Acquired cardiac diseases and their challenges—these cases are probably the most common to present to our facilities in need of anesthesia support for diagnostics or interventional procedures. This session will cover the most common acquired cardiac disease, potential complications, and methods of providing safe anesthesia. Many of these patients are on medications to medically manage their disease so discussion of anesthetic and analgesic agents will be covered as well as agents to manage intra-operative complications relating to their cardiac dysfunction.

In our feline patient, heart muscle diseases with diastolic dysfunction predominate; heart valve diseases are rare although some cats may present with mitral valve dysplasia.

► Cats usually do not cough with cardiac disease.
► Exercise intolerance is difficult to assess in cats.

Not all cats with heart murmurs have heart disease. Simple statement but the fact is that when we do auscult a feline patient with a murmur, we often want to give that patient underlying cardiac disease. The only true way to identify underlying cardiac disease is with an echocardiogram.

Hypertrophic cardiomyopathy (HCM) is the most common feline cardiomyopathy. It is considered an inherited autosomal dominant trait, however; it is considered to be idiopathic.

Domestic varieties: Persian, Maine Coon, Ragdoll, Birman, American Shorthair, Himalayan, Norwegian forest cat, Sphynx and Cornish Rex

Typically, the first sign of a cat with HCM is a heart murmur or gallop discovered during a routine physical examination. Murmurs, however, are not specific for HCM in cats—30 percent are of undetermined origin and some cats with murmurs never develop HCM. In our feline patients, systolic murmurs are best detected close to the apical area and at sternum level.

The gallop sound in cats is most often an S4 gallop heart sound (presystolic). This is caused by the atrial contraction forcibly pushing blood into a relatively high pressure ventricle. Because the heart rate commonly exceeds 160–180 bpm in cats, it is difficult to determine if the gallop sound is due to an S4 or S4 gallop on physical examination. Okay to be honest, with our feline patients the majority of them have rates in the hospital of 180 or above. S4 is the sound that occurs just after atrial contraction (“atrial kick”) at the end of diastole and immediately before S1. Atrial kick is the term used for the approximate 20 percent of additional blood flow from the atria to the ventricles. As the atria contract, the blood pressure in each atrium increases, forcing additional blood into the ventricles. Eighty percent of the blood flows passively down to the ventricles, so the atria do not have to contract a great amount.

Hypertrophic cardiomyopathy (HCM) is seen as an inappropriate hypertrophy of the left ventricle. The right ventricle may occasionally show involvement as well. Hypertrophy may be symmetrical, asymmetrical, or even localized. Cats as a rule do not follow rules and guidelines set forth by text books. Okay, so cats make their own rules.

The majority of HCM cats are classified as primary but HCM can be seen with hyperthyroidism and systemic hypertension. Less common secondary causes may include acromegaly, obesity, muscular dystrophy, and glycogen storage disorders.

The hypertrophy results in abnormal diastolic function while typically systolic function is preserved. The hypertrophied ventricular wall becomes stiff and prevents normal relaxation during diastole. Abnormal ventricular relaxation results in elevated filling pressures with the risk of congestive heart failure.
Feline patients with HCM may be seen as having a variety of clinical outcomes. They may be asymptomatic, develop intermittent or refractory congestive heart failure, have syncope, sudden cardiac death, or develop systemic arterial thromboembolism.

In the hypertrophic heart the heart muscle itself increases in thickness (size), which in turn causes the chambers within the heart to become smaller. Since the chambers are now smaller, they will fill with less blood than normal and consequently the heart will become a much less efficient pump.

The most common age of onset/diagnosis is greater than seven years of age; however, it has been seen as early as three months.

Hypertrophic cardiomyopathy (HOCM) is the second most common feline cardiomyopathy. It is considered an inherited autosomal dominant trait; however, it is considered to be idiopathic.

Domestic varieties, Persian, Maine Coon, Ragdoll, Birman, American Shorthair, Himalayan, Norwegian forest cat, Sphynx and Cornish Rex.

So what is the difference between HCM and hypertrophic obstructive cardiomyopathy (HOCM)? Hypertrophy of the interventricular septum may become severe enough to result in the narrowing of the left and/or right ventricle outflow tract. This form of HCM shows both diastolic and systolic dysfunction, and occurs when the myocytes that hypertrophy reduce the diameter of the outflow tract dynamically or statically. When this happens, stroke volume is reduced because the heart is trying to push the blood out a smaller hole. The result is the same.

A further means of obstruction is the process whereby the mitral valve may be actually sucked into the outflow tract, which is referred to as systolic anterior motion or SAM. This systolic anterior motion of the mitral valve leaflet striking the wall of the outflow tract will eventually create a lesion called a “kissing” lesion.

The heart must pump harder and faster to bring total cardiac output up to feed the body as well as feed the heart itself that has ever increasing oxygen demand as its workload increases.

So looking at the most common feline cardiomyopathies we see that these seem to be in essence relaxation defects. What should our goals be in premedicating, inducing, and maintaining safe anesthesia with these patients?

Anticholinergics—Atropine/glycopyrrolate, we know these will cause an increase in heart rate, contractility, cardiac output, and myocardial oxygen consumption.

Dissociative agents—Ketamine, we know these will indirectly stimulate the cardiovascular system by increasing sympathetic tone, cause an increase in heart rate, cardiac output, mean arterial pressure, pulmonary arterial pressure and central venous pressure and we also know that an increase in rate causes an increase in myocardial work and oxygen demand/consumption.

Most of my feline patients are premedicated with butorphanol. For really cranky, naughty cats we might consider alfaxalone with or without midazolam as a premedicant for IV catheter placement.

As always, hopefully the patient will accept pre-oxygenation with a mask (with or without the diaphragm). Pre-oxygenation of any patient is never a bad idea as long as it is tolerated and doesn’t add to preexisting stress levels. Pre-oxygenation is a technique that can “buy time” before the onset of hypoxemia. In cases where intubation may be difficult or prolonged or if the patient may be become apneic due to induction drugs, pre-oxygenating increases the oxygen content within the functional residual capacity of the lungs. It takes only 90 seconds for a patient that has not been pre-oxygenated to become hypoxic in the event of an airway obstruction as compared to 3–4 minutes in a pre-oxygenated patient.

Canine chronic valve heart disease (CCVHD) is responsible for 75 percent of all canine cardiac disease and affects 85 percent of dogs aged 13 years or older. Scary numbers indeed. CCVHD is also referred to as chronic valve disease (CVD), acquired mitral regurgitation (MR), endocardiosis, chronic myxomatous valve degeneration, and acquired mitral insufficiency (MI). For the purpose of our discussion we will simply refer to it as CVD.
CVD most commonly affects the mitral valve, although in approximately 30 percent of cases the (tricuspid) valve also is involved. The disease is approximately 1.5 times more common in males than in females. Its prevalence is also higher in smaller (less than 20 kg) dogs, although large breeds occasionally are affected. It is highly unusual before the age of 5 years except for Cavalier King Charles Spaniels.

The cause of CVD is unknown, but it appears to have an inheritable component in some breeds. CVD has changes that involve both the collagen content and the alignment of collagen fibrils within the valve itself. We may see endothelial cell changes as well as subendothelial thickening. In the majority of affected patients that we see in practice, the most common complication is mitral valve prolapsed. Cardiac remodeling occurs 2nd to progressive valve incompetence.

- Left atrial ± left ventricular dilation
- Left ventricular systolic dysfunction

The American College of Veterinary Internal Medicine’s Board of Regents selected a group of European and U.S. ACVIM boarded cardiologists to arrive at a consensus as to the diagnosis and treatment of CVD.

The system developed by ACVIM has four basic stages of heart disease and failure.

**Stage A** identifies patients at high risk for developing heart disease but do not have any structural cardiac disorder.

**Stage B** identifies patients with structural heart disease. These are those patients that have a typical mitral valve regurgitation murmur but have never shown clinical signs caused by heart failure. Because of the fact that there are important clinical implications for prognosis and treatment, Stage B has been divided further.

**Stage B1** are those asymptomatic patients that have no radiographic or echocardiographic evidence of cardiac remodeling in response to CVD.

**Stage B2** are also asymptomatic patients but they do have significant valve regurgitation. This is indicated by radiography or echocardiographic findings of left sided heart enlargement. This would be the time to include counting of resting respiratory rates in your client education so they can start the process of home monitoring.

**Stage C** are patients that have had past signs of heart failure or current signs of heart failure. There are treatment differences between patients with Stage C CVD. There are those who present to us in the clinic in acute heart failure that need hospitalization for stabilization and those patients who may be treated on an outpatient basis and dogs with acute heart failure requiring hospitalization.

**Stage D** is the last classification and is reserved for those patients with end-stage disease that exhibit clinical signs of heart failure that are resistant to standard therapy recommendations.

When anesthetizing a patient with mitral valve disease, one must be careful since the vasodilatory effects of many anesthetics may be exaggerated by the patient’s existing medication. On the other hand, untreated mitral valve disease may benefit from the vasodilating effects of anesthetics. When pressors are needed, pure alpha agonists such as phenylephrine that cause vasoconstriction may exacerbate the back pressure and may result in pulmonary edema. Beta agonists such as dobutamine may be used to improve contractility. Fluids must be administered conservatively, to avoid overload and pulmonary edema. However, because these patients may have been on diuretics and also have the same issues as a normal patient with regard to fluid needs, there is a balance that must be achieved. Currently, the most effective way to monitor this balance is by measuring central venous pressure (which approximates the pressure of the blood entering the right atrium). Maintaining heart rate within the normal range for these patients is important. Tachycardia should be avoided as it will increase the oxygen needs of the heart, so an anticholinergic is usually not recommended prophylactically as in a pre-med. However, low doses can be used if indicated by bradycardia that is causing hypotension.

Opioids, benzodiazepines, alfaxalone, and etomidate are considered good choices in an anesthesia protocol for these cases. Ketamine is usually avoided as it increases myocardial oxygen needs. Propofol should be avoided or used with extreme care due to its vasodilating and hypotensive effects. IV administration of an opioid/benzodiazepine combination immediately prior to induction with propofol can greatly reduce the amount of propofol required to capture the airway thus allowing the anesthetist to use this agent while avoiding the majority of the myocardial depressive effects.
The incidence of dilated cardiomyopathy (DCM) in dogs is rare relative to CVD. It is unusual to see in dogs that are younger than 5 years of age. In general, large breed dogs are more apt to develop DCM than small/medium breed dogs. As with CVD, the incidences of idiopathic DCM increases with age. Of our large breed dogs, the Doberman is more often affected than all other breeds combined.

Some American Cocker Spaniels have been reported to develop DCM associated with low taurine levels. Taurine supplementation may result in reversal of the disease and a significantly better prognosis. Although taurine does not appear to be associated with the development of DCM in other commonly affected breeds.

DCM is a disease of the heart muscle that causes the heart to weaken and enlarge. DCM usually affects primarily the left side of the heart but the right side of the heart may be involved as well. Typically, both the ventricles and the atria are enlarged and often the muscular walls of the heart are much thinner than normal. The end result is impairment in the ventricle’s ability to pump blood out to the body and lungs. When the ventricles fail to pump effectively, a backup of blood results in the systemic circulation. If the left heart fails, fluid backs up into the lungs causing coughing and/or difficulty breathing. If the right heart fails, fluid backs up into the abdomen causing abdominal distension or pleural effusion.

The occurrence of DCM increases with age and typically has an age of onset between 4 and 10 years.

This condition is characterized by elongation, weakening of the heart muscle, so that chamber size is increased, but contractility is compromised. Stroke volume is reduced and resulting in decreased blood pressure. Compensatory mechanisms for hypotension ensue. Chamber enlargement may alter the shape and function of the mitral valve, leading to valvular insufficiency. In addition, the chamber may be so enlarged as to affect cell communication and organized depolarization. Atrial fibrillation, premature ventricular contractions, and ventricular tachycardia may develop. Whether heart failure is due to valvular insufficiency or cardiomyopathy, compensatory mechanisms in response to poor cardiac output are similar. The sympathetic nervous system, the renin-angiotensin-aldosterone system is activated. Secretion of antidiuretic hormone increases. Results are increased heart rate and vascular tone, and sodium and water retention.

Digitalis and digoxin are positive inotropes that increase the concentration of calcium within the myocardial cells thus increasing the force of contractions and decreasing heart rate. A relatively new addition to management is pimobendan, which elicits calcium sensitization of the myofilaments. Anti-arrhythmics such as procaainamide, quinidine, or mexiletine may be used if indicated. Calcium channel blockers and beta blockers may be used to help control supraventricular arrhythmias. Diuretics such as furosemide are used for decreasing circulating blood volume. ACE inhibitors cause relaxation of the blood vessels, thereby decreasing systemic vascular resistance. Anesthetic protocols for patients with dilated cardiomyopathy should aim to avoid drugs that would increase heart rate or decrease contractility. Fluids must be delivered with caution to avoid overload. Ideally central venous pressure would be monitored to determine how well the heart is able to handle the circulating volume and fluids may be regulated accordingly.

Hypotension may be corrected with the use of CRI opioid/benzodiazepines to reduce vaporizer setting thus avoiding the dose dependent peripheral vasodilation attributed to inhalant anesthetics. Positive inotropes or beta agonists such as dobutamine may be added if further support of adequate blood pressure is required. These drugs focus on improving contractility without increasing afterload. Dobutamine should be diluted and administered as a constant rate infusion, beginning at 2 to 5 micrograms per kilogram per minute, and increased as needed. The use of dobutamine is not benign as increased rate of administration may cause an increase in heart rate or arrhythmias may be noted as it acts as a positive inotrope as well at higher doses as a vasoconstrictor. Dopamine as a CRI may also be used to support blood pressure and is calculated to be delivered at a starting rate of 2 to 5 mcg/kg/minute. Ventricular arrhythmias may be exacerbated with the use of beta agonists, however, so close monitoring of the EKG is necessary.