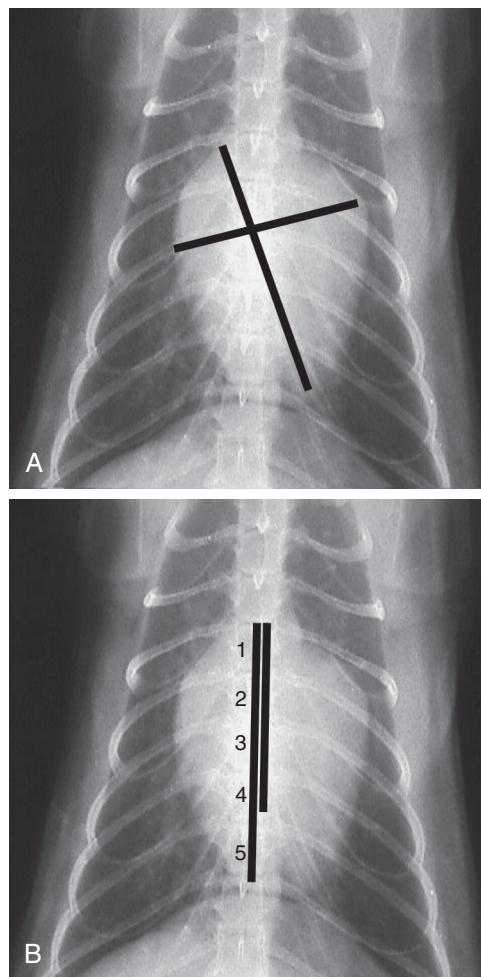


FIGURE 20-1 Measurement of vertebral heart scale on the dorsoventral radiographic view. **A**, A line is drawn along the long axis of the heart from right atrium to left ventricular apex. A second line is drawn perpendicular to the long axis, spanning the atria. **B**, These lines are then transferred to the cranial edge of the fourth thoracic vertebra, and the number of vertebrae spanned by the two lines is summed. In this example the vertebral heart scale is approximately 9 vertebrae.

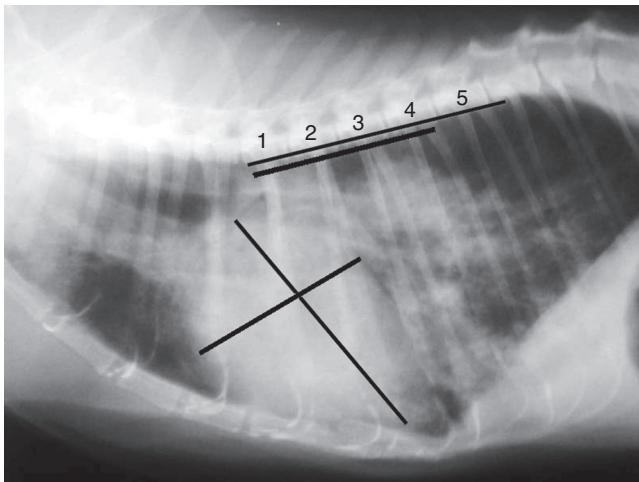


FIGURE 20-2 Measurement of vertebral heart scale on the lateral radiographic view. The same method is used as for the dorsoventral view. In this example the vertebral heart scale measures 9 vertebrae. Note the marked interstitial–alveolar pattern in the caudal and accessory lung lobes, consistent with congestive heart failure (pulmonary edema).

Assessment of pulmonary vasculature is substantially less reliable in cats than in dogs. Venous or arterial enlargement is subject to considerable misinterpretation and rarely accurately reflects the pulmonary hemodynamic state. In some cats with CHF, the pulmonary vasculature on the lateral projection appears to be pronounced, but this is subjective and unreliable.

Diagnosis of CHF in cats is aided by thoracic radiographs. Clinicians should not make a diagnosis of CHF in the absence of supportive clinical signs (i.e., radiographs should not be the primary means by which the diagnosis is made). Ideally, marked cardiomegaly is apparent radiographically to support the hypothesis of severe heart disease underlying the pulmonary changes. However, in many cats severe pulmonary changes (pulmonary edema or pleural effusion) obscure the cardiac silhouette, making interpretation of cardiac size impossible. In contrast to dogs, pulmonary edema in cats has little radiographic consistency.⁵⁰ One study of 23 cats with CHF showed at least six distinct pulmonary parenchymal patterns indicative of pulmonary edema (Figures 20-3 and 20-4).⁷ Thus pulmonary edema cannot be excluded on the basis of a radiographic pattern that is different from that seen in most dogs. This can complicate the diagnosis of CHF in cats when the cardiac silhouette is not clearly visible.

Echocardiography

Echocardiography remains the most useful tool in identifying feline heart disease. Feline echocardiography requires substantial skill in both acquisition and interpretation of data. Additionally, many of the more

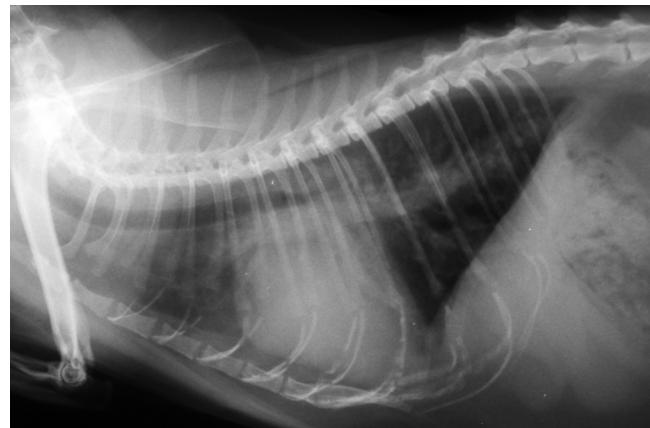


FIGURE 20-3 Lateral radiograph showing congestive heart failure secondary to hypertrophic cardiomyopathy in a cat. Note the prominent caudal lobar vessels with perivascular interstitial pattern. The left atrium is markedly enlarged.



FIGURE 20-4 Dorsoventral radiograph showing congestive heart failure secondary to hypertrophic cardiomyopathy in a cat. Note the marked cardiomegaly and heavy interstitial pattern throughout the lung fields.

common conditions require spectral and color Doppler analysis. Thus feline echocardiography remains largely a specialist diagnostic test. It is important to recognize these requirements when considering echocardiography for a patient because an incomplete or substandard echocardiographic evaluation can impoverish the client without providing a diagnosis.

Because of the cat's high heart rate and small heart size, imaging is usually performed with a high-frequency transducer (7 to 10 MHz). Advances in processing

capabilities of ultrasound machines over the last 20 years have allowed most measurements of chamber and wall dimensions to be made from two-dimensional images rather than M-mode images. This also allows the echocardiographer to measure dimensions in regions not measurable by M-mode echocardiography (e.g., anterior and posterior aspects of the left ventricular wall). Linear and area chamber dimensions can be obtained.

A detailed explanation of echocardiographic technique is beyond the scope of this chapter, and readers should consult cardiology or echocardiography textbooks for additional details.

Biomarkers

Most recently, biochemical indicators of heart disease have been developed and marketed. These include cardiac troponin I; atrial natriuretic peptide (ANP) and its prohormone, NT-proANP; and B-type natriuretic peptide (BNP) and its prohormone, NT-proBNP. These are proteins either secreted or released by cardiomyocytes in response to stretch or injury and can be measured in serum or plasma. In humans these biomarkers have allowed early, rapid identification of acute myocardial injury and stratification of patients for appropriate acute interventions or additional diagnostic testing.

Use of biomarkers in feline medicine has been restricted largely to identification of subclinical heart disease (i.e., as a screening test) and differentiation of causes of acute dyspnea (cardiogenic versus pneumogenic/other).

NT-proBNP as a Screening Test

Several studies have examined the use of NT-proBNP as a screening test for cardiac disease in cats, namely HCM. While the test showed a difference in NT-proBNP concentrations between "affected" and "unaffected" cats in these studies and good or very good sensitivities and specificities for distinguishing "affected" and "unaffected" cats, most of the studies did not stratify the cats according to severity of subclinical disease. Only two small studies have looked at the ability of NT-proBNP to identify cats with HCM with varying degrees of subclinical disease.^{35,78} The authors of the first study showed that only cats with severe myocardial hypertrophy (but not moderate or equivocal changes) could be somewhat confidently identified as being "affected." However, when the authors repeated the study with a new version of the assay, even this ability to detect severely affected cats was compromised. Thus, on the basis of these data, a high NT-proBNP might be expected to help rule in a cat with severe subclinical disease (not many false-positive results) but would not be able to rule out cats with HCM (many false-negative results). Additionally, personal observations by the author suggest that false-positive findings in cats are more common than reported

in these studies. Finally, the assay has undergone substantial modifications since these studies were performed and has not yet been re-evaluated. Therefore substantially larger cross-sectional studies are needed, with patients stratified into degrees of subclinical severity and the modified version of the assay used.

NT-proBNP as a Diagnostic Test for Congestive Heart Failure

An alternative use for this assay has been directed at discriminating causes of dyspnea or respiratory distress. Several studies have shown that populations of unaffected cats or cats with dyspnea resulting from acute respiratory disease have lower NT-proBNP concentrations than cats with dyspnea from CHF. However, some overlap exists. One study, by Connolly and coworkers¹⁶ showed that approximately 80% of cats would be correctly diagnosed on the basis of NT-proBNP concentrations. However, this also suggests that one in five cats would be incorrectly diagnosed and potentially inappropriately treated, with possible life-threatening consequences.

Diagnosis of the cause of tachydyspnea in cats can be difficult when the cardiac silhouette is obscured by pleural effusion, fat, or other pulmonary parenchymal changes. When the "probability of CHF" equals the "probability of not CHF," an NT-proBNP measurement might increase the odds of correct diagnosis. However, because such clinical situations are acute and demand rapid intervention, until the assay becomes available as an in-house rapid assay, it will have little value in diagnosis or treatment of acute tachydyspnea in cats, insofar as a therapeutic decision will likely have been made well before test results become available.

Whether the assay can improve the probability of correct diagnosis beyond what is achieved with current diagnostic tests (physical examination, history, echocardiography, radiography) is not known. Further studies are needed to demonstrate the true clinical efficacy of biomarkers in feline medicine.

Abuses of Biomarker Assays

Any screening test should only be applied to an "at-risk" population, rather than being performed indiscriminately. Furthermore, a screening test should be either specific or sensitive (depending on whether the veterinarian wishes either to rule in a disease or to rule out a disease), or both (which is rare). It should be affordable. An early diagnosis should allow intervention that either alters disease outcome or reduces risk of adverse events. It could be argued that every adult cat is at risk for having subclinical heart disease. However, there are currently no therapies known to alter disease progression in cats with heart disease (with the exception of the almost extinct taurine-deficient myocardial failure). Thus identifying HCM early in the course of the disease

does not allow the clinician to alter the outcome for that patient. Because of this, random testing is not recommended, insofar as the probability of false-positive results (with consequent costly further investigations) far exceeds the probability of true-positive results. One could argue for the use of NT-proBNP as a preanesthetic test, although, as stated earlier, additional studies are required to better evaluate the validity of the assay.

FELINE HYPERTENSION AND HEART DISEASE

Definitions

Systemic hypertension is well documented in cats and can result in significant morbidity and potentially mortality. Clinically, systolic hypertension is identified most commonly, whereas diastolic hypertension has not been reported routinely. Systolic hypertension has been defined as a systolic blood pressure (BP) greater than 160 mm Hg. A grading scheme has been devised, with increasing levels of hypertension presumably associated with worse outcomes ([Table 20-1](#)).¹⁰

Causes

Feline systemic hypertension is generally thought to be associated with underlying systemic diseases, such as renal disease and hyperthyroidism. Essential hypertension and geriatric hypertension (the most common causes of hypertension in humans) have not been recognized as distinct entities in domestic animals. These forms of hypertension are secondary to various, mostly genetic factors (compounded by environmental factors, such as obesity) in the former case and loss of vascular elastance in the latter case. Thus any feline patient diagnosed with systemic hypertension should have an underlying etiology identified. Renal disease is the most common cause of systemic hypertension in cats, resulting in hyperreninemic hypertension. However, measurement of plasma renin activity is not routinely available

TABLE 20-1 Classification of Systolic Blood Pressure by Risk of Target Organ Damage in the Cat

Risk Category	Systolic Blood Pressure (mm Hg)	Risk of Target Organ Damage
I	<150	Minimal
II	150-159	Mild
III	160-179	Moderate
IV	>180	Severe

Modified from Brown S, Atkins C, Bagley R et al: Guidelines for the identification, evaluation, and management of systemic hypertension in dogs and cats, *J Vet Intern Med* 21:542, 2007.

to document a renal etiology. Because renal disease in cats most commonly manifests initially as a loss of urine-concentrating ability, the majority of older cats with hypertension would be expected to have suboptimal urine-concentrating ability.

Clinical Signs

Systemic hypertension has been described as a “silent killer” in humans because clinical signs of disease are often inapparent while end-organ damage is occurring. Similarly, in feline patients, few clinical signs are apparent, and they may be subtle and nonspecific, such as anorexia and lethargy. Renal disease can be extremely mild and not clinically apparent. The most common clinical presentation with severe systemic hypertension is acute retinopathy (retinal separation or hemorrhage) (see [Figure 29-59](#)).⁵² Ocular signs may also include dilated pupils and hyphema. Experimentally, acute severe hypertension has resulted in hemorrhagic encephalopathy (“stroke”), but this is not commonly identified in cats with spontaneous disease. Neurologic signs may include head tilt, ataxia, disorientation, and seizures. Cardiac murmurs have been associated with hypertension; however, there is no physiologic reason for systemic hypertension to produce turbulence that would result in a murmur. Thus it is likely that this association is coincidental rather than causal. Several authors have reported cardiac changes in cats with hypertension, including concentric left ventricular hypertrophy and redundancy of the ascending aorta.^{12,14,34,60} These changes are generally mild but can be confused with a diagnosis of HCM.

Diagnosis

The diagnosis of systemic hypertension in cats is problematic for several reasons. First, the equipment currently available for noninvasive blood pressure (NIBP) measurement is neither accurate nor precise. Despite multiple studies claiming to validate NIBP systems, most have not compared to a true gold standard (direct, invasive telemetric instantaneous BP monitoring). Additionally, many of these studies have examined systems in anesthetized cats rather than conscious animals. Recently, a study comparing oscillometric systems with direct measurements in anesthetized cats found that no system was sufficiently accurate or precise to be useful.² Recommendations for measuring NIBP in cats have been made by multiple investigators who suggest that four measurements should be made, with the first measurement discarded and the remaining three measurements averaged. However, this method does not guarantee either accuracy or precision of the measurement.

Several NIBP systems are currently marketed and used for measuring systolic blood pressure (SBP) in cats:

traditional oscillometric systems; Doppler systems; and, most recently, high-definition oscillometric (HDO) systems. Anecdotal perceptions among clinicians suggested that Doppler-based methodology was more accurate than oscillometric methodology, but unpublished studies comparing both systems in the same animals against invasive measurements have failed to support this perception; both methodologies are equally inaccurate.^{18b} No studies exist detailing performance of HDO systems in cats. However, studies in dogs suggest that these systems would perform no better than standard oscillometric and Doppler systems.⁸⁷

Complicating the accurate measurement of SBP is the lability of feline BP. The author has observed conscious cats, gently restrained and accustomed to handling, with SBP measurements that vary by as much as 100 mm Hg in the space of a few seconds with little change in heart rate or perceived stress level. Many clinicians attempt to measure SBP at the patient's home to reduce the impact of stress, but no studies have demonstrated that this strategy results in more accurate measurement. One study examining the effect of conditioning on SBP in dogs showed that repeated measurements over several weeks resulted in a significant gradual reduction in measured BP as the "white-coat" effect subsided in these patients.⁷³ A similar pattern might be anticipated in cats. A study in cats demonstrated a significant white-coat effect in cats that would result in substantially higher SBP measurements than those obtained at rest.⁶

Sustained SBP above 200 mm Hg generally results in end-organ damage. This can often be appreciated in retinal vascular pathology because retinal blood vessels are exquisitely susceptible to hypertensive injury. Vascular engorgement and tortuosity or retinal hemorrhage should be searched for if repeated SBP measurements exceed 200 mm Hg. If the retinas appear normal, the diagnosis of systemic hypertension should be questioned. However, repeated measurements above 220 mm Hg are likely indicative of true hypertension. Similarly, evidence of severe hypertension in patients with acute retinopathy increases the probability of an accurate diagnosis.

Clinicians should adopt several steps in the diagnosis of feline systemic hypertension:

1. Examine the appropriate target population. Hypertension is mostly a geriatric disorder in cats. Cats younger than 8 or 9 years of age rarely have systemic hypertension. Therefore BP should not be measured in healthy young to middle-aged cats because the prior probability of these patients actually having hypertension is very low, unless they have renal disease. Indeed, the value of routine BP screening in cats is questionable; it might be more prudent to perform routine urinalysis and an ophthalmic examination and restrict BP

measurement to those patients with either inappropriate urine concentrating ability or retinopathy.

2. Obtain multiple measurements over several weeks if an apparently healthy patient is presumptively diagnosed with hypertension on a routine examination. Because hypertension is a chronic progressive disorder, rapid diagnosis is generally not required (unless there is apparent end-organ damage). This can be laborious for both clinician and client, but it reduces the risk of a false-positive diagnosis.
3. Obtain measurements in a quiet environment, using the same technique each time. BP measurement should be performed before the physical examination or any diagnostic procedures, such as blood or urine collection, are performed. Some Doppler machines allow the use of headphones to minimize noise (Figure 20-5). The width of the BP cuff should be 30% to 40% of the circumference of the leg, and it should be positioned at the level of the heart (Figure 20-6). Use the same trained personnel for each measurement to prevent interoperator variability. Record the cuff size and location of the measurement in the medical record along with the BP readings.
4. Accept the lowest reading obtained over several sessions as the most likely.
5. Examine the patient for underlying renal disease (including a urinalysis) and other endocrine



FIGURE 20-5 Use of headphones with Doppler blood pressure units minimizes noise that might be stressful to the patient.

disorders that might result in systemic hypertension. If no underlying cause can be identified, reconsider the diagnosis of hypertension.

6. Perform a retinal examination to identify hypertensive retinopathies, especially if the SBP is consistently above 200 mm Hg.
7. Remain skeptical of the diagnosis in every patient in which the diagnosis was unexpected or unexplained.

One study recently evaluated the use of NT-proBNP in hypertensive cats with chronic kidney disease (CKD).⁴⁴ These authors found elevated NT-proBNP concentrations in hypertensive cats with CKD and occasionally in normotensive cats with severe CKD. Thus NT-proBNP might help identify hypertension in cats with CKD in the absence of cardiac disease. Additional larger studies would be necessary to confirm this initial observation.

Treatment

Treatment of systemic hypertension generally involves administration of arteriodilators (**Table 20-2**). The most



FIGURE 20-6 The width of the blood pressure cuff should be 30% to 40% of the circumference of the leg.

common drug used for management of feline hypertension is amlodipine. This is usually administered at 0.625 mg per cat, every 12 to 24 hours, by mouth. The medication can be administered transdermally, but reductions in SBP are less predictable and of lesser magnitude than with oral administration.³³ Reductions in SBP of 20 to 50 mm Hg can be anticipated with oral amlodipine therapy.

Other drugs that have been examined in management of hypertension include angiotensin-converting enzyme (ACE) inhibitors, beta blockers, and hydralazine. None of these medications has proved to be effective in routine control of SBP or has been associated with undesirable side effects. In cases of hypertension that are refractory to high-dose amlodipine therapy, clinicians can consider adding ACE inhibitors or beta blockers to the therapeutic protocol, although the outcomes are not predictable.

Despite the popularity of both diagnosis and treatment of feline hypertension over the last 10 to 15 years, no studies have documented decreased morbidity or increased survival rates in treated spontaneously hypertensive cats. One study examining the effect of good and poor control of hypertension in cats with renal disease failed to show any increase in survival of cats with good control compared with cats with poor control of SBP, suggesting that management of hypertension in this population of cats might not result in improved clinical outcomes.³⁹ These authors examined survival factors and found that the only variable that predicted survival was proteinuria. The author is unaware of any studies examining rates of nonfatal complications (e.g., retinopathies, progressive renal disease, encephalopathies) in cats with well-controlled versus poorly controlled hypertension. Thus whether morbidity is affected by antihypertensive therapies is unknown. Clinicians should consider that therapy in many patients presumptively diagnosed with hypertension might be of no clinical value. Additionally, therapy of cats with borderline hypertension should be questioned both because of the inherent inaccuracy of diagnosis and the lack of any documented benefit to such therapy.

TABLE 20-2 Drugs Used in the Treatment of Feline Hypertension

Drug	Dose	Mechanism	Comments
Amlodipine	0.625 to 1.25 mg/cat, every 12 to 24 hours, orally	Calcium channel blocker	Preferred first-line therapy; may be combined with ACE inhibitor or beta blocker in refractory cases
Benazepril	0.25 to 0.5 mg/kg, every 12 to 24 hours, orally	ACE inhibitor	Adjunctive therapy, especially for cats with proteinuric renal disease
Hydralazine	1 to 3 mg/kg, every 12 hours, orally	Direct arterial dilator	Primarily used for acute control of hypertension in the hospital setting
Atenolol	6.25 to 12.5 mg/cat, every 24 hours, orally	Beta-adrenoceptor blocker	Primarily used in hyperthyroid cats or as adjunctive therapy for refractory cases

ACE, Angiotensin-converting enzyme.

CARDIOMYOPATHIES

Cardiomyopathies account for most acquired feline cardiac disease. Several types of cardiomyopathy have been described in cats: hypertrophic, dilated, restrictive, unclassified, arrhythmogenic right ventricular cardiomyopathy, excess moderator band, and endomyocardial fibroelastosis. Instances of isolated atrial cardiomyopathy have also been described. Of these, HCM is the most commonly diagnosed.

Hypertrophic Cardiomyopathy

HCM is a concentric hypertrophy of the ventricular myocardium, either diffuse or localized, that is not attributable to any identifiable cause such as hypertension, hyperthyroidism, neoplasia, or increased afterload (e.g., aortic stenosis). Causes of feline HCM are mostly unknown, although, as in humans, genetic mutations likely account for some percentage of cases. Most mutations in humans with HCM have been detected in sarcomeric proteins (proteins associated with the contractile apparatus). A genetic mutation in myosin-binding protein C has been proposed as a cause of HCM in Maine Coons⁵³ and Ragdolls,⁵⁵ although two subsequent studies have disputed the initial observation.^{13,88}

Prevalence of HCM in cats is also poorly estimated. Relatively small studies of apparently healthy cats have identified left ventricular hypertrophy in 7% to 15% of cats.^{18,61} These estimates seem alarming, especially given the fact that most cats with HCM in these studies were random-source, unrelated, domestic shorthair or long-hair cats, rather than breeds predisposed to HCM, and that similar studies in people put the estimate at 0.2% of the general population. Such an “epidemic” of HCM is difficult to explain through genetic causes and requires invoking of either infectious or other environmental etiologies. Indeed, authors have proposed nongenetic causes for feline HCM. Alternatively, because the diagnosis is based on echocardiographic evaluation and measurement, and given that many, if not most, of these cats remain subclinical for their entire life, it is possible that current diagnostic criteria are insufficiently stringent for accurate diagnosis of this condition, resulting in a high percentage of false-positive diagnoses. Furthermore, no studies have examined a large cohort of apparently healthy cats longitudinally to determine whether initial observations of hypertrophy persist over time. Such studies are currently being conducted, but results cannot be expected for several years.

The median survival rate of cats from the time of diagnosis of HCM approaches 5 years. Some studies have suggested an 80% survival rate at 5 years of cats diagnosed with subclinical HCM.^{3,72} Thus the outcome appears to be highly variable. This further supports the

hypothesis that not all idiopathic left ventricular hypertrophy, diagnosed echocardiographically, is HCM. Alternatively, this finding could be attributed to a wide range of expression of the disease in affected individuals; in humans with HCM, identical mutations can cause a wide range of phenotypes, ranging from apparently unaffected to severely affected.

Clinical Signs

Cats with HCM can present with a variety of clinical signs and physical examination findings. Murmurs are present in approximately 50% of cats with HCM. Conversely, cats with murmurs do not necessarily have HCM or even cardiac disease. Therefore presence or absence of a murmur is not useful in identifying cats with HCM. Murmurs in cats with HCM are often associated with systolic anterior mitral valve motion, which produces both a dynamic left ventricular outflow obstruction and mitral regurgitation (Figure 20-7). Some cats with HCM can develop dynamic right ventricular outflow tract obstruction.

Arrhythmias are commonly observed in cats with subclinical HCM. A recent study has suggested that most apparently healthy cats with ventricular arrhythmias have evidence of structural heart disease; however, this study requires additional validation with larger sample populations.¹⁷

Most commonly, cats with clinical HCM present with left-sided CHF. Cats often display subtle signs of CHF until they reach a critical tipping point, at which time they decompensate rapidly. Subtle clinical signs can include mild tachypnea, altered grooming behavior or activity, and decreased appetite. Coughing is a rare clinical finding in cats with CHF; the vast majority of cats with cough have noncardiac disorders, such as asthma. CHF in cats with HCM manifests as pulmonary edema,

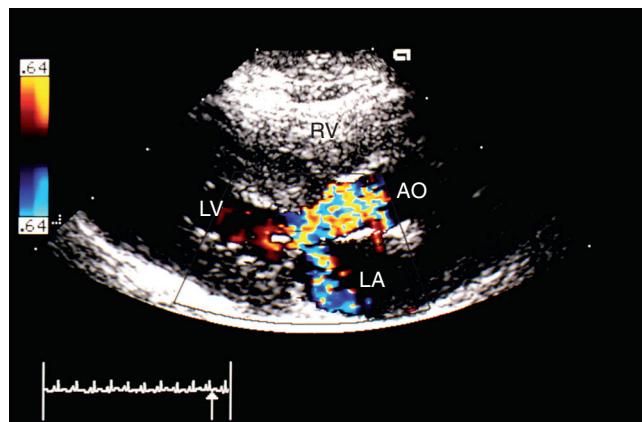


FIGURE 20-7 Right-parasternal long-axis color Doppler echocardiographic image demonstrating the divergent jets characteristic of systolic anterior motion of the mitral valve. Turbulence is seen extending into the aorta, and a second discrete jet is seen extending towards the posterior wall of the left atrium.



FIGURE 20-8 Pleural effusion often accompanies pulmonary edema in cats with congestive heart failure and may make it difficult to visualize the heart as well as the lung fields.

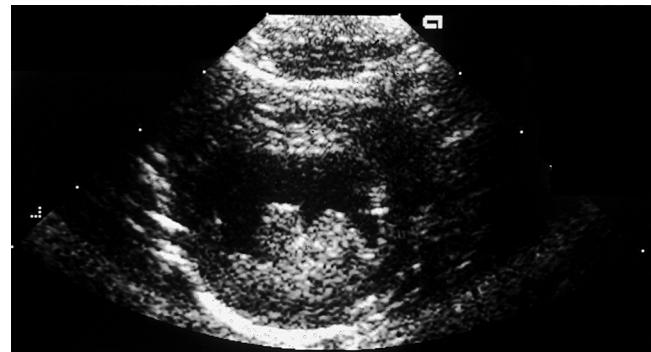


FIGURE 20-9 Right-parasternal long-axis echocardiographic image demonstrating marked papillary hypertrophy and fusion of a cat with hypertrophic cardiomyopathy. Note that the septum and lateral walls do not appear markedly thickened in this patient.

pleural effusion, or both. Rarely, pericardial effusion can be detected, but this is generally mild and of no clinical hemodynamic consequence. Distribution and radiographic pattern of pulmonary edema in cats with CHF is highly variable.⁷ Therefore clinicians should not rely on identification of “typical” radiographic findings when making the diagnosis of CHF. Pleural effusion often accompanies pulmonary edema (Figure 20-8), but substantial effusion will obscure the radiographic pattern of pulmonary edema. Pleural effusion can be a modified transudate or chylous.

Cats with severe CHF exhibit marked tachydyspnea or respiratory distress. Body temperature can be normal or low; hypothermia with CHF is a poor prognostic indicator. Similarly, cats can be tachycardic, have atrial or ventricular arrhythmias, or be normocardic. Cats with an absence of tachycardia at diagnosis of CHF also carry a worse prognosis than those with tachycardia.

Diagnosis

Diagnosis of HCM requires echocardiography. Left ventricular wall thicknesses (either globally or regionally), measured at the standard submitral location, that exceed 6 mm constitute a tentative diagnosis of HCM (Figure 20-9). Cats with wall thicknesses exceeding 7 mm are considered to have moderate left ventricular hypertrophy. Hypertrophy can be focal or diffuse. More controversy exists regarding basilar septal bulges or thickening. This is a common finding in older cats and can cause dynamic left ventricular outflow tract obstruction. However, whether this constitutes HCM or simply is a consequence of aging changes is unclear.

A diagnosis of HCM cannot be made on the basis of ECG and radiography. Many cats with subclinical disease have a normal electrocardiographic reading and a normal cardiac silhouette on thoracic radiographs. Conversely, changes on radiographs consistent with left atrial enlargement (especially on the DV view) are not pathognomonic for HCM but merely indicate

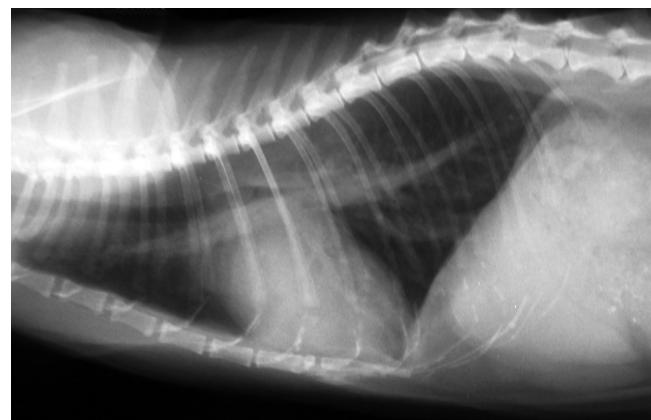


FIGURE 20-10 Lateral radiograph of a cat with severe hypertrophic cardiomyopathy. Note the cardiomegaly, enlarged pulmonary veins, and the moderate generalized interstitial pulmonary pattern, consistent with congestive heart failure.

cardiomegaly and left-sided heart disease (Figures 20-10 and 20-11). ECG is both insensitive and nonspecific for diagnosis of cardiomegaly and should be reserved for diagnosis of arrhythmias.

Biomarkers do not currently appear to be of use in diagnosis of subclinical HCM. Their use in diagnosis of CHF is also unresolved at this time; studies demonstrating true clinical utility of the assay do not exist. However, when ECG is unavailable and radiographs cannot demonstrate cardiomegaly (e.g., owing to obscuring of the cardiac silhouette by pleural effusion or pulmonary edema), NT-proBNP might provide useful information in a subset of cats with CHF, allowing clinicians to increase the probability of a correct diagnosis and institute timely and appropriate therapy.

Genetic testing is available for specific cardiac mutations associated with HCM. These tests are reserved for specific breeds in which the mutations have been identified rather than as a general screening tool. Two



FIGURE 20-11 Dorsoventral radiograph of a cat with severe hypertrophic cardiomyopathy. Note the marked left atrial enlargement and moderate generalized interstitial pulmonary pattern, consistent with congestive heart failure. Note also that left atrial enlargement is significantly more apparent than on the corresponding lateral radiograph seen in Figure 20-10.

mutations have been identified in myosin-binding protein C that have been associated with HCM in Maine Coons and Ragdolls.^{53,55} The investigators who identified these mutations have suggested that cats homozygous for the mutation die in utero (i.e., the mutation is embryonically lethal when both alleles express the mutation). However, recent studies from investigators in Europe have contested these claims, having identified cats homozygous for the mutation both with and without echocardiographic evidence of HCM.^{13,88} These investigators have further contested the hypothesis that the proposed mutation in Maine Coons is associated with HCM in that breed. However, the European investigators used different methodology to identify and determine genotype in their cats and examined predominantly younger cats, in which the disease might not yet be apparent. Thus whether the mutation is causal and differences in phenotype merely reflect expressivity of the trait or whether the mutation is a noncausal polymorphism is still subject to debate. Additional evidence for causality has been proposed in a recent study by the investigators who originally identified the mutation, in which the authors demonstrated altered methylation of CpG sites within the MyBPC gene.⁵⁴

Additionally Maine Coons without the MyBPC mutation have been identified with HCM, both in the colony

where the mutation was initially identified and in the general Maine Coon population.^{13,88} Therefore a normal genotype in this breed does not exclude the diagnosis of HCM.

Treatment

Treatment of HCM is controversial. Currently, no therapeutic studies exist demonstrating clinically important outcomes in cats with subclinical HCM. No drugs have demonstrated a delay in progression or reversal of hypertrophy in cats with subclinical HCM. One study examining ACE inhibitor therapy in subclinical disease failed to show regression of hypertrophy over 1 year; however, the study did not examine whether long-term therapy with ACE inhibitors prevented or delayed the onset of CHF or arterial thromboembolism (ATE).⁵¹ One unpublished study showed that beta blockers reduced the dynamic outflow obstruction in cats with subclinical disease better than calcium channel blockers.⁷⁴ However, whether reducing the obstruction alters clinical outcomes, such as progression to CHF, was not examined. Arguments that reducing obstruction alters "demeanor" or "behavior" are difficult to accept because these cats are, by definition, without clinical signs ("subclinical"). Furthermore, beta blockers have psychotropic effects, so attributing any change in behavior to reduction in left ventricular outflow tract obstruction without a controlled study is improper.

Treatment of CHF in cats is similarly devoid of published evidence. Diuretics are the mainstay of therapy, both in acute and chronic settings. One unpublished study that compared addition of beta blockers, calcium channel blockers, ACE inhibitors, or placebo with furosemide on survival of cats with chronic CHF failed to demonstrate a benefit of any therapy.²⁴ In that study ACE inhibitors tended to improve outcomes, and beta blockers worsened outcomes, compared with placebo. A prior study of calcium channel blockers (diltiazem) and beta blockers in cats with CHF and HCM suggested a survival benefit of diltiazem; however, no placebo group was included in that study to determine whether the difference was due to a benefit of diltiazem or harm from beta blockade.⁹ Recent anecdotal evidence from several investigators has suggested that pimobendan does not appear to dramatically worsen clinical outcomes in cats with HCM and CHF, despite initial concerns of administering a positive inotrope to a cat with HCM. However, no controlled studies have demonstrated a clear benefit of pimobendan therapy in HCM. Thus clinicians should carefully consider their treatment strategies for cats with CHF, remembering that polypharmacy in cats is often substantially more difficult than in dogs and that adding drugs may not improve clinical outcomes and may worsen quality of life for both client and patient.

Acute treatment of CHF in cats is best accomplished by adhering to several simple rules:

- Undue stress can kill a cat with respiratory distress. Therefore physical restraint for diagnostic procedures should be minimal, brief, and gentle. Consider obtaining DV and standing lateral films if necessary (and do not worry excessively about positioning). Do not perform diagnostic tests that require sedation or extensive manual restraint.
- Before any imaging studies, especially if echocardiography is not available, perform a bilateral diagnostic (and potentially therapeutic) pleurocentesis. This can be done with the patient in either sternal recumbency or sitting on the examination table (Figure 20-12). A 23G butterfly needle, coupled to a 10 mL syringe and three-way stopcock, is sufficient for most cases (Figure 20-13). This procedure allows quick and easy identification of severe pleural effusion (especially if prior thoracic auscultation is suggestive) and evacuation of fluid. Even removal of 50% of the volume of pleural effusion can dramatically alleviate dyspnea in feline



FIGURE 20-12 Pleurocentesis should be performed on patients with respiratory distress before imaging studies. The patient can be positioned in sternal recumbency; undue stress should be avoided.



FIGURE 20-13 Pleurocentesis can be performed with a 23G butterfly needle coupled to a 10-mL syringe and three-way stopcock.

patients with CHF, allowing further diagnostic tests to be performed in a hemodynamically more stable patient.

- Do not provide oxygen by face mask; this is generally too stressful for a cat. Consider placing the patient in an oxygen cage to improve oxygenation, and then stage diagnostic procedures such that the patient can be allowed time to recover in the oxygen cage.
- If intravenous catheterization is not easily achievable with minimal patient restraint, administer furosemide intramuscularly. Doses for severe respiratory distress should approach 4 mg/kg every 2 to 4 hours until substantial improvement in respiratory effort and rate are noted. Doses should then be tapered to prevent excessive dehydration and electrolyte depletion.
- If unsure of the diagnosis of CHF, perform a brief echocardiogram with the patient on your lap to determine if there is marked left atrial enlargement. Most cats with HCM and CHF have enlarged left atria. A complete echocardiogram is not required during the acute management period, but a brief gentle evaluation of left atrium size is often beneficial in establishing the diagnosis and directing appropriate therapy.
- Resist the temptation to perform repeated physical examinations of the patient; observe the patient's respiratory character through the cage, and handle the patient only when absolutely necessary.
- Do not administer fluids to *any* cat with CHF while initially attempting to reduce pulmonary edema acutely. It is impossible to dehydrate the pulmonary parenchyma and pleural space while hydrating the rest of the body. Instead, provide water *ad libitum* to allow the patient to drink at will.
- Do not evaluate electrolytes repeatedly; this will stress the patient. Such tests can be performed once the patient is stable.
- Do not become concerned by temporary anorexia. Cats with CHF are often anorexic but will generally begin eating within 3 or 4 days of stabilization. Although potassium depletion is possible during this period, it is rarely of major clinical concern.
- Do not worry about development of mild or moderate azotemia. Acute dehydration is expected to result in azotemia; you are not inducing renal failure or damaging kidneys by dehydrating your patient. Treatment of cats with preexisting chronic kidney disease that develop CHF is difficult, insofar as specific therapies for each condition are diametrically opposed. Such cases are best managed by specialists, or clients should be advised of the difficulty associated with this situation so that informed decisions can be made about both acute and chronic management. For more information on

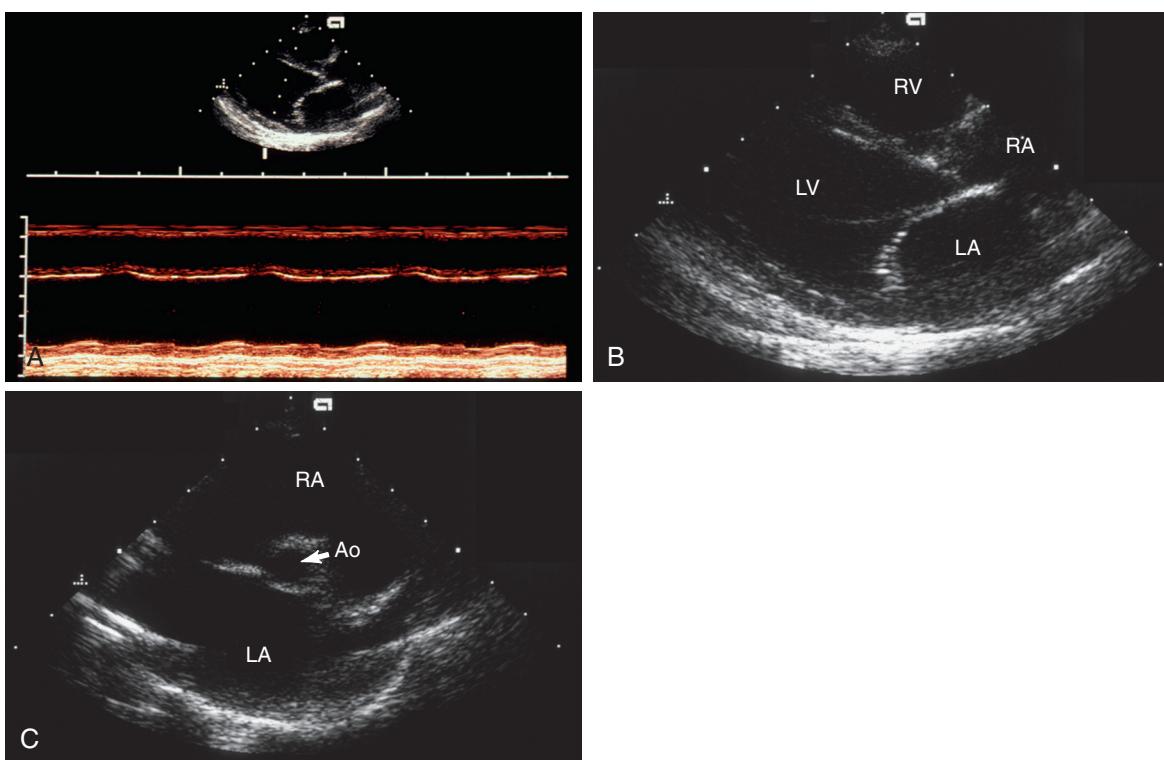


FIGURE 20-14 Echocardiographic images of a cat with dilated cardiomyopathy. **A**, M-mode of the left ventricle showing marked eccentric hypertrophy and myocardial failure. **B**, Right-parasternal long-axis image demonstrating left ventricular and left atrial enlargement (and some right-sided enlargement). **C**, Right-parasternal short-axis image demonstrating a large left atrium.

the management of cats with CHF and chronic renal disease, see Chapter 35.

Clinicians who adhere to these guidelines are likely to resolve an acute CHF crisis in most of their feline patients.

Dilated Cardiomyopathy

Dilated cardiomyopathy (DCM) is an uncommon disease in cats. It is identified echocardiographically as a hypocontractile (usually large) left ventricle (with occasional right ventricular involvement) (Figure 20-14). Subsequent to the identification of taurine-associated DCM in the late 1980s, the prevalence of DCM decreased dramatically. Currently, most DCM in cats is not related to taurine deficiency. However, after the 2008 incident of melamine contamination of pet foods, many clients opted to avoid commercial pet foods and resorted to homemade diets. It is possible that if these diets continue to be fed for prolonged periods, incidence of taurine-associated DCM could increase in cats on homemade diets. Thus clinicians should consider testing any cat diagnosed with DCM for taurine deficiency insofar as such a diagnosis could result in complete cure of the patient. Taurine testing requires specific blood collection if analysis is to be performed on plasma. Heparinized plasma tubes should be prechilled on ice. Blood should

be placed into the chilled tubes and then centrifuged immediately to separate plasma and cells. Plasma can then be drawn off and placed into a regular serum tube (not a separator tube) and shipped cold to laboratories performing taurine analysis.

Cats with DCM present similarly to those with HCM. Many remain subclinical for substantial periods of time. Diagnosis requires echocardiography. Treatment of sub-clinical disease is controversial; no drugs have been studied for their ability to delay onset of clinical signs or reverse disease. Treatment of CHF is the same as for HCM: diuretics, ACE inhibitors, or both. Digoxin can improve contractility in a minority of patients but is associated with substantial risk of toxicity because of its long half-life in cats. Pimobendan has not been critically evaluated in cats with DCM. Anecdotal reports suggest that responses in contractility and clinical picture are underwhelming; however, it can be considered as adjunct therapy if desired.

Other Cardiomyopathies

Several less easily characterized cardiomyopathies exist in cats. Restrictive and unclassified cardiomyopathies (Figure 20-15) are largely indistinguishable antemortem; both result in primary diastolic dysfunction owing to either altered relaxation or altered compliance of the

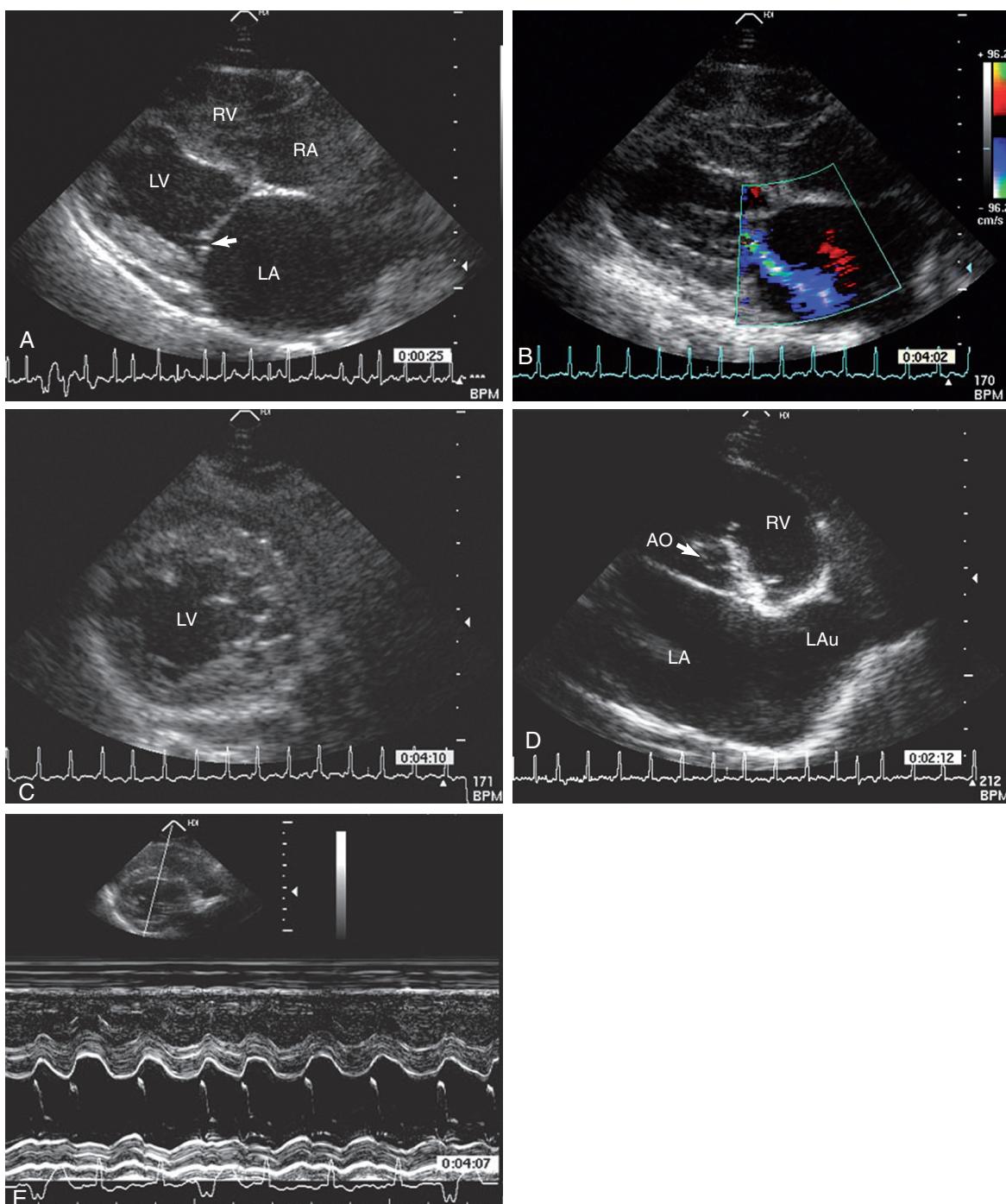


FIGURE 20-15 Echocardiographic images of a cat with unclassified cardiomyopathy. **A**, Right parasternal long-axis view of the heart. Note the four-chamber enlargement. The arrow is pointing to a gap in the coaptation point of the mitral valve, consistent with annular distention. **B**, Color Doppler showing mitral regurgitation. **C**, Right parasternal short-axis view of the left ventricle, showing a normal wall thickness, with marked heterogeneity of the endocardial surface. **D**, Right parasternal short-axis view showing marked left atrial enlargement. **E**, M-mode of the left ventricle at the level of the mitral valve showing moderate chamber enlargement and mild hypokinesis, especially evident in the posterior wall.

ventricles. These two conditions often affect both ventricles, resulting in biatrial enlargement. Diagnosis is by echocardiography. Cats with these cardiomyopathies present with CHF, and personal and anecdotal impressions are that the chance of survival among patients with

these cardiomyopathies is worse than that of patients with HCM.

Arrhythmogenic right ventricular cardiomyopathy (ARVC) is a relatively recently described disorder characterized by fibrofatty infiltration of the right ventricular

wall.²⁷ This results in severe contractile dysfunction of the right ventricle, dilation of the tricuspid annulus, and severe tricuspid regurgitation. The name of this disease is somewhat of a misnomer, insofar as arrhythmias are *not* a feature of the disease; the name was based on a histopathologic similarity to ARVC in humans. Cats with ARVC tend to be older and present with severe right-sided CHF (ascites and pleural effusion). Treatment is generally directed at reducing the effusions by abdominocentesis and thoracocentesis. Survival rates of cats with ARVC have not been extensively examined; however, personal impressions suggest that these patients are relatively resistant to therapy and have a poor quality of life, usually resulting in euthanasia.

Excess moderator band cardiomyopathy is a rare disease of unknown etiology or pathophysiology. It is usually characterized by a rete or network of fibrous bands that traverse the left ventricular chamber and, presumably, alter compliance of the ventricle. Endocardial thickening can be observed in some cases. Additionally, substantial presence of false tendons in cats without apparent heart disease makes the diagnosis of this condition difficult. Treatment is directed against CHF, as described previously.

Atrial standstill has been described in a cat. This is a rare disorder, characterized by atrial enlargement and loss of atrial electrical and mechanical activity with subsequent right-sided CHF. Heart rate is dependent on junctional or ventricular pacemakers. It is different from depressed atrial activity secondary to hyperkalemia in that the latter does not inhibit sinus node activation but merely depresses atrial myocardial depolarization, resulting in a sinoventricular rhythm. Hyperkalemic atrial depression is reversible, whereas atrial standstill is ultimately a fatal condition.

Arterial Thromboembolism

Cardiogenic arterial thromboembolism (ATE) is an uncommon complication of cardiomyopathies in cats.^{4,45,77,82} ATE manifests as sudden occlusive vasculopathy of the systemic arteries. Although the exact pathophysiology of the syndrome is not completely understood, most cardiologists believe that the condition arises when thrombi from the left atrium enter the systemic circulation and lodge in distal locations, with subsequent occlusion of the affected vessels. The distal aorta is the most commonly recognized site of arterial occlusion, and occlusion can be partial or complete. Secondary thrombosis can develop, extending cranially up the aorta to occlude visceral arteries (intestinal, renal). Occasionally, direct splanchnic infarction can occur. Less commonly, thromboemboli will occlude forelimb arteries (right more commonly than left), resulting in unilateral forelimb paresis or paralysis. Cerebrovascular

arterial occlusion is thought to occur on occasion but is difficult to document or substantiate.

Cardiogenic ATE occurs almost exclusively in cats with large left atria. However, atrial enlargement alone is insufficient for development of ATE; this is apparent in the fact that most cats with HCM that develop CHF (and therefore have markedly enlarged left atria) do not develop ATE and that cats with other cardiac diseases, such as VSD and mitral valve disease, can also develop markedly enlarged atria without increased risk of ATE. Thus ATE requires atrial dysfunction, which produces blood stasis or decreased blood flow within the left auricle.⁷⁵ Additionally, alterations in either platelet function or coagulation (or both) might play a role in development of ATE.^{5,84} To date, studies have failed to identify specific risk factors for development of ATE. However, anecdotally, spontaneous echocardiographic contrast (SEC) within the LA is considered by most cardiologists to be a risk factor, insofar as this finding suggests intraatrial blood stasis and aggregation of red blood cells. SEC has been associated with decreased left auricular blood flow and clinicopathologic evidence of hypercoagulability, further supporting the hypothesis that it is a risk factor for ATE.^{75,84} However, not all cats with SEC develop ATE, and cats without SEC can also develop ATE. Presence of an intraatrial thrombus is considered to be a strong risk factor for cardiogenic ATE (Figures 20-16 and 20-17).

Clinical Signs

Clinical signs of ATE depend on the vascular bed occluded and the degree of occlusion. Most commonly, clients observe acute paresis or paralysis, which is accompanied by acute and severe pain in the absence of obvious trauma. The acute pain response is a hallmark of ATE. Pain can persist for several hours and subsides

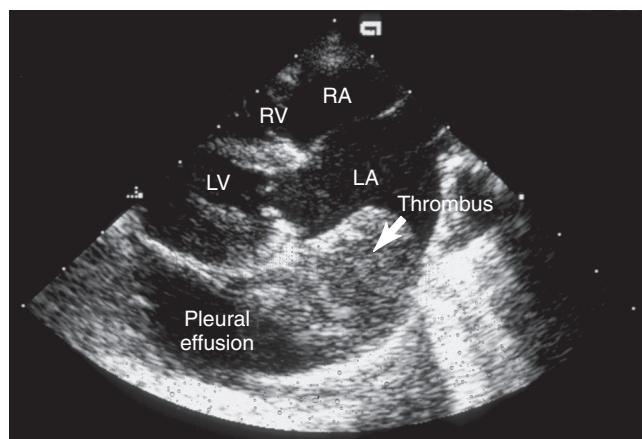


FIGURE 20-16 Right-parasternal long-axis echocardiographic image demonstrating a large left atrial thrombus. A pleural effusion can be observed distal to the heart on the image.

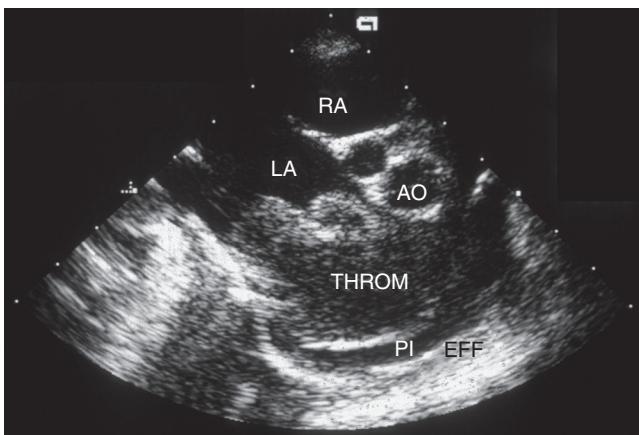


FIGURE 20-17 Right-parasternal short-axis echocardiographic image demonstrating a huge left atrial thrombus.

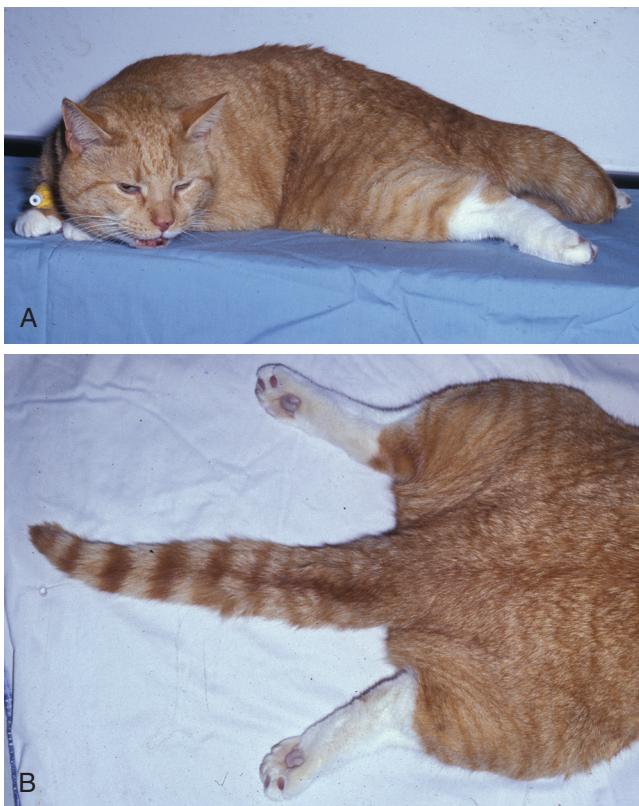


FIGURE 20-18 **A**, A cat with hindlimb paresis secondary to aortic thromboembolism. **B**, Note the cyanotic pads.

as neuronal ischemia develops. Typically, the pain subsides after 24 to 48 hours of complete ischemia. Reperfusion or incomplete occlusion can produce ongoing or recurrent pain. Ultimately, anesthesia of the affected limb (or limbs) ensues. Physical examination can reveal a loss of pulse in the affected limb. Depending on the severity and duration of occlusion, affected limbs can feel hypothermic, with loss of motor and sensory function (Figure 20-18). Nail beds and foot pads can appear

cyanotic. Clipping a toenail on the affected limb to the quick will result in no bleeding or oozing of dark deoxygenated blood. Over several hours swelling and rigidity of affected limb musculature (e.g., gastrocnemius, biceps) can be palpated if the occlusion is severe and persistent. Clinical chemistry analysis will usually reveal elevated creatine kinase and aspartate aminotransferase owing to the ischemic myonecrosis.

CHF is not a necessary feature of ATE; however, in some cats CHF can develop secondary to the stress of the occlusive episode and subsequent hospitalization and therapy. Thus clinicians should carefully monitor respiratory rate and effort in cats presenting and hospitalized for ATE for the possible development of CHF during hospitalization. However, an elevated respiratory rate or mild tachydyspnea could be associated with pain rather than CHF.

If the thromboembolism progresses, renal and gastrointestinal ischemia can ensue. Patients in which this occurs or those with primary splanchnic infarction harbor a grave prognosis and warrant consideration of euthanasia.

The clinical course of ATE is variable. Rarely, complete resolution can be observed in 24 to 48 hours. More commonly, paresis or paralysis persists, with slow return of function or sensation. Occasionally, the larger thromboembolus fragments and dislodges to occlude more distal arteries, resulting in a change in the clinical picture. The more rapidly and completely that function and sensation are regained, the better the short-term prognosis. A partial return of function (sensory or motor) in 48 to 72 hours is encouraging and can be expected in many cases. Persistent complete paralysis beyond this period carries a more guarded prognosis; however, the author has seen cats with complete paralysis that lasted for more than 10 days eventually regain partial motor and sensory function. Provided that the owners are willing to nurse a cat with complete paralysis, including potential bladder expression, clinicians should consider delaying euthanasia in such cases. In general, if complete gangrenous ischemia is not apparent and the toes or foot pads are not completely devoid of blood (black-blue) after 72 hours, the author recommends persisting with therapy if the owners are willing. Additionally, once the acute phase of the syndrome has passed (usually in less than 48 hours), home care can be instituted to minimize stress and costs, with owners being taught to observe for signs of progressive or nonresolving ischemia of the affected limbs.

Treatment

Treatment of ATE is entirely anecdotal and without substantial clinical trials to demonstrate benefit or harm. Most studies of acute therapy consist of small case series. Treatment of acute ATE can be divided into several aims: thrombolysis, promotion of collateral circulation,

prevention of additional thrombosis, control of pain, and treatment of any underlying heart disease.

Thrombolytic therapy has been attempted with various chemical and mechanical approaches, including streptokinase⁵⁸; urokinase⁴³; tissue plasminogen activator⁶³; and, most recently, intravascular thrombectomy.⁶⁷ All these approaches resulted in either substantial peri-interventional mortality (e.g., reperfusion injury) or failure to resolve the thrombus. Thus this author finds it difficult to recommend aggressive thrombolytic therapy in acute ATE.

Fluid therapy is theoretically of benefit (to promote collateral circulation). However, because most of these cats have severe heart disease underlying the ATE, fluid therapy should be used judiciously for fear of producing CHF. Other approaches for improving collateral circulation, such as the use of vasodilators (e.g., acepromazine), have not proved effective. No reports exist regarding the use of more potent arteriodilators such as amlodipine, nitroprusside, and hydralazine, so their use cannot be recommended at this time. Clopidogrel therapy before experimental ATE has been shown to maintain collateral circulation by an as yet unknown mechanism^{34a}; however, whether prophylactic treatment with clopidogrel will reduce episodes of ATE is currently unknown and under investigation.

Prevention of additional thrombosis during the acute phase of the disease has been promoted by various authors. Most commonly, unfractionated heparin can be administered (300 IU/cat, every 12 hours, subcutaneously).¹⁹ However, no clinical studies have demonstrated any benefit to such heparin therapy. Oral antithrombotic therapies have not been shown to be useful in the acute setting of feline ATE.⁸² Experimentally, pretreatment with antithrombotics can reduce ATE; however, this does not reflect the clinical scenario of ATE, in which thrombosis exists at the time of presentation.⁶²

Pain management is paramount in acute ATE. Cats should have routine pain control with fentanyl patches (applied over the more cranial body to ensure absorption) or other acute analgesic therapy such as buprenorphine. Pain control can be reduced after 48 to 72 hours, provided that the patient is comfortable.

Treatment of the underlying heart disease is performed as necessary. Unless there is overt CHF, treatment of the heart disease should be delayed until the patient recovers from the acute ATE episode.

Published case series of ATE suggested that up to 65% of patients can be discharged from the hospital alive (what percentage recovered, and to what degree, was not detailed).^{45,82} Presence of CHF does not appreciably alter these statistics. Hypothermia, loss of anal sphincter, and loss of tail tone are considered poor prognostic indicators (because they demonstrate more profound thromboembolism). Additional studies of prognostic indicators (e.g., return of function) are needed to allow the clinician

to better advise clients before they incur substantial costs.

Once circulation begins to return, clinicians should monitor the color of the foot pads or temperature of the limbs. Furthermore, reperfusion can be painful and associated with hyperkalemia. Therefore serum potassium should be evaluated if clinical signs suggest hyperkalemia is likely.

Chronic therapy of ATE during the recuperative period can include physical therapy. Gentle limb massage and manipulation might help improve circulation or return of function (although there are no studies demonstrating any benefit); in any case, they have little potential for harm, provided the patient tolerates it. Clinicians should advise clients to watch for gangrenous necrosis, the presence of which necessitates either limb amputation or euthanasia. Permanent loss of function in a single limb can be addressed with amputation; however, the underlying heart disease and strong predisposition to recurrent thromboembolism should temper the clinician's eagerness to perform such radical therapy. Clients can be taught to monitor foot pads for changes of color or temperature that might indicate a return of circulation.

Recurrence and Prevention

Limited case series have examined the recurrence of ATE in cats that survive the initial episode.^{45,82} Some of these suggest that most cats will suffer a second event within 6 months of the first, even if they fully recover from the first. There are some patients that have had multiple episodes from which they recovered; however, this is uncommon, at least in part because most owners are unwilling to endure multiple bouts of ATE. Most patients are euthanized if they suffer a second bout of ATE.

Therapy to prevent or delay the occurrence of subsequent episodes of ATE includes the use of aspirin, low-molecular-weight (fractionated) heparin (LMWH)⁷⁹ and clopidogrel. Of these only clopidogrel is being evaluated critically in a randomized trial. This study is currently ongoing at the time of publication of this chapter, so results are not available. One concern with this study is that the comparative group was prescribed aspirin rather than placebo. However, given the lack of evidence of the efficacy of aspirin (low-dose or high-dose), one might argue that aspirin is, in effect, a placebo. Similarly, no clinical studies of the efficacy of LMWH are available. This, coupled with the expense of LMWH therapy and the need for daily injections, makes it difficult to recommend such therapy in cats without additional evidence of efficacy. Some clinicians have considered combining aspirin with clopidogrel to reduce ATE recurrence or occurrence, but no evidence supports this approach.

Prevention of initial episodes of ATE involves the use of the same drugs as for prevention of recurrence: aspirin, clopidogrel, and LMWH, either alone or in combination.

As with recurrent ATE prevention, no clinical trials have demonstrated the value of any of these approaches. Furthermore, clear identification of patients at high risk of ATE that would benefit from preventive therapy has not been studied. Studies evaluating putative risk factors are warranted to help define the subpopulation of cats with cardiac disease that might benefit from early intervention (assuming that early intervention is effective in reducing the risk).

Myocardial Infarction

Myocardial infarction has been documented on necropsy in cats with HCM.²³ However, more recently, cardiologists have recognized what appears to be myocardial infarction in cats without evidence of HCM. Often, these patients have no clinical signs associated with the lesion, which is identified by echocardiography. Affected cats are often older and commonly have other diseases, such as chronic kidney disease. Echocardiographic features include regional thinning of the left ventricular wall or interventricular septum associated with regional dyskinesia or hypokinesia of the affected wall segment. Compensatory eccentric hypertrophy can occasionally occur if the affected region is large. The etiology is usually unknown, and currently no pathologic evidence exists in the veterinary literature to support this antemortem observation. Moreover, no treatment exists to specifically address such presumptive myocardial injury.

ARRHYTHMIAS

Ventricular tachyarrhythmias—ventricular premature contraction (VPC), ventricular tachycardia (VT)—appear to be the most common form of arrhythmias associated with feline heart disease (Figures 20-19 and 20-20). These occur with HCM, unclassified cardiomyopathy, dilated cardiomyopathy, and arrhythmogenic right ventricular cardiomyopathy. Ventricular arrhythmias appear to be common in healthy adult cats. One study of 23 cats showed that 80% had VPCs detectable by 24-hour ambulatory electrocardiography, but 50% of these had less than four VPCs in 24 hours (with a range of 0 to 146 VPCs/24 hours).³⁰ These findings corroborated a previous study of 20 cats wherein VPCs were observed in most healthy cats and increased in frequency with age.⁸⁶

Nonsinus supraventricular tachyarrhythmias are less commonly observed in cats than in dogs. Supraventricular premature complexes, supraventricular tachycardia (Figure 20-21), and atrial fibrillation often require profound atrial enlargement to allow impulse propagation, and in most feline heart disease, unless the disease is severe, the atrial size is insufficient to sustain these arrhythmias. The aforementioned study of 23 healthy cats showed that only one cat had a single atrial premature contraction (APC) over a 24-hour period. The study of 20 apparently healthy cats mentioned previously found a higher rate of APC occurrence, with more APCs

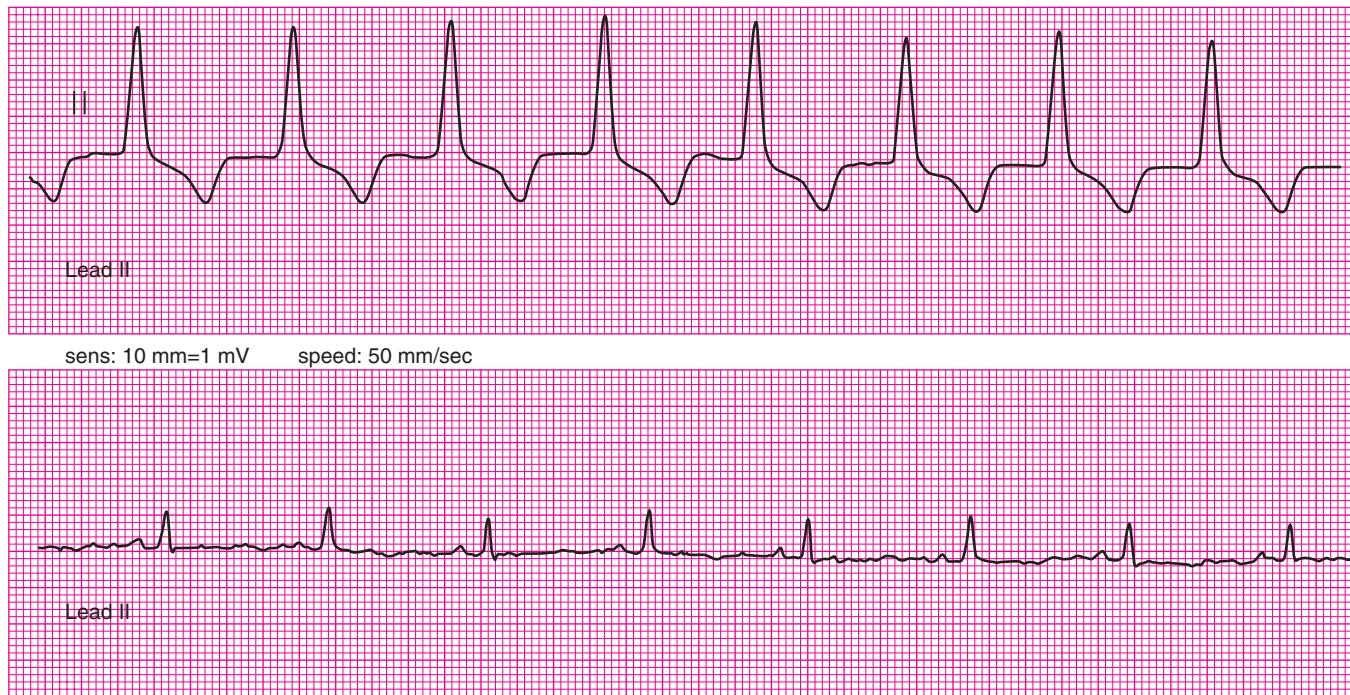


FIGURE 20-19 Electrocardiographic tracing of slow ventricular tachycardia (accelerated idioventricular rhythm) (*top strip*). The rhythm converted spontaneously to sinus rhythm (*bottom strip*).



FIGURE 20-20 Electrocardiographic tracing of nonsustained ventricular tachycardia. *Black arrows* denote normally conducted p-waves; *blue arrows* denote ventricular ectopic complexes.



FIGURE 20-21 Electrocardiographic tracing of a supraventricular tachycardia (*underlined*), which converts spontaneously into a sinus rhythm.

being detected in older cats. Whether these differences can be explained by differences in methodology (at-home monitoring versus in-hospital monitoring) is not clear.

Atrial fibrillation (AF) is almost always associated with severe heart disease in cats,¹⁸ but rare instances of lone or primary atrial fibrillation have been documented.¹⁵ In a study of 50 cats with AF, Côté and coworkers^{18a} identified structural disease in all cases, but 14 of 39 cats (36%) for which left atrial data were available had questionable or mild atrial enlargement, suggesting that factors other than just left atrial size determine the probability of AF in cats. Atrial fibrillation in cats is almost always characterized by a high ventricular response rate, resulting in a tachycardia. Diagnosis is identical to that in other species: a supraventricular tachycardia with no apparent p-waves and an unpatterned irregularity. Diagnosis is complicated in cats because the ventricular rate is often so fast that beat-to-beat irregularity is quite small, and the arrhythmia appears to be regular at first glance. Furthermore, cats often have ventricular conduction disturbances (e.g., left axis deviation), which can make the QRS complexes appear ventricular rather than supraventricular.

Bradyarrhythmias are uncommon in cats. One study of bradyarrhythmias in cats found that cats with complete atrioventricular block could develop escape rates as high as 130 bpm.⁴¹ Thus any cat with a heart rate less

than 130 bpm during a physical examination warrants an ECG investigation.

Atrial standstill has also been documented in cats. Commonly, severe hyperkalemia results in an electrocardiographic reading that resembles atrial standstill; indeed, this term is used by many to describe the sino-ventricular rhythm that develops with severe hyperkalemia. In these cases the atrial myocardium fails to depolarize sufficiently to produce a p-wave, but impulses continue to arise in the sinus node and are propagated to the atrioventricular node and then into the ventricles. The ventricular depolarization is often abnormal, resulting in a wide and bizarre QRS complex. Correction of the hyperkalemia resolves the atrial dysfunction. True, persistent atrial standstill has also been identified in cats and results in a junctional or ventricular escape rhythm.²⁸ It is thought that these cases represent either an atrial cardiomyopathy or right ventricular cardiomyopathy, with progression from the right ventricle to the right atrium.

Conduction Disturbances

Cats frequently have deviations in the overall direction of ventricular depolarization. These usually deviate to the left, with a mean electrical axis of 0 degree to -90 degree—the so-called *left anterior fascicular block (LAFB) type pattern*. Whether such deviations truly represent a

block of conduction through the left anterior fascicle or some other conduction abnormality is not known. This conduction abnormality has traditionally been equated with left-sided heart disease, usually HCM or thyrotoxic heart disease; however, the author has observed this pattern of depolarization in echocardiographically normal cats. Ferasin and coworkers²¹ reported LAFB-type patterns in 17 of 61 (28%) cats with HCM (these were part of a total of 19 of 106 cats with various forms of cardiomyopathy that demonstrated this conduction abnormality). However, Riesen and coworkers⁶⁸ found a lower prevalence of LAFB-type patterns in a study of cats with symptomatic HCM (9 of 169; 5%).

Right axis deviations are less commonly seen in cats; the author has not recognized incidental right bundle branch block (RBBB) in cats that are echocardiographically normal, as occurs in dogs. Ferasin and coworkers²¹ identified RBBB in only 3% of 106 cats with cardiomyopathies.

Atrioventricular Blocks

Atrioventricular blocks are commonly observed in cats and increase in prevalence with increasing age, with 95% of affected cats in one study older than 10 years of age.⁴¹ Second- and third-degree atrioventricular blocks have been described in cats with and without structural heart disease or secondary heart disease (i.e., as a result of systemic disease such as thyrotoxicosis).⁴⁰ These are usually evident by routine electrocardiographic analysis but can occasionally be intermittent and require ambulatory ECG recording to be detected (Figure 20-22). Although pacemaker implantation is advised for cats with clinically significant atrioventricular block, one retrospective study of cats with third-degree atrioventricular block calculated a median survival of 400 days in the absence of pacemaker implantation.⁴¹ In the study 14 of 21 cats died by the time of publication: six died or were euthanized for unknown reasons, five were euthanized for noncardiac causes, one was euthanized because of aortic thromboembolism, and two were euthanized

because of quality-of-life issues. No cats were known to have died suddenly. Additionally, 6 of 21 cats in that study had no clinical signs at presentation. The author's personal experience supports this finding; many, if not most, cats with third-degree atrioventricular block are diagnosed on routine examination, rather than because of specific clinical signs, and appear to require no specific interventions in most cases. The likely cause for this is that the escape rates in cats are often higher than those in dogs (80 to 140 bpm versus 20 to 60 bpm), and old cats are largely sedentary, so a modest bradycardia has little impact on hemodynamics.

Isorhythmic Dissociation

Isorhythmic dissociation is a form of conduction disturbance that is prevalent in cats. It is frequently observed during anesthesia but is occasionally identified in conscious cats and is not necessarily associated with structural cardiac disease. Isorhythmic dissociation is identified by observing a p-wave that "wanders" into and out of the adjacent QRS complex over several seconds, indicating that the two complexes are unassociated; the ventricular depolarizations are independent of the atrial depolarizations but occur at essentially the same rate (Figure 20-23). If the atrial depolarization occurs early enough after the preceding ventricular depolarization, "sinus capture" can occur with a sinus rhythm resuming for an unspecified length of time (and identified because of a shortened R-R interval). Atropine will generally correct the arrhythmia by accelerating the sinus rate above the junctional escape rate; however, the arrhythmia is considered benign and does not warrant therapy.

Treatment of Arrhythmias

Surprisingly little information exists regarding the treatment of feline arrhythmias (Table 20-3). Acute therapy of ventricular arrhythmias requires administration of intravenous agents, most commonly lidocaine. However,

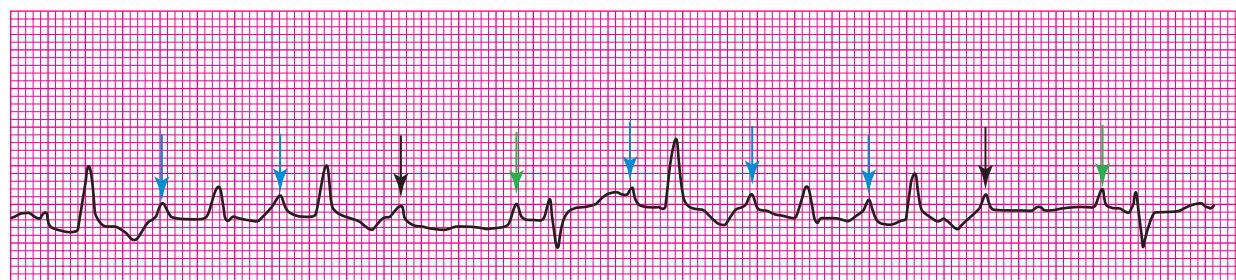


FIGURE 20-22 Electrocardiographic tracing of second-degree atrioventricular block. *Black arrows* indicate unconduct ed p-waves (buried in the preceding T-wave); *green arrows* denote normally conducted p-waves; *blue arrows* denote p-waves conducted with aberrancy (note the longer PR interval—first-degree atrioventricular block—and wide QRS complex).

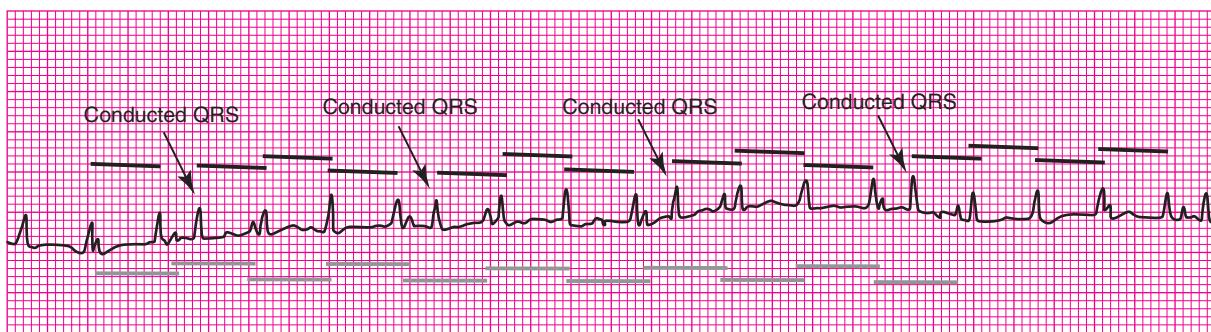


FIGURE 20-23 Electrocardiographic tracing of isorhythmic dissociation. The P-P interval is denoted by the *gray bars* below the electrocardiographic tracing. R-R intervals of nonconducted QRS complexes are denoted by the *black bars* above the electrocardiographic tracing. Sinus complexes are highlighted as “conducted QRS,” with the preceding p-wave being conducted in a normal manner.

TABLE 20-3 Drugs Used in the Treatment of Arrhythmias in the Cat

Drug	Dose	Indications
Atenolol	6.25 to 12.5 mg/cat, PO, once daily	Ventricular tachyarrhythmias
Digoxin	0.005 to 0.008 mg/kg/day, orally, divided twice daily	Atrial tachyarrhythmias
Diltiazem	7.5 mg/cat, orally, every 8 hours	Atrial tachyarrhythmias
Esmolol	Loading dose 200 to 500 µg/kg, slow IV; followed by CRI 25 to 200 µg/kg/minute	Acute management of ventricular arrhythmias
Lidocaine	0.25 to 0.5 mg/kg, slow IV; repeat up to 2 more times if needed	Acute management of ventricular arrhythmias
Propranolol	0.02 mg/kg, slow IV; repeat up to 4 times if needed	Acute management of ventricular arrhythmias
Sotalol	2 mg/kg, orally, every 12 hours	Ventricular tachyarrhythmias

lidocaine has a relatively small therapeutic window in cats and can easily induce neurologic events such as seizures. Acute beta blockade with intravenous propranolol or esmolol can also be attempted, but there is no published literature supporting these strategies.

Atrial tachycardias are often amenable to treatment with diltiazem or digoxin, although the latter drug also has a small therapeutic window in cats and should be administered cautiously.

Beta blockade provides the most common strategy for ventricular tachyarrhythmias. Atenolol has been used extensively in cats and requires twice-daily administration. More recently, sotalol has gained popularity in the management of feline ventricular tachyarrhythmias. However, no studies have demonstrated the natural history of ventricular tachyarrhythmias in cats, so it is impossible to determine if treating these arrhythmias prevents or reduces the risk of sudden cardiac death in these patients.

Feline bradyarrhythmias can, on occasion, require therapy. Pacemaker implantation most commonly resolves clinically apparent atrioventricular block (high-grade second degree or third degree), and can be attempted in cases of persistent atrial standstill.

Isorhythmic dissociation does not require specific therapy because the ventricular rate is sufficient to meet the demands of the patient.

CONGENITAL HEART DISEASES

Very little is known about the prevalence of congenital heart diseases in cats. No studies have examined breed predispositions. In the author’s experience, the most common congenital defect presented for diagnostic evaluation is the VSD. Most congenital defects seen in dogs have also been reported in cats, with a few exceptions. For example, the author knows of no cases of cor triatriatum dexter in cats but has seen several cats with a similar defect affecting the left atrium (cor triatriatum sinister, or supravalvular mitral stenosis), which has not been reported in dogs. Treatment of common congenital defects is similar to that in dogs.

Ventricular Septal Defect

VSDs in cats are similar to those in other species (Figure 20-24). Most are perimembranous (situated just under the aortic valve), and their hemodynamic consequences are determined by the size of the defect. In the author’s experience, many VSDs are incidental findings in cats and cause little hemodynamic perturbation, other than a loud murmur. Most VSDs have a loud right-sided systolic murmur. Larger VSDs generally result in a left-to-right shunt, with pulmonary overcirculation and left-sided heart enlargement. If sufficiently large, left

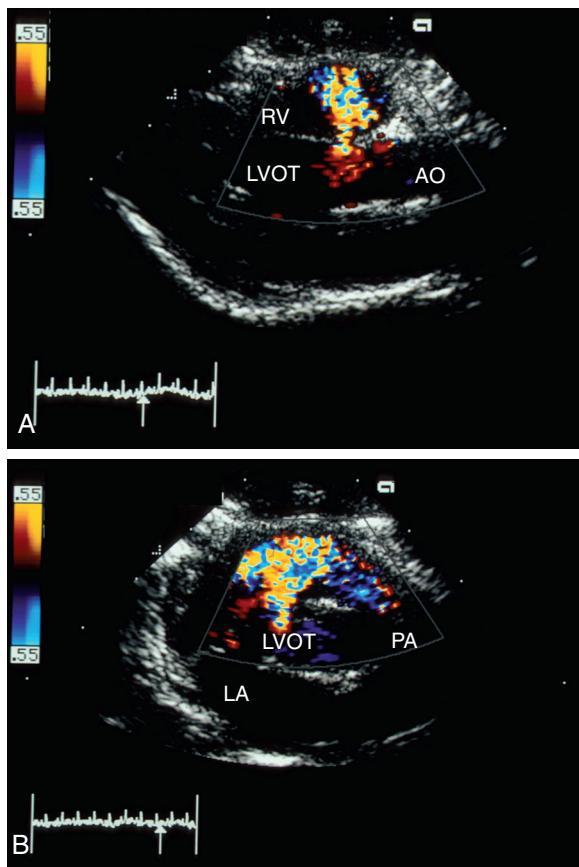


FIGURE 20-24 Echocardiographic images of a cat with a ventricular septal defect. **A**, Right-parasternal long-axis color Doppler image demonstrating left-to-right shunting of blood across the ventricular septal defect. **B**, Right-parasternal short-axis color Doppler image demonstrating left-to-right shunting of blood across the ventricular septal defect. Note that the turbulent jet is directed out into the right ventricular outflow tract in systole.

ventricular and atrial diastolic pressures can increase to the point of CHF (pulmonary edema and pleural effusion). Right-sided changes are uncommon, unless the VSD is extremely large or more ventrally located (in the muscular septum, rather than membranous septum). Because the defect is present at birth and does not generally increase in size as the patient grows, a clinically inconsequential VSD at 3 months of age will not become clinically compromising as the patient matures.

Treatment of VSDs also depends on the size of the shunt. In most instances treatment is reserved for cases with evidence of CHF. Standard diuretic and ACE inhibitor therapy is usually prescribed. Additionally, afterload reduction with arteriolar dilators, such as hydralazine or amlodipine, can decrease the shunt fraction and reduce left ventricular preload. Surgical correction is generally not possible in cats because of the inability to perform open-heart procedures in this species and the lack of suitable endovascular devices that could close the defect by way of a venous approach.

Atrioventricular valvular malformations (so-called “endocardial cushion defects”) are also found in cats, although in the author’s experience, these are less common than VSDs. Signs vary considerably with these defects and depend on the extent of involvement of the valves and the septa. In most cases these defects result in left-sided CHF.

Atrial Septal Defect

Atrial septal defects (ASDs) are relatively uncommonly diagnosed in cats. Septum primum defects appear to be more common than septum secundum defects. Occasionally, ASDs can be seen with supravalvular mitral stenosis. Complete absence of an atrial septum is also described.^{42a} Finally, ASDs can be seen as part of the endocardial cushion defect complex.

Most ASDs do not cause clinical problems. However, with large ASDs, right-sided volume overload occurs (shunting from the left atrium to the right atrium and right ventricle in diastole), resulting in increased end-diastolic right ventricle pressures and right-sided CHF (ascites, pleural effusion). Pulmonary blood flow increases, resulting in pulmonary arterial hypertension.

Closure of clinically relevant ASDs is not currently feasible in most cats. Treatment includes diuretic therapy and abdominocentesis as required.

Supravalvular Mitral Stenosis (Cor Triatriatum Sinister)

Supravalvular mitral stenosis (also identified in some cats as cor triatriatum sinister) is an uncommon defect in which the left atrium has a perforated intraatrial membrane just dorsal to the mitral valve annulus, effectively producing a hemodynamic perturbation similar to mitral valvular stenosis. Flow to the left ventricle is severely restricted. In some cases atrial septal defects can result in left-to-right shunting at the atrial level. An interesting consequence of this defect is the development of severe pulmonary hypertension, rather than left-sided CHF, despite markedly elevated left atrial and pulmonary venous pressures in some cats. Other cats can develop pulmonary edema or pleural effusion.

Surgical correction has been described in one case, where the membrane was torn to allow normal transatrial flow.⁸⁵ The author was involved in an unsuccessful attempt at surgical correction in another case. Medical treatment of the CHF is generally unrewarding because diuretic therapy sufficient to reduce pulmonary venous pressures generally results in underloading of the left ventricle and output failure.

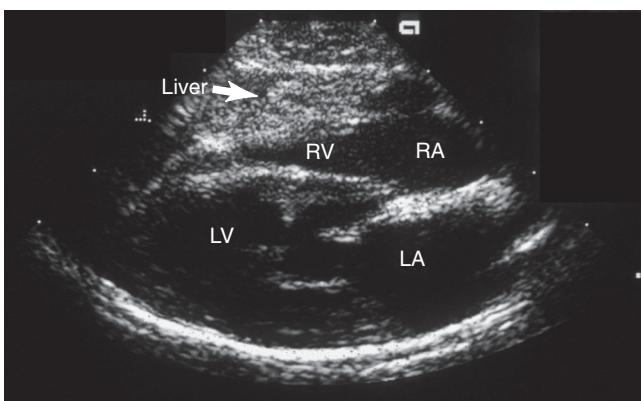


FIGURE 20-25 Echocardiographic right-parasternal long-axis image demonstrating the intraperitoneal presence of liver tissue secondary to a peritoneopericardial diaphragmatic hernia.

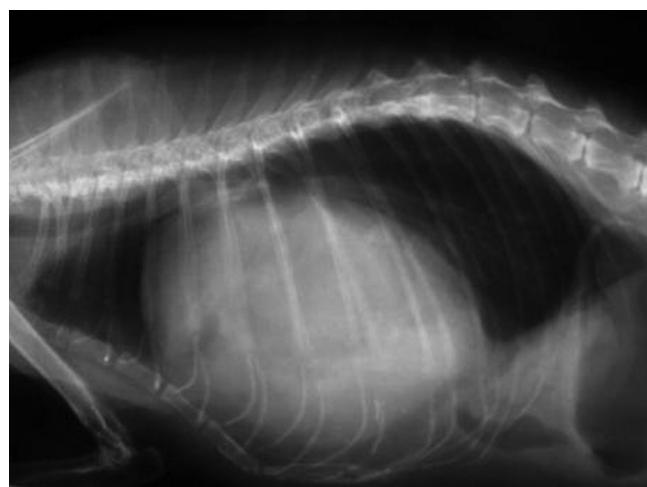


FIGURE 20-26 Lateral radiograph of a cat with a peritoneopericardial diaphragmatic hernia. Note the marked cardiomegaly with heterogeneous opacity of the cardiac silhouette, suggesting presence of fat and soft tissue within the pericardium. Also note the absence of the liver on the abdominal side of the diaphragm.

Peritoneopericardial Diaphragmatic Hernias

Peritoneopericardial diaphragmatic hernias (PPDHs) are congenital defects that are found most commonly as an incidental finding on routine radiography of older patients. Most PPDHs cause no clinical signs. The precordial impulse, or point of maximal intensity of the heart sounds on auscultation, can be displaced (e.g., absent on the left or positioned extremely dorsally in the chest) as a consequence of abdominal viscera displacing the heart within the pericardium. Falciform fat is often found prolapsing through the hernia; however, other organs can also prolapse into the pericardium (Figure 20-25). This can occasionally cause visceral entrapment or strangulation of organs. Some cats develop dyspnea or tachypnea, which is thought to be secondary to PPDH. Radiographic findings usually include a massive cardiac silhouette within which multiple soft tissue structures can be seen (Figures 20-26 and 20-27). Abdominal viscera (e.g., falciform fat, liver silhouette) might be missing from their usual locations. More specific radiographic signs have been described.⁸

Treatment of PPDH generally relies on development of clinical signs that can be attributed to the defect. Elective closure of the defect in cats that have no clinical signs (but where the defect was detected incidentally) is questionable, insofar as the risk associated with this procedure is not minimal. One study showed that postoperative hyperthermia was commonly seen in these cases and perioperative mortality was 14%—not an insignificant complication.⁶⁶ In this author's opinion, surgical correction therefore should be performed if clinical signs warrant intervention or if the patient is undergoing other elective abdominal surgery (e.g., spay) but should not be performed on cats (especially older cats) in which the finding is incidental. One study suggested that only a small percentage of these cases subsequently develop clinical signs requiring intervention,⁶⁶ so the risks



FIGURE 20-27 Dorsoventral radiograph of a cat with a peritoneopericardial diaphragmatic hernia. Note the marked cardiomegaly with heterogeneous opacity of the cardiac silhouette, suggesting presence of fat and soft tissue within the pericardium. Also note the absence of the liver on the abdominal side of the diaphragm.

associated with surgery outweigh the risks of the untreated abnormality in most patients.

Patent Ductus Arteriosus

PDA is less commonly diagnosed in cats than in dogs. However, the hemodynamic consequences of this defect

are identical to those in dogs—namely, volume overload of the pulmonary circulation and left side of the heart. Physical examination findings include a left-sided continuous murmur cranial to the cardiac base, strong pulses and potentially CHF. Treatment of PDA in cats requires surgical thoracotomy and ligation as transvascular occlusion devices are too large to be used in cats. Successful ligation of the PDA will result in resolution of clinical signs and should allow the patient to lead a normal life.

Right-to-left shunting PDA is rarely identified in cats; again, affected cats suffer hemodynamic consequences similar to those of dogs with this defect. Hypoxemia and polycythemia result from blood bypassing the pulmonary circulation. Physical examination findings include a normal auscultation (no murmur) and exercise-induced hindlimb weakness and shortness of breath. Cyanotic mucous membranes can be observed caudally (genital mucosa), but oral mucosa appears normal (differential cyanosis). Treatment can include phlebotomy, although this can be difficult to accomplish in some cats. No other treatment is effective in controlling clinical signs.

MISCELLANEOUS HEART DISEASES

Glucocorticoid-Associated Congestive Heart Failure

In recent years investigators have postulated the possible causal association between the administration of glucocorticoids and subsequent development of CHF in cats. Initial reports found that cats with HCM presented for CHF shortly after either undergoing an anesthetic procedure or being administered glucocorticoids for various unassociated disorders (e.g., asthma, dermatopathies).⁸¹ A subsequent case series reported on 13 cats over a 10-year period that presented to a referral institution with CHF after administration of various glucocorticoids.⁸⁰ Several of these cats succumbed to the CHF, whereas others were reported to have a reversible left ventricular hypertrophy. These authors subsequently suggested that glucocorticoid administration resulted in a hyperosmolar increase in plasma volume (up to 44% in some cats), which could account for the occurrence of CHF. However, they failed to document any changes in cardiac chamber dimensions that would have been anticipated with such increases in plasma volume. Other authors subsequently administered glucocorticoids to healthy cats and performed serial echocardiograms but also failed to document any change in cardiac dimensions that acute volume overload might be expected to produce.⁷⁰

The mechanism by which glucocorticoids could produce CHF remains unresolved. One group of

investigators documented an increase in plasma volume secondary to mild hyperglycemia.⁶⁵ However, this hypothesis is unconvincing. First, glucose is a weak osmole, with an osmotic effect approximately 5% that of sodium or potassium. The plasma glucose concentration that was documented in these cats was less than the renal threshold (180 mg/dL). Thus the osmotic effect would be minimal and would not be able to increase plasma volume by 44%. Second, the hypothesis does not explain the observation of reversible left ventricular hypertrophy. Furthermore, although one study claimed an association between CHF and diabetes mellitus in cats,⁴⁷ this paper has substantial flaws that preclude this author from reaching similar conclusions. Given that CHF is not a common feature of diabetic cats or cats with DKA and up to 20% of diabetic cats would be expected to have subclinical HCM, it is difficult to accept a hypothesis that invokes hyperglycemia as the cause. A mineralocorticoid effect can be excluded because most cats reported with CHF secondary to glucocorticoid administration were injected with methylprednisolone acetate (Depo-Medrol, Pfizer), which has virtually no mineralocorticoid effect.

In all the reports, there is a confounding factor that has not been considered: the hospital visit that accompanies each glucocorticoid prescription. It is possible that the stress of the hospital visit in a cat with subclinical but critical HCM could precipitate an onset of CHF shortly after the visit. The author has observed exactly this situation multiple times. Thus the possibility exists that, in some cats with severe subclinical heart disease, glucocorticoids, through some as yet undefined mechanism, can induce the onset of CHF. However, the evidence is unconvincing and could be an effect of selection bias. Additionally, the incidence appears to be rare (13 cats over 10 years at a single referral institution were identified), so clinicians should continue to use glucocorticoids when indicated in cats. Minimizing the use of glucocorticoids has a clinical rationale (e.g., decreasing the risk of diabetes mellitus) other than avoidance of CHF, so clinicians should prescribe these drugs judiciously in cats regardless of their cardiac status. It might be prudent to avoid glucocorticoids, if possible, in cats with preexisting severe heart disease (e.g., cats with prior episodes of CHF).

Hyperthyroidism and Heart Disease

Hyperthyroidism is the most commonly diagnosed endocrine disease in cats. The increased metabolic demands produced by hyperthyroidism result in cardiac hypertrophy. Additionally, direct stimulation of cardiomyocytes by thyroid hormone results in expression of myocardial genes encoding structural and contractile proteins, again resulting in hypertrophy. Consequently, the heart of a patient with hyperthyroidism has increased

wall thickness, left ventricular chamber volume, and contractility. These changes, when coupled with the tachycardia that is induced by hyperthyroidism, increase cardiac output two to three times above baseline. Thus the heart of a cat with hyperthyroidism is working at a near-maximal capacity, even at rest. "High output failure" can result from prolonged hyperthyroidism because left ventricular diastolic pressures rise because of an increase in preload and blood volume and produce pulmonary edema or pleural effusion.

Reports exist of hyperthyroidism causing HCM in cats.^{48,56} However, there is no rational basis for cats with hyperthyroidism to develop HCM. Instead, these cases likely represent independent comorbidities. Often, the increased preload induced by the hyperthyroid state results in CHF secondary to the previously subclinical HCM. Thus true concentric hypertrophy in a hyperthyroid cat should be considered evidence of HCM independent of the thyroid disease, and regression of the myocardial hypertrophy would not be anticipated in these cases once the thyroid disease is controlled. However, any CHF might resolve after the euthyroid state is achieved because of the reduction in preload and cardiac output.

A single report exists of myocardial failure in cats with hyperthyroidism.³⁶ This presentation is rarely encountered today because the disorder is recognized much more quickly than it was 30 years ago. It is likely that the myocardial failure observed in those cases was a late-stage finding; however, some of the cats in that report might also have had taurine-deficient cardiomyopathy as an independent comorbidity (because the association of taurine and myocardial failure had not yet been made).

Heart rate control in hyperthyroidism is often advocated and is warranted if the rate is exceedingly high or if CHF exists.⁴² In these cases beta blockade is recommended, often resulting in improved clinical signs and hemodynamic stabilization. This is important if the patient is to be admitted to a facility for I-131 therapy, which requires several days of hospitalization because the stress can increase the likelihood of CHF developing. Clinicians should note that propranolol has a *longer* duration of action in hyperthyroid cats than in healthy cats and should adjust doses accordingly.³⁷ Whether similar changes in pharmacokinetics exist for atenolol is not known.

Endocarditis

Endocarditis is a rare condition in cats. Infection of cardiac valves has been reported in several case reports or case series, with the left-sided cardiac valves (aortic, mitral) involved more often than right-sided valves. Infectious agents that have most commonly been associated with feline endocarditis include *Bartonella*

species (*B. henselae*) and gram-positive organisms (*Streptococcus*).^{14a,52a,61a} Clinical signs include malaise, fever, and a new murmur with or without CHF (which depends on the degree of valvular damage). Diagnosis requires culture or amplification of the organism along with echocardiographic evidence of valvular proliferative vegetative or erosive lesions, especially of the aortic or mitral valve. Treatment generally requires aggressive intravenous antibiotic therapy. Prognosis is guarded, although one case report suggested that early and aggressive intervention in endocarditis caused by *B. henselae* infections can have a complete resolution.^{61a} However, most other reports were associated with either acute mortality or persistent clinical signs (e.g., CHF) and requirement for ongoing antibiotic therapy.

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