Meaningful evaluation of the heart is predicated on a sound knowledge of normal anatomy, physiology, and breed variations and adherence to principles of standardized technique and positioning to eliminate non-pathologic variables. Digital radiography has helped improve radiographic technique but appropriate animal positioning remains critical. The ribs and spine should be penetrated well enough to show some bony detail (trabeculation) on the lateral view. On the VD or DV view, the thoracic vertebrae should be just visible but not show detail where they overlie the heart. One should also be able to trace the course of the descending aorta along the left side of the spine on the VD or DV view. Always consider the possibility of noncardiac anatomic or pathophysiologic factors that can alter the appearance of the heart including congenital anomalies of the spine, sternum, or rib cage, intrathoracic masses or fluid, megaesophagus, pneumothorax, lung collapse, a diaphragmatic hernia, trauma (including broken ribs, lung contusion), and pneumonia or other parenchymal lung densities overlapping the heart. Chest radiographs are fairly accurate for identifying left atrial (lateral view), left ventricular and right ventricular enlargement (DV view). Right ventricular and right atrial enlargement are often over-interpreted. Left ventricular eccentric hypertrophy is more visible than concentric hypertrophy.

Lateral view
The normal heart occupies approximately 2.5 to 3.5 intercostal spaces, and the height of the heart is approximately 2/3rds the height of the chest. The trachea typically diverges ventrally from the spine at an angle of about 20°. The caudal waist should be distinctly visualized. The shape of the heart conforms to the general shape of the thoracic cavity. In brachycephalic and other barrel-chested dogs, the heart appears rounder and larger than in the "normal" dog. The trachea may run nearly parallel to the spine on the lateral view. In these dogs, the heart often occupies nearer to 3 1/2 intercostal spaces and it contacts more of the sternum ventrally. In narrow, deep-chested breeds, the heart is more "upright" and slender appearing on the lateral view. Cats have a slender conical-appearing heart that tends to be tipped slightly on the lateral view with the base of the heart lying more anteriorly. In older cats, the heart is less upright and may be inclined almost horizontal to the sternum. Older cats also often have an elongated, tortuous aorta on the lateral view.

An alternative method of evaluating heart size developed by James Buchanan may prove helpful. If the long and short axes of the heart are measured in the lateral views using the thoracic vertebrae as a scale (starting with the cranial margin of the 4th thoracic vertebra), the sum of these measurements (vertebral heart score = VHS) should not exceed 10.5 vertebrae. In 100 normal dogs the average measurement was 9.7 vertebrae. Most normal cats have a short axis dimension of 3.1 to 3.4 vertebrae, and a VHS of 7.2 to 7.8 vertebrae.

Dorsoventral or ventrodorsal view
The width of the heart is approximately 1/2 to 2/3rds the width of the thorax. The right and cranial borders are rounded and the left border nearly straight. The apex points to the left side of the thorax. On the DV view the heart occupies at least 2/3rds of the width of the thorax and the apex of the heart is sometimes directed more to the left. In narrow, deep-chested breeds the heart often appears small and round due to the upright position of the heart.

Radiographic signs of cardiac chamber enlargement
1) Right atrial enlargement
Right atrial enlargement is encountered rarely as an isolated abnormality in the form of congenital tricuspid valve stenosis. It is observed most often with RV enlargement as a result of acquired (valve degeneration) or congenital tricuspid valve insufficiency (tricuspid dysplasia). Right atrial enlargement also develops in dogs with right heart failure due to heartworm disease or dilated cardiomyopathy.

2) Right ventricular enlargement
Right ventricular enlargement develops as a consequence of pressure overload - pulmonic stenosis, tetralogy of Fallot, pulmonary hypertension; as a consequence of volume overload -tricuspid valve insufficiency or an atrial septal defect; and it enlarges in concert with the left ventricle in dogs with dilated cardiomyopathy.

3) Main pulmonary artery enlargement
The main pulmonary artery segment is located between 1:00 and 2:00 o'clock using the clock-face analogy. It enlarges as a consequence of pulmonary hypertension, pulmonic valve stenosis (post-stenotic dilatation), and as a result of volume overload - left to right shunting defects (ASD, VSD, PDA), and pulmonic valve insufficiency.
4) Left atrial enlargement
Left atrial enlargement is usually seen together with LV enlargement as most acquired disorders, such as mitral regurgitation and dilated cardiomyopathy affect both chambers. LA enlargement with no or minimal LV enlargement may be seen with mitral valve stenosis and with those disorders causing concentric LV hypertrophy, e.g. aortic stenosis and hypertrophic cardiomyopathy. Cats frequently display solely left auricular enlargement, a finding that is often absent on the lateral radiograph because of the anatomic location of the left auricle.

5) Left ventricular enlargement
Left ventricular enlargement develops as a consequence of pressure overload (concentric hypertrophy) due to valvular or subvalvular aortic stenosis or systemic hypertension or as a consequence of volume overloading (eccentric hypertrophy) due to mitral valve insufficiency or a left to right shunting VSD or patent ductus arteriosus. Concentric LV hypertrophy also occurs due to hypertrophic cardiomyopathy. The LV undergoes eccentric hypertrophy in concert with the right ventricle in dogs with dilated cardiomyopathy. Some cases of severe concentric hypertrophy have minimal radiographic changes because the sarcomeres are in parallel, thereby increasing the wall thickness but decreasing the radius of the left ventricle.

6) Aortic arch enlargement
The aorta is located between 11:00 and 1:00 o'clock using the clock-face analogy. It enlarges as a consequence of subvalvular aortic stenosis (post-stenotic dilatation), as a result of aortic valve insufficiency, and, more rarely as an idiopathic disorder or secondary to systemic hypertension. The aortic arch is also enlarged in dogs with a patent ductus arteriosus or tetralogy of Fallot.

Radiographic evaluation of the pulmonary arteries and veins
On the lateral view, the pulmonary arteries lie dorsal to the bronchus while the veins are located ventral to the bronchus. In the lateral view, vessels in the cranial and middle lung lobes are most easily seen. The arteries and veins should be approximately the same size. On the dorsoventral view, the pulmonary arteries lie lateral to the bronchus while the pulmonary veins are medial to the bronchus.

1) Enlarged pulmonary arteries and veins
Left to right shunts cause enlargement of both the pulmonary arteries and the pulmonary veins together with an overall increase in pulmonary density. Expiratory radiographs can accentuate the size of the pulmonary vessels and can falsely suggest pulmonary edema.

2) Diminutive pulmonary arteries and veins
Right to left shunts cause a decrease in the size of the pulmonary arteries and veins and a generalized decrease in pulmonary density. Also consider the possibility of hypovolemia.

3) Enlarged pulmonary veins with normal arteries
This finding usually indicates left heart failure or iatrogenic over-hydration. This sign is more reliable in dogs than in cats.

4) Enlarged pulmonary arteries with normal veins
With pulmonary hypertension (heartworms), the proximal branches of the pulmonary arteries are often noticeably larger than the veins. The arteries are also often more tortuous and truncated in appearance.
Wrapping Your Head Around the Pericardium: A Review of Pericardial Disease
Barret Bulmer, DVM, MS, DACVIM
Tufts Veterinary Emergency Treatment and Specialties
Walpole, WA

Congenital and acquired pericardial disease may be encountered in a variety of situations ranging from the asymptomatic animal to the patient in acute shock. Therefore the possibility of pericardial disease should remain at the forefront of many different clinical presentations.

Pathophysiology
The most important pathophysiologic effect of pericardial disease is the reduction of diastolic filling of the heart. Diastolic dysfunction causes a reduction of ventricular stroke volume and cardiac output. The severity of clinical signs usually depends on the rate of fluid accumulation. It is important to realize that the pericardial pressure-volume relationship is such that there is a progressively greater rise in pericardial pressure as pericardial volume increases.

History and clinical signs
Rapidly developing cardiac tamponade can cause acute hypotension, weakness, dyspnea, collapse, and sudden death. Animals with slowly developing and chronic pericardial effusion may present with signs of right-heart failure including abdominal distension, respiratory difficulty associated with pleural effusion, or exertional syncope. The heart sounds are usually muffled, careful examination will often reveal jugular venous distension or a positive hepatojugular reflux, and femoral arterial pulses are often reduced in strength or exhibit pulsus paradoxus.

Diagnostics
1) Electrocardiogram: ECG alterations are variable and non-specific but may often include low amplitude QRS complexes in all leads, sinus tachycardia, ventricular premature complexes (depending on the etiology of the effusion), nonspecific ST segment elevation or depression, and electrical alternans.
2) Thoracic radiographs: Although we often describe a large globular cardiac silhouette rounded in all views as the characteristic finding of pericardial effusion, most studies suggest there are no steadfast radiographic findings for distinguishing cardiac tamponade from various other cardiovascular diseases. With small effusions, changes may be minimal and animals with pleural effusion may have an obscured cardiac silhouette.
3) Echocardiography: Echocardiography is the most sensitive method of detecting pericardial effusion and it often permits visualization of neoplastic lesions that may serve as the etiology for cardiac tamponade.

Pericardiocentesis
Usually with the dog in left lateral recumbency, the right fifth intercostal space at the costochondral junction is clipped and surgically prepared. While monitoring the ECG, an over-the-needle catheter is advanced toward the heart and when fluid is obtained, the catheter is advanced into the pericardial sac. The stylet is withdrawn and most often the catheter is attached to an IV extension set, three-way stopcock and syringe for aspiration. The catheter may be fenestrated to make aspiration of fluid easier. Aspirated fluid can be compared to peripheral blood and monitored for clotting to make certain accidental cardiac catheterization has not occurred. Routine cytology and fluid analysis is generally performed to try and exclude bacterial, fungal, or obvious neoplastic etiologies. But with most hemorrhagic effusions it is impossible to distinguish the neoplastic effusates from idiopathic effusions.

Diseases of the pericardium
Congenital diseases of the pericardium are infrequently encountered but their recognition is gratifying because they are often amenable to surgical correction. These lesions include pericardial defects, peritoneopericardial diaphragmatic hernia and intrapericardial cysts. Acquired pericardial disease is typically manifest as pericardial effusion of neoplastic or idiopathic/inflammatory origin. Less common etiologies for pericardial effusion include uremia, left atrial tear, rodenticide toxicity/coagulopathy, infectious disease, heart failure, hypoalbuminemia, trauma or pericardial foreign bodies. The prognosis and treatment varies with the underlying etiology but in general hemangiosarcoma and mesothelioma carry poor prognoses, while idiopathic disease and effusion related to chemodectoma often carry a better prognosis depending on response to therapy.

References/suggested reading
Reacquainting Yourself with the ECG and Treatment of Arrhythmias
Barret Bulmer, DVM, MS, DACVIM
Tufts Veterinary Emergency Treatment and Specialties
Walpole, WA

A systematic approach to the evaluation of the ECG will prevent overlooking important abnormalities. The following characteristics should be evaluated in every ECG. Familiarity with the normal parameters for the ECGs of the various species is, of course, essential for accurate interpretation.

1) Determine the heart rate
If the heart rate is regular, the number of small boxes (mm) between QRS complexes can be divided into 3,000 (at 50 mm/sec) or 1,500 (at 25 mm/sec) to find the instantaneous heart rate. The heart rhythm in animals, especially in dogs, is frequently irregular. In this circumstance the more accurate average heart rate is found by counting the number of beats in a known time interval and multiplying appropriately. Single channel ECG paper on analog recorders is usually marked by a vertical line at the top of the paper at 75 mm (1 mm = 1 small box) intervals. At a paper speed of 50 mm/sec, 75 small boxes (equivalent to 15 large boxes) represent 1.5 seconds so the heart rate per minute can be calculated by counting the number of QRS complexes in 1.5 seconds and multiplying by 40. At a paper speed of 25 mm/sec, 75 small boxes (15 large boxes) represent 3.0 seconds and the number of QRS complexes in 3.0 seconds is multiplied by 20. Many of the newer digital ECG machines calculate heart rate automatically.

2) Determine the cardiac rhythm
The heart’s rhythm is evaluated by inspection of the ECG and the findings are correlated with the physical findings. Analysis of the heart’s underlying rhythm should include the following steps.

A. What is the rhythm (including the regularity and the relationship among complexes)?
   a. Regular?
   b. Regularly irregular with a consistent and repeating pattern to the variation in the rate?
   c. Irregularly irregular where the rhythm is chaotic and there is no pattern to the irregular nature of the rhythm?
   d. Paroxysmal (which is defined as a sudden outburst)? When applied to the ECG, a paroxysm refers to a series of rapid ectopic beats, which begins and ends abruptly. The series may be as short as 3 beats or may last for minutes to hours.
   e. What is the relationship between the P and QRS complex? Is there a P wave for every QRS complex? Is there a QRS complex for every P wave? Is the duration of time between the various components (P-R interval, Q-T interval) normal? Is the duration of time between the various complexes consistent?

B. Where do the cardiac impulses originate (site of origin)? The four possible choices include:
   a. The sinoatrial (SA) node
   b. The atria
   c. The atrioventricular (AV) node/junctional
   d. The ventricles and His-Purkinje system

Impulses originating from the SA node, atria or AV node are grouped together under the heading supraventricular while impulses from the ventricles or His-Purkinje system are termed ventricular. Supraventricular beats should maintain a relatively tall, upright and narrow QRS complex because the impulse must utilize the His-Purkinje system to transmit the impulse to the ventricles. Therefore the ventricular muscle depolarizes uniformly with a set activation sequence. But when impulses arise from the ventricles or terminal branches of the His-Purkinje system they are slowly transmitted from individual myocardial cell to myocardial cell. This produces a relatively wide and bizarre QRS-T complex.

C. What are the ventricular and atrial rates?
   a. Too fast (tachycardia)
   b. Too slow (bradycardia)

D. What is the temporal relationship between any ectopic beats and the underlying heart rhythm?
   c. Premature beats are defined as ectopic beats that occur early in the sequence of normal beats, meaning the R-R interval from the preceding normal beat to the ectopic beat is shorter than the prevailing R-R interval. Premature beats are formed when the ectopic focus depolarizes more rapidly than normal, overrides the sinus node and assumes control of the heart rate for one or more beats.
   d. Escape beats are defined as ectopic beats that occur after a pause in the sequence of normal beats, meaning the R-R interval from the preceding normal beat to the ectopic beat is longer than the prevailing R-R interval. The ectopic site assumes control of the electrical activity of the heart by default because the SA node fails to discharge or the sinus impulse is not properly conducted to the rest of the heart.
3) Calculate the mean electrical axis (MEA)
One of the most useful applications of vector principles is the calculation of the MEA for the QRS complex in the frontal plane. The MEA is the average of all the instantaneous vectors recorded during the QRS complex. Each species has a range of normal values, for example the MEA of normal dogs is between +40° and +103°. When the MEA is greater than +103° right ventricular enlargement is suggested. The mean electrical axis may be derived in two ways:

- Method 1: Using any two leads in the frontal plane, take the difference between the height of all positive QRS deflections and all negative QRS deflections in the two chosen leads. This calculates the vector for each lead. Plot the appropriate number of units, either positive or negative, on the lead axes. Draw perpendicular lines to the axes at these two points and then draw a vector from the origin of the figure to the point of intersection of these lines. The direction of this vector is the mean electrical axis.

- Method 2: Since the line of the mean electrical axis should have half of the total forces of ventricular depolarization on either side of it, a reasonable estimate of the MEA can be obtained by finding the limb lead which is the most isoelectric (i.e. the difference between the positive and negative QRS deflections in that lead is near 0). The MEA must then be perpendicular to that lead. To determine which direction the MEA takes, look at the lead whose axis is perpendicular to the isoelectric lead. If the lead has mainly positive QRS deflections, the MEA points toward the positive pole of that lead axis, just the opposite if the lead is mostly negative. Occasionally all of the limb leads are equally isoelectric and the MEA is said to be indeterminate in the frontal plane.

4) Measure the ECG waves and intervals.
The duration and amplitudes of the waves of the ECG are important in determining whether chamber enlargement is present. When one or more of the cardiac chambers enlarge, the processes of depolarization and/or repolarization may be altered in 1) magnitude of the vectors, 2) direction of the vectors, 3) rate of activation (duration), and 4) sequence of activation. These changes are reflected in the surface electrocardiogram as alterations in 1) the amplitude in the various leads, 2) the direction of the deflections in the various leads (i.e. change in MEA), 3) the width (duration) of the waves in various leads, and 4) the development of certain abnormal patterns of activation (i.e. S waves with RVH). The duration of the various intervals is important to determine if conduction or electrolyte disturbances are present. By convention the first negative deflection, preceding a positive deflection is termed a Q wave and the first positive deflection is called the R wave. A negative deflection occurring after a positive deflection is called an S wave. A second R wave is termed an r' wave, etc.

Management of arrhythmias
Arrhythmias are clinically important because of their ability to compromise cardiac output and oxygen delivery to the body. The level of cardiac performance during an arrhythmia is dependent on the rate, site of origin, and duration of the arrhythmia, as well as the presence of underlying cardiac or systemic diseases that may adversely affect the patient. Thus, the consequences of an arrhythmia may be clinically undetectable, may produce signs of inadequate cardiac output (weakness, fainting, shock), or may lead to the complete collapse of the circulatory system and sudden death.

Depending on the underlying cause of the arrhythmia, administration of antiarrhythmic drugs may not be needed. Metabolic abnormalities (acid/base or electrolyte disturbances, hypoxia) can contribute to arrhythmia formation and should be corrected. Arrhythmias in patients with concurrent congestive heart failure will often resolve spontaneously once the heart failure is successfully treated. Finally, the clinician must be familiar with the actions and potential side effects of the antiarrhythmic drugs, and must carefully weigh the risks and benefits of treatment. Administration of antiarrhythmics is not a benign procedure. Every agent has the possibility to induce further and perhaps more dangerous arrhythmias (pro-arrhythmia).

Ventricular tachyarrhythmias (VPCs, ventricular tachycardia)
1) No therapy may be required if the VPCs are infrequent and the patient is asymptomatic. However Holter monitoring is often required to confirm the true frequency of the arrhythmia.
2) Withdraw or adjust offending drugs (digitalis) if toxicity is suspected.
3) When associated with congestive heart failure, therapy with positive inotropes under close supervision is indicated along with other measures to treat the CHF.
4) Antiarrhythmic therapy is indicated when VPCs are frequent, multifocal, or occur in rapid groups (ventricular tachycardia). The most commonly employed oral antiarrhythmics include mexiletine and sotalol. Amiodarone may be used in select cases.
5) If life threatening ventricular tachycardia develops, intravenous therapy with lidocaine or procainamide is most often used.

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Supraventricular tachyarrhythmias (frequent APCs, atrial tach, atrial fib)

1) No therapy may be required if the APCs are infrequent and the patient is asymptomatic. However Holter monitoring is often required to confirm the true frequency of the arrhythmia.

2) When frequent APCs are observed in patients with congestive heart failure, digitalis or diltiazem therapy can be considered (may be a precursor for atrial fibrillation).

3) Termination of atrial tachycardia may be accomplished by vagal maneuvers, precordial (chest) thump, or control of the ventricular response rate utilizing digoxin, atenolol, diltiazem, or sotalol. The same agents may be useful for preventing recurrence.

4) Atrial fibrillation.
   a. The usual goal in patients with heart disease is to slow the ventricular response rate. This is often achieved by digitalization +/- the addition of diltiazem, atenolol or sotalol if appropriate rate control is not achieved with digoxin alone. Amiodarone may be used in select cases.
   b. Conversion to sinus rhythm is usually only attempted in patients with a reasonable probability of remaining converted (those with minimal underlying heart disease). Oral quinidine, IV procainamide, or electrical defibrillation have been employed. Intravenous administration of diltiazem, amiodarone or sotalol is occasionally effective.
Myocardial disease is the most frequently diagnosed heart disease in the cat. A study from southwestern Virginia published in 2009 identified 16% of apparently healthy cats (16/103) had echocardiographically demonstrable cardiomyopathy. The prevalence of DCM in cats has drastically declined with identification that most cases were attributable to taurine deficiency. Therefore hypertrophic cardiomyopathy (HCM) is now the most commonly identified feline myocardial disease. Cats with HCM may range from one to 16 years of age, with a large percentage ranging from 4 to 7 years. A genetic alteration in cardiac myosin binding protein C has been identified as a cause of familial HCM in some Maine Coon cats. Similarly an alteration in cardiac myosin binding protein C has been found in Ragdolls with familial hypertrophic cardiomyopathy. There are mutations in 11 or more genes producing >1,400 variants in humans with familial HCM, therefore it is likely there are many additional genetic modifications responsible for feline HCM. Other forms of endomyocardial disease described in cats include arrhythmogenic right ventricular cardiomyopathy, restrictive cardiomyopathy, and endocardial fibroelastosis. Many of these forms of myocardial disease in cats are difficult to distinguish from one another clinically or therapeutically and many cases do not fit neatly into any category. Therefore in many instances the objectives of therapy are uniform across myocardial disease and include (1) to treat the underlying cause, if one can be established, (2) to medically manage congestive heart failure, (3) to control arrhythmias, and (4) to treat or prevent thromboembolic complications.

Treatment of asymptomatic cats
Treatment of any form of cardiomyopathy in the asymptomatic patient is controversial and often depends on the severity of underlying echocardiographic changes, the presence and severity of left ventricular outflow tract obstruction, the rate of disease progression (if known), the presence and severity of other underlying systemic diseases, and the likelihood that medications can be administered easily and with good compliance. 1) Atenolol may be administered to try and resolve significant left ventricular outflow tract obstruction. 2) ACE inhibitors may be used in an effort to blunt the rennin-angiotensin-aldosterone system. 3) Anticoagulant therapy may be administered in an effort to prevent thromboembolism.

Treatment of cats with congestive heart failure
Independent of the form of myocardial disease, many of the priorities and agents used to treat heart failure in cats are similar. These often include: 1) Thoracocentesis is performed to remove large volumes of pleural fluid. 2) Furosemide is used to control edema. Additional diuretics may ultimately be required. 3) ACE inhibitors are used to blunt the activation of the renin-angiotensin-aldosterone system. 4) Anticoagulant therapy is administered in an effort to prevent thromboembolism. 5) Depending on the circumstances atenolol may be used to slow the heart rate and to reduce or eliminate dynamic obstruction in cats with hypertrophic obstructive cardiomyopathy. However caution should be exercised in cats that have active congestive heart failure. 6) Alternatively, diltiazem has been suggested to improve filling (positive lusitropic effect) and to decrease the heart rate in cats with HCM. 7) Pimobendan should be considered experimental therapy at this time but in some cases with myocardial dysfunction, presumed low output, and/or significant renal dysfunction we may use it in cats with HCM and heart failure. Contraindications may include significant left ventricular outflow tract obstruction. But interestingly one potential benefit is the PDE inhibitor action of pimobendan may enhance diastolic function.

Treatment of cats with aortic thromboembolism
Many approaches to this difficult problem have been suggested and none is very satisfactory. The site of thrombosis and duration of the event is critical in determining the clinical outcome. Cats with thrombi occluding the renal arteries or with gastrointestinal infarction have an extremely poor prognosis. Although surgical removal of the clot sounds ideal many cats die when surgery is attempted because of underlying heart disease, from anesthetic depression of the heart, or during the washout phase (of toxins, potassium, etc.) if perfusion is reestablished. Thrombolytic therapy may be accomplished with streptokinase or recombinant tissue plasminogen activator (TPA). Aggressive attempts to dissolve emboli using thrombolytic drugs should be reserved for cats with more serious thromboembolic events. Pion, et. al. reported successful thrombolysis, defined as evidence of reperfusion within 36 hours of TPA (Activase, Genentech) treatment, in 50 per cent of cats with spontaneous aortic thromboembolism that were treated with tissue plasminogen activator. Forty-three percent of the cats walked within 48 hours of presentation. However, 50 per cent of the cats died from either reperfusion syndrome, heart failure, or suddenly.

Many cats with saddle thrombi will regain function of the hind limbs, albeit slowly, with conservative therapy. Recovery takes several weeks to months and residual deficits (peripheral neuropathy, muscle contracture) are common. Conservative management consists of pain management, anticoagulant therapy to prevent additional clot formation and therapies aimed at resolving concurrent heart failure. Pain management is one of the most important goals of treating cats with systemic thromboembolism. Butorphanol,
buprenorphine and oxymorphone are used frequently but more aggressive measures, e.g. morphine epidurals, may be required in some cases.

**Prognosis**

The prognosis for cats with HCM is variable often depending on the stage of disease. Cats with minimal hypertrophy and normal left atrial size may live asymptotically for many years without institution of medications. However once myocardial disease of any form has progressed to congestive heart failure there is overall a guarded to poor long-term prognosis. Some cats respond favorably to drug administration and may live several years, however most others die within 6 to 12 months following development of heart failure.
Dilated Cardiomyopathy: Boxers and Dobies, Oh My!
Barret Bulmer, DVM, MS, DACVIM
Tufts Veterinary Emergency Treatment and Specialties
Walpole, WA

The cause(s) of dilated cardiomyopathy (DCM) in dogs is (are) unknown. Some of the proposed causes of DCM include: genetic defect(s), viral infection, microvascular spasm, chemical toxin(s), dietary deficiency, and immune-mediated processes. There appears to be a familial predisposition to the development of DCM in some breeds of dogs, and many investigators suspect a heritable defect in the metabolic processes of myocardial cells. It is quite possible that DCM is not a single disease, and that there are many etiologies. Taurine deficiency has been convincingly shown to be a reversible cause of DCM in cats and is also a suspected cause of DCM in foxes, but is not an important cause of DCM in dogs except in Cocker spaniels. A number of chemical toxins (anthracycline antibiotics, gossypol, monensin) have been shown to cause myocardial failure. There is evidence that Adriamycin exerts at least some of its toxic myocardial effects by inducing histamine and catecholamine-mediated microvascular spasm.

One of the most frustrating aspects of attempts to identify the etiology behind DCM is determining if changes in protein expression are primary or secondary in nature. Up-regulation and down-regulation of proteins responsible for cardiac contraction ($\beta_1$, $\beta_2$, and $\alpha$ receptors), ventricular relaxation (SERCA2, phospholamban) and energy production (carnitine transport, creatine kinase) occur to equivalent degrees in volume overload, pressure overload, and cardiomyopathy. “In this respect the intracellular biochemical specificity of the response of the myocyte to a chronic insult appears to be relatively restricted. The foremost question remains, which, if any, are the true pathogenic alterations and which are cellular adaptations.”

Dilated cardiomyopathy is a diagnosis arrived at by a process of exclusion. Causes such as infectious myocarditis, chronic volume overload (A-V fistula, valvular insufficiency), heartworm disease (and other causes of cor pulmonale), pericardial disorders, and toxic cardiomyopathy (doxorubicin) must be ruled out before a diagnosis of dilated cardiomyopathy is offered. A provisional diagnosis can be based on the history, physical findings, and typical radiographic and electrocardiographic changes, but echocardiographic evaluation is necessary to establish the diagnosis with certainty.

Most dogs with DCM have an abnormal electrocardiogram, although the changes may be subtle. Dogs may display criteria for left ventricular or left atrial enlargement. There is also a high prevalence of cardiac rhythm disturbances in dogs with DCM. Atrial fibrillation, ventricular premature complexes (VPCs) and ventricular tachycardia are commonly identified. Ventricular rhythm disturbances are most common in Boxer dogs and Doberman pinschers, both of which suffer a high rate of sudden death associated with the development of DCM. Using 24-hour ambulatory EGG (Holter) recordings, 81 percent of asymptomatic Doberman Pinschers with DCM had complex ventricular arrhythmias and almost 30 percent had sustained or non-sustained ventricular tachycardia. The prevalence of ventricular tachycardia and VPCs in Boxer dogs is similar to or greater than that observed in Dobermans.

Radiographic changes in dogs with moderate to severe disease almost always include biventricular or left ventricular and left atrial enlargement as well as evidence of right or left sided heart failure. Pleural effusion is common in dogs with biventricular failure, obscuring thoracic detail and preventing critical evaluation of heart size. Pulmonary edema is present in many dogs with DCM, and is often particularly severe in Boxers and Doberman Pinschers.

Echocardiographic alterations often include larger than normal end-systolic and end-diastolic dimensions of the left ventricle. The interventricular septum and ventricular free walls are hypokinetic, often thinner than normal in diastole, and they fail to thicken normally in systole. The left atrial dimension is increased, and the left atrial to aortic dimension ratio is increased. Fractional shortening, the percent change in short-axis diameter of the contracting left ventricle, is usually markedly decreased. The distance between the interventricular septum and the mitral valve at its maximal opening point in early diastole (EPSS) is increased as a reflection of a reduced ejection fraction.

Breed-specific idiosyncrasies.

Most dogs with dilated cardiomyopathy (classic cardiomyopathy) present with signs of right, left, or biventricular failure, in atrial fibrillation, and with marked weight loss and muscle wasting. In affected Boxers, approximately 20% are presented in predominately left-sided failure, 40% are presented for syncope or collapse secondary to a rhythm disturbance, and 40% are asymptomatic but have rhythm disturbances (primarily ventricular arrhythmias). Doberman pinschers usually present in severe left-sided heart failure, have a slightly lower incidence of atrial fibrillation than other breeds, have a higher incidence of ventricular arrhythmia, and experience a higher incidence of syncope and collapse.

Therapy
Treatment of heart failure in dogs with dilated cardiomyopathy often mimics that of dogs with valvular heart disease and heart failure. Diuretics help control congestion, angiotensin converting enzyme inhibitors are used to blunt activation of the renin angiotensin system, and positive inotropes (pimobendan) are used to enhance systolic performance. Dogs with dilated cardiomyopathy often
require antiarrhythmics to manage ventricular or supraventricular arrhythmias. Caution must be exercised with many of the antiarrhythmics because of their negative inotropic properties.

**Prognosis**
Dogs with echocardiographic evidence of dilated cardiomyopathy, but with no clinical signs of congestive heart failure, may live for a very long period of time. However, most affected dogs with congestive heart failure die within 6 months. Some very ill dogs improve to a remarkable degree with treatment and live comfortably for months or years. Others dogs do not survive the initial 48 hours of hospitalization.
The primary objectives of the cardiovascular evaluation for animals with congenital heart disease are to define the nature and severity of the anatomic defect present. Familiarity with the available therapeutic options, their efficacy and limitations is necessary before an accurate prognosis can be offered to the owner.

Acyanotic congenital heart defects: Left to right shunts

Patent ductus arteriosus (PDA) including right to left shunting lesions

In the fetus the ductus arteriosus serves to shunt the majority of the right ventricular output away from the non-functioning lungs. Expansion of the lungs, increased oxygen concentrations and removal of the umbilical circulation at the time of birth promotes ductal closure. Failure of ductal closure usually results in a left to right shunt from the descending aorta to the pulmonary artery with an excess volume load placed on the pulmonary arteries and veins, left atrium, left ventricle and aortic arch. Histology of the patent ductus reveals a wall structure resembling that of the aorta rather than that of a normal ductus. In the presence of a very large, wide PDA the magnitude and direction of shunted blood is determined by the relative resistance of the pulmonary and systemic circulations. In these dogs the elevated pulmonary vascular resistance present at birth does not fall normally and results in right to left shunting or bidirectional shunting. On rare occasion pulmonary hypertension develops later in life thereby truly reversing the direction of the shunt (Eisenmenger’s physiology).

Clinical features

1. Historically the most common congenital heart defect in dogs although the recent popularity of large breed dogs has resulted in increased prevalence of SAS. PDA is much less common in cats.
2. Females are over-represented.
3. Physical examination findings include:
   a. A continuous “machinery” murmur that is heard best at the left heart base. The continuous murmur may be confined to the heart base while a systolic murmur of mitral insufficiency is ausculted over the left apical region.
   b. Bounding (or waterhammer) pulses are frequently identified because of the increased systolic and decreased diastolic aortic pressures (widened pulse pressure).
   c. Common clinical signs include stunted growth or evidence of left sided heart failure (dyspnea, tachypnea, coughing, exercise intolerance.)
   d. PDA with pulmonary hypertension has no murmur but may have a split S2, differential cyanosis, and hindleg weakness. These dogs often display “differential cyanosis” where the hindlimbs are affected while the forelimbs are normal. This develops because of the communication of the pulmonary artery with the descending aorta.
4. Electrocardiographic findings:
   a. Variable but often marked left ventricular enlargement pattern, possible left atrial enlargement and secondary ST segment changes associated with hypoxia.
   b. Advanced cases may show supraventricular tachyarrhythmias (APCs, A fib) or less frequently ventricular arrhythmias.
   c. A right ventricular enlargement pattern is almost always evident in cases of right to left shunting with pulmonary hypertension.
5. Thoracic radiography:
   a. Enlargement of the left atrium, left ventricle, aortic arch, main pulmonary artery along with pulmonary vascular overcirculation (enlargement of both pulmonary arteries and veins).
   b. Evidence of left sided heart failure may be present.
   c. Dogs with right to left shunting often display pulmonary vascular undercirculation (hypovascularity of pulmonary arteries and veins), a prominent right heart pattern, dilation of the main pulmonary artery and localized dilation of the proximal aorta.
6. Echocardiography: Serves to evaluate the severity of volume overload as reflected by changes in the left heart chamber dimensions, detect other coexisting congenital heart defects, and assess myocardial function.
7. Prognosis:
   a. In dogs with left to right shunts the prognosis is excellent with surgical or transcatheter closure of the defect prior to the development of left-sided heart failure. Without correction puppies with large shunts may die before four weeks of age, dogs with intermediate sized shunts may live for several years although the majority will be dead by 2 years of age. Dogs with small shunts (uncommon) may live normal lives.
   b. In dogs with right to left shunts the prognosis is guarded. Some dogs may survive for long periods of time with exercise restriction and periodic phlebotomy or agents utilized to decrease red blood cell production.
8. Treatment: Ideally involves surgical correction of left to right shunts via thoracotomy or less invasive embolization procedures prior to the development of clinical signs. In cases of left to right shunts with congestive heart failure stabilization is achieved with standard medical therapy followed by closure. Surgery is contraindicated in dogs with right to left PDAs and instead efforts are aimed at preventing hyperviscosity via periodic phlebotomy.

Acyanotic congenital heart defects: Obstructive malformations
Obstructive lesions produce their effects by impeding normal blood flow and causing an increased pressure proximal to the obstruction. The two clinical syndromes identified in small animals include pulmonic stenosis and aortic stenosis. Four anatomic types of obstruction can occur at each location and include: supravalvular, subvalvular, valvular and infundibular. All result in similar degrees of functional impairment but their distinction is important if surgical correction is contemplated.

Pulmonic stenosis
Pathology of the pulmonic valves typically includes variable thickening of cusps and fusion of the cusps at their commissures. Pulmonic stenosis may occur as an isolated lesion or may be combined with other complex defects of the conotruncal septum (Tetralogy of Fallot). The resistance to ejection of blood from the right ventricle induced by the stenotic valve produces elevated RV systolic pressures, right ventricular concentric hypertrophy and in some cases increased right atrial pressure. A post-stenotic dilatation is usually present in the main pulmonary artery.

Clinical features
1. The second or third (because of the increased prevalence of SAS) most commonly diagnosed congenital heart defect in dogs. Uncommon in cats.
2. Physical examination findings include:
   a. Systolic, ejection (crescendo-decrescendo) murmur heard best over the left heart base. A split second heart sound may be obscured by the murmur.
   b. Arterial pulses are usually normal unless severe heart failure is present.
   c. Dogs may be asymptomatic, exhibit exercise intolerance, or in severe cases may exhibit dyspnea and cyanosis from low cardiac output. Syncopal episodes with exercise are occasionally reported. Signs of right-sided heart failure may be present in severe cases.
3. Electrocardiographic findings:
   a. A right ventricular enlargement pattern is usually evident while the rhythm is usually normal. In severe cases complicated by tricuspid dysplasia/insufficiency supraventricular tachyarrhythmias may be identified.
4. Thoracic radiography: Characteristic findings include right ventricular enlargement and dilation of the main pulmonary arterial segment. The pulmonary vasculature is usually normal.
5. Echocardiography: Serves to evaluate the extent of hypertrophy of the papillary muscles, septum and ventricular free wall of the right ventricle. The site of obstruction (valvular, subvalvular, etc.) may be identified via two-dimensional echocardiography and Doppler studies can evaluate the integrity of the tricuspid valve. Spectral Doppler can measure the peak blood flow velocity through the stenotic area and the modified Bernoulli equation (4V²) can estimate the pressure gradient and hence the severity of the stenosis.
6. Prognosis: Many dogs with mild disease appear to do well without therapy while most agree that dogs with a pressure gradient over 80 - 100 mm Hg have a more guarded prognosis without therapy. Dogs with gradients between 40 and 80 mm Hg are more difficult to characterize.
7. Therapy: Balloon valvuloplasty is effective at reducing the pressure gradient significantly in approximately 70% - 85% of cases. In the presence of congestive heart failure exercise restriction and medical therapy are employed followed by consideration for surgery.

Subvalvular aortic stenosis (SAS)
SAS may be the most commonly identified congenital lesion in some regions because of the vast popularity of Golden Retrievers and other large breed dogs predisposed to SAS. In dogs a subvalvular fibrous ring or band partially or completely encircles the left ventricular outflow tract. Small nodules may also occur on the aortic valve cusps. Valvular obstruction results in elevated left ventricular systolic pressures, concentric hypertrophy of the left ventricle and post-stenotic dilatation of the ascending aorta. Increased oxygen requirements of the concentrically hypertrophied left ventricle and disturbances in coronary blood flow may lead to
myocardial ischemia. Histologically, arteriosclerosis of the small coronary arteries, fibrosis, necrosis, and calcification of the myocardium may be observed. SAS usually occurs as an isolated lesion although mitral valve dysplasia has been reported to occur concurrently.

Clinical features

1. A common defect in dogs although it is infrequently recognized in other species.
2. Physical examination findings:
   a. A systolic ejection murmur (crescendo-decrescendo) at the left heart base. It frequently radiates to the carotid arteries at the thoracic inlet and may radiate to the right hemithorax.
   b. Pulses may be weak and late rising due to retarded ventricular ejection.
   c. Young dogs are frequently asymptomatic but may have history of fatigue, dyspnea, or syncope. Sudden death (presumably from ventricular arrhythmias) is one of the most commonly reported events in young dogs with severe SAS.
   d. Arrhythmias (usually ventricular) may be present.
3. Electrocardiographic findings: The ECG may be normal in mild cases or it may display a left ventricular enlargement pattern in more severe cases. ST segment depression may be present due to myocardial hypoxia and arrhythmias (usually ventricular) are common.
4. Thoracic radiography: frequently unremarkable because the left ventricle hypertrophies concentrically. Findings may include left ventricular enlargement, post-stenotic dilatation of the aorta with variable left atrial enlargement. The pulmonary vasculature is normal unless left sided heart failure has developed.
5. Echocardiography: Serves to evaluate the severity of the left ventricular hypertrophy, the area of the obstruction may be directly visualized by two-dimensional echocardiography, and Doppler evaluation can categorize the severity of the stenosis via the modified Bernoulli equation. Myocardial fibrosis, presumably due to ischemia, may be identified as hyperechoic areas within the myocardium. Echocardiography also helps to evaluate for the presence of combined congenital defects. Differentiating normal dogs from dogs with very mild SAS can be difficult even with echocardiography because the subvalvular lesion may be so discrete.
6. Prognosis: The prognosis is guarded in cases of severe SAS (pressure gradient over 80 mm Hg). Owners should be made aware of the possibility of sudden death. SAS appears to be the one cardiac malformation that predisposes dogs to the development of bacterial endocarditis and standard antibiotic administration should be instituted whenever dogs with SAS undergo elective surgical procedures. Congestive heart failure may occur when mitral dysplasia is concurrently present or if myocardial failure develops after long-standing SAS.
7. Treatment: To date surgical resection of the stenotic lesion has not decreased the incidence of sudden death. Balloon valvuloplasty has also proved unrewarding in most cases as the obstruction tends to recur shortly after the valvuloplasty. A cutting balloon technique has started to be employed more recently. Current medical management may include administration of beta-blocking drugs to decrease the heart rate and cardiac contractility thereby decreasing myocardial oxygen demands and hopefully the risk of sudden death. The effectiveness of this therapy is unknown. Arrhythmias should be appropriately treated if present.