

Practical Thoracic Auscultation: Don't throw away your stethoscope

Examination Technique | Transient & Breath Sounds

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. For example, hearing the classic continuous machinery murmur of patent ductus arteriosus or the typical holosystolic left apical murmur of mitral regurgitation allows the clinician to express a “most-likely” diagnosis with high accuracy. Results of auscultation, combined with the signalment, findings from physical examination, and consideration of the clinical situation, often point to a logical cardiac diagnosis. This presumption can then be confirmed, refined, or refuted, as necessary, by Doppler echocardiography (for valvular disease, pericardial disease, cardiomyopathy, or shunts) or by electrocardiography (for arrhythmias).

Essential abnormalities evaluated by cardiac auscultation include: (a) abnormal heart rate or irregular rhythm of heart sounds (arrhythmia); (b) abnormal intensity of sounds (loud, soft, or variable); (c) extra sounds (gallops and clicks); (d) split sounds; (e) cardiac murmurs; and (f) pericardial friction rubs. Respiratory sounds should be classified as well.

Technique

One method of cardiac auscultation is demonstrated in the figures associated with this Practice Pointer article. Auscultation is preceded by palpation of the thoracic wall over the heart (**precordium**) in order to assess the **apical beat** (FIGURE 1). The prominent left apical impulse occurs with opening of the aortic valve (a systolic thrust). The impact is normally strongest at or near the left 5th intercostal space (ICS) near the costochondral junction. A weaker systolic impulse is normally palpable on the right hemithorax at approximately the right 3rd to 4th ICS (FIGURE 2). Dilation of the ventricle displaces the apex beat caudoventrally. Hypertrophy of the ventricle can produce a more prominent than normal impulse. Interpretation of these changes requires considerable experience and practice. A precordial vibration or **thrill** is often palpable over a **point of maximal intensity** of a loud cardiac murmur (also called a “thrilling murmur”).



FIGURE 3



FIGURE 1



FIGURE 2

Stethoscopes // Only some heart sounds fall within the audible frequency-threshold range; accordingly, careful auscultation is necessary to detect those vibrations that can be heard. This requires a stethoscope (FIGURE 3). This instrument should involve materials and construction of acceptable quality and suitable (not too long) length. The binaural orientation should be adjusted towards the nose. Both binaurals and ear pieces should align to the ear canal with the earpiece snugly but comfortably fitted to obtain an airtight seal.

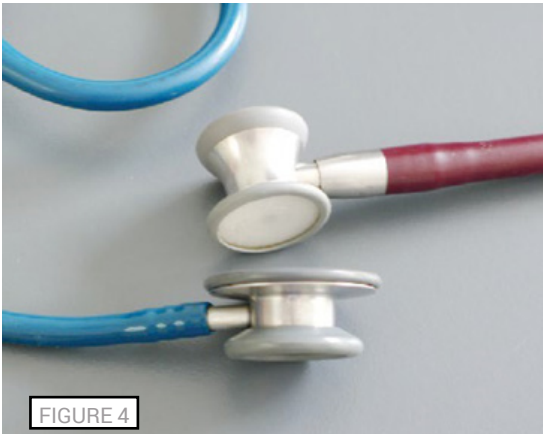


FIGURE 4

Stethoscope chestpieces serve different functions. The *traditional* diaphragm (FIGURE 4) is applied gently but firmly to accentuate higher-frequency sounds such as normal heart and breath sounds. The traditional bell (on the side of the chestpiece opposite the diaphragm) is applied lightly to achieve an airtight seal to enhance auscultation of lower-frequency sounds such as the third and fourth heart sounds as well as some diastolic murmurs. A combination adult- or pediatric-sized chestpiece (“tunable” diaphragm, FIGURE 5) looks like a traditional diaphragm but changes frequency response with varying pressure. Firm pressure on the chestpiece accentuates higher-pitched sounds such as normal heart sounds and most systolic murmurs. Conversely, gentle pressure brings out lower pitched sounds like gallops or diastolic murmurs. The smaller tunable diaphragm is often confused for a bell but is actually a pediatric chestpiece especially useful in cats and in small dogs.

The choice of a stethoscope model and vendor is personal. Again, with the traditional stethoscope there is a diaphragm and a bell, the side is selected by twisting the chestpiece. With a “tunable” chestpiece light pressure creates the “bell” effect, while heavier pressure flattens the chestpiece into a diaphragm. While acoustically superior in some ways, some stethoscope heads designed for use in people are too large for cats and smaller dogs. However, many models now incorporate both “adult” and “pediatric” tunable chestpiece on one rotating head. Some traditional stethoscopes allow the user to swap the adult for the pediatric-sized diaphragm. Lastly, there are various amplified stethoscopes. These are not recommended for

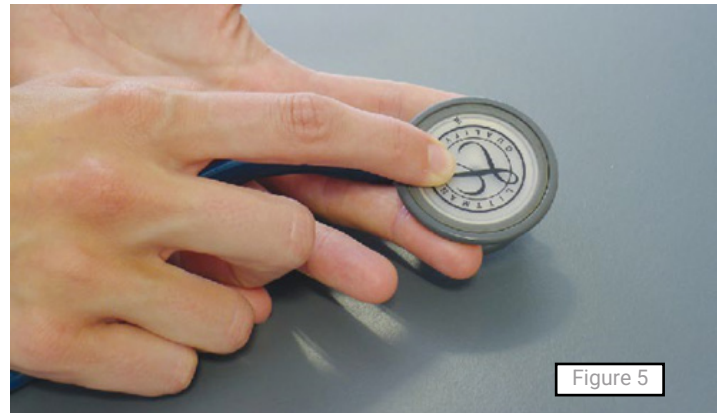


Figure 5

general use because artifacts and sound distortions can occur. However, these instruments can be useful for those hard of hearing or when recording/documenting sounds is important for medical records, distance diagnosis, or teaching. Additionally, the use of digital stethoscopes with artificial intelligence assisted analysis is increasing and include some commercially available systems (e.g., Eko Vet+™ Auscultation System).

► Examination Pointers

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. Minimizing ambient noise in the room is crucial. It is preferable for the dog to **stand** to better locate the relative valve areas. A cat can be restrained with one hand under the abdomen; this encourages the cat to rest on its forelimbs.

Learning to hold the stethoscope with either hand is useful, as is standing behind the patient to allow the arm to naturally curve. This approach is shown in FIGURES 6 – 11. The patient must be calm and ventilation and purring controlled if possible. Synchronous ventilation can mimic cardiac murmurs. Gently holding the mouth closed, whistling, or briefly obstructing the nares are maneuvers for reducing ventilation artifacts. Purring in cats might be reduced by holding the patient, gently touching the skin over the larynx, or turning on a faucet.



Figure 6

The clinician must be aware of **sound artifacts** that are often misinterpreted as abnormal heart or lung sounds. Artifacts include ventilation and panting (mimics murmurs); twitching (sounds like an extra heart sound); and friction from rubbing the chestpiece across hair (sounding like pulmonary crackles or rales). Excessive pressure on the chest should be avoided, as it can distort the thorax of small animals and create abnormal flow patterns and even murmurs.

Auscultation is usually preceded by palpation of the arterial pulse and palpation of the precordium to identify the apical impulse and any precordial vibrations or thrills that would indicate a loud heart murmur. Assessment of pulses is reviewed in another *Practice Pointer* on the Cardiovascular Examination.

Areas for Auscultation // For canine auscultation, the precordium (area over the heart) is examined with attention directed to the **cardiac valve areas**. While the exact anatomic location of the valve areas depends on the species, breed, 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 and other valve listening areas on the left. A useful clinical pointer in dogs is to first palpate the left apical impulse and place the stethoscope there (FIGURE 6) where mitral sounds radiate, and the first heart sound will be prominent. The **mitral valve area** is located over the apex and immediately dorsal to this point. Find other valve areas from the apex. The **aortic valve area** is located craniodorsally to the left apex and the second heart sound is usually loudest there (this change in position of the stethoscope may be subtle, FIGURE 7). Once the aortic second sound is identified, the stethoscope can be moved one intercostal space cranial and slightly ventral (over the **pulmonic valve area**) (FIGURE 8).

The **tricuspid valve area** is over the right hemithorax (FIGURE 9), cranial to the mitral area, and covers a relatively wide area.

The relative position of these valve areas is consistent from dog to dog; however, the exact intercostal space can vary depending on the breed and size of the heart.

The **pulmonary trunk** extends dorsally from the pulmonic valve and is located on the left craniodorsal thorax. The LV outlet is in the center of the heart and aortic murmurs often radiate well to each hemithorax. Left-cranial and dorsal placement of the stethoscope in dogs (FIGURE 10) or left/right-cranial placement along the sternum in cats, often reveals functional outflow tract murmurs (FIGURE 10).

The cardiac apex and the cardiac base are commonly used expressions to designate the regions ventral and dorsal to the atrioventricular groove. Atrioventricular valve sounds from the mitral and tricuspid valves often radiate ventrally towards the apices. Murmurs originating at the semilunar valves and great arteries are detected best over the base.

Feline Examination // Auscultation areas in cats usually not focused on the valve areas. The long-axis of the feline heart is oriented more "horizontally" (FIGURE 11), valve areas are less distinctive, and a chestpiece covers multiple valve areas. Consequently, auscultation sites are designated using terms like *apical* or *caudal* for the location near the palpable apex beat, and *cranial* when listening to areas rostral to the apex. In cats the apical impulse is often felt along both sternum edges (sternal borders). Generally the typical murmur of mitral regurgitation is loudest at the caudal sternal borders, whereas ejection and functional murmurs are often louder along cranial left or right sternal edges. However, these are rough guidelines at best, and loud murmurs are detectable across the precordium.



FIGURE 7



FIGURE 8



FIGURE 9

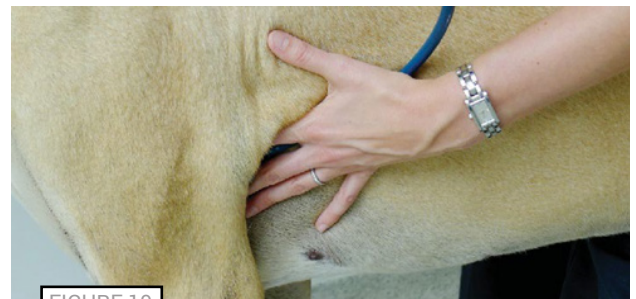


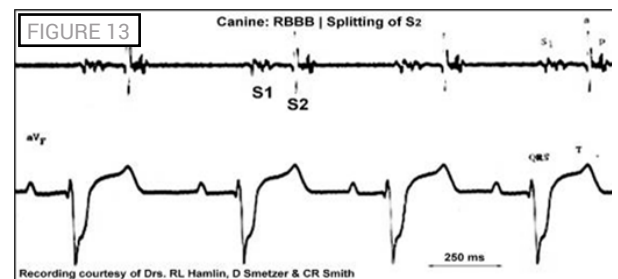
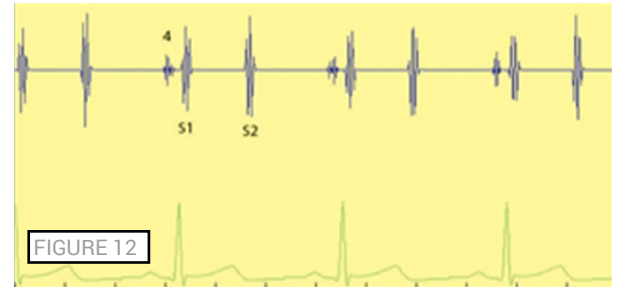
FIGURE 10



FIGURE 11

Transient Cardiovascular Sounds

Transient sounds are caused by vibrations of short duration; their genesis depends on abrupt changes in pressures and blood flow. Following electrical activation (QRS), ventricular pressures rise rapidly, closing the atrioventricular valves, and generating the **first heart sound (S1)** in early systole. The first sound coincides with the external thrust of the left apical impulse. At the end of ventricular ejection, pressures in the great arteries fall, and the semilunar valves close. The **second heart sound (S2)** is generated by the cardio-hemic vibrations surrounding these events (FIGURE 12). Accordingly, clinicians define **ventricular systole** as the period between S1 and S2. The femoral **arterial pulse** is palpated during systole. Other heart sounds and cardiac murmurs are timed relative to S1 and S2. The longer period between S2, and ensuing first sound, represents **ventricular diastole** to the clinician. Transient sounds occurring in diastole are most often third or fourth heart sounds (gallop sounds), and these are abnormal in small animals.



► First & Second Heart Sounds

Both S1 and S2 are high-frequency sounds. Their *timing* correlates to *closure of cardiac valves* (although sound generation is more complicated than just valve closure). The first sound occurs near the moment of atrioventricular (mitral, tricuspid) valve closure, while S2 is timed with closure of the aortic and pulmonic valves (FIGURE 12). These sounds can become abnormal in certain conditions. For example, pericardial and pleural *effusions* decrease the intensity of these sounds. This is also true with *myocardial failure* from dilated cardiomyopathy as the rate of ventricular pressure development is reduced. Conversely, both S1 and S2 are louder in healthy animals under high sympathetic drive and in those with thin body conformation. *Arrhythmias* often create variable-intensity heart sounds from beat-to-beat changes in ventricular filling and pressure. *Splitting* of the first or second sounds occurs with asynchronous ventricular activation as occurs with *ventricular ectopic beats*, *ventricular tachycardias*, and *bundle branch blocks* (FIGURE 13). This can be explained by the different onset or duration of left versus right ventricular ejection. Practically splitting often lead to less distinct or "muddled" sounds. *Severe pulmonary hypertension* augments the pulmonary component of S2 creating a loud, single second sound. This disorder can also cause splitting of aortic and pulmonary components of S2 by creating unsynchronized ventricular ejection times.

► Gallops

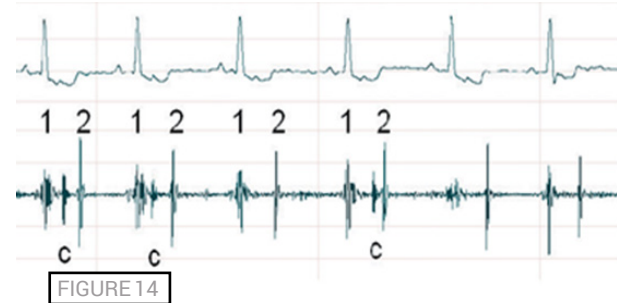
Diastolic sounds or gallops are abnormal in dogs and cats, representing audible manifestations of the third and fourth heart sounds. Gallops are lower-frequency and associated with vibrations surrounding either termination of early ventricular filling (S3) or atrial contraction that fills the ventricle at the end of diastole (S4). If both sounds are present and the heart rate is rapid, the gallops might superimpose, producing a single, combined third heart sound known as a *summation gallop*. Gallops in dogs and cats indicate *ventricular diastolic dysfunction*. Gallops can come and go, and often vary with changes in heart rate. These sounds can be explained (simplistically) as follows: the ventricle *sucks blood into its chamber* during early diastole, while the *atrium pumps blood into the ventricle* at end-diastole. Early diastolic filling is quantitatively most important, while atrial pumping contributes progressively more at higher heart rates or when early diastolic filling is impaired. The latter situation is common with normal aging and with myocardial diseases. It is quite challenging to determine the precise timing of a gallop sound, especially in cats. Nevertheless, there are different clinical associations to these sounds.

The **ventricular gallop (S3)** is associated with a stiff ventricle due to fibrosis, ventricular hypertrophy, or extreme chamber dilation from volume overloads (including mitral regurgitation). In these conditions, early ventricular filling requires higher venous pressures; as such, an S3 gallop (or a summation gallop) is often associated clinically with *overt congestive heart failure* and is frequently related to ventricular *volume overload* (valvular regurgitation, dilated cardiomyopathy, and fluid overloads).

An **atrial gallop (S4)** is associated with impaired early diastolic relaxation that demands ventricular filling shift from early to later in diastole. The S4 represents the *brief, compensatory increase in atrial pressure* needed to complete ventricular filling. Impaired early filling is observed in normal aging as well as with ventricular disease (e.g., feline hypertrophic cardiomyopathy). Thus, the clinical significance of an atrial gallop is more variable. For instance, some older cats without overt cardiomyopathy develop an S4 when hospital-stressed, likely related to age-related, slow ventricular relaxation and concurrent sympathetic activation. (Sympathetic tone increases heart rate, abbreviates diastole, and enhances atrial contraction.) Echocardiography in these cases is often unremarkable, although Doppler studies will reveal impaired ventricular relaxation. However, *gallop sounds are often the only auscultatory abnormality of cardiomyopathy, hypertensive heart disease, or congestive heart failure, so this finding should prompt other diagnostic evaluations.*

► Clicks

Clicks are brief, high-pitched transient sounds. Systolic clicks are common in dogs with mitral or tricuspid valve disease and likely indicate valvular prolapse from abnormal chordae tendineae or valve redundancy. Isolated clicks in older dogs are suggestive of mild myxomatous valve disease. Clicks also can be superimposed over murmurs in dogs with more advanced mitral valve or tricuspid valve degeneration. Although the classic mitral valve click is mid-systolic, these sounds can come and go and move from early to late systole (FIGURE 14).



Systolic clicks have also been reported in dogs and cats with mitral valve dysplasia (malformation). Another potential cause is feline hypertrophic cardiomyopathy, likely related to changes that develop in the mitral valve (in these cases, a systolic click must be distinguished from true diastolic gallop sound). An early systolic click is occasionally related to ventricular ejection. Examples include valvular pulmonic or aortic stenosis if the valve is relatively mobile, but leaflets fused. During ejection, the separation of the valve leaflets can be abruptly checked, and an audible click might be detected. Infrequently, ejection clicks are detected in dogs with severe pulmonary hypertension with associated dilation of the main pulmonary artery.

Arrhythmias // Auscultatory findings associated with cardiac arrhythmias and conduction disturbances include: (a) abnormal heart rate, (b) irregular cadence, (c) irregular intensity of the heart sounds, (d) extra or absent heart sounds, or (e) splitting of S1 or S2 (as discussed previously).

A common example is *atrial fibrillation* wherein a rapid, erratic ventricular rhythm develops in response to chaotic atrial activation and ineffective atrial contraction. Beat-to-beat changes in preload lead to varying intensity of heart sounds and an unpatterned heart rhythm often referred to as “tennis shoes in a dryer.” Other arrhythmias might lead to early beats (as with premature ventricular complexes), marked pauses (sinus arrest or atrioventricular blocks), or bradycardia (atrial standstill; complete AV block). Any of these findings indicate the need for an ECG for verification of the rhythm. Often only the first sound of a ventricular premature beat is heard (S1–S2–S1———S1–S2–S1) because the shortened diastole results in an ineffective ventricular beat. This prevents the aortic valve from opening and precludes development of a second heart sound (while causing a “pulse deficit” – more first heart sounds than arterial pulses). This triple-beat cadence can be easily confused with a true gallop rhythm (S1–S2–S3).

Arrhythmias in cats are often overlooked or the auscultation is confusing. For example, it is common in cats to hear the pause following a ventricular premature beat, as opposed to the premature heart sound itself. Another example is mistaking pathologic arrhythmias for sinus arrhythmia. While a cyclically-irregular rhythm is normal in dogs (usually indicating respiratory sinus arrhythmia), this finding is uncommon in cats examined in the hospital or clinical setting. Thus any irregularity of the heart rhythm in a cat can represent an indication for an ECG. A final feline example is that of complete (third-degree) AV block. Cats have more rapid escape activity than dogs – often leading to heart rates of 110 to 130/minute. This bradycardia for a cat can be readily overlooked, especially if the escape rhythm is regular or a precise heart rate is not obtained.

Thus, the *differential diagnosis of “extra sounds”* includes gallops (atrial & ventricular), systolic clicks, and other sounds. These include widely-split S2, and sounds associated with premature beats. A cardiac murmur is often considered a hallmark auscultatory finding of heart disease, however, many heart diseases are not associated with an audible murmur. However, transient sounds might be abnormal in these patients, and the clinician should be vigilant to detect these abnormal sounds.

RESPIRATORY (BREATH) SOUNDS

The clinician also uses the stethoscope to examine the airways, lungs and thoracic cavity. Auscultation over the larynx (gently!), mid-trachea, and at least 4 to 6 points over each thorax is recommended, depending in part on the medical history. Too often nonspecific terms such as “harsh” are used to describe what is heard; both normal and adventitious breath sounds have better descriptors and are discussed in the following sections.

► Normal Respiratory Sounds

Normal respiratory sounds include bronchial breathing (bronchovesicular sounds) as well as referred tracheal sounds (panting in dogs). *Bronchial sounds* have an inspiratory and expiratory component; inspiration is typically more prominent. Some normal dogs have slightly louder bronchial sounds over the right thorax. Normal breath sounds and abnormal (adventitious) sounds might only become evident after enforcing deeper breathing. This 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 a sigh.

Tracheal sounds have nearly equal inspiratory and expiratory components with a brief pause between the two phases. Panting is usually visually evident and tracheal sounds are audible without a stethoscope. This is a normal ventilation pattern in dogs activated mainly for thermoregulation. It can also be observed in stressed or hyperthermic animals, including highly-stressed cats. Often when a clinician notes “harsh lung sounds” in the record, the examiner is simply reporting normal tracheal sounds referred by major airways into the thorax. This finding is not diagnostic of any disorder (nor is the description “harsh” particularly useful). However, hearing loud tracheal sounds referred across all lung fields usually rules out large pleural effusions or pneumothorax.

► Abnormal Respiratory Sounds

Adventitious breath sounds are abnormal sounds superimposed on normal breath sounds during respiratory auscultation. These include alterations in normal breath sounds, loud sounds emanating from the upper airways, or abnormalities projected from the lungs or pleural space. It is wise to follow up abnormal findings with radiography, especially in symptomatic patients. Absent breath sounds with displaced heart sounds can be associated with a mass lesion, pleural fluid or air, or a diaphragmatic hernia.

Bronchial sounds can become accentuated, especially the expiratory phase, in various diseases of the lungs. These sounds can also be decreased or difficult to hear in the setting of pleural effusions or pneumothorax. An increase in bronchial sounds is often detected as a nonspecific finding of intrapulmonary disease, including pulmonary edema. Marked asymmetry of breath sounds can indicate disease that is localized to either the louder or the softer side of the chest.

Abnormal upper airway sounds include: (a) **stertor** or inspiratory snoring typical of obstructive pharyngeal or nasopharyngeal diseases (e.g., nasopharyngeal stenosis, redundant soft palate); (b) **stridor**, an inspiratory wheeze over the larynx and typical of major upper airway obstruction (e.g., laryngeal paresis or mass); and (c) **tracheal snaps** which may be heard in tracheal collapse. Additionally, prolonged, low- or high-pitched **inspiratory noise** might also indicate upper airway obstruction. Fixed obstructions prolong both inspiratory and expiratory phases of respiration.

Abnormal lower airway (adventitious) sounds include: 1) rhonchi, 2) wheezes, and 3) crackles. A **rhonchus** is an inspiratory or expiratory sound (somewhat similar to soft stertor or whistling; first rule out transmission of upper respiratory noise to the chest). Rhonchi suggest the presence of fluid or exudate in larger airways, and are often prominent at end-inspiration (as with bronchitis or pneumonia). A **wheeze** (previously called sibilant rhonchus) is a high-pitched expiratory sound typical of bronchial narrowing. The usual associations are bronchial disease (bronchitis, asthma) or attenuation of a major bronchus caused by left atrial dilation, hilar lymphadenopathy, primary bronchial collapse (from bronchomalacia), or a pulmonary mass lesion. Pulmonary **crackles** (previously called “rales”) are discontinuous sounds similar to radio static, crinkling cellophane, or Velcro being pulled apart. These sounds are thought to be caused by the explosive opening of collapsed small airways, and are often prominent at end-inspiration. Although there is a tendency to relate crackles to “fluid in the lungs,” there is not a consistent correlation, as crackles might be detected with pulmonary edema, pneumonia, bronchitis, or pulmonary fibrosis. The loudest crackles are typically detected in primary lung diseases, especially idiopathic pulmonary fibrosis. Subtle crackles are evident only after a deep breath and can be absent even if cardiogenic edema is present.

Although there are other abnormal breath sounds that may be identified during thoracic auscultation—including pleural friction rubs—the above sounds are most common.

SUMMARY

1. Effective cardiac auscultation requires knowledge of normal physiology, careful use of the stethoscope, and attention to other examination findings. Normal first and second sounds set the reference platform for auscultation. Abnormal transient sounds include loud or soft first/second sounds; clicks; split sounds; irregular and variable intensity sounds typical of arrhythmias; and extra sounds in diastole (gallops).
2. *Heart murmurs* are often heard in veterinary patients and these are the subject of the accompanying article on auscultation (Part 2).
3. *Breath* (respiratory) sounds correlate to the level of airway or pulmonary disease, and sometimes to relatively specific pathologies; these should be properly described.

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