This session will focus on the three forms of feline cardiomyopathies: hypertrophic, dilated, and restrictive. We will also discuss thromboembolic disease.

Myocardial disease, particularly hypertrophic cardiomyopathy, is the most common heart disease in the adult cat. Remember that valvular disease in the cat is very rare. Cats do not develop endocardiosis, and endocarditis is very rare, so always consider myocardial disease first when considering differentials for feline heart disease.

There are several forms of feline cardiomyopathy: hypertrophic (HCM), restrictive (RCM), dilated (DCM), arrhythmogenic (ARVC), and unclassified. In many cases, there is significant overlap between these different forms with regard to clinical signs, ECG, and radiographic findings. Echocardiography is needed to complete the diagnosis.

**Feline Hypertrophic Cardiomyopathy (HCM)**

Hypertrophic cardiomyopathy is defined by left ventricular hypertrophy without causative systemic or other cardiac disease. It is the most common form of heart disease in the cat.

The etiology for the majority of cases is unknown. However, it is inherited in Maine coon and ragdoll breeds and is believed to be in the American shorthair, sphyx, Norwegian forest, and a few others. In the Maine coon breed it has been shown to be an autosomal dominant trait (both genders are equally affected). In the Maine coon and ragdoll breeds a mutation has recently been identified as causative for the disease. Genetic tests based on a buccal swab are now available for these breeds (www.cvm.ncsu.edu/vhc/csds/vcgl/index.html). It is a different mutation in both breeds and does not appear to be causative in other breeds.

Hypertrophic cardiomyopathy is typically characterized by hypertrophy of the left ventricular free wall and/or interventricular septum. This results in myocardial stiffness and decreased ventricular lumen size and therefore it is a diastolic dysfunction disease. Mitral regurgitation may develop from distortion of the left ventricular cavity and from systolic anterior motion of the mitral valve (SAM). An increased left atrial pressure develops to fill the stiffened left ventricle. Elevated pulmonary venous pressure and pulmonary edema may result. In some cases, pulmonary hypertension and right ventricular enlargement may occur secondary to the left-sided heart disease, and pleural effusion may develop. Thrombi may develop in the stretched, dilated atria and subsequently break free and lodge in the systemic circulation (typically the distal aorta).

The clinical signs of affected cats are quite variable. Affected cats may be asymptomatic, but dyspnea and shortness of breath may be presenting complaints as a result of congestive heart failure. Acute hindlimb paralysis suggests distal aortic embolization. Sudden death can occur.

Physical exam findings may include a systolic murmur consistent with left ventricular outflow tract obstruction and/or mitral regurgitation. A gallop rhythm may be ausculted indicating abnormal left ventricular filling. Since asymptomatic cats may be affected, we recommend screening with an echocardiogram if a murmur or gallop is ausculted. Tachypnea and dyspnea may be observed if heart failure is present, but crackles are uncommon.

The ECG is often within normal limits but conduction disturbances and arrhythmias (ventricular and supraventricular) may be noted.

Radiographs may be useful to evaluate for cardiomegaly, chamber enlargement patterns, and evidence of heart failure but are not diagnostic for the specific form of feline heart disease. Radiographic findings may range from
normal to significant cardiac enlargement depending on the stage of disease and may show evidence of heart failure with pulmonary venous distension and patchy pulmonary edema.

Echocardiography is the best diagnostic test. Generalized concentric left ventricular hypertrophy may be observed, and localized left ventricular free wall and interventricular septal hypertrophy may be noted. Most clinicians use a cut-off of a wall thickness at diastole of greater than or equal to 6 mm as diagnostic in the absence of other causes of left ventricular hypertrophy such as hyperthyroidism or systemic hypertension. Wall-thickness measurements may be made on M-mode echocardiogram or by measurement of the wall on long axis. Multiple modes of echocardiography should be used for a thorough study, including 2D echo to scan for regions of asymmetric hypertrophy, M-mode for LV measurements, and Doppler to look for evidence of SAM and to document obstruction. Left atrial or biatrial dilation may be noted.

Since left ventricular hypertrophy can occur secondary to hyperthyroidism and systemic hypertension, these diseases should always be considered when evaluating a cat with left ventricular hypertrophy.

BNP is a test that is being discussed with increasing frequency in both canine and feline cardiology. Prohormone BNP is released when the ventricles are dilated, hypertrophic, or subjected to increased wall tension. However, it is most specific for identifying cats that are in heart failure. It has not been found to be reliable for assessing the presence or absence of heart disease in asymptomatic cats or in cats with heart murmurs.

**Treatment**

Treatment of hypertrophic cardiomyopathy is directed at decreasing the heart rate to allow for maximum filling time, decreasing the left ventricular outflow tract gradient if SAM is present, and controlling CHF, if present. The optimal therapy for asymptomatic cats is uncertain. In general, mildly affected cats are not treated. Cats that are tachycardic (> 220) and/or have outflow obstruction (SAM) on echo, should probably be treated.

The most commonly recommended treatment is a beta blocker (atenolol, B₁ selective). Atenolol (approximately 3 mg/kg orally, twice a day) should decrease the heart rate and left ventricular outflow tract gradient if SAM is present. However, atenolol should never be started in cats with congestive heart failure (CHF). Supportive treatment for CHF should be given to these cats, and once pulmonary edema is resolved (furosemide, enalapril), atenolol may be started. Calcium channel blockers (diltiazem) are another option, but these are now not frequently used. They may be used to decrease heart rate (perhaps less than beta blockers), but our ability to decrease left ventricular outflow tract gradient (SAM) with oral medications is unclear. Calcium channel blockers should also probably be withheld until the congestive heart failure is resolved.

In one study, the only drug that increased survival in HCM cats with CHF was enalapril (0.25–0.5 mg/kg q 24 hours), and atenolol actually decreased survival—but this was in cats with CHF. Atenolol (beta blockers) should never be started in a cat in CHF but can be used once the heart failure is controlled. However, if the cat is already on atenolol when CHF develops, the atenolol should not be withdrawn.

The prognosis for HCM may vary depending on etiology. Some cats progress rapidly to CHF; others plateau and never progress and live with mild disease for years. It may be best to advise owners that prognosis may be best determined by observing progression of the disease over months, and that cats with CHF or thromboembolic episodes have a poorer prognosis.

**Feline Dilated Cardiomyopathy (DCM) and Feline Restrictive Cardiomyopathy (RCM)**

These are both fairly uncommon forms of cardiomyopathy and will not be discussed today. The following information is provided to you as additional reference material.
Feline Dilated Cardiomyopathy
Feline dilated cardiomyopathy is a functional abnormality of the myocardium causing systolic dysfunction and is similar to the canine form. It is uncommon in cats but can be associated with taurine (an essential feline amino acid) deficiency. Although most commercial cat foods today are well supplemented, special diets or owner-created diets may be deficient. A small percentage of cats have dilated cardiomyopathy and normal plasma taurine levels, and the cause in these cases is unknown. Myocarditis may have preceded development of DCM in some of these cases. Radiographs may provide information about cardiomegaly and heart failure, but the echocardiogram is needed for diagnosis. In additional, taurine levels should be evaluated. Whole blood taurine levels should always be measured (normal mean is > 200 nmol/ml) even if the diet is thought to be balanced. Taurine levels are typically low (< 100 nmol/ml) with taurine deficiency. Since taurine supplementation is safe and inexpensive, taurine supplementation should be given until taurine deficiency is ruled out by blood levels. Taurine is given at 250 to 500 mg PO q 12 hours, orally. Medications for heart failure (Lasix, enalapril) should be provided as needed, and a positive inotrope should be started. Pimobenden (Vetmedin) could be provided (although it is not yet FDA approved for use in cats) at a dose of 1.25 mg/cat orally twice a day.

The prognosis for cats that are taurine deficient and given supplementation is actually quite good. Many cats will eventually be able to be removed from cardiac medications. The prognosis for cats with dilated cardiomyopathy that are not taurine deficient is not good, and many progress into advanced heart failure.

Feline Restrictive Cardiomyopathy
Restrictive cardiomyopathy is an uncommon myocardial disorder characterized by endomyocardial fibrosis, stiffened ventricular wall, and impaired ventricular filling. This is mainly a diastolic disorder. Systolic function may be normal or decreased. Etiology is unknown. Biventricular CHF with pleural effusion and thrombi commonly develop in the markedly dilated atria.
In many cases, there is significant overlap of clinical signs and ECG and radiographic findings between cats with hypertrophic, dilated, and restrictive cardiomyopathy. Echocardiography is needed to complete the diagnosis.

Arterial Thromboembolism
Thromboembolism can occur with all forms of feline cardiomyopathy. Three factors are typically required for clot formation: endomyocardial injury, blood stasis, and altered coagulability. Endomyocardial injury is likely due to injury and fibrosis in the large dilated left atria. Exposed collagenous fibers as well as reactive substances that occur with fibrosis act as reactive substances for platelet adhesion, and large, dilated atria may have areas of decreased contractility where blood pools. Finally, cats have large, sticky platelets, and platelets release serotonin as well as other factors that lead to a hyperaggregable state. Other coagulation abnormalities may be present in cats with cardiomyopathy.

Historically, treatment was directed toward removal or lysis of a clot. This is no longer recommended due to high mortality associated with rapid clot removal that is likely a result of reperfusion injury and hyperkalemia among other factors. Now, treatment is directed towards pain relief, which might include butorphanol (+/- acepromazine as tranquilizer) and an epidural (requires skill in local anesthesia) or a fentanyl patch (does not provide immediate relief)
The prognosis is frequently poor, but approximately 35% to 40% of patients regain use of their limbs. Although many cats will regain use of motor function within 1 or 2 weeks, risk of a second episode is high. Many owners elect euthanasia due to the patient’s discomfort and concurrent heart disease.

The best method of prevention is not known, but one could consider a platelet inhibitor such as clopidogrel (Plavix), 18.75 mg/cat q 24 hrs (more potent than aspirin, described in Hogan et al. 2004). For cats that have had one episode, many suggest adding a low-molecular-weight heparin. A less expensive but likely weaker option would be aspirin (81 mg every 72 hours). There is no clear evidence yet that clopidogrel is any more effective than aspirin, although
studies are ongoing. Generally, some type of anticoagulant preventive therapy is recommended for all cats with atrial enlargement and any form of cardiomyopathy.

**References**