Cardiac Auscultation

John D. Bonagura, DVM, MS, Diplomate ACVIM (Cardiology, Internal Medicine)
The Ohio State University

Auscultation of the heart remains an important examination for the detection of cardiovascular disease. The auscultatory exam is expedient and cost effective. When completed by an experienced clinician, auscultation carries a high predictive value for identification of many—though not all—serious heart diseases. Definitive diagnosis may be possible by auscultation, as when classic murmurs of patent ductus arteriosus or mitral regurgitation are identified. Often the combination of signalment, cardiac and pulmonary auscultation, and general physical examination point to a tentative cardiac diagnosis. This presumption can then be confirmed, refined, or refuted by echocardiography (for valvular disease, pericardial disease, cardiomyopathy, or shunts) or by electrocardiography (for arrhythmias).

The **essential abnormalities** of cardiac auscultation include: abnormal heart rate (bradycardia, tachycardia); irregular cardiac cadence or rhythm; abnormal intensity of heart sounds; extra heart sounds; absent sounds; split sounds; cardiac murmurs; and pericardial friction rubs.[1-5]

TECHNIQUES FOR CARDIAC AUSCULTATION

The choice of a stethoscope is very personal. The traditional stethoscope has an operator selected diaphragm and bell. Some stethoscope designs allow a single chest piece to function as both a bell and a diaphragm. While acoustically superior in some ways, the single chest piece design of some adult stethoscopes is too large for cats and small dogs. However, some of the newer models combine both "adult" and "pediatric" single chest piece stethoscopes into one rotating head. Amplified stethoscopes generally are not recommended because of the potential for blooming artifacts and distortion; however, they can be useful for those with a hearing impairment. Some of the newer models, such as the Welch Allen Meditron®, are accurate and also can provide an instructive computer printout of recorded sounds along with an ECG for timing purposes.

The clinician must understand that many heart sounds fall below the frequency-threshold limit; accordingly, careful auscultation is necessary to detect the vibrations that are audible. The stethoscope tube length should not be excessively long, the binaurals and ear pieces should be directed so that their orientation is rostral and aligned with the ear canals, and earpieces should be inserted snugly but comfortably to obtain an airtight seal. The flat diaphragm chest piece is applied gently but firmly to the chest to accentuate higher frequency sounds such as normal heart and breath sounds and most cardiac murmurs. The bell, which is applied lightly to achieve an airtight seal, enhances detection of lower frequency sounds such as the third and fourth heart sounds. The bell is also useful for detection of the more uncommon diastolic murmurs.

Conditions for auscultation are often overlooked but substantially impact the results of the examination. The room must be quiet, the patient gently restrained, and the examiner relaxed. It is preferable for the animal to stand in order to locate the valve areas accurately. A cat can be gently restrained with one hand under the abdomen; this encourages the cat to stand on the forelimbs. The patient must be calm and ventilation and purring controlled if possible. Gently holding the mouth closed, whistling, or briefly obstructing the nares are effective maneuvers for reducing ventilation artifacts. Showing the feline patient water in a sink, picking up the cat, or gently pressing over the larynx may reduce the degree of purring. Sound artifacts may be misinterpreted as abnormal heart or lung sounds. These include synchronous ventilation and panting (which can mimic murmurs); skin twitching (creating extra heart sounds); and friction from rubbing the chest piece across hair (sounding like pulmonary crackles or "rales"). Excessive pressure on the chest can distort the thorax of small animals and create murmurs.

The entire precordium is examined, with particular attention directed to the cardiac valve areas.[4, 5] While the exact anatomic location of the valve areas depends on the species, chest conformation, and size of the heart, a common relative location is found from cranial to caudal: pulmonic–aortic–tricuspid–mitral with the tricuspid valve on the right. A useful clinical pointer is to first palpate the left apex beat where mitral sounds radiate and the first heart sound is best heard. Find other valve areas from this point. The aortic valve area is located craniodorsal to the left apex and the second heart sound is best heard there. Once the aortic second sound is identified, the stethoscope can be moved one interspace cranial and slightly ventrad (over the pulmonary valve area). The pulmonary artery extends dorsally from the pulmonic valve. The tricuspid valve is over the right hemithorax, cranial to the mitral area, and covers a relatively wide area. The LV outlet is in the center of the heart and aortic murmurs usually radiate well to each hemithorax. Cardiac apex and cardiac base are commonly used expressions to designate the regions ventral and dorsal to the atrioventricular groove. The mitral and tricuspid valvular sounds often radiate ventrally towards the apex. Murmurs originating at the semilunar valves and great arteries are detected best over the base. The use of "valve areas" in cats is less precise. The author typically defines auscultation areas as caudal (chest piece centered over the apex beat), cranial (1–2 intercostal spaces cranial to the apex), left sternal and right sternal. Most apical murmurs in cats indicate pathology; many cranial murmurs in cats are functional.

TRANSIENT CARDIOVASCULAR SOUNDS

Transient sounds are vibrations of short duration because their genesis depends on abrupt changes in blood flow and pressure CV transient sounds, as well as cardiac murmurs, are timed relative to the first (S_1) and second (S_2) heart sounds. Normal heart sounds are dictated by electrical activity of the heart and the cardiac cycle. Following initial activation of the ventricles, the first sound is heard indicating the beginning of mechanical systole. At the end of ventricular ejection, the second sound is generated. The longer period between the second and ensuing first sound represents diastole to the clinician. Any diastolic transient sounds are abnormal in small animals. Both S_1 and S_2 are relatively high-frequency sounds. The first sound is associated with the closure of the AV valves and is slightly longer and lower pitched than the second sound. The second sound is caused by near-synchronous closure of the aortic and pulmonic valves. These sounds may be abnormal in certain conditions. For example, pericardial disease, pleural effusion, or myocardial failure from dilated cardiomyopathy can decrease the intensity of the first heart sound. Arrhythmias typically lead to variable intensity heart sounds, especially when ventricular filling varies across cardiac cycles. Thus, with atrial fibrillation and generally with ventricular tachycardia, the heart sounds will vary in intensity. Both heart sounds tend to be loud in healthy animals under high sympathetic drive or those with an ectomorphic conformation. The second heart sound may be closely split in healthy dogs as resting heart rates,[7] but this is not commonly detected. Pathologic splitting of the sounds may be detected with atrial septal defect or with asynchronous ventricular activation. The latter is typical of ventricular ectopia or bundle branch block. The finding of split sounds during a regular tachycardia more likely indicates an arrhythmia of ventricular, rather than supraventricular, origin. Severe pulmonary hypertension increases the intensity of S_2 or leads to audible splitting of this sound should left and RV ejection times become very disparate.

Figure 1. Transient Heart Sounds

Clockwise from upper left: First and second heart sounds; Split second heart sound; Fourth (atrial) sound and third sound; Third sound or ventricular gallop.
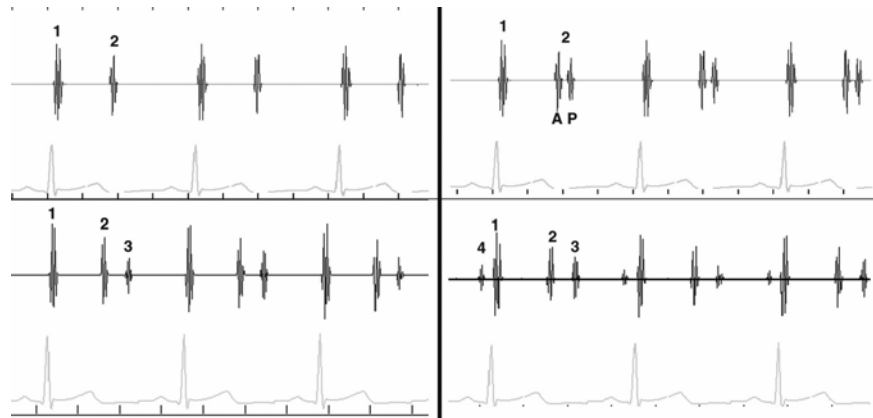
Figure 2. Transient Heart Sounds

Upper panel: Mid-systolic click (c) in a dog with mitral valve disease. Note the variation in the click intensity and timing. Lower panel: Summation (S_3 and S_4) gallop (G) in a cat with dilated cardiomyopathy and heart failure. First and second heart sounds also are indicated.
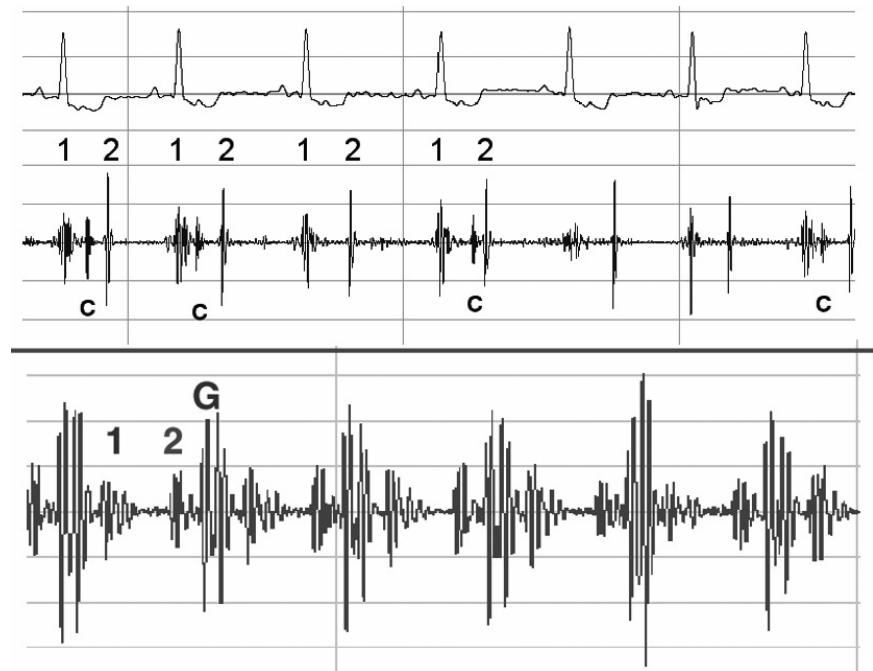
The third and fourth sounds are lower-frequency vibrations associated with termination of rapid ventricular filling and with atrial contraction, respectively. These sounds indicate ventricular diastolic dysfunction when detected in dogs or cats. A third sound is typical of a ventricle with poor compliance that is filling under high venous pressures. Examples include mitral regurgitation or cardiomyopathy in a patient with left-sided CHF. An atrial sound is typical of impaired ventricular relaxation. This sound is often detected in cats with left ventricular hypertrophy, as with hypertrophic cardiomyopathy. It also seems that some older, stressed cats manifest atrial gallops even when there is minimal if any ventricular disease. If both third and fourth sounds are present and the heart rate is rapid, the gallops may be superimposed, producing a summation gallop. Of importance is the

observation that gallops may be the **only** auscultatory abnormality of cardiomyopathy and that these transient sounds can vary with heart rate and filling pressures.

Clicks are high-pitched, systolic transient sounds. Mid-systolic clicks are not uncommon in dogs with mitral or tricuspid valve disease and are probably indicative of prolapse from abnormal (lax) chordae tendineae or valve redundancy. [8,9] Isolated clicks suggest mild disease though clicks are often superimposed over murmurs in dogs with advanced mitral and tricuspid valvular endocardiosis. Ejection clicks are infrequently detected with valvular pulmonic stenosis and with severe pulmonary hypertension. Thus, the differential diagnosis for "extra sounds" includes gallops (atrial and ventricular), split sounds, clicks, and premature beats.

Arrhythmias represent another reason for extra or absent heart sounds. Typically with premature beats the examiner detects an early S_1, followed by a soft (or absent) S_2, and then a pause. Often the premature sound is nearly unapparent, and it's the post-extrasystolic pause that draws attention. In cases of incomplete atrioventricular block, the pauses following blocked P-waves are obvious; differential diagnosis includes post-extrasystolic pauses and sinus arrhythmia in dogs. The latter tends to be cyclical with cardio acceleration usually tied to inspiration and the arrhythmia often abolished following exercise. An ECG quickly answers the question if there is doubt about the cardiac rhythm.

The experienced auscultator learns to be vigilant for abnormal transient sounds. Although a cardiac murmur is often considered the hallmark finding of heart disease, some CV conditions are not be associated with a consistent heart murmur. For example, cardiac murmurs may be inconsistent or absent in tetralogy of Fallot with pulmonary artery hypoplasia and polycythemia, heartworm disease, pulmonary hypertension, systemic hypertension, cardiomyopathy, and pericardial diseases. In the latter, distant heart sounds are most common finding while the diastolic 'knock' of constrictive pericardial disease or the friction rub of pericarditis are rarely detected in small animals. Recurrent or sustained arrhythmias will also be revealed during auscultation.

CARDIAC MURMURS

Cardiac murmurs are prolonged audible vibrations. Murmurs frequently indicate heart disease; however, many heart murmurs are innocent or functional (i.e., the heart is structurally normal). Murmurs are often associated with high velocity blood flow (typically > 1.6 m/sec) or with fluid vibrations that develop about disturbed or turbulent flow. Turbulence tends to develop when the velocity of flow increases or the viscosity of blood decreases. Turbulence is also more likely when blood enters a larger vessel or chamber. Common clinical causes of cardiac murmurs include: 1) sympathetic stimulation as with exercise, fever, or hyperthyroidism (which can increase the velocity of ejection into the great vessels and can also lead to dynamic ventricular outflow tract obstruction); 2) anemia (which decreases viscosity of blood and is also associated with increased sympathetic drive); 3) flow into dilated vessels (especially those that abruptly change in diameter); 4) increased flow volume across otherwise normal heart valves; and 5) abnormal paths of blood flow from high to low pressure. Examples of high velocity flow across otherwise normal heart valves include atrial and ventricular septal defects (increased flow across the pulmonary valve) and complete AV block (increased flow across the aortic valve due to increased stroke volume and lower aortic diastolic pressure). Pathologic causes of increased velocity flow include stenotic valves, incompetent valves, ventricular septal defects, and aortic to pulmonary shunts (PDA). Heart murmurs are often detected in the older cat with a dilated aorta related to either hypertension or degeneration (aortic redundancy). Dynamic right ventricular (RV) outflow tract obstruction has been proposed as another cause of ejection murmurs in stressed cats. Hypovolemia can also predispose to reversible dynamic outflow tract obstruction and murmur development.

The **description of cardiac murmurs** should include timing, intensity, point of maximal intensity (PMI), radiation, pitch and quality. The **timing** of the murmur is systolic, diastolic, continuous, or to-and-fro (systolic–diastolic). The **intensity** of the murmur is arbitrarily graded on a 1–6 scale; we use the following convention:

- **Grade 1** = a very soft, localized murmur detected only in a quiet room after minutes of intense listening.
- **Grade 2** = a soft murmur, heard immediately, localized to a single valve area.
- **Grade 3** = a moderate intensity murmur that is evident at more than one location.
- **Grade 4** = a moderate intensity to loud murmur; radiates well; but a consistent precordial thrill is not present.
- **Grade 5** = a loud murmur accompanied by a palpable precordial thrill.
- **Grade 6** = a loud murmur with a precordial thrill, audible when the stethoscope is removed from the thorax.

The **point of maximal intensity** is communicated by indicating the location, valve area, intercostal space where the murmur is loudest. A murmur usually projects from the PMI in the direction of abnormal blood flow. Murmurs can also radiate through solid structures (e.g., down to the apex). The radiation of a loud cardiac murmur can be extensive. Pitch, quality, and configuration pertain to the frequency and subjective assessment of the murmur by the examiner. Murmurs consisting of one fundamental frequency with overtones are described as "musical**,"** whereas murmurs of mixed frequencies are typically noted to be "harsh." Most murmurs are of mixed frequency. Timing within the cardiac cycle, development, and murmur intensity create the impression of a murmur's "shape" or the vibrations that might be recorded graphically by a phonocardiogram. Ejection murmurs are typically crescendo-decrescendo whereas regurgitant murmurs are longer (holosystolic or pansystolic) and plateau shaped.

Functional murmurs are not associated with any obvious cardiac pathology. Innocent murmurs in puppies should abate by the rabies vaccination. Physiologic murmurs are functional murmurs related to altered physiologic state and include those of the athletic heart[10], anemia, fever, high adrenergic tone, peripheral vasodilation, hyperthyroidism, and bradycardia. Most physiologic murmurs are ejection in timing and detected best over the aortic and pulmonic valves and the great vessels (at the left dorsocranial cardiac base; see Figure 3). Systolic murmurs heard over the right and left cranial sternal borders are common in cats, and probably represent functional murmurs from flow into a dilated aorta or due to dynamic RV outflow tract obstruction.

Figure 3. Systolic heart murmurs in dogs
Upper panel: ejection murmur (EM) in a healthy
dog. The murmur was physiologic. Middle: Loud
ejection murmur in a dog with moderately-severe
subaortic stenosis. Notice that the murmur ends
before the second heart sound (2). Lower: variable
intensity murmur of mitral regurgitation in a dog
with mitral valvular endocardiosis.
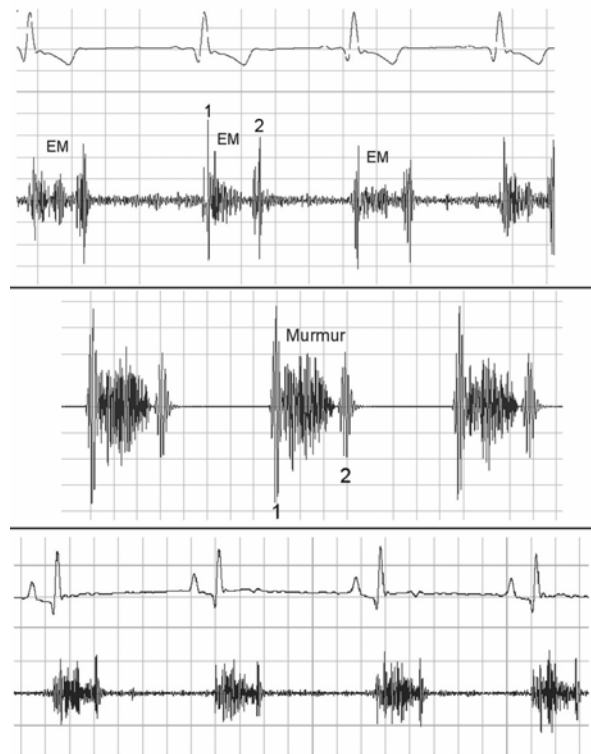
 Mitral regurgitation is one of the most common flow disturbances and develops from malfunction of any
portion of the mitral apparatus. [2,4,5,8,9,11,12] Causes include: congenital mitral dysplasia, degenerative valve disease
(endocardiosis or prolapse) in the dog, bacterial endocarditis, myocardial disease, redundancy or rupture of a
chorda tendineae (in the dog), and causes of LV dilatation or hypertrophy, such as cardiomyopathy,
hyperthyroidism, systemic hypertension, and PDA. This murmur is prominent over the mitral valve area and
transmits prominently down to the left apex where it is often loudest (near the left sternal edge in cats). MR
murmurs radiate both dorsally and to the right (usually one grade softer on the right hemithorax). The MR
murmur can be decrescendo in peracute leakage (from equilibration of LV-LA pressures) or in mild cases (as the
regurgitant orifice closes in late systole). The murmur can be very soft or even absent in **hypotensive** patients;
alternatively, hypertension can increase the intensity. The musical systolic "whoop" is a striking frequency
phenomenon in some dogs. Progressive increase in the intensity of the first heart sound is a unique feature of MR
in dogs with endocardiosis probably indicating cardiac dilatation with maintenance of left ventricular
contractility; furthermore, in typical endocardiosis, the intensity of the MR murmur increases with the degree of
valvular incompetency (assuming normal ABP). The intensity of the murmur in other causes of MR is not as
reliably correlated to the severity. Cats with hypertrophic cardiomyopathy often have murmurs related to MR.[13]
The murmur may have a labile nature related to dynamic LV outflow tract obstruction and systolic anterior
motion of the mitral valve.

      Tricuspid regurgitation is a common cardiac murmur. Much of what was stated concerning mitral
regurgitation is applicable here. Causes include valve malformation, endocardiosis, valve degeneration, RV
enlargement (pulmonic valve stenosis, right sided cardiomyopathy, chronic bradycardia, pulmonary
hypertension), dirofilariasis, and rarely tricuspid endocarditis. The PMI of this murmur is the tricuspid valve area

and dorsal radiation is typical. A very loud murmur in older dogs is typical of endocardiosis complicated by pulmonary hypertension. The following support concurrent TR in the setting of MR: a prominent jugular pulse, right precordial thrill, different frequency murmur than that heard at the left side, or right sided CHF.

Ventricular septal defect is probably the most common congenital heart malformation of cats, and also occurs with some frequency in dogs. A large nonrestrictive VSD (i.e., no substantial pressure difference between the ventricles) is also part of the tetralogy of Fallot. When the defect communicates the LV outlet with the paramembranous or the RV inlet septum, the murmur is loudest just below the tricuspid valve, along the right sternal border. When the defect communicates the LV outlet with the RV outlet septum (a subarterial or supracristal defect), the murmur may be most prominent over the craniodorsal left cardiac base. If the aortic valve has prolapsed into the ventricular septal defect, there may be a diastolic murmur of aortic regurgitation.

Aortic stenosis is most common in dogs as a congenital, subvalvular, fibrous obstruction in the LV outflow tract.[2, 14] The lesion may extend to involve the base of the aortic valve. The valve may be both stenotic and mildly incompetent. Other causes of LV outflow obstruction are hypertrophic cardiomyopathy or mitral valve malformation; these conditions can cause dynamic outflow obstruction. Bacterial endocarditis of the aortic valve generally causes aortic regurgitation before the vegetation is large enough to obstruct the valve. Cases of isolated, aortic valvular stenosis are seen sporadically. The murmur of AS is flow dependent, crescendo-decrescendo in configuration, and like most ejection murmurs, becomes louder following exercise, inotropic stimulation, a ventricular premature beat, and increases in venous return. The intensity of the murmur also depends on the tightness of obstruction and the murmur peaks late in systole when the lesion is severe. When intense, this murmur may be loudest over the right dorsal cardiac base owing to radiation of the murmur up the ascending aorta. Often the murmur of subaortic stenosis projects to the left apex, making it difficult to distinguish from mitral regurgitation. The murmur tends to radiate up the carotids and even up to the head. A diastolic murmur of aortic regurgitation may develop leading to a to-and-fro murmur of AS/AR.

Pulmonic valve stenosis is a congenital malformation of the valve characterized by valve thickening, varying degrees of leaflet fusion, hypoplasia, and thickening at the valve base. It is a common malformation of dogs (but uncommon in cats). The systolic crescendo-decrescendo murmur is heard best over the pulmonic valve (left 2nd–3rd ICS) and radiates prominently in a dorsal direction into the post-stenotic dilatation of the main pulmonary artery. Thus, the murmur tends to be heard very well at the dorsal, left, cardiac base. Valvular PS also may be associated with an early systolic ejection click (fused, doming valve) or a murmur of TR caused by secondary RV enlargement. Tight stenosis is generally associated with a louder and later-peaking ejection murmur. Following balloon catheter valvuloplasty there may be sufficient pulmonary insufficiency to create an audible diastolic murmur.

Aortic regurgitation is the most important diastolic cardiac murmur (Figure 4). Causes include bacterial endocarditis, congenital aortic valve disease, prolapse of an aortic valve cusp into a subaortic ventricular septal defect, and aortic root dilatation in aged cats. This murmur is a long, diastolic, decrescendo murmur heard best over the aortic valve at the left hemithorax. The murmur is also well heard at the right cardiac base. Compared to AR, other diastolic murmurs are uncommon in dogs and cats. The soft, low-pitched rumble of congenital mitral or tricuspid stenosis is very rare (as are these conditions). There is often concurrent valvular regurgitation, which may lead to a systolic murmur.

Patent ductus arteriosus is the most important cause of a continuous murmur and is described as "distant" and "machinery" (like a machine shop) in quality.[15] The murmur must be carefully located on the dorso-cranial left base, over the main pulmonary artery (which is the sink for the shunt). Often there is concurrent mitral regurgitation (due to LV dilatation), which may be responsible for a systolic murmur over the left apex. The stethoscope should be "inched" up and back to the PMI of the continuous murmur at the left base. Continuous

murmurs (bruits) may also be detected over congenital or acquired arteriovenous fistulas, including those associated with thyroid carcinomas or limb injuries.

Figure 4. Cardiac murmurs
Upper panel: Long diastolic murmur of aortic regurgitation. The murmur begins after the second heart sound (2). Lower: Continuous murmur recorded from a dog with patent ductus arteriosus. The murmur peaks about the time of the second heart sound.
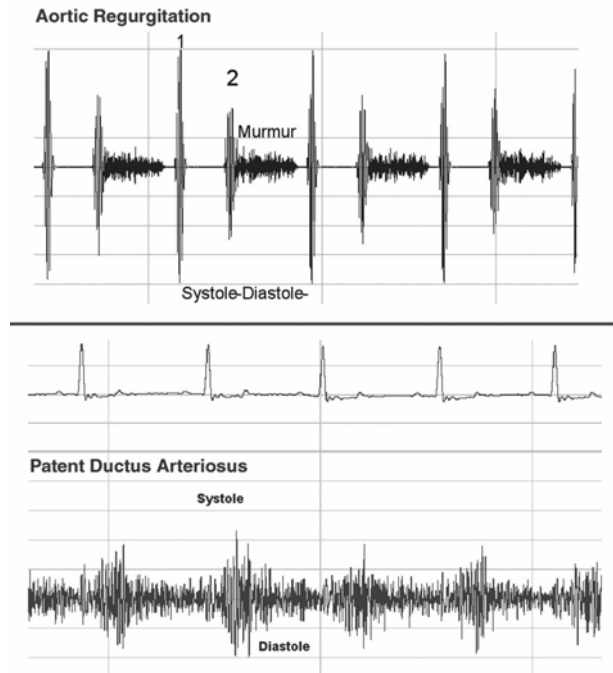
VERIFICATION OF FLOW DISTURBANCES

The identification of flow disturbance leading to murmurs can generally be made through echocardiographic imaging and Doppler techniques. Currently Doppler studies represent the gold standard for diagnosis of valvular lesions and flow disturbances. However, even these sensitive studies are not without controversy. In cases of trivial SAS, where this is no obvious imaging lesion, the diagnosis often hinges on pulsed wave Doppler detection of higher than "normal" outflow velocity. While the velocity measurements are accurate, the significance of a specific value is often subject to debate. It is likely that many dogs with physiological murmurs are labeled as "mild AS" based on these studies. Similarly, the detection of valvular insufficiency by color Doppler techniques can be confusing. Undoubtedly Doppler can detect the earliest signs of subclinical disease as in early mitral regurgitation.[16] However, the clinical significance of valvular regurgitation can be uncertain, especially when valves such as the tricuspid valve–which normally leak–are interrogated. Conversely, Doppler studies are invaluable in deciphering heart murmurs in cats. In about 20–25% of cases we examine, systolic murmurs are either functional or not associated with any substantial pathology. This allows the "cardiac case" to be reclassified and prevents unnecessary therapy.

RESPIRATORY SOUNDS

Auscultation of breath sounds is an integral part of the physical examination.[17] Some key features of pulmonary auscultation are summarized below.

    Normal respiratory sounds include vesicular and bronchovesicular sounds, bronchial breathing, and tracheal sounds (panting). Normal sounds may become accentuated or decreased in diseases of the thorax or in CHF. An increase in bronchial sounds is often detected as a nonspecific finding of pulmonary disease including

pulmonary edema. Decreased sounds may indicate pleural fluid (ventral fluid line) or air. Displaced sounds may indicate a mass lesion or diaphragmatic hernia. Normal sounds and abnormal (adventitious) sounds may only become evident after enforcing deeper breathing which can be encouraged by closing the mouth and holding off one nostril or by occluding both nostrils for a brief period to encourage deep breaths or sighs.

Abnormal upper airway sounds include: tracheal snaps which may be heard in tracheal collapse; stertor (inspiratory snoring) typical of pharyngeal or nasopharyngeal diseases, and stridor, an inspiratory wheeze over the larynx, that is typical of upper airway obstruction. Low-pitched inspiratory noise may also indicate upper airway obstruction.

Abnormal lower airway (adventitious) sounds include rhonchi, wheezes, and crackles. A sonorous rhonchus is an inspiratory or expiratory noise (r/o transmission of upper respiratory stertor), which suggests the presence of fluid or exudate in larger airways, as with bronchitis or pneumonia. Wheezes (sibilant rhonchi) are high-pitched expiratory sounds typical of bronchial narrowing. The usual associations are bronchial disease (bronchitis, asthma) or attenuation of a main bronchus caused by left atrial dilation, hilar lymphadenopathy, primary bronchial collapse, or a pulmonary mass lesion. Crackles ("rales") are discontinuous sounds similar to radio static or the sound of Velcro pulled apart. The explosive opening of collapsed small airways causes these sounds. Though there is a tendency to relate crackles to "fluid in the lungs," there is not a consistent correlation as crackles may be detected with pulmonary edema, pneumonia, bronchitis, or pulmonary fibrosis. The loudest crackles are typically detected in primary lung diseases. Subtle crackles are evident only after a deep breath.

REFERENCES

1. DK, Patterson DF: A phonograph record of heart sounds and murmurs of the dog. *Ann NY Acad Sci* 127: 22-340, 1965.
2. Detweiler DK, Patterson DF: Abnormal heart sounds and murmurs of the dog. *J Small Anim Pract* 8:193-205, 1967.
3. Smetzer DL, Hamlin RL, Smith CR Cardiovascular sounds. In *Dukes' Physiology of Domestic Animals, (Swenson MJ, ed).* Ithaca, Comstock Publishing Company, Inc, 1970, pp 159-168.
4. Ettinger S: Physical examination of the cardiovascular system. *Vet Clin North Am* 1:85-91, 1971.
5. Harpster NK: Clinical examination of the feline cardiovascular system. *Vet Clin North Am* 7:241-56, 1977.
6. Abbott J: Auscultation: what type of practice makes perfect? *J Vet Intern Med* 15:505-506, 2001.
7. Hamlin RL, Muir WW III, Gross DR, Pipers FS: Right and left ventricular systolic intervals during ventilation and sinus arrhythmia in the dog: genesis of physiologic splitting of the second heart sound. *Am J Vet Res* 35:9-13, 1974.
8. Beardow AW, Buchanan JW: Chronic mitral valve disease in cavalier King Charles spaniels: 95 cases. (1987-1991). *J Am Vet Med Assoc* 203:1023-11029, 1993.
9. Pedersen HD, Haggstrom J: Mitral valve prolapse in the dog: a model of mitral valve prolapse in man. *Cardiovasc Res* 47:234-243, 2000.
10. Constable PD, Hinchcliff KW, Olson J, Hamlin RL: Athletic heart syndrome in dogs competing in a long- distance sled race. *J Appl Physiol* 76:433-438, 1994.
11. Darke PG: Valvular incompetence in cavalier King Charles spaniels. *Vet Rec* 120:365-366, 1987.
12. Haggstrom J, Kvart C, Hansson K: Heart sounds and murmurs - changes related to severity of chronic valvular disease in the cavalier King Charles spaniel. *J Vet Internal Med* 9:75-85, 1995.
13. Tilley LP, Liu SK, Gilbertson SR, Wagner BM, Lord PF: Primary myocardial disease in the cat A model for human cardiomyopathy. *Am J Pathol* 86:493-522, 1977.
14. Kvart C, French AT, Fuentes VL, Haggstrom J, et al.: Analysis of murmur intensity, duration and frequency components in dogs with aortic stenosis. *J Small Anim Pract* 39:318-324, 1998.
15. Smetzer DL, Breznock EM: Auscultatory diagnosis of patent ductus arteriosus in the dog. *J Am Vet Med Assoc* 160:80-84, 1972.
16. Pedersen HD, Haggstrom J, Falk T, Mow T, et al.: Auscultation in mild mitral regurgitation in dogs: observer variation, effects of physical maneuvers, and agreement with color Doppler echocardiography and phonocardiography. *J Vet Intern Med* 13:56-64, 1999.
17. Hamlin RL: Physical examination of the pulmonary system. *Vet Clin North Am Small Anim Pract* 30:1175-85, 2000.