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Physical Examination of the Cardiovascular System

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Cardiac Auscultation

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Heart sounds are produced by valve movements and the resultant vibrations in the heart, vascular walls, and blood columns. In the normal dog and cat, heart sounds that are typically audible include $S_1$ (“lub”) and $S_2$ (“dub”) (Fig 1). $S_1$, which is associated with closure of the atrioventricular (mitral and tricuspid) valves, is louder, longer, and of lower pitch than $S_2$, and is best heard over the valve regions (M and T) indicated in Figures 2 and 3. $S_1$ becomes louder with exercise, excitement, or anemia and varies with heart rate and PR interval. The intensity of $S_1$ also varies with atrial fibrillation, atrial or ventricular premature beats, or marked sinus arrhythmia. $S_2$ coincides with semilunar (aortic and pulmonic) valve closure and is best heard over the valve locations (A and P) indicated in Figure 2. The intensity of $S_2$ may increase with pulmonic stenosis, heartworm disease, hyperthyroidism, or pulmonary or systemic hypertension. The intensity is decreased in cardiogenic shock. In normal dogs and cats, $S_3$ (produced by vibrations associated with rapid ventricular filling) and $S_4$ (produced by atrial systole) are not audible (Fig 1). These sounds, termed cardiac gallops, are however, detectable in certain disease states.

Abnormal heart sounds offer the veterinarian an indication of the presence of cardiac disease, as well as specific information as to the underlying diagnosis. In some, but not all, instances, abnormal heart sounds give some indication of the severity of the underlying lesion. Careful auscultation, along with information obtained from the remainder of the physical examination, history, and ancillary diagnostic procedures, provides the means by which a specific diagnosis of cardiac disease is made. It should be kept in mind that, in some instances (e.g., anemia), abnormal cardiac sounds do not represent organic heart disease.

Ideally, auscultation of the heart should be performed in a quiet room, with a standing, cooperative patient. Usually the more dramatic heart sounds are evaluated after listening to the more subtle respiratory sounds. A stethoscope is placed over each heart valve (Fig 2) in turn, as well as over the carotid arteries and other areas of interest. The stethoscope’s diaphragm is pressed firmly to the thorax to maximize high frequency sounds, while the bell portion is lightly applied, maximizing low frequency sounds such as cardiac gallops.

Auscultation is just one portion of the cardiac examination. Palpation of the femoral pulse provides additional information as to pulse quality and strength and, hence, blood pressure and tissue perfusion. Simultaneous auscultation and pulse palpation aids in the detection of pulse variability and deficits, indicating arrhythmias. Information on tissue perfusion, presence of cyanosis, the state of hydration, and the status of the erythron can be obtained by examination of the mucous membranes. The finding of peripheral venous distension or the presence of ascites is compatible with right heart failure. Auscultation of the respiratory tract may reveal
adventitious lung sounds compatible with pulmonary edema of left heart failure or concurrent respiratory disease. Thoracic malformation, cardiac hyperkinesis and enlargement, precordial thrill (palpable murmur), or abnormal cardiac position may be revealed by thoracic palpation, while thoracic percussion is employed to detect pleural effusion.

**Causes, Pathophysiology and Clinical Signs**

Abnormal heart sounds are divided into several categories: murmurs, abnormal splitting of heart sounds, gallops (audible S₃ or S₄), clicks, friction rubs, audible arrhythmias, and muffled heart sounds. Of these, only cardiac gallops and friction rubs are consistently associated with heart disease.

**Murmurs.** Heart murmurs (Fig 1) result when turbulence is produced within the heart or vessels by abnormalities in size of vascular or valve orifices, abnormal communications, and alterations in blood velocity and viscosity. The intensity of a murmur is not a consistently reliable indication of the severity of the causative lesion. Specific causes include valvular incompetence or stenosis, abnormal vascular or cardiac chamber communications, anemia, fever, and anxiety.

Murmurs, which may be innocent (associated with alterations in blood flow, but not with organic cardiac disease) or pathologic (associated with cardiac disease), are classified by their location, duration, timing within the cardiac cycle, intensity, and character. Murmurs may be systolic (occurring during systole-between S₁ and S₂) or diastolic (occurring during diastole-between S₂ and S₁), and are described as continuous or holo- (or pan-), early, mid-, and late systolic or diastolic (Fig 1). The intensity of heart murmurs is graded on a scale of I to VI as described by Ettinger (Table 1). The pitch of the murmur, described as high, low, or mixed frequency, may help to identify the underlying disorder. An important diagnostic clue, especially in the instance of congenital heart disease, is the further characterization of murmurs according to their quality. Regurgitant murmurs are rectangular or plateau-shaped, and ejection murmurs are diamond-shaped or crescendo-decrescendo in nature (Fig 1). A cardiologist’s description of a murmur might read: “a grade V, holosystolic, mixed frequency, regurgitant murmur, most audible over the fifth left intercostal space (mitral valve region).”

Systolic regurgitant murmurs are associated with mitral and tricuspid insufficiency and ventricular septal defects (Figs 1, 4 and 5). Systolic ejection murmurs suggest pulmonic or aortic stenosis or atrial septal defect (Fig 4). Continuous murmurs are produced by arteriovenous connections (patent ductus arteriosus or arteriovenous fistulae; Figs 1 and 4). Diastolic murmurs, which are uncommonly encountered, are associated with semilunar valvular insufficiency or atrioventricular valvular stenosis (Fig 1). Non-continuous, systolic-diastolic murmurs are most often associated with vegetative endocarditis.

Innocent murmurs, characterized by low intensity (lower than grade III) and short duration, are relatively common in young dogs (younger than 6-12 months), and must be distinguished from the murmurs of congenital heart disease. These innocent or functional murmurs, due to turbulent flow in the great vessels during ventricular infection, often change in character or disappear when the patient’s position is altered. Such murmurs usually do not persist into adulthood. Likewise, the murmurs of anemia (packed-cell volume less than 20%), fever, anxiety, or pregnancy are not associated with structural cardiac disease and have been termed **physiologic murmurs**.

**Split Heart Sounds.** Either the first (S₁) or second (S₂) heart sound may be split, producing two nearly simultaneous sounds, similar in quality to the single sound from which
they arose (“lub-lub dub”) or “lub dub-dub” for split S1 or S2, respectively). Splitting of the first heart sound is produced by asynchronous closure of the atrioventricular valves. While it is considered to be normal in large breed dogs, it may be associated with ventricular conduction disturbances, ventricular pacing, or arrhythmias (Fig 1). Spitting of the second heart sound occurs when the semilunar valves close asynchronously (Fig 1). Normally, the aortic valve closes slightly before the pulmonic valve. Splitting of S2 is most frequently associated with delayed closure of the pulmonic valve, as is seen in pulmonary hypertension (usually due to heartworm disease), pulmonic stenosis, or right bundle branch block, or in normal dogs, during inspiration. It is occasionally observed with early closure of the aortic valve in states of diminished left ventricular output. Paradoxical splitting of S2 results when significant prolongation of left ventricular conduction and/or ejection time (left bundle branch block, subaortic stenosis, systemic hypertension, or severe left ventricular hypertrophy) delays closure of the aortic valve until after pulmonic valve closure.

Cardiac Gallops. Cardiac gallops or gallop rhythms, consisting of a series of sounds reminiscent of a galloping horse, are composed of S1, S2, and S3 or S4 (“lub dub thud”); the extra sound is of low frequency and may be difficult to hear. Cardiac gallops occur when S3 (protodiastolic gallop), S4 (presystolic gallop), or a combination of S3 and S4 (summation gallop) is abnormally accentuated (Fig 1). S3 gallops are produced when blood rushes into an incompletely emptied ventricle during rapid ventricular filling (e.g., in mitral insufficiency or dilated cardiomyopathy), while S4 gallops result when a noncompliant ventricle (e.g., hypertrophic cardiomyopathy) receives blood during atrial systole. Cardiac gallops are considered to be pathologic in dogs and cats, and are often associated with a poor prognosis.

Systolic Clicks. Systolic clicks are abnormal, usually midsystolic, high frequency sounds associated with mitral valve prolapse in humans. They are thought to be a preregurgitant phenomenon in dogs or are variably associated with systolic murmurs of mitral regurgitation (Fig 1).

Sounds Associated with Pericardial Disease. Friction rubs, associated with pericarditis and rarely heard in small animals, are “scratchy” biphasic or triphasic sounds audible during portions of both systole and diastole. They occur when inflamed and roughened pericardial and epicardial surfaces contact each other.

A pericardial bump, variable in its number of components and intensity, accompanies early diastolic ventricular filling in the presence of restrictive pericarditis. It occurs when the rapidly filling ventricles are suddenly restricted by the limiting pericardium.

Muffled Heart Sounds. Attenuation of muffling of normal heart sounds may be an important indicator of thoracic disease. Disorders associated with muffled heart sounds include pericardial and pleural effusion, diaphragmatic hernias, thoracic neoplasia, obesity, and hypothyroidism.

Audible Arrhythmias. Certain arrhythmias, such as sinus arrhythmia and atrial fibrillation, are virtually diagnostic upon cardiac auscultation when the femoral pulse is palpated concurrently. Sinus arrhythmia is “regularly irregular” without pulse deficits, and changes in rate are usually associated with respiration. Atrial fibrillation is characterized as being “irregularly irregular,” with marked variability in the intensity of S1, variable pulse strength, and frequent pulse deficits. Unifocal ventricular tachycardia is typically regular, and the pulses, although often weak, are palpable and without deficits. Supraventricular (atrial or junctional) tachycardia tends to be very rapid, but is difficult to distinguish from sinus and ventricular tachycardia without electrocardiographic evaluation. Abrupt cessation of a tachyarrhythmia with
administration of a vagal maneuver is diagnostic of supraventricular tachycardia. Isolated supraventricular and ventricular ectopic beats produce early, abnormal heart sounds (often only \(S_1\) is heard), followed by a pause and typically weak or non-existent pulse. With sinus bradycardia or second- and third-degree heart block, the rate is slow and there is no pulse deficit. Sounds of atrial systole may occasionally be appreciated between the sounds of ventricular systole in high grade AV block. In the case of first- and second-degree atrioventricular block, \(S_4\) (atrial systole) can occasionally be heard.

**Diagnostic Approach and Management**

The diagnostic approach to the finding of cardiac auscultatory abnormalities varies with the abnormality, the accompanying clinical picture, and the client’s wishes. The finding of a supposedly innocent murmur in a healthy pup requires no more than a follow-up examination at the next vaccination appointment. Conversely, the finding of a systolic-diastolic murmur in a dog with fever, joint pain, and dyspnea may require a complete cardiologic and medical workup, blood cultures, and hospitalization.

If the abnormal heart sound(s) cannot be accurately characterized, its exact character can often be determined by obtaining a phonocardiogram (Fig 1). This procedure, although not routinely available in private practice, may be necessary to determine the exact timing or character of a murmur and the type of gallop, or in distinguishing, for example, whether a subtle sound is a gallop, split sound, or systolic click. Phonocardiography is particularly useful in small animal practice where such factors as uncooperative patients, rapid heart rates, and panting or purring decrease the sensitivity and accuracy of cardiac auscultation. Electronic digital stethoscopes, which allow the murmur to be recorded and replayed at half speed, may also employed in cases in which the character of a murmur or exact nature of an abnormal heart sound is difficult to accurately assess.

Once abnormal sounds have been identified, the goal is to determine the presence, severity, and exact nature of underlying cardiac disease. This is accomplished by performing the following procedures, when indicated: thoracic radiography, electrocardiography, extended electrocardiographic (Holter) monitoring, echocardiography with or without Doppler studies, blood gas analysis, and cardiac catheterization (selective or nonselective) with oximetry, pressure measurement, and angiography. The indications for such procedures and the expected results are explored in greater detail in subsequent chapters.

**Patient Monitoring**

Depending on the diagnosis, the patient’s condition, and the prognosis, a follow-up schedule is established. This timing is variable, but may require no more frequent visits than the yearly vaccination appointment. Patients with more severe afflictions (those with impending heart failure, heart failure, or potentially life-threatening arrhythmias) obviously require more frequent reevaluation. It should be emphasized that if the nature of an abnormal heart sound or the resultant diagnosis is unclear, referral to a specialist with the expertise and specialized equipment to effect a more in-depth examination and evaluation is advisable.
Table 1. Grading Heart Murmurs, adapted from Ettinger

<table>
<thead>
<tr>
<th>Grade</th>
<th>Diagnostic Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>I</td>
<td>Murmur barely audible, with minimal radiation</td>
</tr>
<tr>
<td>II</td>
<td>Murmur very soft, but definitely audible, radiates more widely than I</td>
</tr>
<tr>
<td>III</td>
<td>Murmur easily audible but low in intensity, radiates relatively widely</td>
</tr>
<tr>
<td>IV</td>
<td>Murmur moderately intense without precordial thrill, radiates widely</td>
</tr>
<tr>
<td>V</td>
<td>Murmur is loud and associated with precordial thrill, heard over entire thorax</td>
</tr>
<tr>
<td>VI</td>
<td>Murmur very loud and audible, audible with stethoscope removed from thorax</td>
</tr>
</tbody>
</table>
Figure 1
Figure 3
Figure 4
General Inspection of the Cardiorespiratory System

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Inspection of the mucous membranes may reveal cyanosis (a dusky, bluish tint), indicating the presence of >5 g/di of desaturated hemoglobin in the blood. Cyanosis is characterized as central (caused by low arterial oxygen with norm saturation and PaO₂), peripheral (low local tissue PO₂ with normal PaO₂), or differential (cranial mucous membranes pink/caudal cyanotic). Pale mucous membranes (pallor) may indicate anemia or peripheral vasoconstriction from any cause (e.g., volume contraction or heart failure). Like cyanosis, pallor must be interpreted in light of the history and other physical findings. Teeth and gums should be examined as potential sources of infection. Noncardiac abnormalities causing inspiratory dyspnea (e.g., tonsilar tumors obstructing the pharynx, laryngeal edema, etc.) can often be noted during the oral exam. Specific attention should be given to distention of the jugular veins, and the jugular venous pulse should be described completely if visible. The presence of distended jugular veins may be an important sign of pericardial and/or right heart diseases, which are often difficult to diagnose. The hepatojugular reflex can sometimes be elicited by pushing gently on the liver and observing for distention of the jugular vein. Such distention indicates a failing right ventricle that is incapable of pumping out an increased volume of venous return. The general inspection is completed with an examination of the eyes (including the fundus), skin, and lymph nodes.

Palpation of the precordial cardiac impulse often yields much useful information. The location and nature of the impulse may provide information about cardiac enlargement (dilatation or hypertrophy). Loud murmurs may also produce precordial thrills (palpable vibrations on the chest wall at the point of maximal intensity of the murmur), which are useful in localizing the source of the murmur. The cardiac rhythm can be assessed as well as the compressibility of the thorax (cats).

The arterial pulse should be evaluated bilaterally, usually at the femoral artery, for assessment of cardiac rate and rhythm. Remember that the arterial pulse pressure is the difference between the systolic and diastolic arterial pressure. By carefully palpating literally hundreds of normal arterial pulses in both dogs and cats, you will form a foundation of experience by which to judge the peripheral arterial pulses of clinical patients. The pulse quality varies with body condition, species, age, heart rate, hydration, level of excitement and/or activity. Rectal palpation (a routine part of the physical examination in dogs) should include palpation of the iliac arterial pulses (also palpable in normal cats with appropriate sedation). A pulse deficit (heart sound {Sl} occurring without an associated peripheral arterial pulse) usually indicates the presence of some tachyarrhythmia, which should be further defined electrocardiographically. Hyperkinetic pulses (stronger than normal) can be caused by any condition that either augments the stroke volume of the left ventricle (i.e., volume loads the left ventricle), or decreases the diastolic pressure in the arterial system, or both. Common conditions associated with hyperkinetic pulses include fever, hyperthyroidism, aortic regurgitation, and patent ductus arteriosus. Hypokinetic pulses (weaker than normal) are often associated with dehydration (low intravascular volume), shock, heart failure, or aortic stenosis. Variable
intensity pulses are often a sign of arrhythmia (i.e., atrial fibrillation). A special case of variable intensity pulses is associated with pericardial disease and elevated intrapericardial pressures (called pulsus paradoxus, where the peripheral arterial pulse pressure decreases palpably (by more than 10-15%) with inspiration).

**Physical Examination of the Respiratory System**

Thoracic auscultation should be performed as a "mentally separate" event from auscultation of the heart, since you can't concentrate on both things at once. Lung sounds are often difficult diagnostic signs to interpret. Abnormal (called adventitious) lung sounds were first described by Laennec in the 19th century, when he classified them as "rales," adding such modifiers as crepitant, sonorous, and sibilant to further characterize them. For a time, the term "ronchus" was used as a euphemism for rales, because rale was thought to have excessively morbid connotations for bedside use. Adjectives proliferated to further describe these sounds, and you may still hear clinicians talk about a bewildering variety of sounds they have perceived following pulmonary auscultation (including wet, dry, bubbling, consonating, and sticky rales). Unfortunately, none of these terms is associated with a consistent pathophysiologic correlate, and they confuse both physicians and veterinarians alike. Many of these terms are frankly misleading, such as wet rales, which may be produced by a dry, fibrotic lung. In the 13 years since the American Thoracic Society recategorized lung sounds, this confusion has not completely been eliminated (but it's getting better). In general, there are three types of sounds made in the lungs and airways:

1. **Normal breath sounds** (sometimes called vesicular or broncho-vesicular sounds) are soft, rustling sounds produced by turbulent airflow in the large airways and carried down the bronchial tree to the chest wall on inspiration. Conditions that restrict the velocity of airflow into the lung (pleural effusion, pneumothorax, diaphragmatic paralysis, obesity) will make these sounds softer. Conditions that increase the amount of water in the interstitial lung tissue (e.g., pulmonary edema), or increase the velocity of airflow in the large airways (partial upper airway obstruction) make the sounds louder.

2. **Crackles** are brief (transient), non-musical, literally crackling sound, caused by explosive equalization of gas pressure between two previously separated compartments of the lung, usually in the small airways or alveoli. Crackles may be produced in a variety of pathological conditions, including pulmonary edema, pneumonia, interstitial fibrosis, bronchitis, and asthma. The crackles caused by these diseases may vary in timing and intensity, but the actual quality of the sound is identical no matter what the underlying disease. The presence crackles should be interpreted in light of the rest of the history and physical examination, and prompt further investigation to determine the underlying cause of the lung pathology.

3. **Wheezes** are musical sounds usually associated with asthma or chronic bronchitis (although they can occur in pulmonary edema or fibrosis as well). Wheezes are heard less often than crackles in veterinary medicine (probably because of the relatively low incidence of overt reactive airway disease, at least in dogs). The presence of wheezes should also prompt further investigation of their cause.

Percussion is used to elicit areas of dullness (hyperresonance, caused for example by areas of lung consolidation or pleural effusion) or hyperresonance (as might occur with pneumothorax or feline asthma). Percussion has an undeserved reputation as an impractical or
inaccurate diagnostic test. The sound produced when a body surface is struck is termed a percussion note. These notes vary, producing a scale of resonance that ranges from flat notes (relatively high frequency notes of short duration and little or no resonance) to tympanic notes (low frequency notes of long duration and exceptional resonance). Some examples of the underlying tissues that produce the most easily discernable of these notes follow, arranged from the most to the least resonant:

- **tympanic** - the sound produced by percussing an air-filled hollow viscus.
- hyperresonant - the sound produced by percussing pneumothorax or hyperinflated lungs (e.g. feline asthma)
- **resonant** - the sound produced by percussing over normal lungs.
- hyporresonant - the sound produced by percussing over lungs with increased fluid density (e.g. pulmonary edema)
- **dull** - the sound produced by percussing over a significant pleural effusion
- **flat** - the sound produced by percussion of the gluteal muscles

With practice on normal animals of varying haircoats, breeds, body conditions, and confirmations, you will begin to build a mental library of normal percussion notes that you expect to hear, and it will become easy to recognize inappropriately hyper- or hyporresonant notes associated with pulmonary or pleural pathology. By combining the information available from auscultation of the heart and lungs with percussion of the thorax, the clinician's diagnostic acumen rises dramatically.