**HOW TO STAGE HEART DISEASE, PART 1**

**Approach for assessing incidental murmurs**

**Canine valve disease; staging and therapy**

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**Incidental murmurs in dogs and cats**

Introduction

Cardiac murmurs are usually associated with turbulent or high velocity blood flow in the heart or great vessels. A quiet environment is essential for successful auscultation. Extraneous sounds, such as panting or purring, can make auscultation very difficult in some patients. Restraining the animal near a sink with slowly running water or placing a cotton ball soaked with alcohol near the cat’s face can sometimes be used to stop purring. Occasionally, breath sounds or fur rubbing against the stethoscope can be mistaken for sounds originating from the heart. Careful auscultation while watching the dog or cat’s breathing pattern can be useful to differentiate heart sounds from breath sounds.

It is very important that complete auscultation is performed and the animal should be standing. A simple approach is to auscult the left heart base, under the triceps musculature, which is the region of the aortic and pulmonic valve areas and the left apex, which is the mitral valve area. The right heart base and apex should also be ausculted. The right apex, is the area over the tricuspid valve area and tricuspid leaks are usually loudest in this region. Murmurs associated with ventricular septal defects are usually loudest at the right heart base.

Cardiac murmurs are most often caused by the vibrations associated with high velocity, disturbed and turbulent blood flow. Such disturbances may be caused by valvular incompetence, valvular stenosis or the presence of a shunt. Disturbed flow that is low velocity, such as pulmonic valve regurgitation, may not be auscultable. However, pulmonic valve regurgitation is rarely clinically important. Murmurs can also be created by other physiologic or pathological processes. For example, murmurs are often heard with changes in the viscosity of blood (anemia) and high output diseases such as hyperthyroidism can be associated with the development of murmurs due to increased velocity of ejected blood.

Murmurs are classified in several ways:

Timing in the cardiac cycle (systolic, diastolic, continuous). While most common cardiovascular diseases in small animals result in systolic murmurs, occasionally feline patients will present with diseases such as mitral stenosis (which results in a diastolic murmur) and a patent ductus arteriosus results in a continuous cardiac murmur.

Intensity is the loudness of the murmur most often using a 6-tier scale. The intensity of the murmur does not necessarily correlate with the severity of the underlying heart disease as some well tolerated abnormalities (such as restrictive ventricular septal defects) produce very loud murmurs, while some severe lesions may produce soft murmurs (i.e. unrestrictive ventricular septal defect or severe tricuspid regurgitation).

Point of maximal intensity (PMI) is the area at which the murmur is loudest, and it generally relates closely to the underlying source of turbulent flow. For example, a left to right patent ductus arteriosus murmur is usually heard loudest over the left heart base at the pulmonary artery, because high velocity flow from the aorta is shunting through the duct into the pulmonary artery. If the murmur is loud on auscultation over the entire chest, the presence of a precordial thrill will identify the PMI.

Radiation of a murmur refers to how widely the murmur can be heard from the point of maximal intensity. Generally, the wider the radiation of the murmur, the more severe the lesion.

Pitch and quality are subjective descriptions of the character of a murmur (i.e. coarse, musical, etc). These descriptors are not precise and do not have the same objectivity as timing and point of maximal intensity.

Certain murmurs are pathognomonic for specific diseases. For example, a continuous murmur at the left heart base is almost always a patent ductus arteriosus (although rarely an aortic-pulmonary window or an AV fistula can produce a similar murmur). Similarly, a systolic murmur with a point of maximal intensity at the right chest is usually associated with a ventricular septal defect or tricuspid regurgitation.

**Specific considerations in the feline patient**

It is often impossible to differentiate innocent (benign) murmurs from those murmurs secondary to significant heart disease based on auscultation alone, particularly in the cat where innocent murmurs are very common. Benign dynamic right ventricular outflow tract stenosis is an important and common cause of innocent murmurs in cats. Soft (< grade 4/6) murmurs at the heart base or parasternum, which disappear at slower heart rates are more likely to be innocent, particularly if thoracic radiographs reveal a normal heart size. While ausculting the feline patient, it is often possible to slow the heart rate by covering the cat’s face in the examiner’s hand or in the crook of the examiner’s arm. Evaluating the murmur character while the heart rate changes can be very helpful in the feline patient.

Heart murmurs are common findings in normal cats. Depending on the study, roughly 25% of normal cats have auscultable cardiac murmurs and echocardiographic studies have demonstrated that, in cats with cardiac murmurs, approximately 50% of the murmurs are benign findings (i.e. there is no underlying heart disease) and of cats with cardiomyopathy, approximately 50% have murmurs and 50% do not. These statistics make one question whether performing echocardiography in all cats with murmurs is cost effective. Echocardiography is the best diagnostic modality to define underlying cardiac disease and identify the cause of the murmur because in the early stages of disease, hypertrophic cardiomyopathy may be present without radiographic evidence of heart enlargement. However, over time the left (most commonly) atrium enlarges due to elevated ventricular filling pressures associated with cardiomyopathy and usually left atrial enlargement is present prior to the development of congestive heart failure. Therefore, thoracic radiographs are a reasonable screening tool when a murmur is detected on physical examination.

For the owner that wants a definitive answer as to the etiology of a cardiac murmur in a cat, an echocardiogram is recommended. Otherwise, it is reasonable to educate the owner that, although early cardiomyopathy may be present and not recognized with thoracic films, serial thoracic films demonstrating a persistently normal heart size suggest that, if cardiomyopathy is present, it is clinically compensated. There is no evidence that beginning cats on cardiac medications prior to the onset of left atrial enlargement is beneficial. Therefore, cardiac medications would not be recommended for this cat even if early cardiomyopathy were detected. Findings that increase the risk that structural heart disease is present (and therefore the echocardiogram is more likely to yield critical information include):

1. History suggestive of primary heart disease (i.e. possible syncopal events)
2. The murmur is continuous or diastolic.
3. The murmur has a precordial thrill.
4. Heart enlargement is present on thoracic radiographs
5. There are additional physical examination findings consistent with heart disease (i.e. an auscultable gallop sound, arrhythmia)

**Specific considerations in the canine patient**

Caveats to remember:

1: Benign murmurs occur in puppies and adult dogs, but are much less common in adult dogs compared to adult cats.

2. As in cats, diastolic, continuous and murmurs with a precordial thrill in young animals demand further investigation.

 3. Grade I/VI murmurs are rarely clinically significant unless in a breeding animal.

A **new** cardiac murmur is noted on physical examination.

 If the murmur is:

Continuous,

Diastolic, or

> Grade IV/VI, the puppy should be worked up

If murmur persists or is of a character that warrants diagnostics (red text above), thoracic radiographs + echocardiography result in definitive diagnosis and treatment plan.

If murmur disappears as puppy ages, the murmur was benign and no further diagnostics are required.

If the murmur intensity is < Grade IV/VI and it’s systolic, it may be a benign murmur the puppy will outgrow and its reasonable to monitor.

Dog is less than 4 months old

Dog is an adult

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A **new** cardiac murmur is noted on physical examination in an adult dog.

Diagnosis is dilated cardiomyopathy.

Therapy vs. no therapy

Beta blockers

Pimobendan

Diagnostics:

Thoracic radiographs to stage disease

Measure blood pressure

+/- echocardiography

Stage B2

No cardiac medications

Re-evaluate heart size every 6 months

Resting respiratory rate log

#1 differential is degenerative valve disease

Dog is a small breed

Diagnosis is degenerative valve disease

Diagnostics:

Echocardiography

+/- thoracic radiographs

Measure blood pressure

Differential diagnoses:

1. Dilated cardiomyopathy

2. Degenerative valve disease

Dog is a medium to large breed

Stage B1

No cardiac medications

Re-evaluate heart size yearly

Educate owner about heart disease

**Staging and treatment of degenerative valve disease in dogs**

Degenerative valve disease (DVD) is the most common form of heart disease in the dog. It’s common presence in small breed dogs, particularly the cavalier King Charles spaniel, toy and miniature poodle, Bichon frise, etc. suggests a genetic predisposition in some breeds, however a specific genetic mutation has not yet been identified. The ACVIM consensus statement offers a simple classification system for dogs with acquired valve disease that is helpful for prognostic and therapeutic planning.

Stage A- dog is not clinically affected with valve disease, but is of a breed which is at

risk for later valve disease development (i.e cavalier King Charles spaniel)

 Stage B- valve disease is present, but no evidence of congestive heart failure

 Stage B1- no cardiac remodeling (the heart size is normal)

 Stage B2- cardiac remodeling (heart size is increased)

 Stage C- current or historic congestive heart failure

 Stage D- refractory heart failure

**Stage A** dogs are clinically normal, but because of breed predisposition, may develop heart disease in the future. For owners of these dogs it is useful to discuss signs associated with heart disease and depending on their plans, discussion regarding breeding decisions may be warranted.

**Stage B** dogs by definition have a cardiac murmur. For staging purposes, thoracic radiographs, systemic blood pressure and a minimum database (creatinine , urine SG, PCV/TS) is recommended. If the heart size is normal on thoracic radiographs, the dog is considered to be in stage B1 (no cardiac remodeling). No cardiac medications have been shown to alter the progression of disease at this stage of disease. However, regular monitoring for cardiac enlargement with thoracic radiographs is recommended every 12 months. If heart enlargement is detected, the dog is considered to be in stage B2. The EPIC trial results demonstrated that pimobendan therapy resulted in delayed development of congestive heart failure when B2 dogs were started on pimobendan. Based on that trial, B2 criteria included an LA/AO of >1.6 and an LVIDD normalized to body weight > 1.7. Additional ancillary recommendations for B2 dogs include: supplementation with an N-3 fatty acid supplement and counseling the owner to keep a log of the dog’s resting or sleeping respiratory rate. More regular cardiology rechecks are also recommended for dogs in stage B2 because several studies have demonstrated that the onset of congestive heart failure is preceded by a more rapid increase in heart size. Generally rechecks every 6-9 months are recommended for stage B2 dogs (depending on severity of heart enlargement).

Note that an echocardiogram is not necessarily needed in an asymptomatic dog with a signalment and findings highly likely to be degenerative valve disease (for example a small breed dog with a left apical systolic murmur). However, an echocardiogram is recommended in cases that “do not follow the book”. For example, medium size dogs, which may have degenerative valve disease (DVD) or dilated cardiomyopathy (DCM), dogs in which the murmur is loudest on the right, dogs with an unusual pattern of cardiac enlargement, etc.

**Stage C** dogs are dogs that have or have had congestive heart failure due to degenerative valve disease. In the acute stage, treatment of symptomatic congestive heart failure is similar whether the underlying cause is DVD or DCM. For chronic control of CHF, most dogs are treated with pimobendan, an angiotensin converting enzyme inhibitor (ACEI), furosemide and spironolactone. Ongoing owner monitoring of resting respiratory rate is important, and this information is often used for cautious reductions in furosemide dose. It is important to use the lowest dose of furosemide that controls clinical signs of CHF. Regular monitoring of kidney function with bloodwork is also an important part of care in stage C.

For those patients with refractory heart failure (**Stage D**), additional medications may be beneficial in certain cases. For example, further reduction of afterload with amlodipine or heart rate control with digoxin and/or diltiazem may be warranted. Generally, “triple diuretic therapy” (the combination of furosemide, hydrochlorothiazide and spironolactone) and/or torsemide is reserved for this stage of heart failure.

Medical management for stage D dogs often necessitates multiple medications and it is important to balance the effect of these drugs. The drugs available to improve cardiac output work on the following formula:

 Cardiac output = Preload X Contractility X Heart rate

 Afterload

Although reducing preload results in a reduction in cardiac output, because the Starling curve is flattened in patients with heart failure, a reduction in preload will optimally decrease filling pressure to alleviate pulmonary edema with minimal impact on cardiac output. The diuretics are the primary preload reducers with furosemide being the one most commonly used. Nitrates, such as nitroglycerine, are also preload reducers. However, nitroglycerine is of questionable efficacy in dogs and cats.

Drugs that decrease afterload, arterial dilators, result in improved cardiac output. Pimobendan is a vasodilator and one of its beneficial effects is a reduction in afterload. The ACEi agents are also vasodilators. Additional arterial dilators include amlodipine and hydralazine.

The only effective drug for chronic use that improves contractility is pimobendan. Although digoxin was historically considered a positive inotropic agent, the effect is trivial and it should not be chosen over pimobendan for inotropic support. In the acute heart failure setting, sympathomimetic agents, most commonly dobutamine, are often used for inotropic support. This synthetic catecholamine is rapidly metabolized and must be administered as a constant rate infusion.

When the heart rate is either very elevated or very slow, it will negatively impact cardiac output. In dogs with symptomatic valve disease, atrial fibrillation is the most common tachycardia that requires medical management. There are three main classes of drugs that can be considered for heart rate control in dogs with supraventricular tachycardias, such as atrial fibrillation: Beta adrenergic blockers, Calcium channel blockers and Digoxin. Beta blockers should be administered cautiously in dogs with poor myocardial (or uncertain) myocardial function as they reduce contractility. For this reason, beta adrenergic blockers should never be administered to patients in overt heart failure. Digoxin is a very effective negative chronotrope for chronic use, but the risk of toxicity with intravenous administration is so high that this administration route is not recommended. In the emergency setting, intravenous diltiazem can be used to control rapid supraventricular tachycardias.