

FELINE CARDIOVASCULAR DISEASE OVERVIEW OF DIAGNOSIS, TREATMENT, AND MANAGEMENT

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Cardiovascular disease is unfortunately a very common affliction of cats. Congenital malformations and valvular heart disease are relatively uncommon, while myocardial diseases and vascular dysfunction are frequently seen in practice. This review will focus on the spectrum of cardiovascular disease in this species, highlighting pertinent physical findings, diagnostic tests, and therapeutic strategies.

The Spectrum of Feline Cardiovascular Disease

The most common presenting complaint for cats in referral cardiology practice is evaluation of a heart murmur, heard during routine auscultation at a wellness appointment with the general veterinarian. Unfortunately, in contrast to dogs, auscultation alone is seldom a confirmatory test as to the cause of the murmur. Indeed, functional murmurs (a murmur in the absence of structural heart disease) and pathologic murmurs occur with near equal frequency in cats and sound very similar by auscultation. As such, further screening tests are required to evaluate a feline patient with a heart murmur as will be described below. For those cats with structural heart disease, abnormalities of the ventricular myocardium predominate. Hypertrophic cardiomyopathy (HCM) is the most common feline heart disease, while restrictive cardiomyopathy (RCM), dilated cardiomyopathy (DCM), arrhythmogenic right ventricular cardiomyopathy (ARVC), unclassified cardiomyopathy (UCM), and myocarditis are relatively less common. Primary valvular disease is quite rare in cats, though age-related valve degeneration and infective endocarditis are occasionally diagnosed. Congenital malformations are seen in kittens and sporadically diagnosed in adulthood, with atrioventricular valve malformations and septal defects being the most frequently recognized. Other systemic diseases may impact the feline heart and result in pathologic change such as hyperthyroidism, systemic hypertension, severe respiratory disease (cor pulmonale), anemia, diabetes mellitus, growth hormone excess, heartworm infection, neoplastic disease, systemic inflammatory/infectious diseases, and even iatrogenic fluid administration. Lastly, the feline vasculature is a frequent source of morbidity in the older feline with systemic hypertension and arterial thromboembolism (ATE) seen in practice. Each of these conditions will be commented on, though the therapeutic algorithm for many of these diseases will be similar and therefore treatment will be considered more broadly to address clinical problems arising from these underlying conditions – namely, congestive heart failure (CHF), ATE, and dysrhythmias.

Cardiovascular Examination of the Feline Patient

The evaluation of the cat with suspected heart disease should begin with a thorough history and a general physical examination. As a species, cats tend to hide outward signs of disease until late in their disease course and it is therefore not uncommon to diagnose severe cardiovascular disease in a cat with no apparent clinical symptoms. Additionally, as pet cats are frequently indoor sedentary creatures in today's society, subtle

signs of exercise intolerance or fatigue, as can be seen with heart disease in other species, are unlikely to be noticed. That being said, aspects of the history can be pertinent and direct diagnostic investigation. Symptoms of heart disease in the cat are typically respiratory in nature. In contrast to dogs, cats seldom cough with heart disease; the most frequent underlying disorders causing cough in cats are feline asthma/bronchitis and feline heartworm infection. The more common respiratory symptom in the cat with heart disease is an increase in respiratory rate and/or effort. The perceptive client may notice a subtle increase in their cat's breathing rate/effort before fulminant CHF, but many times cats present acutely with severe, open-mouth respiratory distress. Likewise, ATE presents as an acute onset of limb paralysis with severe pain and discomfort and less commonly as nonspecific symptoms of organ infarction (brain, bowel, kidney, etc). The history of cats with heart disease may include symptoms of collapse, sudden weakness, or syncope related to abnormalities of heart rhythm. It is important to note that syncopal cats are frequently mistaken as having a seizure disorder because facial twitching and tonic-clonic motions are not uncommon symptoms in the syncopal cat. The history of the cat with heart disease also may include a recent stressor that precipitated the onset of respiratory symptoms, such as recent travel, introduction of a new pet, or a visit to the veterinarian. Last, the history may provide clues as to the presence of other systemic disease that may impact the cardiovascular system such as polyuria/polydypsia in the cat with diabetes or renal dysfunction, symptoms of a hypermetabolic state (eg, muscle wasting, polyphagia, nocturnal vocalization) in the hyperthyroid cat, symptoms of end-organ damage (eg, sudden onset blindness or seizure activity) in the cat with systemic hypertension, etc.

Physical examination of the cat with suspected cardiovascular disease should focus on documentation of vital parameters, careful auscultation of all heart and lung fields, evaluation of the jugular veins for distension and/or pulsation, palpation of the femoral pulse, inspection of the mucous membranes and capillary refill time, palpation of the ventral neck for a palpable goiter, and optimally a ophthalmic/fundic examination. Vital parameters are important as the cat with CHF frequently is bradycardic and hypothermic, symptoms of cardiogenic shock requiring inotropic support. Auscultation may reveal a heart murmur, gallop sound, or abnormality in cardiac rhythm. The lungs may reveal evidence of cardiac decompensation, with crackles apparent in the setting of fluid accumulation in the alveolar space and dull ventral lung fields present in the cat with pleural effusion. Additionally, wheezes or rhonchi may be a sign of primary pulmonary disease (feline asthma) that may cause secondary pulmonary hypertension and cardiac dysfunction. The jugular veins are useful as a sign of volume overload, which causes visible jugular venous distension if the fur is shaved or wetted with alcohol, or may indicate other pathologies such as dysrhythmias, tricuspid regurgitation, or a poorly compliant ventricle if pulsations are observed. Femoral pulses are important to evaluate as their absence may indicate ATE or hypotension. Very pale membranes may suggest anemia or peripheral vascular constriction, while cyanotic membranes are seen in the setting of some congenital heart diseases with right-to-left shunting of blood. The fundic exam is useful as the retina is often the earliest, and most easily visualized, target organ of systemic hypertension. Additionally, the rare cat with DCM may have retinal lesions consistent with taurine deficiency (hyperreflective elliptical lesions adjacent to the optic disk).

Heart murmurs in cats are almost exclusively systolic; the rare kitten with a patent ductus arteriosus may have a continuous murmur and older cats with aortic dilation and insufficiency will occasionally have a soft diastolic murmur. Localization of the cardiac valves by auscultation and thoracic landmarks is challenging in the cat; rather, murmurs are typically described as parasternal either on the left or right and cranial or caudal. An imperfect approximation is that mitral regurgitant murmurs are often at the left caudal sternal border, dynamic

outflow obstruction is typically cranial, a ventricular septal defect is louder on the right sternal border, and functional murmurs are often localized to the cranial left or right chest. These guidelines are, unfortunately, far from exact. A useful rule of thumb is that functional murmurs are more likely to be soft at rest and increase with excitement. The author will often listen to a cat at rest and then gently lift the cat off the table 3-4 times to stimulate a sympathetic response. This technique is useful to unmask a very soft murmur, but as pathologic murmurs may also increase with excitement it is not able to definitively distinguish functional from pathologic causes.

Gallop rhythms are heard in the setting of myocardial disease in many cats. A thick and poorly compliant ventricle will require a greater proportion of filling to occur late in diastole, thereby increasing the atrial contribution and resulting in an S4 gallop. The cat with a volume-overloaded ventricle may have rapid filling early in diastole due to high left atrial pressure and display an S3 gallop to auscultation. Given the high heart rate of the cat, it is nearly impossible to distinguish S3 from S4 in most patients. It is worth noting that the older cat (>10 years of age) may occasionally have a gallop rhythm related to an aging and stiff ventricle without significant cardiovascular disease. However, a gallop sound in any cat should prompt further diagnostic testing for underlying heart disease.

Diagnostic Testing in the Cat with Suspected Cardiovascular Disease

There are many more diagnostic tests available to today's feline practitioner than at any time in the history of the profession. While beneficial if selected appropriately, this abundance of options sometimes leaves one at a loss to decide which will prove of most benefit to a particular patient. To start, since many feline heart murmurs have functional causes, preliminary testing of the cat with a heart murmur should include measurement of core body temperature, arterial blood pressure, assessment of red blood cell levels (hematocrit or PCV), and a thyroid level for those cats over 6 years of age. Fever, anemia, hypertension, and hyperthyroidism can all contribute to murmur genesis in cats and should be evaluated in a primary care setting. In the kitten, few tests will be useful other than echocardiography in confirming the diagnosis of congenital heart disease and referral to a cardiologist or experienced echocardiographer should be considered.

It is pertinent here to include a few comments on blood pressure (BP) measurement in the cat. It is clear that systemic hypertension occurs in the cat and that the consequence of this elevated BP can be devastating (retinal detachment, hyphema, neurologic sequelae, cardiac hypertrophy, progressive renal dysfunction). The most common causes of systemic hypertension in cats include renal disease and hyperthyroidism. Rare causes include aldosterone-secreting tumors (Conn's syndrome) and essential (eg, idiopathic) hypertension. All cats with elevated thyroid hormone or renal disease, regardless of severity, should have their BP evaluated. Additionally, any cat with a heart murmur or gallop rhythm should have their BP measured to rule out hypertensive heart disease. The author prefers Doppler interrogation using a Parks Model 811-B ultrasonic flow detector with the cat lightly restrained in lateral recumbency. An appropriately-sized Velcro cuff (typically #1 or #2 in cats, determined by the cuff width equal to ~30-40% of the limb circumference) is then placed around the antebrachium of the non-recumbent forelimb.¹ The pulse is found by an audible Doppler signal on the ventral aspect of the carpus and the cuff lightly inflated with a sphygmomanometer until the pulse is lost. The pressure in the cuff is then released until the pulse is again audible, which indicates the cat's systolic blood pressure. This measurement should be repeated 3-5 times until consistent values are achieved (varying by <10%); often, the first measurement is rejected as the cat is not yet used to having the cuff inflated

and the BP may be falsely elevated. Oscillometric devices are also available, which are most useful in very compliant or sedated patients. In the author's experience, such devices are more sensitive to patient movement and can provide false readings in the cat that cannot lie perfectly still. The benefit of the oscillometric devices is that they provide diastolic and mean BP readings in addition to the systolic measurement; however, isolated diastolic hypertension has not been described in the cat and the systolic BP is therefore likely sufficient to screen all cats for systemic hypertension.

Traditional cardiovascular testing has included electrocardiography (ECG) and thoracic radiography. In general, both of these tests are relatively insensitive in confirming heart disease, particularly mild disease, and neither allows full characterization of the underlying condition. However, if an arrhythmia is appreciated on the initial examination, an ECG should be pursued as this remains the best test to evaluate the nature of the rhythm disturbance. Other clues from ECG that there is a cardiac abnormality include elevated amplitudes ($P \geq 0.25\text{mV}$, R or $S \geq 0.7\text{mV}$ in the limb leads), duration ($P \geq 0.4\text{sec}$), or axis deviation. Thoracic radiography is the best test to screen for pulmonary abnormalities and/or decompensation from heart disease, but is not specific in confirming/refuting a diagnosis of cardiomyopathy in the asymptomatic cat as appreciable cardiomegaly occurs later in the course of many cardiomyopathies. Signs of cardiomegaly, such as shifting of the cardiac apex toward midline, pulmonary venous distension, pulmonary edema, or pleural effusion, would support a diagnosis of heart disease based on a thoracic radiograph. It should be noted that cats with CHF may show either pulmonary edema or pleural effusion, even with solely left-sided disease. The proposed explanation for pleural effusion in the setting of left-sided CHF in the cat is that some of the venous drainage from the pleural space enters the bronchial veins, which terminate in the left atrium; as such, elevated left atrial pressure can cause cavitory effusion in the pleural space. In the cat with known heart disease and an increase in respiratory symptoms, thoracic radiography remains the best test.

There is recent interest in the use of cardiac biomarkers in small animal practice. B-type natriuretic peptide (BNP) is a hormone that has natriuretic (salt-wasting) and vasodilating properties. It is released by the myocardium in response to stretch, hypertrophy, and hypoxia. The role of the natriuretic peptides is to counteract the rennin-angiotensin-aldosterone system and their release is up-regulated in the setting of heart disease and CHF. Given these properties, BNP has been utilized as both a therapeutic and diagnostic agent in human medicine and recently as a diagnostic test for heart disease, or as a cardiac biomarker, in veterinary medicine. Currently, the commercially available test is a feline-specific NT-proBNP ELISA (Cardiopet proBNP®, IDEXX Laboratories). Measurement of feline NT-proBNP has some attractive properties in discriminating respiratory from cardiac causes of dyspnea and/or in screening susceptible populations for cardiac disease. In determining the underlying cause of dyspnea in cats presented for respiratory distress, two studies from Europe^{2,3} and one from the US⁴ show fairly consistent results. In cats that were dyspneic because of primary respiratory disease, median NT-proBNP values across studies varied from 45 to 170 pmol/L, while cats with CHF as a cause of respiratory disease had median NT-proBNP values of 532 to 754 pmol/L. These studies suggest that a cut-off value of 250 pmol/L should be reasonably discriminatory in defining which cats are in distress due to primary respiratory disease vs. CHF. Since a high proportion of cats with heart murmurs have no structural disease and, given the difficulty in detecting asymptomatic cardiomyopathy in cats, a blood test that could pick up early cardiomyopathy in cats would be of great benefit. In a study⁵ of 78 cats – of which 28 were normal, 17 had heart disease without CHF, and 33 had CHF – the NT-proBNP had a median concentration of 34 fmol/L in the normal cats, 184 fmol/L in the cats with heart disease but no failure, and 525 fmol/L in the cats in CHF. In

contrast to this, a study⁶ looking at a colony of Maine Coon cats affected with hypertrophic cardiomyopathy (HCM) found NT-proBNP was only able to discriminate cats with severe HCM from controls, while it could not discriminate mild to moderate disease from normal. It seems clear from the data that NT-proBNP is significantly elevated in cats with CHF and can therefore help to distinguish cardiac causes of respiratory distress from pulmonary causes. Unfortunately, the test currently available requires that it be sent out to a commercial lab for analysis with results not available for several days. This delay seriously dampens the clinical utility of the test in directing emergent therapy for the animal with respiratory distress. Using the NT-proBNP test in screening cats for cardiac disease is potentially useful, but some caution is warranted. The predictive value of a result depends on the prevalence of disease in the population studied. We have seen false positive results now that the test is widely available and being performed on low-risk patients. The author advises that NT-proBNP testing should be considered in animals suspected of having heart disease to help guide the need for further testing. This could include breeds at risk for cardiac disease (eg, Maine Coon and Ragdoll cats) or cats with a cardiac murmur on routine exam. If the NT-proBNP is elevated in this setting, further support for a cardiac workup can then be given to the owner. If low or normal, further testing is not urgently required. At this time, initiating cardiac medications based solely on an NT-proBNP level is strongly discouraged.

The gold standard for defining the nature and severity of heart disease in the cat is echocardiography with Doppler. Unfortunately, echocardiography is highly operator dependent and in a structure as small as the feline heart, variability in technical skill will produce variable results. The author advises that those general practitioners with an interest in cardiac disease and echocardiography undergo substantial continuing education courses with guided practice in cardiac ultrasound before attempting to screen cats with cardiomyopathy. With that said, some general guidelines can be instructive in classifying and prognosticating for cats with heart disease by echocardiography. As HCM is the most common heart disease of cats, its echocardiographic features are important to note. These include a concentrically hypertrophied left ventricle (LV), eg a thick-walled chamber with a small ventricular lumen. The normal diastolic wall thickness of the feline LV freewall and/or interventricular septum is 3-5mm. As such, a thickness greater than 5.5 or 6mm at end-diastole implies pathologic hypertrophy. If such a finding is documented, it is important to rule out other causes of LV hypertrophy such as systemic hypertension, hyperthyroidism, or (rarely) aortic stenosis. The author prefers to make measurements of the LV wall thickness from a right-parasternal 2-D long-axis image, though the short-axis image in both 2-D and M-mode is also commonly employed. In all imaging planes, it is important to avoid including a portion of the papillary muscles in one's measurement as this may falsely increase the reported value. Again, proper training and CE can help to minimize measurement error. The other measurement of note for all forms of heart disease in cats is the maximal left atrial (LA) diameter. The LA size can be considered a chronic marker of cardiac dysfunction and risk for CHF or ATE. When severely dilated, the patient is a high risk for either decompensation or thromboembolic disease. The author prefers to measure the maximal LA diameter from a right-parasternal long axis imaging plane optimized to be perfectly sagittal to the heart's axis and highlighting all 4 chambers. The measurement of LA diameter is then taken at the end of ventricular systole, 1-2 frames before the mitral valve opens, from the atrial septum to the posterior wall of the LA and parallel to the mitral annulus. In normal cats, this value is between 12 and 16mm. As such, a value greater than 16mm implies LA enlargement. As a general rule of thumb, 17-18mm is considered mild dilation, 20mm moderate dilation, and >22mm severe dilation. Much more can and should be gained by a full echo-Doppler study, but is beyond the scope of this lecture. Findings specific to certain diseases will be discussed below

where pertinent. In general, one should consider recommending a full echocardiographic evaluation to every cat with a heart murmur or suspected of having cardiovascular disease. Although costly, we currently have no other test as reliable or as sensitive for the detection of heart disease in this species.

Specific Feline Cardiovascular Diseases

Hypertrophic Cardiomyopathy (HCM)

As mentioned above, HCM is the most common heart disease seen in the cat.^{7,8} The disease is genetic, with specific mutations identified in the Maine Coon and Ragdoll, both within the gene encoding myosin binding protein C.⁹ Although some genetic mutations have been identified, there are likely dozens if not hundreds more and comprehensive testing for all breeds is not yet available. For breeding Maine Coons and Ragdolls, however, genetic testing does make sense to help remove homozygous positive animals from the breeding population.

For the vast majority of cats, however, the diagnosis of HCM is made based upon echocardiographic findings of LV hypertrophy while excluding other known causes of such hypertrophy (systemic hypertension, hyperthyroidism, aortic stenosis). HCM is a disease with a highly variable prognosis; many animals with the disease live for years without complication, while others may progress rapidly. In general, a worse prognosis is associated with younger age at diagnosis. In the author's experience, those animals with severe hypertrophy, particularly asymmetric hypertrophy of the LV freewall, tend to also have a more rapidly progressive disease. Cats with HCM commonly have no clinical symptoms and may have no physical exam findings referable to heart disease. A murmur, if present, often derives from turbulent flow associated with mid-ventricular obstruction due to cavitory obliteration or from systolic anterior motion (SAM) of the mitral valve. SAM occurs secondary to altered LV geometry and a portion of the valve becomes displaced into the outflow tract, resulting in obstruction to LV outflow as well as causing mitral regurgitation from the valve being pulled open. If diagnosed early in the course of the disease, a cat may live with HCM for years. The three outcomes to warn clients of HCM cats include development of left-sided CHF, signs of ATE, or sudden cardiac death related to ventricular arrhythmias. Treatment should be directed at each of these clinical syndromes and will be discussed below. Treatment of the asymptomatic cat with HCM is more difficult to define as no studies to date have shown a survival benefit with any therapy. That said, the author does prescribe beta-blocking agents (eg, atenolol) for cats with significant obstruction (either mid-ventricular obstruction or SAM) as this therapy alleviates obstruction and has proven benefit in humans with HCM. The starting dose of atenolol is 6.25mg per cat orally every 12 hours; this dose is a function of the 25mg tablet as the smallest tablet size available of atenolol from the human market. A liquid suspension of atenolol can also be compounded often with added flavoring (eg, tuna, chicken, beef) to improve palatability for cats. Common formulations for compounded atenolol are either 2 mg/mL or 10 mg/mL; it is important to verify the concentration provided by whichever pharmacy is chosen when prescribing this medication. The dose for atenolol suspension in the cat is 1-2 mg/kg PO q12h; the author often starts at a lower dose (0.25 to 0.5 mg/kg) to assure the medication is tolerated before increasing to 1-2 mg/kg q12h. It is important to monitor a cat after starting atenolol therapy, which can be done by auscultation and BP measurement. The normal heart rate for a cat in the hospital is 180-220 beats per minute. On beta-blockade, the author rechecks a cat after starting atenolol in 7-14 days and adjusts the dose of atenolol to an in-hospital heart rate of 120-160bpm. If the cat's heart rate is <120bpm or is hypotensive after starting atenolol, the dose is reduced. If the cat's heart rate on recheck is >170bpm, the dose of atenolol is increased slightly until the optimal heart rate is achieved. Some studies in the past of suggested the use of diltiazem for cats with HCM;

however, the efficacy of diltiazem in HCM cats has never been confirmed and the drug is associated with frequent GI complications and is therefore not prescribed by this author unless the cat is in atrial fibrillation. It is common to note reduction or even resolution of a heart murmur in a cat after starting atenolol as the obstruction is minimized or abated. Whether this translates to improved outcomes is unclear. However, atenolol will provide mild anti-arrhythmic properties, reduces myocardial oxygen consumption, and has anti-anginal properties in humans with obstructive HCM. These comparative benefits underlie the author's recommendation to prescribe it to cats with this disease. For asymptomatic cats with HCM and no obstruction, or for cats that prove highly resistant to medication, the author typically does not treat pre-clinical disease.

A unique form of HCM is occasionally seen in older cats, termed end-stage or "burned-out" HCM.¹⁰ This disease appears as a dilated LV with portions of wall thickening and sections of thin, poorly contracting myocardium. LV systolic function appears depressed and the left atrium is frequently dilated. There are longitudinal studies of HCM in man and cats, which support the theory that this morphology is reflective of chronic HCM with areas of infarction and fibrosis. What was once a thick and hypercontractile ventricle becomes thin and poorly contracting over years of dealing with variable amounts of ischemia and infarction. Treatment is directed to clinical syndromes of CHF or ATE as described below; in addition, the use of a positive inotrope such as pimobendan may be considered. However, the use of pimobendan in the cat is extra-label as this drug is approved solely for dogs; the author has used pimobendan in cats at 0.25 to 0.3 mg/kg q12h, typically 1.25mg per cat q12h, without any apparent adverse effects.

Restrictive Cardiomyopathy (RCM)

RCM is seen much less commonly than HCM, but remains the second most commonly diagnosed myocardial disease in cats. Describing this disease is a bit problematic as the term "restrictive" is more a descriptor of the physiology than of the underlying etiology. That said, RCM reflects a heart afflicted with variable degrees of myocardial and/or endocardial fibrosis.¹¹ In both cases, echo-Doppler studies show a poorly compliant ventricle with elevated filling pressure, normal to slightly thickened wall thickness, normal to mildly depressed systolic function, moderate to severe atrial enlargement, and often profound endocardial scarring and fibrosis, which may be so severe and thick that it bridges the LV cavity. The typical presentation for a cat with RCM is CHF or ATE, the disease is rarely diagnosed in a pre-clinical phase as there is seldom an indication the animal is affected prior to clinical decompensation. Auscultation may reveal a heart murmur, but a gallop sound is more commonly observed. Thoracic radiography and/or ECG may show signs of atrial enlargement and echocardiography reveals the findings described above. Treatment is supportive and directed at management of CHF or prevention of ATE (see below). If diagnosed prior to clinical decompensation, ACE-inhibition may be considered if moderate to severe atrial enlargement is apparent.

Dilated Cardiomyopathy (DCM)

Thankfully, DCM is now a rare disease of cats. After studies in the late 1980s showed that feline DCM was associated with a deficiency of taurine in the diet,¹² the frequency of this disease has fallen dramatically. Rare cases of DCM are still seen in cats, characterized by chamber dilation with thin, poorly contracting LV walls. Physical findings include a soft systolic murmur in most cases, often associated with a gallop rhythm. Symptoms of CHF or ATE are common initial presenting complaints. Treatment includes cardioprotection (eg, ACE-inhibition) for the asymptomatic cat, while those in CHF or with ATE are treated as discussed below. As for RCM, pimobendan should be considered for cats with DCM although this represents off-label use of this medication.

Until plasma taurine levels are known (heparinized blood should be sent to the Amino Acid Laboratory at UC Davis for analysis), taurine supplementation is advised at 250 mg per cat q12h. If taurine deficiency is confirmed, the prognosis for return to normal cardiac function is quite good so long as heart failure can be stabilized. Unfortunately, most cases of DCM seen today in cats are not related to taurine deficiency unless the cat is on a non-commercial diet.

Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

ARVC is a disease seen commonly in dogs of Boxer origin, but also is seen rarely in cats.¹³ The disease is associated with replacement fibrosis and fat infiltration in the right ventricular myocardium. This process, likely related to genetic abnormalities in the structural proteins between cardiac myocytes, results in progressive right ventricular dilation as well as pockets of arrhythmic substrate. As such, presenting complaints typically reflect signs of right heart failure and cavitory effusion, pleural effusion being more common than peritoneal in the cat. Ventricular arrhythmias are also commonly seen. Treatment is directed at therapy for CHF with paracentesis as needed, as well as anti-arrhythmics if life-threatening arrhythmias are observed. Prognosis is variable, but refractory CHF that becomes difficult to control is the most common cause of death or euthanasia in the author's experience.

Unclassified Cardiomyopathy (UCM)

Occasionally, cases of CHF or ATE are seen in cats for which echocardiographic findings reveal a fairly normal-appearing LV. Severe LA dilation may be seen, but the LV shows no apparent hypertrophy, is not overtly dilated, and does not have features consistent with endomyocardial fibrosis. In such cases, the etiopathogenesis of cardiac dysfunction is not known and the diagnosis is given as UCM. While it is somewhat annoying to not have a more complete descriptor, the treatment for these cats will be to address the clinical syndromes of CHF or ATE as for the other diseases. A cardiac troponin I (cTnI) should be considered in these cases to screen for myocarditis with normal cats having values less than 0.1 ng/mL and cats with fulminant myocarditis often having values exceeding 10 ng/mL. cTnI can be measured by sending feline serum to any human reference laboratory as the human test cross-reacts with the feline protein.

Myocarditis

Myocarditis is not well-characterized in the cat. However, occasional cases are seen with chamber dilation, normal to slightly reduced function, ventricular arrhythmias, and CHF or ATE. The author has noted some of these cases have severely elevated levels of circulating cTnI, which implies active myocardial inflammation and necrosis. While the cause is typically unclear, infectious agents are considered as well as auto-immune inflammatory processes, these cats may do well with therapy directed at cardioprotection and supportive care for CHF. Serial cTnI monitoring is useful as normalization of troponin levels seems to be associated with resolving CHF and improvement in cardiac size and function. However, depending on the degree of myocardial damage, persistent dysfunction may occur.

Secondary Cardiomyopathies

As mentioned in the beginning, several other systemic diseases may impair cardiac function in cats. Hyperthyroidism is common in cats over 10 years of age and excessive thyroid hormone causes cardiac hypertrophy, increased plasma volume, increased metabolic demand, increased cardiac output, and dysrhythmias. Long-term thyrotoxicosis can result in CHF or ATE and control is directed at normalization of

thyroid status with methimazole, surgical thyroidectomy, radioactive iodine therapy, or new therapeutic iodine-restricted diets (eg, Hills y/d). Systemic hypertension, as alluded to above, imparts an increased afterload to the left ventricle that results in concentric hypertrophy and can lead to diastolic dysfunction, dysrhythmias, or CHF. Diabetes mellitus in cats also appears to be associated with a 10-fold greater risk of cardiac death compared to the normal population, though the exact pathogenesis involved in this finding is not clear.¹⁴ Occasionally, unregulated diabetic cats may have an excess of growth hormone known as acromegaly. There is evidence that acromegaly does cause cardiac hypertrophy and may result in secondary cardiac disease in this species. Cats with severe anemia may develop high-output heart failure as an increased demand for cardiac output leads to cardiac dilation and, rarely, fluid accumulation. Right-sided heart failure can also develop in cats secondary to severe pulmonary disease. Loss of functional capillary beds in the lungs can lead to increased pulmonary vascular resistance, which may result in pulmonary arterial hypertension. If resistance to blood flow in the lungs becomes severe, it may cause right ventricular failure and cavitory effusions. Last, the cat appears uniquely sensitive to overloading of the vascular space from sodium or fluid administration. The two scenarios in which volume overload cardiomyopathy is appreciated clinically in cats include glucocorticoid administration and iatrogenic fluid delivery. Steroid administration, particularly depo-formulations, appears to be an increased risk factor for CHF in the cat. Studies suggest this is related to hyperglycemia and extracellular fluid shifts from steroids,¹⁵ but remains incompletely resolved. It is not uncommon for cats hospitalized for intravenous fluid therapy, often at clinically appropriate rates of 2-3x maintenance, to develop pulmonary edema or pleural effusion. Many of these cats have subclinical cardiomyopathy, but it may occur in cats with a structurally normal heart. Mild diuresis (eg, furosemide at 1-2 mg/kg per day) is typically sufficient to manage fluid accumulation and can often be tapered and discontinued once the intravenous fluid therapy is done.

Congenital Heart Disease

Congenital heart disease is not common in cats, estimated to be 5% of feline cases presented to a University referral cardiology practice.¹⁶ The most common congenital defects seen in cats include malformations of the mitral (MVD) and/or tricuspid valve (TVD) and ventricular septal defect (VSD).^{16,17} While MVD, TVD, and VSD are most common, nearly all other congenital cardiac malformations have been described in the cat and remain on the differential list for young cats with a heart murmur. Few congenital cardiac malformations in the cat can be definitively treated, though medical management can palliate disease for many cats. Atrioventricular valve malformations typically result in regurgitation, though occasionally stenosis is seen. Severe mitral regurgitation in MVD may lead to left-sided CHF and therapy will be discussed as for other causes of CHF below. Severe TVD will likewise cause tricuspid regurgitation and may lead to right-sided CHF. VSD results in left-to-right shunting of blood, which if moderate or severe in size, may lead to excessive return of blood to the left heart and left-sided CHF. Small defects are well-tolerated and may even close in time in a small percentage of cats. In cats with large defects, the excessive shunting of blood through the lungs leads to an increase in pulmonary vascular resistance, which may cause pulmonary hypertension and reversal of shunt flow. If this occurs, defined as Eisenmenger's physiology, deoxygenated blood may be shunted to the systemic circulation resulting in cyanosis and subsequent polycythemia. A rare complication of a VSD is the development of double-chambered right ventricle. In this situation, the turbulent jet stimulates muscular growth in the mid-right ventricle that may become so severe as to partition the right ventricle into a high-pressure proximal chamber and a low-pressure distal chamber. Beta-blockade is occasionally given in this situation to reduce the pressure gradient across the right ventricle and lessen progressive hypertrophy, but no treatment strategy has

been shown to definitively halt progression of the disease. One congenital defect that can be definitively cured in cats is a patent ductus arteriosus (PDA). The classic PDA murmur is continuous (peaking in systole and maintain a similar quality throughout diastole) and machinery in nature. However, it is not uncommon for cats to have only a systolic component to a PDA murmur, presumably because their pulmonary vasculature is more reactive and the diastolic pressure difference may not be great enough to cause a murmur. Any young kitten with a loud murmur (greater than grade IV/VI) should be evaluated with echocardiography to determine the cause of the murmur as the prognosis may span from grave to excellent and therapeutic options may exist.

Treatment Strategies for Feline Cardiovascular Disease

Therapy for Acute Congestive Heart Failure

CHF is the clinical syndrome most commonly requiring therapy in cats with heart disease. Acute therapy can be remembered with the mnemonic **F-O-N-S**. This stands for furosemide-oxygen-nitroglycerin-sedation. It is important to remember that stress kills the dyspneic cat. As such, handling and diagnostics should be minimized. If there is a suspicion of heart disease in the dyspneic cat (murmur, dull ventral lung fields, pulmonary crackles, etc), the cat should be given 1-2 mg/kg of furosemide IM or SQ and placed in an oxygen cage. Sedation is critical to minimizing stress in these cats and the author prefers butorphanol at 0.3 mg/kg given IM or SQ. Sedation should even precede assessment of vital parameters (eg, rectal temperature) to minimize stress. If dull lung fields are ausculted in the ventral thorax, thoracocentesis should be attempted. The side of dull lung sounds should be tapped, but it is important to remember that cats with heart disease may have severe left atrial enlargement, which could be adjacent to the left chest wall. As such, brief ultrasonographic assessment of the chest can be useful to confirm a safe location for thoracocentesis. Although there is limited evidence in veterinary species, topical nitroglycerin can increase venous capacitance and reduce preload; the author typically applies 1/8" to 1/4" of nitroglycerin paste to the cat's inner ear pinnae at the time CHF is suspected. The initial dose of furosemide may be repeated in 20-30 minutes if the cat's respiratory rate has not reduced with the initial stabilization measures. For those cats that are agonal or so severely dyspneic that respiratory arrest appears imminent, the only option may be rapid sequence intubation. The author prefers IM sedation as noted above, placement of an intravenous line as expeditiously as possible, and administration of low doses of propofol (1-2 mg/kg) to effect to allow intubation. It should be noted, however, that intubation is a last resort as mechanical ventilation will likely be required to maintain the animal's respiratory status. Blood pressure should be assessed and intravenous access obtained as soon as the cat appears stable enough to perform these measures. If hypotension is severe, cardiogenic shock is likely and inotropic support should be considered. The author prefers intravenous dobutamine infusion at 5 mcg/kg/min, escalating by 2-3 mcg/kg/min every 5-10 minutes until a systolic BP of >90mmHg is obtained. Once the cat's respiratory rate has decreased and/or he appears more stable, radiographs may be attempted to evaluate the pulmonary parenchyma, pleural space, and cardiac silhouette. Again, radiographs should not be taken at the expense of the patient's stress level; cats with cardiac failure are tenuous patients and avoidance of loud noises, rapid motions, and excessive handling are important to prevent worsening respiratory status.

Once the radiographs have been performed and initial stabilization achieved, full bloodwork should be obtained to evaluate overall organ function and a schedule for in-hospital therapy should be derived. Depending on the severity of the CHF, pulse IV therapy at 2 mg/kg q12h may be pursued for mild CHF up to a furosemide infusion for severe CHF. In general, the author prefers furosemide infusions be given starting at 0.5

mg/kg/hr for the first 1-3 hours, decreasing to 0.25 mg/kg/hr thereafter. Others will use higher infusion rates, but the risk of excessive volume depletion or acute renal dysfunction is greater at higher doses. Typical hospitalization is in an oxygen-rich environment for this first 12-18 hours, transitioning to room air pending the cat's respiratory status. In the author's opinion, respiratory rate while at rest is one of the most useful parameters to monitor in cats with CHF and a trend over time can be very helpful. Most cats present with initial respiratory rates of 50-100 breaths per minute, which often decline to the 30-40 breaths per minute range within the first 12-18 hours of appropriate therapy. Once the respiratory rate is in the range of 30 breaths per minute, most cats can be transitioned to room air and oral medications. Optimally, the cat's renal function and electrolytes should be reassessed after the initial day of diuresis; rarely, cats with borderline renal function may become severely azotemic after aggressive diuretic therapy. Chest films are also often repeated 24hrs after the time of presentation to confirm the pulmonary edema and/or pleural effusion is improving if not resolved. In the author's institution, most cats are discharged after 24hrs of hospitalization, though severe cases may require longer care. A recheck is scheduled in 5-7 days to reassess renal function, electrolytes, and chest films. An echocardiogram, if not performed at the initial visit, should be considered to confirm the nature of the underlying disease, which may have prognostic implications for the client.

Chronic Home Therapy for Congestive Heart Failure

Typical home therapy for CHF in the cat includes furosemide and an ACE-inhibitor (eg, enalapril or benazepril). Typically, the starting home therapy dose of furosemide in the cat is 1-2 mg/kg q12h. Given the tablet size of 12.5mg, this typically equates to ½ tablet orally twice daily. For large cats or those with severe CHF, a starting dose of 9.375mg (¾ tablet) or a full 12.5mg twice daily may be required. The author typically starts enalapril at a low dose in cats first sent home from an episode of CHF as acute worsening of renal function is possible. If renal function was normal before and after the initial 24hrs of diuresis, enalapril at about 0.25 mg/kg is given twice daily for the first week and then increased to 0.5 mg/kg q12h at the 5-7 day recheck. If moderate azotemia developed with diuresis or renal function was borderline at the outset, a starting dose of 0.1 mg/kg is usually pursued and gradually increased pending renal parameters over time. Spironolactone has been shown to be beneficial in chronic CHF therapy in other species, but no studies exist in the cat. Additionally, roughly 15% of cats on spironolactone develop dermatologic symptoms and self-excoriation; as such, the author seldom prescribes spironolactone in this species. If echocardiography shows borderline or depressed systolic function (as with RCM, DCM, and ARVC), pimobendan may be considered. The use of pimobendan is off-label in this species and there are no scientific studies to guide pharmacokinetics or pharmacodynamics in the cat. However, a comparable dose to that given in dogs, 0.25 mg/kg q12h, appears well-tolerated in cats in the author's experience. Lastly, most cats with heart disease significant enough to cause CHF warrant anti-thrombotic therapy to lessen the chance of ATE.

Preventative Therapy for Arterial Thromboembolism

Moderate to severe left atrial enlargement is a risk factor for ATE and most cats in CHF have at least moderate left atrial enlargement. There are few studies proving efficacy of anti-thrombotic medications in cats at risk for ATE, but as the consequences of ATE are so devastating preventative therapy is warranted in most cases. Anti-thrombotic options include platelet inhibitors or anticoagulants. The author uses platelet inhibitors for nearly all cases as the safety profile appears preferable to anticoagulants. Clopidogrel (Plavix®) is well-tolerated by cats, has proven anti-platelet effects in this species, and is dosed at 18.75mg per cat orally once

daily ($\frac{1}{4}$ of a 75mg tablet). Aspirin is typically given at a micro-dose of $\frac{1}{4}$ to an $\frac{1}{8}$ th of an 81mg tablet every 72hrs. Typically, the author advises clients to choose 2 days per week to give the aspirin (eg, Wednesday and Sunday) to improve compliance. For those clients with severe cost concerns, aspirin is given as sole therapy. For those that can afford it, the author typically gives clopidogrel as sole therapy for those with moderate left atrial enlargement and dual-therapy with both aspirin and clopidogrel for cats with severe left atrial enlargement. Side effects of either drug appear to be primarily gastrointestinal, with roughly 10-15% of cats on aspirin developing inappetence, vomiting, diarrhea, melena, or hematochezia. If this develops, the aspirin is discontinued and sole-therapy with clopidogrel continued.

If echocardiography shows an active thrombus in the left atrial appendage and the risk for ATE appears imminent, the author will occasionally consider anticoagulant therapy in addition to anti-platelet therapy. Although data in cats is sparse, low-molecular-weight heparin may be given at 1 mg/kg q12h enoxaparin (Lovenox[®]) or 100 U/kg q12h deltaparin (Fragmin[®]) with an apparently fair safety profile. There are some studies that question the appropriateness of this dosing schedule in the cat based upon Factor Xa inhibitory activity, but the issue remains unresolved. The author has noted resolution of thrombi in the left atrial appendage of cats on this dosing schedule, but this is anecdotal evidence at best. The downside to low-molecular-weight heparin is the high cost of these medications, which are limiting for most clients. Home therapy with unfractionated heparin may be considered, but the dosing effects are more variable than the low-molecular-weight products and therefore there is a greater risk of under-dosing or causing bleeding tendencies with long-term therapy. As such, the author does not recommend home therapy with unfractionated heparin. The other anticoagulant prescribed to cats at risk for ATE is warfarin, a vitamin-K antagonist. At this time, the author does not recommend home warfarin therapy for cats given the narrow therapeutic index of this medication and the high degree of monitoring required.

Treatment of Acute ATE

The most important therapy for cats that present with signs consistent with acute ATE (cold, pulseless, cyanotic limbs with elevated muscle enzymes) is analgesia. The loss of the vascular supply to one or more limbs is unquestionably painful and pure μ -agonist opioids should be given. The author prefers fentanyl as a constant rate infusion (typically 2-5 mcg/kg/hr), given its potent analgesic properties, short half-life, and ease of dose adjustment pending the cat's response. Once the cat's pain is addressed, consideration should be given to whether further treatment is appropriate. Therapy for acute ATE is expensive and most cats are left with severe, life-threatening heart disease if they are able to live through the acute event. Studies suggest roughly $\frac{1}{3}$ of cats presented to a University referral hospital will be euthanized at the time of diagnosis, $\frac{1}{3}$ of cats will succumb to the disease during treatment, and $\frac{1}{3}$ will live through the event.¹⁸ It is important to assess the cat's rectal temperature as there is a correlation between rectal temperature and survival, with more profound hypothermia associated with higher mortality.¹⁸ Additionally, those with a single limb affected have greater survival than those with two or more limbs afflicted. Full bloodwork should be performed to confirm elevated muscle enzyme activities, monitor for evidence of end-organ damage secondary to thromboembolism, and assess for early evidence of reperfusion (hyperkalemia, acidemia). In addition to analgesia, blood-thinning agents should be given to prevent progression of thrombus formation. The author typically gives unfractionated heparin at 250 U/kg q6hrs for the first 24-48hrs. After 24-36hrs, most cats do not appear painful and the analgesia can be gradually tapered. In-hospital monitoring is critical as most cats die from reperfusion injury as blood flow is restored to ischemic muscle; they develop acute hyperkalemia and acidemia. The author typically

monitors cardiac rhythm and ECG changes continuously in these cats to detect early signs of reperfusion (peaked T waves, widening of the QRS, absent P waves). Blood gases are monitored q8-12h to assess rising potassium levels and/or declining blood pH.

Gentle physical therapy of the affected limbs is recommended so long as the cat's pain is controlled. Once the cat has some use of the limb and a pulse is palpated, discharge is considered. For those cats that do not succumb to reperfusion injury, most will regain some limb function. Many cats have persistent neurologic deficits in the distal limb and some develop gangrenous changes to the distal limb related to absent perfusion. In these cases, distal amputation may be the only therapeutic option. The long-term survival of cats that leave the hospital is reported as 223 days, though those that were also in CHF at the time of the ATE event have a median survival of only 77 days.¹⁸

Treatment of Feline Arrhythmias

Feline arrhythmias remain a challenging aspect of care for this species. Ventricular arrhythmias may not require treatment; in general, the author recommends treatment for cats that are symptomatic from their arrhythmia (syncope, weakness) or for those cats whose arrhythmia is frequent with rapid runs of ventricular tachycardia, closely coupled ectopic beats (so called R-on-T phenomenon), or polymorphic ectopy. Few antiarrhythmic medications are studied in cats. In-hospital therapy for life-threatening ventricular arrhythmias is lidocaine at 1-1.5 mg/kg IV. The author's preferred oral ventricular antiarrhythmic medication for cats is sotalol, which is a class III (potassium-blocking) and class II (beta-blocking) antiarrhythmic. Sotalol tablets are too large for cats and so the drug must be compounded to a suspension and is dosed at 1-3 mg/kg q12h. Optimally, a 10-15min ECG should be performed before and 1-2 weeks after starting the drug to assess response. Holter monitoring (24 hour ambulatory ECG) can be performed in cats, but is expensive and often challenging for the client and the patient. A 24hr monitor, however, is the best means to assess the frequency and malignancy of the arrhythmia in the animal's home environment as well as the best means to compare the efficacy of a drug before and after treatment. For lack of a more scientific guideline, the author typically prescribes atenolol for "mild" ventricular arrhythmias (single PVCs, no runs of ventricular tachycardia) and sotalol for apparently life-threatening dysrhythmias.

Supraventricular arrhythmias (SVT) are less common than ventricular arrhythmias in cats. Severe, life-threatening SVT can be treated with IV diltiazem or esmolol infusion. Diltiazem is typically given as a slow IV push of 0.05 to 0.1 mg/kg repeated every 5-10 minutes until the rhythm is controlled or a total dose of 0.5 mg/kg is reached. Esmolol, a rapidly acting beta-blocker with a short half-life, is given at 500 mcg/kg initial dose, followed by 50-100 mcg/kg/min infusion. Given the small size of the feline heart, atrial fibrillation is a relatively uncommon rhythm disturbance. However, cats with severe atrial enlargement will occasionally present with atrial fibrillation and require therapy to normalize and control heart rate. The author prefers diltiazem for heart rate control in atrial fibrillation, with digoxin added for cases not sufficiently controlled with diltiazem. Diltiazem is typically dosed at 1 mg/kg q8hrs, though current tablet sizes make this more difficult and typically ¼ of a 30mg tablet is chosen for most cats; extended release versions are also available, typically given as 15mg every 12hrs. Digoxin has a long half-life in the cat and is typically dosed as ¼ of a 0.125mg tablet q48hrs. A serum digoxin level should be taken at 10-12h post-pill in 5-7 days, targeting a level of 2-2.5 ng/mL. Signs of digoxin toxicity in cats are typically anorexia and vomiting.

Treatment for the Cat with Systemic Hypertension

As noted above, systemic hypertension is common in cats with renal disease or hyperthyroidism. Therapy is directed at reducing systemic vascular resistance and the preferred medication in the cat is amlodipine, which is a calcium channel blocker with vascular selectivity. Amlodipine is typically given at ¼ of a 2.5mg tablet per cat q12-24hrs (roughly 0.1 mg/kg). Higher doses are occasionally required for cats with resistant hypertension (rarely exceeding 0.4 mg/kg per day). Transdermal amlodipine administration has been described in the cats with fair efficacy, though appears to be not as effective as oral administration.¹⁹ The normal BP reported from several studies in cats is 125/90 mmHg by direct measurement; 115 to 140 / 75 mmHg by oscillometric testing; and roughly 140 mmHg systolic by Doppler.²⁰

The initiation of therapy should be reserved for cats with evidence of end-organ damage or those cats for which the risk of injury is likely. In general, this means that treatment should be initiated for hypertensive cats with retinal hemorrhage, LV hypertrophy, and/or signs of neurologic dysfunction. Treatment for the cat with no evidence of organ dysfunction secondary to hypertension is more problematic. Once initiated, anti-hypertensive therapy will almost certainly require life-long medication and, as such, the diagnosis should be confirmed prior to initiating treatment. Cats, perhaps even more than dogs and man, develop false elevations in BP related to stress in the hospital environment – the “white coat effect”. As such, care should be taken to confirm the measured BP is repeatable, optimally in a setting of low stress. The author recommends BP measurement in the room with the client, once the cat is acclimated to her surroundings. Admittedly, this will not eliminate white coat hypertension for all cats, but expending the effort to minimize stress and handling is critical to accurate measurement of BP. Once systemic hypertension is diagnosed, defined as a BP of greater than 160 mmHg, screening tests for diseases that may cause hypertension as well as tests to evaluate for end-organ dysfunction are advised. In general, this should include renal parameters, urinalysis, thyroid hormone panel, and a fundic evaluation. If a disease known to be associated with systemic hypertension is documented, then therapy for systemic hypertension is warranted. If there is no evidence of underlying disease to explain the systemic hypertension and no evidence of target organ injury, the author recommends repeat BP measurement to confirm the diagnosis prior to starting therapy. In general, this means bringing the cat back in 7-10 days and repeating the BP measurement at the very start of the visit, with the client present to minimize stress. If the BP remains elevated, a discussion about antihypertensive therapy is begun. In general, the author strongly advises treatment if the BP is >180 mmHg and will often reevaluate the BP in another 2-3 weeks if it is between 160 mmHg and 180 mmHg. As many cats develop systemic hypertension secondary to renal disease, ACE-inhibition is also prescribed to most cats with systemic hypertension for renal-protective and antihypertensive properties. ACE-inhibitors, by themselves, have only weak antihypertensive properties in cats and should not be used as sole therapy for systemic hypertension. Benazepril is often considered the preferred ACE-inhibitor for cats with renal dysfunction as it is partially metabolized by the liver, rather than solely by the kidneys as is the case with enalapril. The standard dose for benazepril is 0.5 mg/kg orally q12h; however, the author often starts at a lower dose (0.1 to 0.2 mg/kg q12h) in cats with renal dysfunction and escalates to the full dose in 10-14 days if renal function remains stable.

For the cat with an acute hypertensive crisis (eg, BP >200 mmHg with evidence of target organ injury), few rapidly acting antihypertensive agents are available. Sodium nitroprusside is the preferred intravenous antihypertensive in dogs, and can be used as an infusion in cats. However, nitroprusside is a highly potent arterial vasodilator and the author typically avoids its use without invasive arterial monitoring, which is seldom

feasible in cats. Instead, the author typically gives cats oral amlodipine (0.625mg orally, repeated in 3-4hrs pending response) and hospitalizes for 24-48hrs until BP control is achieved. It is important to remember that amlodipine reaches peak effect in dogs at 6hrs post-pill; pharmacokinetic studies have not been performed in the cat. Given this long duration to peak effect, the author advises caution against dosing schedules that call for q2hr dosing of amlodipine until BP control. The initial regulation of BP in an acute hypertensive crisis should strive for a target BP of 170-190mmHg in the first 24hrs, with a gradual reduction to normal BP thereafter. Resetting of the arterial baroreceptors occurs with systemic hypertension, such that cerebral perfusion may be compromised at otherwise normal BP.

Summary

In summary, cardiovascular disease is common in cats. Often, cardiovascular disease is asymptomatic and physical findings (murmur, gallop) may be the only indication of underlying cardiomyopathy or other systemic disease (hyperthyroidism, systemic hypertension). Therapy is targeted at resolving/preventing clinical syndromes of CHF and/or ATE. Prognosis is variable depending on the underlying diagnosis and disease severity.

****Note: All doses are believed accurate, but should be confirmed via a veterinary specific formulary prior to distribution****

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