Feline Myocardial Disease
Cardiomyopathy (CM) is the most common cardiac disease seen in cats. This disease can lead to the development of congestive heart failure, which is the major cause of cardiac mortality.

Echocardiography is the most important diagnostic test for the identification of myocardial disease in cats

Clinical signs
Abnormal heart sounds are the most frequent clinical findings. These include heart murmurs (60% of CM cats), gallop sounds (20%) and muffled heart sounds (5%)

Murmurs in our feline patients are most commonly heard along the mid sternum or left apex. These murmurs are often dynamic and increase intensity as contractility increases (seen with increased sympathetic tone and heart rate).

The most common cause of a heart murmur with CM is systolic anterior motion of the mitral valve (SAM) into the left ventricular outflow tract (LVOT) due to papillary muscle enlargement or mitral regurgitation.

Gallop sounds are diastolic sounds (S3/S4), which become audible due to reduced compliance of the myocardium

Muffled heart sounds could indicate the presence of pleural or pericardial effusion.

Dyspnea/Tachypnea are common clinical signs seen with CM induced CHF. Radiographically recognized as pleural effusion, pulmonary edema or both. Cats with CM that present for simple clinical procedures (e.g. examinations, venipuncture) or stressful events (e.g. car rides, hospitalization) can acutely develop CHF due to catecholamine release causing vasoconstriction and increased cardiac output leading to pressure overload.

Tachycardia occurs in one-third of CM. Cats with heart rates over 200 bpm can worsen CM by affecting diastolic dysfunction and reducing coronary blood flow

Electrocardiograms are a relatively insensitive diagnostic test in cats with myocardial disease even though most cats with CM will show some form of arrhythmia on a 24-hour Holter recording. There is no evidence based medicine to support the statement that cats with CM can be diagnosed with an ECG.

Limb paresis/paralysis caused by an arterial thromboembolism can occur in 5-10% of the patients with CM.

Currently no evidence exists that any drug therapy alters the natural progression of HCM until the patient is in heart failure
Types of Cardiomyopathy in cats

**Hypertrophic cardiomyopathy** (HCM) is the most common form and seen in two-thirds of cats with CM

**Restrictive cardiomyopathy**

**Dilated cardiomyopathy**

**Arrhythmogenic right ventricular cardiomyopathy**

**Unclassified cardiomyopathy including left ventricular noncompaction**

It is very important for the clinician to rule out two diseases that may mimic CM in cats, these diseases are **Systemic Hypertension** and **Hyperthyroidism**

**How should we treat cats that present for CHF induced by cardiomyopathy?**

Acute therapy

Follow the FONS protocol as you would for a dog in CHF

**Furosemide** 2-4mg/kg IM or IV may be repeated in 1-2 hours

**Oxygen therapy**

**Nitroglycerin 2%** cream 1/8”-1/4” of cream applied to inner ear every 4-6 hours

**Sedatives** - Butorphanol 0.08 mg/kg IV or 0.36 mg/kg q 4 hrs subq or acepromazine 0.04-0.1 mg/kg subq

**Pleural effusion** - perform **Pleurocentesis**

Once drugs therapy is instituted the cat should be left to rest in quiet oxygen enriched environment. Respiratory rate and respiratory character should be assessed every 30 minutes. Furosemide should be continued until the respiratory rate and character starts to decrease. Once the cat is stable the furosemide dose and dosage should be curtailed to the lowest possible dosage to keep the patient stable and out of CHF.

Chronic Therapy

In a recent survey of veterinary cardiologists regarding therapy for HCM cats with CHF most treated these patients with **furosemide** and an **ACE inhibitor**.

In cats that present in CHF due to HCM furosemide therapy should almost always be maintained for the patient’s life. This maintenance dose usually ranges from 6.25mg q 24 hrs to 12.5 mg q 8 hrs. Higher dosages can be used if necessary (up to 37.5 mg q 12 hrs) without severe consequences as long as the patient is eating and drinking. Cats on high dose furosemide may become mildly dehydrated and azotemic (prerenal) but as long as a reasonable quality of life is maintained these clinical parameters can be ignored

The furosemide dosage needs to be titrated carefully for each patient. This is best accomplished by having the owner keep a daily log of the resting (sleeping) respiratory rate at home (normal 15-30 breaths per minute with some cats being up to 40 breaths per minute). See link to home monitoring in reference section
**Ace Inhibitors**
Many cardiologists believe that Ace inhibitors improve the quality and quantity of life for cats with HCM. There is no evidence-based medicine to support this claim it is only anecdotal.
Benazepril 0.25-0.5mg/kg q 24 hrs or Enalapril at 1.25-2.5mg q 24 hrs

**Atenolol** *(Specific Beta-1 blocker)*
70% of veterinary cardiologists use Atenolol to treat cats with severe SAM, while 50% used it to treat cats w/ mild SAM. SAM can only be diagnosed by an echocardiogram. Dosage 6.25-12.5 mg /cat q 12 hrs

**Diltiazem**
Recent evidence suggests that using Diltiazem has no effect on survival time in cats with severe HCM and heart failure. It appears that many veterinary cardiologists have abandoned its use

**ASA or Plavix** *(clopidogrel)*
90-95% of cats with HCM never form a clot. There are no studies to support the use of ASA or clopidogrel to prevent ATE. If a clot or smoke is seen in the left atrium on echo one could consider using ASA, clopidogrel or both with the knowledge that we do not know if it helps or hinders.
Dosages ASA 5mg/ cat q 3 days or 81mg/cat q 3 days. Clopidogrel 18.75 mg/cat q 24 hrs

**Prognosis**

The prognosis is determined by clinical presentation and echocardiographic severity of the disease but it is highly variable. Adult cats that show no clinical signs and have mild-to-moderate disease and no-to-mild left atrial enlargement have a good short-term and possibly a good long-term prognosis. Many, however, progress to more severe disease, and some may die suddenly. Cats with severe wall thickening and mild-to-moderate left atrial enlargement but no clinical signs have a guarded prognosis for developing heart failure at some point in the future. They are also at risk of developing thromboembolism and are at risk for sudden death. Cats with severe wall thickening and moderate-to-severe left atrial enlargement but no clinical signs are at risk for developing heart failure or often already have mild-to-moderate heart failure that has gone undetected. These cats are at risk for developing thromboembolic disease and are at risk for sudden death. Cats presented in heart failure usually have a poor prognosis. In one study they had a median survival time of 3 months although in another it was 563 days (range 2 to 4,418 days). In the first study most died of intractable heart failure; some developed thromboembolism, and some died suddenly. However, some cats (about 20% in this study) in this class stabilize and do well for prolonged periods for unknown reasons. Cats in the first aforementioned study that had severe HCM and aortic thromboembolism had a poor prognosis, with a median survival time of 2 months while in the second study it was 184 days (range 2 to 2,278 days). In the second study, cats that presented with syncope had a median survival time of 654 days
(range 28 to 1,505 days). In this study (260 cases), 56 cats died of systemic thromboembolism, 49 of heart failure, and 13 suffered sudden death. This study also included 87 cats with no clinical signs at presentation. Their median survival time was 1,129 days (range 2 to 3,778 days).

**Nt-proBNP** concentrations correlate with left atrial size and pressure and may have utility in assessment and monitoring of cardiac disease. Nt-proBNP concentration may prove to be clinically useful as a screening test especially when an “in hospital test” is developed. It is important to evaluate for systemic hypertension and renal disease since both can induce elevated concentrations of BNP.

Feel free to contact me with any questions.

**Suggested Readings**

1. ACVIM/CVMA 2009 Proceedings ACVIM Cardiology Section Mark D. Kittleson, DVM, PhD, DACVIM pg 111-123
2. Luca Ferasin, Myocardial Disease Classification, Pathophysiology and clinical presentation, Journal of Feline Medicine and Surgery (2009); 11,3-13
3. Luca Ferasin, feline Myocardial Disease, Diagnosis, prognosis and clinical management, Journal of Feline Medicine and Surgery (2009); 11,183-194
4. Link to Resting Respiratory Rate

Doug Casey, DVM, DABVP Canine/Feline See my BCVMA Locum add
778-866-9465
dougcasey@shaw.ca