Contents

Video Contents

PART ONE ▪ Cardiovascular System Disorders

Wendy A. Ware and Jessica L. Ward

CHAPTER 1

Clinical Manifestations of Cardiac Disease

SIGNS OF HEART DISEASE

Several signs can indicate the presence of heart disease, even if the animal is not clinically in "heart failure." So-called objective signs of heart disease are, for the most part, cardiac specific. These are cardiac murmurs, rhythm disturbances, jugular pulsations, and cardiac enlargement. Notable exceptions to this generalization include murmurs that are functional (nonpathologic) in nature and the normal rhythm irregularity of sinus arrhythmia. Other clinical signs may indicate heart disease but can occur with noncardiac diseases as well. These include syncope, excessively weak or strong arterial pulses, cough or respiratory difficulty, exercise intolerance, abdominal distention, and cyanosis. Further evaluation using thoracic radiography, cardiac biomarker tests, echocardiography, electrocardiography (ECG), and sometimes other tests usually is indicated when signs consistent with cardiovascular (CV) disease are present.

SIGNS OF HEART FAILURE

Heart failure generally is considered to occur when the heart cannot adequately meet the body's circulatory needs or can do so only with high filling (venous) pressures. Not all animals with heart disease will develop heart failure. Of those that do, the majority show clinical signs (Box 1.1) related to high venous pressure behind one or both ventricles (congestive signs), and some also manifest signs of inadequate blood ejection from the heart (low output signs). Congestive signs associated with right-sided heart failure stem from systemic venous hypertension and the resulting increase in systemic capillary hydrostatic pressure. High left-heart filling pressure causes pulmonary venous engorgement and edema. Signs of biventricular failure develop in some animals. Chronic left-sided congestive heart failure (CHF) can promote the development of right-sided congestive signs when pulmonary venous hypertension markedly raises pulmonary arterial pressure. Signs of low cardiac output are similar regardless of which ventricle is affected, because output from the left heart is coupled to that from the right heart. Heart failure is discussed further in Chapter 3 and within the context of specific diseases.

WEAKNESS AND EXERCISE INTOLERANCE

Animals with heart failure often cannot adequately raise cardiac output to sustain increased levels of activity. Furthermore, vascular and metabolic changes that occur over time impair skeletal muscle perfusion during exercise and contribute to reduced exercise tolerance. Increased pulmonary vascular pressure and edema also lead to poor exercise ability. Episodes of exertional weakness or collapse can relate to these changes or to an acute decrease in cardiac output caused by arrhythmias (Box 1.2).

SYNCOPE

Syncope is characterized by transient unconsciousness, with loss of postural tone (collapse), from insufficient oxygen or glucose delivery to the brain. Various cardiac and noncardiac abnormalities can cause syncope and intermittent weakness (see Box 1.2). Syncope can be confused with seizure episodes. A careful description of the animal's behavior or activity before the collapse event, during the event itself, and following the collapse, as well as a drug history, can help the clinician differentiate among syncopal attacks, episodic weakness, and true seizures. Syncope often is associated with exertion or excitement. The actual event can include rear limb weakness or sudden collapse, lateral recumbency, stiffening of the forelimbs with opisthotonos, and micturition (Fig. 1.1). Vocalization is common; however tonic/clonic motion, facial fits, and defecation are not. An aura (which often occurs before seizure activity), postictal dementia, and neurologic deficits are not expected in dogs and cats with CV syncope. Sometimes profound hypotension or asystole causes hypoxic "convulsive syncope," with seizure-like

BOX 1.1

Clinical Signs of Heart Failure

Congestive Signs—Left (↑ Left Heart Filling Pressure)

Pulmonary venous congestion
Pulmonary edema (causes tachypnea, ↑ respiratory effort, cough, orthopnea, pulmonary crackles, tiring, cyanosis, hemoptysis)
Postcapillary pulmonary hypertension
Secondary right-sided heart failure
Cardiac arrhythmias

Congestive Signs—Right (↑ Right Heart Filling Pressure)

Systemic venous congestion (causes ↑ central venous pressure, jugular vein distention)
Hepatic ± splenic congestion
Pleural effusion (causes ↑ respiratory effort, orthopnea, cyanosis)
Ascites
Small pericardial effusion
Subcutaneous edema
Cardiac arrhythmias

Low Cardiac Output Signs

Tiring
Exertional weakness
Syncope
Prerenal azotemia
Cyanosis (from poor peripheral circulation)
Cardiac arrhythmias

FIG 1.1
Syncope in a Doberman Pinscher with paroxysmal ventricular tachycardia. Note the extended head and neck with stiffened forelimbs. Involuntary micturition also occurred, followed shortly by return of consciousness and normal activity.

activity or twitching; however, these convulsive syncopal episodes are preceded by loss of muscle tone. Presyncope, where reduced brain perfusion (or substrate delivery) is not severe enough to cause unconsciousness, may appear as transient "wobbliness" or weakness, especially in the rear limbs.

BOX 1.2

Causes of Syncope or Intermittent Weakness

Cardiovascular Causes

Bradyarrhythmias (second- or third-degree AV block, sinus arrest, sick sinus syndrome, atrial standstill)
Tachyarrhythmias (paroxysmal atrial or ventricular tachycardia, reentrant supraventricular tachycardia, atrial fibrillation)
Congenital ventricular outflow obstruction (pulmonic stenosis, subaortic stenosis)
Acquired ventricular outflow obstruction (heartworm disease and other causes of pulmonary hypertension, hypertrophic obstructive cardiomyopathy, intracardiac tumor, thrombus)
Cyanotic heart disease (tetralogy of Fallot, pulmonary hypertension, and "reversed" shunt)
Impaired forward cardiac output (severe valvular insufficiency, dilated cardiomyopathy, myocardial infarction or inflammation)
Impaired cardiac filling (e.g., cardiac tamponade, constrictive pericarditis, hypertrophic or restrictive cardiomyopathy, intracardiac tumor, thrombus)
Cardiovascular drugs (diuretics, vasodilators)
Neurocardiogenic reflexes (vasovagal, cough-syncope, other situational syncope)

Pulmonary Causes

Diseases causing hypoxemia
Pulmonary hypertension
Pulmonary thromboembolism

Metabolic and Hematologic Causes

Hypoglycemia
Hypoadrenocorticism
Electrolyte imbalance (especially potassium, calcium)
Anemia
Sudden hemorrhage

Neurologic Causes

Cerebrovascular accident
Brain tumor
(Seizures)

Neuromuscular Disease

(Narcolepsy, cataplexy)

AV, Atrioventricular.

Tests to explore the cause of intermittent weakness or syncope usually include ECG recordings (during rest, exercise, and/or after exercise or a vagal maneuver), complete blood count (CBC), serum biochemical analysis (including electrolytes and glucose), heartworm testing, neurologic examination, thoracic radiographs, and echocardiography. Other studies for neuromuscular or neurologic disease also may be valuable. Intermittent cardiac arrhythmias that are not apparent on resting ECG may be uncovered by ambulatory ECG monitoring, using a 24-hour Holter, event, or

implantable loop recording device. In-hospital continuous ECG monitoring for a period of time sometimes reveals a culprit arrhythmia also.

Cardiovascular Causes of Syncope

Various arrhythmias, obstruction to ventricular outflow, cyanotic congenital heart defects, and acquired diseases that impair cardiac output are the usual causes of CV syncope. Activation of vasodepressor reflexes and excessive dosages of CV drugs also can induce syncope. Arrhythmias that provoke syncope usually are associated with either very fast or very slow heart rates and can occur with or without identifiable underlying organic heart disease. Ventricular outflow obstruction can provoke syncope or sudden weakness if cardiac output becomes inadequate during exercise or if high systolic pressure activates ventricular mechanoreceptors, causing inappropriate reflex bradycardia and hypotension. Both dilated cardiomyopathy and severe mitral insufficiency can impair forward cardiac output, especially during exertion. Vasodilator and diuretic drugs can induce syncope if given in excess.

Syncope caused by abnormal peripheral vascular and/or neurologic reflex responses is not well defined in animals, but is thought to occur in some patients. Syncope associated with sudden bradycardia after a burst of sinus tachycardia has been documented, especially in small breed dogs with advanced atrioventricular (AV) valve disease; excitement often precipitates such an episode. Doberman Pinschers and Boxers similarly may experience syncope after sudden bradycardia. Postural hypotension and hypersensitivity of carotid sinus receptors infrequently can provoke syncope by inappropriate peripheral vasodilation and bradycardia.

Fainting associated with a coughing fit (cough syncope or "cough-drop") occurs in some dogs with marked left atrial (LA) enlargement and bronchial compression, as well as in dogs with primary respiratory disease. Several mechanisms have been proposed, including an acute decrease in cardiac filling and output during the cough, peripheral vasodilation after the cough, and increased cerebrospinal fluid pressure with intracranial venous compression. Severe pulmonary disease, anemia, certain metabolic abnormalities, and primary neurologic diseases also can cause collapse that resembles CV syncope.

COUGH AND OTHER RESPIRATORY SIGNS

CHF in dogs produces tachypnea, respiratory distress, and sometimes coughing. These signs also can occur with the pulmonary vascular pathology and pneumonitis of heartworm disease in both dogs and cats. Noncardiac conditions, including diseases of the upper and lower airways, pulmonary parenchyma (including noncardiogenic pulmonary edema), pulmonary vasculature, and pleural space, as well as certain nonrespiratory conditions, also should be considered in patients with cough, tachypnea, or dyspnea (see Chapter 19).

The cough associated with cardiogenic pulmonary edema in dogs often is soft and moist; it sometimes sounds like gagging. Cats, in contrast, rarely cough from pulmonary edema. Tachypnea progressing to dyspnea occurs in both species. Pleural and pericardial effusions occasionally are associated with coughing as well. Mainstem bronchus collapse or compression associated with severe LA enlargement can stimulate a dry or hacking cough in dogs with chronic mitral valve disease, even when pulmonary edema or congestion is absent. Concurrent bronchomalacia is likely to be a contributing factor in these cases. A heart base tumor, enlarged hilar lymph nodes, or other masses that impinge on an airway also can stimulate this type of cough.

When respiratory signs have a cardiac cause, other evidence of heart disease usually is evident, such as generalized cardiomegaly, LA enlargement, pulmonary venous congestion, lung infiltrates that resolve with diuretic therapy, or a positive heartworm test. Findings on physical examination, thoracic radiographs, cardiac biomarker assays, echocardiogram, and sometimes an ECG, help in differentiating cardiac from noncardiac causes.

CARDIOVASCULAR EXAMINATION

The medical history (Box 1.3) is an important part of the CV evaluation and can help guide the choice of diagnostic tests because it may suggest various cardiac or noncardiac diseases. The patient's signalment is useful because some

BOX 1.3

Important Historic Information

Signalment (age, breed, gender)?
Vaccination status?
What is the diet? Have there been any recent changes in food or water consumption?
Where was the animal obtained?
Is the pet housed indoors or outdoors?
How much time is spent outdoors? Supervised?
What activity level is normal? Does the animal tire easily now?
Has there been any coughing? When? Describe episodes.
Has there been any excessive or unexpected panting or heavy breathing?
Has there been any vomiting or gagging? Diarrhea?
Have there been any recent changes in urinary habits?
Have there been any episodes of fainting or weakness?
Do the tongue/mucous membranes always look pink, especially during exercise?
Have there been any recent changes in attitude or activity level?
Are medications being given for this problem? What? How much? How often? Do they help?
Have medications been used in the past for this problem? What? How much? Were they effective?

congenital and acquired abnormalities are more prevalent in certain breeds or life stages, or because specific findings are common in individuals of a given breed (such as a soft left basilar ejection murmur in normal Greyhounds and other sighthounds).

Physical evaluation of the patient with suspected heart disease includes observation (for example, of attitude, posture, body condition, level of anxiety, respiratory pattern) and a general physical examination. The CV examination itself consists of evaluating the peripheral circulation (mucous membranes), systemic veins (especially the jugular veins), systemic arterial pulses (usually the femoral arteries), and the precordium (left and right chest wall over the heart), as well as auscultation of the heart and lungs, and palpating or percussing for abnormal fluid accumulation (e.g., ascites, subcutaneous edema, pleural effusion). Proficiency in all aspects of the CV examination requires practice but is important for accurate patient assessment and monitoring.

RESPIRATORY PATTERNS

Respiratory difficulty (dyspnea) usually causes the animal to appear anxious. Increased respiratory effort, flared nostrils, and often a rapid rate of breathing are evident (Fig. 1.2). Increased depth of respiration (hyperpnea) can result from hypoxemia, hypercarbia, or acidosis. Pulmonary edema (or other pulmonary infiltrates) increases lung stiffness; the rapid and shallow breathing (tachypnea) that results helps minimize the work of breathing. In the absence of primary lung disease, an increase in resting respiratory rate often is an early indicator of pulmonary edema. Lung stiffness also increases with pleural fluid or air accumulation and can produce tachypnea, too. However, with large-volume pleural effusion or pneumothorax, respiratory motions become increasingly labored and exaggerated as the animal struggles to expand the collapsed lungs; the respiratory rate often is not elevated in these cases.

It is important to note whether the respiratory difficulty is more intense during a particular phase of respiration. Prolonged, labored inspiration usually is associated with upper airway obstructive disorders, whereas prolonged expiration occurs with lower airway obstruction as well as pulmonary infiltrative disease (including edema). Animals with severely compromised ventilation may refuse to lie down; rather, they stand or sit with elbows abducted to allow maximal rib expansion, and they resist being positioned in lateral or dorsal recumbency (orthopnea). Cats with dyspnea often crouch in a sternal position with elbows abducted. Open-mouth breathing usually is a sign of severe respiratory distress in cats (Fig. 1.3). The increased respiratory rate associated with excitement, fever, fear, or pain generally can be differentiated from dyspnea by careful observation and physical examination.

MUCOUS MEMBRANES

Mucous membrane color and capillary refill time (CRT) are used to evaluate peripheral perfusion. The oral mucosa usually is assessed, although caudal mucous membranes (prepuce or vagina) also can be evaluated. The CRT is determined by applying digital pressure to blanch the membrane; color should return within 2 seconds. Slower refill times occur as a result of dehydration and other causes of decreased cardiac output because of high peripheral sympathetic tone and vasoconstriction. Pale mucous membranes occur with either anemia or peripheral vasoconstriction. The CRT is normal in anemic animals unless hypoperfusion also is present. However, the CRT can be difficult to assess in severely anemic animals because of the lack of color contrast. In animals with polycythemia (erythrocytosis) or exercise-induced rear limb weakness, the color of the caudal membranes should be compared with that of the oral membranes for evidence of differential cyanosis (see p. 115 in Chapter 5). If oral membranes are heavily pigmented, the ocular

FIG 1.2
Dyspnea in an older male Golden Retriever with dilated cardiomyopathy and fulminant pulmonary edema. The dog appeared highly anxious, with rapid labored respirations and hypersalivation. Respiratory arrest occurred within minutes of this photograph being taken, but the dog was resuscitated.

FIG 1.3
Severe dyspnea is manifested in this cat by open-mouth breathing, infrequent swallowing (drooling saliva), and reluctance to lie down. Note also the dilated pupils associated with heightened sympathetic tone.

BOX 1.4

Abnormal Mucous Membrane Color

Pale Mucous Membranes

Anemia
Poor cardiac output/high sympathetic tone

Injected, Brick-Red Membranes

Polycythemia (erythrocytosis)
Sepsis
Excitement
Other causes of peripheral vasodilation

Cyanotic Mucous Membranes*

Pulmonary parenchymal disease
Airway obstruction
Pleural space disease
Pulmonary edema
Right-to-left shunting congenital cardiac defect
Hypoventilation
Shock
Cold exposure
Methemoglobinemia

Differential Cyanosis

Reversed patent ductus arteriosus (head and forelimbs receive normally oxygenated blood, but caudal part of body receives desaturated blood via the ductus, which arises from the descending aorta)

Icteric Mucous Membranes

Hemolysis
Hepatobiliary disease
Biliary obstruction

*Anemic animals might not appear cyanotic even with marked hypoxemia because 5 g/dL of desaturated hemoglobin is necessary for visible cyanosis.

conjunctiva can be evaluated. Box 1.4 outlines causes for abnormal mucous membrane color. Petechiae in the mucous membranes might be evident in animals with platelet disorders (see Chapter 87). In addition, oral and ocular membranes often are areas where icterus (jaundice) is first detected; a yellowish cast to these membranes should prompt further evaluation for hemolysis (see Chapter 82) or hepatobiliary disease (see Chapter 33).

JUGULAR VEINS

Systemic venous and right heart filling pressures are reflected at the jugular veins. These veins should not be distended when the animal is standing with its head in a normal position (jaw parallel to the floor). Persistent jugular vein distention occurs in patients with right-sided CHF (because of high right heart filling pressure), external compression of the cranial vena cava (as from a cranial mediastinal mass), and jugular vein or cranial vena cava thrombosis (Fig. 1.4).

Jugular pulsations that extend higher than one third of the way up the neck from the thoracic inlet also are

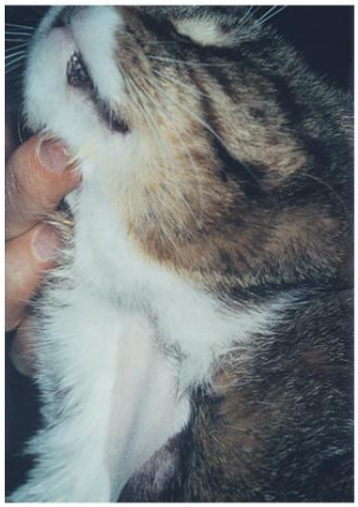

FIG 1.4
Prominent jugular vein distention in a cat with signs of right-sided congestive heart failure from dilated cardiomyopathy.

abnormal. Sometimes the carotid pulse wave is transmitted through adjacent soft tissues, mimicking a jugular pulse in thin or excited animals. To differentiate a true jugular pulse from carotid transmission, lightly occlude the jugular vein below the area of visible pulsation. If the pulse disappears, it is a true jugular pulsation; if the pulse continues, it is being transmitted from the carotid artery. Jugular pulse waves are related to atrial contraction and filling. Visible pulsations occur in animals with tricuspid insufficiency (after the first heart sound, during ventricular contraction), conditions causing a stiff and hypertrophied right ventricle (just before the first heart sound, during atrial contraction), or arrhythmias that cause the atria to contract against closed AV valves (so-called cannon "a" waves). Specific causes of jugular vein distention and pulsations are listed in Box 1.5. Impaired right ventricular (RV) filling, reduced pulmonary blood flow, or tricuspid regurgitation can cause a positive *hepatojugular* (*abdominojugular*) *reflux* test even in the absence of jugular distention or pulsations at rest. To test for this reflux, apply firm pressure to the cranial abdomen while the animal stands quietly with head and neck in normal position. This transiently increases venous return. Jugular distention that persists while abdominal pressure is applied constitutes a positive (abnormal) test. Normal animals have little to no change in the jugular vein with this maneuver.

ARTERIAL PULSES

The strength and regularity of the peripheral arterial pressure waves and the pulse rate are assessed by palpating the femoral or other peripheral arteries (Box 1.6). Subjective evaluation of pulse strength is based on the difference between the systolic and diastolic arterial pressures (that is,

BOX 1.5

Causes of Jugular Vein Distention/Pulsation

Distention Alone

Pericardial effusion/tamponade
Right atrial mass/inflow obstruction
Dilated cardiomyopathy
Cranial mediastinal mass
Jugular vein/cranial vena cava thrombosis

Pulsation ± Distention

Tricuspid regurgitation of any cause (degenerative, cardiomyopathy, congenital, secondary to diseases causing right ventricular pressure overload)
Pulmonic stenosis
Heartworm disease
Pulmonary hypertension
Ventricular premature contractions
Complete (third-degree) heart block
Constrictive pericarditis
Hypervolemia

BOX 1.6

Abnormal Arterial Pulses

Weak Pulses

Dilated cardiomyopathy
(Sub) aortic stenosis
Pulmonic stenosis
Shock
Dehydration

Strong Pulses

Excitement
Hyperthyroidism
Fever
Hypertrophic cardiomyopathy

Very Strong, Bounding Pulses

Patent ductus arteriosus
Fever/sepsis
Severe aortic regurgitation

the pulse pressure). When the difference is wide, the pulse feels strong on palpation; abnormally strong pulses are termed *hyperkinetic.* When the pressure difference is small, the pulse feels weak *(hypokinetic).* If the rise to maximum systolic arterial pressure is prolonged, as with severe subaortic stenosis, the pulse also tends to feel weak *(pulsus parvus et tardus).* Both femoral pulses should be palpated and compared; absence of pulse or a weaker pulse on one side could be caused by thromboembolic disease. Femoral pulses can be difficult to palpate in cats, even when normal. Often an elusive pulse can be found by gently working a fingertip between the dorsomedial thigh muscles toward the femur, in the area of the femoral triangle, where the femoral artery enters the leg.

The femoral arterial pulse rate should be evaluated simultaneously with the direct heart rate, which is obtained by auscultation or chest wall palpation. Fewer femoral pulses than heartbeats constitute a pulse deficit. Various cardiac arrhythmias induce pulse deficits by causing the heart to beat before adequate ventricular filling has occurred. Consequently, minimal or even no blood is ejected for those beats, and a palpable pulse is absent. Other arterial pulse variations also occur occasionally. Alternately weak then strong pulsations can result from severe myocardial failure *(pulsus alternans)* or from a normal heartbeat alternating with a premature beat *(bigeminy),* which causes reduced ventricular filling and ejection. An exaggerated decrease in systolic arterial pressure during inspiration occurs from cardiac tamponade *(pulsus paradoxus)*; a weak arterial pulse strength may be detectable during inspiration in those patients.

PRECORDIUM

The term "precordium" refers to the area of the chest wall that overlies the heart on both sides of the thorax. To palpate the precordium, place the palm and fingers of each hand on the corresponding side of the animal's chest wall over the heart (e.g., right hand over the right precordial area and left hand over the left precordial area). Normally the strongest systolic impulse is felt over the area of the left cardiac apex (located at approximately the fifth intercostal space near the costochondral junction). Cardiomegaly or a space-occupying mass within the chest can shift the precordial impulse to an abnormal location. Decreased intensity of the precordial impulse can be caused by obesity, weak cardiac contractions, pericardial effusion, intrathoracic masses, pleural effusion, or pneumothorax. The precordial impulse should be stronger on the left chest wall than on the right. A stronger right precordial impulse can result from RV hypertrophy or displacement of the heart into the right hemithorax by a mass lesion, lung atelectasis, or chest deformity. Very loud cardiac murmurs cause palpable vibrations on the chest wall known as a *precordial thrill.* This feels like a "buzzing" sensation to the hand. A precordial thrill usually is localized to the area of maximum murmur intensity.

EVALUATION FOR FLUID ACCUMULATION

Right-sided CHF promotes abnormal fluid accumulation within body cavities (Fig. 1.5; see also Fig. 9.3) and occasionally in the subcutis of dependent areas. Palpation and ballottement of the abdomen, percussion of the chest in the standing animal, and palpation of dependent areas are used to detect effusions and subcutaneous edema. Fluid accumulation secondary to right-sided heart failure usually is accompanied by abnormal jugular vein distention with or without pulsations, unless the animal's circulating blood volume is diminished from diuretic administration or other cause. Hepatomegaly and splenomegaly also may be noted in cats and dogs with right-sided CHF.

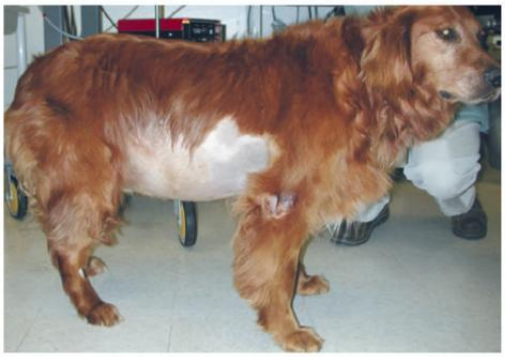

FIG 1.5
Abdominal distention caused by ascites from right heart failure in a 7-year-old Golden Retriever.

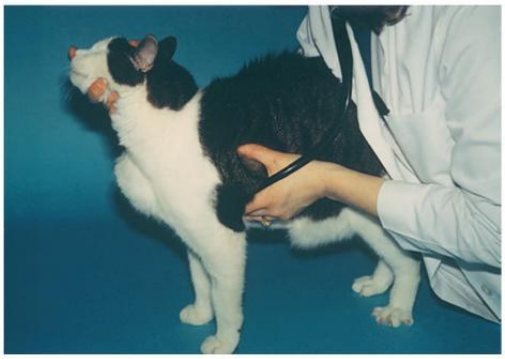

FIG 1.6
During cardiac auscultation, respiratory noise and purring often can be decreased or eliminated by gently placing a finger over one or both nostrils for brief periods of time.

AUSCULTATION

Thoracic auscultation is used to assess heart rate and rhythm, identify normal heart sounds, determine the presence or absence of abnormal sounds, and evaluate pulmonary sounds. Heart sounds are created by turbulent blood flow and associated vibrations in adjacent tissue during the cardiac cycle. Although many of these sounds are too low in frequency or intensity to be audible, others can be heard with the stethoscope or even palpated. Heart sounds are classified as transient sounds (those of short duration) and cardiac murmurs (longer sounds occurring during a normally silent part of the cardiac cycle). Cardiac murmurs and transient sounds are described by their timing within the cardiac cycle and by general characteristics of sound: frequency (pitch), amplitude of vibrations (intensity/loudness), duration, and quality (timbre). Sound quality is affected by the physical characteristics of the vibrating structures.

Because many heart sounds can be difficult to hear, a cooperative animal and a quiet room are important during auscultation. The animal should be standing, if possible, so that the heart is in its normal position. Panting in dogs is discouraged by holding the animal's mouth shut. Respiratory noise can be decreased further by placing a finger over one or both nostrils for a short time. Purring in cats often can be stopped by briefly holding a finger over one or both nostrils (Fig. 1.6), gently pressing the cricothyroid ligament region with a fingertip, waving an alcohol-soaked cotton ball near the cat's nose, or turning on a water faucet near the animal. Various other artifacts can interfere with auscultation, including respiratory clicks, air movement sounds, shivering, muscle twitching, hair rubbing against the stethoscope, gastrointestinal sounds, and extraneous room noises.

The traditional stethoscope has both a stiff, flat diaphragm and a bell on the chestpiece. The diaphragm, when applied firmly to the chest wall, allows better auscultation of higher-frequency heart sounds than those of low frequency. The bell, applied lightly to the chest wall, facilitates auscultation of lower-frequency sounds such as S_3 and S_4 (see the section on Gallop Sounds). Stethoscopes with a single-sided chestpiece are designed to function as a diaphragm when used with firm pressure against the skin and as a bell when used with light pressure. Ideally the stethoscope should have short double tubing and comfortable eartips. The binaural eartubes should be angled rostrally to align with the examiner's ear canals (Fig. 1.7).

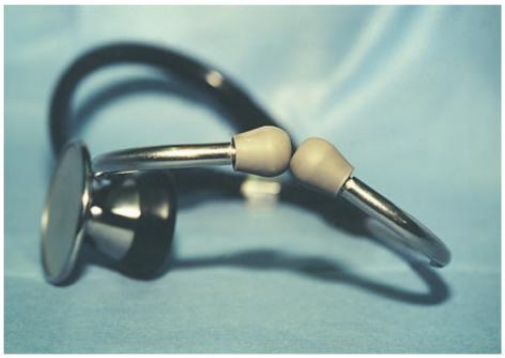

FIG 1.7
Note the angulation of the stethoscope binaurals for optimal alignment with the clinician's ear canals (top of picture is rostral). The flat diaphragm of the chestpiece faces left, and the concave bell faces right.

Both sides of the chest should be carefully auscultated, with special attention to the valve areas (Fig. 1.8). The stethoscope is moved gradually to all areas of the chest. The examiner should concentrate on the various heart sounds, correlating them to the events of the cardiac cycle, and listen for any abnormal sounds in systole and diastole successively. The normal heart sounds (S_1 and S_2) are used as a framework for timing abnormal sounds. The point of maximal intensity (PMI) of any abnormal sounds should be located. The examiner should focus on cardiac auscultation separately from

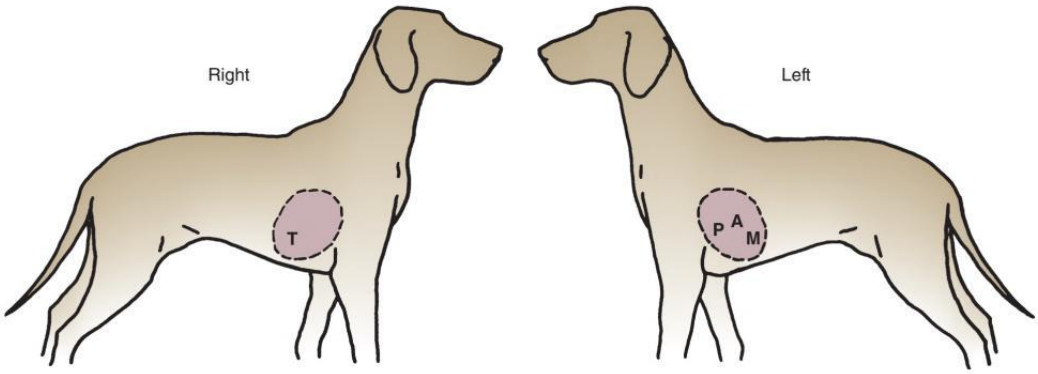

FIG 1.8
Approximate locations of various valve areas on the chest wall. *T,* Tricuspid; *P,* pulmonic; *A,* aortic; *M,* mitral.

pulmonary auscultation because full assimilation of sounds from both systems simultaneously is unlikely. Pulmonary auscultation is described further in Chapter 20.

Transient Heart Sounds

The heart sounds heard normally in dogs and cats are S_1 (associated with closure and tensing of the AV valves and associated structures at the onset of systole) and S_2 (associated with closure of the aortic and pulmonic valves following ejection). The diastolic sounds (S_3 and S_4) are not audible in normal dogs and cats. Fig. 1.9 correlates the hemodynamic events of the cardiac cycle with the ECG and timing of the heart sounds. It is important to understand these events and identify the timing (from a clinical perspective) of systole (between S_1 and S_2) and diastole (after S_2 until the next S_1) in the animal. The precordial impulse occurs just after S_1 (systole), and the arterial pulse occurs between S_1 and S_2.

Sometimes the first (S_1) and second (S_2) heart sounds are altered in intensity. The normal heart sounds may be louder in dogs and cats with a thin chest wall, high sympathetic tone, tachycardia, or systemic arterial hypertension. Shortened PR intervals increase the intensity of S_1. Muffled sounds can result from obesity, pericardial effusion, diaphragmatic hernia, dilated cardiomyopathy, hypovolemia/poor ventricular filling, or pleural effusion. A split or sloppy-sounding S_1 may be normal, especially in large dogs, or it may result from ventricular premature contractions or an intraventricular conduction delay. The intensity of S_2 can be increased by pulmonary hypertension of any cause (see Chapter 10). Cardiac arrhythmias often cause variation in the intensity (or even absence) of heart sounds.

Normal physiologic splitting of S_2 occasionally is heard in some (larger) dogs because of variation in stroke volume during the respiratory cycle. During inspiration, increased venous return to the right ventricle tends to delay closure of the pulmonic valve, whereas reduced filling of the left

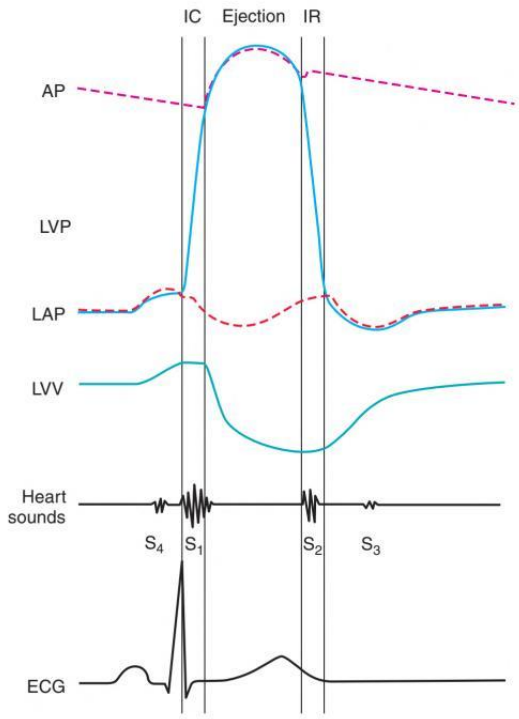

FIG 1.9
Cardiac cycle diagram depicting relationships among great vessel, ventricular and atrial pressures, ventricular volume, heart sounds, and electrical activation. *AP,* Aortic pressure; *ECG,* electrocardiogram; *IC,* isovolumic contraction; *IR,* isovolumic relaxation; *LAP,* left atrial pressure; *LVP,* left ventricular pressure; *LVV,* left ventricular volume.

ventricle accelerates aortic closure. Pathologic splitting of S_2 can result from delayed ventricular activation or prolonged RV ejection secondary to ventricular premature beats, right bundle branch block, a ventricular or atrial septal defect, or pulmonary hypertension.

Gallop Sounds

The third (S_3) and fourth (S_4) heart sounds occur during diastole (see Fig. 1.9) and are not normally audible in dogs and cats. When an S_3 or S_4 sound is heard, the heart can sound like a galloping horse, hence the term *gallop rhythm*. This term can be confusing because the presence or absence of an audible S_3 or S_4 has nothing to do with the heart's rhythm (that is, the origin of cardiac electrical activation and the intracardiac conduction process). Gallop sounds usually are heard best with the bell of the stethoscope (or by light pressure applied to a single-sided chestpiece) because they are of lower frequency than S_1 and S_2. At very fast heart rates, differentiation of S_3 from S_4 may be impossible. If both sounds are present, they may be superimposed, which is called a *summation gallop.*

The S_3 gallop, also known as an *S_3 gallop* or *ventricular gallop,* is associated with low-frequency vibrations at the end of the rapid ventricular filling phase. An audible S_3 in the dog or cat usually indicates ventricular dilation with myocardial failure. The extra sound often is very subtle although sometimes it can be fairly loud and easily detected; it is heard best over the cardiac apex. This sound may be the only auscultable abnormality in an animal with dilated cardiomyopathy. An S_3 gallop also may be audible in dogs with advanced mitral valve disease and CHF.

The S_4 gallop, also called an *atrial* or *presystolic gallop,* is associated with low-frequency vibrations triggered by blood flow into the ventricles during atrial contraction (just after the P wave of the ECG). An audible S_4 in the dog or cat usually is associated with increased ventricular stiffness and hypertrophy, such as with hypertrophic cardiomyopathy or hyperthyroidism in cats. A transient S_4 gallop of unclear significance sometimes is heard in stressed or anemic cats.

Other Transient Sounds

Other brief abnormal sounds are audible in some cases. Systolic clicks are mid-to-late systolic sounds that usually are heard best over the mitral valve area. These sounds have been associated with degenerative valvular disease (endocardiosis), mitral valve prolapse, and congenital mitral dysplasia; a concurrent mitral insufficiency murmur can be present. In dogs with degenerative valvular disease, a mitral click might be the first abnormal sound noted, with a murmur developing over time. An early systolic, high-pitched ejection sound at the left base can occur in animals with valvular pulmonic stenosis or other diseases that cause dilation of a great artery. The sound is thought to arise from either the sudden checking of a fused pulmonic valve or the rapid filling of a dilated vessel during ejection. Rarely, constrictive pericardial disease causes an audible pericardial knock. This diastolic sound arises from sudden checking of ventricular filling by the constrictive pericardium; its timing is similar to the S_3.

Cardiac Murmurs

There are many causes for cardiac murmurs. Most involve a structural cardiac abnormality and are considered pathologic murmurs. However, some murmurs do not and are considered nonpathologic. Nonpathologic murmurs are systolic in timing. They can occur for physiologic reasons, for example, when blood viscosity is reduced by anemia, or when cardiac output is increased from fever, hyperthyroidism, etc.; these are known as *functional* murmurs. Sometimes a soft murmur is heard in an animal without evidence for structural cardiac disease or physiologic alteration. These are considered *innocent* murmurs and often are found in young puppies. Many animals with a pathologic murmur also have other clinical signs consistent with heart disease. However, pathologic as well as nonpathologic murmurs often are discovered as incidental findings on physical examination. In these cases, it is important to determine if a clinically important cardiac disease or physiologic abnormality is the cause or not. Careful physical examination and auscultation can help the clinician decide how aggressively (or whether) to immediately pursue additional diagnostic testing.

Cardiac murmurs are described by their timing within the cardiac cycle (systolic or diastolic, or portions thereof), intensity, PMI on the precordium, radiation over the chest wall, quality, and pitch. Systolic murmurs can occur in early (protosystolic), middle (mesosystolic), or late (telesystolic) systole or throughout systole (holosystolic). Diastolic murmurs generally occur in early diastole (protodiastolic) or throughout diastole (holodiastolic). Murmurs at the end of diastole are termed *presystolic.* Continuous murmurs begin in systole and extend through S_2 into all or part of diastole. Murmur intensity generally is graded on a scale of 1 to 6 (sometimes written I to VI) (Table 1.1). The PMI usually is indicated by the hemithorax (right or left) and valve area or intercostal space where it is located, or by the terms *apex* or *base.* Because murmurs can radiate extensively, the entire thorax, thoracic inlet, and carotid artery areas should be auscultated. The pitch and quality of a murmur relate to its frequency and subjective assessment. "Noisy" or "harsh" murmurs contain mixed frequencies. "Musical" murmurs are of essentially one frequency with its overtones; these can sound like a "whoop" or "honk."

Murmurs also can be described by their phonocardiographic configuration (Fig. 1.10). A plateau-shaped or "regurgitant" murmur begins at the time of S_1 and remains of fairly uniform intensity throughout systole. Sometimes this murmur configuration also is called *holosystolic*, because it generally is consistent throughout systole. Loud regurgitant/holosystolic murmurs can mask the S_1 and S_2 sounds. AV valve insufficiency and interventricular septal defects commonly cause this type of murmur because turbulent blood flow begins at the time of AV valve closing and continues throughout ventricular systole. A crescendo-decrescendo or diamond-shaped murmur starts softly, builds intensity in